

Unravelling the Influence of Right Ventricle Presence on Paced ECGs

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Abstract

Background: *In-silico* pace-mapping uses personalized computational models and patient ECGs to create virtual pace maps, aiding in ventricular arrhythmias (VT) ablation. Due to challenges in accurately defining the right ventricle (RV) boundaries, most computational studies focus only on left ventricle (LV) anatomy. The impact of the RV's presence on simulated ECGs during pacing remains unexplored.

Objective: To compare ECGs during pacing with and without the RV.

Methods: Twenty biventricular (BiV) computational models were created from computer tomography (CT) data, and a LV-only version was derived. Stimuli were delivered at 17 LV locations (AHA segments). ECGs from pacing BiV and LV models were correlated. The top 10 ECG lead correlations (ccm10) were averaged.

Results: The mean ccm10 across all models was 0.97 ± 0.02 , with the lowest and highest values in segments 8 (0.92) and 15 (0.99), respectively.

Conclusion: *In-silico* pacing showed strong ECG correlation (ccm10 > 0.90) between BiV and LV models, suggesting that RV inclusion may not be necessary for simple pace-mapping applications.

ablation targets, its invasive and time-consuming nature introduces limitations and increases procedural risk. *In-silico* pace mapping, using image-based computational models of the heart, offers a promising alternative [2]. This virtual approach allows for the rapid exploration of a wide range of pacing locations without exposing the patient to any risks, providing an efficient and safer means of guiding ablation therapy. *In-silico* pace mapping integrates personalized computational heart models with clinical electrocardiograms (ECGs) to generate virtual 3D pace maps; however, accurately defining the boundaries of the right ventricle (RV) wall remains a challenge due to the limitations of standard imaging resolutions, which can affect the precision of these personalized models. Furthermore, since infarct scars are primarily located within the left ventricle (LV) myocardium, many computational studies have focused exclusively on modelling the LV anatomy. This LV-centric approach has been practical for investigating areas most affected by scar-related arrhythmias[3]. However, the role of the RV in shaping the ECG during pacing is still poorly understood. The potential influence of the RV's anatomical presence or absence on ECG patterns during pace mapping remains an unexplored area of research, which could have implications for the accuracy and efficacy of computational models in guiding ablation therapy. The goal of this study is to compare ECGs generated during pacing simulations with and without the RV in the computational models. This will help assess whether the inclusion of the RV significantly impacts ECG outcomes in *in-silico* pace mapping.

1. Introduction

Catheter ablation is a well-established therapy commonly recommended for managing incessant, drug-resistant ventricular tachycardia (VT). Its success relies on accurately identifying the conduction isthmuses or focal sources that sustain the VT, which then serve as key targets for ablation. Pace mapping plays a crucial role in this process by helping to pinpoint these arrhythmogenic sites, guiding effective ablation treatment [1]. Although pace mapping is a valuable technique for identifying potential

2. Methods

2.1. Model generation

Twenty patient-specific biventricular (BiV) computational models were created using four-chamber

heart meshes derived from cardiac CT images of asymptomatic subjects. These models, which included detailed anatomical representations of both the LV and RV, were generated in a previous study from our group [4]. From each BiV model, an LV-only version was derived by isolating the LV sub-mesh as illustrated in Figure 1, allowing for a comparison of ECG outcomes during pacing simulations. Full details on the model generation process can be found in Rodero et al. [4].

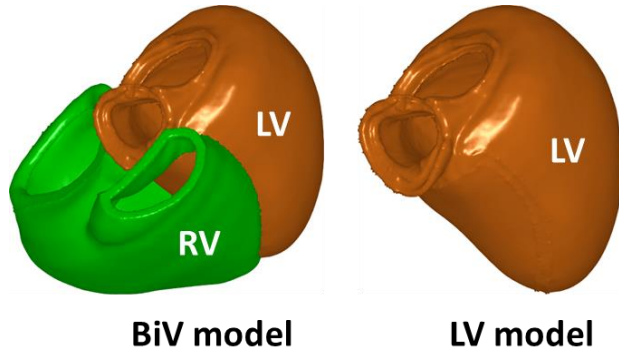


Figure 1. BiV and derived LV submesh of a representative patient.

2.2. Biophysical simulations

Biophysical simulations were performed using the monodomain model implemented within the publicly-available openCARP simulation framework [5]. Conduction velocity in the ventricular myocardium was set to 0.63cm/s in the myofibre direction and 0.30cm/s in the transverse direction [6]. The ten Tüscher ventricular action potential cell model was used to represent ionic cellular dynamics [7].

Extracellular potentials were computed at the 9 electrodes required to produce a 12-lead ECG from a generic torso model [8].

2.3. Pacing protocol

Stimuli were delivered at 17 specific locations within the LV, corresponding to the centers of each of the 17 American Heart Association (AHA) segments as illustrated in Figure 2. These targeted pacing sites were strategically chosen to comprehensively assess cardiac activation across the entire LV. Simulations of cardiac activation were conducted in both the BiV meshes, which included the RV, and the LV-only meshes, allowing for a detailed comparison of how activation patterns differ between the two models.

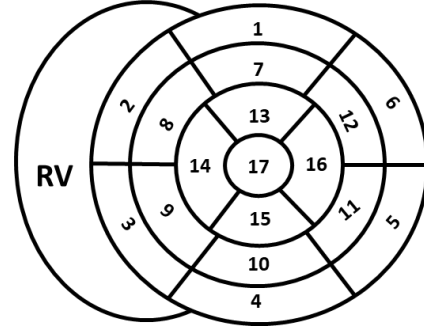


Figure 2. Pacing locations: stimuli were delivered at the center of each 17 AHA segment of the LV.

2.4. Data analysis

12-lead ECGs for each of the 17 simulated paced beats within the each BiV model were sequentially correlated with the corresponding ECGs in the LV-only model using the Pearson product-moment. 12 separate correlation coefficients were derived (one for each lead). The mean of the top 10 correlation coefficients (ccm10) across leads was used to give a single correlation coefficient value for each pacing site. Furthermore, the average of the ccm10 values for each of the $N = 20$ patients at each of the $N = 17$ AHA segment was calculated. This approach generates a correlation map for the LV illustrating how the ECG characteristics compare between the BiV and LV-only models across different pacing locations.

3. Results

Figure 3 presents the activation maps and derived ECGs simulated within both models of a single patient. Cardiac activation was initiated by pacing each model at the center of segment 7 (see Figure 2). These results illustrate the findings within one model from the overall cohort of $N = 20$ virtual patients. In The correlation was highest and lowest in leads LI (1.0) and LIII (0.27), respectively. The ccm10 between the ECGs from the BiV and LV models in this instance was notably high (0.98), indicating a strong similarity in the electrical signals generated at this pacing location.

The mean ccm10 across all models was 0.97 ± 0.02 , demonstrating consistent ECG characteristics when comparing the BiV and LV-only simulations. The lowest and highest ccm10 values across the cohort were observed in segments 8 and 15, with values of 0.92 and 0.99, respectively. These findings are summarized in Figure 4, which illustrates the distribution of ccm10 values across the various AHA segments, further emphasizing the relationship between anatomical considerations and pacing outcomes in the computational models.

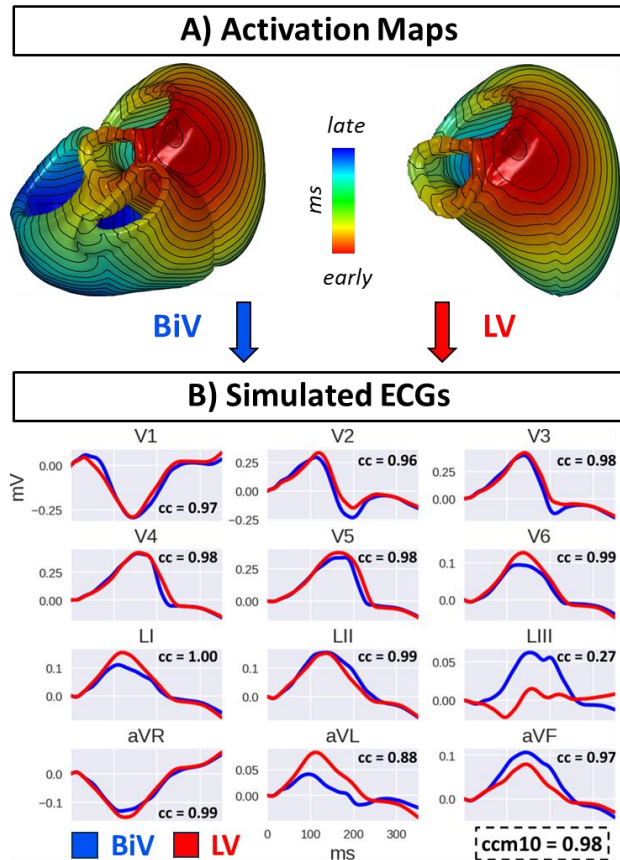


Figure 3. Simulated activation sequences and ECGs following pacing at segment 7 in both BiV and LV-only models of a representative patient.

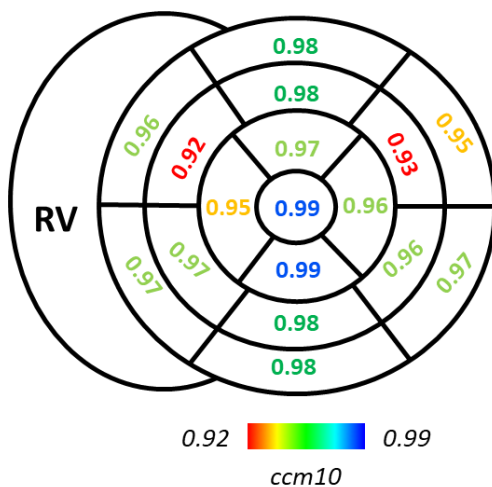


Figure 4. Correlation map: average of the ccm10 values calculated for each patient (N = 20) at each AHA segment (N = 17).

4. Discussion

This study investigated the effects of including the RV in *in-silico* pacing. *In-silico* experiments were conducted within a virtual cohort (N = 20) of patient-specific BiV and derived LV-only computational models. Stimuli at 17 designated LV locations based on AHA segments were simulated and the resulting ECGs correlated from both BiV and LV models. The high ccm10 values across all models indicates a strong correlation between the ECGs of the BiV and LV models.

The majority of cardiac computational studies have predominantly utilized LV-only meshes due to 1) challenges associated with accurately defining the boundaries of the RV wall. Standard imaging resolutions often fall short in capturing the precise contours of the RV, which can compromise the accuracy of personalized models; 2) infarct scars are mainly found within the LV myocardium. This LV-centric approach, while practical, has led to a limited understanding of the RV's role in cardiac activation and its potential impact on electrocardiographic outcomes during pacing.

A previous study from our group has investigated the role of the RV on simulation of paced activation sequences and VT inducibility in porcine heart models [9]. These *in-silico* experiments suggested that the RV should be included in patient specific models used to simulate VT, whereas this likely not required for simulating paced activation [9]. In this study, our main goal was not to investigate differences in VT inducibility between BiV and LV models, but rather to investigate whether the inclusion of the RV significantly impacts ECG outcomes in *in-silico* pacing. As shown in Figure 3, despite differences in morphology, especially in LIII and aVL leads, the paced ECGs in both BiV and LV-only models of the were strongly correlated (ccm10 = 0.98). Within the virtual cohort, simulated paced ECGs in BiV and LV-only models remained strongly correlated (ccm10 > 0.90, Figure 4) indicating that the RV may not be required for basic pacing applications.

5. Conclusions

In-silico experiments showed that while paced ECGs in BiV and LV-only models can differ in morphology, they are still strongly correlated (ccm10 > 0.90). This finding suggests that the RV may not be necessary in applications using simple pacing such as *in-silico* pace-mapping.

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