Implantable cardioverter defibrillators (ICDs) can effectively terminate ventricular tachycardia (VT) when it occurs, but they do not prevent initiation of VT. There are several reasons why prevention of episodes is desirable. When VT is promptly and painlessly terminated by antitachycardia pacing, it can be asymptomatic. However, patients describe defibrillator shocks as terrible events that engender fear of future shocks and reduced quality of life. VT episodes are associated with increased risk of mortality and heart failure hospitalizations, although it is not clear whether the arrhythmia is merely a marker for worsening heart disease, or whether episodes of VT and possibly ICD shocks actually have an adverse effect themselves. Antiarrhythmic drugs, notably sotalol and amiodarone, reduce VT episodes, but inefficacy and adverse effects lead to discontinuation in ≈20% of patients, and long-term toxicities are a particular concern with the most effective drug, amiodarone. Catheter ablation offers the potential to prevent VT without the adverse effects of antiarrhythmic drugs, but technical challenges limit efficacy, and ablation has procedural risks.

Sustained monomorphic VT can often result in recurrent ICD shocks in patients with structural heart disease. The most common cause is scar-related reentry through myocyte bundles bordered by fibrosis from previous infarction, surgical scars, or replacement fibrosis in nonischemic cardiomyopathies. Interruption of these circuits requires ablation across the reentry path. But the circuits can be large in relation to ablation lesions, and may extend deep into the midmyocardium or epicardium. In elegant studies in explanted human hearts, de Bakker and colleagues demonstrated reentry circuits containing a circuitous narrow path through an infarct scar. A narrow isthmus or channel that is a critical segment of the reentry circuit is a desirable ablation target, where a focal lesion can interrupt reentry. These channels can be difficult to identify. During VT, they have low-amplitude electrograms that can be distinguished from surrounding bystanders in the scar, because pacing at sites in the isthmus entrains the VT in a characteristic manner (entrainment mapping). Termination of VT is observed when radiofrequency ablation is applied to the critical site. This approach is effective for targeting a specific VT, particularly one that is sufficiently hemodynamically tolerated to allow mapping during the VT. However, most patients with scar-related VT have other VT circuits, with a different QRS morphology on the surface ECG, and frequently a different tachycardia rate. It is often unclear which VTs have occurred spontaneously, designated clinical VTs, but even those VTs that have not occurred before ablation likely represent some degree of ongoing risk, such that targeting these VTs for ablation is also common practice. Complicating the attempted identification of reentry circuit channels is the fact that extensive mapping cannot generally be performed during VT because of its hemodynamic consequences, and attempts to maintain the patient in VT for an extended period raises concern about inducing hemodynamic instability or heart failure. Hemodynamic assist devices have the potential to extend the mapping time during VT, but also have risks, and some interfere with mapping in proximity to the device.

Attempting to identify and ablate the VT substrate during stable sinus rhythm is an alternative strategy. This substrate mapping approach often includes identifying the area of scar as an area of low-amplitude electrograms. A number of additional features that can indicate a potential channel have been described: isolated late potentials inscribed after the QRS, regions of relatively greater voltage flanked on either side by lower voltage, sites where pacing produces a QRS similar to that of VT and with a delay from pacing stimulus to QRS onset that is due to slow conduction away from the pacing site, and sites where pacing captures between 2 electrically unexcitable regions.

In this issue of Circulation, Jaïs and colleagues describe a substrate ablation approach that targets sites they define as having local abnormal ventricular activity (LAVA) during sinus rhythm. LAVA characteristics are those expected for a channel through a scar. There is a sharp, generally low-amplitude electrogram that, during VT or pacing in comparison with sinus rhythm, is shown to have a different timing relationship to other potentials recorded at the site, consistent with impaired coupling to the surrounding myocardium. LAVAs were late potentials or diastolic isolated potentials in some cases, but were also inscribed coincident with the QRS at sites that would not necessarily have been targeted by using electrogram criteria described previously. To facilitate identification of these potentials, mapping often included use of a 5-spline electrode array catheter (in 35 of the 70 endocardial cases and all 21 cases in which mapping was performed in the epicardium). Voltage maps to identify the area of scar, a
staple of most substrate mapping, were not routinely used, but LAVA sites had electrogram amplitudes well below the 1.5-mV lower limit of normal, and only 2 LAVA sites had greater amplitude signals. It seems likely that LAVA sites were in the scar region.

All LAVA potentials were targeted for ablation, and the resulting findings bring into focus challenges for substrate mapping and ablation of scar-related VTs.

In some patients, the substrate could not be identified. LAVA sites were not seen in 3 patients. Intramural and epicardial (when epicardial access is not possible) substrate locations, inadequate sampling during mapping because of difficult anatomy, such as interfering papillary muscles, and VT circuits without LAVA are possible causes, as well.

The anatomy is complex and 3-dimensional. In some cases, a LAVA electrogram was eliminated by ablation at a remote site (an example is shown of a LAVA in the epicardium eliminated during endocardial ablation).

Even when the substrate is identified, it can be difficult to eliminate. All LAVAs were eliminated in only 70% of cases. This suggests that, in many cases, the recording site was sufficiently close to detect the potential, but radiofrequency catheter ablation could not completely damage the reentry path, perhaps because of overlying fibrosis or fat (in the epicardium).

When ablation is acutely successful, the potential for recovery of incompletely therapeutically damaged tissue, as is commonly recognized in atrial fibrillation ablation, is a potential problem. Of 6 patients in whom a repeat procedure was performed for VT recurrence after LAVA elimination, sites with LAVA had recovered in 5 patients and were eliminated by repeat ablation.

It is difficult to abolish all reentry circuits, but this is not always necessary and absence of inducible VT at the end of the procedure is not a guarantee of success. At the end of the ablation procedure, at least 1 morphology of sustained monomorphic VT remained inducible in 30% of patients regardless of whether all LAVAs were eliminated. Because these patients have multiple inducible VTs, and an ICD that promptly elicits ICD therapies.7 Awareness of this approach is clearly lagging behind in many regions as centers continue to see patients who are referred late, after they have posttraumatic stress disorder from multiple defibrillator shocks during trials of ineffective medical therapy. Further advances are clearly required to improve success; these will likely involve additional mapping tools and more effective ablation lesions, the same fundamental issues that confronted Drs Hartzler and Josephson 3 decades ago.

Disclosures
Dr Stevenson is coholder of a patent for needle ablation that has been assigned to Brigham and Women’s Hospital. Dr Tedrow has received consulting moneys from St. Jude Medical and Boston Scientific, and nonsalary research dollars from Biosense Webster and St Jude Medical, as well.

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