Simulation of Lung Edema in Impedance Cardiography

Mark Ulbrich[1], Jens Mühlsteff[2], Marian Walter[1], Steffen Leonhardt[1]

[1]Philips Chair for Medical Information Technology, RWTH Aachen University, Germany
[2]Philips Research, Eindhoven, The Netherlands

Abstract

Impedance cardiography (ICG) is a simple and non-invasive method to assess hemodynamic parameters. Unfortunately, this technology does not work for all patient groups. This work aimed to identify the reasons for the inaccuracy of ICG to assess the stroke volume (SV) for heart failure patients. Therefore, the effect of lung edema on the impedance cardiogram was analyzed by simulations using the finite element method (FEM). The simulation model, based on human MRI data, includes volumetric changes of heart beat and aortic expansion as well as changes during lung perfusion and erythrocyte orientation. In addition, important static tissues have been implemented. Lung tissue has been substituted stepwise by body fluid. The model had excellent correlation (r = 0.94) with measured signals and a decrease of Z_0 by 6.8% and a decrease of the peak-to-peak impedance by 54% could be observed. Hence, a decrease of the computed SV according to the standard algorithms and models is the consequence.

1. Introduction

Hemodynamic parameters such as stroke volume (SV) or cardiac output (CO) can be used to quantify the severity of cardiovascular diseases, such as heart failure (HF), which is one of the most common causes of death in Western Europe. These parameters are commonly measured utilizing a pulmonary artery catheter, which is an invasive technique. Risks of estimating CO via catheters include infections, sepsis, and arrhythmias, as well as increased morbidity and mortality [1].

Hence, an alternative method to assess hemodynamic parameters non-invasively and cost-effectively is preferred. Such an alternative could be ICG, which is not commonly used as diagnosis method, because it is not considered to provide sufficient accuracy for particular patient groups [2]. One reason is the inaccuracy of the technology itself concerning SV calculations in HF patients. Another possible reason is that the interaction and compostition of processes in the human body during ICG measurements are widely unknown. One way to analyze effects inside the human body contributing significantly to the measurement result, is to use computer simulations. Other researchers have already examined multiple sources of the ICG signal, using different approaches: some works are based on simple geometries [3], others on real anatomical data, such as MRI data [4].

To our knowledge, the influence of pathologies on the ICG signal has never been taken into account. Within this work, lung edema as a symptom of HF shall be analyzed according to its impact on the impedance signal using a dynamic human thorax model based on MRI data.

2. Methods

In this section, the basics of ICG as well as the FEM simulation approach, the simulation model and its parameterization will be explained.

2.1. Impedance cardiography

ICG is a bioimpedance measurement technique which aims to assess the cardiac health status. The impedance is measured on the thorax by injecting a low harmless current by two outer electrodes and two other inner electrodes measure the voltage drop. Standard adhesive electrodes, known from ECG, are placed on neck and abdomen. By measuring the impedance continuously at one fixed frequency around 100 kHz, time-dependent hemodynamic parameters can be extracted from the measured impedance curve using its temporal derivative (see fig. 1).

To extract the SV using these characteristic points, several model assumptions have been made by different research groups. The oldest model approximates the human thorax by one outer cylinder with a mean conductivity and permittivity with respect to the thorax' tissues, and one inner cylinder representing the aorta. With this assupmtion, the SV can be calculated using the following equation:

$$SV = \frac{1}{\sigma_b} \cdot \left(\frac{l}{Z_0}\right)^2 \cdot \left|\frac{dZ}{dt}\right|_{max} \cdot t_{LVET} \qquad (1)$$

Here, σ_b is the conductivity of blood, l the distance between upper and lower electrodes, t_{LVET} the left ventric-

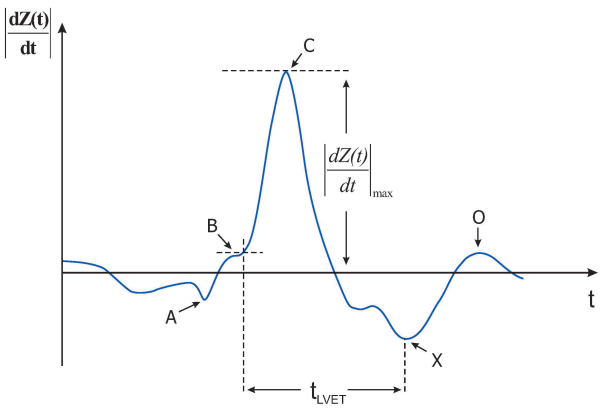

Figure 1. ICG wave ($|Z(t)|'$) with its characteristic points [5]

ular ejection time (X minus B-point), Z_0 the static base impedance and $\left|\frac{dZ}{dt}\right|_{max}$ the maximum of the impedance's temporal derivative [6]. There are other models providing other equations, but they all have in common that they depend on Z_0, $\left|\frac{dZ}{dt}\right|_{max}$, t_{LVET} and individual body characteristics.

2.2. FEM simulations

2.2.1. Simulation setup

The finite element method (FEM) has been chosen to simulate current flow and potential distribution during an ICG measurement on the human thorax. CST EM Studio® from Computer Simulation Technology, Darmstadt, Germany was used to perform the simulations which have been computed on a personal computer with a 64 bit operating system, an Intel® Xeon® 5240 processor with 2 cores and 24 GB RAM. The excitation frequency has been set to 100 kHz. Since this wavelength is much higher than our measuring volume, the human thorax, the low frequency electroquasistatic solver has been employed.

To calculate the complex impedance, a voltage source has been used to define the voltage at the electrode sites. In addition, the current has been calculated by integrating the current density and the displacement current over a predefined area, assuming a harmonic oscillation ($\vec{D} = |D| \cdot e^{j\omega t} \cdot \vec{e}_z$). Thus, the complex current was calculated using the following equation:

$$\underline{I} = \int_A \left(\vec{J} + \frac{\partial \vec{D}}{\partial t} \right) \cdot d\vec{A} = \int_A \left(\vec{J} + j\omega \vec{D} \right) \cdot d\vec{A} \quad (2)$$

Using a discretization density of 50 units, each model comprises 1.5 million tetrahedrons. Besides this global discretization, a finer local discretization has been used for shape changing organs in order to obtain accurate results for small geometry changes. The temporal resolution has been set to 125 Hz.

2.2.2. Simulation model

Our thorax model is composed of simple geometries, such as frustums, spheres and cylinders. This approach ensures a fast calculation time and a fast model creation. This is important, since for every point in time a new model has to be created because geometric changes were incorporated to model phsyiological processes. This results in 103 models for the chosen temporal resolution. Important tissues have been realized: fat, muscle, bone, lung tissue, abdominal organs and blood vessels. In addition, dynamic volumes for aorta and heart have been considered as well. Rebuilding all organs situated in the abdomen by importing surface polygons would have been too complex for the desired model. In addition, the impedance change of these organs plays a subordinated role for the signal during one heartbeat so that the simplification has been made to fill the abdominal part of the thorax with a uniform "tissue mixture" by using Boolean operations. An average permittivity and conductivity of all abdominal organs has then been assigned to this tissue (see tab. 1).

Table 1. Permittivity and conductivity values at 100 kHz [7].

	σ [S/m]	ϵ_r
Blood	0.70292	5120
Myocard	0.21511	9845.8
Bone	0.020791	227.64
Fat	0.024414	92.885
Muscle	0.36185	8089.2
Abdomen	0.2	4000
Lung	0.10735	2581.3
Body fluid	1.5	97.99

All shapes of organs and tissues are based on the dataset from the Visible Human Project© from the National Library of Medicine in Maryland, USA [8]. It provides voxel data with different resolutions from 1x1x1 mm^3 to 8x8x8 mm^3. The origin of this data is an executed prisoner who has been frozen into gelatin after death. His frozen body was then cut into slices, so-called cryosections. These slices have been digitized by using MRI and CT with an high spatial resolution.

To simulate aortic expansion and contraction and relaxation of the heart, dynamic volumes have been implemented. Since the expansion of the aorta is proportional to the aortic blood pressure (see eq. 3), real measured data from PhysioNet [9] has been used as basis for the aortic

expansion [10].

$$\Delta R = \frac{\Delta P \cdot R_0 \cdot extensibility}{100} \quad (3)$$

Here R_0 is the diastolic radius of the aorta. The aortic blood pressure has then been scaled to fit the requirement for the maximum aortic expansion so that the diameter of the aorta varies between 25 and 30 mm.

The radius of a sphere representing the heart has been altered using the left ventricular volume change obtained by MRI data while assuming a stroke volume of 80 ml [11]. The radius varies between 38.5 mm and 33.8 mm and shall mimic the contraction of the heart.

The conductivity change due to lung perfusion has been implemented by assuming a maximum conductivity change of 10 % [12]. Based on measured conductivity changes with electrical impedance tomographic spectroscopy (EITS), a dataset comprising 103 points in time has been created using Matlab [13]. Due to the alignment of erythrocytes along a laminar flow during high velocities inside blood vessels, blood conductivity changes during the heart beat. Hence, a blood resistivity change of 15% due to erythrocyte orientation has been implemented using published data [14]. Using a diastolic conductivity of 0.70292 S/m, a maximum conductivity of 0.80836 S/m during systole is achieved.

To simulate cardiac lung edema as a result of left heart failure, lung tissue was substituted in four steps with body fluid by 14%, 42%, 70% and 85%. Figure 2 shows the thorax model with semi-transparent tissues and highlighted lung tissue. In this figure, 42 % of the caudal lung tissue have been substituted with body water, assuming that the person is in upright position.

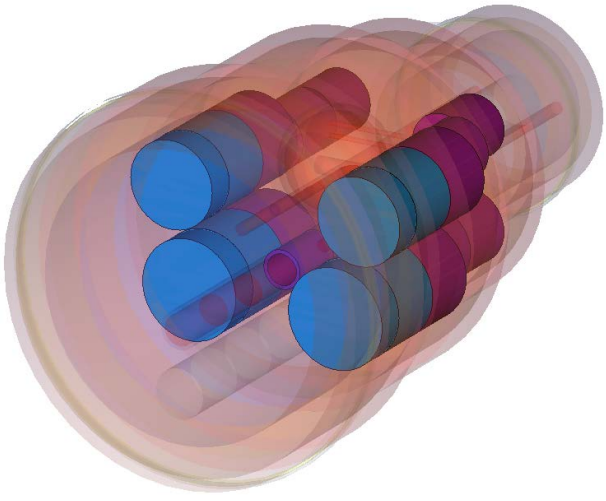

Figure 2. Thorax model showing lung with substituted body water

3. Results

For every filling stage, 103 simulations have been conducted, resulting in 515 simulations. Figure 3 shows the normalized impedance changes of the absolute value of the impedance during one heartbeat. The healty state (0%) as well as the four pathologic stages are plotted versus time.

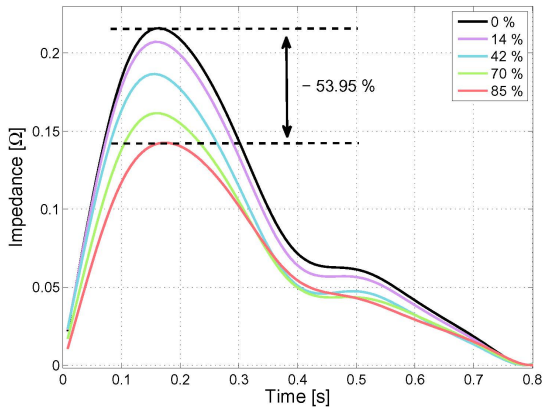

Figure 3. Simulated impedance changes

The peak-to-peak value of the healty state ($0.21\ \Omega$) lies within the range of normal measured values. One can see, that for every subsequent filling stage, the impedance decreases. Comparing healty and maximum filling stage (85%), an impedance decrease of 54% can be observed. The morphology of the signal changes only slightly. The base impedance Z_0, which is not included in fig. 3, decreases by 6.8% from $58.9\ \Omega$ to $54.9\ \Omega$. To calculate t_{LVET}, the temporal derivative of all simulations has been calculated an plotted against time (see fig. 4).

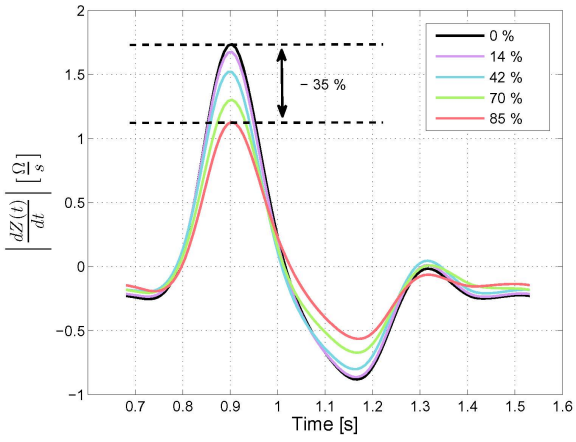

Figure 4. Simulated ICG changes

As for the impedance progression, the maximum of its

temporal derivative $\left|\frac{dZ}{dt}\right|_{max}$ decreases for every filling stage of the lung. In total, a change of 35% could be observed. To analyze the influence of lung edema on the t_{LVET}, B and X-point of the ICG curve have been extracted using standard algorithms found in literature. Comparing the t_{LVET} for every stage, no significant change could be observed. All results are summarized in the following table:

Table 2. Simulation results.

	0%	14%	42%	70%	85%
$\Delta Z[m\Omega]$	215	199	165	129	99
$Z_0[\Omega]$	58.86	58.38	57.25	55.97	54.85
$\left\|\frac{dZ}{dt}\right\|_{max}$	1.74	1.68	1.52	1.3	1.13
$B[ms]$	730.3	722.1	722.1	722.1	738.6
$X[ms]$	1167.7	1167.7	1159.5	1167.7	1176
$LVET[ms]$	437.4	445.6	437.4	445.6	437.4

4. Discussion and conclusion

The task of this work was to analyze the influence of lung edema on the impedance cardiogram using FEM simulations.

First, a model has been modified to suit the needs for the simulation of lung edema by implementing a substitution of lung tissue by body water using four filling degrees. This model provides a high temporal resolution to extract important characteristic points of the ICG curve. Second, these characteristic points were successfully extracted using standard algorithms. Thus, the influence of lung edema on the parameters t_{LVET}, Z_0 and $\left|\frac{dZ}{dt}\right|_{max}$ could be analyzed. These parameters are important, since they contribute in all models essentially to the calculated SV. Since t_{LVET} remained constant for every filling stage and Z_0 as well as $\left|\frac{dZ}{dt}\right|_{max}$ decreased, a decrease of the computed SV according to the standard algorithms and models is the consequence. This is a possible explanation for the inaccuracy of ICG concerning heart failure patients.

Acknowledgements

This work was funded by "HeartCycle", an EU-project about compliance and effectiveness in Heart Failure (HF) and Coronary Heart Disease (CHD) closed loop management and has been supported by Philips Research Europe.

References

[1] Darovic G. Hemodynamic Monitoring: Invasive and Noninvasive Clinical Application. Saunders, 2002. ISBN 978-0721692937.

[2] Cotter G. Impedance cardiography revisited. Physiological Measurement 2006;27:817–827.

[3] Kosicki J. Contributions to the impedance cardiogram waveform. Annals of Biomedical Engineering 1986;14:67–80.

[4] Patterson R. Sources of the thoracic cardiogenic electrical impedance signal as determined by a model. Medical Biological Engineering Computing 1985;23(5):411–417.

[5] Packer M. Utility of impedance cardiography for the identification of short-term risk of clinical decompensation in stable patients with chronic heart failure. Journal of the American College of Cardiology 2006;47:2245–2252.

[6] Bernstein D. Stroke volume equation for impedance cardiography. Medical Biological Engineering Computing 2005; 43:443–450.

[7] Gabriel C. The dielectric properties of biological tissues. Physics in Medicine and Biology 1996;41:2231–2249.

[8] National Library of Medicine. The visible human project. URL `http://www.nlm.nih.gov/research/visible/visible_human.html`.

[9] National Institute of Biomedical Imaging and Bioengineering. Physionet - the research resource for complex physiologic signals. URL `http://www.physionet.org/`.

[10] Greenfield J. Relation between pressure and diameter in the ascending aorta of man. Circulation Research 1962; 10:778–781.

[11] Feng W. A dual propagation contours technique for semiautomated assessment of systolic and diastolic cardiac function by cmr. Journal of Cardiovascular Magnetic Resonance 2009;11.

[12] Brown B. Simultaneous display of lung ventilation and perfusion on a real-time eit system. Engineering in Medicine and Biology Society Proceedings of the Annual International Conference of the IEEE 1992;5:1710–1711.

[13] Zhao T. Modelling of cardiac-related changes in lung resistivity measured with eits. Physiological Measurement 1996;17:227–234.

[14] Raaijmakers E. The influence of pulsatile blood flow on blood resistivity in impedance cardiography. 18th International Conference of the IEEE Engineering in Medicine and Biology Society 1996;5:1957–1958.

Address for correspondence:

Mark Ulbrich
Lehrstuhl für Medizinische Informationstechnik
Pauwelsstrasse 20, Room 0.11A
52074 Aachen, Germany
ulbrich@hia.rwth-aachen.de