Does asthma-like increased breathing load influence impedance pneumography signal?

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Abstract— **Asthma is a chronic disease characterized by recurrent attacks of breathlessness and wheezing. During an asthma attack, a bronchospasm occurs. In order to overcome increased resistance of airways forced breathing is necessary. It affects the intrapleural pressure, blood volume within the thoracic cavity and therefore it may affect also impedance pneumography signal. We tried to imitate asthma-like conditions by increasing breathing load in to find out, whether it influences impedance pneumography signal. Forced breathing was performed as a respiration through adjustable respiratory loads. The study subjects breathed through 4, 7 and 10 cmH_2O*s/liter breathing loads, at 6 and 12 breathes per minute rates. A calibration coefficient between tidal volume and impedance pneumography and the effect of breathing load on cardiac component of impedance pneumography signal were determined. We found that forced respiration did not significantly affect the impedance pneumography signal, thus it seems unlikely that asthma-like bronchospasms will disturb calibration of the impedance pneumography signal. Thus it is likely, that this technique can reliably measure respiratory parameters during long-term ambulatory monitoring of patients with asthma.**

Keywords—**Impedance pneumography, Forced breathing, Asthma, Ambulatory monitoring**

I. Introduction

WHO estimates show that 235 million people currently suffer from asthma. It is a chronic disease characterized by recurrent attacks of breathlessness and wheezing. During asthma attack, bronchospasm occurs.

Measurement of thoracic electrical impedance is used to determine patient's breathing function, the phase of breathing and tidal volume. The impedance pneumography can be useful for long-term ambulatory measurements [1]. In patients with asthma, in whom the episodes of bronchospasms occur at random, this technique allows to record such episodes and provide information on individual susceptibility to triggering factors and effectiveness of treatment.

The impedance pneumography signal amplitude shows good agreement with tidal volume after calibration [2]. The calibration must be performed individually for every patient and for given body position, as the position of the body has dominant influence on calibration coefficients [3]. On the other hand, calibration coefficient exhibits moderate variability over time (e.g., days) [4]. Furthermore, the disadvantage of impedance pneumography is its lack of resilience to interferences, caused by motion and speaking [5].

Thoracic impedance signal encompasses both respiratory as well as cardiac components. A method to separate these components, based on their inherent frequencies, has been proposed [8,9].

It is not known at present, how changes of lung volume cause the changes in thorax electric impedance. For instance, during inspiration lung volume increases, and it may increase electrical impedance of the thorax [6]. It is also possible, that the respiratory induced shift in blood volume within the thoracic blood vessels affects the impedance of the thorax.

Respiration with breathing load causes much greater changes in lung and pleural pressures, and clearly affects heart rate [7,10], stroke volume (SV) [11] and arterial blood pressure [10,14]. Such conditions could also cause considerable shift in blood volume in the lungs [11].

The aim of the study was to evaluate the feasibility of impedance pneumography technique in monitoring respiratory function of asthma patients. Subjects performed forced rhythmic respiration through increased breathing load in order to imitate bronchospasm during asthma's attack. The influence of breathing load on impedance pneumography signal was also evaluated.

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II. Method

In the preliminary study, 3 patients participated: 33 years old women and two men (28 and 62 years old). The subjects did not have asthma or other respiratory disease. Patients breathed at 6 and 12 breaths per minute rates. For each breathing rate, 3 breathing loads were arbitrarily set. Each test lasted 1 minute. Between tests, patient were breathing freely without measuring device. Patients stood during the tests. The measurement scheme was presented in the Figure 1.

Two breathing parameters were measured: respiratory airflow (Medikro Oy, Kuopio, Finland) and pressure in the mouth (MPX2050, Freescale Semiconductor, Inc, Arizona USA). Respiration load was calculated as the ratio of air mouth pressure to respiratory airflow. Three breathing loads were applied: 4, 7 and 10 cmH_2O*s/l, clearly greater than respiratory system own breathing load, which is about 2-4 cmH_2O*s/l [12]. Tidal volume was calculated as an integral of respiratory airflow.

Impedance pneumography signal (ΔZ) and the ECG were recorded with the device constructed by us (Pneumonitor 3) [13]. Application electrodes (1) were placed on the arms, sensing electrodes (2) were placed on the thorax; ECG reference electrode (3) was placed on right side of thorax at the level of 11th-12th rib (Fig. 2). Radial blood pressure was continually measured using finger cuff with Portapres (Finapres Medical System, Holland).

We estimated the slope value of linear regression between impedance pneumography signal and tidal volume at different breathing loads; the coefficient of determination (R^2) of linear model value were calculated.

We also estimated the relative share of cardiac component in the recorded impedance pneumography signal using the method described by *Pandey et al*. [14]. Respiratory and cardiac components were separated in ΔZ signal using variable length averaging time windows. In addition, time window must be matched to actual respiratory rate. The cardiac component was estimated as subtraction of estimated respiratory components from acquired signal. The amplitude of cardiac components for every cardiac cycle was determined.

All signals were recorded with USB-6001 data acquisition card. We used Signal Express (National Instruments Corporation, USA) application to control measurements. Sampling frequency for every channel was set at 1000 Hz.

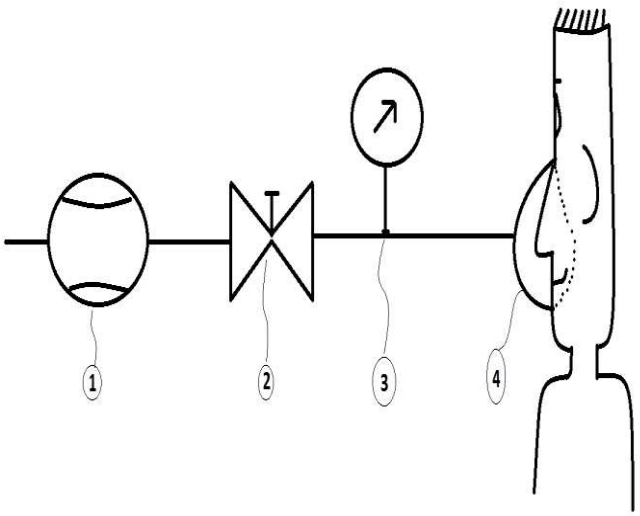

Figure 1: The measurement scheme. During the trial, the patient breathed through a mask (4), tightly adjacent to the skin, next through the flow transducer (1), adjustable diaphragm (2) which affected breathing load, and the pressure sensor (3).

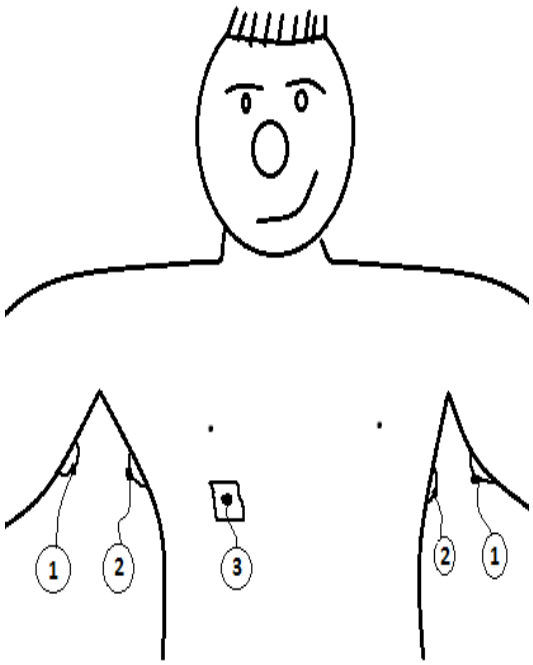

Figure 2: Location of the electrode on the body. 1 – application electrodes, 2- sensing electrodes, 3 – ECG reference electrode

III. Results

The relationship between tidal volume and amplitude of impedance pneumography signal is presented in Fig. 3. All obtained results are gathered in the Table 1.

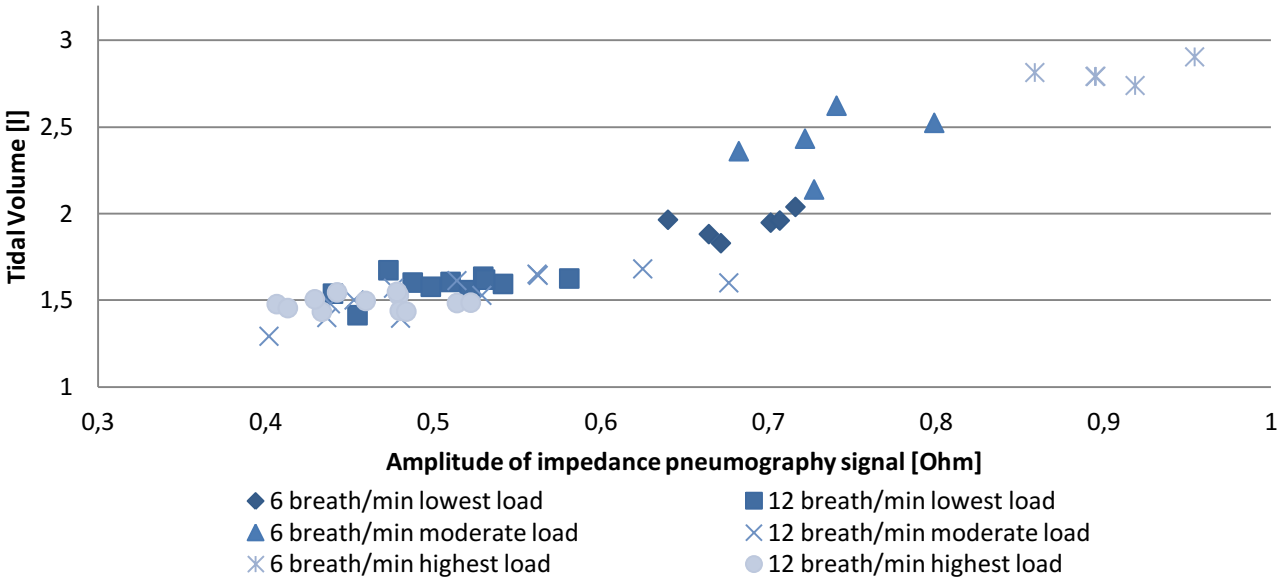

Figure 3: Relationship between tidal volume and amplitude of impedance pneumography signal for all 6 tests for 63 years old man. At slow respiratory rate an increase of breathing load resulted in an increase of tidal volume and amplitude of impedance signal, this effects do not occurs for rate of 6 breath per minute.

Table 1: Slope value of linear regression model, the coefficient of determination (R^2) of linear model and percentage of cardiac component amplitude in impedance signal for 6 tests with different breathing rates and loads; I patient is 33 years old women, II 28 years old men and III 63 years old men.

Conditions	**Slope value of linear regression**			**The coefficient of determination (R^2)**			**Percentage of cardiac component amplitude in impedance signal**		
	I	**II**	**III**	**I**	**II**	**III**	**I**	**II**	**III**
6 BPM; lowest load	0,92	0,28	0,35	0,95	0,69	0,94	9,9%	24,5%	7,3%
6 BPM; medium load	0,89	0,28	0,31	0,95	0,69	0,95	8,3%	11,2%	8.5%
6 BPM; highest load	0,88	0,22	0,33	0,95	0,61	0,95	7,5%	17,3%	9,1%
12 BPM; lowest load	0,78	0,19	0,24	0,64	0,43	0,52	13,5%	20,0%	7.7%
12 BPM; medium load	0,77	0,27	0,26	0,61	0,57	0,59	9,9%	16,5%	8,8%
12 BPM; highest load	0,77	0,29	0,24	0,64	0,95	0,60	12,2%	11,3%	14,3 %

IV. Discussion and conclusions

At slow respiratory rate, an increase of breathing load resulted in an increase of tidal volume. This effect was absent at high respiratory rate. Each patient puts more effort in breathing when higher load is applied. The inertia of muscle work can results in an increase in tidal volume, it may be a reason in observed differences.

The slope of the linear relation between impedance pneumography signal and lung volume change was not affected by breathing load. Value of a slope varied between subjects. These results are similar to those described in our previous work [4]. Obtained results indicate that tidal volume and respiration rate have a greater impact on impedance pneumography signal than a value of breathing load.

The ratio between cardiac and respiratory components in the impedance signal changes with the breathing load, however in an individual way. We would like to focus on that issue in our further analyses.

Brown et al. [16] suggest that both cardiac and impedance pneumography signal stem from blood volume change in the pulmonary tree. In our work forced breathing weakly affected impedance signal, what suggests that either blood volume changes in the pulmonary tree were not affected by this kind of breathing.

Concluding, our results indicate the insignificant impact of breathing load on impedance pneumography signal. Thus, it seems likely that readings of tidal volume would not be compromised by episodes of bronchospasm in asthma patients. Obviously, these very preliminary results have to be validated in actual asthma patients.

CONFLICT OF INTEREST

Authors declare no conflict of interest.

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