International Journal of EXERCISE SCIENCE

Original Research

The Influence of a Slow-Breathing Protocol on Heart Rate and Blood Pressure from Exercise in Moderately Trained Females

EMILY L. ZUMBRO†[1], GREG A. RYAN‡[2], STEPHEN J. ROSSI‡[2], and JOHN L. DOBSON‡[2]

[1]Department of Kinesiology, Texas Woman's University, Denton, TX, USA; [2]Department of Kinesiology, Georgia Southern University, Statesboro, GA, USA

†Denotes graduate student author, ‡Denotes professional author

ABSTRACT

International Journal of Exercise Science 12(2): 714-725, 2019. Heart rate recovery (HRR) and blood pressure recovery (BPR) from exercise are both important indicators of health and fitness and are strongly associated with cardiovascular disease. The purpose of this study was to compare the effects of a slow-breathing technique, upright passive recovery (PASS), and active recovery (ACT) on HRR and BPR from exercise. Nine moderately trained, college-aged (20.22 ± 0.97 yrs) female participants cycled three times on an ergometer for 15 mins at 70% of their heart rate maximum (HR_{max}), each of which was followed by one of three 5 min recovery interventions with heart rate (HR) and blood pressure (BP) objectively measured. Each participant completed all three recovery protocols. One recovery protocol consisted of breathing at a rate of 6 breaths per minute (BRE), another involved PASS and the third was ACT at 60 RPM and 25 W. A repeated measures ANOVA revealed there was a significant effect of protocol (p = 0.00, η_p^2 = 0.67) with HRR. BRE resulted in the fastest HRR of 69 ± 9.31 bpm (40.12%) at the end of the 5 min recovery compared to 63 ± 10.60 bpm (36.57%) and 47 ± 12.54 bpm (27.34%) for PASS and ACT, respectively. A second repeated measures ANOVA indicated there was no effect of protocol (P = 0.43), nor was there a significant interaction with time (p = 0.68), for BPR. The results indicated that BRE increased HRR after exercise more rapidly than PASS or ACT with no influence on BPR. These findings lead to future research needed to explore different breathing protocols following exercise in at-risk populations, such as individuals with cardiovascular disease.

KEY WORDS: Active, passive, recovery techniques, acute exercise

INTRODUCTION

Endurance exercise induces several systemic challenges that require elevations in the pumping activity of the heart and arterial blood pressure (BP). The autonomic nervous system (ANS), comprised of the sympathetic nervous system (SNS) and parasympathetic nervous system (PNS) (7, 35), influences cardiovascular function by controlling the force of contraction of the heart, heart rate (HR), and blood vessel constriction (15). More precisely, the SNS stimulation increases HR, contractile force and vasoconstriction, while the PNS stimulation induces opposite effects (7) to conserve and restore energy (15). During exercise, HR initially increases by way of

a reduction in PNS stimulation (27, 34, 44) up to approximately 30 to 50 beats per minute (bpm) (15), and then increases up to maximum HR (HR_{max}) via increased SNS stimulation (42, 36, 38, 44, 45, 46). Conversely, BP increases during exercise primarily by SNS stimulation and secondarily by way of the renin-angiotensin system through its vasoconstriction properties (33).

Once HR and systolic BP have been increased during a bout of exercise, they will remain in an elevated state several minutes after a bout of exercise has concluded (16). This post-exercise elevated state is due to elevated SNS and adrenal activity (30), body temperature (34) and the need to restore both phosphocreatine (PC) and oxygen levels in muscle (9, 32). HRR from exercise has become an important biomarker of the health of the cardiovascular system and, in particular, PNS control of the heart (5) due to its easy, noninvasive approach. HRR occurs primarily by way of PNS reactivation (18, 31) through the loss of central command (29), and secondarily by reduction in SNS (28, 31) at the cessation of exercise. The rapid stimulation of PNS is thought to be caused by the removal of inhibitory motor cortex signals to the PNS (31). Akyuz, Alpsoy, Akkoyun, Degirmenci, & Guler (2014) found that a normal HRR in 1 min (HRR1) throughout passive recovery was > 21 bpm following exercise at 85% HRmax (1), whereas other investigators (10, 11, 24, 25) observed HRR1 values of > 12 bpm during active recovery on a treadmill following exercise at 80-100% HR reserve as normal. An abnormal HRR, which is defined as HRR1 < 21 bpm at passive recovery (1), is due to insufficient PNS stimulation and is strongly associated with coronary artery disease, pre-hypertension, hypertension, and all-cause mortality (1, 4, 6). Furthermore, cardiac PNS activity declines with age, resulting in a slower HRR, but there is evidence that the decline can be attenuated with regular exercise (15).

Turning to the control of BP, this cardiovascular parameter is proportional to blood flow and resistance, as is described by Poiseuille's Law (40). During exercise, systolic BP is increased, in part, by enhancing cardiac output (CO) and to meet the demands of the active muscles (37). Systolic BP typically increases by 10 ± 2 mmHg per metabolic equivalent (MET) increase while diastolic BP slightly increases or does not change at all during aerobic activity (2). Systolic BP generally returns to resting values within 5 to 6 minutes of recovery following low- to moderate-intensity exercise and declines in a stepwise fashion during passive recovery (3, 14).

BP recovery (BPR) is another important indicator of autonomic cardiovascular control with delayed BPR values linked to cardiovascular diseases and all-cause mortality (17, 21, 24, 26, 41). A normal BPR in the first minute of recovery (BPR1) has been found to be an average of ~ 36 mmHg in young men (18-35 yo) during passive recovery after a VO_{2max} cycle ergometer test with BP measured by an electronic sphygmomanometer within the first min of passive recovery and every 2 min afterwards until pre-exercise BP values were achieved (12). Subsequently, a separate study by Dimkpa & Ugwu (2010) observed mean BPR1 values of approximately 20 mmHg in males (18-66 yo) and 7 mmHg in females (18-65 yo) during passive recovery after cycling at 80% of age-predicted HR_{max} (13). Although the age range in the study above is wide, this is one of few studies that examined BPR in females. In a recent study, Soares et al. (2017) examined the effect active recovery on a cycle ergometer (30-35% VO_{2max}) had on BPR and found that there was no significant difference in BPR between active recovery and passive recovery

after 30 minutes of moderate-intensity (60-70% VO_{2max}) aerobic exercise on a cycle ergometer (42).

Both HRR and BPR may be influenced by breathing depth and rate as HR is known to increase during inspiration as vagal activity (PNS) is blocked in the brain stem while PNS stimulation is restored during expiration leading to a decrease in HR (15). Nevertheless, few studies have observed HRR and BPR using a deep breathing relaxation technique in an attempt to enhance the reactivation of cardiac PNS stimulation during recovery, thus increasing HRR and BPR (19, 43). For example, Jones, Sangthong, Pachirat, & Jones (2015) aimed to determine the effects slow breathing has on BP (19). Results indicated that a slow breathing protocol reduced resting systolic BP by 10 mmHg. The investigators also found that the strategy reduced the systolic BP response to a sustained 2-minute isometric handgrip contraction by an average of 12 mmHg. Whereas Sugimoto (2015) used a recovery protocol of 6 BrPM for 10 minutes and found evidence that the technique did accelerate the reactivation of the PNS immediately after exercise at 50% VO_{2peak}, but no differences were reported compared to spontaneous breathing during recovery (43).

In summary, there is some indication that slow breathing techniques may enhance HRR and BPR following endurance exercise, but the evidence is limited and requires further investigation (19, 43). Due to the differences in recovery values for females compared to males (13) and lack of female research within this area, there is a need to further explore the effects of recovery modes on HR and BP in females. Therefore, the purpose of this study was to determine the effectiveness of a slow-breathing technique on HRR and BPR within a healthy female population during a recovery period following aerobic exercise on a cycle ergometer compared to more commonly used active and upright passive recovery strategies. It is hypothesized that slow breathing at 6 breaths per minute will result in an increased HRR and BPR.

METHODS

Participants

Nine college-aged, moderately active females were recruited through the use of flyers and word-of-mouth for this study. Eligibility criteria included being a female between the ages of 18 and 35 years, not on any medication that would have an effect on HR or any other cardiovascular functioning (i.e. beta blockers), not a current smokers, and being moderately active. Moderately active was defined as having exercised at moderate intensity at least 150 minutes per week for the past 3 months as per American College of Sports Medicine criteria (2). Prior to participation in this study, participants signed an informed consent waiver and completed a Physical Activity Readiness Questionnaire health questionnaire (PAR-Q+). The PAR-Q+ was used to determine ineligibility if any medical issues were known to the participants. Participants were questioned on their physical activity participation to meet minimum activity requirements for this study. Mean age, height and mass were 20.2 ± 1.0 years, 167.2 ± 6.1 cm and 68.9 ± 11.9 kg, respectively.

Protocol

Participants were asked not to eat within 2 hours, nor consume caffeine within 6 hours, of each testing session, and they were instructed not to participate in vigorous physical activity 24 hours before each session. Each participant was fitted with a Polar Team 2 HR monitor (Polar Electro Oy, Professorintie 5, Kempele, Finland) and resting HR (HR_{rest}) was measured to establish a baseline and determine 70% of the individual's HR_{max} using the Karvonen formula (%HRreserve = [220 – age – HRrest] x % + HRrest) (20). Each participant then exercised on an electronically braked cycle ergometer (Monark 939 E, Monark Exercise AB, Vansbro, Dalarna) at a cadence of 70 revolutions per minute (RPM), with increasing workload every two minutes until 70% of their predicted HR_{max} was achieved.

On three subsequent occasions with a minimum of 48 hours apart, participants returned to the lab to conduct the aerobic exercise and three recovery protocols in a randomized order. During each of these testing sessions, participants were first fitted with a Finometer PRO system finger BP cuff (Finapres, Medical Systems, Amsterdam, The Netherlands) with their arm in a sling to establish a more accurate reading for BP and HR before the beginning of each exercise protocol. The Finapres system is used for continuous valvular measurements of HR and BP in clinical settings (22). HR and BP were recorded to establish a baseline for each participant 10 seconds immediately prior to exercise, every 60 seconds throughout exercise, and during the recovery periods.

Before each recovery intervention, the participants first performed a 5 min warm-up with a 25 W load and a cadence of 60 RPM, followed by cycling for 15 min at 70 RPM and maintained a load that elicited a HR within 5 bpm of 70% of their predicted HR_{max}. This protocol was chosen as to have a consistent cadence across all trials and participants at a comfortable pace for the participants while also eliciting appropriate responses for HR and BP at the desired intensity (70% HR_{max}) with differences in resistance based on individualized assessments. During each 15 minute exercise session, the Borg's modified rate of perceived exertion (RPE) scale was used to establish one's exertion level (8) at the end of each minute. The first minute of recovery had HR and BP measured every 20 seconds and then every 60 seconds each subsequent minute for a total of 5 minutes of recovery during each visit. $HR_{average}$ was established as the average of HR recorded during the last 5 minutes of the exercise protocol for each exercise session. HRR was established as the difference in HR post-exercise from $HR_{average}$ during exercise and was measured each minute during the recovery period for a total of 5 minutes (HRR1, HRR2, HRR3, HRR4, and HRR5). $BP_{average}$ was established as the average of BP recorded during the last 5 minutes of the exercise protocol for each exercise session. BPR was defined as the difference in systolic BP post-exercise from systolic $BP_{average}$ during exercise and was measured every minute during the recovery period for a total of 5 minutes (BPR1, BPR2, BPR3, HRR4, and HRR5).

All participants used the following three recovery protocols in a randomly assigned order. The PASS protocol consisted of the participant sitting on the cycle ergometer with their feet propped onto the bike comfortably and were instructed to breathe at a comfortable rate for 5 minutes. The ACT protocol consisted of the participant pedaling at a cadence of 60 RPM with a load of 25 W for 5 min. The BRE protocol required the participant to sit on the cycle ergometer with

their feet propped onto the bike comfortably, and they were instructed to breath at a rate of 6 BrPM for 5 min. A metronome was used to accurately count breaths with the participant. An investigator also watched each participant's chest rise and fall and she counted aloud the time between breaths to ensure the participants maintained the proper rhythm was maintained. HRR and BPR were measured throughout the 5 min PASS and BRE protocols, whereas values were measured during a 5 min period in a rested upright position immediately after 5 min of cycling at a reduced load for the ACT protocol.

Statistical Analysis

All data were analyzed using the Statistical Package for Social Sciences software (SPSS 23.0, Chicago, IL) and reported as mean ± standard deviation (SD). The effect of the three recovery sessions (ACT vs. PASS vs. BRE) on HRR and BPR was determined using repeated measures ANOVAs with a Greenhouse-Geisser correction due to violation of sphericity in both models. Bonferroni's post-hoc analyses were used to further distinguish significant differences. Effect sizes were determined using partial eta squared. Statistical significance was set at $p < 0.05$.

RESULTS

HRR: The HRR scores associated with each recovery protocol are presented in Figure 1. HRR values expressed over time are presented in Table 1. Results indicated significant main effect of time [$F(1.61, 12.91) = 110.63$, $p < 0.001$, $\eta_p^2 = 0.93$]. Post hoc tests revealed that HRR values significantly increased at time points 20 sec, 40 sec, 1 min, and 2 min ($p < 0.01$), with no significant differences at time points 3 min, 4 min, and 5 min ($p > 0.05$), as expressed in Table 2.

Table 1. Heart rate recovery values over time.

Time	HRR20	HRR40	HRR1	HRR2	HRR3	HRR4	HRR5
Values	20.08 ± 11.14	31.38 ± 12.49	38.41 ± 14.72	49.30 ± 14.52	55.16 ± 14.73	58.23 ± 14.37	59.86 ± 14.08

Data is expressed as mean ± SD. Values expressed as beats per minute (bpm); HRR = heart rate recovery; HRR20 = HRR at 20 seconds after exercise; HRR40 = HRR at 40 seconds after exercise; HRR1 = HRR at 1 minute after exercise; HRR2 = HRR at 2 minutes after exercise; HRR3 = HRR at 3 minutes after exercise; HRR4 = HRR at 4 minutes after exercise; HRR5 = HRR at 5 minutes after exercise.

Table 2. Effect of time on heart rate recovery.

Time	HRR20	HRR40	HRR1	HRR2	HRR3	HRR4	HRR5
HRR20		< 0.01*	< 0.01*	< 0.01*	< 0.01*	< 0.01*	< 0.01*
HRR40	< 0.01*		< 0.01*	< 0.01*	< 0.01*	< 0.01*	< 0.01*
HRR1	< 0.01*	< 0.01*		< 0.01*	< 0.01*	< 0.01*	< 0.01*
HRR2	< 0.01*	< 0.01*	< 0.01*		< 0.01*	< 0.01*	< 0.01*
HRR3	< 0.01*	< 0.01*	< 0.01*	< 0.01*		0.33	0.28
HRR4	< 0.01*	< 0.01*	< 0.01*	< 0.01*	0.33		1.00
HRR5	< 0.01*	< 0.01*	< 0.01*	< 0.01*	0.28	1.00	

* = significant interaction; HRR = heart rate recovery; HRR20 = HRR at 20 seconds after exercise; HRR40 = HRR at 40 seconds after exercise; HRR1 = HRR at 1 minute after exercise; HRR2 = HRR at 2 minutes after exercise; HRR3 = HRR at 3 minutes after exercise; HRR4 = HRR at 4 minutes after exercise; HRR5 = HRR at 5 minutes after exercise.

Results also indicated a significant main effect of protocol [$F(1.07,8.55) = 16.15$, $p = 0.003$, $\eta_p^2 = 0.67$]. BRE resulted in the highest HRR (54.91 ± 2.67 bpm) compared to PASS (46.54 ± 2.05 bpm) and ACT (32.54 ± 3.89 bpm). Post-hoc comparisons between protocols are as follows: $p = 0.01$ for BRE vs. PASS, $p = 0.01$ for BRE vs. ACT, $p = 0.02$ for PASS vs. ACT. There was no significant interaction between time and protocol for HRR values [$F(12,96) = 1.56$, $p = 0.12$].

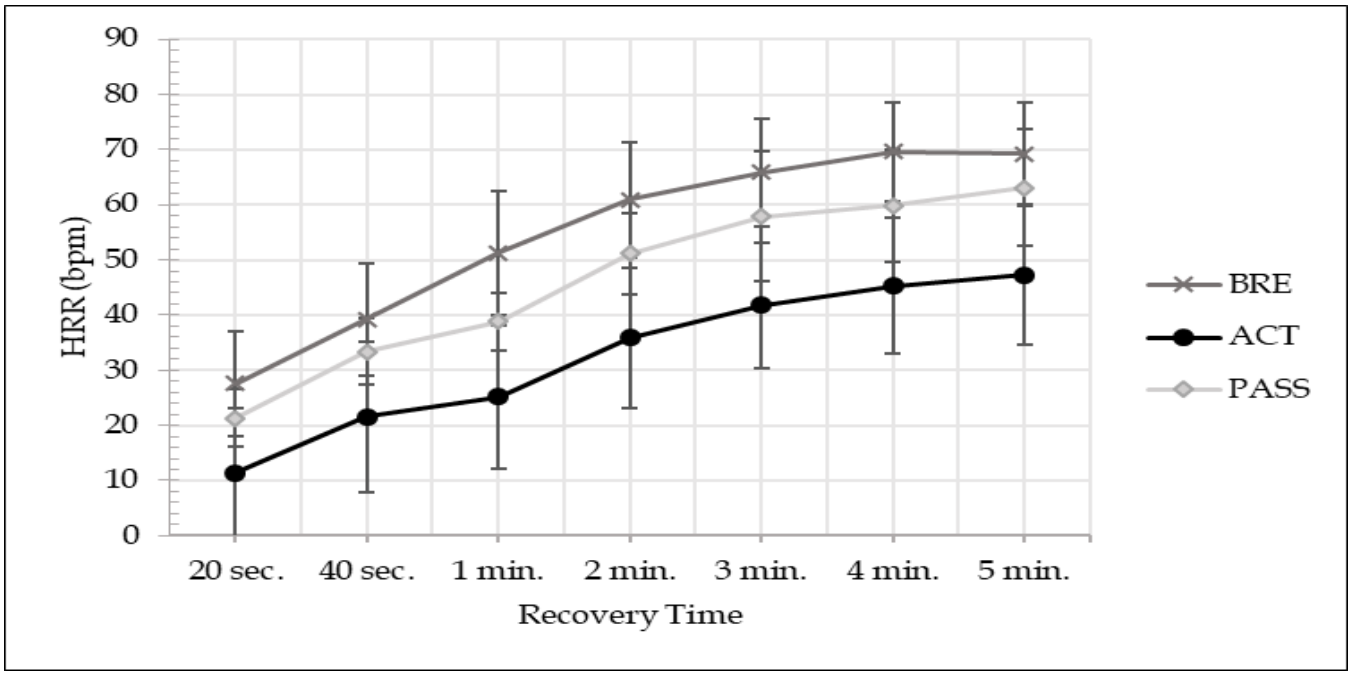

Figure 1. The effect of recovery protocol and time on heart rate recovery. Data is expressed as mean ± SD. n = 9; Values expressed as beats per minute (bpm); HRR = heart rate recovery; PASS = passive recovery; ACT = active recovery; BRE = breathing at 6 breaths per minute during recovery.

BPR: The BPR scores associated with each recovery protocol are presented in Figure 2. Results indicated a significant main effect of time [$F(2.45,19.59) = 21.95$, $p < 0.001$, $\eta_p^2 = 0.73$]. Table 3 lists significant differences by time with BPR time points 3 min, 4 min, and 5 min being significantly larger versus all other time points. No significant main effect of protocol was found [$F(2,16) = 0.90$, $p = 0.43$], nor was there an interaction between time and protocol [$F(12,96) = 0.77$, $p = 0.68$]. It is worth noting that the analyses for the main effect for protocol and the interaction effect were constrained by low power (0.18 and 0.42, respectively).

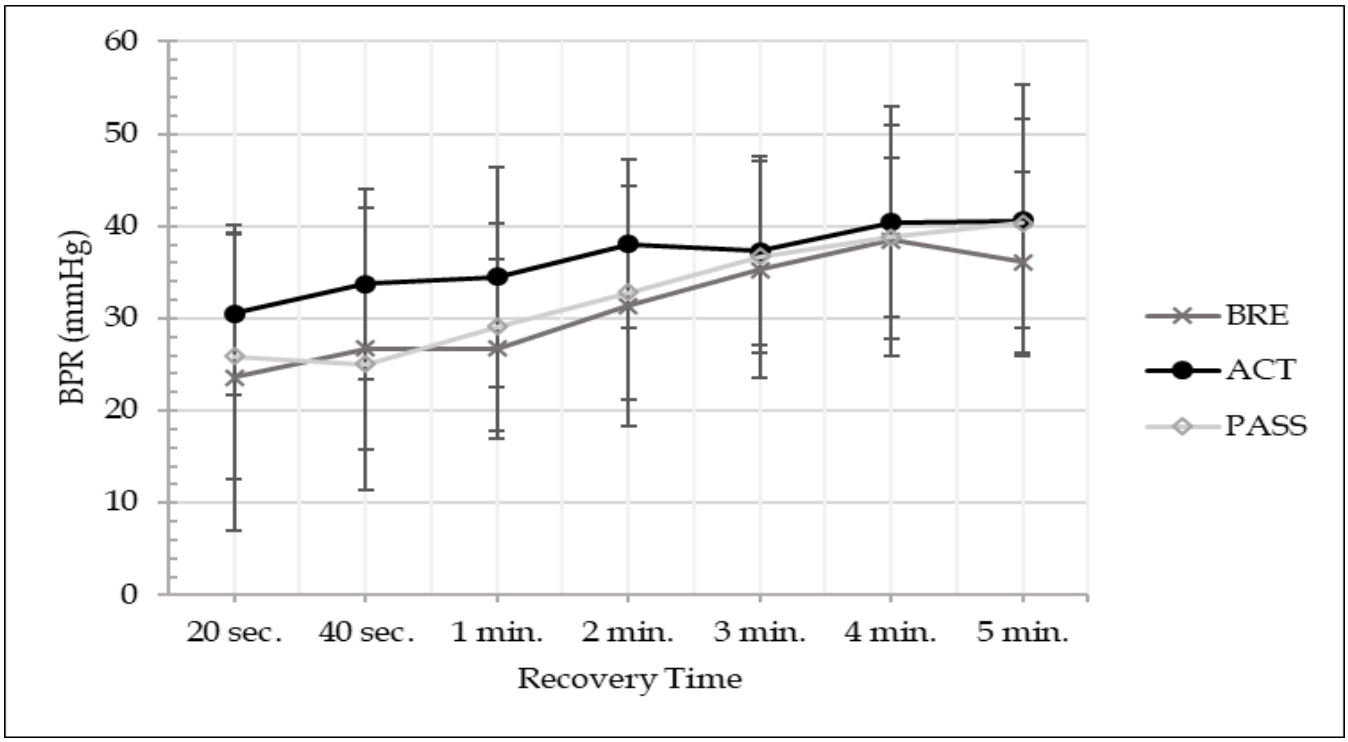

Figure 2. The effect of recovery protocol and time on systolic blood pressure recovery (BPR). Data is expressed as mean ± SD. n = 9; Values expressed as mmHg; BPR = systolic blood pressure recovery; PASS = passive recovery; ACT = active recovery; BRE = breathing at 6 breaths per minute during recovery.

Table 3. The effect of time on blood pressure recovery.

Time	BPR20	BPR40	BPR1	BPR2	BPR3	BPR4	BPR5
BPR20		1.00	0.35	0.06	0.01*	< 0.01*	0.01*
BPR40	1.00		1.00	0.11	0.01*	< 0.01*	0.04*
BPR1	0.35	1.00		0.12	0.03*	< 0.01*	0.05*
BPR2	0.06	0.11	0.12		0.02*	0.01*	1.00
BPR3	0.01*	0.01*	0.03*	0.02*		0.09	1.00
BPR4	< 0.01*	< 0.01*	< 0.01*	< 0.01*	0.09		1.00
BPR5	0.01*	0.04*	0.05*	1.00	1.00	1.00	

* = significant interaction; Values expressed as p-values; BPR = systolic blood pressure recovery; BPR20 = BPR at 20 seconds after exercise; BPR40 = BPR at 40 seconds after exercise; BPR1 = BPR at 1 minute after exercise; BPR2 = BPR at 2 minutes after exercise; BPR3 = BPR at 3 minutes after exercise; BPR4 = BPR at 4 minutes after exercise; BPR5 = BPR at 5 minutes after exercise.

DISCUSSION

The purpose of this study was to determine the effectiveness of a slow breathing recovery protocol on HRR and BPR from exercise within a healthy female population compared to ACT and PASS. It was hypothesized that using a slow breathing technique during the recovery period would result in a faster HRR and BPR compared to ACT and PASS. Based on the results in this study, a BRE recovery protocol did increase HRR, but it had no influence on BPR compared to ACT recovery and PASS recovery. This is the first investigation to have demonstrated that BRE recovery can enhance HRR compared to the more commonly used PASS and ACT recovery strategies in women.

Results in this study are consistent with previous studies in HRR response during the first minute of recovery during passive and active recovery. In the current study, the mean HRR during the first minute was 39 bpm during PASS recovery compared to > 21 bpm reported by Akyuz et al. (2014) following aerobic exercise conducted at 85% of the participant's age-predicted HR_{max} (1). Furthermore, the mean HRR of 25 bpm following one minute of ACT in the current study was comparable to the > 12 bpm values reported by previous studies following maximal intensity exercise (10, 11, 23, 25). The relatively large HRR values observed in each recovery protocol, compared to those established by previous researchers (1, 10, 11, 23, 25) were likely due to the participants used herein were more fit and therefore resulted in faster ANS recovery (48). More specifically, the faster HRR seen in this study, particularly that which was observed with the BRE protocol, was likely primarily the result of faster reactivation of the PNS and secondarily the deactivation of the SNS after exercise (39). The faster mean HRR observed with BRE protocol compared to the ACT and PASS protocols may be explained by greater enhancement of vagal inhibition of HR during the periods of prolonged expiration (15). However, this finding differs from that of Sugimoto (2015), who did not observe any differences in HRR following BRE and PASS recovery protocols (43). These inconsistencies may be due to the participants in Sugimoto's study (2015) exercised at a relatively low workrate (50% VO_{2max}). The low workrate may not have effectively facilitated a recovery of sufficient magnitude to be able to distinguish between recovery protocols.

BP results observed during aerobic exercise closely resembled those previously found in young females that were reported by both Sharman & LaGerche (2015) and Wielemborek-Musial, Szmigielska, Leszczynska, & Jegier (2016) (40, 47). While there were no significant differences in BPR between the three recovery protocols, the values associated with each recovery protocol did steadily decline back to baseline 5 min post-exercise, which is consistent with previous studies (3, 14). To the best of our knowledge, only one study has examined BPR from aerobic exercise in similar groups of females. Dimpka and Ugwu (2010) observed markedly lower BP values during the first minute of recovery (7 ± mmHg vs 29 ± 11.26 mmHg in this study), and yet there were no clear differences in the results pertaining to the latter stages of recovery (13).

The most significant limitation of this study, which may also help to explain both the lack of any differences in BPR between the three recovery protocols was the small sample size. It is evident from the very low statistical power values reported above that the small sample size severely constrained the analysis of the BPR results. Another limitation to this study could be the lack of familiarity with the breathing technique, as the participants were not accustomed to breathing at a rate 6 breaths per minute regularly. Nevertheless, the investigators did ensure all participants conformed to the breathing protocol during the BRE recovery protocol. A third limitation is the generalizability of the results because the participants were all moderately active, college-aged females. Future studies related to this topic should be performed in a broader range of groups, including individuals suffering from high blood pressure, sedentary individuals, and athletes looking to enhance their recovery for HR and BP following bouts of exercise or in between bouts. One final limitation resulted from the use of the Finapres, which, although it provides highly accurate continuous measurements of BP and HR, does require

participants to restrict the movement of their non-dominant arm (i.e., the arm in which the data are collected). To minimize measurement error, restriction was achieved in this study using an arm sling that reduced movement in the non-dominant arm. Therefore, the participants were able to use only one hand on the handle bars to maintain balance when they were riding the cycle ergometer. This caused the participants to use a more upright position and produced more swaying when they were cycling; nevertheless, all the participants had good body control throughout the study, and none had a problem maintaining the required 70% HR_{max} workrate.

Strengths associated with this study include the counterbalanced, repeated-measures design of the study and continuous measurement of the outcome variables. Each participant completed all three recovery protocols expressed in this study (PASS, ACT, and BRE). The order in which the recovery protocols were performed was randomly assigned to reduce the chance of an order effect of the protocols. The use of the same participants for all three recovery protocols also strengthens this study rather than including different participants for each recovery protocol. The continuous measurement of the outcome variables strengthened this study due to a reduced number of data points and sample size needed for analyses. Continuous measurements are also high in sensitivity to the target data. It should also be noted that the current authors did not control for the menstrual cycle as recent evidence has suggested the menstrual cycle does not influence HR responses (49) and previous studies involving women and BP responses following exercise did not control for the menstrual cycle (13).

In conclusion, this study found that a slow breathing recovery protocol (BRE) enhanced HRR when compared to ACT and PASS. These findings may benefit recreational moderately-active individuals looking to enhance recovery from exercise between bouts of moderate-to-high-intensity exercise. Individuals with cardiovascular disease could also potentially benefit from the results of this study by increasing their HRR and BPR after exercise and reduce the stress placed upon the cardiovascular system. More research is needed to understand the physiological changes associated with slow breathing recovery techniques. Future studies should focus on comparing multiple slow breathing techniques (e.g., 6 BrPM, 7 BrPM, 8 BrPM, etc.) during recovery and their effect on HRR and BPR following lower (e.g. 60% HR_{peak}) and higher (e.g. 80% HR_{peak}) exercise intensities. Such studies may help further improve our understanding of cardiovascular health and help establish better methods of recovery following exercise.

REFERENCES

1. Akyuz A, Alpsoy S, Akkoyun DC, Degirmenci H, Guler N. Heart rate recovery may predict the presence of coronary artery disease. Anatol J Cardiol 14: 351-356, 2014.

2. American College of Sports Medicine. ACSM's guidelines for exercise testing and prescription. Philadelphia: Lippincott Williams & Wilkins: 2018.

3. Amon KW, Richard KL, Crawford MH. Usefulness of the post exercise response of systolic blood pressure in the diagnosis of coronary artery disease. Circulation 70: 951-956, 1984.

4. Aneni E, Roberson L, Shaharyar S, Blaha M, Agatston A, Blumenthal R, Meneghelo R, Conceicao R, Nasir K, Santos R. Delayed heart rate recovery is associated with early and late-stage prehypertension during exercise stress testing. Am J Hypertens 27(4): 514-521, 2014.

5. Arai Y, Saul JP, Albrecht P, Hartley LH, Lilly LS, Cohen RJ, Colucci WS. Modulation of cardiac autonomic activity during and immediately after exercise. Am J Physiol Heart Circ Physiol 256: H132-H141, 1989.

6. Arbit B, Azarbal B, Hayes S, Gransar H, Germano G, Friedman J, Thomson L, Berman D. Prognostic contribution of exercise capacity, heart rate recovery, chronotropic incompetence, and myocardial perfusion single-photon emission computerized tomography in the prediction of cardiac death and all-cause mortality. Am J Cardiol 116: 1678-1684, 2015.

7. Aubert A, Seps B, Beckers F. Heart rate variability in athletes. Sports Med 33(12): 889-919, 2003.

8. Borg GAV. Psychophysical bases of perceived exertion. Med Sci Sports Exerc 14(5): 377-381, 1982.

9. Brooks GA, Hittelman KJ, Faulkner JA, Beyer RE. Temperature, skeletal muscle mitochondrial functions, and oxygen debt. Am J Physiol 220: 1053-1059, 1971.

10. Cole CR, Blackstone EH, Pashkow FJ, Snader CE, Lauer MS. Heart-rate recovery immediately after exercise as a predictor of mortality. N Engl J Med 341(1): 1351-1357, 1999.

11. Cole CR, Foody JM, Blackstone EH, Lauer MS. Heart rate recovery after submaximal exercise testing as a predictor of mortality in a cardiovascularly healthy cohort. Ann Intern Med 132(1): 552-555, 2000.

12. Dimkpa U, Ugwu AC. Age-related differences in systolic blood pressure recovery after a maximal-effort exercise test in non-athletic adults. Int J Exerc Sci 1(4): 142-152, 2008.

13. Dimkpa U, Ugwu AC. Independent multiple correlates of post-exercise systolic blood pressure recovery in healthy adults. Int J Exerc Sci 3(1): 25-35, 2010.

14. Fletcher GF, Balady GJ, Amsterdam AE, Chaitman B, Robert E, Jerome F, et al. Exercise standards for testing and training. A statement for health care professionals from the American Heart Association. Circulation 104: 1694-1740, 2001.

15. Freeman JV, Dewey FE, Hadley DM, Myers J, Froelicher VF. Autonomic nervous system interaction with the cardiovascular system during exercise. Prog Cardiovasc Dis 48(5): 342-362, 2006.

16. Gaesser GA, Brooks GA. Metabolic bases of excess post-exercise oxygen consumption: a review. Med Sci Sports Exerc 16: 29-43, 1984.

17. Hashimoto M, Okamoto M, Yamagata T, Yamane T, Watanabe M, Tsuchioka Y, et al. Abnormal systolic blood pressure during exercise recovery in patients with angina pectoris. J Am Coll Cardiol 22: 659-664, 1993.

18. Imai K, Sato H, Hori M, Kusuoka H, Ozaki H, Yokoyama H, Takeda H, Inoue M, Kamada T. Vagally mediated heart rate recovery after exercise is accelerated in athletes but blunted in patients with chronic heart failure. J Am Coll Cardiol 24(6): 1529-1535, 1994.

19. Jones CU, Sangthong B, Pachirat O, Jones DA. Slow breathing training reduces resting blood pressure and the pressure responses to exercise. Physiol Res 64: 673-682, 2015.

20. Karvonen MJ, Kentala E, Mustala O. The effects of training on heart rate; a longitudinal study. Ann Med Exp Bil Fenn 35: 307-315, 1957.

21. Kurl S, Laukkanen JA, Rauramma R, Lakka TA, Sivenius J, Salonen JJ. Systolic blood pressure response to exercise stress test and risk of stroke. Stroke 32: 2036-2041, 2001.

22. Langewouters GJ, Settels JJ, Roelandt R, Wesseling KH. Why use Finapres or Portapres rather than intraarterial or intermittent non-invasive techniques of blood pressure measurement? J Med Eng Technol 22(1): 37-43, 1998.

23. Lauer MS, Francis GS, Okin PM, Pashkow FJ, Snader CE, Marwick TH. Impaired chronotropic response to exercise stress testing as a predictor of mortality. JAMA 281: 524-529, 1999.

24. Laukkanen JA, Kurl S, Salonen R, Lakka TA, Rauramaa R, Salonen JT. Systolic blood pressure during recovery from exercise and the risk of acute myocardial infarction in middle aged men. Hypertension 44: 820-825, 2004.

25. Maddox TM, Ross C, Ho PM, Masoudi FA, Magid D, Daugherty SL, et al. The prognostic importance of abnormal heart rate recovery and chronotropic response among exercise treadmill test patients. Am Heart J 156: 736-744, 2008.

26. McHam SA, Marwick TH, Pashkow FJ, Lauer MS. Delayed systolic blood pressure recovery after graded exercise: an independent correlate of angiographic coronary disease. J Am Coll Cardiol 34: 754-759, 1999.

27. Mitchell JH, Reeves DR, Rogers HB, Secher NH, Victor RG. Autonomic blockade and cardiovascular responses to static exercise in partially curarized man. J Physiol – London 413: 433-445, 1989.

28. Niewiadomski W, Gasiorowska A, Krauss B, Mroz A, Cybulski G. Suppression of heart rate variability after supramaximal exertion. Clin Physiol Funct Imaging 27: 309-319, 2007.

29. O'Leary DS. Autonomic mechanisms of muscle metaboreflex control of heart rate. J Appl Physiol 74(4): 1748-1754, 1993.

30. Pierpont GL, Adabag S, Yannopoulos D. Pathophysiology of exercise heart rate recovery: a comprehensive analysis. Ann Noninvasive Electrocardiol 18(2): 107-117, 2013.

31. Perini R, Orizio C, Gamba A, Veicsteinas A. Kinetics of heart rate and catecholamines during exercise in humans. The effect of heart denervation. Eur J Appl Physiol 66: 500-506, 1993.

32. Powers SK, Howley ET, Cox R. Ventilatory and metabolic reactions to heat stress during prolonged exercise. J of Sport Med and Phys Fit 22: 32-36, 1982.

33. Reid IA. Interactions between ANG II, sympathetic nervous system, and baroreceptor reflexes in regulation of blood pressure. Am J Physiol 262(25): E763-E778, 1992.

34. Rowell LB, O'Leary DS. Reflex control of the circulation during exercise: chemoreflexes and mechanoreflexes. J Appl Physiol 69: 407-418, 1990.

35. Saul JP. Beat-to-beat variations of heart rate reflect modulation of cardiac autonomic outflow. Physiology 5(1): 32-37, 1990.

36. Scherrer U, Pryor SL, Bertocci LA, Victor RG. Arterial baroreflex buffering of sympathetic activation during exercise-induced elevations in arterial pressure. J Clin Invest 86: 1855-1861, 1990.

37. Schultz MG, Otahal P, Cleland VJ, Blizzard L, Marwick TH, Sharman JE. Exercise-induced hypertension, cardiovascular events, and mortality in patients undergoing exercise stress testing: a systematic review and meta-analysis. Am J Hypertens 26(3): 357-366, 2012.

38. Seals DR. Sympathetic neural discharge and vascular resistance during exercise in humans. J Appl Physiol 66: 2472-2478, 1989.

39. Sears CE, Choate JK, Paterson DJ. Inhibition of nitric oxide synthase slows heart rate recovery from cholinergic activation. J Appl Physiol 47: 1311-1314, 1998.

40. Sharman JE, LaGerche A. Exercise blood pressure: clinical relevance and correct measurement. J Hum Hypertens 29(1): 351-358, 2015.

41. Singh JP, Larson MG, Manolio TA, O'Donnell CJ, Lauer M, Evans JC., et al. Blood pressure response during treadmill testing as a risk factor for a new-onset hypertension. The Framingham heart study. Circulation 99: 1831-1836, 1999.

42. Soares AHG, Oliveira TP, Cavalcante BR, Farah BQ, Lima AHRA, Cucato GG., et al. Effects of active recovery on autonomic and haemodynamic responses after aerobic exercise. Clin Physiol Funct Imaging 37(1): 62-67, 2017.

43. Sugimoto T, Sugiyama K, Takamori K, Michiue K, Ono K, Ishikawa A. Effects of consciously controlled breathing at 6 breaths per minute on cardiac parasympathetic nervous activity after exercise in healthy adult men. Adv Exerc Sport Physiol 21(4): 85, 2015.

44. Victor RG, Pryor SL, Secher NH, Mitchell JH. Effects of partial neuromuscular blockade on sympathetic nerve responses to static exercise in humans. Circ Res 65: 468-476, 1989.

45. Victor RG, Seals DR, Mark AL. Differential control of heart rate and sympathetic nerve activity during dynamic exercise. J Clin Invest 79: 508-516, 1987.

46. Wallin BG, Victor RG, Mark AL. Sympathetic outflow to resting muscles during static handgrip and postcontraction muscle ischemia. Am J Physiol 256(25): H105-H110, 1989.

47. Wielemborek-Musial K, Szmigielska K, Leszczynska J, Jegier A. Blood pressure response to submaximal exercise test in adults. BioMed Res Int 1: 1-8, 2016.

48. Yamamoto K, Miyachi M, Saitoh T, Yoshioka A, Onodera S. Effects of endurance training on resting and post-exercise cardiac autonomic control. Med Sci Sports Exerc 33(9): 1496-1502, 2001.

49. Yazar S, Yazici M. Impact of menstrual cycle on cardiac autonomic function assessed by heart rate variability and heart rate recovery. Med Princ Pract 25(4): 374-377, 2016.