

WEBVTT

Episode Title: Heart Failure with Preserved Ejection Fraction: Updates in Definition, Diagnostic Testing, and Phenotyping

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Meg Fraser: Welcome to the American Heart Association's Heart Failure Podcast Series. This episode is titled heart failure with preserved ejection, fraction updates and definition, diagnostic testing and phenotyping. This program has been created and directed by a volunteer planning committee and is made possible by support from Bayer.

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Meg Fraser: I'm Meg Fraser, and I'll be moderating today's discussion. I'm a nurse practitioner at the University of Minnesota, specializing in advanced heart failure, cardiac transplant and genetic cardiomyopathies I'll next ask my colleagues to introduce themselves.

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Kavita Sharma: Hi, Meg, thanks so much for having me. It's really a pleasure to be here. This is Kavita Sharma. I am the director of heart failure and cardiac transplantation at the Johns Hopkins University School of Medicine. I also direct a dedicated heart failure with preserved ejection fraction program which is a clinical and translational science program focused on care and research of HFpEF patients. Thank you. Again.

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Sanjiv J Shah: And I'm Sanjeev Shah. I'm a heart failure cardiologist at Northwestern University in Chicago, and I'm the Director of Research for our Bloom Cardiovascular Institute and director of our HFpEF program over here.

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Meg Fraser: Thank you both for being here

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Meg Fraser: before we get started. It's important to give the disclaimer the recommendations and opinions presented by faculty today may not represent the official position of the American Heart Association. The materials are for educational purposes only, and do not constitute an endorsement or instruction by the AHA/ASA

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Meg Fraser: The AHA does not endorse any product or device.

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Meg Fraser: So let's get started, Dr. Sharma, hoping we can start the discussion by reviewing HFpEF definition risk factors for its development and some diagnostic criteria.

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Kavita Sharma: Great thanks again, Megan. It's great to be here. The definitions of HFpEF have certainly evolved over the years. If you look back 1520 years ago the definition was really quite broad, and of course this used to historically be called diastolic heart failure, but all of really the kind of academic guidelines, and certainly in practice we have moved to heart failure with preserved ejection fraction, and that was really in the early 2 thousands.

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Kavita Sharma: If you were to look at the accha guidelines today. Generally this is a syndrome defined by having clinical signs and symptoms of heart failure. Now it may not be all of the signs and symptoms. But there ought to be some that are suggestive of a heart failure, syndrome.

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Kavita Sharma: And with that an injection fraction, typically by echocardiography of 50% or greater. There are now other sort of sub guidelines that are incorporated. It's more in the European society guidelines than in the American guidelines, but that might include biomarkers that are elevated, such as NT-proBNP or markers of diastolic dysfunction by echo. But the key is to recognize that not all are required to make the diagnosis.

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Kavita Sharma: which, of course, can make this rather tricky to diagnose in certain patient populations, and we'll talk about that more. But those are really the general sort of contracts around how we make the diagnosis. There are many patients at risk for this, as it turns out historically again, commonly seen in older patients over the age of, say, 65, those with longstanding hypertension. But increasingly, we're seeing this in our patients who have

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Kavita Sharma: obesity diabetes and metabolic syndrome, or what we now term cardio-kidney-metabolic syndrome, and we'll talk about phenotypes as well more. But those are the general guidelines around diagnosis.

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Sanjiv J Shah: Yeah, just, I'd add to that that, you know. When I teach my fellows about this, I like to tell them that I think as clinicians. We're all well aware of what heart failure looks like or what heart failure. At least it's on the differential diagnosis. So sometimes it's just shortness of breath. Other times it's more obvious where the neck veins are elevated and there's lower extremity swelling whatnot.

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Sanjiv J Shah: So it's this thought that there's heart failure. And maybe it's there. The ejection fraction is preserved, and the key thing to think about is that you have to have some way of diagnosing that filling pressures are elevated either at rest or with exertion. And so, you know, sometimes it's real easy. The Bnp is real elevated. They've got leg swelling. Maybe the chest X-ray shows pulmonary edema. Maybe they've got grade 2 or 3 diastolic dysfunction on the echo. But a lot of times it's not so obvious.

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Sanjiv J Shah: And I think that's where we're going to talk about. What are some of the nuances on echocardiography, on invasive hemodynamic testing, that we can use the key thing that we need to tell our audience today is that don't rule out the diagnosis. Just because, for example, the Bnp is normal. I mean, I think we really, the onus is on us, especially now that we have treatment for HFpEF, that we really need to make the diagnosis of it or its mimickers. But the very 1st step is, do they have heart failure?

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Sanjiv J Shah: And is the ejection fraction preserved, and don't get so bogged down on all the other details. Once we know that that they really have the heart failure syndrome. Then we can go about figuring out what kind of phenotype is it? A mimicker, etc? But that's what I think is really important as well.

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Meg Fraser: Yes, thank you. I think that's a kind of perfect segue into. If we could now discuss, maybe, Dr. Shah, you could start the predominant Hf. Phenotypes that we see.

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Sanjiv J Shah: Yeah, you know, we've been thinking a lot about this over the years. And I'll say, from a historic standpoint, it started with just the notion, I think, that we all recognize that this is a very heterogeneous syndrome, I mean heart failure in and of itself is very heterogeneous. But HFpEF, in particular, I think that is really quite varied in its presentation and its phenotypes. So the 1st thing I would say is, how do they come to us in clinic.

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Sanjiv J Shah: and I tend to think of 3 different

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Sanjiv J Shah: phenotypes. So one, in terms of their presentation is, you have a patient who comes in, and they're just very short of breath, I mean, they can't even walk from the waiting room to the clinic room without getting short of breath, and yet their neck veins aren't elevated. They don't have a lot of swelling or any lower extremity swelling.

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Sanjiv J Shah: They're not. They haven't been hospitalized for heart failure, but it sure does seem like they could have heart failure, and those are patients with sort of exercise induced elevations in left atrial pressure or exercise induced left atrial hypertension. We call that the Isla syndrome that's really hard to diagnose, but it is a real syndrome, and I'm sure Dr. Sharma would agree. We have these patients that come to the Cath lab.

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Sanjiv J Shah: Their pulmonary capillary wedge pressure is 13/4 at rest, and just with a little bit of exercise, or putting their legs up on a passive leg raise. The filling pressures go through the roof 30, 40 mercury with these huge V waves

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Sanjiv J Shah: signifying a stiff left atrium. So there is that phenotype.

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Sanjiv J Shah: Then there's the more sort of common phenotype of the patient who comes in. Maybe they've had a hospitalization for heart failure, or they've got clear lower extremity, swelling their neck veins are elevated. They may have some bibasilar crackles, that sort of overt HFpEF/HFpEF, or resting left atrial hypertension, and then at the next extreme. It's sort of the

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Sanjiv J Shah: later stage or end stage HFpEF, where they have significant pulmonary hypertension, right ventricular dysfunction. They have the cardiorenal syndrome often, and so I find it helpful when they're coming in to see me in clinic about thinking of those 3 phenotypes, because, as we'll see in future episodes. The treatment may differ. The kind of clinical trials we

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Sanjiv J Shah: we refer them to could differ. But I think that's very helpful, and the other thing to recognize is those 3 types of you know how they present to us in clinic, also really relate quite well to what their prognosis is and how sick they're going to be, you know, with the lowest risk that exercise induced left atrial hypertension to the highest risk.

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Sanjiv J Shah: A Ph with Rv. Failure type, phenotype. Now, there's others that we really have gotten to know about more recently. I mean, we've sort of understood that they're present all the time. But all this time. But it really, I think, in today's day and age there's a reason to diagnose them, and I think the 1st one is sort of the cardiometabolic or obese HFpEF phenotype, just because we have treatments for that now.

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Sanjiv J Shah: And those patients typically are morbidly obese, and they tend to have lower natriotic peptides. So sometimes it's harder to diagnose, but they clearly have plasma, volume, expansion, pericardial constraint.

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Sanjiv J Shah: and they really can be difficult to treat if we don't treat their underlying obesity and cardiometabolic syndrome.

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Sanjiv J Shah: We've talked about the Phrv failure phenotype, that being a high risk phenotype. What I find in that one is typically there is often if they have severe pulmonary hypertension, and their pulmonary vascular resistance, for example, is significantly elevated. Either they've had very long standing left atrial hypertension, or longstanding, afib

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Sanjiv J Shah: or they have often a second risk factor. So they might have autoimmune disease. They might have something else like chronic thromboembolic disease or chronic lung disease. That's sort of like a multiplier.

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Sanjiv J Shah: A few other phenotypes to recognize are the left atrial myopathy phenotype. These patients may or may not have overt afib, but you can recognize them on the echo. The left atrium is sort of like the biggest chamber on the echo, and we've done studies, and others have as well that this phenotype, this left atrial myopathy. The Lv. Actually is

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Sanjiv J Shah: healthier than we would imagine, based on how sick these patients are. But the left atrium is really sick, and it seems like their syndrome is coming from left atrial failure. They tend to have higher pulmonary pressures.

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Sanjiv J Shah: Lower stroke volume, lower cardiac output augmentation, and that, too, is another phenotype. And finally, there's sort of the hypertensive heart disease. You know, this sort of overlaps a bit with supernormal ef

phenotype, these patients typically have an Lvh and they have an ejection fraction of 60 to 65% or even higher. It sort of looks like hypertrophic cardiomyopathy in a way, but they've had a longstanding history of hypertension.

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Sanjiv J Shah: It turns out that we are seeing less and less of that with the cardiometabolic and obesity epidemics. But we do still see quite a few patients with this hypertensive heart disease phenotype, which has its own nuances in treatment as well because they have the higher ejection fraction.

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Meg Fraser: So we've mentioned Ntprobnp a couple times, and how it's not sort of a perfect biomarker, because it can be normal in some hfef patients. I'm wondering, Dr. Sharma, how do you use biomarkers in clinical practice, whether it be Ntprobnp or others? And are there any that you order routinely.

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Kavita Sharma: Yeah, that's a great question, Meg, you know, this is becoming an increasingly challenging area. Specifically in HFpEF, because of the paradox we see in our obese patients where anti-probing p is often low or normal, so certainly it's a helpful tool. And I'm going to speak specifically to Antiprobion Peak, because we don't have too many other really validated proven biomarkers in clinical practice that are routinely obtained. But there are some that are investigational that are helpful.

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Kavita Sharma: But I guess I would look at it from the standpoint of it's helpful if it's elevated to support a diagnosis, but it by no means rules out

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Kavita Sharma: heart failure in the various phenotypes that Sanjeev just very nicely outlined. If you are still suspicious of a heart failure syndrome, so I wouldn't use it to sort of negate clinical presentation history, and what your clinical judgment is in the patient. And again, most

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Kavita Sharma: institutions use around a level of 150, and above that being elevated for Nt. Pro bnp. Below that being quote unquote, normal. What we are learning more and more is that we probably ought to have a scale that is specific to Bmi and or a calculator, if you will, where we adjust for the Bmi we've published on this and others have, and we know from large clinical trials that even a low, level elevation in an obesity

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Kavita Sharma: patient can be significant. And interestingly. When, for example, those patients are in the hospital and they get diuresis. It's actually the percent change in anti-probnp that we see is markedly elevated in our most obese patients. And so we probably need to shift in our way of utilizing biomarkers in our actual algorithms, of how we use these for making diagnosis and for prognostication. And it is different from Hfref.

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Kavita Sharma: We know again from many who do hemodynamic studies, that you can have low or low, normal anti-probnp and significantly elevated filling pressures in the FpEF syndrome, and so again, helpful, if it's positive and elevated, doesn't rule out. The syndrome can help with prognostication, but we often use the Delta more than the Absolute. So over time, in the outpatient setting, or in the inpatient setting, in the setting of treatment and looking at

response to GDMT therapies, for example.

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Meg Fraser: Great. Thank you.

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Meg Fraser: Dr. Shah. Could you please discuss the importance of echocardiographic measurements when evaluating for HFpEF? So for our audience, what are some of the most important echo parameters in helping develop the diagnosis and distinguishing HFpEF from its mimickers.

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Sanjiv J Shah: You know, it's really important to look at the whole picture together, especially when we have this Bnp issue. And I call Bnp. Biomarker not perfect because of all the things that Dr. Sharma just talked about. And when you think about the echocardiogram of a patient with HFpEF, you know, we typically think of a small Lv cavity and a thickened Lv wall thickness. But it really can be quite variable, and that

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Sanjiv J Shah: suffice it to say, there are many different sort of echocardiographic phenotypes. So the things that we look for are like Dr. Sharma said, preserved ejection fraction. So latest consensus is that the ejection fraction is 50% or greater.

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Sanjiv J Shah: and that the filling pressures are elevated. So what do we mean by that? Well, E to E prime is elevated. So we typically use E to E. Prime greater than 15 at the septum, greater than 10 to 12, depending on the guidelines you read at the lateral wall, lateral mitral annulus, and I think it's helpful. I have this 1113, 15 rule for E to E prime at the septum. If E to E. Prime is greater than 11 in. Afib.

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Sanjiv J Shah: greater than 13 at peak exercise, and greater than 15 at rest. This is septal E to E. Prime. Then the filling pressures are likely to be elevated, and I find that to be helpful, or if the average E to E prime at rest between the septal or mitral and the lateral mitral annulus greater than 13. That also is quite helpful. Left. Atrial enlargement is also another helpful sign. We call it the hemoglobin, a 1 c of left atrial pressure. It's sort of our

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Sanjiv J Shah: biomarker of left atrial pressure chronicity. Just recognize that there are patients who are quite morbidly obese as we've discussed. So if you use the left atrial volume Index greater than 34, you might not catch everyone who has an elevated left atrial volume.

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Sanjiv J Shah: So what I typically try to look at is what's the left atrial size in comparison to the left ventricular size, even if the left atrium is not greater than 34 milliliters per meter squared.

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Sanjiv J Shah: which is the cutoff for increased size. Look at how it compares to the left ventricle. If the left atrium is bigger than the left ventricle

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Sanjiv J Shah: patient probably has elevated left atrial pressure.

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Sanjiv J Shah: I think other things that have been helpful and been shown over the years are an elevated PA systolic pressure. Obviously, you think pulmonary hypertension, but the vast majority of patients who have a PA systolic pressure over let's say 40 on the echo. Really actually have HFpEF, especially if the Ef is preserved and you're seeing left atrial enlargement, etc.

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Sanjiv J Shah: And then I look at the Ivc is the Ivc dilated. Is it not collapsing? Very well, all of these things tell us about filling pressure elevation. Now, the one thing you

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Sanjiv J Shah: did not hear me talk about is, what's their grade of diastolic function? And that's because a lot of times diastolic function grading is misinterpreted, uninterpreted. It can't be interpreted, etc. Intermediate and so don't get too bogged down on that. And remember that this is a diagnosis where it really matters what's happening during exercise. So I'd encourage the audience if it looks like the echo is relatively normal. The filling pressures are normal at rest.

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Sanjiv J Shah: Do an exercise echo? Do diastolic stress testing? Do a bike echo? For example, to get more information. Finally, we're using global longitudinal strain quite a bit. One of the 1st things to get abnormal in a patient with HFpEF is global longitudinal strain. It's not universal.

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Sanjiv J Shah: But looking at that, if a patient has reduced global longitudinal strain, and especially as we talk about mimickers, the patterns on the bullseye map of the longitudinal strain can really help us. So the numbers to recognize there is the normal, I think, of global longitudinal strain, even though we report it out as negative numbers. Think of it as the absolute value.

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Sanjiv J Shah: If that absolute value of global longitudinal strain is greater than 18%, it's normal 16 to 18%. It's borderline less than 16%. It's definitely abnormal. So these are all the things that I look at on a routine echo to see if the patient has Hfpep.

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Meg Fraser: That's fantastic. Thank you. I will definitely store the 1113 15 in my bank and remember that, Dr. Sharma, I'd like to circle back to invasive hemodynamics. So could you please comment on the importance of hemodynamic assessment, and specifically how invasive hemodynamics can be helpful in differentiating HFpEF from the mix.

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Kavita Sharma: Sure. So, Meg, maybe before we get to invasive human dynamics, we can talk a little bit about some of the some of the HFpEF risk tools that are out there just for the listeners. There are a couple of

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Kavita Sharma: helpful practical scores that likely most are familiar with. One is the H. 2 Paf. Score that comes from the Mayo clinic. The second is the Hfa Paf score from the European Society of Cardiology. There are now some modifications of these scores that are really trying to improve both positive predictive value, but also to help rule out HFpEF if you will. We have a score from our center that

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Kavita Sharma: actually found that that left atrium to left ventricular volume ratio which Sanjeev was just alluding to, is actually one of the strongest predictors of Hfpaf, particularly in an obese patient cohort. And so now, in a simplified score, with a couple of other comorbidities like Bmi, we, too, have a score that's out there. And there are other scores that you can find online. But the idea is to take

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Kavita Sharma: readily available comorbidity history. Maybe some laboratory tests, biomarkers and be able to really get a sense of what's the likelihood that my patient has HFpEF the challenge, though with still most of these is that the younger patients who fit that cardiometabolic or ckm phenotype that Sanjeev was referring to often still land in the intermediate score or low score categories.

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Kavita Sharma: So what do you do with that patient if they don't have some of the echocardiographic key signs and abnormalities that we were just hearing about. Where do you go from there. And so that's where most of us in the field still see hemodynamic testing to be the gold standard to make the diagnosis. Now, there are challenges intrinsic to that, because not all centers have accessibility to a cath lab for

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Kavita Sharma: readily available right heart catheterization testing. But nevertheless, this is where we are in the field, and so it might be a send out or referral out.

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Kavita Sharma: We've also learned over the years that when we get to the point of hemodynamic testing, resting. Hemodynamics alone are often not sufficient, and we can still miss HFpEF as a diagnosis if we only measure pressures at rest. Sometimes they're borderline. Other times. The patient's already in a diuretic, and there's just an entire likely phenotype of patients that we were sort of hearing about, that have exercise induced. Whether you

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Kavita Sharma: left atrial hypertension, or simply a milder phenotype of HFpEF. That's just not manifest until you perturb the system. Truly, those patients are then often missed. There's a missed opportunity, if you will, and they don't present until for years later, when they now have more clinically manifest disease.

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Kavita Sharma: So we, as a program have really moved towards exercise hemodynamic testing in all of our referrals for this, if we can. Obviously, if a patient cannot exercise. And just so, you know we do. Supine bicycle exercise. Other centers do upright bicycle exercise. There are various ways to do it, and of course you have exercise echocardiographic testing that you heard about.

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Kavita Sharma: But the idea is to really provoke and to mimic what is happening in real life as best possible for completeness sake. The numbers that we typically use are a pulmonary capillary or PA wedge pressure of 15 or greater at rest.

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Kavita Sharma: If you were to just do a leg raise, then that going up to 18 or so is our criteria with leg raise and then with exercise, a wedge pressure of 25, or greater, and those are generally the numbers that we use. There's obviously a lot more we can glean from hemodynamic testing, and that should also just be a point made that it's not just about the wedge pressure. We also learn about concomitant pulmonary hypertension. We learn about cardiac power. We learn about other metrics

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Kavita Sharma: that can help predict outcomes and what is always striking to me is that there is rarely very little that's truly preserved or normal about the hearts in these patients, whether it is just severe left atrial hypertension, whether actually they have cardiac output and cardiac indices that are actually quite low and often as low as our HFrEF patients with advanced heart failure. Whether there's very significant, right-sided concomitant

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Kavita Sharma: which we know portends worse prognosis. There's just quite a lot of information that we can get from these tests. And it's not just about assigning a diagnosis. It really can help to tailor treatment. I can't tell you how many patients we start to pull back beta blockers off of, because we find that the resting cardiac output is far from normal, where, in fact, many of these patients without a class, one indication

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Kavita Sharma: might be on Beta blocker therapy, so a lot that can be learned from the test then used to modify treatment if it hasn't been initiated, and also now increasingly to help with enrollment in clinical trials, as many of the trials are relying on this tool to really identify the correct phenotype for therapies.

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Kavita Sharma: so we strongly recommend it, obviously quite biased as it's our go to at our center. But to encourage other centers to send out for referral, especially in the complex patients.

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Sanjiv J Shah: Yeah, I think that's a great summary. And I really think that our audience needs to understand that we have no qualms about doing a coronary angiogram

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Sanjiv J Shah: in the setting of a suspicion of coronary artery disease. Why would we do anything different here? It is not a morbid test. It's not a test that has a high complication rate, and it can give us such great information. And so, you know, there are nuances in doing it, as Dr. Sharma said, but I think that it's something that we should be doing to really seal the diagnosis. It's much less risky to do a right heart, Cath.

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Sanjiv J Shah: than to leave the patient undiagnosed, and some of the other pearls that I find helpful are when you're doing the right heart catheterization. Have the patient breathe freely. Don't try to control their breathing. That always

ends up, I think, making the data very difficult. If you have them hold their breath, they could be doing valsalva, etc, and then just measure the pressures at end expiration with them, breathing freely.

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Sanjiv J Shah: It's really helpful to see if they have a Kussmaul sign. So if the right atrial pressure goes up instead of down with inspiration, that's really helpful, because that can tell us that the right ventricle is stiff, that they might have restrictive cardiomyopathy constriction, severe. Tr, if they have big V waves.

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Sanjiv J Shah: And then on the pulmonary capillary wedge pressure side, one of the things that we typically see is big V waves, especially with exercise. And that's because they don't have severe Mr. The most common cause for a big V wave. A tall V wave, especially during exercise in these patients, is a stiff left atrium. So we find that to be very helpful as well, and I can't agree more with this cardiac output, and looking at what happens to the cardiac output with exercise.

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Sanjiv J Shah: and does it go up, etc? I think that it's really important for people to recognize that this can really help in the management, as Dr. Sharma said, and really tell us what is going on with the patient in terms of their phenotypes. As we discussed earlier, so it can be really helpful, and not every place is going to have exercise hemodynamics.

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Sanjiv J Shah: But the wedge pillow is on the order of 30 to \$40, and it's easily available. So there's really no excuse. It just adds a few extra minutes to put the wedge pillow under the patient's legs.

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Sanjiv J Shah: Then, you can see, does the wedge pressure? Is it above 18/18 or higher? Then you're done? And if you're in a restricted situation. You don't have a lot of time, but you have the capability to exercise. Then, if the resting wedge pressure is less than 15 passive leg raise is less than 18. Then you go on to exercise, and most places will typically do about 3 min exercise stages and 20 to 25 watts per exercise

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Sanjiv J Shah: stage, and you start measuring the wedge pressure. PA, pressure and thermodilution cardiac outputs about halfway through each stage, and it's totally doable. And you can get a really good sense of what's going on with the patient that way.

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Meg Fraser: Thank you both. That was a fantastic discussion.

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Meg Fraser: I'd like to end with what's next for HFpEF diagnostics. Are there any anticipated diagnostic tools that are on the horizon.

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Sanjiv J Shah: Well, I guess I could start. I mean, I think it's a really exciting time for HFpEF.

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Sanjiv J Shah: You know. Of course, we have a limited amount of time today. So we didn't get to discuss everything. But I will say that we already have some tools that help us diagnose mimickers, and when I'm taking care of patients with HFpEF. All I care about is

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Sanjiv J Shah: finding patients who have heart failure with a preserved ef and then figuring out, do they have HFpEF? Do they have cardiac, amyloid? Do they have constriction? Do they have hypertrophic cardiomyopathy? Do they have this preload insufficiency, syndrome? Do they have pulmonary arterial hypertension. What do they have? And if they have HFpEF, what is the

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Sanjiv J Shah: subtype of HFpEF they have, and we've talked a lot about that today? But what's really exciting, I think, is AI and machine learning. So there's already a commercially available algorithm for echocardiography where it can give you the probability that the patient is at high risk of having HFpEF.

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Sanjiv J Shah: Of course an echo itself can't diagnose a clinical syndrome, but I find that to be very useful as kind of a barometer that this patient either has HFpEF, or is at high risk of developing the HFpEF syndrome. There are Ecg tools that are coming out. That also will be able to do the same. There's also, as we discussed earlier on the echo itself, just looking at the global longitudinal strain map.

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Sanjiv J Shah: you have the cherry on the top, where we see that in cardiac amyloid you might see a decrement in the basal lateral wall in fabry disease, or a basal anterior or anterolateral wall in hypertrophic cardiomyopathy. Or maybe the center of the bullseye is where the problem is in apical hcm, so that can be very helpful. And I think that there's always

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Sanjiv J Shah: new data coming out on biomarkers. There's several that are promising. It may be a collection of biomarkers, but hopefully, we'll have a lot more as we go forward in the future. Over the next few years.

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Kavita Sharma: Yeah, I couldn't agree more with Sanjeev. It is a very exciting time, mostly because we now finally have treatments for these patients. And so the impetus is even higher to make the diagnosis and get it correct, whereas before we would identify these patients and say, we'll work on your blood pressure, and maybe some of your comorbidities. But now it's so important to know and to get treatment going early. And you know he's absolutely right. We're moving increasingly in all of medicine

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Kavita Sharma: towards having readily available algorithms at our fingertips, driven by AI, mostly to be able to help, risk, predict, risk, predict prognosis. I think really, what is going to be a very helpful tool down the road is if we can identify metrics that really mimic, what a catheterization study can provide, or the closest thing to, especially for centers in rural areas who truly

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Kavita Sharma: cannot access these diagnostic tools to be able to come as close as possible to getting that information and to making that diagnosis. And so it's a really truly exciting time, both on the diagnostic front and on the treatment front in a disease that's not going away anytime soon. So really, the many centers that have been engaged in this type of work are really working hard to try to really improve these tools for all of us.

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Meg Fraser: I'd like to thank Dr. Shah and Dr. Sharma for their expertise in this discussion, and thank our listeners for joining us for this conversation on the diagnosis and phenotypes of HFpEF.

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Meg Fraser: Reminder that this episode is a part of the American Heart Association's heart failure podcast series. More episodes can be found@learn.heart.org. Thank you.