

Heart rate variability during breathing at 0.1 Hz frequency in the standing position

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Abstract

Background: Respiratory sinus arrhythmia (RSA) is a major component of heart rate variability (HRV) in the high frequency (HF) range (0.15 – 0.4Hz). It occurs due to the modulation of cardiac vagal outflow by respiration. The longer response time of sympathetic nervous system does not allow for a similar respiratory modulation. Instead, cardiac sympathetic outflow is modulated only in the low frequency (LF) range (0.04 – 0.14Hz), by arterial baroreflexes.

Assessment of HRV during deep breathing at 0.1Hz frequency (HRVdb) in the supine resting state is a standard test to assess the cardiac parasympathetic function. At this frequency, there is a possibility of respiration modulating the sympathetic outflow. This study explores the same during standing, which is a sympathoexcited state.

Materials and Methods: ECG and respiration were recorded continuously as ten healthy subjects breathed spontaneously and at 0.1Hz frequency in both supine and standing positions for 5 minutes each. The mean and standard deviation of the normal to normal RR intervals (meanRR, SDNN) and the average of the differences between the maximum and minimum RR intervals of 6 consecutive respiratory cycles during 0.1Hz breathing (HRVdb) were compared.

Results: Upon standing from the supine position, HRV during spontaneous breathing decreased significantly ($p < 0.05$) while there was no significant change in the HRVdb

Conclusion: Although vagal activity is reduced in the standing position, HRVdb did not show a corresponding decrease. Hence, HRVdb is not a very reliable test of cardiovagal activity when there is sympathoexcitation.

Key Words 0.1 Hz breathing, cardiovagal function, heart rate variability (HRV), HRVdb, respiratory sinus arrhythmia (RSA)

I. Introduction

Heart rate variability (HRV) is the variation in the interval between consecutive heartbeats occurring due to the changes in the autonomic flow to the sinoatrial node. This variability occurs at different frequencies and is due to many factors. HRV at lower frequencies (LF) (0.04 – 0.14 Hz) is caused by changes in sympathetic and parasympathetic outflows, while high frequency (HF) variability (0.15 to 0.4 Hz) is due to modulation of parasympathetic outflow [1,2]. Respiratory sinus arrhythmia (RSA) is a major contributor to HRV. Normal breathing frequency falls within the HF range and hence the HRV due to RSA also occurs in the HF range and is thought to be due to respiration modulating the cardiac vagal activity [3]. The longer response time of the sympathetic nervous system prevents its modulation at higher frequencies [4].

HRV varies with the frequency and depth of respiration and is found to be maximum at a breathing frequency of 6 breaths per minute (0.1 Hz) [3,5]. Infact, HRV during breathing at 0.1Hz frequency (HRVdb) has been used as a standard test to assess cardiac vagal control [6]. Here, the fact that the breathing is falling in the LF range and hence, the possibility of respiration modulating the cardiac sympathetic outflow is not taken into consideration. To explore the same, in this study, HRVdb is done during standing, when cardiac sympathetic tone is known to increase and vagal activity is reduced, and is compared against the HRVdb during the resting supine state.

II. Materials And Methods

The ECG and the respiratory signals were acquired through a physiograph and digitized by a PC with a 16 bit data acquisition card (National Instruments, USA). A custom software was used for peak detection. For HRV analysis, HRV analysis software version 1.1 from Biomedical Signal Analysis group, Department of Applied Physics, University of Kuopio, Finland, was used [7].

Ten healthy participants (6 males, 4 females) of age 28.5 ± 7 years (mean \pm SD) and BMI of 23.65 ± 4.35 Kg/m² (mean \pm SD) volunteered for the study, which was approved by the institutional research committee.

Protocol

The participants were allowed to rest in the supine position for 5 minutes after instrumentation. ECG and respiration were recorded continuously as the subjects breathed spontaneously for 5 minutes. Then they were instructed to do deep breathing at 0.1Hz frequency (6 breaths / minute) for 5 minutes prompted by verbal commands. During this deep breathing, each breathing cycle consisted of 4 seconds of inspiration followed by 6 seconds of expiration.

Then the participants were instructed to stand up from the supine position. After allowing 2 minutes for hemodynamic stabilization, their ECG and respiration were recorded for 5 minutes during normal breathing after which they were instructed again to do 0.1 Hz breathing for 5 minutes.

The mean and standard deviation of the normal to normal RR intervals (meanRR, SDNN) and the average of the differences between the maximum and minimum RR intervals of 6 consecutive respiratory cycles during 0.1Hz breathing (HRVdb) were calculated and statistically analysed.

III. Results

The results are presented in Table 1. Wilcoxon’s signed rank test was used for statistical analysis. A p-value of less than 0.05 was considered significant.

Table 1 Comparison of the parameters between supine and standing positions (n=10)

Parameter	Supine position	Standing position
MeanRR during Spontaneous breathing	0.897 ± 0.10	0.747 ± 0.05*
MeanRR during Deep breathing	0.873 ± 0.10	0.773 ± 0.07*
SDNN during Spontaneous breathing	0.055 ± 0.02	0.035 ± 0.01*
SDNN during Deep breathing	0.083 ± 0.03	0.072 ± 0.02 [#]
HRV db	0.25 ± 0.12	0.20 ± 0.07 [€]

All values are mean ± SEM

* *Significantly different P < 0.05*

Not significantly different P = 0.359

€ *Not significantly different P = 0.241*

IV. Discussion

In the SA node of the heart, the acetylcholine secreted by the vagus nerve, acts by directly opening the potassium channels and hence the response time is brief. Whereas, the norepinephrine secreted by the sympathetic nerve endings brings about its effects by binding to β1 receptors resulting in an increase in intracellular cAMP. This takes a longer time, compared to the vagal effect. Hence, sympathetic control of the heart rate can happen only at lower frequencies, while vagus can bring about high frequency as well as low frequency changes in heart rate.

Normal breathing frequency falls in the HF range. So, the respiration is thought to modulate only the cardiac vagal activity. Eckberg et al. [8] have challenged the notion that RSA is mediated only by vagal cardiac nerve activity and have provided evidence for sympathetic modulations contributing to RSA. This is corroborated by another study, in which slow breathing training decreased the spontaneous respiratory rates and increased the HRV in the standing position [9]. In this study, when the respiratory frequency is voluntarily reduced to the LF range, it is most probable that the cardiac sympathetic activity also got modulated, which became very obvious in the standing position when the sympathetic tone is increased. Hence, HRVdb did not show a significant decrease upon standing, despite the decrease in vagal activity. This implies that the HRVdb cannot be used as a test to measure the extent of cardiac vagal modulation and in conditions where the basal sympathetic tone is increased (anxiety, cardiac failure etc.) the HRVdb might include the sympathetic activity also.

V. Conclusion

The HRV during breathing at 0.1 Hz during standing, does not differ significantly from that of the supine position, inspite of the sympathoexcitation during standing. The use of HRVdb as a tool to evaluate the cardiac vagal control may not be relevant in conditions where there is increased sympathetic activity.

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