Sustained monomorphic ventricular tachycardia (VT) in patients with heart disease is usually due to reentry involving a region of myocardial scar. Most commonly, the scar is due to prior myocardial infarction and associated with significantly depressed left ventricular function. Scars create a very stable substrate for VT, such that up to 70% of patients who present with sustained monomorphic VT late after myocardial infarction will have another episode within the next 2 years.1,2 Scars also occur in patients with nonischemic myocardial infarction will have another episode within the next 2 years.1,2 Scars also occur in patients with nonischemic cardiomyopathies (NICM) but are generally small, averaging less than 4% of the ventricular myocardium in one study using MRI.3 Replacement fibrosis is a likely mechanism. Consistent with the less frequent and smaller areas of confluent scar in nonischemic cardiomyopathies, sustained monomorphic VT is relatively infrequent. In patients with depressed left ventricular function who received an implantable cardioverter-defibrillator (ICD) for primary prevention, 15% had spontaneous VT or ventricular fibrillation detected by the ICD during a follow-up of 29 months, and approximately half of these arrhythmias were monomorphic VT.4

Because large scars are not common in NICM, the possibility of bundle-branch reentry, which may occur without large scars, is an important consideration when sustained VT is encountered but accounts for fewer than 10% of VTs in this patient population.5 It has become increasingly clear that ventricular scars are a major factor causing sustained monomorphic VT in patients with NICM.6,7 Of patients who have recurrent episodes of VT, who are considered for catheter ablation, the vast majority have evidence of ventricular scars, based on voltage mapping or late Gd enhancement (LGE) on MRI.8–11 In a series of 61 patients with NICM (mean left ventricular ejection fraction of 25%) without a history of sustained VT, who were receiving ICDs, 51% had areas of LGE on MRI; during follow-up, ICD-treated VT or ventricular fibrillation occurred in 29% of those with LGE but in none of those without LGE.6

In contrast to the usually large endocardial scars in patients with postinfarction VT, scars causing VT in nonischemic cardiomyopathy are more variable in location.5–11 They are often intramural and extend to the epicardium. Those associated with VT often abut a valve annulus.12 When endocardial ablation fails, epicardial mapping often identifies areas of low-voltage scar, where the VT can be ablated. Multiple morphologies of inducible VT are common, indicating multiple potential reentry circuits and a complex arrhythmia substrate. Ablation for sustained monomorphic VT in NICM is often more challenging and less successful than for patients ischemic cardiomyopathy.1,13

Anatomic obstacles appear to be an important cause of failure of catheter ablation. Circuits that are intramural in location may escape detection.9,11,14,15 When a circuit is identified or suspected, it may not be possible to create ablation lesions that reach the target. Some epicardial circuits are beneath epicardial fat or protected by an overlying coronary artery.16 Scar-related VTs in the aortic mitral continuity region at the base of the heart and deep to papillary muscles are particularly challenging.14

A direct cardiac surgical approach offers a potential solution to these obstacles. Deep lesions can be achieved with surgical application of cryoablation, which achieves temperatures substantially colder (∼160°C or colder) than those that can be achieved with present catheter cryoablation. Dissection through epicardial fat and myocardium can reach intramural circuits. Coronary arteries can be avoided under direct vision or occasionally mobilized for ablation of tissue beneath the vessel.

Although surgery can potentially treat regions that are inaccessible to catheter ablation, surgical ablation for sustained monomorphic VT is now relatively rare.17 Surgical morbidity compared with percutaneous procedures is a major concern. Mapping to identify the ablation target can be more difficult in the operating room. General anesthesia and cooling of the heart limit arrhythmia inducibility. The tools for operative mapping have not kept up with the capabilities of catheter mapping.18 Although the epicardium is easily explored at surgery, endocardial mapping requires cardiac incisions for access and cardiopulmonary bypass, which may also affect VT inducibility. Assessment of inducibility of VT after ablation, when the heart is warm, is also a challenge in the operating room, impeding assessment of end points of ablation.

In this issue of Circulation: Arrhythmia and Electrophysiology, Anter and colleagues19 report their experience with the use of surgical ablation in 8 patients with recurrent VT caused by nonischemic cardiomyopathy. All had failed attempted endocardial radiofrequency ablation, and percutane-
ous epicardial ablation either failed or was not feasible. Of the 6 patients with follow-up, spontaneous VT was absent in 4 and substantially improved in 2 patients, respectively.

Interestingly, ablation was not guided by intraoperative mapping. Surgical freezing was performed at sites identified by prior catheter mapping and ablation. In one case, empirical ablation of visually identified epicardial scar was performed after emergency surgery for right ventricular laceration and tamponade. The effectiveness of this approach suggests that failure of catheter techniques was related to inability to create lesions that reached arrhythmia substrate rather than a failure to identify the region involved. The observation that in 3 patients, no VT was inducible at the end of the procedure but VT recurred soon after, suggesting transient injury of the arrhythmia substrate, further supports this consideration.

Although these findings show that surgery guided by prior endocardial/epicardial mapping without intraoperative mapping can be effective in some patients, the possibility remains that intraoperative mapping might achieve success with smaller areas of ablation. Despite the advantages of surgical ablation for creating deep, large lesions, VT was not completely abolished; VT recurred during follow-up in 2 patients, and 5 of 6 patients remained on antiarrhythmic medications. It is possible that intraoperative mapping would have identified other target areas for ablation in these patients.

Catheter-based substrate mapping now allows identification of scar and targeting of potential scar-related channels during stable sinus rhythm. However, the less precise targeting of reentry circuit channels is accompanied by more extensive ablation, increasing the likelihood that the ablation area will include the reentry circuit. When confined to low-voltage areas of infarct scar, substrate-guided ablation does not appear to adversely affect ventricular function.20 There is a trend for increasingly extensive catheter ablation to abolish all regions of conduction through scars in nonischemic as well as ischemic cardiomyopathies. Extension of this approach to methods that create larger ablation lesions, such as surgical ablation, probably will have greater risks of damage to functioning myocardium. One could conceive of freezing areas of visually identifiable scar in the operating room. However, intramural scars, present in 4 patients on MRI in this series, would escape detection by this method. The risk of extensive “substrate ablation” may be of greater concern in nonischemic cardiomyopathy, where areas of scar may be smaller and the target for ablation may be intramural, beneath areas of normal myocardium. Incipient VT and recurrent episodes of life-threatening VT warrant aggressive therapy. Application of aggressive ablation to less severe situations requires careful assessment of risks and potential benefit and will warrant ongoing study.

As emphasized by the authors, these patients are drawn from the severe end of the VT spectrum and represent a small portion (8 of 144) of patients with nonischemic cardiomyopathy referred for ablation. They had failed catheter ablation and antiarrhythmic drug therapy and probably were thought to be acceptable surgical candidates. The authors have extensive experience with management of VT and with arrhythmia surgery, yet risks are clearly substantial. Two patients died in the postoperative period of heart failure and sepsis, respectively.

Anter and colleagues19 show that surgical ablation by an experienced team is an option for selected patients with difficult-to-control VT caused by nonischemic cardiomyopathy. The development and expansion of hybrid laboratories, where mapping tools from the electrophysiology laboratory may be incorporated into hybrid procedures, may further facilitate surgical ablation approaches. The capability for this and other approaches to achieve greater tissue destruction warrants careful patient selection, consideration of risks, and ongoing study.

Disclosures
Dr Stevenson is co-holder of a patent for cardiac needle ablation that has been consigned to Brigham and Women’s Hospital.

References


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