EFFECTS OF AEROBIC EXERCISE ON NOCTURNAL BLOOD PRESSURE DIPPING IN PREHYPERTENSIVE INDIVIDUALS

A Thesis by BENJAMIN WILLIAM CARTNER

Submitted to the Graduate School Appalachian State University in partial fulfillment of the requirements for the degree of MASTER OF SCIENCE

December 2011 Department of Health, Leisure, and Exercise Science

EFFECTS OF AEROBIC EXERCISE ON NOCTURNAL BLOOD PRESSURE DIPPING IN PREHYPERTENSIVE INDIVIDUALS

A Thesis by BENJAMIN WILLIAM CARTNER December 2011

APPROVED BY:

Scott Collier, PhD Chairperson, Thesis Committee

__

__

__

__

N. Travis Triplett, PhD Member, Thesis Committee

David Morris, PhD Member, Thesis Committee

Edelma Huntley, PhD Dean, Research and Graduate Studies Copyright by Benjamin William Cartner 2011 All Rights Reserved

FOREWORD

The research detailed in this thesis will be submitted to *Medicine & Science*, the official journal of the American College of Sports Medicine. The thesis has been prepared according to the guidelines set forth by the Graduate School of Appalachian State University.

ABSTRACT

EFFECTS OF AEROBIC EXERCISE ON NOCTURNAL BLOOD PRESSURE DIPPING IN PREHYPERTENSIVE INDIVIDUALS. (December 2011)

Benjamin William Cartner, B.S., Appalachian State University

Chairperson: Scott R. Collier

During the typical nocturnal sleep cycle, blood pressure (BP) will "dip" or decrease when compared to diurnal blood pressure reducing stress on the cardiac system. Compared with a reference group of normotensive dippers, hypertensive non-dippers may experience a much higher relative risk of cardiovascular mortality than normotensive non-dippers and hypertensive dippers. Post exercise hypotension may increase the dipping response; however, exercise timing on sleeping blood pressures has never been elucidated. Therefore, the purpose of this study was to examine how exercise timing affects circadian blood pressure and specifically nocturnal blood pressure. METHODS: Fifteen prehypertensive subjects, 7 male and 8 female (mean age 40 ± 3 years), with no self-reported cardiac disorders participated in this study. Following a graded exercise test during visit 1, subjects reported for 3 exercise times at 7 AM (aerobic exercise completed at 7 AM), 1 PM (aerobic exercise completed at 1 PM), and 7 PM (aerobic exercise completed at 7 PM) in random order to perform a 30 min treadmill protocol at 65% of their VO2 max (maximal oxygen uptake). An Oscar 2™ ambulatory blood pressure cuff was used to monitor blood pressure responses for the 24 hours immediately following exercise. RESULTS: Aerobic exercise at 7 AM invoked the greatest dip in nocturnal systolic blood pressure (SBP; 16.7% drop at 7 AM, 12.3% at 1 PM, 15.2% at 7 PM) than exercise in the afternoon or evening time slots and 7 PM invoked the greatest dip in nocturnal diastolic blood pressure (DBP; 20.9% drop at 7 AM, 15.4% at 1 PM, 21.1% at 7 PM). CONCLUSION: When looking at the overall benefit of the blood pressure variables measured,

v

exercising in the morning at 7 AM may be the most beneficial epoch of time for nocturnal blood pressure changes.

ACKNOWLEDGMENTS

At this point I would like to thank my mentors, Dr. Scott R. Collier, Dr. N. Travis Triplett and Dr. Jeffrey T. Soukup for their guidance and support throughout my graduate studies at Appalachian State University. To my thesis committee members, Dr. Scott R. Collier, Dr. N. Travis Triplett, and Dr. David M. Morris, I express my utmost gratitude for your involvement in the fruition of this project. To my fellow graduate students, I thank you for your unyielding support and friendship over these past two years and I anticipate the opportunity when we may again work together in the future. I also would like to thank the Office of Student Research for their financial support of this project by the Graduate Student Association Senate Research Award and Graduate School Research Grants.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

It is well known that blood pressure (BP) increases with age and, as a result, the incidence of hypertension increases. Hypertension also remains the most prevalent cardiovascular disease that afflicts the elderly population. Further, hypertension-associated cardiovascular disease is the leading cause of morbidity and mortality in the Western world (4) .

There is mounting evidence correlating the benefits of aerobic exercise to decreases in resting BP, which overall will lend to the prevention and treatment of cardiovascular disease (CVD) and all-cause mortality [\(12\)](#page-34-0). Aerobic exercise has been shown to reduce blood pressure in more than 75% of the hypertensive population and has become a preferred method of non-pharmacologic treatment for the lowering and maintenance of blood pressure [\(7\)](#page-33-1). Thirty minutes of moderate intensity, aerobic exercise can decrease blood pressure in hypertensive individuals when compared to otherwise non-exercise controls [\(12\)](#page-34-0). Individuals with undiagnosed hypertension share the same 1.5 to 3 fold higher risk of contracting cardiovascular disease as individuals with chronic elevated blood pressure [\(1\)](#page-33-2). Studies have also shown that the risk of end target organ damage is comparable for both individuals with masked hypertension and chronic hypertension [\(1\)](#page-33-2). Recent studies have concluded that moderate intensity aerobic exercise may have more cardiovascular benefit than higher intensity exercises for individuals with hypertension [\(7\)](#page-33-1). The training effect of moderate intensity aerobic exercise has been shown to decrease systolic blood pressure an average of 7 to10 mmHg systolic and 4 to 8 mmHg diastolic in hypertensive individuals [\(12\)](#page-34-0).

1

Additionally, it has been shown that the blood pressure lowering effects for hypertensive individuals participating in a moderate intensity aerobic exercise regimen continue to remain steadfast even with a 33% reduction in blood pressure lowering medications [\(12\)](#page-34-0). With a consistent 16 week moderate intensity exercise program, daily blood pressure has been shown to be significantly reduced in individuals with Stage 1 and 2 hypertension [\(5,](#page-33-3) [12\)](#page-34-0). This information continues to support current aerobic exercise prescription as an effective, initial form or appendage to existing hypertensive blood pressure mediation [\(7\)](#page-33-1).

Calhoun and Harding describe that even the smallest elevations in BP during sleep project a marked increase in the risk for cardiovascular morbidity and mortality [\(3\)](#page-33-4). During the typical nocturnal sleep cycle, BP will decrease (dip) by 10-20% compared to the awake BP [\(25\)](#page-35-0). If an individual's BP does not experience this "dipping" pattern, he or she is termed a "non-dipper" [a "non-dipper" is arbitrarily defined as an individual with a nocturnal decline in systolic or diastolic blood pressure of less than 10% [\(25\)](#page-35-0)]. This non-dipping archetype ultimately results in poor prognosis for lifespan and greater risk for future cardiovascular complications [\(28\)](#page-36-0). Compared with a reference group of normotensive dippers [< 135/85 mmHg out-of-office, < 140/90 mmHg clinical [\(19\)](#page-35-1)], hypertensive non-dippers experience a more than five-fold increase in the relative hazard rate of cardiovascular mortality; whereas, normotensive non-dippers' and hypertensive dippers' relative risk rose only two- to threefold [\(15\)](#page-34-1). These data infer that for each 5% increment in the systolic or diastolic night-today ratio, there is a 20% rise in the risk for cardiovascular death, even when 24 hour ambulatory blood pressure is within the normotensive range [\(15\)](#page-34-1).

2

Upon review of the existing research, Pickering and colleagues have suggested that at least 25% of the general hypertensive population suffers from a non-dipping BP pattern; however, no studies have ever investigated the effect of exercise timing on sleep BP [\(20\)](#page-35-2). Therefore, the purpose of this study is to investigate the effects of aerobic exercise timing on ambulatory BP throughout a 24 hour cycle in middle aged pre- to stage-one adult hypertensives. We hypothesize that aerobic exercise completed at 7 PM will allow the subsequent post exercise hypotension (PEH) phenomenon to reduce BP in the early stages of sleep causing a more pronounced "dip" in the nocturnal BP pattern.

METHODS AND PROCEDURES

Subjects

Fifteen prehypertensive men and women (systolic blood pressure (SBP) range of 120- 139 mmHg, diastolic blood pressure (DBP) range of 80-89 mmHg) between the ages of 30 and 60 years old were recruited from the local community. The guidelines of the study included that no subjects had any self-reported sleep disorder, and all were non-smokers and were not on any medications, including aspirin therapy or sleep aids as identified in health and activity history questionnaires. Investigators reviewed the screening questionnaire to ensure subjects met the required guidelines of the study. The investigation was approved by the Institutional Review Board of Appalachian State University.

Experimental design

Subjects reported to the laboratory on four separate occasions. On the first visit, each subject completed a written informed consent as well as a physical activity and health history questionnaire (American College of Sports Medicine guidelines, 2010). Age, height, weight, and blood pressure were all recorded, followed by an assessment of peak aerobic capacity. A graded treadmill (Quinton, Bothell, WA) exercise test to volitional exhaustion with metabolic measurements was employed to determine each subject's VO_{2peak} (Parvo Medics, Sandy, UT). This investigation utilized a randomized, crossover design where each subject performed each separate time-of-day exercise protocol. Additionally, subjects were familiarized with all of the data collection equipment utilized within the study.

During visits two, three, and four, participants completed the randomized aerobic exercise protocol at either 7 AM, 1 PM, or 7 PM. An average washout period of 72 hours was allotted between subsequent exercise sessions which all took place during a weekday (Monday, Thursday, Monday). Subjects were asked to refrain from alcohol and caffeine 12 hours prior to each exercise session and throughout the duration of the data collection. Following each exercise session, participants wore an ambulatory blood pressure device (SunTech Medical Oscar 2, Morrisville, NC) for the next 24 hour period as well as an ambulatory sleep monitoring system (Zeo, Newton, MA) on the nights following exercise. Subjects were instructed to maintain the same time to bed and time to wake following each exercise bout in order to control for total sleep time. All subjects slept at least 7 hours each night following exercise as confirmed by the Zeo monitors.

Anthropometric assessment

Body weight and body composition were calculated to the nearest 0.01 kg using a calibrated laboratory electronic scale (Tanita bioelectrical impedance analysis (BIA), Tokyo, Japan). Height was calculated using a stadiometer and recorded to the nearest 0.5 cm.

Maximal aerobic capacity

Peak oxygen consumption (VO_{2peak}) was assessed using a customized treadmill protocol (Modified Armstrong protocol). Intensity started at 2.5 miles per hour (mph) and increased by 1 mph every two minutes until a comfortable pace was established. If additional intensity was required, the grade of the treadmill was increased (2.5 %) at two-minute intervals until volitional fatigue was reached. Heart rate was recorded once per minute during the protocol, and a minimum of four minutes into recovery, using a Polar Heart Rate Monitor (Polar Electro Incorporated, Woodbury, NY). Ratings of perceived exertion (RPE) were also

assessed once per stage using the 6-20 Borg Scale. Expired gases were analyzed using a True One metabolic system (Parvo Medics, Sandy, UT). Exercise capacity was assessed by exercise time and total workload expressed in metabolic equivalents (METs). A 12-lead electrocardiogram (ECG) was used to monitor the test for men and women greater than 45 and 50 years of age, respectively.

Exercise protocol

Values attained from the preliminary exercise testing session were used to design the aerobic exercise prescription. All individuals were provided with a 30 minute, aerobic exercise bout on a motorized treadmill at an intensity that elicited a HR consistent with 65% of their VO_{2peak} . Heart rate was monitored throughout every 30 minute session using a Polar Heart Rate Monitor to ensure that subjects remained at 65% of their corresponding VO_{2peak} HR. Each subject completed each bout of exercise at all three time points.

Ambulatory blood pressure measurement (ABPM)

Upon completion of each exercise session, subjects were outfitted with a SunTech Medical Oscar 2 ambulatory blood pressure device. The device was programmed to take oscillatory blood pressure measurements every 20 minutes throughout the 24 hour postexercise time and every 40 minutes during sleep. Data was stored within the device until it was later uploaded onto the laboratory computer.

Ambulatory sleep stage measurement

On nights following the completed exercise sessions, subjects wore a ZeoTM ambulatory sleep monitoring headband. The system consists of a soft headband (metallic fibers woven into the material) and a bedside display. The two-piece system utilizes dry sensor electroencephalogram (EEG) technology to transmit brainwave data wirelessly to the

6

bedside display where it is stored for later analysis. The ZeoTM system has been validated against an in-laboratory polysomnography and shown to be an accurate and easy way to measure sleep stages [\(23\)](#page-35-3).

Treatment of the data

Data were gathered as mean +/- standard error of the mean (SEM) for SBP, DBP, and percent dip throughout the 24 hour cycle. Data were divided into wake blood pressure and sleep blood pressure groups. All data were analyzed by SPSS version 15 (SPSS, Chicago, IL, USA) using a 1 x 3 (exercise x time) analysis of variance (ANOVA) with repeated measures. A Bonferroni post-hoc test was applied to determine significance between pairwise comparisons.

RESULTS

Subjects each completed their non-exercise control session to be compared with their individual exercise data. All of our subjects were classified as dippers following the initial lab assessment. Subject descriptive characteristics are presented in Table 1. The timing of aerobic exercise has various marked effects on ambulatory BP. These significant differences arose during times of sleep with no significant BP changes during awake times. These timing differences allowed for comparison of absolute differences in sleep time BP among the separate exercise timing bouts.

Nocturnal blood pressure variables

Sleep SBP was significantly augmented by the timing of exercise as demonstrated in Figure 1. Aerobic exercise completed at 7 AM revealed a significant reduction in sleep systolic pressure (107.00 mmHg \pm 9.08 mmHg) when compared to sleep systolic blood pressure when aerobic exercise was completed at the 1 PM (114.8 mmHg \pm 15.53 mmHg) time point $(p < 0.05$; Figure 1). There were no significant differences in sleep systolic blood pressure between 7 AM and 7 PM (110.0 mmHg \pm 12.8 mmHg) or between 1 PM and 7 PM $(p < 0.05)$.

Sleep diastolic blood pressure also presented with a significant pressure decrease at 7 AM (60.80 mmHg \pm 1.61 mmHg) when compared to aerobic exercise completed at the 1 PM (65.67 mmHg \pm 1.99 mmHg) time point ($p \le 0.05$; Figure 2). Sleep diastolic blood pressure was significantly lower following aerobic exercise completed at 7 PM (61.87 mmHg \pm 1.99 mmHg) compared to sleep diastolic blood pressure when aerobic exercise was completed at 1 PM (65.67 mmHg \pm 1.99 mmHg; $p \le 0.05$; Figure 2). There was no significant sleep diastolic pressure difference between aerobic exercise completed at 7 AM and 7 PM (*p* < 0.05).

Nocturnal blood pressure dipping

The percent dip for systolic pressure was significantly altered between aerobic exercise completed at the 7 AM (16.67 % \pm 0.92 %) and the 1 PM (12.34 % \pm 1.24 %) exercise time points ($p < 0.05$; Figure 3). There was no significant difference in systolic blood pressure dip between 1 PM and 7 PM (15.23 $% \pm 5.23$ %) or 7 AM and 7 PM ($p <$ 0.05).

The percent dip for diastolic pressure was significant between aerobic exercise completed at the 7 AM (20.90 % \pm 1.14 %) and the 1 PM (15.44 % \pm 1.67 %) exercise time points ($p \le 0.05$) as well as the 7 PM (21.16 % \pm 1.91 %) and the 1 PM (15.44 % \pm 1.67 %) exercise time points ($p \le 0.05$; Figure 4). There was no significant difference in the diastolic blood pressure dip between 7 AM and 7 PM.

Diurnal blood pressure variables

There were no significant changes in the mean awake systolic (7 AM 123.2 ± 11.01) mmHg, 1 PM 126.4 ± 12.3 mmHg, 7 PM 124.3 ± 15.8 mmHg) or diastolic (7 AM 72.8 ± 7.2 mmHg, 1 PM 75.0 \pm 6.7 mmHg, 7 PM 73.8 \pm 9.9 mmHg) pressures at any time point.

Age (years)	Height (cm)	Weight (kg)	$VO2$ (ml·kg·min)	BMI (kg/m^2) Pressure	Blood (mmHg)
40 ± 3	173 ± 2.2	75 ± 3.2	40 ± 3.3	25.3 ± 2.5	$133/80 \pm 3.1$

Table 1. Descriptive Characteristics $(n = 15)$

Values are mean ± SEM.

Figure 1. Changes in systolic blood pressure at night following the exercise protocol at 7 AM, 1 PM, and 7 PM. The data shown are nocturnal systolic blood pressures 107.00 mmHg, 114.80 mmHg, and 110.00 mmHg compared to exercise time intervals 7 AM, 1 PM, and 7 PM, respectively. Data are presented as mean ± SEM in millimeters of mercury (mmHg). *Significance at $p \le 0.05$ noted at 7 AM versus 1 PM.

Figure 2. Changes in diastolic blood pressure at night following the exercise protocol at 7 AM, 1 PM, and 7 PM. The data shown are nocturnal diastolic blood pressures 60.80 mmHg, 65.67 mmHg, and 61.87 mmHg compared to exercise time intervals 7 AM, 1 PM, and 7 PM, respectively. Data are presented as mean \pm SEM in millimeters of mercury (mmHg). *Significance at $p \le 0.05$ noted at 7 AM versus 1 PM and 7 PM versus 1 PM.

Figure 3. Changes in systolic blood pressure dip at night following the exercise protocol at 7 AM, 1 PM, and 7 PM. Exercise timing resulted in differential nocturnal systolic dips of 16.67 %, 12.34 %, 15.23 % at 7 AM, 1 PM, and 7 PM, respectively. Data are presented as mean \pm SEM in percent dip (%).

*Significance at $p \le 0.05$ noted at 7 AM versus 1 PM.

Figure 4. Changes in diastolic blood pressure dip at night following the exercise protocol at 7 AM, 1 PM, and 7 PM. Exercise timing resulted in differential nocturnal diastolic dips of 20.90 %, 15.44 %, and 21.16 % at 7 AM, 1 PM and 7 PM, respectively. Data are presented as mean \pm SEM in percent dip (%).

*Significance at $p \le 0.05$ noted at 7 AM versus 1 PM and 7 PM versus 1 PM.

DISCUSSION

The current study investigated the effects of aerobic exercise timing on nocturnal BP using the variables of 24-hour ambulatory systolic and diastolic pressures as well as the percent dip of systolic and diastolic pressure during sleep. Significant differences in systolic BP, diastolic BP and dipping parameters were shown between exercise times during times of sleep with no significant blood pressure changes during awake times. This study revealed that aerobic exercise conducted at 7 AM produced the most beneficial changes in ambulatory blood pressure when compared to aerobic exercise completed at either the 1 PM or 7 PM time points. Aerobic exercise completed at 7 AM elicited a significantly lower nocturnal systolic pressure, nocturnal diastolic pressure, nocturnal systolic BP dip, and nocturnal diastolic BP dip compared to aerobic exercise completed at 1 PM or 7 PM. These data agree with a study by Jones et al. that showed moderate intensity aerobic exercise decreased nocturnal blood pressure (9).

The most likely cause of the overall decreases in nocturnal blood pressures is a reduction in sympathetic nervous activity [\(2,](#page-33-5) [18,](#page-35-4) [21\)](#page-35-5). Although there are many physiological factors that influence blood pressure change, we can speculate a possible

mechanism for the decreased nocturnal pressures we recorded in this study to be related to the post exercise hypotension phenomenon.

Moderate intensity aerobic exercise has been shown to reduce ambulatory systolic BP for 11 to 12 hours and diastolic BP for 4 to 8 hours post-exercise (post-exercise hypotension), which is propagated by the increased peripheral vasodilation $(7, 11, 17)$ $(7, 11, 17)$ $(7, 11, 17)$ $(7, 11, 17)$. Post exercise hypotension occurs as a product of reduced vascular resistance mediated by the autonomic nervous system and up regulation of multiple vasodilatory mechanisms. Post exercise hypotension has been shown to be present in both normotensive and hypertensive individuals, but it has been observed in greater magnitude in prehypertensive and hypertensive individuals [\(13\)](#page-34-4). Additionally, it has also been suggested that the magnitude of PEH is unrelated to the size of the muscle group being exercise, but the greater the mass of the muscle group being exercised could have an augmented effect on the duration of the PEH phenomenon [\(13\)](#page-34-4). This projected increase in the PEH response may explain why we recorded lower nocturnal blood pressure values following morning exercise using a treadmill protocol instead of during arm or leg ergometry. A recent study conducted by Jones et al. concluded that moderate aerobic exercise conducted in the morning showed the highest reactivity of the PEH phenomenon with a differential of 8 to 14 mmHg in mean arterial BP from diurnal to nocturnal pressure values [\(8\)](#page-33-6). This finding is consistent with the lowered nocturnal BP responses we recorded following the morning exercise protocol. It has been

shown that the reduction in blood pressure due to PEH is directly related to the current level of BP; that is to say that the higher the blood pressure is initially, the greater the PEH response and the greater the reduction in blood pressure [\(13\)](#page-34-4). This may further explain why the prehypertensive group that we studied had an augmented blood pressure response to the exercise protocol.

BP and heart rate both experience a spike upon wakening known as the "morning pressure surge" and can be attributed to circadian rhythms of many other physiological parameters including hemodynamic, hematological, and humoral factors as well as postural changes [\(29\)](#page-36-1). If an exercise treatment is to be implemented at this morning time of heightened BP, then the resulting PEH would exhibit greater reductions in blood pressure following exercise [\(13\)](#page-34-4). The effects of PEH on sleep pattern are not well understood, but the limited research has concluded that exercise in the morning shows the largest fluctuation in BP due to PEH [\(11\)](#page-34-2). Further, a greater magnitude of PEH has been documented following periods of nocturnal sleep and was absent when exercise was completed after day time sleep [\(8\)](#page-33-6).

The benefit of aerobic exercise on blood pressure and hypertension has been well documented, but little data exist on how the timing of aerobic exercise can influence these benefits. A recent study conducted by Shiotani et al. shows that aerobic exercise conducted in the morning elicits a significantly favorable decrease in heart rate throughout the day [\(24\)](#page-35-6). The Shiotani et al. study did not, however, find significance in any parameter of blood pressure but attested that aerobic exercise completed during morning hours may have the most benefit to the cardiovascular circadian rhythm [\(24\)](#page-35-6). The findings from this study support the theory that blood pressure reactivity to physical activity is greater during bouts of morning exercise [\(10\)](#page-34-5).

A recent study investigating the effects of morning aerobic exercise on vascular shear stress reported that brachial shear stress was significantly higher in the morning at 8 AM than at 2 PM following moderate intensity aerobic exercise protocols [\(10\)](#page-34-5). This increase in shear stress may contribute to the decreased nocturnal blood pressure values we recorded following aerobic exercise conducted at 7 AM in our current study. The decreased sheer stress rate at 2 PM may also validate why we recorded a decreased benefit of aerobic exercise conducted at 1 PM.

Park et al. investigated the circadian blood pressure difference between dippers and non-dippers following aerobic exercise (30 minutes of intermittent training on a motorized treadmill at 50% VO_{2peak}) during morning (6 AM to 8 AM) and evening (5 PM to 7 PM) exercise [\(16\)](#page-34-6). Park et al. ultimately concluded that aerobic exercise in the evening exhibited a greater reduction in systolic pressure for non-dippers and morning exercise produced similar systolic blood pressure responses for both dippers and non-dippers [\(16\)](#page-34-6). These data are similar to the findings in the current study that there is a benefit of aerobic exercise at 7

PM; however, the present study supports that the most beneficial blood pressure response follows aerobic exercise completed at 7 AM. These disparities may be partially due to the fact that the subjects in the Park et al. study were hypertensive and were on anti-hypertensive medications which may produce lower blood pressure responses throughout the day and alternate the overall blood pressure response to exercise. A large difference between the Park et al. study and the present study may be the training intensity since they used 50% of VO_{2peak} , yet we used 65% VO_{2peak} , which may elicit divergent BP responses [\(6\)](#page-33-7).

Sleep is the most important recovery period from the elevated pressures associated with daily activities and it has been reported that regular physical activity enhances overall sleep quality [\(14\)](#page-34-7). However, the most appropriate time period of exercise for cardiovascular benefit during sleep has yet to be established. Recent studies have shown that vigorous activity right before bed time may not be beneficial to the cardiovascular system [\(14\)](#page-34-7). When comparing a vigorous exercise group to a non-exercise control group, the exercise group experienced a greater proportion of Non-Rapid Eye Movement sleep and maintained an elevated HR throughout their sleep cycle, especially within the first 3 hours of sleep when compared to the non-exercising control group [\(14\)](#page-34-7).

Another seemingly potential mechanism for the augmented BP response to exercise timing may be the influence of catecholamines and the subsequent circadian rhythms. It has been documented that moderate intensity training increases baroreflex sensitivity (BRS)

throughout the day [\(27\)](#page-36-2). This increased BRS has been associated with lower arterial pressures, especially at night during sleep [\(26\)](#page-35-7). BRS has been documented as significantly higher during dreaming sleep and that arterial pressures are inversely correlated with BRS [\(26\)](#page-35-7). It is possible that the acute bout of aerobic exercise completed at 7 AM heightened the BRS response and provided a cascade for lower pressures throughout the remainder of the day and into the evening. The lower arterial pressures we recorded during sleep may be actively maintained in individuals through a heightened BRS [\(26\)](#page-35-7).

There is a measurable increase in catecholamines during the morning hours which contribute to the increases in cardiac output, heart rate, and blood pressure. It has been shown that the "morning surge" in catecholamines is evident from 6 AM to 12 PM, increasing the risk for cardiovascular events (29). The circadian peak for vagal cardiac markers that are cardioprotective and the trough in SBP and DBP occurs during the biological night corresponding to 12 AM to 8 AM [\(22\)](#page-35-8). The existing research continues to support that this vagal peak overlaps with the sympathetic rise from 6 AM to 8 AM and may possibly counterbalance the sympathetic peak and ultimately prove to be more cardioprotective [\(22\)](#page-35-8). With regard to the heightened BRS and this overlap in sympathetic and vagal circadian rhythms, performing moderate intensity aerobic exercise at 7 AM may explain the lowered SBP and DBP values we recorded throughout the night.

Data from the present study indicated that exercise at 7 AM had the most favorable influence on sleep systolic and diastolic pressure change benefits for all prehypertensive subjects. The benefit of diastolic dipping may be most beneficial for clinical populations suffering from congestive heart failure or any other cardiovascular diseases that relate to ventricular filling.

CONCLUSIONS AND IMPLICATIONS

The results from the present study show that partaking in a moderate bout of aerobic exercise in the morning (7 AM) can have marked effects on the circadian BP rhythm. It is well known that aerobic exercise contributes to decreased cardiovascular risk; however, no studies have been conducted pertaining to a specific epoch that may produce the most beneficial response to the blood pressure archetype. Aerobic exercise completed at 7 AM and 7 PM both yielded beneficial results with the most wide ranging benefits coming from exercise at 7 AM. A specifically timed bout of moderate intensity aerobic exercise completed at 7 AM may be an appropriate treatment to absolve or at least alleviate some the problems associated with cardiovascular disease. The current investigation suggests that the timing of aerobic exercise may influence the nocturnal BP system and may be a useful intervention in treating cardiovascular disease.

REFERENCES

- 1. Angeli F, Reboldi G, Verdecchia P. Masked hypertension: evaluation, prognosis, and treatment. *Am J Hypertens*. 2010;23(9):941-8.
- 2. Bristow JD, Honour AJ, Pickering TG, Sleight P. Cardiovascular and respiratory changes during sleep in normal and hypertensive subjects. *Cardiovasc Res*. 1969;3(4):476-85.
- 3. Calhoun DA, Harding SM. Sleep and hypertension. *Chest*. 2010;138(2):434-43.
- 4. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL, Jr., Jones DW, Materson BJ, Oparil S, Wright JT, Jr., Roccella EJ. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. *JAMA*. 2003;289(19):2560-72.
- 5. Collier SR, Kanaley JA, Carhart R, Jr., Frechette V, Tobin MM, Hall AK, Luckenbaugh AN, Fernhall B. Effect of 4 weeks of aerobic or resistance exercise training on arterial stiffness, blood flow and blood pressure in pre- and stage-1 hypertensives. *J Hum Hypertens*. 2008;22(10):678-86.
- 6. Fagard RH. Exercise characteristics and the blood pressure response to dynamic physical training. *Med Sci Sports Exerc*. 2001;33(6 Suppl):S484-92
- 7. Hagberg JM, Park JJ, Brown MD. The role of exercise training in the treatment of hypertension: an update. *Sports Med*. 2000;30(3):193-206.
- 8. Jones H, George K, Edwards B, Atkinson G. Effects of time of day on post-exercise blood pressure: circadian or sleep-related influences? *Chronobiol Int*. 2008;25(6):987-98.
- 9. Jones H, George K, Edwards B, Atkinson G. Exercise intensity and blood pressure during sleep. *Int J Sports Med*. 2009;30(2):94-9.
- 10. Jones H, Green DJ, George KP, Black MA, Atkinson G. Evidence for a greater elevation in vascular shear stress after morning exercise. *Med Sci Sports Exerc*. 2009;41(6):1188- 93.
- 11. Kenney MJ, Seals DR. Postexercise hypotension. Key features, mechanisms, and clinical significance. *Hypertension*. 1993;22(5):653-64.
- 12. Kokkinos P, Sheriff H, Kheirbek R. Physical inactivity and mortality risk. *Cardiol Res Pract*. 2011;2011:924945.
- 13. MacDonald JR. Potential causes, mechanisms, and implications of post exercise hypotension. *J Hum Hypertens*. 2002;16(4):225-36.
- 14. Myllymaki T, Kyrolainen H, Savolainen K, Hokka L, Jakonen R, Juuti T, Martinmaki K, Kaartinen J, Kinnunen ML, Rusko H. Effects of vigorous late-night exercise on sleep quality and cardiac autonomic activity. *J Sleep Res*. 2011;20(1 Pt 2):146-53.
- 15. Ohkubo T, Hozawa A, Yamaguchi J, Kikuya M, Ohmori K, Michimata M, Matsubara M, Hashimoto J, Hoshi H, Araki T, Tsuji I, Satoh H, Hisamichi S, Imai Y. Prognostic significance of the nocturnal decline in blood pressure in individuals with and without high 24-h blood pressure: the Ohasama study. *J Hypertens*. 2002;20(11):2183-9.
- 16. Park S, Jastremski CA, Wallace JP. Time of day for exercise on blood pressure reduction in dipping and nondipping hypertension. *J Hum Hypertens*. 2005;19(8):597-605.
- 17. Pescatello LS, Fargo AE, Leach CN, Jr., Scherzer HH. Short-term effect of dynamic exercise on arterial blood pressure. *Circulation*. 1991;83(5):1557-61.
- 18. Pickering TG. The clinical significance of diurnal blood pressure variations. Dippers and nondippers. *Circulation*. 1990;81(2):700-2.
- 19. Pickering TG, Davidson K, Gerin W, Schwartz JE. Masked hypertension. *Hypertension*. 2002;40(6):795-6.
- 20. Pickering TG, Kario K. Nocturnal non-dipping: what does it augur? *Curr Opin Nephrol Hypertens*. 2001;10(5):611-6.
- 21. Ruger M, Scheer FA. Effects of circadian disruption on the cardiometabolic system. *Rev Endocr Metab Disord*. 2009;10(4):245-60.
- 22. Scheer FA, Hu K, Evoniuk H, Kelly EE, Malhotra A, Hilton MF, Shea SA. Impact of the human circadian system, exercise, and their interaction on cardiovascular function. *Proc Natl Acad Sci U S A*. 2010;107(47):20541-6.
- 23. Shambroom JR, Johnstone J, Fabregas SE. Evaluation of portable monitor for sleep staging. *Sleep (Rochester)*. 2009;32:(Suppl.): A386. Abstract 1182.
- 24. Shiotani H, Umegaki Y, Tanaka M, Kimura M, Ando H. Effects of aerobic exercise on the circadian rhythm of heart rate and blood pressure. *Chronobiol Int*. 2009;26(8):1636- 46.
- 25. Smolensky MH, Hermida RC, Castriotta RJ, Portaluppi F. Role of sleep-wake cycle on blood pressure circadian rhythms and hypertension. *Sleep Med*. 2007;8(6):668-80.
- 26. Smyth HS, Sleight P, Pickering GW. Reflex regulation of arterial pressure during sleep in man. A quantitative method of assessing baroreflex sensitivity. *Circ Res*. 1969;24(1):109- 21.
- 27. Somers VK, Conway J, Johnston J, Sleight P. Effects of endurance training on baroreflex sensitivity and blood pressure in borderline hypertension. *Lancet*. 1991;337(8754):1363- 8.
- 28. Tsioufis C, Andrikou I, Thomopoulos C, Syrseloudis D, Stergiou G, Stefanadis C. Increased nighttime blood pressure or nondipping profile for prediction of cardiovascular outcomes. *J Hum Hypertens*. 2011;25(5):281-93.
- 29. White WB. Cardiovascular risk and therapeutic intervention for the early morning surge in blood pressure and heart rate. *Blood Press Monit*. 2001;6(2):63-72.

VITA

Benjamin William Cartner was born in Gastonia, North Carolina. His parents are Dr. Larry and Marvie Cartner. He attended high school in Wilkesboro at Wilkes Central High School and graduated in the spring of 2004. He entered Appalachian State University the summer of 2004 and graduated in 2009 with a Bachelor of Science degree in Strength and Conditioning. He began his graduate coursework at Appalachian State University the fall of 2009 and completed his Master of Science in Exercise Science concentrating in Clinical Exercise Science in December 2011. Upon completion of his graduate coursework Mr. Cartner will continue his education in a Physician Assistant program where he plans to practice, research, and educate others about his chosen field.