Intracardiac Impedance as a Method for Ventricular Volume Monitoring - Investigation by a Finite-Element Model and Clinical Data

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Abstract. A method for monitoring left ventricular (LV) volume changes of the human heart by intracardiac impedance measurement was developed. In order to model this method, we simulated the ventricular contraction using a finite-element model (FEM). The myocardium comprised three layers with anatomical fiber orientation. During excitation propagation contraction forces were applied, taking into account the myocardial elastic properties and the blood pressure time course. For a set of 21 contraction stages we calculated the intracardiac impedance \(Z\) between the right ventricular (RV) and LV leads for a set of common LV lead positions. The FEM results were compared to clinical data. Impedance and stroke volume were determined during overdrive pacing and end diastolic volume (EDV) at rest in 14 patients. All clinical EDV values were in the range of 147-394ml. Both the clinical data and the FEM in this volume range showed a linear correlation between admittance \(Y=1/Z\) and ventricular volume. For a quantitative comparison end diastolic impedance EDZ and the slope \(dY/dV\) were calculated. The model results across all LV lead positions were \(EDZ=0.16-1.2\ \Omega\), and \(dY/dV=3.3-21\text{mS/ml}\), corresponding to clinical values of \(EDZ=0.14-1.46\ \Omega\) and \(dY/dV=1-64\text{mS/ml}\). In conclusion, the FEM resembled the clinical measurement data and serves as theoretical basis for ventricular volume monitoring via intracardiac impedance.

1. Introduction
Monitoring of the hemodynamic state of the circulatory system is essential for assessment of cardiovascular diseases. In particular, for heart failure (HF) patients hemodynamic monitoring is of great relevance as the ventricular pumping efficacy is impaired in these patients. A biventricular pacemaker or defibrillator frequently is implanted in HF patients with ventricular asynchrony for cardiac resynchronization therapy (CRT). CRT devices comprise pacing leads implanted in the RV and LV for synchronous stimulation of the ventricles. A method to monitor LV volume changes based on intracardiac impedance measurements between the implanted RV and LV leads was proposed [1]. The LV intracardiac impedance \(Z\) or admittance \(Y\), respectively, reflects LV volume and can be used for hemodynamic longterm monitoring with implanted CRT devices.

This paper presents a finite-element model of the human ventricles based on geometry obtained from MRI data. The model served to investigate the impedance method and to evaluate the sensitivity on
several influence parameters. LV volume and the related intracardiac impedance were computed for
the different contraction states during the heart cycle and compared to clinical data from HF patients.
A hybrid model approach was chosen to simulate the heart contraction based on a realistic ventricular
geometry and myocardial fiber structure.

2. Methods

2.1. The hybrid model
A tetrahedral mesh obtained from a static 3D-model of the human right and left ventricles in the
diastolic state was the basis for modelling the mechanical contraction of the human heart, see figure
1a. The geometry was based on cardiac MRI data. The passive mechanical parameters of the
myocardium Young’s Modulus E = 600 kPa and a Poisson Ratio $\mu = 0.485$ were used. The myocardial
surface was fixed at the rigid valve plane.

![Figure 1](image_url)

**Figure 1.** Model of the ventricular myocardium (posterior-inferior view).

a.) Outer and inner surfaces. b.) The myocardium divided into three layers to model
the anisotropic muscle fiber structure.

The bulk myocardium was divided into three layers [2], see figure 1b, to simulate the anisotropic
spatial arrangement of muscle fibers in the ventricles: an inner layer of longitudinal, a middle layer of
circular and an outer layer of diagonal fibers [3,4]. The internal layer boundaries were rigid so that no
slip between neighboring layers occurred. The fiber structure was contained in a second geometrical
model consisting of dense sets of line segments that represent the corresponding local fiber direction,
separately for each of the three myocardial layers, see figure 2.

In order to simulate the contraction of the ventricles forces resulting from the isotropic blood pressure
in the ventricles were applied on the inner layer boundary, while the outer boundary was free to
deform without additional constraints, except for the area of the valve plane, where no displacements
could occur. The time courses of the blood pressures in the ventricles were obtained from [5]. The
corresponding pressure ranges for the LV were 1.3-18.7 kPa (10-140 mmHg) and for the RV 0.7-3.7
kPa (5-28 mmHg), respectively. The directions of the forces that were applied to the tetrahedral nodes
were calculated from the fiber line segments. After deformation of the tetrahedral mesh the fiber
structure was morphed in order to adjust the line segments to the new positions of the tetrahedral
nodes. This sequence was repeated for all contraction stages. When calculating the direction of the
nodal contraction forces only line segments were considered that belonged to the same layer as the
node itself. For nodes on the rigid boundaries of the middle layer both adjacent layers contributed to
the calculation. The direction was obtained by averaging the individual directions of all fiber segments
within a given distance around the node. Having defined the nodal forces in this way the deformation
of the tetrahedral mesh was calculated by standard finite element code. In this step the myocardium was considered to be mechanically isotropic, the applied force field, however, was highly anisotropic.

**Figure 2.** Simulated fiber structures in each myocardium layer. 

- **a.)** Outer longitudinal layer.  
- **b.)** Circular fibers of the middle layer.  
- **c.)** Inner longitudinal layer.

In order to mimic the propagation of the electrical excitation over the heart, the excitation, and thus nodal force generation, started at the nodes nearest to the source of excitation in the RV apex. The excitation propagation was modeled by delaying the excitation according to the geometrical distance to the primary excitation source in the RV apex. The internal ventricular pressure was isotropically applied to the innermost fiber layer during the contraction. Eighteen discrete stages of the active contraction phase of the ventricles and three dilated stages representing heart failure conditions were calculated with a resulting ejection fraction baseline of 55%. Only the active contraction phase of the heart cycle was simulated. Passive filling and isovolumetric relaxation were not included as the main interest was the impedance-volume relation.

For impedance simulation the mechanical hybrid model was embedded in a conducting sphere that simulated the tissues of the thorax. For each step of the mechanical contraction impedance was calculated for the selected current injection configuration and various LV lead positions. As a result dynamic impedance curves were obtained. The current was injected between tip and coil of an intracardiac RV defibrillation lead and the voltage was measured between tip and ring of an epicardial LV lead (config1), see also [1]. In a second configuration current was injected between RV-coil and LV-ring and voltage was determined between RV-tip and LV-tip (config 2). Impedance was simulated for nine LV lead positions: all combinations of anterior/lateral/posterior and basal/medial/apical.

### 2.2. Clinical Study

An acute clinical study was performed to investigate the intracardiac impedance approach in heart failure patients. 14 patients with non-ischemic cardiomyopathy were included. Impedance and stroke volume were measured during graded overdrive pacing. The measurements were repeated with different LV lead positions. EDV was determined via echocardiography. For more details and patient characteristics see [6].
3. Results
The linear correlation coefficient between admittance \( Y \) (config 1) and ventricular volume of the model was \( r = 0.9 \pm 0.12 \), computed for nine different simulated LV lead positions. This result was in excellent agreement with the correlation results found in an earlier experimental animal study \( r = 0.89 \pm 0.15 \) [1]. The simulation of config2 in mid-lateral LV lead position resulted in a lower correlation coefficient of \( r = 0.74 \) compared to \( r = 0.99 \) in config1, see figure 3.

The FEM results were compared to a clinical study with 14 heart failure patients [6]. All clinical EDV values were in the range 147-394 ml (mean: 260±70 ml). The model results across all LV electrode positions were \( EDZ = 0.16-1.2 \ \Omega \), and \( dY/dV = 3.3-21 \ \text{mS/ml} \) corresponding to clinical values of \( EDZ = 0.14-1.46 \ \Omega \) (mean 0.57±0.32 \( \Omega \)) and \( dY/dV = 1-64 \ \text{mS/ml} \) (mean 14±18 mS/ml). Figure 3 depicts the simulation results of the clinically most important lateral LV lead positions in the volume range up to 300 ml.

![Figure 3. Simulated admittance for config1 and config2 in lateral LV-lead positions.](image)

4. Summary
A hybrid model was constructed to mimic the macroscopic mechanical contraction of the human ventricles to simulate intracardiac impedance signals. In the absolute impedance level and the sensitivity to cardiac volume changes a reasonable agreement between simulation results and clinical impedance measurements was achieved. Thus, the model is suitable for simulating the influence of various physiological and pathophysiological conditions on the contraction and the measured intracardiac impedance.

5. References
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