Estimation of the PVC Origin from Simulations Using Cellular Automaton

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Abstract

The ability of the patient-specific heart model with activation propagation simulated using a cellular automaton principle to mimic premature ventricular contraction (PVC) and corresponding surface ECGs was examined on ten patients.

Body surface mapping was performed for 5-20 minutes on patients with PVC. Their specific torso geometry models were created from CT scans. The PVC simulations were computed from 100-300 starting points in the whole ventricular myocardium model, and 12 leads of standard ECG were simulated/calculated. The measured and simulated signals were compared and evaluated by the Pearson correlation coefficient (PCC) in five intervals of the QRS duration, and the mean PCC for all 12 leads was computed. The distance between the stimulation point with the best mean PCC and the true ablation point was computed as a localization error (LE).

The best mean PCC (0.95) was obtained for the shortest evaluated interval of 20 % of QRS duration. However, the LE for this interval was the worst one (42.9 mm). The best mean LE (27.8 mm) was obtained for 80% and 100% of the QRS duration, with a mean PCC of 0.80.

The obtained LE was comparable with the results of the inverse solution for the same patients' cohort.

1. Introduction

The idea of using a heart and torso model and simulation of ventricular activation to localize the origins of premature ventricular contractions (PVC) has been around for a long time [1]. It was shown that the patient-specific geometry is important for obtaining good simulation results. Until now, many heart models and mathematical formulations with various complexities have been developed for activation propagation description [2,3]. On the other hand, such models are also computationally demanding. Finding a balance between the model's complexity and the goal of its usage is a challenge. The next open question is the number of measuring electrodes needed for PVC origin identification. Because multi-lead ECG measurements are still not introduced in clinical practice, several new methods are focused on the standard 12-lead measurements [4,5]. Simulations of ventricular activation using a cellular automaton principle are simple; however, they are fast and allow a principal insight into some changes in corresponding ECG measurements [6,7].

Recently, a thorough study aiming to fit the simulated and recorded 12-lead ECG from patients with PVC using a patient-specific heart-torso model was performed in [8]. In the presented study, we examined the possibility of such an approach to mimic the spontaneous PVCs using an activation propagation model based on the cellular automaton principle.

2. Materials and Methods

2.1. Measured Data

Multilead ECG measurements were performed on ten patients with PVCs indicated for invasive intervention by radiofrequency ablation (RFA) procedure. The measured signals were recorded in 128 electrodes on the torso together with standard limb leads. The electrodes were placed evenly on the torso using vertical strips with eight electrodes on each. Immediately after the measurement, the patients underwent a CT scan of the whole torso with the electrodes still positioned on the body. Thus, by segmenting the CT scans, we were able to create the patient-specific geometry model of the torso and also the actual positions of the measuring electrodes. The models of the lung lobes and ventricular cavities were generated to assume the main inhomogeneities in the torso.

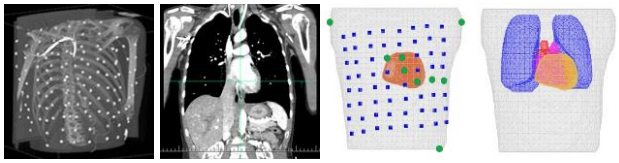

Figure 1. CT scan of the patient‘s torso with patient-specific electrode positions, and created homogeneous and inhomogeneous torso model. Green circles indicate the electrodes corresponding to the standard 12-lead ECG used in the study.

The duration of the signal recordings varied from 5 to 20 minutes to capture several PVC heartbeats. The

representative morphology of the patient's PVC heartbeat was defined as the average signal from all PVC heartbeats.

2.2. Heart Model

In each specific patient's heart model, simulations of PVC were performed using a cellular automaton principle [9]. The ventricular surface obtained from the CT scan was filled with points evenly distributed at a 1 mm distance, creating a three-dimensional grid. The starting points of PVC activation were chosen within the ventricular volume at a 10 mm distance from each other. The activation propagation was simulated from each starting point using the Huygens principle of a wavefront. The ventricular myocardium was assumed to be a homogeneous medium with constant activation propagation velocity in all directions. The final equivalent electrical generator was computed as a multiple dipole in each time step of the propagation. To compute the potential distribution on the torso, the transfer matrix between the multiple-dipole heart generator and the torso was computed by the boundary element method, assuming a homogeneous or inhomogeneous torso model.

2.3. Evaluation of Simulations

For each patient, the ECG signal from the PVC simulations was computed in the electrodes representing standard 12-lead ECG measurement. The QRS duration of the simulated signal was adjusted to the true QRS duration of the patient for each simulation by linear interpolation. To evaluate the similarity of the simulated signals with the measured ones, Pearson's correlation coefficient (PCC) was computed for each lead and then the average of them. The starting point with the highest/best mean PCC of simulated signals in 12-lead ECG was considered to be the presumed origin of the measured PVC. The localization error (LE) of the computed PVC origin was calculated as the distance to the true point of successful ablation.

We hypothesized that the simulation could be more similar to the reality at the beginning of the QRS interval when only a part of the ventricular volume is activated. Therefore, the PCC was computed and evaluated for five time intervals from the beginning of activation: 20%, 40%, 60%, 80%, and 100% of the QRS interval.

3. Results

The multi-lead ECG measurements were performed on ten patients, eight men and two women. The mean age of the patients was 49 (17-72). The number of captured and later averaged PVC heartbeats in each of the ten patients varied from 9 to 616 (218±184). In dependence on the size of the modeled ventricular myocardium the number of predefined starting points inside the model was from 103 to 306 (232±58). From each predefined position of the starting point, the activation propagation was simulated, and the forward problem was solved by computing ECG potentials at specific electrode positions on the patient's torso. The activation propagation velocity was set to 1 mm/ms. Because the simulation using a cellular automaton principle is a linear problem, the appropriate activation propagation velocity can be derived from the coefficient used to adjust the simulated QRS duration to the real one. The best mean PCC for each patient in each time interval of the QRS duration was computed. The values of the best PCC for all patients are depicted in Figure 2 for the homogeneous and in Figure 3 for the inhomogeneous torso model, respectively.

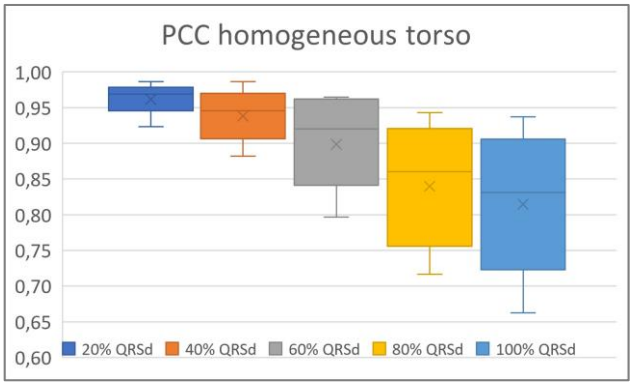

Figure 2. The best mean PCC from 12-lead ECG between the measured and simulated PVC signals in homogeneous torso for ten studied patients when PCC was computed for five time intervals of QRS duration.

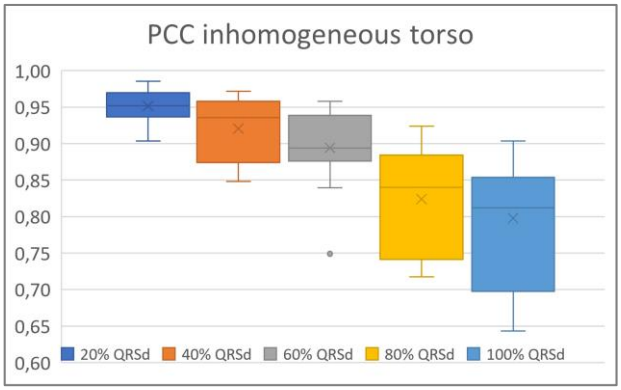

Figure 3. The best mean PCC from 12-lead ECG between the measured and simulated PVC signal in inhomogeneous torso for ten studied patients when PCC was computed for five time intervals of QRS duration.

Table 1 summarizes the obtained LEs for a homogeneous torso model and Table 2 for an inhomogeneous torso model for all studied intervals of QRS duration.

Table 1. LE for a homogeneous torso model.

	20%	40%	60%	80%	100%
P001	37,6	27,3	27,3	34,9	34,9
P002	23,8	23,8	28,0	31,6	31,6
P004	24,5	13,2	16,0	26,9	26,9
P008	44,9	20,3	15,2	18,4	18,4
P010	69,9	66,3	61,4	12,1	21,5
P020	40,5	20,9	40,5	30,7	30,7
P021	40,7	45,0	53,5	45,5	45,5
P023	13,4	13,4	13,4	23,1	23,1
P029	75,9	42,0	31,0	40,9	40,9
P036	57,6	14,1	14,1	14,1	4,7
mean	**42,9**	**28,6**	**30,0**	**27,8**	**27,8**
std	**20,1**	**17,3**	**17,0**	**11,1**	**11,7**

Table 2. LE for an inhomogeneous torso model

	20%	40%	60%	80%	100%
P001	62,4	15,5	27,3	27,3	27,3
P002	28,0	26,8	11,4	18,9	18,9
P004	46,7	15,4	21,5	27,0	27,0
P008	44,9	20,3	14,3	18,4	15,2
P010	69,9	62,3	61,4	5,4	29,5
P020	50,6	30,8	40,5	24,3	40,5
P021	39,2	51,8	53,5	45,5	45,5
P023	24,0	24,0	18,4	23,1	33,0
P029	65,8	32,4	31,0	31,0	32,4
P036	61,1	38,6	34,0	14,1	17,2
mean	**49,3**	**31,8**	**31,3**	**23,5**	**28,6**
std	**15,7**	**15,4**	**16,5**	**10,7**	**9,8**

4. Discussion and Conclusion

In the presented study, we searched for a simulation starting from a single point in the ventricular model that best mimics the measured 12-lead ECG signals during PVC. We also investigated the influence of the length of the time interval used for the evaluation.

As expected, the best values of PCC were obtained for the shortest time interval for the 20% QRS duration for the homogeneous (0.99–0.92) as well as for the inhomogeneous (0.99-0.90) torso model. The longer time interval was considered, the smaller mean PCC between the measured and simulated signals was obtained, so for the whole QRS interval, the PCC varied from 0.94 to 0.66 for the homogeneous torso model and from 0.90 to 0.64 for the inhomogeneous one. This is apparent in Figure 2 and Figure 3. Surprisingly, high values of PCC did not correspond with the small LE, and considering the original intention of this study, the smallest LE was obtained if 80% or 100% of the QRS interval was evaluated. The mean LE for all patients and the homogeneous torso model was 42.9 mm for 20% of QRS duration and 27.8 mm for both longest time intervals. Similarly, for the inhomogeneous torso model, the LE was 49.3 mm for 20% of QRS duration and 23.5mm and 28.6 mm for 80% and 100% of QRS duration, respectively. The largest mean LEs from all patients in 20% QRS time interval were 75.9 mm for the homogeneous and 69.9 mm for the inhomogeneous torso model. These values decreased rapidly for 80% and 100% of the QRS time interval to 45.5 mm for both torso models. In Figure 4, the distribution of the PCC values for 12 leads, giving the best average value for each patient, and a 100% QRS time interval is depicted for the homogeneous torso model. Figure 5 illustrates the ECG signals for patients with the highest and lowest mean PCC for both torso models.

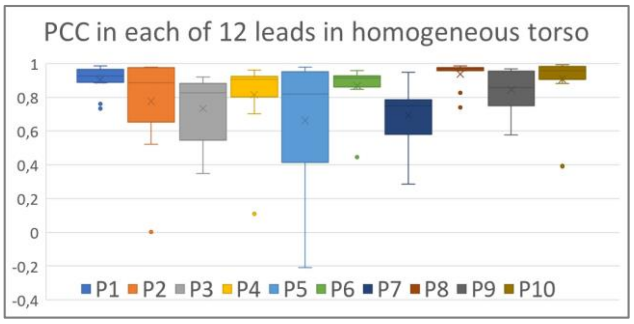

Figure 4. PCC distribution in 12 leads for the best mean PCC for each patient and 100% time interval of QRS duration.

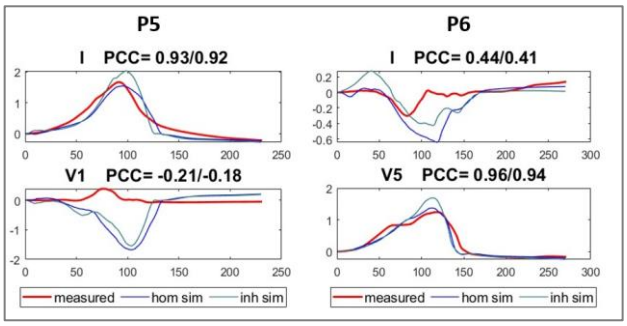

Figure 5. Examples of measured and simulated signals for the lowest mean PCC (P5) and one of the highest mean PCC (P6) for both torso models. For both patients, the signals with the best and the worst PCC were selected out of the standard twelve leads. The values of PCC are depicted for each signal for the homogeneous/ inhomogeneous torso model.

The hypothesis that evaluating the shorter time interval from the QRS duration will lead to a better representation of the measured ventricular activity was not confirmed. We suppose that this results from the fact that the signal-to-noise ratio at the beginning of the QRS interval is the smallest, 7.5 dB vs. 21.1 dB for the whole interval, and therefore, the measured signal in this time interval is not reliable (Figure 6).

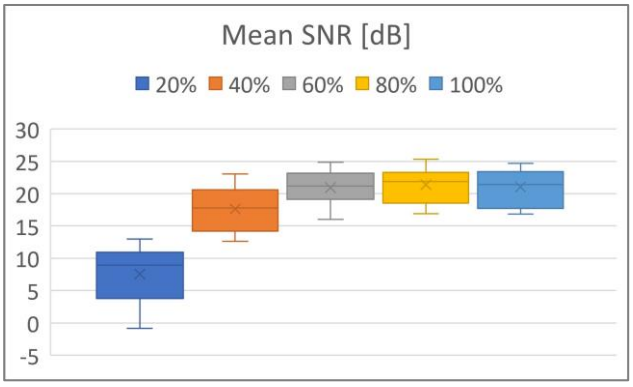

Figure 6. Mean signal-to-noise ratio of measured signals for all patients and five intervals of QRS duration.

The PVC origin was searched on the same patient cohort by two methods of the inverse solution (potential-based and dipole-based) in [10], where the mean LE for all patients was 37.6 mm/31.3 mm for a potential-based solution and homogeneous/inhomogeneous torso model and 27.9 mm/29.5mm for a dipole-based solution. The results obtained in the presented study 27.8/28.6 mm correspond to the dipole-based inverse solution.

The used method has several limitations, the overcoming of which can lead to better results in the future. First, the density of the grid in the ventricular volume used for activation propagation, and number of starting points can be increased. Next, the cells with high activation propagation velocity modeling conduction system on the endocardial layer of the ventricles can be assumed. Finally, no anisotropy in activation propagation and no additional comorbidities of the modeled ventricular muscle of the patients were taken into account. Also, the same evaluation could be performed for all measured 128 leads instead of the standard 12 leads.

Despite the limitations of the simulations in the heart and torso model, sparse positioning of the stimulation points, and simple activation propagation rules, the method of forward solution based on cellular automaton allows the estimation of the PVC origin with accuracy comparable to that of the inverse methods.

Acknowledgments

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