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Released: 01/21/2025
Valid until: 01/21/2026
Time needed to complete: 60 minutes

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Right Heart Catheterization: Mastery of Precision Techniques

Announcer:
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This replay of a live broadcast discusses strategies for enhanced precision, efficiency, and safety during right heart catheterization procedures.

Dr. McLaughlin:
Good evening, and welcome to this right heart catheterization masterclass webinar. We're so excited that you joined us tonight. I'm Val McLaughlin. I direct the Pulmonary Hypertension Program at the University of Michigan, and I'm joined tonight by my dear friend, Rich.

Would you like to introduce yourself, Rich?

Dr. Krasuski:
Hey, I'm Rich Krasuski. I'm from Duke University. I run the Adult Congenital Heart Disease Program, and I do heart catheterization. So I do a lot of interventional work in the cath lab as well.

Dr. McLaughlin:
And Rich will offer a really practical perspective. He spends a ton of time in the right heart cath lab. So let's go on with it.

I want to show you our disclosures.

So we are here tonight to talk about pulmonary hypertension, and we're going to focus on right heart catheterization, but let's talk a little bit about how we get there. Obviously, the symptoms of pulmonary hypertension are very nonspecific: exertional dyspnea, fatigue, sometimes lightheadedness or chest pain. And the differential for that is quite broad. And usually we start to think about pulmonary hypertension based on an echocardiogram. That usually is what gets patients in our doors. And so it's important to remember that while the echo can give an estimated PA pressure, which we calculate from the tricuspid regurgitant jet, there are other echo signs of pulmonary hypertension that need to be considered: the size and function of the right ventricle, the motion of the intraventricular septum, notching, a lot of different signs that reflect the severity of the pulmonary hypertension. And so we need to integrate that TR velocity and the other echo signs to end up with a probability of pulmonary hypertension.

And if we think that there's a high probability of pulmonary hypertension, we move to a right heart catheterization, which is what we're here to discuss tonight. If we think the probability is really low, then we look at alternative diagnosis. And of course, if it's more intermediate, sometimes we just follow those patients with an echocardiogram.

So the right heart catheterization is really required for the diagnosis of pulmonary arterial hypertension. It's generally done as an outpatient procedure, but it should be done in experienced hands. Of course, we will talk about how to float the catheter in more detail, and Dr. Krasuski will give some very important tips and tricks for that. But there are a lot of hemodynamic measurements that need to be made along the way, generally with a very specific Swan-Ganz catheter that was designed to help navigate the way through an enlarged right heart in patients with pulmonary hypertension.

So the right heart catheterization is required to confirm the echo findings when we suspect pulmonary hypertension. As good as the echo is at looking for left heart disease, it's really critical to measure the wedge pressure, and it's really critical to calculate the cardiac output, and we'll spend some time talking about that today, because we need that to calculate the pulmonary vascular resistance. I think one of the worst experiences I have with my patients is when they have a cath at an outside institution before they get to me, and there's a pulmonary artery pressure and a right atrial pressure, and there's no wedge, or there's no cardiac output. We need all of that information together. It's important to screen for shunts. And Dr. Krasuski will talk about that in some detail.

The right heart catheterization is also important in terms of the severity and prognosis, and some patients may need an acute vasodilator challenge, which, of course, we'll talk about as well.

So this is the updated hemodynamic definition of pulmonary hypertension. This was changed a little bit at the 6th World Symposium, and then a little bit more at the ESC/ERS guidelines, and then that was accepted at the 7th World Symposium and further changes were not made. So any type of pulmonary hypertension is a mean pulmonary artery pressure over 20. To be considered precapillary pulmonary hypertension, one has to have a mean pulmonary artery pressure over 20 and a wedge pressure of less than or equal to 15 and a calculated pulmonary vascular resistance of greater than 2 Wood units. So patients who have Group 1 pulmonary arterial hypertension fall into that category, but sometimes patients with Groups 3 or 4 or 5 fall into that category as well. Isolated postcapillary pulmonary hypertension, so that's Group 2 left heart disease, has a high mean pulmonary pressure but also a high wedge pressure, and the transpulmonary gradient and the pulmonary vascular resistance are normal. And then the very challenging combined pre- and postcapillary pulmonary hypertension is that the wedge pressure is high. There's something wrong on the left side of the heart, but the pulmonary artery pressure is a lot higher than you'd expect for that; the transpulmonary gradient and the pulmonary vascular resistance are elevated.

And then exercise PH was reintroduced at the ESC/ERS guidelines, but I love this. They didn't like just pick a number, say it's exercise mean PA greater than 35; they really emphasize the physiology. So they described it as the slope of the mean pulmonary artery pressure over cardiac output between rest and exercise being more than 3 mmHg/L/min. But it's important to recognize that even though this is back in the definition, there's still a lot of limitations with exercise. Hemodynamics, probably one being is that there's no uniform way that people do it. There's not a uniform protocol. And another thing that I think is really important is that the most common cause of exercise pulmonary hypertension is actually diastolic heart failure, the wedge pressure going up with exercise. So we can probably talk about that a little bit more at some point, if we have time.

We do right heart catheterizations all the time. We don't think that it's a particularly risky procedure. Certainly, there are some rare contraindications. So if there's a mechanical tricuspid or pulmonary valve or if there's a thrombus or tumor in the right side of the heart or there's tricuspid or pulmonic valve endocarditis, you really shouldn't do a right heart cath in that situation. And then there are some relative contraindications, coagulopathies, recent pacemakers, infections, certainly concern for ventricular arrhythmias or heart blocks, so we have to keep that in mind.

But in general, the procedure is relatively safe. This was a study that was done, gosh, almost two decades ago now that Marius Hoeper led, and he just collected up data from right heart caths at big centers. So I think that's an important point to make. This is right hearth caths done in centers of experience, operators like Rich doing these procedures, and the complication rate was quite low, 1.1% complication risk. And most of the complications were access related, arterial puncture or bleeding or pneumothorax, and then some of them were PA catheter related, PA rupture, pulmonary infarction, arrhythmias, and perforation and pacemaker lead displacement. But the serious complications were really quite rare.

So, Rich, with that a little bit as background, maybe you can walk our viewers through your top tips to get through a right heart catheterization.

Dr. Krasuski:
Sure, Val. So I wouldn't necessarily say this is a benign procedure, but I would say that it's a safe procedure, as you've mentioned. The things that can go wrong, I often say, are usually related to sedation that I've seen. I think this is a procedure that you don't need the patient very sedated. There's not much uncomfortable about doing a right heart catheterization, and if you over-sedate, the numbers that you get aren't always all that accurate. So I do tend to emphasize the low risk of this particular procedure, but not that it's no risk.

You want to think about where you're going to get access from. And there's a lot that goes into this decision. Certainly, if I'm doing simply a right heart catheterization, neck is perfectly fine. But if I'm going to plan on doing something where I want to get arterial access as well, let's say it's the first catheterization, I'm probably going to go femoral. And as I've learned at Duke, they do a lot of brachial caths too. So you can do brachial and radial alternatively. But I do think it's nice to be able to have one access site where you're not looking at multiple sites and worrying about complications.

Generous local anesthesia, very, very important. I really numb it up really well, and usually you can get away without doing much in the way of conscious sedation when you do that. I want to collect all the equipment that I need before so that we're not kind of hunting around. If I'm anticipating it's going to be a challenging procedure and I'm going to need additional equipment, I want to have that available so I can just tell my tech to go grab whatever it is that I've left on the counter for them, rather than having to go out of the room and look in other places. The Swandom, if you're going to leave the Swan in, has to go on the catheter before it's floated. I think we've all had that situation where we've forgotten about that and then have to go back and refloat the Swan. You really don't want to do that, particularly if it's challenging cases.

And then a real simple rule is always advance the catheter with the balloon up. You should not be advancing it without the balloon up. And withdraw it with the balloon down, because if you pull it back without deflating the balloon, you can damage valves and other structures, so you want to be very careful. And similar, you can cause a lot of arrhythmias if you're not utilizing the balloon at the tip.

You want to advance the catheter with purpose. I think we've learned this over time. I had a fellow tell me once that the biggest difference he noted is when the attending comes in the room, they start moving at a much quicker pace. But it is a balloon, and a balloon is sort of sailing in the bloodstream, so using that cardiac output to drive you downstream can sometimes be extremely helpful. If you're just kind of spinning without purpose, then you're not going to get anywhere.

If coiling occurs, it's always important to use a wire to try to avoid tying a knot with a catheter. Don't let the balloon go all the way down. Certainly, if you're advancing, you never want to advance without that balloon being up at least partially.

You want to decide whether you're going to need arterial access, as I mentioned. Again, I usually do my cases by the femoral, but a lot of folks do neck access as well, particularly if they want to leave the catheter in.

And then we've already gone over the equipment.

So I do say that with wedge position, you really want to make absolutely certain that when you're going forward, again, I gently deflate that balloon when I'm down. If I'm not getting a good wedge tracing, a lot of times what happens if you just gently deflate the balloon with just a little bit of positive pressure, so you're letting the balloon down slowly, what you'll see is the balloon will jump forward just a little bit, and often that'll create a much cleaner tracing, and you'll see a very nice wedge tracing. Sometimes the balloons tend to catch right at the spot where there's bifurcations and stuff in vessels, and if you don't occlude them properly, you end up with a hybrid tracing. And that can be a real problem. If you're uncertain whether you have a good wedge, you can have the patient do some deep respirations. There should be pretty dramatic respiratory variations in the wedge, and if they're not, then you're probably not adequately wedged. Or you can draw wedge saturation. You can also place a pigtail in the left ventricle if you have an arterial access that's placed. So for the first catheterization, I always will place that just so I have that as backup. And I can also draw my sats that way.

Always make sure you're making your measurements at end expiration. So if somebody's on positive ventilation, that's going to be down, that's going to be the lower number. When you've got a patient that's awake and breathing spontaneously, that's going to be the higher number. And you want to avoid really deep breathing. Sometimes the patient will take a big, deep breath and blow it out, and they'll Valsalva, and that's going to make your numbers inaccurate.

Again, this is very similar. The only tip, I would say, is the final point here is that use an angled pigtail catheter. I just did this twice today. If you really run into trouble, and this is one of my tricks, and it means I get fewer calls about this, but if nothing else works, I use an angled pigtail catheter, and I advance that and then advance the wire. Usually, it's easy to get across the tricuspid valve that way. Then I take the catheter into the right ventricle, turn it upward, advance the wire. I can get across the pulmonic valve, and I'm in the pulmonary artery. I exchange out then for a balloon wedge and so I can get a balloon wedge catheter. So that's one of those little tricks that I think is invaluable.

So now, Val, I think you wanted to go over the interpretation of the hemodynamics.

Dr. McLaughlin:
Yeah. I mean, it's really important to use all the data that you have in the cath lab. So looking at the waveforms is actually really critical to making the correct diagnosis. You're looking at where the catheter is under fluoro, but you should really also look at the waveforms. And look at them yourself; don't just trust the computer mean, because that's going to give you wrong information sometime. As Rich already emphasized, end expiration is the time that we're going to make our measurements.

And so here's a right atrial pressure tracing. And most of the time our patients aren't in atrial fibrillation, so you should see that very nice A wave right after the P wave and then the V wave right after the T wave. And so you have that little AV pattern, and that's what the right atrial pressure tracing should look like. So you want to make sure you have something that looks like that. And then, as you advance, then you'll see the right ventricular tracing where you have a much higher systolic and then you have that little end diastolic blip, which is

very, very typical of the right ventricular pressure.

Dr. Krasuski:
Just to interject, if it's okay, Val, that the A wave is going to be greater than your V wave. It almost always is the higher pressure that's present. And then in the wedge tracing the V wave is going to be a little bit higher than the A wave. And it almost invariably is the case. And you'll also see the end diastolic will kind of correlate with that A wave in the right ventricular diastolic pressure as well. So that A wave when it's like, let's say it's 11, 10, or 8 or something, the A wave, V wave, and mean, and you'll see that that's your end diastolic is going to be 11 and not 8. So when somebody says their mean or atrial pressure was 8, but their end diastolic was 11, it's because of that A wave that you're seeing that correlates with that end diastolic pressure.

Dr. McLaughlin:

Great. Thanks. And then as you advance further, you get the pulmonary artery tracing, and you see that nice dicrotic notch. The difference, again, is the pulmonary [artery] systolic pressure should be similar to the right ventricular systolic pressure, but obviously the diastolic pressure is higher. And this is that typical waveform.

Anything to add about the PA tracing, Rich?

Dr. Krasuski:
Yeah, just remember you've got the mean line there that's present. And a lot of times people will report the means. And one of the ways to distinguish yourself from folks who are sort of novices, a lot of times people will try to put a mean line on a RV tracing. And I say no, no, no, we don't have means on RV tracing. But that's one of the ways you can clearly tell.

Now, sometimes there can be a lot of whip in the catheter or the tracing can not be adequate in terms of the way it looks. Sometimes it's a matter of the filters that they're using. If you're working in the cath lab, you ask them to change filters, and often they'll give you a cleaner-looking tracing.

And there's some patients who have very significant respiratory variations. People with very significant lung disease will sometimes have these just tracings that go up and down. So not just the wedge that's going to have that respiratory variation but also the PA tracing. This is a nice one here that doesn't have that much variation.

Dr. McLaughlin:
The wedge tracing you already spoke to. And again, this one doesn't have a lot of respiratory variation. Sometimes it's a little bit more apparent.

Dr. Krasuski:
Could be asleep.

Dr. McLaughlin:
They got a lot of sedation, huh? We don't use sedation.

Dr. Krasuski:
We shouldn't.

Dr. McLaughlin:
Right. And here's a directly measured left atrial pressure and a wedge pressure. And you see that little bit of time delay in the wedge pressure, huh, Rich?

Dr. Krasuski:
Yes. That's important to recognize that it's a reflected tracing. So we used to, particularly if we're measuring, let's say, a valve case, and we're trying to look at, like, for instance, mitral stenosis, you have to then, if you're measuring your wedge, be able to transpose that back. So.

Dr. McLaughlin:
So we all really focus on the wedge pressure. Like, that's so critical in terms of the diagnosis, what the wedge is. And Rich alluded to earlier, something that we do the same thing, we do our first right heart catheterizations prepared to do an LVEDP if we don't get a good wedge pressure tracing.

And there's some tips on collecting a proper wedge. Some of these were in the top tips that Rich went through, but they all bear repeating. If the waveform isn't perfect, sometimes if you deflate just a little bit of air from the balloon, you'll see it pop forward, and then you can inflate and get a good wedge pressure tracing. You've got to look for those A and B waves. Remember, the wedge pressure

should be for sure lower than the mean pulmonary artery pressure and it cannot possibly be higher than the end diastolic pressure. In pulmonary arterial hypertension, usually the end diastolic pressure is much higher than the wedge pressure.

There's also the possibility of overwedging, as opposed to underwedging. And sometimes you can see that number creep up and get really high. And that is actually a little concerning that you're overwedged, and you're impinging on the vascular wall. You can also look at the catheter under fluoro, see if there's movement, perform a wedge angiogram, and, of course, draw a wedge saturation. If your balloon is inflated and you're truly measuring that reflection from the left atrium, that saturation should be high. It should be a systemic arterial saturation.

And I think this is – do you want to walk us through this image, Rich? I think –

Dr. Krasuski:
Yeah, so this is basically the balloon is up here, and you can see we're injecting a little bit of contrast dye. And it's because the tracing didn't make sense. It was somebody basically who expected PAH, but what it looked like when we did this was that the wedge pressure was super high. But you can see that we're at a branch. So this is why it's so important, when you deflate that balloon, it'll take you into the more distal branch, where you're more likely to be occlusive. Because you see, if there's runoff to the other side, you're going to get a hybrid tracing.

Dr. McLaughlin:
Yeah, it's a great example. And large V waves. Let's walk through those, Rich.

Dr. Krasuski:
So when you see those big V waves, and in this patient, all you see is a V wave essentially. There's probably a little A wave in there. It's hard to tell. It could be – I'm looking at that EKG, wondering if it's Afib or something that we've got here, but there are very large V waves. The most common thing this is going to be, is going to be mitral regurgitation. People often say, oh, it's pathognomonic. It's not really pathognomonic. Anything that gives you a stiff left side can give you those larger V waves. But it's going to be classically with mitral regurgitation.

Dr. McLaughlin:
And then this is the respiratory variation you were referring to earlier. And it's often worse in patients with lung disease, with COPD, ILD. But this is tremendous amount of respiratory variation here. And you can see how different an end expiratory wedge would be from a mean wedge in a patient like this.

Dr. Krasuski:
I'm amazed how sometimes that the mean line will go out there and it's a straight line. So you really want to make sure that you're measuring the end expiratory and you're not measuring the mean as much, because I think you can be really fooled, right? Because in this case, the mean here probably is about 15, but you can see the end expiratory is really about 22-25 here, range. So this is somebody who has left ventricular diastolic dysfunction, but you could get fooled and call them 14 or 15 if you just looked at their mean.

Dr. McLaughlin:
Sometimes it's really hard to get into the PA in these folks. The combination of the big right heart and the TR and put low flow on top of it, like you just don't have as much flow pushing that balloon forward. So what are your tips to get into the PA, Rich?

Dr. Krasuski:
Yeah. So one of the keys, and we mentioned this earlier, is to really use as much as possible the technique of kind of using the flow and going with the flow so that you're using the balloon kind of like a sail or whatever on a sailboat so it helps you to move distally. That'll help you to get over. Using fluoroscopy so you can actually see where you think the tricuspid valve is and aiming in the correct direction. If you have lateral imaging available, it's really neat because you can actually look at the lateral and you know that you're going to go anterior, to go from the right atrium to the right ventricle. So you want to make sure you're facing in the correct direction.

People will use wires. I'm a little bit careful with wires. I think that there's such a tendency potentially to induce arrhythmias and other complications that you ideally would like to avoid that.

The PA can be challenging too. I think one of the things I've learned, particularly when I'm teaching the fellows, people want to torque the catheter so much and just they end up spinning it around and around. And the real key is that you don't transmit torque unless you're advancing the catheter or pulling the catheter back. So you can put all the rotation and spin that you want on the catheter, if it's not moving forward or back, you're not transmitting that torque. And so if you're spinning, spinning, spinning and then pulling back, it's going to keep spinning around. And you really want to try to avoid that. So you want to do a controlled spin so that it'll turn up towards the right ventricular outflow tract and then advance when that starts to head in that particular direction.

So you can do other things. I mean, some people have tried to reshape the tips and do all sorts of other stuff, or try different catheters, different Swans. And then asking the patient to take a deep breath, that's another way to kind of increase the flow so that it helps you to move forward. And a lot of times getting up into the PA, that's a great tip. So if you tell somebody to take a breath in and hold it, and then you try to advance the catheter then, the flow is going to take you up into the pulmonary artery.

Dr. McLaughlin:
Those are all good tips. And we've talked a little bit about getting the wedge as well. I think one thing that's important is having that pretest index of suspicion as well. Like, do you expect this to be left heart? Do you expect this to be straightforward pulmonary arterial hypertension? And looking at the tracing is using fluoro, getting a wedge sat, that's all important. And here you can see, there you go, you can see that advance, and now the balloon is not moving, the catheter is not moving, but you can see under fluoro, that can help guide you to get the wedge pressure.

Dr. Krasuski:
That was not my slide, by the way. Just to make that clear, I don't move that catheter forward without the balloon up.

Dr. McLaughlin:
And then the catheter could be overwedged as well. If you don't see respiratory variation, if you just see that number going up, and I think I have an example of that coming later.

So cardiac output. Man, Rich, this is really an important issue, and I see too many caths that don't have cardiac output. This is absolutely critical for the PVR calculation. So it is really required for the diagnosis, and it helps us assess the severity. In fact, the severity and the prognosis in pulmonary hypertension is dictated more by how the right ventricle is functioning against that high pressure, against that high resistance, than the absolute pressure itself. So the indices of right heart function, such as cardiac output, are really, really critical.

There are a number of ways to measure cardiac output. The gold standard is a direct Fick. So the Fick formula basically looks at the patient's oxygen consumption and saturations before and after the lungs. And to really do this well, you need to put a hood on and directly measure the VO2 at the time of the procedure. Now, Rich probably does this not infrequently, certainly, I bet he does this in all his congenital cases, but most adult labs rarely put that hood on and measure the oxygen consumption. And instead they use a calculation, an assumption to calculate the VO2 based on the patient's age, height, gender, what have you. And those are notoriously inaccurate. So the indirect Fick is the least accurate measure of cardiac output.

And then kind of in the middle better, I think, than indirect Fick, but not as good as a good measure of oxygen consumption, is thermal dilution, where we inject saline into the proximal port, into the RA port, and then the sensor measures the temperature later at the PA. And you calculate the cardiac output by looking at that speed of that temperature change. So this is really, really important.

What do you use regularly, Rich? Do you direct Fick on everyone, or just the congenitals? What do you do?

Dr. Krasuski:
I do it on the more complicated cases. I think it'd be nice if we could do that easily. It does take a little bit of extra time to get the hood and to do all the calculations, and so it is time-consuming. I think that's the biggest thing. And everybody always rolls their eyes in the cath lab when you bring the hood over, and people aren't too pleased with me when I do. But I hate to say it, a lot of times we do use indirect Ficks. They're just easy. But it's absolutely a great point, right? Not only do we know that there's a lot of inaccuracies in those formulas, but we also know that when people are sick, their oxygen consumption is very different, right? When you're well, it's one thing. When you're in the hospital and you're ill, your oxygen consumption is very, very different. It can be significantly higher. And you're introducing a lot of error by simply making an assumption based on a resting status that's been calculated for other patients. So it's really important to get the numbers correct.

Dr. McLaughlin:
Yeah, and again, I can't emphasize how important it is to have a good, accurate cardiac output. I mean, it is very important in terms of prognosis. It's very important in terms of calculating the PVR. It's very important in terms of helping us decide what medication a patient should be on.

And then there's also the other end. There are certain conditions like cirrhosis and hyperthyroidism and anemia, where the cardiac output is very high, and then you calculate a normal PVR, and the treatment is much different. So it's really important to focus on that.

Rich, you want to talk about vasodilator challenges?

Dr. Krasuski:

Sure. I think it's important. If you look at the guidelines, basically they're going to recommend that this be done predominantly in Group 1 patients. And specifically, it's going to be idiopathic disease. It's going to be people with drug-induced – it's going to be people – but we've learned now that actually it's helpful in a lot of other patients. And I don't necessarily use it necessarily to see who I'm going to treat with a calcium channel blocker. I think you could say that reactivity, which has been defined as a drop in the mean PA pressure of greater than 10 mmHg down to a value less than 40 mm would be considered a positive challenge. But I think if you have that kind of a challenge, most people are still going to use the newer medications to treat the pulmonary hypertension and not necessarily put somebody in a calcium blocker. I mean, every once in a while, I see somebody and they have just a dramatic change, and I think in that person, it makes total sense to put them on a calcium blocker. Because in general, they're fairly well tolerated drugs. They're extremely inexpensive, but we also know that they're not disease modifying like some of these other agents are.

Now, how do we assess the vasodilatory response? The most common thing is to use inhaled nitric oxide. I give 40 parts per million. It takes 5 minutes. You get that; you repeat your data. And I'm really doing it for two purposes. One is I want to see the drop, but secondly, I want to measure that wedge, because in some patients, I do recognize their wedge goes up, and we can unmask people who have Group 2 disease. And sometimes you just don't know until you see that wedge rise that they have a Group 2 component to their pulmonary hypertension. And it's a lot easier, I always tell folks, to recognize that in the cath lab than it is to send somebody home with a medication that's going to be in their system for a significantly longer period of time, and they're going to feel worse on that.

So again, a positive response is that drop that I mentioned to the mean less than 40. No drop in the cardiac output or index, because in some patients we see that, and that obviously negates the effect that we've seen. And ideally, no rise in the pulmonary capillary wedge pressure. Again, recommended for idiopathics and other Group 1 disease, in particular, drug induced.

But there's another, and we're going to talk about this more, about there's some data suggesting repeating this, and if you lose vasoreactivity, it may be bad. We've also looked at what the impact is in terms of responders versus nonresponders for patients with idiopathic, with non-idiopathic Group 1 PH, and we found, again, that there was a statistically significant improvement in survival. Now, obviously patients are also going to be receiving other therapies. It's a little harder to control for that, but I do think it gives you a good prognosticator. In addition to everything else you're looking at, 6-minute walk distance, echocardiographic findings, etc. Here is basically a survival of Group 1 versus non-Group 1, which is maybe a little taboo here. But we found in our patients, again, that that vasoreactivity kind of predicted a group of patients with – other groups, like Group 3, Group 4, again, suggesting a better clinical outcome if they're responders than if they're nonresponders.

Dr. McLaughlin:
And so, Rich, you spent a lot of your life doing congenital heart disease, and it's something we should always be thinking about. Because people are surviving into adulthood. Sometimes their diagnosis is missed, or sometimes they're living with the residual or sequelae of interventions they had younger, so why don't you talk us through that?

Dr. Krasuski:
Yeah, so I mean, if you're seeing somebody that is going to undergo a heart catheterization and they have a history of congenital heart disease, as a flat statement, it's always smart to involve an adult congenital heart expert. It's in the guidelines that if you have a patient, that before they undergo a heart catheterization, it's always important to involve the congenital team. But if it's undiagnosed, and we see this periodically, I'm sure you've had patients just like I have that have had heart catheterization, multiple heart catheterizations, and then at some point you recognize, boy, that high cardiac output is not really – or normal cardiac output is not a normal cardiac output, it's a shunt that's present. So I think when you see somebody who has elevated pulmonary pressures, no progression in their heart disease despite perhaps a lack of therapy, you've really got to keep it in mind that you might be missing a shunt. So doing that first.

Now, this patient here, you can see on the right, they have an occluded femoral vein. And so that's important to get too. If you get the old records, you can find out who has had prior interventions, prior catheterizations, and where the access was from. And reviewing old records is important. Deciding on the access sites is important. And ruling out, especially in somebody who doesn't have a diagnosis, ruling out the most common lesion of all, which are shunt lesions.

So I have made it part of my routine first heart catheterization to at least get an SVC saturation. You want to wire up into the SVC, get that saturation, and it should ideally be about the same as your pulmonary artery. If the pulmonary artery is higher, then you've got to step up. And I think when there's a step-up, you should ideally do a full saturation run. Again, normal or supernormal cardiac output in somebody with elevated pulmonary pressures, think about this. You might miss an undiagnosed shunt. A shunt run, again, should be part of the initial catheterization, and at the least get that SVC sat because ASDs and partial anomalous veins, they're the most common thing that you're going to see.

I like to get at least two samples from the PA and the femoral artery. And again, they want to be within 2%. Because sometimes people

will draw one sat, and it's really hard to make all your assessment looking at all this based on a single saturation. And as you mentioned earlier, review the data when the patient's on the table. There's nothing more embarrassing for me to send somebody back to the floor, or even worse, send them home, the shee's pulled and I realized they had a step-up that I missed.

The RA is the area where you get mixing from the SVC, the IVC, the coronary sinus. So depending on where you grab your sat, you may find that it's not the proper mix. So some people do multiple RA sats. I don't do that. I think there's enough data collected from the downstream sats to help you decide. And you can see you need a significant step-up. The more distal the shunt is, the less of a step-up you need to make that particular diagnosis. And here is how I do a sat run. So here is, first thing I want to do is I want to get up above the innominate vein here so that I'm certain that I'm not missing a shunt. And why do I do that? Because anomalous pulmonary veins can come in to either the SVC/RA junction or they can come into the innominate vein. So you want to get that sat first. So here is anomalous right upper pulmonary vein. You don't want to miss that sat, stepping up at that level. So you want to be above that.

And then the next one here you'll see, this is the anomalous left upper pulmonary vein draining into the innominate vein. So again, if you get above that innominate vein and get a saturation, you're not going to miss that particular step-up, but you can easily miss it.

Then I go in and I get an IVC sat, and then I pop right back into the right atrium. And I do these 3 sats, boom, boom, boom, in succession. You don't need to disconnect; you don't need to connect. You don't need to do all this. Just grab your sat. Come back, grab your sat. Go forward, grab your sat, and then you connect to the transducer to get a right atrial pressure.

Again, when you're doing saturations, particularly in the right ventricle, you want to pull very gently, because I've seen people get ectopy when they pull a little too hard. It's not unusual. And then a PA saturation. And depending on where you're looking, for instance, sometimes you worry about a patent ductus. If you look in the middle of that picture, there's the duct, that you can see that duct is closed. But if there's a patent ductus, you want to get a sat distal into the left pulmonary artery, because that's where you'll see the step-up.

I only do a high and low right atrium if I'm really worried about where the ASD location is. And like I said, I don't think that that is all. I've been led astray by getting multiple atrial sats. So I don't think it's all that helpful to get that.

Dr. McLaughlin:
These patients often have TR. And I think it's difficult to make sense of that data. And if you know the step-up is at the atrial level, they're generally going to have a TE or something. So I agree with you on that one.

Dr. Krasuski:
Yep. Yep. And then for calculating shunts, ideally, you want to get all those sats, and then you want to calculate your mixed venous, right? So your mixed venous by convention that's been calculated, is 3 times the SVC plus the IVC over 4. Why is that calculation? Because we're not measuring a coronary sinus, and so that coronary sinus is, if you really wanted to measure properly and figure out the percentages, you'd have to include that too. But this does a very good, accurate way of doing it.

Now, if you don't have an IVC sat, you can use the SVC saturation as the, quote/unquote, poor man's or whatever mixed venous saturation. And again, if there's no shunt, your SVC sat, your mixed venous sat should be the same as your PA sat. If you see a step-up, you know that you have a saturation, that there's a step-up there; there's a shunt.

So I do this all the time when I get my calculations. If you want to figure out your QPQS, so QPQS is Q is blood flow in the pulmonary artery, QS is blood flow in the systemic. So if you want to know your pulmonary over your systemic, and you see all the other numbers can't cancel out, all you need to know is your arterial sat and your mixed venous saturation, then your pulmonary vein, which, if you don't have it, you can assume it's 95%, and your PA sat. And with that, you can calculate your QPQS. So if you're worried about an ASD or whatever, you can calculate that QPQS and know if it's over 1.5, that it's significant.

So let's quickly do a shunt run here. And you can see this is a patient who we've measured their SVC sat at 64, their IVC at 68, and you see there's a significant step-up at the ventricular level here because this patient has a VSD, and so their PA sat is now 80%. And if you stick those numbers in the calculation, you've got your aortic sat, you've got your pulmonary vein, which you're estimating at 95, so it's 95 minus your mixed venous or 65 and then you've got 95 over 80. And because we've made this such a simple equation, this ends up being 2 to 1, which we know is a significant shunt. So this is somebody potentially we'd be worried about a significant VSD that needs to be corrected.

Dr. McLaughlin:
Yeah, that was a great high-level overview on shunts.

Let's talk a little bit about left heart disease, because this is the most common cause of pulmonary hypertension worldwide. And this is something that we always need to think about going into the cath lab. And really it starts with that pretest probability. And there's lots of

tables like this. This happens to be from the 2018 World Symposium, published in 2019, where you look at some of these risk factors that predict the probability of a left heart phenotype, things like systemic risk factors, hypertension, diabetes, obesity, previous cardiac interventions. Afib is like, really, we need to think about left heart disease a lot. There's some echo findings, some EKG findings, some CPET findings that can push us to a higher or a lower probability of left heart disease.

We now have some objective scores for this too. I love this H2FPEF score. It's so easy to do. You have all the information you need when you're seeing a patient in clinic. So the things are 2 H's, so heavy and hypertension more than 2, anti-hypertensive, atrial fibrillation, pulmonary hypertension on echo, older, it kind of sucks getting older, but the diastolic dysfunction is common the older you get, and then E/e' on Doppler. And you can see the point values there, the maximum point values. You sum up the points, and then you get the risk of HFpEF on the bottom, based on the total points. So that, I think, is really helpful in clinic, and it's helpful to explain to referring docs, and it's helpful to decide who to cath and who not to cath. If someone's score is super high, then there's always the opportunity to say let's maximize your medical therapy of your HFpEF. Let's diurese you more. Make sure your blood pressure is controlled. Maybe add an SGLT2. So I think we have to think about those things before getting to the right heart cath.

And then, when you're in the cath lab, you need to know the patient. You need to know their risk, their probability of diastolic heart failure, and take that into account when you do their hemodynamics. And you need to, we can't say this enough, you need to really pay close attention to that wedge. Measure at end expiration. If you don't think you've got a perfect wedge pressure tracing, then get an LVEDP. So I think that's really, really critical.

And here are some examples. We've talked about this a little bit. This is an example of the respiratory variation. And measuring at end expiration is really where you should measure it, and that is often very different than the digital mean. Here's a nice example of a catheter being underwedged. So on the far left you see a PA pressure tracing with a nice dicrotic notch. You advance the catheter and, gosh, that tracing looks different. The numbers are different, but you don't see beautiful A and V waves, and that's actually an underwedged catheter. And if you see that, and a wedge pressure of 22 in a young woman with a family history of PAH and no risk factors, you've got to look at yourself and say, That is not what I was expecting. Is that really a good wedge pressure? And then you could use some of the tips and tricks that Rich talked about earlier, play with that balloon and look under fluoro, maybe get a wedge sat, and really get a good wedge pressure tracing. And so that is a common error in the cath lab.

So you need to know the patient going in, know what you think their likelihood of left heart disease is, and then look at those tracings right there in real time. Think about it right there in real time to make sure you get the correct measurements. Last thing you want to do is take them back to the cath lab.

So that's my little soapbox on left heart disease and wedge pressure.

Rich, I'm going to give you the opportunity to add anything to that that you'd like. I'm just really passionate about that.

Dr. Krasuski:
No. I totally agree, Val. And I can tell the passion there. It's so important, the pretest probability that you talked about very early is so important. So if you see somebody that sort of matches who you think is going to have HFpEf and you get numbers that are different, you've really got to ask yourself, do you trust your numbers or not? And similarly, if you see somebody who matches the phenotype of idiopathic PAH and you get a really elevated wedge, you really have to question yourself and make sure you get that additional data so it fits. You really want to come out of there – because people think, oh, cath is the gold standard. But I've seen so many times caths lead people astray, and then they treat the patient incorrectly, right? The difference between giving somebody a medication to treat their pulmonary hypertension versus giving them diuretics and heart failure medications, is a big one. You can actually do a lot of harm by treating somebody with the wrong line of therapy. So that is so, so important. It's the most important thing I think we do in the cath lab of anything we do, and this is coming from somebody who likes to implant valves and close ASDs and things like that. I don't think those are as important as differentiating a pulmonary capillary wedge to know how to treat somebody.

Dr. McLaughlin:
And then in terms of prognosis, the hemodynamic measures that are most important, not the pulmonary artery pressure. In fact, sometimes as you get really, really sick, the pulmonary artery pressure goes down because the right ventricle fails and it can't generate a pulmonary artery pressure. The most important hemodynamic indicators of prognosis is how the right ventricle is functioning against that high PVR. So right atrial pressure, cardiac index, SVO2, some more recent data suggests that SVI should be in there too. And so I'll just remind you of those hemodynamic cutoffs from the ESC/ERS guidelines. And this is a schematic that really talks about as the disease progresses, it's the patients really get sick because the right ventricle fails and the cardiac output goes down, not because their PA pressure goes up and up and up.

And then let's just say a couple last words about follow-up right heart catheterizations before we get to your questions.

I always repeat a right heart cath around 6 months or so in patients who had an acute response in the cath lab to a vasodilator and I treat with calcium channel blockers. You want to confirm that their hemodynamics have normalized or near normalized. We also often repeat right heart catheterizations on our patients who are on parenteral prostacyclins, because I think it's really important to help dose. We want to get that patient into the range of a normal cardiac index. And sometimes we overshoot, sometimes we undershoot, but I think it's really valuable in helping us manage their prostacyclin dosing.

We have great risk stratification tools, but they're not perfect, and some of the factors in the risk stratification tools like Functional Class and 6-minute hall walk can be impacted by other variables, like the older patients might have worse data because they've got arthritis or because they're obese or any number of other diagnoses. And on the other hand, the younger patients may perform really well on those risk scores despite having really bad RV function. So I think sometimes right heart catheterization is important as an adjunct to the objective risk stratification scores that we get.

And then, of course, sometimes patients progress despite all of our therapies, and we need to make decisions around lung transplantation. So I think repeat right heart caths are important there as well.

And again, this is the more recent hemodynamic measures from the ESC/ERS guidelines that portend prognosis.

Now, I think there's a lot of variability as to how often right heart caths are repeated. I kind of personally find this table from the ESC/ERS guidelines a little disappointing, or maybe I should say it's not exactly how I practice. Certainly they agree that a right heart cath is indicated at baseline, but they don't give as strong of indications for other points, whether it be clinical worsening or changes in therapy. And I know there's a tremendous variation in how experts practice there.

So I don't know, Rich, when do you repeat right heart caths?

Dr. Krasuski:
Yeah, I think, Val, anytime that I'm looking through and assessing the patient and there's incongruities between the different findings. So I've been taught by super smart people like you that if I'm looking at an echo and it doesn't match what I think is going on clinically, or if the patient maybe feels a little bit better but their echo is getting worse, these are the things where there's incongruities so I really want to get the hemodynamics to help me to know where I am. And because it's such a confusing picture, sometimes, right? The RVSP may have improved, but it may be because the numbers are down because the heart is actually getting sicker. And this is what you don't want to miss.

And patients will often downplay their symptoms. They'll say, oh, I feel better, and you're like, better? Like, how? Tell me what... So I think anytime that I'm stumped and the data is not consistent, I'm repeating a right heart cath.

The other thing that I found recently is these patients that get sent to me with elevated pulmonary vascular resistance with shunt lesions, and I will often repeat their caths because I'm pleasantly surprised sometimes how much their resistance will go down with medical therapies, and then we'll consider whether we can close their defects. So those are always ones that I like to repeat.

And like you, I was a little disappointed. I like to be a little bit more aggressive than this. certainly having a IIA recommendation is nice if the patient – but that should be a pretty much a Class I. If your patient is getting sicker, you need to know hemodynamics. I think you want to know which direction things are going and what your impact of therapy has been. And there's no better way to figure that out than to know the cardiac output and the PVR and the numbers, the right atrial pressure. That really helps me the most.

Dr. McLaughlin:
And that's one of the downsides of having something that has to be evidence based, right? Because we don't have a lot of evidence here, but if you put us in a room, I think we could agree on things. So it's a little bit of a methodology thing.

So we have some questions, and I want to try to get to as many of them as we can. The first question is: What do you do when there's a significant discrepancy between the cardiac output by Fick versus thermal dilution? And my answer to that would be, what kind of Fick is it? Right? So if it is a measured direct Fick, that's the gold standard, and that's what I would believe. If it's an indirect Fick, and they're using one of these formulas that has flaws, especially as patients are sick, as Rich mentioned, we can't rely on that data, and I'd rather have the thermal dilution. So in order of accuracy, it is direct measured Fick, highest, take that if you have it; next, thermal dilution; if you don't have the directly measured Fick, thermal dilution is what you should use. And then bottom of the list is indirect Fick.

Rich, there's also a question: What is the best way to address a patient with hypoxia during the right heart catheterization? And you probably deal with this for lots of different reasons. I'd be interested in your take on this.

Dr. Krasuski:
Yeah. No, obviously, the differential diagnosis here is pretty large, right? People can be hypoxic. The two big differentiators are whether

it's heart disease or lung disease. Before the patient gets even into the cath lab, you have some idea of what type of lung disease they have. Hopefully you've gotten PFTs in that particular patient so you have an idea of whether or not they've got bad COPD or bad interstitial lung disease or whatnot. So that's step one.

Step two is, if you're really worried about, let's say, somebody who is – you're worried about a shunt lesion and that their pulmonary hypertension is so severe now, like the Eisenmenger patient, you're shunting now right to left, that's pretty rare to see that. Now, one thing we have, and we've described this, is exercise desaturation. And some patients will either open up or start using their foramen ovale. And the question becomes, what should you do about that? And I can tell you almost 100% treat their pulmonary hypertension. Don't worry about - because if you treat their pulmonary hypertension, the right atrial pressure is going to go down, and that little valve that's opened up, because the PFO is essentially a one-way valve, it allows it to open up, to allow blood to go from right to left. But when their left atrial pressure is higher, it should sort of snap shut. There are exceptions to that stretched foramen and whatnot. But in general, it's treating the pulmonary hypertension. You can get yourself into a lot of trouble if you're trying to close PFOs in patients like this, but we see that. It's not an unusual referral that I see. And there, if it's an atrial septal defect, then you think about it a little bit more. Can you put a device and fenestrate it so they still have a pop-off valve for their right side and close off the shut from left to right? But those are definitely rarer cases.

The other thing I always worry about is that undiagnosed left heart disease, right? I've given them agents which have raised their left ventricular end diastolic pressure, and now they've got some pulmonary edema. And the oxygen doesn't get to the alveoli as well when you're filled with fluid in the lungs. So that can also be another sign of clinical worsening, or somebody who's gotten a vasodilator and they're now sending blood to unhealthy parts of their lung. We always worry about that too, for instance, in somebody with Group 3 type disease.

So these are all different things that should go through your head. And again, having an idea before you go in of what you want to find and what you want to look for with a catheterization can be very helpful. A lot of these diagnoses are made outside of heart catheterization. I get asked to do shunt runs all the time, and I say, okay, I'm looking for a step-up. And they say, “No, can you look for a step-down.” it is very rare that you're going to find a right to left shunt in the catheterization lab. I can count on my fingers, and I've been doing this now for 25+ years, almost 30 years I've been doing heart caths for congenital heart disease, and it's almost never that we make that diagnosis that way.

Dr. McLaughlin:
Great. And last question, very brief answer: What's the best way to address or prevent overwedging?

Dr. Krasuski:
Yeah, do you want to take that first? Do you want me to? Yeah. So, I mean, again, one of the ways that I prevent it is I tend to inflate the balloon and then move forward. The way you overwedge is you will get more distal balloon position, and then you'll inflate the balloon beyond the size of the pulmonary artery. You’ve got to be very careful. I've never had a pulmonary hemorrhage, and I hate to knock on wood because I don't want to have a pulmonary hemorrhage tomorrow simply because I'm tempting the cath lab gods here with my comment. Avoiding going distal and then inflating the balloon up. You always head first with the balloon, and then it's okay to deflate, because the balloon will move forward again as you deflate. But if you're inflating in the distal part of the pulmonary artery, you can overwedge, and even worse, you can damage the blood vessel. And we've seen that even in cases where people were going in for things like balloon pulmonary angioplasty, the damage occurred before the actual angioplasty. Because when we're doing angioplasty, we've taken pictures before; we know how big the vessel is. We're inflating the balloons to the size of the vessel and no bigger. When we're inflating that wedge balloon, even though it's not a very strong balloon, when you inflate it full strength in that little, tiny vessel, that's where problems can occur.

Dr. McLaughlin:
Yeah, and I agree with exactly what you said. But the other thing I would say is keep your eyes on that tracing, right. It's that characteristic. You see that wedge pressure kind of just float up.

Well, Rich, this was highly informative. I hope our audience enjoyed it. I certainly enjoyed talking to you and learning some of your pearls from your day-to-day practical adventures in the cath lab. So I really enjoyed presenting this with you. Thanks so much.

Dr. Krasuski:
Yeah, that was so great, Val. I always enjoyed learning from you. I think all of us have learned from you over the years. So pretty much all I know in pulmonary hypertension probably comes from Val.

Dr. McLaughlin:
All right. Stop it.

All right, well, thank you everyone, and have a good evening.

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