Analyzing the Atrial Depolarization Wavefront Triggered from Sinus Node and Coronary Sinus for Identification of the Arrhythmogenic Substrate

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Abstract

The success rate of the cardiac ablation procedure to cure atrial fibrillation is moderate and depends on the experience and expertise of the physicians. It could be increased by precisely locating arrhythmogenic substrates. The aim of this work is to present a simple and feasible method to analyze intraatrial electrograms to identify the arrhythmogenic substrate on the atrium, under sinus rhythm and pacing sequences. The change in the depolarization wavefront propagation, resulting from consecutive triggering at a point in the coronary sinus (CS), can be an indication of the arrhythmogenic substrate. The region specific study enables the localization of critical sites in the patient specific atrial anatomy. This could aid the physicians in ascertaining the efficacy of cardiac therapies. In this work the point-to-point analysis of the intraatrial electrograms was carried out.

1. Introduction

The changes in the normal heart rate and rhythm due to physiological or pathological reasons are termed as cardiac arrhythmias. The most common cardiac arrhythmia is atrial fibrillation (AF) [1]. AF increases with age and can lead to stroke as well as reduced quality of life [3]. In this work the concept of triggering has been used to identify the arrhythmogenic substrate. The depolarization wavefront originating from sinus node (SN) and electrodes on the CS catheter were analyzed. The tissue behavior has been analyzed on the basis of the differences in the depolarization wavefront propagation, which originated from the two triggers given at the same location from CS catheter after a time difference of 350 ms. In arrhythmogenic tissue, the change of wavefront propagation over the atrium can be expected. The regions in which the change in the propagation wavefront (that originated from the same location) is observed, could be suspected to be arrhythmogenic in nature and therefore are the regions of interest for further ablation therapies. The conduction properties are studied in terms of the conduction timings obtained using Non-linear Energy Operator (NLEO) [4]. Since routine clinical recordings were used for analysis, therefore the method is easy to integrate in routine clinical environment. These clinical studies could help in better clinical procedure adaptation to cure the atrial arrhythmia on a long run.

2. Method

2.1. Patient data

The recordings of two patients were carried out at Städtisches Klinikum Karlsruhe with a written informed consent. The details of both patients are stated in table 1. St. Jude Velocity system was used for the electroanatomical mapping of both patients. Spiral catheter was used for recording. Patient P1 had one ablation procedure history while for patient P2; there has been no ablation history previously.

Table 1. Patient details

Patient	Age, Sex, Wt. (kg)	Protocol (ms)	Prior ablation, Fibrillation
P1	62, M, 80	400-350	Yes, Paroxysmal
P2	67, F, 60	400-350	No, Paroxysmal

2.2. Electroanatomical mapping

The Velocity system (St. Jude Medical) was used to record the intraatrial electrograms and their location information for both the patients.

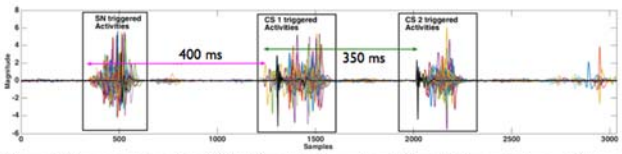
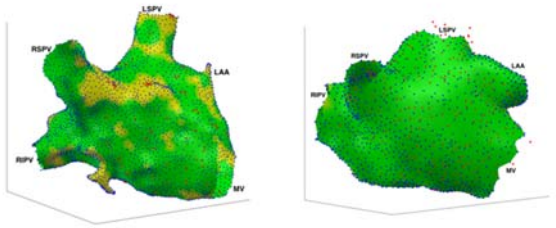


Figure 1: Representation of the 400- 350ms stimulus protocol. Time difference between CS trigger 1 and Sinus trigger was 400 ms, while time difference between CS trigger 2 and CS trigger 1 was 350 ms. Therefore, at each location three activities could be observed.

Surface ECG signals and the CS signals have also been recorded. The physicians selected the points of interest out of the entire data, were used for analysis. Figure 2, represents the endocardial geometry for both patients with the recorded points marked blue and the physician-selected points marked red. At all the red marked recorded locations, the intraatrial electrogram of 1.5 second was obtained and analyzed. The 400-350 ms stimulus protocol was used for pacing. The protocol was such that the time delay between sinus trigger and CS 1 trigger was 400 ms and the time delay between CS1 trigger and CS 2 trigger was 350 ms as represented in figure 1. CS catheter is used for triggering because it remains stationary throughout the recording. The surface ECG signals and the CS signals are used as references signals to remove Ventricular Far Field (VFF) and align the CS triggered activities.



a: Patient P1 Geometry b: Patient P2 Geometry

Figure 2: 3D endocardial surface point representation for a) Patient P1, b) Patient P2. Blue are the points recorded using the velocity system. Red are the points of interest marked by the physicians.

2.3. Data analysis

The data selected by the clinicians were taken into consideration. At every point, marked red in figure 2, the 1.5 second long electrograms, as represented in figure 3, were taken into consideration for analysis. These comprise the activities triggered from SN and CS. The stimulus in patient 1 was given at CS electrode 7-8, while in patient 2, at D-2 electrodes.

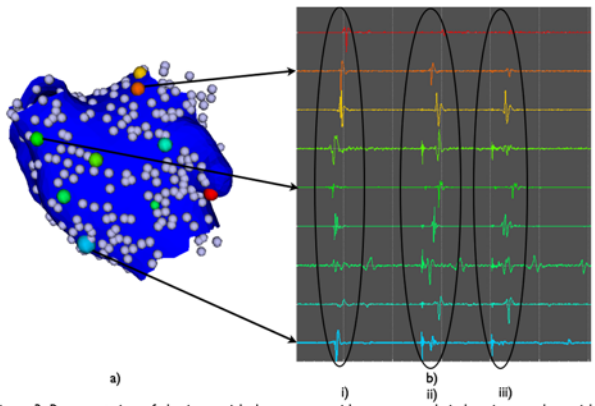


Figure 3: Representation of the intraatrial electrograms with respect to their location on the atrial geometry. a) 3D atrial geometry, b) intraatrial electrograms with i) SN triggered activities, ii) CS 1 triggered activities, iii) CS 2 triggered activities.

The intracardiac electrograms along with the surface ECG signals and CS signals were analyzed with respect to the atrial locations. The surface ECG was used to remove VFF. The CS signals were taken as the reference to align the triggering activities, which are later on used to calculate the local activation time (LAT) for each location.

2.4. Data analysis

The parametric analysis was done on the desired segments out of the entire data set. To avoid the patient going into fibrillation and to get the clear activities, the steady state pacing sequence of 400–350 ms was used. Figure 3 gives a clear representation of the three activities one after the other with respect to the atrial locations. For analysis purposes, the activities because of three triggers were analyzed separately. The time taken by the depolarization wavefront resulting from individual trigger for both the patient has been calculated at each measured point and interpolated for visualization purposes over the atrium. The local activation time (LAT) is the timing that the depolarization wavefront took to travel from the point of its origin to a particular location. The LAT at each location was calculated using the NLEO [4, 5]. The NLEO for the sample j of signal x was calculated as:

$$E_j = x_j^2 - x_{j+1}\, x_{j-1}$$

For each activity, the LAT is the time where maximum of the NLEO was detected as represented in figure 4. The LAT interpolation over the atrial geometry gives the information about the regions of slow and fast conduction over the atrium. The morphology was also analyzed in terms of the similarities in the activities from different triggering at specific location.

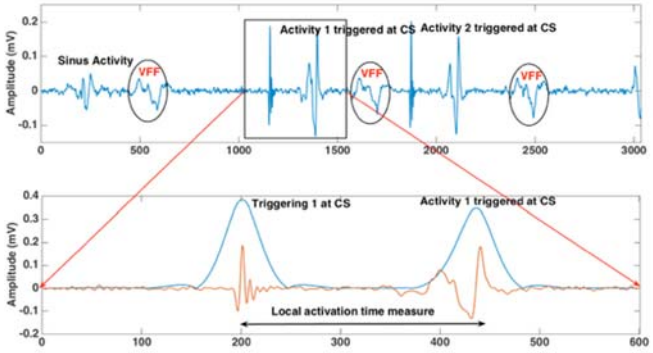


Figure 4: a) Representation of the LAT selection from CS1 triggered activity. The maxima from the stimulus artifact to NLEO maxima of the activity is the LAT.

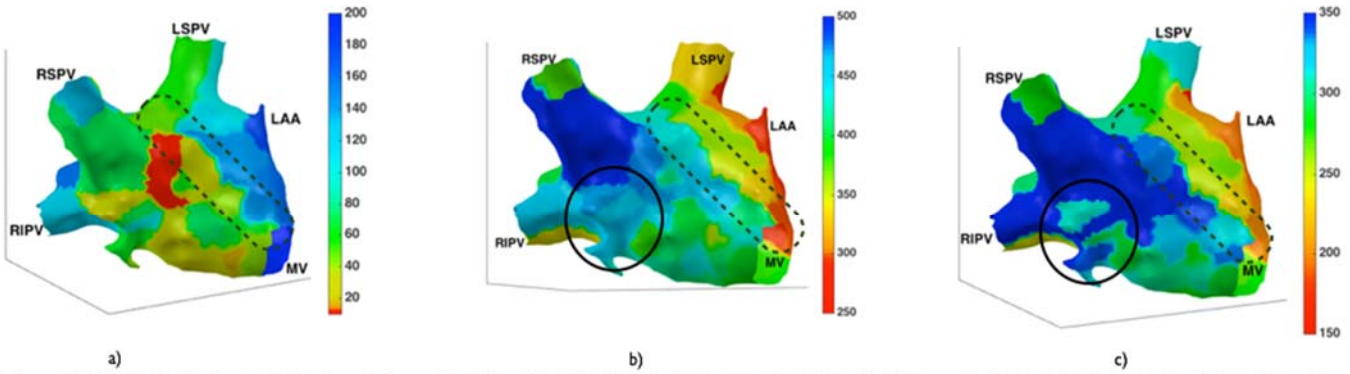


Figure 5: LAT depolarization wavefront excitation pattern for patient P1. Depolarization wavefront travelled from red to blue region in case of : a) Sinus triggering, b) CS triggering 1 and c) CS triggering 2. The difference in propagation pattern are encircled for CS1 and CS2 triggered depolarization wavefronts. The offset in terms if alignment between CS triggering 1 and CS triggering 2 was 100 samples. The slow conduction region is represented inside the dashed lines.

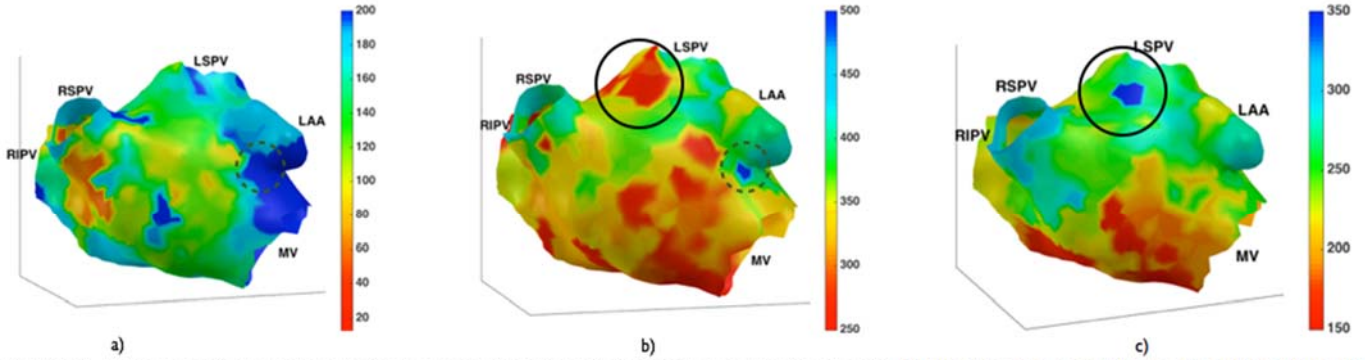


Figure 6: LAT depolarization wavefront excitation pattern for patient P2. Depolarization wavefront travelled from red to blue region in case of : a) Sinus triggering, b) CS triggering 1 and c) CS triggering 2. The difference in propagation pattern are encircled for CS1 and CS2 triggered depolarization wavefronts. The offset in terms if alignment between CS triggering 1 and CS triggering 2 was 100 samples. The slow conduction region is represented inside the dashed lines.

3. Results

3.1. Data analysis

The interpolation of the LAT in figure 5 and 6 were done to represent the depolarization wavefront propagation over the atrium for patient P1 and P2 respectively. In figure 5a, the initiation of the depolarization wavefront could be seen clearly, as represented by red region. Heart is a heterogeneous tissue; therefore there are slow and fast conduction areas. The region highlighted inside the dotted lines, represents the slow conduction region. This is also observed in figure 5b, 5c and 6c for both the patients. The activity due to CS 1 and CS 2 triggers were compared to see the region in which the propagation direction of the depolarization wavefronts changes. The region that is encircled in figure 5b and 5c represents the region with differences in the depolarization wavefront propagation pattern and therefore, could be an indication for the arrhytmogenic substrate. Similarly, the region encircled by solid lines in figure 6b and 6c is an indication for the arrhythmogenic substrate, while encircled by dashed lines represents the slow conduction areas. Along with the LAT interpolations, the morphology-based analysis was also done between the SN triggered and the CS triggered activities. To mark the regions with very low amplitude, the peak-to-peak value interpolation is also done on the atrium.

3.2. Parameter analysis

The time taken by the depolarization wavefront after initiation from respective triggers to spread over the entire atrium is represented in table 2. The correlation coefficients for patient 2 are represented in figure 7. Correlation coefficients were used to comment on the depolarization wavefront propagation patterns. Correlation coefficient value 1 signifies that the wavefront propagation is in the same direction, while zero signifies that the propagation is in different directions, from the particular location at which it is calculated. This indicates that the depolarization wavefront triggered from SN and CS 1 took different directions to propagate over the atrium. Figure 7b represents the correlation coefficient values between the depolarization wavefronts triggered at CS 1 and CS 2. This also is in favor that there was a change in the depolarization wavefront propagation initiated from the same point in the stimulus, which could

possibly be because of the arrhythmogenic substrate. The interpolation of these values over the atrium, for patient P1, is represented in figure 8.

Table 2. Representing the activity spread time taken by the depolarization wavefront to cover the entire atrium.

Patient	Stimulus electrode	Sinus Trigger (ms)	Stimulus 1 trigger (ms)	Stimulus 2 trigger (ms)
P1	7-8	97.7887	88.9423	90.9091
P2	D-2	101.7190	103.6855	91.8919

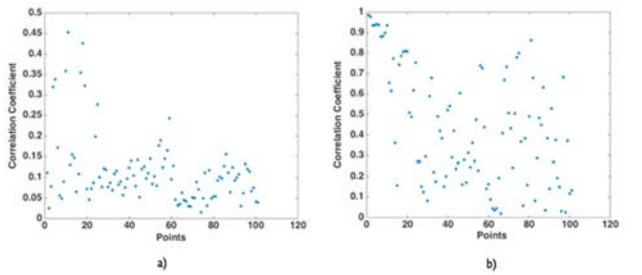


Figure 7: a) Correlation coefficients between SN triggered and CS1 triggered Activities, b) Correlation coefficient between CS2 triggered and CS1 triggered activities.

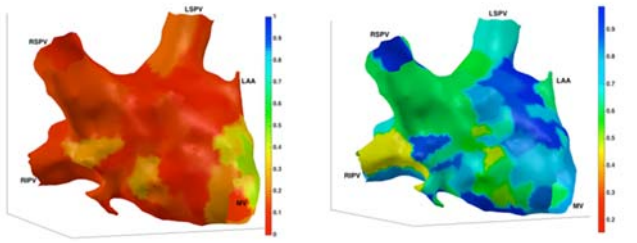


Figure 8: a) Correlation coefficient interpolated over the atrium for SN triggered and CS1 triggered activities, b) Correlation coefficient interpolated over the atrium for CS2 triggered and CS1 triggered activities.

4. Discussion and conclusion

In this study, the presence of arrhythmogenic substrates in both patients have been marked, by analyzing the LAT, depolarization wavefront propagation pattern and correlation coefficient. Figure 5 and 6 are also in agreement to the fact that the stimulus is given in electrode 7-8 for patient 1 and electrode D-2 for patient 2. By comparing the intracardiac signals along with the ECG signals, the VFF are removed before taking the individual segments into consideration. To remove VFF, the golden truth has been followed that it appears after the atrial activities. Faster we simulate less would be the clearer activation sequence. Therefore in the pacing protocol 400-350 ms protocol was used, so as to get the clear activation sequences and also avoiding the patient to go into fibrillation. The presence of double potentials and the late potentials was also done, which were absent in both the clinical cases. The slow conduction areas were marked in figure 5 and 6. The arrhythmogenic areas were also indicated figure 5 and 6. Figure 7 along with figure 5 and figure 6 respectively favors the presence of arrhythmogenic substrates in both the patients, resulting the change in depolarization wavefront propagation pattern. The final interpretations were done manually. For better understanding, the peak-to-peak values are calculated and compared to look for the low contact region or the low potential presence. The correlation coefficients calculated were also in favor of the fact that the direction of propagation for SN triggered depolarization wavefront and CS triggered depolarization wavefront was different. Since routine clinical recordings have been used to find the arrhythmogenic substrate therefore this method is easy to be integrated with the clinical environment.

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