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Clinical Cardiology [ALERT]

Critical analysis of the latest clinical research in cardiovascular medicine

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ABSTRACT & COMMENTARY

Should We SPRINT to Lower Blood Pressure Targets?

By Michael Crawford, MD, Editor

SYNOPSIS: In a subgroup of the Systolic Blood Pressure Intervention Trial (SPRINT), patients ≥ 75 years of age randomized to intensive hypertension treatment (target < 120 mmHg) or standard therapy (< 140 mmHg), intensive treatment resulted in 33% fewer major adverse cardiovascular events.

SOURCES: Williamson JD, Supiano MA, Applegate WB, et al. Intensive vs standard blood pressure control and cardiovascular disease outcomes in adults aged ≥ 75 years: A randomized clinical trial. *JAMA* 2016;315:2673-2682.

Chobanian AV. SPRINT results in older patients: How low to go? *JAMA* 2016;315:2669-2670.

Since the latest guidelines were released, two new trials have been published that advance the debate over blood pressure targets in hypertension: SPRINT and HOPE. We discussed the initial Systolic Blood Pressure Intervention Trial (SPRINT) data in the November 2015 issue of *Clinical Cardiology Alert* prior to publication of the full paper. These data showed that intensive therapy to a systolic blood pressure (SBP) goal of < 120 mmHg (intensive treatment) vs. < 140 mmHg (standard treatment) resulted in a 25% reduction in major adverse cardiac events (MACE) and death. The most recent publication reports on the prespecified subgroup of those ≥ 75 years of age. A previous trial (HYVET) suggested that a SBP target of < 150 mmHg was adequate in patients ≥ 80 years of age, and this target was recommended in some of the recent guidelines. Subjects enrolled in SPRINT had to be at increased risk for cardiovascular (CV) disease based on a history of CV disease (25%), chronic kidney disease, or a Framingham 10-year risk of CV disease of 15% or more. Exclusions included diabetes, stroke, heart failure, reduced left ventricular systolic function, standing SBP < 110 mmHg and an expected life expectancy of less than three years. SBP had to be between 130-180 mmHg for those on zero to one drug, 130-170 mmHg on two drugs, 130-160 mmHg on three drugs, or 130-150 mmHg on four drugs. Almost 4,000 patients > 75 years old were evaluated for

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trial entry, and 2,636 were randomized to the two treatment groups. The primary CV outcome was a composite of acute myocardial infarction, unstable angina, stroke, heart failure, and death from CV causes. The choice of antihypertensive treatment was up to each investigator at 100 sites. Maximum follow-up duration was six years. The mean SBP was 123 mmHg in the intensive treatment group and 134 mmHg in the standard treatment group. The primary outcome was achieved in 102 of the intensive group (2.6%/year) and in 148 of the standard group (3.9%/year) (hazard ratio [HR], 0.66; 95% confidence interval [CI], 0.51-0.85). At three years, the number needed to treat (NNT) was 27. However, CV death was not statistically significant, with an NNT of 116. More patients in the intensive therapy group with a normal estimated glomerular filtration rate (eGFR) at baseline exhibited a 30% decrease in eGFR to < 60mL/min/1.73m^2 (HR, 3.14; CI, 1.66-6.37). All serious adverse events were higher in the intensive treatment arm, but not statistically significant: hypotension (2.4% vs. 1.4%), syncope (3.0% vs. 2.4%), electrolyte abnormalities (4.0% vs. 2.7%), and acute kidney injury or renal failure (5.5% vs. 4.0%). Researchers noted orthostatic hypotension in about 21% of both groups, but injurious falls were somewhat less common in the intensive group (4.9% vs. 5.5%). The authors concluded that an SBP target of 120 mmHg vs. 140 mmHg resulted in fewer MACE in hypertensive patients ≥ 75 years of age.

■ COMMENTARY

Before applying trial results to your patients, it is important to understand who was in the trial and who wasn't. The exclusion criteria included diabetes, stroke, heart failure, low ejection fraction (< 35%), SBP < 110 mmHg standing, and residence in a nursing home. This is a lot of non-applicable patients. The characteristics of those in the trial also are interesting: 60% had an abnormal eGFR (< 60). Also, all were supposed to have or be at high risk for CV disease, yet only half were on statins and 60% on aspirin. The authors claimed that their patients' characteristics match those of 60% of the general population, based on National Health and Nutrition Examination Survey data. I find this hard to believe.

The hypertension treatment was not predetermined, and each physician enrolling patients in the 100 centers could use whatever drugs he or she wished. The researchers managed to get the target < 140 mmHg SBP patients to a mean of 134 mmHg, but the patients targeted to < 120 mmHg only reached a mean of 123 mmHg for a difference of 11 mmHg. Thus, more than half of the intensive group did not get below 120 mmHg, and the mean difference between groups was less than the overall trial (11 mmHg vs. 15 mmHg). Yet there was a 33% reduction in the primary endpoint, which is amazing. When individual components of the primary composite endpoint are examined, only heart failure and all-cause mortality were significantly reduced. The other CV endpoints, including CV death, were not.

What was the cost of attaining these meager results? There were more adverse effects, as you would expect, but particularly worrisome is the three-fold increase in those who experienced a 30% drop in eGFR to < 60. Even the authors agreed this finding requires a longer follow-up to assess whether this would lead to end-stage renal disease. The authors did not assess cognitive function, a big concern in elderly patients. Also, the authors did not explore the cost of the extra medications and office visits to achieve more aggressive SBP targets. Finally, compliance with aggressive regimens outside the confines of a clinical trial would certainly be an issue. Interestingly, concerns about myocardial infarction and ischemia occurring because of lower diastolic pressures in the aggressive treatment arm were not manifested. About 25% of the subjects had known coronary artery disease, and there was no increase in cardiac events with aggressive therapy to a mean diastolic BP of 62. However, mean diastolic BP in the standard group was 67, so there may not have been enough of a separation to see a difference in cardiac events.

So what are we to do with these results? Clearly, there is a J-shaped curve. Too high an SBP is bad, as is a too low SBP, but where is the sweet spot for each patient?

SPRINT clearly shows that in patients > 75 years of age with a high incidence of chronic kidney disease but no diabetes, a target SBP of < 130 mmHg is reasonable. This is supported by other studies in renal disease patients, where target SBPs of < 135 mmHg have been recommended. However, for patients who don't fit the narrow entry criteria of SPRINT, higher targets in those > 75 years of age may be appropriate, as other studies with broader patient populations have shown. ■

ABSTRACT & COMMENTARY

Is There HOPE for Blood Pressure Targets in Primary Prevention?

By Michael Crawford, MD, Editor

SYNOPSIS: A large randomized trial of fixed-dose antihypertensive treatment in patients at intermediate risk of cardiovascular events with systolic blood pressure <160 mmHg showed no difference in outcomes vs. placebo.

SOURCE: Lonn EM, Bosch J, López-Jaramillo P, et al. Blood-pressure lowering in intermediate-risk persons without cardiovascular disease. *N Engl J Med* 2016;374:2009-2020.

The role of antihypertensive therapy in intermediate-risk patients, defined as 1%/year risk of major cardiovascular (CV) events, with systolic blood pressure (SBP) < 160 mmHg, is unclear. Thus, the Heart Outcome Prevention Evaluation (HOPE)-3 trial was conceived. In 228 centers in 21 countries, men ≥ 55 years of age with at least one CV risk factor or women ≥ 60 years of age with two risk factors were randomized to candesartan 16 mg plus hydrochlorothiazide 12.5 mg a day or placebo. Excluded were patients with known CV disease, symptomatic hypotension, or moderate to advanced renal dysfunction. There also was a rosuvastatin 10 mg vs. placebo arm to the trial that was reported in this same journal issue. The primary efficacy outcomes were CV death, myocardial infarction, or stroke, and the composite of these events plus resuscitated cardiac arrest, heart failure, or revascularization. There were several secondary outcomes and safety outcomes. More than 6,000 patients were randomized to each arm, and the median follow-up was 5.6 years. In the whole population, mean BP was 138/82 mmHg. It decreased in both groups, but to a larger degree in the active treatment group (mean difference -6/3 mmHg). There was no significant difference in either the first or the second primary outcomes (hazard ratio [HR], 0.93; 95% confidence interval [CI], 0.79-1.10; $P = 0.4$, and HR, 0.95; CI, 0.81-1.11; $P = 0.5$). However, in the prespecified subgroup with SBP > 144 mmHg (upper one-third of population), mean 154, who were in the active treatment group, there were significantly fewer primary events (first primary endpoint HR, 0.73; CI, 0.56-0.94, and second HR, 0.76; CI, 0.60-0.96). In the subgroups of the population with baseline SBP < 143 mmHg, there was no significant difference in outcomes between active treatment and placebo. Symptomatic hypotension, dizziness, or lightheadedness was more common in the active treatment group (3.4% vs. 2.0%; $P < 0.001$), but syncope, renal dysfunction, or abnormal potassium levels were not. The authors concluded that fixed-dose dual antihypertensive therapy in subjects at intermediate risk for CV disease did not lower the rate of major CV events, despite lowering mean SBP 6 mmHg to an average of 128 mmHg.

■ COMMENTARY

The subjects enrolled in this trial were at increased risk for CV events (about 10% over 10 years) and worthy of treatment consideration. In fact, the rosuvastatin arm of the study showed a significant reduction in CV events. This study population differed from SPRINT in several ways, but most importantly only 3% had evidence of mild renal dysfunction as compared to 60% in SPRINT. Clearly, these were lower-risk patients. This conclusion is supported by the primary outcome incidence in the two studies — 2.2% for SPRINT and 0.8% for HOPE. Also, SPRINT used more extensive and complicated treatment regimens and observed more adverse events. Neither study included many diabetics (excluded in SPRINT, and 6% in HOPE). In addition, the baseline mean SBP in SPRINT was higher (155 vs. 138 mmHg) and the mean reduction in SBP was more in SPRINT (11 mmHg vs. 6 mmHg). So one could argue that less aggressive therapy in HOPE eliminated the potential benefit of treatment. However, other studies of hypertension treatment have shown that even a 5 mmHg average reduction with therapy can produce significant benefits. Perhaps the baseline SBP is the issue. In the subgroup analysis of HOPE, the upper one-third of SBP patients (> 143 mmHg, mean 154 mmHg) showed a significant decrease in the coprimary outcomes. This mean SBP is similar to that in SPRINT (154 vs. 155 mmHg). Since the lower two-thirds of baseline SBP subjects in HOPE (SBP

< 143 mmHg) did not exhibit a benefit from SBP-lowering therapy, the HOPE results contradict the lower is better hypothesis and support the J curve concept. The HOPE results support treatment for SBP > 140 mmHg, but not lower values in patients with low to moderate CV risk. Thus, it may be that treatment targets depend on CV risk and baseline blood pressure, and that universal SBP targets for all are not the correct approach. Based on HOPE, SPRINT, and other studies, one could conclude that SBP > 160 mmHg should be treated, but in low-risk patients, perhaps < 150 mmHg is adequate; in intermediate-risk patients, < 140 mmHg; and in high-risk patients, < 130 mmHg. This type of approach is similar to our current approach to cholesterol management. ■

ABSTRACT & COMMENTARY

The Double-edged Sword of Public Mortality Reporting

By Jeffrey Zimmet, MD, PhD

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Dr. Zimmet reports no financial relationships relevant to this field of study.

SYNOPSIS: Since the exclusion of cardiogenic shock from public mortality reporting in New York in 2006, rates of intervention in these patients have risen dramatically. However, these rates remain below those in non-reporting states, suggesting continued reluctance to treat the highest-risk patients due to public reporting.

SOURCES: McCabe JM, Waldo SW, Kennedy KF, Yeh RW. Treatment and outcomes of acute myocardial infarction complicated by shock after public reporting policy changes in New York. *JAMA Cardiology*. doi:10.1001/jamacardio.2016.1806.

Bangalore S, Guo Y, Xu J, et al. Rates of invasive management of cardiogenic shock in New York before and after exclusion from public reporting. *JAMA Cardiology*. doi:10.1001/jamacardio.2016.0785.

Today's healthcare environment is marked by efforts to improve quality and increase transparency, especially in regard to invasive procedures. In the early 1990s, New York became the first state to publicly report mortality figures following percutaneous intervention (PCI), and it continues to do so at an individual-operator level. Amid concerns that public reporting could lead PCI operators to avoid the highest-risk patients, the New York State Department of Public Health ultimately chose to exclude patients with cardiogenic shock from its publicly reported analysis of PCI. This began on a trial basis in 2006-2007 and became permanent in 2008. Two recently published reports examined the results of this policy change.

The analysis by McCabe et al identified patients presenting with acute myocardial infarction (MI) and shock from statewide databases between 2002 and the end of 2012. Massachusetts, Michigan, New Jersey, and California data were used as comparators. Across all states, the number of patients receiving PCI for cardiogenic shock increased when comparing the baseline reference period of 2002 through 2005 to the post-policy change period from 2006 through 2012, and in-hospital mortality also decreased. In New York specifically, the authors noted an increase in the use of coronary angiography (63.6% vs. 67.9%; $P < 0.001$), an increase in the use of PCI (30.5% vs. 39.7%; $P < 0.001$), and a decrease in the rate of in-hospital death (47.1% vs. 35.5%). After the reporting policy changes, operators in New York were 28% more likely to perform PCI on patients with acute MI and cardiogenic shock than they were previously (adjusted relative risk, 1.28; 95% confidence interval, 1.19-1.37; $P < 0.001$). By comparison, operators in the comparator states only were 9% more likely to perform PCI in the later era relative to the former. The "difference in differences" also was statistically significant ($P < 0.001$ for interaction). These data suggest that the policy change had the desired effect of reducing avoidance of these high-risk cases, allowing more patients to be offered PCI. However, the data also show that New York began with lower rates of PCI and higher mortality for cardiogenic shock compared to other states. Even after the policy change, New York continued to lag behind other states in terms of rates of revascularization for cardiogenic shock throughout the study period. The authors interpreted this finding as indicative of "continued risk aversion on the part of PCI operators in a public reporting environment."

In a separate analysis, Bangalore et al used propensity score matching to identify cardiogenic shock patients with similar baseline characteristics in New

York and Michigan. The matched cohort contained 1,063 patients in New York and 1,063 patients in Michigan. Events were compared among three time periods: the era prior to censoring of cardiogenic shock from public data (2002-2005), the trial censoring period (2006-2007), and the period after permanent exclusion of cardiogenic shock from reporting in New York (2008-2011). An analysis of New York patients revealed a graded increase in the proportion of patients undergoing PCI, invasive management, and revascularization over the three time periods. However, comparison to the propensity-matched cohort showed that during the same periods, a greater proportion of patients underwent PCI, invasive management, or revascularization in Michigan. Sensitivity analyses comparing New York to other non-reporting states, including California and New Jersey, led to similar results. The authors noted that the relatively strict definition of cardiogenic shock qualifying for exclusion leads to other changes in operator behavior geared toward meeting exclusion criteria. For example, they reported that while overall rates of right heart catheterization in MI patients has decreased over time, the proportion of patients with cardiogenic shock who underwent right heart catheterization (one method to qualify patients for cardiogenic shock data censoring) increased significantly after the exclusion of cardiogenic shock from public reporting. As in the first paper, these authors posited that persistent gaps in intervention between New York and non-reporting states may be due to "continued reluctance to perform interventions on high-risk cases."

■ COMMENTARY

On the face of it, examination of outcomes data from invasive cardiac procedures appears to be a good idea. Clearly, it is within the public interest to identify variability in care and to address deficiencies in poorly performing operators and institutions. The cardiogenic shock example illustrates some basic fallacies in the approach of using mortality as the primary metric to measure quality in PCI when patient presentation has such a dramatic effect on outcomes. For example, contemporary figures for mortality in elective PCI hover around 0.6%, while PCI in ST elevation myocardial infarction carries a mortality risk approaching 5%, and in cardiogenic shock averages more than 40%. Conflating these very different scenarios in the evaluation of a common procedure carries obvious objections. These studies show how profoundly such reporting affects operator behavior, often to the detriment of patient care. Although the 2006 reporting change in New York effected a nearly immediate and positive change in the invasive treatment of patients with cardiogenic shock, rates of intervention still lag behind those in non-public reporting states. The patients who are most at risk and have the most to gain from appropriate intervention ultimately are affected by these policies. ■

ABSTRACT & COMMENTARY

Ventricular Tachycardia Ablation vs. Escalation of Antiarrhythmic Drugs in Ischemic Cardiomyopathy

By Cara Pellegrini, MD

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Dr. Pellegrini reports no financial relationships relevant to this field of study.

SYNOPSIS: VANISH, a multicenter, randomized, controlled trial, demonstrated a significant reduction in composite of death, ventricular tachycardia storm, or appropriate implantable cardioverter defibrillator shock in ischemic cardiomyopathy patients with ventricular arrhythmias on antiarrhythmic drug who underwent ablation as compared to those who were treated with escalation of antiarrhythmic drug therapy.

SOURCE: Sapp JL, Wells GA, Parkash R, et al. Ventricular tachycardia ablation versus escalation of antiarrhythmic drugs. *N Engl J Med* 2016;375:111-121.

Although an implantable cardioverter defibrillator (ICD) can prevent sudden cardiac death in patients with an ischemic cardiomyopathy and ventricular arrhythmias, recurrent arrhythmias and ensuing ICD shocks are unpleasant and potentially harmful. Many patients also are placed on antiarrhythmic medications to suppress arrhythmias. Nonetheless, ventricular tachycardia (VT) recurs frequently. Whether VT ablation or escalation of antiarrhythmic drug therapy is the most appropriate management in this situation has not been clear, prompting the Tachycardia Ablation versus Escalated

Antiarrhythmic Drug Therapy in Ischemic Heart Disease (VANISH) trial.

Sapp et al performed a multicenter, randomized, controlled trial of patients with a history of myocardial infarction, ICD placement, and an episode of VT during treatment with amiodarone or another antiarrhythmic medication within the previous six months. Patients randomized to ablation continued on the same antiarrhythmic drug regimen. Those assigned to drug escalation received amiodarone if previously treated with another agent, high-dose amiodarone if they had been receiving a dose < 300 mg, or amiodarone plus mexiletine if their arrhythmia had recurred despite at least 300 mg of amiodarone daily. VT ablation approach and ICD settings were standardized. The primary outcome was a composite of death at any time after randomization, VT storm occurring after a 30-day blanking period, and an appropriate ICD shock occurring after a 30-day blanking period.

Researchers enrolled 259 patients, and mean follow-up was about 28 months. Significantly fewer patients in the ablation group experienced the primary outcome, 68.5% vs. 59.1%. There was no significant difference between groups in mortality (the study was not powered to detect a difference in mortality). The other two components of the composite outcome trended toward significance individually and drove the overall distinction. Notably, the planned subgroup analysis of the two-thirds of the study group who were on, and had failed, amiodarone prior to ablation showed a marked distinction between the patients (a 45% reduction in endpoints) vs. the group who were previously on another agent, mostly sotalol (no significant difference between treatment groups). Adverse events were present in both groups, with the ablation arm experiencing two cardiac perforations and three cases of major bleeding, and the drug escalation group experiencing three deaths thought to have been potentially related to antiarrhythmic drug use. The authors concluded that among patients with an ischemic cardiomyopathy and an ICD, catheter ablation was more effective than escalated antiarrhythmic drug therapy in reducing the rate of death at any time or VT storm or ICD shocks after a 30-day blanking period.

■ COMMENTARY

This study's findings are noteworthy for several reasons. The management of these patients is critical, as they are truly a high-risk group. More than half the patients continued to experience VT, and more than one-quarter died during the just over two-year follow-up period. There was a relatively low number needed to treat of 11 patients to prevent one death/VT storm/ICD shock. The findings of this study are consistent with those of previous related trials, such as VTACH and SMASH-VT, and add to the growing body of evidence supporting an increased role for VT ablation in the management of patients with ischemic, scar-based VT.

In addition, this study's interpretation has numerous limitations, first and foremost generalizability of the results. Although the study involved multinational cooperation (with researchers from Canada, Europe, the United States, and Australia), all but nine of the 259 patients were enrolled at Canadian centers. Access to an electrophysiologist highly skilled at VT ablation may be limited for many patients. This study was restricted to patients who already had experienced VT despite antiarrhythmic medication and does not address the potential role of VT ablation as a primary prevention, something that would reach an even larger population. Even in the ablation arm, more than half the group still reached the primary endpoint, leaving clinicians seeking more effective treatments for these patients.

It is interesting to ponder why those who failed to achieve VT control with amiodarone derived more relative benefit from ablation. Although not explicitly discussed in the paper, it appears that those who were already on amiodarone had an extremely low probability of event-free survival (i.e., mexiletine didn't add much), rather than a magical greater effect of ablation in this group. This is perhaps the clearest take-home point of this study: If amiodarone has failed, it is time for a new strategy. ■

ABSTRACT & COMMENTARY

Screening for Coronary Artery Disease Is Underused in Heart Failure

By Van Selby, MD

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Dr. Selby reports no financial relationships relevant to this field of study.

SYNOPSIS: In a large retrospective cohort of patients hospitalized for new-onset heart failure, the majority did not receive testing for ischemic heart disease.

SOURCES: Doshi D, Ben-Yehuda O, Bonafede M, et al. Underutilization of coronary artery disease testing among patients hospitalized with new-onset heart failure. *J Am Coll Cardiol* 2016;68:450-458.

Coronary artery disease (CAD) is the most common cause of heart failure (HF). Current practice guidelines recommend screening for CAD in patients with newly diagnosed HF. However, few studies have evaluated how often clinicians perform diagnostic testing for CAD on patients hospitalized with newly diagnosed HF.

Doshi et al analyzed a large commercial administrative claims database, supplemented by Medicare data. They evaluated the frequency of diagnostic testing for CAD, both during the index hospitalization for new-onset HF and within 90 days of hospitalization. Between 2010 and 2013, the authors identified 67,691 patients.

Overall, 17.5% of patients underwent any testing for ischemic CAD during the index hospitalization, and 27% were evaluated for CAD within 90 days. The most common evaluation method was stress testing, followed by coronary angiography. In a multivariable analysis, predictors of undergoing noninvasive testing for CAD included baseline CAD (odds ratio [OR], 1.25; $P < 0.001$), hypertension, hyperlipidemia, and reduced ejection fraction. Patients who were > 70 years of age and those with prior stroke, peripheral arterial disease, prior arrhythmia, renal disease, or a prior workup for CAD were less likely to receive noninvasive testing for CAD.

Only 2% and 4.3% of patients underwent coronary revascularization during the index hospitalization and at 90 days, respectively. Baseline CAD (OR, 9.27; $P < 0.001$), male sex, diabetes, and smoking all were associated with greater odds of coronary revascularization, and percutaneous coronary intervention was used more commonly than coronary artery bypass grafting (CABG). The authors concluded that diagnostic testing for ischemic CAD is underutilized significantly among patients hospitalized for new-onset HF.

■ COMMENTARY

Not every patient hospitalized for new HF requires evaluation for CAD within 90 days, and the exact percentage of patients who should be evaluated is unknown. However, considering CAD is the most common cause of HF and present in more than half of patients with HF, the rate of CAD testing reported in this study (27% of all patients were evaluated within 90 days) is surprisingly low and suggests, as the authors concluded, that diagnostic testing for CAD is underutilized significantly in this population. Part of the explanation for low use of ischemic evaluation in new HF may be a perceived lack of evidence showing clear benefit of revascularization in patients with HF and CAD. Perhaps because of this lack of data, the most recent HF guidelines from the American College of Cardiology/American Heart Association only make a class IIa recommendation (meaning it is reasonable) to evaluate for CAD in the diagnostic evaluation of new HF. The recent publication of 10-year follow-up from STICHES, showing a mortality benefit associated with coronary artery bypass grafting in patients with CAD and systolic HF, may strengthen the argument for CAD testing. Regardless of the benefits associated with revascularization, identification of CAD also may guide medical therapy of a given patient.

When evaluating findings of large retrospective cohort studies, it is important to acknowledge limitations. Analyses of insurance claims databases depend on accurate coding, and it is possible that diagnostic testing for CAD was not always coded properly. Furthermore, complete clinical information is not available for these patients — some may have had a contraindication to diagnostic testing or other explanation for why testing was not performed. Despite these limitations, given the size of the cohort and the magnitude of the findings, it appears the majority of eligible HF patients are not screened for CAD.

Identifying which HF patients will have underlying CAD is difficult based on clinical history of risk factors alone. Many will not have angina or other clear ischemic symptoms. Once CAD is identified, it may be difficult to determine whether it is the cause of a given patient's HF; however, this should not deter clinicians from testing. The authors suggested an increasingly cost-conscious medical environment, eager to minimize unnecessary diagnostic testing, is responsible for the low rates of CAD evaluation. By their estimate, clinicians miss 325,000 cases of CAD in patients with congestive HF every year due to underutilization of diagnostic testing. Whatever the reason, the rates of CAD testing reported in this study would strike most cardiologists as inappropriately low, and suggest those of us who evaluate and treat HF should consider screening for CAD more frequently in patients with newly diagnosed HF. ■

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CME/CE QUESTIONS

1. **Recurrent ventricular tachycardia despite antiarrhythmic therapy in ischemic cardiomyopathy patients is best treated with:**
   a. increasing amiodarone to at least 400 mg/day.
   b. the addition of another antiarrhythmic drug.
   c. ventricular tachycardia ablation.
   d. placement of an antitachycardia pacemaker.

2. **Public reporting of percutaneous intervention (PCI) mortality by operators can lead to:**
   a. fewer physicians performing PCI.
   b. fewer high-risk patients receiving PCI.
   c. improved outcomes in shock patients.
   d. improved physician morale.

3. **What was the most common characteristic of patients > 75 years of age enrolled in SPRINT?**
   a. Known cardiac disease
   b. Diabetes
   c. Reduced left ventricular function
   d. Chronic kidney disease

4. **In a large insurance survey study, approximately what proportion of newly diagnosed heart failure patients were evaluated for coronary artery disease within 90 days?**
   a. 25%
   b. 50%
   c. 75%
   d. 95%

5. **The HOPE-3 study of intermediate cardiovascular risk patients supported a systolic blood pressure treatment goal of:**
   a. < 120 mmHg.
   b. < 130 mmHg.
   c. < 140 mmHg.
   d. < 150 mmHg.