Elimination of the Respiratory Effect on the Thoracic Impedance Signal with Whole-body Impedance Cardiography

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Abstract

The study presents a method for quantification of the respiratory effect on the thoracic impedance signal. We used measurements with 18 simultaneously scanning impedance signal locations. Elimination of breathing is based on the assumption that the neck and limb impedance signal measures only the variability in systemic blood pressure and is not affected by respiration. The results show approximately double the respiration variability in thorax impedance parameters in comparison with legs and neck. Conclusion: Whole-body impedance signal measurements and processing reflect changes in the hemodynamic system not affected by respiration. By using retrospective reconstruction we can eliminate the influence of respiration on the impedance signal from the chest.

1. Introduction

Respiratory sinus arrhythmia (RSA) is heart rate variability in synchrony with respiration, by which the R-R interval on an ECG is shortened during inspiration and prolonged during expiration. Although RSA has been used as an index of cardiac vagal function, it is also a physiologic phenomenon reflecting respiratory-circulatory interactions universally observed [1].

The rhythm of the heart is primarily under the control of the vagus nerve, which inhibits heart rate and the force of contraction. When we inhale, vagus nerve activity is impeded and heart rate begins to increase. When we exhale this pattern is reversed. The degree of fluctuation in heart rate is also significantly controlled by regular impulses from the baroreceptors (pressure sensors) in the aorta and carotid arteries. When RSA is enhanced by biofeedback, the goal is usually to reinforce the natural feedback activity of the baroreceptors by our breathing pattern.

Piepoli [2] have shown that during normal respiration, in healthy subjects, the baroreflex is the major system for generating RSA and also slow oscillations of heart rate (HR) and blood pressure (BP). This means that changes in air pressure in the lungs when breathing are reflected in changes in arterial BP and subsequently, through the baroreflex mechanism, in HR.

Important parameters describing the hemodynamic are stroke volume (SV) and cardiac output (CO). SV represents the volume of blood pumped from the left and right ventricles of the heart with each beat. CO is the volume of the blood being pumped during a time interval of one minute. Non-invasive measurement of these fundamental hemodynamic parameters is complicated and inaccurate. One of the methods used for measuring SV and CO is thoracic impedance cardiography (TIC). TIC estimates cardiac function from a single impedance waveform measured at the thorax surface that reflects an integrated combination of complex thoracic sources. The first practical method for determination of cardiac function in a clinical setting was introduced by Kubicek et al. in the 1960s [3]. Several variations of electrode configurations and CO equations have been presented over the years to improve the method [4]. Although much work has already been done, the development of TIC is still principally based on the same concept introduced decades ago.

Calibrated cardiac output values are, unfortunately, dependent on factors related to sex, weight, age, etc. These factors are the same (fixed) for one subject during one measurement. Two parameters are crucial for comparison of relative changes of blood flow: the LVET interval (left ventricle ejection time) and the maximum amplitude of the negative first derivative of the IS signal (–dZ/dtmax). These two parameters provide information about hemodynamic changes (primarily stroke volume).

Respiration not only modulates systemic blood pressure and HR, but also strongly affects the thoracic impedance signal (IS). This effect is often used for noninvasive measurement of the respiration pattern. Respiratory time intervals derived from TIC closely

corresponded to those derived from plethydmographs, thermocouples or spirometers. A good cross-method comparison was also found for RSA parameters derived using both the peak-to-trough and spectral analyses methods. Slow respiratory oscillations of the impedance signal are most often separated by high frequency filters with a pass band of 0.1–0.5 Hz.

Unfortunately, what can be positive for respiration detection may not be positive for SV computation. TIC measures not only systemic BP respiration changes but also impedance changes due to different thorax volume and pressure during ventilation. Consequently, we measure additional artificial variability unrelated to changes of hemodynamic. This artificial variability is reflected by the –dZ/dtmax parameter, not the LVET interval. In such circumstances it is not possible to evaluate stroke volume beat-to-beat, and averaging over a larger number of heartbeats is necessary. TIC provides completely incorrect results in the case of the Valsalva or Mueller maneuver.

To estimate artificial oscillation in TIC we used a multichannel impedance monitor (MBM ISI Brno) and analyzed IS signals from different parts of the body simultaneously.

There are significant differences between the thorax, neck, arms and legs. Thorax localization accumulates a number of influences – pulmonary circulation, heart filling, aortal circulation and respiration. On the other hand, the signal from the neck doesn't include the influence of respiration and pulmonary circulation. It primarily reflects carotid artery volume change. The same is true for locations on the limbs. The thoracic (descending) aorta is much more elastic and can change volume relatively more than the brachial artery and deep and femoral artery. These properties can be reflected in whole-body IS.

2. Methods

There are not many papers dealing with multichannel bioimpedance. Stanley [5] describes a method based on simultaneous IS measurement. Stanley used 10 voltage electrodes and four current injecting electrodes located at each distal extremity. The alternating current was of a fixed frequency. He received IS simultaneously from R and L arm, R and L leg and thorax. Stroke volume is calculated by averaging the IS from 40 heartbeats.

The main feature of the MBM is the use of three independent current sources operating at different frequencies – three current generators with adjustable frequency and amplitude. Current sources are positioned to cover the entire body without mutual interference. Electrode localization is presented in Fig. 1. The MBM includes the desired number of scanning channels that enable the detection exclusively of those signals to which the frequency is adjusted. Here we present hemodynamic measurements with 18 simultaneously scanning MBM sites – left and right carotid artery (1,2), left and right part of the chest (3,4), left and right thigh (5,6), left and right calf (7,8), left and right part of the abdomen and the abdominal artery (9,10), left and right upper part of the chest (11,12), left and right arm (13,14), left and right forearm (15,16), left and right part of the chest at the level of the heart (17,18) – Figure 1. Sites 1,3,5,7 and 9 are adjusted to current G1. Sites 2,4,6,8 and 10 are adjusted to current G2. Sites 11,12, 13,14, 15,16 and 17,18 are adjusted to current G3.

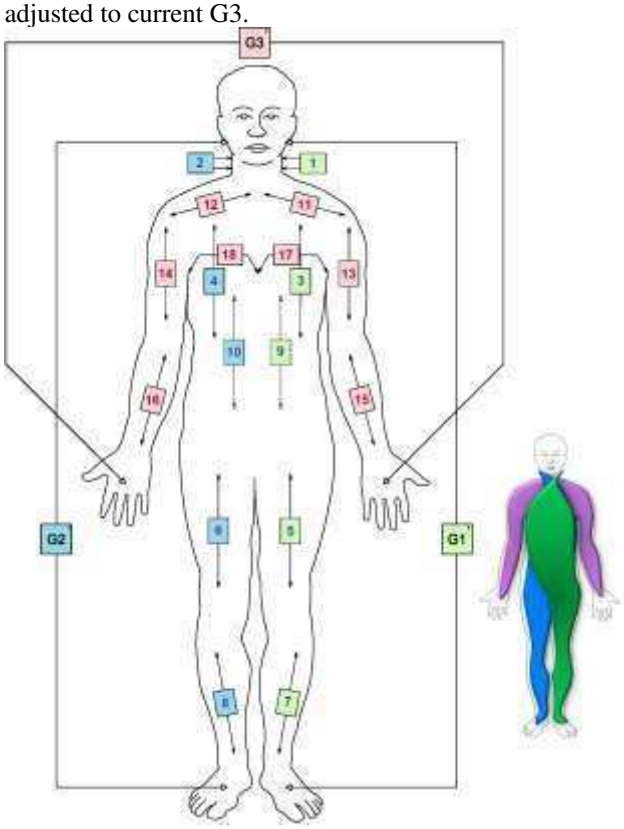

Figure 1. Placement of voltage electrodes 1-18 and current electrodes G1, G2, G3 – three independent current sources operating at different frequencies.

We measured six healthy volunteers (male, age 30-50) in resting supine position with spontaneous breathing. One subject was removed in view of a lack of sufficient signal quality. 12-lead ECG, continuous blood pressure, phonocardiography, and breathing rate and depth were recorded simultaneously with IS. Impedance signal Z was filtered in three pass bands: 0.1–0.5 Hz, 0.1–14 Hz and non-respiratory 0.5–14 Hz. We detected beat-to-beat maxima from Z (Zmax) and from negative derivative impedance signal -dZ/dt (-dZx/dtmax). The results were a 100s time series from the thorax, neck and extremities. Locations are identified by number – Fig. 1. At each location we computed standard deviation – SD Zmax and

SD -dZx/dtmax. To obtain results independent of the different IS amplitude we performed normalization of standard deviation: SD_n = SD/mean.

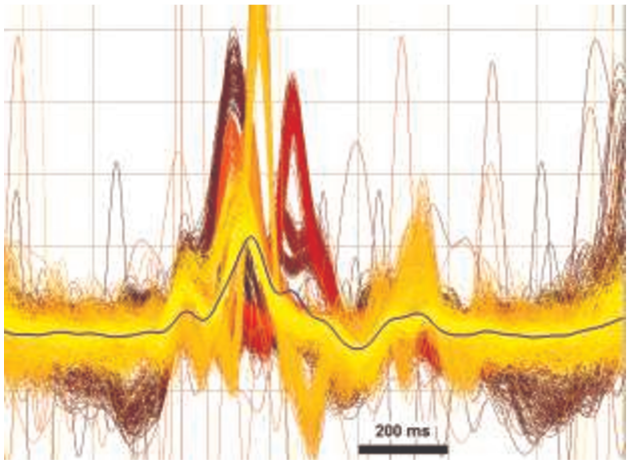

Figure 2. Derivative impedance signal -dZ/dt, 0.5-14 Hz, 18 locations, 100-second interval, overlapped beat-to-beat draw.

3. Results

Elimination of breathing is based on the assumption that neck and limb IS measures only the variability in systemic blood pressure and is not affected by impedance change in the thorax due to respiration.

The results are demonstrated in paired figures (3-4, 5-6 and 7-8) with variability of IS in both the respiratory and non-respiratory pass band.

Fig. 3. includes SD Zmax at respiratory frequency. It is clearly seen in high variability in thoracic locations – 3,4,11,12,17,18. SD Zmax in the respiration free pass band is given in Fig. 4. The SD and differences between locations are lower. There is also high SD in the arms (13, 14, 15, 16).

Figs. 5 and 6 include the SD of negative derivative impedance signal -dZx/dtmax. Due to the insufficient quality of the derivative signal, subject 5 wasn't included in these results. In comparison with figures 3 and 4, there is lower reduction of respiration. The SD in the arms is higher in both figures. -dZx/dtmax in the 0.5-14 Hz pass band (Fig. 6) represents an essential parameter for SV computation.

Figs. 7 and 8 include relative changes of normalized variability SD_n in the extremities to normalized variability SD_n in the thorax ($SD_n x/SD_n 3$). Normalization of SD to the mean value eliminates the possible effect of different amplitude of derivative IS. Relative changes are computed as the quotient of extremities location to thorax location 3. Value 1 means the same variability as in the thorax, value 0.5 means half the variability.

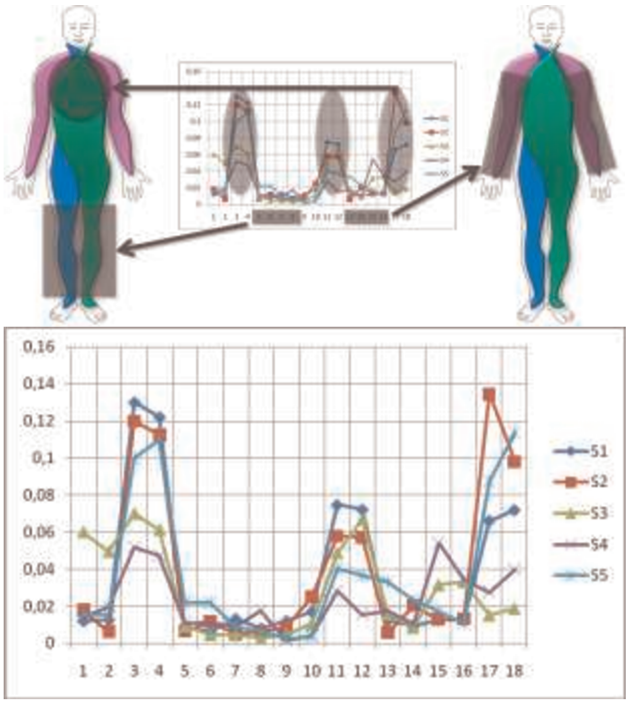

Figure 3. SD Zmax, pass band 0.1–0.5 Hz. Horizontal axis - 1, 2, …, 18 electrode position. S1, … S5 – subject identification.

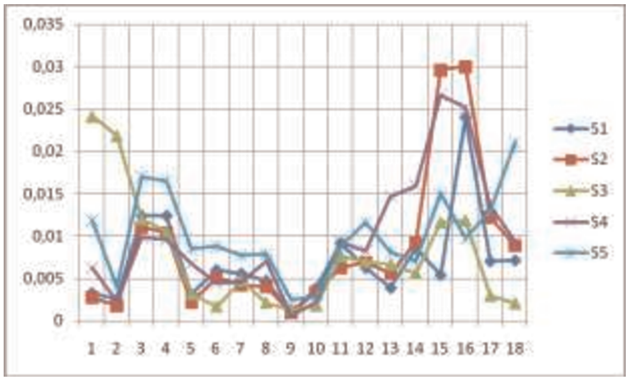

Figure 4. SD Zmax, pass band 0.5–14 Hz.

4. Discussion

The presented study demonstrates the artificial effect of respiration on thoracic IS. Commonly used filters are not able to eliminate it. Whole-body IS measurement and computation shows a decrease in respiratory variability in the extremities. This decrease in variability can be reliably monitored, especially in the legs (locations 5, 6, 7, 8) and the neck (location 1, 2). Surprisingly, measurements of the arms show differences between locations within one subject and between subjects inter-individually (locations 13, 14, 15, 16).

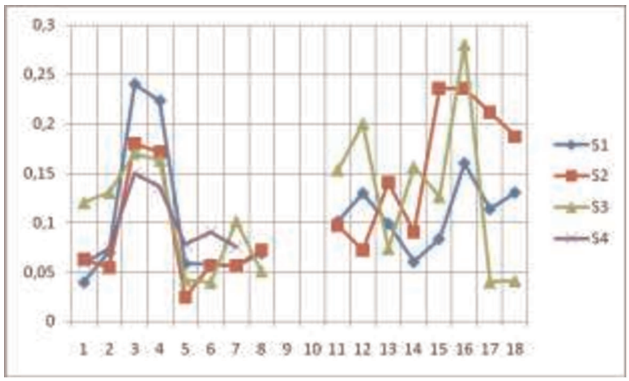

Figure 5. SD –dZ/dtmax, pass band 0.1–14 Hz.

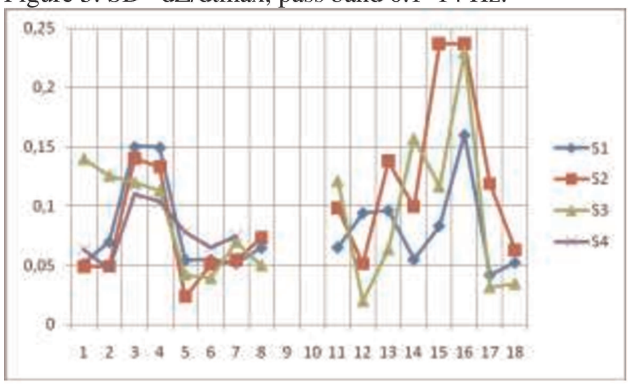

Figure 6. SD –dZ/dtmax, pass band 0.5–14 Hz.

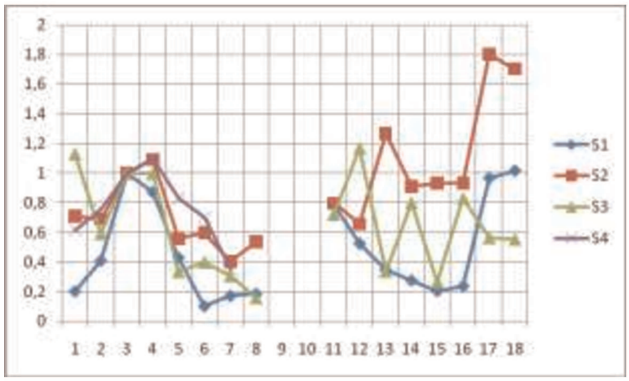

Figure 7. Normalized SD_nposition/SD_nthorax, –dZ/dtmax, pass band 0.1–14 Hz.

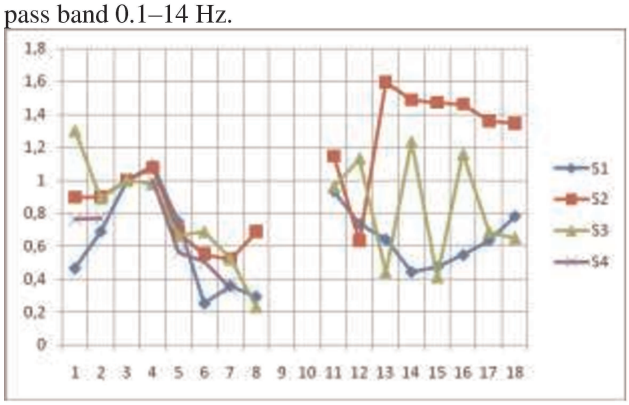

Figure 8. Normalized SD_nposition/SD_nthorax, –dZ/dtmax, pass band 0.5–14 Hz.

The -dZx/dtmax variability in the 0.5–14 Hz pass band in the legs represents 44.6 % and in the neck 58.3 % of the thoracic signal variability – Fig. 6. If we use normalized variability – Fig. 8., this reduction is 49.1 % and 81.8 %. Normalized variability eliminates the influence of different signal amplitude in the legs and provides reliable results of respiratory effect.

5. Conclusions

The results of this first pilot of a whole-body impedance study are the following: there are two sources of respiratory induced IS variability in the thorax; the variability in derivative thoracic impedance signal after respiration filtration is approximately double in comparison with the neck and legs; stable results can be obtained from the lower extremities and from the neck.

Moreover, whole-body IS recording can provide additional information such as analysis of the pressure-wave propagation in the arteries and the characteristic shape of IS. This shape provides information about the dynamic change in vascular volume during one heartbeat in different locations.

Acknowledgements

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