

Mapping of a postinfarction left ventricular aneurysm–dependent macroreentrant ventricular tachycardia



Elad Anter, MD,^{*†} Jianqing Li, MD,^{*†} Cory M. Tschabrunn, CEPS,^{*†} Reza Nezafat, PhD,[†] Mark E. Josephson, MD^{*†}

From the ^{*}Harvard-Thorndike Electrophysiology Institute, and [†]Cardiovascular Division, Department of Medicine, Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, Massachusetts.

Introduction

Activation mapping of ventricular tachycardia (VT) is rarely accomplished owing to limited temporal and spatial resolution, unacceptably long mapping time, and hemodynamic instability.¹ Entrainment mapping is a reasonable approach to identify targets for ablation in patients with tolerated postinfarction reentrant VTs; however, it often does not allow delineation of the entire VT circuit.^{2,3} Introduction of newer mapping technologies capable of rapid and high-resolution electroanatomic mapping may allow detailed activation mapping of macroreentrant VTs, enhancing our understanding of macroreentrant circuit geometry and electrophysiology to facilitate ablation.⁴

Case report

We present the case of a 77-year-old man transferred to our institution for the management of recurrent monomorphic VT. The patient has a history of hypertension, hypercholesterolemia, diabetes, and multivessel coronary artery disease with prior inferior myocardial infarction. The baseline sinus 12-lead electrocardiogram (ECG) is shown in [Figure 1A](#). Six weeks earlier, he underwent coronary artery bypass graft surgery. At the time of surgery, a thin-walled large aneurysm at the base of the inferior wall was identified and a pericardial patch was placed over it. The aneurysm contained thin fibrous, noncontractile material that was associated with dyskinetic wall motion abnormality, consistent with a contained ruptured wall from his old transmural myocardial

infarction. A cardiac magnetic resonance of the left ventricle (LV) including the basal inferior aneurysm is depicted in [Supplemental Figure 1](#) (available online).

The patient recovered well from surgery with only mild congestive heart failure symptoms (New York Heart Association class I–II) and a left ventricular ejection fraction of 35%–40%, as was shown on transthoracic echocardiography 1 month after surgery. However, he developed frequent and recurrent episodes of sustained monomorphic VT requiring external shocks due to hemodynamic instability. He failed therapy with antiarrhythmic drugs including amiodarone, quinidine, and mexiletine. The 12-lead ECG of the clinical VT is shown in [Figure 1B](#). The VT cycle length was 360 milliseconds and it had a left bundle branch block pattern with left superior axis, suggestive of a basal inferior wall exit.

In an attempt to obtain detailed mapping of the VT circuit with as short as possible mapping duration, we elected to use the Rhythmia mapping system with its proprietary Orion mini-basket catheter (Boston Scientific, Cambridge, MA).⁴ The mini-basket consists of 8 splines, each containing 8 very small electrodes of 0.4 mm² that are separated by 2.5 mm from center to center, and with an overall extended basket diameter of 18 mm.⁵ Activation mapping is automated and is determined based on the combination of the bipolar and unipolar electrograms and timed at the maximal (–) dV/dt of the local unipolar electrogram.

A pentapolar catheter was placed in the right ventricular apex (RVA) with its proximal electrode in the inferior vena cava serving as an indifferent unipolar electrode. An intracardiac ultrasound catheter was placed at the base of the right ventricle in order to visualize the LV and confirm tissue contact of the mini-basket catheter. Heparin was administered to maintain an activated clotting time of 300–350 seconds for the duration of the procedure. The 8F mini-basket bidirectional catheter was introduced into the LV using a retrograde transaortic approach. The mini-basket catheter was placed in the aneurysm and the clinical VT was induced with single extrastimuli from the RVA. Pacing from the RVA during VT showed ECG fusion, consistent with a reentrant mechanism. The entire reentrant circuit was

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ABBREVIATIONS ECG = electrocardiogram; LV = left ventricle; RVA = right ventricular apex; VT = ventricular tachycardia
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Elad Anter receives research grants from Biosense Webster and Boston Scientific. Mark Josephson receives research grants and speaking honoraria from Medtronic. **Address reprint requests and correspondence:** Elad Anter, Harvard-Thorndike Electrophysiology Institute, Beth Israel Deaconess Medical Center, 185 Pilgrim Rd, Baker 4, Boston, MA 02215. E-mail address: eanter@bidmc.harvard.edu.

KEY TEACHING POINTS

- High-resolution mapping technologies can improve visualization of reentrant ventricular tachycardia (VT) circuits. They allow detailed mapping of the entire reentrant circuit, including the “common channel.”
- These technologies often use multielectrode catheters with automated annotation to allow rapid mapping of VTs. These may permit mapping of VTs that were previously unmappable because of hemodynamic instability or circuit complexity.

mapped and demonstrated a continuous loop around the base of the aneurysm (Figure 2A). Supplemental Video 1

(available online) shows the complete propagation map of the VT circuit. During a mapping time of 7 minutes and 12 seconds, 4264 activation points were acquired. The area of slowest conduction velocity was at the junction between the base of the aneurysm and the mitral annulus (Figure 2A, yellow star). This area served as the protected common pathway of the tachycardia that propagated clockwise around the edge of the aneurysm. A fractionated mid-diastolic signal was recorded at this site. Figure 2B shows an example of activation time at this area of slow conduction as determined by the automated mapping algorithm. Entrainment from this site showed a concealed QRS fusion with postpacing interval that was identical to the tachycardia cycle length and with a stimulus-to-QRS interval of 182 milliseconds (50% of the tachycardia cycle length) that was the same as the electrogram-to-QRS interval, consistent with a protected

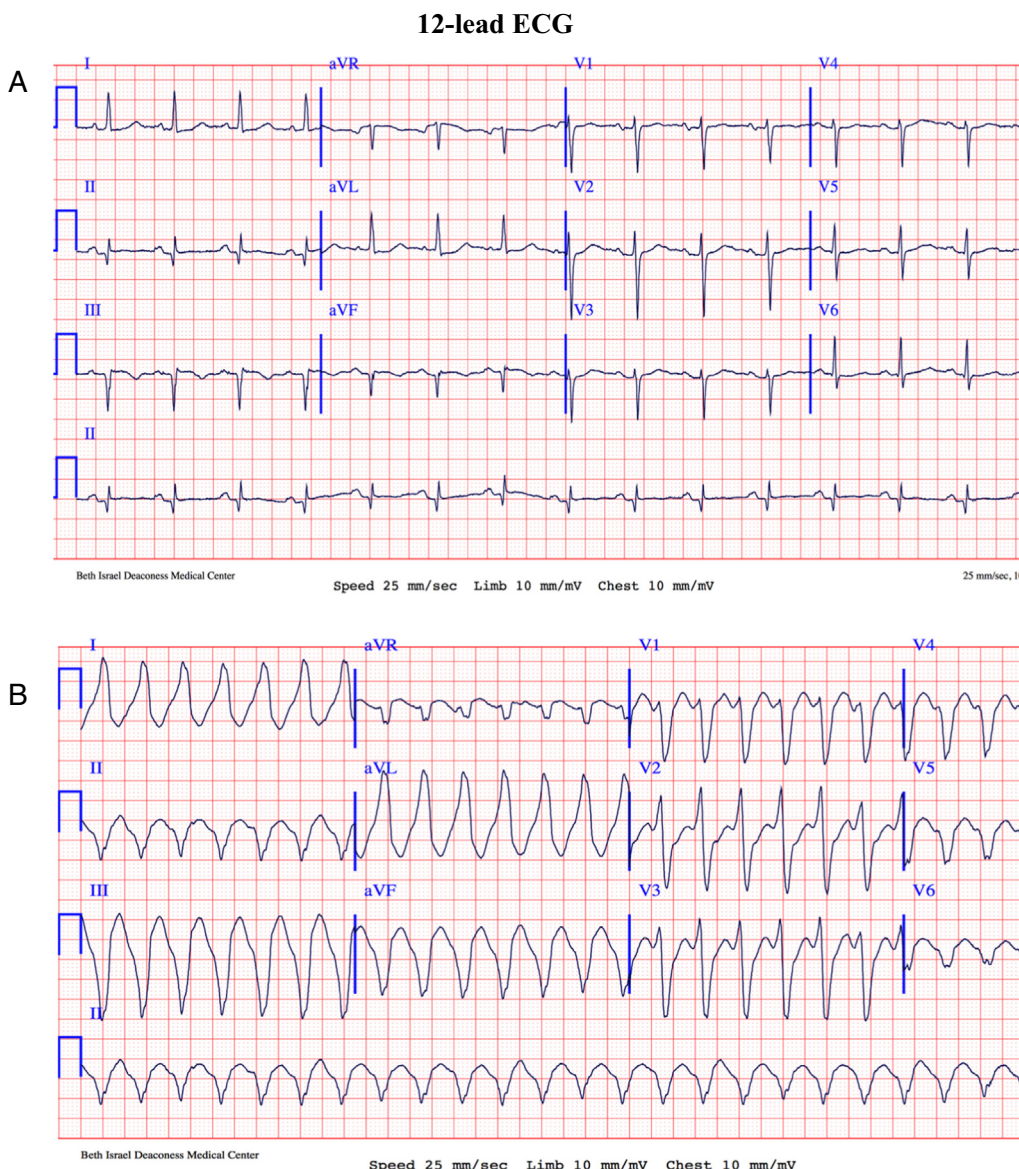


Figure 1 Twelve-lead electrocardiograms. **A:** A 12-lead electrocardiogram (ECG) during sinus rhythm. **B:** The 12-lead ECG of the ventricular tachycardia (VT). The VT cycle length was 360 milliseconds with a left bundle branch block pattern and a left superior axis, suggestive of a left ventricular basal septal site. Lead II shows atrioventricular dissociation.

Activation map of the VT around the base of an LV aneurysm

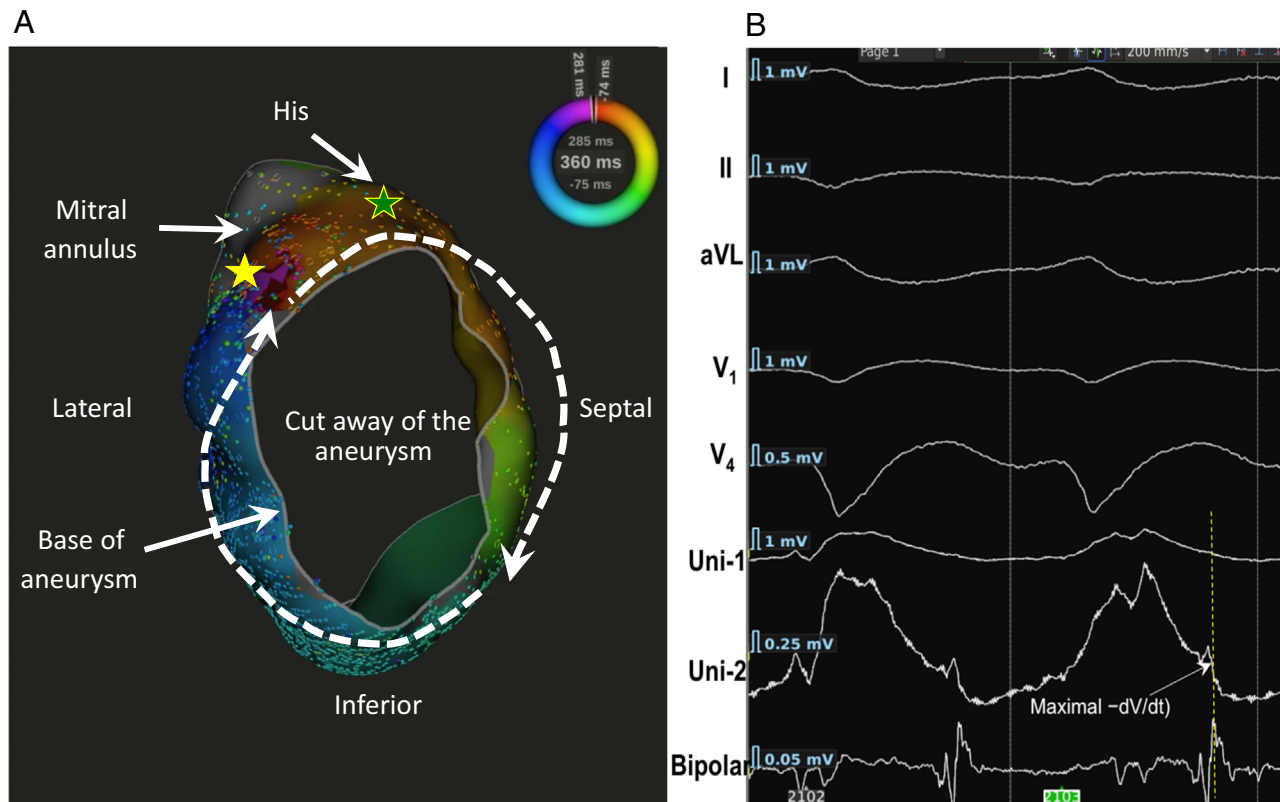


Figure 2 Activation map of the ventricular tachycardia (VT) circulating around the base of the aneurysm. **A:** Color-coded activation map of the reentrant VT circulating around the edge of the aneurysm. Red represents early activation and purple represents late activation. The range of colors between red and purple represent intermittent activation time as depicted in the circular color bar. The impulse propagated in a clockwise orientation (*dashed arrows*) exiting the zone of slowest activation (*yellow star*) at the septal aspect of the aneurysm, resulting in a left bundle branch block pattern with a left superior axis. **B:** Example of local activation time annotation by the automated software. The panel shows 4 electrocardiogram leads, 2 unipolar electrograms (Uni-1 and Uni-2), and a resultant bipolar signal (Bipolar). The bipolar signal is multicomponent and fractionated. Determination of activation time (*yellow dotted line*) was based on the maximal negative slope ($-dV/dt$) of the unipolar electrogram (Uni-2).

isthmus site (Figure 3A). In addition, pacing outside the basal rim of the aneurysm, including the anterior and lateral mitral annulus, resulted in QRS fusion and postpacing intervals that were significantly longer than the tachycardia cycle length, suggesting that the remainder of the LV was not part of the VT circuit. A single radiofrequency ablation application at the protected isthmus between the base of the aneurysm and the mitral annulus slowed and terminated the tachycardia with block after the mid-diastolic electrogram (Figure 3B). The local electrogram at the termination site demonstrated atrial and ventricular signals, consistent with a mitral annulus site. In addition, it showed a late potential. Following termination of the tachycardia, pacing just medial to the ablation lesion resulted in clockwise propagation around the edge of the aneurysm with QRS morphology similar to the VT, while pacing just lateral to the ablation lesion resulted in counterclockwise propagation around the edge of the aneurysm with QRS morphology opposite to the VT with a right inferior axis, consistent with block across the isthmus line. Following ablation at the isthmus site, the VT was not inducible.

Discussion

We present high-resolution activation mapping of the entire reentrant VT circuit in a patient with inferior infarction and aneurysm. We demonstrated that the macroreentrant circuit circulated around the edge of the aneurysm with the area of slowest conduction velocity at the edge of the aneurysm adjacent to the mitral annulus. This was confirmed to be a central isthmus site by entrainment mapping ablation at that site, resulting in slowing and termination of the VT with a single radiofrequency ablation application.

Macroreentrant circuits around the edge of an LV aneurysm have been described with a wavefront propagation that can be either clockwise or counterclockwise.⁶ However, detailed activation maps of such arrhythmias have been limited by inadequate spatiotemporal resolution. New mapping technologies using catheters with small multielectrodes in conjunction with automated annotation of local activation time allow rapid mapping of reentrant electrical circuits in unprecedented detail.

This mapping technology may be particularly useful for mapping reentrant VTs. It may permit rapid mapping of VTs that were previously considered unmappable because of

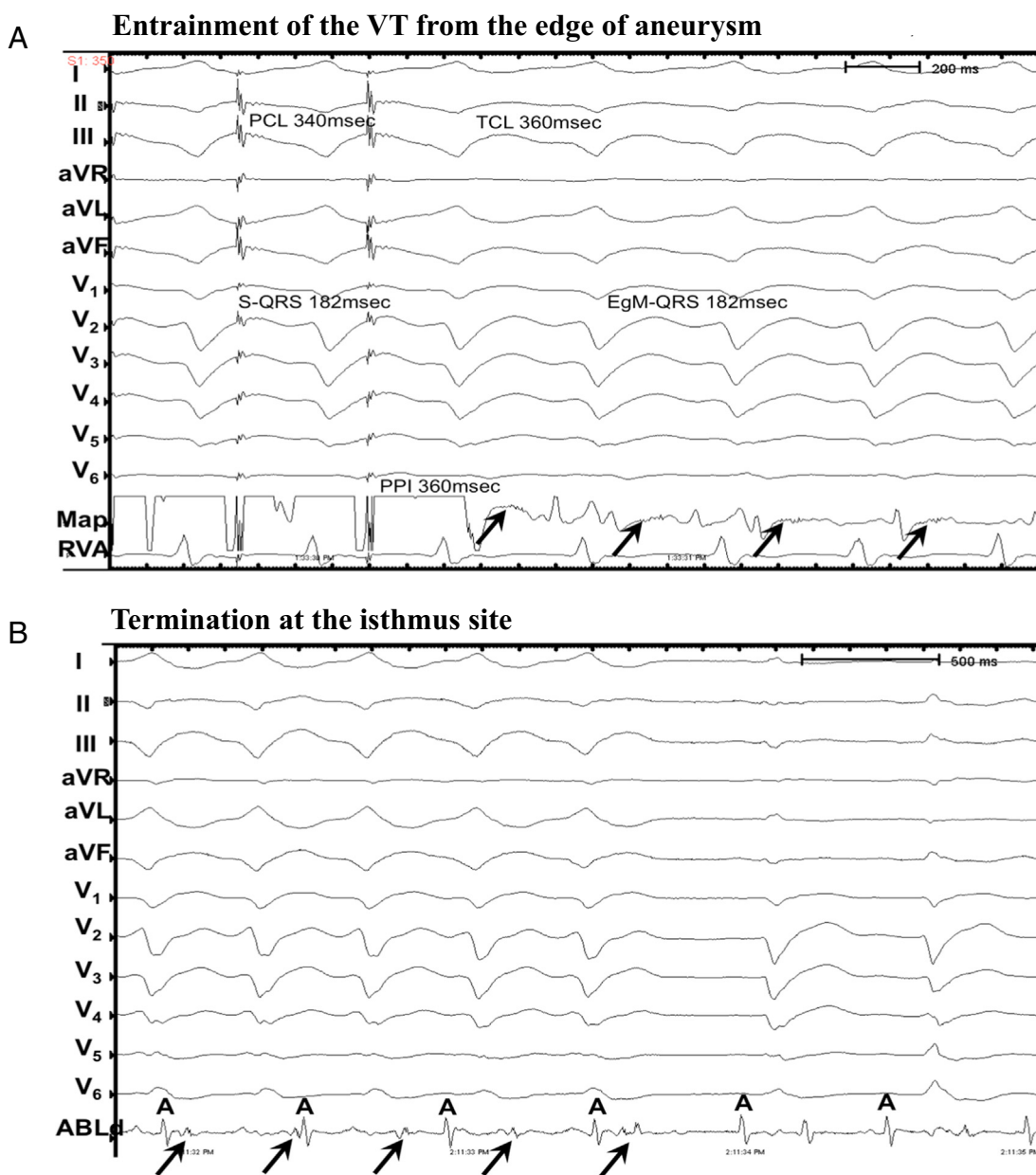


Figure 3 Entrainment and termination of the ventricular tachycardia (VT) at the protected isthmus. **A:** Entrainment from the area of slowest conduction velocity at the superior aspect of the aneurysm below the septal aspect of the mitral annulus (Figure 2 yellow star). Pacing at 20 milliseconds faster than the tachycardia entrained the VT with concealed QRS fusion and a postpacing interval that equaled the tachycardia cycle length. In addition the stimulus-to-QRS was similar to the electrogram-to-QRS interval and was 50% of the tachycardia cycle length, consistent with a protected isthmus site. **B:** Termination of the VT with a single radiofrequency application. The tachycardia progressively slowed from 360 milliseconds to 420 milliseconds and terminated with the nonpropagated electrogram (solid black arrow). The ablation site was at the base of the aneurysm below the septal aspect of the mitral annulus as it showed atrial (A) and ventricular electrograms.

hemodynamic instability or circuit complexity. It may also improve our understanding of circuit geometry and physiology that can better guide targeted ablation strategy.

Conclusions

High-resolution mapping technologies can improve visualization of reentrant electrical circuits. This case illustrates the clinical utility of this technology to improve mapping accuracy to facilitate ablation therapy.

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Appendix

Supplementary data

Supplementary data associated with this article can be found in the online version at <http://dx.doi.org/10.1016/j.hrcr.2015.07.011>.

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