Do patients with hemodynamically tolerated ventricular tachycardia require implantable cardioverter-defibrillators?

Patients With Hemodynamically Tolerated Ventricular Tachycardia Require Implantable Cardioverter–Defibrillators

David J. Callans, MD

Conventional wisdom has long held that patients with tolerated ventricular tachycardia (VT) in the setting of chronic coronary heart disease are at low risk of arrhythmic death. This logic held that arrhythmia recurrence, although reasonably likely, could be predicted to be well tolerated. As with all truisms, it is good to reexamine this belief periodically. Disease states change; more information becomes available; and the general context of medical care improves. Expectations and capacity to tolerate risk, which are essentially societal rather than medical considerations, continue to evolve. Situations exist such as the present case in which this process of reexamination rather than the results of well-constructed randomized trials will determine the correct course.

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Many past studies have looked at this question at least tangentially. Despite this experience, no study directly answers this question in the present day. Our contemporary strategies for treating patients with advanced structural heart disease, including more aggressive revascularization and prevention of remodeling in heart failure, were not available during the time when these studies were performed. These strategies certainly have antiarrhythmic effects but more profoundly influence the natural history of progressive heart failure. There also have been marked changes in strategies for specific antiarrhythmic therapy. For all of these reasons, the data from trials performed in the 1980s and 1990s are reviewed for themes rather than direct answers.

Despite these disclaimers, critical review of the evidence demonstrates that patients with structural heart disease (primarily healed myocardial infarction) who present with tolerated VT require implantable cardioverter-defibrillator (ICD) therapy. The foundations for this conclusion are as follows. First, tolerated VT is not a benign condition but signals a risk of life-threatening ventricular arrhythmias. Second, the benefit of secondary-prevention ICD therapy is difficult to challenge. Finally, successful catheter ablation does not sufficiently reduce residual risk. These considerations in the context of our current societal expectations for medical care make ICD therapy difficult if not impossible to avoid.

Tolerated VT Signals a Risk of Life-Threatening Arrhythmias

The logical bases for many ideas widely accepted in the “oral history” of electrophysiology are often difficult to determine. In my mind, the foundation of the idea that patients with tolerated VT will do well is formed by a series of early studies comparing patients who present with resuscitated cardiac arrest and tolerated VT.1–3 These studies demonstrated that presentation with cardiac arrest was a major risk for sudden and total mortality; however, given the lack of appropriate...
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treatment for this condition at this time, these data are not surprising. The study by Saxton and coworkers5 presents data to support that the sudden and total mortality rates in patients with tolerated VT (presenting with palpitations and/or dizziness) are significantly better than those with cardiac arrest (4-year survival from sudden death and total mortality, 87±6% versus 59±11% and 67±8% versus 45±10%; P<0.05 for both comparisons). These early studies identified other predictors for mortality such as presentation with VT/ventricular fibrillation early after myocardial infarction, severe left ventricular (LV) dysfunction, and symptomatic heart failure. A study by Waller et al4 provides some basis for the idea that patients with tolerated VT have a good prognosis, if only tangentially. In this study, 258 patients with a variety of arrhythmia presentations underwent serial electrophysiologically guided drug testing. Group 1 patients (n=103) were noninducible on the final regimen; group 2 patients (n=51) were inducible, but a drug regimen conferred a beneficial response (VT cycle length increased by >100 ms and no severe symptoms during VT); and group 3 patients (n=52) had no response to antiarrhythmic drugs. Although group 2 patients had a 39% incidence of recurrent sustained VT over 19.9 months, the total and sudden death mortality rates were substantially reduced compared with group 3 (12% and 4% compared with 39% and 34%) and were not different from group 1. However, patients in this study were censored at the time of VT recurrence if changes in antiarrhythmic therapy were made, artificially making them “immune” from mortality end points.

An important pre-ICD study that directly evaluated this question was presented by Sarber et al.5 This study retrospectively analyzed the outcome of 124 patients who presented with hemodynamically tolerated VT and were treated with arrhythmia surgery (46 patients) or antiarrhythmic drugs (78 patients) between 1981 and 1990. The duration of follow-up was 36±30 months. Most patients had multivessel coronary artery disease and depressed LV ejection fraction (mean, 31%); 60% had an LV aneurysm. Total mortality was 29%, including a discouraging 20% operative mortality. Sudden death mortality was 7% at 3 years (2.4% per year), although it was higher in medically treated (9.1%) than surgically treated (5.4%) patients.

Although not necessarily flawed in terms of construction, the information provided by these studies may be less applicable to present-day patients. Background cardiovascular care has improved dramatically, with infarct revascularization and aggressive treatment of LV remodeling. LV aneurysms are unusual. Arrhythmia surgery is seldom used, largely owing to concerns over operative mortality. The number of patients included in these single-center studies was small, and follow-up was limited in duration. Many have questioned the accuracy of cause-of-death assessment in single-center, retrospective trials; this is essentially why major ICD trials have focused on the end point of total mortality. Finally, the widespread applicability of ICD therapy has changed expectations about residual risk considerably.

In this context, the first observation that weakened the status quo was by Bocker et al.6 These investigators reported a 22% incidence of rapid VT (<250-ms cycle length) in 17 months documented by ICD interrogation in a group of 50 patients who received ICDs after presenting with tolerated VT (Figure 1). Furthermore, the results of programmed stimulation were not helpful in predicting patients at risk. Glickson et al7 reported that 12% of 82 patients treated with ICDs for hemodynamically well-tolerated VT developed unstable ventricular arrhythmias over 23.6±21.5 months. No one would contend that ICD shocks are equivalent to arrhythmic death had the ICD not been present (see below); however, these observations imply that patients with stable VT are at risk for poorly tolerated arrhythmia recurrence.

Several large and more current albeit observational studies appear to advance this same point. Caruso and coworkers8 analyzed the predictors of sudden cardiac death in patients enrolled in the Electrophysiology Study Versus Electrocardiographic Monitoring (ESVEM) over a 6-year follow-up period. This study included 381 patients who presented with sustained VT and 105 cardiac arrest survivors. The mean LV ejection fraction was 32%, and 84% had coronary artery disease. Although univariate analysis suggested a relationship between presentation and outcome, the multivariable analysis did not, implying that LV function has a stronger influence than presentation on outcome. Comparison between the groups aside, 18% of patients who presented with VT suffered arrhythmic death or cardiac arrest in the short-term follow-up; however, it must be acknowledged that the use of potentially harmful, type I antiarrhythmic agents may have contributed to this result. Raett et al9 compared total mortality in 440 patients with tolerated VT and 1029 patients with unstable VT in the Antiarrhythmics Versus Implantable Defibrillator (AVID) Registry. It is important to realize that this analysis was retrospective and that patients in the registry were not randomized in terms of treatment strategy; in the tolerated VT group, treatment included ICDs with or without antiarrhythmic drugs (32%), antiarrhythmic drugs (52%), and catheter ablation (14%) as determined by physician preference. Significant differences existed between the groups in incidence of heart failure and the use of ICD therapy, but no difference existed in ejection fraction or the number with coronary disease. Total mortality at 3 years was 33.6% in the tolerated VT group, higher than the 27.6% of the unstable VT group (Figure 2). Multivariable analysis identified that treatment with antiarrhythmic drugs was associated with an increased risk and that ICD therapy was protective. In a similar manner, a retrospective multivariable analysis of the entire AVID Registry (3559 patients) demonstrated that prognosis was determined predominantly by the severity of structural heart disease and the presence of heart failure.10 Arrhythmia presentation had no impact on survival, and ICD therapy was protective (hazard ratio, 0.51; P<0.001). The
potential confounding influence of selection bias for prescription of ICDs notwithstanding, these studies support the hypothesis that tolerated VT is not a benign condition and that arrhythmic death contributes to total mortality.

The Benefit of Secondary-Prevention ICD Therapy Is Difficult to Challenge

ICD therapy has been demonstrated to result in a reduction in total mortality in patients with hemodynamically unstable arrhythmias in well-designed, randomized clinical trials.11–13 The logic in assuming that this benefit would extend to patients with tolerated VT may be questioned. However, because the total mortality is higher in patients with tolerated VT than unstable VT and because ICD therapy reduces total mortality in patients with unstable VT, this “leap of faith” does not seem farfetched. Whether the magnitude of this ICD benefit is worth the cost—to society, in terms of resource allocation, or to individual patients, in terms of enduring potential adverse consequences of ICD therapy—remains worth examining. To this end, what arguments have been raised against secondary-prevention ICD therapy?

The foundation of secondary-prevention therapy was the AVID study, which demonstrated that ICD therapy reduced total mortality by 26% at 2 years compared with antiarrhythmic drug therapy (primarily amiodarone) in patients with aborted cardiac arrest or VT with severe symptoms.13 Detrac-
tors often focus on the small increase in life expectancy afforded by ICD therapy (2.7 months at 3 years) and marginal cost efficacy based on this calculation ($66,677 per life-year saved). However, it is important to consider how the limitations of randomized clinical trials in general dilute the benefit of ICD therapy. Although intention to treat is the scientifically correct analysis, 18.4% of the antiarrhythmic drug group crossed over to receive ICDs. In addition, particularly in light of subsequent, longer-term ICD trials, the duration of follow-up was too short to realize the greatest portion of the benefit of ICD therapy.

To this last point, a recent report about longer-term follow-up in the Canadian Implantable Defibrillator Study (CIDS) is illustrative. In the main trial, follow-up of 659 patients for a mean of 3 years demonstrated that ICD therapy resulted in a nonsignificant reduction in total mortality (8.3% versus 10.2% per year; \( P = 0.142 \)) compared with amiodarone. However, in a single enrolling center, follow-up was maintained in 120 patients (60 in each treatment assignment) for a mean of 5.6 years. This experience showed a more dramatic benefit of ICD therapy compared with amiodarone (total mortality, 27% versus 47%; \( P = 0.0213 \)), which appeared to increase over time (Figure 3). The time dependence of the ICD benefit also was demonstrated in an analysis of 8 landmark ICD trials by Salukhi and coworkers. This study demonstrated that the marginal effectiveness (in life-years gained) with ICD therapy increased in a nonlinear fashion, at least over the amount of time provided by clinical trial data (maximum, 3 years). Although longer-term time frames are not economically feasible in randomized controlled trials, the time-weighted benefit of ICD therapy also has been observed in primary prevention trials such as the Second Multicenter Automated Defibrillator Implantation Trial (MADIT II) and the Sudden Cardiac Death in Heart Failure Trial (SCD-HeFT). From a patient standpoint, one of the biggest problems associated with ICD therapy is inappropriate shocks. Traditionally, inappropriate shocks occur in approximately one third of ICD patients; the chance of these shocks occurring can be reduced with improved diagnostic algorithms and proper attention to programming. A new twist on this issue is the concept that appropriate therapy delivery may be “inappropriate” in that the device itself may be somehow proarrhythmic. In a compelling analysis of the major ICD trials, Germano et al. raise the point that the frequency of appropriate ICD therapy exceeds the control group sudden death rate by a factor of 2 to 3. They propose that several factors may be responsible, including treatment of VT that would have been nonsustained and actual ICD proarrhythmia (caused by right ventricular pacing, lead-induced tricuspid regurgitation, and other factors). It is important to recognize that the majority of “excess” therapy delivered took the form of antitachycardia pacing as opposed to high-energy shocks. Recent research has demonstrated that the delivery of appropriate shocks is a significant risk for subsequent development of heart failure and death. However, the substantial benefit of ICD therapy demonstrated in most major trials persisted despite the concern over potential device proarrhythmia.

As discussed above, the vast majority of the therapy delivered by ICDs is in the form of antitachycardia pacing. This therapy is highly effective; even in nonselected patients with VT (faster VTs are less amenable to successful antitachycardia pacing), antitachycardia pacing terminates 78% to 94% of episodes, typically before patients even develop symptoms. Although this is too self-evident to submit to formal study, antitachycardia pacing is helpful in
preventing symptoms and repeat hospitalization in patients who are bound to have recurrent VT.

No one would argue that ICDs have been conclusively demonstrated to be effective in reducing arrhythmic death. Observations available from the trials discussed above suggest that arrhythmic death contributes to the high total mortality in patients with tolerated VT (although the extent of this contribution has not been determined accurately). No compelling medical reason exists to not want to protect patients from this residual risk.

Successful Catheter Ablation Does Not Sufficiently Reduce Residual Risk

Other nonpharmacological therapy strategies for the treatment of patients with tolerated VT have not consistently eliminated the risk of death. Although technically not restricted to patients with tolerated VT, most ablation trials have focused on such patients, at least before the development of substrate-based ablation for unmappable VT. Many of these studies were focused on the evaluation of mapping/ablation strategy or end point (eg, evaluation of whether ablation of all inducible VT morphologies influenced outcome), but some provide insight into prognosis in patients with tolerated VT. Independently of procedural strategy, these studies typically demonstrate a high incidence of recurrent VT, some rapid, and a high total mortality, even in short-term follow-up (Table 1). Two studies that are often presented as supporting the idea that successful ablation procedures and the absence of inducible VT predict a good outcome in fact are not convincing. Rothman et al23 studied 35 patients with hemodynamically well tolerated VT after myocardial infarction. In a “complete success” subgroup of 11 patients, the clinical VT was successfully ablated, and no VT was inducible at the conclusion of the procedure and at a predischarge electrophysiological study; in addition, programmed stimulation was repeated late (4.2 months) after the procedure in 8 of the 11 patients. One patient who had no inducible VT after the procedure and only polymorphic VT at late follow-up died suddenly at 18 months after ablation. Della Bella and coworkers24 evaluated the strategy of catheter ablation often followed by treatment with amiodarone and β-blockers (86%) in 124 patients who presented with hemodynamically tolerated VT. These investigators estimated that this represented 30% of the total VT/ventricular fibrillation population treated at 4 participating referral centers over the time period of interest (1992 to 1997). Over a 41.5-month follow-up period (range, 1 to 86 months), low sudden and total mortality rates were observed (2.4% and 12%, respectively), perhaps reflecting the exclusion of patients with active coronary disease and the small number of patients with severely reduced LV ejection fraction. The authors conclude that catheter ablation (if successful) is appropriate treatment for this group of patients and that ICD therapy is not required. However, 24 patients were treated with ICDs in this study, 11 before study entry and 13 after less than completely successful ablation procedures or VT recurrence.

Despite increasing use of optimal therapy for heart failure and coronary heart disease, most contemporary studies of catheter ablation for VT are more sobering. In a multicenter evaluation of internally cooled RF ablation,25 124 patients with healed infarction and spontaneous sustained VT were followed up after ablation for 243±153 days. All patients had ICDs with electrogram storage capabilities. Despite successful elimination of all mappable VTs in 75% of patients, recurrent VT was observed in

![Figure 4. Kaplan-Meier survival probabilities in patients with or without ICDs after VT ablation. Although a trend is present toward improved survival in the ICD group (hazard ratio, 0.54), it does not reach statistical significance in this relatively small study. In addition, ICD prescription was not randomized and was subject to selection bias. Nonetheless, this graph suggests the time-dependent benefit of ICD therapy in patients after VT ablation. Adapted from Segal et al.,26 copyright © 2005, with permission from Elsevier.](image-url)
46%. Furthermore, total mortality at 1 year was 25%, mostly because of progressive heart failure. A recent study by Segal and coworkers investigating a noncontact mapping strategy in 40 patients is of interest because they distinguished between arrhythmia recurrence (ie, of targeted VT morphologies) and the development of new arrhythmias. Of the 140 VTs induced, 81 were targeted; “rapid and nonclinical” VT morphologies were not targeted, and patients in whom these arrhythmias were induced received an ICD (n=13; in addition, 13 had a preexisting ICD). Ablation was successful in 33 patients. Programmed stimulation after ablation demonstrated no inducible VT in 24 patients, and only rapid, nonclinical VT was induced in 9 patients. At 36±21 months, recurrence of an ablated VT was observed in 7.5%, a new or nontargeted VT in 37.5%, and ventricular fibrillation in 7.5% of patients. Total mortality was 32.5%, and although the suggestion existed that the presence of an ICD curtailed mortality (hazard ratio, 0.54), it did not reach statistical significance (Figure 4). The frequency of ICD therapy was markedly reduced after ablation in this study.

Catheter ablation for VT does not appear to be sufficiently protective against VT recurrence or risk of total mortality even in short-term follow-up. In general, programmed stimulation after ablation has not proved helpful in assessment of subsequent arrhythmia risk. Given the complexity of the arrhythmia substrate (Figure 5) and the typically inexorable
TABLE 2. Points That Favor Use of ICD Therapy in Patients With Tolerated VT

<table>
<thead>
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<th>Points</th>
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<tr>
<td>Patients with tolerated VT have a high mortality, even in short-term follow-up.</td>
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<td>Secondary-prevention ICD trials show consistent benefit (reduction in total mortality) in similar populations.</td>
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<td>The relative benefit of ICD therapy improves with increasing duration of follow-up.</td>
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<td>Antitachycardia pacing is an effective means of preventing symptoms of recurrent VT.</td>
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<td>Catheter ablation of VT is successful in reducing recurrence of individual VT morphologies; VT recurrence and total mortality remain high.</td>
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<tr>
<td>Programmed stimulation is not a sufficiently precise means of predicting risk of sudden death.</td>
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<tr>
<td>Structural heart disease progresses over time; the risk of hemodynamically untolerated VT is almost certainly increased by the progression of heart disease.</td>
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progression of severe structural heart disease, the outlook over the longer term is certainly even less favorable.

Summary

Patients who present with tolerated VT in the setting of structural heart disease should be treated with ICD therapy independently of the other strategies used to care for them. The points that lead to this conclusion are summarized in Table 2. One of the most compelling concepts that I hope is clear from the discussion above is the influence of the duration of follow-up on our expectations for patient well-being. Over the short term, patients with tolerated VT do well, and ICD therapy appears less necessary; with passing years, disease processes progress, risk escalates, and the benefit of ICD therapy increases. These factors are unlikely to be captured in the framework of randomized controlled studies.

Patients who present with tolerated VT in the setting of structural heart disease with relatively preserved ejection fractions (≥40%) represent a special, although rather unusual, circumstance in which more thought is required. Even fewer data exist to guide this specific decision, although the powerful effect of ejection fraction on arrhythmic and cardiac mortality is well understood. In this setting, it is my opinion and practice that primary catheter ablation without ICD therapy may be the most reasonable approach. As mentioned in the prior paragraph, it is important to consider the possibility of progressive structural heart disease complicating the course of this initial therapy.

Although intellectually interesting, this exercise of comparing one therapy with another is distinctly different from how physicians practice. The question is not which therapy in isolation is best for the patient but rather which of these potentially complementary strategies will provide some possibility of improved outcome, both now and in the future. In my own practice, patients with tolerated VT receive an ICD. If they have symptoms from frequently recurrent VT, most patients benefit from concomitant VT ablation.

Many of us began practicing electrophysiology before its interventional era, when this field was intensely intellectual but less successful at protecting patients from future harm. The unappealing part of this exciting present era is the fact that many problems are approached exactly the same way in all patients (ie, all patients with structural heart disease and ventricular arrhythmias receive ICDs). I look forward to the time when our understanding is sufficient to offer ICD therapy to only those specific patients who will benefit. That time is not now. Our present focus should not be on wishing that things were different but instead on working to improve all applicable strategies that are useful in caring for patients with this complex, dynamic, and high-risk condition.

Disclosures

Dr Callans has received honoraria from Boston Scientific, Medtronic, and St Jude Medical and is on an advisory board for St Jude Medical. His program receives fellowship support from Boston Scientific, Medtronic, and St Jude Medical.

References

Response to Callans

Jesús Almendral, MD, PhD; Mark E. Josephson, MD

Although conventional wisdom might be against the use of implantable cardioverter-defibrillators (ICDs) for well-tolerated ventricular tachycardia (VT), the reverse (ie, implantation of ICDs) is intuitively more likely. The logic would be as follows: If we are implanting ICDs for primary prevention because patients are at risk of malignant ventricular arrhythmias, how could we not implant in patients who have already had a sustained VT (ie, the paradigm of a malignant arrhythmia)? Such a dilemma needs the discourse of scientific information that demonstrates that tolerated VT, adequately treated, is truly a malignant arrhythmia. Despite Dr Callans’ convincing discussion of secondary-prevention ICD trials, these trials do not help scientifically simply because patients with tolerated VT were explicitly excluded from them. The most important information derives from observational studies on catheter ablation, including almost 800 patients altogether. It is claimed that ablation does not “appear sufficiently protective” on the basis of a high rate of recurrent VT and total mortality. However, recurrent tolerated VT is overestimated by the ICD and is not a catastrophic event; it allows further therapy. Total mortality is high, but sudden death is low (2.5% after procedures considered successful). One of the series quoted as having high mortality (that of Calkins et al) is the series with the highest ICD implantation rate (79% had ICDs), suggesting that most deaths were nonarrhythmic and/or that the ICD contributed to the outcome. Thus, catheter ablation series are consistent with observational series from the 1980s and perhaps with the Antiarrhythmics Versus Implantable Defibrillator Registry, pointing toward a high total mortality but a low sudden death risk if the VT substrate can be substantially modified with catheter ablation or surgery. Why should we add the risks and complications of ICDs to all of these patients?
All Patients With Hemodynamically Tolerated Postinfarction Ventricular Tachycardia Do Not Require an Implantable Cardioverter-Defibrillator

Jesús Almendral, MD, PhD; Mark E. Josephson, MD

Several randomized clinical trials have been published in the last decade to evaluate the clinical efficacy of the implantable cardioverter-defibrillator (ICD) for secondary prevention of sudden cardiac arrest.1–4 However, none of those trials included patients with hemodynamically tolerated ventricular tachycardia (VT). In the largest of these trials, the Antiarrhythmics Versus Implantable Defibrillators (AVID) trial, patients with VT were included only if the VT produced syncope or if the “ejection fraction was 0.40 or less and symptoms suggesting severe hemodynamic compromise” were present.1 The Canadian Implantable Defibrillator Study (CIDS) trial included patients with VT if it produced syncope or caused presyncope or angina.2 The other 2 trials included only cardiac arrest survivors.3,4 Thus, no information is available from randomized clinical trials about outcome in patients with hemodynamically tolerated VT.

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Information About Clinical Outcome of Patients With Tolerated Post–Myocardial Infarction VT Is Limited

Despite decades of active research focused on post–myocardial infarction (MI) VT, information on the clinical outcome of sustained monomorphic VT patients is limited. Several reasons account for the limited information. First, because post-MI VT has always been considered a life-threatening condition, it has been considered unethical to leave these patients without specific antiarrhythmic therapy for group comparisons. Second, VT recurrences during follow-up have been considered in some studies together with sudden cardiac death (SCD)5 despite the fact that both events are distinctly different; the recurrence of a well-tolerated VT may require medical attention, but in most instances, the patient leaves the hospital alive and without any sequelae. Third, because ICD and catheter ablation therapy have been used increasingly in recent years, outcome information is likely to be more homogeneous in relatively old studies. Fourth, several outcome studies have included patients with a variety of sustained ventricular arrhythmias, but the prognosis of patients with ventricular fibrillation (VF) may not be the same as that of patients with VT, and within VT, different patient categories may have different prognoses. In fact, although most patients in the series referenced in this article suffered post-MI VT, some patients in some series have other substrates. A recent study, however, showed that prognosis was rather similar for patients with different sustained ventricular arrhythmias,6 particularly if the left ventricular (LV) ejection fraction...
fraction was ≥0.35, but in this study, ICD implantation rate was half in patients with asymptomatic VT compared with other arrhythmia categories (27% in patients with asymptomatic VT, 53% in patients with VF, and 58% in patients with syncpe and inducible arrhythmia), suggesting a more benign prognosis for asymptomatic VT. Moreover, no data on SCD and arrhythmia recurrence were given in this study. Finally, total mortality should be distinguished from arrhythmia-related mortality, which is all that can be decreased by the ICD. Although total mortality is the most important outcome variable for population studies, it should not be ignored that most of these patients have severe ventricular damage and may succumb to pump failure, which could be a "confounding variable" when one tries to evaluate the results of an antiarrhythmic intervention. Terminal VT in the setting of end-stage heart failure should not be considered a sudden cardiac arrest because the event must be unexpected. Moreover, the efficacy of an ICD in such instances may be limited. Such patients make up much of the group of patients in whom death occurs with VT despite ICD therapy (successful or unsuccessful).7

The study by Sarter et al8 is one of the few studies that analyzed the SCD rate in patients with stable post-MI VT. This retrospective cohort of 124 patients with a mean LV ejection fraction of 0.31 who were followed up for a mean of 36 months received electrophysiologically guided therapy with either antiarrhythmic drugs or arrhythmia surgery. In an actuarial analysis, the cumulative rate for total mortality was 32±5% at 3 years. However, the cumulative rate for SCD in the same period was 7±3%, with an average annual SCD rate of 2.4%. Brugada et al9 reported that in a cohort of 140 patients with post-MI VT followed up for a mean of 2 years and treated with antiarrhythmic drugs, the SCD rate was 2%. In contrast, Olson et al10 found a high SCD rate of 25% over a mean follow-up of 19 months in 122 patients with sustained VT treated with amiodarone (51 of those had tolerated VT and a 25% SCD rate). In the Electrophysiologic Study Versus Electrocardiographic Monitoring (ESVEM), an intermediate 14% risk of SCD at 1 year was found.11 A more recent study analyzed the outcome of patients in the AVID Registry according to whether their index arrhythmia was stable or unstable VT.12 The authors found that total mortality at 3 years was 33.6% in patients with stable VT compared with 27.6% in patients with unstable VT. Despite the fact that no further attempt was made to classify the cause of death, the authors suggested that stable VT may be a marker for faster, poorly tolerated VT and thus for SCD; ie, stable VT is not a benign rhythm. However, it is interesting to note that total mortality at 3 years was virtually identical to that in the study by Sarter et al (33.6% versus 32%).8 Because in the population reported by Sarter et al the SCD rate was low despite a high total mortality, it is difficult to assume what the SCD rate might have been in the AVID Registry population. In addition, in patients receiving an ICD in the AVID Registry, total mortality was as high as 23% (in the total VT group; no information was provided for the specific group of stable VT).12 A similar phenomenon was observed in our surgical ablative cohort at the University of Pennsylvania (see below) in whom the 5-year mortality was almost 45% but the incidence of SCD was only 4% over the same time period (MEJ, unpublished observations, 1978–1992). Because most of these series reflect data from the 1980s, one could question whether this information applies to our contemporary patients, most of them revascularized and treated with modern pharmacological therapy for LV dysfunction. While recognizing that no answer to this issue can be complete and that tolerated VT may be less frequent in recent years, both the AVID Registry12 and 2 recent ICD contemporary series13,14 suggest that tolerated (slow) VT may still be a clinical problem. It is likely that a specific substrate is required for monomorphic VT, and although it may be less frequent in recent years, once a patient develops monomorphic VT, his/her substrate is similar to what was found in VT patients years ago.

Thus, although it is clear that post-MI tolerated VT carries a high mortality, the contribution of arrhythmic death is unknown but likely far less than suspected. Tolerated VT also can be a clinical problem in the setting of ICD therapy because it can be symptomatic even if it is slow enough to overlap with sinus or supraventricular arrhythmia rates.13,14

**ICD Therapy as a Surrogate for Sudden Arrhythmic Death**

ICDs provide information about their therapeutic interventions. The notion that this information could establish the natural history of ventricular tachyarrhythmias is an old wish. In the initial ICD studies, it was thought that if an ICD discharged and the discharge was preceded by symptoms, an arrhythmia had appeared that would have been lethal had the ICD not been implanted.13 When technological advances allowed the storage and subsequent recording of electrograms preceding ICD therapies, it became clear that some of these therapies were in fact triggered by nonlethal supraventricular arrhythmias.16 Admitting that electrogram analysis allowed precise differentiation between supraventricular and ventricular tachyarrrhythmias,16 “real” ventricular arrhythmias were considered to measure the degree of protection conferred by the ICD. However, it is quite clear that sustained VT cannot be equated with SCD, so some qualification to electrogram analysis was needed in addition to tachycardia origin to consider an ICD arrhythmia a surrogate for SCD.

Bocker et al17 reported on a retrospective group of 50 patients with hemodynamically tolerated VT treated with an ICD. During a mean follow-up of 17 months, 11 patients (22%) had VT with a cycle length <250 ms.17 It was postulated that such fast VT would have been life-threatening, and a supposed survival benefit in these patients was postulated by subtracting survival free of fast VT from total mortality, giving 27% at 4 years.17 In a more recent study, with a larger retrospective series of 82 patients with
stable VT and a longer follow-up of 23 months, it also was found that a significant number of patients (10 of 82, 12%) developed a VT that was considerably faster than the index VT and was regarded as an unstable VT.18 The authors came to a 4-year actuarial estimation of developing an unstable VT of 25%. It is of note, however, that the criteria used in this study to consider that the ICD recordings represented an unstable VT were different and less strict than those used in the previous study, pointing out that any ICD criteria for stability of a VT are arbitrary. Moreover, if the criteria of Bocker et al (even less strict) were applied in the latter study, the number of patients with unstable VT would be only 5 (6%)18 and the ICD benefit much less. Both of these studies fail to provide information about the clinical situation of the patients at the time of the unstable VT (ie, whether they were hospitalized or clinically decompensated).

Several limitations exist to using ICD electrograms as surrogates for SCD and spontaneous ventricular arrhythmias. First, what was going to be a nonsustained VT would be classified as sustained if the device provides therapy; in fact, it has been suggested that ICD therapy frequently can be caused by nonsustained VT.19 Second, VT tolerance is a complex issue, and rate is only one of the determinants.20 In our own series of 243 patients with spontaneous tolerated VT and coronary artery disease at the University Hospital Gregorio Marañon, 46 (19%) had a VT cycle length <270 ms, and 32 (13%) had a VT cycle length <250 ms (JA and Mercedes Ortiz, PhD, unpublished observations, 2000), so at least 13% of tolerated VT would have been classified as SCD if an ICD were in place, attesting to the complexity of the mechanisms influencing VT tolerance. A third limitation is ICD-related proarrhythmia, in which the ICD can induce, facilitate, or aggravate ventricular arrhythmias by several mechanisms, usually described in observational studies21–27 (Figure 1). Although the incidence of each of these mechanisms is unknown, they clearly limit the assumption that ICD-observed arrhythmias are spontaneous arrhythmias. Finally, pacing-related ventricular deterioration is possible, with an increase in ventricular arrhythmias.28

The possibility that the device could be proarrhythmic also has been analyzed on clinical grounds. We recently evaluated the results of several randomized ICD trials (primary and secondary prevention) and compared the appropriate ICD therapies with SCD in the control group as an arrhythmic manifestation.29 In all studies, a 2-fold increase was present in arrhythmic events in the ICD group. Because patients with tolerated VT typically have sustained episodes, one could not assume that the device was just “oversensing” episodes that would have terminated spontaneously. Ellenbogen et al30 performed an elegant analysis on data from a randomized ICD trial in patients with dilated cardiomyopathy and low ejection fractions (the Defibrillators in Non-Ischemic Cardiomyopathy Treatment Evaluation [DEFINITE] trial). These investigators hypothesized that because patient allocation was random, spontaneous arrhythmic mortality should have been essentially the same in both groups. Thus, if appropriate ICD shocks were a reliable surrogate for arrhythmic death, appropriate ICD shocks plus SCD in the ICD group would have equaled SCD (arrhythmic death) in the standard therapy group. However, the number of appropriate ICD shocks (n=33) was more than double the number of arrhythmic deaths in the conventional arm (n=15), providing evidence that “counting (appropriate) ICD shocks is not equivalent to counting lives saved by ICD therapy.”30 These data are
similar to the data of Germano et al.29 Somewhat similar observations have been made in at least 2 studies involving VT ablation; patients with an ICD implanted had more VT recurrences than those without an ICD (see below).31,32 In one of these studies, having an ICD implanted was shown to be an independent predictor of VT recurrence.31 Two conclusions can be reached from these studies. First, it seems unjustified to infer clinical consequences (ie, prognosis or probability of recurrence) to a general VT population from the recorded ICD electrograms from those who had the device implanted. Second, the ICD could be producing arrhythmias that it then treats, leading to both an assumed higher incidence of spontaneous arrhythmias and an imagined efficacy.

**VT Surgery as a Model For Substrate Ablation: Reduction of VT Recurrences and SCD**

VT surgery was actively performed at a number of centers in the 1980s when the use of ICDs was limited and catheter ablation for VT was nonexistent. Unfortunately, use of this procedure has nearly vanished because of its relatively high operative mortality and complexity compared with ICD implantation and catheter ablation. However, the results of a combined series of 3 centers showed that SCD in 229 patients rendered noninducible after VT surgery was as low as 4% after a 5-year follow-up.33 In a follow-up of patients undergoing surgical ablative therapy for tolerated and untolerated VT at the University of Pennsylvania, the incidence of SCD was 4% over 5 years and was 0% in patients with single-vessel disease (unpublished observations). As stated earlier, these patients still have a considerable mortality as a result of heart failure. VT surgery included revascularization in a significant number of patients (61% in the Pennsylvania series34), and it could be suggested that revascularization was the curative mechanism. However, the poor results of previous series including revascularization alone or with aneurysmectomy35 along with the scar nature of the VT substrate suggest that revascularization is not the main mechanism of benefit of surgical techniques. Thus, the high antiarrhythmic efficacy of surgical ablative techniques can be regarded as proof of concept for all ablative therapies in the sense that removing the substrate not only suppresses VT recurrence but also lowers arrhythmic mortality in the absence of an increase in the contractile muscle mass and without the improvements in pharmacological therapy developed in subsequent years.

**Antiarrhythmic Drug Therapy for Ventricular Arrhythmias**

The above discussion about the clinical outcome of patients with tolerated VT applies mostly to patients treated with antiarrhythmic drugs and to relatively old series. Three main messages have evolved in recent years in regard to antiarrhythmic drug therapy for ventricular arrhythmias. First, certain drugs such as sotalol, azimilide, and amiodarone can significantly decrease the number of ICD-treated ventricular arrhythmias, demonstrating that they in fact have some antiarrhythmic effects.35–37 Second, in contrast, amiodarone, the most popular drug for ventricular arrhythmias, has repeatedly failed to decrease total mortality in patients with structural heart disease with or without heart failure.38,39 Third, compared with ICD therapy, amiodarone provided worse results in terms of total mortality as both primary prevention40 and secondary prevention in patients having a variety of ventricular tachyarrhythmias except tolerated sustained VT1,2; sotalol was shown to be of no practical use in a general way because in the AVID trial it was used in only 2% of the group assigned to drug therapy.3 Although none of these messages apply specifically to patients with tolerated VT, they certainly would suggest more efficacy in decreasing number of episodes than in reducing the SCD rate.

**Catheter Ablation Abolishing VT**

For more than a decade, radiofrequency catheter ablation has been used to abolish postinfarction VT. Several groups have reported their experience, in most cases as a single-center experience,31,32,41–50 in an observational format, including cases in which the clinically documented VT could be induced and mapped during electrophysiological evaluation. These studies are summarized in the Table, totaling 735 patients in whom post-MI VT ablation was attempted.

**Short-Term Results**

Some differences exist in the ways in which different groups define whether the procedure has achieved short-term success. In general, if the VTs that were induced, mapped, and terminated during radiofrequency application could not be induced afterward, the procedure was considered successful. Some groups added the requirement for noninducibility in a second evaluation a few days after the ablation procedure. A matter of controversy has been the significance of additional VT morphologies different from the clinically documented VT. A full discussion of this subject is beyond the scope of this article. We will say only that in most of these studies, success has referred only to the VTs that were treated with ablation, and nonclinical VT morphologies were approached only if hemodynamically tolerated. If we accept the criteria for success used in each of the studies referred to in the Table, the short-term success rate was 76% if those studies are pooled together.31,32,41–50

Major complications ranged from 0% to 15%, with an average of 6%, including a mortality rate that averaged 1.5% (Table). Although these figures are by no means insignificant, they have to be considered in light of the low LV ejection fraction of the patients in these series (Table) and, perhaps most important, in light of the inclusion of patients in incessant VT and poor clinical condition in whom procedural risk is highest.48,49

**Long-Term Results**

Most of these series have a mean follow-up that extends for >1 year, and some of them extend their mean follow-up for
If all patients submitted to an ablation attempt are considered on an intention-to-treat principle, the probability of a recurrence of a sustained VT during follow-up is significant at a mean value of 33% (Table). However, most investigators have found that the short-term response is predictive of the long-term outcome. In addition, in all series, a large proportion of patients had ICDs implanted (Table), particularly those patients in whom the procedure was thought to be unsuccessful, and in these patients, recurrences had to be assessed by the stored electrograms retrieved from the device with the limitations discussed above. Moreover, it has been shown that VT recurrences are significantly more frequent in patients who have an ICD after VT ablation. It could be argued that the investigators chose to implant an ICD after ablation in patients who were more likely to have VT recurrences, but it has been shown that the implantation of an ICD is an independent predictor of VT recurrence after VT ablation, raising the alternative explanation, as discussed above, that the ICD may in fact be arrhythmogenic in these patients. A third potential explanation is misclassification of nonsustained VT as sustained because the device provided therapy, but we feel that this is unlikely on a significant scale because patients suffering from sustained VT tend to recur with sustained VT. As shown in Figure 2, the overwhelming majority of VT recurrences occurred in patients with an ICD implanted. In this study, with a 46% recurrence rate in the total population, those without an ICD had <10% recurrence rate. It is of note that 106 of the 115 ICDs implanted in this study had been implanted before the ablation procedure, thus excluding a bias in the postablation ICD indication as an explanation for the higher recurrence rate in ICD patients.

### Table: Summary of Published Results in Patients Undergoing Catheter Ablation of Post-MI VT

<table>
<thead>
<tr>
<th>Authors</th>
<th>Patients, n</th>
<th>Mean LVEF, %</th>
<th>Short-Term Success, %</th>
<th>Procedural Mortality, %</th>
<th>Major Morbidity, %</th>
<th>Patients With ICD, %</th>
<th>Mean Follow-Up, mo</th>
<th>VT Recurrences, %</th>
<th>VT Recurrences SCD Total Mortality During Follow-Up</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morady et al</td>
<td>15</td>
<td>27</td>
<td>73</td>
<td>0</td>
<td>0</td>
<td>20</td>
<td>9</td>
<td>13</td>
<td>0/8</td>
</tr>
<tr>
<td>Kim et al</td>
<td>21</td>
<td>32</td>
<td>81</td>
<td>5</td>
<td>0</td>
<td>43</td>
<td>13</td>
<td>45</td>
<td>2/12</td>
</tr>
<tr>
<td>Rothman et al</td>
<td>35</td>
<td>24</td>
<td>86</td>
<td>0</td>
<td>11</td>
<td>51</td>
<td>14*</td>
<td>31</td>
<td>0/16</td>
</tr>
<tr>
<td>Stevenson et al</td>
<td>52</td>
<td>33</td>
<td>71</td>
<td>2</td>
<td>6</td>
<td>44</td>
<td>18</td>
<td>31</td>
<td>NA</td>
</tr>
<tr>
<td>Ortiz et al</td>
<td>34</td>
<td>31</td>
<td>62</td>
<td>0</td>
<td>3</td>
<td>44</td>
<td>26</td>
<td>38</td>
<td>6/19</td>
</tr>
<tr>
<td>El-Shalakany et al</td>
<td>15</td>
<td>26</td>
<td>93</td>
<td>0</td>
<td>NA</td>
<td>13</td>
<td>15</td>
<td>27</td>
<td>2/12</td>
</tr>
<tr>
<td>Calkins et al†</td>
<td>146</td>
<td>31</td>
<td>75</td>
<td>2.7</td>
<td>5</td>
<td>79</td>
<td>8</td>
<td>46</td>
<td>3/27</td>
</tr>
<tr>
<td>O’Callaghan et al††</td>
<td>55</td>
<td>32</td>
<td>82</td>
<td>1.8</td>
<td>7</td>
<td>70</td>
<td>39</td>
<td>NA‡</td>
<td>NA‡</td>
</tr>
<tr>
<td>Borger et al§‡</td>
<td>151</td>
<td>29</td>
<td>79</td>
<td>2.2</td>
<td>8</td>
<td>55</td>
<td>34‡</td>
<td>23</td>
<td>5/38**</td>
</tr>
<tr>
<td>Della Bella et al§</td>
<td>124</td>
<td>34</td>
<td>73</td>
<td>0.8</td>
<td>3</td>
<td>20</td>
<td>41</td>
<td>28</td>
<td>19/91</td>
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<tr>
<td>O’Donnell et al§‡</td>
<td>109</td>
<td>NA†</td>
<td>72</td>
<td>0</td>
<td>6</td>
<td>26</td>
<td>61</td>
<td>23</td>
<td>3/63</td>
</tr>
<tr>
<td>Segal et al</td>
<td>40</td>
<td>36</td>
<td>82</td>
<td>2.5</td>
<td>15</td>
<td>65</td>
<td>36</td>
<td>57</td>
<td>NA</td>
</tr>
<tr>
<td>Total</td>
<td>797</td>
<td>708</td>
<td>76</td>
<td>1.5</td>
<td>6</td>
<td>48</td>
<td>33</td>
<td>14%</td>
<td>2.5%</td>
</tr>
</tbody>
</table>

LVEF indicates LV ejection fraction; NA, not applicable.
*Approximately.
†Only 62% with coronary artery disease.
‡Recurrences described in an actuarial fashion.
§The denominator cannot be established precisely from the article; it is ≤17.
Data from this series refer to the 89 patients with post-MI VT except when mentioned otherwise.
||The whole group of 151 patients.
††The numerator is ≤5.
**One patient died of an unknown cause.
††This value is <0.35 in 74%.

≥3 years. If all patients submitted to an ablation attempt are considered on an intention-to-treat principle, the probability of a recurrence of a sustained VT during follow-up is significant at a mean value of 33% (Table). However, most investigators have found that the short-term response is predictive of the long-term outcome. In addition, in all series, a large proportion of patients had ICDs implanted (Table), particularly those patients in whom the procedure was thought to be unsuccessful, and in these patients, recurrences had to be assessed by the stored electrograms retrieved from the device with the limitations discussed above.

Moreover, it has been shown that VT recurrences are significantly more frequent in patients who have an ICD after VT ablation. It could be argued that the investigators chose to implant an ICD after ablation in patients who were more likely to have VT recurrences, but it has been shown that the implantation of an ICD is an independent predictor of VT recurrence after VT ablation, raising the alternative explanation, as discussed above, that the ICD may in fact be arrhythmogenic in these patients. A third potential explanation is misclassification of nonsustained VT as sustained because the device provided therapy, but we feel that this is unlikely on a significant scale because patients suffering from sustained VT tend to recur with sustained VT. As shown in Figure 2, the overwhelming majority of VT recurrences occurred in patients with an ICD implanted. In this study, with a 46% recurrence rate in the total population, those without an ICD had <10% recurrence rate. It is of note that 106 of the 115 ICDs implanted in this study had been implanted before the ablation procedure, thus excluding a bias in the postablation ICD indication as an explanation for the higher recurrence rate in ICD patients.

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**Figure 2.** Kaplan-Meier curve showing freedom from arrhythmia recurrence among patients who underwent catheter ablation of VT subclassified by whether the patient was discharged with (Yes) or without (No) an ICD. Reprinted from Calkins et al, with permission of the publisher. Copyright © 2000, Elsevier.
For these reasons, a search was made through each study in the Table to detect the proportion of patients having VT recurrences and/or SCD among those who had a procedure with short-term success and no ICD implanted. The VT recurrence rate in 286 such patients was 14%, less than half that in the total population. However, the most striking finding in these people is the low SCD rate of 2.5% among 319 patients (Table).

Additional information comes from patients having an ICD before the ablation procedure in whom the indication for ablation comes from the intolerable number of ICD shocks. In these patients, the arrhythmic activity can be judged quantitatively and in a similar fashion before and after ablation by the number of ICD therapies. A successful ablation procedure dramatically decreased ICD therapies$^{52,53}$ from a mean of 60 therapies per month before ablation to a mean of 0.1 therapies per month after ablation.$^{51}$ Interestingly, ICD therapies decreased even when the procedure was thought to be unsuccessful.$^{51}$

The above studies, although not controlled, strongly suggest that ablation therapy can be efficacious in most tolerated post-MI VT patients and that this can be predicted by the short-term result. Most important, they show that although recurrences can occur, SCD risk is quite low if the ablation procedure is successful in the short term.

### Catheter Ablation of the VT Substrate

It is clear that both the need for inducibility of the clinical arrhythmia$^{52}$ and the need for clinical tolerance to allow mapping limit the proportion of patients amenable to VT ablation if energy application has to be delivered during ongoing VT.$^{53}$ The introduction in clinical practice of a number of nonfluoroscopic navigation systems and the recognition of additional criteria to identify the substrate$^{54–56}$ have allowed more precise identification of the VT substrate and consequently ablation of VT during sinus rhythm. Since the pioneer work of Marchlinski et al,$^{57}$ several other groups have used a variety of criteria to recognize and ablate the substrate, reporting good results and creating the expectation that an ablation approach also can be offered to post-MI VT patients in a more general fashion.$^{58–64}$

Very recently, the results of the first randomized controlled trial involving catheter ablation for ventricular arrhythmias have been presented, so far only as an abstract.$^{65}$ This interesting trial randomized 126 ICD candidates to undergo or not undergo a substrate ablation procedure in addition to conventional therapy, including the ICD. During a follow-up of 24 months, the ablation group had a significant decrease in appropriate ICD therapies (31% versus 15%) and appropriate shocks (24% versus 8%), with a tendency (nonsignificant) toward a decrease in total mortality (17% versus 8%). This randomized controlled trial, along with previous work, provides support for the concept that catheter ablation approaches the arrhythmia substrate is an efficacious protection against ventricular arrhythmias.

### SCD and Complications in Patients With an ICD

ICDs have been proved to be efficacious to decrease SCD and total mortality in patients at a risk of SCD. However, despite the tendency to believe that they offer complete protection against arrhythmic death, SCD can occur in ICD-treated patients.$^{7,21,66}$ In a controlled randomized trial that included patients with coronary artery disease and a similar degree of LV dysfunction that had SCD or cardiac arrest as its main end point with a blinded assignment of mode of death, the 3-year actuarial incidence of cardiac arrest/SCD in the ICD group was 6%,$^{67}$ certainly not lower than the 2.5% found in successfully ablated patients.

It could be argued that even that 2.5% SCD rate could have been decreased further with the ICD. However, this should be balanced against the risks related to ICD therapy. Although some clinical trials have reported up to >800 ICD implants without operative mortality,$^{70,68}$ a prospective multicenter trial from the late 1990s that included 778 patients$^{69}$ and a recent study reporting on 30,984 unslected patients implanted with an ICD in 2003$^{70}$ observed an almost 1% operative mortality. This operative mortality increased to 11% in the case of resynchronization therapy.$^{70}$ Significant complications of ICD therapy are invariably reported even in recent series, ranging from 3% in clinical trials with short follow-up$^{69}$ to 31% in a single-center prospective experience with a mean follow-up of almost 4 years.$^{71}$ In the Medicare population of 30,984 patients, the acute complication rate was 10.8%,$^{70}$ and Moreover, some studies have found that among ICD patients, those having therapies (shocks in some studies) have higher mortality.$^{66,72,73}$ Although patients having shocks may be sicker, in a subanalysis from the Multicenter Automatic Defibrillator Trial II (MADIT-II),$^{66}$ only marginal differences existed between patients having and not having ICD therapies (shocks in 144 of 169 VT patients), and prognosis was worse for the former, suggesting the alternative hypothesis that ICD shocks could be harmful. Additional support for this idea comes from the observations in several studies that patients having frequent shocks (in the form of electrical storm or multiple consecutive discharges) have a poor prognosis.$^{74–77}$

The long-term effects of the not-so-infrequent lead complications, leading to inappropriate shocks and proarrhythmia, may add to the ICD risk,$^{27}$ making ICD therapy even less attractive for a population with severe structural heart disease but low SCD risk. In addition, it has been demonstrated that both adverse symptoms and ICD shocks decrease the quality of life$^{78}$ in ICD patients, an important consideration in a patient population likely to experience ICD shocks. In contrast, quality of life improves in ICD recipients experiencing frequent shocks after a successful ablation procedure but not after an unsuccessful ablation.$^{51}$

Probably because of all the above considerations, in the recently published American College of Cardiology/American Heart Association Task Force/European Society of Cardiology guidelines, the therapeutic indications in patients
with post-MI VT (both ICD and catheter ablation) have a level of evidence C. 79

Final Considerations

Despite our limited knowledge about the outcome of patients with tolerated post-MI VT, the information available at the present time suggests that catheter ablation, if successful in the short term, confers both qualitative and quantitative protection against VT recurrence and SCD. Of note, although recurrence of a tolerated VT is not so rare, the SCD rate in these patients is extremely low. The recently introduced technique of substrate ablation for untolerated VT offers ablative options to a more general post-MI VT population and has been shown in a randomized controlled trial to decrease ICD therapies and shocks. Thus, catheter ablation can be considered a therapeutic alternative for those patients with post-MI tolerated VT in whom the procedure produces a satisfactory short-term result. The virtual absence of SCD in patients with tolerated VT and single-vessel disease makes such patients ideal candidates for ablation as a first-line therapy.

Disclosures

Dr Almendral has received honoraria from Medtronic, Guidant, Johnson & Johnson, and St Jude Medical for educational activities and lectures. Dr Josephson is consultant for Medtronic and has received honoraria for educational activities from Biosense-Webster.

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Response to Almendral and Josephson

David J. Callans, MD

The elegant article by Almendral and Josephson makes many important points, but I am concerned about 2 of their assumptions. The first is keeping score by means of arrhythmic instead of total mortality. All major implantable cardioverter-defibrillators (ICD) trials have used the metric of total mortality for the following reasons: Even with a blinded review panel, the treachery of assigning a specific cause of death is well known; this problem is greatly magnified when investigators are directly caring for the patients in the trial. In addition, the concept of competing mortality risk, in which a patient is saved from sudden death only to succumb to heart failure death soon after, is not compelling. Finally, it has been demonstrated (as in the Defibrillators in Acute Myocardial Infarction Trial [DINAMIT]) that ICDs may have a negative effect on nonsudden mortality despite reducing arrhythmic death. Second, although I share their enthusiasm for catheter ablation, it is not clear that this strategy protects patients against sudden death. It may be that such patients have a low incidence of sudden death, but it does not necessarily follow that this is an effect of ablation, given the incidence of recurrent ventricular tachycardia. However, most patients who present with tolerated ventricular tachycardia meet the criteria for primary-prevention ICD therapy. Does it make any sense to worry less about electrical storm in patients with transvenous implantable cardioverter-defibrillators: incidence, management and prognostic implications. J Am Coll Cardiol. 1998;32:1909–1915. It may be that such patients have a low incidence of sudden death, but it does not necessarily follow that this is an effect of ablation, given the incidence of recurrent ventricular tachycardia. However, most patients who present with tolerated ventricular tachycardia meet the criteria for primary-prevention ICD therapy. Does it make any sense to worry less about electrical storm in patients with transvenous implantable cardioverter-defibrillators: incidence, management and prognostic implications. J Am Coll Cardiol. 1998;32:1909–1915.

All Patients With Hemodynamically Tolerated Postinfarction Ventricular Tachycardia Do Not Require an Implantable Cardioverter-Defibrillator

Jesús Almendral and Mark E. Josephson

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