Heart Failure Treatment: A Focus on Treatment in Black Patients

Moderated by Keith C. Ferdinand, MD

Discussants: Christopher Leggett, MD; Ileana L. Piña, MD, MPH; Frank W. Smart, MD

DR. FERDINAND: I am Dr. Keith C. Ferdinand, Professor of Clinical Medicine, Tulane University School of Medicine, Tulane Heart and Vascular Institute, New Orleans, Louisiana. I have with me Christopher Leggett, MD, Ileana Piña, MD, and Frank W. Smart, MD.

DR. LEGGETT: I am Christopher J.W.B. Leggett, Director of Interventional Cardiology, Director of the Cardiac Catheterization Laboratory, and Director of the Cardiovascular Institute of Georgia at East Georgia Regional Medical Center.

DR. SMART: I’m Frank W. Smart from the Louisiana State University School of Medicine, Professor and Chief of the Section of Cardiology, and Director of the Cardiovascular Center of Excellence.

DR. PIÑA: I am Ileana Piña, Professor of Medicine, Epidemiology and Population Health at Einstein College of Medicine and Associate Chief of Cardiology for Academic Affairs at Montefiore-Einstein Medical Center.

DR. FERDINAND: Frank, I’m going to start with you. Give me a basic definition of heart failure (HF) and the risk factors for HF.

DR. SMART: The basic definition, Keith, is when there’s not enough cardiac output to meet the metabolic needs of the body. That can manifest as poor function and poor exercise tolerance and congestion. The risk factors are those risk factors that we know for ischemic heart disease because ischemic heart disease, along with hypertension, is one of the classic problems causing HF.
DR. FERDINAND: Okay, so that’s in the general population. We always talk about, although we don’t see them as often, certain rare forms of heart disease such as Chagas disease, which is a common condition in Mexico, Central and South America. With migration of persons from endemic areas, some of whom may have the disease and not know it, we have begun to see cases. In fact, as many as 300,000 people in the United States may have chronic Chagas disease.1-4

DR. SMART: There is, as you said, a myriad of other things that are associated with HF. Actually, it’s now probably close to 200 different diagnoses related to HF so that HF is a syndrome and not really a diagnosis. There are dilated cardiomyopathies, infiltrative cardiomyopathies, and infectious cardiomyopathies. Those are all systolic HF. We’ve not even touched on HF with preserved ejection fraction with its multitude of causes. We are also realizing that HF of all types has a strong genetic preponderance and many single nucleotide polymorphisms have already been associated with various cardiomyopathies.

DR. FERDINAND: I think for the purpose of this discussion, since you have appropriately suggested that HF can be quite complex, let’s focus mainly on systolic HF. Chris, in view of our focus mainly on systolic HF, can you give us some background on HF specifically in identified black patients or African Americans.

DR. LEGGETT: Well, in the identified United States black patient population, there is a higher prevalence of HF nationally. When you focus primarily on systolic HF, in the majority, the number one cause remains coronary artery disease in the majority population. When you examine this issue from the standpoint of the prevalence of coronary disease and myocardial infarctions as a backdrop for causing systolic HF, black individuals are clearly disproportionately affected. African American women, when compared with white women, continue to carry the highest risk in the United States for having these conditions. However, beyond the spectrum of pure coronary disease, especially in African Americans, we should consider underlying hypertensive cardiovascular disease or poorly controlled hypertension as another major culprit that, over the years, leads to systolic HF. Once again, when we look at hypertensive patients, nationally and probably worldwide, blacks carry a much higher burden of hypertension and hypertensive cardiovascular disease than other groups.

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DR. FERDINAND: Frank, when you look at HF—and we’re going to talk a little bit about various therapies, none in detail at this point but just as an overview—give us the basic approaches to patients who have symptomatic HF. Later, we’ll discuss whether these approaches have mortality benefit. So what do you use if a person presents with systolic HF?

DR. SMART: There have been three drug classes that have been identified to make patients live longer and to some degree feel better, and two other drug classes that don’t really impact survival but do make patients feel better. The first, the mainstay of therapy are the angiotensin-converting enzyme (ACE) inhibitors and, in the absence of being able to tolerate them, the angiotensin receptor blockers (ARB). The second is a beta-blocker; in particular, three beta-blockers that have been shown to work in HF are carvedilol, bisoprolol, and metoprolol succinate. Finally, an aldosterone blocker, whether it’s eplerenone or spironolactone. The feel-better drugs are diuretics and digoxin in fairly low doses.

DR. FERDINAND: Now the reason you use the term “feel better,” is because we don’t have actual mortality benefit with either diuretics or digoxin at this point?

DR. SMART: Correct. There is no mortality benefit, but the Digitalis Investigation Group (DIG) trial and then the Randomized Assessment of Digoxin on Inhibitors of Angiotensin-Converting Enzyme (RADIANCE) trial and the Prospective Randomized Study of Ventricular Function and Efficacy of Digoxin (PROVED) trial all show that patients who were taking digoxin felt better and even exercised longer. Even patients with HF and preserved ejection fraction feel a little bit better with digoxin.

DR. FERDINAND: Another controversial point would be if a patient was eu- volemic; hence, we tend to stay away
HF Treatment in Black Patients • Ferdinand

from the term congestive HF, where they have significant systolic dysfunction or history of HF with or without congestion. Can diuretics actually be harmful in those patients?

DR. SMART: The point is far less contentious with HF physicians. Because of the fact that diuretics stimulate the renin-angiotensin system and the sympathetic nervous system, and cause electrolyte imbalances, we all recognize that diuretics may be harmful but are a necessary component to a treatment plan. As you mentioned a minute ago, however, if the patient is euvolemic, you should reduce or even stop the diuretics if you can.

DR. FERDINAND: Chris, are there any clinical pearls that you would like to discuss before we specifically talk about the application of some of these pharmacologic management options in black patients?

DR. LEGGETT: In general, I believe that the evidence-based proven therapies that we’ve prescribed to patients over the years still remain effective today. Overall, we find ourselves clinically complementing the basic cornerstones of HF therapy—ACE inhibitors, beta-blockers, and the diuretics—with additional medications that may provide some added symptomatic relief, as well as a possible survival benefit. These therapies continue to evolve.

DR. FERDINAND: You know again, in most of the major HF trials, non-whites or self-identified blacks have, until recent years, been much under-represented and, indeed, I’ve practiced long enough to remember the Studies of Left Ventricular Dysfunction (SOLVD) trial in which there was a lack of either hospitalization or mortality benefit in the black cohort. Now in SOLVD, 12% were black; it may have been underpowered. There was also another study called the Beta-Blocker Evaluation of Survival Trial (BEST) with bucindolol, which was a National Heart, Lung, and Blood Institute–sponsored trial that did appear to have adequate sample size, and the black cohort did not appear to do as well with the trend toward an increased mortality with bucindolol versus the predominantly white population. We do understand that to the large extent these are very artificial terms when we talk about blacks, whites, and so on. I’ll ask Frank first: Is it disconcerting that we have the under-powering of some of these sub-populations in some landmark studies and then when we do get some of the studies, the data are not convincing?

"African Americans, and in particular African American women ... carry the highest risk in the United States for having [CAD, MI, and CV morbidity and mortality]. Unfortunately African Americans, Hispanics and women of all ethnicities suffer from a disparity in access to life saving technologies and pharmacologic therapies."

- Christopher Leggett, MD

DR. PIÑA: Right, I think that we need to include a wide variety of patients, whether it’s by sex, ethnicity, or age. If you look at the ACE inhibitor trials and separate the women, the hazard ratio in women is not as impressive as it is in the men. Similarly with the black subjects in those studies, ACE inhibitors may not be as effective in that racial/ethnic group.

Now why aren’t we getting more women into trials? Well, we exclude women of childbearing age, so that excludes younger women. Also, if you look at the SOLVD trial, those patients were found, not because we had HF programs back then, but because we were trolling the echocardiography lab looking for HF and post–myocardial infarction patients who were, of course, primarily men. So, fewer women got into the trials. Women may also provide a greater challenge in that they rely more on consent from their families before they agree to participate in a clinical trial. Therefore, it may be advisable to include a family caregiver when attempting to enroll women in clinical trials.

DR. SMART: The large randomized trial gives us the best information we have, but occasionally a sub-group analysis does not show the same benefit or risk as the entire study cohort. We usually believe this is due to a lack of prospective design and under-powering of the particular subgroup to answer the question with statistical certainty. We do know, however, from the bucindolol trial, that there is a genetic predisposition that determines whether or not bucindolol, as well as perhaps other beta blockers, will benefit. That has spurned what I think is the next wave in medicine, which is more of a personalized medicine approach using a patient’s genetics to design a treatment. The field of pharmacogenomics will help to decide what drugs people should get in the near future.
DR. FERDINAND: There have been some small studies looking at pharmacogenomics specifically in self-identified blacks showing some single nucleotide polymorphisms related to beta receptors and showing less response based on that. I’m certainly not making the case that we shouldn’t offer morbidity/mortality-proven drugs to anyone regardless of self-identified race, ethnicity, age, or sex, but I was pointing out that in the landmark studies, we often under-represent certain subpopulations and I’m always a little bit cautious with extrapolation of data.

DR. SMART: I agree.

DR. FERDINAND: So that brings us to other pharmacologic options and again, we are going to revisit the conventional medications, including the ACE inhibitors, the ARBs, and the aldosterone antagonists, but before we do that, is there anything else specifically as it relates to treating self-identified black patients we should discuss, Chris?

DR. LEGGETT: Certainly, in the context of the current therapies that we have discussed, without introducing new therapies, such as the fixed dose combination hydralazine and isosorbide dinitrate (BiDiL®), I think that our biggest hindrance in achieving therapeutic success with our patients is not the lack of available therapeutic options, but rather, often, poor patient adherence, combined with our collective inability to simplify their dosing regimens and persistently pursuing basic clinical and pharmacogenomics research trials to better understand every aspect of the disease process.

DR. PIÑA: I just came from the inpatient service and it is hard to get patients to take so many pills. We always say, “Oh, the patient is not adherent.” But I really think that we have a responsibility to try to find a regimen that they are more likely to be adherent to by reviewing their medications at each visit and removing drugs that are not necessary. Sometimes we add medications to handle side effects of other drugs.

In the case of hypertensive patients, we keep adding drugs before we even optimize one of them. Even though I’m not a big combination pill prescriber, very often for these patients with severe hypertension, the combination of an ACE or an ARB with a hydrochlorothiazide-like drug, in one pill, may improve adherence. So rather than always pointing the finger at the patient, I think we have to find tech-

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-Ileana L. Piña, MD

niques to get them engaged and make their regimen a lot easier for them, with education as to why they’re taking what they’re taking. And, when we have the opportunity to remove drugs, try to leave them with those that are most essential.

DR. FERDINAND: There have been some data from the American College of Cardiology’s PINNACLE Registry along with the American Heart Association’s guidelines programs where they specifically examine registries of electronic health records and subsequently provide report cards to providers and inpatient facilities, pointing out gaps in the application of evidence-based medications for HF or post-myocardial infarction. For instance, data reports appropriate use of ACE inhibitors, or ARBs if not tolerated, or beta-blockers or aspirin. There does appear to be an ability to close some of those gaps when those data are available. Do you think going forward, Chris, that’s going to be a way to decrease some of these disparities in the application of evidence-based medicine?

DR. LEGGETT: Yes, I absolutely think that will be helpful in guiding clinicians and other health care professionals to adhere to guideline-based HF therapies, which have the collateral benefit of helping to reduce racial, ethnic, and gender disparities in access to evidence-based proven medications and life saving technologies. We may need an added layer of electronic health record trained personnel to note data in the medical record so the clinician can remain engaged with the patient. This approach should be effective in both the inpatient and outpatient setting. This will almost certainly reduce the component of racial, ethnic, and gender disparity in access to appropriate care related to physician bias.

DR. FERDINAND: Frank, you’ve had a wealth of experience in the field of HF, and I know working and living in Louisiana you treat a lot of high-risk patients of all races including blacks. Are there any clinical pearls that you would like to add to Ileana and Chris’s concept that we need to do more to increase adherence to medications?

DR. SMART: Keith, it is the Achilles’ heel of what we do, as you well know. I can sit there and write prescriptions all day long, and if people don’t get them filled, then it doesn’t help them. Because of the cost of medications, particularly for the population that we have here in Louisiana with a very high self-pay and Medicaid rate, those patients are tough to take care of. You have to factor cost into your pharmacologic choice all the time. We have to recommend low-salt diets and low-fat diets to these patients, but that’s not necessarily similar to what the normal
diet for the rest of the family is and it becomes a major lifestyle shift for some of our patients.

Yes, it’s a big problem. I wish I had pears to make it better. I think that the problem with adherence is that everybody has their own individual issue and you have to speak to them. I think that my patients who are of a different race/ethnicity do not trust me as much when we first meet. I have to earn their trust a little bit more than I do with some of my Caucasian patients. Maybe that’s just historic. I don’t really know. It may be that I don’t come across as well as I should but I think that there are things that enter into a decision process, when you’re trying to talk to people to take an extra couple of minutes and explain things a little bit more as opposed to just dashing out of the room.

DR. FERDINAND: You know, Frank, the social scientists have a term for that. It’s called cultural humility, understanding that there may be barriers in our communication methods to persons of various backgrounds, many of whom may have limited literacy or have cultural values related to medication use, diet, and exercise that may be different from our own. The practitioner always needs to take the time to sit down at eye level and listen to that patient. When you provide instructions, give them an idea of what they think you’ve said versus mumbling something and scribbling it on a prescription pad and say, take this to the pharmacist. Would you agree with that?

DR. SMART: Absolutely.

DR. FERDINAND: So let’s go back to Chris. Would you like to add anything?

DR. LEGGETT: Yes, it’s an interesting dialogue because there is an automatic assumption that because you are of the same race/ethnicity that there are no communication problems and that you understand each other just because of your genetic racial/ethnic background similarities. There are so many layers to race and/or ethnicity that are economic, social, and environmental, which clearly play a central role in that communication process. So I do agree that taking the extra minute, while challenging, to sit down and maybe put your hand on their back, and look into their eye and connect with them as a human being, often yields more towards compliance than anything that you ever write down on a prescription pad. I believe that the time performance pressures that we all experience in modern-day clinical practice minimizes those important touchy-feely interactions, and the majority of our patient interaction is now becoming computer-based.

DR. FERDINAND: There have been increasing reports of patient dissatisfaction with clinicians who sit at a computer and type into an electronic health record with their backs to the patient.

DR. LEGGETT: Absolutely, and it’s increasingly becoming part of our culture. I was reading in one of the cardiology journals about how handheld devices are going to become such an inseparable dimension of our lives with patient interaction, that we’re going to be constantly entering data. Those data that we’re entering, while useful, are going to take our eyes off the patient.

DR. FERDINAND: Those are all very good points. I’m going to loop back to the pharmacologic management because we do have some additional outcome data in a self-identified black patient population. That was the African American Heart Failure Trial (A-HeFT) and I was one of the steering committee members and an investigator at two sites. But before I give my opinion, Chris, do you want to give us any of your take on the background of A-HeFT and what it was all about?

DR. LEGGETT: A-HeFT was an opportunity to examine the therapeutic benefit of a fixed dose combination of hydralazine and isosorbide dinitrate on class 3 and 4 HF patients of a self-identified black population on standard therapy. It also allowed for an increased representation of women in the trial. This trial provided a clinical opportunity to make more specific comments about whether a particular therapy worked or didn’t work and whether there was a survival, hospitalization, or quality of life benefit. I think beforehand, as you stated earlier, that we just simply did not have that sort of representation in many of the older trials that we’ve relied heavily on.

We have slowly learned that all medication therapies for a particular illness do not clinically provide the same benefit across racial/ethnic and gender lines. There are genetic, biologic, and metabolic factors that contribute to how our bodies absorb, metabolize, and utilize medications. The scientific opportunity to increase our clinical understanding and search for added therapeutic survival and quality of life benefit in African American men and women with HF was hugely important.

DR. FERDINAND: Chris, I agree and hold the same opinions regarding the benefits of the A-HeFT trial. The reason for these positive findings are unclear and perhaps, in blacks, less nitric oxide availability and increased endothelial dysfunction. We should note...
The A-HeFT trial was stopped early because of a significant and clear mortality benefit.

**DR. PIÑA:** The A-HeFT trial did a good job including women, and in the Heart Failure: A Controlled Trial Investigating Outcomes of Exercise Training (HF-ACTION), as many as 35% women in my center, so it can be done. Getting women to consent may take a little bit longer. I don’t have women consent unless either the significant other or the family are in the room, to give the family full understanding of the trial rather than being relayed through the patient, where often the family will say, “Oh no, mom, you’re going to be a guinea pig—don’t get into that trial.” So I think there are different sensitivities to the different gender approaches and care that would help. Providing the appropriate and factual information about the trial to both the woman patient and the family can be rewarding.

**DR. FERDINAND:** That brings us to the next point, and I’ll give some of the data as the background. We’ve already introduced A-HeFT. This was based on some subgroup analyses in earlier studies, the Vasodilator-Heart Failure Trial I (V-HeFT-I) and Vasodilator-Heart Failure Trial II (V-HeFT-II), where black patients appeared to have a benefit with the combination of isosorbide dinitrate, a vasodilating drug and a nitric oxide donor, and hydralazine, an arteriovascular dilator, which also has antioxidant properties. Our purpose was to study a self-identified black population and to see whether there would be a benefit when conventional therapy was added in patients with New York Heart Association class III and IV.

The primary endpoint was a combination of mortality, hospitalization, and quality of life, and 1050 black patients were entered into the study. As I already pointed out, approximately half of them were women and we did, indeed, show an outcome that was beneficial—a survival increase of 43% with the combination of isosorbide dinitrate added to conventional therapy, 33% reduction in the rate of first hospitalization for HF and significant improvement in quality of life, based on the Minnesota Living with HF Questionnaire. The theoretical background was that it may be more than just a hemodynamic effect; it may be related to nitric oxide enhancement therapy by giving the isosorbide dinitrate and an antioxidant effect by giving the hydralazine. That, of course, is unproven.

So we’ll start with Frank, then we’ll go to Chris, and Ileana. What’s your take on A-HeFT and its application to treating HF in black patients?

**DR. SMART:** As you described, Keith, it is very challenging to find any fault with the study. It was well done and I think that I certainly believe that in those self-identified blacks the use of hydralazine and isosorbide dinitrate as an adjunct once they’re on the other big three, if you will, is very much indicated. The only problem is in patients with really bad HF where their systolic performance was quite low, it may become challenging from a blood pressure standpoint—in dropping the blood pressure too much. But in those patients who have reasonably good blood pressure, I think it’s very appropriate and I use it routinely.

**DR. FERDINAND:** You would suggest that if we use that combination of drugs, we should start slow, especially in patients with low systolic blood pressure, to avoid excessive hypotension and dizziness.

**DR. SMART:** Absolutely. I think we underplay, particularly in the HF community, how important blood pressure and feeling bad is. The other thing that we typically don’t do is talk to our patients enough about the effects of their medications on the way they feel and when they should be taken. We talked about adherence before, but many times patients will take isosorbide dinitrate, hydralazine, carvedilol, and lisinopril together and 2 hours later, they feel like they’ve been hit by a bus. Just by spacing the drugs out over the course of the day and avoiding that pharmacologic peak of all of them at the same time is important in managing people.

**DR. FERDINAND:** Yes. Those are good observations. Nevertheless, there are data that A-HeFT patients with low systolic blood pressure <100 mm Hg did well on the fixed dose isosorbide dinitrate/hydralazine, and overall was well-tolerated, even with some increase in blood pressure in these patients on this medication. Chris, what’s your take on A-HeFT?

**DR. LEGGETT:** I think that the 43% survival improvement and 39% reduction in the risk of a first hospitalization is huge. Another, and more global, benefit is that we’ve taken the opportunity to really try and identify a specific population of people to see if the therapy would be useful in that population. After the results were published, many discussions occurred surrounding the study design and whether it was race specific, to the point where it excluded other races and ethnicities from potential study benefit. The reality is that it was an opportunity to really focus on underrepresented study populations with a high prevalence of HF disease to see whether a survival, hospitalization, or quality of life benefit could be achieved with an added therapeutic combination to standard therapy. The benefit was clearly desirable. With that said, I am happy that a relevant HF study of a self-identified black population, with an increased representation of women, was performed. I’m also delighted that significant survival and hospitalization benefit was realized with added therapy.
I am always of the mindset that we need to appropriately maximize our medical and technologic armamentarium as much as possible so that we can look in that therapeutic treasure chest for every patient who walks in our room. The appropriate therapies coupled with knowledgeable clinicians and motivated compliant patients will give our patients the best opportunity for improved survival, fewer hospitalizations, and a better quality of life.

**DR. FERDINAND:** Ileana, any different comments related to A-HeFT?

**DR. PIÑA:** Yes, my concerns have always been, as we’ve discussed before, with adherence, the difficulty for patients to take two additional pills three times a day, and cost of medication. How can we get twice a day dosing? Because if I can convince them at least to take a pill in the morning and take another one in the evening, we may be able to get the full dosage. It’s really a shame if adherence is part of the problem. In addition, I don’t think the usage is being appropriately recommended; the gap is huge. But we really need to push the pharmaceutical industry a bit to come up with different formulations that would make it easier to take, because reduction in mortality of 43% is significant and not seen in other HF trials.

**DR. FERDINAND:** In those clinicians and researchers involved with the early investigation of the use of fixed dose ISDN/HYD, we did entertain having a longer-acting fixed-dosed combination, but it was curtailed perhaps because of some of the problems related to the uptake of the agent and the availability. The other thing is that none of the main investigators, whom I know personally, had really intended to limit it to that population, and even the Heart Failure Society recommendations describe the combination of fixed-dose isosorbide dinitrate for self-described black patients.

That brings us to the last topic, and I would like some real pointed discussion related to performance measures. The American College of Cardiology Foundation, the American Heart Association, and the American Medical Association have performance measures. In the past, they didn’t list the application of isosorbide dinitrate and hydralazine in self-identified blacks. Any take on that and why that is?

**DR. PIÑA:** I was on the writing committee for the upcoming 2012 performance measures. A measure to add the vasodilator combination was strongly considered and discussed extensively. However, the definition of the denominator, i.e., racial data, would be difficult given the current status of racial data collection in most practices. The advent of electronic health systems should improve markedly the racial and ethnic data fields making this measurement achievable. It was very strongly supported around the table, nonetheless.

**DR. FERDINAND:** So you believe that a fixed-dose combination of isosorbide dinitrate/hydralazine should be listed in terms of the application of a performance measure post hospitalization in the future with electronic health records? Do you think that will give us even more impetus for the use of this since it does decrease hospitalization, which, as you know, would be a cost issue for integrated health systems if patients come back within 30 days?

**DR. PIÑA:** Yes, absolutely. We dropped the measure of discharge instructions. A new and more meaningful performance measure is an outpatient visit scheduled at discharge, and reads: “Post discharge appointment for HF patients. No measure for 2005.” This visit post-hospitalization should occur at 7 to 10 days after a hospitalization for HF, and is a wonderful opportunity to go over all the medications again, and if the patient has not had this combination added, that’s a good time to do so. That appointment can also be modified to include more patient education and once again try to engage patient adherence. The use of combination vasodilator therapy needs patient agreement and adherence.

**DR. FERDINAND:** I would like to make one point that’s often mistaken by clinicians. Again, as a person who helped with the design of the trial, we wanted to have patients taking conventional medicines and the misgivings about the under-representation of blacks in landmark studies were understood. Still, if you look at the data, 69% of patients were taking ACE inhibitors and 17% were taking ARBs. So we did use the appropriate medications. I still think regardless of race, ethnicity, sex, or age, we should try to use those medications that are shown to be beneficial in the evidence-based outcome studies. That being said, we do know specifically in the HF population, the addition of the fixed dose of isosorbide dinitrate and hydralazine gave clear benefit, and the benefit was not only in terms of hospitalization and quality of life, but overall all-cause mortality. Although clinicians attempt to replicate dosages of the individual medications hydralazine and isosorbide dinitrate, the Food and Drug Administration has clearly indicated based upon pharmacokinetic studies, that there is no therapeutic substitute for the fixed-dose combination isosorbide dinitrate/hydralazine.

So we’ll go around the table for parting comments. I’ll let Ileana go first and then I’ll let Frank go, the two HF specialists, and Chris, since you are a practicing clinician who sees a lot of high-risk patients. I would like you to conclude.
**DR. PIÑA:** My take-home message is that we have a lot of medications in our current armamentarium. Now we also have devices such as cardiac resynchronization therapy and implantable-cardioverter defibrillators (ICDs). We really need to start using approaches where we have the evidence and where we have the data. To simply say, my patients are not like the ones in the trials, is really not acceptable, and we need to continue to utilize evidence-based therapies for all patients to further close this gap that exists.

**DR. SMART:** I will echo what Ileana said, that we need to apply the therapies that we know work. We did not talk about device therapy and, in particular, ICD therapy, and in my population here in Louisiana, I can tell you that blacks are not being implanted with ICDs at the same rate as their Caucasian counterparts.²⁰

So there is a disparity, and in women as well. We need to eliminate those differences and have, if you would, a check sheet or a thought process of “Did I look at this; did I consider this?” in every person, whether it’s an actual sheet or something in our mind when we see somebody in the clinic and make sure that we’ve covered all the bases. Because I think that it is, as Ileana said, infinitely important to the well being of our patients.

**DR. FERDINAND:** Frank, does that disparity in the use of devices and technology extend to very costly interventions such as left ventricular assist devices (LVADs)?

**DR. SMART:** I don’t know the specific data on LVADs, Keith. I do know that we have looked at ICDs. We have a study that’s currently ongoing to look at cultural bias and race bias in ICD applications.

**DR. FERDINAND:** Okay. Chris, last comments.

**DR. LEGGETT:** Certain educational initiatives have focused on the areas of community education, healthcare professional education, and adherence to guidelines, and specifically on the issue of a gap in access to care, whether it’s ICD therapy, interventional therapy, or cardiac surgery. What we’re seeing is a 30% less likelihood of minorities, blacks and Hispanics, and women of all races, to have access to these technologies. There are many factors that contribute to this disparity, such as physician bias, lack of cultural competence, lack of insurance, poor economics, and decreased access to informed knowledgeable providers with readily available access to life saving therapies and technologies. As clinicians, we must minimize our learned and inherent biases, simplify patient medication regimens, follow evidence-based guidelines, improve our cultural sensitivity and competence, and remember that all men are created equal and therefore deserving of excellent appropriate therapy for their condition irrespective of race, ethnicity, or gender. We must explore all avenues to improve patient compliance, as our best therapies are only as good as our patients, understanding of the personal benefit of the medication or technology to them.

**DR. FERDINAND:** I’d like to thank Dr. Christopher Leggett, Dr. Frank W. Smart, and Dr. Ileana Piña for their very insightful comments as they relate to HF treatment. We didn’t just discuss treatment and identification and increasing outcomes specifically in self-identified black patients, but for all patients. HF morbidity mortality is higher in US blacks. A significant component of this disparity is driven by underutilization of evidence-based medications and devices where evidence regarding positive outcomes is available. Some of it affected by socioeconomic status, some by ability to afford medications, and then even some by mistrust of conventional providers caused by cultural barriers.

I’ve also noted that minorities, along with the elderly and women, have in the past been under-represented in HF trials and we make assumptions of efficacy often based on limited clinical data. A-HeFT gives us an excellent opportunity to apply evidence-based medicine in self-identified blacks already on conventional treatment including ACE inhibitors, ARBs, beta-blockers, aldosterone antagonists, and diuretics if needed for symptoms, with a fixed-dose combination of isosorbide dinitrate and hydralazine. Nevertheless, in order to curtail the epidemic of HF and the disparities in cardiovascular death and disability, it’s going to take the recognition and the early treatment of risk factors such as hypertension, diabetes, dyslipidemia, smoking, physical inactivity, and obesity, because that is going to be the only way we’re going to be able to prevent HF in the future.

I thank all of the faculty for their participation.

Faculty disclosures: Keith Ferdinand, MD discloses that he has received research grants from AstraZeneca, Daiichi Sankyo, and Novartis. He has acted as a consultant to AstraZeneca, Daiichi Sankyo, Novartis, Forest, Roche, and Sanofi Aventis. Christopher J.WB. Leggett, MD, has no relevant financial relationships to disclose. Ileana L. Piña, MD, MP has no relevant financial relationships to disclose. Frank Smart, MD discloses that he has received and honorary from Scios, Inc.

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HF Treatment in Black Patients • Ferdinand


