

Respiration-induced changes in tissue blood volume distal to occluded artery, measured by photoplethysmography

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Abstract. Photoplethysmography (PPG) measures the cardiac-induced fluctuations and other changes in tissue blood volume by light transmission measurement. In the current study, light transmission was simultaneously measured in the two index fingers of healthy subjects, while the brachial artery in the left arm was occluded by a pressure cuff, so that no PPG signal appeared in the left finger. Correlated respiratory-induced changes in the PPG baseline in the right hand and in the light transmission in the left hand were found, indicating respiratory-induced blood volume changes in the finger distal to the occluded artery. The blood volume changes under the PPG probe distal to the occluded artery are interpreted as transition of blood volume from small arteries into big veins, mediated by the sympathetic nervous system. © 2006 Society of Photo-Optical Instrumentation Engineers. [DOI: 10.1117/1.2236285]

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1 Background

The origin of the respiratory-induced high-frequency (HF) fluctuations in arterial blood pressure was attributed either to direct mechanical effect of respiratory-induced thoracic pressure changes on the arteries or to sympathetic or parasympathetic tone oscillations.^{1–4} The latter can be either direct, through central coupling of respiratory drive to the autonomic nervous system, or modulated by the baroreceptors.

Respiratory-induced fluctuations have also been shown in the peripheral hemodynamics. Deep inspiration demonstrated lower skin blood flow due to higher sympathetic activity.^{5–7} The photoplethysmographic (PPG) signal is also modulated by respiration. The PPG photodetector output, which is proportional to light transmission through the tissue, oscillates in the heart cycle rate due to the cardiac-induced increase in the tissue blood volume during systole. The PPG baseline, which is inversely related to tissue blood volume has shown respira-

tory HF fluctuations^{8–11} and significantly decreased after deep inspiratory gasp.¹²

The respiratory-induced changes in PPG baseline can originate from arterial blood pressure changes caused mechanically by thoracic pressure changes and passively transmitted from the large arteries to the small arteries⁹ or from peripheral sympathetic activity oscillations.¹¹ In the following we present PPG examinations, demonstrating respiration-induced changes in tissue blood volume in a finger distal to occluded artery under a pressure cuff. These tissue blood volume changes cannot be directly originated from hemodynamic changes in the body proximal to the pressure cuff.

2 Materials and Methods

2.1 Subjects

Transmission PPG was simultaneously measured in the right and left index fingers of 10 nonsmoker male subjects aged 21–63 years. The subjects were normotensive (of blood pressure not higher than 140/90 mmHg), and had no known cardiovascular or neurological disease. During the examination the subjects sat comfortably, with their hands laid on the table, at about heart level. After a rest period of 5 min the subjects were asked to breathe with a period of 10–15 s, and PPG measurement started. After 2 min, the cuff pressure was raised to above systolic blood pressure, and PPG measurement continued for an additional 2 min. Only the data after the occlusion was used in the offline analysis, and because of great change in the light transmission data in the first 30 s after the occlusion only the last 90 s were used for the analysis. Room temperature was 21–24 °C.

2.2 The PPG Device

The PPG probe consisted of infrared light-source and photodetector of pulse-oximeter probe (Oxisensor N25, Nelcor). A low-pass filter (0–40 Hz) reduced high-frequency noise. The signal was also high-pass filtered (with cutoff frequency of 0.03 Hz) and further amplified ($\times 25$) and inverted for better presentation of the PPG pulses [Fig. 1(b)]. The latter signal (AC) was used for the determination of end-diastole time (AC signal minimum) and the signal (DC) before the high-pass filter was used for baseline measurement [DC signal maximum; see Fig. 1(a)]. The signals were sampled at a rate of 500 samples/s (12 bit) and digitally stored for offline processing.

2.3 Data Analysis

After the examination, the stored data was digitally analyzed for detecting the minimum of each PPG pulse¹⁴ in the right hand. No PPG pulses appeared in the left hand (distal to the cuff) for cuff pressure above systolic blood pressure. For each PPG pulse (in the right hand) the baseline (BL) of the pulse and the value of the photodetector output in the left hand for the time of end-diastole (the inverted PPG pulse minimum, see Fig. 1) were determined.

In the 90-s examination data a region of 40–80 PPG pulses was determined after discarding data points affected by movement of the subject. From the light transmission curves for the two hands two series of 40–80 BL values were obtained and

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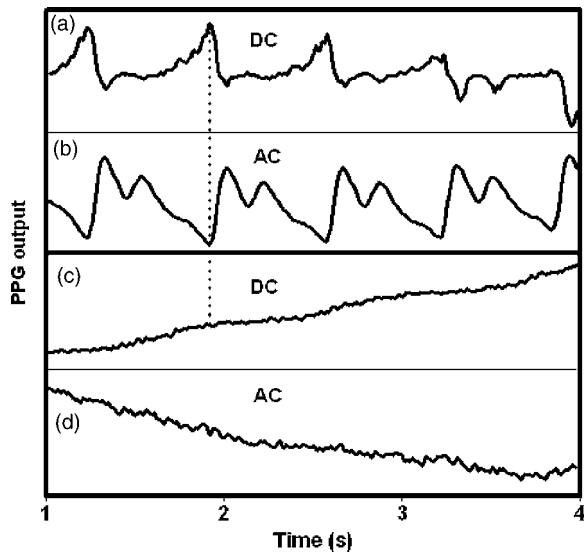


Fig. 1 PPG signal in the right hand, (a) before and (b) after high-pass filtering and inversion, and the photodetector output in the left hand, distal to the occluded artery, (c) before and (d) after high-pass filtering. No PPG pulses appear in the photodetector output in the left hand. The end-diastole time, obtained from the pulse minimum in (b), is shown.

the data were smoothed by moving average of three points. Lower frequency fluctuations were removed (trend removal) by subtracting the best-fit fifth-order polynomial from the original BL curve. The cross-correlation coefficient was calculated for the two BL series, allowing lag of 20 pulses between them.¹⁴ The intensity of the respiration-induced changes in the blood volume in the left and right fingers was assessed by the standard deviation of the corresponding BL curve after smoothing and trend removal.

2.4 Statistical Analysis

The Student t-test was used for evaluating the significance of the difference between the respiratory fluctuations intensities in the two hands. The significance of the correlation between the fluctuations in two hands was assessed by correlation coefficient and t-test; $p < 0.05$ was considered statistically significant.

3 Results

The finger PPG BL curves for the two hands of one of the subjects are shown in Figs. 2(a) and 2(b). After smoothing the curves and trend removal, BL fluctuations in respiratory rate were found in both hands [Figs. 2(c) and 2(d)], though left-hand fluctuation amplitude, distal to the pressure cuff, was smaller than for the right hand. Figure 3 presents the cross-correlation between these two curves against the lag (in pulses number) between them, showing maximal correlation coefficient for a lag of -1 pulse (negative lag means left-hand preceded right-hand oscillations).

Similar results were obtained for the other subjects. All values of maximal correlation coefficient between the two hands curves were above 0.5 ($p < 0.001$), with mean \pm SD values of 0.71 ± 0.10 . The mean lag was -1.2 pulse (say about 1 s), but was not statistically significant. The mean standard

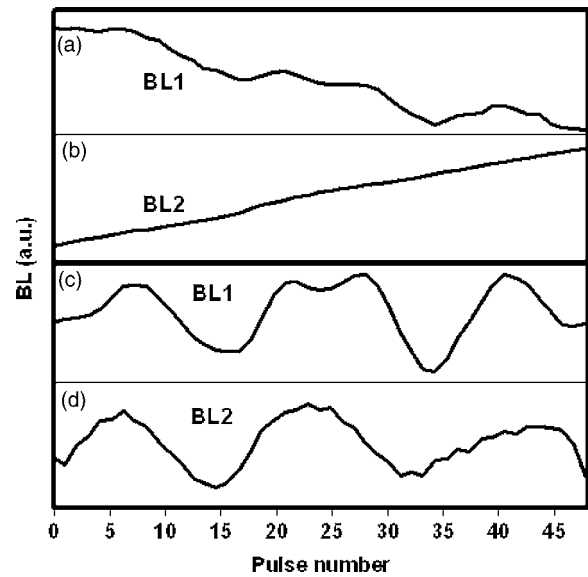


Fig. 2 Curves of PPG baseline in the right (BL1) and left (BL2) fingers for one of the subjects before (a,b) and after (c,d) detrending and smoothing. Note the high correlation between the lower curves.

deviation of the BL respiratory fluctuations (measuring fluctuations intensity) in the free hand was about five times higher than distal to the occluded artery. The mean difference between the standard deviations (in arbitrary units) in the two hands was significantly higher than zero (0.34 ± 0.32 , $p < 0.001$).

These fluctuations, of 10- to 15-s periods, were demonstrated in the occluded arm only during deep breathing of a 10- to 15-s period. During regular breathing, no light transmission changes in the respiratory rate were found on the background of other spontaneous fluctuations.

4 Discussion

Two possible mechanisms can induce the PPG baseline fluctuations in the respiratory rate: direct transmission of arterial blood pressure changes caused mechanically by thoracic pressure changes⁹ or sympathetic activity oscillations.¹¹ From the phase relation between PPG baseline and arterial and venous blood pressure oscillations in the respiratory rate, Nilsson

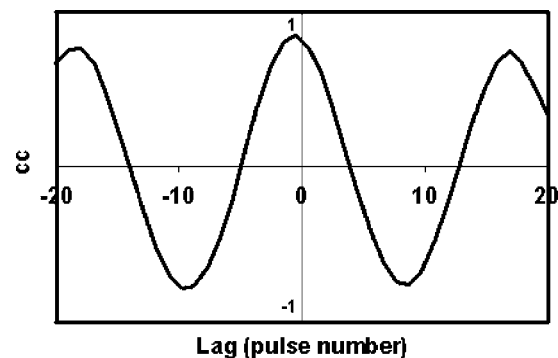


Fig. 3 Coefficient of correlation between PPG baseline curves in the two hands against the lag between them for the same subject.

et al.¹¹ inferred that the arterial and venous blood pressure changes are not the sole determinant of the respiratory-induced variations in the PPG signal.

In our study we found light transmission changes induced by deep breathing even in the finger distal to the occluding cuff. These light transmission changes—which indicate blood volume changes—appear only during deep breathing, so that they cannot be identified as the low-frequency fluctuations. Since the arteries conveying blood to the left hand were occluded by the pressure cuff, these changes cannot be attributed to respiratory-induced fluctuations in arterial blood pressure, but to the vasoconstrictive effect of the sympathetic nervous system. The effect of regular respiration or deep breathing on sympathetic activity can be direct, through central coupling of respiratory drive to the autonomic nervous system or modulated by the baroreceptors.^{2,10,11,13} Since blood cannot leave or enter the limb through the occluded artery under the cuff, explanation regarding possible mechanism for sympathetically-induced blood volume changes in the left-hand finger, under the PPG probe, is still required.

We suggest the following explanation. When cuff pressure increases to above systolic blood pressure value both arteries and veins under the cuff are occluded and blood drains from arteries into veins due to the pressure difference between them, until equalization of the blood pressure in the distal vascular system is achieved.¹⁵ The higher sympathetic activity during inspiration constricts the arteries under the PPG probe, transferring blood from them into the big veins in the hand or in the forearm, which are not under the finger probe. During expiration sympathetic activity, and consequently arterial muscle tone are lower, and blood is allowed to flow back into the arteries under the PPG probe.

The values of maximal correlation coefficient between the two hands curves were high enough (above 0.5, $p < 0.001$) to prove that the fluctuations in both hands are related to the same origin, say respiration. However the mean values of maximal correlation coefficient, 0.71, was not very high, indicating that the mechanisms responsible for the fluctuations in the two hands are not identical, as explained above.

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