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Effects of Deep Breathing on Blood Pressure Measurement in Healthy Subjects

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Abstract

The aim of this study was to investigate the effects of deep breathing on blood pressure parameters: systolic (SBP), diastolic (DBP) and pulse pressure, mean pulse rate (MPR), and the amplitude of Korotkoff sounds.

Manual SBP and DBP were measured from 20 healthy subjects (age: 44±13 years) during normal (NB) and deep (DB) breathing. A chest magnetometer was used to acquire the respiratory depth, and an electronic stethoscope for the Korotkoff sound. All data were sampled at 2 kHz. MPR was derived from the oscillometric pulse waveform. For each oscillometric pulse, time-frequency analysis of the Korotkoff sound located the time of the peak energy (Tp), and Korotkoff sounds amplitude was defined as the maximum peak-to-peak amplitude in the window T_P±50ms. The respiratory rate was calculated from both the magnetometer (f_R) and Korotkoff amplitude series (f_K) for the period between the manually measured SBP and DBP, with a power spectral density resolution of 0.016 Hz in the physiological respiratory frequency range at rest.

DB compared to NB increased respiratory regularity (quantified by spectral concentration) by 9±10% (mean±SD, $p<0.005$), and decreased f_R from 0.24±0.06 to 0.20±0.05 Hz ($p<0.0005$). SBP and pulse pressure also decreased, from 116±12 to 113±11 mmHg ($p<0.005$) and 41±9 to 37±9 mmHg ($p<0.01$) respectively, and MPR increased from 64±8 to 68±9 pulse/min ($p<0.0005$). In 70% of recordings f_R differed from f_K by 0.05 Hz or less. The results show an effect of DB on blood pressure, and the presence of a modulating effect of respiration on the Korotkoff sound.

1. Introduction

Several studies have shown the influence of deep breathing on systolic (SBP) and diastolic (DBP) blood pressure in healthy subjects [1-4]. It has also been shown that deep breathing is one of the conditions influencing the auscultatory blood pressure (BP) measurement [5].

The auscultatory method is the clinical standard for BP measurement, based on the auscultation of Korotkoff sounds (KS) during cuff deflation by means of a stethoscope placed on the *antecubital fossa* of the arm wearing the cuff. Although the genesis of KS is still a matter of debate, several hypotheses have been proposed in the past decades [6-9], the most accredited being wall distension of the compressed artery, turbulent flow downstream of the compressed brachial artery, and cavitation.

The aim of this study was twofold: *i*) to investigate the presence of a modulating effect of respiration on the Korotkoff sound amplitude; *ii*) to assess the effect of deep breathing on mean pulse rate (MPR) in the systolic-to-diastolic blood pressure measurement, in healthy subjects.

2. Methods

2.1. Data acquisition

SBP and DBP were measured manually from 20 healthy subjects (age: 44±13 years) sitting quietly, during normal (NB) and deep (DB) breathing, with the subjects breathing at their own comfortable rate.

Pulse pressure (PP) was calculated as difference between SBP and DBP.

The cuff was inflated to 200 mmHg then deflated linearly at 2-3 mmHg/s. A chest magnetometer was used to acquire the respiratory depth (RD), and an electronic stethoscope was used to record KS. All data were sampled at F_S = 2 kHz, 16-bit/sample, and stored to a computer for offline processing.

2.2. Data processing

The oscillometric waveform was segmented to determine the foot of each pulse.

The time interval between SBP and DBP (T_{SD}) was annotated. MPR was defined as the inverse of the mean inter-pulse interval in T_{SD}.

For each oscillometric pulse, joint time-frequency analysis was applied to its corresponding KS following the criterion proposed by Allen *et al* [10] based on the short-time Fourier transform (STFT) spectrogram, to locate the time (T_P) of the peak energy. For each KS, namely for each segmented pulse, the spectrogram was

calculated using a sliding time window of 40 ms with 70% overlap over the original signal (sampled at F_S). The sliding window was smoothed by the Hamming window.

For each oscillometric pulse, KS amplitude was defined in the time domain as the maximum peak-to-peak amplitude in the interval [T_P-50ms, T_P+50ms].

The KS amplitude time series was evenly resampled at 4 Hz, together with RD, using an anti-alias low-pass filter. The two synchronous signals (KSA and RESP, respectively) were used in subsequent processing.

The respiratory rate was calculated from both RESP (f_R) and KSA (f_K), in the T_{SD} interval, as the peak frequency in the power spectral distribution (PSD) between 0.1 Hz and the lowest value between 0.5 Hz and half the lowest pulse rate, with a resolution of 0.016 Hz.

In a preliminary stage, it had been verified that all RESP signals had a respiratory rate within the above mentioned range, and that T_{SD} was 10 s or longer, to comply with the spectral low frequency resolution of 0.1 Hz.

Respiratory regularity ("localization" in frequency) was quantified by the spectral concentration (SC) of RESP at the respiratory rate f_R. Mathematically:

$$SC = \frac{\sum_{f=f_R-0.1}^{f_R+0.1} \Gamma_{RESP}(f_i)}{\sum_{f=0}^{F_S/2} \Gamma_{RESP}(f_i)} \qquad (1)$$

where $\Gamma_{RESP}(f)$ is the power spectral distribution of RESP estimated by the Welch periodogram, in the T_{SD} interval.

2.3. Statistical analysis

Analysis of variance for repeated measurements with a significance level α=0.05 (two-tail) was used to assess differences in respiratory and BP parameters between the two breathing patterns.

3. Results

3.1. Respiratory parameters

Table 1 shows mean±SD of the respiratory parameters estimated from RESP in the T_{SD} interval, for the two breathing patterns.

Table 1. Respiratory parameter statistics.

Parameter	Normal	Deep	p value
f_R [Hz]	0.24±0.06	0.20±0.05	<0.0005
SC [%]	81±14	90±7	<0.005

The respiratory regularity (spectral concentration of RESP) was significantly higher in deep than in normal breathing (ΔSC = 9±10%, p<0.05).

3.2. BP parameters

Table 2 shows mean±SD of the BP parameters, for the two breathing patterns.

Table 2. BP parameter statistics.

Parameter	Normal	Deep	p value
SBP [mmHg]	116±12	113±11	<0.005
DBP [mmHg]	75±9	75±9	N.S.
PP [mmHg]	41±9	37±9	<0.01
MPR [ppm]	64±8	68±9	<0.0005

ppm = pulse/min

3.3. Respiratory modulation of KSA

Figures 1 and 2 show an example of RESP and KSA synchronous signals from the same subject in T_{SD} interval (top panels) and their PSD (lower panels), for the normal and deep breathing patterns respectively..

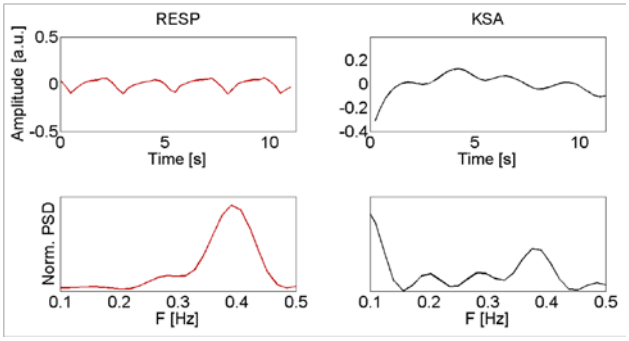

Figure 1. Example of normal breathing RESP and KSA in T_{SD} interval (top panels) and normalized PSD (bottom panels). f_R = 0.39 Hz (bottom left panel), f_K = 0.38 Hz (bottom right panel).

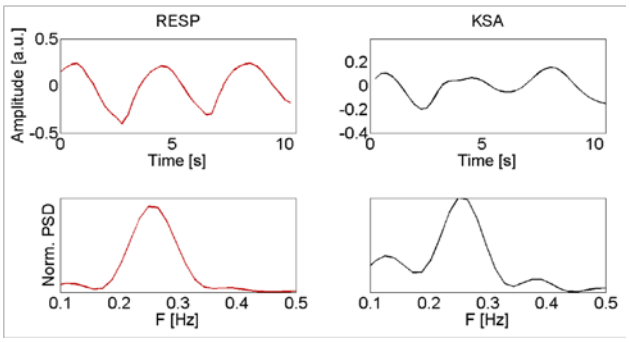

Figure 2. Example of deep breathing RESP and KSA in T_{SD} interval (top panels) and normalized PDS (bottom

panels). f_R = 0.25 Hz (bottom left panel), f_K = 0.25 Hz (bottom right panel).

Table 3 shows the count (percentage) of recordings in which the absolute difference $|\Delta f|$ between f_R and f_K did not exceed 0.05 Hz, for each breathing pattern, and in aggregate. The histogram of the respiratory rate estimation error $\Delta f = f_R - f_K$ is shown in Figure 3, and the Bland-Altman plot (2-SD limits of agreement) of the two different estimates in Figure 4.

Table 3. Estimated respiratory rate agreement

Parameter	Normal	Deep	Total
#{ $\|\Delta f\|$ <0.05 Hz }	12(60%)	16(80%)	28(70%)

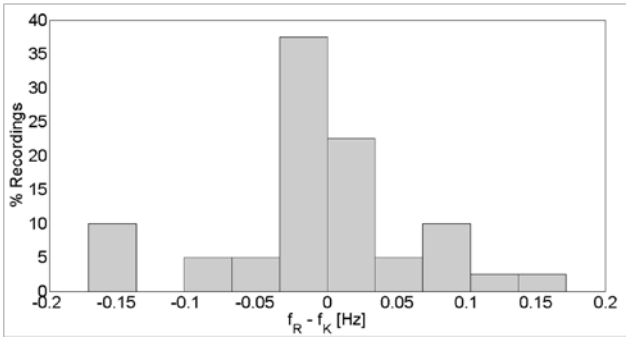

Figure 3. Histogram of respiratory rate estimation error $\Delta f = f_R - f_K$. The width of each bin is 0.034 Hz.

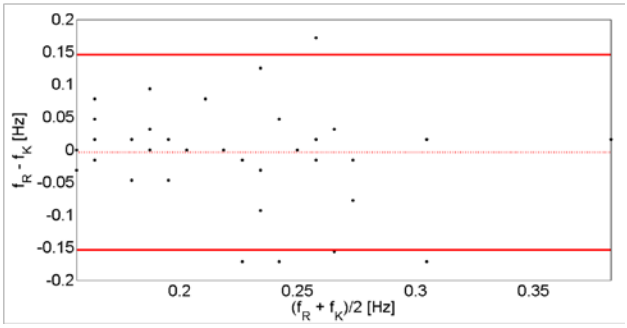

Figure 4. Bland-Altman plot of respiratory rate estimation. Solid red lines indicate 2-SD limits of agreement. Dotted red line indicates bias (bias=-0.0043 Hz)

4. Discussion

The primary finding of this study was the presence of a modulating effect of respiration on the KS in the systolic-to-diastolic BP interval, during BP measurement in healthy subjects.

Two breathing patterns were adopted: normal and deep, at a comfortable rate for each subject. As expected, in deep breathing respiratory rate was significantly lower (0.20±0.05 vs. 0.24±0.06 Hz, p<0.0005), and spectral concentration of RESP was significantly higher (90±7% vs. 81±14%, p<0.005). The agreement between the two independent estimates of the respiratory rate (f_R, f_K) was higher in deep breathing (80% vs. 60% of recordings agreed within 0.05 Hz, Table 3), possibly because of the increased respiratory regularity which made the respiratory component (spectral peak) prominent, both in RESP and KSA, thus avoiding potential estimation errors caused by multiple peaks of similar height in the frequency band of interest.

The agreement between the two estimations is fairly good as 70% of recordings agree within 0.05 Hz (Table 3) and Δf was not significant (p=0.718, not reported in tables). The Bland-Altman plot (Figure 4) shows no trends nor bias (bias = -0.0043 Hz), however the 2-SD limits of agreement are quite large, indicating that KSA may not in general be used as surrogate tool to estimate the respiratory rate.

The secondary finding of the study was the effect of deep breathing on MPR during cuff deflation between SBP and DBP, as this parameter might influence the systolic and diastolic values. A significant decrease was found in SBP in deep breathing (Table 2) which was consistent with previous findings [1, 5], while DBP did not change significantly. MPR was found significantly higher (64±8 vs. 68±9 pulse/min, p<0.0005, Table 3) in deep than in normal breathing. This could be caused by hyperventilation occurring in deep breathing, as hyperventilation causes CO_2 partial pressure to decrease, pH to consequently increase causing arteries to dilate and resulting in an increase in pulse rate to compensate SBP decrease.

5. Conclusions

The analysis of respiratory effects on auscultatory BP measurement presented in this study showed: *i*) a modulating effect of respiration on Korotkoff sounds amplitude, in the T_{SD} interval; *ii*) a significant increase in the mean pulse rate in the T_{SD} interval during deep breathing, potentially as a consequence of hyperventilation.

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