Cardiac magnetic resonance imaging for coregistration during ablation of ischemic ventricular tachycardia for identification of the critical isthmus

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Introduction

Catheter ablation of ventricular tachycardia (VT) has been proven to be an effective therapy in patients with ischemic cardiomyopathy.[1] Activation mapping for stable VT and substrate-based mapping during sinus rhythm have become the mainstay of VT ablation.[2] Preprocedural high-resolution late gadolinium–enhanced cardiac magnetic resonance (LGE-CMR) imaging may be used for the purpose of
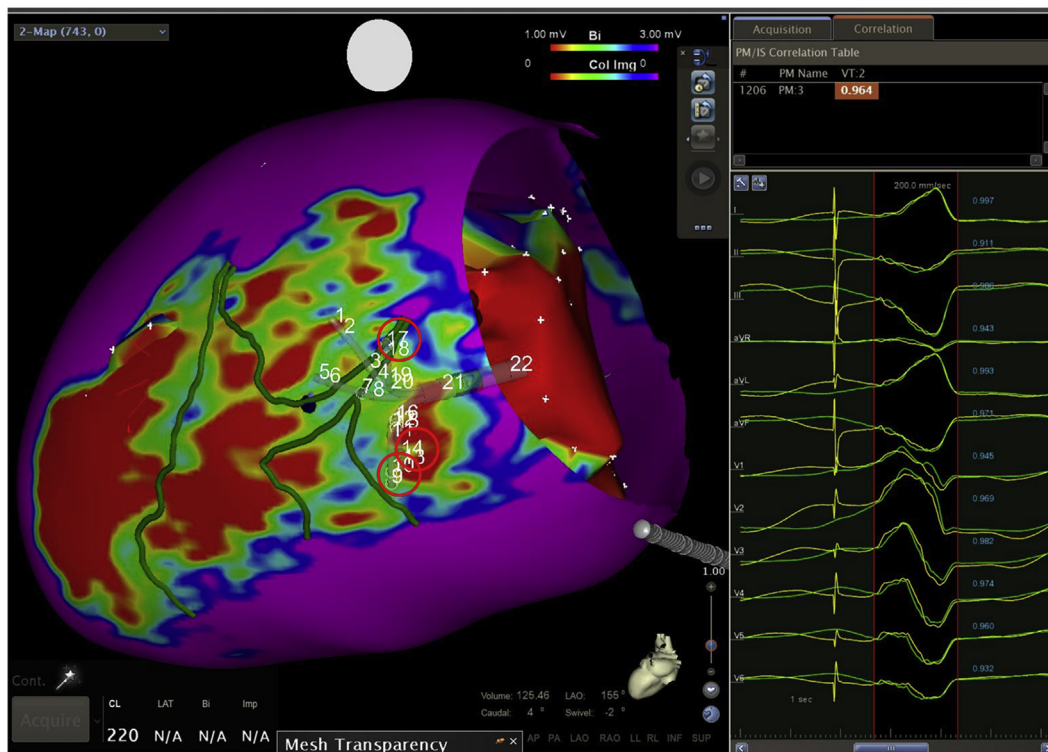

Figure 1 Three-dimensional scar reconstruction of a high-resolution late gadolinium–enhanced cardiac magnetic resonance image merged with the CARTO-derived electroanatomic voltage map. The position of the PentaRay catheter is demonstrated, with red circles indicating respective electrodes in the ventricular tachycardia channel from the entrance to the exit: 17-18, 13-14, and 9-10. Green lines indicate conducting channels as detected on late gadolinium–enhanced cardiac magnetic resonance images (*left*); pace map from electrodes 13-14 resulted in a PaSo score of 96.4% (*right*).

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scar characterization and for the detection of conducting channels on the basis of the 3-dimensional (3D) distribution of signal intensity variation within the left ventricular myocardium.[3] In the present case, we report on VT ablation guided by LGE-CMR imaging, which is coregistered with electroanatomic voltage and activation maps.

KEY TEACHING POINTS

- Magnetic resonance imaging is a useful method to predict the location of the arrhythmogenic substrate of ischemic ventricular tachycardia (VT).

- The Automatic Detection of Arrhythmic Substrate system can help understand the 3-dimensional scar anatomy and can predict the location of an inhomogeneous conducting channel in the scar area.

- Pace mapping can help to individuate the channel responsible for the clinical VT and the VT exit site.

Case report

An 83-year-old man with a history of inferolateral myocardial infarction was referred to our hospital with recurrent hemodynamically stable VT unresponsive to antiarrhythmic medications. On admission, initial echocardiography showed a reduced left ventricular ejection fraction of 40%. Preprocedural 3D high-resolution LGE-CMR imaging was performed (Ingenia 1.5T MRI system, Philips, Eindhoven, The Netherlands) 10 minutes after the application of intravenous contrast (gadolinium-DTPA [diethylenetriaminepentaacetic] 0.2 mmol/kg; free-breathing, navigator-gated 3D inversion recovery sequence with individually adapted inversion delay and near isotropic spatial resolution of $0.7 \times 0.7 \times 1.0$ mm) (Supplemental Figure 1). LGE-CMR images were processed off-line using a dedicated software package (Automatic

Detection of Arrhythmic Substrate, ADAS-VT, Galgo Medical SL, Barcelona, Spain) with semi-automatic determination of endo- and epicardial borders of the left ventricular myocardium and subsequent automatic characterization of an internal 3D scar architecture on the basis of the CMR-signal intensity distribution pattern. Left ventricular myocardial wall was split into layers using 10% steps from the endocardium to the epicardium. Dense scar, heterogeneous tissue ("border zone"), and normal tissue were differentiated using prescribed thresholds of >60% of the maximum pixel intensity, 40% to 60%, and <40%, respectively.[4] Within the scar area, conducting channels were defined as a border zone corridor connecting normal tissue ("healthy-to-healthy"). In a matching location, VT QRS morphology on the surface electrocardiogram and ADAS-VT were suggestive of a VT-related channel in the inferolateral wall, with the scar area extending to the mitro-aortic continuity. Consequently, after an uncomplicated transseptal puncture, substrate mapping (electroanatomic mapping) of the left ventricle was performed during sinus rhythm using a 1-mm multielectrode mapping catheter (PentaRay, Biosense Webster Inc., Diamond Bar, CA). Bipolar signals were filtered on a Prucka Cardiolab system (Prucka Inc., Milwaukee, WI) with a range of 30–500 Hz. Voltage map was created in sinus rhythm, with the commonly applied thresholds of 0.5–1.5 mV for scar and normal tissue[5] (Supplemental Figure 2). Afterward, the electroanatomic map was coregistered manually with the LGE-CMR–based ADAS-VT shell using a 3D mapping system (CARTO 3, Biosense Webster). During ongoing clinical VT, recordings from the PentaRay catheter located in the scar area showed diastolic potentials in the PentaRay splines 9-10, 13-14, and 17-18 (Figure 1). All mid-diastolic
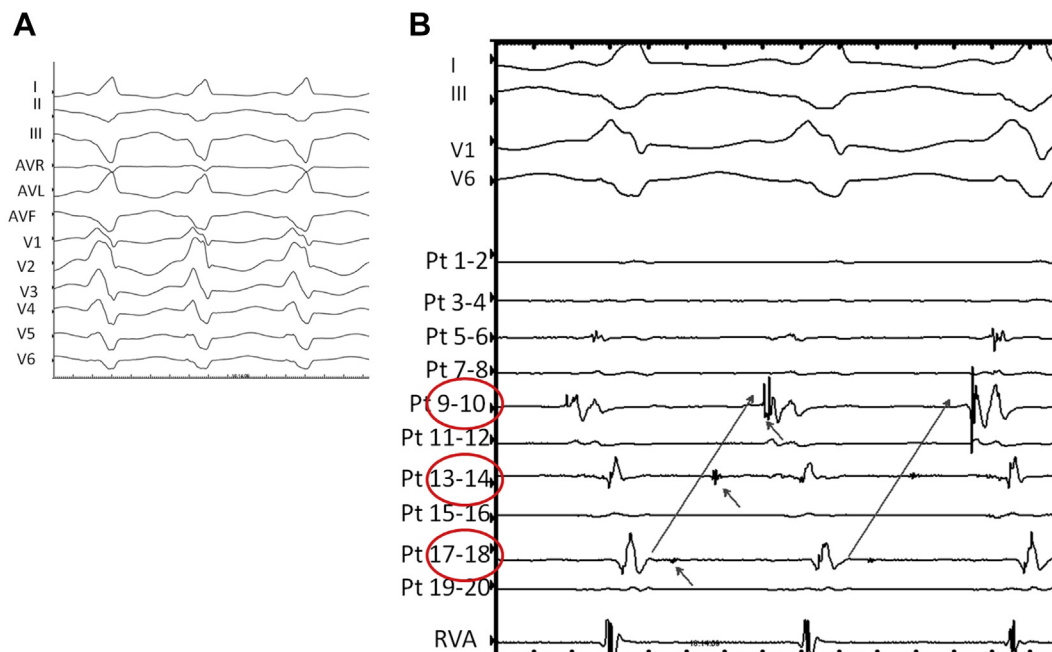

Figure 2   A: Ventricular tachycardia (VT) morphology on the 12-lead electrocardiogram. **B:** Electrograms from the PentaRay splines during VT demonstrating activation through the VT-protected isthmus: from 17-18 (prediastolic) to 13-14 (mid-diastolic) and 9-10 (end-diastolic).
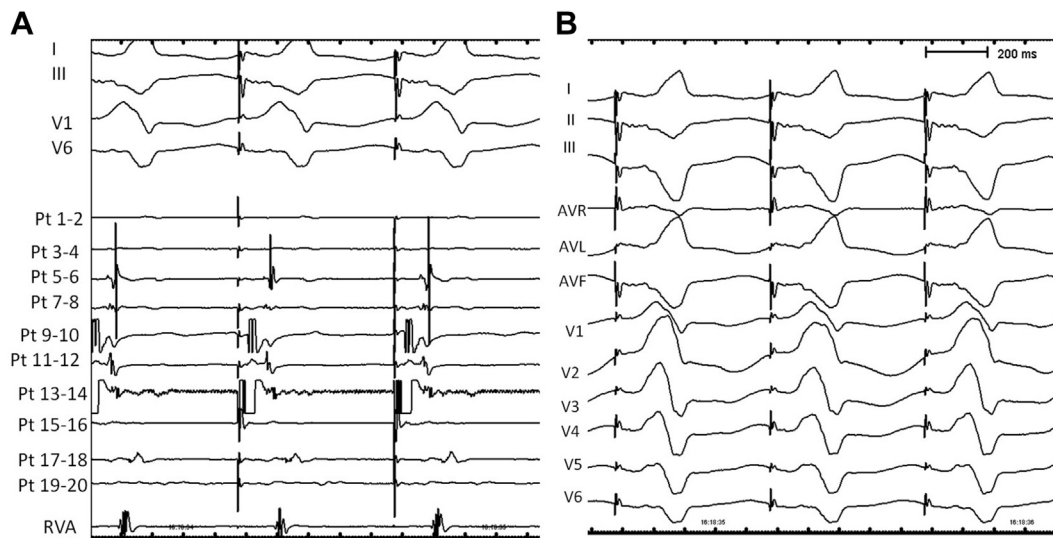
Figure 3   **A:** During sinus rhythms, pacing from PentaRay electrodes 13-14 located in the middle of the protected isthmus during ventricular tachycardia. **B:** Twelve-lead electrocardiogram during stimulation from PentaRay 13-14 showing a clinical ventricular tachycardia and paced QRS correlation of 96% (Figure 2) and a long S-QRS interval.

potentials were located within the ADAS-VT–identified channel (Figure 2). In addition, using pace mapping from the PentaRay splines we could identify the entrance and exit sites of the clinical VT at the corresponding exit and entrance of the culprit channel on the LGE-CMR–based ADAS-VT image (Figures 1 and 3). After reinduction of VT, a radiofrequency line crossing the VT channel between the mitral annulus and the inferior portion of the scar led to successful VT termination. Finally, further ablation was carried out in order to eliminate late potentials in the scar area, resulting in complete noninducibility of any VT.

Discussion
LGE-CMR imaging is the recognized standard of reference for myocardial scar detection and proved useful for substrate characterization before electrophysiological procedures. Continuous corridors of intermediate signal intensity on LGE-CMR images interspersed within dense LGE areas most likely represent the CMR equivalent of slow conduction zones during VT. Using the ADAS-VT software, dedicated 3D reconstructions of left ventricular myocardial layers in patients with post–myocardial infarction VT can identify conducting channels and facilitate VT ablation. In the present case, simultaneous recordings from the multielectrode mapping catheter located within the coregistered LGE-CMR–defined 3D substrate provided electrophysiological evidence that the CMR-defined channel architecture matched the critical isthmus of the clinical VT.

Conclusion
ADAS-VT software has the ability to characterize the myocardial fibrosis and potential critical isthmus and recon-

struct in 3-D the scar that could then be integrated in a 3D-mapping system. This system seems to have also high capacity to detect the arrhythmogenic substrate and the isthmus of the reentry circuits critical for VT generation. Further studies are warranted to assess the feasibility of this technology.

Appendix
Supplementary data
Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2017.11.008.

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