Computer Simulation of Bundle Branch Re-entry in a 3D Cellular Automata Model of the Heart

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Abstract: A 3D cellular automata model of the entire heart was used for modelling the spread of excitation and repolarization in the heart. By modelling ventricular pacing and premature stimuli the inducibility of bundle branch re-entry was investigated. Under normal conditions bundle branch re-entry of type A and B were induced. A reduction of conduction velocity in the bundle branches resulted in multiple re-entrant echo beats as well as a wider zone of re-entrant activation.

INTRODUCTION

Akhtar et al. [1] was the first to report electrophysiologic findings in human studies that supported the hypothesis of macro re-entry via the His-Purkinje system. More complex pattern resulting in fascicular re-entry have recently been reported and show the importance of the stimulus site [2]. In diseased hearts, sustained bundle branch re-entrant tachycardia can occur when there is sufficient delay in His-Purkinje conduction such that the re-entrant impulse can continue to propagate around the bundle branches. Serious hemodynamic consequences, such as sudden cardiac death or syncope, are frequently the initial clinical representation of this arrhythmia.

In the last decade the increase of computational power supported the development of several 3D cellular automata models [3-6] for calculating the spread of excitation and the body surface electrocardiogram of the human heart. The purpose of this study was to simulate the spread of excitation in a 3D computer model of the entire heart and to test the inducibility of bundle branch re-entry.

METHOD

The described 3D cellular automaton model is an extended version of the previously published computer model [5-6]. In brief the heart anatomy was discretized in a Cartesian co-ordinate system by a 2.5 10^-3m grid including 11 different cardiac tissues for the normal and 4 for pathologic conditions. To each element of heart anatomy an effective refractory period (ERP) value was assigned. A conduction velocity was assigned for propagation inside as well as between different cardiac tissues. In addition the conduction velocity was assumed to increase linearly after end of the ERP till the end of the relative refractory period.

The earliest regions to excite the ventricle were the conjunction elements at the terminations of the bundle branches where the excitation propagated to the Purkinje fibre network and ventricular myocardium. The left bundle is separated into three branches which excite the high anterior paraseptal region, the midseptum and the posterior paraseptal apical region of the ventricle 0.153s, 0.155s and 0.165s after start of sinus node activation. In the right ventricle a single bundle excited the lower right apex and the right ventricular free wall 0.004s and 0.01s after left high anterior paraseptal activation. These locations were all close to the sites mentioned in literature [7] as being activated first.

The Purkinje fibre network was represented by a sheet of conducting tissue on the endocardial surface of the ventricle. Anisotropic conduction of the ventricular myocardium was modelled by different conduction velocities in direction of fibres and perpendicular to it.

The ERP dynamic depending on pacing cycle length was taken into consideration by assuming ERP dependence on the preceding time interval in the atrium whereas in the ventricle the formula published by Gulrajani [8] was used for all ventricular tissues by taking values from literature [9-10]. Figure 4 shows the ERP values of ventricular myocardium and purkinje fibre after a step increase of pacing frequency. Similar measurements were first reported by M.J. Janse in mongrel dogs [11].

The algorithm for calculating the excitation process is based on a modified version of Huygen's principle for constructing wavefronts. Each element of the heart
geometry can be in either of three states "excitable", "excited" or "waiting" during the computer simulation process [8]. Ventricular pacing at a basic cycle length of 0.7s in combination with premature stimuli at different coupling intervals and different anatomical locations was modelled for testing the inducibility of bundle branch re-entry. According to reference [2] the ERP of the right bundle branch was assumed to be longer compared to the left bundle.

Results

In case of left and right apical ventricular premature stimuli (S2) the preferred retrograde route of impulse propagation was through the left bundle branches. In addition the increment of S2H2 conduction time between premature stimulus (S2) and His bundle (H2) tended to have a linear pattern in relationship to decreasing S1H1 coupling intervals similar to the results described in literature [2].

In a next step ventricular stimuli were applied near the terminations of the bundle branches for inducing re-entrant activation. Single premature stimuli near the right ventricular free wall resulted in single ventricular echo beats. The premature beats were retrograde blocked at the right bundle branch but sufficiently delayed to excite the left sided His-Purkinje system in retrograde and the right bundle branch in anterograde direction resultant in V3. These excitation front was then finally blocked in the His-Purkinje system.

In the left ventricle the high anterior paraseptal region was selected for inducing re-entrant activation. This time the premature stimulation resulted in fascicular re-entry. The excitation travelled in retrograde direction along the posterior bundle and excited the ventricle (V3) through the anterior bundle. Then the excitation was finally blocked in the His-Purkinje system.

A reduction of conduction velocity (as found under pathologic conditions) in the bundle branches resulted in multiple re-entrant echo beats as well as in a wider S1S2 time interval of re-entrant activation. In addition the anatomical region where re-entrant activation occurred increased.

DISCUSSION

Several limitations and simplifications of the electrophysiologic process must be kept in mind. The excitation process was modelled on a macroscopic level and omitted electrotonic interactions between cardiac fibres. A coarse 2.510^3/m grid was used for simulating the excitation process in the entire heart. These simplifications seemed to be justified due to the scope of modelling macro re-entrant activation. In addition the difference between antegrade and retrograde conduction velocity and effective refractory period was not taken into consideration.

According to reference [2] the re-entrant circuit of His-Purkinje system re-entry can be classified into three types. Type A is the typical re-entrant phenomenon seen with right ventricular stimulation where retrograde conduction is via the left bundle branch and anterograde conduction is via the right bundle branch. Type B is typical seen with left ventricular stimulation. Type B is a fascicular re-entry where retrograde conduction occurs via a left sided fascicle and anterograde conduction is via the other fascicle. The reversal of re-entrant circuit of type A was rarely seen with left ventricular stimulation at least in the normal human heart. This reversal re-entrant circuit was labelled type C and involved retrograde propagation via the right bundle branch and antegrade activation via the left bundle branch. In our model type A and B were found.

A possible application of the model is to use it for patient oriented simulation. By this way the computer model can be used as an additional decision tool for pharmacological antiarrhythmic therapy or ablation procedure. Further the model can be used for calculating the magneto- and electrocardiogram based on a known pathologic substrate.

REFERENCES


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