A MODEL OF THE HEART'S CONDUCTION SYSTEM USING A SELF-SIMILAR (FRACTAL) STRUCTURE

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Abstract

A model of the heart's ventricles which consists of a conduction system and muscle cells is described. A self-similar bifurcating fractal structure simulates the conduction system of the heart (Bundle branches and Purkinje fibers). The model is used to describe (1) a normal heart's ventricles and (2) a defective heart with a damaged mass of ventricular tissue. The damaged tissue are regions of cells having a substantially lower conduction velocity with delayed activity. As the model is activated a dipole potential is generated and a simulated QRS complex is obtained. The results show that using the computer model, a normal QRS complex and late potentials at the end or after the QRS complex in a defective heart can be simulated. This model can be used as an investigative tool to study pathological phenomena in the heart.

Introduction

The heart contains a complex conduction network, which carries the electrical signals which activate the myocardium, to many points almost simultaneously. Histological research reveals the recursive bifurcating nature of this system. It starts with the Bundle of His, which bifurcates into the left and right bundle branches, which bifurcate again into finer and finer fibers. This intricate structure can be described using a self-similar (fractal) bifurcating 'tree'[1].

Model Description

The model described in this work is a discrete cell model. It contains about 20,000 rectangular cells, arranged inside a rectangular 'space'. Conduction from each cell is to its 8 nearest neighbors. The self-similar conduction system is defined by calculating the positions of the appropriate cells, and then defining them as conduction cells by giving them a higher conduction velocity than that of the 'muscle' cells. Fig. 1 shows the model as it is graphically displayed on a computer screen. Various parameters of the model and, in particular, of the conduction system, can be modified to create different versions of the model. Parameters which can be varied are the tree's basic angle of bifurcation, the factor of branch shortening from one generation to the next, the size of the model, the ratio between conduction velocity in the conduction system and in 'muscle' cells.

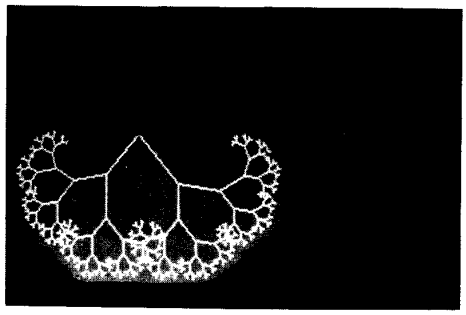

Fig.1
A graphic representation of the model

When an initial signal is introduced, the model causes it to propagate at different speeds through the conduction system and the muscle cells. The conduction system cells, transferring the signal at a higher speed, cause different areas of the model to be 'activated' at nearly the same time. The dipole potential generated by neighboring 'activated' and 'resting' cells is measured at a predetermined 'vantage-point'. A 'QRS complex' is thus obtained.

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QRS Complex and Late Potentials Simulation

The model was used to simulate a normal QRS complex and a QRS with delayed activity (late potentials) . During the last decade, low level high frequency electrical activities (late potentials), which represent an area of myocardium that shows delay in activation, were recorded at the end or after the QRS complex. It was found[2] that such signals appear to be specific for patients with recurrent ventricular tachycardia. In our simulation a rectangular area within the model was defined as having a lower conduction velocity than the 'normal' cells. Conduction velocity was reduced both in the 'damaged muscle' and 'damaged conduction system' cells. To obtain the late potential activity the QRS complexes were band-pass filtered between 60-200 Hz using non-recursive digital filter and fast Fourier transform algorithm. Two parameters which have been used as markers for the presence of late potential activity were measured: (1) the filtered QRS duration (FQRSD) and (2) the root mean square voltage of the last 40 mSec of the filtered QRS complex (RMS40).

In order to demonstrate the importance of the conduction system in the production of the normal QRS complex, we ran a simulation of the model with and without the conduction system.

Results

Fig. 2 (upper trace) shows a QRS complex obtained under normal conditions from the model configuration shown in Figure 1. The lower trace shows the high frequency (60-200Hz) content of this QRS as a function of time. A narrow FQRSD and a high RMS40 values, 82 mSec and 110 arbitrary units (AU) respectively, were measured. Fig. 3 shows the non-filtered and filtered (60-200 Hz) QRS complexes obtained from the model with (traces a and b) and without (traces c and d) the conduction system. Due to the slow and uniform advance of the signal throughout the model without the conduction system, the duration of the QRS is increased, and the high frequency content of the QRS complex is very low.

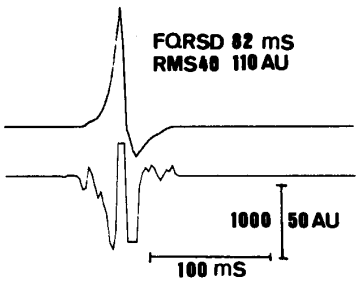

Fig. 2
A 'normal' QRS simulation as obtained from the model represented in Fig. 1. Short FQRSD and high RMS40 can be seen.

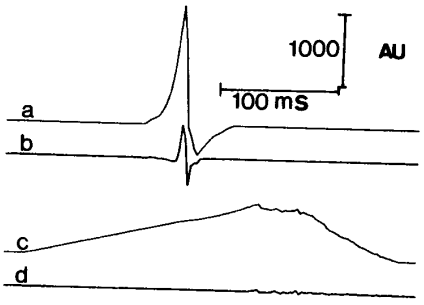

Fig 3
Non-filtered and filtered QRS complexes obtained with a conduction system (traces a,b) and without the conduction system (traces c,d).

Figure 4 shows the graphic representation of the model with a damaged block of cells close to the end of the conduction system, (the damaged part is in a region which is normally activated late in the QRS complex). As can be seen most of the cells are activated and the 'damaged tissue' is depolarized at the last part of the QRS complex . Figure 5 is the output QRS complex. The filtered QRS complex (Figure 5 lower trace) contains low level signals at the end of the QRS complex with FQRSD of 110 mSec and RMS40 of 15 AU. Figure 6 shows a graphic representation of the model with a damaged block of cells in a part of the conduction system normally activated earlier in the QRS cycle. This configuration results in a longer delay in the activation of the damaged tissue and late potentials can be seen (Figure 7 lower trace) after the end of the QRS complex with a wide FQRSD of 122 mSec and a low RMS40 of 5 AU.

Discussion

The results show the importance of the conduction network to the normal

function of the cardiac system. This network is responsible to the near-simultaneous activation of muscle cells at many points in the myocardium. This order of activation contributes to the high-frequency content of the QRS complex.

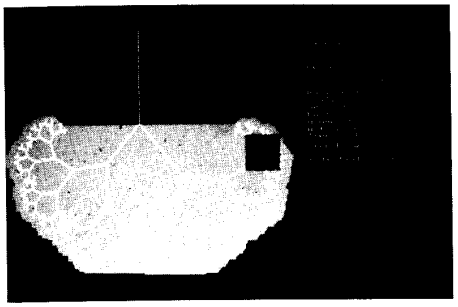

Fig 4
A model containing a damaged block of cells near the end of the conduction system.

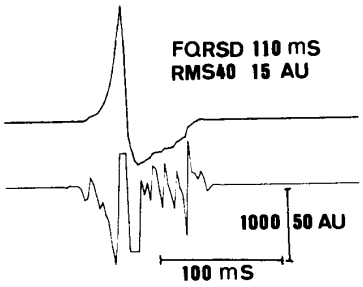

Fig. 5
QRS obtained from model represented in fig. 4. Upper trace:non-filtered QRS. Lower trace:filtered QRS. Wide FQRSD and low RMS40 were obtained.

The results also indicate that late potential phenomena can indeed be attributed to reduced conduction velocity in regions of the heart. In the model, as in real hearts, the conduction disturbance creates in the QRS complex low level signals with high-frequency content. The duration and amplitude of this late ventricular activity in the QRS complex depends on the size and location of the damaged area as well as the conduction velocity in this area.

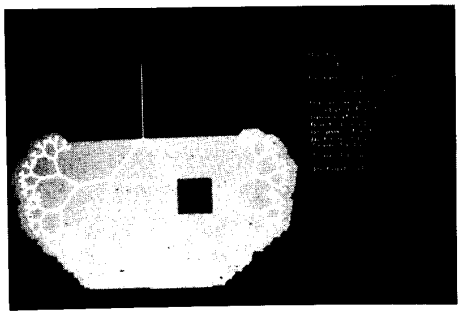

Fig. 6
A model containing a damaged block of cells in a middle section of the conduction system.

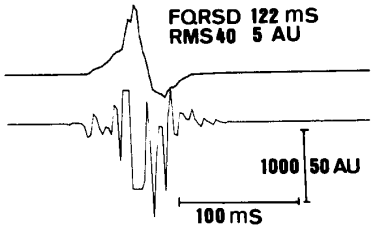

Fig. 7
QRS obtained from model represented in fig. 6. Late potentials can be seen after the end of the QRS complex.

The effect of a damaged conduction system on the heart can also be inferred from the simulation results. Delay in the activity of the damaged region can cause disorder in the activation of the myocardium. This in turn can cause reduced efficiency of the heart. In extreme cases, phenomena such as reentry and ventricular fibrillation can occur. We intend to investigate these phenomena in later versions of the model.

References

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2. Breithardt, G., and Borggrefe, M. Recent advances in the identification of patients at risk of ventricular tachyarrythmias: role of ventricular late potentials. Circulation 75: 1091-96. 1987.