Implantable Cardioverter-Defibrillators

Rachel Lampert, MD and Zachary Goldberger, MD

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Abstract

Sudden cardiac death (SCD) causes 300,000–400,000 deaths annually in the United States and is responsible for nearly 50% of all cardiovascular mortality worldwide (1–3). Ventricular tachycardia (VT) degenerating into ventricular fibrillation (VF) causes two-thirds of SCD. Among patients with heart failure (HF), one-third to one-half of deaths are sudden. Because there are often no warning symptoms to identify potential victims of SCD, successful therapy has focused on identifying high-risk patients and implanting cardioverter-defibrillators (ICDs), which continuously monitor the heart rhythm and deliver therapy (a shock or an antitachycardia pacing) upon detection of a sustained ventricular arrhythmia. This chapter focuses on the clinical indications and technical aspects of ICDs for heart failure patients.

Key Words: Heart failure; Implantable defibrillator; Sudden death; Ventricular tachycardia; Ventricular fibrillation.

1. SUDDEN CARDIAC DEATH IN HEART FAILURE

Sudden cardiac death (SCD) causes 300,000–400,000 deaths annually in the United States and is responsible for nearly 50% of all cardiovascular mortality worldwide (1–3). Ventricular tachycardia (VT) degenerating into
ventricular fibrillation (VF) causes two-thirds of SCD (4–6). Among patients with heart failure (HF), one-third to one-half of deaths are sudden (7). While overall mortality increases as functional status worsens, the proportion of deaths which are sudden is highest in those patients with less-severe signs and symptoms (8). This holds true for both ischemic and nonischemic etiologies (7). While more recent pharmacological advances have improved overall mortality, the percentage of deaths which are sudden remain similar (7, 9, 10). Of sudden deaths in patients with HF, about half are due to ventricular tachyarrhythmias and half due to bradycardia or electromechanical dissociation (11). Because there are no warning symptoms to identify potential victims of tachyarrhythmic SCD (4), successful therapy has focused on identifying high-risk patients and implanting cardioverter-defibrillators (ICDs), which continuously monitor the heart rhythm and deliver therapy (a shock or an antitachycardia pacing) upon detection of a sustained ventricular arrhythmia (12).

2. INDICATIONS FOR ICD IMPLANTATION IN PATIENTS WITH HF

The introduction of the ICD into clinical practice has been a process in evolution. While secondary prophylaxis for patients surviving a life-threatening ventricular arrhythmia has long been the standard of care, more recent trials demonstrate the survival benefit of the ICD in expanding groups of patients at high risk (primary prophylaxis) for SCD as well.

2.1. Secondary Prevention Trials

Several trials have investigated the role of the ICD in secondary prevention of SCD (Table 1) (13–17). The largest include the Antiarrhythmics Versus Implantable Defibrillators (AVID) trial (13), the Cardiac Arrest Study – Hamburg (CASH) (14), and the Canadian Implantable Defibrillator Study (CIDS) (15). Each randomized patients to ICD vs pharmacologic therapy, and in each, the ICD reduced total mortality, although only AVID reached statistical significance.

AVID was also the largest and best designed, including nearly exclusive use of transvenous defibrillators and comparing the ICD against the best available antiarrhythmic drug therapy (amiodarone and sotalol) unlike CASH and CIDS. Further, a meta-analysis of AVID, CASH, and CIDS confirmed that ICD therapy resulted in significant relative reductions in total mortality (27%) and arrhythmic mortality (51%) (18). The ICD improved survival regardless of beta-blockade, surgical revascularization, or presenting arrhythmia (VT or VF). There was no difference in benefit gained from ICD implantation between those patients with coronary disease and those with nonischemic cardiomyopathies (18).
## Table 1

<table>
<thead>
<tr>
<th>Study</th>
<th>Randomization</th>
<th>CHF enrollment by class</th>
<th>N</th>
<th>Inclusion criteria by class</th>
<th>Mean follow-up (months)</th>
<th>Main findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>AVID (13)</td>
<td>Antiarrhythmic medications (97% amiodarone, 3% sotalol) vs ICD</td>
<td>None 42% I/I 48% I/II 10%</td>
<td>1016</td>
<td>Survived VT/VF/cardiac arrest; VT with syncope; VT with LVEF ≤ 0.40</td>
<td>18</td>
<td>27% relative risk reduction in total mortality with ICD therapy (P &lt; 0.02)</td>
</tr>
<tr>
<td>CASH (14)</td>
<td>Antiarrhythmic medications propafenone (withdrawn early), metoprolol, or amiodarone vs ICD</td>
<td>None 42% I/I 48% I/II 10%</td>
<td>288</td>
<td>Survived VT/VF/cardiac arrest</td>
<td>57</td>
<td>23% relative risk reduction in total mortality with ICD therapy (P = 0.08)</td>
</tr>
<tr>
<td>CIDS (15)</td>
<td>Amiodarone vs ICD</td>
<td>None 50% I/I 38% I/II 11%</td>
<td>659</td>
<td>Survived VT/VF/cardiac arrest; VT with syncope; VT with LVEF ≤ 0.35 and cycle length ≤ 400 ms</td>
<td>35</td>
<td>19.7% relative risk reduction in death from any cause with ICD therapy (P = 0.142); 32.8% reduction in the risk of death from arrhythmia with ICD therapy (P = 0.094)</td>
</tr>
</tbody>
</table>
### Table 1 (Continued)

<table>
<thead>
<tr>
<th>Study</th>
<th>Randomization</th>
<th>N</th>
<th>Inclusion criteria</th>
<th>CHF enrollment by class&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Mean follow-up (months)</th>
<th>Main findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>DEBUT&lt;sup&gt;(16)&lt;/sup&gt;</td>
<td>Beta-blocker vs ICD</td>
<td>86</td>
<td>Survived VT/VF/cardiac arrest; no structural abnormalities</td>
<td>I 100%</td>
<td>36</td>
<td>Seven total deaths, all of which occurred in the beta-blocker group – (three deaths during 2-year follow-up, <em>P</em> = 0.07; four deaths during 3-year follow-up, <em>P</em> = 0.02)</td>
</tr>
<tr>
<td>MAVERIC&lt;sup&gt;(17)&lt;/sup&gt;</td>
<td>EP-guided therapy (antiarrhythmic, revascularization, or ICD) vs amiodarone</td>
<td>214</td>
<td>Survived VT/VF/cardiac arrest</td>
<td>Any CHF 26%</td>
<td>60&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Lower mortality with ICD therapy compared to non-ICD therapy (<em>P</em> = 0.04). No advantage to EP testing</td>
</tr>
</tbody>
</table>

Abbreviations: AVID, Antiarrhythmics Versus Implantable Defibrillators; CASH, Cardiac Arrest Study – Hamburg; CIDS, Canadian Implantable Defibrillator Study; DEBUT, Defibrillator Versus beta-Blockers for Unexplained Death in Thailand; MAVERIC, The Midlands Trial of Empirical Amiodarone Versus Electrophysiologically Guided Intervention and Cardioverter Implant in Ventricular Arrhythmias; EP, electrophysiologic; ICD, implantable cardioverter-defibrillator; LVEF, left ventricular ejection fraction; VF, ventricular fibrillation; VT, ventricular tachycardia.

<sup>a</sup>As defined for the trial.

<sup>b</sup>Median follow-up.
Interestingly, the AVID registry, enrolling patients not qualifying for randomization, showed that some groups previously considered at lower risk for SCD – patients with hemodynamically stable VT or arrhythmias attributed to “reversible causes” – actually had significantly worse survival than did randomized ICD-treated patients. In particular those with lower ejection fractions fared poorly, suggesting that these groups may also benefit from ICDs (19).

2.2. Primary Prevention Trials

The dismal survival rate after cardiac arrest (20–22) provides strong impetus to identify high-risk patients who might benefit from an ICD, before a first life-threatening arrhythmia. How best to stratify risk has been a process in evolution. Historically, patients with LV dysfunction, postmyocardial infarction, and a history of nonsustained VT underwent electrophysiology (EP) testing to identify higher risk patients with inducible, nonsuppressible, ventricular tachyarrhythmias (23, 24). This population was the target for the first primary prevention trial (Table 2), the Multicenter Automatic Defibrillator Trial (MADIT) (25), which compared ICDs to conventional therapy (mainly amiodarone) (ejection fraction (EF) ≤35%), and the Multicenter Unsustained Tachycardia Trial (MUSTT) (26), which compared EP-guided therapy (ICDs or drug therapy) vs no EP-guided therapy (EF ≤40%).

MADIT was prematurely aborted after enrolling only 196 patients, when preliminary analysis revealed a dramatic benefit of ICD therapy in reducing overall mortality by 54% ($P = 0.009$). MADIT had no placebo group, raising the question of whether the trial proved benefit of ICD or detriment of amiodarone. Also, beta-blocker use was higher in the ICD arm. However, MUSTT, while not designed to evaluate the ICD, supported the MADIT findings. The original hypothesis of MUSTT was that EP-guided therapy, either pharmacological or device based, could reduce arrhythmic and total mortality in high-risk patients who had arrhythmias induced at EP study. In patients randomized to EP-guided therapy, antiarrhythmic drugs were tested first and, at the physician’s discretion, nonresponders received ICDs. MUSTT did show a decrease in arrhythmic death/cardiac arrest with EP-guided therapy, supporting the primary hypothesis. However, subgroup analysis revealed that the benefits were due entirely to the ICD: at 5 years, there were absolute reductions in total mortality of 31% when compared to those receiving pharmacological therapy and of 24% when compared to those receiving no therapy (mortality 24% in the ICD group, 55% with pharmacological therapy, and 48% with no therapy). In MUSTT, few patients received amiodarone, and ICD use was not randomized. However, taken together, MUSTT and MADIT clearly demonstrate the benefit of the ICD in the relatively small population of patients with coronary artery disease (CAD), low LVEF, and inducible ventricular arrhythmia.

The MUSTT registry (27), however, followed patients who had clinical criteria for the trial but had no inducible arrhythmias. Surprisingly,
### Table 2
Primary prevention of sudden cardiac death in ischemic cardiomyopathy – randomized trials

<table>
<thead>
<tr>
<th>Study</th>
<th>Randomization</th>
<th>N</th>
<th>Population</th>
<th>HF NYHA Class</th>
<th>Mean F/U (months)</th>
<th>Main findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>MADIT (25)</td>
<td>Antiarrhythmic therapy (74% amiodarone) vs ICD</td>
<td>196</td>
<td>Prior MI; LVEF ≤ 0.35; asymptomatic NSVT; NYHA Class I–III; inducible VT refractory to intravenous procainamide on EPS</td>
<td>II/III 65%</td>
<td>27</td>
<td>56% RR reduction in mortality with ICD therapy ($P = 0.009$)</td>
</tr>
<tr>
<td>MUSTT (26)</td>
<td>EP-guided therapy (AAD or ICD) vs conventional therapy</td>
<td>704</td>
<td>Prior MI; LVEF ≤ 0.40; CAD; NSVT; inducible VT on EPS</td>
<td>I 37%, II 38%, III 25%</td>
<td>39a</td>
<td>60% RR reduction in mortality with ICD therapy ($P &lt; 0.001$)</td>
</tr>
<tr>
<td>MADIT II (28)</td>
<td>Conventional therapy vs ICD</td>
<td>1232</td>
<td>Prior MI; LVEF ≤ 0.30</td>
<td>I 37%, II 34%, III 24%, IV 5%</td>
<td>20</td>
<td>31% RR reduction in mortality with ICD therapy ($P = 0.016$)</td>
</tr>
<tr>
<td>SCD-HeFT (29)</td>
<td>Conventional therapy vs amiodarone vs ICD</td>
<td>2521</td>
<td>NYHA Class II–III CHF (ischemic and nonischemic); LVEF ≤ 0.35</td>
<td>II 70%, III 30%</td>
<td>45.5a</td>
<td>Overall: 23% RR reduction in mortality with ICD therapy ($P = 0.007$)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Ischemic heart disease: 21% relative reduction in mortality with ICD therapy ($P = 0.05$)</td>
</tr>
<tr>
<td>Study</td>
<td>Intervention</td>
<td>Patients</td>
<td>Design Criteria</td>
<td>Follow-up</td>
<td>Findings</td>
<td></td>
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</tr>
<tr>
<td>CABG Patch (44)</td>
<td>CABG vs CABG with an ICD</td>
<td>900</td>
<td>Patients scheduled for CABG; LVEF ≤ 0.35; positive SAECG</td>
<td>II/III 72%</td>
<td>32 No reduction in mortality with ICD therapy ($P = 0.64$)</td>
<td></td>
</tr>
<tr>
<td>DINAMIT (33)</td>
<td>Conventional therapy vs ICD</td>
<td>674</td>
<td>Recent MI (within 4–40 days), LVEF ≤ 0.35; impaired cardiac autonomic modulation (HRV)</td>
<td>I 13%, II 59%, III 27%</td>
<td>39 No reduction in death with ICD therapy ($P = 0.66$); risk of arrhythmic death lower with ICD therapy ($P = 0.009$)</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: MADIT, Multicenter Automatic Defibrillator Trial; MUSTT, Multicenter Unsustained Tachycardia Trial; SCD-HeFT, Sudden Cardiac Death in Heart Failure Trial; CABG Patch, Coronary Artery Bypass Graft Patch; DINAMIT, Defibrillator in Acute Myocardial Infarction Trial; AAD, antiarrhythmic drug; CAD, coronary artery disease; EPS, electrophysiological study; HRV, heart rate variability; ICD, implantable cardioverter-defibrillator; LVEF, left ventricular ejection fraction; MI, myocardial infarction; NSVT, nonsustained ventricular tachycardia; NYHA, New York Heart Association; RR, relative risk; SAECG, signal-averaged electrocardiogram; VF, ventricular fibrillation; VT, ventricular tachycardia.

*Median follow-up.*
while 5-year mortality for these patients was statistically lower than that for inducible patients randomized to no therapy (44% and 48%, respectively, \( P = 0.005 \)), it was significantly higher than that for the inducible, ICD-treated patients (24%). These data implied that noninducible patients with LV dysfunction and nonsustained ventricular tachycardia (NSVT) may also benefit from a prophylactic ICD and that EP study may be an inadequate risk stratifier.

The second MADIT trial (MADIT II) (28) directly addressed the value of prophylactic ICD implantation in patients with CAD and EF $\leq 30\%$, without EP risk stratification. The ICD showed a 31% relative reduction in mortality at any interval \( (P = 0.016) \). Of note, among 593 patients in the ICD arm who underwent peri-implant EP testing (not an entry criterion), inducibility did not predict later ventricular arrhythmia, supporting a low sensitivity for EP testing.

The more recently published Sudden Cardiac Death in Heart Failure Trial (SCD-HeFT) (29) enrolled patients with either ischemic or nonischemic cardiomyopathy, New York Heart Association (NYHA) Class II or III heart failure, and LVEF $\leq 35\%$. The results confirmed the benefit of ICD in ischemic patients as found in MADIT II, as well as the findings of a previous smaller study of nonischemic cardiomyopathy patients, the Prophylactic Defibrillator Implantation in Patients with Nonischemic Dilated Cardiomyopathy (DEFINITE) trial (30). (Selected studies of primary prophylaxis of SCD in patients with nonischemic cardiomyopathy can be found in Table 3 (29, 31–33).) In SCD-HeFT, ICD-treated patients lived longer than those treated with amiodarone (which had no benefit) or conventional medical therapy. While previous studies have shown the benefits of pharmacological therapy of HF in preventing sudden death (9, 10), the majority of patients in SCD-HeFT were receiving standard HF therapies (87% receiving an ACE inhibitor or ARB, 78% receiving beta-blockers), implying an incremental benefit of the ICD even among appropriately treated HF patients. Further, while the concern has been raised that ICD benefit in MADIT II may have been skewed by the short follow-up (34), SCD-HeFT showed ICD benefit extending to 5 years, independent of heart failure etiology (ischemic vs nonischemic). These studies suggest that patients with LVEF $\leq 35\%$ and NYHA Class II or III HF are candidates for an ICD (based on SCD-HeFT) as are patients with LVEF $\leq 30\%$ and history of MI (based on MADIT II).

However, whether ejection fraction and heart failure functional class alone should be the primary factor determining ICD eligibility remains somewhat controversial (35–38). In MUSTT, left ventricular EF had poor specificity in predicting SCD (39), and in other studies, combinations of factors were more predictive (40, 41). Limiting both the MADIT II and SCD-HeFT study designs, neither evaluated ICD benefit in patients known to be noninducible. This may explain why the absolute reductions in all-cause mortality for MADIT II and SCD-HeFT (6% and 7%, respectively) are much smaller than that for MADIT I and MUSTT (23% and 31%, respectively), which selected
Table 3
Primary prevention of sudden cardiac death in nonischemic dilated cardiomyopathy – randomized trials

<table>
<thead>
<tr>
<th>Study</th>
<th>Randomization</th>
<th>N</th>
<th>Population</th>
<th>CHF enrollment by class</th>
<th>Mean follow-up (months)</th>
<th>Main findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>CAT (31)</td>
<td>Conventional therapy vs ICD</td>
<td>104</td>
<td>NYHA Class II–III, NIDCM; LVEF ≤ 0.30; asymptomatic NSVT</td>
<td>II 66%</td>
<td>66</td>
<td>No reduction in total mortality with ICD therapy (P = 0.554)</td>
</tr>
<tr>
<td>AMIOVERT (32)</td>
<td>Amiodarone vs ICD</td>
<td>103</td>
<td>NYHA Class I–III, NIDCM; LVEF ≤ 0.35; asymptomatic NSVT</td>
<td>I 15%</td>
<td>36</td>
<td>No reduction in total mortality with ICD therapy (P = 0.80)</td>
</tr>
<tr>
<td>DEFINITE (30)</td>
<td>Conventional therapy vs ICD</td>
<td>458</td>
<td>NIDCM; LVEF &lt; 0.36; NSVT or PVCs</td>
<td>I 22%</td>
<td>29</td>
<td>35% relative risk reduction in total mortality with ICD therapy (P = 0.08); 80% reduction in death from arrhythmia with ICD therapy (P = 0.006)</td>
</tr>
</tbody>
</table>

(Continued)
Table 3
(Continued)

<table>
<thead>
<tr>
<th>Study</th>
<th>Randomization</th>
<th>N</th>
<th>Population</th>
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</thead>
<tbody>
<tr>
<td>SCD-HeFT (29)</td>
<td>Conventional therapy vs amiodarone vs ICD</td>
<td>2521</td>
<td>NYHA class II–III CHF (ischemic and nonischemic); LVEF ≤ 0.35</td>
<td>See above</td>
<td>45.5a</td>
<td>Overall: 23% relative risk reduction in mortality with ICD therapy ($P = 0.007$) Nonischemic heart disease: 27% relative reduction in mortality with ICD therapy ($P = 0.06$)</td>
</tr>
</tbody>
</table>

Abbreviations: CAT, Cardiomyopathy Trial; AMIOVERT, Amiodarone Versus Implantable Defibrillator in Patients with Nonischemic Cardiomyopathy and Asymptomatic Nonsustained Ventricular Tachycardia; DEFINITE, Prophylactic Defibrillator Implantation in Patients with Nonischemic Dilated Cardiomyopathy; SCD-HeFT, Sudden Cardiac Death in Heart Failure Trial; CAD, coronary artery disease; ICD, implantable cardioverter-defibrillator; LVEF, left ventricular ejection fraction; NIDCM, nonischemic dilated cardiomyopathy; NSVT, nonsustained ventricular tachycardia; NYHA, New York Heart Association; PVC, premature ventricular contraction; VF, ventricular fibrillation; VT, ventricular tachycardia.

aMedian follow-up.
higher risk patients using EP criteria. As a result, SCD-HeFT/MADIT II had a much higher number needed to treat than did MUSTT/MADIT (15–17 vs 3–4). Thus, the benefit of ICD therapy may be greater in patients with SCD risk beyond low LVEF (although these differences in absolute risk reduction may also be due to better medical therapy in the control groups in the later trials (42). The validity of ICD implantation in all post-MI patients with reduced LVEF has been questioned (35, 43, 44), suggesting that further risk stratification is still needed (35). The potential role of noninvasive risk stratifiers shown to have good predictive value, such as T-wave alternans (45, 46), remains undetermined.

Two studies failed to show benefit of ICD for primary prophylaxis in specific populations: the Coronary Artery Bypass Graft (CABG) Patch Trial (47) and the Defibrillator in Acute Myocardial Infarction Trial (DINAMIT) (33). CABG Patch randomized patients with an abnormal signal-averaged electrocardiogram undergoing CABG to treatment with an ICD, implanted during surgery, or to treatment with no ICD. The ICD showed no benefit, likely due to the lower risk profile of the patient population. While preoperative ejection fractions were low, they may improve with surgery. Also, the signal-averaged electrocardiogram may lack specificity (23, 24). Revascularization itself may have protected against arrhythmia, although in AVID, the ICD offered similar survival rates independent of revascularization (13).

A recent substudy of the large Valsartan in Acute Myocardial Infarction Trial (VAlIANT) (48) demonstrated that SCD risk is highest in the first 30 days after MI complicated by reduced LVEF and/or HF, suggesting that early ICDs might save lives. However, the DINAMIT study, which randomly assigned such patients to receive an ICD or conventional medical therapy, showed no ICD benefit early after MI, possibly due to the low event rate in this small study. It is also possible that the VAlIANT patients with SCD were sicker than the survivors, with competing risks. DINAMIT supports this theory, as the decrease in arrhythmic deaths in the ICD group was offset by an increase in nonarrhythmic cardiac death. Whether further risk stratification or noninvasive measures such as an external wearable vest or an automatic external defibrillator might be more beneficial and cost-effective early after MI (49) is unknown.

2.3. Benefit of the ICD in Patients with Advanced Heart Failure

The benefit of the ICD for primary prevention of SCD in patients with more severe HF has been mixed among the different studies. In SCD-HeFT, analysis of prespecified subgroups showed that while patients with NYHA Class II HF (70% of the study population) showed an absolute reduction of mortality of 12% at 5 years, there was no apparent reduction in risk of death with an ICD for those with Class III HF (30% of the population, hazard ratio 1.16, 97.5% CI 0.84–1.61) (29). Other studies, however, have shown
equal or greater benefit in patients with more advanced HF. Among the secondary prevention trials, most patients had NYHA Class I–II HF, with Class III comprising just 9% of AVID patients, 19% of CASH, and 11% of CIDS. However, meta-analysis of the three trials revealed that patients with the lowest left ventricular ejection fraction (LVEF) and more advanced HF benefited most (18). In a subanalysis of the MADIT II trial, while patients with Class III HF (29% of the population) had overall higher mortality and higher risk of arrhythmic events than those with Class I–II HF, there was no interaction between functional class, ICD treatment, and mortality, implying similar benefit of ICD treatment regardless of NYHA class (50). Further, in the DEFINITE trial, there was a greater benefit for those with Class III HF.

The role of the ICD for patients with Class IV HF has not been well studied, as most trials of standard defibrillators have excluded these individuals. However, the Comparison of Medical Therapy, Pacing, and Defibrillation in Heart Failure (COMPANION) trial (51) randomized patients with NYHA Class III or IV HF, reduced EF, and conduction delays (QRS > 120 ms) to receive conventional medical therapy alone, cardiac resynchronization therapy (CRT) alone, or CRT incorporated in an ICD. While patients in the CRT-alone arm had a 19% risk reduction in the primary end point of death or hospitalization ($P = 0.014$), patients in the CRT plus ICD arm had a significant improvement in total mortality, with a relative reduction of 36%. (A limitation of COMPANION was its lack of power to directly compare CRT with vs without defibrillation.) While the number of Class IV patients was small (14%), the mortality benefit with the combined CRT-ICD device was similar between Class III and IV patients.

Overall, these trials strongly support ICD implantation for secondary prevention in patients with prior life-threatening arrhythmia and as primary prophylaxis for many patients with a low LVEF and CAD and/or HF (52–54). Current ACC/AHA/HRS guidelines reflect the inclusion and exclusion criteria from the landmark studies and are displayed in Table 4 (53, 54). Importantly, the 2008 AHA/ACC/HRS guidelines note that Class IV heart failure may be a “heterogeneous and dynamic state,” requiring a careful individualized approach to decisions about further invasive interventions. ICD therapy is generally not recommended, however, for patients with severe, persistent Class IV symptoms who are not candidates for CRT and are already receiving optimal medical (54). The Centers for Medicare and Medicaid Services has expanded coverage for ICDs for most heart failure patients based on the results of MADIT II and SCD-HeFT and for patients with more Class IV HF meeting criteria for CRT (Table 5) (55).

ICDs are also used to prevent SCD in other high-risk patient subsets, such as those with ion-channel abnormalities [i.e., Brugada syndrome, long QT syndrome (LQTS)] or other structural heart disease (i.e., RV dysplasia and hypertrophic cardiomyopathy). Although prospective randomized trials in these rare conditions are not likely to be pursued (56), case series
Table 4
ACC/AHA/HRS guidelines (2008): indications for ICD therapy

<table>
<thead>
<tr>
<th>Class I indications</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. ICD therapy is indicated in patients who are survivors of cardiac arrest due to VF or hemodynamically unstable sustained VT after evaluation to define the cause of the event and to exclude any completely reversible causes <em>(Level of Evidence: A)</em></td>
<td></td>
</tr>
<tr>
<td>2. ICD therapy is indicated in patients with structural heart disease and spontaneous sustained VT, whether hemodynamically stable or unstable <em>(Level of Evidence: B)</em></td>
<td></td>
</tr>
<tr>
<td>3. ICD therapy is indicated in patients with syncope of undetermined origin with clinically relevant, hemodynamically significant sustained VT or VF induced at electrophysiological study <em>(Level of Evidence: B)</em></td>
<td></td>
</tr>
<tr>
<td>4. ICD therapy is indicated in patients with LVEF less than 35% due to prior MI who are at least 40 days post-MI and are in NYHA functional Class II or III <em>(Level of Evidence: A)</em></td>
<td></td>
</tr>
<tr>
<td>5. ICD therapy is indicated in patients with nonischemic DCM who have an LVEF ≤ 35% and who are in NYHA functional Class II or III <em>(Level of Evidence: B)</em></td>
<td></td>
</tr>
<tr>
<td>6. ICD therapy is indicated in patients with LV dysfunction due to prior MI who are at least 40 days post-MI, have an LVEF less than 30%, and are in NYHA functional Class I <em>(Level of Evidence: A)</em></td>
<td></td>
</tr>
<tr>
<td>7. ICD therapy is indicated in patients with nonsustained VT due to prior MI, LVEF less than 40%, and inducible VF or sustained VT at electrophysiological study <em>(Level of Evidence: B)</em></td>
<td></td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Class IIa indications</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. ICD implantation is reasonable for patients with unexplained syncope, significant LV dysfunction, and nonischemic DCM <em>(Level of Evidence: C)</em></td>
<td></td>
</tr>
<tr>
<td>2. ICD implantation is reasonable for patients with sustained VT and normal or near-normal ventricular function <em>(Level of Evidence: C)</em></td>
<td></td>
</tr>
<tr>
<td>3. ICD implantation is reasonable for patients with HCM who have one or more major risk factors for SCD <em>(Level of Evidence: C)</em></td>
<td></td>
</tr>
<tr>
<td>4. ICD implantation is reasonable for the prevention of SCD in patients with ARVD/C who have one or more risk factors for SCD <em>(Level of Evidence: C)</em></td>
<td></td>
</tr>
<tr>
<td>5. ICD implantation is reasonable to reduce SCD in patients with long QT syndrome who are experiencing syncope and/or VT while receiving beta-blockers <em>(Level of Evidence: B)</em></td>
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*(Continued)*
<table>
<thead>
<tr>
<th>Class I indications</th>
<th></th>
</tr>
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<tbody>
<tr>
<td>6. ICD implantation is reasonable for nonhospitalized patients awaiting transplantation <em>(Level of Evidence: C)</em></td>
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<tr>
<td>7. ICD implantation is reasonable for patients with Brugada syndrome who have had syncope <em>(Level of Evidence: C)</em></td>
<td></td>
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<tr>
<td>8. ICD implantation is reasonable for patients with Brugada syndrome who have documented VT that has not resulted in cardiac arrest <em>(Level of Evidence: C)</em></td>
<td></td>
</tr>
<tr>
<td>9. ICD implantation is reasonable for patients with catecholaminergic polymorphic VT who have syncope and/or documented sustained VT while receiving beta-blockers <em>(Level of Evidence: C)</em></td>
<td></td>
</tr>
<tr>
<td>10. ICD implantation is reasonable for patients with cardiac sarcoidosis, giant cell myocarditis, or Chagas disease <em>(Level of Evidence: C)</em></td>
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</table>

<table>
<thead>
<tr>
<th>Class IIb indications</th>
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<tbody>
<tr>
<td>1. ICD therapy may be considered in patients with nonischemic heart disease who have an LVEF ≤ 35% and who are in NYHA functional Class I <em>(Level of Evidence: C)</em></td>
<td></td>
</tr>
<tr>
<td>2. ICD therapy may be considered for patients with long QT syndrome and risk factors for SCD <em>(Level of Evidence: B)</em></td>
<td></td>
</tr>
<tr>
<td>3. ICD therapy may be considered in patients with syncope and advanced structural heart disease in whom thorough invasive and noninvasive investigations have failed to define a cause <em>(Level of Evidence: C)</em></td>
<td></td>
</tr>
<tr>
<td>4. ICD therapy may be considered in patients with a familial cardiomyopathy associated with sudden death <em>(Level of Evidence: C)</em></td>
<td></td>
</tr>
<tr>
<td>5. ICD therapy may be considered in patients with LV noncompaction <em>(Level of Evidence: C)</em></td>
<td></td>
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</tbody>
</table>

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<thead>
<tr>
<th>Class III indications</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. ICD therapy is not indicated for patients who do not have a reasonable expectation of survival with an acceptable functional status for at least 1 year, even if they meet ICD implantation criteria specified in the Class I, IIa, and IIb recommendations above <em>(Level of Evidence: C)</em></td>
<td></td>
</tr>
<tr>
<td>2. ICD therapy is not indicated for patients with incessant VT or VF <em>(Level of Evidence: C)</em></td>
<td></td>
</tr>
<tr>
<td>3. ICD therapy is not indicated in patients with significant psychiatric illnesses that may be aggravated by device implantation or that may preclude systematic follow-up <em>(Level of Evidence: C)</em></td>
<td></td>
</tr>
<tr>
<td>4. ICD therapy is not indicated for NYHA Class IV patients with drug-refractory congestive heart failure who are not candidates for cardiac transplantation or CRT-D <em>(Level of Evidence: C)</em></td>
<td></td>
</tr>
</tbody>
</table>
5. ICD therapy is not indicated for syncope of undetermined cause in a patient without inducible ventricular tachyarrhythmias and without structural heart disease (Level of Evidence: C)

6. ICD therapy is not indicated when VF or VT is amenable to surgical or catheter ablation (e.g., atrial arrhythmias associated with the Wolff–Parkinson–White syndrome, RV or LV outflow tract VT, idiopathic VT, or fascicular VT in the absence of structural heart disease) (Level of Evidence: C)

7. ICD therapy is not indicated for patients with ventricular tachyarrhythmias due to a completely reversible disorder in the absence of structural heart disease (e.g., electrolyte imbalance, drugs, or trauma) (Level of Evidence: B)

---

Class I: Conditions for which there is evidence and/or general agreement that a given procedure or treatment is beneficial, useful, and effective.
Class II: Conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of a procedure or treatment.
Class IIa: The weight of evidence/opinion is in favor of usefulness/efficacy.
Class IIb: The usefulness/efficacy is less well established by evidence/opinion.
Class III: Conditions for which there is evidence and/or general agreement that a procedure/treatment is not useful/effective and in some cases may be harmful.

Level of Evidence A: Data derived from multiple randomized clinical trials or meta-analyses.
Level of Evidence B: Data derived from a single randomized trial or nonrandomized studies.
Level of Evidence C: Consensus opinion of experts, case studies, or standard of care in the absence of the above.
Table 5
Centers for Medicare and Medicaid Services (CMS) coverage requirements for ICD implantation (53)

(1) Patients with IDCM, documented prior MI, NYHA Class II and III heart failure, and measured LVEF ≤ 0.35
(2) Patients with NIDCM > 3 months, NYHA Class II or III heart failure, and measured LVEF ≤ 0.35
(3) Patients who meet all current CMS coverage requirements for a CRT device and have NYHA Class IV heart failure
For all groups, patients must not have:

Cardiogenic shock or symptomatic hypotension while in a stable baseline rhythm
A CABG or a PTCA within the past 3 months
An acute MI within the past 40 days
Clinical symptoms or findings that would make them a candidate for coronary revascularization
Irreversible brain damage from preexisting cerebral disease
Any disease, other than cardiac disease associated with a likelihood of survival less than 1 year

Abbreviations: CABG, coronary artery bypass grafting; CAD, coronary artery disease; ICD, implantable cardioverter-defibrillator; IDCM, ischemic dilated cardiomyopathy; LVEF, left ventricular ejection fraction; NIDCM, nonischemic dilated cardiomyopathy; NSVT, nonsustained ventricular tachycardia; NYHA, New York Heart Association; PTCA, percutaneous transluminal coronary angioplasty; PVC, premature ventricular contraction; VF, ventricular fibrillation; VT, ventricular tachycardia

show efficacy of the ICD in patients with LQTS (57), Brugada syndrome (58), hypertrophic cardiomyopathy (59, 60), and arrhythmogenic RV dysplasia (61). Current guidelines support its use in selected patients with these disorders (54).

An evaluation of the cost-effectiveness of the ICD using a Markov model (62) revealed that prophylactic ICD implantation was both more effective and more expensive than control therapy. Consistent with the lower number needed to treat, the populations showing greatest cost-effectiveness of the ICD were those in MADIT I and MUSTT, with approximately $25,000 per life-year added. The ICD was less cost-effective in the MADIT II and SCD-HeFT populations, with an estimated cost of $40,000–$50,000 per life-year added, respectively. Direct data from SCD-HeFT showed a cost-effectiveness of just $30,000–$40,000, assuming that the mortality benefits extend to at least 8 years (63). Costs per quality-adjusted life-year ranged from $34,000 to $70,000 (62). Not surprisingly, lowering the cost of the device or increasing longevity would improve cost-effectiveness (62). These costs are well within the range considered acceptable to society (64).
3. ICD FUNCTION AND TECHNOLOGY

3.1. Treatment

The three main functions of the ICD are detection of arrhythmia (tachycardia or bradycardia), delivery of appropriate electrical therapy (high-voltage shock or low-energy pacing), and storage of diagnostic information, including electrograms and details of treated episodes. The device consists of two components: the pulse generator and the lead (electrode) system. Current ICDs are only slightly larger than a pacemaker (25–45 cm³) and are similarly implanted in a subcutaneous pectoral pocket (65). Leads are inserted transvenously through the subclavian, axillary, or cephalic vein into the right ventricular apex (66) (and the right atrial appendage for atrial sensing/pacing in dual-chamber systems). Lead characteristics are described in Table 6. Chest roentgenogram demonstrating positioning of the leads and generator is shown in Fig. 1.

<table>
<thead>
<tr>
<th>Type</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Fixation mechanism</strong></td>
<td>Tines become lodged in trabeculations. Lower pacing threshold, higher dislodgement rate</td>
</tr>
<tr>
<td>Transvenous, passive fixation</td>
<td>Helix or screw extends into the endocardial tissue to secure lead. Higher pacing thresholds, lower dislodgement rate</td>
</tr>
<tr>
<td>Transvenous, active fixation</td>
<td>Surgically implanted on outer surface of heart. Typically higher pacing thresholds. May be used for patients with mechanical tricuspid valve, those with difficult coronary sinus access requiring LV pacing, or those with high defibrillation thresholds requiring epicardial patches</td>
</tr>
<tr>
<td>Epicardial</td>
<td></td>
</tr>
<tr>
<td><strong>Insulation</strong></td>
<td>Easier to pass, more rigid, less durable, thinner</td>
</tr>
<tr>
<td>Polyurethane</td>
<td>Higher coefficient of friction, therefore less “slippery,” less rigid, more durable, thicker</td>
</tr>
<tr>
<td>Silicone</td>
<td></td>
</tr>
<tr>
<td><strong>Configuration</strong></td>
<td>Sensing and pacing between the lead tip (distal electrode) and the pacemaker pulse generator. Bigger “antenna,” therefore more prone to oversensing</td>
</tr>
<tr>
<td>Unipolar</td>
<td></td>
</tr>
<tr>
<td>Bipolar</td>
<td>Sensing and pacing between two electrodes separated by several millimeters (proximal and distal) located on a single lead. Smaller “antenna,” therefore less prone to oversensing</td>
</tr>
<tr>
<td><strong>Connector type</strong></td>
<td>Lead connector and generator header must be compatible. IS-1 is the most common connector in use today for pace/sense connection; DF-1 is most commonly used for high-voltage connections</td>
</tr>
<tr>
<td>IS-1 bipolar, IS-1 unipolar, DF-1, IS-4</td>
<td></td>
</tr>
</tbody>
</table>
Fig. 1. Chest roentgenograms of standard ICD systems are shown. (A) Single-chamber ICD; (B) Dual-chamber ICD; (C) Biventricular ICD. Single-chamber systems consist of a right ventricular (RV) electrode (white arrow head). Dual-chamber systems have a right atrial (black arrow head) and RV electrode, while biventricular systems have a third lead positioned in the coronary sinus (black arrow).

The modern ICD combines high-energy defibrillation with two other electrical therapies, low-energy cardioversion and antitachycardia pacing (ATP), to terminate ventricular arrhythmias. Figure 2 shows a stored electrogram of a rapid ventricular tachycardia terminated with a high-energy shock. ATP terminates VT without delivering a painful shock by pacing at a rate faster than the intrinsic tachycardia, entering and interrupting the re-entrant circuit (67, 68), as shown in Figs. 3 and 4. As shown in Fig. 5, demonstrating device programming options, tiered therapy allows programming these electrical modalities to treat tachycardias with rates within defined zones of detection (69). For example, a slower arrhythmia might be treated by ATP followed by low-energy and then high-energy shock, if needed. VF falls in a faster zone,

Fig. 2. Stored electrogram of a shock-terminated rapid ventricular tachycardia. Top tracing represents atrial electrogram, second tracing represents ventricular electrogram. Marker annotation describes device-defined event; “AS,” atrial sensed event; “VS,” ventricular sensed event; “CD,” charge delivered.
Fig. 3. Surface tracing recorded during standard ICD testing demonstrating anti-tachycardia pacing. Shown are surface leads I and V₁. One short burst (four beats) of antitachycardia pacing (ATP) is able to terminate a ventricular tachycardia (VT), restoring normal sinus rhythm (NSR).

Fig. 4. (A) Stored electrogram of ATP-terminated ventricular tachycardia. “VT Rx 1 Burst” marks the beginning of delivered ATP. Top tracing represents atrial electrogram, second tracing represents ventricular electrogram. Marker annotation describes device-defined event; “AS,” atrial sensed event; “TS,” ventricular sensed event in “tachy” zone; “TD,” arrhythmia has met tachycardia detection criteria; “TP,” antitachycardia pacing, i.e., ATP; “VS,” ventricular sensed event. (B) Text description of the same event with quantified cycle length and interval stability.
prompting high-energy defibrillation. While ATP was initially used only to treat slower, stable VT, the recent Pacing Reduces Shocks for Fast VT II trial (PainFREE Rx II) (70) found that empirically programmed ATP delivered during device charging (a function available in some devices) could treat faster VTs, reducing shocks by 70% with no increase in adverse outcomes. Attempted ATP can accelerate VT into VF, leading to defibrillation.

### 3.2. Detection

As shown in Fig. 5, the primary criterion for arrhythmia detection is heart rate. However, other rhythms may result in heart rates above the defined rate cutoff, resulting in inappropriate shocks, occurring in up to 15% of patients with ICDs, and representing more than one-third of shocks received, regardless of ICD indication (71). Figure 6 shows an example of atrial fibrillation with a rapid ventricular response triggering a high-energy shock. Programmable detection criteria, described in Table 7, can improve discrim-
Table 7

Arrhythmia discrimination

<table>
<thead>
<tr>
<th>Discriminator</th>
<th>How it works</th>
<th>Clinical implications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset</td>
<td>Measures how sudden the arrhythmia onset was. Withholds therapy if onset is gradual</td>
<td>Effective at discriminating sinus tachycardia, which accelerates gradually, from VT, of usually sudden onset</td>
</tr>
<tr>
<td>Stability</td>
<td>Measures beat-to-beat variation in arrhythmia cycle length. Withholds therapy for irregular rhythms</td>
<td>Effective at discriminating between VT (regular) and rapid atrial fibrillation (irregular)</td>
</tr>
<tr>
<td>Morphology</td>
<td>Compares the QRS morphology during tachycardia to the baseline morphology. Withholds therapy if the tachycardia morphology is similar</td>
<td>Can discriminate supraventricular arrhythmias from VT</td>
</tr>
<tr>
<td>V&gt;A</td>
<td>Compares the measured ventricular rate to the atrial rate. Treats as VT if ventricular rate greater than atrial</td>
<td>Can identify VT rapidly</td>
</tr>
<tr>
<td>AV interval</td>
<td>Evaluates the AV during tachycardia to differentiate sinus or atrial tachycardias from VT (short R-P/long P-R)</td>
<td>Can differentiate supraventricular tachycardias from VT with retrograde conduction</td>
</tr>
<tr>
<td>(dual-chamber)</td>
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VT – ventricular tachycardia.

inination between atrial and ventricular arrhythmias. For example, the device can be programmed to withhold therapy for a set amount of time if the onset of tachycardia is gradual, as is the case with sinus tachycardia (an example of this programming is shown in Fig. 5) In most, although not all studies, the dual-chamber ICD, incorporating a right atrial lead, can further decrease inappropriate shocks due to rapid supraventricular rhythms or physiologic sinus tachycardia using specific algorithms (72–74), such as those which analyze the relative timing of atrial and ventricular electrograms. Or, when the atrial sensed rate is faster than the ventricular rate, as in atrial fibrillation, ICD discharge may be inhibited (74).

Inappropriate detection, in which the device senses noncardiac electrical events, can occur due to either lead insulation disruption, as shown in Fig. 7, or electromagnetic interference (EMI) in the environment and can also result in inappropriate shock. Sources of environmental EMI that can be sensed by the ICD include medical procedures using cautery, radiofrequency abla-
Observations

- V. pacing lead impedance is > 2000 ohms.

- 726 V-V intervals have been sensed at 120 or 130ms since Dec 28, 2006 21:08:40. Check for sensing issues (e.g. double counting of R-waves, lead fracture, loose set screw).

- Patient Alert triggered - V. Pacing lead impedance >2000 ohms.

- 2 VF episodes were longer than 30 seconds. Most recent was Episode #12, Dec 29, 2006 11:17:11.

Fig. 7. (A) Stored electrogram from shock delivered to otherwise asymptomatic patient. Electrogram clearly shows high-frequency variation in the signal consistent with electrical noise (noncardiac electrical activity) triggering the shock. (B) Observations recorded by the ICD at the time of interrogation demonstrating the electrogram above. High-pacing impedance implies lead fracture. Multiple, very short (120–130 ms) V–V intervals suggest inappropriate sensing of noise. VS, ventricular sensing; FS, sensing of ventricular signal in VF zone; TS, sensing of ventricular signal in VT zone; FD, detection criteria met to declare VF episode in progress.

3.3. Management Issues in the Care of the CHF Patient with an ICD

3.3.1. Programming of the ICD: Bradycardia Pacing

One concern following MADIT II was the higher rate of HF in the ICD group (28, 78). The later Dual Chamber and VVI Implantable Defibrillator (DAVID) trial (79), which randomized patients receiving ICDs to either dual- or single-chamber devices, showed higher incidence of HF with the dual-chamber device, and it was later determined that the percent of RV pacing was more important than pacing mode in determining adverse out-
come (HF or death) (80). The mechanism by which RV pacing effectively causes iatrogenic dyssynchrony likely explains the higher incidence of HF in ICD-treated patients in MADIT II, as this concern was not appreciated at that time. Based on these and other data showing deleterious effects of RV pacing (81, 82), dual-chamber devices are now programmed to minimize RV pacing, using recently developed algorithms incorporating AV search hysteresis to provide atrial-based pacing (83, 84).

3.4. Management of Frequent Shocks

While an ICD shock effectively terminates ventricular tachyarrhythmias and improves mortality, shocks are painful. One survey asking patients to describe the sensation of a shock yielded responses of “a blow to the body,” “a punch in the chest,” “being hit by a truck,” “kicked by a mule,” and “putting a finger in a light socket” (85). In the Canadian Implantable Defibrillator Study (CIDS), patients who received 0–4 shocks had significant improvement in QOL over time, but those with 5 or more shocks did not improve (86). Similarly, in the AVID trial, the occurrence of even one shock was associated with reduction in mental well-being and physical function, even after controlling for multiple clinical factors such as HF; the reduction in QOL grew greater as shocks were more frequent (87). Thus, decreasing shock frequency is critical to maintaining quality of life in patients with ICDs.

In patients with ischemic cardiomyopathy, increases in BNP predict appropriate ICD shocks (88), and therapies effective for HF decrease incidence of sudden cardiac death (9, 10, 88). These findings suggest that the first step in the prevention of frequent shocks for patients with HF is maximization of heart failure therapy. For patients requiring specific antiarrhythmic therapy, amiodarone has been shown to decrease appropriate ICD shocks (89) and has neutral effects on survival in patients with heart failure (29, 90). In patients suffering from shocks for atrial fibrillation, dofetilide also does not worsen survival or heart failure in patients with HF (91) and can be used safely as long as renal function and QT interval are normal. Class I antiarrhythmics, both Ia agents, such as quinidine and procainamide, and Ic agents, such as flecainide, increase mortality in patients with HF (92).

3.5. Device Malfunction

With the expanse in device technology has come an increase in malfunctions resulting in advisories and recalls and with increasing indications for the ICD, the number of patients affected by advisories is expected to increase (93). Actual device malfunction requiring device replacement, which can be due to either physical or mechanical factors or software failure, is estimated to be about 20 per 1000 implants (94–96). Like ICD generators, leads may also experience performance concerns, most commonly due to insula-
tion degradation or lead fractures (97, 98). Fortunately, death due to device malfunction is rare (59, 93, 99, 100). Each major ICD manufacturer has experienced product advisories and malfunctions (101).

Patients and physicians faced with an advisory must weigh the risks of malfunction, the nature of the specific advisory, the patient’s underlying arrhythmia and clinical condition, and the risks of replacement. The Heart Rhythm Society has published recommendations for management of device performance issues (94), emphasizing greater transparency in postmarket surveillance, analysis, and reporting as well as cooperation among industry, regulators, and physicians. Further, ongoing efforts to improve detection, reporting, and management of device performance and malfunction information will improve patient safety (102). Specific device algorithms to automatically measure performance-related variables such as lead and battery impedance on a regular basis, automated patient and physician alert systems, and the advent of remote ICD monitoring will further improve patient safety (103).

3.6. Management of the ICD in End-of-Life Care

HF is a progressive disease with many interventions providing palliative, but not curative, benefit. The ACC/AHA heart failure guidelines stress that the possible reasons and process for potential deactivation of defibrillator features should be discussed long before functional capacity or outlook for survival is severely reduced (54). The dying process of patients with ICDs can be accompanied by multiple shocks, to the distress of patient and family (104, 105), and conversations between physicians and patients regarding the option of deactivation of the shocking functions have taken place only rarely, even among patients who have chosen a “do not resuscitate” order (105). There is solid legal basis for deactivating an ICD should this be the patient’s wishes (106), and multidisciplinary strategies to identify patients with terminal illnesses and initiate withdrawal of ICD shock therapy as part of a comprehensive comfort care approach can decrease painful shocks as the patient ultimately succumbs to heart failure or another terminal illness (107).

4. CONCLUSIONS

Preventing sudden SCD remains a major challenge for physicians treating patients with heart failure. ICDs are a remarkable technology, clinically and scientifically proven to improve survival in appropriately selected patients. Although the precise indications for ICD implantation continue to evolve, the therapy will undoubtedly remain an important complement to comprehensive medical treatment of patients with heart failure.
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35. Buxton AE. Should everyone with an ejection fraction less than or equal to 30% receive an implantable cardioverter-defibrillator? Not everyone with an ejection fraction < or = 30% should receive an implantable cardioverter-defibrillator. Circulation 2005; 111:2537–49; discussion 2537–49.


