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Influence of controlled breath on healthy adult autonomic heart modulation

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ABSTRACT. Heart rate variability (HRV) is a technique that indirectly assesses the activities of the autonomic nervous system in the heart. However, during data collection there are controversies regarding the use of spontaneous or controlled breath. The aim of this research was to compare the effects of controlled and spontaneous breath on HRV in healthy adults.The present cross-sectional study held a sample of 52 healthy volunteers (22 male, 42.3%), average of 25 (±4) years old and body mass index 22.4 (±2.7) kgm^{-2}. All the volunteers were evaluated in 2 days (at most three days between the evaluations). On each day the data were collected with spontaneous and then controlled breath (12 breaths per minute - bpm: Inspiration/Expiration: 2/3). Data were collected with a pulse frequency meter (Polar brand, model 810i). The HRV was evaluated for time and frequency domain and was analyzed using an area corresponding to 5 minutes (containing at least 256 beats per minute) by Kubios program version 2.1. In the time domain, no differences were observed between spontaneous and controlled breath. In the frequency domain, the controlled breathing reduced sympathetic activity ($p<0.001$) in approximately 14% and increased ($p<0.001$) parasympathetic activity proportionally. The autonomic modulation evaluated by low frequency/ high frequency(LF/HF) ratio decreased ($p<0.001$) in average -0.6 on the days evaluated. The controlled breathing (12 bpm) decrease sympathetic activity and increases parasympathetic by modifying the heart autonomic modulation.

Keywords: autonomic nervous system; sympathetic nervous system; parasympathetic nervous system; heart rate variability; respiration; respiratory rate.

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Introduction

Autonomic nervous system (ANS) is an important tool of homeostasis (Sasaki & Maruyama, 2014) and its activity is a neurohumoral regulation (Chang, Liu, & Shen, 2013). Its sympathetic and parasympathetic portions regulate cardiac and circulatory function, modulating heart rate, blood flow and systemic blood pressure (Vaseghi & Shivkumar, 2008). ANS activity in heart can be evaluate in a non-invasive method by the heart rate variability (HRV), and its dysfunctions are mortality and cardiovascular events predictor (McCraty & Shaffer, 2015; Quintana, Alvares, & Heathers, 2016; Sassi et al., 2015).

HRV can be evaluated in time domain (beats per minute) and frequency domain (ms^2) (McCraty & Shaffer, 2015; Quintana et al., 2016; Sassi et al., 2015). However, several factors can interfere in ANS and HRV, such as heart implants, age, gender, body mass index (BMI), medicine use, functional capacity, smoking, body position during collection, and so on (Dinas, Koutedakis, & Flouris, 2013; Karmali, Sciusco, May, & Ackland, 2017; Vidigal et al., 2016). The ANS has a relation with respiratory system, vagus nerve mediated (Sasaki & Maruyama, 2014), by respiratory sinus arrhythmia (RSA) mechanism, where inspiration and expiration inhibit or excite (respectively) vagal control over cardiac autonomic function (Berntson, Cacioppo, & Quigley, 1993).

Previous studies in HRV in healthy volunteers showed results only with spontaneous breath (Brown, Barnes, & Mündel, 2014; Landreani et al., 2017; Sima et al., 2017; Williams et al., 2015) and other ones only with controlled breath (Nardi et al., 2017; Liu et al., 2016; Stein, Dal Lago, Ferreira, Casali, & Plentz, 2011; Oliveira et al., 2022), and some studies with controlled and spontaneous breath (Kim, Bae, & Park, 2016; Levin & Swoap, 2019; Melo et al., 2018; Tavares et al., 2017). Other studies evaluated patients with schizophrenia (Liu et al., 2016), hypertension (Li, Chang, Zhang, & Chai, 2018), human immunodeficiency virus (Casarin et al., 2019) and asthma (Franco, Júnior, Signori, Prietsch, & Zhang, 2020), and the results were compared with

healthy volunteers. These differences in data collection can interfere in heart ANS activity, because variation and/or the breath control can produce autonomic heart changes (Li et al., 2018).Previous studies (Nardi et al., 2017; Liu et al., 2016; Stein et al., 2011; Oliveira et al., 2022), that even within physiological parameters reduced the respiratory rate (10–12 breaths per minute - bpm), may change the autonomic heart modulation, but the influence of this intervention is not yet determined, requiring further investigation. The objective of this study was to compare the effects of spontaneous (between 12 and 20 bpm) and controlled breath (12bpm; inhalation / exhalation: 2/3) on HRV of healthy young adults.

Methods

Design overview and Settings

The present cross-sectional cohort was carriedat the Federal University of Santa Maria and approved by the institutional Ethics Committee (Protocol: 2.180.257). The research follows the resolution nº 510/2016 of the Nacional Health Council and Helsinque declaration. All volunteers were informed about the study protocol and signed the free and informed consent form clarified before participation.Data were collected between October 2017 and April 2019 at the Clinical Research Laboratory of Federal University of Santa Maria.

Participants

All enrolled volunteers were literate, both genre, aged between 20 and 30 years-old, body mass index lower than 30 kgm^{-2}; non-smokers; and free of skeletal muscle, rheumatic, cardiovascular, metabolic, neurologic, oncologic, immune, hematologic, psychiatric or cognitive disorders. The enrolled volunteers were not taking any type of medication (except contraceptive).

Participants were instructed not to perform exhaustive exercises (48 hours before) and not to drink beverages containing caffeine or alcohol 12 hours before the exams. On the day of the exams, volunteers who presented values of blood pressure above normal (SBP > 120mmHg and DBP > 80mmHg) (Whelton & Carey, 2017), bradypnea (< 12 breaths per minute), tachypnea (> 20 breaths per minute) and tachycardia (> 90 beats per minute) were excluded from the study. Volunteers who reported stressful events and ingest of alcohol beverages that occurred in the last 48 hours also excluded from the study.

Outcomes and follow-up

The primary outcome measure was autonomic function, which was assessed by the heart rate variability in the time-domain and frequency-domain.

Evaluation

The volunteers were fasting for 8 hours. Before data collection, all volunteers performed three days of controlled breathing adaptation. Data collections (first and second evaluations) were performed within a maximum interval of 3 days between collections (Figure 1). Collections were always performed after spontaneous breathing and after controlled breathing. The volunteers were placed in the supine position and remained in this position for one hour (rest: 30min., data collection: 10min. spontaneous and 10min. controlled breath). The room temperature was maintained between 21 to 24ºC. During the spontaneous breath the volunteers did not suffer any interference, and the breath was monitored between 12 and 20 bpm by visual and multi-parameter monitor (Dixtal, model 2021, Manaus, Brazil) inspection. Data were collected with controlled breath (12bpm; inhalation/exhalation: 2/3) per 10 min.(Nardi et al., 2017; Liu et al., 2016; Stein et al., 2011; Oliveira et al., 2022). The control of breathing occurred through verbal stimulus performed by prior recording.

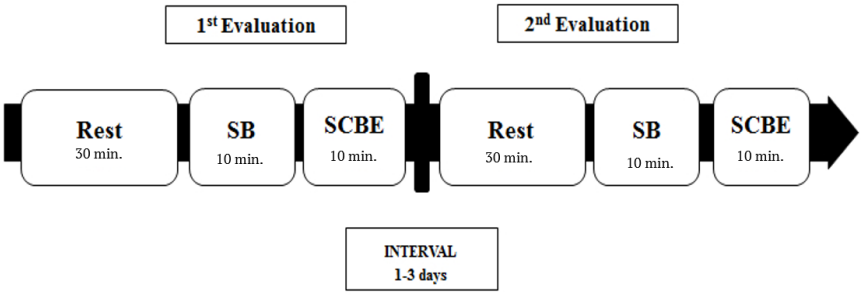

Figure 1. Experimental draw. min. = minutes; RE = rest; EB = spontaneous breath and CB = controlled breath.

Heart Rate Variability

The autonomic function was evaluated through the heart rate variability (Task Force ESC/NASPE, 1996) technique using a pulse frequency meter (Polar brand, model 810i, Kempele - Finland). Heart rate acquisition (sample rate – 1000 Hz) was performed in time series of the R-R intervals and acquired at continuous intervals (10 min.) before and immediately after the interventions. Data were downloaded on computer with Polar ProTrainer 5 software. Intervals R-R of 5 min. were selected (with at least 256 beats consecutive per minute) with a stable heart frequency, usually between 3 and 8 minutes of the entire 10 min. record. Each R-R serial of 5 min. was corrected by beats and aberrant errors using the correction algorithm of Polar ProTrainer 5 standard software (in other words, moderate power of the filter and minimal protection zone of 6bpm). Also, we inspected visually temporal serials by the R-R intervals and all the remaining artefacts were manually removed (Franco et al., 2020; Casarin et al., 2019; Oliveira et al., 2022).

Data were transferred for a computer and the R-R intervals were processed for heart rate variability calculation using the parameters of Kubios version 2.1 program (Biosignal Analysis and Medical Image Group, Department of Physics, University of Kuopio, Finland, 2012).

Spectral analysis of power was estimated using the fast Fourier transform algorithm. This analysis decomposes the heart rate variability into fundamental oscillatory components, the main ones being: high frequency component (HF) of 0.15 to 0.4 Hz, corresponding to respiratory modulation and to the indicator of the vagus nerve acting on the heart; low frequency component (LF) of 0.04 and 0.15 Hz, which is due to the joint action of the vagal and sympathetic components on the heart, predominantly sympathetic. Normalized units (n.u.) were obtained by dividing the power of a given component by the total power (from which VLF has been subtracted) and multiplying it by 100 (LF or HF/(Total Power – VLF) x 100). The LF/HF ratio reflects the absolute and relative changes between the sympathetic and parasympathetic components of the autonomic nervous system, characterizing the sympatho-vagal influence on the heart (McCraty & Shaffer, 2015).

The variables in the time-domain were the heart rate (HR), standard deviation of all normal-to-normal R-R (NN) interval (SDNN), square root of the mean of the squares of successive R-R interval differences (rMSSD), percentage of intervals differing more than 50ms different from preceding interval (PNN50%) and Triangular Index. At the frequency-domain were total power (TP), low frequency (LF), high frequency (HF) and sympatho-vagal function ratio (LF/HF).

Statistical analysis

Descriptive data were presented as mean and standard deviation (SD). Effects of breaths (spontaneous vs controlled) were compared (breath, time and interaction) by two-way analysis of variance for repeated measures (ANOVA), and post-hoc analysis was carried out by the Bonferroni test. Variations between significant results are reported as mean differences (MD) and 95% of confidence intervals (95%CI). All the statistical analyses were performed using the GraphPad Prism 5.0 software (San Diego, CA, EUA).

Ethics approval

The study was approved by the Human Research Ethics Committee (Protocol: 2.180.257). It was in accordance with the Declaration of Helsinki (2013) and Resolution 510/2016 of the National Health Council for research with human beings. All participants after explaining the study, signed the informed consent form.

Results

Initially 64 healthy volunteers were recruited, but eight were excluded (three had elevated arterial pressure, three performed exhausting exercises before the evaluation and two used medicines between the collects) and four did not attend the second evaluation. 52 healthy adults were included in this study (Figure 2). The sample had a mean age of 25 (±4) years, bodymass 69.8 (±10.3) kg, height 1.69 (±0.08) meters and body mass index (BMI) 24.4 (±2.7) kgm^{-2}. Male represented 42.3% (n=22) of the sample.The respiratory rate developed by the volunteers during the spontaneous breathing data collection was similar on the two days (1st day: 16.5 ± 1.7; 2nd day: 16.3 ± 1.5; p = 0.358).

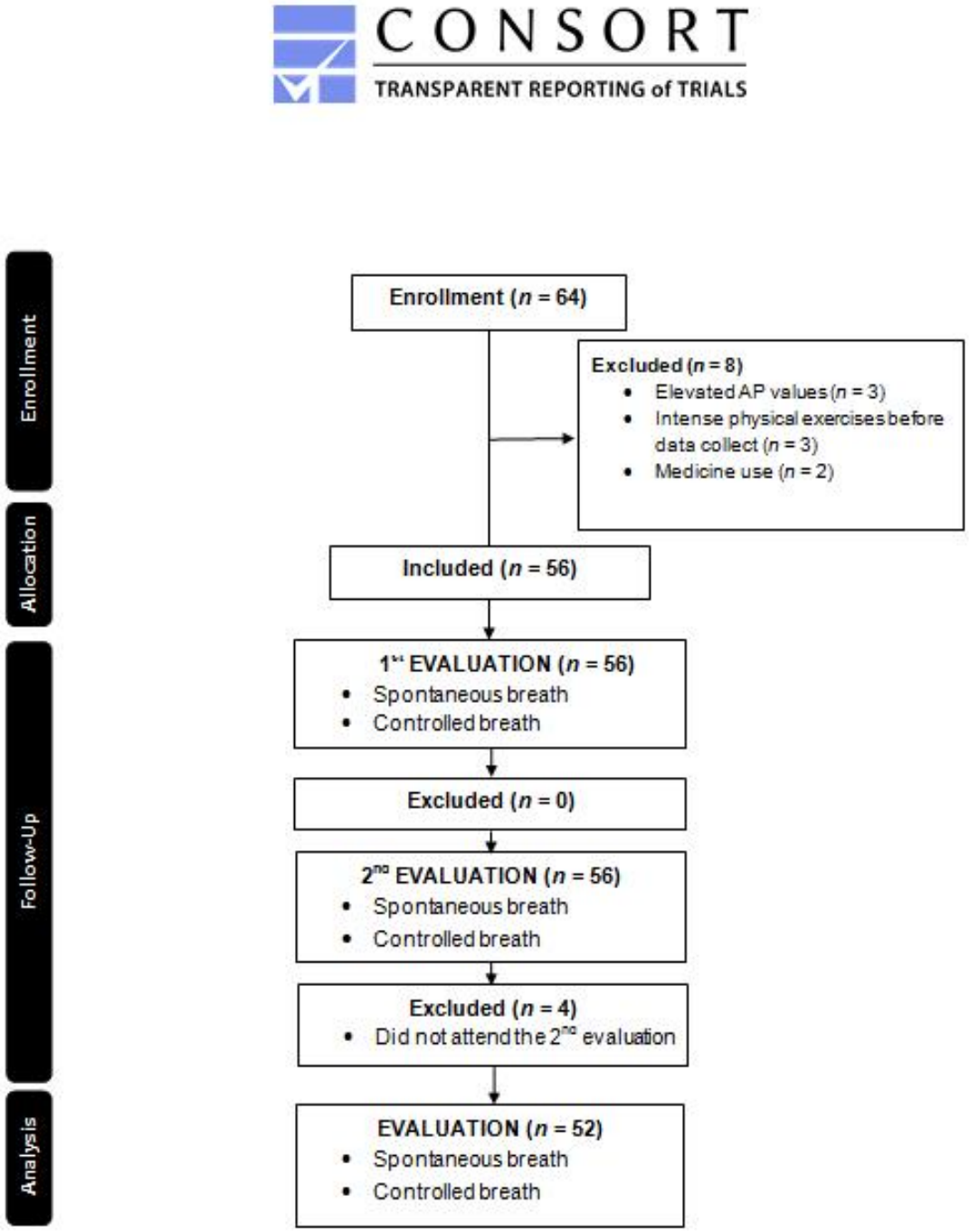

Figure 2. Study flowchart.AP = arterial pressure.

The results of heart rate variability performedare shown in Table 1. Time domain analysis showed no differences (breath, time and interaction) between analyzed variables (mean heart rate, pNN50 and triangular index). Frequency domain had no differences between Total Power (TP ms^2), High Frequency power (HF ms^2) and Low Frequency (LF ms^2) in two evaluations. The High frequency band power presented differences between breaths($p<0.036$), but there was no difference in confidence intervals (1^{st} evaluation MD: 665.3; 95%CI: -354.1 to1696 ms^2; 2^{nd} evaluation MD: 901.3; 95%CI: -129 to 1953 ms^2). After the unit's normalization, controlled breath ($p<0.001$) reduced sympathetic activity in approximately 14% (1^{st} evaluation MD: 13.6%; 95%CI: -7 to -20; 2^{nd} evaluation MD: -14.5%; 95%CI: -8 to -21%), and increased the parasympathetic activity (1^{st} evaluation MD: 13.6%; 95% CI: 7 to 20; 2^{nd} evaluation MD: 14.5%; 95% CI: 8 to 21%; Figure 3A and Figure3B). Controlled breathing decreased autonomic modulation by the reduction of the rate LF/HF (1^{st} evaluation MD: -0.65%; 95%CI: -0.26 to -1.04; 2^{nd} evaluation MD: -0.60%; IC95%: -0.19 to -0.97%; Figure 3C).

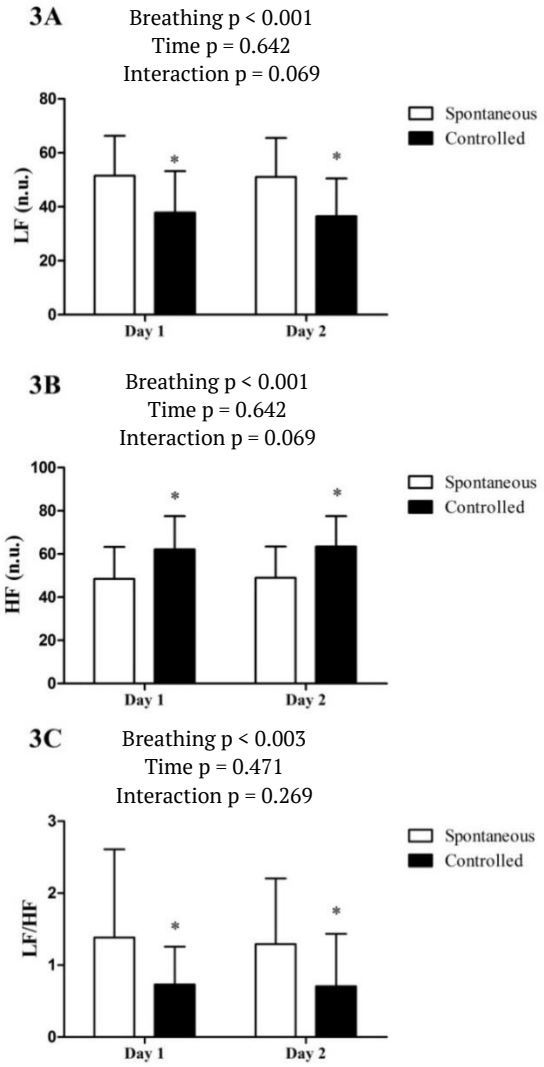

Figure 3. Normalized results of heart rate variability. LF power in normalized units; HF (n.u.) = HF power in normalized units; LF/HF = LF (ms^2)/HF (ms^2) ratio; *p < 0.05 vs breath.

Table 1. Summary of heart rate variability data.

Variable	Day 1		Day 2		p Value		
Time–Domain	Spontaneous Breathing	Controlled Breathing	Spontaneous Breathing	Controlled Breathing	Breathing	Time	Interaction
Mean HR (bpmmin.$^{-1}$)	67.1 ± 11	67.4 ± 11	67.1 ± 12	66.5 ± 11	0.648	0.219	0.476
SDNN (ms)	69.4 ± 33	75.9 ± 31	69.5 ± 30	72.2 ± 28	0.349	0.607	0.594
rMSSD (ms)	61 ± 40	65.8 ± 29	61.2 ± 37	67.9 ± 36	0.317	0.784	0.819
PNN50 (%)	32.3 ± 22	36.5 ± 18	32.6 ± 20	37.6 ± 21	0.166	0.807	0.813
Triangular index	14687 ± 5573	16037 ± 5517	14203 ± 3841	15112 ± 4502	0.059	0.588	0.588
Variable	Day 1		Day 2		p Value		
Frequency–Domain	Spontaneous Breathing	Controlled Breathing	Spontaneous Breathing	Controlled Breathing	Breathing	Time	Interaction
TP (ms^2)	5367 ± 5403	6443 ± 5807	5333 ± 6204	5533 ± 4649	0.479	0.442	0.869
LF (ms^2)	1672 ± 1721	1513 ± 1234	1570 ± 1790	1392 ± 1392	0.513	0.494	0.954
HF (ms^2)	1881 ± 2696	2546 ± 2152	1631 ± 2050	2533 ± 2356	0.036	0.626	0.662
LF (n.u.)	51.5 ± 15	37.8 ± 15*	51 ± 15	36.5 ± 14*	<0.001	0.533	0.753
HF (n.u.)	48.5 ± 15	62 ± 15*	48.9 ± 14	63.5 ± 14*	<0.001	0.536	0.758
LF/HF	1.38 ± 1.2	0.73 ± 0.53*	1.29 ± 0.9	0.71 ± 0.7*	<0.001	0.597	0.753

Data are presented as mean ± Standard Deviation (SD); HR = Heart Rate (bpmmin.$^{-1}$), SDNN = standard deviation of all normal to normal R-R (NN) intervals; rMSSD = Square root of the mean of the squares of successive R-R interval differences; pNN50 = the percentage of intervals differing more than 50ms different from preceding interval; Total power (TP ms^2) = The variance of RR intervals over the temporal segment; LF(ms^2) = Power in low frequency range (0.04-0.15Hz); HF(ms^2) = Power in high frequency range (0.15-0.4Hz); LF(n.u.) = LF power in normalized units; HF(n.u.) = HF power in normalized units; LF/HF = Ratio LF(ms^2) / HF(ms^2); * p < 0.05 vs Breathing.

Discussion

In the frequency domain, controlled breath decreased sympathetic activity (LF) and increased parasympathetic (HF) which modified the heart autonomic modulation (LF/HF ratio). Our results showed controlled breath (12 bpm; inhalation/expiration: 2/3) do not change HRV in time domain. The absence of changes in time domain possibly occurred because of the data collect (10 min. in our research) and, it was recommended at least 24 hours of analyses (Task Force ESC/NASPE, 1996). However, only the square root of the mean squares of successive R-R interval differences (rMSSD) can be considered in a short-term evaluation, in this case, spontaneous is recommended (Melo et al., 2018; Williams et al., 2015), but this variable did not change in this study.

Also, in the frequency domain, controlled breath induced a decrease in sympathetic activity, as showed in previous studies (Kim et al., 2016; Melo et al., 2018; Sasaki & Maruyama, 2014). Otherwise, the volunteers were not in supine position, and did notadapt a controlled breath previously to the data collection as in our study. Sasaki and Muruyama (2014) used mask in data collection, and probably it changed the results in sympathetic activity (Sasaki & Maruyama, 2014).

This study showed that the parasympathetic activity increases during the controlled breath. Spontaneous breath reflects the physiological function of the organism, nevertheless in normality parameters the evidences did not show the changes in autonomic function (Druschky, Lorenz, & Druschky, 2019). Otherwise, previous studies (Chang et al., 2013; Russo, Santarelli, & O'Rourke, 2017) showed the controlled breath can increase the parasympathetic activity, mainly in expiration phases, inducing a sympathetic decrease in heart muscle, by the respiratory sinus arrythmia (RSA) mechanism (Mestanik et al., 2019), corroborating with our research data.

It was shown that the controlled breath modifies the autonomic modulation, as it was found in previous studies (Li et al., 2018). Some studies showed different results comparing with our research (Levin & Swoap, 2019; Tavares et al., 2017). Levin and Swoap (2019) (n=55 volunteers) observed a parasympathetic activity increase during controlled breath (Levin & Swoap, 2019). However, differences in the data were observed, as the position of the collection, Levin and Swoap (2019) uses siting positions and the alternated nostril breath every 5 minutes (Levin & Swoap, 2019). Another study diverges from ours, such as Tavares et al. (2017).They used the controlled breath (5-6 bpm) and did not change HRV in healthy volunteers, but in this case, they used a verbal guide breath (Tavares et al., 2017). These differences between the results were because of the different body positions used, the recommended is using the supine position (Task Force ESC/NASPE, 1996) and, the alternated nostril breath and non-standard verbal instruction may induce stress/anxiety in ANS interfering in the results (Dishman et al., 2000). Nevertheless, Li et al. (2018) compared different breath frequencies (16 vs 8 bpm) in healthy volunteers (n=60) and hypertensive patients and showed that slow breath changes (decreases sympathetic and increases parasympathetic) cardiac autonomic modulation (LF/HF ratio) as found in our study. This study also showed that the slow breath (8 bpm) reduced cardiac frequency, arterial pressure and baroreflex activity in hypertensive patients (Li et al., 2018).

The decreased of sympathetic activity it isdue to the physiologic feedback mechanism (Berntson et al., 1993), possibly occurred in other studies (Li et al., 2018; Melo et al., 2018; Sasaki & Maruyama, 2014). The sympathetic system innervates the ventricles, characterizing the cardiovascular inotropic effect. On the other hand, parasympathetic system innervates atriums controlling the cardiac frequency (Liu et al., 2016; Lopes et al., 2014). The ANS regulate cardiac and respiratory function, and that function is influenced by the RSA (Mestanik et al., 2019). Respiratory changes send signal to the nucleus tractus solitarius and that excite (expiratory phases) or inhibits (inhalation phase) stimulating cranial nerves (especially valgus nerve) changing the HRV (Berntson et al., 1993). Therefore, the slow and controlled breath decreases the sympathetic and increases the parasympathetic activity, modifying the cardiac autonomic modulation (Russo et al., 2017), as observed in this study.

This study some limitations throughout the invasive evaluation ofthe ANS with the microneurography (Wehrwein, Orer, & Barman, 2016). Another one was the catecholaminesplasma concentration measurements (acetylcholine, adrenaline and norepinephrine) (Stacey et al., 2018) and the baroreflex sensibility (Ditterline et al., 2018). However, this technique is recommended by the Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology (Task Force ESC/NASPE, 1996). Another limitation is that the research was carried out with healthy volunteers, and care should be taken not to extrapolate these results for other populations.

Conclusion

Controlled breathing (12 bpm; inhalation/expiration: 2/3) even within parameters of normality, performed acutely changes the HRV in healthy adults. That was showed by a reduction of sympathetic activity and an increase of parasympathetic, which interfered in the autonomic heart modulation. These findings should be considered in future researches, since the controlled breathing modulates the autonomic responses during the HRV measurement. Furthermore, our data suggest the acute reductionon the breath movements, even within a normal range, can be a non-pharmacologic tool in the control of sympathetic hyperactivity in hypertensive individuals and especially in patients with hypertensive crises.

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