Postural Effect on Respiratory Sinus Arrhythmia with Various Respiratory Frequencies

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Abstract. Heart rate variations during steady state respiration with various frequencies were studied on seven healthy male students at two different body positions. Respiration was controlled at four different frequencies (0.083, 0.100, 0.200, 0.250Hz), and the tidal volume was simultaneously controlled at 1500ml (0.083, 0.100Hz) or 1000ml (0.200, 0.250Hz). A tilting bed was used for changing body position, and the measurements were conducted at horizontal and vertical position. RSA (respiratory sinus arrhythmia) amplitude at 0.250Hz was significantly decreased at vertical position compared with horizontal position. At 0.200Hz the significant decrease could not be obtained although some tendency of decrease appeared. Contrary to these high frequencies, the amplitudes at low frequencies (0.083, 0.100Hz) were significantly increased (p<0.01) during vertical position. This postural effect on the low frequency RSA could be regarded as a similar result on MWSA (Mayer wave relate sinus arrhythmia) which reflects sympathetic nervous activity. Furthermore, the ratio between the amplitude at 0.100Hz and that at 0.250Hz was significantly correlated with mean heart rate (n= 56, r = 0.73). From these results it was assumed that the RSA amplitude at low frequency associate a with not only parasympathetic nerves but also sympathetic nerves whereas the amplitude at high frequency was solely mediated by parasympathetic nerves.


Keywords: respiratory sinus arrhythmia, heart rate variability, spectral analysis, posture.

Introduction

Heart rate variability could be divided into two main components: Mayer wave (periodical oscillation of arterial blood pressure) related sinus arrhythmia (MWSA) and respiratory sinus arrhythmia (RSA). MWSA appears at approximately 0.1Hz which is identical to the frequency of Mayer Wave. The frequency of RSA is generally 0.2-0.4Hz, and it corresponds with the respiratory frequency. It has been noticed that RSA is mediated by parasympathetic nerves and MWSA is mediated by both sympathetic and parasympathetic nerves. Recently, a number of attempts have been made to estimate the autonomic balance noninvasively using these two components of HRV. In these studies, the spectral power of MWSA and that of RSA are named as LF and HF respectively, and the ratio of LF/HF has been used as the index of human autonomic balance.

The change of posture causes a widespread readjustment process to a cardiovascular system. Changing from supine to standing, mean heart rate increases, cardiac output decreases, and total peripheral resistance increases (Burke et al., 1977). It has been also noticed that the spectral components of HRV are effected by postural change. At standing position, LF increases and HF decreases compared to supine position. Since it is considered that sympathetic nervous activity becomes relatively predominant over parasympathetic nervous activity, therefore also supposed that these changes of the spectral components of HRV should be attributed to the change of autonomic balance.

This change of the spectral components due to postural change has been reported by many researchers. However, most of these studies have measured RSA only at high frequency ranging from 0.2 to 0.33Hz. For example, Pomerantz et al. (1985) used 0.25Hz and Pagani et al. (1986) used 0.33Hz. Although human breathing frequency at rest generally lies in this frequency band, it is possible that the frequency does operate below 0.1Hz. Consequently the decrease of RSA at standing has been studied only at narrow frequency bands.

Therefore, in this experimentation, the postural effects on the RSA amplitude were studied not only at high respiratory frequencies but also at low respiratory frequencies.
Method

Subjects and experimental conditions

Seven healthy young males volunteered as subjects. Their age ranged from 21 to 24 years with an average of 22.5 years. RSA amplitudes were measured at two body positions (horizontal and vertical), and four respiratory frequencies (0.083, 0.100, 0.200, 0.250Hz). In addition to the respiratory frequency, tidal volume was also controlled at 1500ml (0.083, 0.100Hz) or 1000ml (0.200, 0.250Hz).

A tilting bed was used for changing their body position, and the moving time from horizontal to vertical was 23 seconds. Measurements were conducted after five and eight minutes rest at horizontal position. Then the body position was changed to vertical, and the measurements were conducted in the same procedure as at horizontal position.

In addition to the RSA measurements, HRV was measured for comparing with the results of RSA amplitude measurements. The measurements were carried out in a similar procedure to the RSA measurements, whereas the respiratory frequency was fixed at 0.25Hz, with no control of the tidal volume. All conditions of both measurements were repeated on the other day.

Control of respiration

In order to control both the respiratory frequency and the tidal volume simultaneously, the following procedures were used; the instantaneous respiratory volume was measured with a hot-wire spirometer (MINATO Med. Sci., RF-H) whose sensor was attached on a subject's mask. This volume was feedback to the subject on real-time by a cursor on a TFT-LCD display. The cursor moved up and down according to the subject's instantaneous ventilatory volume, and also shifted towards right in the horizontal direction as the time elapsed. The subject was required to trace a sinusoidal curve shown on the screen with the cursor, by controlling his respiration.

The frequency, tidal volume and a pattern of respiration were able to be controlled by changes in the amplitude, and frequency of the sinusoidal curve. The subjects performed the respiratory control for one minute, and during this, an electrocardiogram and instantaneous ventilatory volume were recorded into the computer's memory. In this experiment, every subject could control their respiration within 5% of the designated values. A schematic diagram of this measurement was shown in Fig. 1.

Data processing

Data of electrocardiograms and ventilation volume were feed into a personal computer via a 12-bit AD converter. The R-R interval sequences were obtained by detecting a peak of the R wave in ECG. Afterwards the R-R interval sequences were converted into beats per minute and interpolated into equidistant data of 10Hz, according to the instantaneous heart rate method described by DeBoer et al. (1985).

In this study, the RSA amplitude was defined as the difference between the maximal and minimal values of the heart rate, during the steady-state respiration. Digital filtering processing was applied to 512 points interpolated heart rate, to extract the wave-form in accordance with the respiratory frequency. RSA amplitude was calculated from this filtered wave-form.

Power spectra were calculated from the interpolated HRV data using FFT processing. LF-HRV and HF-HRV were the integrations of HRV power spectra from 0.05 to 0.15Hz and from 0.20 to 0.30Hz respectively.

Statistical analysis

The results of RSA and HRV measurements were statistically analyzed with ANOVA. With respect to the factor of time lapse since postural change, significant differences were not obtained between five and eight minutes. Consequently, this factor has been incorporated into the factor of repeat in the statistical analysis.
Results

Spectral components of HRV

Figure 2 shows the change of spectral components of HRV (left figure) and corresponding mean heart rate (right figure). Mean heart rate at the vertical position was larger than that at the horizontal position (p < 0.01). With respect to the spectral components, HF power decreased and LF power increased significantly at the vertical position compared with the horizontal position (p < 0.01). These results are in agreement with the results of the previous studies (Pomerantz et al., 1985; Pagani et al., 1986).

RSA amplitude

Four samples of RSA of one subject are shown in Fig. 3A-D. Both Fig. 3A and B, are samples obtained under the condition of 0.250Hz and 1000ml, whereas Fig. 3A is a sample at the horizontal position, and Fig. 3B is a sample at the vertical position. Changing from horizontal to the vertical position, mean heart rate increased from 71.0 to 88.3bpm, and RSA amplitude decreased from 5.5 to 2.9bpm.

With respect to lower frequency, the different effect of postural change on RSA amplitude has been observed. Both Fig. 3C and D, are samples obtained under the conditions of 0.100Hz and 1500ml, whereas Fig. 3C is a sample at the horizontal position, and Fig. 3D is a sample at the vertical position. When changing from horizontal to vertical position, the mean heart rate increased from 62.9 to 82.3bpm which was similar to the higher frequencies. Contrary to the higher frequencies, RSA amplitude increased from 11.4 to 18.2bpm.

As shown in these samples, RSA amplitude was affected by postural change, yet the postural effect was different between high and low frequency. The mean RSA amplitudes and corresponding heart rate of seven subjects are shown in Fig. 4. At every frequency, the mean heart rate was significantly increased at the vertical position. The RSA amplitude at 0.25Hz was significantly decreased at the vertical position (p < 0.01), although the decrease at 0.20Hz was not statistically significant. Contrary to the changes at high frequency, both the amplitude at 0.100Hz and 0.083Hz were increased at the vertical position, and these changes were statistically significant (p < 0.01).

At lower frequencies, the RSA amplitudes were larger than those at higher frequencies. This result may be attributed to the frequency dependent characteristics of RSA amplitude reported by Angelone and Coulter (1964), Hirsch and Bishop (1981), Womack (1971) and Sone et al. (1991).

Correlations with heart rate

Correlations of autonomic indexes calculated from RSA and HRV with corresponding mean heart rate were examined. In this analysis, RSA amplitudes at 0.100Hz and those at 0.250Hz were used as LF and HF respectively. LF/HF ratio obtained from RSA was positively correlated with mean heart rate (r = 0.73, n = 56, Fig. 5A). With respect to HF/(LF + HF) ratio from RSA, a negative correlation with mean heart rate was obtained (r = -0.75, n = 56, Fig. 5B). When the ampli-

![Fig. 3 Samples of RSA measured from one subject. Upper thin and thick traces of each figure are instantaneous heart rate and band-pass filtered heart rate (bpm) respectively. Lower trace of each figure is instantaneous ventilatory volume (litter).](image-url)
Fig. 4 Changes of RSA amplitudes and mean heart rates.

The amplitudes at 0.083Hz were used as LF, LF/HF and HF/ (LF+HF) also correlated with mean heart rate as well as those of 0.100Hz (r=0.70 and r=−0.76). However, when the amplitudes at 0.200Hz were used as HF, the correlations were weakened (r=0.42 and r=−0.42).

The ratio of LF/HF and HF/(LF+HF) obtained from HRV spectra also correlated with mean heart rate (Fig. 6A and Fig. 6B); however, the correlation coefficients were smaller than that of RSA amplitude (r=0.46 and r=−0.47). On both RSA and HRV, LF and HF did not correlate with mean heart rate as well as the above autonomic indexes.

Discussion

Recently, in the field of physiological anthropology (e.g., Mukae and Sato, 1992; Lee et al., 1994), many attempts have been made to evaluate autonomic balance using spectral components of heart rate variability. In these cases MWSA and RSA are designated as LF and HF respectively, and the ratio LF to HF are used as an index of autonomic balance. However, note that some researchers designate MWSA and RSA as MF and HF (or RF), and describe thermoregulatory component around 0.03Hz as LF (e.g., Weise and Heydenreich, 1989; Yeragani et al., 1994).

A number of pharmacological studies have revealed that the RSA component relates to parasympathetic activity, while the MWSA component relate to both sympathetic and parasympathetic activities. In the case of RSA, the relationship between RSA amplitude and parasympathetic activity was quantitatively examined (Katona and Jih, 1976; Fouad et al., 1984).

In most of these studies the experimental conditions of respiratory frequency were set on the frequency from 0.2Hz to 0.3Hz. In fact, the frequency of human spontaneous breathing at rest generally lies in this frequency band; however, the frequency of controlled respiration could be lowered to below 0.1Hz. Thus it can be regarded that the relationship between RSA and parasympathetic nervous activity was examined only at high frequency in the previous studies. Consequently, the control mechanism of low frequency RSA might be different from high frequency.

In this study, the postural effect on high frequency RSA was similar to the previous studies, but on the contrary, the amplitude of low frequency RSA was increased at the vertical position. Since the sympathetic nervous activity is predominant during the vertical position, this increase of low frequency RSA at the vertical position is hard to explain, especially if it is considered that RSA is mediated solely by parasympathetic nerves over the broad frequency band.

Furthermore, this change of low frequency RSA seemed to show a similar change of MWSA. Therefore, it is possible to make a hypothesis that RSA at low frequency is mediated by sympathetic nerves in addition to parasympathetic nerves. Hence, we can presume that the low frequency RSA becomes a more accurate measurement than MWSA because low frequency RSA is more stable, and a more apparent phenomena than MWSA.

Some studies have been made on the postural effect on RSA at a wide frequency band. Weise and Heydenreich (1989) studied the effect at 0.12, 0.20 and 0.30Hz, yet in their experiments, tidal volume was not controlled. Saul et al. (1989) has proposed a different method to study the relationship between the RSA and respiratory frequency. They have called their new method a "broad-band approach". In this method, the subjects synchronized their respiration with a predeter-

Fig. 5 Correlations of LF/HF and HF (LF+HF) with mean heart rate. LF and HF means the RSA amplitudes at 0.10Hz and 0.25Hz respectively.

Fig. 6 Correlations of LF/HF and HF (LF+HF) with mean heart rate. LF and HF means the spectral powers integrated 0.05-0.15Hz and 0.20-0.30Hz respectively.
mined random interval sequence, which has a “whitened” frequency characteristic. The advantage of this method is that the relationship between the RSA and respiration in a broad frequency band, could be examined from one heart rate sequence. However, it seems to be impossible to control the tidal volume in their method. Although these studies had problems such as the absence of the control of tidal volume, they did report the increase of low frequency RSA amplitude at the standing position, which is similar to our results.

Saul et al. (1989) proposed that the contribution of sympathetic nerves to RSA amplitude might have a characteristic of low pass filter. It has been reported that low frequency RSA (especially around 0.1Hz) shows a large amplitude compared to high frequency. The mechanisms of this phenomena have not been identified (Sone et al. 1991). Our hypothesis, that sympathetic participation in the generation of RSA at low frequency, might account for this phenomena. Since sympathetic nervous activity is relatively predominant when the mean heart rate is increased, it can be considered that the positive correlation between LF/IF of the RSA and the mean heart rate gives a support to the above hypothesis.

Although the LF / HF ratio derived from HRV spectra also correlated with mean heart rate, the correlation coefficient was smaller than that of RSA. Using the spectral power of HRV, Lee et al. (1994) reported significant correlations of LF/IF, and IF/(LF + HF) respectively with mean heart rate; however, the correlation coefficients of their results were smaller than that of RSA amplitude of our study.

From this discussion, it is concluded that RSA amplitude at lower frequency (around 0.1Hz) could be used as an autonomic index identical to MWIA power of the HRV spectrum, and, therefore it can be further proposed that this measurement could make a better estimation of human autonomic balance.

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References


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