The Editor's Roundtable: Implantable Cardioverter-Defibrillators in Primary Prevention of Sudden Cardiac Death and Disparity-Related Barriers to Implementation

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Disclosures

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Objectives

Upon completion of the activity, the physician should be able to:

1. Diagnose patients with congestive heart failure who are candidates for implantable cardioverter-defibrillator (ICD) therapy.
2. Explain the risks and benefits of ICD therapy to patients.
3. Decrease gender and ethnic disparities in treatment with ICD therapy.

Target Audience: This activity is designed for cardiologists and all other health care specialists caring for patients with acute and chronic coronary heart disease.

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Combination of Media: Print and Internet
Computer Requirements: Windows 2000, Pentium 3 or greater, 512 ram, 80 gigabytes storage
Estimated Time to Complete: 1 hour
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Introduction

The ICD was first placed into human subjects in 1980 by Mirowski, after several years of nonhuman animal testing.[1,2] In the 30 years since the introduction of ICDs, ICD therapy in the United States has become commonplace, with 2 broad categories of use for preventing sudden cardiac death (SCD): *primary prevention* involves the prevention of SCD in patients without histories of cardiac arrest or sustained ventricular tachycardia, and *secondary prevention* involves the prevention of SCD in patients who have survived prior cardiac arrest, sustained ventricular tachycardia, or other major cardiac events. This Editor's Roundtable focuses on ICD therapy for primary prevention, which mainly involves patients with ischemic and nonischemic

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heart failure "who are receiving optimal medical therapy and have a reasonable expectation of survival with good functional status for >1 year."[3]

Discussion

Dr. Friedewald: When did ICD therapy appear?

Dr. Olshansky: ICD therapy was developed and first tested in nonhuman animal models by Michel Mirowski in the 1970s.[2] Although the concept of an ICD was not initially well received, Mirowski paved the way for clinical acceptance when he performed the first human implantation in 1980.[1] The first ICD was large, weighing about 9 oz, with a large battery and a capacitor to shock the heart. It had few settings, its sensing ability was rudimentary, and it lasted only about 18 months. It required major surgery because the device was implanted in the abdomen and the chest had to be opened, requiring at least 1 week of hospitalization. The first ICD devices were handmade, so the supply was limited, and patients sometimes had to wait for weeks before 1 could be obtained. The technology progressed dramatically over the next 30 years as the devices became multiprogrammable, used smaller batteries with longer battery life, had better capabilities to defibrillate with biphasic shocks, were made programmable for cardiac pacing, had better leads that could be placed intravenously, and were implanted into the upper chest, an easy procedure to perform. Today, ICD implantation is a low-risk procedure carried out worldwide.

Dr. Yancy: What are the current risks of ICD use?

Dr. Olshansky: There are several. Although the focus in the lay press has been placed on device recalls and lead problems, improper working devices are rare, maybe 1 in 10,000 implants. Other complications including myocardial lead perforation, infection, pneumothorax, lead dislodgement, and inappropriate shocks (a shock delivered for a reason other than a life-threatening ventricular tachyarrhythmia). The ICD is designed, however, to protect life at the expense of an occasional inappropriate shock. The risk for inappropriate shock is about 25%.

Dr. Yancy: What is the frequency of serious problems with ICDs?

Dr. Olshansky: Serious problems such as infection and device failure occur in about 1% of ICD implants.

Dr. Yancy: What is the current role of the ICD in preventing SCD?

Dr. Olshansky: There has been a significant movement to ICD use for primary prevention of SCD. At 1 time, before receiving an ICD, patients had to experience 2 separate out-of-hospital cardiac arrests, so few patients used to qualify for an ICD. We no longer require a prior event, only that a patient is "likely to have cardiac arrest" (Appendix[3]). Although there is some controversy about the criteria, most ICD implantations are for primary prevention, not secondary prevention.

Dr. Fonarow: Much of the growth of ICD utilization is due to the recognition that most antiarrhythmic drugs are ineffective for both primary and secondary prevention and are sometimes proarrhythmic, thereby increasing the risk for SCD. Two decades ago, flecainide and encainide were 2 of the top 10 cardiac medications prescribed, but they subsequently were found to increase all-cause mortality and are proarrhythmic.[4] Other antiarrhythmic drugs, such as amiodarone, also fail to protect against SCD.

Dr. Friedewald: What is the relation between left ventricular (LV) dysfunction and SCD?

Dr. Fonarow: Patients with significant LV dysfunction—even in the absence of a prior cardiac event, ventricular ectopic beats on ambulatory monitoring, or inducible arrhythmia on electrophysiologic study—are at increased risk for SCD. Because up to 1/2 of deaths in patients with LV dysfunction are sudden, prophylactic ICD placement in this patient population is often indicated. Prospective randomized clinical trials in patients receiving optimal heart failure (HF) treatment with subsequent placement of the ICD demonstrated that they aborted SCD when compared to drug treatment alone, in patients with both ischemic and nonischemic forms of cardiomyopathy.

Dr. Friedewald: Do drugs that are not directly antiarrhythmic, but proven beneficial in treating patients with HF (i.e., β blockers and renin-angiotensin aldosterone inhibitors) reduce the risk of SCD in patients with HF?

Dr. Fonarow: Beta blockers reduce death from progressive HF as well as SCD in patients with LV dysfunction. Patients on β blockers, however, have a greater relative reduction in death from progressive HF, resulting in increased incidence of SCD in this population. The predominant effect of angiotensin-converting enzyme inhibitors is on death from progressive HF with possibly a slight reduction in the frequency of SCD. Aldosterone antagonists also decrease the risk of death from progressive HF, with possibly a slight reduction in the frequency of SCD. Thus, patients on optimal medical therapy for LV dysfunction and HF have enough residual risk for SCD that usually justifies primary ICD placement.

Dr. Yancy: Is there a role for antiarrhythmic drugs in patients with an ICD?

Dr. Olshansky: There may be a role for the use of antiarrhythmic drugs in addition to ICD therapy in patients who receive multiple ICD shocks for ventricular and atrial tachyarrhythmias. As primary therapy to reduce total mortality or arrhythmic death, however, antiarrhythmic drugs have no role. The important point is that ICD therapy reduces the incidence of both SCD and overall mortality.

Dr. Fonarow: It is important to separate absolute risk and proportional risk. The proportion of SCD relative to death from progressive left ventricular dysfunction is higher in patients with less severe HF symptoms—New York Heart Association class I or II—compared to patients in class III or IV HF, in which a greater proportion of deaths are due to progressive HF. Thus, although SCD occurs in patients in class III and IV HF, the absolute risk for deaths both from progressive HF death as well as SCD rises with increasing severity of HF. There is little benefit in preventing SCD in a patient who shortly thereafter dies from progressive HF. Thus, identifying patients who derive the greatest absolute benefit from the therapy and in whom the benefit outweighs the potential ICD risks is essential. In patients with class I to III HF treated with an ICD, the benefit outweighs the risk and prolongs survival. In class IV patients, however, because of the ICD impairment on quality of life and functional status, HF not amenable to optimal medical therapy precludes ICD therapy. Class II and III HF