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Heart Rate Recovery and Blood Pressure Recovery Influenced by a Slow-Breathing Protocol After Exercise in Moderately Trained Females

Emily Layne Zumbro

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ABSTRACT

Heart rate (HR) and blood pressure (BP) increases in order to meet metabolic demands as a result of exercise primarily through the sympathetic nervous system (SNS) and parasympathetic nervous system (PNS). This study aimed to build on previous research in finding the best method to rapidly decrease HR and BP after exercise during the recovery period. PURPOSE: The purpose of this study was to determine the effectiveness of a breathing technique on HRR and BPR within a healthy female population during an exercise recovery period compared to active recovery and upright passive recovery. METHODS: Nine moderately trained female participants were recruited to complete this study. Participants cycled at 70% of their predicted HRmax for 15 minutes followed by one of three recovery interventions for five minutes: 6 breaths per minute (BRE), active recovery (ACT), and passive recovery (PASS). Participants completed all three recovery protocols following exercise on three separate visits. HR and BP were measured before, during, and after exercise up to 5 minutes. HR and BP after exercise were converted into HR recovery (HRR) and BP recovery (BPR) by subtracting the recovery values from peak HR and peak BP. RESULTS: Statistical analysis yielded a significant (P ≤ 0.05) effect of time for HRR up to 3 minutes and a significant (P ≤ 0.05) effect of protocol for HRR with BRE resulting in the fastest HRR. Statistical analysis also yielded a significant (P ≤ 0.05) effect of time for BPR but found no significant (P ≤ 0.05) effect of protocol nor a significant interaction.
between time and protocol for BPR. CONCLUSIONS: The results of this study suggest that BRE can increase HRR after exercise more rapidly than PASS or ACT but none of the three protocols have an influence on BPR.

INDEX WORDS: Heart rate recovery (HRR), Blood pressure recovery (BPR), Slow-breathing, 6 breaths per minute, Active recovery, Passive recovery
HEART RATE RECOVERY AND BLOOD PRESSURE RECOVERY INFLUENCED BY A SLOW-BREATHING PROTOCOL AFTER EXERCISE IN MODERATELY TRAINED FEMALES

by

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HEART RATE RECOVERY AND BLOOD PRESSURE RECOVERY INFLUENCED BY A SLOW-BREATHING PROTOCOL AFTER EXERCISE IN MODERATELY TRAINED FEMALES

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CHAPTER I

INTRODUCTION

Exercise induces stress on one’s body by increasing heart rate and blood pressure, just to name a couple of resulting factors. The autonomic nervous system (ANS) influences the cardiovascular system through the sympathetic nervous system (SNS) and parasympathetic nervous system (PNS) (Aubert, et. al., 2003; Saul, 1990) by way of controlling the force of contraction of the heart, heart rate (HR), and blood vessel constriction and dilation (Freeman, et. al., 2006). The SNS typically increases heart rate and constricts blood vessels when stimulated, while the PNS typically stimulates opposite effects on the cardiovascular system (Aubert, Seps, & Beckers, 2003) in order to conserve and restore energy (Freeman, et. al., 2006). With exercise, heart rate of an individual increases by way of a reduction in stimulation of the PNS primarily (Mitchell, et. al., 1989; Rowell & O’Leary, 1990; Victor, et. al., 1989) by approximately 30 to 50 beats per minute (bpm) (Freeman, et. al., 2006) and SNS stimulation secondarily up to maximum HR (HR\text{max}) (50 bpm – max) (Rowell & O’Leary, 1990; Scherrer, et. al., 1990; Seals, 1989; Victor, et. al., 1989; Victor, et. al., 1987; Wallin, et. al., 1989). Blood pressure increases during exercise primarily by SNS stimulation and secondarily by way of the renin-angiotensin system through its vasoconstriction properties (Reid, 1992).

HR and BP are in an elevated state after exercise along with excess post-exercise oxygen consumption, or EPOC (Gaesser, G. A. & Brooks, G. A., 1984), other than being due to SNS and PNS activity. After exercise, there is an elevated oxygen uptake when compared to resting values and this elevation in oxygen uptake contributes to restoring oxygen stores within the muscle and resynthesizing stored adenosine triphosphate (ATP) and phosphocreatine (PC) initially and can be completed within two to three minutes (Harris, R. C., Edwards, R. H., Hultman, E., Nordesjo,
L. O., Nylind, B., & Sahlin, K., 1976). This is considered to be the rapid portion due to the rapid decline in oxygen consumption within the first few minutes, leading to an initial decrease in HR and BP after exercise. Other than restoring PC and oxygen in the muscle, elevated body temperature and elevated epinephrine/norepinephrine levels also contribute to an increase in HR and BP and can last for as long as 30 minutes post-exercise (Brooks, G. A., et al., 1971; Powers, S. K., Howley, E. T., & Cox, R., 1982). Body temperature rises during exercise causing an increase in HR and stays elevated after exercise, contributing to the elevated HR levels after exercise (Rowell, L. B., 1990). Epinephrine and norepinephrine are characterized by increasing HR by stimulating the SNS and peaks within the first minute of post-exercise (Peirpont, G. L., Adabag, S., & Yannopoulos, D., 2013).

Heart rate recovery (HRR) has been used to determine the health of the cardiovascular system post-exercise and of the PNS (Arai, et. al., 1989). HRR is primarily defined as the rapid return of heart rate after the cessation of exercise during the first 60 seconds of recovery (HRR1) (Buchheit & Grindre, 2006). A second definition for HRR is defined as the time constant decay during the first 5 minutes of recovery (Buchheit & Grindre, 2006; Pierpont, et. al., 2000; Pierpont and Voth, 2004). For the sake of this study, HRR1 and subsequent minutes (HRR2, HRR3,…) up to 5 minutes will be used to define HRR. A normal HRR1 value during passive recovery has been established as > 21 bpm (Akyuz, et al., 2014), whereas > 12 bpm during active recovery on a treadmill at 1.5 mph and 2.5% grade has been established as a normal HRR1 value (Cole et al., 1999; Cole et al., 2000; Lauer et al., 1999; Maddox et al., 2008). HRR occurs primarily by way of PNS reactivation (Imai, et al., 1994; Perini, et. al., 1993) through the loss of central command (O’Leary, 1993) and secondarily by SNS weakening (Niewiadomski et. al., 2007; Perini, et. al., 1993) at the cessation of exercise. The rapid stimulation of PNS immediately
after the cessation of exercise is thought to be primarily caused by the removal of inhibitory motor cortex signals to the PNS (Perini, et. al., 1993).

An abnormal HRR has been shown to predict, or is strongly associated with, coronary artery disease, pre-hypertension, hypertension, and all-cause mortality (Akyuz, et. al., 2014; Aneni, et. al., 2014; Arbit, et. al., 2015) and could be associated with insufficient stimulation of the PNS after exercise. As one ages, PNS activity declines leading to a slower HRR but can be improved through regular exercise (Freeman et al, 2006). Poor sleep has also been shown to influence HRR negatively by reducing the rate at which HRR occurs after exercise (Yuksel et al., 2014). The intensity and duration of exercise have been shown to have an influence on how fast HR values can return to baseline with short-duration (<15 minutes), low-intensity exercise (<30% VO₂max) having the fastest response back to baseline of HR within 5 minutes (Seiler et al., 2007; Terziotti et al., 2001) and high-intensity exercise (>70% VO₂peak) resulting in a slow decline back to baseline post-exercise for HR ranging from 10 minutes (Arai et al., 1989; Pober et al., 2004; Takahashi et al., 2000) to over an hour (Furlan et al., 1993; James et al., 2002; Mourot et al., 2004; Terziotti et al., 2001) but has a longer PNS activation period.

BP is proportional to blood flow and resistance as described by Poiseuille’s Law (Sharman & LaGerche, 2015). During exercise, BP increases in a stepwise progression along with an increasing exercise intensity in order to meet oxygen demand to the active muscles through an increase in cardiac output (CO) (Schultz et. al., 2012) and typically tapers off after 2-3 minutes of exercise (O’Brien, et al., 2002; Yamaguchi, et al. 2001). Systolic BP typically increases by 10 ± 2 mmHg per metabolic equivalent (MET) increase and can reach a plateau at peak exercise while diastolic BP changes slightly or does not change at all (Sharman & LaGerche, 2015). CO increases during exercise due to an increase in HR and stroke volume (SV)
After exercise, BP levels sometimes have the tendency to be lower than BP readings prior to exercise; this phenomena is termed post-exercise hypotension (PEH) (Cardoso, et. al., 2009). The reduction in SNS activity (Halliwell, et al., 2013) leading to a reduction in CO and peripheral vascular resistance (PVR) (Brito, Queiroz, & Forjaz, 2014) has been suggested as the primary determinant of PEH.

BP recovery (BPR) has been defined as systolic BP measured as the difference in the first minute of recovery post-exercise and BP_{peak} during exercise, or BPR1 (Dimkpa & Ugwu, 2008; Dimkpa & Ugwu, 2010). A normal BPR1 has been found to be an average of ~ 36 mmHg in young men (18-35 yo) during passive recovery after maximum effort cycle ergometer exercise testing (Dimkpa & Ugwu, 2008). A normal BPR1 after cycling at 80% of age-predicted HR_{max} was found to be an average of ~ 20 mmHg in males (18-66 yo) and an average of ~ 7 mmHg in females (18-66 yo) during passive recovery (Dimkpa & Ugwu, 2010). Soares et al. (2017) set out to determine the effect of active recovery (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. Similar to HRR, abnormal BPR values have been shown to be linked to cardiovascular diseases and all-cause mortality (Hashimoto, et al., 1993; Kurl, et al., 2001; Laukkanen, et al., 2004; McHam, et al., 1999; Singh, et al, 1999).


Respiration has been shown to increase HR during inspiration by way of vagal activity (PNS) being blocked in the brain stem with PNS activity being restored during expiration causing a decrease in HR (Freeman et. al., 2006), hence leading to HR variability (HRV).
Sangthong, Pachirat, & Jones (2015) aimed to determine the effects slow breathing had on BP through way of the central nervous system and found that a slow breathing protocol reduced resting systolic BP. This study also found a reduction in pressor response to a single handgrip exercise, or a reduction in BP response to exercise (Jones et al., 2015). These findings suggest that slow breathing results in decreasing BP and can be used to alleviate hypertension. Slow breathing has also been suggested to alleviate anxiety, depression, and stress and could be due to the reduction in BP from slow breathing treatment (Jerath, Crawford, Barnes, & Harden, 2015).

Few studies have observed HRR and BPR using relaxing techniques such as participants lying in a supine position post-exercise (Barak et al., 2010; Barak et al., 2011; Buchheit et al., 2009; Larson et al., 2013; Takahashi, et al., 2000), but even fewer studies have used a deep breathing technique as a relaxing technique in order to further reactivate the PNS in normal subjects and increase HRR and BPR. Gupta (2014) used a breathing technique at 6 breaths per minute (BrPM) for 2 sets of 5 repetitions along with a progressive muscle relaxation technique in order to reduce blood pressure (BP) in hypertensive participants after low-intensity exercise (<30% VO$_{2max}$). They found that the relaxation techniques significantly reduced BP compared to a passive recovery protocol in a sitting position by 16 mmHg during the recovery phase. Sugimoto (2015) used a recovery protocol of 6 BrPM for 10 minutes and found that breathing at 6 BrPM accelerated the reactivation of the PNS after exercise at 50% VO$_{2peak}$ within the first 2 minutes of recovery, but no changes in HR were observed compared to spontaneous breathing during recovery. Jones et al. (2015) found that with breathing at 6 BrPM for 30 minutes twice a day over the course of 8 weeks, resting systolic BP levels and the BP response to a sustained 2-minute isometric handgrip contraction were both lower compared to systolic BP levels before the slow breathing training. Resting systolic BP was 10 mmHg lower while BP response to the
sustained isometric handgrip contraction was 12 mmHg lower after the slow breathing training compared to before training. Due to the lack of current literature, further investigation into HRR and BPR involving slow breathing after exercise is needed.

Purpose of the Study

The purpose of this study was to determine the effectiveness of a breathing technique on HRR and BPR within a healthy female population during an exercise recovery period compared to active recovery and upright passive recovery.

Research Questions

The research questions for this study are:

- RQ1 – Will a breathing technique during recovery facilitate a more rapid heart rate recovery within participants compared to passive and active recovery?
- RQ2 – Will a breathing technique during recovery result in a faster blood pressure recovery within participants when compared to active and passive recovery?

Hypothesis

The hypothesis for this study is:

- Slow breathing will decrease heart rate and blood pressure at a faster rate than passive and active recovery, resulting in an increase in heart rate recovery and blood pressure recovery.

Limitations

The following are limitations of the study:

- Participants are not familiar with the testing nor breathing technique.
- The sample size of participants is low (n = 9)
- The use of equipment
Delimitations

The following are delimitations of the study:

- The selection of participants is from a college campus in south Georgia.
- Participants are between the ages of 18 and 35.
- Only female participants

Assumptions

The following are assumptions of the study:

- Equipment will be functioning properly and calibrated for testing.
- Participants will give 100% effort during VO$_{2peak}$ testing.
- Participants will not perform vigorous physical activity 24 hours prior to testing.
- All participants are non-smokers.
- Participants will not ingest food 2 hours prior to testing.
- Participants will not consume caffeinated drinks 6 hours prior to testing.
- Participants will provide honest and accurate answers on surveys.

Operational Definitions

**Parasympathetic Nervous System (PNS):** reduces HR and BP while also controlling digestion and absorption of nutrients and excretion of wastes leading to the preservation and restoration of energy (Freeman et al, 2006).

**Sympathetic Nervous System (SNS):** responses that include an increase in HR, BP, and CO within the cardiovascular system along with other responses throughout the body in order to ensure survival (fight or flight) from external and internal sources of the body (Freeman et al, 2006).
Heart Rate Recovery (HRR): peak HR during exercise subtracted by the HR after the cessation of exercise, usually at the 30-second, 1 minute, 2 minute, or 4 minute mark (Aneni et al, 2013).

Post-Exercise Hypotension (PEH): BP after a single bout of exercise that are lower than resting values before exercise and is sustained for a period of time after exercise (Cardoso et al, 2009).
CHAPTER II

REVIEW OF LITERATURE

Heart rate recovery (HRR) and blood pressure recovery (BPR) are fairly popular topics among researchers looking at its effect on the specific mechanisms behind the phenomenon. Few studies, however, have been conducted in order to potentially accelerate HRR and BPR. The health of the parasympathetic nervous system and the cardiovascular system post-exercise can be determined by calculating HRR during recovery from exercise (Arai, et. al., 1989). Parasympathetic nervous system (PNS) activity declines as one ages leading to a slower HRR but can be improved through regular exercise (Freeman et al, 2006). Coronary artery disease (CAD), pre-hypertension, hypertension, and all-cause mortality is strongly associated with an abnormal HRR (Akyuz, et. al., 2014; Aneni, et. al., 2014; Arbit, et. al., 2015) and could be associated with insufficient stimulation of the PNS after exercise. Similar to HRR, abnormal BPR values have been shown to be linked to cardiovascular diseases and all-cause mortality (Hashimoto, et al., 1993; Kurl, et al., 2001; Laukkanen, et al., 2004; McHam, et al., 1999; Singh, et al, 1999).

Parasympathetic Reactivation after Exercise

A systematic review of 26 studies by Freeman et al. (2006) analyzed how the autonomic nervous system (ANS) influences the cardiovascular system during rest and exercise. The ANS system is known to control HR and heart contraction force, the constriction and dilation of blood vessels, the contraction and relaxation of smooth muscle in organs, and certain secretions by glands. The PNS is responsible for conserving and restoring energy through way of reducing HR and BP while the SNS is responsible for increasing HR, BP, and CO. This review found that
during exercise, the withdrawal of the PNS significantly increases HR by approximately 30-50 bpm while further HR increases is due to SNS stimulation.

Yamamoto et al. (2001) studied the effects of a 6 week cycling protocol and its influence on the ANS. Participants (n = 12) were assigned to either an endurance-trained group (n = 7) or a control group (n = 5), matching as closely as possible in age, height, weight, and VO\textsubscript{2peak}. The endurance-trained group participated in the cycling protocol while the control group did not participate in any type of training. Each group was tested throughout the study to note changes in HR variability and VO\textsubscript{2peak}. This study found that endurance training influenced changes in ANS modulation of the cardiovascular system. These ANS changes were observed immediately after exercise during the recovery period and resulted in a faster HRR compared to the beginning of the study for those in the endurance-trained group. A slower resting HR was observed in the endurance-trained group, further resulting from ANS remodeling.

Goldberger et al. (2005) used an electrocardiograph (ECG) to determine if parasympathetic activity influenced HRR. This study used PNS blocking drugs to compare ECG screenings to non-PNS blocking testing. The findings resulted in the confirmation that PNS plays a major role in decreasing HR immediately after exercise, thus indicating that PNS reactivation is a prime factor in HRR.

Along with SNS and PNS activity, excess post-exercise oxygen consumption, or EPOC, has been shown to be associated with HR and BP elevation after exercise compared to resting values (Gaesser, G. A. & Brooks, G. A., 1984). When compared to resting values, oxygen uptake is elevated after exercise and contributes to restoring oxygen stores within the muscle and resynthesizing stored adenosine triphosphate (ATP) and phosphocreatine (PC) initially and can be completed within two to three minutes (Harris, R. C., Edwards, R. H., Hultman, E., Nordesjo,
The first few minutes of recovery is considered to be the rapid portion of EPOC due to the rapid decline in oxygen consumption, leading to an initial decrease in HR and BP after exercise. Elevated body temperature and elevated epinephrine/norepinephrine levels also contribute to an increase in HR and BP and can last for as long as 30 minutes post-exercise (Brooks, G. A., et al., 1971; Powers, S. K., Howley, E. T., & Cox, R., 1982). Body temperature increases during exercise leading to an increase in HR and stays elevated after exercise (Rowell, L. B., 1990). Epinephrine and norepinephrine stimulates the SNS and peaks within the first minute of post-exercise contributing to an increase in HR (Peirpont, G. L., Adabag, S., & Yannopoulos, D., 2013).

Heart Rate Recovery

HRR was defined by Lipinski et al. (2004) as the HR at 2 minutes post-exercise subtracted from the peak HR during exercise, or HRR2, and is able to predict the presence of CAD (Akyuz et al., 2014). This measurement is used in many HRR studies (Akyuz et al., 2014; Aneni et al., 2013). Akyuz et al. (2014) also used the measurement HRR1, or HRR after 1 minute post-exercise. This study established that a HRR1 ≤ 21 bpm was abnormal in the seated position with no active recovery, whereas other studies established a HRR1 ≤ 12 bpm as abnormal in a sitting upright position during recovery with an active recovery on a treadmill 1.5 mph with a 2.5% grade (Cole et al., 1999; Cole et al., 2000; Lauer et al., 1999; Maddox et al., 2008). Shetler et al. (2001) established that HRR1 and HRR2 are valid testing protocols to determine cardiovascular disease presence and all-cause mortality.

Dimkpa and Ugwu (2008) studied the effects age had on HRR within males that were split into three groups: young (18-35 yo), middle-aged (36-58 yo), and elderly (60-68 yo). This study defined HRR as recovery during the first minute of recovery after exercise (HRR1) or
during the third minute of recovery after exercise (HRR3) subtracted from \( HRR_{\text{peak}} \) during exercise. 96 participants conducted exercise on a cycle ergometer to maximum effort in a stepwise fashion by having the workload increase by 20 Watts every minute until exhaustion. HR and BP were measured before, during, and after exercise. This study found that a normal HRR in young men was \(~ 64 \text{ bpm}\) during the first minute of recovery and \(~ 107 \text{ bpm}\) during the third minute of recovery. A normal HRR for middle-aged men was \(~ 39 \text{ bpm}\) during the first minute of recovery and \(~ 56 \text{ bpm}\) during the third minute of recovery. A normal HRR for elderly men was \(~ 34 \text{ bpm}\) during the first minute of recovery and \(~ 54 \text{ bpm}\) during the third minute of recovery.

Aneni et al. (2013) studied HRR tendencies with pre-hypertension (PHT). PHT is associated with overstimulation of the SNS and PNS dysfunction. This study included 683 participants who conducted a Bruce protocol having their HR and BP recorded prior to exercise, during, and after exercise. HRR was defined in the study as the peak heart rate minus the heart rate at 2 minutes post-exercise (HRR2), whereas BPR was defined as the BP decline at 2 minutes and 4 minutes after exercise from BP\(_{\text{peak}}\). This study concluded that HRR for those with hypertension was slower at the 2-minute mark post-exercise compared to those with PHT. With BPR, there was no significant difference at the 2-minute nor the 4-minute marks. This study also showed that a delay in HRR2 was present in early- (60.4 ± 13.2 bpm) and late-stage PHT (57.7 ± 20.7 bpm), leading to suggest abnormal PNS function being present in the early stages of developing hypertension. Early-stage PHT is defined as systolic BP being 120-129 mmHg and diastolic BP being 80-84 mmHg. Late-stage PHT is defined as systolic BP being 130-139 mmHg and diastolic BP being 85-89 mmHg. Hypertension has been shown to be associated with an
overstimulation of the SNS (Julius, 1991) and an understimulation of the PNS (Langewitz, Ruddel, & Schachinger, 1994).

Freeman et al. (2006) analyzed HRR through a systematic review of 26 studies and found that the reactivation of the PNS primarily influences the decline in HR immediately after exercise and the deactivation of the SNS secondarily influences the decline in HR immediately after exercise (Sears, Choate, & Paterson, 1998). Further studies within this review then established that a slow HRR (≤ 12 bpm at 1 minute immediately after exercise) was associated with a shorter life-span (Cole, et al., 1999). This review also noted that those who smoke had a slower HRR immediately after exercise leading to the conclusion of poor health for these individuals (Gordon, et al., 1987).

Systolic Blood Pressure Recovery

During exercise, systolic BP increases in a stepwise fashion by approximately 10 ± 2 mmHg per MET until peak exercise is reached while diastolic BP stays relatively the same as its resting value (Sharman & LaGerche, 2015). This review paper also noted that a normal maximal response for systolic BP in young adults is < 210 mmHg for men and <190 mmHg for women with these numbers increasing as one ages.

Wielemborek-Musial et al. (2016) conducted a study aimed to address BP response to submaximal exercise testing in healthy adults during recovery. 1015 participants partook in this study and were subjected to exercise tests on a cycle ergometer pedaling at a cadence of 60 RPM with workload increasing every 3 minutes until at least 85% of maximum age-predicted HR was reached. BP and HR were monitored at rest and during the exercise tests. The results showed that systolic BP increased significantly as workload increased, but systolic BP values were lower in females (162.4 ± 25.6 mmHg) when compared to men (173.7 ± 28.8 mmHg). This could be due
to the secretion of estrogens in women by playing a role in arterial BP regulation. There were no significant differences between men and women when comparing HR values at rest or post-exercise.

Dimkpa and Ugwu (2008) studied the effects age had on BPR within males with the participants split into three groups: young (18-35 yo), middle-aged (36-58 yo), and elderly (60-68 yo). This study established BPR as recovery during the first minute of recovery after exercise (BPR1) or during the third minute of recovery after exercise (BPR3). 96 participants conducted exercise on a cycle ergometer to maximum effort in a stepwise fashion by having the workload increase by 20 Watts every minute until exhaustion. HR and BP were measured before, during, and after exercise passively. This study found that a normal BPR for systolic BP in young men was ~ 36 mmHg during the first minute of recovery and ~ 55 mmHg during the third minute of recovery. A normal BPR for systolic BP in middle-aged men was ~ 27 mmHg during the first minute of recovery and ~ 41 mmHg during the third minute of recovery. A normal BPR for systolic BP in elderly men was ~ 23 mmHg during the first minute of recovery and ~ 34 mmHg during the third minute of recovery. This study described that an increase in systemic vascular resistance, a decrease in activity of the PNS, an elevation in activity of the SNS, and a reduction in baroreflex sensitivity are all factors associated with aging and results in slower systolic BPR.

Dimkpa and Ugwu (2010) set out to establish normal values for BPR for males and females. This study consisted of participants (n = 337) aged 18 to 66 years, with 172 of the participants being male and 165 participants being female. Participants conducted exercise at 80% of their age-predicted HR\text{max} on a cycle ergometer with BP and HR being measured at rest, during exercise, and post-exercise passively. The results of this study found that normal BPR was ~ 20 mmHg for males and ~ 7 mmHg for females calculated as the difference in the first
minute of recovery from peak systolic BP (BPR1). A normal BPR was ~ 44 mmHg for men and ~ 39 mmHg for women after three minutes of recovery compared to peak systolic BP (BPR3).

An acute bout of aerobic exercise can result in the promotion of lowering BP after exercise when compared to normal resting BP values in hypertensive (Pescatello, L. S. & Kulikowich, J. M., 2001; Pescatello, L. S., et al., 1999; Quinn, T. J., 2000; Syme, A. N., et al., 2006; Taylor-Tolbert, N. S., et al., 2000; Wallace, J. P., et al., 1997) and normotensive (Bermudes, A. M., et al., 2004; Forjaz, C. L., et al., 2000) participants and is referred to as PEH (Pescatello, et al., 2004). Chronic aerobic training has been shown to decrease BP in hypertensive participants (Pescatello, L. S., Franklin, B. A., Fagard, R., Farquhar, W. B., Kelley, G. A., & Ray, C. A., 2004) but the literature lacks in studies that have examined ambulatory BP response to chronic aerobic exercise in normotensive subjects. The reduction in BP with chronic aerobic exercise is primarily associated with a reduction in peripheral vascular resistance (Fagard, R. H., 2006). SNS activity reduction (Halliwell, et al., 2013) leading to a reduction in CO and peripheral vascular resistance (PVR) (Brito, Queiroz, & Forjaz, 2014) has been suggested as the primary determinant of PEH.

Recovery Techniques

There are multiple techniques in order to facilitate recovery after exercise, ranging from active recovery to many different postural passive recoveries.

Soares et al. (2017) conducted a study of 19 male participants cycling on an ergometer for 30 minutes at moderate-intensity (60-70% VO\textsubscript{2max}) with an active recovery or a passive recovery on two separate visits for 5 minutes comparing BP and HR responses during recovery. The active recovery was performed at 30-35% of VO\textsubscript{2max} on a cycle ergometer while passive recovery included the participants sitting on the cycle ergometer with no movement. This study
found that there were no differences in BPR and HRR between the active recovery and passive recovery after moderate-intensity aerobic exercise.

Takahashi et al. (2000) aimed to determine if lying in a supine position during recovery from submaximal exercise exhibited cardiovascular benefits through an increase in HRR. Resting stroke volume (SV), HR, BP, and CO was measured in both resting supine position and resting upright sitting position before exercise. Participants cycled at 80% VO2peak for 5 minutes at 60 RPM for each day the participants participated in the study followed by one of two recovery protocols: sitting position on the cycle ergometer or lying supine on a bed. The recovery protocols were performed for 10 minutes after exercise each on separate days. HRR1 in the supine position (47 bpm) was found to be faster than HRR1 in the upright position (33 bpm) during recovery. SV decreased gradually post-exercise in the upright sitting position below resting upright sitting resting values whereas SV increased in the supine position to similar SV resting values in the supine resting position. At the end of the 10-minute recovery period, SV and CO were found to be significantly greater in the supine recovery position compared to the upright recovery position. TPR was found to be significantly lower in the supine recovery position at the end of the 10-minute recovery period also when compared to upright sitting recovery. These findings have lead to the belief that lying in a supine position immediately after exercise will elicit a faster HRR along with other cardiovascular health benefits.

Buchheit, Al Haddad, Laursen, & Ahmaidi (2009) followed the Takahashi et al. (2000) study and aimed to distinguish the differences in HRR through way of four relaxation techniques after exercise: upright standing, sitting, lying supine with legs parallel to the floor, and lying supine with legs inclined at 70°. This study had participants perform 6 intermittent 25-yard sprints every 25 seconds in order to elevate HR and participants then proceeded to attain recovery
positions on four separate days. The results indicated that the supine only position accelerated HRR during the first 60 seconds of recovery (HRR1) by 43 bpm and parasympathetic reactivation compared with all other recovery positions, the upright position showed minimal parasympathetic reactivation and had a slow HRR1 (20 bpm), and the supine position with legs raised showed no HRR1 improvement (42 bpm) compared to the supine only position. The slow HRR1 associated with the upright standing position is believed to be associated with an increase in sympathetic activity in order to prevent syncope and preserve BP due to the effects of blood pooling in the lower extremities.

Barak et al (2011) conducted a similar study to Buchheit et al (2009) in that four recovery protocols were conducted after a submaximal exercise. This study had participants perform a 5-minute submaximal cycling exercise at 80% of the participant’s HRpeak followed by a 5-minute recovery in one of four recovery positions: passive upright seated recovery, active recovery with no workload at 20 RPM, lying supine on a bed, and lying supine with thighs in an elevated vertical position and the knees bent at a 90° angle. Barak et al (2011) found that HRR was faster within both supine recovery positions with no difference between the two (41 bpm) compared to the two seated recovery positions within the first minute of recovery (HRR1). Passive sitting recovery resulted in a HRR1 of 30 bpm. Active recovery was associated with a slower HRR1 of 26 bpm than the other three recovery conditions, though it should be noted that HRR was only measured during the active recovery and no measurements were taken after the active recovery. The slower HRR is described in this study to be associated with parasympathetic withdrawal in order to continue blood being pumped from the heart to the working muscles and back to the heart during active recovery. With an active recovery, lactate is prevented from accumulating within the muscle cells and is likely metabolized and is regarded as the best recovery method in
order to reduce fatigue and enhance the capability of the athlete to perform multiple bouts of exercise within a short period of time. Further impacts on HRR while in an upright sitting position involve blood being shifted to the lower extremities leading to decreased filling in the ventricles and decreased stroke volume and CO. To accommodate these changes, SNS activity is slightly activated in order to prevent syncope and prevent a fast HRR. These results suggest that lying in a supine position is best performed for a more rapid HRR whereas active recovery is best performed in order to increase the rate of lactate removal.

Larson et al. (2013) set out to determine the differences of HRR and power output between active recovery and recovery in a supine position. Participants (n=14) performed three Wingate sprints with 2-minute recovery intervals between each sprint. This protocol was followed for two separate days, with six Wingate sprints totaled. An active recovery was performed on one day during the 2-minute recovery periods and a supine passive recovery was performed on the second day during the 2-minute recovery periods between sprints. The active recovery consisted of walking on a treadmill at 1.5 miles per hour (mph) at a 2.5% grade during each 2-minute recovery while the supine passive recovery consisted of the participants lying supine on a table during each 2-minute recovery. The results of the study found that lying in a supine position during resting periods produced a faster average HRR back to baseline (10 minutes) while active recovery produced a slower average HRR back to baseline (30 minutes). There was no significant difference in power output between lying in a supine position during recovery or performing an active recovery. The faster HRR seen in the supine passive recovery closely relates to previous findings as discussed in this paper. The non-significant difference in power output expressed in this study between the two recovery protocols can be due to the nature of the study using anaerobic exercise. Anaerobic exercise is characterized as not needing oxygen
to replenish energy stores for the type of exercise and performance is maintained by having enough recovery time in between bouts. HR is not a factor that plays into recovery for repeated anaerobic exercise bouts.

**Breathing Protocol**

Hypertension is a worldwide issue affecting millions of people and is primarily managed through the use of anti-hypertensive medication. Gupta (2014) set out to examine if a deep breathing protocol during the recovery period from exercise along with a muscle relaxation technique would provide as an acceptable relief of acute hypertension. This study recruited 40 individuals in India aged 40-70 yo of both genders diagnosed with hypertension and were on medication for 1-2 years. Participants performed 15 minutes of low-intensity aerobic exercise (<30% VO_{2max}) at the participant’s preferred pace followed by a 5-minute cool down of slow walking. BP was then recorded immediately after the cool down multiple times over a resting 15-minute period. On the second day, participants performed the same exercise protocol with BP being recorded before exercise. During the 15 minutes of the resting period, participants were instructed to relax their muscles progressively in the supine position for each muscle group. Participants then proceeded to breath at a rate of 6 BrPM after the conclusion of the progressive muscle relaxation protocol at 2 sets of 5 repetitions. BP was measured immediately after the end of the deep breathing protocol. A significant higher reduction in systolic BP (16 mmHg) was found after the progressive muscle relaxation and deep breathing recovery protocol compared to no recovery protocol.

Jones et al. (2015) set out to determine if slow breathing training influenced BP responses to exercise. Participants (n=30) were split into a control group and a training group and participated in the study for 10 weeks. HR and HRV were recorded at rest and during the
exercise protocol before and after the exercise intervention. The training group performed a slow breathing protocol at 6 BrPM for 30 minutes twice a day over the course of 8 weeks. Before and after the 8-week intervention, the participants conducted a sustained isometric handgrip contraction on their dominant side. A significant reduction in resting systolic BP of 10 mmHg was found within the training group after the slow breathing training was completed. This study also found that slow breathing influenced BP responses to exercise by causing a lower increase in BP normally found due to exercise with a significant difference of 12 mmHg compared to pre-training values.

Sugimoto et al. (2015) is the only study found in the literature to use a slow breathing protocol during recovery in a healthy population after exercise. Participants (n=15) exercised on a cycle ergometer at 50% VO$_{2\text{peak}}$ for 10 minutes followed by a 10-minute recovery. This protocol was performed on two separate occasions with separate recovery protocols, spontaneous breathing or slow breathing at 6 BrPM. The results concluded that breathing at 6 BrPM during recovery can reactivate PNS faster post-exercise within the first two minutes of recovery compared to spontaneous breathing rates.
CHAPTER III

METHODS

Experimental Overview

This study consisted of recruiting 12 college-aged females aged 20 ± 1 years old. Participants cycled on a cycle ergometer at 70% of their measured predicted maximum heart rate (HR$_{\text{max}}$) for 15 minutes at 70 RPM on three separate visits with at least 48 hrs between each visit. Each visit contained one of three recovery protocols: breathing at 6 breaths per minute (BRE), active recovery (ACT), and passive recovery at a regular breathing rate (PASS). The recovery protocols were in a randomized order for each participant and lasted for 5 minutes after exercise. Heart rate (HR) and blood pressure (BP) were measured before and during exercise with additional measurements during the first minute of recovery every 20 seconds and then every 60 seconds each subsequent minute for a total of 5 minutes. HR recovery (HRR) was calculated as the difference between the HR measured each minute during recovery and HR$_{\text{peak}}$ as the average of HR during the last five minutes of exercise. BP recovery (BPR) was calculated as the difference between the systolic BP measured each minute during recovery and systolic BP$_{\text{peak}}$ as the average of BP during the last five minutes of exercise.

Participants

Participants of the study (n=12) were college-aged females (20 ± 1 years old). Three participants dropped out of the study, two due to injury caused by participation in activities outside of the study and one due to personal matters. Participants were recruited through word of mouth and recruitment flyers within a southern Georgia university campus. Prior to participation, participants were required to complete a written statement of informed consent and a PAR-Q+ questionnaire. It was assumed participants gave full effort during each testing period listed in this
study and all questions were answered truthfully. Participants were asked to not ingest food within the last 2 hours and no caffeine for 6 hours prior to each exercise session and not partake in vigorous physical activity within the previous 24 hours to ensure cardiovascular measurements were as close to normal as possible.

Inclusion/Exclusion Criteria

The inclusion/exclusion criteria for participation in this study are provided in Table 1.

Instrumentation

**Polar heart rate monitor.** The Polar Team 2 heart monitor system was used in this study during HR<sub>peak</sub> testing. This system included 30 Team 2 transmitters with straps and a charger (Polar Electro Oy, Professorintie 5, Kempele, Finland). HR was recorded using the Polar Team 2 heart monitors and PC software.

**Finapres.** The Finometer PRO system was used to record HR and BP before, during, and after exercise during this study. This system included a monitor and a finger BP cuff (Finapres Medical Systems, Amsterdam, The Netherlands). Each participant was fitted with a finger cuff on their middle finger on their non-dominant hand with cables attaching from the finger cuff to the Finapres. The Finapres was configured to each participant’s gender, height, and age and a height correction was performed prior to collecting data in the position the participant were stationed in during the data collection, which was in a sitting position during this study. Calibration of the Finapres system was then performed in order to ensure the equipment was functioning properly and was performed prior to each data collection. Once calibration was finished, data was able to be collected by pressing the “Start/Stop” button on the Finometer. During data collection, the participant’s arm and hand were as still as possible. This was ensured
by having the non-dominant arm of each participant in a sling that was placed around their neck to minimize movement.

*Cycle ergometer.* The Monark 939 E cycle ergometer (Monark Exercise AB, Vansbro, Dalarna) was used in this study during the exercise protocol and the active recovery protocol.

**Measurements**

- Resting, active, and recovery HR (beats per minute, bpm) and BP (mmHg)
- Height
- Weight

**Procedure**

*Screening:* Prior to participation in this study, participants signed an informed consent waiver and filled out a PAR-Q+ health questionnaire. The participants were informed about the extent of the study’s content and also signed a University research participation release form prior to the start of the study. Each participant was fitted with a Polar HR monitor and resting HR ($HR_{rest}$) was measured prior to exercise. Each participant then exercised on a Monark 939 E cycle ergometer at a cadence of 70 revolutions per minute (RPM) with increasing workload every two minutes until 70% of their predicted HR$_{max}$ was reached and maintained for at least 2 minutes. 70% of predicted HR$_{max}$ was established using the Karvonen method, which is $HR_{target} = ((HR_{max} - HR_{rest}) \times \% \text{Intensity}) + HR_{rest}$. This method was used in order to accurately measure the workload in watts (W) associated with 70% of predicted HR$_{max}$. The workload measured during this screening was used for each subsequent laboratory visit included in this study along with the same seat height used.

*Measurements of cardiovascular variables:* HR and BP were recorded to establish a baseline for each prior to exercise, during exercise, and during the recovery periods. HR and BP
were measured immediately prior to exercise and every 60 seconds during exercise. 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. The participants were fitted with a Finapres finger BP cuff with their arm in a sling to establish a more accurate reading for BP and HR before the beginning of each exercise protocol. HR\textsubscript{peak} 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\textsubscript{peak} during exercise and was measured every minute during the recovery period for a total of 5 minutes (HRR1, HRR2, HRR3, etc…). BP\textsubscript{peak} 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\textsubscript{peak} during exercise and was measured every minute during the recovery period for a total of 5 minutes (BPR1, BPR2, BPR3, etc…).

**Exercise protocol:** Before each recovery intervention, the participants performed exercise at their 70% predicted HR\textsubscript{max} for 15 minutes at a pedaling cadence of 70 RPM at a set resistance with a 5 minute warm-up with a 25 W load before the exercise protocol at a cadence of 60 RPM. The participants performed this exercise protocol on three separate occasions, one for each recovery protocol. During each exercise session, the Borg’s modified rate of perceived exertion (RPE) scale was used to establish one’s exertion level (Borg, 1982). The Borg’s RPE scale is numbered 6-20 with 20 being the highest level of exertion and 6 being the lowest level of exertion and participants were asked how they feel on a tiredness scale of 6-20 during exercise. The reliability of the Borg’s RPE scale is established as high \((r = 0.80-0.90)\) (Chen, Xitao, and Moe, 2002) and the validity is established as high also \((r = \sim 64)\) (Karavatas and Tavakol, 2005). At least 48 hours of rest were established between each test period.
Recovery protocols: The PASS protocol consisted of the participant sitting on the cycle ergometer with their feet propped onto the bike comfortably and was instructed to breathe as they normally would for 5 minutes while HR and BP was monitored. The ACT protocol consisted of the participant pedaling at a cadence of 60 RPM with a load of 25 W for 5 minutes. HR and BP were recorded during ACT for 5 minutes. The BRE protocol consisted of the participant sitting on the cycle ergometer with their feet propped onto the bike comfortably and were instructed to breath at a rate of 6 BrPM for 5 minutes. The person conducting the study and collecting data counted breaths with the participant to ensure proper rhythm was being conducted and visually watched the participant’s chest rise and fall. A metronome was also used in order to accurately count breaths with the participant. The recovery protocols were performed in a randomized order for each participant with at least 48 hours between protocols.

Data Analysis

Independent variables:

- Recovery protocol
  - PASS
  - ACT
  - BRE

Dependent variables:

- HR
- BP
Statistical Analysis

Each dependent variable (HRR and BPR) was measured and compared between recovery sessions (ACT vs. PASS vs. BRE) using a repeated measures ANOVA. The assumptions associated with this type of testing and with the statistical analysis are the following:

- All samples are drawn from normally distributed populations.
- All populations have a common variance.
- All samples are drawn independently of each other.
- Within each sample, the observations are sampled randomly and independently of each other.

A statistical significance was set at \( p < 0.05 \). Bonferroni’s post-hoc analysis was used to further distinguish differences between the recovery periods.
CHAPTER IV
RESULTS

Participants

Participants whom completed the study included 9 college-aged females recruited from a southern Georgia university. Basic demographics are shown in Table 1 displayed in Appendix C. It should be noted that heart rate (HR) and blood pressure (BP) were not significantly different during exercise when comparing each protocol. HR and BP reacted in a similar fashion for each protocol.

Heart Rate Recovery Analysis

Mean and standard deviation heart rate recovery (HRR) values over time for each protocol during recovery are listed in Table 3 and shown graphically in Figure 1. Sphericity was found to be violated after a repeated measures ANOVA was measured on the data for the effect of time and the effect of protocol resulting in the use of the Greenhouse-Geisser correction when reporting significance for time and protocol. A repeated measures ANOVA indicated there was a significant effect of time up to 3 minutes in post hoc testing during each protocol $F(1.61,12.91) = 110.63$, $p = 0.00$, $\eta_p^2 = 0.93$, which is the time difference of the averages of all protocols. HRR was significantly different from one another ($p \leq 0.00$) at time points 20 seconds, 40 seconds, 1 minute, and 2 minutes; but time points 3 minutes, 4 minutes, and 5 minutes were not significantly different from one another ($p > 0.06$). There was also a significant effect of protocol $F(1.07,8.55) = 16.15$, $p = 0.00$, $\eta_p^2 = 0.67$, which is the average HRR in total for each protocol. The slow breathing recovery protocol (BRE) resulted in the highest HRR at an average of 54.91 compared to 46.54 and 32.54 for passive recovery (PASS) and active recovery (ACT).
respectively. There was no significant interaction between time and protocol for HRR values 
\[ F(12,96) = 1.56, \ p = 0.12, \ \eta_p^2 = 0.16 \] and was associated with a moderate power of 0.79.

Systolic Blood Pressure Recovery Analysis

Mean and standard deviation systolic BP recovery (BPR) values over time for each 
protocol are displayed in Table 4 and shown graphically in Figure 2. Sphericity was found to be 
violated after a repeated measures ANOVA was measured on the data for the effect of time and 
the effect of protocol resulting in the use of the Greenhouse-Geisser correction when reporting 
significance for time and protocol. A repeated measures ANOVA indicated there was a 
significant effect of time during each protocol 
\[ F(2.45,19.59) = 21.95, \ p = 0.00, \ \eta_p^2 = 0.73 \] 
which is the time difference of all protocols. Systolic BP interactions and the significance for each 
interaction are listed in Table 5. No significant effect of protocol was found 
\[ F(2,16) = 0.90, \ p = 0.43 \] and was associated with a low power of 0.18. There was no significant interaction between 
time and protocol for BPR values 
\[ F(12,96) = 0.77, \ p = 0.68, \] and was associated with a low power of 0.42.
The purpose of this study was to determine the effectiveness of a slow breathing recovery protocol (BRE) on heart rate recovery (HRR) and blood pressure recovery (BPR) within a healthy female population compared to active recovery (ACT) and passive recovery (PASS) after exercise. 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 can increase HRR but had no influence on BPR compared to ACT recovery and PASS recovery.

Heart Rate Recovery

HRR measurements can indicate cardiovascular health through non-invasive techniques (Arai et. al., 1989; Shetler et al., 2001). Results in this study were consistent to previous studies in how fast HRR occurred during the first minute of recovery during passive recovery and active recovery. HRR in this study during the first minute resulted in ~39 beats per min (bpm) during PASS recovery compared to > 21 bpm reported by Akyuz et al. (2014) during recovery after exercise. During ACT in this study, a HRR of ~25 bpm occurred compared to > 12 bpm established during the first minute of active recovery after exercise by similar studies (Cole et al., 1999; Cole et al., 2000; Lauer et al., 1999; Maddox et al., 2008). HRR as seen in this study declined in a smooth, consistent fashion but cannot be compared to any other study. The differences in HRR values during ACT and PASS seen in this study could be due to gender, exercise intensity, and recovery process differences. The values of HRR seen in this study for each recovery protocol can be concluded to be a result of the reactivation of the parasympathetic nervous system (PNS) primarily and the deactivation of the sympathetic nervous system (SNS)
secondarily to decrease HR immediately after exercise (Sears, Choate, & Paterson, 1998). Results indicated there were significant differences in mean HRR over time up to 3 minutes, as seen in Table 3. Along with PNS and SNS activity, the rapid HRR within the first few minutes of recovery followed by the plateau of further HRR after 3 minutes as observed within this study could be associated with the rapid restoration of oxygen stores within the muscle, resynthesis of stored adenosine triphosphate (ATP) and phophocreatine (PC), and the decline of epinephrine and norepinephrine (Harris et al., 1976; Peirpont, G. L., Adabag, S., & Yannopoulos, D., 2013). The high values of HRR during each recovery protocol compared to normal values established by previous researchers (Akyuz et al., 2014; Cole et al., 1999; Cole et al., 2000; Lauer et al., 1999; Maddox et al., 2008) can be associated with the participants in the current study being more fit resulting in a faster ANS reactivation (Yamamoto et al., 2001).

The BRE recovery protocol resulted in the highest mean HRR compared to ACT and PASS recovery. This could be associated with the breathing slower during recovery therefore lengthening expiration and leading to a further decrease in HR due to the removal of vagal inhibition during expiration (Freeman et al., 2006). This is inconsistent with previous findings by Sugimoto (2015) where no changes in HR were found between a BRE recovery protocol and PASS recovery protocol. The differences between the two studies could be associated with the difference in exercise protocol, with Sugimoto (2015) having participants exercise at 50% \( \text{VO}_2\text{max} \) compared to 70% \( \text{HR}_{\text{peak}} \) in the current study. The difference in intensity could have resulted in significantly different peak HR responses therefore resulting in significantly different HRR.
Systolic Blood Pressure Recovery

Results indicated there were significant differences in mean BPR after 2 minutes of recovery over time, as seen in Table 4. The comparison between BPR20 and BPR4 resulted in the largest difference of 12.56 mmHg and the comparison between BPR20 and BPR5 resulted in the second highest difference of 12.37 mmHg. These results are associated with a reduction in SNS activity and increased PNS activity (Halliwell et al., 2013) with values returning back to baseline after 5 minutes of recovery compared to resting values before exercise in each recovery protocol, which is consistent with previous studies (Amon, K. W., Richard, K. L., & Crawford, M. H., 1984; Fletcher, G. F. et al., 2001).

BP values closely resembled results from Sharman & LaGerche (2015) with the maximal response for systolic BP in young females being < 190 mmHg. Systolic BP levels were also consistent with Wielemborek-Musial et al. (2016) with submaximal values not exceeding 162.4 ± 25.6 mmHg. In the current study, BPR was markedly higher (29.11 mmHg) than previous findings (~7 mmHg) during the first minute of passive recovery for females (Dimkpa and Ugwu, 2010). This could be a result of post exercise data collection procedures resulting in extra movement, differences in fitness levels of participants, and higher workloads compared to the current study. During the third minute of PASS recovery, BPR was found to be similar to previous findings (Dimkpa and Ugwu, 2010).

The BPR results from the current study do not match the previous results reported by Gupta (2014) which could be due to the participants in the current study being moderately active healthy females compared to hypertensive participants. Gupta (2014) used a progressive muscle relaxation technique with participants lying on their backs immediately after exercise and then tensing each muscle group one at a time and releasing the tension after a few seconds along with
a slow breathing recovery protocol at 6 BrPM. The difference in relaxation protocols could have resulted in the differences in results. Gupta (2014) reported a significant difference in BPR between passive recovery and slow breathing recovery after exercise, whereas the current study found no significant differences in BPR between recovery protocols after exercise.

Limitations of the Study

The main limitation of this study was the sample size. The study began with 12 participants total recruited. As testing continued, 3 participants dropped from the study before the conclusion of data collection. Two of these participants dropped out of the study due to injury caused by sources outside of the study and 1 other participant dropped from the study due to personal reasons. Due to the small sample size, power within the statistical analysis was greatly affected and also resulted in high variance within measures. Analysis was constrained as the result of low power and high variance within this study. Another limitation to this study could be the lack of familiarity with the breathing technique used during recovery and the lack of familiarity with equipment. Most, if not all, participants within this study were not familiar with slow breathing at 6 breaths per minute (BrPM) and complained of the breathing technique being harder than expected to conform to immediately after exercise, though it should be noted that all participants did conform to the breathing protocol during the BRE recovery protocol. The unfamiliarity with this type of slow breathing could have resulted in abnormal PNS and SNS responses involving HRR and BPR. The unfamiliarity of equipment could have also played a role in the findings due to the participants being unfamiliar with maintaining a set revolutions per minute (RPM) with as little fluctuation in RPM as possible on the cycle ergometer. The use of equipment could have also played a role in the findings within this study due to the nature of the Finapres. The Finapres was used to measure HR and BP before exercise, during exercise, and
during recovery through way of a finger cuff. While using this equipment, the arm being used to measure HR and BP had to have movement restricted as much as possible in order to measure accurate readings. This was achieved by using an arm sling in order to reduce the movement of the arm, but not all movement was able to be eliminated from the arm. Participants were able to cycle on the ergometer using only one hand on the handle bars to maintain balance. This change in posture during cycling resulted in the participants cycling in a more upright position and produced more swaying of the participants in order for the participants to sustain the work rate established to achieve their 70% $\text{HR}_{\text{max}}$. This increase in movement could have influenced the data collected during exercise and during ACT, but it should be noted that the participants were able to maintain their work rate and had good body control during the study.

Conclusions

This study investigated the effects of a slow breathing recovery protocol (BRE) compared to ACT and PASS on HRR and BPR during 5 minutes of recovery. HR, according to this study, is greatly influenced by the type of recovery protocol used after exercise with BRE resulting in the fastest HRR when compared to PASS and ACT. It should be noted that the type of recovery one wants to use after exercise should be based on one’s goals for recovery after exercise. BPR was not altered by any of the three recovery protocols. These findings suggest that a slow breathing recovery protocol can greatly increase HRR and should be used if the goal of the recovery is to rapidly decrease HR after exercise closer to resting values, but they also suggest that any of the three recovery protocols can result in normal BPR responses to return BP back to near baseline within the first 5 minutes of recovery. The results of this study could be beneficial to cardiovascular patients participating in a cardiovascular rehabilitation program after exercise in order to learn how to control their heart rate better, for chronic obstructive pulmonary disorder.
(COPD) patients having trouble breathing after exercise, the normal population learning how to exercise and control their breathing during exercise, and for athletes needing to lower their HR as fast as possible. More research is needed to understand the changes in cardiovascular variables during recovery involving a slow breathing technique after exercise. Future studies could also focus on comparing multiple slow breathing techniques (6 BrPM, 7 BrPM, 8 BrPM, etc…) during recovery and the influence of HRR and BPR each technique has after different exercise intensities (50% HR\textsubscript{peak}, 60% HR\textsubscript{peak}, etc…). Future studies should also investigate the mechanisms of recovery including PNS and SNS innervations and how each type of recovery influences these mechanisms. This could result in improved cardiovascular health and further establish the best method for recovery after exercise.
REFERENCES


APPENDIX A

INFORMED CONSENT

COLLEGE OF HEALTH AND HUMAN SCIENCES

DEPARTMENT OF HEALTH AND KINESIOLOGY

1. The primary investigator for this study is Emily Zumbro. She is a Master’s Exercise Science student within the Department of Health and Kinesiology at Georgia Southern University and is conducting this research in completion of her Master’s degree.

2. Purpose of the Study: The purpose of this research is to determine what type of recovery is best in order to accelerate heart rate recovery and blood pressure recovery.

3. Procedures to be followed: You will be asked not to ingest food two hours prior to each visit nor caffeine 6 hours prior to each visit in this study. You will also be asked not to partake in vigorous exercise within 24 prior to each visit in this study. Participation in this research will include completion of an initial screening to fill out a health questionnaire (PAR-Q+) and a cycling exercise protocol in order to establish your 70% age predicted heart rate workload that will last no longer than 15 minutes with increasing workload every 2 minutes to be used for each subsequent visit. Three separate cycling visits will follow after this initial screening with at least 48 hours in between visits. The cycling exercise visits will consist of 15 minutes of exercise cycling at 70 RPM at 70% of HRmax. Each visit will have a randomized recovery protocol: an active recovery, passive recovery sitting, and a slow breathing recovery protocol. Heart rate and blood pressure will be measured during each visit.

4. Discomforts and Risks: Risks associated with this study include discomforts due to moderate-intensity exercise associated with an increase in heart rate. I understand that medical care is available in the event of injury resulting from research but that neither financial compensation nor free medical treatment is provided. In the case of injury, you will be sent to East Georgia Regional Medical Center for assessment.

5. Benefits: The benefits to participants include an assessment of cardiovascular fitness.

6. Duration/Time required from the participant: You will be asked to participate in this study on four separate occasions each lasting approximately 30 minutes with at least 48 hours between each visit participating in an initial cycling exercise screening for the first
visit and three subsequent cycling exercise sessions at 70% of your age-predicted heart rate max.

7. Statement of Confidentiality: Emily Zumbro and Dr. John Dobson will have access to the data collected during this study. The data will be recorded on paper using assigned numbers for each participant and converted onto an encrypted hard drive on a computer behind closed doors that only the investigators will have access to. Data will be discarded 3 years following this study along with the PAR-Q+. Deidentified or coded data from this study may be placed in a publically available repository for study validation and further research. You will not be identified by name in the data set or any reports using information obtained from this study, and your confidentiality as a participant in this study will remain secure. Subsequent uses of records and data will be subject to standard data use policies which protect the anonymity of individuals and institutions. The PAR-Q+ health questionnaire is the only document which requires your signature related to your health and will be kept in a filing cabinet behind locked doors along with the informed consent forms. Name and signature is required for the PAR-Q+ in order to ensure that you understand any risk associated with moderate-intensity exercise and to ensure the investigator that you are not contraindicated to exercise. This form also requires your name and signature in case of emergency to ensure emergency personnel that you and the investigator were not aware of any pre-existing conditions that would be contraindicated to exercise.

8. Right to Ask Questions: You have the right to ask questions and have those questions answered. If you have questions about this study, please contact the researcher named above or the researcher’s faculty advisor, whose contact information is located at the end of the informed consent. For questions concerning your rights as a research participant, contact Georgia Southern University Office of Research Services and Sponsored Programs at 912-478-5465.

9. Compensation: Wal-Mart gift cards (one for $20 and 4 for $5) will be available through a drawing to those whom have completed all steps included in this study. You must have completed all steps in order to be considered for a Wal-Mart gift card.

10. Voluntary Participation: This study is completely voluntary. If, for any reason, you do not want to complete the entirety of this study, you have the right to do so at any time and all of the data collected from you will be terminated. You also have the right to not answer any questions you do not feel comfortable answering.

11. Penalty: There will be no penalty if you choose not to participate in this study but you will not be considered for a Wal-Mart gift card in the drawing.

12. “All information will be treated confidentially. There is one exception to confidentiality that we need to make you aware of. In certain research studies, it is our ethical responsibility to report situations of child or elder abuse, child or elder neglect, or any life-threatening situation to appropriate authorities. However, we
are not seeking this type of information in our study nor will you be asked questions about these issues.”

13. You must be 18 years of age or older to consent to participate in this research study. If you consent to participate in this research study and to the terms above, please sign your name and indicate the date below.

You will be given a copy of this consent form to keep for your records. This project has been reviewed and approved by the GSU Institutional Review Board under tracking number H17330.

Title of Project: “Heart rate recovery and blood pressure recovery influenced by a slow-breathing protocol after exercise in moderately trained females”

Principal Investigator: Emily Zumbro  
(601) 384-0479  
ez00252@georgiasouthern.edu

Faculty Advisor: Dr. John Dobson  
(912) 478-8541  
jdobson@georgiasouthern.edu

____________________________________  ____________________  
Participant Signature  Date

I, the undersigned, verify that the above informed consent procedure has been followed.

____________________________________  ____________________  
Investigator Signature  Date
APPENDIX B

PAR-Q+

The Physical Activity Readiness Questionnaire for Everyone

Regular physical activity is fun and healthy, and more people should become more physically active every day of the week. Being more physically active is very safe for MOST people. This questionnaire will tell you whether it is necessary for you to seek further advice from your doctor OR a qualified exercise professional before becoming more physically active.

SECTION 1 - GENERAL HEALTH

<table>
<thead>
<tr>
<th>Question</th>
<th>YES</th>
<th>NO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Has your doctor ever said that you have a heart condition OR high blood pressure?</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>Do you feel pain in your chest at rest, during your daily activities of living, OR when you do physical activity?</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>Do you lose balance because of dizziness OR have you lost consciousness in the last 12 months? Please answer NO if your dizziness was associated with over-breathing (including during vigorous exercise).</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>Have you ever been diagnosed with another chronic medical condition (other than heart disease or high blood pressure)?</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>Are you currently taking prescribed medications for a chronic medical condition?</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>Do you have a bone or joint problem that could be made worse by becoming more physically active?</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>Please answer NO if you had a joint problem in the past, but it does not limit your current ability to be physically active. For example, knee, ankle, shoulder or other.</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>Has your doctor ever said that you should only do medically supervised physical activity?</td>
<td>☐</td>
<td>☐</td>
</tr>
</tbody>
</table>

If you answered NO to all of the questions above, you are cleared for physical activity.

- Go to Section 3 to sign the form. You do not need to complete Section 2.

  - Start becoming much more physically active – start slowly and build up gradually.
  - Follow the Canadian Physical Activity Guidelines for your age (www.csep.ca/guidelines).
  - You may take part in a health and fitness appraisal.
  - If you have any further questions, contact a qualified exercise professional such as a CSEP Certified Exercise Physiologist® (CSEP-CEP) or CSEP Certified Personal Trainer® (CSEP-CPT).
  - If you are over the age of 45 yrs. and NOT accustomed to regular vigorous physical activity, please consult a qualified exercise professional (CSEP-CEP) before engaging in maximal effort exercise.

If you answered YES to one or more of the questions above, please GO TO SECTION 2.

- Delay becoming more active if:
  - You are not feeling well because of a temporary illness such as a cold or fever – wait until you feel better
  - You are pregnant – talk to your health care practitioner, your physician, a qualified exercise professional, and/or complete the PARmed-X for Pregnancy before becoming more physically active OR
  - Your health changes – please answer the questions on Section 2 of this document and/or talk to your doctor or qualified exercise professional (CSEP-CEP or CSEP-CPT) before continuing with any physical activity programme.

CSEP|SCPE

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## SECTION 2 - CHRONIC MEDICAL CONDITIONS

Please read the questions below carefully and answer each one honestly: check YES or NO.

<table>
<thead>
<tr>
<th>Question</th>
<th>YES</th>
<th>NO</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Do you have Arthritis, Osteoporosis, or Back Problems?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1a. Do you have difficulty controlling your condition with medications or other physician-prescribed therapies? [Answer NO if you are not currently taking medications or other treatments]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1b. Do you have joint problems causing pain, a recent fracture or fracture caused by osteoporosis or cancer, displaced vertebra (e.g., spondylolisthesis), and/or spondylolysis/par defect (a crack in the bony ring on the back of the spinal column)?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1c. Have you had steroid injections or taken steroid tablets regularly for more than 3 months?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Do you have Cancer of any kind?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2a. Does your cancer diagnosis include any of the following types: lung/bronchogenic, multiple myeloma (cancer of plasma cells), head, and neck?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2b. Are you currently receiving cancer therapy (such as chemotherapy or radiotherapy)?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Do you have Heart Disease or Cardiovascular Disease?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3a. Do you have difficulty controlling your condition with medications or other physician-prescribed therapies? [Answer NO if you are not currently taking medications or other treatments]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3b. Do you have an irregular heart beat that requires medical management? [e.g. atrial fibrillation, premature ventricular contraction]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3c. Do you have chronic heart failure?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3d. Do you have a resting blood pressure equal to or greater than 160/90 mmHg with or without medication? [Answer YES if you do not know your resting blood pressure]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3e. Do you have diagnosed coronary artery (cardiovascular) disease and have not participated in regular physical activity in the last 2 months?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Do you have any Metabolic Conditions?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4a. Is your blood sugar often above 13.0 mmol/L? [Answer YES if you are not sure]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4b. Do you have any signs or symptoms of diabetes complications such as heart or vascular disease and/or complications affecting your eyes, kidneys, and the sensation in your toes and feet?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4c. Do you have other metabolic conditions (such as thyroid disorder, pregnancy-related diabetes, chronic kidney disease, liver problems)?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Do you have any Mental Health Problems or Learning Difficulties?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5a. Do you have difficulty controlling your condition with medications or other physician-prescribed therapies? [Answer NO if you are not currently taking medications or other treatments]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5b. Do you also have back problems affecting nerves or muscles?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Question</td>
<td>YES</td>
<td>NO</td>
</tr>
<tr>
<td>-------------------------------------------------------------------------</td>
<td>-----</td>
<td>----</td>
</tr>
<tr>
<td>6. Do you have a Respiratory Disease? This includes Chronic Obstructive</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pulmonary Disease, Asthma, Pulmonary High Blood Pressure</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6a. Do you have difficulty controlling your condition with medications</td>
<td></td>
<td></td>
</tr>
<tr>
<td>or other physician-prescribed therapies? (Answer NO if you are not</td>
<td></td>
<td></td>
</tr>
<tr>
<td>currently taking medications or other treatments)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6b. Has your doctor ever said your blood oxygen level is low at rest</td>
<td></td>
<td></td>
</tr>
<tr>
<td>or during exercise and/or that you require supplemental oxygen</td>
<td></td>
<td></td>
</tr>
<tr>
<td>therapy?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6c. If asthmatic, do you currently have symptoms of chest tightness,</td>
<td></td>
<td></td>
</tr>
<tr>
<td>wheezing, laboured breathing, consistent cough (more than 2 days/week),</td>
<td></td>
<td></td>
</tr>
<tr>
<td>or have you used your rescue medication more than twice in the last</td>
<td></td>
<td></td>
</tr>
<tr>
<td>week?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6d. Has your doctor ever said you have high blood pressure in the</td>
<td></td>
<td></td>
</tr>
<tr>
<td>blood vessels of your lungs?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Do you have a Spinal Cord Injury? This includes Tetraplegia and</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Paraplegia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7a. Do you have difficulty controlling your condition with medications</td>
<td></td>
<td></td>
</tr>
<tr>
<td>or other physician-prescribed therapies? (Answer NO if you are not</td>
<td></td>
<td></td>
</tr>
<tr>
<td>currently taking medications or other treatments)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7b. Do you commonly exhibit low resting blood pressure significant</td>
<td></td>
<td></td>
</tr>
<tr>
<td>enough to cause dizziness, light-headedness, and/or fainting?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7c. Has your physician indicated that you exhibit sudden bouts of high</td>
<td></td>
<td></td>
</tr>
<tr>
<td>blood pressure (known as Autonomic Dysreflexia)?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. Have you had a Stroke? This includes Transient Ischemic Attack (TIA)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>or Cerebrovascular Event</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8a. Do you have difficulty controlling your condition with medications</td>
<td></td>
<td></td>
</tr>
<tr>
<td>or other physician-prescribed therapies? (Answer NO if you are not</td>
<td></td>
<td></td>
</tr>
<tr>
<td>currently taking medications or other treatments)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8b. Do you have any impairment in walking or mobility?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8c. Have you experienced a stroke or impairment in nerves or muscles in</td>
<td></td>
<td></td>
</tr>
<tr>
<td>the past 6 months?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. Do you have any other medical condition not listed above or do you</td>
<td></td>
<td></td>
</tr>
<tr>
<td>live with two chronic conditions?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9a. Have you experienced a blackout, fainted, or lost consciousness as</td>
<td></td>
<td></td>
</tr>
<tr>
<td>a result of a head injury within the last 12 months OR have you had a</td>
<td></td>
<td></td>
</tr>
<tr>
<td>diagnosed concussion within the last 12 months?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9b. Do you have a medical condition that is not listed (such as</td>
<td></td>
<td></td>
</tr>
<tr>
<td>epilepsy, neurological conditions, kidney problems)?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9c. Do you currently live with two chronic conditions?</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Please proceed to Page 4 for recommendations for your current medical condition and sign this document.
PAR-Q+

If you answered NO to all of the follow-up questions about your medical condition, you are ready to become more physically active:

- It is advised that you consult a qualified exercise professional (e.g., a CSEP-CEP or CSEP-CPT) to help you develop a safe and effective physical activity plan to meet your health needs.
- You are encouraged to start slowly and build up gradually – 20-60 min. of low- to moderate-intensity exercise, 3-5 days per week including aerobic and muscle strengthening exercises.
- As you progress, you should aim to accumulate 150 minutes or more of moderate-intensity physical activity per week.
- If you are over the age of 45 yrs. and NOT accustomed to regular vigorous physical activity, please consult a qualified exercise professional (CSEP-CEP) before engaging in maximal effort exercise.

If you answered YES to one or more of the follow-up questions about your medical condition:

- You should seek further information from a licensed healthcare professional before becoming more physically active or engaging in a fitness appraisal and/or visit a or qualified exercise professional (CSEP-CEP) for further information.

Delay becoming more active if:

- You are not feeling well because of a temporary illness such as a cold or fever – wait until you feel better.
- You are pregnant - talk to your health care practitioner, your physician, a qualified exercise professional, and/or complete the PARmed-X for Pregnancy before becoming more physically active OR
- Your health changes - please talk to your doctor or qualified exercise professional (CSEP-CEP) before continuing with any physical activity programme.

SECTION 3 - DECLARATION

- You are encouraged to photocopy the PAR-Q+. You must use the entire questionnaire and NO changes are permitted.
- The Canadian Society for Exercise Physiology, the PAR-Q+ Collaboration, and their agents assume no liability for persons who undertake physical activity. If in doubt after completing the questionnaire, consult your doctor prior to physical activity.
- If you are less than the legal age required for consent or require the assent of a care provider, your parent, guardian or care provider must also sign this form.
- Please read and sign the declaration below:

I, the undersigned, have read, understood to my full satisfaction and completed this questionnaire. I acknowledge that this physical activity clearance is valid for a maximum of 12 months from the date it is completed and becomes invalid if my condition changes. I also acknowledge that a Trustee (such as my employer, community/fitness centre, health care provider, or other designee) may retain a copy of this form for their records. In these instances, the Trustee will be required to adhere to local, national, and international guidelines regarding the storage of personal health information ensuring that they maintain the privacy of the information and do not misuse or wrongfully disclose such information.

NAME ___________________________ DATE ___________________________

SIGNATURE ___________________________ WITNESS ___________________________

SIGNATURE OF PARENT/GUARDIAN/CARE PROVIDER ___________________________

For more information, please contact:
Canadian Society for Exercise Physiology
www.csep.ca

KEY REFERENCES

The PAR-Q+ was created using the evidence-based AGREE process (1) by the PAR-Q+Collaboration chaired by Dr. Damien E. R. Warburton with Dr. Norman Gledhill, Dr. Veronica Jamnik, and Dr. Donald C. McKenize (2). Production of this document has been made possible through financial contributions from the Public Health Agency of Canada and the BC Ministry of Health Services. The views expressed herein do not necessarily represent the views of the Public Health Agency of Canada or BC Ministry of Health Services.
## APPENDIX C

### TABLES AND FIGURES

**Table 1: Inclusion/Exclusion Criteria**

<table>
<thead>
<tr>
<th>Inclusion Criteria</th>
<th>Exclusion Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Females age 18-35 years old</td>
<td>Outside 18-35 years old</td>
</tr>
<tr>
<td>Participant is willing and able to complete the protocol</td>
<td>Males</td>
</tr>
<tr>
<td></td>
<td>Aware of pre-existing conditions (diabetes, hypertension, etc…)</td>
</tr>
<tr>
<td></td>
<td>Contraindicated to exercise (Appendix D)</td>
</tr>
<tr>
<td></td>
<td>Smokes cigarettes or uses any other drugs</td>
</tr>
</tbody>
</table>

**Table 2: Demographic Characteristics of Participants**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean</th>
<th>Standard Deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>20.22</td>
<td>0.97</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>167.22</td>
<td>6.14</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>68.93</td>
<td>11.87</td>
</tr>
</tbody>
</table>

n = 9
Figure 1: Heart rate recovery (HRR) over time per recovery

n = 9; Values expressed as beats per minute (bpm); PASS = passive recovery; ACT = active recovery; BRE = breathing at 6 breaths per minute during 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 3: Effect of recovery protocol on heart rate recovery (HRR)

<table>
<thead>
<tr>
<th></th>
<th>20 sec.</th>
<th>40 sec.</th>
<th>1 min.</th>
<th>2 min.</th>
<th>3 min.</th>
<th>4 min.</th>
<th>5 min.</th>
<th>Protocol mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>PASS</td>
<td>21.33 ± 5.15</td>
<td>33.44 ± 5.98</td>
<td>38.89 ± 5.33</td>
<td>51.22 ± 7.53</td>
<td>57.89 ± 11.86</td>
<td>59.89 ± 10.23</td>
<td>63.11 ± 10.79</td>
<td>46.54</td>
</tr>
<tr>
<td>ACT</td>
<td>11.33 ± 11.81</td>
<td>21.44 ± 13.69</td>
<td>25.11 ± 12.99</td>
<td>35.78 ± 12.72</td>
<td>41.67 ± 11.40</td>
<td>45.22 ± 12.31</td>
<td>47.22 ± 12.47</td>
<td>32.54</td>
</tr>
<tr>
<td>BRE</td>
<td>27.67 ± 9.54</td>
<td>39.33 ± 10.14</td>
<td>51.33 ± 11.27</td>
<td>61.00 ± 10.51</td>
<td>66.00 ± 9.79</td>
<td>69.67 ± 8.92</td>
<td>69.33 ± 9.31</td>
<td>54.91</td>
</tr>
<tr>
<td>Time mean</td>
<td>20.11</td>
<td>31.41</td>
<td>38.44</td>
<td>49.33</td>
<td>55.19</td>
<td>58.26</td>
<td>59.89</td>
<td></td>
</tr>
</tbody>
</table>

n = 9; Values expressed as beats per minute (bpm); PASS = passive recovery; ACT = active recovery; BRE = breathing at 6 breaths per minute during recovery.
Figure 2: Systolic blood pressure recovery (BPR) over time per protocol

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; 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
Table 4: Effect of recovery protocol on blood pressure recovery (BPR)

<table>
<thead>
<tr>
<th></th>
<th>20 sec.</th>
<th>40 sec.</th>
<th>1 min.</th>
<th>2 min.</th>
<th>3 min.</th>
<th>4 min.</th>
<th>5 min.</th>
<th>Protocol mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>PASS</td>
<td>25.89 ± 13.01</td>
<td>25.00 ± 8.96</td>
<td>29.11 ± 11.03</td>
<td>32.78 ± 11.34</td>
<td>36.67 ± 10.16</td>
<td>38.78 ± 8.45</td>
<td>40.33 ± 11.09</td>
<td>32.65</td>
</tr>
<tr>
<td>ACT</td>
<td>30.56 ± 8.75</td>
<td>33.78 ± 10.27</td>
<td>34.56 ± 11.91</td>
<td>38.11 ± 9.05</td>
<td>37.33 ± 10.09</td>
<td>40.44 ± 12.50</td>
<td>40.67 ± 14.57</td>
<td>36.49</td>
</tr>
<tr>
<td>Time  mean</td>
<td>26.67</td>
<td>28.48</td>
<td>30.11</td>
<td>34.07</td>
<td>36.41</td>
<td>39.22</td>
<td>39.03</td>
<td></td>
</tr>
</tbody>
</table>

n = 9; Values expressed as mmHg; PASS = passive recovery; ACT = active recovery; BRE = breathing at 6 breaths per minute during recovery

Table 5: Systolic blood pressure recovery (BPR) interactions over time

<table>
<thead>
<tr>
<th>(A) Time</th>
<th>(B) Time</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>BPR20</td>
<td>BPR40</td>
<td>1.00</td>
</tr>
<tr>
<td>BPR1</td>
<td>0.35</td>
<td></td>
</tr>
<tr>
<td>BPR2</td>
<td>0.06</td>
<td></td>
</tr>
<tr>
<td>BPR3</td>
<td>0.01 *</td>
<td></td>
</tr>
<tr>
<td>BPR4</td>
<td>0.00 *</td>
<td></td>
</tr>
<tr>
<td>BPR5</td>
<td>0.01 *</td>
<td></td>
</tr>
<tr>
<td>BPR40</td>
<td>BPR20</td>
<td>1.00</td>
</tr>
<tr>
<td>BPR1</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>BPR2</td>
<td>0.11</td>
<td></td>
</tr>
<tr>
<td>BPR3</td>
<td>0.01 *</td>
<td></td>
</tr>
<tr>
<td>BPR4</td>
<td>0.00 *</td>
<td></td>
</tr>
<tr>
<td>BPR5</td>
<td>0.04 *</td>
<td></td>
</tr>
<tr>
<td>BPR1</td>
<td>BPR20</td>
<td>0.35</td>
</tr>
<tr>
<td>BPR40</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>BPR2</td>
<td>0.12</td>
<td></td>
</tr>
<tr>
<td>BPR3</td>
<td>0.03 *</td>
<td></td>
</tr>
<tr>
<td>BPR4</td>
<td>0.00 *</td>
<td></td>
</tr>
<tr>
<td></td>
<td>BPR5</td>
<td></td>
</tr>
<tr>
<td>-----</td>
<td>-------------</td>
<td></td>
</tr>
<tr>
<td>BPR2</td>
<td>BPR20 0.05*</td>
<td></td>
</tr>
<tr>
<td></td>
<td>BPR40 0.06</td>
<td></td>
</tr>
<tr>
<td></td>
<td>BPR1 0.11</td>
<td></td>
</tr>
<tr>
<td></td>
<td>BPR3 0.12</td>
<td></td>
</tr>
<tr>
<td></td>
<td>BPR4 0.02*</td>
<td></td>
</tr>
<tr>
<td></td>
<td>BPR5 0.01*</td>
<td></td>
</tr>
<tr>
<td>BPR3</td>
<td>BPR20 0.01*</td>
<td></td>
</tr>
<tr>
<td></td>
<td>BPR40 0.01*</td>
<td></td>
</tr>
<tr>
<td></td>
<td>BPR1 0.03*</td>
<td></td>
</tr>
<tr>
<td></td>
<td>BPR2 0.02*</td>
<td></td>
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<tr>
<td></td>
<td>BPR4 0.09</td>
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<tr>
<td></td>
<td>BPR5 1.00</td>
<td></td>
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<tr>
<td>BPR4</td>
<td>BPR20 0.00*</td>
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<td></td>
<td>BPR40 0.00*</td>
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<tr>
<td>BPR5</td>
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<td>BPR40 0.04*</td>
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<td>BPR3 1.00</td>
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<tr>
<td></td>
<td>BPR4 1.00</td>
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</tr>
</tbody>
</table>

n = 9; BPR = 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; * = p ≤ 0.05
### APPENDIX D

**CONTRAINDICATIONS TO EXERCISE**

<table>
<thead>
<tr>
<th>Absolute</th>
<th>Relative</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute myocardial infarction (3-5 days)</td>
<td>Left main coronary stenosis or its equivalent</td>
</tr>
<tr>
<td>Unstable angina</td>
<td>Moderate stenotic valvular heart disease</td>
</tr>
<tr>
<td>Uncontrolled arrhythmias causing symptoms or hemodynamic compromise</td>
<td>Severe untreated arterial hypertension at rest (&gt;200 mg Hg systolic, &gt;120 mm Hg diastolic)</td>
</tr>
<tr>
<td>Sycope</td>
<td>Tachyarrhythmias or bradyarrhythmias</td>
</tr>
<tr>
<td>Active endocarditis</td>
<td>High-degree atrioventricular block</td>
</tr>
<tr>
<td>Acute myocarditis or pericarditis</td>
<td>Hypertrophic cardiomyopathy</td>
</tr>
<tr>
<td>Symptomatic severe aortic stenosis</td>
<td>Significant pulmonary hypertension</td>
</tr>
<tr>
<td>Uncontrolled heart failure</td>
<td>Advanced or complicated pregnancy</td>
</tr>
<tr>
<td>Acute pulmonary embolus or pulmonary infarction</td>
<td>Electrolyte abnormalities</td>
</tr>
<tr>
<td>Thrombosis of lower extremities</td>
<td>Orthopedic impairment that compromises exercise performance</td>
</tr>
<tr>
<td>Suspected dissecting aneurysm</td>
<td>Untreated anemia (hemoglobin level between 8 and 11 gm/dL)</td>
</tr>
<tr>
<td>Uncontrolled asthma</td>
<td></td>
</tr>
<tr>
<td>Pulmonary edema</td>
<td></td>
</tr>
<tr>
<td>Room air desaturation at rest ≤ 85%</td>
<td></td>
</tr>
<tr>
<td>Respiratory failure</td>
<td></td>
</tr>
<tr>
<td>Acute noncardiopulmonary disorder that may affect exercise performance or be aggravated by exercise (i.e., infection, renal failure, thyrotoxicosis)</td>
<td></td>
</tr>
<tr>
<td>Mental impairment leading to inability to cooperate</td>
<td></td>
</tr>
<tr>
<td>Evidence of extensive visceral or skeletal metastases, or both</td>
<td></td>
</tr>
</tbody>
</table>