Ventricular tachycardia (VT) and ventricular fibrillation (VF) are important causes of mortality and morbidity in a wide variety of heart diseases. Implantable cardioverter-defibrillators (ICDs) are now widely used in patients who survive sustained VT or VF that is not attributable to a transient correctable cause, or who are at high risk for recurrent arrhythmia.1,2 ICDs are dramatically effective for terminating VT and VF. The extent to which VT termination extends survival, however, depends on the severity of the underlying heart disease and associated comorbidities.3–6 The occurrence of VT or VF is associated with increased mortality and heart failure hospitalizations in ICD patients, despite effective termination of the arrhythmia.7 VT or VF is associated with heart disease severity and, in some patients, is a marker for deterioration or intermittent illness. Whether short VT or VF episodes and the hemodynamic and neurohormonal responses they may elicit contribute directly to adverse outcomes is not known.7,8 Frequent or incessant VT can cause hemodynamic deterioration, and death from uncontrollable VT occasionally occurs. ICD shocks in response to VT can damage myocardial cells and elicit sympathetic responses.8 ICD shocks have been associated with mortality in post hoc analyses of ICD trials, but whether there is a direct causal relationship of shocks to cardiac mortality and heart failure remains a controversial topic.8,9

Patients With Arrhythmia Symptoms or ICD-Detected Arrhythmia

A patient who reports arrhythmia symptoms or a perceived shock should be promptly evaluated with an ICD interrogation to determine whether the event was truly a ventricular arrhythmia, rather than an inappropriate ICD therapy in response to rapid atrial fibrillation, or electric noise from a lead malfunction, which warrants further intervention.10 Those who have had VT or VF will broadly fall into 1 of 3 groups. The first are typically stable with modest heart disease and preserved functional capacity, who return to a stable state following ICD termination of an arrhythmia. The VT or VF episode is an isolated event, and a substantial extension of survival is likely. The second group comprises patients for whom the VT/VF event signifies an unfavorable change in their previously stable clinical status. The arrhythmia may be the sentinel marker for heart failure, myocardial ischemia, worsening valve function, or an intercurrent illness, and they are typically worse after the event. The third group are patients who have severe heart disease and a poor prognosis. Following arrhythmia termination, the competing risk of progressive heart disease limits survival, and for many, the VT/VF episode also marks the onset of a rapid decline in their clinical status.6

Monomorphic VT Versus Polymorphic VT/VF

Details of the arrhythmia episode that can be retrieved from the ICD can be important for suggesting precipitating factors and guiding evaluation and additional therapy. VT can be monomorphic, with each QRS resembling the preceding and following QRS, or polymorphic, with a continually changing QRS configuration that usually degenerates rapidly to VF if it does not terminate promptly. It is important to recognize, however, that arrhythmia detection algorithms in ICDs primarily define the VT rate (usually expressed as cycle length) and cannot discriminate between various ventricular arrhythmia types. The ICD is typically configured for a VF zone for fast VTs (eg, >220/min) and 1 or 2 VT zones for slower arrhythmias. A fast monomorphic VT with a rate falling into the VF zone is often erroneously reported to the clinician as VF, whereas a polymorphic VT with a rate that falls in a designated VT zone may be reported as VT. Review of the stored electrograms is required to distinguish the difference. Electrograms in an ICD derive from various bipolar configurations involving the tip and ring electrodes or shocking coils on the pacing/defibrillating lead, or a wide bipolar recording between one of the ventricular electrodes and the ICD casing. Monomorphic VTs typically have constant electrogram morphology, and the rate usually stabilizes within a few beats.11 Polymorphic VTs have a changing rate and usually demonstrate varying electrogram morphology. Exceptions do occur, in particular with rapid VTs, and there is often some variability at the onset of a monomorphic VT. Sustained monomorphic VT is usually due to reentry in a region of myocardial scar in patients with structural heart disease. The scars associated with VT are a mix of fibrosis and surviving myocytes that provide the path for reentry.12 In ischemic heart disease, the area of scar is usually evident as an old infarction. In nonischemic disease, scars are also present, likely related to areas of replacement fibrosis, the causes for which are not well defined. The scars are evident on MRI as areas of late gadolinium enhancement.13,14 Scars are a durable arrhythmia substrate, such that the likelihood of a VT recurrence following the initial episode of scar-related VT can exceed 20% per year.15,16 ICDs, however, often can terminate monomorphic VTs by applying a burst of pacing.
(antitachycardia pacing,) that is painless.17–19 Appropriate ICD programming to allow this option is therefore important. A shock is applied if antitachycardia pacing fails, accelerates VT, or causes VT to transition to VF.

Polymorphic VT indicates a continually changing ventricular activation sequence, often associated with a changing arrhythmia substrate. Acute myocardial ischemia and metabolic derangements are the major concerns, although migrating reentry circuits in a region of scar can be the cause. Polymorphic VT is often associated with disorders that increase dispersion of repolarization, such as the acquired and congenital long-QT syndromes and Brugada syndrome. Myocardial hypertrophy and failure predispose to these arrhythmias. In some cases, the pacing features of ICDs designed to minimize ventricular pacing can result in long-short R-R interval sequences, and may initiate polymorphic VT in a susceptible heart.20–22 Antitachycardia pacing is unlikely to be effective for terminating polymorphic VT. Thus, the diagnostic and therapeutic considerations for polymorphic VTs are different than those for scar-related monomorphic VTs.

**Therapy to Reduce VT**

Episodes of VT that cause symptoms or perceptible hemodynamic effects usually warrant therapy to reduce or prevent recurrences, although an impact of antiarrhythmic therapy on mortality has yet to be proven. It should also be recognized that, although occurrence of VT or VF may be a marker for disease severity and deterioration, therapies initiated in response to arrhythmias also have the potential to adversely affect outcomes. In patients with significant structural heart disease, β-blockers and amiodarone are the common pharmacological options. Amiodarone combined with a β-blocker was more effective than sotalol or an alternative β-blocker.16 Amiodarone has well-known noncardiac toxicities and was associated with increased mortality in patients with class III heart failure who did not have ICDs.4 The most common cardiac adverse effect of amiodarone is bradycardia, which has the potential to impair ventricular function by increasing ventricular pacing from the ICD in patients who do not otherwise require pacing.23

For patients with monomorphic VT, catheter ablation is often an option. In multicenter trials, catheter ablation reduces VT recurrences in over two thirds of patients with recurrent drug-refractory VT due to a previous myocardial infarction.24 Outcomes for VT due to nonischemic cardiomyopathies and arrhythmogenic RV cardiomyopathy are less well studied. The arrhythmia substrate is more variable and more likely to require epicardial ablation, which requires more specific expertise and is associated with additional risk.25,26 In postinfarction patients with frequent, recurrent VT, procedure-related mortality is ≈3%, but most deaths are attributable to continued uncontrollable VT when the procedure fails. Catheter ablation can be life-saving in patients with incessant VT or very frequent VT, often referred to as electrical storms.27 Technologies for guiding ablation of the VT substrate during stable sinus or paced rhythm are now widely available, allowing treatment of VTs that are not hemodynamically stable. Specific expertise is required.28 A recent consensus group recommended that catheter ablation be considered early for patients with recurrent monomorphic VT, before multiple recurrences and drug failures that may lead to posttraumatic stress disorder and serious psychological disability, provided that it can be performed with low risk, in general, at an experienced center.28,29 Catheter ablation is not usually an option for treating infrequent, polymorphic VT, but small series have shown that ablation targeting premature ventricular complex foci that initiate recurrent VT/VF,30,31 or the epicardial outflow tract scar in Brugada syndrome, can control recurrent polymorphic VT/VF in selected patients.32

**Further Needs**

Many issues require further study. Improvements in cardiac therapies and the widespread use of ICDs are increasing the number of patients with severe cardiac disease, for whom recurrent episodes of VT and VF are prominent. Large scars, intramural reentry circuits, and evolution of a new arrhythmia substrate are features that limit the efficacy of available therapies for these patients. Arrhythmia control without improvement in cardiac function has limited benefit, and often arrhythmia control cannot be achieved. End-of-life care for this population is challenging and guidance to physicians and families is needed.33,34

The relation of ventricular arrhythmias to mortality and heart failure suggests that therapies that prevent VT/VF might improve outcome, but all therapies have the potential for adverse effects that could negate benefit. Early use of catheter ablation, after an initial episode of sustained VT, has been evaluated in 2 trials in patients with ischemic heart disease.15,35 Ablation reduced VT recurrences, but neither trial was of sufficient size to assess the impact on survival. Ventricular scars are metabolically active and dynamic, suggesting other potential approaches to modifying the electrophysiological substrate for VT.36–40 In addition, the potential exists to identify patients who have scar-related reentry circuits, before the occurrence of a first VT event, which could theoretically allow early implementation of therapies to select patients.34

The availability of home-monitoring systems for ICD patients will further inform our understanding of the relation between ventricular arrhythmias, cardiac remodeling, and mortality as heart disease evolves.41–43 Arrhythmia episodes are identified and characterized early, often shortly after the episode, facilitating early patient assessment and treatment. Use of this technology has the potential to facilitate the definition of arrhythmia management strategies in clinical trials. Such strategies could include the early use of ablation or antiarrhythmic drugs, and will hopefully focus not only on mortality, but also on the prevention of heart failure and the improvement of quality of life for patients at risk for life-threatening arrhythmias.

**Disclosures**

Dr Stevenson is a coholder on a patent for needle ablation that is consigned to Brigham and Women’s Hospital. Dr John has received speaking honoraria from St. Jude Medical.
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