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https://reachmd.com/programs/cme/chronic-heart-failure-primary-care-diagnosis-treatment-referral/9568/

Released: 07/17/2017
Valid until: 07/16/2018
Time needed to complete: 30 minutes

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Chronic Heart Failure in Primary Care: Diagnosis, Treatment, Referral

Voice Over:
Welcome to CME on ReachMD. This activity, “Chronic Heart Failure in Primary Care: Diagnosis, Treatment, and Referral”, is jointly sponsored by the University of Cincinnati and CORE Medical Education, LLC and supported by an educational grant from Novartis Pharmaceuticals.

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Here is your host, Dr. John Russell.

Dr. Russell:
Heart failure is the only cardiovascular disease that continues to increase in both prevalence and incidence, and as our population continues to age, these trends are expected to rise further. Since most patients with heart failure will present in general practice, the community management of heart failure has become increasingly important and the role of primary care providers even more crucial. Collaboration between primary care practitioners and specialists further enhances optimal patient management and outcome.

This is CME on ReachMD, and I am Dr. John Russell. Joining me today is Dr. James L. Januzzi, Jr. Dr. Januzzi is Hutter Family Professor of Medicine at Harvard Medical School and practices at the cardiology division of Massachusetts General Hospital. He also serves as Senior Medical Director for the Baim Institute for Clinical Research in Boston, Massachusetts.

Dr. Januzzi, welcome to the program today.

Dr. Januzzi:
Thank you very much for having me.

Dr. Russell:
Dr. Januzzi, could you go over the epidemiology of heart failure that we’re seeing today in the United States?

Dr. Januzzi:
Sure thing. So, heart failure is a major epidemic in cardiovascular disease in the United States. Currently, there are estimates that in 2017, the number of patients living with heart failure is approaching 6 million patients with probably about a million new cases every year. In addition to the fact that it’s the number one reason for hospitalization among those over age 65 years, it comes with substantial morbidity and mortality, which is a real problem. The mortality after a hospitalization for heart failure approaches 50% at 5 years, and this is despite available effective treatments, and that’s quite a shame. A large number of patients that could be receiving more optimal care are not receiving that care, which in theory could reduce both the incidence of new heart failure, the prevalence of existing cases and reduce the mortality associated with the diagnosis. And, indeed, if you think about the outlook of what we’re facing moving forwards, when we think about heart failure, presently it causes more deaths than all forms of cancer combined, and that’s not the worst part. The worst part is that we’re expecting the prevalence to increase substantially in the upcoming decades given the aging population and our

ability to care for the various diseases that might lead to heart failure better. So, as we keep people alive longer from their coronary artery or valvular heart disease, we expect that the diagnosis of heart failure will continue to grow.

Dr. Russell:
So, as a primary care doctor, are there things I can do to prevent this instead of waiting for this very serious disease to develop?

Dr. Januzzi:
Really important question. It's important to emphasize that with heart failure, there are different forms. There are circumstances where the ejection fraction is normal, and that comes with its own set of risk factors, and then there are forms of heart failure where the ejection fraction, the squeezing strength of the left ventricle, is reduced, and that comes with various risk factors as well. But, you know, it's actually for the primary care physician important to really remember the comprehensive list of risk factors, because both forms of heart failure, reduced ejection fraction and preserved ejection fraction, are equally important, and so things that lead to coronary artery disease—so, elevated lipids, elevated blood pressure, tobacco use, diabetes mellitus—these are all important modifiable or preventable risk factors for heart failure. For those patients that have had a myocardial infarction, it's important to have good follow-up to make sure that their function of the left ventricle is continuing to hold okay. If a patient has heart valve disease or heart muscle disease, careful follow-up and management of their medications is important. Really, all of the various risk factors—congenital heart disease, lung disease, obesity, sleep apnea—these are all things that are really in the hands of the primary care physician in order to best prevent the onset of heart failure.

Dr. Russell:
So, as a primary care doctor, this is probably folks that are in my office every single day. What do you view as the important things that I need to be doing in taking care of folks with heart failure?

Dr. Januzzi:
That's a really important question, and the primary care physician is really central to the management of patients with heart failure. Besides their role for prevention, it's important to emphasize that once heart failure is present, early diagnosis and treatment is critical to improve clinical outcomes. It's not just improvement in quality of life. It's also length of life. The primary care physician is an integral part of the team, and so understanding the role of the primary care physician relative to the cardiologist, for example, is really important. I really say to my colleagues here in the Corrigan Minehan Heart Center at Mass General that the primary care doctor is like the quarterback of the football team. They establish the diagnosis and then pass the patient over to the specialist. In the office the clinician should be sensitive to signs and symptoms of heart failure, and they play a really important role in the longitudinal management of patients across the whole journey of the diagnosis from beginning all the way to the end.

So, relative to recognizing the diagnosis, the Heart Failure Society of America talks about the faces of heart failure, that being fatigue, activities being limited, chest congestion, edema or ankle swelling, as well as shortness of breath, and those are some of the cardinal signs or symptoms of patients that have heart failure. And there are many others including various forms of shortness of breath including orthopnea—that's shortness of breath when lying flat—is very specific for heart failure. Paroxysmal nocturnal dyspnea, waking up in the middle of the night short of breath is also very specific for heart failure, and it's related to pooling of blood in the lungs when a person lies down. Shortness of breath when bending to tie shoes has recently been described as being called bendopnea. This is a circumstance where venous return gets influenced by flexing at the waist and causing shortness of breath. And then there are other cardinal signs and symptoms including right upper quadrant discomfort from congestion of the liver, swelling of the abdomen, of the legs. From the fluid that patients retain, a very common sign is weight gain that's hard to explain, and indeed, a lot of the times patients try to come up with reasons why they're gaining water weight besides the fact that this may be from heart failure. These are all signs and symptoms.

The clinician, if they have got a heightened sensitivity that the patient may have heart failure, may reach for diagnostic testing, and so the PCP can get an electrocardiogram. This is useful because it's rapidly available, and it really doesn't cost very much, and it allows us not only to identify the potential cause of heart failure—so, for example, if a patient has coronary ischemia, or very importantly, if they've developed atrial fibrillation, a common trigger for heart failure—it also might identify what we say the substrate for heart failure. In other words, if the primary care doctor sees Q-waves suggesting a prior myocardial infarction or left ventricular hypertrophy that might suggest hypertrophic cardiomyopathy, hypertensive heart disease, valvular heart disease, or, interestingly, the increasing incidence of amyloidosis. If the clinician sees low voltage on the ECG in somebody with heart failure, that might suggest that the patient has an infiltrative cardiomyopathy. And then the next step is to refer the patient for diagnostic testing, including echocardiography, really the gold standard for visualizing the left ventricle. There are pros to echo. It's widely available, it's reproducible, it's portable, in the hospital at least, and it gives you functional information about cardiac size, function of the left ventricle, valve disease. It tells you about filling pressures, and it can also give you something about prognosis, which is useful, especially if you measure it repeatedly over time. The down side, of course to echo, is that it's costly. It requires referral. There can be a wait for getting the test, and it requires specialized

interpretation.

The other thing is it's important to emphasize that half of the patients with heart failure have preserved left ventricular function, and so the more subtle imaging techniques that we use to measure diastolic function, which is typically impaired in these patients, may be somewhat challenging for some labs. So that moves us to other types of testing, and there's been a growing use of blood tests to help support the decision-making of the clinician who might be suspicious of heart failure, and the use of natriuretic peptides, BNP and NT-proBNP, has really grown for the diagnostic and prognostic evaluation of patients with suspected and proven heart failure. And so, BNP and NT-proBNP both have a Class 1 level of evidence A, so, really, the highest level of support in the guidelines for establishing diagnosis, as well as prognostication in heart failure.

Dr. Russell:
So, I have a patient in my office and I have clinical suspicion that they might have heart failure, so I get an EKG; I get an echo; I get a BNP; I get the patient a scale; I counsel them on diet; maybe I start a little bit of medication. we don't want to refer that person too early and waste your time. We also don't want to refer that person when it's too late , when do you think is the right time for them to land in your office?

Dr. Januzzi:
You want to make sure that you refer patients in a timely fashion so that certain things that a heart failure specialist or cardiologist has access to can be employed before the patient's too far down the road. I work at the American College of Cardiology, and we have a consensus document coming out that really addresses this very issue. What we have focused on is this great acronym that primary care physicians should remember, which is I NEED HELP.

So, I NEED HELP, what does that mean? Well, the "I" means that if a patient requires intravenous inotropes—let's say that they're in the hospital and their heart failure requires the use of drugs like dobutamine or milrinone, commonly used, actually, in the acute setting—that's clearly somebody who would merit consideration of referral. That's unusual, but there are others.

So, patients with New York Heart Association Class III or IV, so these are patients that are short of breath with really minimal exertion but despite good medical management, if their BNP and NT-proBNP remains elevated, that identifies a high-risk patient no matter how the patient may tell you they're feeling, and so persistently elevated natriuretic peptides is the "N" in I NEED HELP.
The first "E" is for end-organ dysfunction. You know, the classic one that you probably see is worsening kidney function. That's a real sign that the patient may be heading in a bad direction and might benefit from advanced cardiovascular evaluation.

The second "E" is a low ejection fraction. So, ejection fraction left ventricular function less than 25 percent. These are patients that are really in a situation where it's unlikely that medical management is going to necessarily reverse, remodel their ventricle sufficiently to improve their ejection fraction to normal. And so, again, someone might be a candidate for transplantation or advanced mechanical circulatory support in that setting, but if they're not in the hands of a cardiologist, the opportunity might be lost before they unfortunately pass.

The "D" stands for defibrillator shocks. So, if a patient has an implantable cardioverter defibrillator -- and they should if their ejection fraction is less than 40%, definitely if it's under 35% -- if they receive a defibrillator shock, that identifies a patient that's probably in need of consideration of referral.

The "H" in HELP stands for hospitalizations greater than one. It's well understood that the frequency of hospitalization directly predicts mortality in patients with heart failure, and so repeated hospitalization should buy a patient referral.

The "E" in HELP stands for edema or escalating diuretics. If a patient is having worsening troubles with fluid retention and you're increasing their loop diuretic consistently, once again identifies a need for referral.

The "L" stands for low blood pressure, high heart rate. These are patients in whom titration of medical therapy will be increasingly challenging.

And that's the "P" in HELP, which is prognostic medication. In other words, the meds that we really want to try to increase to as high a dose as possible, if these prognostic medications are not tolerated or if you have to down-titrate guideline-directed medical therapy, those are all reasons to keep in mind for referral to a cardiovascular specialist.

Dr. Russell:
So, now that I've referred a patient on to you and they're sitting there in your office, how do you start your approach to thinking through a pharmacopeia for medicines for your patients with heart failure?

Dr. Januzzi:
This is a really challenging circumstance for managing patients, because heart failure is a diagnosis that involves a layering of multiple different therapies, and let's specifically talk about reduced ejection fraction patients, because here we have a well-defined list of medications that can reduce mortality, whereas with preserved EF heart failure, it's really about treating the individual risk factors. We don't yet have a defined pharmacopeia for preserved EF heart failure, but focusing on reduced ejection fraction, we know that patients with reduced EF have substantial risk despite current, everyday, guideline-directed medical therapy. For example, in the most recent contemporary heart failure study, about 1 in 4 patients in the control arm of a study that compared high-dose enalapril—ACE inhibitor, standard therapy—to a new drug called sacubitril/valsartan, which we'll talk about, 1 in 4 patients actually experienced a cardiovascular event, either death or heart failure hospitalization. As their first event, nearly half of them that was the first event they had ever suffered, and so, this really shows that despite conventional therapy, we have a lot of work to do with respect to treating patients better and getting them on therapies that will reduce their risk for death.

So, common issues that might lead to inadequate treatment: It's a real challenge, as I said, sometimes to manage these patients with advanced heart failure, so it may be that there's inability to reach the guideline-directed drug and dose targets, clinicians might be uncertain about what drugs to use, and understanding the guidelines can be challenging in terms of the list of drugs. There's often great uncertainty about target doses. Heart failure is a unique circumstance where our target is the dose of the medicines rather than a biological target like blood pressure, and so we frequently will up-titrate therapies despite what we would call conventionally "normal" blood pressure. Patients don't like taking lots and lots and lots of meds, nor do they like the titration of meds that sometimes needs to occur in heart failure and, indeed, these therapies may lead to side effects as well. All of these things may challenge our ability to get to the target doses and get on a comprehensive list of medications.

And then, we have new guideline recommendations for newer drugs including ivabradine, a heart rate reducing agent, as well as sacubitril/valsartan, which is a new drug that helps to vasodilate and improve cardiac function and cardiac output in patients with heart failure as a replacement for ACE inhibitors or angiotensin receptor blockers.

Each of these various therapies that we use, whether they're beta blockers, ACE inhibitors, angiotensin receptor blockers, sacubitril/valsartan, mineralocorticoid blockers like eplerenone or spironolactone, all of these drugs have doses. They've got starting doses. They've got target doses. And what I try to tell my colleagues is find the drugs that you're most comfortable with initiating and titrating and try to remember the drugs as best you can. Know that you should start, for example, if you're starting carvedilol as a beta blocker at 3.125 mg twice daily, but you don't stop there. You should never stop there. You should try to get to the doses of the drugs that were looked at in clinical trials that exerted the mortality reduction that each of these therapies may bring. And so, for carvedilol, for example, the target dose is 25 to 50 mg twice daily. Understanding the target doses for all these meds is really important.

Now, I mentioned earlier that the guidelines for heart failure have recently been updated. I really would love to be able to spend a couple moments talking about the new guideline updates because they incorporate a couple of new medications including ivabradine, as I said, which is a drug that has now Class IIa incorporation in the guidelines, and this is a drug that particularly helps to reduce heart failure hospitalization thanks to effects on heart rate, and then another drug called sacubitril/valsartan, which is a combination of 2 drugs—sacubitril, which is a neprilysin inhibitor, and valsartan, which is an angiotensin receptor blocker. So, this angiotensin receptor blocker neprilysin inhibitor, or ARNi, has been actually given a Class I level of recommendation in the guidelines for heart failure, really important therapy. So, I think that a good understanding of both of these drugs is really important for clinicians so they really can take advantage of the opportunities to reduce risk in their patients.

Dr. Russell:
So, you mentioned ivabradine. And how does that work, and what are some of the safety issues and data that I need to know about for my patients?

Dr. Januzzi:
Thanks for the opportunity to talk about this. So, ivabradine is kind of a unique drug that works with a specific channel in the sinus node. It is a pure heart rate reducing agent, so it doesn't have any effect on blood pressure, and it only works when patients are in sinus rhythm. So, in a patient with sinus rhythm, through inhibition of the IF channel—or current, rather, the IF current in the sinus node—use of ivabradine reduces heart rate. So, this was examined in a clinical trial called the SHIFT study, because we know that high heart rates are a risk for bad outcome in heart failure, and the SHIFT study enrolled patients with Class II through IV adult heart failure cases with different etiologies. Again, these are patients with reduced ejection fraction. And these patients had to have a heart rate over 70, in sinus rhythm, and had to have some evidence that their heart failure was not stable, including a recent hospitalization. In the SHIFT study patients were randomized to receive ivabradine versus placebo, and they were followed over a 4-year period or so. And, you know, the drug did what it was supposed to do. It reduced heart rate by about 10 beats per minute from a baseline around 75 or so

down to 64 on average, and by the end of the trial, there was a 20% reduction in cardiovascular death, hospital admission for worsening heart failure, which was driven by a reduction in hospitalization for heart failure, about a 26% reduction, highly statistically-significant. There was an impact on mortality, actually, but it was only seen in patients with very high heart rates at baseline. So, it's important to view heart rate as almost like a biomarker that we're targeting with this drug.

Now, of course, every drug comes with potential side effects, and you asked about what the risks of the drug are, and it's actually reasonably well tolerated. As you'd imagine, because it's a drug that reduces heart rate—bradycardia, low heart rate is one of the more common things that occurs in patients treated with ivabradine—so one needs to monitor the heart rate. There's something called phosphenes. This is sort of a rare, but important, side effect to remember. It's basically due to the fact that the IF current is found in other places in the body, particularly in the retina, and so it can cause flashing lights, particularly in the dark if you rub your eyes. Nobody knows exactly whether this has any long-term ramifications, and, indeed, if you continue the drug, they tend to go away, but it's important to let the patient know that they might develop this.

When we dosed ivabradine, the first and most important thing to say is we have another drug that lowers heart rate, and that's beta blockers. And so, I tell my colleagues in the heart center here, "Before you start ivabradine, , be absolutely certain that you've got the patient up to a well-tolerated high dose of beta blocker, the highest tolerated dose you can. Once you do, if your patient still has a heart rate over 70, then consider ivabradine." And it started at 5 mg twice a day, and you follow them up in about 2 weeks or so, and if their heart rate is below 60, you're done. If the heart rate remains above 60, then titrate up to 7.5 mg twice a day. If their heart rate is below 50, then down-titrate to 2.5 mg. And you can also start at a lower dose in older patients because older folk often tend to be more sensitive to the effects of any drug, including ivabradine.

Dr. Russell:
And this medicine can be used with our other medicines that we use for congestive heart failure, correct?

Dr. Januzzi:
Correct. It's important to emphasize that this is an add-on. .

Dr. Russell:
So, I'm reading lots of stuff about sacubitril/valsartan and how it might replace ACEs and ARBs. Can you kind of walk me through, how this mechanism of action, of this new class of medicines?

Dr. Januzzi:
Sure, yes, so this is a revolution in the care of patients with heart failure. We haven't had a drug that you have to switch from your traditional therapies to something new in years and years. So, this is kind of a big change that's sort of upended how we manage patients with heart failure. Again, this is for reduced ejection fraction, and sacubitril/valsartan, as I said, is a combination of an angiotensin receptor blocker, good old-fashioned valsartan, and the way that works is it just blocks the angiotensin 1 receptor, but it also contains a neprilysin inhibitor called sacubitril. Sacubitril gets converted after it is taken as a pill into another compound that inhibits something called neprilysin. Neprilysin in the body is an enzyme that degrades a number of different hormones in the body, and by blocking neprilysin, what you do is you cause a rise in all of these various hormones that are actually effective for treating heart failure. These peptides, these proteins that are vasoactive—in other words they're vasodilators—when you inhibit their breakdown rise in concentration, so we get higher concentrations of the natriuretic peptides as well as a number of other substances in the body that are good for heart failure. So, what I tell people when I explain the drug is you're blocking the bad by blocking the angiotensin 1 receptor while raising the good, all of the various circulating vasodilator substances that the body wants in heart failure.

Dr. Russell:
So, talking about this new medicine, how has it worked against the standard stuff that we're using each and every day and have been using?

Dr. Januzzi:
This is something that I know sometimes bothers people because we've really become so comfortable with ACE inhibitors and angiotensin receptor blockers. So, what are the data about sacubitril/valsartan? Well, it was studied in a trial called the PARADIGM-HF study, and in PARADIGM, what the investigators did was they first got patients titrated up to a healthy dose of enalapril. So, the first step is they took patients with reduced EF heart failure and got them up to 10 mg twice a day of enalapril. Just to emphasize, that's the highest dose of enalapril examined in any clinical trial of heart failure. For people that sniff at the PARADIGM study and say that the dose of enalapril wasn't high enough, I'd really encourage them to go to the library and pull all the papers on enalapril and heart failure, and they'll realize that that is by no means a weak comparator. They then up-titrated sacubitril/valsartan to target dose and then randomized patients to receive one or the other. So, it was a real head-to-head comparison of well-dosed sacubitril/valsartan versus enalapril.

The study was terminated early, very unusual in large pivotal studies for this, because of a remarkable reduction in the primary endpoint of cardiovascular death and heart failure hospitalization, substantial reductions in both endpoints. If you just want to talk about cardiovascular death, there was a 20% reduction in CV death with a number needed to treat of only 32 patients in order to reduce 1 endpoint. Now, you say, "Well, 32 patients, that sounds like a lot," but that's one of the smallest number needed to treat that you see in any clinical trial in cardiology. Statins have a much higher number needed to treat, for example. It's important to emphasize this is a really huge step forwards with respect to the care of our patients with heart failure with reduced ejection fraction.

Now, just like ivabradine, it's important -- like any therapy really -- it's important to remember therapies come with potential side effects. Right? And so, sacubitril/valsartan, given the fact that it's a combination of 2 vasodilating substances, the most common side effect associated with its use is symptomatic hypotension. Patients tend to get a little bit of a rush to the head if they jump up out of their chair too quickly. On the other hand, discontinuation for low blood pressure was no different than enalapril in the clinical trial. I warn my patients that they may feel a little more head rushy, so to speak, they may feel a little more lightheaded when they get out of a chair, for example, but it's something that patients get acclimated to, and it often goes away over time. To be very clear, because patients tend to become lower risk on sacubitril/valsartan, one of the things that we do to manage the lower blood pressure it causes is we actually lower the dose of diuretic in our patients. So we're often able to reduce the loop diuretic.

One other side effect to remember, so neprilysin inhibition may be associated with a low, but measurable, risk for angioedema. Clinicians may recognize it as a side effect from ACE inhibitors and angiotensin receptor blockers as well, but neprilysin inhibition may also increase the risk for angioedema. In the PARADIGM-HF study, there was a numerically higher rate of angioedema, 16 cases versus 9 in the enalapril arm, but that was not statistically significant. It was less than 0.1% of the overall study population. No one needed any kind of airway management. In other words, no one had airway compromise from angioedema. When contemplating use of sacubitril/valsartan, prior history of angioedema is an absolute contraindication for use.

Dr. Russell:
I think dosing of ACEs and ARBs is in our DNA by now, but with this new sacubitril/valsartan, and it's a combination, how do we figure out dosing? And if I have that patient already on an ACE or an ARB, how do I have to transition?

Dr. Januzzi:
As much as ACEs and ARBs are in our DNA, sacubitril/valsartan is going to be in the DNA soon. We'll get used to how to use it. I know it seems like a spooky new therapy that's come along. And we haven't had to switch, and we're switching away from our security blanket, right? ACEs and ARBs, we know these drugs through and through. Well, any new drug...

Dr. Russell:
We're not used to odd numbers.

Dr. Januzzi:
Right, exactly.

Dr. Russell:
Odd numbers in doses.

Dr. Januzzi:
It's a strange dose, isn't it?

Dr. Russell:
Yes, yes.

Dr. Januzzi:
Right, it's the combination of the sacubitril and the valsartan. That's why those numbers look the way they do. So, let me explain the doses and how we think about changing them. You know, what's interesting is it's already second nature for us cardiologists who frequently use the drug. Couple of basic thoughts first: One, remember the whole angioedema thing? So, ACE inhibitors can cause angioedema, and if you combine ACE inhibitors plus neprilysin inhibition, it increases the risk even further. So, if your patient takes an ACE inhibitor, you've got to get it out of their system before you start sacubitril/valsartan. If a person is taking an ACE inhibitor, the recommendation is to stop it for 36 hours—and patients don't suffer this, we've got plenty of experience doing this now—to prevent the potential risk of angioedema. And then, based on the dose of ACE inhibitor they were taking, you can choose a starting dose. If a patient was taking a big whopping dose of an ACE inhibitor and they have got good blood pressure, you can start on the intermediate dose of sacubitril/valsartan, which is 49/51—it's 100 mg if you put them together—twice a day. And then you should try to increase them up to the 97/103 dose, which is 200 mg twice a day, within 2 to 4 weeks. If, however, they are taking a low dose of an ACE inhibitor or if

they have got softer blood pressures, then start them at the lower dose, 24/26, so 50 mg twice a day of sacubitril/valsartan, and increase it gradually. If they are taking an angiotensin receptor blocker, there's no risk from being switched right over to sacubitril/valsartan. When I say risk, I mean from an angioedema perspective. Once again, if they are taking a big healthy dose of an ARB, you can jump right to the middle dose of sacubitril/valsartan and titrate up to target within 2 to 4 weeks, and then if they are on a lower dose or if they have got softer blood pressures, again, start at the lowest dose. Importantly, if you run into trouble with blood pressure, try that trick that I mentioned earlier, which is down titrating the loop diuretic. It really does actually work quite safely in most cases.

And lastly, you know, there's currently no published consensus about this, although the current consensus document we're working on at the American College of Cardiology, acknowledges that you're going to see patients that are not on an ACE or an ARB. And then the question is, should we just jump right to sacubitril/valsartan? As written, the current guidelines say that a patient should be on an ACE inhibitor or ARB stably so for 4 weeks, but many of us are now jumping straight to sacubitril/valsartan starting at the lowest dose and titrating up because we really feel like it's onerous to the patient to have to be started on one drug and then switched to another just 4 weeks later.

Dr. Russell:
So, Dr. Januzzi, thank you so much. That's an important topic for both of our patient populations, right? We're seeing them at both ends of the spectrum. Are there some final things you really want to go over for our audience to really hit on some of these new points for heart failure?

Dr. Januzzi:
Again, as a consulting cardiologist, I really want to reiterate just what an important role the PCP plays in the management of patients with heart failure and what a critical role they play across the whole journey from beginning to the end of the diagnosis. Patients with heart failure sadly have a high risk for morbidity and mortality, and so, I really come back to the point that the primary care physician who may know their patients much, much longer than us consultants really plays an important role in terms of the management of patients, collaborating with the specialists, because that's really pivotal for optimal care.

When we think about how we might put a dent in this epidemic that we're dealing with, it really comes down to early recognition of heart failure and excellent medical care, achievement of guideline-directed medical therapy and trying to get patients to target doses. New treatment options, as I said, exist for patients with reduced ejection fraction heart failure, which are either added to our baseline guideline-directed therapy, so ivabradine. Just remember, if your patient's heart rate is over 70, in sinus rhythm, and they're on a good dose of beta blocker, then they might be eligible for ivabradine. And then, of course, the big switch, which is changing patients from ACEs and ARBs over to sacubitril/valsartan. Once again, treatment with ivabradine is really an important therapy for those patients on maximally tolerated beta blocker therapy with a heart rate that remains above 70, 75—77 is what the guidelines say. This reduces risk for hospitalization. With respect to switching patients to sacubitril/valsartan, this is a Class I recommendation. In other words, folks, you really should do it if patients are eligible for the therapy, so this is patients with Class II, III, so mild/moderate symptoms, reduced ejection fraction heart failure and ejection fraction less than 40. Being switched to sacubitril/valsartan reduced a broad range of adverse outcomes compared to well-dosed ACE inhibitor, and that includes an improvement in mortality. So, this drug is really a powerful weapon in our armamentarium from chronic heart failure management.

Dr. Russell:
So, hopefully better days ahead for our patients with heart failure.

With that I'd like to thank our guest, Dr. James Januzzi, for discussing heart failure management in primary care.

Dr. Januzzi, it was great having you with us today.

Dr. Januzzi:
Thanks so very much.

Voice Over:
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