Ventricular Tachycardia Associated With Myocardial Infarct Scar
A Spectrum of Therapies for a Single Patient

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Case: A 73-year-old woman is referred for management of recurrent ventricular tachycardia (VT). She had suffered an inferior wall myocardial infarction in 1970. Fifteen years later, she presented with a wide QRS tachycardia, palpitations, and dizziness. Therapy with amiodarone was initiated but discontinued in 1997 because of toxicity, and she received an implantable cardioverter-defibrillator (ICD). She did well until July 2000, when she had several shocks from the ICD, all of which were preceded by syncope. Interrogation of the ICD confirmed 23 episodes of VT, 20 asymptomatic runs terminated by antitachycardia pacing (ATP), and 3 episodes requiring cardioversion from the ICD. Her left ventricular ejection fraction was 25%. Sotalol failed to prevent VT recurrences and mexiletine produced nausea and tremor.

She was referred for catheter ablation. An echocardiogram revealed akinesis of the inferior wall and no left ventricular thrombus. In the electrophysiology laboratory, programmed stimulation induced 5 different morphologies of VT (Figure 1) with rates ranging from 180 to 220 bpm. Because the induced VTs were unstable, producing hypotension and often changing from one VT to another, catheter mapping and ablation were performed largely during sinus rhythm, guided by electrogram characteristics and pacing during sinus rhythm (pace-mapping) that marked the location of the infarct scar and likely reentry paths in the subendocardium. After placement of lines of radiofrequency (RF) lesions through these abnormal regions, only ventricular flutter (280 bpm) was inducible; the slower VTs were no longer inducible. There have been no VT recurrences in the 18 months of follow-up after ablation.

Discussion

Ventricular arrhythmias associated with myocardial infarction (MI) occur in 2 distinct phases. During the acute phase of infarction, polymorphic VT that degenerates to ventricular fibrillation is most common. In the weeks that follow, the healing infarct undergoes structural changes. Fibrosis creates areas of conduction block and also increases separation of myocyte bundles, slowing conduction through myocyte pathways in the border of the infarct.1,2 These pathways or channels can support stable reentry circuits, leading to monomorphic VT, when an appropriate trigger (such as a change in sinus rate or a premature depolarization) occurs. After surviving the acute phase of the infarct, monomorphic VT may emerge at any time. With present management of myocardial infarction, the incidence of sustained VT is relatively low, and fewer than 5% of infarct survivors have inducible VT when studied early after the infarct.3 Patients with large infarcts, often those who are not successfully reperfused, are at greatest risk for VT. Although the first 6 months after infarction is thought to be the period of greatest risk for VT and sudden death, some patients develop VT much later, as in the case presented above. Whether late development of VT is related to electrical and mechanical remodeling or additional ischemic events contributing to the development of the substrate for the VT is not known. Because the arrhythmia substrate for late VT is relatively fixed, this type of VT tends to be recurrent and difficult to suppress with medications.

Antiarrhythmic Drugs and ICDs

Antiarrhythmic drugs are frequently prescribed because they alter the electrophysiological properties of the reentrant circuit and suppress potential triggers for the development of VT. However, within 2 years, >40% of patients being treated for sustained VT will experience recurrences.4 There is a risk that a VT recurrence will cause sudden death, particularly in
patients with depressed ventricular function and those who have presented with a hemodynamically poorly tolerated VT.5

Three recent trials support the superiority of ICDs over antiarrhythmic drug therapy for prolonging survival and preventing sudden death in survivors of sustained ventricular arrhythmias.6,7,8 Thus, an ICD is first-line therapy for these patients. For many patients, placement of an ICD prevents the side effects of antiarrhythmic drugs. The most effective drug, amiodarone, produces side effects in almost 75% patients within 5 years. These side effects include hypothyroidism (5% to 25%), blue skin (1% to 6%), corneal pigmentation (1%), pulmonary toxicity (1% per year), tremor, or other neurological toxicity. ICD risks include device failure, lead fractures, and infection, but these are infrequent. ICDs also provide back-up pacing that protects against bradyarrhythmia.

Although ICDs extend survival, they only treat the arrhythmia when it occurs, and do not prevent arrhythmia recurrences. Follow-up is required for the infrequent possibility of device malfunction. Within a year of ICD implantation, 68% of patients have recurrent episodes of VT.6 Most monomorphic VTs can be terminated by antitachycardia pacing, which is painless and often asymptomatic, but some patients require electrical cardioversion via the ICD. When VT initially recurs, and particularly when it becomes frequent, an evaluation is required to address potential aggravating factors, such as myocardial ischemia, electrolyte abnormalities, or decompensated heart failure. Most patients with frequent monomorphic VT require additional therapy to reduce VT episodes.

Interactions Between ICDs and Antiarrhythmic Agents
Antiarrhythmic drug therapy decreases the frequency of VT episodes in patients with ICDs and may make the VT more amenable to antitachycardia pacing therapy. For some patients, drug therapy is problematic. The antiarrhythmic agent may slow the sinus rate, causing the patient to be paced, potentially with loss of AV synchrony, or producing adverse hemodynamic effects from right ventricular pacing. Antiarrhythmic agents may slow the rate of VT when it occurs such that it falls below the detect rate of the ICD, or falls into the range where sinus tachycardia can also occur, making distinction of sinus tachycardia from VT difficult. Some drugs,
notably amiodarone, can increase the energy required for defibrillation, theoretically reducing the likelihood that ventricular fibrillation would be effectively treated by the ICD.

**Ablation**

RF catheter ablation is a useful adjuvant therapy for frequent episodes of symptomatic VT. Initial ablation studies used careful mapping during VT to identify a critical part of the VT reentry circuit where the relatively small RF ablation lesions could interrupt reentry. The presence of hemodynamically stable VT facilitated mapping and ablation attempts. Patients with unstable VTs that did not allow detailed mapping were largely excluded from initial ablation attempts. Developments in the understanding of the nature of reentrant circuits and in methods to identify the region of the infarct scar and potential reentrant circuit paths through the scar now allow catheter ablation to be effective for many patients who have multiple and unstable VTs.9,10

Catheter mapping systems allow electrophysiological data to be integrated in a 3-dimensional anatomic reconstruction of the ventricle (Figure 2A). The map of the left ventricle in Figure 2A, was created during sinus rhythm. The catheter was moved from point to point around the ventricle. At each point, the electrogram amplitude was plotted and color coded, with normal amplitude areas (>1.5 mV) indicated as purple and progressively lower-amplitude regions indicated by blue, green, yellow, and red regions. This patient has a large infero-posterior low-amplitude region consistent with her prior infarction. The area is much larger than that which can be completely ablated by RF energy; however, additional data can be obtained to focus the ablation to an appropriate region.10

Inducing VT once in the electrophysiology laboratory allows confirmation of the diagnosis. In addition, the QRS morphology of the VT is obtained for use as a rough guide to the location of the reentry circuit in the infarct. In lead V1, a right bundle-branch block–like morphology VT suggests a left ventricular origin, and left bundle-branch block–like morphology predicts an origin in the right ventricle or in the interventricular septum. Dominant S waves in V2, V3, and V4 suggest an exit near the apex. Dominant R waves in these leads suggest an exit closer to the mitral annulus. Then, during sinus rhythm, pacing from the mapping catheter (pace-mapping) at sites around the infarct region and comparing the paced QRS with the VT morphology helped identify the VT reentrant circuit.11 The circuits can be large and multiple circuits are common.

In the case presented, 5 different VTs were inducible. Figure 2A shows that pace mapping at a site in the low-voltage infarct region, located between two areas of dense unexcitable scar (gray regions), produced a QRS morphology similar to that of one of the VTs. To gain further confirmation that this region was involved in VT, the mapping catheter was placed at the site and VT was induced. After assessing the pattern of electrical activation, burst pacing was initiated to terminate VT. The effects of pacing (entrainment mapping) confirmed that this site was in the circuit (Figure 3). During stable sinus rhythm, a line of RF lesions (line 1) was then created through the target region. After the initial RF line was created, programmed stimulation induced other VT morphologies. On the basis of pace-mapping, additional RF lesions (line 2) were created (Figure 2B), which abolished inducible monomorphic VT.

**The Role of VT Ablation**

ICDs are first-line therapy for many patients with recurrent VT. When antiarrhythmic drug therapy fails to control symptomatic recurrences of VT, catheter ablation should be considered and can be expected to reduce the frequency of recurrent VT in >75% of patients.9,10,13,14 In experienced centers, ablation is now performed regardless of whether the VT rate is rapid and is associated with hemodynamic collapse. The major procedural risks are related to thromboembolism (1.2%), perforation (0.3%), and vascular access complications.15 The procedures can be long and are facilitated by the use of 3-dimensional reconstructions of the ventricular anatomy.

When ablation fails, it is usually because of existence of portions of the reentrant circuits deep to the endocardium where they cannot be interrupted with standard endocardial ablation techniques. Ablation with saline-irrigated cooled ablation catheters and percutaneous epicardial mapping and ablation approaches are being evaluated that may allow some of these VTs to be ablated.16,17 Nonpharmacological therapies, such as RF ablation, have an increasingly important role in the management of VT after myocardial infarction, thus expanding the array of options available to clinicians.

**References**

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