Maximizing Survival Benefit With Primary Prevention Implantable Cardioverter-Defibrillator Therapy in a Heart Failure Population

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Background—Although implantable cardioverter-defibrillator (ICD) therapy reduces mortality in moderately symptomatic heart failure patients with an ejection fraction $\leq 35\%$, many such patients do not require ICD shocks over long-term follow-up.

Methods and Results—Using a modification of a previously validated risk prediction model based on routine clinical variables, we examined the relationship between baseline predicted mortality risk and the relative and absolute survival benefits of ICD treatment in the primary prevention Sudden Cardiac Death in Heart Failure Trial (SCD-HeFT). In the placebo arm, predicted 4-year mortality grouped into 5 equal-sized risk groups varied from 12% to 50% ($c$ statistic $= 0.71$), whereas the proportion of SCD in those same risk groups decreased from 52% to 24% of all deaths. ICD treatment decreased relative risk of SCD by 88% in the lowest-risk group versus 24% in the highest-risk group ($P = 0.009$ for interaction) and decreased relative risk of total mortality by 54% in the lowest-risk group versus no benefit (2%) in the highest-risk group ($P = 0.014$ for interaction). Absolute 4-year mortality reductions were 6.6%, 8.8%, 10.6%, 14.0%, and 4.9% across risk quintiles. In highest-risk patients (predicted annual mortality $> 20\%$), no benefit of ICD treatment was seen. Projected over each patient’s predicted lifespan, ICD treatment added 6.3, 4.1, 3.0, 1.9, and 0.2 additional years of life in the lowest- to highest-risk groups, respectively.

Conclusions—A clinical risk prediction model identified subsets of moderately symptomatic heart failure patients in SCD-HeFT in whom single-lead ICD therapy was of no benefit and other subsets in which benefit was substantial. (Circulation. 2009; 120:835-842.)

Key Words: arrhythmias ■ death, sudden ■ defibrillators, implantable ■ electrophysiology ■ heart failure ■ electrophysiology ■ survival

Both the 2005 clinical practice guidelines on the management of chronic heart failure$^1$ and the 2008 guidelines on pacemakers and implanted devices from the American College of Cardiology and the American Heart Association rate as Class I the use of prophylactic implantable cardioverter-defibrillator (ICD) therapy in heart failure (HF) patients with New York Heart Association (NYHA) class 2 to 3 symptoms and ejection fraction $\leq 35\%$, suggesting that ICDs should routinely be placed in such patients as a part of evidence-based medicine. $^2$ However, actual use of ICD treatment in this large population appears to have lagged behind these recommendations.$^3$ Several reasons for this slow adoption can be offered, but 2 may be particularly relevant. First, patients with chronic HF and a depressed ejection fraction are prognostically heterogeneous for both overall mortality and sudden death mortality. $^4$ Second, because only $\approx 20\%$ to 25% of primary prevention ICD patients receive appropriate shocks within 5 years of implantation, many nominally eligible patients appear not to actually need this therapy. $^5,6$ Although much interest exists in developing various novel testing strategies to identify subsets of patients most likely to benefit, to date, none of these prediction strategies has proved

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sufficiently discriminative or received independent validation for use in general clinical practice.7

The Seattle Heart Failure Model (SHFM) is a multivariable risk model that predicts both all-cause and cause-specific mortality in HF patients. The model was developed in the Prospective Randomized Amlodipine Survival Evaluation (PRAISE I) trial cohort and prospectively validated in 5 additional cohorts derived from both large clinical trials and outpatient community practice settings in the United States and Europe.8,9 The SHFM uses routinely collected clinical variables to make predictions without requiring specialized or costly testing. We postulated that ICDs may be more beneficial in relatively lower-risk patients, in whom the predominant mode of death would be sudden cardiac death (SCD) and in whom SCD may more often be due to ventricular tachycardia/ventricular fibrillation (more amenable to ICD therapy) than electromechanical dissociation, pulmonary embolus, or ventricular tachycardia storm (less amenable to ICD therapy). We used patient-level data from the Sudden Cardiac Death in Heart Failure5 (SCD-HeFT) randomized trial to test the hypothesis that among patients with moderate systolic HF, the SHFM-predicted risks could identify subsets of patients in whom clinically relevant differences in single-lead ICD treatment benefit would be present.

### Methods

#### Study Patients

Patients were eligible for SCD-HeFT if they had NYHA class 2 or 3 HF with an ejection fraction ≤35%.5 Compared with medical therapy alone, randomization to single-lead ICD therapy (829 patients) reduced total mortality by 23% (P=0.007) in the overall trial, whereas amiodarone had no benefit. Of the 2521 enrolled patients, 38 were excluded from the present analysis because of missing baseline variables for SHFM calculation.

#### Calculation of the SHFM

The SHFM is a validated risk prediction model based on routinely collected clinical variables.6 In SCD-HeFT, most SHFM variables were available, including age, gender, ischemic origin, systolic blood pressure, ejection fraction, medication use (angiotensin-converting enzyme inhibitor, angiotensin receptor blocker, β-blocker, statin, and daily diuretic dose), and serum sodium, but data were not available on allopurinol use, total cholesterol, hemoglobin, percent lymphocytes, or uric acid. To account for the impact of these missing variables, we used a separate derivation cohort of 10 038 HF patients from 5 other studies including 23 037 patient-years of observation10–14 to develop a modified version of the SHFM that included the SHFM predictor variables available in the SCD-HeFT population and additional prognostic variables using the Cox proportional-hazards model and previously described methods.6 This new model, SHFM-D (differential ICD benefit), is abbreviated SHFM here for simplicity. The final model, SHFM-D, derived in the separate derivation data set, included the original SHFM variables of age, gender, systolic blood pressure, ischemic origin, NYHA class, ejection fraction, angiotensin-converting enzyme inhibitor use, angiotensin receptor blocker use, β-blocker use, statin use, furosemide equivalent daily dose in milligrams per kilogram, and serum sodium.
was estimated by the Gompertz method using the SHFM estimated total life expectancy: reduction for ICD versus placebo at 4 years. Total life expectancy for 4 years to save 1 life was calculated as 1 divided by absolute risk estimates of annual survival through year 5. Number needed to treat applied prospectively to patients in SCD-HeFT to provide individual years, fit using a third-degree polynomial curve. The SHFM was then derived from measured survival in the derivation cohort between 0 and 5 years. Vital status for all patients was known.

Figure 1. Survival predicted by the SHFM and the observed (Kaplan–Meier) survival are shown for quintiles of the placebo group at 1 and 4 years. The predicted and observed mortality at 4 years was 71%. The diagonal line is the line of identity.

As a percentage of all deaths, the proportion of SCDs in the Ascertainment of Mortality A centralized adjudication committee classified modes of death in SCD-HeFT. For this analysis, the primary outcomes were all-cause mortality, SCD, and all other deaths, which include pump failure deaths (non-SCD). Patients who underwent transplant (n = 61) or crossed over to an ICD (n = 188) were analyzed using intention-to-treat principles. Median follow-up was 3.8 years (range, 2.1 to 6.0 years). Vital status for all patients was known.

Statistical Analysis The SHFM regression coefficients derived on the external data set were used to calculate a risk score and predicted survival for each SCD-HeFT patient using each individual’s specific values of the variables included in the SHFM. Quintiles of SHFM-predicted survival were plotted against observed (Kaplan–Meier) survival for the placebo group at 1 and 4 years. The ability of the risk score to provide different predictions for patients who lived versus those who died (ie, discrimination) was evaluated using the c statistic for time-to-event data. Confidence intervals (CIs) for c statistics were generated by drawing 200 bootstrap samples from the placebo group, fitting a Cox model using the SHFM risk score, and calculating the c statistic for each sample. The CI for the c statistic was then calculated as ±1.96 times the SD of the 200 c statistics. The SHFM score and randomization group (ICD, amiodarone, or placebo) were entered into a Cox model to determine the risk-adjusted effects (hazard ratio [HR], hereafter called relative risk) of ICD and amiodarone therapy on all-cause and cause-specific mortality. We used the same SHFM risk score (derived using all-cause mortality in the external data set) for examining all 3 outcomes (all-cause mortality, SCD, and all other deaths) rather than building a separate cause-specific model for each outcome. Potential interaction (effect modification) between SHFM-predicted mortality and randomization group was evaluated by adding multiplicative interaction terms (SHFM score × amiodarone, SHFM score × ICD) to the Cox model as continuous variables. Potential interaction between SHFM-predicted risk and ICD therapy was further evaluated in stratified analyses by quintiles of SHFM-predicted mortality. We used Statview 5 (SAS Institute, Inc, Cary, NC) for the external derivation of the SHFM and SAS version 8.2 for analyses in SCD-HeFT. Statistical significance was defined as α < 0.05 (2 tailed).

The authors had full access to and take full responsibility for the integrity of the data. All authors have read and agree to the manuscript as written.

Results The baseline variables in ascending quintiles of the Seattle HF score (lower to higher risk) are shown in Table 1. QRS width and 6-minute walk distance were not part of the model but showed higher risk values with higher risk quintiles. The SHFM had excellent model calibration, with overall 4-year predicted and actual survival of 71% (Figure 1). The c statistic was 0.71 in the external derivation data set and 0.71 (95% CI, 0.69 to 0.73) in the SCD-HeFT cohort. Although the SHFM was designed to estimate all-cause mortality, when applied to the SCD-HeFT data, it was more accurate in predicting pump failure death (c statistic = 0.79; 95% CI, 0.73 to 0.82) and non-SCD (which includes pump failure death; c statistic = 0.74; 95% CI, 0.72 to 0.77) but still discriminative for predicting SCD (c statistic = 0.66; 95% CI, 0.63 to 0.70).

As a percentage of all deaths, the proportion of SCDs in the placebo group decreased with increasing annual SHFM-predicted mortality, from 52% in low-risk patients (quintiles

<table>
<thead>
<tr>
<th>Quintile (n)</th>
<th>Total Deaths, n</th>
<th>Placebo Mortality Rate, Events per 1000 Person-y</th>
<th>Relative Risk Comparing ICD Therapy and Placebo</th>
<th>Relative Risk (95% CI) Comparing ICD Therapy and Placebo Using a Linear Interaction Term in the Model (SHFM × ICD)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>I (497)</td>
<td>42</td>
<td>33</td>
<td>0.46 (0.20–1.04)</td>
<td>0.46 (0.30–0.70)</td>
</tr>
<tr>
<td>II (497)</td>
<td>73</td>
<td>51</td>
<td>0.48 (0.26–0.92)</td>
<td>0.19 (0.03–0.73)</td>
</tr>
<tr>
<td>III (496)</td>
<td>106</td>
<td>73</td>
<td>0.63 (0.39–1.01)</td>
<td>0.54 (0.30–0.90)</td>
</tr>
<tr>
<td>IV (496)</td>
<td>178</td>
<td>113</td>
<td>0.69 (0.46–1.03)</td>
<td>0.07 (0.57–0.86)</td>
</tr>
<tr>
<td>V (497)</td>
<td>256</td>
<td>176</td>
<td>0.98 (0.73–1.32)</td>
<td>0.89 (0.63–1.14)</td>
</tr>
</tbody>
</table>

*Quintile point estimate, 95% CI, and P value derived from the SHFM, ICD, and SHFM × ICD interaction terms in the Cox model using continuous variables.
The Kaplan–Meier survival curves according to both ICD treatment and the 5 quintiles of SHFM-estimated risk are shown in Figure 3. The ICD had a survival advantage in quintiles 1 through 4, but in quintile 5, the survival curves were not different at 4 years.

Absolute 4-year reductions in mortality with ICD treatment were 6.6%, 8.8%, 10.6%, 14.0%, and −4.9% across SHFM quintiles 1 through 5, respectively. The number needed to treat to add 1 year of life over 4 years of follow-up was 15.2, 11.4, 9.4, 7.1, and −20.4 (no benefit in quintile 5; Figure 4). Treatment with an ICD added 6.3, 4.1, 3.0, 1.9, and 0.2 additional years of life in the low to high risk quintiles when projected over the patients’ predicted lifespans (Figure 5). Assuming a 7-year ICD battery life, for each ICD, one would

Table 3. SCD According to ICD Therapy by Quintiles of SHFM-Predicted Risk

<table>
<thead>
<tr>
<th>Quintile (n)</th>
<th>Total Deaths, n</th>
<th>Placebo Mortality Rate, Events per 1000 Person-y</th>
<th>Relative Risk Comparing ICD Therapy and Placebo</th>
<th>Relative Risk (95% CI) Comparing ICD Therapy and Placebo Using a Linear Interaction Term in the Model (SHFM×ICD)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>I (497)</td>
<td>17</td>
<td>15</td>
<td>0.12 (0.016–0.97)</td>
<td>0.047</td>
</tr>
<tr>
<td>II (497)</td>
<td>29</td>
<td>29</td>
<td>0.12 (0.028–0.53)</td>
<td>0.005</td>
</tr>
<tr>
<td>III (496)</td>
<td>42</td>
<td>29</td>
<td>0.47 (0.21–1.08)</td>
<td>0.076</td>
</tr>
<tr>
<td>IV (496)</td>
<td>52</td>
<td>46</td>
<td>0.30 (0.13–0.69)</td>
<td>0.005</td>
</tr>
<tr>
<td>V (497)</td>
<td>52</td>
<td>41</td>
<td>0.76 (0.39–1.47)</td>
<td>0.42</td>
</tr>
</tbody>
</table>

*Quintile point estimate, 95% CI, and P value derived from the SHFM, ICD, and SHFM×ICD interaction terms in the Cox model using continuous variables.
add 2.0, 1.9, 1.8, 1.5, and 0.2 years of life across the 5 quintiles. The years needed to treat to add 1 year of life with an ICD were 4.0 for the overall trial and 3.5, 3.8, 3.9, 4.6, and 21.5 in the low to high risk quintiles.

Amiodarone had no significant effect on all-cause mortality, SCD, or non-SCD. There was no significant interaction of amiodarone with SHFM score for any mode of death (data not shown).

Discussion

The primary finding of our study is that an externally derived risk stratification model containing only routine clinical variables can accurately partition and quantify the treatment benefit from primary prevention ICD therapy in systolic HF patients. In particular, the model identified subsets with large differences in both relative and absolute risk reduction. For example, numbers needed to treat for 4 years to save 1 life

### Table 4. Non-SCD According to ICD Therapy by Quintiles of SHFM-Predicted Risk

<table>
<thead>
<tr>
<th>Quintile (n)</th>
<th>Total Deaths, n</th>
<th>Placebo Mortality Rate, Events per 1000 Person-y</th>
<th>Relative Risk Comparing ICD Therapy and Placebo</th>
<th>Relative Risk (95% CI) Comparing ICD Therapy and Placebo Using a Linear Interaction Term in the Model (SHFM×ICD)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>I (497)</td>
<td>25</td>
<td>18</td>
<td>0.76 (0.29–1.96)</td>
<td>0.57</td>
</tr>
<tr>
<td>II (497)</td>
<td>44</td>
<td>22</td>
<td>0.90 (0.42–1.95)</td>
<td>0.79</td>
</tr>
<tr>
<td>III (496)</td>
<td>64</td>
<td>44</td>
<td>0.73 (0.41–1.30)</td>
<td>0.28</td>
</tr>
<tr>
<td>IV (496)</td>
<td>126</td>
<td>67</td>
<td>0.96 (0.60–1.54)</td>
<td>0.86</td>
</tr>
<tr>
<td>V (497)</td>
<td>204</td>
<td>134</td>
<td>1.05 (0.75–1.47)</td>
<td>0.79</td>
</tr>
</tbody>
</table>

*Quintile point estimate, 95% CI, and P value derived from the SHFM, ICD, and SHFM×ICD interaction terms in the Cox model using continuous variables.

Figure 3. Kaplan–Meier survival curves for SHFM-predicted quintiles are shown for the placebo and the ICD groups. The HR and P values using a linear interaction model for SHFM×ICD are shown for each quintile.
than would appear to be warranted from the evidence.3 Expert clinicians remain less enthusiastic about this therapy eligible patients are not currently receiving ICDs, and many practice guideline recommendations.1,2 Nonetheless, many evidence has been used as the basis for Class I clinical tested in several decades. Effectiveness has been demonstrated in large contemporary clinical trials, and the resulting evidence has been used as the basis for Class I clinical practice guideline recommendations.1,2 Nonetheless, many eligible patients are not currently receiving ICDs, and many expert clinicians remain less enthusiastic about this therapy than would appear to be warranted from the evidence.3 Although much of the focus on refining the use of primary prevention ICD has been on trying to identify some novel test-based measure of the risk of SCD (eg, microvolt T-wave alternans), no study to date has provided evidence that any single test can serve that purpose.4,7 Our results suggest that a regression-based risk model that uses only standard clinical variables, without specialized or expensive testing, can identify clinically useful and statistically valid risk subsets that have different levels of benefit from ICD therapy. Providing clinicians with a simple quantitative tool that can identify the patients in whom ICD therapy offers little potential of benefit and can quantify the anticipated additional life expectancy of patients who would be expected to benefit, offers a cost-effective method for matching patient preferences and tolerance of risk with an invasive but highly effective therapy that is currently significantly underused according to the present guidelines.

Clear variation of primary prevention ICD efficacy based on estimated annual mortality has not previously been demonstrated in a large clinical trial population, although the patients at lower risk of total mortality die mainly from SCD.9 In the present analysis, these relatively lower-risk groups (estimated annual mortality, ~2.5 to 4.5%) made up ~40% of all patients, and a single-lead ICD therapy was 88% effective in reducing SCD and decreased all-cause mortality by ~50%. These patients were projected to gain on average ~5 years of life with an ICD. Patients with higher annual mortalities (up to ~11%) had less relative risk reduction but greater absolute risk reduction with ICD therapy. Conversely, patients with in the highest quintile of predicted annual mortality (~19%) did not benefit from ICD therapy; exploratory analyses suggested that a threshold of benefit may be present at an annual mortality of >20% to 25% for primary prevention ICD therapy. In these patients, we found no significant benefit of the ICD in preventing SCD and no overall benefit on all-cause mortality.

Current guidelines suggest that ICDs are indicated in Class II and III patients but not in Class IV patients.1,2 Our results suggest that a multivariable risk model can provide a more nuanced and likely more reproducible method of assessing candidacy for ICD therapy. The standard SHFM includes hemoglobin, percent lymphocytes, uric acid, and total cholesterol (commonly available clinical variables), with a 1-year receiver-operating characteristic of 0.68 for SCD and 0.85 for

Figure 4. Observed (Kaplan–Meier) mortality at 4 years for the placebo and ICD groups is shown for each SHFM-estimated quintile of risk. The absolute reduction in mortality (shown above each quintile) ranged from ~7% to 14% in quintiles 1 to 4 with no benefit in quintile 5.

Figure 5. A, Projected total lifespan estimate (Gompertz method) for each patient within each quintile was averaged for all placebo and ICD patients within the quintile according to SHFM-predicted risk. B, The difference in total lifespan between the placebo and ICD group averaged over a lifetime is shown. In quintile 1, the average patient will live ~6 years longer but will require ~3 ICDs over the 22-year projected lifespan. Assuming a 7-year ICD battery life, 2.0 life-years were saved per ICD for patients with an average SHFM-predicted 2.5% annual mortality but decreased to 0.2 life-years for quintile 5.
As a result of absent data for these laboratory variables in the present cohort, the SHFM was modified (SHFM-D) for this analysis but had similar overall results, although the c statistic was modestly lower than for the original model for all-cause mortality (0.71 versus 0.73), pump failure death (0.79 versus 0.85), and SCD (0.66 versus 0.68). Having complete covariate information on these patients would likely have strengthened the discriminative properties even further.

Our findings are consistent with an analysis of Acute Decompensated Heart Failure National Registry Longitudinal Module (ADHERE LM) registry, in which an ICD in stage D HF patients was not associated with improved survival; only 17% of deaths in this high-risk population (annual mortality, 28%) were due to arrhythmia.\(^1\)\(^8\) Our results also are consistent with a Multicenter Automatic Defibrillator Implantation Trial (MADIT II) analysis in which patients who were at highest risk for 2-year all-cause mortality had no benefit from the ICD.\(^6\) Results similar to ours were found in a recent ICD propensity analysis in which an increasing number of comorbidities was associated with increased mortality (4.5% to 13.8%), along with a trend for diminishing ICD benefit (53% to 11%; \(P=0.18\)).\(^1\)\(^9\)

In the SCD-HeFT population, we did not find a subgroup of patients who were at such a low risk of SCD that they did not derive benefit from the ICD. This differs from MADIT II, in which a U-shaped relationship of ICD benefit was seen, with no benefit in either high- or low-risk patients.\(^6\) The low-risk group in MADIT II, in whom no ICD benefit was seen, had 4% annual mortality. In comparison, the lowest risk quintile in SCD-HeFT, in whom substantial ICD benefit was seen, had a 3% annual mortality. The reasons for the different results of MADIT II versus SCD-HeFT for these low-risk patients are not clear; the MADIT II model results should likely be validated in an independent cohort before low-risk patients otherwise meeting criteria are denied ICD therapy.

Risk stratification with the SHFM-D should be most beneficial in NYHA class III patients because 98% of the NYHA class II patients in the derivation cohorts and 99% in SCD-HeFT had a <20% annual mortality compared with \(\approx85\%\) of NYHA class III patients. The present analysis cannot determine whether patients with severe symptoms (NYHA class IV) but at lower risk (\(\approx15\%\) SHFM-D estimated annual mortality) would benefit from an ICD; this is not a small subgroup in clinical practice, making up, for example, \(\approx20\%\) of the NYHA class IV patients in the derivation cohorts. These patients in the derivation cohorts (\(\approx15\%\) SHFM-D annual mortality) had a similar ratio of sudden death to pump failure death at 2 years whether they were NYHA class II to III (2.4) or IV (2.6).

The 1-year mortality in Medicare patients who received an ICD is 13.5%, \(\approx2.5\)-fold higher than the patients in SCD-HeFT. It is quite likely that a significant proportion of Medicare patients have an estimated 1-year mortality of >20% to 25%, the point at which the benefit of a primary prevention ICD may be minimal.\(^2\)\(^0\)

Strengths of this analysis include external derivation of the modified model in a large separate cohort of HF patients that preceded prophylactic ICD use. This differs from the MADIT II,\(^6\) Antiarrhythmics Versus Implantable Defibrillators (AVID) trial,\(^2\)\(^1\) and Multicenter Unsustained Tachycardia Trial (MUSTT),\(^4\) in which the risk models were derived within the same database and not externally validated. Several caveats should also be considered. Although the SHFM-D performed well in this analysis, addition of other variables such as brain natriuretic peptide might improve the predictive accuracy of the model even further. Additionally, all trials and cohort studies are subject to the possibility of varying amounts of unrecognized misclassification of SCD. However, the benefit of ICD therapy for total mortality also varied with SHFM-D-predicted risk. This study also does not address the effect of 2-lead and 3-lead systems on outcome; only single-lead, conservatively programmed devices were included in this analysis. Some comorbidities may increase the risk of all-cause mortality without a corresponding increase in risk of preventable SCD.\(^2\)\(^0\),\(^2\)\(^2\) These may include, for example, cancer, stroke, lung disease, peripheral vascular disease, dementia, and cirrhosis.\(^2\)\(^3\) HF populations with an increased prevalence of \(\approx1\) of these conditions may experience diminished benefits from an ICD by increasing the non-SCD rate. Caution should be exercised if this approach is used in the general population, which often has more comorbidities than patients in clinical trials.

Conclusion

A clinical risk prediction model that was externally derived using HF patient cohorts from the pre–primary prevention ICD era and validated in the present cohort was able to identify subgroups of moderately symptomatic HF patients in whom clinically relevant differences were seen in the therapeutic benefit of primary prevention ICD therapy.

Acknowledgments

The authors would like to acknowledge all the subjects, clinical investigators, and clinical coordinators who participated in SCD-HeFT.

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This analysis developed and validated a multivariate risk model (Seattle Heart Failure Model–D) in \( \approx 10,000 \) heart failure patients. We prospectively applied the model to the Sudden Cardiac Death Heart Failure Trial to determine whether the benefit of a primary prevention implantable cardioverter-defibrillator (ICD) varies with the estimated annual mortality. The percentage of sudden death was inversely proportional to estimated annual mortality (low risk had a higher proportion of sudden death). The ICD benefit was greatest (\( \approx 90\% \) reduction in sudden death and \( \approx 50\% \) reduction in all-cause mortality) in the lowest-risk patients who had an estimated annual mortality of \( \approx 3\% \) to \( \approx 5\% \). In the highest-risk patients (\( \approx 20\% \) annual mortality), the ICD was only \( \approx 25\% \) effective in reducing sudden death and had benefit in reducing total mortality. The years needed to treat 1 patient to add 1 year of life was 3.5 to 4.6 in 80% of patients but was 21.5 in the highest-risk patients (\( \approx 20\% \) annual mortality). Each ICD adds 1.5 to 2 years of life in patients with an annual mortality of \( < 15\% \). Use of a validated multivariate risk model may allow healthcare providers to better select patients for primary prevention ICDs and to describe the potential ICD benefit to patients in easily understood terminology.

CLINICAL PERSPECTIVE
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