Changes in Heart Rate and Tissue Blood Volume Induced by Inspiration and Expiration

M Nitzan[1], D Dayan[1], A Babchenko[1], A Murray[2]

[1]Jerusalem College of Technology, Jerusalem, Israel
[2]Medical Physics, Freeman Hospital, Newcastle University, Newcastle upon Tyne, UK

Abstract

Respiration induces fluctuations in heart rate and arterial blood pressure, originating from either mechanical effect of respiration or from changes in autonomic nervous system activity. In order to study the relationship between cardiovascular hemodynamics and the two phases of respiration, inspiration and expiration, we used the photoplethysmographic (PPG) signal, which reflects the cardiac-induced changes in the tissue blood volume. The time of inspiration and expiration was determined by a novel depth-of-breath sensor. The PPG and the depth-of-breath signals were simultaneously recorded during long breathing period of 12 s duration per breath. In most examinations the finger blood volume increased during inspiration, probably due to higher sympathetic activity during expiration. In a minority of examinations tissue blood volume decreased during inspiration probably due to the mechanical effect of low pressure in the thoracic cavity during inspiration.

1. Introduction

The fluctuations in heart rate due to respiration – respiratory sinus arrhythmia – and the similar fluctuations in the arterial blood pressure have been intensively investigated. Heart rate increased and systolic blood pressure decreased during inspiration [1,2]. These respiratory-related fluctuations were attributed to fluctuations in central autonomic activity caused by spontaneous oscillations in the respiratory center activity or by respiratory-induced mechanical effects on the aortic baro-receptors and the pulmonary stretch receptors [2,3]. The direct mechanical effect of respiratory-induced thoracic pressure changes on the arterial blood pressure and on central veins may also be significant [4-6].

Respiratory-induced fluctuations have also been shown in the peripheral circulatory system. Deep inspiration demonstrated lower skin blood flow, which was measured by laser Doppler flowmetry [7-9] and was attributed to higher sympathetic activity. The photoplethysmographic (PPG) signal is also modulated by respiration. The PPG photodetector output, which is proportional to light transmission through the tissue, oscillates at the heart cycle rate due to the cardiac-induced increase in the tissue blood volume during systole. The PPG amplitude, which is related to the arterial compliance and the pulse pressure [10], changes with the respiration cycle [11,12]. The PPG baseline, which is inversely related to tissue blood volume has shown fluctuations in the respiratory rate [11,13-15] and significantly decreased after a deep inspiratory gasp [8,9] The respiratory-induced oscillatory changes in PPG pulse transit time, which is related to the compliance of the conduit arteries, were also studied and correlated with the heart rate variability [16].

The respiratory-induced changes in PPG baseline can originate from arterial blood pressure changes caused mechanically by thoracic pressure changes [13] or from peripheral sympathetic activity oscillations [14].

In the following we describe the temporal relationship between changes in PPG baseline and the phase of respiration.

2. Methods

2.1. Subjects and examination

Non-smoker male subjects, with no known cardiovascular or neurological disease were examined in this study. The subjects were examined in the sitting position, with their right hand comfortably laid on the table, at about heart level. A reflection PPG probe was attached to the right index finger and an optic-fiber sensor for the measurement of the chest-circumference changes during respiration (see later) was applied around their chest.

After a rest period of five min the subjects were asked to breathe three series of 5 regular and 5 long respirations. The subjects started inspiration and expiration according to a light point moving on a computer screen in the form of triangular waves. The

long breathing consisted of inspiration of 4s and expiration of 3s followed by 5s of no-breath.

2.2. PPG and chest circumference sensors

The reflection PPG probe consisted of an infrared light-source and a photodetector of pulse-oximeter probe (Oxisensor N25, Nelcor). A low-pass filter (0-40 Hz) reduced high frequency noise. The changes in light transmission were inverted so that a higher signal level corresponded to higher blood volume. The light transmission signals were sampled at a rate of 500 samples per second.

In order to obtain the relationship between PPG changes and time of inspiration and expiration, we used an optic-fiber sensor for the measurement of the respiratory-induced changes in chest-circumference. The sensor was developed by us and has already been described [12]. The sensor is based on the dependence of light transmission through a bent optic-fiber, and on its radius of curvature and on the change of the latter when chest-circumference changes. Some light rays, which are totally reflected in the core-cladding surface when the fiber is straight or slightly bent, may escape through the cladding when the fiber bending is higher, if the angle to the surface normal decreases below the critical angle.

The optic-fiber was connected to an elastic chest belt which was wrapped around the chest. The radius of curvature of the fiber increased due to chest-circumference increase during inspiration, resulting in higher light transmission. Infrared light from a light emitting diode (LED) was introduced into the sensor fiber and the light transmitted through the fiber was measured by PIN photo-detector. The detector output was also sampled at a rate of 500 samples per s.

3. Results

In this pilot work almost all subjects showed physiological changes with respiration, and in over 70% of examinations tissue blood volume increased during inspiration and decreased during expiration. Figure 1 shows an example for this pattern. The upper curve displays light transmission changes, which reflect tissue blood volume changes. The latter include relatively slow changes due to respiration and relatively fast changes due to cardiac activity (PPG). The middle curve is the chest-circumference change due to respiration. Upward direction is related to higher tissue blood volume and higher chest-circumference change (during inspiration). Note that the respiratory increase in tissue blood volume started before the start of inspiration, as determined from the chest-circumference change curve.

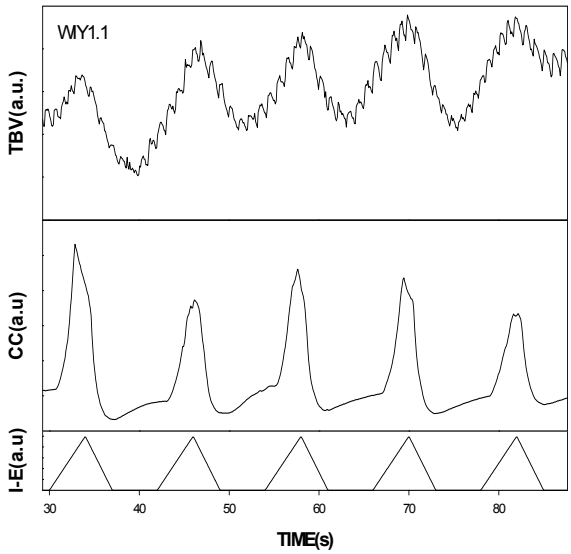

Figure 1. Respiratory changes in tissue blood volume (TBV) (top) and chest-circumference (CC) (middle) for one of the subjects. The instruction pattern for inspiration and expiration (I-E) is shown in the lowest curve. Each cycle of inspiration-expiration-pause is of 12 s. Upward direction is related to higher TBV and CC. TBV increased during inspiration and decreased during expiration.

The pattern of Figure 1, in which tissue blood volume started to increase before the start of inspiration was found in about half of the examinations. In some examinations there was a sharp increase in tissue blood volume at the start of inspiration. The start of the decrease in the tissue blood volume occurred 0-2 s after the start of expiration.

In a minority of examinations an inverse relationship was found between the tissue blood volume increase and chest-circumference increase: blood volume decreased during inspiration and increased during expiration. Figure 2 presents an example for such a pattern. The curves of two other examinations could not be described by a direct or inverse relationship. One of them displayed a direct relationship with significant delay of the tissue blood volume curve relative to the chest-circumference curve and the other showed two pulses of blood volume change for each cycle of respiration.

The respiratory changes in heart period were also measured from the PPG signal, assuming that the PPG period is equal to the heart period. In all examinations, including those of inverse relationship between the tissue blood volume and chest-circumference, the heart period increased during inspiration and decreased during expiration.

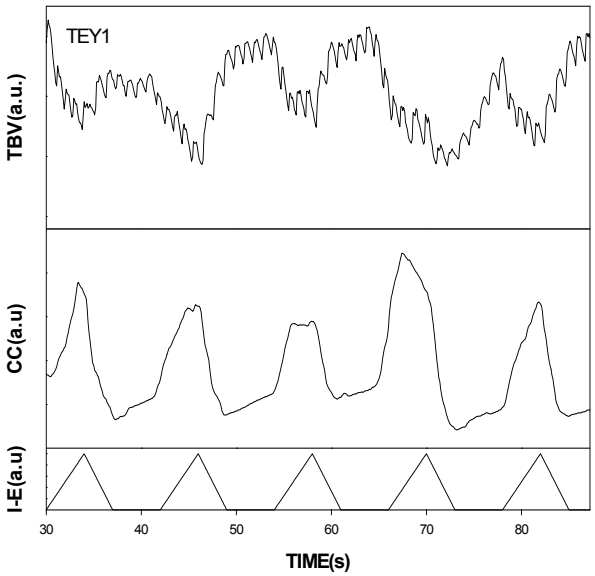

Figure 2. Inverse relationship between respiratory changes in tissue blood volume (TBV) (above) and chest-circumference (CC) (middle) for one of the subjects.

4. Discussion and conclusions

The main finding obtained in this study was that in long breathing of 12 s duration, the respiratory-induced finger blood volume changes can appear in one of two opposing patterns: in direct relationship with chest-circumference change, where tissue blood volume increased during inspiration and decreased during expiration, and in inverse phase to chest circumference change, where tissue blood volume decreased during inspiration and increased during expiration. In most of the examinations the direct relationship pattern was found; the inverse pattern was found in only a minority of examinations.

Two possible mechanisms were suggested as origin of the tissue blood volume fluctuations in the respiratory rate: consequence of the mechanical effect of the negative thoracic pressure during inspiration on the arteries and veins in the thorax [13] or result of sympathetic activity oscillations [14,15].

The effect of respiration on sympathetic activity was demonstrated by several studies [17-19] which showed higher muscle sympathetic nerve activity (MSNA) during expiration and very low MSNA at end-inspiration, when lung volume is maximal. The effect can be either direct, through central coupling of respiratory drive to the autonomic nervous system or through indirect modulation by the aortic baro-receptors or pulmonary stretch receptors. Since sympathetic activity constricts skin blood vessels, the decrease of sympathetic activity during inspiration is expected to increase fingertip blood volume, which can explain most of our results. Though those studies measured the respiratory changes in MSNA in spontaneous breathing, say in respiration frequency which is lower than our 12 s period breathing, it seems reasonable to suggest a hypothesis that sympathetic activity is similarly modulated for the respiratory pattern of our long-breathing examinations.

The infrequent exceptional results obtained in examinations, when tissue blood volume decreased during inspiration, can be attributed to the mechanical effect of inspiration. The negative thoracic pressure during inspiration can decrease arterial blood pressure in the aorta and consequently in the whole body and can increase venous blood volume in the thorax at the expense of venous blood volume in the rest of the body. These two effects can result in decrease in peripheral tissue blood volume during inspiration, and can therefore explain our results for the two exceptional examinations of inverse pattern in long-breathing. Our hypothesis is that in this case the peripheral blood vessels are more influenced by the mechanical effect of the negative thoracic pressure during inspiration than by the sympathetic activity reduction during inspiration.

The heart period increased during inspiration in all examinations, indicating different mechanism than that of peripheral tissue blood volume change. It is accepted that the high-frequency respiratory-related heart rate variability is related to parasympathetic activity [3,20]. The increase of heart period during inspiration was attributed to decrease in parasympathetic activity, originating from the respiratory-induced changes in the thoracic pressure or the output of pulmonary stretch receptors [3, 18].

Conclusion: In the current study different patterns of tissue blood volume change during respiration were found. While in most examinations tissue blood volume increased during inspiration, in a minority of examinations tissue blood volume decreased during inspiration. The different mechanisms are probably due to the different mechanisms in which respiration affect the cardiovascular system.

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Address for correspondence

Name: Prof. Meir Nitzan
Full postal address: Dept. Applied Physics/Medical Engineering, Jerusalem College of Technology, POBox 16031, Jerusalem 91160, Israel
E-mail address: nitzan@jct.ac.il