Enhanced Cellular Automation Model
For Detecting Heart Fibrillation

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Abstract: A new technique for human heart diseases diagnosis is presented. The technique is based upon the cellular automation and cellular membrane models. The coupling between the two models represents a key step in determining the action potential distribution, and hence a better view of the diagnosis. Beside ventricular arrhythmia other heart instabilities and ultimately chaotic behaviors can be detected.

1-Introduction: Heart disorders are due to conditions of abnormal impulse generation or abnormal impulse conduction in the myocardium. Abnormal impulse is manifested in either an enhanced automacity or a triggered activity due to some nonlinearities [1]. Abnormal impulse conduction is caused by circus movements of the depolarization wave in the form of a re-entrant wave form of excitation[2]. The spread of the depolarization from the Sinuatrial node (SA-node), to the atrioventricular node (AV-node) via the bundle of His, to the right bundle branch (L.B.B) and finally the perkenji fibers is an ordered process following a time schedule. The heart function integrity requires the above events to occur sequentially with correct timing. Many of the above events are subject to non-linear behavior, so the heart is susceptible to electrical instabilities leading to chaotic behavior [3]. Many researchers have investigated this behavior through the qualitative study of the cellular membrane models [4], that describe the cell behavior as microscopic events. The cellular automation model describes the propagation of this local microscopic event throughout the heart conduction network. We claim in this paper that the coupling between the two methods gives a better insight about the heart disorders and the early detection of the heart diseases.

2-Membrane Models of Cell activity
The electrical activity of the myocardial cell is controlled by some gating variables, these variables control the inflow and outflow of the important ions, namely; (Na), (k), (CL), the following models are generic systems that show excitability, oscillatory activity and membrane action potential.

2-1- Hill Model:
\[ V' = \frac{-1}{k} (V-V_0) + I(t) \]
\[ U' = \frac{-1}{l} (U-U_0) + \frac{1}{B} (V-V') \]

2-2-Fitz Hugh-Nagomo Model:
\[ V' = V - \frac{1}{3} U + I(t) \]
\[ U' = \varphi(V-bU+a) \]

2-3-Hind marsh & Rose Model:
\[ V' = -aV^3 + bV^2 + y - z + I \]
\[ Y' = c - dV^2 - y \]
\[ Z' = r[s(V-V_1) - Z] \]
Where:
V: Membrane potential, Y: Recovery variable
I: Total synaptic current & a, b, c, d, are constants
U: Time-varying threshold.

3-Cellular Automation and Recursive Growth

3-1- Finite State Automata (FSAs)
(FSAs) Are a family of mathematical constructs that are defined by a finite set of states, output alphabets and rules that take the automation from its current states to the next state. One of the states is designated the initial state from which the execution of the (FSA) begins.

A Cellular Automation (CA) is a spatially explicit form of a (FSAs). A set of cells are defined in a space, each cell is a (FSA) whose transition function depends on the cells own state and those of neighboring cells. Typically the symbolic output of (CAs) is the state of each cell.
The cellular automata employs a symbolic output to describe the growth of biological structures, it’s a spatially explicit form of a (FSA). When the machine changes state it produces a symbol, the dynamics of the machine are reflected in the sequence that it produces. The set of sequences of symbols can be considered to be the language that the (FSA) produces and the state transition rules constitute the grammar that underlies the language.

3-2- Cellular Automata Model of Ventricular Excitation

The cellular automata model developed by Mitchel et.al in (1992) in which each grid cell of the automation represents a homogeneous group of excitable units. Each grid cell is one of four possible states that define the cardiac action potential: (Quiescent, Excited, Absolute Refractory{can not respond to any excitation}, Relative Refractory). The relation of the four states to the ventricular action potential is shown in Figure.1

4- The Proposed Model

4-1- Physiological Basis of the Model

To investigate the nature of impulse propagation and the mechanisms of electrical instability, which precipitates arrhythmias, a model has been devised which simulates heart conduction processes. This modeling process has proven to be valuable in revealing the mechanisms of abnormal modes of impulse conduction.

There are generally two main methods of modeling complex cardiac tissue. One uses 'phenomenological' models based on an inductive approach where the system is considered as a black box. The other is based on the deductive approach where the tissue behaviour is described by general biophysical laws. The deductive model for excitable media tends to be much more complex and relies on membrane theory according to which the action potential generation is described by the charging of the cell membrane capacitance by ionic currents. Because of the complexity of the deductive approach, which limits simulations to small areas of cardiac muscle, an inductive approach to modeling has been chosen which allows the development of a basic conduction model. The properties of the proposed conduction model are fundamentally determined by both the properties of the individual elements and the nature of the propagation between them. The concept of a formalized excitable medium is used to define the behaviour of individual elements and their interaction. The excitable medium comprises an array of discrete elements, with the physiological behaviour of each element defined in terms of a number of discrete states.

At any time an element may be in one of four possible states. These are:

- Quiescent (at rest)
- Excited (depolarized)
- Absolute refractory (depolarized and in excitable)
- Relative refractory (recovering excitability)

The relative refractory phase is characterized by a gradual recovery of excitability, the simulation threshold falls from infinity (absolute refractoriness)
to a steady value corresponding to the quiescent level. This is simulated in the model by defining an exponentially falling relationship for the number of excited neighboring elements required to depolarize a relative refractory element with increasing refractory duration. The transition of an element between the states is related to the cardiac action potential. An element will remain in the quiescent state until it receives excitation from a neighboring element. Once a stimulus is conducted to the element, it depolarizes and passes into the excited state. In the excited state it has the ability to excite any of the eight neighbors, which becomes quiescent. In this state it can also contribute to the depolarization of a relative refractory element Figure.1-b. After a while, the element enters the absolute refractory state, during which it neither excites quiescent neighbors nor be excited itself. It then goes through the relative refractory state and eventually becomes quiescent again. It is assumed that all excited elements have action potentials of unit amplitude. This simple algorithm defines the basic neighborhood rule which controls the interaction of elements and the spread of conduction in the model.

4-2- Cylindrical Surface Model With Spatial Dispersion (Gaussian) of Refractoriness:
To simulate the spatial dispersion of heart depolarization the refractory periods of the elements are defined in terms of a normal Gaussian distribution. The Gaussian probability function is selected because the variation in properties of many natural processes is known to follow this natural distribution. In this simulation the period values for each element are initially defined and remain fixed for the course of simulation. A procedure generates normally distributed random numbers for which a mean (M) and standard deviation (SD) have been defined to generate independent relative refractory period for each element.

The structure of the excitable medium is represented initially as a two-dimensional network comprising an array of (50*50) square elements. The scale is chosen such that the properties of individual cardiac cells are not simulated; rather the model is macroscopic and hence represents the bulk properties of myocardial tissue. The two dimensional sheets are configured as cylindrical shell structure to simulate atrial and ventricle surface conduction. Simulation of the model is controlled by a pacemaker element situated at the upper edge of each cylinder as shown in Figure.2.

4-3- Model Electrocardiogram (ECG)
The model simulates the electrical activity of the heart (ECG), which is derived from the vector summation of dipole sources between any adjacent quiescent and polarized element. By using the (ECG) data (durations, amplitudes) provided by the amplitude detector, the (ECG) can be constructed and simulated.

5-Results
5-1-Normal Heart Rhythm
The simulated conditions of normal excitation and repolarization in the heart model are illustrated in Figure.3 The mean refractory period is taken as 250ms, with conduction velocity (CV) of 50cm/s, which are realistic values. The spatial dispersion of refractoriness is specified with a standard deviation.
(SD) of 70ms. The excited state duration (EP) is 10ms, and the stimulation rate is set at 120 beats/min (period SP of 500ms). The model activity is displayed as a profile map of element states with the cylindrical surface opened out as a two-dimensional sheet Figure.2.

The discrete states are shown as different colors:
- Excited: Red
- AR: Violet
- RR: Brown
- Quiescent: White

The model (ECG) is plotted underneath with markers on the time scale to indicate the time of state propagation. In the ventricular sheet the wave front propagates uniformly downwards and also radially to converge in the middle. The initial spread of depolarization through the ventricular surface produces a sharp positive deflection in the (ECG) followed by a corresponding downward deflection to the baseline as the wave front merges in the center, this well defined component of the (ECG) is the model equivalent of the (QRS) complex. The wave fronts continues to conduct downward and decays when reaching the lower edge Figure.3. Leaving in its wake a totally refracted region. After a time the refractory elements begin to repolarize. This repolarization progresses nonuniformly due to the spatially distributed refractoriness of the medium.

Figure.3-a

Figure.3-b

Figure.3-c

Figure.3 Normal Ventricular Activity

SP=500ms
RP(M)=250ms
SD=70ms
Ep=10ms
5-2-Rhythm Disturbance & Electrical Alternans

A variety of rhythm disturbances are observed in the simulations as the stimulation rate is raised. These are dependent on parameter conditions as illustrated in Figure.4 - 7.

Figure 4-a

Figure 4-b

Figure 4-c

Figure 4

SP=200ms
RP(M)=250ms
SD=70ms
Ep=10ms

It's seen that the normal rhythm of Figure.3 is transformed to a condition of electrical alternans followed by 2:1 conduction block as the driving interval is reduced from 500ms to 200ms, with a stimulation period of 200ms, the depolarization wave fronts facing nearly most of the area in Absolute Refractory states which appear to stop its propagation, the diagnosis is (Ventricular Tachy Cardia).
Figure 5 illustrates a more severe alternans condition which exhibits gross rhythm disturbance, produced by increasing the excited state duration from 10ms to 20ms. The activity is instable and the diagnosis is (Premature Ventricular Contraction).
Figure 6-a

Figure 6 The effect of reducing the stimulation period to 200ms and the increasing the excited period to 20ms at the same time, the diagnosis is (Ventricular Tachycardia, Premature Ventricular Contraction).

Figure 6-b

Figure 6-c

Figure 6

SP=200ms
RP(M)=250ms
SD=70ms
Ep=20ms
Figure.7 The effect of increasing the stimulation period to 1600ms with $EP=20\text{ms}$ can be seen and the diagnosis is (Idio Ventricular Rhythm).
The simulation in Figure.8 shows that the normal rhythm has been interrupted by a stimulus delivered during the phase of maximum ventricular repolarization, the resulting wave front has spread out radially and become fractionated at many points around islands of refractory states. The activity begins to appear totally unsynchronized, and fibrillatory.

**6-Conclusion**

A new technique for the human heart diagnosis is presented. The technique is based upon the cellular automation and the cellular membrane models. The coupling between the two models represents a key step in determining the action potential distribution. The model can be used to investigate the mechanisms involved in normal conduction as well as those which lead to electrical alternans and chaotic behaviors, a better view of the heart diagnosis can be obtained.

**6- References**


