Analysis of the Aortic Influence on the Impedance Cardiography Signal by a Simple Model using Finite Integration Technique

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Chronic heart failure is a serious disease in our society which can potentially be diagnosed by impedance cardiography (ICG). However, there is a strong debate about using this technology. In this work, the capability of a simple model to generate the ICG signal by taking the aorta as major signal source has been analyzed. Therefore, a simulation with a high temporal resolution has been conducted using the finite integration technique (FIT). It has been shown that although neglecting other physiologic signal sources, the results obtained from the simulations produce an ICG signal which correlates very well with measured ICG signals.

Introduction

Since the beginning of the 20th century, a demographic change is observable in Europe which leads to a steadily aging society. Hence, society will have to face more and more geriatric patients and diseases, which leads to increasing costs and burdens on medical personnel. Thus, it is reasonable to establish methods to treat elderly people more cost-effectively and more easily by improving diagnostics and monitoring for certain diseases that prevalently occur among elderly people.

Cardiovascular diseases are the most common cause of death in Western Europe. One of these diseases is chronic heart failure (CHF) from which 2 million people are suffering and this number increases every year by half a million. The basic definition of CHF is a state in which the heart is not able to pump enough blood into the periphery to supply it with oxygen. Reasons for that can be dysfunctions concerning filling, contractility or emptying of the ventricle. Measures for these dysfunctions are hemodynamic parameters such as cardiac output (CO) or stroke volume (SV).

Until now, the gold standard for measuring these parameters is the thermodilution technique which utilizes a pulmonary artery catheter [1]. However, risks of estimating CO via catheters include infections, sepsis, and arrhythmias, as well as increased morbidity and mortality. A technology to acquire SV or CO non-invasively is echocardiography. Here ultrasound images are used to calculate these parameters by a physician.

A very promising technology to measure cardiac output and other hemodynamic parameters easily and non-invasively is impedance cardiography (ICG). Although this technology is known since the 1960s, it is not used in clinical practice today. One reason is that processes in the human body during ICG measurements are widely unknown, which means e.g. that the composition of physiological processes as contributors to the ICG signal is very complex. Another reason is that impedance cardiography is considered to provide unreliable results [2]. One way to analyze where the current paths run, and which tissue contributes significantly to the measurement result, is to use computer simulations employing FIT.

Other researchers have already examined multiple sources of the impedance cardiography signal, using different approaches. Some works are based on simple geometries [3], others on real anatomical data, such as MRI data [4, 5]. The examined sources comprise heart volume and position, diameter of large vessels, changes in conductivity of the lungs as they fill with blood, and shear rate dependent changes in conductivity of the blood in large vessels. Besides identifying sources, it is important to know to which extent the various sources contribute to the resulting signal. A recent study suggests, that the genesis of the impedance cardiogram is attributed primarily to volumetric expansion of the aorta [6].

Since controverse results have been obtained before, a simple model shall be analysed within this work to clarify if it is sufficient to explain the origin of the ICG signal. Therefore, physiologic signals with a high temporal resolution shall be used.

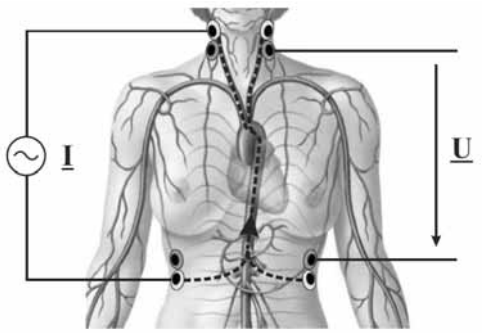

Figure 1. ICG measurement

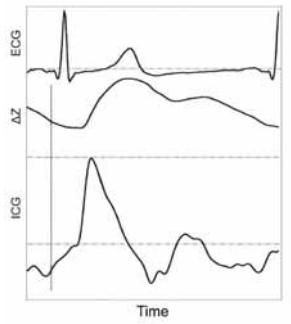

Figure 2. ECG, ΔZ and ICG waves

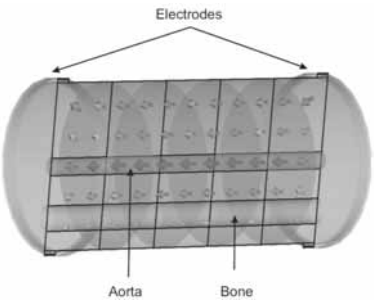

Figure 3. Simulation model.

1 Impedance Cardiography

ICG is a method for measuring hemodynamic parameters at one fixed frequency between 20 and 100 kHz. Similar to a bioimpedance analysis, tetrapolar electrodes are used to measure the complex bioimpedance. Therefore, one electrode pair is used to inject the current and the other to measure the voltage of the thorax so that four electrodes have to be used for one measurement (cf. Figure 1). In practice, 8 electrodes are used, because the pairs of current injecting electrodes act as one current source and the voltage sensing electrodes lie on the same equipotential lines.

The complex impedance is dependent on electrode position and quality, skin thickness, sweat and pathologies. In contrast to bioimpedance analysis, the thoracic impedance has to be acquired continuously because its temporal derivation contributes to the stroke volume.

The measured stroke volume according to Bernstein and Sramek can be described by the following equation:

$$SV = \delta \cdot \frac{(0.17)^3}{4.2} \cdot \left|\frac{dZ}{dt}\right|_{\max} \cdot \frac{t_e}{Z_0} \qquad (1)$$

Here the factor δ is the actual weight divided by the ideal weight, t_e the left ventricular ejection time (LVET) and Z_0 the thoracic base impedance [7]. Two local minima of the ICG signal are used to calculate the LVET [8]. Figure 2 shows a typical impedance signal and its temporal derivation.

2 Methods

Classical impedance cardiography analyzes the thoracic impedance assuming the thorax to be composed of two cylinders: one inner cylinder representing the aorta and outer cylinder with one conductivity for all other tissues.

This is of course an assumption which leads to modelling errors [10]. Hence, new models with different approaches have been created.

In this work, the accuracy of the classical ICG model shall be analyzed using FIT simulations with a high temporal resolution and an anatomical data set of a male human to create an improved model. Other works mostly concentrate on systolic and diastolic events only [11]. The simulation program used for this is CST EM Studio® from Computer Simulation Technology in Darmstadt, Germany.

The simulation setup is based on the original model of Kubicek et al. and contains three cylinders [10]. The diameter of the outer cylinder (260 mm) is based on the size of the visible male's thorax, the diameters of the inner cylinders on the size of his aorta (25 mm) and spine (40 mm). The male data set is based on the Visible Human Data Set from the National Library of Medicine in Maryland [12]. The model used for the simulation results is shown in Figure 3. It consists of the materials bone, blood and tissue with the conductivities:

$$\sigma_{\text{bone}} = 0.05 \frac{\text{S}}{\text{m}}\,\sigma_{\text{blood}} = 0.7 \frac{\text{S}}{\text{m}}$$

$$\sigma_{\text{tissue}} = 0.25\ \text{S/m}.$$

These values are based on empirically assessed values by Gabriel et al. at 70 kHz [13]. For the value for the bone tissue, it has been taken into account that bone consists of bone marrow, cancellous and cortical bone tissue.

For every expansion step a new model has been created using an aortic diameter increase of 20% as the maximum [14]. In addition, the expansion of the surrounding tissue has been taken into account by increasing its radius by the following equation:

$$r_{\text{tissue}} = \sqrt{(130\ \text{mm})^2 - (12.5\ \text{mm})^2 + r_{\text{aorta}}^2} \qquad (2)$$

To facilitate the creation of the different models, a script has been written using WinWrap Basic included in CST Studio, so that all 103 points in time could be simulated automatically. Every model had a mesh density of approx. 980000 tetrahedrons.

Since the expansion of the aorta is proportional to the aortic blood pressure (cf. eq. 3), real measured data from PhysioNet [15] has been used as basis for the aortic expansion [16].

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

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. For every point in time, the impedance of the whole setup has been calculated so that the impedance depends on the expansion of the aorta only.

3 Results

For every point in time, the complex impedance of the simulation model has been calculated. To get correct impedances, a correction factor has to be considered resulting from an increase in simulated impedances due to the size of electrodes [17]. In addition, the resulting curve for the real values of the impedances has been derivated to get the impedance cardiogram. Both curves are shown in Fig. 4. The magnitudes of impedances were found to be in the range of physiological values ($37.3 < Z\,[\Omega] < 37.8$).

The last curve comprises three heart cycles and one cycle has been used for a cross correlation analysis with ICG raw data from a measured male subject (cf. Fig. 5). The thoracic impedance was acquired using a "niccomo" patient monitor [18].

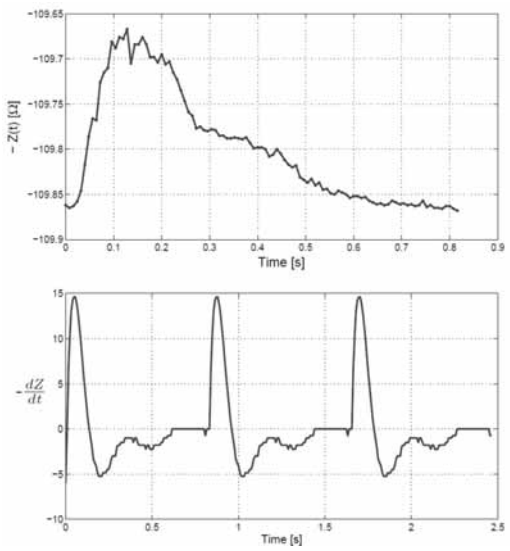

Figure 4. Simulated resistance and ICG

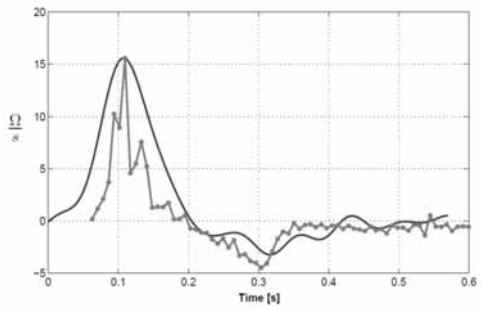

Figure 5. Comparison between simulated (red) and measured ICG (blue)

For the analysis, the height of the simulated curve has been scaled to fit the height of the measured curve. This must not be considered as a source of error because it is only taken into account that hemodynamic parameters vary interindividually. Comparing the two time signals, the correlation coefficient without lag is 0.88, proving what Fig. 5 suggests visually.

4 Discussion

The task of this work was to find out whether the assumption to take the aorta as the major contributor for the impedance cardiography signal is correct or not.

A simple dynamic simulation model has been created to simulate the aortic expansion during a heartbeat using a high temporal resolution. The dynamics of this model is based on measured data of a subject.

Despite the very simple model of the human thorax, comprising only a cylindric aorta and an outer cylinder representing all other tissues, it could be shown that the aorta plays indeed a very important role in the physiological ICG signal generation as the major contributor, which corresponds to a recent study in which the impedance change of a dog's aorta has been measured in vivo [6]. This is an astonishing result because other works have shown that only about 0.7 % of the measured impedance change of the thorax are caused by the aorta [5].

5 Outlook

Models being created to overcome the inaccuracy of today's ICGs should take into account that the aorta is the major contributor of the ICG signal. In addition, future works shall show to which extent other dynamic signals inuence the ICG signal (e.g. volume changes of heart and lung) and which static values contribute to the non-dynamic part of the signal. What is more, changes in the ICG signal caused by pathologies such as lung edema and the possibility of the simulated signal to reect the characteristic points should be analyzed.

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