

Slow breathing influences cardiac autonomic responses to postural maneuver

Slow breathing and HRV



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ABSTRACT

Chronic slow breathing has been reported to improve Heart Rate Variability (HRV) in patients with cardiovascular disorders. However, it is not clear regarding its acute effects on HRV responses on autonomic analysis. We evaluated the acute effects of slow breathing on cardiac autonomic responses to postural change manoeuvre (PCM). The study was conducted on 21 healthy male students aged between 18 and 35 years old. In the control protocol, the volunteer remained at rest seated for 15 min under spontaneous breathing and quickly stood up within 3 s and remained standing for 15 min. In the slow breathing protocol, the volunteer remained at rest seated for 10 min under spontaneous breath, then performed slow breathing for 5 min and rapidly stood up within 3 s and remained standing for 15 min. Slow breathing intensified cardiac autonomic responses to postural maneuver.

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1. Introduction

Slow breathing has gained consideration because the literature reported positive physiological effects, including blood pressure reduction and improvement in chronic heart failure [1–3]. Reduction in the respiratory rate also acutely reduced anxiety and, it was considered sufficient to control physiological arousal during stressful conditions in musicians [4].

The effects of slow breathing include beneficial changes in the Autonomic Nervous System (ANS) [5]. It was demonstrated that slow breathing increases parasympathetic cardiac modulation and reduces sympathetic cardiac control [6]. In this manner, Heart Rate Variability (HRV) [7,8] is a simple and non-invasive method that analyzes cardiac autonomic regulation. It measures the fluctuation

of the intervals between consecutive heart beats (RR intervals) [9].

Previous studies have illustrated increases in HRV caused by chronic guided breathing sessions in hypertensive subjects. These studies support slow breathing as a complementary and alternative intervention for cardiovascular disorders [10,11].

Autonomic tests are applied to evaluate the adequate function of the ANS. A standard test applied in the clinical daily routine is the postural change maneuver (PCM). This test is based on the measurement of heart rate reflex changes in response to postural change stimulation [12].

A further study demonstrated that slow breathing training decreased baseline blood pressure and declined the pressor response to handgrip exercise [13]. Nonetheless, the acute effects of slow breathing on HRV responses to different autonomic tests are unclear. Additionally, non-pharmacological intervention is of assistance to add new elements in alternative and complementary therapies. In this situation, we investigated the acute effects of slow breathing on cardiac autonomic responses to PCM.

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2. Methods

2.1. Study population

The subjects participating in the study were 21 healthy male students - all non-smokers, aged between 18 and 35 years old. All volunteers were informed about the procedures and the objectives of the study and gave written informed consent. All study procedures were approved by the Ethics Committee in Research of our Institution (No. 2014-953), and were in accordance with Resolution 196/96 National Health 10/10/1996.

2.2. Non-inclusion criteria

We excluded subjects under the following conditions: Body Mass Index (BMI) > 35 kg/m²; systolic blood pressure (SBP) > 140 mmHg or diastolic blood pressure (DBP) > 90 mmHg (at rest); cardiovascular, respiratory, endocrine and reported neurological disorders that did not allow the volunteers to perform the procedures. Subjects under medication that influence the ANS were excluded.

2.3. Initial evaluation

Baseline information collected included age, gender, mass, height and Body Mass Index (BMI). Mass was determined using a digital scale (W 200/5, Welmy, Sao Paulo, Brazil) with a precision of 0.1 kg. Height was determined using a stadiometer (ES 2020, Sanny, Sao Paulo, Brazil) with a precision of 0.1 cm and 220 cm of extension. BMI was calculated as weight/height [2], with weight in kilograms and height in meters.

2.4. HRV analysis

HRV was analyzed according to instructions from the Task Force guidelines [7]. Instantaneous RR intervals (RRi) were recorded with a digital telemetry system, consisting of a transmitter placed on the patient's chest and a heart rate monitor (Polar® RS800CX; Polar Electro Oy, Kempele, Finland). This system detects ventricular depolarization, corresponding to the R wave on the electrocardiogram. This was achieved at a sampling rate of 500 Hz. It had been previously validated [14] and further downloaded to the Polar Precision Performance program (v.3.0, Polar Electro, Finland). The software enabled the visualization of heart rate and the extraction of a cardiac period (RR interval) file in "txt" format. Subsequent digital filtering complemented with manual filtering for the elimination of premature ectopic beats and artefacts, 256 RR intervals were applied for the data analysis. Only series with sinus rhythm greater than 95% were included in the study.

2.5. Time and frequency domain indices of HRV

For HRV analysis in the frequency domain we applied the spectral components of low frequency (LF: 0.04–0.15 Hz) and high frequency (HF: 0.15–0.40 Hz) in absolute (ms [2]) and in normalized units. The spectral analysis was calculated with the Fast Fourier Transform (FFT) algorithm [15].

Time domain analysis was accomplished through the SDNN (average standard deviation of normal RR intervals), pNN50 (percentage of adjacent RR intervals lasting more difference than 50 ms), RMSSD (square root of the average square differences between normal adjacent RR intervals) and SDNN/RMSSD ratio [16]. For analysis of linear indices in the frequency and time domain we applied the Kubios HRV® analysis software [17].

2.6. Slow breathing protocol

The slow breathing protocol was based on the literature which emphasized cycles with 10–12 s duration, corresponding to a breathing rate of 5–6 cycles per minute [18]. In this *modus operandi* the volunteers performed about 6 cycles per minute each with 10 s duration (0.1 Hz) for 5 min. The researcher guided the volunteer's breathing with a metronome. The volunteers were instructed to perform deep, but slow inspirations, and similar expirations with lung volumes ranging from the total lung volume to residual volume.

2.7. Experimental protocols

Data collection was undertaken in the same sound-proofed laboratory for all volunteers with the temperature between 20 °C and 26 °C; and relative humidity between 40% and 60%. Volunteers were instructed not to drink alcohol and/or caffeine for 24 h before evaluation and 24 h with no extenuous exercise, with a light meal at least 2 h before conducting tests. Datasets were collected on an individual basis, between 18:00 h and 21:00 h to standardize the circadian cycle. All procedures necessary for the data collection were explained on an individual basis and the subjects were instructed to remain at rest and avoid conversation during the data collection.

In the slow breathing protocol, the subject remained at rest seated for 10 min under spontaneous breathing. After 10 min, the volunteers performed slow breathing for 5 min and promptly stood up from a seated position within 3 s according to verbal command and remained standing for 15 min. In the control protocol, the subject remained at rest seated for 15 min under spontaneous breathing (Table 1). The sequence of the protocols was randomized.

2.8. Statistical analysis

Standard statistical methods were applied for the calculation of means and standard deviations. Normal Gaussian distribution of

Table 1
Experimental protocols.

Control protocol	10 min	10–15 min	15 min	15–20	20–25	25–30
	HRV recording under spontaneous breathing at seated rest.	HRV recording under spontaneous breathing at seated rest.	Change from seated to standing.	HRV recording at standing under spontaneous breathing.	HRV recording at standing under spontaneous breathing.	HRV recording at standing under spontaneous breathing.
Paced breathing protocol	10 min	10–15 min	15 min	15–20	20–25	25–30
	HRV recording under spontaneous breathing at seated rest.	Slow breathing at seated rest.	Change from seated to standing.	HRV recording at standing under spontaneous breathing.	HRV recording at standing under spontaneous breathing.	HRV recording at standing under spontaneous breathing.

Table 2

Baseline diastolic (DAP) and systolic arterial pressure (SAP), heart rate, mean RR interval, weight, height and body mass index (BMI) of the volunteers. Mean \pm standard-deviation. m: meters; ms: millisecond; kg: kilograms; bpm: beats per minute; mmHg: millimeters of mercury.

Variable	Value
Age (years)	21.5 \pm 2.5
Height (m)	1.78 \pm 0.1
Weight (kg)	78.3 \pm 17.9
BMI (kg/m ²)	24.5 \pm 4.9
Heart Rate (bpm)	77.8 \pm 10
Mean RR (ms)	788.108 \pm 61
SAP (mmHg)	118.3 \pm 10.9
DAP (mmHg)	72.9 \pm 11

the data was verified by the Shapiro–Wilk goodness-of-fit test (z value > 1.0). HRV was analyzed in the last 5 min of seated rest under spontaneous breathing, 0–5 min, 5–10 min and 10–15 min at standing under spontaneous breathing. For parametric distributions we applied paired one-way ANOVA followed by Bonferroni posttest. For non-parametric distributions we applied the Newman–Keuls posttest. Differences were considered significant when the probability of a Type I error was less than 5% ($p < 0.05$). We used the Biostat® Software 2009 Professional v5.8.4 (Analysis Soft, Walnut, California, USA).

3. Results

We observe data in Table 2 regarding baseline systolic arterial pressure (SAP) and diastolic arterial pressure (DAP), heart rate and mean RR interval, age, height, body mass and Body Mass Index (BMI).

Fig. 1 illustrates the time domain indices of HRV for the control protocol (at seated control) and during the 15 min after the volunteer stood up. We observed that the RMSSD and pNN50 reduced 10–15 min after the subjects stood up compared to control (seated vs. 10–15 min). The SDNN/RMSSD ratio was increased during the 15 min after the subjects stood up compared to control (seated vs. 0–15 min).

Regarding spectral analysis of HRV in the control protocol, the LF (nu) increased and HF (nu) declined 0–5 min and 10–15 min after the subjects stood up compared to control (Fig. 2).

Regarding the slow breathing protocol, the SDNN index increased 0–5 min after the subjects stood up compared to control. The RMSSD and pNN50 lowered 5–10 min and 10–15 min after the subjects stood up compared to control, whereas the SDNN/RMSSD index improved 0–15 min after the subjects stood up compared to control (Fig. 3).

Concerning the frequency domain analysis in the slow breathing protocol, the LF/HF ratio increased 0–5 min after the subjects stood up compared to control, the LF (nu) also increased 0–15 min after

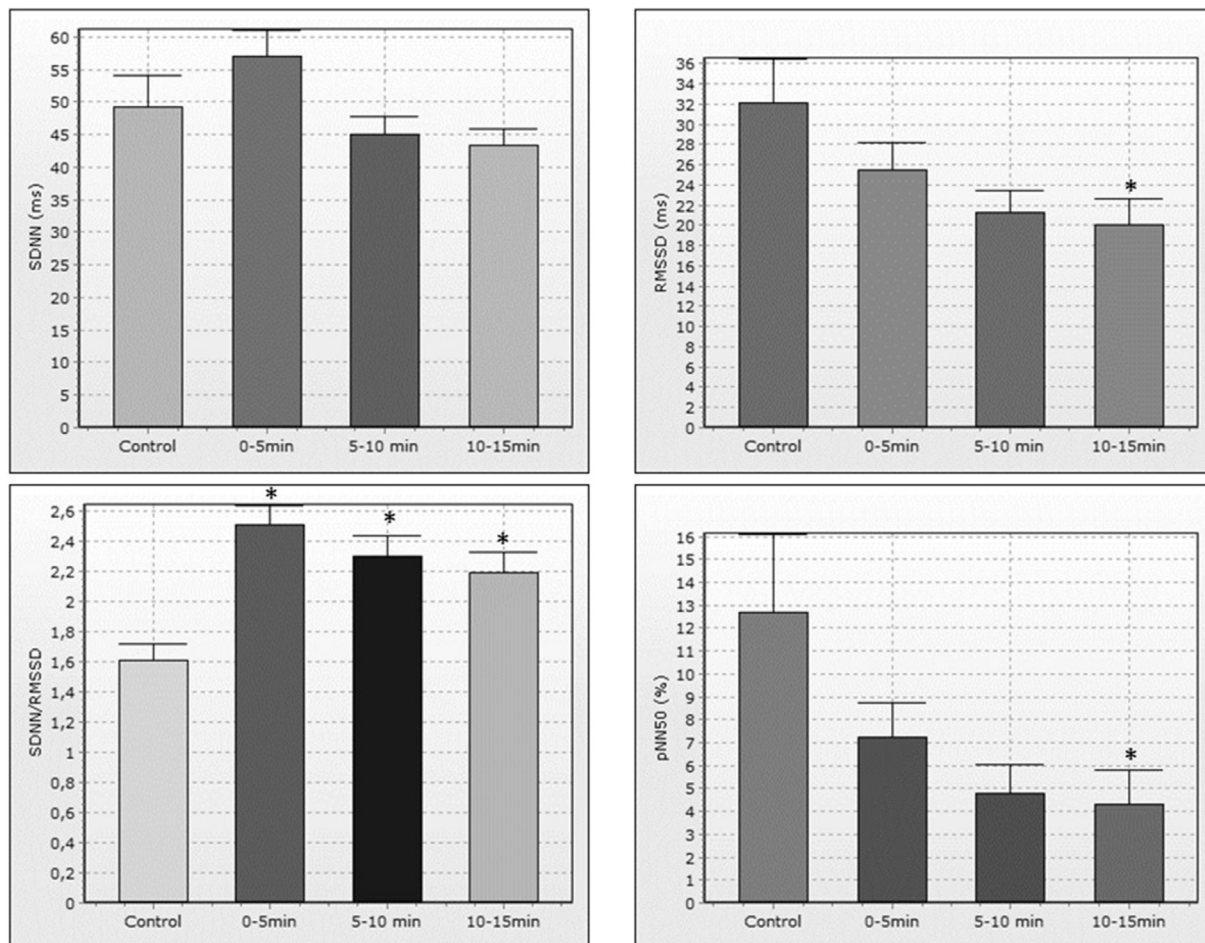


Fig. 1. Time domain indices of HRV before (Pre) and 0–5 min, 5–10 min and 10–15 min after PCM in the control protocol. SDNN: standard deviation of normal-to-normal RR intervals; pNN50: the percentage of adjacent RR intervals with a difference of duration greater than 50 ms; RMSSD: root-mean square of differences between adjacent normal RR intervals in a time interval; ms: milliseconds. * $p < 0.05$: vs. Control.

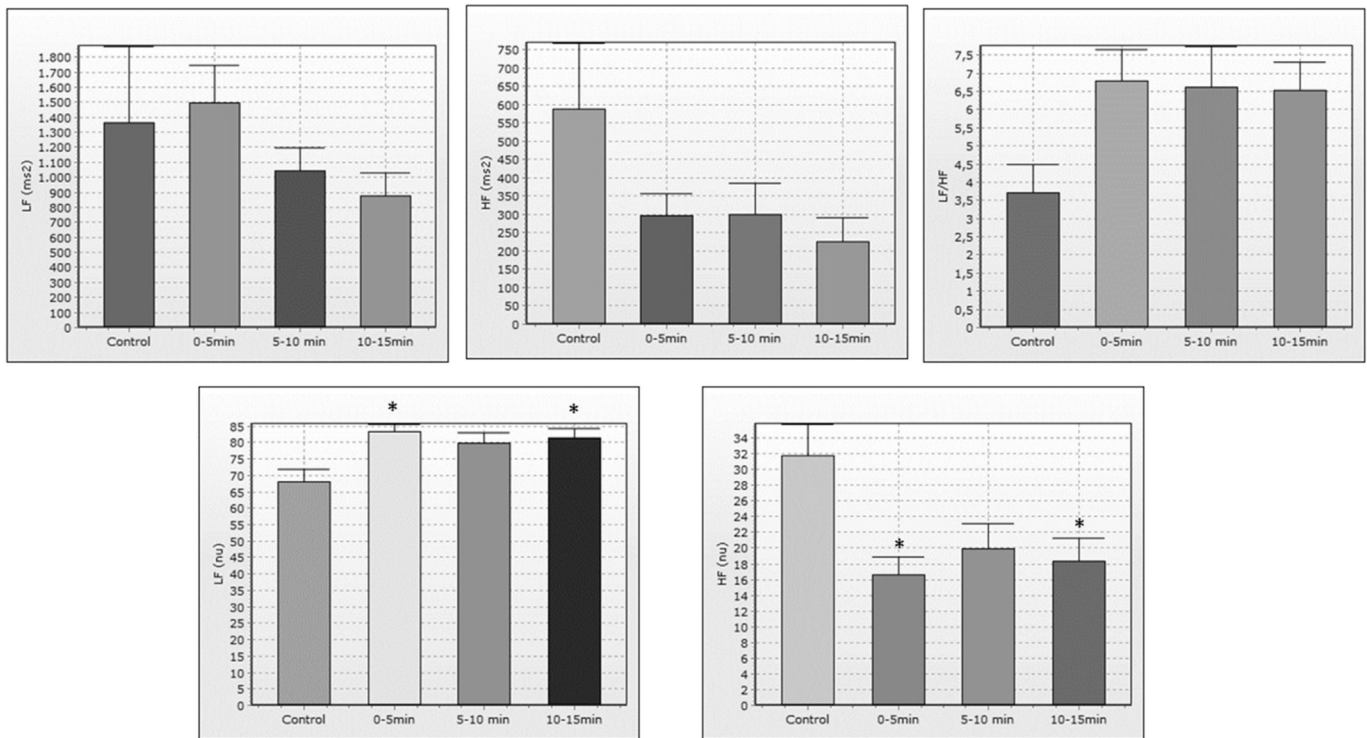


Fig. 2. Frequency domain indices of HRV before (Pre) and 0–5 min, 5–10 min and 10–15 min after PCM in the control protocol. LF: low frequency; HF: high frequency; LF/HF: low frequency/high frequency ratio; ms: milliseconds. *p < 0.05; vs. Control.

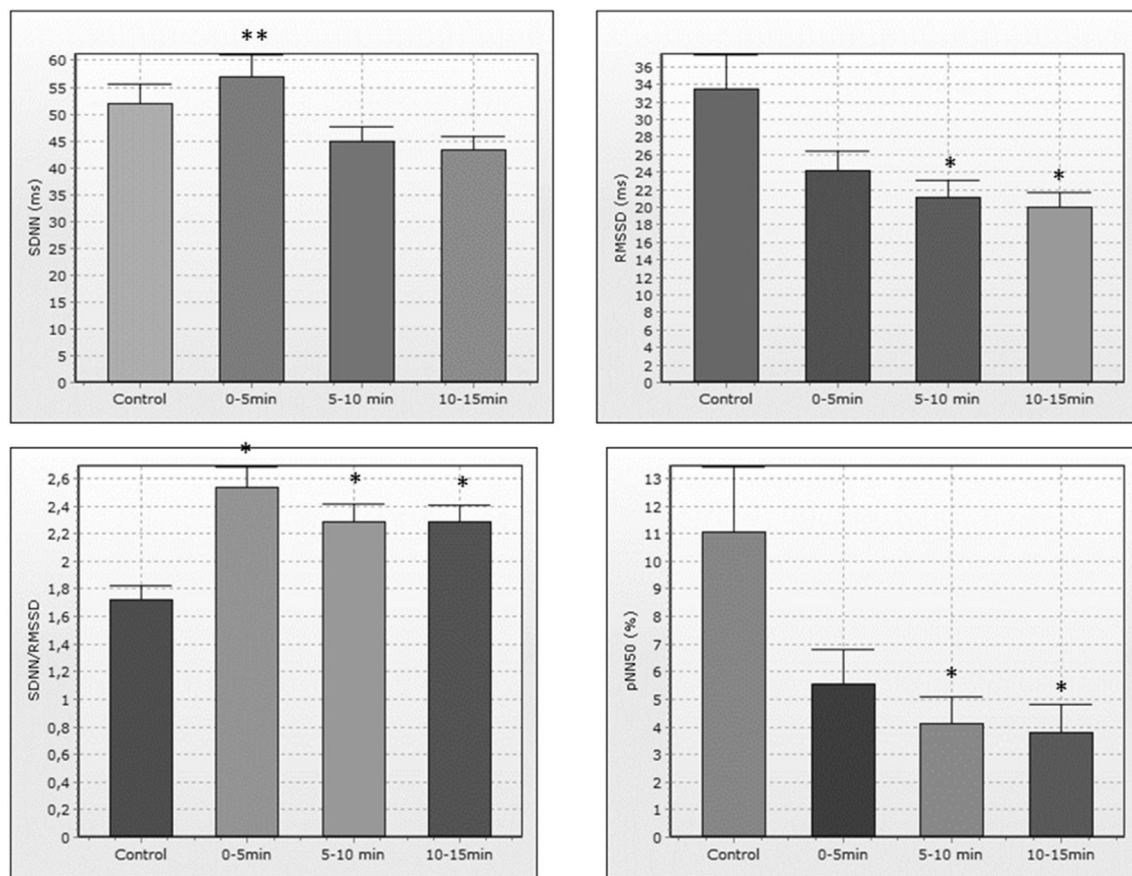


Fig. 3. Time domain indices of HRV before (Pre) and 0–5 min, 5–10 min and 10–15 min after PCM in the slow breathing protocol. SDNN: standard deviation of normal-to-normal RR intervals; pNN50: the percentage of adjacent RR intervals with a difference of duration greater than 50 ms; RMSSD: root-mean square of differences between adjacent normal RR intervals in a time interval; ms: milliseconds. *p < 0.05; vs. Control; **p < 0.05; vs. 10–15 min.

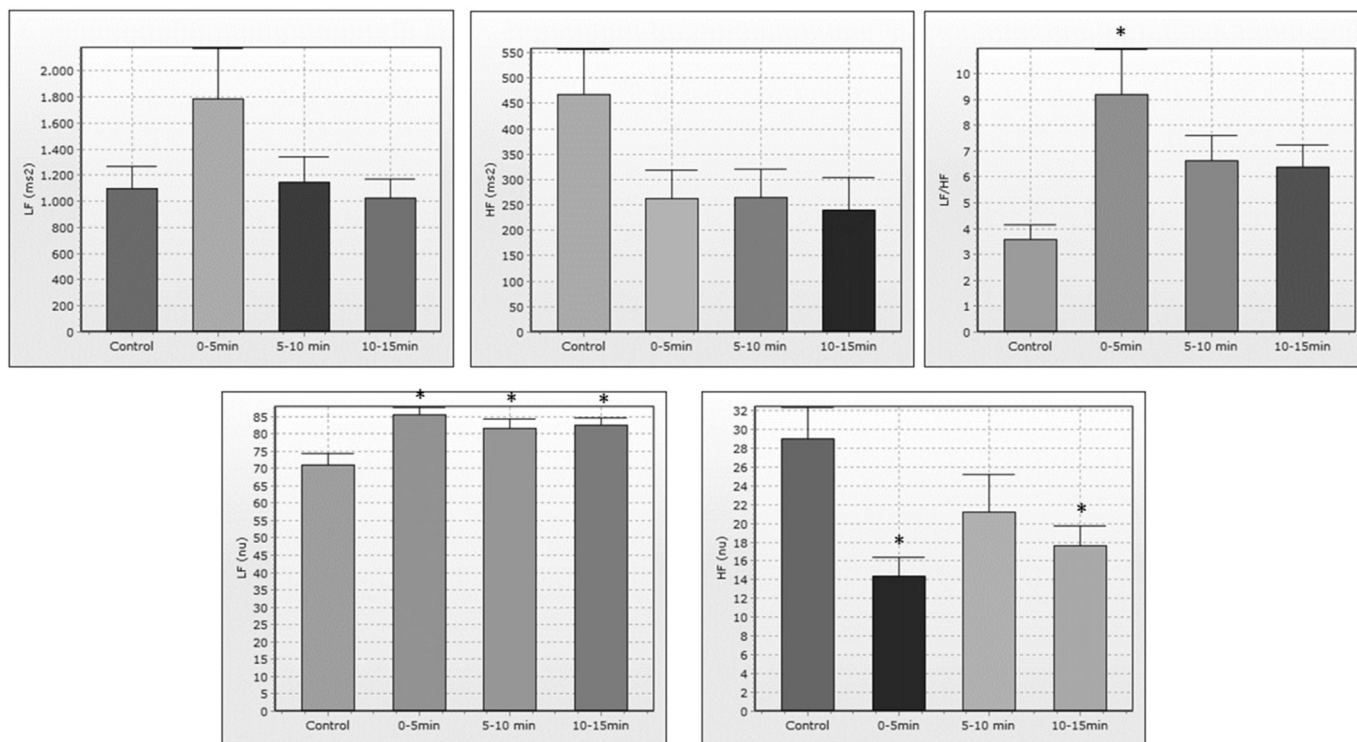


Fig. 4. Frequency domain indices of HRV before (Pre) and 0–5 min, 5–10 min and 10–15 min after PCM in the slow breathing protocol. LF: low frequency; HF: high frequency; LF/HF: low frequency/high frequency ratio; ms: milliseconds. * $p < 0.05$: vs. Control.

the subjects stood up compared to control and the HF (nu) decreased 0–5 and 10–15 min after the subjects stood up compared to control (Fig. 4).

4. Discussion

At this point we investigated the acute effects of slow breathing on cardiac autonomic responses elicited by varying from seated to orthostatis. Standard cardiac autonomic mechanisms involved in the PCM were evaluated in the control protocol that included spontaneous breathing instead of slow breathing. We noted more intense responses of time and frequency domain indices of HRV in the slow breathing protocol, indicating that it intensified cardiac autonomic reactivity.

Here the slow breathing protocol was based on 6 cycles per minute. Breathing patterns of approximately 6 cycles per minute coincides with spontaneous Mayer wave frequency, increasing cardiac oscillation by synchronizing sympathetic and vagal systems [19].

There is a synchrony between breathing and cardiac cycle, this phenomenon is called respiratory sinus arrhythmia [20]. Acute changes in breath causes alterations in heart rate and blood pressure. During inspiration heart rate increases due to the vagal withdrawal and during expiration the parasympathetic cardiac regulation rises over the sinus node and the heart rate decreases [21]. The slow breathing influences the cardiac regulation because it is associated with increased baroreflex sensitivity, HRV and cardiac input. It increases the parasympathetic modulation and/or reduces the sympathetic component. Thus, slow breathing is proposed as an alternative therapy to reduce blood pressure [22].

Based on our results, the SDNN index was not altered in the control protocol, while it significantly distorted in the initial 5 min after the PCM in the slow breathing protocol, indicating that slow breathing increased its responses. The SDNN index corresponds to

global heart rate modulation [7]. However, it does not provide information regarding whether the sympathetic or parasympathetic component of the ANS is modified.

The parasympathetic cardiac regulation is represented by RMSSD and pNN50 indices in the time domain and, by the HF index in the frequency domain [23]. It is well documented in the literature that those indices are reduced at standing compared to seated [24,25]. We reported reduced values of all aforesaid indices at orthostatic position compared to seated in the control and slow breathing protocols. More intense responses were observed in the slow breathing protocol, supporting that slow breathing at 6 cycles per minutes intensifies HRV responses to PCM.

Slow breathing has received consideration since it has been used as an alternative or complementary therapy for cardiovascular disorders. A previous study reported that slow breathing reduced arterial blood pressure and improved baroreflex sensitivity in essential hypertensive patients [26]. Another study found that slow breathing at the same rate used in our analysis decreased chemo-reflex responses induced by hypercapnia and hypoxia [27].

With regards to the sympathetic cardiac responses to PCM, the spectral analysis indicated that the LF/HF ratio presented significant responses 0–5 min after the volunteers stood up in the slow breathing protocol. Alternatively, the same index did not change in the control protocol, reinforcing the acute effects of slow breathing on cardiac autonomic responses.

Previous studies illustrated the involvement of slow breathing in sympathetic responses. Fonoberova et al. [28] performed a specific computational physiological technique to model cardiac autonomic regulation during slow breathing in humans. The authors reported beneficial effects of slow breathing on sympathetic component of the baroreflex. As follows, our study provides additional effects of slow breathing, since it improved cardiac sympathetic and parasympathetic reactivation induced by a specific autonomic test.

The higher cardiac autonomic responses to PCM caused by slow breathing observed in our study may be explained by its effects on controlling physiological arousal. Wells et al. [4] investigated the influence of acute slow breathing on cardiac autonomic modulation and anxiety. The authors reported that positive efficacy of slow breathing on HRV values in response to stress followed by reduction in anxiety levels in anxious subjects. Overall, we may hypothesize that slow breathing influenced cardiac autonomic responses to PCM due to its interaction with physiological stress reactivity.

Alterations in HRV patterns represents an early and sensitive indicator of cardiovascular events. Increased HRV corresponds to good adaptation of the ANS and, it is related to a healthy individual with well-organized autonomic activity [29]. Moreover, attenuated HRV responses to postural changes maneuver were found in patients with cardiovascular disorders [30,31].

Subjects with autonomic failure presented impaired cardiac autonomic responses to orthostatism, inducing postural hypotension caused by injury in parasympathetic activity [32]. Orthostatic hypotension is considered the hallmark of autonomic dysfunction, which was related to hypertension and pathologic ventricular hypertrophy [33]. Moreover, orthostatic hypotension was also designated as a risk factor for developing atrial fibrillation, coronary disorders, stroke, heart failure and chronic kidney disease [34–36]. Similarly, the slow breathing protocol applied in our study acutely improved autonomic responses, supporting its beneficial effects on ANS.

We may propose a hypothesis to explain the increase in HRV reactivity to postural change. It is feasible that previous parasympathetic activation induced by slow breathing intensified cardiac autonomic responses to PCM. The brainstem plays an important role in autonomic regulation. Areas such as surrounding the fourth cerebral ventricle – related to the integration between cardiopulmonary and baroreflex mechanisms. [37] In this circumstance we suggest that slow breathing changes baroreflex responses to PCM, leading to amplification of HRV responses to this maneuver.

Our results highlight the technique of slow breathing as an alternative or complementary therapy. It acutely increased parasympathetic cardiac regulation and improved the autonomic adaptation to respond to internal stimuli.

5. Conclusion

Slow breathing intensified cardiac autonomic responses induced by PCM from seated to orthostatic position. Our data supports the beneficial acute effects of slow breathing on HRV responses to a specific autonomic test.

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