Feasibility of Impedance Cardiography to Assess Hemodynamic Changes and Fluid Loss Related to Pleural Drainage

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Abstract

This work aims to verify if impedance cardiography (ICG) as a non-invasive measurement method for hemodynamic parameters is feasible for the detection of hemodynamic changes and fluid loss due to pleural drainage in heart failure (HF) patients with pleural effusion. Thus, stroke volume (SV) and thoracic fluid content (TFC) before and after pleural drainage were compared. 6 patients with pleural effusion as implication were treated and by the insertion of an intercostal drain, between 1 and 2 liters of fluid were removed from the pleural space in sitting position. ICG measurements were made before and after the drainage by injecting a sinusoidal current of 1.5 mA at a frequency of 85 kHz into the body. SV was calculated according to Kubicek, Sramek and Bernstein. TFC was calculated using the static base impedance (Z_0). A paired-sample t-test was made at 5 % significance level. The calculated SV difference varies between 45 ml and 17 ml, whereas Z_0 changes between $0.9\ \Omega$ and $2\ \Omega$. The t-tests showed significant changes of SV ($p \leq 0.0035$) and TFC ($p \leq 0.0014$). All measured and calculated values show significant changes before and after pleural drainage.

1. Introduction

Cardiac decompensation and hospitalization are responsible for 75 % of the costs caused by heart failure. Hence, a goal should be to establish an early warning system to prevent cardiac decompensation. Hemodynamic parameters such as stroke volume or cardiac output can be used to quantify the severity of heart failure. These parameters are commonly measured by an invasive pulmonary artery catheter [1]. An alternative method to assess hemodynamic parameters non-invasively and cost-effectively is preferred. ICG could be such a technology; integrated in daily life, e.g. into a textile or furniture, measured hemodynamic parameters by ICG could predict cardiac decompensation. However, ICG is not commonly used as a diagnostic method because it is not considered to be valid for heart failure patients [2]. In this work, the potential of ICG to assess hemodynamic parameters in heart failure patients with pleural effusion as specialized setting is analyzed.

2. Methods

A clinical trial was set up to verify whether ICG could be used as preventive measure for high risk patients of decompensation. The trial was approved by the local ethics commitee (Clinicaltrials.gov ID: NCT01778270) and a written informed consent was obtained from patients. Six patients suffering from excessive pleural effusion as comorbitity of heart failure were examined using ICG. The patient data including the amount of drained fluid is shown in table 1.

Table 1. Patient data.

Patient	Age	Height [cm]	Weight [kg]	Gender	Drained fluid [l]
1	73	165	66	w	1.5
2	62	178	71	m	1.2
3	66	180	89	m	1.5
4	64	176	107	m	2
5	68	163	63	w	1.7
6	58	173	101	m	1.7

Measurements were made in sitting position before and after pleural drainage. Electrode positions were cleaned with alcohol before electrodes were attached at neck and abdomen (standard positions). Subsequently, relevant basics related to the trial will be explained.

2.1. Impedance cardiography

ICG is a bioimpedance measurement technique which aims to assess the cardiac health status by estimating stroke volume and other hemodynamic parameters. The impedance is measured on the thorax by injecting a current by two outer electrodes and two other inner electrodes

measure the voltage drop with standard adhesive Ag/AgCl electrodes placed on neck and abdomen. By measuring the impedance continuously at one fixed frequency, time-dependent hemodynamic parameters are extracted from the measured impedance curve using its temporal derivative (see fig. 1).

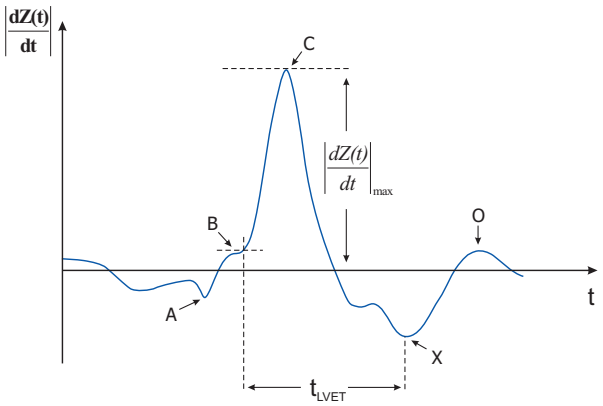

Figure 1. ICG wave ($-Z(t)'$) with its characteristic points [3]

As shown in this figure, the B-point, reflecting the aortic valve opening, is assumed to be located at the inflection point before the maximum of the curve. However, other locations are assumed to be more precisely related to their physiological correspondent within the cardiac cycle. The same applies to the X-point since the absolute minimum after the C-point is only one possible extraction point [4]. To estimate the stroke volume using these characteristic points, several model assumptions have been proposed. The oldest model approximates the human thorax by cylinders representing thorax and aorta. Other models use a frustum to represent the thoracic volume.

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

$$SV = \frac{(0.17 \cdot H)^3}{4.2} \cdot \left|\frac{dZ}{dt}\right|_{max} \cdot \frac{t_{LVET}}{Z_0} \tag{2}$$

$$SV = \delta \cdot \frac{(0.17 \cdot H)^3}{4.2} \cdot \left|\frac{dZ}{dt}\right|_{max} \cdot \frac{t_{LVET}}{Z_0} \tag{3}$$

$$SV = \frac{V_{ITBV}}{\zeta^2} \cdot \sqrt{\frac{\left|\frac{dZ}{dt}\right|_{max}}{Z_0}} \cdot t_{LVET} \tag{4}$$

Equations 1 to 3 are based on the assumption that impedance changes are mainly caused by volume changes in the thorax while equation 4 relates the impedance change mainly to blood acceleration [5]. Other models provide 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. Here, σ_b is the conductivity of blood, l the distance between upper and lower electrodes, H the height of the patient, δ a body mass correction factor, V_{ITBV} the intrathoracic blood volume, ζ the index of transthoracic aberrant conduction, t_{LVET} the left ventricular 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 [5]. To calculate TFC, the inverse of Z_0 is multiplied with 1000:

$$TFC = \frac{1000}{Z_0} \tag{5}$$

2.2. Pleural effusion

The inner and outer pleura form a continuous double layered membrane which envelops the lungs and lines the thoracic cavity. The pleural space normally contains 10 ml of pleural fluid produced by the membranes. This allows the pleural layers to slide easily over each other during respiration. A negative pressure exists between the inner and outer pleura which provides suction and results in the lung being held close to the chest wall [6].

Pleural effusion develops by the collection of large amounts of free fluid in the pleural space because more fluid enters the pleural space than is removed. It is analogous to edema fluid in tissues and can be called edema of the pleural cavity. A potential mechanism of pleural fluid accumulation is increased interstitial fluid in the lungs secondary to increased peripheral and pulmonary capillary pressure (a symptom of heart failure) or permeability. Other mechanisms are: decreased intrapleural pressure, decreased plasma oncotic pressure, increased pleural membrane permeability and obstructed lymphatic flow, diaphragmatic defects and thoracic duct rupture [7, 8].

Common symptoms of an effusion are dyspnea, cough, and pleuritic chest pain and these vary depending on the underlying disease.

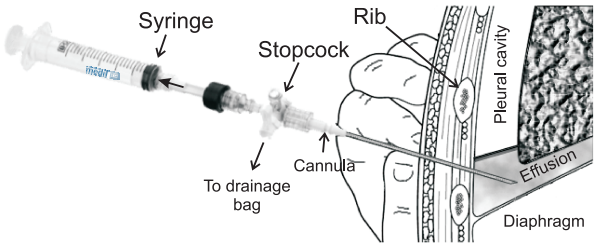

Figure 2. Thoracentesis (pleural drainage)

The treatment of a severe pleural effusion involves draining it. This intervention is called thoracentesis. When the insertion area has been anaesthetised, a very small cut in the chest is applied and a cannula inserted until the needle reaches the effusion area. The cannula is attached to a

tube and drainage bag. The fluid is drained out of the chest by a syringe and is collected in a bag (see figure 2). In an animal trial it was shown that stroke volume increases after pleural drainage [9].

3. Results

Figure 3 shows measured impedance curves normalized by subtracting Z_0 of one patient before and after drainage.

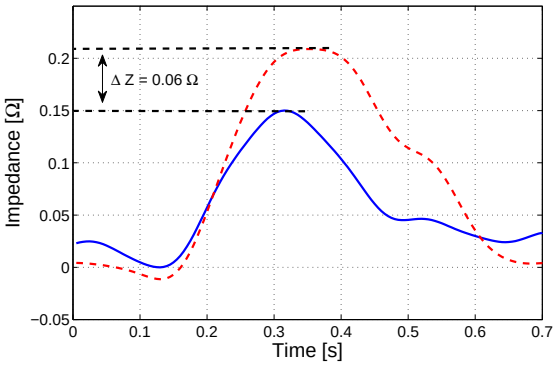

Figure 3. Measured impedance before and after drainage (dashed)

The peak-to-peak impedance increased after drainage, here by 0.06 Ω. In addition, the morphology of the measured impedance curves allowed the extraction of all standard characteristic points in order to calculate stroke volume by using the zero-crossing of the ICG curve as B-point and the global minimum of the curve as X-point (see figure 4).

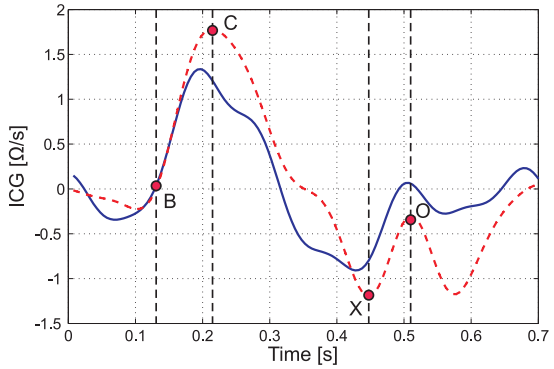

Figure 4. Calculated ICG before and after drainage with characteristic points (dashed)

This figure also shows that not only $|dZ/dt|_{max}$ increases after drainage, but also a temporal shift of characteristic points influencing e.g. LVET.

In table 2, mean values of all patients of LVET, Z_0, TFC and stroke volume are listed.

Table 2. Mean values before and after drainage.

		Before	After
LVET [ms]		306.7	330.5
$\|dZ/dt\|_{max}$ $[\Omega/s]$		1.33	1.7
Z_0 $[\Omega]$		29.9	31.7
TFC $[1/\Omega]$		33.4	31.5
SV [ml]	Kubicek	46.2	72.6
	Sramek	77.8	106.3
	Bernstein-Sramek	76.8	101.7
	Bernstein-Osypka	86.1	101.4

In general, all measured values lie in normal physiological ranges. LVET varied between 197 ms and 413 ms, Z_0 between 21.2 Ω and 45.4 Ω, and stroke volume between 40 ml and 140 ml. Figure 5 shows a boxplot of Z_0 values for all patients.

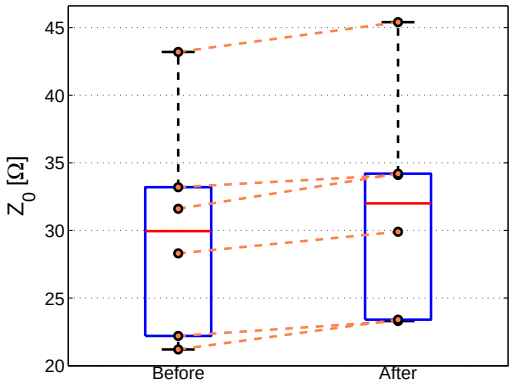

Figure 5. Measured base impedance before and after drainage

Although there is a big overlap of measured values before and after drainage, a positive trend for each patient can be observed (dashed lines between data points). The stroke volume depicted in figure 6 also has a positive trend for each patient, but this trend is already visible when comparing all values before and after drainage since there is a minimal overlap.

Although figure 7 visually implies positive trends for each data pair, there is no positive LVET trend for every patient.

Since the Lilliefors-test yielded that all parameters are normally distributed, a paired-sample t-test was made at 5 % significance level. The results rejected the null hypothesis and thus proved that stroke volume ($p \leq 0.0035$) and TFC ($p \leq 0.0014$) before and after pleural drainage are significantly different.

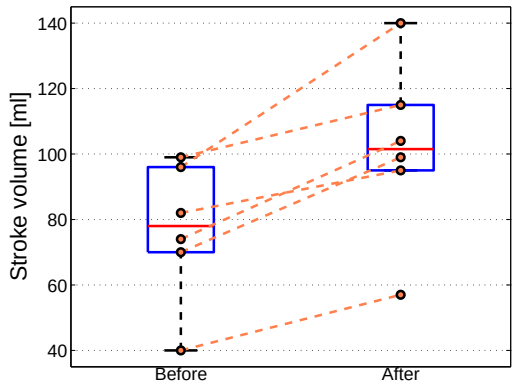

Figure 6. Measured SV (Bernstein-Sramek) before and after drainage

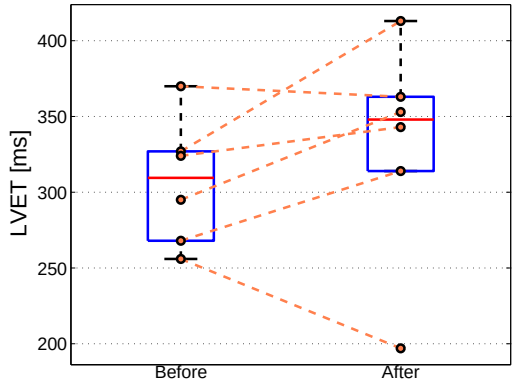

Figure 7. Measured LVET before and after drainage

4. Discussion and conclusion

The potential of ICG to predict cardiac decompensation by detecting pleural effusion was analyzed in this work. A clinical trial with heart failure patients and pleural effusion was conducted by measuring ICG before and after drainage. Since a t-test revealed that calculated TFC and stroke volume show significant changes before and after pleural drainage, ICG reflects the influence of the removed fluid of the thorax. It should be noted that the absolute stroke volume might not reflect the real stroke volume since no reference measurements could be conducted due to ethical reasons.

Interestingly, time seems not to have a high impact on the measurement results since some patients could be measured directly after drainage and some only on the next day. During the time between drainage and last measurement one could expect lots of other influences on the impedance, e.g. ingestion or a fluid redistribution in the body but the results of all patients show the same trend.

Since ICG obviously is suitable to detect heart recovery after the drainage of a pleural effusion, ICG might be a valuable technology to predict decompensation in HF patients prospectively by detecting pleural effusion. This could be accomplished by integrating this technology in the daily life of high risk patients in a home care scenario.

Acknowledgements

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