Simplified Flexible Model of Human Heart Atria

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Abstract. It is known that origin of atrial activation has impact on ECG signals. We developed a simple computer model of heart atria that allows changing the geometry of atria and location of the sino-atrial (SA) node and conduction pathways. We used it to study the impact of the location of the SA node within the atria on the ECG signals. Although the developed model was quite simple, we were able to reproduce the natural variability in body surface potential maps observed in healthy young adults.

Keywords: Atrial Activation, Body Surface Potential Mapping, Mathematical Model, Simulations

1. Introduction

Electrocardiography is one of the most frequently used non-invasive diagnostic techniques in cardiology. Although, it is widely and often used in the clinics, many phenomena are not fully understood or explained yet. Because of that, it is very useful to apply mathematical modelling in this field. In silico approach has many advantages, which are useful especially in research that involves humans. With this tool one can perform experiments and evaluate hypotheses that cannot be done on living subjects.

It is known that origin of the atrial activation has impact on surface ECG signals [1]. In [2] it was showed that ectopic pacing of atria produced different patterns in the body surface potential maps (BSPM). Also in [3] it was shown that young adults and children have different BSPM, although they all had no history of cardiovascular disease and normal electrocardiographic and echocardiographic findings. Atrial function can be impaired by atrial enlargement, inter-atrial conduction delay or block, fibrosis and changes in electrical properties of atrial cells. That can result in serious medical problem – atrial fibrillation. In clinics, it is often difficult to recognize, if the P-wave abnormalities are due to inter-atrial conduction delay or atrial enlargement [1], which has different treatment strategies.

Because of that, we developed a transformable model of the heart atria that allow us to change the geometry of atria and the location of the SA node and conduction pathways. We used this model to study the impact of the SA node location on ECG signals and BSPM.

2. Subject and Methods

In our in silico experiments we have used a new mathematical model of the human heart atria developed in Matlab. It was inspired by the model of the heart ventricles developed earlier by Szathmáry and Osvald [4]. Basic geometry of the atria was created from two groups of ellipsoids. The first group represent outer surface and the second group, with slightly smaller dimensions than the first one, represents inner surface of the atria. Space between these two groups of ellipsoids was filled with cubic elements; each has size 1x1x1 mm and represents a piece of the atrial tissue. In each group of ellipsoids there were four ellipsoids, two bigger represent the body of the atria and two smaller represent the appendices of the atria, see
Fig. 1. In the body of the atria there are fenestrae corresponding to intrusions of large blood vessels. This configuration allows us to change the size and the shape of the model.

In recent anatomical studies [6] it was shown that SA node has bigger size than what is usually used on schematics and simplified illustrations. Therefore in the model the SA node was defined as an intersection of the atrial wall and another ellipsoid with the size of approximate 15x10x5 mm. Since the position of SA node is not very strict [5], at first, we placed it in correspondence with literature at the most anterior position in the right atrium and then gradually moved it more posteriorly. Thus we created five positions of the SA node as it can be seen in Fig. 2.

Preferential conduction pathways were simulated like areas with 3 time faster velocity of propagation of activation front like in normal tissue. These areas were located in the appendices, in the septum, and between the bodies of the atria. They were created as intersections of the atrial mass and several ellipsoids with desired shapes and positions.

To simulate the shape of the action potential we used the Courtemanche-Ramirez-Nattel model of human atrial cell [7]. The action potential propagation was simulated using the Huygens principles implemented by a cellular automaton [4]. The surface potentials were computed by solving the forward problem of electrocardiology. The equivalent multiple dipole model described in details elsewhere [8] was used to represent the cardiac electric generator and potential were computed on the surface on an realistic inhomogeneous torso model.

3. Results

Results of the simulations are shown in Fig. 2. There are visible differences in the patterns of BSPM corresponding to different SA node positions. These results are similar to the data measured in [3]. Also in individual leads there are some visible differences, e.g. in lead V1 the signal shapes in cases D and E are different from those in cases A, B and C.
Fig. 2. Results of simulations – A to E represent different positions of the SA node. In each picture from left to right: model of the atria with the location of the SA node (black), simulated BSPM and simulated signals of ECG leads I, II, V1, V6.

Differences between integral BSPM corresponding to atrial models with different SA position are visible, although the differences in ECG signals are not so evident. All ECG signals are with the same scale and integral BSPM were calculated from the whole atrial activation sequence (contain the P-wave and the Ta-wave corresponding to the atrial depolarization and repolarization).

4. Discussion

Despite the presented model of the atria is quite simple, we have found it sufficient to simulate distributions of the body surface potentials that are in agreement with the data measured in [3]. The variations in body surface potential maps could be explained by different locations of the SA node. As it is stated in [9], for detailed study of the atrial activity based on surface ECG signals modified position of ECG leads should be used. In our study we simulated only signals in positions of standard 12-lead ECG. When simulating the preferential conduction pathways, we assigned the same velocity of activation propagation to all these regions regardless of the position of the SA node.

5. Conclusions

Using the developed simple model of the atria, we were able to reproduce the observed natural variability in body surface potential maps recorded in healthy young adults. We demonstrated the impact of the location of the SA node on the BSPM. Variations in body
surface potential maps in healthy subjects can be explained by this. This can be one of the many possible reasons, why patient specific approach in interpreting the surface ECG is important. These first results suggest that our simple model can be used to study the variability of ECG signals caused by changed configuration of the atria.

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References


