A Cellular Automaton Based 2-D Electromechanical Heart Tissue Model

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1 Introduction

Computer modeling and simulations have great role in clinical diagnosis and therapy to bring down the increasing mortality rate due to heart diseases, besides clinical research and animal experimentation [1, 2, 3, 4]. Beginning with the Hodgkin-Huxley excitable tissue model many other models using ion exchanges through the cell membrane [1]. Experimental studies for ventricular muscle stress analysis [2] show that the mechanical properties of myocardium are nonlinear, anisotropic and viscoelastic [2, 5]. In this study, the main purpose is to analyze and model the electrical and mechanical aspects of the heart myocard tissue beginning with 2-dimensional geometry, which represents the simplified left ventricle tissue. The “cellular automaton” method was selected for this modeling, since both electrical excitation and propagation can be represented by this method. The algorithm can also express the synchronization of the electrical and mechanical activities. Furthermore, in addition to the normal behavior of the tissue, the ischemic/damaged heart tissue parameters can easily be implemented with this algorithm. Also its relative simplicity and short computation time are its other advantages.

2 Method

The electrical excitation and the resulting mechanical contractions have been simulated on the 2-D layers. The effect of the fiber orientation (anisotropy) and also ischemic (damaged) tissue properties are applied to the model to observe re-entry (a commonly encountered disorder) through this simplified model. Cellular Automaton Method is a mathematical model that explains the propagation of the electrical
Table 1: AP values and durations at each state

<table>
<thead>
<tr>
<th>States</th>
<th>Membrane Potential (mV)</th>
<th>Duration (ms)</th>
</tr>
</thead>
<tbody>
<tr>
<td>State 1 (S1)</td>
<td>+25</td>
<td>50</td>
</tr>
<tr>
<td>State 2 (S2)</td>
<td>0</td>
<td>80</td>
</tr>
<tr>
<td>State 3 (S3)</td>
<td>-25</td>
<td>80</td>
</tr>
<tr>
<td>State 4 (S4)</td>
<td>-50</td>
<td>30</td>
</tr>
<tr>
<td>State 5 (S5)</td>
<td>-80 (resting potential)</td>
<td></td>
</tr>
</tbody>
</table>

Figure 1: Myocardial transmembrane membrane potential variation (action potential) (shown in red) and the associated contracting force function (shown in blue) with respect to time. Left: Continuous potential and force functions due to ionic exchanges through the membrane [1]. Right: State changes according to the cellular automaton model.

...activity in macroscopic scale [6, 7]. In 1942, Wiener and Rosenblueth used this mathematical model to simulate the cardiac electrical activity for the first time [8] and many others followed this study [9]. In the cellular automaton model, complex ionic phenomena are represented in terms of changes of state of each model unit. Each state is assigned a physiological significance and specific rules govern the state transitions and propagation of the activation from one element to its neighbors. As the transmembrane potential passes the threshold value, the excitation begins and generates an action potential (AP) (in Figure 1(a)) and in our model it follows the rule on Figure 1(b). The potential values and durations of each AP state are given in Table 1. In a short time after initiation of the AP, a mechanical force is applied on the muscle cells that results in mechanical contraction. After a delay, each cell excites its 8 neighbors. In the isotropic case, all of the neighboring cells are stimulated with the same time delay; there is no directional preference of propagation. However, propagation is faster along the fiber directions in the heart; and this directional propagation variability should be considered in the simulations (along the fiber direction 1 m/s, and in the transverse direction 0.3 m/s [9]).
Figure 2: (a) Electrical excitation propagation in 2D anisotropic case. Colors represent the voltage changes: Blue nodes are in resting voltage (-90mV), new excited points are dark red (+20mV). (propagation at t=20, 150, 300 msec), (b) Isotropic case: Electrical propagation in 0°, 45°, and 90° fiber directions, (c) Ischemic tissue electromechanical wave propagation, isotropic tissue model.

3 Results

If the anisotropy is neglected the propagation is in a circular form. Color change in Figure 2(a) shows the first excited point reaches its resting value in the first instance. Each of the nodes have the same AP length and after their excitation, each point reaches the end of its action potential with the same delay with which it was excited. In the anisotropic case, delays are calculated according to the neighborhood and fiber angle (Figure 2(b)). For modeling the mechanical behavior, synchronization was set as it is shown in Figure 1(b). After a node is stimulated, contraction of the cell begins in its second state (S2) and all neighbors follow with appropriate delays. Ischemic tissue also has a property that causes re-entry; which is called electric accumulation. An electrical activation originates in ischemic tissue and begins to propagate. Because of the difference in action potential of normal and ischemic tissue (See Figure 2(c)), excitation of the nodes in resting state forms re-entrant electrical waves.

4 Conclusion

The design of computational models of human organs is a new research field, which opens new possibilities for medical analysis, therapy, and understanding of the biological phenomena. This study does not include complex ionic differential equations from the electrical point of view or the non-linear, viscoelastic properties of the tissue from a mechanical point of view. However, the conduction of electrical wave and the deformation can still be seen by using rule based cellular automaton model, which is accepted as the easiest and fastest approach in modeling the heart functions. The model can be used to better understand physiology and pathophysiology of the heart, to improve diagnostics of infarction and arrhythmia and to enable quantitative therapy planning.
References


