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Noninvasive Epicardial and Endocardial Electrocardiographic Imaging of Scar-Related Ventricular Tachycardia

Linwei Wang^{a,*}, Omar A. Gharbia^a, B. Milan Horáček^b, and John L. Sapp^b

^aRochester Institute of Technology, Rochester, NY, USA

^bDalhousie University, Halifax, NS, Canada

Abstract

Background—The majority of life-threatening ventricular tachycardias (VTs) are sustained by heterogeneous scar substrates with narrow strands of surviving tissue. An effective treatment for scar-related VT is to modify the underlying scar substrate by catheter ablation. If activation sequence and entrainment mapping can be performed during sustained VT, the exit and isthmus of the circuit can often be identified. However, with invasive catheter mapping, only monomorphic VT that is hemodynamically stable can be mapped in this manner. For the majority of patients with poorly tolerated VTs or multiple VTs, a close inspection of the reentry circuit is not possible. A noninvasive approach to fast mapping of unstable VTs can potentially allow an improved identification of critical ablation sites.

Methods—For patients who underwent catheter ablation of scar-related VT, CT scan was obtained prior to the ablation procedure and 120-lead body-surface electrocardiograms (ECGs) were acquired during induced VTs. These data were used for noninvasive ECG imaging to computationally reconstruct electrical potentials on the epicardium and on the endocardium of both ventricles. Activation time and phase maps of the VT circuit were extracted from the reconstructed electrograms. They were analyzed with respect to scar substrate obtained from catheter mapping, as well as VT exits confirmed through ablation sites that successfully terminated the VT.

Results—The reconstructed reentry circuits correctly revealed both epicardial and endocardial origins of activation, consistent with locations of exit sites confirmed from the ablation procedure. The temporal dynamics of the reentry circuits, particularly the slowing of conduction as indicated by the crowding and zig-zag conducting of the activation isochrones, collocated well with scar substrate obtained by catheter voltage maps. Furthermore, the results indicated that some reentry circuits involve both the epicardial and endocardial layers, and can only be properly interpreted by mapping both layers simultaneously.

*Corresponding author. c/o PhD Program of Computing and Information Sciences, 74-1075, 102 Lomb Memorial Dr., Rochester Institute of Technology, Rochester, NY 14623, United States of America. linwei.wang@rit.edu.

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Conclusions—This study investigated the potential of ECG-imaging for beat-to-beat mapping of unstable reentrant circuits. It shows that simultaneous epicardial and endocardial mapping may improve the delineation of the 3D spatial construct of a reentry circuit and its exit. It also shows that the use of phase mapping can reveal regions of slow conduction that collocate well with suspected heterogeneous regions within and around the scar.

Keywords

Electrocardiographic imaging; scar-related ventricular tachycardia; catheter ablation; phase mapping; inverse solution

Introduction

Sustained ventricular tachycardia (VT) contributes to over 350,000 sudden cardiac deaths each year in the US [1]. More than 90% of the sustained monomorphic VTs are due to reentry involving an area of ventricular scar with narrow strands of surviving tissue [2]. While the use of implantable cardioverter defibrillators (ICDs) provides protection from sudden death, it does not prevent VT. Recurrent shocks are also associated with reduced quality of life and increased risk of death [3]. To treat VT and prevent its recurrence, catheter ablation is an important therapeutic option to target and modify the isolated channels of surviving myocardium within the scar [4].

Successful ablation requires an effective identification of myocardial sites critical to the VT circuit. If the reentry circuit can be mapped with a combination of activation and entrainment mapping, its exit and isthmus can often be identified [5]. However, with invasive catheter mapping, this is only feasible in a small percentage of VTs that are hemodynamically stable [1]. For the majority of patients with ill-tolerated VTs or multiple VTs, the alternative of substrate mapping must be used to identify potential critical sites during sinus or paced rhythm [5]. In comparison to VT/entrainment mapping, substrate mapping provides a less specific identification of circuit exits/isthmuses.

Noninvasive ECG-imaging involves the computational reconstruction of cardiac electrical activity, using high-density body-surface ECG data in combination with heart-torso tomographic scans [6] [13]. Collectively, ECG-imaging techniques have demonstrated potential in a variety of clinical applications, such as the mapping of atrial flutter and fibrillation [14], the localization of premature ventricular contractions [15], and the detection of myocardial ischemia [13]. For scar-related VT, there has been an interest in using ECG-imaging to electrically delineate the myocardial scar as an arrhythmogenic substrate for VT [16], [17]. Although 3D scar anatomy can be reliably delineated by imaging modalities such as delayed contrast-enhanced MRI [18], ECG-imaging also has the unique ability to reveal the arrhythmia dynamics of VT. In the latter context, some studies have reported the use of ECG-imaging in reconstructing the activation isochrones and localizing the origin of VT [19], [20]. These initial studies focused on VT mapping on only the epicardium of the heart and the reentry circuit was studied in the form of a static map of activation sequence. The temporal dynamics of the reentry circuit, its epicardial and endocardial involvement, and its relation to the myocardial scar substrates still remain largely unexplored.

In the present study, we investigated the clinical potential of ECG-imaging in rapid mapping of unstable scar-related VT. We hypothesized that: 1) simultaneous epicardial and endocardial reconstructions by ECG-imaging will improve the delineation of the 3D construct of the reentry circuit and its exit; and 2) the use of phase mapping [21] in addition to a static map of activation sequence will enhance the visualization of the temporal dynamics of the reentry circuit. Experiments were conducted on patients who underwent catheter ablation of scar-related VT. The reconstructed reentry circuits were examined with respect to VT exits confirmed during ablation procedures, as well as scar substrates identified from invasive mapping.

Materials and Methods

Patients & Clinical Methods

Patients were enrolled from those undergoing epicardial and endocardial catheter mapping and ablation of VT. As detailed in our previous study [20], in a protocol approved by the institutional Research Ethics Board, axial CT (0.8–3 mm, Siemens Sonata, Erlangen, Germany) was performed within 24 hours before the procedure. Body surface electrodes (Foxmed, Idstein, Germany) were applied immediately before the clinical procedure according to the previously published Dalhousie protocol [22]. 120-lead ECGs were recorded during induced VT using a 128-channel acquisition system (Mark 6, BioSemi, Amsterdam, the Netherlands).

The electrophysiology study and catheter ablation were performed according to usual clinical protocols and included venous and arterial vascular access with catheters placed at the His bundle region and in the right ventricle. LV endocardial mapping was performed by the retrograde aortic approach. The pericardial space was entered percutaneously using previously published techniques.[23]. A three-dimensional electroanatomic mapping system was utilized (CARTO, Biosense Webster, Inc., Diamond Bar, CA) to acquire substrate maps during sinus or paced rhythm. An irrigated ablation catheter (Thermocool Navistar, Biosense Webster, Diamond Bar, CA, USA) was used for mapping and ablation. VT was induced by programmed stimulation from the RV apex or outflow tract.

Data Processing

Off-line processing of 120-lead ECG recordings was performed using custom software [24]. Faulty leads were manually identified and discarded. Noise was removed using wavelet filters with the following parameters customized: level of wavelet decomposition, the type of orthogonal wavelets (Symlet wavelets were used in this study), and the cut-off frequency threshold. Baseline drift was corrected by fitting a low-order polynomial (with a customizable order of the polynomial) to the signal and subtracting it from the signal. All customizable parameters were determined by the user based on visual inspection of the resulting signals, via interactive windows. Finally, representative beats were manually selected for ECG imaging.

Patient-specific geometrical models of the heart and torso were created from axial CT scans using custom-written Matlab routines. The heart model included the epicardial and the

endocardial surfaces of both ventricles. The torso model was a homogeneous volume conductor model in the form of a triangulated body-surface mesh with 120 nodes corresponding to the surface electrodes [17]. Although this model neglects the effect of tissue inhomogeneity, the trade-off is a simplified modeling procedure that can better fit into clinical routines.

ECG-Imaging

To obtain electrograms on both epicardial and endocardial surfaces of the heart, the unknown variable was formulated as the extracellular potential on the nodes of the entire bi-ventricular mesh, *i.e.*, the inverse solution solves for the unknown potential throughout the epicardial, left-endocardial, and right-endocardial surface of the ventricles. This inverse solution was calculated from the body-surface ECG at each time instant independently. As an initial feasibility study, a simple second-order Tikhonov regularization was used to obtain the inverse solution:

$$\hat{\mathbf{s}} = \underset{\mathbf{s}}{\operatorname{argmin}} \{ \|\phi - \mathbf{H}\mathbf{s}\|_2^2 + \lambda \|\mathbf{L}\mathbf{s}\|_2^2 \} \quad (1)$$

where $\mathbf{s}$ represents the unknown potential on the ventricular surface at a given time instant, ϕ the ECG data on the body surface at the given time instant, and $\mathbf{H}$ the transfer matrix constructed for the subject-specific geometrical model. $\mathbf{L}$ is a Laplacian matrix on the ventricular surface. The first term of equation (1) seeks a solution of $\mathbf{s}$ that will best fit the surface ECG data; the second term of equation (1) enforces a local smoothness of the spatial distribution of $\mathbf{s}$. The regularization parameter λ controls the relative importance of these two terms. In all the results reported in this paper, the value of λ was fixed at 0.5. The computation time for reconstructing one complete beat of λ VT is on the order of 10^{-1} seconds. Therefore, in comparison to sequentially assembled electrograms at different locations of the heart over multiple beats, ECG-imaging simultaneously constructs all electrograms on the heart in a single beat in real time. It thus allows rapid mapping of unstable VTs.

Post-Processing: Phase Mapping and Extraction of Activation

To track the progression and spatiotemporal organization of reentry circuits during VT, we adopted the phase-mapping approach [21]. A standard approach which is prone to errors caused by the variable and complex morphology of the electrograms is to determine the time of the steepest negative deflection on each local electrogram. Phase mapping uses the Hilbert Transform to generate phase-shifted signals from the reconstructed electrograms, and then computes the instantaneous phase from these two signals for each node of the cardiac mesh at each time instant. The resulting maps track the progression of the phase from $-\pi$ to π at each location of the cardiac mesh. The depolarization wave front (local activation time) is then defined as the time when the local phase crosses zero from negative to positive values. This operation can be executed in real time.

Results

As a feasibility study, three detailed VT reentry circuits reconstructed by ECG-imaging on two patients are reported here.

VT 1 Epicardial Reentry Circuit

The first VT was from a patient with non-ischemic cardiomyopathy. Sinus-rhythm substrate mapping revealed an infero-lateral scar located at the basal region of the LV epicardium (Fig. 1A). During ablation, the VT exit was identified to be at the basal-lateral region of the LV epicardium.

In the map of activation sequence (Fig. 1B) reconstructed by ECG-imaging, the exit (white star) is shown at the high basal-lateral region of the LV epicardium, consistent with the actual VT exit identified by contact mapping. The activation sweeps toward the apex at the anterior part of the heart, and rotates back toward the base at the inferior-lateral part of the epicardium. Slow conduction (crowding of isochrones) can be observed at regions consistent with the margin of the scar. An especially slow conduction can be noticed at the basal-lateral region of the LV before the circuit exits, consistent with the location of a heterogeneous zone at the ablation sites.

To better appreciate the temporal dynamics of the reentry circuit, snapshots of phase maps of the reconstructed electrograms are shown in Fig. 1C. The depolarization wave front (locations with phase = 0) is tracked by the light blue contour and annotated by small white arrows. The timing of each snapshot with respect to the onset of the QRS is labeled and tracked on an ECG trace on lead V2 (*i.e.*, -20 ms = 20 ms before the QRS onset). Low-voltage regions identified from catheter maps (Fig. 1A) are registered to the patient-specific ventricular model and displayed as a semi-transparent gray patch.

As shown, the reentry circuit exits at the high basal-lateral LV, which is consistent with the VT exit confirmed from the ablation procedure. In agreement with clinical knowledge, it is identified at 35 ms prior to the QRS onset during presystolic activation [1]. The propagation was relatively slow before the QRS onset as the wave front approached the scar exit. After the QRS onset, the propagation shows a macro-rotation around the inferior lateral scar (0–70 ms): note the relatively smooth propagation at the anterior LV where the extent of scar tissue is minimal, versus a notable delay of activation of the wave front at mid-inferior LV at the site of the myocardial scar (70–80 ms). After the circuit reenters the scar and before it exits, there is a long period (105–175 ms) when the activation wave front exhibits a zig-zag course of propagation inside the scar. This is followed by very slow conduction at the epicardium of the lateral LV throughout the diastolic phase of the activation, until the wave front exits again and repeats the next VT beat. Interestingly, as shown in the last two snapshots of the phase-map sequence, during the very-slow epicardial conduction before the next exit (occurring during the T-wave of the VT cycle), the same circuit also quickly depolarizes the endocardium of the LV with a sweep from inferior base down to apex and then up to the anterior base. This beat-to-beat, 3D image reveals the complete epicardial and endocardial involvement of the circuit, as well as the continuous nature of reentry-circuit dynamics in time.

VT 2 – Epicardial & Endocardial Figure-of-Eight Reentry Circuit

The second VT under study came from a patient with prior infarction. As revealed by the bipolar contact signal amplitude (voltage) map, the myocardial scar is primarily located at inferior endocardium of the LV and extends somewhat to the inferior epicardium (Fig. 2A). A large area of low voltage and unexcitable tissue (gray) was also found on the anterior surface of the epicardium, although it is not clear whether it was due to the presence of myocardial scar or a fat layer. This clinical VT had a confirmed exit site at the high basal anterior region of the LV endocardium below the aortic valve.

ECG-based reconstruction of the activation sequence of this VT is shown in Fig. 2B. From the epicardial view, the earliest epicardial breakthrough can be observed at high basal anterior LV consistent with an endocardial exit at the same location. However, the overall propagation pattern on the epicardium is suggestive of a focal rather than macro-reentrant arrhythmia. To appreciate the complete dynamics of this VT circuit, however, an understanding of endocardial involvement is necessary.

Fig. 2C shows snapshots of the arrhythmia dynamics throughout the epicardial and endocardial layer. As shown, within 100 ms prior to the QRS onset of this VT, the exit of the circuit can be seen at the high basal region of the LV endocardium. After breaking through the epicardium, one part of the circuit (Fig. 2C, row 2) sweeps through the basal-anterior region of the epicardium at a slower conduction velocity (0–30 ms, crowded isochrones), followed by a faster sweep through the rest of the anterior epicardium (30–90 ms) and rotates back to the basal epicardium of the RV (90–120 ms). At the same time, the other part of the circuit (Fig. 2C, row 1) involves a counter-clock-wise macro-circuit across the lateral LV and anterior region of the epicardium, until it rotates back to the base of the RV epicardium (0–120 ms). These two waves meet, propagate through the endocardium of the RV (120–180 ms), the septum (180–240ms), and reenter the endocardium of the LV, until it is ready to exit again in the next VT beat. This epicardial and endocardial activation sequence suggests a stable figure-of-eight reentry circuit that exits at the basal-anterior part of the LV, which can be appreciated in the endocardial view of activation sequence shown in Fig. 2A. This can only be appreciated by examining its dynamics at both epicardial and endocardial layers.

VT 3 – Epicardial & Endocardial Figure-of-Eight Reentry Circuit

The third clinical VT came from the same patient as VT 2, although with a different ECG morphology and an exit confirmed at the basal infero-lateral region of the LV endocardium. From the epicardial view of the reconstructed activation sequence (Fig. 3A), an early breakthrough at the infero-lateral region of the LV can be seen within 100 ms preceding the QRS onset during VT. Similar to VT 2, the epicardial dynamics would suggest a focal rather than a reentrant mechanism. Incorporation of the endocardial view, however, reveals a figure-of-eight reentry circuit involving both epicardial and endocardial layers. As shown in Fig. 3B, this circuit exits at the basal infero-lateral region of the LV endocardium. It then breaks into two waves, one activating the inferior epicardial LV until it rotates back to the RV epicardium (0–180 ms). The other wave, at a much later stage (120–180 ms), quickly depolarizes the anterior LV epicardium and the RV lateral wall. These waves then meet (at

180 ms) and rotate back through the RV endocardium, the septum, and reenter the endocardium before exiting at the infero-lateral region again. The figure-of-eight reentry circuit with its endocardial exit site is summarized in the endocardial view of the activation sequence in Fig. 3A. Similar to VT 2, the reentry dynamics of this VT involves the endocardial layer and would not be properly interpreted without knowledge of endocardial activation.

Discussion

This study demonstrated the feasibility of ECG-imaging in fast mapping of patient-specific reentry circuits for complex, unstable, scar-related VT. Firstly, this study demonstrates – to our knowledge for the first time – the noninvasive mapping of reentry circuits throughout both epicardial and endocardial layers. The examples of VT 2 and VT 3 show that, in reentry circuits that involve both epicardial and endocardial layers, only a complete epi-endo reconstruction can reveal the underlying dynamics of the circuit (*e.g.*, a figure-of-eight reentry throughout the epi-endo layers may appear to be a focal arrhythmia on the epicardial layer). Furthermore, by including endocardial reconstruction, we can differentiate between endocardial and epicardial exits, which may have important clinical implications for planning ablation treatment. Secondly, this study shows that the use of phase mapping can facilitate the visualization and tracking of a reentry circuit from beat to beat; it enables a complete inspection of the reentry dynamics not only three-dimensionally in space but also continuously in time. Finally, the dynamics revealed by phase maps also appear to be well correlated with the location of myocardial scar substrate and confirmed VT exit sites. In particular, the slowing of conduction (crowding of isochrones) co-locates very well with the margin of the myocardial scar. The spatial meandering of the isochrones is seen within the dense scar region near the exit, indicating a zig-zag slow-conduction path as expected inside the scar. Additionally, in VT 1 and VT 2, the anchor of the reentry circuit tends to meander around the scar margin in a region close to the expected VT exits, which is consistent with previous findings that the anchors of reentrant circuits may co-localize with anatomical heterogeneities and their spatial meandering is modulated by these heterogeneities [21].

Limitations

This feasibility study has several limitations. First, as a first demonstration of VT mapping throughout epicardial and endocardial layers, we focused on a retrospective analysis of the reconstructed circuit dynamics. While good correlation between the reconstructed circuit and its underlying substrates is found, future studies should focus on how to prospectively identify the critical components (*e.g.*, exit, entrance, isthmus, *etc.*) from the reconstructed circuit. Second, while locations of scar substrate and VT exits were available as reference data from invasive procedures, this study lacked a “gold standard” for the analysis of the VT circuit. This gold standard is clinically difficult to establish due to the difficulty to map a complete VT circuit. In the future, experiments will be designed to include better reference data, such as more specific knowledge about the VT circuit obtained through entrainment mapping, as well as 3D data of myocardial scar obtained from DCE-MRI. Third, this study focused on epicardial and endocardial activation analysis of the reentry circuit; in the future, it would be interesting to compare with a transmural ECG-imaging technique [11] for

analyzing the transmural activation within the scar. Finally, a larger study is needed to establish the potential clinical use of ECG-imaging in fast mapping of unstable VTs and eventually in guiding catheter ablation of VT.

Conclusion

This study demonstrated that simultaneous epicardial and endocardial mapping by ECG-imaging may improve the delineation of the 3D construct of an arrhythmic reentry circuit and its exit. In addition, the use of phase mapping was shown to not only facilitate the tracking of reentry circuit dynamics from beat to beat, but also reveal regions of slow conduction that collocate well with suspected heterogeneous within and around the scar.

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Figure 1.

ECG-imaging results for VT1. Panel A: Bipolar voltage map of myocardial scar on the epicardium by catheter mapping (scar core: ≤ 0.5 mV, red; scar border: 0.5–1.5 mV; healthy myocardium: >1.5 mV, purple). Panel B: Maps of activation time sequence re-constructed from 120-lead ECG during VT. Origin of activation is labeled by a white star at high basal lateral region of the LV epicardium, consistent with the exit site confirmed by ablation. A macro-reentry is seen counter-clock-wise anchored at lateral region of the LV epicardium. Panel C: Snapshots of the depolarization sequence of the reentry circuit reconstructed from 120-lead ECG. The color encodes the phase of the reconstructed electrograms. Isochrones overlaying the color of light blue, corresponding to the zero-crossing from negative to positive phase, represent the depolarization wave front. This wave front is tracked by white arrows throughout the sequence to facilitate visualization. In this sequence, a counter-clock-wise reentry circuit can be seen anchored around the high basal lateral region of the LV epicardium.

Figure 2.

ECG-imaging results for VT2. Color maps in all panels are as described in Figure 1. Panel A: Bipolar voltage map of myocardial scar on the epicardium and endocardium by catheter mapping. Panel B: Maps of depolarization sequence re-constructed from 120-lead ECG during VT. Origin of activation is labeled by a white star at high basal anterior region of the LV endocardium below aortic valve, consistent with the exit site confirmed by ablation. A figure-of-eight reentry is seen involving both the epicardial and endocardial layer of the ventricles. Panel C: Snapshots of the depolarization sequence of the reentry circuit reconstructed from 120-lead ECG. After exiting from the anterior endocardium of the LV, part of the circuit depolarizes the anterior epicardium in a clock-wise rotation (0–120 ms), while the other depolarizes the inferior epicardium in a counter-clock-wise rotation (0–120 ms). This circuit then reenters through the RV endocardium, the septum, toward the LV endocardium, completing a figure-of-eight reentry circuit (150–270 ms).

Figure 3.

ECG-imaging results for VT3. Panel A: Maps of depolarization sequence re-constructed from 120-lead ECG during VT. Origin of activation is labeled by a white star at high basal inferior-lateral region of the LV endocardium, consistent with the exit site confirmed by ablation. A figure-of-eight reentry is seen involving both the epicardial and endocardial layer of the ventricles. Panel B: Snapshots of the depolarization sequence of the reentry circuit reconstructed from 120-lead ECG. After exiting from the infero-lateral endocardium of the LV, part of the circuit depolarizes the inferior epicardium in a counter-clock-wise rotation (0–150 ms), while the other depolarizes the anterior epicardium in a clock-wise rotation (120–210 ms). This circuit then reenters through the RV endocardium, the septum, toward the LV endocardium, completing a figure-of-eight reentry circuit (210–270 ms).