

# Analysis of Body Surface Potential Maps in Cardiac Resynchronization Therapy

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## Abstract

*Cardiac resynchronization therapy (CRT) aims at improving systolic function in patients with heart failure. We investigated the use of body surface potential mapping (BSPM) in the understanding of the activation sequence in the myocardium during biventricular (BiV) pacing.*

*We acquired BSPM recordings in 29 patients with an implanted BiV device during normal sinus rhythm, right ventricular (RV) pacing, left ventricular (LV) pacing and BiV pacing. We compared potential maps during BiV pacing to those during RV pacing and LV pacing by a 2-D correlation.*

*By studying BSPM maps, we observed that 38% of our patients presented potential maps similar to those observable when pacing with a single lead. These patients may not be taking full advantage of the potential benefits of CRT.*

## 1. Introduction

Cardiac resynchronization therapy (CRT) aims at improving systolic function in patients with heart failure [1]. Dyssynchrony in the contraction of left and right ventricles caused by abnormalities in the cardiac conduction in this group of patients can be reduced with the implantation of a pacemaker which stimulates both ventricles (biventricular pacing) [2]. In order to increase synchrony both ventricles can be paced either simultaneously or with some delay between them. Restoration of contraction synchrony has been shown to be beneficial to patient's quality of life and mortality [3]. However, trials of long-term therapy have shown that response to CRT varies among individuals and at least 20% to 30% of patients are nonresponders [4].

Determination of the optimal interventricular delay for each patient has been shown to increase synchrony and thus improve patient's cardiac function [5]. Another critical question in CRT therapy is the identification of patients that will benefit from CRT. One of the most extended inclusion criteria for CRT is the duration of the QRS complex. A wide QRS complex is associated with a

conduction disturbance and a subsequent lost in synchrony. Although most patients with a wide QRS complex benefit from CRT, some of these patients are nonresponders. Also, a few patients with a narrow QRS can benefit from CRT if they present dyssynchrony in the contraction of both ventricles.

Further investigation in the optimal programming of the pacing delay of both ventricles may benefit the outcome of nonresponders. Recently, electrocardiographic imaging (ECGI) has been proposed as a new tool for assessing interventricular synchrony during CRT [6;7]. ECGI makes use of Body Surface Potential Mapping (BSPM) recordings which consist in the recording of electrocardiographic signals at many locations on the body surface [6;7]. Additionally, it requires precise information about the geometry of the patient that can be obtained from a computer tomography scan. Although ECGI appears as an interesting tool for the individual optimization of the programmed interventricular delay in biventricular (BiV) devices, the requirement of a CT image and an intensive computing workload that supposes the resolution of the inverse problem for every patient, are some of the obstacles for its implementation in clinical practice.

The objective of the present study is the analysis of body surface potential maps for a better understanding of the activation sequence in the myocardium during BiV pacing. We studied surface maps during BiV pacing and compared these maps to those observable during lone right ventricular (RV) pacing and left ventricular (LV) pacing. We hypothesized that body surface maps during correct BiV pacing should differ from those during RV only or LV only pacing. In case a BiV stimulation pattern resembles an LV or RV pattern, little benefit is expected from that obtained with a single-chamber pacemaker.

## 2. Methods

Body Surface Potential mapping recordings were performed on 29 patients with implanted CRT devices at Montreal Heart Institute. We made use of an ActiveTwo acquisition system (Biosemi, Amsterdam, The Netherlands) with a sampling frequency of 2048 Hz and a

quantization of 24 bits. Sixty-seven electrodes were attached to the patient's body by using an elastic vest specifically designed for BSPM applications. One-minute recordings were acquired during normal sinus rhythm, RV pacing, LV pacing and BiV pacing in 20 patients (Group 1). Delay in the activation of both ventricles was determined for each patient in order to improve their ventricular function. There was no programmed delay in the activation of both ventricles in 17 patients while in 3 patients the left ventricle was activated first (+30 ms, +35 ms and +45ms). In 9 patients (Group 2) recordings were acquired during normal sinus rhythm, RV pacing, LV pacing, and BiV with varying activation delays: -70 ms, -60 ms, -40 ms, -20 ms, 0 ms, +20 ms, +40 ms, +60 ms and +70 ms, where a positive delay time refers to an earlier LV activation.

Tracings were visually inspected during acquisition and processed off-line using Matlab 7.0 (The Mathworks, Natick, Massachusetts, USA). Thirty seconds of each activation mode were analyzed avoiding the use of the first and last 15 seconds of the recording. ECG signals were high-pass filtered ( $F_c = 0.7$  Hz) in order to reduce baseline wandering. An averaged cardiac cycle was calculated by template-matching averaging (correlation coefficient threshold equal to 0.97). Time intervals corresponding to pacemaker spikes were manually selected and samples belonging to these time intervals were interpolated by cubic spline interpolation. QRS onset and offset points were semi-automatically detected in averaged cardiac signals free from pacemaker artifact. QRS duration was defined as starting in the QRS onset found in the 67 leads and ending in the last QRS offset found in all leads. All ECG signals were visually inspected and those leads that presented noticeable noise were discarded and replaced by an interpolated version using neighboring leads. No patient presented more than 5 discarded leads.

Twenty BSPM maps were computed equally distributed along the QRS complex. Eighteen surface maps (discarding the first and last maps which correspond to the QRS onset and offset times) during to BiV pacing were compared to those obtained for native rhythm, RV pacing and LV pacing by a 2-D correlation. Mean correlation indexes during the QRS complex were computed for comparing BiV and RV pacing maps ( $\rho_{BR}$ ) and BiV and LV pacing maps ( $\rho_{BL}$ ). Dissimilarity index ( $\Delta\rho$ ) was computed as the absolute difference between  $\rho_{BR}$  and  $\rho_{BL}$ . BiV pacing maps were classified into three groups according to their computed mean correlations and dissimilarity indexes: RBiV (BiV maps similar to RV maps;  $\rho_{BR} > 0.7$  and  $\Delta\rho > 0.2$ ), LBiV (BiV maps similar to LV maps;  $\rho_{BL} > 0.7$  and  $\Delta\rho > 0.2$ ) or IBiV (otherwise). Comparisons between correlation indexes of BiV and LV

pacing and BiV and RV pacing were obtained with an independent sample t-test. Comparisons among QRS durations were performed with a paired sample t-test. A significance value of  $p < 0.01$  was considered.

### 3. Results

QRS duration in normal sinus rhythm in our patients was found to be  $146 \pm 32$ ms. Twelve patients (41%) presented a narrow QRS ( $< 140$ ms) while seventeen patients (59%) presented a wide QRS ( $> 140$  ms). QRS duration in 11 patients with a narrow QRS during sinus rhythm (92%) increased during BiV pacing. Widening of the QRS in patients with a narrow QRS was  $17 \pm 18$  % ( $p < 0.01$ ). QRS duration in 14 patients with a wide QRS during sinus rhythm (82%) decreased during BiV pacing. Shortening of the QRS in patients with a wide QRS was  $16 \pm 16$  % ( $p < 0.01$ ).

Distribution of patients into RBiV, LBiV and IBiV groups is summarized in Figure 1. BiV surface potential maps of 3 patients (10%) were classified into RBiV group. Although potential maps during BiV pacing were similar to maps during RV pacing in these three patients, the QRS was shortened 41% in the case of BiV pacing. BiV maps of 8 patients (28%) were classified into LBiV group. In these patients, QRS during BiV pacing was 8% narrower than during LV pacing. The remaining 18 patients (62%) were classified into IBiV group.

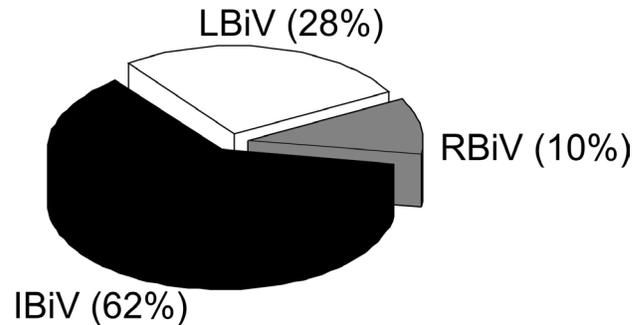


Figure 1: Distribution of patients according to the resemblance of their body surface potential maps during BiV stimulation with the stimulation of a single ventricle.

Correlation coefficients corresponding to BiV map comparison with LV pacing maps and RV pacing maps of three patients belonging to RBiV, LBiV and IBiV groups are shown in Figure 2. Patient in Figure 2.A showed a BiV activation patterns on the body surface similar to those caused by RV pacing ( $\rho_{BR} = 0.81$ ,  $\rho_{BL} = 0.07$ ,  $p < 0.01$ ). Patient in Figure 2.B showed a BiV activation pattern similar to activation pattern during LV pacing ( $\rho_{BR} = 0.40$ ,  $\rho_{BL} = 0.94$ ,  $p < 0.01$ ). Patient in Figure 2.C showed an intermediate BiV activation pattern ( $\rho_{BR} = 0.55$ ,  $\rho_{BL} = 0.73$ ,  $p = ns$ ).

## BiV vs Left and Right activations

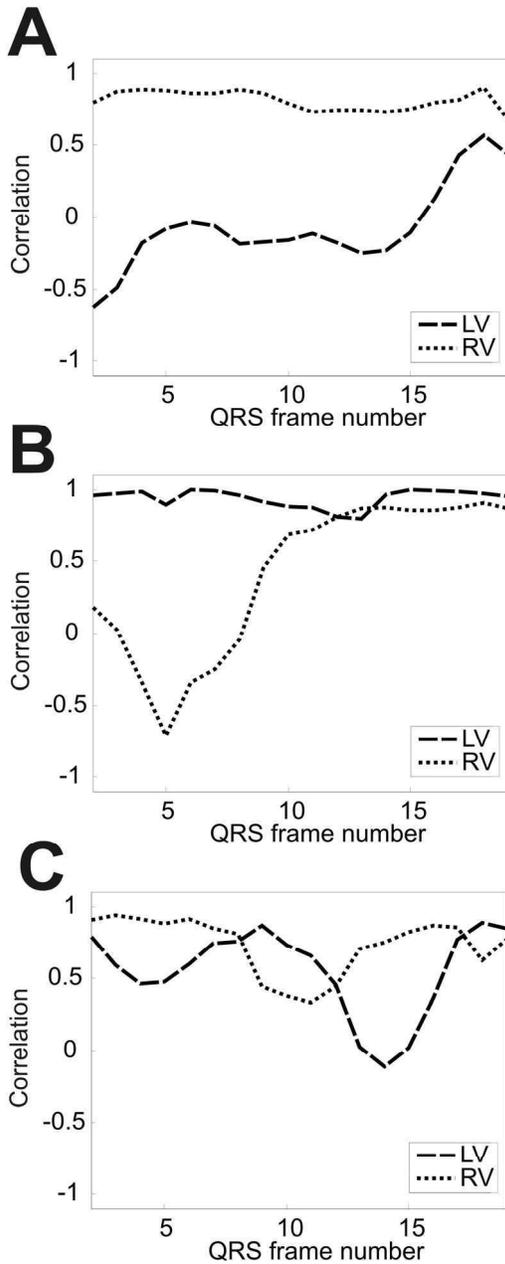


Figure 2. Comparison among BiV pacing and LV and RV pacing. Correlation coefficients corresponding to eighteen maps during BiV pacing vs. RV pacing and LV pacing are presented. In Panel A correlation coefficients of each map are presented for a patient classified as RBiV. Panel B shows correlation coefficients for a patient classified as LBiV. Panel C shows correlation coefficients for a patient classified as IBiV.

An example of the analysis in Group 2 patients with varying activation delays in the stimulation of both ventricles can be observed in Figure 3. In this patient, stimulation of the right ventricle 40ms prior to the stimulation of the left ventricle or earlier produced surface potentials maps almost identical to surface maps produced during RV stimulation (see Figure 3.A). Pacing in the left ventricle 40ms prior to right ventricular pacing or earlier produced surface potentials maps almost identical (correlation > 0.94) to surface maps produced during LV stimulation (see Figure 3.B).

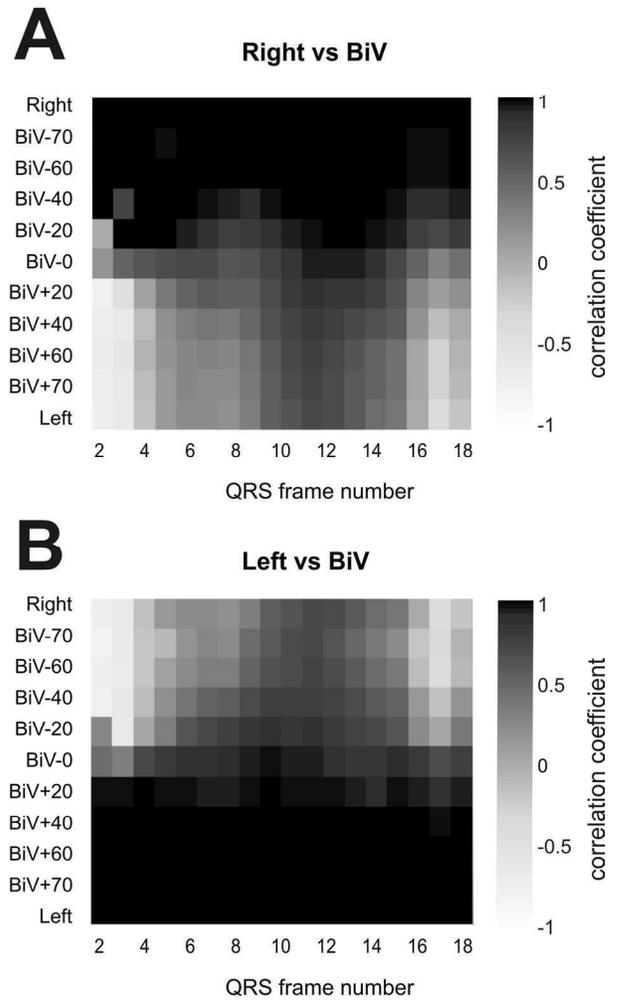


Figure 3. Comparison among BiV pacing and LV and RV pacing in Group 2. Delay in the stimulation of both ventricles ranged from -70 ms (RV paced first) and +70 ms (LV paced first). Correlation coefficients corresponding to eighteen maps during BiV pacing vs. RV pacing and LV pacing are presented for each programmed delay. Panel A shows correlation coefficients of BiV pacing vs. RV pacing. Panel B shows correlation coefficients of BiV pacing vs. LV pacing.

Analysis of all patients in Group 2 revealed that stimulations of either right or left ventricle 60ms or earlier than the other ventricle produced body surface maps with 80% correlation or higher with maps produced during the stimulation of only one ventricle in all patients. Also, stimulations of either right or left ventricle 40ms or earlier than the other ventricle produced body surface maps with 80% correlation or higher with maps produced during the stimulation of only one ventricle in 7 patients (78%).

#### 4. Discussion and conclusions

Eight patients presented surface maps similar to those observed during LV pacing. Although LV pacing may be beneficial to some patients [8], if an LV stimulation pattern is obtained, the RV lead appears to have little use. Three patients presented BiV maps similar to those during RV pacing, which is usually not the most suitable stimulation in patients with heart failure. These patients are clearly obtaining little benefit of their LV lead.

BiV stimulation produced surface potential patterns similar to those produced during the stimulation of a single ventricle in 38% patients. These patients may not be taking full advantage of the potential benefits of CRT and a reprogramming in their stimulation delay may change their activation patterns to those expected when pacing both ventricles.

BiV stimulation narrowed the QRS of patients with a wide QRS except for three patients. Interestingly, two of these patients presented surface potential maps similar to those obtained with the stimulation of a single ventricle. Even for the rest of the patients in which QRS width is reduced by using a second lead, the global activation pattern is unchanged as opposed to what could have been expected.

We have not investigated whether these patients that present activation patterns as visible from the body surface similar in BiV pacing and pacing in a single ventricle will benefit from a reprogramming of their interventricular delay but we focus in solving this question in future studies.

Another interesting observation that arises from this study is the similarity of BiV map patterns to RV or LV map patterns when the interventricular delay is 60 ms or more. Stimulation of one ventricle 60 ms or earlier than the other ventricle makes the stimulation of the second ventricle have no effect on the activation pattern.

Study of body surface maps during biventricular activation may help in the understanding of the activation patterns in the myocardium, allowing the discrimination of BiV patterns to those of RV or LV activations. Further study of these body surface maps may help in the optimization in the programming mode of BiV devices individualized for each patient.

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