Inverse Solution Electrocardiographic Mapping of Epicardial Pacing Correlates with Three-Dimensional Electroanatomic Mapping

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Abstract

We hypothesized that the calculation of epicardial potentials from body-surface potential maps (BSPMs) could aid ablation of ventricular tachycardia (VT). BSPMs were recorded during epicardial catheter mapping and pacing in 2 patients. Single-beat epicardial maps were calculated by inverse solution using customized torso/cardiac geometry, discretized from a CT scan. During pacing from 48 epicardial sites, we observed stimulus-QRS delay (Stim-QRS) of 27 ± 7 ms and a difference between known pacing locations and calculated sites of earliest potential minima of 1.6 ± 1.4 cm. Pacing in scar and scar-border zones had longer Stim-QRS delay (51 ± 24 ms and 35 ± 23 ms, respectively, p=0.004), and greater distances between known pacing sites and known locations (3.0 ± 1.6 cm and 4.6 ± 2.0 cm, respectively, p=0.0004). BSPM with inverse solution mapping can identify sites of earliest epicardial activation and thus could have clinical utility.

1. Introduction

Prior myocardial infarction is the most common cause of sustained ventricular tachycardia (VT). Management options include implantable defibrillators (ICDs), antiarrhythmic drugs, arrhythmia surgery, and catheter ablation [1,2]. Although ICDs reduce mortality, they do not prevent recurrent VT, which occurs frequently in ICD recipients [3-5]. Catheter ablation has a role as an alternative or adjunct to pharmacologic therapy to prevent recurrent VT [6]. One such technique involves identification of the infarct substrate with pace-mapping around the edges of the infarcted myocardium [7,8] until a match for QRS morphology of VT is seen. An intuitive interpretation of the surface ECG is required for this approach, which is often challenging. A technique to rapidly identify the scar exit sites of VT circuits could facilitate catheter ablation and direct more detailed point-by-point mapping. An endocardial array is available for this purpose, but is costly and requires the introduction of a large intracardiac balloon-mounted array [9].

Body-surface potential mapping (BSPM) is an extension of the standard 12-lead ECG incorporating the recording of multiple surface ECG leads arrayed over the torso. Computerized analysis of BSPM recordings has permitted the extraction of data unavailable from the standard ECG [10-12], matching of recorded paced activation patterns with VT and, with the incorporation of body-surface and heart-surface reconstruction from CT imaging, calculation of epicardial potentials [13,14] and isochrones of activation time [15,16]. We have calculated, by means of inverse solution, epicardial potentials from BSPMs during percutaneous epicardial mapping and compared sites of early minimum of epicardial potentials with pacing site determined with electroanatomic mapping [17].

2. Methods

2.1. Epicardial mapping and ablation

Two patients undergoing epicardial mapping and ablation procedures for recurrent VT were enrolled in a study protocol of BSPM during mapping and ablation of VT approved by the institutional research ethics board. A CT scan of the chest was performed prior to the procedure for image integration with our 3-dimensional mapping system (Carto, Biosense Webster, Diamond Bar, CA). Access to central vasculature was obtained in the usual fashion and electrode catheters were advanced to the right ventricle and His bundle position. Programmed stimulation (up to triple ventricular extrastimuli at two drive cycle lengths from two intracardiac sites) was performed to induce VT. Unstable VTs were terminated with burst pacing or DC cardioversion. The sub-xiphoid

region was prepared and draped and the pericardial space was entered [18-20]. Nonfluoroscopic electroanatomic point-by-point mapping of the epicardium of the left and right ventricles was performed during sinus rhythm. The electroanatomic map was annotated with peak-to-peak bipolar signal amplitude (filtered 30–400 Hz) and values < 1.5 mV were considered consistent with ventricular scar [21]. Areas which could not be captured during pacing at 10 mA (pulse width 2 ms) were considered "dense scar" (which is electrically unexcitable) [22]. Sites widely separated over the epicardial surface were selected for pacing. Pacing at 10 mA, 2-ms pulse width, cycle length 600 ms was performed at each site.

2.2. Body-surface mapping

A standard torso lead set was applied according to our previously published protocol [12]. In brief, a 120-lead array of radiolucent skin electrodes (FoxMed, Idstein, Germany) was applied to the thorax immediately before the EP study and catheter ablation procedure. Signals were acquired using a 128-channel acquisition system (Mark 6, BioSemi, the Netherlands), sampling at 2 kHz. The system's front end was affixed to the procedure table and connected by fibre-optic cable to a laptop computer running custom software (Mapper, Dalhousie University, Canada). Signals were displayed in real time, recorded to hard disk and uploaded after the procedure to our IBM RS/6000 server system. Recordings of 15-s duration were recorded at each pacing site.

2.3. Data processing

During processing, leads with significant artifact were interpolated. Maps were generated for every sample during the paced activation sequence, with the starting point set immediately following the pacing artifact. The CT scan was analyzed with commercial software (Amira, Mercury Computer Systems, Inc) to identify both the torso surface and cardiac surface. These surfaces were discretized and used to customize the inverse solution. Epicardial potential maps were calculated using Tikhonov 2nd order regularization and the regularization parameter was chosen according to the *L*-curve method. For each paced activation sequence, the site of the earliest calculated epicardial minimum potential was identified from a single beat. The discretized cardiac surface was registered with the epicardial map derived from the electroanatomic mapping system using anatomic landmarks (ventricular apex, aortic valve, and mitral valve annulus). Cartesian distances of known pacing sites identified from the electroanatomic mapping system were measured to the site of earliest calculated epicardial minimum potential. Distances were compared for each site.

3. Results

Two patients were studied. Clinical characteristics are summarized in Table 1. Epicardial mapping of both patients revealed localized areas of contiguous low-amplitude (<1.5 mV) bipolar electrograms. For patient 1, 25 epicardial pacing sites were studied. Of these, 20 sites were within areas of diminished signal amplitude, 10 of which resulted in Stim-QRS delay (≥ 60 ms). The remaining 5 sites were within areas of normal signal amplitude. Two of these were at scar margin (≤ 1 cm to sites of multiple contiguous low-amplitude signal sites), one of which resulted in Stim-QRS delay. For patient 2, recordings were made during pacing at 23 widely dispersed epicardial sites, 6 of which were within areas of scar (none with Stim-QRS delay ≥ 60 ms) and 7 of which were at the scar margin. These did not result in any Stim-QRS delay.

Table 1: Characteristics of Patients

Pt	Age/Sex	Disease	Presentation	Findings
1	29/M	Dilated CM EF 40%	VT Storm, recurrent ICD shocks, amiodarone thyroiditis, prior failed endocardial catheter ablation	Posterobasal epicardial scar with central area of unexcitable scar. 2 VTs induced, not tolerated, successfully ablated from epicardium
2	70/M	Ischemic CM EF 81%	Recurrent sustained VT with near syncope, failed endocardial catheter ablation	3 VTs induced, one sustained. Basal inf LV epicardial scar, with VT exit from inferior margin. VT1 term with ablation, VT3 rendered noninducible, VT2 nonsustained

Stim-QRS delay was longer in areas of scar (51 ± 24 ms) and in scar border zone (35 ± 23) in comparison with normal myocardium (27 ± 7 ms, p = 0.004). Sites of early minima were identified 2.9 ± 1.9 cm from the pacing site determined from the registered electroanatomic and computed tomographic maps. Inverse solution sites of early minima were significantly closer to anatomic correlate sites when pacing was performed over normal myocardium (1.6 ± 1.4 cm) than when pacing over scar (3.0 ± 1.6 cm) or scar-margin sites (4.6 ± 2.0 cm, p = 0.0004).

4. Discussion and conclusions

This study takes advantage of clinically indicated closed-chest catheter mapping of the epicardial surface to record BSPMs during pacing from multiple epicardial sites. A pre-procedure CT scan registered with the

electroanatomic (point-by-point catheter) map was used to derive a customized cardiac and torso geometry. Sites of inferred early minima were identified from the inverse solution map and compared with the known site of pacing.

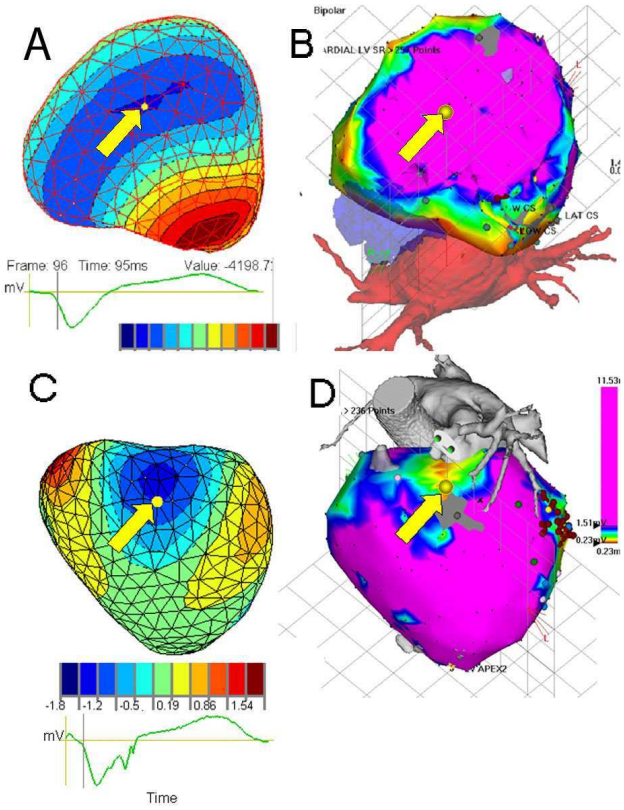

Figure 1: Correlation of inverse solution electrocardiographic mapping and electroanatomic mapping. A: Epicardial potential map obtained by inverse solution for patient 2 early within the QRS complex; a potential minimum (arrow) is seen in the mid-inferior wall, immediately below the septum. This correlates closely with the anatomic identification of the pacing site (arrow, Panel B) derived from the electroanatomic map registered to the CT scan. C: Epicardial potential map for patient 1 early within the QRS complex. An early potential minimum at the basal anterior LV is seen. This correlates closely with the anatomic identification of the pacing site (Panel D, arrow). Panels B & D demonstrate electroanatomic maps of bipolar signal amplitude over the epicardium; signals >1.5 mV are displayed in purple; the colour scale shows lower amplitude signals, and gray represents unexcitable scar.

Inverse solution mapping most accurately identified pacing sites when pacing was within areas of normal signal amplitude. As expected, discrepancy was introduced when pacing within scar or the scar border zone in association with Stim-QRS delay. Stim-QRS delay may result when pacing captures a protected channel within myocardial scar, and would be expected to result in myocardial activation remote from the pacing site. The relatively good correlation obtained in this study when capturing normal myocardium suggests that inverse solution mapping is a feasible method for identification of epicardial events using single-beat mapping.

Several potential mechanisms may explain discrepancies between pacing sites identified by either method. Error in registration of the electroanatomic map with the 3D reconstruction of the epicardial surface using the CT scan could introduce a systematic error into the comparison. Likewise, it is possible that inaccuracies in identification of pacing sites using electroanatomic mapping could have contributed to mismatch. The electroanatomic map has been reported to have accuracy within 1 mm [17], but significant error may be introduced by cardiac movement, including respiration, which can introduce an error of several centimeters. This error was minimized by observation of the catheter position for stability during pacing. Other possible sources of error include potential variation in lead position. Body-surface leads were applied according to a protocol using bony landmarks. For logistic reasons, we were unable to perform the CT scan with the body-surface leads in position. This potential error was attenuated by careful attention to bony landmarks, and matching of the CT-derived landmarks with lead positions in the solution.

This study was performed during epicardial catheter mapping and pacing. This results in a relatively easily interpreted inverse solution, since myocardial activation propagates away from the site of pacing, and the site of earliest activation is represented by an early minimum potential. Most catheter ablation procedures for ablation of VT, however, are performed from the endocardium, and this may limit interpretation of inverse solution maps.

Currently, inverse solution mapping requires extensive signal processing, and time-consuming generation of customized geometry. If these tasks could be completed in real time during catheter ablation, it is possible that this technique could have clinical utility to assist in cardiac mapping during catheter ablation procedures.

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