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Noninvasive electrocardiographic imaging of chronic myocardial infarct scar☆,☆☆

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Abstract

Background: Myocardial infarction (MI) scar constitutes a substrate for ventricular tachycardia (VT), and an accurate delineation of infarct scar may help to identify reentrant circuits and thus facilitate catheter ablation. One of the recent advancements in characterization of a VT substrate is its *volumetric* delineation within the ventricular wall by noninvasive electrocardiographic imaging. This paper compares, in four specific cases, epicardial and volumetric inverse solutions, using magnetic resonance imaging (MRI) with late gadolinium enhancement as a gold standard.

Methods: For patients with chronic MI, who presented at Glasgow Western Infirmary, delayed-enhancement MRI and 120-lead body surface potential mapping (BSPM) data were acquired and 4 selected cases were later made available to a wider community as part of the 2007 PhysioNet/Computers in Cardiology Challenge. These data were used to perform patient-specific inverse solutions for epicardial electrograms and morphology-based criteria were applied to delineate infarct scar on the epicardial *surface*. Later, the Rochester group analyzed the same data by means of a novel inverse solution for reconstructing intramural transmembrane potentials, to delineate infarct scar in three dimensions. Comparison of the performance of three specific inverse-solution algorithms is presented here, using scores based on the 17-segment ventricular division scheme recommended by the American Heart Association.

Results: The noninvasive methods delineating infarct scar as three-dimensional (3D) intramural distribution of transmembrane action potentials outperform estimates providing scar delineation on the epicardial surface in all scores used for comparison. In particular, the extent of infarct scar (its percentage mass relative to the total ventricular mass) is rendered more accurately by the 3D estimate. Moreover, the volumetric rendition of scar border provides better clues to potential targets for catheter ablation.

Conclusions: Electrocardiographic inverse solution providing transmural distribution of ventricular action potentials is a promising tool for noninvasively delineating the extent and location of chronic MI scar. Further validation on a larger data set with detailed gold-standard data is needed to confirm observations reported in this study.

Keywords: Electrocardiography; Myocardial infarction; Inverse solution

Introduction

Chronic myocardial infarct scar can be detected, and its location and extent can be determined, by means of a variety of invasive and non-invasive methods, including standard 12-lead electrocardiography [1,2] and cardiac magnetic resonance imaging [3]. Scarring from previous myocardial infarction (MI) constitutes a substrate for ventricular arrhythmias; in patients with postinfarction ventricular tachycardia (VT), accurate delineation of myocardial infarct scar facilitates catheter ablation [4–7].

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For many years electrocardiography has been widely used for detecting and assessing both acute and chronic MIs. In particular, modern electrocardiographic imaging technology has emerged as a promising noninvasive approach to mapping the electric activity of the heart and to delineating arrhythmogenic substrates in humans [8]. The present paper reviews recent collaborative studies of the Dalhousie and Rochester groups that are aimed at advancing the ability of noninvasive electrocardiographic imaging to delineate arrhythmogenic substrates. The studies reported here use a common set of electrocardiographic and gold-standard data, providing material for assessing merits and limitations of different algorithms.

Electrocardiographic assessment of myocardial infarction

It is difficult to obtain detailed information on the extent and location of the damaged myocardium from the standard ECG. Once the repolarization abnormalities (ST-segment and T-wave changes) subside after an acute infarction resolves, the Q waves and a fragmented QRS remain as the only recognized signs of infarction. A fragmented QRS is defined by the presence of an additional R wave or notching in the S wave, or the presence of several additional R waves (fragmentation) in two contiguous leads corresponding to a major coronary artery territory [9].

Various algorithms have been proposed to define location and extent of MI necrosis from the 12-lead ECG. One notable approach to addressing this problem is the Selvester QRS scoring system [1,10], developed with the aid of a computer model of ventricular activation. The Selvester scoring uses amplitude and duration criteria derived from the QRS complexes in 10 of the standard 12 ECG leads (I, II, aVL, aVF, and V1–V6) for a total of 31 points, each equivalent to the necrosis of approximately 3% of the LV mass. The Selvester scoring system was automated in our Dalhousie lab [11] to facilitate its clinical use. Until recently, myocardial necrosis has been assessed by the Selvester scoring only in the absence of QRS confounders (e.g., bundle branch block, left ventricular hypertrophy, and Wolff–Parkinson–White syndrome). These "no-confounder" criteria have been updated to accommodate cases with hypertrophy and conduction defects [12,13].

MRI imaging of myocardial infarction

Delayed contrast enhancement magnetic resonance imaging (DCE-MRI) is very effective in determining the presence, location, and extent of MI, in both the acute and chronic stages. This imaging technique works by injecting the subject with an enhancement agent, such as gadolinium, and acquiring a scan within 10–20 minutes after injection. Damaged tissue with poor blood perfusion takes a longer time to absorb the contrast agent and is seen as enhanced areas on delayed scanning. An important advantage of the DCE-MRI is that its high spatial resolution allows accurate delineation of 3D transmural infarcted myocardium [14,15].

Methods

Patients

Four patients, who initially presented with acute MI at Glasgow Western Infirmary, without hypertrophy or bundle branch block, were selected for this study from a cohort of 38 patients; all patients provided written informed consent. Each patient underwent clinical assessment, a cardiac MRI for reconstructing ventricular geometry within the chest, a DCE-MRI for 3D delineation of the infarcted region, and 120-lead body-surface potential mapping (BSPM) according to the Dalhousie protocol [16]. Both DCE-MRI and BSPM used in this study were performed at the chronic stage of MI, 6 months after the acute event.

Assessment by noninvasive electrocardiographic imaging

ECG data were recorded from 120 locations on the torso sampled at 1 kHz [16], using a Mark 6 acquisition system (BioSemi, Amsterdam, The Netherlands). Off-line processing produced an averaged complex for each lead from the 15-second recording. Faulty leads were identified and a 3D interpolation algorithm [17] produced body-surface potentials at 352 nodes of the Dalhousie standard torso. Inverse solutions were obtained by three different methods that calculated epicardial electrograms or volumetric transmembrane potentials (TMP). Individual inverse solutions used patient-specific anatomy derived from MRI data; at the Dalhousie lab, models of the epicardial surface and of the body surface with positions of electrodes were extracted using Amira 4.1 software (Mercury Computer Systems, Chelmsford, MA); at the Rochester lab, volumetric biventricle models of the heart were constructed by custom-written Matlab (Mathworks Inc., Natick, MA) routines. In both labs, all data processing and analysis were performed by custom-written Matlab routines.

The first method of inverse solution, used by the Dalhousie group, calculates *epicardial potential distributions* from ECG data and patient-specific anatomical data [18]. Briefly, transfer coefficients relating potentials on the epicardial surface to measured body-surface potentials are calculated by using the Radon formula for numerical quadrature and then epicardial electrograms are calculated, instant by instant, from body-surface potential distributions by solving the inverse problem using Tikhonov second-order regularization, with the regularization parameter estimated by the *L*-curve method [19].

The second method of the inverse solution—referred to as noninvasive transmural electrophysiological imaging (TEPI)—was introduced by the Rochester group of Wang et al. [20]. This method involves patient-specific reconstruction of TMP dynamics inside 3D ventricular myocardium by means of the Bayesian estimation, where the priors are generated by a Monte-Carlo type simulation of a physiological model of cardiac electrical activity to constrain the TMP solution. The TEPI method is applied to the entire sequence of ECG data, and produces as a solution the spatiotemporal activity of TMP throughout the myocardium. This method was subsequently applied, by the same group, as an imaging