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Clinical Cardiology [ALERT]

Critical analysis of the latest clinical research in cardiovascular medicine

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ABSTRACT & COMMENTARY

Who Really Needs Intensive Blood Pressure Control?

By Michael H. Crawford, MD, Editor

SYNOPSIS: A patient baseline characteristics level analysis of the SPRINT and ACCORD trials resulted in the creation of a simple algorithm for identifying high-risk patients who experienced fewer major cardiac events without increased serious adverse events from intensive blood pressure therapy.

SOURCE: Wang S, Khera R, Das SR, et al. Usefulness of a simple algorithm to identify hypertensive patients who benefit from intensive blood pressure lowering. *Am J Cardiol* 2018;122:248-254.

Recent large randomized trials have driven a move toward more aggressive blood pressure (BP) control. However, the downsides to such an approach have been downplayed. Investigators from Dallas sought to develop an algorithm that would inform physicians about which patients were most likely to benefit from intensive BP lowering using patient-level data from two large randomized trials: SPRINT and ACCORD. The authors of both trials compared intensive treatment (systolic BP < 120 mmHg) to standard treatment (SBP < 140 mmHg) but enrolled different patient populations. In both trials, investigators enrolled about 10,000 patients, all diabetic in ACCORD and all nondiabetic in SPRINT. All available patient characteristics were included in developing the risk prediction model. The primary outcome was major adverse cardiovascular events (MACE).

In SPRINT, a subset of patients with high MACE was used to develop a decision tree that was tested on the remaining lower MACE risk patients (n = 8,357) and in 2,258 ACCORD patients in the standard glycemic control group. A decision tree model using three variables (age > 74 years, urinary albumin to creatinine ratio > 34, and history of clinical cardiovascular disease) identified 49% of SPRINT patients and 55% of ACCORD patients considered high risk for MACE. Intensive BP lowering reduced MACE in these high-risk patients in SPRINT (hazard ratio [HR], 0.66; 95% confidence interval [CI], 0.52-0.85) and ACCORD (HR, 0.67; 95% CI, 0.50-0.90), but not in the remaining lower-risk patients (SPRINT: HR, 0.83; 95% CI, 0.56-1.25; ACCORD: HR, 1.09; 95% CI, 0.64-1.83). Importantly, intensive BP therapy in the high-risk group did not increase the risk of serious adverse events.

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The authors concluded that this simple three-factor risk prediction model identified high-risk patients with systolic hypertension in whom the benefits of intensive therapy outweighed the risks.

■ COMMENTARY

One of the criticisms of the SPRINT study was the increased incidence of renal insufficiency and orthostatic symptoms in the intensive treatment arm. There was legitimate concern that older patients with stiff blood vessels would experience more harm than benefit. Also, the overall results of ACCORD suggested that there was no difference in MACE between the standard and intensive arms of the study. Thus, some clinicians ignored SPRINT and the new guidelines it spawned.

The hypothesis of the Wang et al study was that perhaps there is a high-risk group among the SPRINT and ACCORD patients who would benefit from more aggressive targets. The investigators analyzed the myriad clinical data in both trials and discovered a simple decision tree model that identified a high-risk group that benefited from intensive therapy in both trials. Also, intensive therapy did not increase the risk of serious adverse events in this same high-risk group.

The differences in four-year MACE were impressive. In SPRINT, 9.5% of the high-risk group experienced a MACE compared to 2.9% of lower-risk patients. In ACCORD, MACE was 11.5% in the high-risk group and 4.3% in the rest. The number needed to treat (NNT) in high-risk SPRINT patients was 39 and 29 in high-risk ACCORD patients. In the lower-risk SPRINT patients, the NNT was 244 but was not calculable for lower-risk ACCORD patients since the HR was > 1.0. The degree of BP-lowering in the high- and lower-risk patients in both trials was equivalent, so a treatment effect difference does not explain the results. Serious adverse events were not higher in the high-risk groups but were among lower-risk patients (HR, 1.16; 95% CI, 1.03-1.3).

This risk predictor is unique because it targets patients with hypertension, not the general public. SPRINT and ACCORD patients already were a higher-risk group among hypertension patients. Despite this, further risk stratification was possible. Also, the new algorithm does not require extensive data. The only unusual aspect of the algorithm is the urine albumin to creatinine ratio, which clinicians usually do not obtain from hypertensive patients without chronic kidney disease. However, it is an inexpensive, easy-to-conduct test.

The major limitation to this study is that it was a retrospective analysis of two trials that only included higher-risk patients. Thus, we do not know if the new algorithm would perform as well in low-risk hypertensive patients. Also, only simple clinical and laboratory data were included in the trial databases. No sophisticated cardiovascular imaging or stress testing was available consistently; consequently, those were not included. It is possible that these more sophisticated and expensive tests would further stratify patients.

At this juncture, I agree with the authors. Despite the limitations of this study, this new algorithm and other insights potentially could inform hypertension management decisions. At the least, the concept that among hypertensive patients there are higher-risk patients who should have lower BP targets seems established. Exactly how to identify these higher-risk patients is evolving. ■

ABSTRACT & COMMENTARY

MitraClip Falls Short in First Randomized Trial Specific to Functional Mitral Regurgitation

By Jeffrey Zimmet, MD, PhD

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

SYNOPSIS: The first randomized trial of MitraClip for secondary mitral regurgitation showed no benefit over medical therapy.

SOURCE: Obadia JF, Messika-Zeitoun D, Leurent G, et al. Percutaneous repair or medical treatment for secondary mitral regurgitation. *N Engl J Med* 2018; Aug 27. doi: 10.1056/NEJMoa1805374. [Epub ahead of print].

In 2013, the FDA approved the MitraClip device for the treatment of significant *primary* (also known as degenerative) mitral regurgitation (MR) in patients at high surgical risk. The approval was based in large part on the results of the 2011 EVEREST II trial. The authors of that trial randomized 279 MR patients 2:1 to percutaneous repair or to open surgery. Approximately 75% of the patients in this trial had primary MR, with secondary MR patients making up the remainder. Despite the relative lack of data on this population, most MitraClip procedures performed outside the United States involve cases of secondary MR. Subsequent prospective registries have suggested that these patients can benefit in terms of symptoms and functional capacity, but no dedicated randomized trials have been available until now.

The authors of the MITRA-FR trial randomized 307 patients with severe secondary MR to either MitraClip or to medical therapy. The primary outcome measure was a composite of all-cause mortality and unplanned heart failure hospitalization at 12 months. These were very sick patients; the trial inclusion criteria called for patients with left ventricular ejection fraction between 15% and 40%. A significant number fell into the lower end of that range.

Of the 152 patients assigned to receive the procedure, 14 did not experience a successful implant, either because the procedure was never attempted (eight patients) or because the procedure failed (six patients). Although the technique was effective in most cases in reducing MR in the short term (95% of patients in the MitraClip group showed an MR reduction of at least one grade. Seventy-five percent of those patients exhibited an MR grade of ≤ 1+ at the time of discharge after the procedure), the hard outcomes were disappointing. The composite primary outcome occurred in 83 patients in the intervention group and in 78 patients in the control group (odds ratio, 1.16; 95% confidence interval [CI], 0.73-1.84; $P = 0.53$). Death occurred in 24% of patients in the intervention group and in 22% of patients in the medical control group. The results were not different when viewed in a per-protocol analysis (according to whether patients actually received the device) vs. the intention-to-treat analysis.

Unfortunately, a substantial amount of follow-up data at one year were missing regarding echo, functional status, and quality of life outcomes (follow-up was 99% complete for the primary outcome). Regardless, analysis of the available data did not show significant improvements in New York Heart Association class, six-minute walk test, or global quality of life scores in the intervention group. Regarding procedural safety, 21 out of 144 patients in the intervention group experienced periprocedural complications. These included implant failure (4.2%), vascular complications or hemorrhage (3.5%), cardiogenic shock (2.8%), embolism and stroke (2.8%), and tamponade (1.4%).

The authors concluded that percutaneous mitral valve repair with MitraClip did not significantly alter rates of death and heart failure hospitalization at one year over medical therapy alone.

■ COMMENTARY

By definition, in secondary MR, the mitral leaflets and chordae are structurally normal. In such cases, MR occurs because of left ventricular and annular dilatation and associated functional abnormalities. It is worth repeating that these patients' MR occurred because of a poor ventricle. Although severe, MR is associated with worse outcomes in these patients. Rectifying the MR does not change the underlying disease. In the MITRA-FR trial, improvements in MR following percutaneous mitral valve repair did not translate to improved hard outcomes. In fact, one

could argue that because of the non-negligible rate of serious periprocedural complications, patients in the intervention group experienced net clinical harm from the procedure.

Close examination of the data from this trial leaves open the possibility that certain subgroups of patients may benefit. For example, the echo data indicate that the average patient in this trial exhibited truly severe left ventricular dilatation. One could argue many of these patients were beyond the point where a procedural intervention could be expected to produce benefits. The authors of the highly anticipated Cardiovascular Outcomes Assessment of the MitraClip Percutaneous Therapy for Heart Failure Patients With Functional Mitral Regurgitation (COAPT) trial, whose results are expected in the near future, chose to exclude patients with very low ejection fraction or very severe dilatation. Thus, the possibility of a different outcome remains.

In the meantime, the results of MITRA-FR demonstrate that patients with advanced heart failure and secondary MR will not benefit from the MitraClip procedure. It remains to be seen whether this will change procedural indications outside the United States and if the results of forthcoming trials will further alter this landscape. ■

ABSTRACT & COMMENTARY

Tafamidis Improves Survival in Transthyretin Cardiac Amyloidosis

By Van Selby, MD

Assistant Professor of Medicine, University of California, San Francisco Cardiology Division, Advanced Heart Failure Section

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

SYNOPSIS: Treatment with tafamidis in transthyretin amyloid cardiomyopathy patients produced reductions in all-cause mortality and cardiovascular hospitalizations. This was the first medical therapy to demonstrate improved survival for patients with this condition.

SOURCE: Maurer MS, Schwartz JH, Gundapaneni B, et al. Tafamidis treatment for patients with transthyretin amyloid cardiomyopathy. *N Engl J Med* 2018;379:1007-1016.

Transthyretin amyloid cardiomyopathy (ATTR-CM) is an under-recognized condition caused by myocardial deposition of amyloid fibrils composed of transthyretin. Currently, there are no approved therapies to treat ATTR-CM. Tafamidis is a transthyretin stabilizer that reduces the formation of pathologic amyloid fibrils in patients with ATTR-CM.

The authors of the Transthyretin Amyloidosis Cardiomyopathy Clinical Trial (ATTR-ACT) randomized 441 patients with confirmed ATTR-CM to tafamidis (20 mg or 80 mg daily) vs. placebo. The median age was 75 years. Most patients were male. Patients with both wild type (76%) and mutant (24%) ATTR-CM were enrolled. Investigators followed patients for 30 months. The primary outcome was all-cause mortality or cardiovascular hospitalization.

Tafamidis was associated with lower all-cause mortality compared to placebo (29.5% vs. 42.9%; hazard ratio, 0.7; 95% confidence interval [CI], 0.51-0.96) as well as a lower rate of cardiovascular hospitalization (relative risk ratio, 0.68; 95% CI, 0.56-0.81). The survival curves began to diverge at approximately 12-18 months, suggesting the drug takes time before demonstrating a favorable impact on outcomes. Tafamidis also was associated with significantly lower rates of decline in quality of life ($P < 0.001$) and six-minute walk distance ($P < 0.001$). Generally, tafamidis was well tolerated, with no significant differences in adverse events or discontinuation between the two groups.

The authors concluded treating patients with heart failure due to ATTR-CM with tafamidis reduces all-cause mortality and cardiovascular hospitalizations vs. placebo.

■ COMMENTARY

Generally, ATTR-CM affects patients > 60 years of age and is far more common in men. Amyloid fibrils form because of misfolded monomers or oligomers of the protein transthyretin. Transthyretin misfolding can occur either because of a mutation in the transthyretin gene (an autosomal dominant condition) or wild-type transthyretin that becomes unstable and misfolds (formerly referred to as senile amyloidosis). Amyloid fibrils deposit in the heart, leading to heart

failure and arrhythmias. Tafamidis acts by binding to circulating transthyretin and stabilizing it in the tetramer form, preventing breakdown into pathologic, amyloid-forming monomers.

Long considered a rare disease, recent studies have demonstrated that ATTR-CM is much more common than previously believed, especially among older patients with heart failure and preserved ejection fraction. Patients often present with left ventricular thickening in the absence of significant hypertension, sometimes accompanied by low QRS voltage on ECG. A history of bilateral carpal tunnel syndrome, neuropathy, or autonomic dysfunction also should raise suspicion. Diagnosing ATTR-CM previously required tissue confirmation, usually through endomyocardial biopsy. However, technetium-labeled bone scintigraphy (such as technetium pyrophosphate scan) now allows providers to make a diagnosis of ATTR-CM without tissue biopsy in many cases.

Traditionally, treatment has been supportive, with no medical therapy shown to alter the underlying course of the disease. Without effective therapy, the prognosis generally was poor, with an average survival of approximately three years from diagnosis. Therefore, the results of ATTR-ACT mark a new era in the management of ATTR-CM, demonstrating impressive improvements in survival, hospitalizations, and quality of life. In a prespecified subgroup analysis, the benefit of tafamidis was not observed among patients with New York Heart Association (NYHA) functional class III heart failure. In fact, the rate of cardiovascular-related hospitalization among this subgroup was higher with tafamidis compared to placebo. Generally, subgroup analyses should be interpreted with caution. There is little reason to suspect tafamidis would worsen patients with NYHA class III symptoms. Maurer et al speculated that hospitalizations increased because of longer survival during a period with severe disease. In any case, this finding underscores the importance of early diagnosis of ATTR-CM, as the data suggest treatment is most efficacious when initiated before patients become markedly symptomatic.

The FDA has granted fast-track and breakthrough therapy designations for tafamidis, which could lead to the drug's final approval in the coming months. Meanwhile, if patients are referred to a local amyloidosis specialty center, they may be able to obtain access to tafamidis through an open-label extension study that the drug's manufacturer will offer. There are many more drugs in development for treatment of ATTR-CM, using several approaches to reduce amyloid deposition in the heart. In the coming years, we will see multiple options for this not-so-uncommon disease. It is more important than ever that providers maintain a high index of suspicion and know the red flag signs and symptoms. ■

ABSTRACT & COMMENTARY

Clinical Context and Outcomes in Stress Cardiomyopathy

By Michael H. Crawford, MD, Editor

SYNOPSIS: An analysis of the International Takotsubo Registry showed that long-term mortality is similar to that observed in patients with acute coronary syndromes but varies widely depending on the triggering event. Emotional trigger patients receive the best prognosis, and acute neurological events receive the worst prognosis. Investigators recommended categorizing stress cardiomyopathies by the triggering event rather than wall motion distribution.

SOURCES: Ghadri JR, Kato K, Cammann VL, et al. Long-term prognosis of patients with Takotsubo syndrome. *J Am Coll Cardiol* 2018;72:874-882.

Sharkey SW, Maron BJ. Survival after Takotsubo, revisited. *J Am Coll Cardiol* 2018;72:883-884.

Although initially viewed as a relatively benign condition, it is becoming clear that stress cardiomyopathy (SCM) often resembles acute coronary syndromes (ACS) clinically. Investigators from the International Takotsubo Registry (InterTAK) sought to determine the relationship between the triggering event and outcome in SCM. The InterTAK database includes patients from 25 hospitals in nine countries in Europe and the United States who were recruited between 2011 and 2014. These patients were compared to an age- and sex-matched cohort of ACS patients in the Zurich ACS Registry. The SCM patients were categorized by their triggering event into four groups: emotional stress; physical activities, medical illness, or procedures; neurologic disorders; or no identifiable trigger. The primary endpoint was

mortality. There were 455 patients in the SCM group and 455 in the ACS group, including 233 with ST-segment elevation myocardial infarction (MI), and 222 with non-ST-elevation MI. Long-term mortality was similar between SCM patients and ACS patients ($P = 0.49$).

To assess the effects of triggers on outcomes, 1,613 patients with SCM with clear differentiation of the triggers, including no trigger, were selected from the InterTAK database. There were 485 patients in the emotional trigger group, 532 with a physical trigger, 98 with a neurologic disorder, and 498 with no trigger. Patients with emotional triggers experienced better outcomes than ACS patients. Patients with physical or neurological triggers received a worse prognosis compared to ACS patients. Physical stress (hazard ratio [HR], 3.78; 95% confidence interval [CI], 2.21-6.44), neurologic disease (HR, 5.76; 95% CI, 2.96-11.2), and no trigger (HR, 2.14; 95% CI, 1.2-3.82) were potent predictors of five-year mortality compared to emotional trigger patients. The authors concluded that, overall, SCM patients demonstrated long-term outcomes similar to ACS patients, but certain triggering conditions support a benign vs. life-threatening course.

■ COMMENTARY

In the 1990s, reports appeared describing a unique basal left ventricular (LV) cardiomyopathy with apical sparing that was seen in some cases of intracerebral hemorrhage. This is the opposite of the more common apical wall motion abnormalities in coronary artery disease. Also, the LV areas involved did not conform to the usual coronary artery territories. In the early 2000s, reports appeared, mainly from Japan, of an apical ballooning cardiomyopathy with clinical features suggestive of acute MI but with normal coronary arteries. Because of the LV angiographic appearance that resembled an octopus-trapping jar, Japanese researchers called it Takotsubo syndrome. In many of these early reports, the syndrome seemed to be precipitated by emotional stress and was observed more often in women. In the last decade, a mid-LV wall version of Takotsubo was described by Mayo Clinic physicians. Now, it is clear that all these conditions are manifestations of some emotional or physical stress and are more properly called SCM, even though the particular stress is not always easily discernible.

Instead of using the wall motion abnormality pattern, Ghadri et al proposed classifying SCM based on the initiating stress. They identified three basic stress categories: I-emotional, II-physical (which is divided into two subcategories, IIa physical activity, medical illness, or postprocedural, and IIb, which is neurologic disease), and III-no identified stress. Their argument for this classification is their data showed different mortality outcomes for these three categories. Type II is divided because neurologic disease patients receive a worse prognosis than other physical stress SCM patients. Ghadri et al believe this categorization based on prognosis makes more clinical sense than one based on where the wall motion abnormalities are.

The data supporting this categorization from the InterTAK Registry are not surprising since physical trauma, such as surgery, and serious diseases, such as subarachnoid hemorrhage, affect prognosis regardless of the presence or absence of SCM. Clearly layering a cardiomyopathy on top of these conditions will only increase mortality. Interestingly, even when no trigger is found, the prognosis is worse than if there was a clear emotional trigger. Ghadri et al also confirmed what others have shown: Long-term mortality for SCM is equal to that of ACS (20% at five years for both), but varies by stressor. The five-year mortality for SCM in the registry was 30% for physical trauma, 15% for no triggering event, and 9% for emotional stress. Thus, there seems to be two drivers of mortality: the cardiomyopathy and the triggering illness. Cardiomyopathy usually improves or normalizes, especially in the emotional stress category. When it does not, mortality increases. Also, when SCM is so profound as to cause cardiogenic shock, mortality naturally increases. ■

ABSTRACT & COMMENTARY

MRI for Patients With Pacemakers and Defibrillators

By Joshua D. Moss, MD

Associate Professor of Clinical Medicine, Cardiac Electrophysiology, Division of Cardiology, University of California, San Francisco

Dr. Moss reports he is a consultant for Biosense Webster and Abbott.

SYNOPSIS: Performing an MRI on patients with pacemakers and defibrillators that were not MRI-conditional was not associated with any clinically significant changes in device parameters.

SOURCE: Shah AD, Morris MA, Hirsh DS, et al. Magnetic resonance imaging safety in nonconditional pacemaker and defibrillator recipients: A meta-analysis and systematic review. *Heart Rhythm* 2018;15:1001-1008.

The major cardiac implantable electronic device (CIED) companies have engineered and tested pacemaker and implantable cardioverter-defibrillator (ICD) pulse generator and lead systems with demonstrated safety in MRI scanners, classified as MRI-conditional devices. However, performing an MRI on a patient with a nonconditional device remains relatively contraindicated over concerns regarding lead malfunction, battery depletion, device damage, and inappropriate shock. Scanning of patients with nonconditional pacemakers and defibrillators has been limited to tertiary academic centers that follow specific MRI protocols. Although studies have shown these protocols to be safe, without significant adverse events, performing an MRI with a nonconditional CIED is not covered by the Centers for Medicare & Medicaid Services (CMS) due to safety concerns.

Shah et al sought to further characterize the clinical risks associated with performing an MRI on nonconditional CIED recipients by conducting a meta-analysis of 70 observational studies. More than 5,000 patients with nonconditional devices who underwent MRI were included (3,692 patients with a pacemaker, 1,440 patients with an ICD). Roughly 10% of all patients were reported to be pacemaker-dependent. The brain and cervical spine were imaged most frequently. Most MRI scanners used a ≤ 1.5 Tesla magnet. In all but six observational studies, one of several periprocedural MRI protocols was followed, including the American Heart Association 2007 protocol and the John Hopkins MRI protocol. In older studies, various therapies simply were disabled. Notably, there were several scenarios included that often are avoided, even with MRI-conditional devices. These included scans of 100 abandoned leads, a small number of externalized leads connected to pulse generators taped to the chest wall, very recent implants, and thoracic isocenter imaging.

Statistically significant changes in lead parameters, such as sensing and pacing impedance, were observed, but none were clinically significant. There also was a statistically significant but clinically insignificant acute decrease in battery voltage of 0.002 ± 0.001 V. There were three lead failures, none of which were clearly directly attributable to the MRI study. There was one inappropriate ICD shock, which occurred after an MRI was performed without knowledge that the patient had an implantable defibrillator. Electrical reset to factory settings was observed in 1.6% of scans, but only in devices that were implanted before 2006. Symptoms possibly attributable to MRI-induced torque, current induction, or lead heating were reported in 0.3% of scans. There was an increased risk of a safety event when a > 1.5 Tesla MRI magnet was used, driven by power or device resets. The authors concluded that clinical events after MRI of nonconditional CIEDs are rare, and that such scans can be performed safely in appropriately screened patients.

■ COMMENTARY

Although MRI-conditional CIEDs are increasingly common in clinical practice, nonconditional devices still are encountered frequently in both inpatient and outpatient settings. It is estimated that a large number of these patients will warrant an MRI in their lifetime. Concerns about performing an MRI on patients with nonconditional devices arise from both observed and theoretical interactions between the device and the surrounding magnetic field emitted by the MRI. The dynamic nature of MRI-induced magnetic fields can result in heating of the myocardium via the implanted lead, inappropriate sensing of ambient signals leading to inhibition of pacing or inappropriate defibrillation, and hardware resets or damage within the pulse generator. As a result, guideline and regulatory statements have recommended against MRI scans in those with a nonconditional CIED, even when an MRI may be the imaging modality of choice. Several protocols have been developed to optimize safety, including the American Heart Association 2007 protocol and the Johns Hopkins MRI protocol. The authors of large studies have tested these protocols and found a low rate of clinical events during and after MRI. Still, scanning nonconditional devices has not been covered by CMS or adopted widely.

This well-conducted meta-analysis adds to the growing body of evidence that performing an MRI on patients with nonconditional CIEDs is safe and only rarely associated with adverse clinical events. It is important to note that most patients who underwent MRI were managed with a specific MRI protocol, including periprocedural device interrogation and reprogramming, cardiac telemetry or plethysmography monitoring during the scan, and verbal contact during the entirety of the scan. An electrophysiologist or CIED-trained nurse or technician also was available immediately. Patients with unique situations, including abandoned leads, epicardial leads, and very recently implanted devices (< 24 hours), also were included without any significant events. However,

they were not well represented, and safety results should not necessarily be extrapolated to these unique scenarios.

In our own practice, patients with nonconditional devices are scanned according to an evidence-based protocol that includes pre- and post-MRI device interrogation with reprogramming *(Learn more at: https://bit.ly/2p1OZRQ.)* Careful screening of these patients remains critical, as data on the overall safety of scanning patients with abandoned leads, externalized leads, and very recently implanted devices remain limited. ■

CME/CE QUESTIONS

1. **Use of the mitral valve clip to reduce regurgitation secondary to left ventricular disease resulted in:**
   a. reduced mortality.
   b. less regurgitation.
   c. fewer heart failure admissions.
   d. None of the above

2. **Although rare, MRI scanning of patients with nonconditional implanted electrophysiological devices can lead to:**
   a. lead malfunction.
   b. battery depletion.
   c. inappropriate shock.
   d. All of the above

3. **Which of the following features characterize transthyretin amyloid cardiomyopathy?**
   a. Women affected more often
   b. Age > 80 years
   c. Heart failure with preserved left ventricular ejection fraction
   d. Left ventricular hypertrophy on ECG

4. **Compared to acute coronary syndromes, stress cardiomyopathy (Takotsubo syndrome) long-term mortality is:**
   a. lower.
   b. the same.
   c. higher.
   d. rare.

5. **Systolic blood pressure targets should be lower in patients with:**
   a. known cardiovascular disease.
   b. age > 74 years.
   c. urinary albumin to creatinine ratio > 34.
   d. All of the above

CME/CE OBJECTIVES

Upon completion of this educational activity, participants should be able to:

- discuss the most current information related to cardiac illness and the treatment of cardiac disease;
- explain the advantages and disadvantages, as well as possible complications, of interventions to treat cardiac illness;
- discuss the advantages, disadvantages, and cost-effectiveness of new and traditional diagnostic tests in the treatment of cardiac illness; and
- discuss current data regarding outpatient care of cardiac patients.