Proceedings of the 28th IEEE
EMBS Annual International Conference
New York City, USA, Aug 30-Sept 3, 2006

Simulation of Intra-Cardiac Catheter Complex Impedance Signals with Variable Stroke Volume.

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***Abstract*—In this study computer models are used for simulating electrical impedance signals from intracardiac catheter inserted to the bottom of the left ventricle of the heart. Frequency dependent models are used for tissue impedance simulation that produce complex impedance signals. 3D dynamic heart is composed of dynamic spline surfaces. Five different stroke volumes of the heart have been studied. Impedance signals from the catheter electrodes are calculated with Finite Difference Method using complex tissue impedance values at frequencies 1 kHz and 1 MHz. We were able to see clear correlation between the volume change of the left ventricle and the simulated signal. The signal magnitude change was really small – less than 0.3 %. The phase angle of the simulated signal and its' dynamic change was more pronounced at 1kHz frequency and had a small change but was a lot higher overall at 1Mz frequency. The correlation of phase angle with left ventricle volume change was clearer than signal magnitude and appeared to be less influenced by electrode positioning. We therefore conclude that phase angle of the impedance signal can contain valuable information that can be used additionally to the signal magnitude when estimating cardiac stroke volume.**

I. Introduction

Cardiac output is a direct indicator of energy demand of the organism. Cardiac output can be divided into two distinct parameters – heart rate and stroke volume. When measuring cardiac output, it is easy to determine the former but quite difficult to reliably estimate stroke volume. Stroke volume information is a valuable asset for setting heart rate by the cardiac pacemaker. It has long been proposed that electrical impedance measurement can be used for stroke volume assessment of the heart. Impedance measurement is expected to give good correlation with stroke volume if it is performed invasively by intracardiac catheter [1, 2, 3, 4, and 5]. Stroke volume information can be used then to accurately record cardiac output. It can also be used to adaptively set pacing rate by a cardiac pacemaker [6]. Computer modeling can be used for research on intracardiac impedance signals as a flexible and inexpensive tool. In this study the focus is on simulating complex-valued impedance signals consisting of modulus and phase. The complex values of the signal can also be measured in-vivo with dedicated instrumentation [7, 8]. We propose that using dynamic complex impedance information gives additional benefit in finding correlations between stroke volume and impedance signal behavior.

Manuscript submitted April 24, 2006. Resubmitted June 30, 2006. This work was supported in part by The Estonian Information Technology Foundation and Archimedes Foundation of Estonia.

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II. Methods

In this study models are used on 2 different levels. As a first level we model electrical impedance of tissues. For accurate impedance response of a tissue, electrical equivalent circuits are used. Basic idea of an electrical equivalent circuit is given on **figure 1**. Biological tissues usually have a capacitive effect when measured at higher frequencies. Low frequency current can pass only through extracellular media but higher frequencies can also penetrate the cellular walls and therefore at higher frequencies tissues have lower impedances. Cellular membranes act as capacitors, allowing more current to pass and also create a phase shift in the passing current.

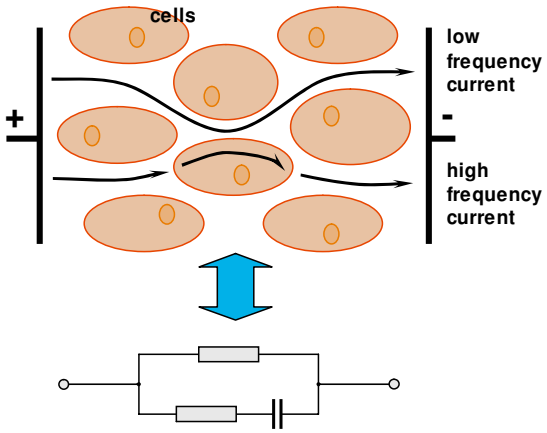

Fig. 1. Basic principle of tissue electric equivalent circuit.

In our simulation, three tissue types are used – blood, cardiac muscle and lung. Due to different cell size, shape, density and distribution, they each have different frequency characteristic of electrical impedance. In this simulation system we want to be able to use a very wide frequency range, therefore tissue equivalent circuits have been developed that cover the range 10Hz to 5MHz. To accurately represent a real tissue frequency characteristic, a lot more electric components have to be used and an alpha-parameter has to be introduced [9]. Those tissue impedance models are based on data from [10]. In this study only 2 frequencies are used for comparison - 1 kHz and 1 MHz. They were selected because of simplicity but also because they produce very

different impedance values in case of pure tissues. Frequency characteristics of the three tissues are represented in figure 2.

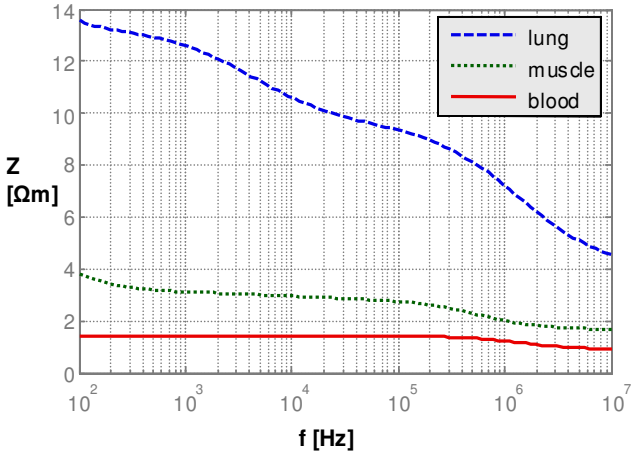

Fig. 2. Electrical impedance frequency characteristics of the three tissue models used in simulation.

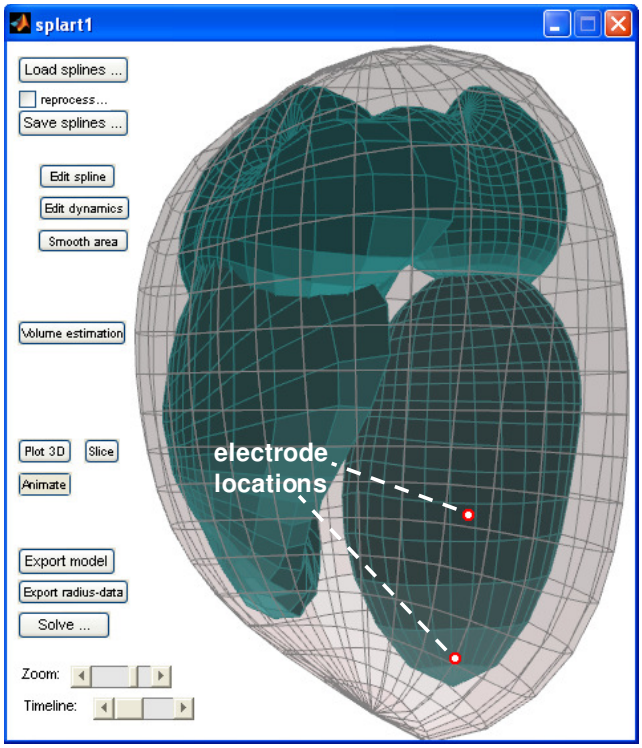

Fig. 3. GUI for visualizing and modifying the dynamic heart geometry model. Electrode locations are highlighted in the left ventricle of the heart.

The second level of modeling is the geometrical presentation of a dynamic heart anatomy. This is done by custom created GUI in MATLAB. A crude 3D heart shape was used as initial bases for creating spline surfaces for the four chambers of the heart and epicardium surface. The main interface for visualizing and modifying the spline geometry can be seen on figure 3. These tools also provide functions for introducing dynamic contractions to the heart shape. For this study five different contractions were introduced with realistic chamber volume dynamics. The cardiac cycle was chosen as in a textbook to last 0.7 seconds (the whole timescale of the signal in figures 4 and 5). The five contractions were of decreasing stroke volumes (decreased end-diastolic volumes and increased end-systolic volumes). Left ventricle volume dynamics are shown in figure 4 and described in table 1. The right ventricle volume was modeled equal to the left ventricle volume.

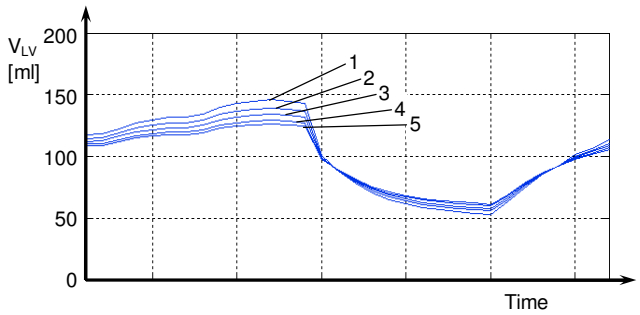

Fig. 4. Volume changes of the left ventricle with five decreasing stroke volumes.

model	end-syst. volume	end-diast. volume	ejection fraction
1	53 ml	147 ml	64 %
2	56 ml	139 ml	60 %
3	58 ml	135 ml	57 %
4	60 ml	129 ml	54 %
5	61 ml	125 ml	51 %

Table 1. Volumes and ejection fractions of the left ventricle with five decreasing stroke volumes.

Impedance signal simulation was carried out with Finite Difference Method. The volume data was composed of cubic volume elements. The modeling space was chosen a box with resolution 50-51-54 elements in x, y and z – directions. The modeling space contained the heart and some of the lung tissue surrounding it. Voxel size was 2.77 mm and the system contained 137700 elements. That resolution was close to the limit of the complex Conjugate Gradient solver in MATLAB (cgs). The solver had to be used together with incomplete Choleski preconditioner (cholinc) to achieve reasonable conversion rate. The size of the preconditioning matrix is the limiting factor in this solving method. The creation of the preconditioning matrix has heavy memory requirements and takes five times more time than the solving function itself.

Intracardiac catheter electrodes were placed close to the vertical axis of the left ventricle with the bottom electrode 5.54 mm above the left ventricle bottom and second electrode 28 mm above the first one. Electrode locations are marked on figure 3. Current was chosen to be fixed at 10 μA. Based on the impedance of the tissues, potentials where

calculated in all the nodes of the modeling space with 32 frames of motion and at the two frequencies mentioned previously.

III. Results

Results of the five cases of cardiac dynamics simulation can be viewed as voltage drop between the two electrodes used for signal input (figure 5). Other locations on the catheter can also be used for signal pickup by additional electrodes when our understanding of the system improves. Presented here is signal picked up at the electrodes in voltages. Below that is phase shift of measured potential compared to input current. Due to capacitive effects in tissues, the phase shift is negative. We present here the results of simulation as voltage drop between the electrodes. The current was fixed in all simulation cases: 32 frames of cardiac dynamics, two frequencies and five different contraction scenarios. All the results can therefore be directly translated to impedance in ohms. At 1 kHz the voltage drop magnitude has a very small change over the cardiac cycle (0.3 %) and even smaller at 1 MHz (0.1 %). This can be explained by the fact that electrodes are at a distance from the cardiac wall and majority of the current from one electrode to the other passes through the blood. The endocardial walls do not come very close to the electrodes during the cardiac cycle and so the much lower impedance of the blood plays a leading role. Same can be said for the 1 MHz case, but other factors also become evident here. The maximum difference in the signal between the five decreasing stroke volumes at 1 MHz (dynamics frame 13 in Fig. 5 C) has the same overall magnitude as the whole signal change during the cardiac cycle. The reasons for this effect are not clear at first. They might have more to do with the capacitive effects of the tissues than with resistance. On the higher frequency of 1 MHz the phase shift is generally a lot bigger (around -10 degrees) than at 1 kHz (around -0.1 degrees). Although the phase shift at 1 kHz is changing a lot more with respect to cardiac volume and exhibits a 29 % change during cardiac cycle. The higher frequency phase shift at the same time changes only 0.05 % having a lot higher value throughout the cardiac cycle. We assume this arises from the fact that at 1 kHz the blood does not produce phase shift and so the distance of the cardiac muscle to the electrodes has a big effect. At higher frequency the blood has a considerable phase shift and gives a signal with a large phase shift. Meanwhile the change of phase shift is very small because the big variability of tissue phase shift (like muscle distance at 1 kHz produced) is not present and the distance of muscle from the electrodes has much less effect.

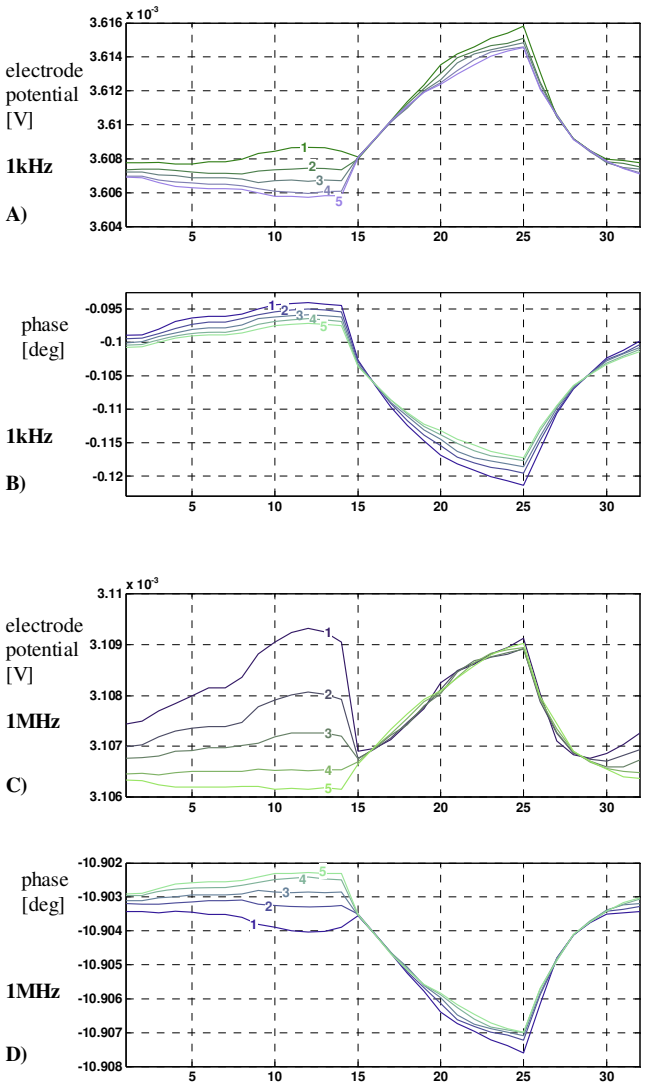

Fig. 5. Simulation results: A) impedance signal magnitude at 1 kHz, B) signal phase angle at 1 kHz, C) signal magnitude at 1 MHz, D) signal phase angle at 1 MHz.

IV. Conclusions

This modeling and simulation study has left many details unspecified and several components oversimplified. The electrode size and shape has not been introduced into the system yet. The electrode-tissue contact has not been assessed carefully. Many details are missing from the heart model and cardiac dynamics like papillary muscles, arteries, aorta and valves. Cardiac muscle impedance is still considered as isotropic. Also the pericardial sack and epicardial surface are poorly modeled. Until the modeling system has not improved in all these areas, we would not get benefit from comparing the simulated results with actual signals measured in-vivo. Nevertheless the results presented here have already provided us insight to the origins of intracardiac impedance signals. Furthermore, these results clearly show that instead of measuring only resistance, complex impedance measurement can give us much more

information on the dynamics of the heart and provide us with more data for stroke volume estimation or cardiac muscle condition monitoring. When monitoring the intracardiac impedance at several frequencies simultaneously, it should be possible to distinguish between the volume change of the ventricle and the measurement electrode position change inside the ventricle.

V. Discussion

Showing the results with magnitude and phase shift might not reveal the reasons behind certain behavior of the signal. In that case there is also the option of looking at the signal behavior as real and imaginary part of the voltage drop. Because the signals were calculated as real and imaginary parts in the first place, this view will contain the same information as magnitude-phase view. But this might give different opportunities for human intuitive reasoning.

To finally compose a stroke-volume estimation algorithm, the impedance signals have to be simulated with a wide range of possible heart shapes and motion variations. Correlations between volume and impedance signal can be searched with automatic methods. The automatically generated algorithms can then be tested with another variety of heart models. The actual 3D dynamic segmented models of cardiac dynamics anatomy are not very easy to acquire and will not be abundant enough for this type of research. Therefore this correlation research program would need to have a very flexible dynamic heart modeling system to produce all possible kinds of dynamic hearts. Several independent parameters should be taken into account, like inter-patient variability, possible pathologic conditions and also various loads and beating rates of the same heart.

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