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Modulation of Parasympathetic Reactivation Post-Exercise via Slow Breathing

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AN ALL COLLEGE THESIS

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by

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Abstract:

During exercise sympathetic nervous activity increases and parasympathetic nervous activity decreases to fuel energy demands. Heart rate variability (HRV) indicates activity of the autonomic nervous system and the balance of its two branches. Slow breathing (SB) (6 breaths/minute) increases parasympathetic activity both spontaneously during practice and at rest after long-term practice. Thus, SB may increase parasympathetic activity after exercise. PURPOSE: This study aimed to determine whether post-exercise SB increases parasympathetic activity as measured by HRV. METHODS: Ten recreationally active college aged males (21 +/- 1 years old) completed the study. A VO_2 max test was used to determine 70% of VO_2 max for subsequent testing sessions. Each individual completed a control trial consisting of a 5-minute ECG (from V5) recording to measure baseline HRV, a 30-minute treadmill run at 70% VO_2 max, and a seated 30-minute recovery during which ECG was recorded. The experimental trial was completed after at least 48 hours and was identical to the control trial except for a SB intervention of 6 breaths/minute conducted during the first 10-minutes of recovery. HRV data were analyzed using frequency and time-domain analyses as well as non-linear analysis. 2x7 repeated measures ANOVAs and simple effects tests were conducted to determine differences between control and experimental trials in high frequency (HF) activity, low frequency (LF) activity, LF/HF ratio, standard deviation between consecutive R waves (SDRR), root mean square of standard deviation (RMSSD), and percent of consecutive R wave intervals greater than 50 milliseconds (PRR50) HRV variables. RESULTS: HF activity and LF/HF ratio did not significantly differ between control and experimental trials over time ($F(1.039, 9.35) = 2.178$; $p = 0.267$ and $F(1, 9) = 0.074$; $p = 0.792$). SDRR ($F(2.150, 19.351) = 17.359$; $p = <0.001$),

RMSSD (F(1.869, 16.819) = 7.464; p = 0.005), PRR50 (F(2.303, 20.727) = 7.186; p = 0.003), and LF (F(1.737, 15.629) = 9.107; p = 0.003) activity were all greater in the experimental trial than the control trial during the slow breathing intervention intervals of 0-5 minutes (SDRR p = 0.002; RMSSD p = 0.013; PRR50 p = 0.007; LF p = 0.01) and 5-10 minutes (SDRR p = 0.001; RMSSD p = 0.005; PRR50 p = 0.001; LF p = 0.004) . SDRR was also greater in the experimental trial than the control trial during the interval of 10-15 minutes (p = 0.049). CONCLUSION: SB post-exercise increased parasympathetic activity during the SB intervention as indicated by higher SDRR, RMSSD, and PRR50; but not after cessation of the SB intervention in SB naive participants. Elevated LF activity as well as nonsignificant HF and LF/HF ratio values indicate traditional frequency-domain HRV analysis was not effective in analyzing HRV during SB of 6 breaths/minute. SB may benefit athletes by increasing parasympathetic activity after exercise, but immediate post-exercise .appears to only have a reflexive effect.

Introduction:

Exercise reduces the activity of the parasympathetic nervous system (PNS) both during exercise and for up to an hour after cessation in healthy individuals (Weberruss et al., 2018). The parasympathetic nervous system is one branch of the autonomic nervous system (ANS) and operates alongside the sympathetic nervous system (SNS). The sympathetic branch of the ANS increases excitatory response to stressors while the parasympathetic branch stimulates functions generally related to resting and recovery. The activity of the ANS can be measured via heart rate variability (HRV). HRV measures the variation in time between consecutive heartbeats defined at the R wave in an ECG (Kleiger et al., 2005). High HRV corresponds to higher

parasympathetic output from the ANS (Draghici & Taylor, 2016). Because of its non-invasive nature HRV has traditionally been used to measure ANS activity in clinical settings and recently begun to be used to monitor athletic training (Kiviniemi et al., 2007; Kleiger et al., 2005). Cardiac chronotropy is controlled by the sinoatrial node which receives input from both branches of the ANS, PNS, and SNS (Draghici & Taylor, 2016). Increased SNS or decreased PNS activity cause cardio acceleration and lower HRV (Rajendra Acharya et al., 2006). Increased parasympathetic activity signifies a recovered state (Gifford et al., 2018). Therefore, HRV can be used to measure ANS activity, also known as sympathovagal balance.

Slow breathing, defined as breathing at 6 breaths per minute in this study, increases parasympathetic activity relative to sympathetic activity during breathing (Aysin & Aysin, 2006; Chang et al., 2015). Additionally, prolonged slow breathing practice, ranging from 6-8 breaths per minute, improves autonomic function by increasing parasympathetic activity and decreasing sympathetic activity at rest in healthy individuals (Pal et al., 2004; Turankar et al., 2013). Therefore, practicing slow breathing after exercise may instantly increase parasympathetic activity. The exact effects of SB after exercise on ANS activity must be characterized before the efficacy of post-exercise SB can be tested. It is unknown whether slow breathing has a purely reflexive influence on HRV or actually induces prolonged alterations in ANS activity after exercise.

The exact physiology of how slow breathing modulates the ANS remains unknown. Jerath et al., hypothesize that slow breathing produces two distinct signals which modulate the ANS: first, it activates slowly adapting stretch receptors (SARS) in the lungs as inhalation greater than tidal volume generates inhibitory impulses and the stretching of fibroblasts around the lungs generates hyperpolarization currents (Jerath et al., 2006). The combination of increased

inhibitory impulses and hyperpolarization currents lead to synchronization of neural tissues including the hypothalamus and brainstem increasing PNS tone (Jerath et al., 2006; Lutz et al., 2004).

Slow breathing's specific effects on the ANS via HRV measurement are more well defined. Carotid baroreflexes generate the majority of the variation in HRV during slow breathing in response to blood pressure changes as venous return increases during inspiration and decreases during expiration (Bernardi et al., 2001). These changes in venous return result from diaphragm contractions decreasing intrathoracic pressure and increasing the pressure gradient between the right atrium and systemic circulation (Wise et al., 1981). Increased pulmonary resistance occurs at the same time causing blood to pool in the pulmonary system, increased stroke volume during the next heartbeat, and a reduction in heart rate (HR) (Wise et al., 1981). These effects reverse during expiration and are relayed to the medulla oblongata via the carotid baroreceptors eliciting an increase in HR (Russo et al., 2017). As a result, HR increases during inspiration and slows during expiration. Rhythmic influence of respiration on HRV is termed respiratory sinus arrhythmia (RSA) (Russo et al., 2017). Variations in RSA and other cardiovascular fluctuations are maximized at 6 breaths per minute in the healthy human due to synchronization of blood flow and heart beat (Russo et al., 2017) (Bernardi et al., 2001). The PNS affects HRV at much higher frequencies (>0.1 Hz) because it acts through acetylcholine and therefore can pose a beat to beat effect (Russo et al., 2017). RSA is largely characterized as a vagal phenomenon because of this greater ability for the PNS to impose a beat to beat effect on HR (Zhang et al., 1997).

Jones et al. found that SB practice decreased resting heart rate and systolic blood pressure both at rest and during recovery after a hand grip exercise signifying that SB strains autonomic

balance via central regulatory pathways (Jones et al., 2015). The addition of SB with an exercise protocol may speed training of autonomic balance, by training central regulatory pathways, allowing athletes to maintain autonomic balance during high training loads. However, little research into the effects of slow breathing immediately after exercise exists. Tatsuya Sugimoto et al. found that after low intensity (50% peak oxygen uptake) and short duration (10 minutes) exercise slow breathing increased parasympathetic reactivation by increasing coefficient of variance between RR intervals and total power of HRV activity (Tatsuya Sugimoto et al., 2015). It is unknown whether slow breathing after exercise produces significant changes in the more common measures of HRV activity including HF power, LF power, LF/HF ratio, SDRR, rMSSD, or pRR50. It is also unknown whether changes in HRV parameters after exercise results in improved parasympathetic recovery after cessation of slow breathing.

Time-domain, frequency-domain, and non-linear analysis are all utilized throughout the literature for HRV analysis. Frequency domain analysis has shown to display an increase in LF activity when used in conjunction with SB (Sasaki & Maruyama, 2014). Increased LF activity during SB may be because of HF activity falling into lower frequencies due to cardiorespiratory coupling during RSA (Aysin & Aysin, 2006). Aysin and Aysin suggest that the use of breathing frequency analysis simultaneous with HRV to distinguish HF from LF activity during SB.

This study investigated the effects of slow breathing (6 breaths per minute) post-exercise on parasympathetic reactivation and ANS activity. It is important to identify the extent of the effects of slow breathing on parasympathetic reactivation after exercise to determine whether slow breathing should be tested to determine if it can be of benefit for athletes improving ANS fitness via central regulatory methods in addition to peripheral adaptations. The improvement of central regulatory methods may help athletes recover during high training loads (Jones et al.,

2015). Additionally, SB may be of benefit to athletes in recovering from exercise as it increases gastrointestinal activity and glycogenesis (McCorry, 2007). Higher intensity exercise of longer duration elicits greater parasympathetic withdrawal. Subjects were monitored after cessation of the slow breathing protocol to determine whether any changes in ANS activity remained once slow breathing was stopped. It was hypothesized that slow breathing after exercise would speed parasympathetic reactivation leading to increased indices of parasympathetic HRV activity across the entire recovery of the slow breathing condition.

Methods:

Participants:

Participants of this study were ten male college students (21 $\pm$ 1 years old) and were recreationally active (participating in at least 150 minutes of moderate to vigorous physically activity per week) and without any current musculoskeletal injuries. All methods were approved by the Institutional Review Board and all subjects gave informed consent before participation.

Methods:

Data were collected over three exercise sessions: an initial graded VO_2max test to identify the speed and treadmill grade necessary to elicit 70% of VO_2max and two experimental sessions during which baseline HRV data (one 5 min. epoch) was collected and the participants ran for 32 minutes at 70% of VO_2max followed by 30 minutes of recovery monitored by ECG. Both experimental sessions took place at the same time of day (within one hour) to control for circadian fluctuations in HRV (Kleiger et al., 2005). The first session served as a familiarization

trial during which the participants were shown how the ECG data would be collected (via a 5-lead system) and instructed in the slow breathing technique and visual metronome (https://www.grc.com/breathe.htm). Criteria for confirmation that a true VO_2max was reached during the familiarization and VO_2max testing session were the same as used by Chapman and Stager (Chapman & Stager, 2008). These criteria for assessment of VO_2max included 1) a heart rate (HR) of $\pm$ 10% of age-predicted maximum, 2) a respiratory exchange ratio (RER) of 1.10 or higher, and 3) a plateau ($\leq$150-mL increase) in VO_2 with an increase in workload. If two of the three criteria were met, the highest VO_2 recorded was chosen as the subject's VO_2max (Chapman & Stager, 2008). All participants met at least two of these inclusion criteria for a maximal VO_2max test.

The procedures for the familiarization trial was as follows. Demographic and physical data including age, height, weight, and physical activity level were collected. The researcher instructed the participant through a total body dynamic warm up including high knees, butt kickers, lunges, and arm circles prior to testing. Participants were then hooked up to the metabolic cart via a facemask and mouthpiece. A nosepiece and heart rate monitor were also applied, and participants were secured in a harness for safety in the event that they fell during the test. The test was a standard incremental treadmill test at a speed chosen by the participants with the slope of the treadmill increasing 2 percent every two minutes until volitional fatigue or at least two of the inclusion criteria were met (Chapman & Stager, 2008)

The procedures for the ECG data collection methods for the experimental and control trials were as follows: ECG recordings were collected via a five-lead system in five minutes intervals. The five ECG leads were attached to the underside of each wrist of the participant, the fifth intercostal space, and the lateral side of each ankle.

The second session entailed a 32-minute treadmill run at 70% of VO_2max established in the familiarization trial. The intensity and duration were chosen to elicit post-exercise hypotension and parasympathetic withdrawal (MacDonald et al., 2000; Michael et al., 2017). Higher intensity exercise of longer duration was chosen in order to elicit greater parasympathetic withdrawal than previous research. Baseline HRV and HR values were recorded via ECG (one 5-minute epoch) following the same procedures detailed in the familiarization trial. The researcher instructed the participant through a total body dynamic warm up including high knees, butt kickers, lunges, and arm circles prior to testing. The participants then completed the treadmill run. After the completion of the test, participants were seated in a chair and toweled off to remove sweat then hooked up to the ECG. HRV data was collected for 30 minutes. Participants remained seated quietly with their eyes closed for the duration of the recovery.

The final session was identical to the second with the addition of a slow breathing protocol using a visual metronome (https://www.grc.com/breathe.htm) for the first 10 minutes of data collection and beginning approximately 2 minutes after exercise. HRV measurement continued for 30 minutes just as during the control experimental session. All testing took place in the College of St. Benedict exercise science and sports studies laboratory.

HRV data were recorded before (one 5-minute interval) and after (six 5-minute intervals) treadmill exercise at 70% of VO_2max to determine whether slow breathing (experimental session) increased parasympathetic reactivation after exercise as compared to simply quiet seated recovery (control). HRV data recorded during the control (no slow breathing) and experimental (slow breathing for the first 10 minutes of recover) sessions were compared using 2x7 repeated measures ANNOVAs and a simple effects test. Significance was determined to be at p values <

0.05. Outputs were measured using time-domain (SDRR, RMSSD, and PRR50), frequency (LF activity, HF activity, and LF/HF ratio), and non-linear analysis (SD1 and SD2).

Results:

No significant interaction between time and treatment was found for HF activity ($F(1.039, 9.35) = 2.178$; $p = 0.267$). Also, no significant interaction between treatments was found for LF/HF ratio ($F(1, 9) = 0.074$; $p = 0.792$).

RMSSD (p: 5 = 0.013 and 10 = 0.005) (figure 2), PRR50 (p: 5 = 0.007 and 10 = 0.001) (figure 3), SD1 (p: 5 = 0.013 and 10 = 0.005) (figure 4), and LF activity (p: 5 = 0.01 and 10 = 0.004) (figure 6) were all greater in the experimental trial than the control trial during the slow breathing technique intervals of 0-5 minutes and 5-10 minutes at the beginning of the recovery. None of them were significantly different between trials at baseline or time intervals 10-15 minutes, 15-20 minutes, 20-25 minutes, or 25-30 minutes.

SDRR (p: 5 = 0.002, 10 = 0.001, and 15 = 0.049) (figure 1) and SD2 (p: 5 = 0.002, 10 = 0.001, and 15 = 0.047) (figure 5) were both greater in the experimental trial than the control trial during the slow breathing technique time intervals of 0-5 minutes and 5-10 minutes and the subsequent five-minute interval of 10-15 minutes. Neither were significantly different between trials at baseline or time intervals 15-20 minutes, 20-25 minutes, or 25-30 minutes.

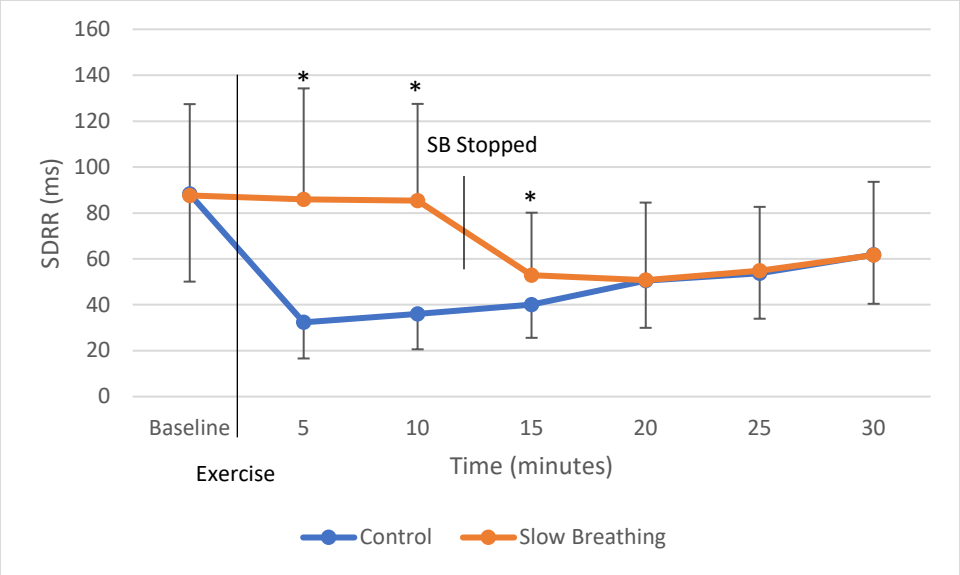

Figure 1. Displays the interaction between SDRR in milliseconds across time in minutes from baseline (before exercise) until 30 minutes after exercise cessation. There was a significant interaction between treatment and time for SDRR (F (2.150, 19.351) = 17.359; p = <0.001). * indicates statistical significance at the level of $p < 0.05$ (5 = 0.002, 10 = 0.001, and 15 = 0.049).

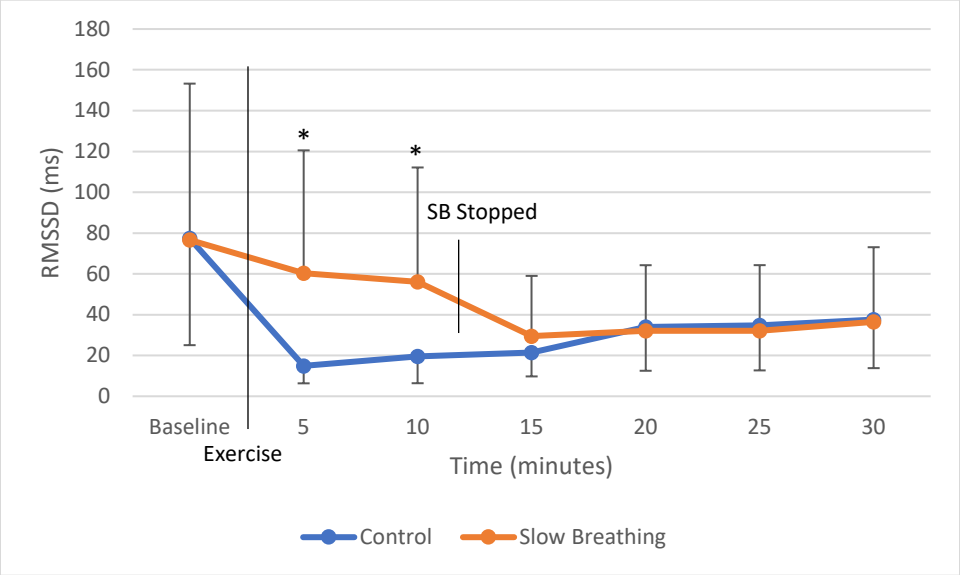

Figure 2. Displays the interaction between RMSSD in milliseconds across time in minutes from baseline (before exercise) until 30 minutes after exercise cessation. There was a significant

interaction between treatment and time for RMSSD (F (1.869, 16.819) = 7.464; p = 0.005). * indicates statistical significance at the level of $p < 0.05$ (5 = 0.013 and 10 = 0.005).

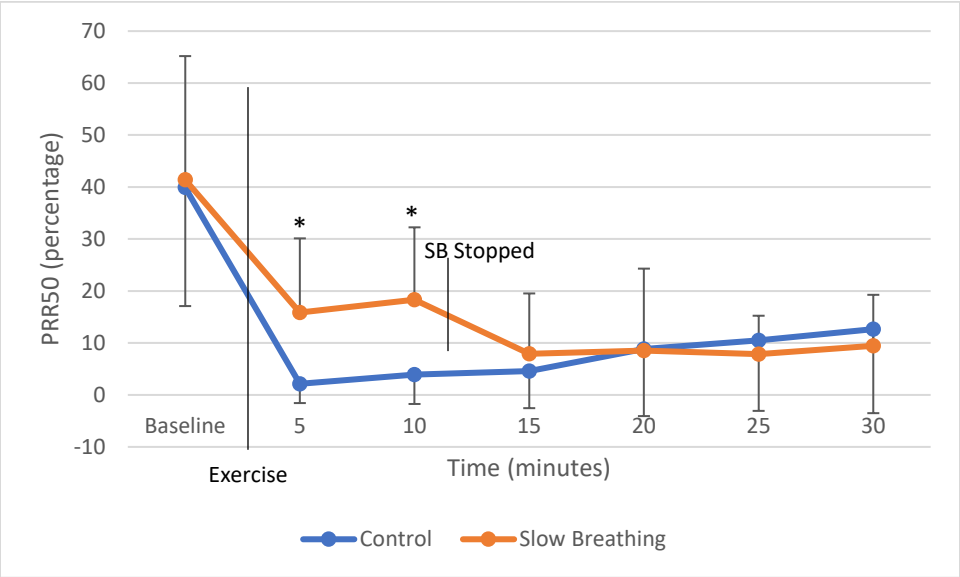

Figure 3. Displays the interaction between PRR50 in percent across time in minutes from baseline (before exercise) until 30 minutes after exercise cessation. There was a significant interaction between treatment and time for pRR50 (F (2.303, 20.727) = 7.186; p = 0.003). * indicates statistical significance at the level of $p < 0.05$ (5 = 0.007 and 10 = 0.001).

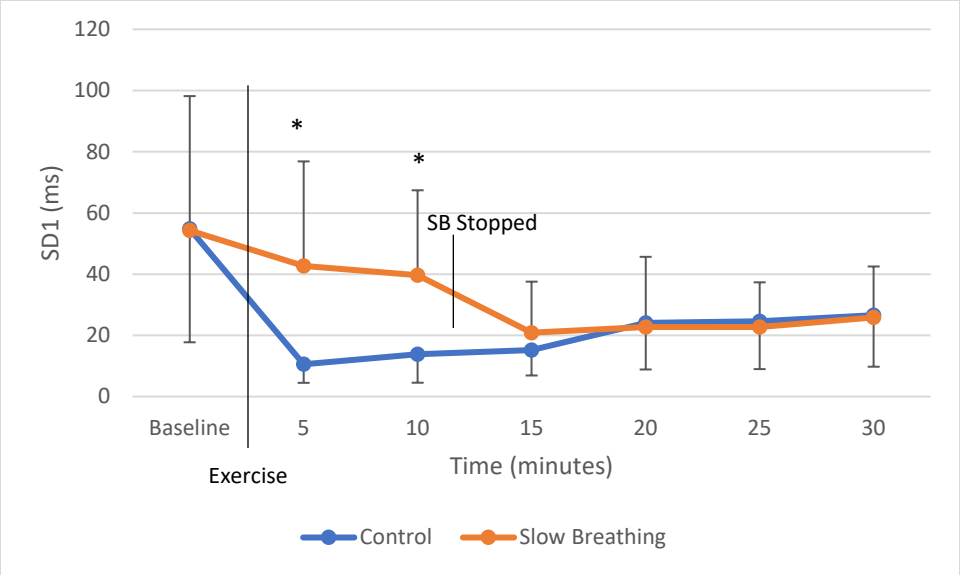

Figure 4. Displays the interaction between SD1 in milliseconds across time in minutes from baseline (before exercise) until 30 minutes after exercise cessation. There was a significant interaction between treatment and time for SD1 ($F(1.869, 16.825) = 7.462$; $p = 0.005$). * indicates statistical significance at the level of $p < 0.05$ (5 = 0.013 and 10 = 0.005).

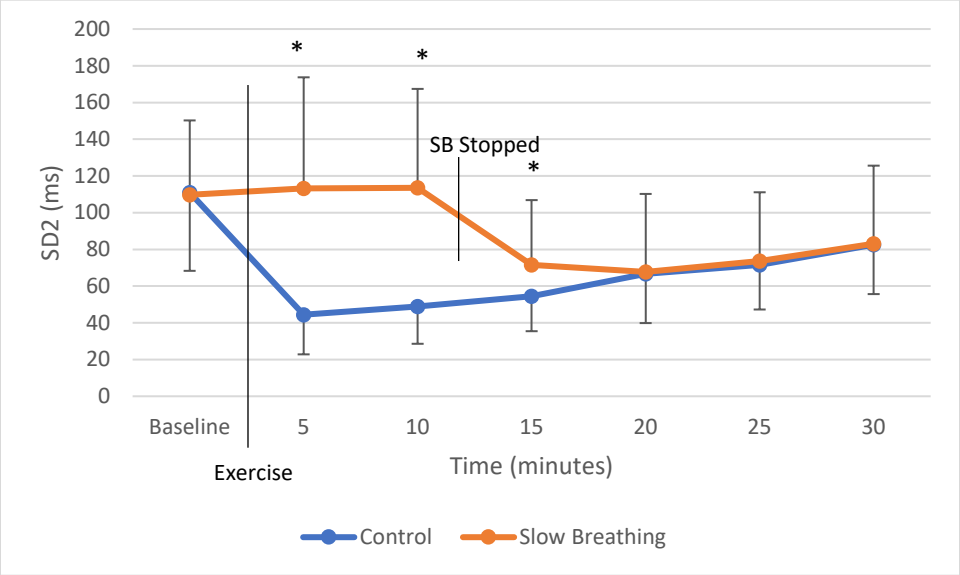

Figure 5. Displays the interaction between SD2 in milliseconds across time in minutes from baseline (before exercise) until 30 minutes after exercise cessation. There was a significant

interaction between treatment and time for SD2 (F (2.324, 20.919) = 18.556; p = >0.001). * indicates statistical significance at the level of $p < 0.05$ (5 = 0.002, 10 = 0.001, and 15 = 0.047).

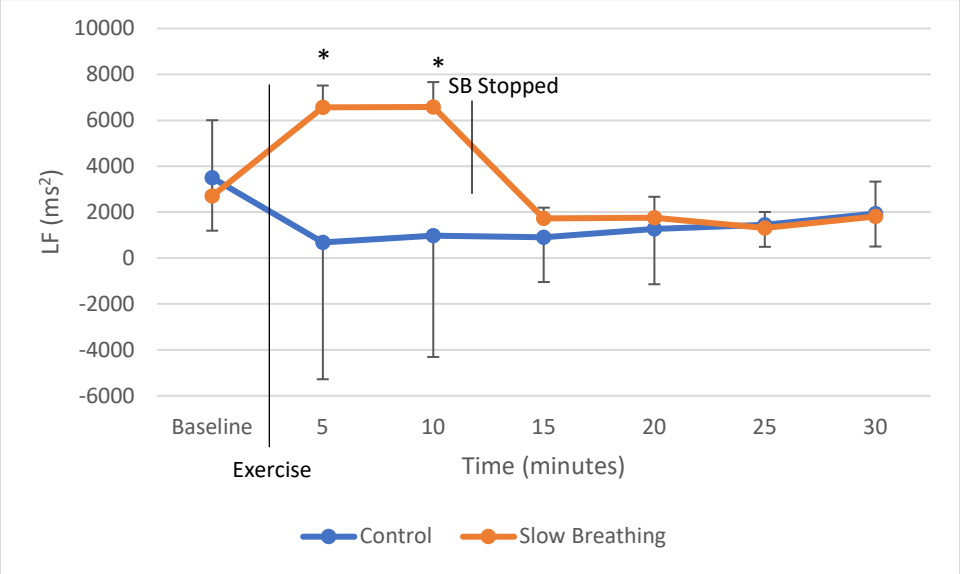

Figure 6. Displays the interaction between LF activity in milliseconds^2 across time in minutes from baseline (before exercise) until 30 minutes after exercise cessation. There was a significant interaction between treatment and time for LF activity (F (1.737, 15.629) = 9.107; p = 0.003). * indicates statistical significance at the level of $p < 0.05$ (5 = 0.01 and 10 = 0.004).

Discussion:

Parameters of parasympathetic activity increased across all measurements except HF activity, LF activity, and LF/HF ratio during the slow breathing protocol compared to the control recovery. Slow breathing did successfully increase parasympathetic activity during the slow breathing practice immediately after cessation of exercise confirming prior findings by Aysin and Aysin and Chang et al. (Aysin & Aysin, 2006; Chang et al., 2015). However, the effects of slow breathing diminished after cessation of the technique as only SDRR and SD2 were significantly different between the control and experimental treatments after the slow breathing

stopped. Furthermore, none of the observed parameters were statistically significantly different between control and experimental trials across the final three 5-minute recordings of the recoveries. Therefore, it appears that SB had a purely reflexive effect on parasympathetic reactivation and ANS activity after exercise.

Interestingly, LF activity increased during the slow breathing technique and was significantly different from the control recovery. This would indicate increased sympathetic activity and seems to directly contradict the increase in SDRR, RMSSD, PRR50, SD1, and SD2 indicated during the slow breathing protocol. Increases in these variables all indicate an increase in parasympathetic activity. Sasaki and Maruyama found that slow breathing causes an increase in LF activity (Sasaki & Maruyama, 2014). Although, as mentioned before, frequency domain analysis should be analyzed carefully as the preset parameters for LF and HF activity describe activity during spontaneous breathing. Aysin and Aysin suggest the use of breathing frequency analysis simultaneous with HRV to determine the frequencies over which LF and HF activity are actually displayed across during slow breathing (Aysin & Aysin, 2006). Increased LF activity during SB should not be misconstrued as it may be due to shifting of parasympathetic activity to lower frequencies than the normal HF range seemingly increasing sympathetic and decreasing parasympathetic activity (Aysin & Aysin, 2006). The findings of this study appear to support their conclusion that slow breathing does not necessarily increase sympathetic activity despite an apparent increase in LF activity as every time-domain and non-linear measurement indicates the opposite, increased parasympathetic activity. Frequency domain analysis should be used cautiously in interpreting HRV during slow breathing unless breathing frequency is also analyzed and used to correct analysis of HRV (Aysin & Aysin, 2006). The use of corrected HRV analysis should be a target of future research.

Prior researchers have found slow breathing increases parasympathetic reactivation after exercise, similar to the findings of this study via time-domain and non-linear analysis (Tatsuya Sugimoto et al., 2015). However, prior researchers had not continued to measure HRV after cessation of the slow breathing technique. Additionally, greater intensity and longer duration exercise was utilized than previous studies (Tatsuya Sugimoto et al., 2015). Therefore, future research should delve into the combination of SB and exercise without necessarily focusing on immediate SB after exercise. Importantly, this study found that there was no prolonged benefit to ANS activity after cessation of SB practice immediately following exercise which runs directly counter to prior findings (Tatsuya Sugimoto et al., 2015).

Future research should explore the effects of slow breathing in conjunction with exercise and not necessarily immediately afterwards. This research would allow for the elucidation of whether the combination of SB and exercise has a greater impact on ANS balance for athletes as immediate post-exercise SB appears to have only reflexive effects. It should also monitor HRV over the course of a 24-hour ambulatory ECG interval to determine whether parasympathetic recovery after slow breathing post-exercise has effects on recovery over longer periods of time or possibly very-low frequency (VLF) HRV activity. All HRV data should also be collected at the same time of day rather than just the same for control and experimental sessions for each individual participant to control for fluctuations in total HRV activity due to circadian rhythm fluctuations. Also, future research should use non-naïve SB practitioners and/or use long-term SB practice in conjunction with exercise to determine whether these alterations elicit prolonged benefits to parasympathetic reactivation after cessation of the SB technique.

In conclusion, this study confirmed that slow breathing increases parasympathetic activity during the slow breathing protocol (Aysin & Aysin, 2006; Chang et al., 2015). Potential

for traditional frequency-domain analysis to show an increase in LF activity, and therefore sympathetic activity, despite other forms of analysis indicating an increase in parasympathetic activity was also confirmed. Such findings may occur especially if breathing frequency is not measured and used to adjust frequency domain intervals (Aysin & Aysin, 2006; Sasaki & Maruyama, 2014). The ability for post-exercise SB to significantly increase parasympathetic activity after cessation of slow breathing could not be confirmed; thus, future research should explore the combination of non-post-exercise SB practice and exercise on ANS control at rest and during recovery from exercise.

Appendix:

HRV Measurement Key:

SDRR: standard deviation between consecutive normal heart beats as measure from R, greater SDRR indicates a parasympathetic state

RMSSD: root mean square of successive RR interval differences, greater RMSSD indicates a parasympathetic state

PRR50: percentage of RR intervals that vary by greater than 50 milliseconds, greater PRR50 indicates a parasympathetic state

HF: high frequency HRV activity between 0.15 and 0.40 Hz; roughly equivalent to parasympathetic activity

LF: low frequency HRV activity between 0.04 and 0.15 Hz; roughly equivalent to sympathetic activity

LF/HF: ratio of low frequency activity to high frequency activity; roughly equivalent to sympathovagal balance

SD1: standard deviation of instantaneous beat-to-beat variability via Poincare plot*, greater SD1 indicates a parasympathetic state

SD2: continuous RR interval variability via a Poincare plot*, greater SD2 indicates a parasympathetic state

*The Poincaré plot is a scatter plot of RR_n vs. RR_{n+1} where RR_n is the time between two successive R peaks and RR_{n+1} is the time between the next two successive R peaks (Bhaskar & Ghatak, 2013).

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