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Critical analysis of the latest clinical research in cardiovascular medicine

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Angiotensin Receptor Blockade, Renal Function, and Outcomes in Chronic Heart Failure

By Van Selby, MD

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

SOURCE: Kiernan MS, et al. Early and late effects of high- versus low-dose angiotensin receptor blockade on renal function and outcomes in patients with chronic heart failure. *JACC Heart Failure* 2015;3:214-223.

Renin-angiotensin-aldosterone (RAAS) blockade is an important component of guideline-recommended therapy for heart failure with reduced ejection fraction (HFrEF). ACE inhibitors (ACEIs) and angiotensin receptor blockers (ARBs) often cause a reduction in the glomerular filtration rate (GFR). The relationship between ACEI or ARB dose and changes in renal function and the long-term implications of these changes is not well-described. To address this issue, Kiernan and colleagues performed a secondary analysis of the Heart Failure Endpoint evaluation of Angiotensin II Antagonist Losartan (HEAAL) study. HEAAL randomized 3834 patients with HFrEF to either 150 mg or 50 mg losartan daily. Patients with serum creatinine > 2.49 mg/dL, potassium > 5.7 mmol/L, or renal artery stenosis were excluded.

Compared to the 50 mg dose, patients receiving 150 mg losartan had a greater reduction in GFR over time (mean difference -3.79 mL/min/1.73 m^2, $P < 0.0001$). The difference was driven by early worsening renal function (WRF), defined as an increase in creatinine > 0.3 mg/dL. After the first 4 months of therapy, there was no significant difference in GFR between the two doses ($P = 0.016$). WRF in the first 4 months was not associated with an increased risk of death

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or hospitalization for heart failure (HF) (HR [hazard ratio], 1.09, P = 0.20). Overall, losartan 150 mg was associated with reduced risk of death or hospitalization for HF (HR, 0.85, P < 0.0001). Among patients with chronic kidney disease (CKD) at baseline, there was no significant difference in change in GFR between the two doses. The authors concluded that compared with 50 mg losartan, 150 mg losartan is associated with an increased risk of early WRF, but this appears to be a benign event. Losartan 150 mg daily retains its net clinical benefit and is associated with reduced risk of death or hospitalization for HF in patients with HFrEF.

■ COMMENTARY

It is well known that initiation of ACEI or ARBs can cause a rise in serum creatinine, and WRF is a common reason for ACEI/ARB discontinuation or dose reduction. Although WRF, in general, has been associated with adverse outcomes, the clinical significance of this early WRF, following ACE or ARB initiation, is less clear. This is among the first studies to examine the dose effect of ACEI or ARB therapy on renal function in patients with HFrEF, as well as the association with long-term outcomes.

The acute rise in serum creatinine following ARB initiation is likely related to alterations in hemodynamics, related to the role angiotensin II plays in regulating renal blood flow. Longer-term declines in renal function, on the other hand, generally reflect disease progression, and not surprisingly are associated with worse outcomes. Thus, when patients with HFrEF develop WRF, it is crucial the clinician carefully evaluate the cause of the decline before adjusting the patient's medication. Current ACC/AHA guidelines recommend an angiotensin receptor blocker for patients with HFrEF who cannot tolerate ACE inhibitors. For losartan, the target dose is 150 mg daily, and given the clear long-term benefit associated with this higher dose, it is important to consider whether dose modification or discontinuation is truly necessary when patients develop WRF.

The findings among patients with baseline CKD are important to note. In the HEAAL study, patients with baseline mild-to-moderate CKD saw smaller average changes in GFR following ARB initiation compared to patients without baseline CKD. Prior studies have shown that CKD patients are less likely to receive target doses of RAAS inhibitors compared to patients with HFrEF and normal renal function, likely related to concern for WRF. These findings support the same target dose of 150 mg in patients with mild-to-moderate CKD (remember that patients with serum creatinine > 2.49 mg/dL were excluded from HEAAL). Of course it is critical that patients be monitored closely for worsening renal failure or hyperkalemia after ACEI or ARB initiation.

This was a retrospective, secondary analysis with important limitations. Study investigators were not blind to changes in renal function, and it is possible that treatments were modified in response to changes in serum creatinine or potassium. The study also was not powered to detect differences in clinical events according to presence of WRF. The authors could not determine what was considered a "safe" increase in creatinine following ARB initiation, and what degree of early WRF should prompt drug discontinuation.

Despite these limitations, the findings suggest the early decline in GFR associated with initiation of high-dose losartan does not increase adverse outcomes, and the net clinical benefit favors targeting the 150 mg dose in patients with HFrEF. It is important to consider the cause of WRF in all HF patients, and strive to maintain the guideline-recommended dose of ACEI or ARBs whenever possible, rather than discontinuing or reducing the dose as an automatic reaction to a rise in serum creatinine. ■

Is it Time to Give Up on Systemic Cooling in ST Segment Elevation MI?

By Jeffrey Zimmet, MD, PhD

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

SOURCE: Nichol G, et al. Prospective, multicenter, randomized, controlled pilot trial of peritoneal hypothermia in patients with ST-segment-elevation myocardial infarction. *Circ Cardiovasc Interv* 2015;8(3). pii: e001965. doi: 10.1161/CIRCINTERVENTIONS.114.001965. Epub 2015 Feb 19.

Systemic hypothermia as a means to protect against tissue damage is a strategy that has found its way into clinical practice guidelines in the post-cardiac arrest population, in which cooling has demonstrated a neuroprotective effect in multiple contemporary clinical trials. Countless animal models have suggested a similar profound myocardial protective effect in acute myocardial infarction (MI). This explains the surfeit of interest in hypothermia for ST elevation MI. External cooling is both slow and uncomfortable; thus, opening the door for other innovative methods for achieving this end.

The VELOCITY trial used a novel automated peritoneal lavage system to induce rapid hypothermia in ST-elevation MI patients undergoing primary percutaneous intervention. Fifty-four patients with STEMI presenting within 6 hours of symptom onset were randomized to control or to peritoneal cooling before and for 3 hours after cardiac catheterization. Infarct size was assessed by cardiac MRI between 3 and 5 days post-infarct. Safety, as assessed at 30 days, was a composite of death, reinfarction, ischemia-driven target vessel revascularization, major bleeding, sepsis, pneumonia, peritonitis, severe arrhythmia, or renal failure. Hypothermia was successfully achieved in more than 96% of patients, in most of them prior to first balloon inflation. Unsurprisingly, this was achieved at the expense of a small but significant increase in door-to-balloon times — median door-to-balloon times were 62 [51-81] minutes in the hypothermia group and 47 [37-55] minutes in the control group ($P = 0.007$). Infarct size was not reduced significantly in the peritoneal lavage group. On the contrary, the primary safety endpoint occurred in six of the hypothermia patients and in none of the controls. Of these six events, three were definite stent thrombosis. The authors also reported one case each of cardiac death, major bleeding, ventricular arrhythmia, and sepsis — all in the hypothermia group.

The authors concluded that, although hypothermia by the peritoneal lavage system was feasible and effective, it resulted in an increase in treatment times and safety events, without a measured benefit in infarct size.

■ COMMENTARY

Controlled hypothermia in STEMI patients has had a rather long and storied history. More than 10 years ago, the COOL-MI trial reported on a larger population (n = 392) of similar patients randomized to control treatment or to automated endovascular cooling via the femoral vein, in addition to primary PCI. In that trial, as in this one, hypothermia resulted in no significant difference in infarct size. In COOL-MI, however, the non-prespecified anterior MI group showed an apparent decrease in infarct size, keeping the dream of hypothermia in STEMI alive. Despite the addition of an invasive procedure in the setting of primary PCI and systemic anticoagulation, there was no price paid in terms of major adverse cardiac events. Similar results were found in the ICE-IT trial of the same era. More recently, the CHILL-MI trial (published in early 2014) used a different endovascular cooling system to achieve rapid and safe hypothermia in STEMI patients, but again, without demonstrating a decrease in infarct size for the entire cohort (the subset of early anterior infarctions again suggested a small benefit).

The addition of invasive whole-body cooling to a time-sensitive procedure such as primary PCI is challenging indeed, and the investigators are to be commended for their work. The search for interventions to reduce infarct size and improve outcomes in ST-segment-elevation MI has yielded multiple promising candidates. This includes interventions as varied as ischemic post-conditioning and remote ischemic conditioning, as well as simple measures such as pre-reperfusion beta blockade and adenosine infusion. Most of these are less cumbersome and time consuming

than systemic hypothermia and do not carry the necessary penalty in terms of time to reperfusion. In the case of controlled peritoneal hypothermia, not only was the sought-after reduction in infarct size not realized, but there was a safety penalty as well. Although the investigators suggest the need for an additional larger-scale, appropriately powered trial, I cannot help but wonder whether we should close the book on systemic hypothermia in acute MI. ■

Are Atrial Premature Complexes Benign?

By Michael H. Crawford, MD

SOURCE: Murakoshi N, Xu D, et al. Prognostic impact of supraventricular premature complexes in community-based health checkups: The Ibaraki Prefectural Health Study. *Eur Heart J* 2015;36:170-178.

Atrial premature complexes (APCs) are commonly observed on routine ECGs and believed to be harbingers of atrial fibrillation, especially in patients with cardiovascular disease. However, little is known about the long-term prognosis of APCs in the general population. Thus, these investigators from Japan analyzed the database of a large community-based cohort from 1993 to 2008 to determine the risks of APCs seen on the subjects' baseline ECGs. There were 63,197 subjects without heart disease or atrial fibrillation (AF) who were followed for at least 1 year (20,492 men and 42,705 women, mean age 58 years at baseline). The primary endpoint was mortality and the secondary endpoint was AF. The mean follow-up was 14 years, but if censured by AF occurrence on the yearly follow-up exam, it was 6 years. In addition to analyzing the raw data, the data were adjusted for age and other potential confounders such as blood pressure, body mass index, alcohol use, and other ECG findings. Also, a propensity-matched analysis was done matching subjects with APCs to those without.

Results: APCs were observed in 6%, and these subjects were more likely to be older and have other risk factors for AF and mortality. APCs were significantly associated with death from stroke, cardiovascular death, and all-cause mortality in women, but only cardiovascular death in men. AF occurred in 1 per 1000 person years, and APCs were a significant predictor of AF (hazard ratio [HR], 4.87 men and 3.87 women). In the propensity score-matched subjects, APCs were significantly associated with AF and cardiovascular death in all subjects and stroke death in women, but not all-cause mortality. The authors concluded that in a general population free of AF or cardiovascular disease, the presence of APCs on a routine ECG is associated with AF and cardiovascular death.

■ COMMENTARY

This study affirms what has been seen in smaller studies of higher-risk patients, that APCs predict future AF. Why APCs would predict cardiovascular death in a general population is not clear from this study. It could be simply that by being associated with AF, you are more likely to have a stroke or develop heart failure. On the other hand, APCs may be markers of underlying cardiovascular disease. This makes sense since in the baseline data, APC subjects were older and had more risk factors for cardiovascular disease. That APCs are strong predictors of AF is not surprising. Pathophysiology studies show that APCs originating in the pulmonary vein orifices can trigger AF. Also, when the number of APCs per ECG was evaluated, more APCs increased the risk of AF. This is remarkable given that we are talking about a routine ECG, approximately 15 seconds of monitoring. Since APCs are frequently seen on ambulatory ECG monitoring done for a variety of reasons, one wonders if there is some threshold for APCs 24 hours above which the risk of AF and cardiovascular disease increases significantly in a general population.

In addition to the limitations of an ECG as a monitoring device for APCs, it was also the way AF was confirmed. Thus, asymptomatic intermitten AF was unlikely to be detected. Also, the subjects were not extensively evaluated for cardiovascular disease on the yearly exams, so some subclinical disease may have been present and unaccounted for in the propensity analysis. In addition, there were twice as many women as men in the study. The authors don't offer an explanation for this, but since subjects with known cardiovascular disease were excluded in this older middle-aged population, many men may have been excluded. The main clinical message of this study is that patients with APCs on a routine ECG should undergo screening for heart disease and asymptomatic intermitten AF. ■

SIMPLE Is Better – ICD Defibrillation Testing Does Not Improve Outcomes

By Cara N. Pellegrini, MD

Assistant Professor of Medicine, UCSF, Cardiology Division, Electrophysiology Section, San Francisco VA Medical Center

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

SOURCE: Healey JS, et al. Cardioverter defibrillator implantation without induction of ventricular fibrillation: A single-blind, non-inferiority, randomized, controlled trial (SIMPLE). *Lancet* 2015;385:785-791.

Traditionally, the placement of an implantable cardioverter-defibrillator (ICD) concludes with induction of ventricular fibrillation (VF) to confirm that the new device is able to appropriately sense the arrhythmia and terminate it while there is still the opportunity to reposition the ventricular lead or add an additional lead if necessary. This defibrillation testing has never been shown to independently decrease mortality or improve outcomes. A growing number of implanting physicians are moving away from performing defibrillation testing routinely due to greater confidence in the reliability of modern-day, high-energy devices and increasing concern about the potential deleterious effects of even brief VF and/or the high-current density of the defibrillation itself.

The multinational SIMPLE study, led by Drs. Healey and Connolly at McMaster University in Ontario, Canada, examined whether abstaining from defibrillation testing at initial device implant was noninferior to testing, with regard to success of first appropriate shock and occurrence of arrhythmic death. They enrolled 2500 patients into this randomized, controlled trial, with broadly defined inclusion criteria. Over a mean follow-up of 3.1 years, 90 patients who did not undergo defibrillation testing had experienced a failed appropriate shock or arrhythmic death, as compared to 104 patients in the testing group, a nonsignificant difference (hazard ratio [HR], 0.86; confidence interval [CI], 0.65–1.14, P non-inferiority < 0.001). Safety endpoints and overall mortality were similar between groups, with no deaths in this study directly attributable to defibrillation testing. Notably, about 1% of all patients having defibrillation testing needed chest compressions or an unplanned intubation, compared to 0.1% in the no-testing group (P = 0.007). The authors concluded that there was no clear benefit to routine defibrillation testing with regard to shock efficacy or arrhythmic death prevention, and that complications from testing appeared to be rare.

■ COMMENTARY

This well-conducted study has two main messages. First, not testing should probably be the default. They made a special effort to minimize exclusion criteria and make their study very generalizable. A bit more than one-third of their patients were receiving an ICD for secondary prevention, 30% had NYHA class III heart failure symptoms, and almost 30% received a resynchronization device (biventricular defibrillator). These patients are among those who are most likely to have higher defibrillation thresholds, making testing theoretically more important, and the lack of difference between groups despite their meaningful inclusion is notable. As those getting a resynchronization device have the distinction of receiving disease-modifying therapy with the potential for improvement of their ventricular function, and also being at higher risk for heart failure exacerbation with defibrillation testing in subgroup analysis, this group should be specifically spared from defibrillation testing where possible. Some benefits of not testing are not even accounted for in this study, in which presumably both groups underwent similar anesthesia protocols to achieve patient blinding. By not performing defibrillation testing, the risks and costs of deeper levels of sedation can be eliminated.

The second important lesson is that, should testing be judged appropriate for a certain patient, it can probably be performed with minimal additional risk. Patients receiving right-sided devices — where the vector of defibrillation may be less favorable — putting them at higher risk for a failed shock, were explicitly excluded from this study (although they did constitute about 1% of each arm). Patients with subcutaneous ICDs were also excluded. Data are sparse for those with hypertrophic cardiomyopathy, channelopathies, or sarcoidosis; congenital heart disease patients also may be expected to have higher defibrillation thresholds and were not studied.

Finally, those with a recalled lead or starting a new antiarrhythmic medication that could increase defibrillation threshold may merit testing. Indeed, individualized risk-benefit calculus is appropriate here.

Not addressed by this study is the role of other potentially safer methods of assessing the integrity of the ICD system, such as upper limit of vulnerability testing. This technique utilizes the relationship between the energy required for VF induction and that required for VF termination to determine the presence of an adequate safety margin, with VF being induced in only the minority of procedures. Where device testing is believed to be necessary, this practice may further lower risk. For the majority of patients, though, we now have data that are at least just as good to keep it SIMPLE. ■

Cardiac Sarcoidosis: Rare or Underdiagnosed?

By Michael H. Crawford, MD

SOURCE: Kandolin R, et al. Cardiac sarcoidosis: Epidemiology, characteristics, and outcome over 25 years in a nationwide study. *Circulation* 2015;131:624-632.

There is increasing evidence that sarcoidosis can be confined to the heart, but disagreements abound about the diagnosis and treatment of cardiac sarcoidosis (CS). Thus, these investigators examined all the cases of histologically confirmed CS in Finland over the past 25 years to gain insights into this issue. The myocardial inflammatory diseases in Finland study group focuses on CS and giant cell myocarditis and collects data on such patients from 17 Finnish hospitals. This report includes cases identified from 1988 on; after 2008, the cases were collected prospectively and outcomes were determined up to 2013. Patients entered had histologic evidence of sarcoidosis on endomyocardial biopsy (55) or an extracardiac source plus clinical evidence compatible with CS, including 18F-FDG PET, gadolinium MRI, or echocardiography. Among the 110 patients identified, most were diagnosed after 2003 and 102 were diagnosed before transplantation or autopsy. The prevalence rate in 2012 was 2.2 per one million persons. Isolated CS was found in 65% of the patients. They were more likely to be females with a low left ventricular ejection fraction (EF) and late gadolinium enhancement (LGE) on MRI. Almost all the patients were treated with steroids, and many were treated for heart failure and arrhythmias, including ICD placement. Survival rate free of transplantation was 97% at 5 years and 93% at 10 years. Survival was highly correlated with low EF, heart failure, and isolated CS, all confirming worse prognosis. Steroid therapy appeared to improve EF on follow-up if the EF was < 35% initially, but not in those with EFs between 35% and 49%. The authors concluded that CS detection has increased markedly over the past two decades, and that the prognosis for treated patients is better than anticipated.

■ COMMENTARY

This large and well-done database study of CS in Finland confirms that it is a rare disease (2.2 per 1 million people). Since the overall incidence of sarcoidosis in Finns is similar to other white European populations, this estimate of CS prevalence would probably be similar in other white populations. Also, the marked increase in detection of CS over the past decade is not surprising, since it corresponds to the deployment of LGE-MRI and 18F-FDG PET in the diagnostic evaluation of suspected sarcoid. In addition, the fact that 58% had a reduced EF and that this and the presence of heart failure were predictive of survival is no surprise. What is interesting is that in 65%, sarcoid was clinically confined to the heart. These patients had no mediastinal involvement on chest X-ray or clinical evidence of other organ involvement. However, by PET, 71% had mediastinal lymph node involvement and 41% had other organ involvement. Also of interest was the higher than expected survival of these patients over 10 years. Since almost all of them received steroids and many got other immunosuppressive drugs, it is difficult to say if this treatment was the reason. In addition, almost all received beta-blockers and renin angiotensin system blockers. ICDs were implanted in 54%, and 25% received a pacemaker alone for AV conduction block. Only 11 patients had cardiac transplantation and four of them died during the study period. So either this is a relatively benign disease or modern therapy is highly effective.

CS should be suspected in young to middle-aged

patients who present with complete heart block, ventricular tachyarrhythmias, or sudden cardiac arrest and heart failure. CS can mimic coronary artery disease and arrhythmogenic right ventricular cardiomyopathy. Often there is basal septal thinning on echocardiography or an apical aneurysm. Cardiac MRI should be done in suspected cases. It can show acute inflammatory edema and LGE due to fibrosis. MRI has a high specificity for CS, but is not as sensitive as PET for detecting it. Also, PET can detect other organ involvement. Some believe the ideal approach is to do both of these imaging tests. ■

Athlete's Heart vs Brugada ECG

By Michael H. Crawford, MD

SOURCE: Zorzi A, et al. Differential diagnosis between early repolarization of athlete's heart and coved-type Brugada electrocardiogram. *Am J Cardiol* 2015;115:529-532.

The normal variant ECG seen predominantly in highly trained athletes of domed ST segment elevation, and negative T waves in V1-V3 can be confused with some of the ECG patterns in Brugada syndrome patients. Clearly this distinction is of importance. Thus, these investigators from Italy compared the ECGs from 61 healthy endurance athletes who exhibited this normal variant of early repolarization with domed ST elevation and a negative T wave to 92 age- and sex-matched patients with Brugada syndrome and the type 1 ECG pattern of ≥ 2 mm of coved ST elevation, followed by a negative T wave in at least two contiguous leads between V1-V3. None of the subjects in either group had demonstrated structural heart disease. All were in sinus rhythm and were not taking drugs that affect the ECGs. All of the athletes had an uneventful, average 4-year follow-up. All the ECGs were digitally analyzed by two observers, with disagreements adjudicated by a third observer.

Results: Although the heart rate was significantly slower in the athletes (56 vs 73 bpm, $P < 0.001$) paradoxically, the PR interval was longer in the Brugada patients (181 vs 160 ms athletes, $P = 0.04$) and the QRS duration was longer (112 vs 91 ms athletes, $P = 0.01$). As expected, more athletes met criteria for left ventricular hypertrophy (Sokolow-Lyon index 36 vs 29 Brugada, $P < 0.001$). The QTc interval was normal and similar in both groups. Athletes had a lower ST segment height above the baseline at the J point (1.5 mm vs 3.3 mm Brugada $P < 0.001$) and a lower ST J point/ST at 80 ms after the J point ratio (0.8 vs 1.6 Brugada, $P < 0.001$). Only 3% of athletes had a ST J/ST80 ratio of > 1.0, whereas 100% of the Brugada patients did. Receiver operating curve analysis showed that an STJ/ST80 > 1.0 had a sensitivity of 97%, a specificity of 100%, and a diagnostic accuracy of 99% for detecting Brugada syndrome patients. The authors concluded that the STJ/ST 80 ratio is highly accurate for distinguishing the athlete's heart from Brugada syndrome on the resting ECG and could be used to reduce the number of athletes who undergo further evaluation or are unnecessarily disqualified from competition.

■ COMMENTARY

Now that the European Society of Cardiology has recommended resting 12-lead ECGs be done in all athletes before competitive sports participation (not endorsed by ACC or AHA), we are seeing more athletes with "ECG abnormalities" referred to cardiologists in the United States and elsewhere. Early repolarization (J point elevation > 1 mm) in leads V1-V3 is seen in a majority of endurance athletes. However, in the typical pattern, J point and ST elevation are followed by a peaked and upright T wave. One series showed that 9% of highly trained athletes have a pattern with upward ST doming and T wave inversion that can resemble the type I Brugada pattern. It is stated that this pattern is more common in athletes of Afro-Caribbean origin, but the racial classification of the athletes in this study is not reported.

This large study with two selected groups validates the use of the STJ/ST80 ratio, according to the authors. In their multivariate analysis, STJ/ST80 ratio was superior to all the other ECG findings for distinguishing the two conditions. Of course, the selection of 61 athletes with this ECG pattern and 92 well-evaluated Brugada patients is a strength and weakness of the study. Selection bias always inflates the value of any discriminator between two groups. Only large all comers type studies in athletes will confirm the utility of the STJ/ST80 ratio. At this point, the authors suggest that an athlete with no family history of sudden collapse or death and a STJ/ST80 ratio < 1.0 does not need further tests for Brugada syndrome.

QUESTIONS & COMMENTS:
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CME QUESTIONS

1. In a general population, APCs are associated with which of the following?

A. Atrial fibrillation

B. Stroke death

C. Cardiovascular death

D. A & C

2. The ECG parameter with the highest accuracy for diagnosing Brugada syndrome in athletes is:

A. J point elevation >1 mm in V1-V3.

B. criteria for left ventricular hypertrophy absent.

C. ST segment height at the J point/ST at 80ms later ratio >1.0 in V1-V3.

D. PR interval > 170 ms.

3. Which of the following is correct concerning cardiac sarcoidosis?

A. It is rare

B. It is clinically isolated in 2/3 cases

C. The prognosis if treated is very good

D. All of the above

4. Which of the following is correct concerning VF induction to test a new ICD in the lab?

A. It predicts ICD efficacy

B. It predicts arrhythmic death despite the device

C. It has a high complication rate

D. None of the above

5. Which of the following is correct concerning worsening renal function after initializing high-dose angiotensin receptor blocker therapy for heart failure?

A. It usually self corrects within 4 months

B. It predicts early mortality

C. It predicts early rehospitalization

D. It usually worsens over time

6. Cooling by peritoneal lavage in acute STEMI patients undergoing primary PCI vs controls results in:

A. smaller MI size.

B. longer door-to-balloon time.

C. fewer early complications.

D. All of the above

CME 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.