Precise Noninvasive ECG Mapping Derived Localization of the Origin of an Epicardial Ventricular Tachycardia

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Invasive electrophysiological study with subsequent radiofrequency ablation is the most common therapeutic approach for the management of idiopathic ventricular tachycardias (VTs). Different algorithms based on the standard 12-lead ECG have been described for localizing the origin of ventricular arrhythmias. The standard surface ECG has limitations of spatial resolution, whereas an electrophysiological study, requiring invasive access, does not provide global activation patterns and cannot easily track changing arrhythmia patterns. Invasive mapping remains, however, the gold standard of spatial localization.

See Editor's Perspective by Asirvatham and Stevenson

A novel approach for diagnosing the source of ventricular arrhythmias was demonstrated in recent years. This method, noninvasive electrocardiographic imaging, is based on the numeric reconstruction of epicardial electrograms (based on epicardial potentials) derived from computed tomography (CT) or magnetic resonance imaging–based thoracic imaging and multichannel body surface EGG recording. This case study demonstrates the feasibility of a novel noninvasive epicardial and endocardial electrophysiology system (NEEES) for the diagnosis of an epicardially originating idiopathic VT.

We present here the case of a 61-year-old female patient who was referred to our center for the investigation of symptomatic sustained VT. The 12-lead surface ECG showed a monomorphic VT with a QRS duration of 120 ms, a left superior axis, a right bundle branch morphology, and an early septal transition in the precordial leads suggesting a posteroseptal origin (Figure 1). No detectable anomalies were found on transthoracic echocardiographic examination.

Noninvasive Electrocardiographic Imaging Findings

She then underwent a noninvasive electrocardiographic imaging study (NEEES system Amycard 01C electrophysiology [EP] laboratory; EP Solutions SA, Yverdon-les-Bains, Switzerland) followed by an invasive electrophysiological and ablation procedure. Two-hundred twenty-four body surface mapping electrodes were applied on the patient's torso and connected to a multichannel ECG amplifier (EP Solutions SA). ECG recording was performed during 30 minutes, and 10 to 15 s ECG segments of monomorphic VT were selected and exported into the Amycard 01C EP laboratory software. The patient then underwent an ECG-gated CT of the thorax performed on a second-generation dual-source CT system (Somatom Definition Flash; Siemens Healthcare, Forcheim, Germany). Scanning of the torso and heart was performed simultaneously with prospective ECG gating and an injection of 100 mL intravenous contrast medium (Iohexol, 350 mg of iodine/mL; GE Healthcare) during 15 s breath hold. The obtained data were imported into Amycard 01C EP laboratory software in DICOM format and 3-dimensional torso and heart geometry were reconstructed from the CT acquisition. Accurate epicardial and endocardial heart and ventricular models were obtained by the segmentation and polygonal mesh reconstruction. Epicardial and endocardial, unipolar and bipolar electrograms, and isopotential and isochronal maps based on unipolar (first negative deflection of unipolar electrograms) and bipolar (activation directional mapping of bipolar electrograms) electrograms were reconstructed by NEEES inverse problem solution software by an analyst blinded to the invasive procedure findings. Multiple noninvasive electroanatomical maps (isopotential, isochronal based, and propagation maps) were calculated and analyzed. On the CT scan, a diverticulum was found at the ostium of the coronary sinus (CS; measuring 16×22×21 mm) and was included in the reconstructed heart volume produced by the segmentation and was also meshed and included into the subsequent calculations. Noninvasive epicardial isochronal and isopotential maps of the complete heart showed a single zone of earliest activation under the CS diverticulum in the posterobasal segment of left ventricle (Figure 2A). Endocardial maps showed the zone of the earliest activation inside the CS diverticulum (69 ms preceding the QRS peak in lead I), next at the epicardial surface of the CS (60 ms preceding the QRS in lead I), then the endocardial right ventricle (RV; 50 ms before QRS peak in lead I), and followed by the endocardial posterobasal segment of left ventricle at 38 ms before QRS peak in lead I (Figure 2B and 2C).

Findings of Endocardial Mapping and Ablation Procedure

Invasive activation mapping was performed under mild sedation and local anesthesia during the EP study. Point-by-point

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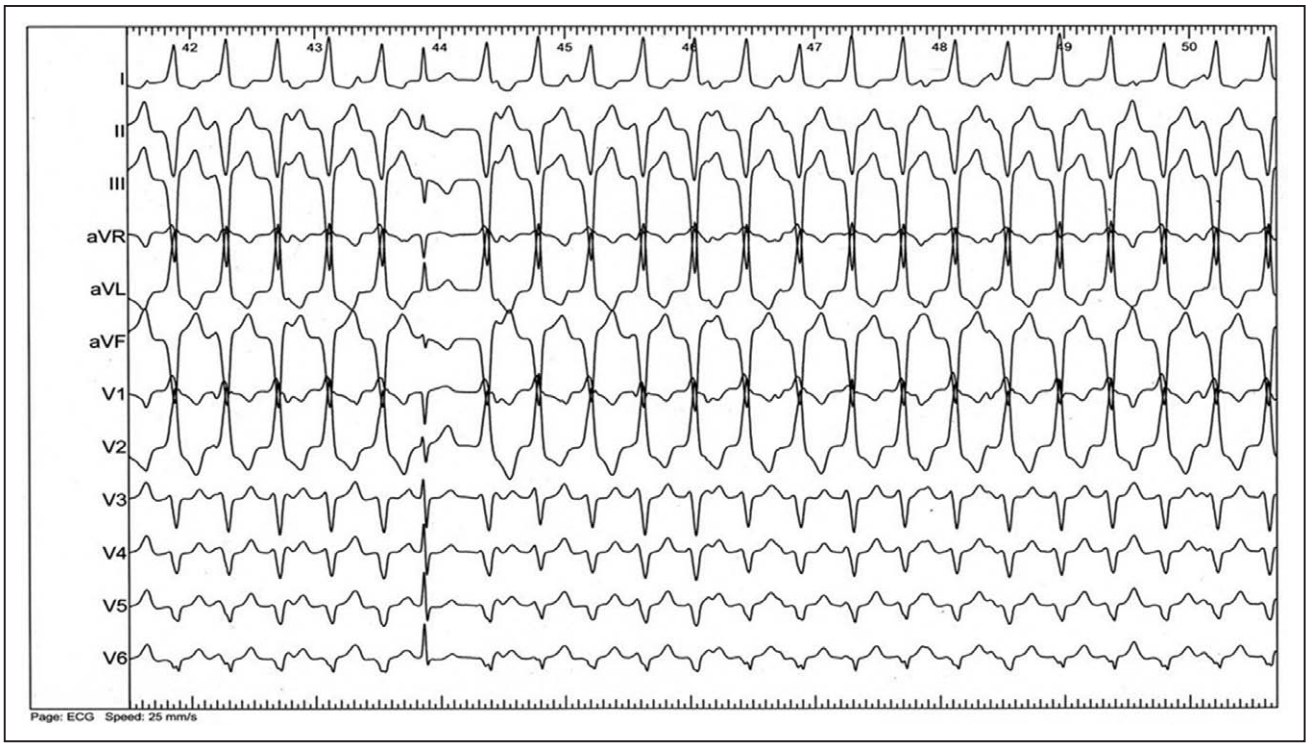

Figure 1. Twelve-lead surface ECG of the clinical ventricular tachycardia.

activation mapping was performed in both ventricles during well-tolerated ongoing VT and the earliest endocardial activation (100 ms before the peak of the QRS complex on the surface ECG [lead I]) and a deep QS complex in the unipolar electrogram were recorded in the right posteroseptal region close to the ostium of the CS with later activation in the right ventricle (72 ms) and left ventricle (86 ms). The VT terminated mechanically at this site as indicated by the sudden restoration of sinus rhythm without change in activation (Figure 3). A high baseline impedance was noted (279 Ω), and, therefore, low power radiofrequency energy was delivered at this site (20 W titrating down to 10 W after the first 5 s with an impedance drop to 208 Ω and 105 s with a mean impedance of 223 Ω). Two additional radiofrequency applications were delivered (12 W and 33 W) in the immediate vicinity with progressive withdrawal of the ablation catheter, and an elevated impedance was observed during the first of these 2 applications. A contrast injection close to the ablation catheter revealed the ablation site to be situated within the diverticulum of the proximal CS (Figure 4). These observations correspond well with the endocardial/epicardial activation maps generated from the noninvasive electrocardiographic imaging mapping data.

Discussion

Value of Standard 12-Lead ECG and Conventional Mapping

Various ECG criteria and algorithms have been published in the past decades to predict the localization of VT and ectopies originating from the right ventricular/left ventricular outflow tract, the aortomitral continuity, coronary cusps, and epicardium.[1,2] However, not all of these algorithms could prove their accuracy and reproducibility when applied independently. Furthermore, loss of reliability with increasing numbers of algorithmic steps to reach localization diagnosis is a well-known epiphenomenon, as has been demonstrated in a recently published comparison.[3] Anatomic variations of the thoracic wall, the cardiac chambers, and thus inconsistencies in their spatial interrelationship are likely to hamper diagnostic accuracy. However, distinct ECG patterns and morphological refinements still may facilitate an invasive strategy, which remains required for proper diagnosis, mapping, and eventually guidance of catheter ablation therapy and its anatomic approach. This has significant importance in particular for idiopathic epicardial VTs originating from the vicinity of the CS system (ca. 9% of all idiopathic VTs) because the vast majority of them can be identified and completely ablated in the CS system avoiding a transthoracic and more invasive approach.[4] Different ECG characteristics have been analyzed to predict and differentiate various epicardial origins of VTs and particularly those arising from the CS system.[5] VT originating from the proximal CS typically shows a left superior axis (with a QS in II<III), a transition zone in V1 (V1 and V2), a tall R in I and aVL and a QS in V2 through V6 [4,6]; distinct patterns, which correlate well with the ECG morphology we observed. Moreover, similar ECG patterns have been shown to occur in patients with accessory atrioventricular pathways mapped epicardially in CS diverticula.[7] On the basis of the body surface mapping and tomography, the electrocardiographic imaging is a noninvasive technique providing global electroanatomic maps by projecting body surface electrode signals onto the epicardial geometry of the heart derived from imaging modalities with a high spatial resolution such as cardiac CT or magnetic resonance imaging.[8]

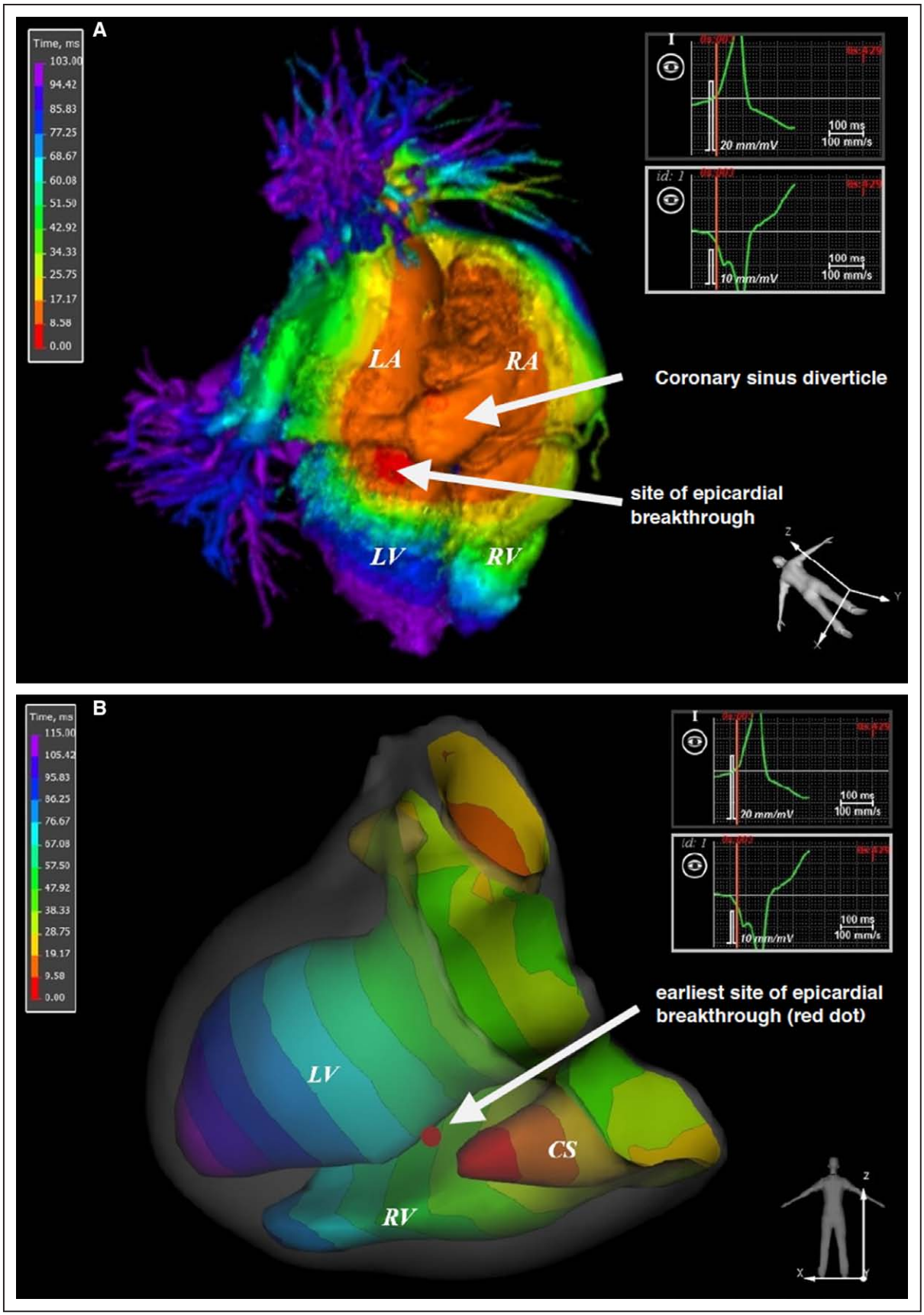

Figure 2. A, Isochronal epicardial map superposed on computed tomography 3-dimensional whole heart anatomy. **B**, Isochronal endocardial map depicted on the epi/endocardial ventricular model in the translucent epicardium mode. Coronary sinus (CS) diverticulum, ventricular tachycardia focus (red zone), and site of epicardial breakthrough posterobasal left ventricle (LV; red dot). (*Continued*)

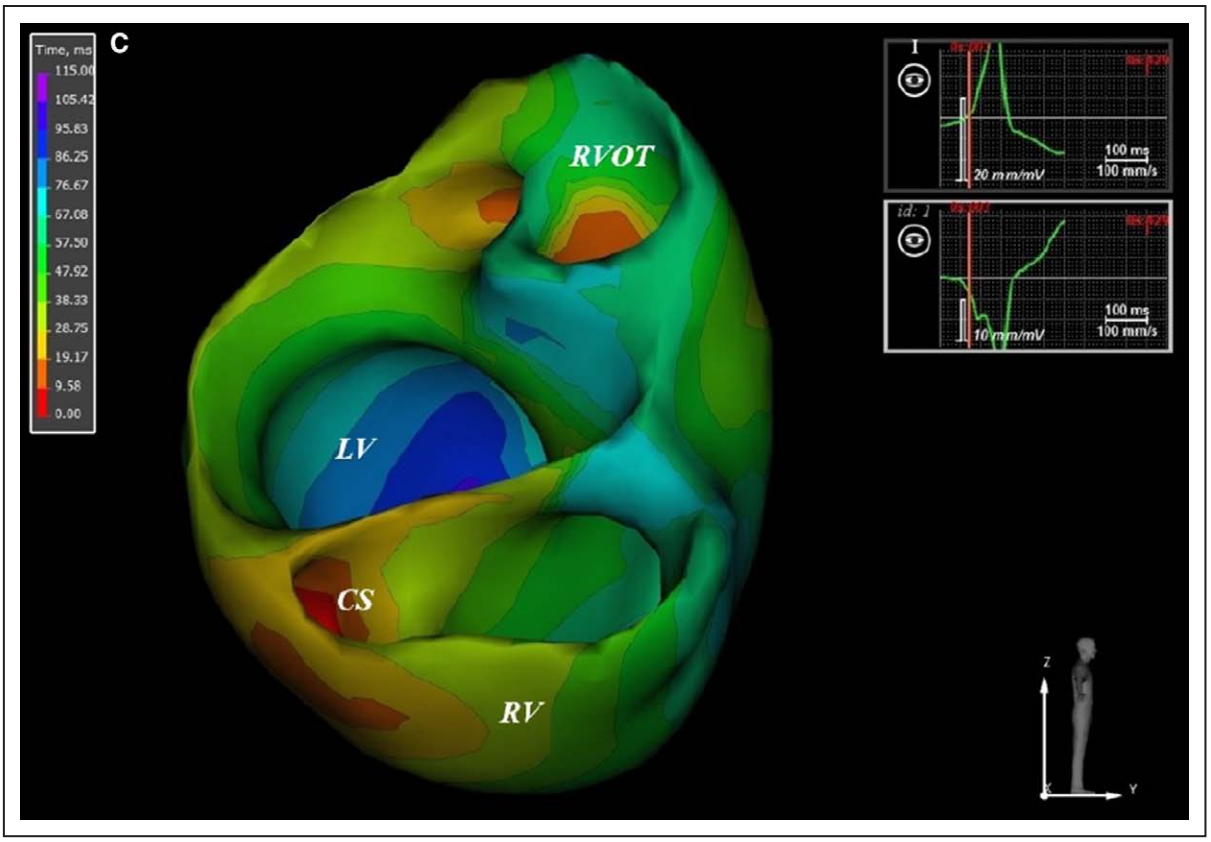

Figure 2 Continued. C, Isochronal endo/epicardial map on the epi/endocardial ventricular model. LA indicates left atrium; RA, right atrium; RV, right ventricle; and RVOT, right ventricular outflow tract.

This technology has the potential to localize and visualize the onset and electric propagation of various arrhythmias not only recorded in the preprocedurally but also in a real-time fashion onto the whole cardiac geometry to guide ablation, as recently reported.[9,10]

Accurate Mapping Using Noninvasive Electrocardiographic Imaging

The NEEES used in this report (EP Solutions SA) provides both epicardial and endocardial activation mapping by the use of different proprietary acquisition and processing algorithms when compared with existing electrocardiographic imaging systems. The main advantages of NEEES are its noninvasive nature, simultaneous mapping of all 4 chambers of the heart in only 1 cardiac cycle and of epicardial and endocardial surfaces.[11] Such information may be important to guide the identification and ablation of challenging arrhythmias and their sources and potentially of more complex irregular arrhythmias such as atrial and ventricular fibrillation. The higher source voltage of ventricular activation certainly facilitates the noninvasive localization of the origin of ventricular arrhythmias such as this VT. The imaging modalities identified the CS diverticulum to be the anatomic correlate of the observed impedance rise during radiofrequency, which allowed the operators to adjust settings for ablation based on the intraoperative findings. The discrepancy between the relative timings of the right versus left ventricular endocardial breakthroughs as measured by NEEES versus invasive mapping may simply represent undersampling during the EP study because even detailed high-density point-by-point mapping in the ventricles is sequential, often incomplete or nonuniform. Of note, the relatively early and isochronous activation of the right ventricular outflow tract represents a so-called far-field artifact. Noninvasively reconstructed electrograms may create this artifact on the isochronal maps in some nonconductive areas, around vessels, or of complex geometry but do not call into question the accuracy of the earliest negative potential (source of activation). Furthermore, because of the tight real-time conditions during the procedure, we did not complete exclusion of this area from the isochronal map.

To the best of our knowledge, this is also the first report of a VT origin precisely localized within a CS diverticulum, which is better known for their relationship with accessory AV connections. One could speculate whether our patient actually could have presented a ventricular pre-excitation through the fascicule localized epicardially in the CS diverticulum. However, ventricular arrhythmias dissociation was obvious during the VT. Although ventricular ectopies and even idiopathic VT have been traced and ablated within the coronary venous system, the only other report of a VT associated with a CS diverticulum described neither its ECG morphology, localization of origin, nor the activation mapping.[12] Strands of myocardium within the diverticulum (which more frequently present as accessory pathways because of connections to the nearby atrium and ventricle) connected only to the ventricles

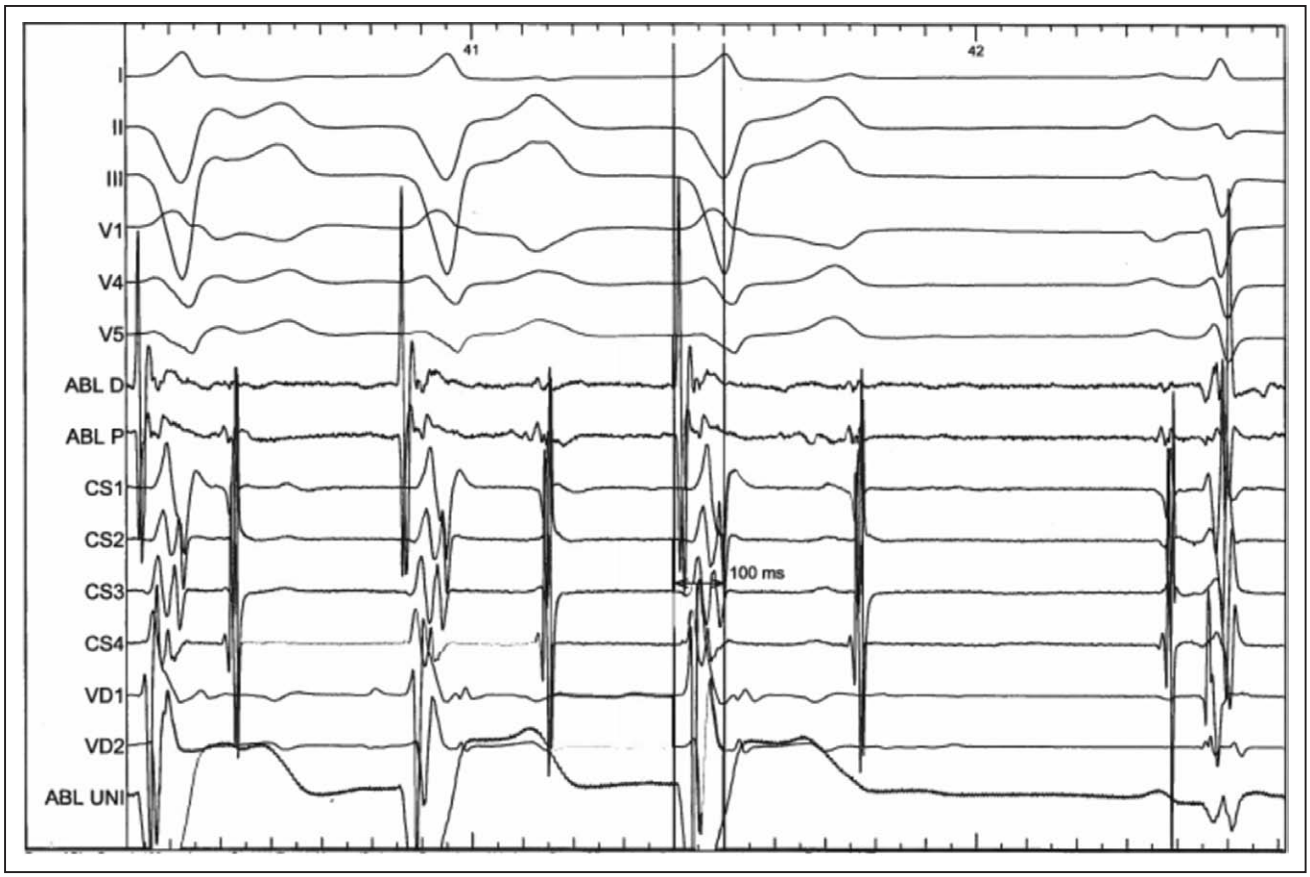

Figure 3. Site of mechanical termination as indicated by the sudden termination without change in activation. ABL D indicates distal electrode of the ablation catheter; ABL P, proximal electrode of the ablation catheter; ABL UNI, unipolar recording of the ablation catheter; CS, coronary sinus; and V_D, right ventricle.

probably provided the source of this VT, which was successfully ablated by focal ablation.

When anticipated by a meticulous ECG analysis, catheter ablation of idiopathic endocardial and epicardial VTs is usually relatively straightforward and reported to be associated with excellent success rates, with or without the use of currently available 3-dimensional electroanatomical mapping systems. Nevertheless, anatomically challenging cases with unusual ventricular endocardial localizations and epicardial breakthroughs and rarely occurring ectopies or poorly tolerated arrhythmias during the mapping procedure highlight the limitations of current strategies and may be overcome with the use of concomitant imaging and mapping technologies such as noninvasive electrocardiographic imaging systems, as demonstrated in our report.

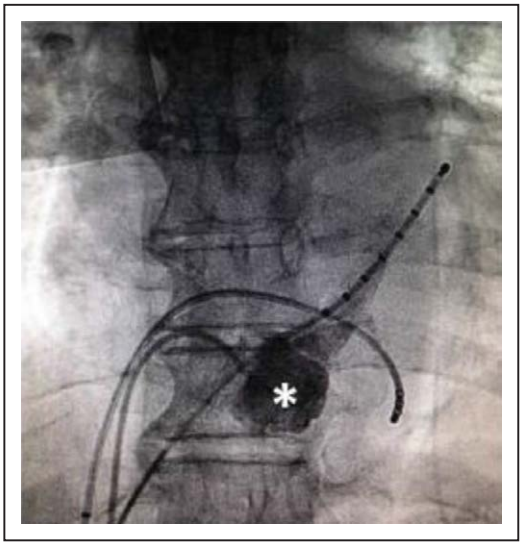

Figure 4. Coronary sinus aneurysm confirmed by the angiography (*).

Conclusions

This case report describes the unusual origin of a focal VT from a CS diverticulum. Furthermore, we show that noninvasive electrocardiographic imaging using NEEES is feasible and precise for endocardial and epicardial localization particularly of ventricular focal arrhythmia sources, permitting a safe successful ablation.

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Disclosures

None.

References

1. Betensky BP, Park RE, Marchlinski FE, Hutchinson MD, Garcia FC, Dixit S, Callans DJ, Cooper JM, Bala R, Lin D, Riley MP, Gerstenfeld

EP. The V(2) transition ratio: a new electrocardiographic criterion for distinguishing left from right ventricular outflow tract tachycardia origin. *J Am Coll Cardiol*. 2011;57:2255–2262. doi: 10.1016/j.jacc.2011.01.035.
2. Park KM, Kim YH, Marchlinski FE. Using the surface electrocardiogram to localize the origin of idiopathic ventricular tachycardia. *Pacing Clin Electrophysiol*. 2012;35:1516–1527. doi: 10.1111/j.1540-8159.2012.03488.x.
3. Jamil-Copley S, Bokan R, Kojodjojo P, Qureshi N, Koa-Wing M, Hayat S, Kyriacou A, Sandler B, Sohaib A, Wright I, Davies DW, Whinnett Z, S Peters N, Kanagaratnam P, Lim PB. Noninvasive electrocardiographic mapping to guide ablation of outflow tract ventricular arrhythmias. *Heart Rhythm*. 2014;11:587–594. doi: 10.1016/j.hrthm.2014.01.013.
4. Chen YH, Lin JF. Catheter ablation of idiopathic epicardial ventricular arrhythmias originating from the vicinity of the coronary sinus system. *J Cardiovasc Electrophysiol*. 2015;26:1160–1167. doi: 10.1111/jce.12756.
5. Chen YA, Nguyen ET, Dennie C, Wald RM, Crean AM, Yoo SJ, Jimenez-Juan L. Computed tomography and magnetic resonance imaging of the coronary sinus: anatomic variants and congenital anomalies. *Insights Imaging*. 2014;5:547–557. doi: 10.1007/s13244-014-0330-8.
6. Doppalapudi H, Yamada T, Ramaswamy K, Ahn J, Kay GN. Idiopathic focal epicardial ventricular tachycardia originating from the crux of the heart. *Heart Rhythm*. 2009;6:44–50. doi: 10.1016/j.hrthm.2008.09.029.
7. Scanavacca MI, Sternick EB, Pisani C, Lara S, Hardy C, d'Ávila A, Correa FS, Darrieux F, Hachul D, Marcial MB, Sosa EA. Accessory atrioventricular pathways refractory to catheter ablation: role of percutaneous epicardial approach. *Circ Arrhythm Electrophysiol*. 2015;8:128–136. doi: 10.1161/CIRCEP.114.002373.
8. Ramanathan C, Jia P, Ghanem R, Ryu K, Rudy Y. Activation and repolarization of the normal human heart under complete physiological conditions. *Proc Natl Acad Sci USA*. 2006;103:6309–6314. doi: 10.1073/pnas.0601533103.
9. Berger T, Fischer G, Pfeifer B, Modre R, Hanser F, Trieb T, Roithinger FX, Stuehlinger M, Pachinger O, Tilg B, Hintringer F. Single-beat noninvasive imaging of cardiac electrophysiology of ventricular preexcitation. *J Am Coll Cardiol*. 2006;48:2045–2052. doi: 10.1016/j.jacc.2006.08.019.
10. Haissaguerre M, Hocini M, Shah AJ, Derval N, Sacher F, Jais P, Dubois R. Noninvasive panoramic mapping of human atrial fibrillation mechanisms: a feasibility report. *J Cardiovasc Electrophysiol*. 2013;24:711–717. doi: 10.1111/jce.12075.
11. Revishvili AS, Wissner E, Lebedev DS, Lemes C, Deiss S, Metzner A, Kalinin VV, Sopov OV, Labartkava EZ, Kalinin AV, Chmelevsky M, Zubarev SV, Chaykovskaya MK, Tsiklauri MG, Kuck KH. Validation of the mapping accuracy of a novel non-invasive epicardial and endocardial electrophysiology system. *Europace*. 2015;17:1282–1288. doi: 10.1093/europace/euu339.
12. Mandegar MH, Saidi B, Oraii S, Tehrai M, Roshanali F. Giant diverticulum causing ventricular arrhythmia. *J Card Surg*. 2011;26:117–118. doi: 10.1111/j.1540-8191.2010.01083.x.

KEY WORDS: algorithms ■ coronary sinus ■ diverticulum ■ electrophysiology ■ tomography

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