Epicardial Isochrones from a New High-Frequency ECG Imaging Technique

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Abstract

The aim of this study is to compare epicardial isochrone maps (EIM) derived from electrocardiographic (ECG) imaging with a new technique based on high-frequency ECG isochrone maps (HFEIM) computation.

We analyzed three subjects – normal, left bundle branch block (LBBB) and right bundle branch block (RBBB). Body surface potentials were measured: 5-minute supine, 2KHz sampling, 184 electrodes. These potentials were used for inverse reconstruction of EIM using patient-specific torso-heart geometry (CT). HFEIM was determined as follows: averaged body surface QRS amplitude envelopes 150–400 Hz (HFQRS) were projected onto the epicardium, the time delay from the onset of the QRS complex to centers of mass of projected HFQRS was computed.

The EIM and HFEIM pattern of electrical activation was similar, especially for LBBB and RBBB subjects. The correlation between EIM and HFEIM activation times was 0.42, 0.82 and 0.83 for the NORMAL, LBBB and RBBB subjects respectively. Maximal dyssynchrony was about 40 ms lower for HFEIM than for EIM.

EIM and HFEIM provide comparable distribution of electrical delays but different reference values. Lower HFEIM dyssynchrony may reflect the electrical activation in an entire ventricular wall segment and may better correlate with local electro-mechanical function.

1. Introduction

Electrocardiographic imaging (ECGI) is a non-invasive cardiac electrical procedure for imaging epicardial potentials and electrical activation times (isochrones) using inverse reconstruction from body surface electrograms (ECGs) [1,2].

Epicardial isochrone maps (EIMs) provide information about the activation timing of ventricular regions with high value for cardiac resynchronization therapy and arrhythmias.

However, EIMs represent activation of the epicardial surface only [3]. The purpose of this study is to compare EIM with a new technique based on isochrone computation from high-frequency (HF) ECG components projected onto the epicardium (HFEIM). Those HF components contain information on the depolarization wavefront across the ventricular walls.

2. Data recording

We compared three subjects – normal healthy (N), left bundle branch block (LBBB) and right bundle branch block (RBBB). Body surface potentials were recorded from 184 sites around the patients' torso using BioSemi (Amsterdam, the Netherlands) hardware. Measurements were performed supine over 5 minutes with a sampling rate of 2 kHz and frequency range up to 400 Hz. A thoracic computed tomography (CT) scan was performed with the electrodes attached to the patient.

3. Methods

3.1. EIM computation

The body surface potentials and CT images were processed to reconstruct patient-specific epicardial unipolar electrocardiograms at 2100 virtual points [3]. Depolarization times were determined as the time delay from the onset of the QRS complex and the maximal negative deflection of the potentials within the QRS complex [3].

3.2. High-frequency epicardial activation time computation (HFEIM)

V1-V6 QRS complexes were detected and clustered by morphology [4,8]. The time position of sinus (dominant) rhythm QRS complexes were used for further averaging.

The amplitude envelopes of the signals of all 184 electrodes were computed using the Hilbert transform in five frequency bands starting at 100–200 Hz and ending at 300–400 Hz with a step of 50 Hz. The averaged QRS amplitude envelopes were computed for each frequency band. Averaged envelopes were smoothed using a 0–40 Hz passband filter. High-frequency body surface QRS (HFQRS) was determined as the mean of normalized averaged envelopes over five frequency bands. The single-band HFQRS calculation procedure is detailed in [5] and multi-band computation is reported in [6].

Body surface HFQRS were projected onto the epicardium through the heart geometry center. Each epicardial virtual point was computed as a weighted average of 3 body surface electrodes. The virtual points generated for the inverse reconstruction were used for HF projection location.

The HF depolarization activation times of a single virtual point on the epicardium were determined as the time delay from the QRS onset (estimated from precordial leads) to the center of mass of HFQRS above the 50 percent threshold of baseline to peak magnitude – Figure 1. HF depolarization activation times in each virtual point create an HFEIM – Figure 2.

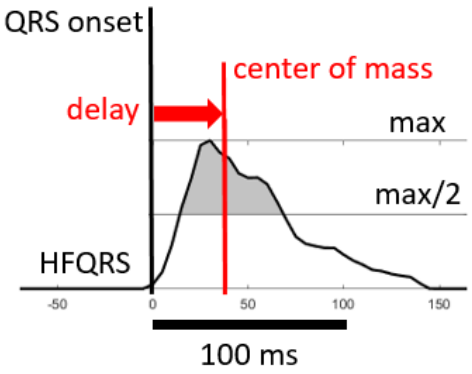

Figure 1. A single virtual point HFQRS time position is defined as the time distance between QRS onset (black vertical line) and virtual point HFQRS center of mass (red vertical line). Center of mass was computed in a signal above the threshold defined as half of the maximal value of the signal (gray area).

4. Results

For each patient we computed EIM and HFEIM and maximal dyssynchrony as total activation time (TAT, HFTAT) in milliseconds. Figure 3 presents the comparison between EIM and HFEIM. Figure 4 compares scatter plots and histograms of delay distribution.

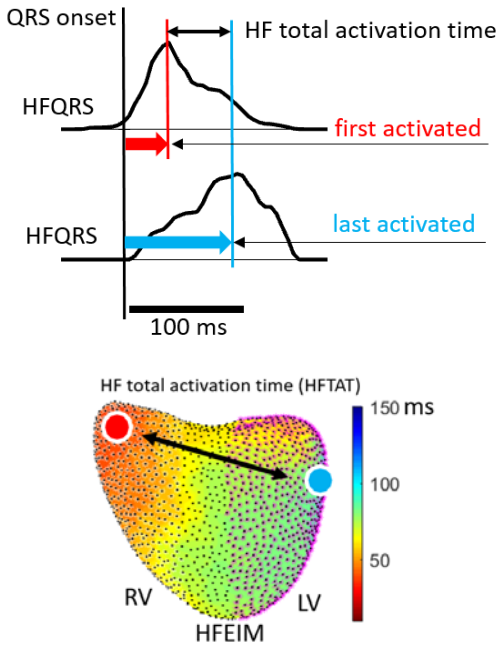

Figure 2. High-frequency ECG isochrone map (HFEIM) computation. Upper panel: delays of two (first and last activated) virtual points. Bottom panel: HFEIM of the LBBB patient. The color of each virtual point corresponds to its delay. Maximal difference between delays is marked as HF total activation time (HFTAT). Anterior view, LV – left ventricle, RV – right ventricle.

The pattern of electrical activation created by EIM and HFEIM per subject was similar. The linear Pearson correlation coefficient R between EIM and HFEIM activation times was R = 0.42, 0.82 and 0.83 for the NORMAL, LBBB and RBBB subject, respectively, $p<0.001$, example shown in Figure 4. The maximal HFEIM dyssynchrony was about 40 ms lower than maximal EIM dyssynchrony: TAT was 77, 96, -121 ms and HFTAT 27, 57 and -78 ms (the negative sign for RBBB means that RV was activated later than LV). QRS duration was 82, 138 and 160 ms.

5. Discussion

The results show that RBBB and LBBB EIM and HFEIM correlate and the time-spatial distribution of images provides comparable information. Importantly, there are differences that point to the distinct nature of the information.

1: While the activation sequence is similar in TAT and HFTAT, absolute values show lower HFEIM delays. The biggest difference is in a normal heart with a low

correlation between EIM and HFEIM, where HFTAT represents only 35 percent of the TAT value, while a TAT of 77 ms is comparable with the QRS duration 82 ms. TAT reflects the total activation time on the epicardium. In contrast, the HFTAT value of 27 ms indicates the small dyssynchrony between transmural ventricular segments. For LBBB and RBBB, the HFTAT is about 40 ms lower than TAT, but the correlation is high due to added LBBB and RBBB dyssynchrony.

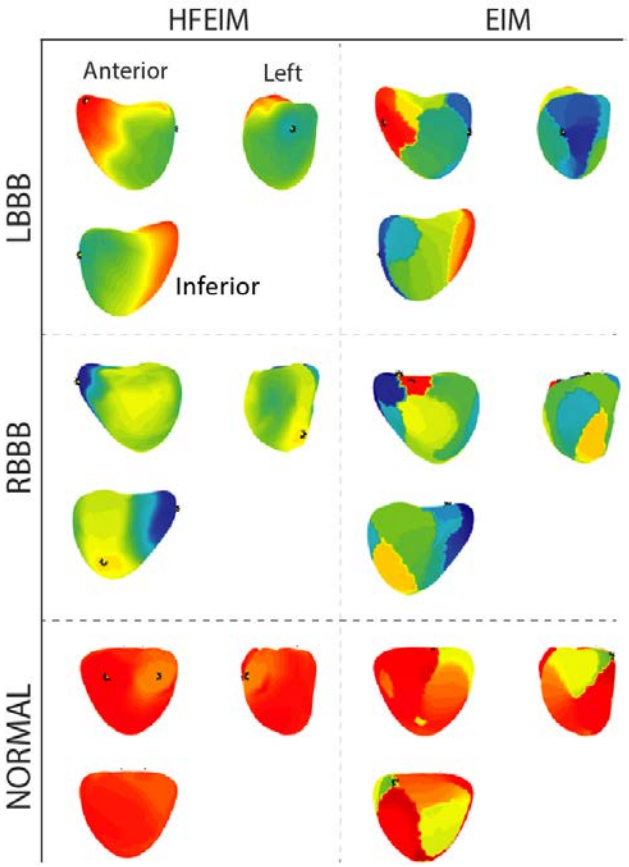

Figure 3. Epicardial isochrone maps (EIMs) and high-frequency EIM (HFEIMs) in patients with left bundle branch block (LBBB), right bundle branch block (RBBB) and with synchronous heart. Each map is shown from three projections (anterior, inferior and left). Dark red – 0 ms, dark blue – 150 ms.

2: EIM provides maps with sharp color edges. This feature is shown in Figure 4 for the RBBB patient. The scatter plot and histograms in the bottom part of the figure demonstrate the distribution of the delays in virtual points. This clearly shows the clustering of the epicardial delays in EIM – peaks in the histograms.

These two differences can be explained by the methods determining the delay related to QRS onset. In the case of EIM, the time position of the maximum negative derivative of epicardial potential is used [3]. The reconstructed epicardial potential morphology significantly affects the measured delays. The time position of the maximum of the negative derivative of the epicardial potentials creates large areas of the same value and the transitions between the areas are steep. This leads to the appearance of monochromatic areas in the EIM [7].

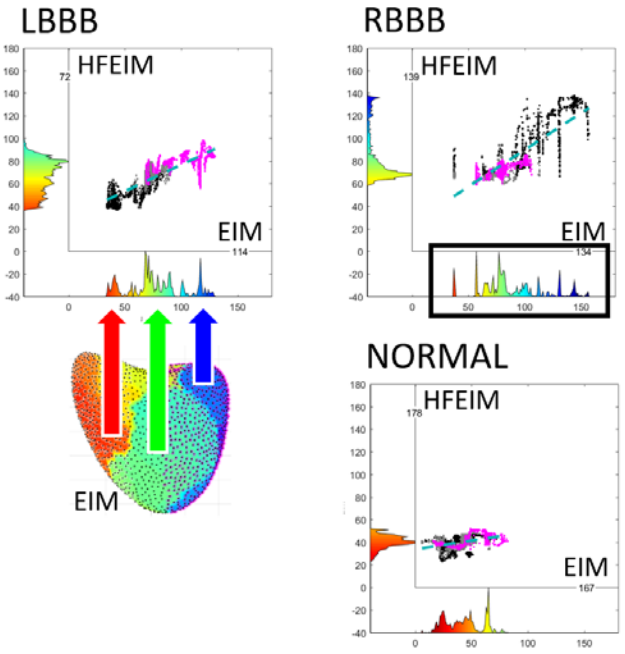

Figure 4. Scatter plots demonstrating the association between HFEIM vs. EIM delay for every virtual point. Black points correspond to the right ventricle (RV), violet points correspond to the left ventricle (LV) and gray points correspond to the middle area between LV and RV. The blue dashed line defines a linear regression slope. The scatter plots simultaneously include histograms of the virtual points' distribution in time. The time scale for both the EIM and HFEIM is 0–180 ms. Scatter plots and histograms show clustering of epicardial delays in EIM (peaks in the histogram). Clustering is caused by sharp color edges in EIMs. This EIM phenomenon is most striking in the RBBB patient (black rectangle).

HFQRS morphology provides information about the time-spatial distribution of the depolarization activation – depolarization wavefront. It was demonstrated in [5] that HF components acquired from the body surface consist mainly of a steep change of current and voltage during phase 0 of the myocardial action potential. The HF component properties are different from low-frequency (LF) potentials. The LF potentials form a main electrical vector and its projection to body surface electrode creates ECG morphology including its negativity and positivity.

We assume that HFQRS covers the temporal and spatial distribution of depolarization in the volume of RV and LV, rather than the epicardium alone from EIM. The resulting HFQRS shape is the sum of HF potentials, whose contribution is determined mainly by the distance of the activated myocardial cells from the electrode and the number (volume) of the activated cells at a given time.

In this study we use the direct projection of body surface HFQRS (3 electrodes mean) onto the epicardium (single virtual point). Body surface HFQRS is the sum of HF signals from many sources (depolarization wavefront), not just from a single point on the epicardium. The projected HF potential onto a virtual epicardial point does not, therefore, represent HF oscillations from this virtual point only, but a sum of HF oscillations from ventricular volumes contributing to the originated body surface electrode.

5. Conclusion

We can assume that lower HFEIM delays based on the depolarization activation center of mass computation in comparison to the maximal negative slope (EIM) reflect more physiologically the electrical activation distribution within the ventricles. The dyssynchrony computed from HFEIM could probably better correspond to the electrical activation abnormalities throughout the ventricular volume.

HFEIM is a supplementary technique to EIM, not an alternative computational technique. The HFEIM solution exploits a new source of information which is related to the high-frequency signals originated in the depolarization wavefront. Further studies will be required to determine the appropriate method to display HF components measured on body surface electrode arrays.

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