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Clinical Cardiology [ALERT]

Critical analysis of the latest clinical research in cardiovascular medicine

ABSTRACT & COMMENTARY

# Management of Severe Ischemic Mitral Regurgitation

*By Michael H. Crawford, MD, Editor*

SOURCE: Acker MA, et al. Mitral-valve repair versus replacement for severe ischemic mitral regurgitation. *N Engl J Med* 2014;370:23-32.

Practice guidelines recommend mitral valve repair or replacement for severe ischemic mitral regurgitation (MR) that is causing symptoms refractory to best available medical therapy. However, few data exist to aid the selection of repair vs replacement. Thus, these investigators from the Cardiothoracic Surgical Trials Network conducted a randomized, multicenter trial of repair vs replacement in 22 centers involving 251 patients eligible for surgical treatment of their severe MR with or without coronary artery bypass grafting (CABG). Each surgeon decided on the best prosthetic valve or annuloplasty ring for the patient, but all the replacements had chordal sparing. The primary endpoint was the left ventricular end-systolic volume index (LVESVI) by echocardiography at 12 months. Secondary endpoints included mortality, major adverse cardiac or cerebral events, serious adverse events, recurrent MR, rehospitalization, and quality of life.

Concomitant CABG was performed in 75%, and 11 of 126 patients in the repair group (9%) were converted to valve replacement. The ESVI at 12 months had decreased 7 mL/m$^2$ in both groups. Mortality was 14% in the repair group and 18% in the replacement group (hazard ratio, 0.79; 95% confidence interval, 0.42-1.47; $P$ = 0.45). Recurrence of moderate-to-severe MR at 12 months was 33% in the repair group and 2% in the replacement group ($P$ < 0.001). Major and serious adverse events, functional status, and quality of life did not differ between the two groups. The authors concluded that there was no significant

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difference in LV function, mortality, or other clinical outcomes in patients who underwent repair vs replacement for severe symptomatic ischemia MR, but replacement resulted in a more durable result.

## ■ COMMENTARY

The management of severe symptomatic ischemic MR is a challenge. Most of these patients have low LVEFs, which are known to increase the risk of surgery, yet rarely does maximal medical therapy for LV dysfunction improve the MR and there is no specific medical therapy for MR. Although successful mitral valve surgery would eliminate MR, it is unclear whether the benefit would outweigh the risks of surgery. Consequently, the decision to operate for ischemic MR is usually in the context of CABG surgery. It has been said that severe MR with unrevascularized coronary artery disease is good news, since there is a good chance revascularization will improve or at least maintain LV function. In this study, 75% had concomitant CABG.

Based on the excellent results repairing mitral valves with degenerative disease, the majority of surgeons select mitral valve repair over replacement for ischemic MR. This practice is supported by observational studies that show superior results for repair. However, these observational studies require adjustments for baseline differences in repair vs replacement patients. One major difference is that repair patients have tended to be younger. Thus, a randomized trial was certainly in order and the results of this trial are contradictory to the observational studies. In addition to better matched groups with a randomized design, there are probably other differences that help explain the results. The 30-day mortality was lower in this trial (2% repair, 5% replacement) than in the Society of Thoracic Surgeon's database (5%, 9%). One reason for lower mortality in this trial may have been the exclusive use of a chordal sparing operation for replacement and the use of rigid or semi-rigid complete annuloplasty rings in all cases.

One fear in MR surgery cases is that once you close the low impedance leak to the left atrium, the ischemia weakened LV may be unable to handle the increased afterload and the EF will fall postoperatively. This was not the case in this study where LVEF remained almost the same postoperatively at around 40% in both groups. Although the 30-day mortality was higher with valve replacement, in the ensuing 11 months the death rate was the same in the two groups. Also, the most common causes of death were multiorgan failure (38%), heart failure (13%), and renal failure (10%), suggesting that the underlying ischemic cardiomyopathy played a big role in survival.

The primary endpoint of the study was LVESVI by echocardiography, which is an indirect marker of clinical outcomes. However, the authors point out that using mortality as an endpoint would have required 4000 patients and taken years to complete enrollment. A larger study that included a CABG alone arm would have been interesting. The investigators plan to follow the patients for at least another year, which may shed more light on the durability of repairs. As it is, at 1 year one-third of the repair group had moderate-to-severe MR. Given the poor durability of repair vs replacement and no other differences in outcomes, it would seem that surgeons should give more consideration to valve replacement for ischemic MR. ■

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ABSTRACT & COMMENTARY

# Mechanical Chest Compressions in CPR

*By Michael H. Crawford, MD, Editor*

SOURCE: Rubertsson S, et al. Mechanical chest compressions and simultaneous defibrillation vs conventional cardiopulmonary resuscitation in out-of-hospital cardiac arrest: The LINC randomized trial. *JAMA* 2014;311:53-61.

Effective cardiopulmonary resuscitation (CPR) is partly dependent on the adequacy of manual chest compressions, but they are limited by interruptions and less than ideal conditions such as during transport. Mechanical chest compression devices have been developed that improve organ perfusion vs manual compressions in experimental studies, but there is little evidence of their clinical effectiveness and safety compared to manual compressions. Thus, these investigators from Sweden, the Netherlands, and the United Kingdom conducted a randomized trial to compare whether CPR using a mechanical chest compression device (LUCAS) resulted in superior 4-hour survival in patients with out-of-hospital cardiac arrest as compared to standard CPR with manual chest compressions. All patients in whom CPR was performed by six emergency medical systems in three countries between January 2008 to August 2012 were entered. Exclusion criteria were traumatic cardiac arrest, known pregnancy, age < 18 years, or body size inappropriate for the device (too large or small). Several secondary outcomes were evaluated, including survival with good neurologic outcome at 6 months.

A total of 4998 cases were screened and 2593 were enrolled. Informed consent was done after successful resuscitation and four patients withdrew consent, leaving 2589 study subjects. The first defibrillation was delivered 1.5 minutes later and there were more defibrillations in the mechanical compression group, but otherwise the groups were well matched. The primary outcome of survival to 4 hours was not different between the two groups (both 24%). Nor was there a significant difference in any of the secondary outcomes. There were 23 device-related adverse events among 1282 deployments of the device and eight of these required discontinuing use of the device. There were seven serious adverse events with the device vs three with manual compressions; these included pneumothorax and flail chest. The authors concluded that there was no significant difference in 4-hour post out-of-hospital cardiac arrest survival between those in whom a mechanical chest compression device was used vs manual compressions during CPR.

## ■ COMMENTARY

This seems to be the glass half full or empty parable. One could look at this study and conclude that these mechanical chest compression devices are not worth buying or could say that they are just as good as the manual way, so what could an emergency medical technician (EMT) do to benefit the arrest victim if he/she is not pushing on the chest. Also, they produce consistent excellent chest compressions with a compression fraction of 0.84 vs 0.78 for manual. So, this might allow highly skilled but smaller people with less arm strength to be EMTs. One of my colleagues was urging us to buy these devices because he didn't want a small relatively weak resident doing chest compressions on him when he collapses during rounds (he is a large man). In addition, these devices seem safe; serious complications from the device were unusual and not statistically different from manual compressions. Also, device malfunction was rare (< 1%). There were issues with very large and small people with device fit, but overall it fit 95% of people. This could easily be corrected by having different sized devices or other technical improvements.

Given all these pluses, why didn't use of this device improve outcomes? There are several plausible reasons. First, the device group's CPR protocol was different to try to take advantage of the device's strengths. Chest compressions were done in 3-minute intervals rather than 2 minutes with manual compressions to take advantage of the lack of fatigue with the device. Second, the first defibrillation shock was given without stopping compressions in the device group, again to take advantage of the automatic chest compressions without human contact with the victim during the shocks. Third, since compressions were not stopped for the first shock, it was given as quickly as possible without efforts to determine the patient's rhythm. Fourth, the first shock was delivered 1.5 minutes later in the device group due to the time needed to employ the device. Finally, since this study was done in the field by EMS personal, it is likely that all the EMTs were excellent at chest compressions, which may not be the case in less selective environments. Which of these potential explanations for the failure to show better outcomes

with the device is the most important is difficult to determine. However, I believe the device has promise and is at least as good as manual compressions. Further work to capitalize on the advantages of mechanical chest compressions seems warranted. If your CPR results aren't what you want them to be, perhaps looking into deploying a mechanical chest compression device makes sense. ■

ABSTRACT & COMMENTARY

# Prognosis of Ventricular Fibrillation in Acute Myocardial Infarction

*By Michael H. Crawford, MD, Editor*

SOURCE: Bougouin W, et al. Incidence of sudden cardiac death after ventricular fibrillation complicating acute myocardial infarction: A 5-year cause-of-death analysis of the FAST-MI 2005 registry. *Eur Heart J* 2014;35:116-122.

At this time, ventricular fibrillation (VF) early after acute myocardial infarction (MI) is not an indication alone for an implantable cardioverter-defibrillator (ICD) therapy. However, there is concern that despite the efficacy of mechanical and pharmacological therapy for acute MI, the risk of subsequent sudden cardiac death (SCD) in patients with VF complicating acute MI may be higher and the guidelines should be revisited. Thus, these investigators from the French registry on Acute ST-elevation and non-ST elevation Myocardial Infarction (FAST-MI) registry enrolled 3670 patients with acute MI in October 2005 and reported the 5-year follow-up data from this study population. Enrolled patients had to present within 48 hours of symptom onset and meet the international definition of acute MI. Excluded were iatrogenic MIs (e.g., postsurgical). The primary endpoints were deaths, classified as sudden cardiac death (SCD), non-SCD, and non-cardiac deaths, and related to early (< 48 hours after admission) or late (prior to hospital discharge) VF. A variety of clinical and demographic features were used in a multivariate model.

The incidence of in-hospital VF was 3.2% with 79% being early. VF patients were younger and more often smokers compared to the rest of the MI population. Beta-blocker therapy did not differ according to VF occurrence. Only anterior MI location was associated with VF occurrence. Interestingly, the presence of atrial fibrillation on the first ECG was associated with VF by multivariate analysis (hazard ratio [HR], 2.5; 95% confidence interval [CI], 1.4-4.4; $P = 0.003$). In- hospital mortality was higher in the VF group (25% vs 5%; $P < 0.001$) with an adjusted HR of 7.38 (95% CI, 4.27-12.75; $P < 0.001$). Also, mortality was higher with early vs late VF (33% vs 23%; $P < 0.001$). In-hospital death in the VF group was mainly from arrhythmias (82%) whereas cardiogenic shock was the most common cause in non-VF patients (62%). The overall survival at 5 years was 74% and was not associated with the occurrence of in-hospital VF on multivariate analysis. Also, the incidence of SCD was not more frequent in the VF group (13% in both groups at 5 years) despite a very low rate of ICD placement (1.2% overall). The authors concluded that the development of VF in the acute phase of MI was associated with a higher in-hospital mortality, but not long-term mortality or SCD.

## ■ COMMENTARY

It seems that the indications for ICD placement just keep expanding, so it is interesting to see a slight reversal in this trend. The VF group in this study was different from the rest of the MI patients in that they had lower LVEFs and were more likely to be on ACE/ARB and amiodarone. Also, they were more likely to have an ICD (3.4% vs 0.2%, $P < 0.001$) but the overall ICD rate in this study was low (about 1%). Despite these differences, neither the raw or adjusted HRs reached significance for a worse prognosis in the VF patients who survived the initial MI and could be followed long-term. Prior studies employing ICDs early (< 40 days) after MI, so-called primary prevention, showed no benefit. Now this study, which could be viewed as an observational study of secondary prevention of SCD, has shown that even in patients with VF early post MI, no benefit is likely to be obtained from ICD placement. This result is in agreement with the current guidelines and prior smaller studies.

The results also support our practice of ECG monitoring of all patients after an acute MI, since those who developed VF had a higher in-hospital mortality. However, it leaves the duration of

such monitoring unclear. This is an issue since the deployment of primary PCI has decreased hospital stays for acute MI patients who are treated promptly and successfully. Bucking this trend may be difficult in the current hospital cost-containment era, and perhaps we should be giving more thought to placing a defibrillator vest on higher-risk patients being discharged early. My current practice is to keep all acute MIs in the hospital on ECG telemetry monitoring for a minimum of 48 hours. Those deemed at higher risk, such as patients with LVEF < 35% who may meet ICD criteria later (> 40 days), I am sending out with a defibrillator vest. However, this is a fast-moving field and further guidelines are sure to be emerging.

This study has limitations. It is an observational registry study, but the numbers of subjects are large, permitting robust statistical adjustments. It is a multicenter study and no control was exercised on the protocols used at each hospital. Also, categorizing cause of death can be challenging, but standard definitions were used and carefully adjudicated. Thus, for this type of study, it was well conducted. ■

ABSTRACT & COMMENTARY

# Are Implanted Defibrillator Therapies Deactivated at End of Life?

***By Edward P. Gerstenfeld, MD***

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Dr. Gerstenfeld does research for Biosense Webster, Medtronic, and Rhythmia Medical.

SOURCE: Kinch Westerdahl A, et al. Implantable cardioverter-defibrillator therapy before death: High risk for painful shocks at end of life. *Circulation* 2014;129:422-429.

This study aimed to determine the incidence of appropriate shocks, inappropriate shocks, and device malfunction prior to death in patients with implantable cardioverter-defibrillators (ICDs). The study was performed in Sweden where it is mandatory that all ICDs are explanted after death. The 26 participating pathology departments contributed 130 devices (5 nonfunctioning). Medical records were reviewed and autopsies and death certificates were obtained to determine the cause of death. ICDs were interrogated and electrograms from the 24 hours prior to death were retrieved and adjudicated by blinded investigators to determine whether shocks were appropriate. Death was classified as "sudden" if it occurred within 1 hour of symptoms. If true ventricular tachycardia/fibrillation (VT/VF) was present within 1 hour of death, the death was classified as arrhythmic. Electrical storm was defined as ≥ 3 shocks in the 24 hours prior to death. The majority of devices (82%) were implanted for secondary prevention and 35% were biventricular devices. At the time of death, 80% of patients were in a hospital or care facility. Although 52% of patients had do-not resuscitate (DNR) orders, ICD shock therapies remained active in half of these patients up to 1 hour before death. Twenty-five devices had been programmed off. There were 38 patients with VT/VF in the final 24 hours and 24 who had VT storm before death. A DNR order was present at the time of ICD shocks in 10 VT storm patients, and six of these patients received an average of 5.6 ± 6.5 shocks. Four patients received inappropriate shocks because of SVT or T wave oversensing. There were four deaths related to device malfunction, three due to undersensing of VF, and one due to a lead malfunction. The cause of death was determined to be sudden in 29 patients and arrhythmic in 16 (13%). The most common cause of death was congestive heart failure (37%). The authors concluded that most ICD patients died in the hospital and the devices remained active in more than half of the patients with a DNR order.

## ■ COMMENTARY

ICD therapy can be lifesaving, and is now standard of care for patients with ischemic or nonischemic cardiomyopathy with reduced ejection fraction and for secondary prevention of ventricular tachycardia in patients with structural heart disease. After a decade of experience with ICDs, we now discuss both the benefits and risks of ICD placement with patients prior to implantation, including the risk of lead/device malfunction, and appropriate and inappropriate shocks. However, this study shows that we are often remiss in incorporating the ICD into end-of-life decisions with our patients. Although the presence of ICD shocks at the "end of life" is not surprising, it is striking that of 52 patients with DNR orders, ICD therapies remained

on in half the patients. There are several reasons why these devices may not be deactivated. First, implanting electrophysiologists are often not involved in palliative or end-of-life care with their patients. Internists or family practitioners may not even know of an ICD being present, or consider it in their end-of-life planning. Finally, patients may not know of the option for deactivating the ICD at end of life.

What should we learn from this study? First, as implanting electrophysiologists and cardiologists, the ability to turn off the ICD should be discussed with the patient before or shortly after implantation. As difficult as the discussion is when considering life-saving therapy, a simple mention that the device can be turned off when no longer needed can actually be reassuring to patients. Patients should be encouraged to incorporate decisions regarding ICD therapy into living wills. Finally, internists, cardiologists, oncologists, and all those involved in end-of-life care should be educated to ask patients whether an ICD is present and if deactivation of shock therapies is appropriate. According to the Heart Rhythm Society consensus statement,[1] electrophsyiologists should turn off device therapies when requested by a patient in the appropriate circumstance, or find a colleague to perform this action. Device representatives also have the ability to deactivate ICDs at remote sites, including patient homes, when ordered by an attending physician. ICDs have become an important tool in our fight against sudden death. However, this study reminds us that death is inevitable in all our patients. Deactivation of ICD therapies at end of life should be part of the commitment to our patients who have undergone ICD implantation. ■


REFERENCE

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ABSTRACT & COMMENTARY

# Prognosis of First-Degree AV Block

***By Michael H. Crawford, MD, Editor***


SOURCE: Aro AL, at al. Prognostic significance of prolonged PR interval in the general population. *Eur Heart J* 2014;35:123-129.


Although long believed to be benign, first-degree AV block has recently been shown to be associated with atrial fibrillation development, pacemaker need, and all-cause mortality. This group of investigators from Finland had access to a 30-year follow-up study of almost 11,000 apparently healthy, middle-aged Finns where the prognostic value of the ECG PR interval could be assessed. In 10,957 men and women aged 30-59 years at entry between 1966-1972, a complete history, targeted physical examination, routine laboratory studies, and an ECG were done. They were followed for a mean of 30 ± 11 years until 2007. Less than 2% were lost to follow up. The endpoints of death, cardiovascular death, sudden death, and hospitalizations were obtained from Finland government data and medical record reviews. Also, a second ECG was performed after a mean of 6 years into the study.

A PR interval > 200 msec was observed in 2.1% of the subjects and was more common in obese older men and related to a slower heart rate and suspected cardiac disease. At the 6-year ECG, 71% of the subjects with a prolonged PR at entry still had it. After adjustment for age and sex, prolonged PR interval was not associated with all-cause cardiovascular or sudden death, and these results were not changed by multivariate adjustment, including use of a PR interval of > 220 msec. Also, the risk of hospitalization for atrial fibrillation, CAD, heart failure, or stroke did not differ from the rest of the population. The authors concluded that in an apparently healthy, middle-aged population, a prolonged PR interval can normalize over time in almost one-third of subjects, and even if persistent, is not associated with death or cardiovascular morbidity and mortality.

## ■ COMMENTARY

Occasionally, we see isolated PR interval prolongation on ECG in an otherwise healthy individual without overt cardiac disease and wonder if we should be concerned. This study in almost 11,000 middle-aged (30-59 years) subjects followed for a mean of 30 years sheds considerable light on the issue. When combined with prior studies, several conclusions can

be reached. Overall, in an otherwise healthy, middle-aged population, first-degree AV block is not a harbinger of death, sudden death, or cardiovascular (CV) morbidity when adjusted for age, sex, and risk factors for CV disease. So why is the PR interval prolonged in some healthy people? It is related to heart rate and really should be adjusted for heart rate the way the QT interval is, but since this is only relevant at heart rates < 60 beats per minute we don't bother. It is influenced by autonomic nervous system tone. Increased PR can be seen in athletes and others with high vagal tone. It has a circadian variation, so the time of day the ECG is done can be important. These observations probably explain the disappearance of first-degree block in this study at the 6-year follow-up in about one-third of the population studied. There is also a genetic component and it can be associated with atrial arrhythmias and the development of AV block. This is not surprising because we know there is a large genetic influence on the risk of developing atrial fibrillation.

Clearly a prolonged PR interval can be a harbinger of future conduction system disease in some people, especially older subjects where cardiac degenerative diseases are more common. Also, if a patient develops diastolic dysfunction, a prolonged PR interval can perturb diastolic filling and lead to diastolic mitral valve regurgitation, which under the right conditions could contribute to the development of heart failure. So how should we handle patients with first-degree heart block? If they are young and overtly healthy, I would repeat the ECG in 1-3 years because it may just disappear with age or deconditioning. In older patients, it would prompt me to repeat the ECG at the time of their routine health maintenance follow-up exams, which is usually every 1-2 years depending on age. However, patients should be reassured that most people with this finding have normal longevity and are free from CV disease. ■

ABSTRACT & COMMENTARY

# Risk of Early Repolarization ECG Patterns

***By Michael H. Crawford, MD, Editor***

SOURCE: Muramoto D, et al. Patterns and prognosis of all components of the J-wave pattern in multiethnic athletes and ambulatory patients. *Am Heart J* 2014;167:259-266.

ECG early repolarization patterns were long thought to be benign normal variants until recent papers purported to show a relationship between these patterns and the risk of malignant arrhythmias and cardiac death. Since these patterns are common in young athletes, concern has arisen about their interpretation. Thus, these investigators from Stanford University performed a retrospective study of more than 5000 consecutive ECGs performed at a Veterans Affairs hospital between 1997-1999. They excluded patients with atrial fibrillation or flutter, QRS duration > 120 msec, paced rhythm, pre-excitation, or acute myocardial infarction. Also, routine screening ECGs of Stanford athletes were recorded in 2007-2008. There were 4041 patient ECGs (90% male, average age of 57 years), and there were 1114 athlete ECGs (57% male, average age 19 years). Rather than using the ambiguous term "early repolarization" they preferred the term "J-wave pattern," which included elevation of the QRS-T junction of at least 0.1 mV from baseline in the inferior or lateral leads manifested as terminal QRS slurring or notching of the R or S wave downstroke, respectively. The notching was termed a J-wave. The primary outcome was time to cardiovascular death in the patients; the athletes had no cardiovascular events after 3 years.

ST elevation was more common in the lateral vs inferior leads in both groups: patients 4.8% lateral vs 1.0% inferior; athletes 21 vs 1.3%, respectively. J-waves were equally present in lateral and inferior leads and were < 5% of both groups. Terminal QRS slurring was more common in the inferior leads in both groups: inferior < 10% and lateral < 5% of both groups. The presence of J-wave patterns in both lead groups was infrequent in both populations (2.4% of patients, 5.5% of athletes). Most of the components of the J-wave pattern were more frequent in men. All components of the J-wave pattern in the lateral leads were more frequent in African American patients and athletes. No single component or combination of the J-wave patterns was associated with an increase in cardiovascular death. The authors concluded that their large, multiethnic, ambulatory population J-wave patterns were not predictive of cardiovascular outcomes and that their high prevalence, especially in men and

athletes, would make them poor screening parameters even if they were predictive.

## ■ COMMENTARY

A few years ago, Haissaguerre and later Nam described cases of young men with J-waves and unprovoked ventricular fibrillation (VF). This rare syndrome is of unknown etiology as it does not fit into the usual channelopathies. This created a "fear of J-waves" that has created considerable controversy.[1] Also, other prior reports have suggested that the early repolarization pattern, especially if confined to the inferior leads, is associated with higher rates of cardiovascular events and death. This study refutes these assertions in a large multiethnic population with long-term follow-up. Also, they analyzed all components of the J-wave pattern, not just ST elevation or J-waves. Some prior studies used selective criteria for the early repolarization pattern, but the main weakness of prior studies is selection biases. Also, some studies had very long follow-up periods, up to 30 years. It is difficult clinically to deal with a death 20 years after an ECG is done.

Their analysis of an athlete population showed that these J-wave patterns are prevalent (5-20%). So, even if a J-wave identifies subjects at risk for a rare genetic disorder associated with VF (high sensitivity), the specificity is horrible. You can't put an ICD in even 5% of athletes or exclude them from sports participation. Despite this flurry in the literature over the last decade, currently we should view these J-wave patterns or early repolarization as largely benign entities again and remove them as triggers for further evaluation. ■

### REFERENCE

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## CME Questions

1. Which of the following is most correct concerning end-of-life patients with ICDs?
   a. Most die of VT/VF storm
   b. The majority have an active device
   c. Electrophysiologists are actively involved in their care
   d. Device malfunction is a common cause of death

2. Which of the following is the major benefit of mitral valve replacement vs repair in ischemic mitral regurgitation?
   a. Regurgitation is almost completely eliminated
   b. LV function is better preserved
   c. In-hospital mortality is less
   d. Fewer major adverse events occur postoperatively

3. Mechanical vs manual chest compression for CPR results in:
   a. fewer adverse events.
   b. lower survival rates.
   c. limited applicability due to body size in 5%.
   d. less deep chest compressions.

4. In a general, apparently healthy population, a prolonged PR interval:
   a. is common.
   b. is associated with sudden cardiac death.
   c. is associated with atrial fibrillation development.
   d. resolves over time in one-third.

5. Ventricular fibrillation in hospitalized patients with acute MI is associated with:
   a. higher in-hospital mortality.
   b. higher long-term mortality.
   c. higher long-term risk of sudden death.
   d. All of the above

6. Early repolarization or J-wave patterns are *not* associated with:
   a. athletic activity.
   b. African Americans.
   c. cardiovascular death.
   d. men.