Radiofrequency ablation of premature ventricular contractions originating from the aortomitral continuity localized by use of a novel noninvasive epicardial and endocardial electrophysiology system

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Introduction

Accurate localization of the origin of focal ventricular arrhythmias is a prerequisite to successful catheter ablation. However, ventricular arrhythmias arising from complex anatomic sites render mapping and ablation difficult. On the 12-lead surface electrocardiography (ECG), the QRS morphology of ventricular arrhythmias may be difficult to discern because of their anatomic vicinity, in particular if arising from the right ventricular outflow tract, aortic sinus cusps, and aortomitral continuity (AMC).[1–3] A new approach to accurately diagnose ventricular arrhythmias based on noninvasive electrocardiographic imaging was recently introduced.[4,5] This case report demonstrates the feasibility of a novel noninvasive epicardial and endocardial electrophysiology system (NEEES) to identify the origin of premature ventricular contractions (PVCs) arising from the AMC.

Case report

A 54-year-old male patient was referred for the treatment of symptomatic monomorphic PVCs (12,607/24 h or 17.8% of all QRS complexes). Transthoracic echocardiography showed no structural heart disease. The 12-lead ECG demonstrated sinus rhythm and frequent monomorphic PVCs with an inferior axis and right bundle branch block QRS morphology (Figure 1).

The patient underwent noninvasive mapping using the NEEES (EP Solutions SA, Yverdon-les-Bains, Switzerland) prior to an invasive electrophysiology study. Custom-made magnetic resonance imaging (MRI)-compatible electrode arrays with a total of 224 contacts were placed onto the patient's torso followed by same-day contrast MRI (Magnetom Avanto 1.5T; Siemens, Erlangen, Germany) of the heart and torso. Scanning of the torso and heart was performed simultaneously using the vibe mode without ECG gating and included an intravenous contrast agent injection (Dotarem, Guerbet, France; 20 mL) during a 30-second breath hold. The MRI data were imported in DICOM format and semi-automatically processed by the NEEES to reconstruct realistic 3-dimensional models of the torso and heart. In the electrophysiology laboratory, the body-surface electrode arrays were connected to the NEEES amplifier and 224-channel ECG recording was performed to capture the clinical PVC. The body-surface ECG data were processed by the NEEES using its inverse problem solution software in combination with anatomic data from the heart and torso in order to reconstruct local unipolar electrograms (EGs) in more than 2500 nodes at the epicardium and endocardium.[6–9] Based on the unipolar EGs, the noninvasive isopotential and isochronal maps were built and analyzed to localize the PVC origin. Unexcitable tissue including the aorta was excluded from noninvasive EG reconstruction in order to avoid mapping inaccuracies from far-field projection.

During the next step, invasive endocardial activation mapping was performed using the CARTO 3 system (Biosense Webster, Inc, Diamond Bar, CA) as previously described.[10] In brief, during episodes of spontaneous PVCs, activation mapping was performed with an irrigated 7.5F deflectable quadripole 3.5-mm-tip ablation catheter (Navistar

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KEY TEACHING POINTS

- Noninvasive mapping will direct the operator to the region of interest during invasive mapping.
- A novel noninvasive cardiac mapping system allows for accurate localization of premature ventricular contractions arising from the aortomitral continuity.
- Preprocedural magnetic resonance imaging can be used for noninvasive cardiac mapping, avoiding additional radiation exposure.

ThermoCool; Biosense Webster, Inc). Mapping of the left ventricle was performed via a combined transseptal and retrograde approach, and mapping of the aortic root was performed via the retrograde approach. Bipolar EGs were filtered at 30–400 Hz. The arrhythmia origin and ablation target were defined by the earliest activation (local bipolar ventricular EGs preceding the onset of the surface QRS complex and a QS complex on unipolar recordings). Radiofrequency energy was delivered using a power of 30 W and increased up to 40 W with a maximum temperature of 43°C.

Analyzing the noninvasively reconstructed unipolar EGs, a QS complex was noted at the AMC on the epicardial and endocardial 3-dimensional anatomic models. Similarly, the noninvasive isochronal maps demonstrated earliest activation at the AMC area (Figure 2, online Supplementary Videos 1 and 2). Invasive activation mapping confirmed the zone of earliest activation close to the AMC. The earliest intracardiac

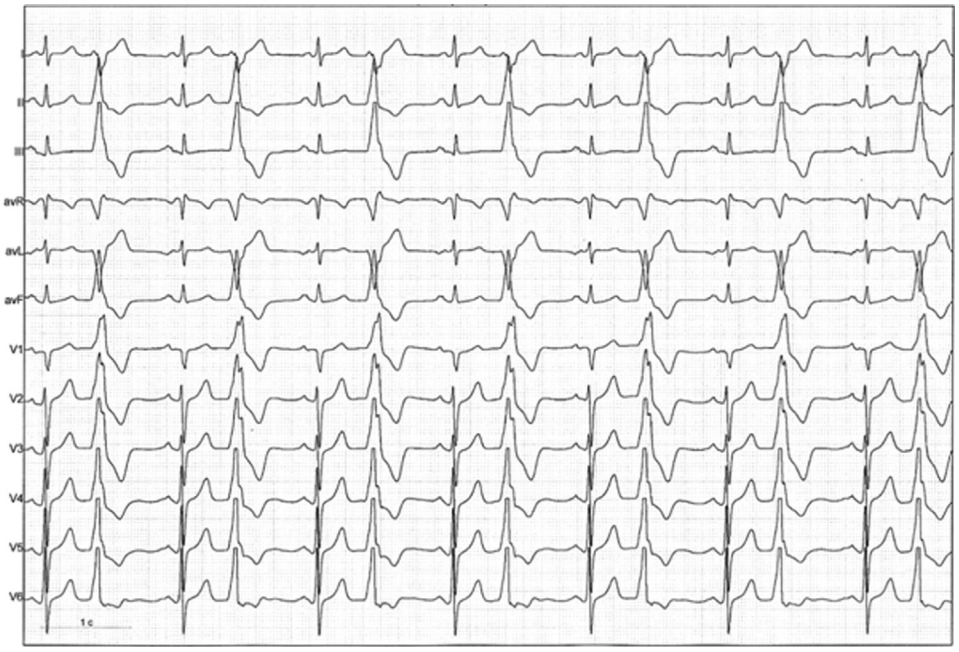

Figure 1 The 12-lead surface electrocardiography showing ventricular bigeminy with right bundle branch block morphology, inferior axis, positive concordance, and negative QS-complex in lead aVL.

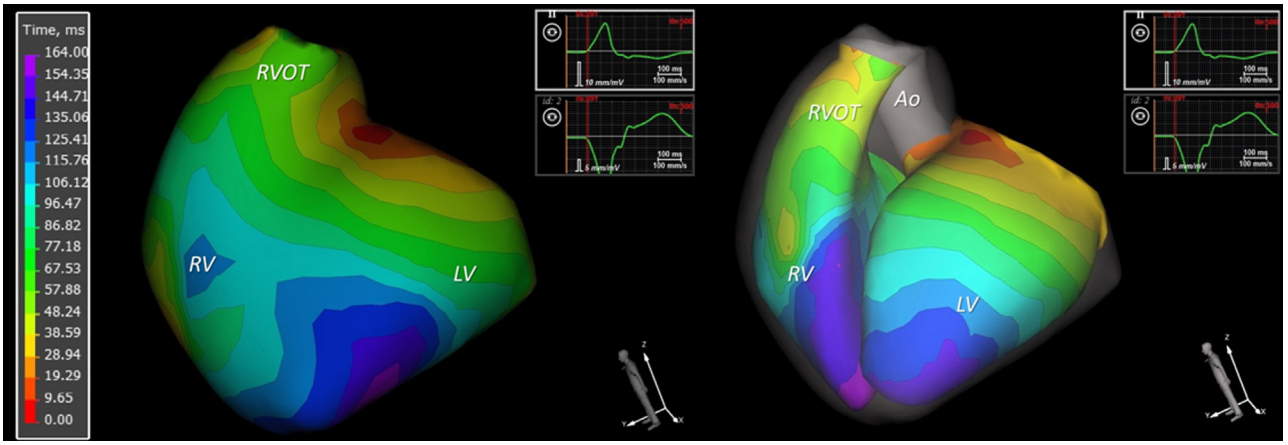

Figure 2 Image on left shows noninvasive isochronal epicardial map reconstructed on the 3-dimensional (3-D) biventricular heart model. The zone of earliest activation (*red*) was identified close to the aortomitral continuity on the epicardial surface. Image on right shows noninvasive isochronal endocardial map reconstructed on the 3-D biventricular heart model in posterior-anterior projection. The zone of earliest activation (red) was identified close to the aortomitral continuity on the endocardial surface. Ao = aorta; LV = left ventricle; RV = right ventricle; RVOT = right ventricular outflow tract.

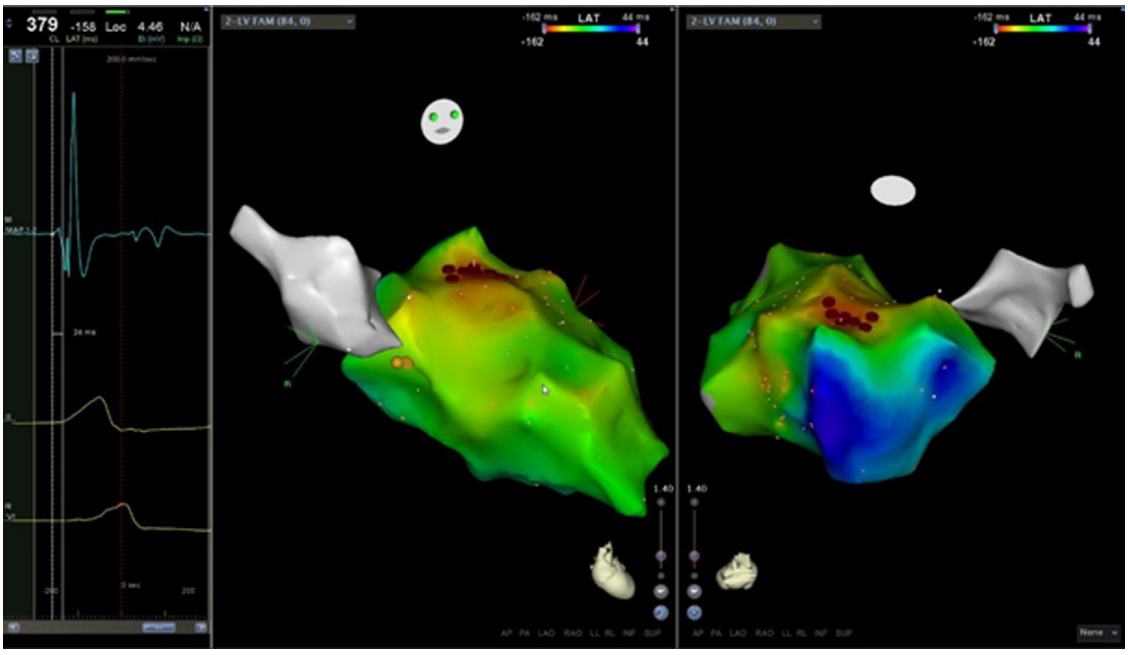

Figure 3 Invasive endocardial 3-dimensional (3-D) activation map of the left ventricle (*colored*) and invasive 3-D anatomic map of the aortic root (*gray*). The zone of earliest activation (*orange*) during premature ventricular contraction (PVC) (preceding the QRS-complex in surface lead II by 24 ms) was identified close to the aortomitral continuity, correlating well with the noninvasive maps (mitral valve indicated by the *blue area*). Ablation was performed in this area (*red dots*) and abolished the PVC.

bipolar EG preceded the onset of the surface QRS complex in lead II by 24 ms (Figure 3, online Supplementary Video 3). Radiofrequency ablation performed in this area abolished the clinical PVC at the end of the procedure. After a follow-up period of 6 months the patient remained asymptomatic, and Holter monitoring revealed a significant reduction in PVC burden from 17.8% to 1.4% of all QRS complexes.

Discussion

Noninvasive electrocardiographic mapping allows for accurate localization of the PVC origin. The NEEES displays electrical wavefront propagation of various arrhythmias without the need for an invasive procedure and directs the physician to the region of interest during invasive mapping and ablation. Other noninvasive mapping systems only provide epicardial mapping, and utilize computed tomography scanning for acquisition of anatomic data, which may be associated with substantial radiation exposure. The NEEES can process anatomic data reconstructed from computed tomography or MRI, and allows for both epicardial and endocardial activation mapping using different acquisition and processing algorithms. In the present case, using epicardial and endocardial isopotential maps rendered by the NEEES, the earliest site of activation was identified close to the AMC, which correlated with the earliest activation during invasive mapping and was verified by successful ablation in this area.

Conclusion

Using data from preprocedural cardiac MRI, the NEEES allowed accurate, noninvasive definition of the origin of monomorphic PVCs arising from the AMC.

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.2016.02.004.

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