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Eduardo Sosa and Mauricio Scanavacca

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Percutaneous Pericardial Access for Mapping and Ablation of Epicardial Ventricular Tachycardias

Eduardo Sosa, MD, PhD; Mauricio Scanavacca, MD, PhD

A 26-year-old male athlete was admitted in 1997 for exercise-induced ventricular tachycardia (VT). Physical examination was unremarkable. Magnetic resonance imaging and angiogram (Figure 1) showed a mild left-ventricle dilation, with left-ventricle ejection fraction of 58% and normal coronary arteries. In an electrophysiological study, programmed stimulation induced monomorphic VT with right bundle-branch block morphology (Figure 2). Endocardial mapping of both ventricles failed to localize an adequate site for ablation. Amiodarone was initiated, and the patient became asymptomatic until April 2000 when he developed recurrent VT. Baseline ECG showed an “epsilon” wave in lead V1, and late potentials were demonstrated in the signal average ECG (Figure 2), consistent with conduction delay, which has been associated with VT in arrhythmogenic right-ventricle dysplasia.

In a repeat electrophysiological study, endocardial mapping did not identify any low-voltage areas to suggest scarring. A subxiphoid approach was taken to achieve pericardial access. Epicardial mapping during sinus rhythm identified an area of isolated delayed potentials 110 ms after the end of the epsilon wave. During induced monomorphic VT, this isolated delayed potential became middiastolic and presystolic (180 ms before QRS complexes). One pulse of radiofrequency current at this site promptly interrupted VT after 4.5 s (Figure 3) and was continued for 60 s. Three additional radiofrequency applications were placed closely adjacent to this site. VT could no longer be provoked, and the

patient has remained asymptomatic without antiarrhythmic drugs. The findings are consistent with a small, epicardial scar causing VT, possibly related to a mild idiopathic cardiomyopathy.

Accessing the epicardial space is a useful tool for dealing with epicardial arrhythmias, especially VT, as in the presented case. Although the subxiphoid approach previously has been described extensively,¹ many electrophysiologists are still not comfortable performing this procedure with only the guidance of static images. Moving additions to the images may allow better understanding of the technique and confer more confidence in performing it (see Movies I through III in the online-only Data Supplement).

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Disclosures

None.

Reference

1. Sosa E, Scanavacca M. Epicardial mapping and ablation techniques to control ventricular tachycardia. *J Cardiovasc Electrophysiol*. 2005;16(4):449–452.

From the Heart Institute, INCOR, School of Medicine, São Paulo University, São Paulo, Brazil.

The online-only Data Supplement, consisting of movies, is available with this article at <http://circ.ahajournals.org/cgi/content/full/115/21/e542/DC1>.

Correspondence to Eduardo Sosa, MD, Av Dr E.C. Aguiar 44-São Paulo, Brazil, CEP 05403–000. E-mail sosa@incor.usp.br (*Circulation*. 2007;115:e542-e544.)

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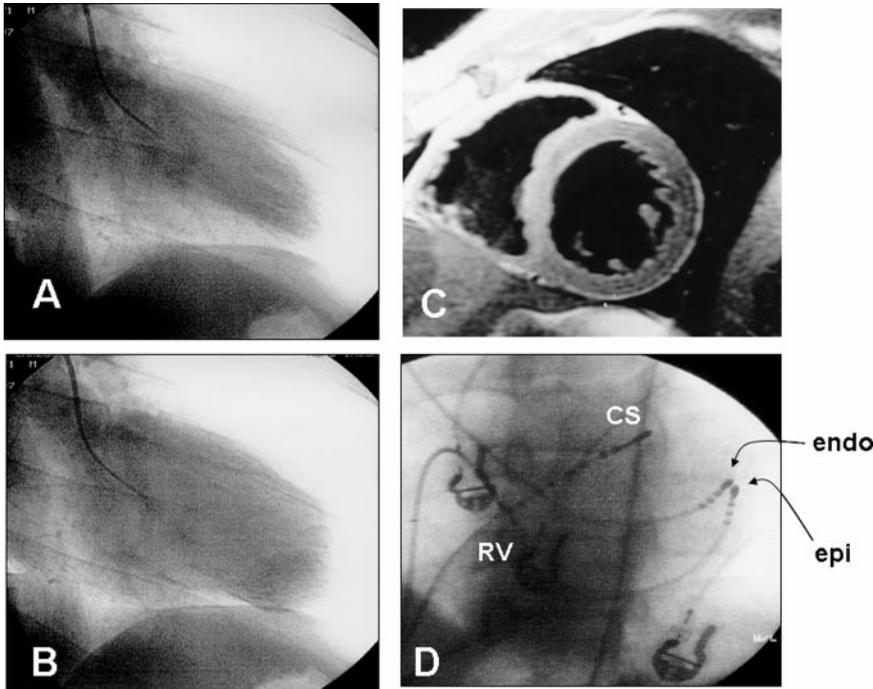


Figure 1. A and B, Left ventricle angiogram in right anterior oblique XR projection during systole and diastole. Note moderate diffuse hypocontractility. C, Magnetic resonance image and D, left anterior oblique XR projection, showing the catheter position. CS indicates sinus coronary; RV, right ventricle; endo, endocardial; epi, epicardial placed where ventricular tachycardia was interrupted with radiofrequency pulse.

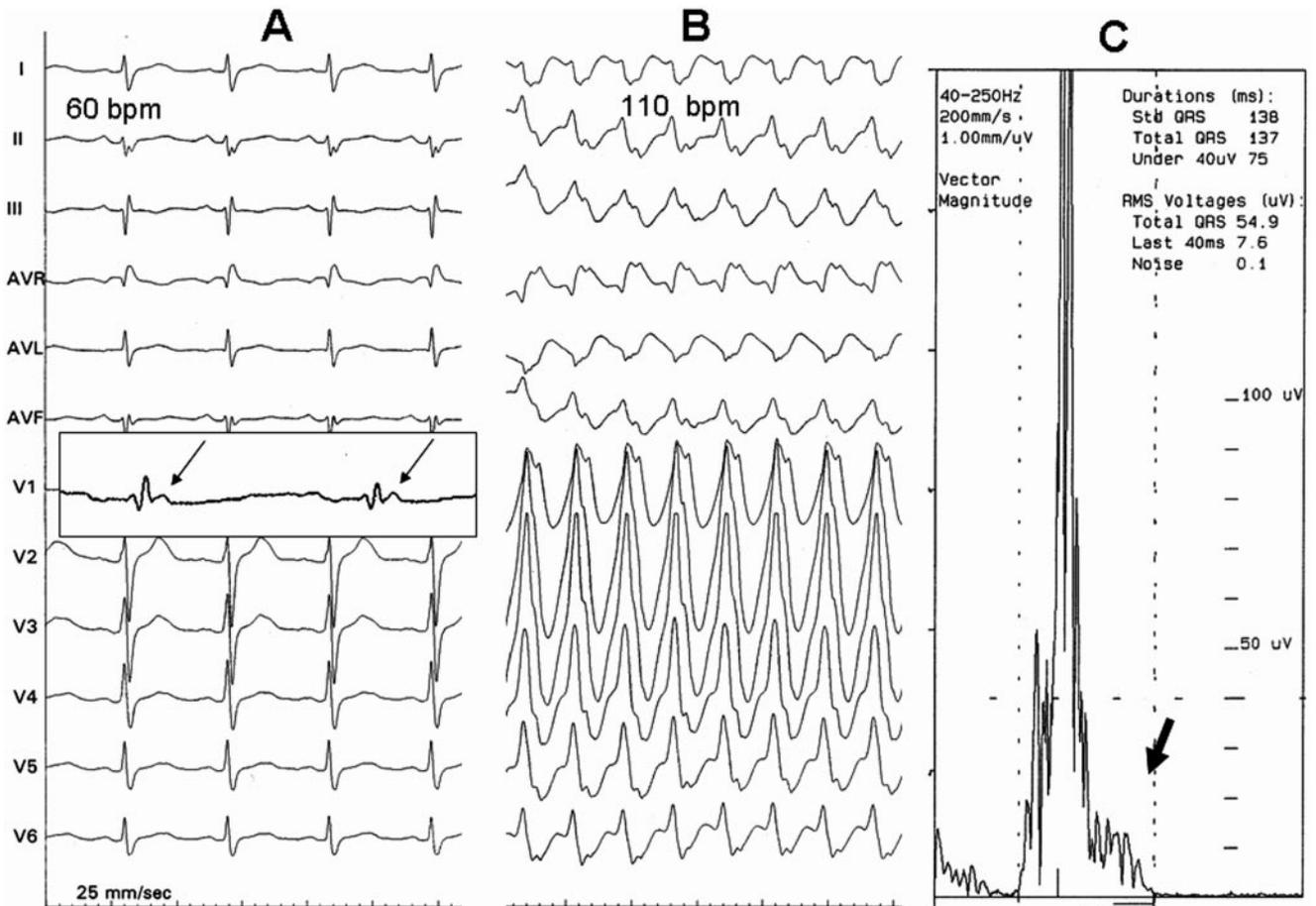


Figure 2. A, Twelve-lead ECG; the epsilon wave in lead V1 is indicated by arrows. B, Twelve-lead ECG during ventricular tachycardia. Note the right bundle-branch morphology of the QRS complex. C, Signal-averaged ECG showing late potentials, consistent with delayed activation of a portion of the ventricle, which can be associated with reentrant ventricular tachycardia (arrows).

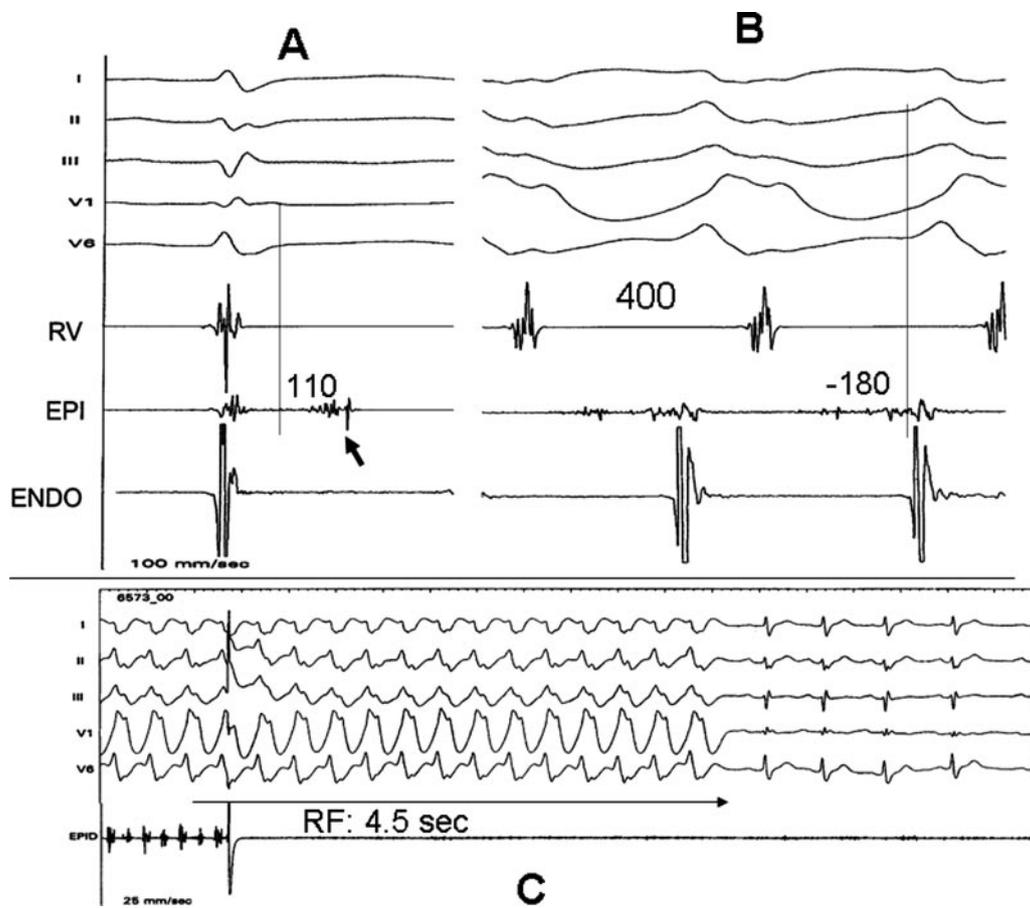


Figure 3. Findings during epicardial mapping. A, Surface ECG leads and endocardial recordings from the right ventricle (RV), from the epicardium of the lateral wall of the left ventricle (EPI), and from the endocardial of the left ventricle (ENDO) obtained during sinus rhythms. Note the normal left-ventricle endocardial electrogram, but in the epicardium an isolated delayed potential 110 ms after the end of “epsilon” wave (inset). B, Tracings from the same sites, shown during VT. The isolated potential in the epicardium occurs 180 ms before QRS complex. C, Ablation at the epicardial site terminates ventricular tachycardia in 4.5 ms, consistent with participation of the site in the ventricular tachycardia.