Who Should Receive an Implantable Cardioverter-Defibrillator After Myocardial Infarction?

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Despite a decline in overall cardiovascular mortality, the incidence of sudden cardiac death (SCD) continues to rise. Patients who survive a myocardial infarction (MI) with depressed ejection fraction are at particularly high risk for SCD. The development of implantable cardioverter-defibrillators (ICDs) has revolutionized SCD prevention; however, despite the current fervor for device implantation, many unresolved questions remain about risk stratification in post-MI patients. This review presents the current indications and timing of ICD implantation for primary and secondary prevention of SCD after MI. Several conventional and investigational methods of risk stratification after MI, as well as current controversies regarding device implantation in specific patient populations, are also reviewed.

Introduction

Modern pharmacologic therapies and the broad adoption of early reperfusion strategies have significantly decreased morbidity and mortality from acute myocardial infarction (MI). Despite remarkable progress, about 500,000 patients still die annually from coronary heart disease. More than two-thirds of these deaths occur suddenly, without prior recognition of cardiac disease [1]. Disturbingly, the incidence of sudden cardiac death (SCD) continues to rise [2]. SCD also accounts for more than 50% of 30-day mortality after MI [1]. In selected patient groups, particularly those with left ventricular (LV) dysfunction or clinical heart failure, SCD risk remains elevated during the 12 months after infarction [1,3].

Mechanisms of SCD After MI

The most common mechanism of SCD is spontaneous ventricular tachycardia (VT) or ventricular fibrillation (VF) causing hemodynamic collapse and subsequently asystole [1]. The mechanism of ventricular arrhythmias after an acute MI depends largely upon the temporal relationship between the events. For example, ventricular arrhythmias or SCD as the initial presentation of acute MI are typically related to electrical instability from acute ischemia that resolves after coronary reperfusion. These patients often present with polymorphic VT or VF. Factors that contribute to SCD during the first 30 days after MI include recurrent infarction, reperfusion injury, or autonomic instability [1]. In the weeks and months after infarction, the arrhythmic substrate changes because of remodeling of the peri-infarct region. The creation of fixed (fibrosis/scarring) and functional (hibernating/stunned myocardium with slow conduction) barriers to conduction within the infarct area creates the "perfect storm" for the development of ventricular arrhythmias. The typical mechanism is myocardial reentry around these electrical barriers. However, some patients also present with tachycardias resulting from abnormal automaticity within the reperfused myocardium. Because of ongoing ventricular remodeling, both the initiation and morphology of arrhythmias after acute MI can be quite unpredictable. In addition, dilation of the ventricle that usually accompanies large infarcts facilitates the temporal dispersion of repolarization that further predispose to reentrant arrhythmias. Because scar tissue is an area with minimal metabolic turnover, scar-related VTs are generally monomorphic and often reproducible with electrophysiologic testing (EPS). Because most monomorphic tachycardias are caused by scar-based reentry, the appearance of monomorphic VT in the early post-infarct period should raise concern that the patient has already developed fixed arrhythmia substrate and may be at higher risk for arrhythmia recurrence.

Trials for SCD Prevention After MI: Facts and Controversies

Secondary prevention

Three landmark, randomized clinical trials have established the use of implantable cardioverter-defibrillators

(ICD) for survivors of SCD [4–6]. Patients with a history of MI were mostly represented in the Canadian Implantable Defibrillator Study (CIDS), with 75% of participating patients having a history of MI. A pooled analysis from all three trials showed an overall reduction in mortality of 27% and a decrease in arrhythmic death of 51% compared with amiodarone [7]. Based on these results, an ICD should be considered for all SCD survivors.

Despite the wide acceptance of ICD for secondary prevention, a few points are important to emphasize. First, patients with ventricular arrhythmias with recent (< 72 h) MI were excluded from those trials. Despite the increased incidence of arrhythmias early after infarction, this event alone is not sufficient to justify ICD implantation for secondary prevention based on prevailing evidence that arrhythmias in the peri-MI period do not predict future SCD.

Second, it is important to emphasize that ejection faction (EF) is the most important predictor for arrhythmic death even for secondary prevention indications. Whereas CIDS included only patients with depressed EF, the Antiarrhythmic Versus Implantable Defibrillators (AVID) trial showed that only patients with depressed LV function (EF < 35%) derived significant benefit from an ICD (> 40% mortality reduction). Similar outcomes were noted when a pooled analysis of the three secondary prevention trials was performed.

Third, patients with hemodynamically stable or asymptomatic ventricular arrhythmias were not included in the secondary prevention trials and evidence for ICD implantation in these patients is lacking. Although subgroup analysis of the AVID database showed similar mortality for patients presenting with stable versus unstable VT, the routine use of ICDs for hemodynamically tolerated VT remains an area of uncertainty for many investigators [8].

Primary prevention

The observation that ventricular ectopy in the early post-MI period predicted arrhythmia-related deaths prompted several trials examining the benefit of empiric antiarrhythmic agents in this setting [9]. Randomized trials with class Ic antiarrhythmics (flecainide and encainide) not only failed to show mortality benefit but also demonstrated an unexpected increase in mortality, likely caused by the proarrhythmic properties of these drugs [10]. Sotalol, a class III antiarrhythmic drug, also had disappointing results in the Survival With Oral d-Sotalol (SWORD) trial [11].

The role of amiodarone in preventing SCD after MI was examined in two randomized trials [12,13]. The European Myocardial Infarct Amiodarone Trial (EMIAT) enrolled 1485 patients with depressed EF and the Canadian Amiodarone Myocardial Infarction Arrhythmia Trial (CAMIAT) enrolled 1202 patients with frequent ventricular ectopy. Both studies demonstrated a reduction in arrhythmic death; however, EMIAT failed to show a reduction in all-cause mortality. This was attributed to an unexpectedly high incidence of "non-sudden" or "unwitnessed" deaths in the amiodarone-treated group. Nevertheless, in both EMIAT and CAMIAT, patients receiving β-blockers appeared to have decreased mortality compared with patients treated with amiodarone alone. The results of these trials, the multitude of side effects attributable to amiodarone, and the advent of device-based therapies have limited the utility of amiodarone in primary prevention of SCD.

The paradigm shift toward device-based prevention of SCD has paralleled significant advancement in the form and function of the modern ICD. The transition from large, abdominal pulse generators and epicardial patches to smaller, subpectoral systems with transvenous leads has greatly facilitated device implantation and revision. Improvements in device sophistication and reliability have also made the ICD a more palatable primary prevention strategy. Over the past decade, much attention has been focused on identifying high-risk patients who would derive benefit from prophylactic ICD implantation. Five major trials have evaluated the ICD versus antiarrhythmic medications in the coronary artery disease population [14–18].

The first Multicenter Automatic Defibrillator Implantation Trial (MADIT I) and the Multicenter Unsustained Tachycardia Trial (MUSTT) included patients with prior MI, depressed LV systolic function, documented ventricular arrhythmias and inducible VT during EPS, which were not suppressible with antiarrhythmic drugs [14,15]. MUSTT was a larger trial than MADIT I (n = 704 vs 196, respectively), used a higher EF cutoff of 40%, and was unique in its use of randomized EPS-guided therapy. MADIT I showed a large, 54% relative risk reduction (RRR) in mortality for the ICD-group. A similar RRR of 49% was found in MUSTT when comparing patients who did and did not receive an ICD. Although high-risk patients were identified with great specificity in these studies due to stringent patient selection criteria, they represented only a miniscule proportion of at-risk patients in common clinical practice.

In an effort to increase sensitivity and obviate the need for invasive risk stratification, two subsequent trials were designed: the Second Multicenter Automatic Defibrillator Implantation Trial (MADIT II) and the Sudden Cardiac Death Heart Failure Trial (SCD-HeFT) [16,17]. In MADIT II, patients with MI and EF ≤ 30% were randomly assigned to receive ICD implantation or conventional medical therapy only. ICD implantation produced a significant (though more modest) absolute risk reduction (ARR) in overall mortality versus conventional medical therapy (14.2% vs 19.8%; ARR, 5.6%) during a mean follow-up of 20 months.

SCD-HeFT randomly assigned patients with New York Heart Association (NYHA) class II or III heart failure symptoms and an LVEF ≤ 35% to conventional medical therapy alone versus amiodarone versus implantation of an ICD. The EF cutoff in SCD-HeFT was higher

Table 1. Summary of the inclusion criteria and the primary outcomes for the major primary prevention ICD trials after MI

Study	Patients, *n*	Groups compared	Inclusion criteria					Primary outcomes			
			EF	NYHA	NSVT	EPS	Other criteria	Follow-up, *mo*	ARR, %	RRR, %	NNT
MADIT [14]	196	ICD vs placebo	0.35	I-III	Yes	Yes	Q wave MI > 3 wk; CABG > 3 mo	27	22.8	54	4
MUSTT [15]	704	ICD vs antiarrhythmics	0.4	I-III	Yes	Yes	MI > 4 d	39	23	51	4
MADIT II [16]	1232	ICD vs placebo	0.3	I-III	No	No	MI > 30 d	20	5.6	28	18
SCD-HeFT [17]	1676	ICD vs amiodarone; ICD vs placebo*	0.35	II or III	No	No	HF of > 3 mo on optimal treatment; MI > 30 d; PCI/CABG > 30 d	45.5	7.2	25	14
COMPANION [19]	903	BiV ICD vs placebo	0.35	III or IV	No	No	QRS > 120 ms; HF > 6 mo; MI or CABG > 60 d	15	7.3	36	14

*Nonrandomized groups
ARR—absolute risk reduction; BiV—biventricular; CABG—coronary artery bypass graft; COMPANION—Comparison of Medical Therapy, Pacing, and Defibrillation in Heart Failure; EF—ejection fraction; EPS—inducible ventricular tachycardia on electrophysiology study; HF—heart failure; ICD—implantable cardioverter-defibrillator; MADIT—Multicenter Automatic Defibrillator Implantation Trial; MI—myocardial infarction; MUSTT—Multicenter Unsustained Tachycardia Trial; NNT—number needed to treat; NSVT—nonsustained ventricular tachycardia; NYHA—New York Heart Association Heart Failure class; PCI—percutaneous coronary intervention; RRR—relative risk reduction; SCD-HeFT—Sudden Cardiac Death Heart Failure Trial.

than in MADIT II, and it was the first primary prevention ICD trial to include patients with nonischemic cardiomyopathy. The study showed a 7.2% ARR at 5 years in the ICD group compared with placebo. Interestingly, the study showed a lack of benefit from the ICD in patients with NYHA class III, whereas class II patients had a 46% RRR with ICD implantation. These subgroup analyses should be interpreted with caution in light of the results from the Defibrillators in Nonischemic Cardiomyopathy Treatment Evaluation (DEFINITE) trial, which showed a substantial therapeutic benefit from ICD implantation in class III patients [18]. SCD-HeFT is not only the largest of all primary prevention trials but also had the longest follow-up period (median, 45.5 mo). It is also important to emphasize that SCD-HeFT was conducted in the modern era of medical therapy for heart failure with the vast majority of patients on β-blockers, angiotensin-converting enzyme (ACE) inhibitors, and statins. Thus, the additive survival benefit of the ICD to optimal medical therapy is clearly demonstrated in this population.

Because patients with severe heart failure symptoms were excluded from SCD-HeFT, their suitability for ICD implantation was not clear. The Comparison of Medical Therapy, Pacing, and Defibrillation in Heart Failure (COMPANION) trial approached a subset of these patients with LV dyssynchrony. COMPANION randomly assigned patients with NYHA class III and IV heart failure, ischemic, or nonischemic cardiomyopathy, an EF ≤ 35%, and a QRS duration of ≥ 120 ms in a 1:2:2 fashion to optimal medical therapy, a biventricular pacemaker, or a biventricular ICD [19]. The primary end point of death from hospitalization for any cause was significantly lower in patients receiving either a biventricular pacemaker or ICD compared with medical therapy (HR, 0.81–0.80). Although all-cause mortality was not significantly lower in the biventricular pacemaker cohort (ARR, 4%; $P = 0.057$), patients receiving a biventricular ICD has significantly lower overall mortality compared with patients with medical therapy (ARR, 7%; number needed to treat [NNT], 14; $P = 0.003$). Table 1 shows a comparison of all five primary prevention trials with regard to their inclusion criteria and subsequent outcomes.

The shift toward using EF as the main risk stratification tool for primary prevention ICD implantation has greatly increased the population of patients at increased risk for SCD, while raising important concerns regarding the cost efficacy of broad application of ICD therapy. Because of more stringent selection criteria, the NNT with an ICD in MUSTT was four, compared with 14 in SCD-HeFT. Despite this disparity, the cost per quality-adjusted life year (QALY) in the primary prevention ICD trials has been estimated to be significantly less than $100,000 ($34,000–$70,200) [20]. Given the significant

health care economic impact of ICD therapy, there has been renewed interest in the evaluation of adjunctive SCD risk factors.

Identifying Patients at Increased Risk of SCD After MI

EF as SCD predictor

A major problem in preventing SCD is the lack of a reliable test to identify patients at elevated risk. All major trials of SCD prevention have focused primarily on EF because of its proven association with overall mortality after MI [7]. However, EF alone lacks sensitivity for predicting SCD because less than 50% of patients with prior MI and SCD have an EF less than 30% [21•]. Data from the Maastricht registry of SCD found twice as many events in patients with no or mild LV dysfunction; of these patients, 72% had coronary artery disease [22]. Because depressed LV function confers similar risk for arrhythmic and nonarrhythmic death, one would not anticipate that EF alone is an effective predictor for ICD benefit [23••]. In addition, the risk of SCD among patients with similar EF varies significantly; therefore, using EF alone to assess risk lacks specificity. A recent subanalysis of MUSTT showed that by using simple clinical characteristics, one can effectively increase the sensitivity of EF in identifying high-risk patients for SCD [23••]. This particular analysis showed that age, the presence of atrial fibrillation, the presence of heart failure (NYHA class II or III), inducible VT during EPS, and the absence of coronary artery bypass graft (CABG) can significantly improve the SCD risk assessment in patients with similar EF. This study underscores the importance of individualizing therapy by integrating patient characteristics and sound clinical judgment.

Microvolt T-wave alternans

Microvolt T-wave alternans (MTWA) is defined as a microvolt alteration in the amplitude of the T wave on a beat-to-beat basis. Early studies found that MTWA strongly predicted arrhythmic events in patients with ischemic heart [24]. Analysis of the MADIT II population based on MTWA status showed that the NNT in MTWA-positive patients was nine, compared with 76 in the MTWA-negative patients [25•]. The negative predictive value (NPV) of MTWA has been previously reported as high as 95%, which makes it a potentially useful tool in identifying patients unlikely to benefit from ICD implantation [25•]. In another prospective trial, MTWA and EPS were found to have similar NPVs, suggesting that MTWA could possibly replace an EPS as a noninvasive risk stratification tool [26]. Another observational study reported an NPV with MTWA of 90%; however, there was a significant event rate in the MTWA-negative patients calling into question the utility of MTWA alone as a deciding factor for ICD implantation [27]. The largest series to date using MTWA was a subanalysis of 490 patients from SCD-HeFT, which failed to predict both arrhythmic events and all-cause mortality [28•]. A major criticism of that particular substudy was the high prevalence of intermediate test results (41%), which may have confounded the analysis. In addition, MTWA result is affected by some pharmacologic agents, and it is unclear to what extent the broad use of β-blockers and amiodarone (41%) in this substudy may have affected the testing results. Given the mixed results from the above studies, the use of MTWA alone as a risk stratification technique has lost favor.

Other noninvasive risk-stratification tools

QT dispersion

Several tests have been used to document heterogeneity of ventricular repolarization as a predictor of SCD. In early studies, QT dispersion on surface ECG was found to predict SCD; however, its utility is limited by its lack of reproducibility and contradictory results in the ischemic heart disease population [29].

Signal-averaged electrocardiography

Signal-averaged electrocardiography (SAECG) records low-amplitude potentials occurring after ventricular depolarization; they may indicate slow electrical conduction, often associated with scarred myocardium, which serves as the anatomical basis of reentrant arrhythmias. In early studies, abnormal SAECG has been associated with increased mortality after MI [30]. A more recent study has challenged this association, suggesting that current medical treatment and early reperfusion strategies could affect SAECG results [31].

QRS duration

QRS width as a marker of conduction system disease or electrical conduction outside the Purkinje system has also been associated with increased mortality after MI [32•]. Interestingly, the survival benefit from an ICD in MADIT II was greater in patients with a wide QRS (> 120 ms); patients with normal QRS duration did not benefit [16]. In contrast, a retrospective analysis of 431 patients with ICD for primary prophylaxis did not find QRS duration to be predictive of tachyarrhythmias [33]. Present guidelines do not include QRS width as a requirement for ICD implantation. It is important to remember that a QRS duration longer than 120 ms is an electrocardiographic marker for ventricular dyssynchrony, and selected patients with heart failure symptoms may benefit from cardiac resynchronization therapy with a biventricular device [18].

Heart rate variability

Autonomic imbalance often accompanies MI and clinical heart failure, and likely plays a significant role in arrhythmogenesis [1]. Decreased heart rate variability (HRV) measured as the standard deviation of a patient's R-R intervals has been used as a surrogate of autonomic imbalance in the setting of increased sympathetic activation. Although HRV has been shown to predict arrhythmic

death, it also predicts death from progressive cardiac failure [34]. Therefore, using HRV alone may not provide adequate specificity to identify patients at elevated risk.

Myocardial imaging

Structural heterogeneity within the infarct region on MRI has been associated with appropriate ICD therapies. This finding may provide a method to noninvasively correlate the relationship between structural and electrical remodeling, which facilitates postinfarction ventricular arrhythmias [35•].

Cardiac imaging with the norepinephrine analogue, iodine-123 (^{123}I) metaiodobenzylguanidine (MIBG) has also been validated as a useful method in estimating cardiac sympathetic activity. In a recent meta-analysis, increased cardiac sympathetic activity resulting in abnormal cardiac MIGB uptake was associated with poor clinical outcome [36•]. A different study found MIBG independently predictive of SCD [37•].

The observation that infarct mass measured by MRI is highly correlated with VT inducibility during EP study has created interest in using this measurement in SCD risk stratification [38]. The same study also found that infarct mass and surface area were more highly correlated with monomorphic VT inducibility than EF. These observations serve as the basis for the multicenter Defibrillators to Reduce Risk by Magnetic Resonance Imaging Evaluation (DETERMINE) trial. DETERMINE will randomize postinfarction patients with both an EF ≥ 35% and a more than 10% infarct mass to an ICD versus optimal medical therapy [39].

In summary, several noninvasive tests have been proposed as prognostic tools in identifying patients who are at high risk for SCD. Most of those tests have been studied in small observational studies, often with contradictory results. Although they should not be used independently as risk-stratifying tools, they contribute incrementally when assessing risk in an individual patient.

Role of invasive electrophysiologic testing

The role of EPS in guiding management of patients with ischemic cardiomyopathy was initially tested in MUSTT. This was the only trial to randomly assign patients to receive EPS-guided therapy. In the EPS-guided cohort, patients who had an inducible, sustained VT were started on antiarrhythmic agents. Serial EPS was performed with multiple agents and an ICD was implanted only in patients with nonsuppressible arrhythmias. Although EPS-guided therapy was superior, the benefit was seen only in patients receiving an ICD [15]. A MUSTT substudy showed that, although patients with inducible VT on EPS had a higher 5-year mortality rates, the absolute difference between the two groups was more modest (48% vs 44%) [40]. A subanalysis of MADIT I also showed that inducibility at EPS did not predict higher risk for SCD [41•]. The major limitation of EPS is its low NPV; as shown in MUSTT, a negative EPS was not a protective finding, rendering it an unacceptable test to identify patients at low risk for SCD [40]. In clinical practice, EP studies may be useful for patients with an EF between 35% and 40% and a high-risk clinical profile. For these patients, a positive EPS indicates a high risk for SCD and warrants implantation of ICD.

Timing of ICD Implantation After MI

The optimal timing of ICD implantation remains unclear. Current guidelines advocate a 40-day waiting period after an acute MI before ICD implantation. This recommendation is largely based on the results of the Defibrillator in Acute Myocardial Infarction Trial (DINAMIT). DINAMIT randomly assigned patients to receive an ICD or medical therapy 6 to 40 days (mean, 18 d) after MI who had an EF ≤ 35% and either reduced HRV or an elevated resting pulse (> 80 beats per minute) [42]. Although one-third of patients in the ICD group received an appropriate therapy, there was no overall survival benefit from ICD implantation. The findings from DINAMIT seem to contradict the findings of the Valsartan in Acute Myocardial Infarction (VALIANT) trial, which showed a higher risk of sudden cardiac death in the first 30 days after MI. Further analysis of the DINAMIT data showed that most patients (> 75%) who were rescued from an arrhythmic death subsequently died of nonarrhythmic deaths [42]. The above finding illustrates that ventricular arrhythmias early after an MI can represent an ominous portent of imminent pump failure. Under such circumstances, treating ventricular arrhythmias with an ICD may simply transform an arrhythmic death into one from heart failure. Patients requiring permanent pacing were excluded from the above analysis, and it would be quite reasonable to consider an ICD implantation in patients with large infarctions who have bradyarrhythmia pacing indications.

In contrast to DINAMIT, the average times from MI to enrollment were 39 months in MUSTT and 81 months in MADIT II. Moreover, in both MADIT II and SCD-HeFT, the protective effect of the ICD (divergence of the Kaplan-Meier survival curves) began around 18 months after implantation. Even patients more than 120 months after MI showed a significant survival benefit from ICD implantation [43]. These data demonstrate that, although ICDs may not be effective in preventing mortality in the early (< 40) days after MI, patients more remote from their event are not protected. Based on these data, there is a relative lack of evidence to guide ICD therapy between 40 days and 18 months after MI.

Regardless of the timing of ICD implantation after a specific coronary event, it is crucial to emphasize the importance of revascularization before ICD implantation. In the Coronary Artery Surgical Study (CASS) registry, 5-year survival from sudden cardiac arrest was better in the revascularized group [44].

The CABG Patch trial examined the role of epicardial ICD implantation during bypass surgery [45]. The study enrolled 900 patients with depressed EF and abnormal SAECG undergoing CABG. No significant reduction in

overall survival was seen in the ICD group. Subgroup analysis showed a significant reduction in SCD counterbalanced by an unexpected increase in nonarrhythmic death (71%) in patients receiving an ICD [46]. Patients are generally not offered ICD therapy until 3 months after coronary revascularization because such patients were excluded from the larger ICD trials such as MADIT II.

In addition to coronary revascularization, optimal medical therapy with β-blockers and ACE inhibitors should be instituted to ensure that the maximum possible benefit in positive LV remodeling has been achieved before implanting an ICD, especially for patients qualifying via SCD-HeFT criteria (EF ≤ 35%, NYHA class II or III). For patients with nonrevascularizable coronary disease, the decision for ICD implantation is based on clinical judgment.

ICD Implantation in Unique Patient Populations

When evaluating patients for ICD implantation, it is important to account for overall mortality risk from cardiovascular causes. In a recent analysis of MADIT II, risk for all-cause mortality significantly impacted the benefit derived from ICD therapy. Patients with intermediate clinical predictors had the most benefit from an ICD (60% survival reduction) whereas patients with extremely high or low risk received no benefit [47••]. The 2008 American Heart Association/American College of Cardiology scientific statement on ICD implantation for primary prevention acknowledges that the current guidelines may not apply to certain subgroups of patients underrepresented in randomized trials. The guidelines also limit ICD implantation to patients with more than 12 months of life expectancy.

Elderly patients were underrepresented in most primary prevention ICD trials. The mean patient age from many of these studies was 65 years old. This contrasts with current clinical practice in which more than 40% of new ICDs are implanted in patients ≥ 70 years old and 10% in patients ≥ 80 years old [48•]. Recent observational studies have shown a significant reduction in mortality in the elderly from ICDs and support the extrapolation of ICD guidelines to the elderly patients without clinically advanced heart failure [49•].

The presence of renal failure has been long associated with higher cardiovascular mortality. Patients with severely impaired renal function were also not included in the main ICD trials. Observational studies have shown that despite ICD therapy, patients with severe renal impairment still manifest a threefold increase in overall mortality [50]. In addition, a subanalysis of MADIT II showed that patients with advanced renal disease (blood urea nitrogen > 50 mg/dL and serum creatinine > 2.5 mg/dL) did not demonstrate a mortality benefit from ICD implantation [47••]. The role of primary SCD prevention in patients with advanced renal disease requires further investigation.

Patients awaiting cardiac transplantation are a unique group at particularly high risk of SCD who may benefit from ICD even without fulfilling the conventional indications for ICD placement. Early practice favored external defibrillator systems in such patients. Because of practical inconveniences of external defibrillators such as cost and patient compliance, many centers place ICDs in patients awaiting cardiac transplantation [51].

Conclusions

In summary, provision of an ICD for the primary prevention of SCD is reasonable to consider in stable patients with at least 12 months of life expectancy who are 1) at least 40 days post-MI or 3 months post–coronary revascularization; 2) on optimal medical therapy; and 3) have either an EF ≤ 30% in the absence of heart failure symptoms or an EF ≤ 35% with heart failure symptoms. All survivors of sudden cardiac arrest in the setting of coronary disease should be considered for ICD implantation for secondary prevention of SCD provided that 1) active coronary ischemia has been excluded; 2) the sudden cardiac arrest occurs more than 48 hours after an acute MI; and 3) severe metabolic abnormalities and drug toxicities have been excluded. The survival benefit for secondary prevention may be attenuated in patients with relatively preserved LV function (EF ≥ 40%) or with severe medical comorbidities.

LVEF remains the dominant clinical risk assessment for patients with ischemic heart disease. The presence of clinical heart failure symptoms and the inducibility of arrhythmias during EPS are also significant predictors of increased SCD risk and especially useful in patients with moderately reduced EF. The results of other testing such as SAECG, MTWA, or myocardial imaging should be integrated with individual patient data and may provide additive risk assessment. Figure 1 shows a proposed algorithm for SCD prevention in patients with a history of MI based on current evidence.

Although the incidence of SCD is proportionally higher in patients with coronary artery disease with LVEF ≤ 30%, most sudden deaths occur in patients with preserved or only mild LV dysfunction. Current risk stratification indices identify only the "tip of the iceberg" of patients at risk for SCD. Ongoing clinical trials such as DETERMINE may provide novel screening methods to identify patients to whom we currently fail to offer this life-saving therapy.

Disclosure

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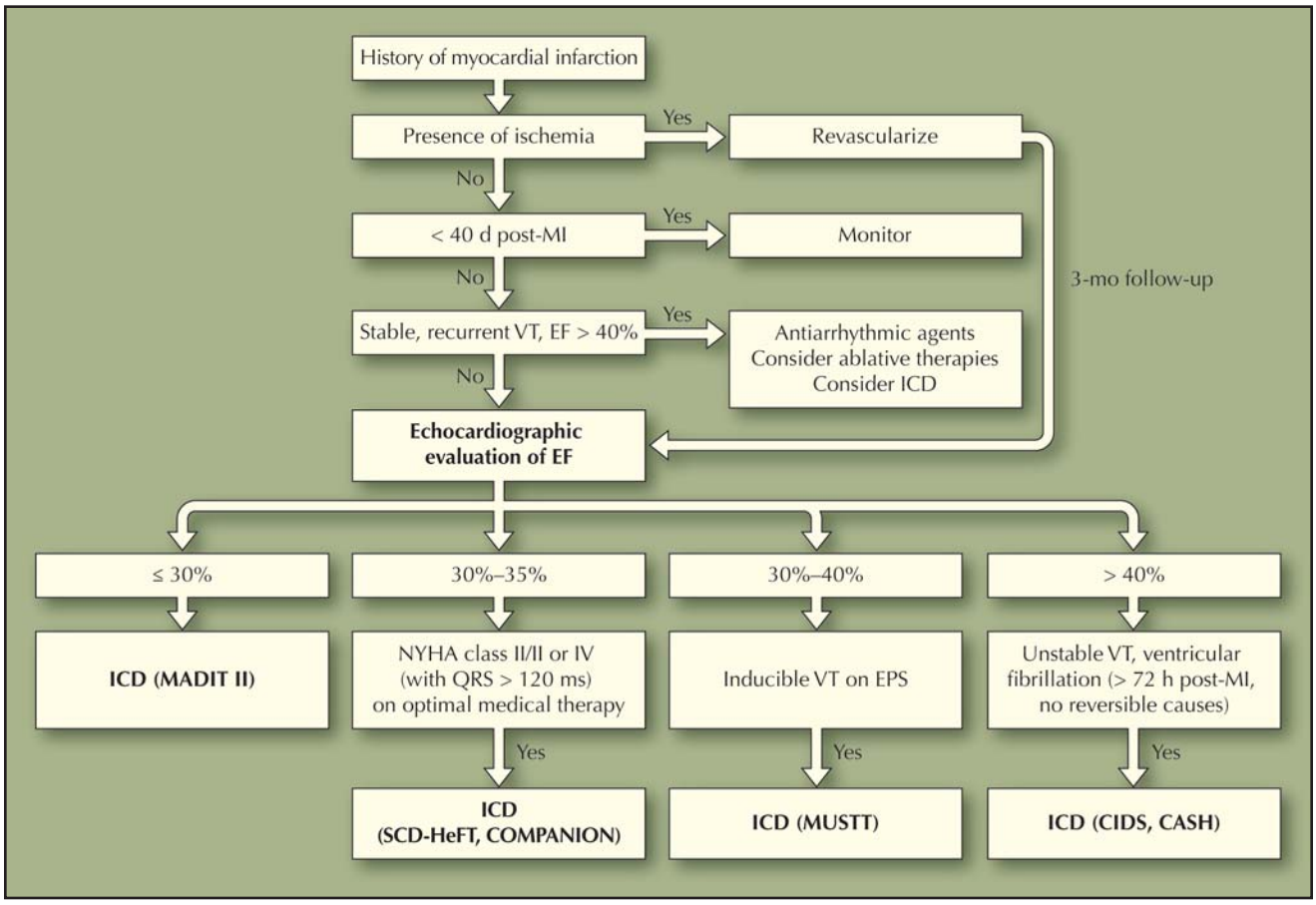

Figure 1. Algorithm for sudden cardiac death prevention in a postinfarction patient. CASH—Cardiac Arrest Study Hamburg; CIDS—Canadian Implantable Defibrillator Study; COMPANION—Comparison of Medical Therapy, Pacing, and Defibrillation in Heart Failure; EF—ejection fraction; EPS—electrophysiology study; ICD—implantable cardioverter-defibrillator; MADIT—Multicenter Automatic Defibrillator Implantation Trial; MI—myocardial infarction; MUSTT—Multicenter Unsustained Tachycardia Trial; NYHA—New York Heart Association; SCD-HeFT—Sudden Cardiac Death Heart Failure Trial; VT—ventricular tachycardia.

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