

A Model for Simulating Bundle Branch and Fascicular Block

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Abstract

Objective: To develop a model for simulation of Bundle Branch and Fascicular block. The model accounts for the contribution of fiber orientation to the velocity of signal propagation through the myocardium as well as "reentry" of the signal into the subendocardial purkinje system..

Methods: The Selvester/Soloman method for simulation of normal myocardial activation was adapted to the ECG-TECH Heart-to-loop-to leads software. The software connects the intercardiac activation to the body surface vector and ECG recordings and was modified for this study to simulate complete Right and Left Bundle Branch and Left Anterior and Left Posterior fascicular block. The process of developing models that simulated typical recorded QRS waveforms of these four conduction abnormalities involved iterative interaction among the investigators.

Results: Accurate simulations of each of these interventricular conduction abnormalities was accomplished by utilizing published data on the purkinje and myocardial conduction properties, especially at the purkinje myocardial junction. Agreement between the simulated and recorded waveforms was accomplished by varying 1) the locations of the initial purkinje-myocardial activation sites and 2) the relative contributions of intra-myocardial conduction and reentry to the subendocardial purkinje system to activation of myocardium past the point of conduction block.

Conclusions: Accurate simulation of intraventricular conduction abnormalities caused by bundle branch and fascicular block abnormalities was accomplished. These results were then used to add conduction defects to the 3D Heart training program used to train medical students to better diagnose heart disease.

1. Introduction

This program was inspired by the need to provide a realistic simulation of the activation of the right and left ventricles in the setting of a significant conduction defect. The original program simulated both the normal and infarcted heart. The new program builds on the original program to include:

1. Left Bundle Branch Block – LBBB.
2. Right Bundle Branch Block - RBBB
3. Left Anterior Fascicular Block - LAFB
4. Left Inferoposterior Fascicular Block. - LIFB

These additional instructional simulation aids should make the program a more complete guide to the understanding of many types of heart problems. The simulation program shows a 3 dimensional view of the left ventricle with an orientation as seen in the human chest. The 3D wave fronts generated in this model will be the source of the electrical dipole vector signals that transfer to the surface electrodes. The proof will be in the generation of realistic electrocardiograms which will be checked for the validity of both the vector and 12-lead results [5,6].

2. Methods

The simulation involves the use of a realistic heart model with an activation sequence reflecting the type of conduction abnormality present. This model includes the development of a wave front surface that is propagated through the myocardium. This surface is the basis for a perpendicular vector that represents the electrical signal recorded by the surface leads of the electrocardiograph [2, 3]. The method was developed in a previous paper [1] and was modified to include the considerable effects of fiber orientation on the velocity of propagation Agreement between simulated and measured ECGs

required numerous model iterations, each resulting in a modification of a simulation parameter.

The LV was divided into 12 segments in accordance with the ISCE nomenclature of myocardial wall segments. This division was adapted from the Ideker pathoanatomic subdivisions and used in the Solomon-Selvester model. There are 3 subdivisions (apical, middle, and basal) in each of the four LV quadrants (anteroseptal, anterosuperior, posterolateral, and inferior). In each cross-section, the vector groupings for the RV are assigned to one of the four LV vector groups on the basis of the angles of the vectors projecting from the RV endocardium (Figure 1).

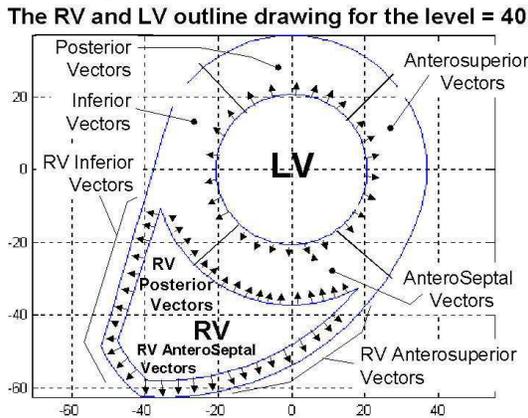


Figure 1. Cross section of the human heart at a level of 40 mm from the base. The small arrows represent the vectors directed perpendicular to the endocardial surfaces of both ventricles generated by the initial electrical forces (dipoles) in each local area. The vectors from the 4 LV quadrants represented are labeled, as are those from the RV that share the same spatial direction. For example, both the LV and RV vectors labeled “AnteroSeptal” are directed anteriorly. The grid indicates the location in mm from the center of the left ventricle.

3. Results

Each type of conduction defect was simulated. A series of 3 movies of up to 60 frames was created, each depicting the sequential activation of the Left and Right ventricles during the QRS cycle. The vectors generated by each of the 12 segments were illustrated. The vector sum and its resolution into the frontal, sagittal, and horizontal planes were also shown. For the frontal and horizontal planes, the vectors comprising the loops are projected onto the lead vectors highlighting the relationship between the activation sequence and the 12 lead ECG.

Left bundle branch block

LBBB was the most difficult of the conduction defects to simulate. When Purkinje excitation of the Left Ventricle is completely blocked, all activation originates in the Right Ventricle. The activation signal travels to the LV through the myocardium, primarily septal myocardium. It was necessary to develop a whole new set of algorithms to propagate the wave fronts from the RV to the LV. This is shown in the figure below where we observe the wave fronts as a function of time originating at both the lower part of the RV as well as at the middle of the RV free wall. These surfaces propagate across the septum to the LV where they reenter the Purkinje system. The surfaces at the edges of the LV propagate at a velocity that is 2 times the velocity seen in the middle of the septum. This represents the difference between the longitudinal and transverse fiber conduction velocities. This difference made a large contribution to the vector magnitude and direction over the middle part of the simulated ECG.

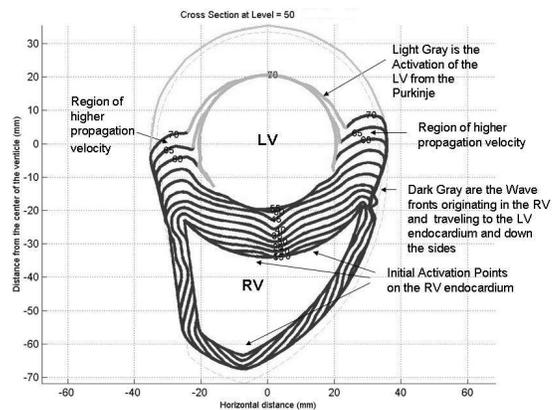


Figure 2. This shows a cross section of the Left and Right Ventricles of the heart at a level that is 50 mm from the base of the LV. The excitation is initiated at the middle of the RV septal endocardium. Lines depicting the propagating wave front are shown at 5 millisecond intervals up to 70 ms from the start. The light gray lines represent the activation of the LV resulting from the reactivation of the LV Purkinje system by the RV wave fronts.

The figure below shows the ECG results achieved for LBBB.

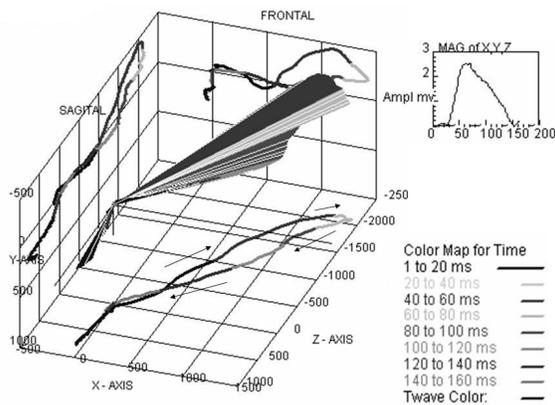


Figure 3: Simulated LBBB. A 3D Vectorcardiograph and its projection onto 3 planes: Frontal, Horizontal and Sagittal. The indicated intervals approximate the measured ECG. More importantly, the indicated vector directions are in agreement with measured values.[5]

Simulation of right bundle branch block

In Right Bundle Branch Block (RBBB), the activation of the LV proceeds normally via the Left Bundle Branch of the purkinje system whereas normal activation of the RV via the Right Bundle Branch is blocked.

RBBB is simulated by considering normal LV activation and eliminating the small RV contribution. Thus, the initial signals and vectors in RBBB approximate that seen in the normal heart. This greatly simplified the simulation problem since LV excitation software was used with little modification. Once the wave front crosses the septum from the LV endocardium, the purkinje system of the RV is reentered.

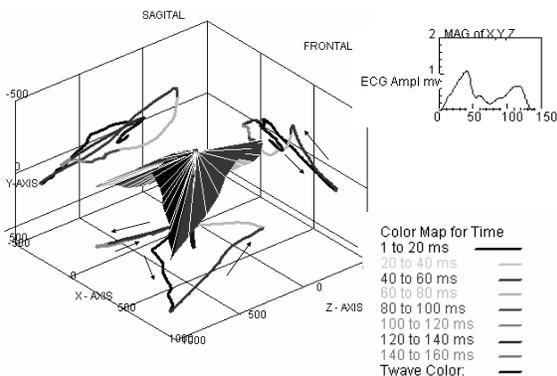


Figure 4. This is the 3D Vectorcardiograph of the simulated RBBB .

The simulated output was compared to both textbook examples of RBBB and hospital derived RBBB ECG data [5]. The results of the simulation are shown in figure 4.

Simulation of left anterior fascicular block

In Left Anterior Fascicular Block (LAFB), initial LV excitation arises solely from the Right Posterior Fascicle. The RV is activated in its normal sequence. The spreading of the surface of activation starts from the LV endocardium at points between the posterior papillary muscle and the intersection of the septum and the anterior LV free wall. This results in initial vectors pointing inferiorly and anteriorly. Shortly after this initial period, the surface of excitation begins to spread in the myocardium in a direction that is superior. This occurs as a result of the superior orientation of the longitudinal axes of the fibers in the un-activated region. These fibers conduct at a high velocity along their long axes and initially suppress the spread of activation from the purkinje fibers located on the endocardial surface of the LV. This process is responsible for the rapid change in the vector sum from inferior to superior directions.

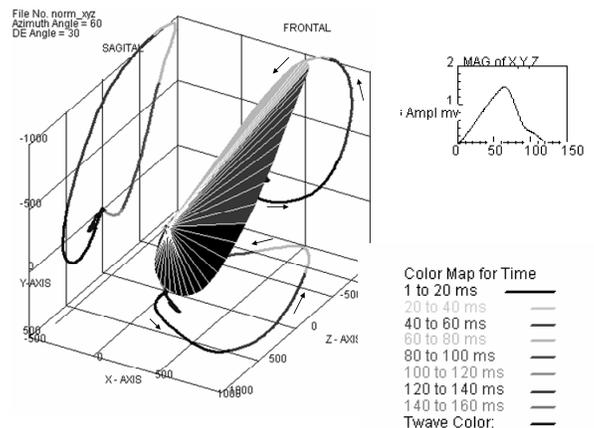


Figure 5.The 3D Vectorcardiograph of the simulation of LAFB.[5,6]

Simulation of left posterior fascicular block

In Left Posterior Fascicular Block (LPFB), initial LV excitation arises solely from the Left Anterior Fascicle. As in LAFB the RV is activated in its normal sequence. The surface of activation starts from the LV endocardium at points between the anterior papillary muscle and the midpoint of the septum. This results in initial vector pointing superiorly and anteriorly. Shortly after this initial period, the surface of excitation begins to spread

through the myocardium in an inferior direction. This occurs as a result of the inferior orientation of the longitudinal axes of the fibers in the un-activated region. As previously mentioned, these fibers conduct at a high velocity along their long axes and initially suppress the spread of activation from the purkinje fibers located on the endocardial surface of the LV. This process is responsible for the rapid change in the vector sum from superior to an inferior/posterior direction.

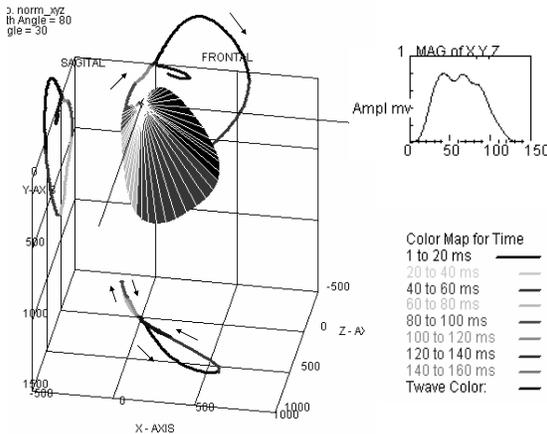


Figure 6. A 3D Vectorcardiograph of the simulated LPFB [5,6]

The plane of the vectors in the simulation is seen to be nearly vertical. This characteristic of LPFB is also seen in real patients. However, we have noted variability in the angle of the plane. This could be due to variations in the orientation of the heart in the chest, or to differences in the initial points of activation. It has recently been found that there is considerable variation in the location of papillary muscles between patients. Since the anterior papillary muscle is a key anatomic boundary of initial excitation of the LV in LPFB, this variability would explain changes in the angle of the plane between patients, as noted above.

4. Discussion and Conclusions

The investigators have developed a realistic simulation of the 4 types of conduction defects noted.

This was accomplished by detailed analyses of the influence of initial points of activation and wave front propagation dynamics on the simulated ECG. The contributions of myocardial fiber orientation and the purkinje system reentry were found to be of great significance. Comparison of the simulated VCGs and ECGs with the corresponding ECGs of real patients showed excellent agreement. A goal of the program was to generate a model that displayed the sequence of activation of the heart for use as a learning tool. This goal was also achieved and the 3D Heart software program is available to train students in understanding and recognizing conduction defects.

Acknowledgements

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