Body Surface Electrical Activation Delay Computation From High-Frequency ECGs

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Abstract

The ventricular electrical activation delay is a valuable piece of information in cardiac resynchronization therapy and arrhythmias. The purpose of this study is to introduce electrical activation delay obtained from high-frequency body surface maps (HFBSM).

We studied seven patients – three normal (N), three left bundle branch block (LBBB) and one right bundle branch block (RBBB). The amplitude envelopes of the QRS complex in each lead were computed in a frequency band of 100–400 Hz and were averaged (HFQRS). Activation times were defined as the delay from the beginning of the QRS complex to the center of mass of HFQRS, and activation (isochrone) maps were produced. Three different methods of electrical dyssynchrony computation were chosen: maximal activation time difference from the whole surface (HFTAT), delay between lead V1 and V6 (VED) on the 12-lead ECG, and standard deviation of activation times from all electrodes (SDAT).

For N/LBBB/RBBB patients, the mean values were: SDAT 3.2/18.3/20.6 ms, VED 3/54/-65 ms, HFTAT 18/71/-83 ms and QRSd 83/141/160 ms.

An HFBSM allows easy calculation of HFTAT and SDAT directly from the torso surface. VED provides directional distinction between LBBB and RBBB.

1. Introduction

Cardiac resynchronization therapy (CRT) improves outcomes in heart failure patients with reduced left ventricular (LV) ejection fraction and conduction abnormalities, thereby reducing mortality and morbidity [1, 2]. The recommended selection criteria are currently based on a 12-lead electrocardiogram (ECG): prolonged QRS duration and morphology. However, approximately one third of patients show no response to CRT, probably due to insufficient electrical dyssynchrony prior to CRT or persistent electrical dyssynchrony afterwards [3]. This points to an inadequate ability to detect electrical dyssynchrony using QRS-duration and morphology.

Technology for assessing electrical activation patterns in ventricular dyssynchrony from high-frequency (HF) ECGs has been introduced and has shown that high-frequency components in a QRS complex region can provide information about the temporal-spatial distribution of ventricular depolarization [4]. The diagnostic contribution of HF of QRS complexes (HFQRS) was verified in the MADIT-CRT trial (Multicenter Automatic Defibrillator Implantation – Cardiac Resynchronization Therapy) and an optimized computerized approach measuring ventricular electrical activation delay (VED) from body surface 12-lead ECGs was introduced [5, 6].

The aim of this work was to introduce time-varying distribution of the HFQRS over the whole torso surface and parameters defining VED obtained from high-frequency body surface maps (HFBSM).

2. Method

2.1. Data recording and subjects

Body surface potential data was collected from two centers. Three subjects are from Maastricht University. Body surface potentials were recorded from 184 sites over the entire surface of the human torso with BioSemi (Amsterdam, the Netherlands) hardware. The average duration of supine and resting measurement was 5 minutes, the sampling rate was 2 kHz and the frequency range was up to 400 Hz.

Another four subjects were taken at the International Clinical Research Center at St. Anne's University Hospital, Brno, Czech Republic. 96-lead ECG was collected at 5 kHz with a high dynamic range and a 2 kHz pass band (M&I Prague, CZ). The average duration of supine and resting measurement was 5 minutes.

There were seven patients in the study: three normal (N), three left bundle branch block (LBBB) and one right bundle branch block (RBBB).

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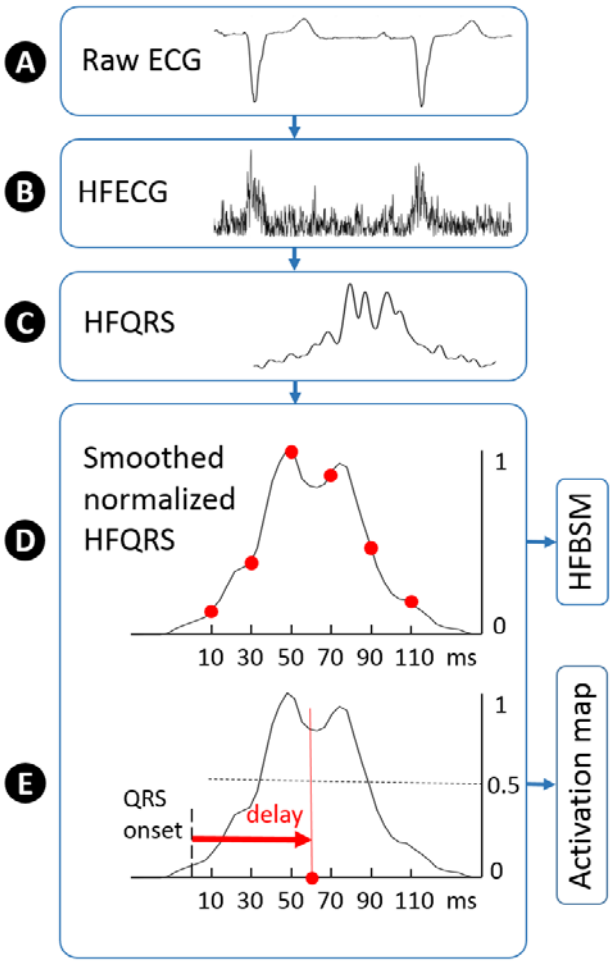

Figure 1. Computation of the high-frequency body surface maps. The raw ECG signal (A) is transformed into an amplitude envelope in the frequency range 100–400 Hz (B); averaging of QRS complexes is used (C) and HFQRS is smoothed and normalized (D, E). HFQRS can be sampled (in this example at 20 ms interval) to plot the HFBSM (D). The activation time is defined as the delay from the beginning of the QRS complex to the center of mass above the 50 percent threshold of HFQRS (E). This process is applied to each chest lead.

2.2. ECG signal transformation

QRS complexes were detected and sorted into categories using a robust multichannel approach. This technique was used to distinguish a regular rhythm from abnormal QRS shapes to focus the analysis primarily on the sinus (dominant) rhythm.

High-frequency ECG (HFECG) was calculated for each lead as follows. Signals from the lead are transformed into amplitude envelopes in five frequency ranges (100–200, 150–250, 200–300, 250–350 and 300–400 Hz) using fast Fourier and Hilbert transforms (Fig. 1 A, B). A total of 5 amplitude envelopes are obtained on which signal averaging is applied.

Due to the low signal-to-noise ratio of amplitude envelopes, the signal averaging of QRS complexes is used. HFQRS was calculated as the median shape of all QRS complexes from HFECG.

In the last step, HFQRS was smoothed. Each point of the HFQRS curve was computed as the median of its 10 ms surroundings. HFQRS was normalized between 0 and 1 for each lead separately.

2.3. High-frequency body surface maps

HF body surface potential measurements (HFBSM) show the time-varying distribution of the HFQRS on the whole torso surface. For any time position HFQRS values from all leads can be measured and plotted to the body surface as one frame of HFBSM. HFQRS can be sampled at a regular time interval, as shown in Figure 1D. This creates a series of frames that form an HFBSM. Figure 2 shows an HFBSM for an LBBB patient.

Activation times were defined as a delay from the beginning of the QRS complex to the center of mass above the 50 percent threshold of HFQRS – Figure 1E. These activation times were mapped at body surface nodes to construct an HFBSM activation map.

Data for electrodes with an artificial signal or low signal-to-noise ratio were replaced by the weighted average of the four nearest body surface electrodes.

2.4. HFBSM-derived markers for dyssynchrony

Electrical dyssynchrony on a high-frequency body surface activation map was quantified by the following parameters: ventricular electrical activation delay (VED) and high-frequency total activation time (HFTAT). VED was measured from body surface 12-lead ECGs as the delay between lead V1 and V6. HFTAT was measured from HFBSM as the maximal activation time difference from the whole surface.

Body surface electrode arrays were divided into two parts, the left and the right side, by a plane passing through the longitudinal axis of the chest. If activation occurred first on the right side, the HFTAT takes a positive sign; if activation occurred first on the left side, the HFTAT takes a negative sign. The sign of VED and HFTAT shows which ventricle was activated first. A positive sign means that the right ventricle was activated before the left ventricle. A negative sign means that the left ventricle was activated before the right ventricle.

The standard deviation of activation times (SDAT) quantifies the heterogeneity of electrical activation. SDAT was computed as the standard deviation of activation times from all BSPM electrodes.

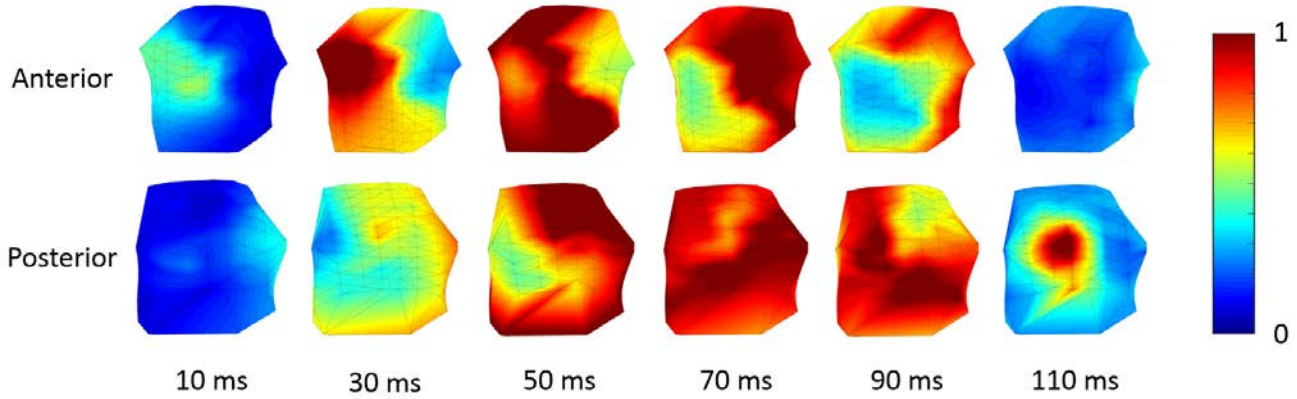

Figure 2. High-frequency body surface maps (HFBSM) of one left bundle branch block (LBBB) patient. Anterior and posterior view. The figure shows the time-varying distribution of high-frequency components over the whole torso surface during QRS complex duration. High-frequency components were normalized between 0 and 1 for each lead separately. The red color represents the activated part of the torso in a specific time position. Earlier activation of the anterior region and late activation of the posterior region is typical for LBBB patients.

3. Results

HFBSM and activation maps demonstrated the varying dyssynchrony of electrical activation across different pathologies. Figure 4 compares HFBSM activation maps for N, LBBB and RBBB patients. In LBBB patients there is a similar pattern: early, rapid activation of the anterior region (right ventricle) and late activated posterior region (left ventricle) – Figure 2. Maps for the RBBB patient have the opposite pattern than that for LBBB. Maps for both LBBB and RBBB show that the whole torso was never activated at the same time. In contrast to LBBB and RBBB, the whole torso was activated at nearly the same time for all subjects with a normal synchronous heart. Only in the area around the V2 and V3 leads was activation slightly earlier – Figure 4.

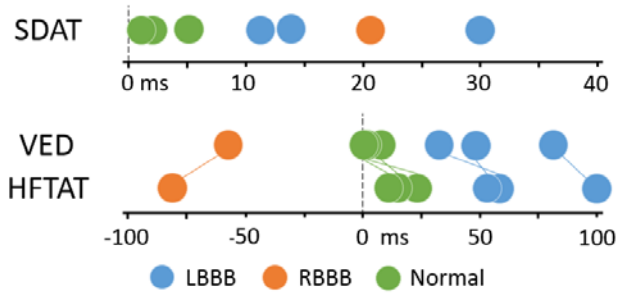

Figure 3: Distribution of the standard deviation of activation times (SDAT), ventricular electrical activation delay (VED) and high-frequency total activation time (HFTAT) value for LBBB (blue), RBBB (orange) and normal (green) patients. The sign of VED and HFTAT shows which ventricle was activated first. A negative sign means that the left ventricle was activated before the right ventricle.

For N/LBBB/RBBB, the mean values of SDAT 3/18/21 ms, VED 3/54/-65 ms, HFTAT 18/71/-83 ms and QRSd 83/141/160 ms were computed. Parameters VED, HFTAT and SDAT differ between all groups of subjects. Figure 3 shows the distribution of all parameters for all patients.

4. Discussion

In this study we describe a new methodology for computation of body surface activation maps and quantification of electrical dyssynchrony.

The high-frequency dyssynchrony represents a clearly interpretable numerical parameter. Nevertheless, the graphical inspection is irreplaceable and offers important information about the time-spatial distribution of activation.

The “gold standard” for measuring electrical activation non-invasively in humans is electrocardiographic imaging (ECGi). Body surface potential measurements, a heart-torso CT, and inverse epicardial potential reconstruction provide detailed information on epicardial activation [7]. Similarly to ECGi, this high-frequency technique records anterior and posterior torso body surface electrograms. However, the advantages of the HFBSM technique over ECGi are ease of use and no need for CT scans. On the other hand, there are technical limitations: a higher sampling rate, frequency range and the dynamics of the ECG devices are required.

The ability of body surface mapping using a high frequency to image the ventricular electrical activation dyssynchrony could be used to facilitate proper patient selection for CRT and potentially to guide LV lead placement. High-frequency activation maps provide

information on electrical wavefront propagation that can be helpful in understanding the mechanisms contributing to improved electrical dyssynchrony under different pacing conditions and provide a physiological approach to optimizing CRT programming.

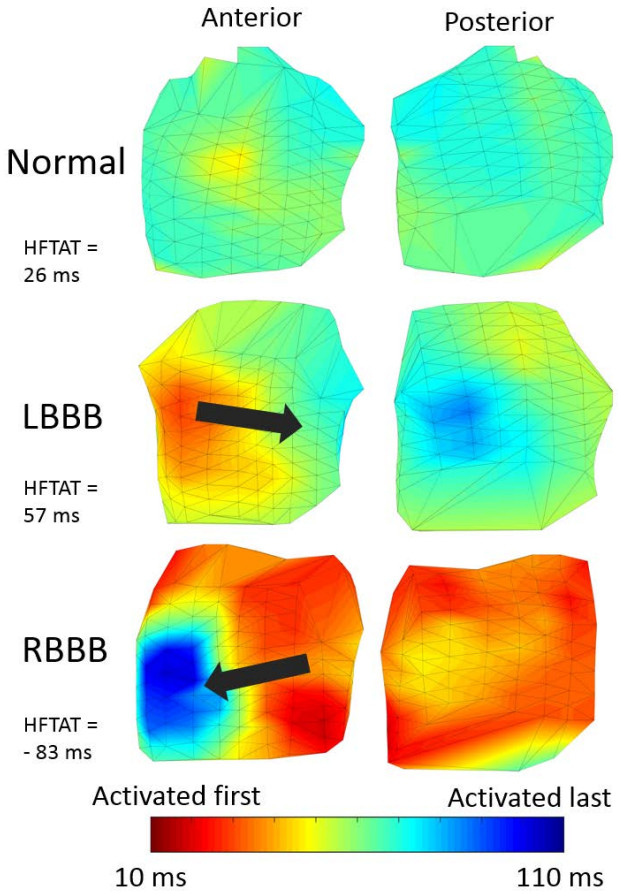

Figure 4. Activation maps using high-frequency ECG. From the top: normal synchronous heart, left bundle branch block (LBBB) patient and right bundle branch block (RBBB) patient. Each map is shown from the anterior and posterior view.

5. Conclusion

HFBSM allows easy calculation of ventricular electrical delays directly from the torso surface. Electrode reduction and significant simplification by the use of 12-lead ECG provides a lower value of dyssynchrony. However, the division into normal, LBBB and RBBB remains differentiated. Studies with more subjects will be required to determine the appropriate additional value of complete torso maps in comparison with 12-lead ECG.

Acknowledgements

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