1-2015

The Acute Effects of Aerobic Exercise Durations on Arterial Compliance in Recreationally Active Males

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THE ACUTE EFFECTS OF AEROBIC EXERCISE DURATIONS ON ARTERIAL COMPLIANCE IN RECREATIONALLY ACTIVE MALES

BY

Joe-Angel Lopez

A THESIS PRESENTED TO THE GRADUATE FACULTY OF THE COLLEGE OF EDUCATION IN PARTIAL FULLFILLMENT

OF THE REQUREMENTS FOR THE DEGREE OF

MASTER OF SCIENCE IN EXERCISE SCIENCE

IN THE FIELD OF HEALTH AND HUMAN PERFORMANCE

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January 2015

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DEDICATION AND ACKNOWLEGEMENT

This thesis is dedicated to my late grandfather Salvador Lopez who passed away from a heart attack when I was a teenager. Always he encouraged me to perform well in school. I hope that I make him proud with through this and my future accomplishments.

I would like to thank my lab supervisor Dr. Murat Karabulut for all the work he has done to help me succeed throughout my academic career. Although I may have not had the confidence in my abilities, he saw potential in me and has been an outstanding mentor of mine. I like to give thanks to Dr. Christopher Ledingham. His supervision has benefitted my critical thinking skills as well as the quality and organization of my writing. I would also like to thank Dr. Sue Ann Chew for stealing from her own valuable time in order to serve on my thesis committee.

I thank the many students who volunteered time to assist me in my data collection over the past few months. Especially, Margarita Gonzalez who volunteered her time to come in, often earlier than she would have liked, to assist me. I would also like to thank Guillermo Perez, who has been a colleague of mine for the past three years for helping me out with formatting of this body of work.

I would like to thank my family for supporting me and encouraging me to continue my education. Had they not, I likely would not have gone to college, like many of my friends. Finally, I thank “my wife” (Borat voice) and best friend in this entire

planet of Earth, Christa Jean Gunn for her support, patience, love and for motivating me to pursue my goals.

Abstract

PURPOSE: The purposes of this study were to 1) examine the acute effects of different aerobic exercise durations on large (LAC) and small (SAC) arterial compliance, 2) examine the acute effects of different aerobic exercise durations on central pulse wave velocity (cPWV), peripheral pulse wave velocity (pPWV), Aortic Index (AIx), and 3) to examine the acute effects of aerobic exercise durations on hemodynamics.

METHODS: Eighteen male subjects (age= 23.4±2.0) performed a maximal aerobic stress test (Bruce protocol) in order to estimate VO2max. Participants were required to meet in the lab fasted for at least 8 hours for three sessions. While in the supine position LAC, SAC, and AIx were then assessed. The velocity of pressure waveforms between the carotid and femoral arteries, and the femoral and pedal arteries were measured to assess cPWV and pPWV, respectively. Participants then performed aerobic exercise on a treadmill at 65% of their respective VO2max for 30 min, 45 min, or 60 min in a randomly assigned ordered. Assessments of arterial compliance were then performed immediately post, 10 min, 20 min, and 40 min post exercise.

RESULTS: No significant difference was seen between conditions in compliance of large arteries. A condition main effect was seen suggesting an increase SAC ($p<0.05$) occurred 10 min following the 60 min condition. Heart rate, stroke volume, and systemic vascular resistance significantly decreased ($p<0.05$) immediately post exercise following the 60 min session. No significant condition effect was seen in either cPWV or pPWV. There was a condition*time interaction ($p<0.04$) and a time main affect ($p<0.01$) for AIx. Further analyses showed a significant decrease in AIx ($p<0.05$) and was seen 45 min post exercise following the 60 min condition.

CONCLUSION: Different durations of moderate intensity aerobic exercise did not provoke changes in large arterial elasticity. Longer durations of aerobic exercise may be necessary to modify vascular tone and vessel diameter resulting in reduced small arterial stiffness. The response may be dissimilar for populations with different activity levels. More research is required to examine the effects of different intensities in combination with different durations of aerobic exercise on arterial compliance.

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CHAPTER I

INTRODUCTION

Arterial stiffness is increasingly being recognized as a marker of future cardiovascular disease (Mattace-Raso et al., 2006; Laurent et al., 2001; Blacher et al., 1999; Zoungas, & Asmar, 2007). Cardiovascular disease is a term that refers to a number of health risk conditions such as myocardial infarction, atherosclerosis, and stroke (O'Rourke et al., 2002). Although mortality rates associated with cardiovascular disease in developed nations have decreased, mainly due to available treatments to myocardial infarction and stroke, it remains to be a significant cause of mortality events (Gaziano, 2007). For this reason, there are a number of ongoing suggestions for active lifestyles and recognition of its significance not only for fitness, but also in order to prevent the onset of cardiovascular diseases.

Studies have been conducted regarding intensive, resistance exercise program on arterial stiffness in patients. In a study by DeVan et al. (2005), 16 participants underwent a single bout of resistance exercise protocol consisting of 9 exercises at 75% of their 1 repetition maximum (1RM). Central arterial compliance, as assessed by B-mode ultrasound, and applanation tonometry, was demonstrated to acutely decrease for up to an hour post exercise. A number of studies support the contention that resistance exercise negatively impacts arterial compliance (Miyachi et al., 2004; Yoon et al., 2010). However, a study by Rakobowchuk et al. (2005) demonstrated conflicting results in which a resistance training program did not cause decreased arterial compliance in sample of young healthy males.

In contrast, aerobic exercise has been shown to increase arterial compliance (Tanaka, et al., 2000; Edwards, Schofield, Magyari, Nichols & Braith, 2004). In particular, the heart rate, carotid femoral pulse wave velocity (PWV) and aortic arterial stiffness all significantly improve

for 20 min, 30 min and 40 min following a 20 min period of aerobic exercise (Blacher, Asmar, Djane, London & Sa-far, 1999). Aerobic exercise may be effective in the treatment and prevention of cardiovascular disease. Presently, most studies focus on the acute effect of aerobic exercises on arterial stiffness. A study by Tanaka et al. (2000) observed that short-term aerobic exercises that last from about 20 min to 40 min enhanced the function of blood vessels.

A decrease in arterial stiffness subsequent to a long duration of aerobic exercise is preventative of cardiovascular risk factors (Tanaka et al., 2000). These observations help establish that aerobic exercises help decrease arterial stiffness (Donato et al., 2000). In a study by Kingwell et al. (1997), an acute 30-min bout of moderate intensity aerobic exercise (65% of VO2max) on a cycle ergometer was demonstrated to increase arterial compliance and decrease peripheral resistance in a sample of sedentary male participants. Similarly an acute 30-min session of aerobic exercise increased small artery compliance for 30-min following exercise, but did not exhibit changes in large arterial stiffness.

Conversely, McClean et al. (2010) demonstrated that a moderate intensity bout of exercise (60% VO2max) lasting for a period of one hour did not cause any changes to arterial stiffness. However, arterial stiffness of the limbs has been demonstrated to significantly decreased following participation in long duration aerobic exercise such as a marathon race (Phillips et al., 2012). The problem is that there remains variability in the literature regarding the acute effect of aerobic exercise on arterial stiffness. Furthermore, no study has examined the acute effects of differing durations of aerobic exercise on arterial stiffness.

Heart rate and plasma catecholamine concentrations have been observed to increase over the course of aerobic exercise (Urhausen et al., 1994). Furthermore, increased heart rate may be indicative of increases in sympathetic tone, which directly causes elevated arterial stiffness

(Mahmud et al., 2001). Longer durations of aerobic exercise may possibly provoke a greater sympathetic response, which may negative impact on arterial compliance.

Study Purposes

The purposes of this study were to 1) examine the acute effects of different aerobic exercise durations on large and small arterial compliance, central pulse wave velocity (cPWV), peripheral pulse wave velocity (pPWV), Aortic Index (AIx), and 2) to examine the acute effects of aerobic exercise durations on hemodynamics by measuring blood pressure, mean arterial pressure (MAP), Pulse Pressure (PP), heart rate (HR), cardiac output (CO), cardiac ejection time (CET), stroke volume, (SV) systemic vascular resistance (SVR) and total vascular impedance (TVI).

Significance of the Study

Arterial stiffness has been increasingly shown to be a risk factor contributing to the development of cardiovascular disease (Laurent et al., 2001). Even though a considerable percentage of incidences of cardiovascular disease can be prevented, the frequency of occurrences continues to climb largely due to insufficient available preventive measures. A single bout of aerobic exercise has been demonstrated to acutely decrease arterial stiffness. However, the duration of aerobic exercise, as it impacts arterial compliance, has not been considered. Some studies have demonstrated that longer duration of aerobic exercise prolonged excess post-exercise oxygen consumption following exercise (Maresh et al., 1992; Sedlock et al., 1989). The evaluation of the effects of various exercise durations on arterial compliance would provide valuable information regarding exercise prescription for individuals with cardiovascular disease.

Findings from this study would also build upon the literature regarding the effects of exercise on arterial health and cardiovascular disease treatment and prevention. This would be significant because cardiovascular disease remains the leading cause of death in the United States (Trogdon et al., 2007).

Delimitations

The study was delimited as follows:

1) Individuals with signs or symptoms of cardiovascular disease were not permitted to participate in the study.
2) Individuals younger than 18 and older than 40 were not permitted to participate in the study.
3) Individuals with joint or muscle problems were excluded from the study.
4) Individuals afflicted with symptoms of respiratory disease, and diabetes were excluded from participation.
5) Individuals taking medication that may interfere with vascular function were excluded.
6) Individuals with chronic back pain were excluded from the study.

Assumptions

The following assumptions were made for this study:

1) All participants completed the study in a timely manner.
2) Participants provided accurate information about medical and health history.
3) The equipment used provided accurate results following proper calibration.

4) Subject have been fasted and abstained from the consumption of caffeine and alcohol for at least eight hours prior to participation.
5) Participants performed testing and exercise sessions at a maximal effort.

Research Questions

In order to test the hypotheses, the following research questions were addressed:

1) What changes in large and small arterial compliance would be induced as a result of different aerobic exercise durations?
2) What changes in central pulse wave velocity (cPWV) and peripheral pulse wave velocity (pPWV) would be induced as a result of different aerobic exercise durations?
3) What changes in systolic and diastolic blood pressure, mean arterial pressure (MAP), pulse pressure (PP), cardiac output (CO), cardiac ejection time (CET), stroke volume (SV), systemic vascular resistance (SVR) and total vascular impedance (TVI) would be induced as a result of different durations of aerobic exercise durations?

Hypotheses

The hypotheses guiding this study were as follows:

1) Aerobic exercise duration of 30 min would yield greater increases in large and small arterial elasticity in contrast to the longer exercise durations of 45 min and 60 min.
2) Aerobic exercise duration of 30 min would yield greater decreases in central pulse wave velocity (cPWV), peripheral pulse wave velocity (pPWV), and augmentation index (AIx) in contrast to the longer exercise durations of 45 min and 60 min.

3) Differences in hemodynamics including systolic (SBP) and diastolic blood pressure (DBP), mean arterial pressure (MAP), pulse pressure (PP), cardiac ejection time (CET), cardiac output (CO), and stroke volume (SV), systemic vascular resistance (SVR), and total vascular impedance (TVI) would be induced as a result of different durations of aerobic exercise.

Operational Definitions

To aid the reader, the following terms are defined as used in this study:

1) **PAR-Q:** PAR-Q (Physical activity readiness questionnaire) is a screening tool that is designed to determine whether a subject may perform the exercise in a safe and risk free manner.
2) **Arterial compliance:** the measurement of the elastic properties of the arteries, which has an inverse relationship with arterial stiffness.
3) **Hemodynamics:** Analysis of physical aspects of blood circulation and blood flow.
4) **Augmentation Index:** Indicator of arterial stiffness.
5) **Pulse Wave Analysis:** Noninvasive method of measuring systemic arterial stiffness by assessing the shape of arterial pressure waveform.
6) **Pulse Wave Velocity:** Noninvasive assessment of arterial compliance in which velocity of blood pressure wave forms traveling between two different sites are measured.

Summary

Arterial stiffness has been demonstrated to be a risk factor in the development of cardiovascular disease (Matace-Raso et al., 2006). If current trends in treatment and prevention persist, incidences of death related to cardiovascular disease is expected to rise 10% over the next 20 years (Heidenreich et al., 2011). Selection of the proper method of exercise is crucial in order to accomplish intended physiological goals. While high intensity resistance exercise is known to increase arterial stiffness, low-intensity aerobic exercise has been demonstrated to decrease arterial stiffness post-exercise (Aizawa et al., 2009).

There remains variability in the literature regarding the effects of aerobic exercise duration on arterial stiffness. Furthermore, no study has examined the acute effects of differing aerobic exercise durations on arterial stiffness. Therefore the purposes of this study were to 1) examine the acute effects of different aerobic exercise durations on large and small arterial compliance, central pulse wave velocity (cPWV), peripheral pulse wave velocity (pPWV), Aortic Index (AIx), and 2) to examine the acute effects of aerobic exercise durations on hemodynamics by measuring blood pressure, mean arterial pressure (MAP), Pulse Pressure (PP), heart rate (HR), cardiac output (CO), cardiac ejection time (CET), stroke volume, (SV) systemic vascular resistance (SVR) and total vascular impedance (TVI).

Chapter 2 contains a review of selected literature related to arterial stiffness. Chapter 3 contains a discussion of the methodology used in this study. In chapter 4, the results of this study are presented and discussed. Chapter 5 contains a summary of the study, conclusions that were drawn, and recommendations for future research related to arterial stiffness prevention programs.

CHAPTER II

REVIEW OF THE LITERATURE

The purpose of this study is to 1) investigate the acute effects of different aerobic exercise durations on large and small arterial elasticity, 2) to investigate the acute effects of different durations of aerobic exercise on cPWV, pPWV, and AIx, 3) to examine how various aerobic exercise durations elicit changes in hemodynamics by measuring resting heart rate (RHR), blood pressure, mean arterial pressure (MAP), cardiac output (CO), stroke volume (SV) systemic vascular resistance (SVR) and total vascular impedance (TVI).

Arterial Compliance

Arterial compliance is defined as the measure of the elastic properties of the arteries, which is negatively related to arterial stiffness (O'Rourke and Mancia, 1999). Arterial compliance can be reliably assessed noninvasively through a method known as pulse wave analysis (PWA) (Wilkinson et al., 2002). This technique involves monitoring arterial circumference and pressure waveforms through applanation tonometry of the radial artery (O'Rourke and Mancia, 1999). A growing body of literature has identified increased arterial stiffness as a marker for cardiovascular disease development (Laurent et al., 2001; Blacher et al., 2003). Cardiovascular disease is a general term that consists of a number of complications of the heart and the blood circulatory system including myocardial infarction, stroke, and atherosclerosis. Cardiovascular complications are a mounting health problem and a significant cause of mortality rates in developed nations (Gaziano, 2007). Even with remarkable and continued reduction in mortality rates arising from cardiovascular disease, the intensity of the problem is still on the rise (Heidenreich et al., 2011). Exercise is recognized for having a protective effect on the heart and overall cardiovascular function. American College of Sports

Medicine guidelines recommend between 30 min and 60 min of exercise most days of the week. No study has examined how differing durations of aerobic exercise effects arterial compliance. Therefore, it is the purpose of this review to investigate the acute effects that the duration of aerobic exercise exacts on arterial compliance as measured by PWA and PWV.

Cardiovascular Disease Risk Factors Related to Arterial Stiffness

Arterial stiffness is a condition that is associated with advancement of age and cardiovascular diseases such as atherosclerosis, myocardial infarction, and stroke (O'Rourke et al., 2002; Ziemen et al., 2005). Moreover, two of the most significant causes of death in the developed nations include myocardial infarction and stroke (Laurent, 2001). Arterial stiffness is a developing problem connected with advanced risk of cardiovascular disease occurrence, dementia and loss of life (Zoungas, & Asmar, 2007). Reduced compliance of the core vasculature varies the arterial pressure and the flow patterns and affects cardiac functioning and coronary perfusion (Covic, Gusbeth-Tatomir & Goldsmith, 2005). Patients with end stage renal diseases managed by persistent dialysis have remarkable mortality where more than 50% of this mortality is related to cardiovascular disease (Foley, Parfrey & Sarnak, 1998). In spite of stratification for sex, race and the incidence of diabetes, cardiovascular mortality is 10-30 times greater in dialysis patients when measured up to wide-ranging population (Foley et al., 1998).

An elevation in arterial stiffness can lead to advanced risk of stroke via a number of mechanisms. A commonly known mechanistic view is that an increase in arterial stiffness leads to early return of reproduced waves in late systole, advancing core pulsing pressure, and systolic blood pressure (Covic et al., 2007). Additionally, increased indices of arterial stiffness are associated with increases in intima-media thickness and the development of stenosis and plaques

(Popele et al., 2001). It has also demonstrated that arterial stiffness separately plays a significant role in aggravating recurring kidney disease progression (Zieman et al., 2005).

Hypertension is a strong risk factor for cardiovascular diseases in addition to coronary heart disease. Approximately 50 million people have high blood pressure, marked as an intensity measured up to or more than 140 mmHg systolic pressure or 90 mmHg diastolic pressures (Anderson, Odell, Wilson, & Kannel, 1991). Primary epidemiologic information established that obesity is a significant risk factor for coronary heart disease. Successive research suggests that obesity is not a significant risk factor, but somewhat acts indirectly via increase in blood pressure and cholesterol. More research with extensive patient follow up has established that obesity is a significant risk factor that acts autonomously through the other risk factors (World Health Organization, 2000).

A number of studies have shown that individuals living a less engaging lifestyle are at high risk of contracting coronary heart disease (Arsenault et al., 2010). This is associated with exercises and considered one of the four significant changeable risk factors for coronary heart disease. For this reason, there are a number of ongoing suggestions for engaging in active lifestyles and recognition of its significance not only to fitness but also further in the deterrence of diseases. From the above-mentioned risk factors, it is clear that exercising helps in the reduction of arterial stiffness, a developing problem that is as a result of advanced cardiovascular occurrence.

Effect of Aerobic Exercise on Arterial Stiffness

Aerobic exercise has been shown to induce benefits to arterial compliance following as little as a single bout of exercise. Several research studies have demonstrated that aerobic exercise decreases cardiovascular risk factors and have associated aerobic exercise with

decreased arterial stiffness and mortality rates attributable to cardiovascular disease (Tanaka, et al., 2000; Edwards, Schofield, Magyari, Nichols & Braith, 2004). Changing vascular structural and performing factors can effectively reduce arterial stiffness. According to Seals et al. (2000), long duration of aerobic exercise can decrease or lead to improvement from structural disintegration of the vascular walls, which is connected to aging, and therefore decrease arterial stiffness.

Following a 30 min bout of moderate intensity cycling aerobic exercise (65% of VO2max) arterial compliance was shown to increase while decreasing peripheral resistance in a sample of sedentary male participants (Kingwell et al., 1997). In this study, both cPWV and pPWV were demonstrated to decrease 30 min following exercise before returning to resting levels within an hour. A graded exercise protocol on a cycle ergometer which terminated was upon volitional fatigue demonstrated a reduction in pulse wave velocity in both sedentary and resistance trained males (Heffernan et al., 2007). In this study, both Aix and pPWV was shown to significantly decrease in both trained and untrained groups.

In contrast to these studies, an acute bout of moderate intensity (60% VO2max) aerobic exercise lasting for duration of one hour induced no significant effects on arterial compliance (McClean et al., 2010). However, arterial stiffness was only assessed immediately following exercise, and thus did not allow for assessment of several time intervals following exercise. Long duration aerobic exercise, such as a marathon race, has been demonstrated to cause decreases to arterial compliance (Phillips et al., 2012). Phillips (2012) showed that long durations of aerobic exercise causes a significant decrease in pPWV, yet did not cause a significant change to cPWV. Decrease in arterial stiffness subsequent to a long duration of aerobic exercises could be preventive of cardiovascular risk factors. Furthermore, in a study by Naka et al. (2003)

following a maximal aerobic exercise protocol (Bruce Protocol) on a treadmill, 25 normal male subjects were shown to decrease in arterial distensibility as assessed by pulse wave velocity. This study reported a decrease in both aortofemoral (central) and lower limb (peripheral) PWV 30 min post exercise. As in other investigations of exercise on arterial compliance, Naka et al. (2003) reported that arterial compliance indices returned to baseline levels within an hour following exercise.

Conversely, resistance exercise programs have put emphasis on development of musculoskeletal performance rather than cardiovascular function. Resistance exercises have been valuable on protection of performance capability and on cardiovascular risk factors (Braith & Stewart, 2006). That is, moderate intensity resistance exercise in tandem with aerobic exercise is recommended to aide in the maintenance of healthy blood pressure. Resistance exercise combined with aerobic exercise is not demonstrated to increase arterial stiffness. The American Heart Association recommends that resistance exercises be integrated in exercise programs for avoiding and managing cardiovascular disease (Haskell, Lee & Pate et al., 2007). On the other hand, there is inadequate evidence to illustrate a preventive effect of resistance exercise on cardiovascular diseases as weighed against 20 min, 40 min or longer durations of aerobic exercises. In addition, a number of studies have analyzed the relationship between resistance exercises and arterial stiffness where the outcomes are conflicting. There are studies that have demonstrated that resistance exercise increases arterial stiffness (Miyachi, Kawano & Sugawara et al., 2004). In this study a intervention group under went a resistance exercise program for 4 months resulting in a 19% reduction in arterial elasticity. Additionally, arterial compliance was reduced in young men as a result of resistance exercises in comparison with inactive men in similar age group (Kawano, Tanaka & Miyachi, 2006).

Even though the mechanism responsible for increases in arterial stiffness subsequent to resistance exercises is not known, there exist probable justifications founded on preceding research. The variance in arterial stiffness after resistance exercises and short versus long duration of exercises are established to have originated from variance in blood pressure during the time of the exercises. Due to the fact that exercises significantly apply pressure to large muscles in a distinctive pattern, variances in blood pressure are mild in times of aerobic exercises. Conversely, resistance exercise elevates blood pressure as high as 310/250 mmHg in times of exercises (Tanaka et al., 2006). Long-term resistance exercises leads to added content of smooth muscle cells on the vascular walls and variances in the loading bearing features of elastin and collagen. This can lead to a pathophysiologic function where resistance exercises intensify arterial stiffness.

Conclusions

In conclusion, it is recognized that arterial stiffness acutely reduces following a single bout of aerobic exercises. Still, many inconsistencies remain in the literature. McClean et al. (2010) observed no significant effect following an exercise protocol lasting for duration of 60 min. However, this study only assessed arterial elasticity immediately post exercise, and did not continue to monitor arterial compliance for a period following exercise. It might be possible that significant changes in the elastic properties might have been seen had their protocol called for further assessment of arterial compliance following exercise treatment. However, single bouts of exercise can be efficacious in acutely reduce arterial stiffness. Increased arterial compliance was demonstrated to occur following in long duration aerobic exercise such as participation in marathon events lasting several hours (Philips et al., 2012). In this study, PWV of both the limbs of the upper and lower body were shown to significantly decrease as a result of participation in

an ultramarathon. However, central PWV was not shown to significantly change following the ultramarathon. No study has been conducted investigating the acute effects of various aerobic exercise durations on arterial stiffness. The hypotheses of this study were that 1) a 30 min duration of aerobic exercise would induce greater increases in large and small arterial elasticity than durations of 45 min and 60 min, and 2) aerobic exercise duration of 30 min would yield greater decreases in cPWV, pPWV, and AIx in contrast to the longer exercise durations of 45 min and 60 min, and 3) Differences in hemodynamics including systolic (SBP) and diastolic blood pressure (DBP), mean arterial pressure (MAP), cardiac ejection time (CET), cardiac output (CO), and stroke volume (SV), systemic vascular resistance (SVR), and total vascular impedance (TVI) would be induced as a result of different durations of aerobic exercise. Therefore, it was the purpose of this study to examine the responses of large and small arterial stiffness, Aix, cPWV, pPWV, SBP, DBP, MAP, CO, SV, SVR, and TVI resulting from various aerobic exercise durations. The data of this prospective study might yield valuable information regarding the most effective duration on aerobic exercise for populations with or at risk of cardiovascular diseases. The results of this study might provide insightful data that can be useful for exercise prescription of aging populations as well as for individuals with symptoms of cardiovascular diseases that might benefit from exercise method that reverse arterial dysfunction.

CHAPTER III

METHODS

Subjects

Twenty healthy male subjects between the ages of 18 and 40 were recruited for this research study. All subjects were part of a within subject design. The University of Texas-Brownsville Institutional Review Board approved the study procedure for Human Subjects.

Inclusion Criteria

1) Individuals with no signs or symptoms of cardiovascular disease.
2) Individuals younger than 18 and older than 40.
3) Individuals without joint or muscle problems.
4) Individuals without symptoms of respiratory disease, and diabetes.
5) Individuals not taking medication that may interfere with vascular function.
6) Individuals without chronic back pain were excluded from the study.

Exclusion Criteria

1) Individuals with signs or symptoms of cardiovascular disease were not permitted to participate in the study.
2) Individuals younger than 18 and older than 40 were not permitted to participate in the study.
3) Individuals with joint or muscle problems were excluded from the study.
4) Individuals afflicted with symptoms of respiratory disease, and diabetes were excluded from participation.
5) Individuals taking medication that may interfere with vascular function were excluded.

6) Individuals with chronic back pain were excluded from the study.

Recruitment

Participants were recruited from amongst the local population and campus community of the University of Texas at Brownsville by word of mouth and via flyers.

Experimental Protocol

On the first day, the participants filled out questionnaires and were familiarized with the study procedures before starting the exercise sessions. Following initial screening (PAR-Q and health questionnaire) and familiarization, anthropometric measurements including resting heart rate (RHR), blood pressure (BP), height, and weight were performed. Weight and body fat percentage were measured using the Tanita Body Composition Analyzer. Once body anthropometric and body composition measurements have been taken, subjects performed a VO2max test on a treadmill. Testing consisted of running on a treadmill, with increasing speeds and incline, until exhaustion. Subjects were fitted with a mask for the MOXUS metabolic cart. Gas exchange, heart rate were monitored continuously by the metabolic cart machine while performing VO2max testing and endurance exercise on a treadmill. Participants arrived at the lab fasted for a minimum of eight hours on three separate days to complete the testing sessions.

Participants were required to show up to the laboratory at least 8-hour fasted during the three separate randomized sessions. The subjects were instructed to lie down in the supine position on a bed for a minimum of 10 min. Participants were then asked to lift their shirts and then three small areas of their chests shaved and wiped with alcohol just below the supra-sternal notch, over their sternum, and over their left ribcage. ECG electrodes were then applied over each of the shaved areas and attached to the leads. The carotid, femoral, and pedal arteries of the

participants were palpated, and marked with a felt marker. The distance from the supra-sternal notch to each of the marked arteries were measured and recorded.

A wrist stabilizer was strapped to the right arm of the participants. The radial artery was then palpated and marked. Baseline indices of arterial elasticity and hemodynamics were then measured via PWA. PWA was then assessed using the SphygmoCor^{TM} tonometer at the radial artery. Central PWV (cPWV) was then measured by placing the tonometer at the carotid artery (site A) directly on top of the skin and recording waveforms for a minimum of 11 seconds. The participants were then asked to bend their right knee and rest it laterally. The tonometer was then placed at the femoral artery (site B) and pressure waveforms were recorded for a minimum of 11 seconds. Peripheral PWV (pPWV) was then measured by recording waveforms at the femoral artery (site A). Participants were then asked to extend their right leg, and the tonometer was then placed at the pedal artery (site B) and waveforms were recorded.

Testing sessions consisted of running on a treadmill at a moderately intense speed (65% VO_2max). Conditions were randomized into three session, 30 min, 45 min and 60 min of aerobic running on a treadmill. Upon completion of exercise, participants were instructed to lie back down and post exercise arterial compliance, hemodynamics, PWA and PWV were then assessed immediately post, 10 min, 20 min, 30 min, and 40 min following the same procedure described earlier. Calibration of all the equipment was performed regularly according to instructions provided by the manufacturer.

Instrumentation

Moxus VO_2 Metabolic Cart

Participants were required to wear a breathing mask that was connected to the metabolic cart through a breathing tube. The metabolic cart computer collected inspired oxygen and expired carbon dioxide. The software analyzed and performed calculations of energy expenditure and oxygen consumption. Heart rate was assessed continuously by wearing a Polar Heart Rate Monitor E600 series and recorded in the data collection sheet.

Pulse Wave Analysis

Analysis of arterial stiffness was measured noninvasively through applanation tonometry using Pulse Wave Analysis using HDL/PulseWave CR-2000TM Research Cardio Vascular Profiling System (Hypertension Diagnostic, Inc. Eagan, MN, USA)

Pulse Wave Velocity

Assessment of arterial elasticity through measurements of pulse wave velocity, and AIx were performed using SphygmoCor® CVMS, Software Version: 9.0. Copyright © 2011 AtCor Medical Pty. Ltd., Sydney Australia.

Statistical Analysis

A 2-way analysis of variance (ANOVA) with repeated measures were used to determine if significant differences existed in large and small arterial elasticity, cPWV, pPWV, AIx, SBP, DBP, MAP, PP, CET, CO, SV, TVI, and SVR. Differences in physiological response to different durations of aerobic exercise were analyzed using least significant difference test. An alpha of 0.05 was used to determine statistical significance and data was analyzed using SPSS 22.0 for Windows.

CHAPTER IV

RESULTS

The purposes of this study was to 1) examine the acute effects of different aerobic exercise durations on large and small arterial compliance 2) to examine the acute effects of different aerobic exercise durations on cPWV, pPWV, and AIx, and 3) to examine the acute effects of aerobic exercise durations on hemodynamics by measuring resting heart rate (RHR), blood pressure, mean arterial pressure (MAP), cardiac output (CO), cardiac ejection time (CET), stroke volume (SV) systemic vascular resistance (SVR) and total vascular impedance (TVI).

Subject Characteristics

This study consisted of eighteen recreationally active male (age= 232.4) (2.0) participants. Table 1 displays anthropometric measurements of the research sample. Subjects were recruited from among the campus community of University of Texas at Brownsville and surrounding community via flyers and word of mouth.

Table 1. Anthropometric Data

Variables	(N=18)
Age (yr)	23.4 (2.0)
Height (cm)	175.7 (1.9)
Mass (kg)	82.9 (2.9)
RHR (bpm)	56.7 (8.3)
SBP (mmHg)	122 (2.9)
DBP (mmHg)	61.6 (3.4)

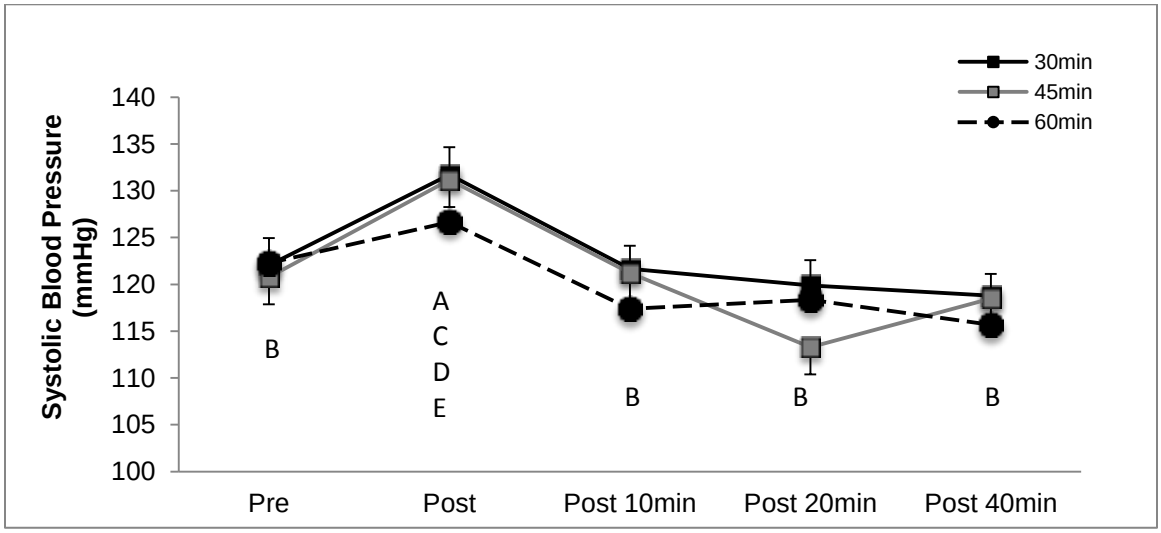
Values Reported as means (SD)

Hemodynamic Response

Systolic Blood Pressure (SBP)

No condition main effect or condition*time interaction was seen in SBP following aerobic exercise treatments. However, a follow up analysis indicated that a significant time effect was seen between pre and post exercise ($p < 0.05$). Immediately Post was shown to be significantly greater than all of the other time points (Figure 1).

Figure 1. Systolic Blood Pressure Response

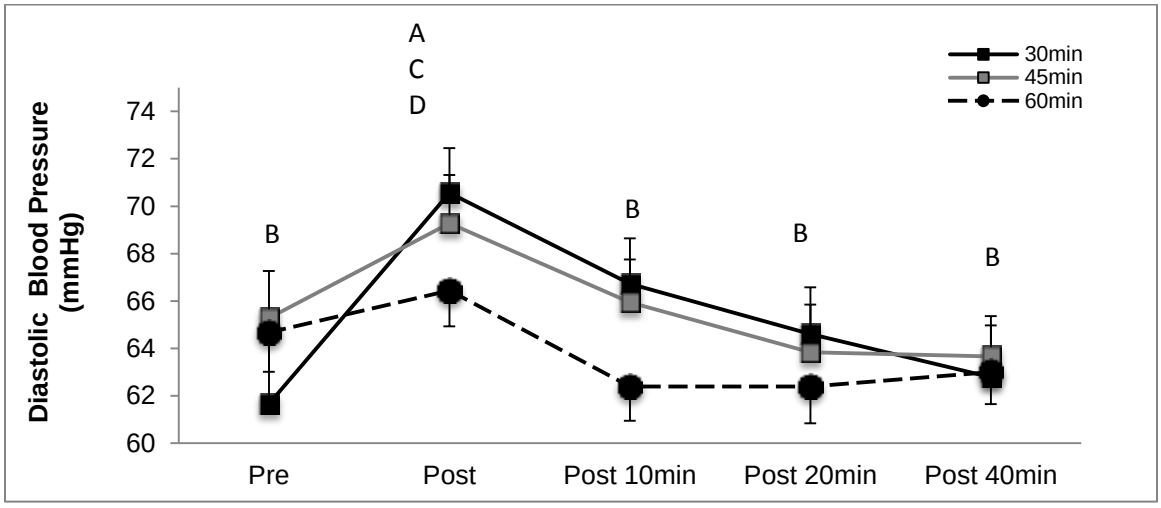


Systolic Blood Pressure values before and after exercise. [A] Represents time significant difference ($P<0.05$) from pre. [B] Represents significant time difference ($P<0.05$) from post. [C] Represents significant difference ($P<0.05$) from post 10 min. [D] Represents significant time difference ($P<0.05$) from post 20 min. [E] Represents significant time difference from post 40 min. Values reported as Mean ± SE.

Diastolic Blood Pressure (DBP)

No condition main effect or condition*time interaction was seen in DBP following aerobic exercise treatments. However, a follow up analysis demonstrated a significant time effect between pre and immediately post exercise ($p < 0.05$). Immediately Post was shown to be significantly greater than all of the other time points (Figure 2).

Figure 2. Diastolic Blood Pressure Response

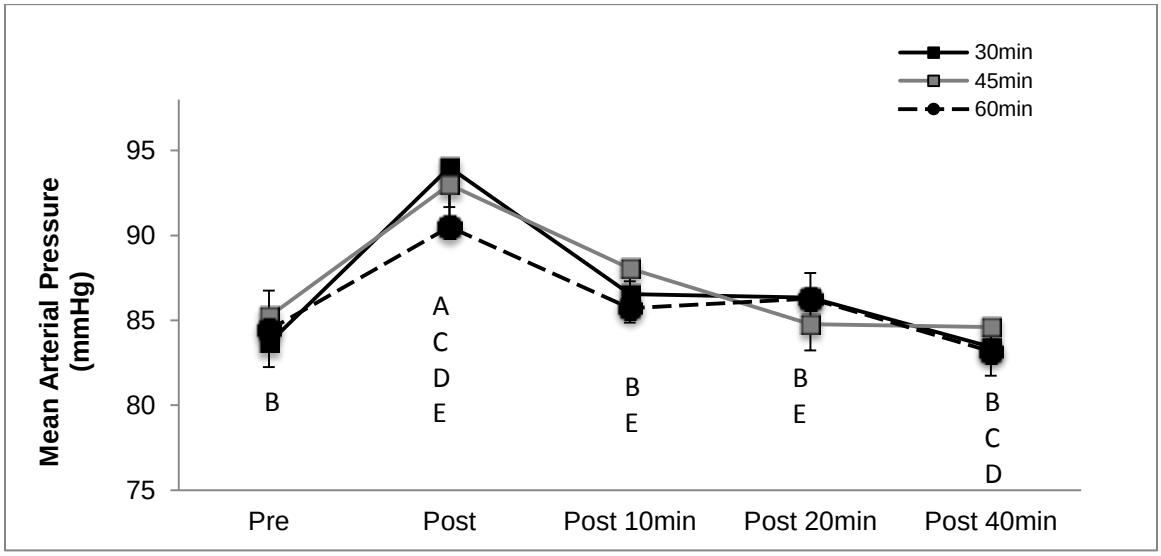


Diastolic Blood Pressure values before and after exercise. [A] Represents significant time difference ($P<0.05$) from pre. [B] Represents significant time difference ($P<0.05$) from post. [C] Represents significant time difference ($P<0.05$) from post 10 min. [D] Represents significant time difference ($P<0.05$) from post 20 min. [E] Represents significant time difference from post 40 min. Values reported as Mean ± SE.

Mean Arterial Pressure (MAP)

For MAP, no condition main effect was detected in response to different aerobic exercise durations. However, a follow up analysis demonstrated a significant time effect between pre and immediately post exercise ($p < 0.05$). Immediately Post was shown to be significantly greater than all of the other time points. 40 min post exercise was shown to have time difference between post, 10 min and 2 min post (Figure 3).

Figure 3. Mean Arterial Pressure Response

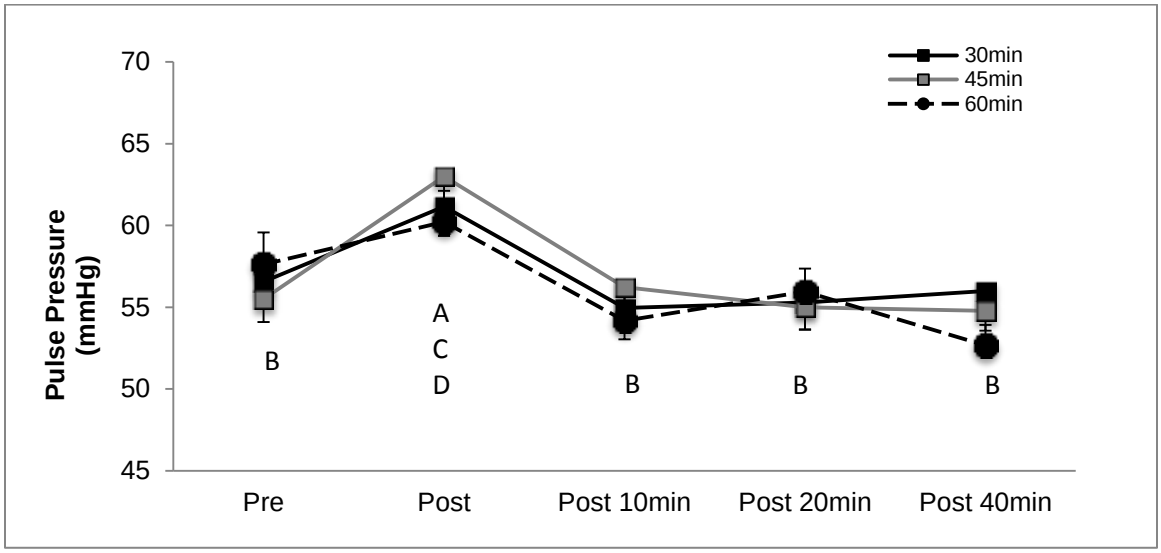


Mean Arterial Pressure values before and after exercise. [A] Represents significant time difference ($P<0.05$) from pre. [B] Represents significant time difference ($P<0.05$) from post. [C] Represents significant time difference ($P<0.05$) from post 10 min. [D] Represents significant time difference ($P<0.05$) from post 20 min. [E] Represents significant time difference from post 40 min. Values reported as Mean ± SE.

Pulse Pressure (PP)

No condition main effect or condition*time interaction was seen in PP following aerobic exercise treatments. However, a follow up analysis demonstrated a significant time effect between pre and immediately post exercise ($p < 0.05$). Immediately Post was shown to be significantly greater than all of the other time points (Figure 4).

Figure 4. Pulse Pressure

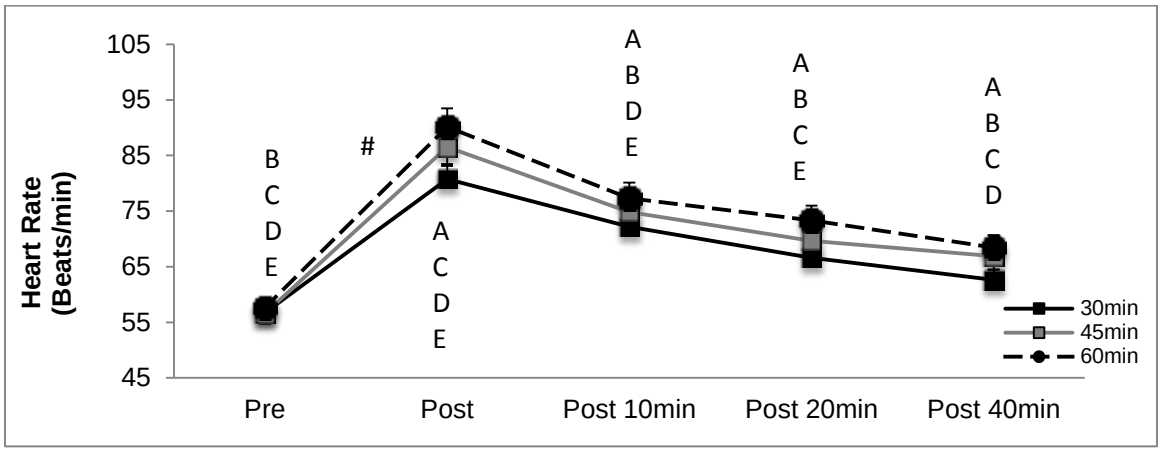


Pulse Pressure values before and after exercise. [A] Represents significant time difference ($P<0.05$) from pre. [B] Represents significant time difference ($P<0.05$) from post. [C] Represents significant time difference ($P<0.05$) from post 10 min. [D] Represents significant time difference ($P<0.05$) from post 20 min. [E] Represents significant time difference from post 40 min. Values reported as Mean ± SE.

Heart Rate (HR)

For HR a condition main effect was seen indicating an increase in heart rate occurred between the 30min and 60 min aerobic exercise conditions ($p < 0.05$). No condition*time interaction was detected. Follow up analysis demonstrate significant time changes between all time points (Figure 5).

Figure 5. Heart Rate Response

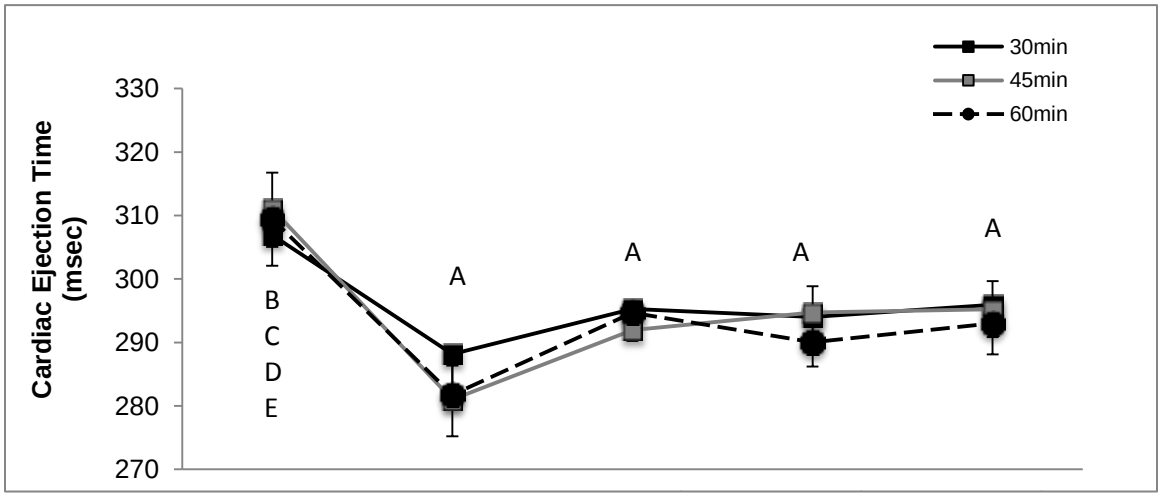


Heart Rate values before and after exercise. # Indicates a significant condition main effect ($p<0.05$). A Represents significant time difference ($P<0.05$) from pre. B Represents significant time difference ($P<0.05$) from post. C Represents significant time difference ($P<0.05$) from post 10 min. D Represents significant time difference ($P<0.05$) from post 20 min. E Represents significant time difference from post 40 min. Values reported as Mean ± SE.

Cardiac Ejection Time (CET)

No condition main effect, or condition*time interaction was seen in CET following aerobic exercise treatments. However, a follow up analysis demonstrated a significant time effect between pre and immediately post exercise ($p < 0.05$). Pre exercise CET was demonstrated to be significantly greater than all four of the post exercise time points (Figure 6).

Figure 6. Cardiac Ejection Time

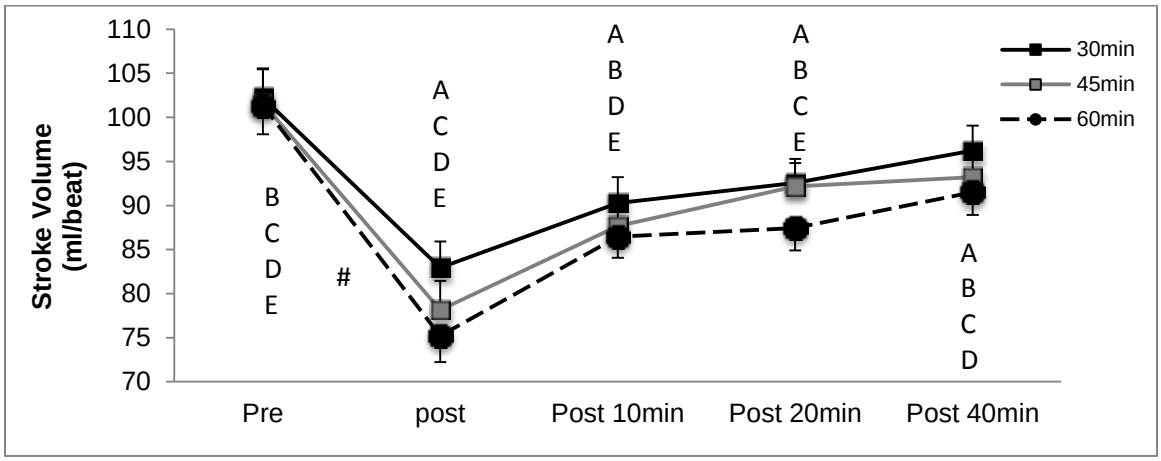


Cardiac Ejection Time values before and after exercise. [A] Represents significant time difference ($P<0.05$) from pre. [B] Represents significant time difference ($P<0.05$) from post. [C] Represents significant time difference ($P<0.05$) from post 10 min. [D] Represents significant time difference ($P<0.05$) from post 20 min. [E] Represents significant time difference from post 40 min. Values reported as Mean ± SE.

Stroke Volume (SV)

For SV a condition main effect was observed indicating a greater decease occurring between the 30min and 60 min aerobic exercise conditions ($p < 0.05$). No condition*time interaction was detected. Follow up analysis demonstrated significant time changes between all time points (Figure 7).

Figure 7. Stroke Volume

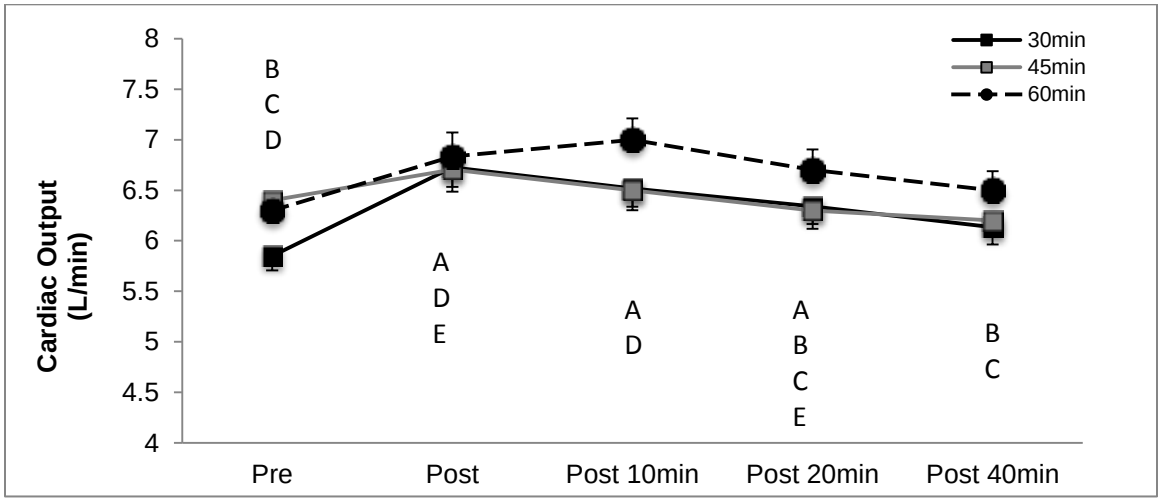


Stroke Volume values before and after exercise. # indicates a significant condition main effect ($p<0.05$). A Represents significant time difference ($P<0.05$) from pre. B Represents significant time difference ($P<0.05$) from post. C Represents significant time difference ($P<0.05$) from post 10 min. D Represents significant time difference ($P<0.05$) from post 20 min. E Represents significant time difference from post 40 min. Values reported as Mean ± SE.

Cardiac Output (CO)

No condition main effect or condition*time interaction was seen in CO following aerobic exercise treatments. However, a follow up analysis demonstrated a significant time effect between pre and immediately post, 10 min and 20 min post exercise ($p < 0.05$) (Figure 8).

Figure 8. Cardiac Output

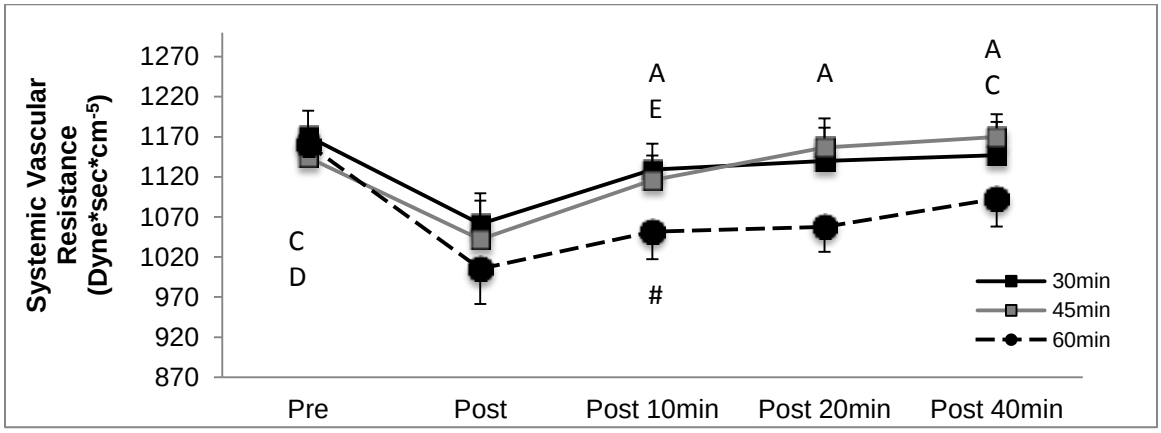


Cardiac Output values before and after exercise. [A] Represents significant time difference ($P<0.05$) from pre. [B] Represents significant time difference ($P<0.05$) from post. [C] Represents significant time difference ($P<0.05$) from post 10 min. [D] Represents significant time difference ($P<0.05$) from post 20 min. [E] Represents significant time difference from post 40 min. Values reported as Mean ± SE.

Systemic Vascular Resistance (SVR)

For SVR a condition main effect was seen indicating a greater decease occurred between the 60 min aerobic exercise conditions and both of the 45 and 30 min conditions ($p< 0.05$) 10 min post exercise. A significant decrease in SVR ($p<0.05$) was seen resulting from the 60 min condition in comparison to the 30 min condition 20 min post exercise. No condition*time interaction was detected. Follow up analysis demonstrate significant time changes between pre exercise and 10 min, 20 min and 40 min measurements (Figure 9).

Figure 9. Systemic Vascular Resistance

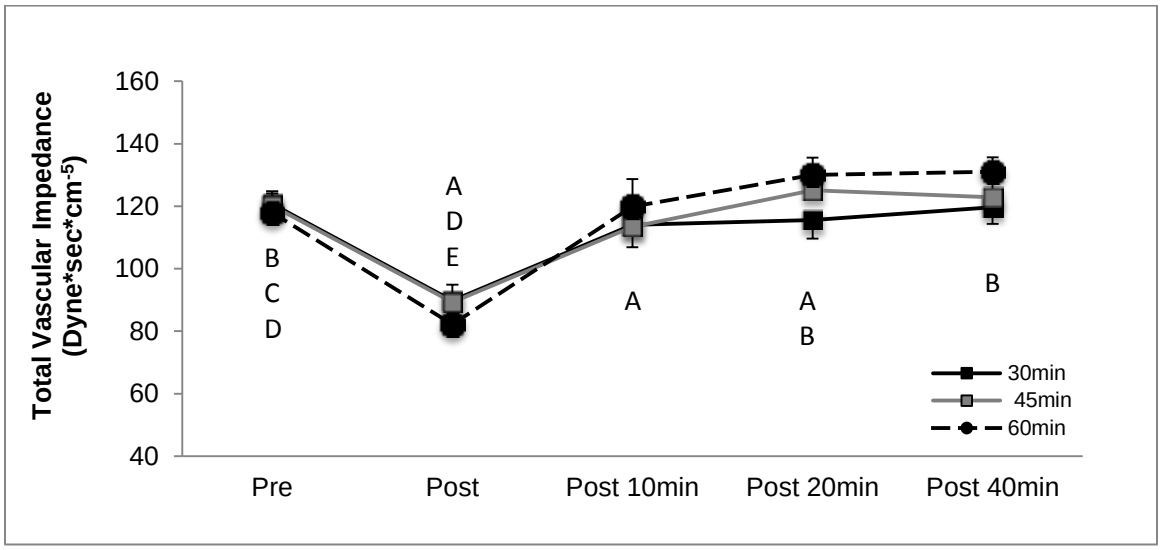


Systemic Vascular Resistance values before and after exercise. # indicates a significant condition main effect ($p<0.05$). A Represents significant time difference ($P<0.05$) from pre. C Represents significant time difference ($P<0.05$) from post 10 min. D Represents significant time difference ($P<0.05$) from post 20 min. E Represents significant time difference from post 40 min. Values reported as Mean ± SE.

Total Vascular Impedance (TVI)

No condition main effect or condition*time interaction was seen in TVI following aerobic exercise treatment. However, a follow up analysis demonstrated a significant time effect between pre and post, 10 min and 20 min post exercise. Immediately post exercise was shown to a have a significant time change between 20 min and 40 min post exercise (Figure 10).

Figure 10. Total Vascular Impedance



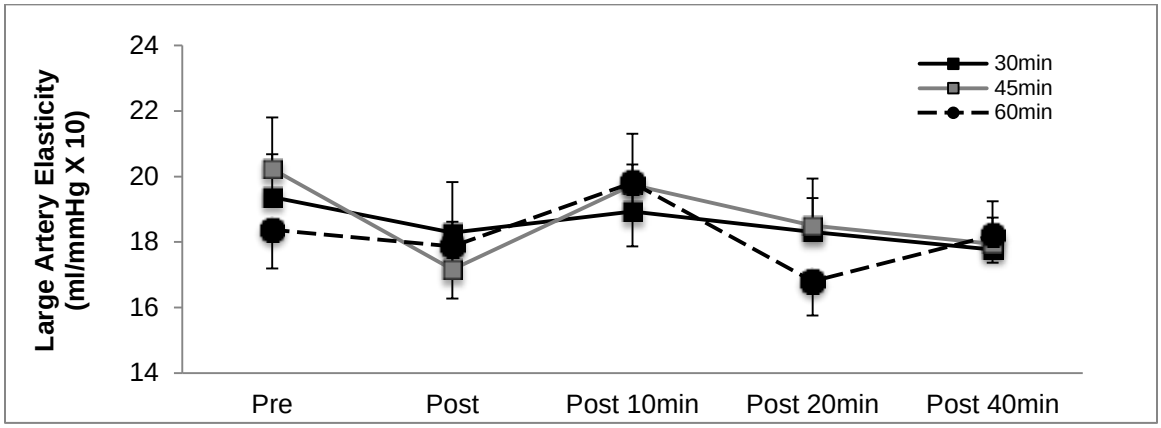
Total Vascular Impedance values before and after exercise. [A] Represents significant time difference ($P<0.05$) from pre exercise. [B] Represents significant time difference ($P<0.05$) from post. [C] Represents significant time difference ($P<0.05$) from post 10 min. [D] Represents significant time difference ($P<0.05$) from post 20 min. [E] Represents significant time difference from post 40 min. Values reported as Mean ± SE.

Arterial Elasticity Response

Large Arterial Elasticity (LAE)

For LAE, there was no significant condition* time interaction and no significant main effect for condition or time (Figure 11).

Figure 11. Large Arterial Elasticity Index

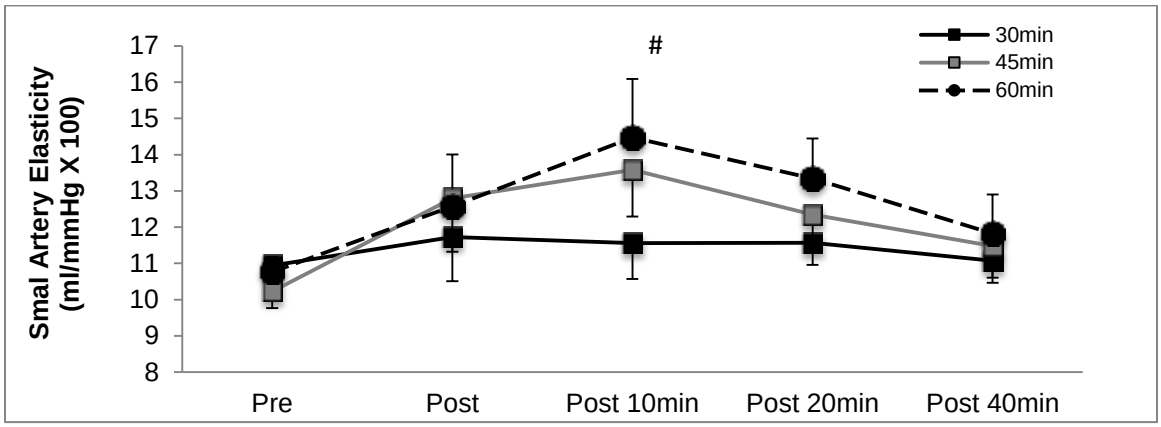


Large Arterial Elasticity values before and after exercise. No significant time or condition main effect shown. Values reported as mean ± SE.

Small Arterial Elasticity (SAE)

There was a condition main effect for SAE (p= 0.041) indicating that the 60 min condition provoked a significant increase in small arterial elasticity in contrast to the 30 min condition. No condition*time interaction was detected (Figure 12).

Figure 12. Small Arterial Elasticity Index

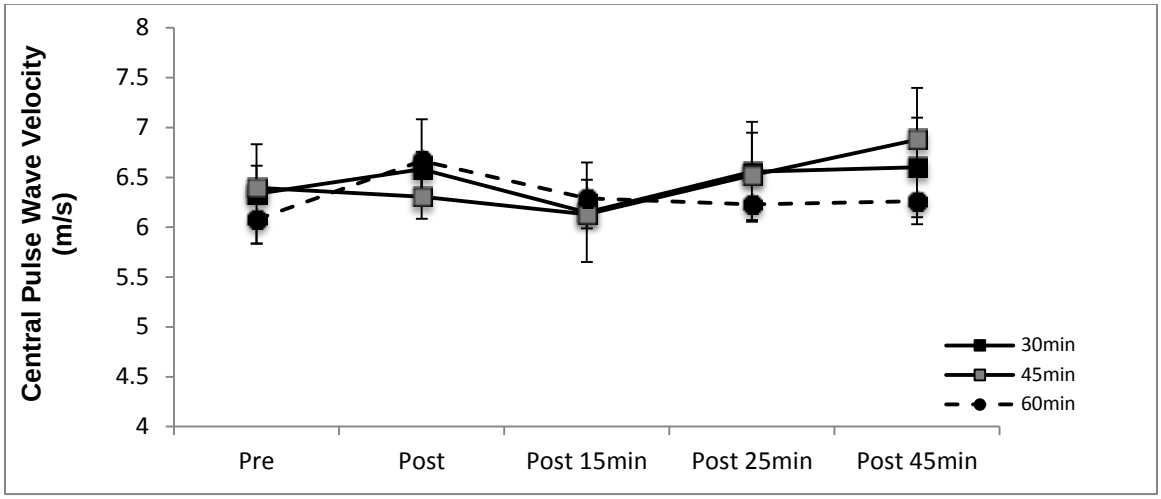


Small Arterial Elasticity values before and after exercise. # Indicates a significant difference was seen for main condition effect (p<0.041). Values reported as Mean ± SE.

Central Pulse Wave Velocity (cPWV)

For cPWV, there was no significant condition* time interaction and no significant main effect for condition or time (Figure 13).

Figure 13. Central Pulse Wave Velocity

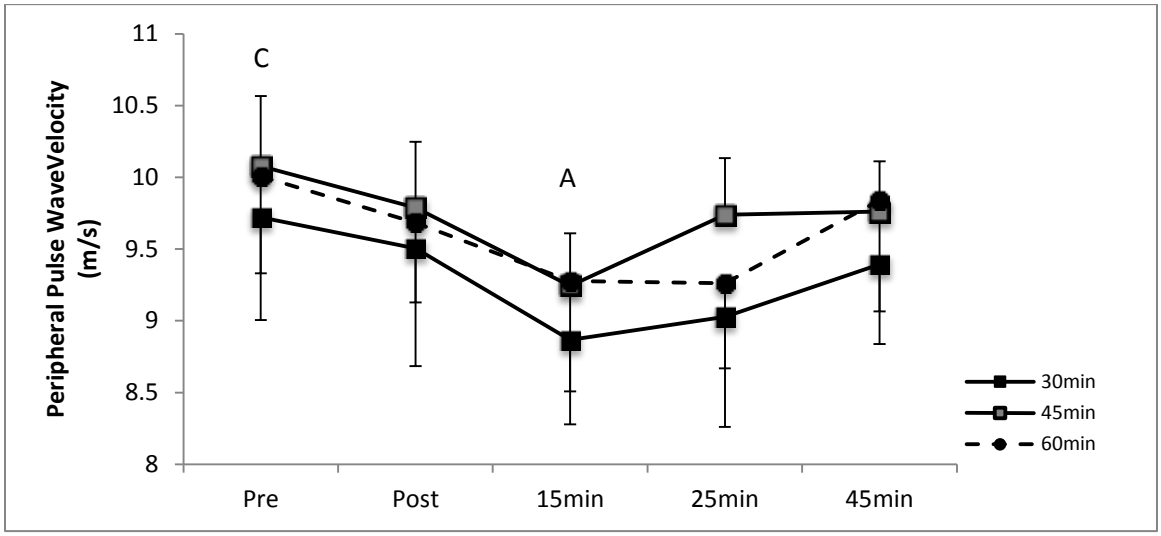


Central Pulse Wave Velocity values before and after exercise. No significant condition*time interaction, time, or condition, main effect found. Values reported as mean ± SE.

Peripheral Pulse Wave Velocity (pPWV)

For LAE, there was no significant condition* time interaction and no significant main effect for condition (Figure 11). Follow up analysis demonstrated a significant time change between pre exercise and 15 min post exercise (Figure 14).

Figure 14. Peripheral Pulse Wave Velocity

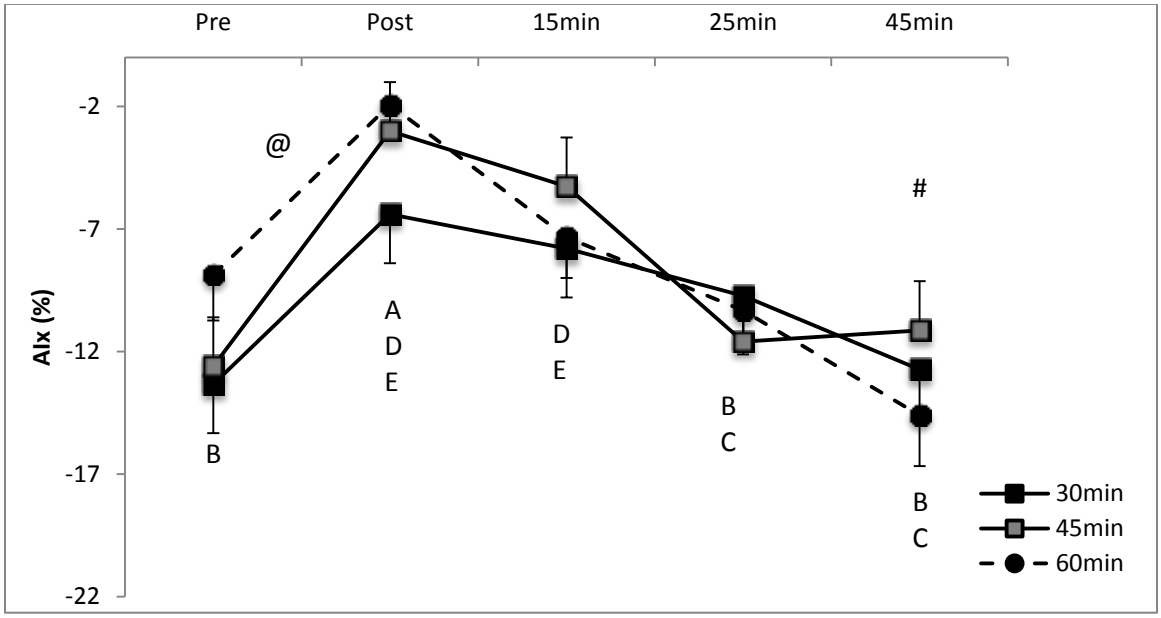


Peripheral Pulse Wave Velocity values before and following exercise. [A] Represents significant time difference (P<0.05) from pre. [C] Represents significant time difference (P<0.05) from post 10 min. Values reported as Mean ± SE.

Augmentation Index (AIx)

For AIx, a condition* time interaction was observed following the 60 min condition ($p<0.05$). A significant condition main effect ($p=0.04$) was demonstrated between the 60 and 45 min conditions 45 min post exercise. A significant time effect was seen between immediately post exercise and pre, 25 min, ad 45 min conditions. Due to unreliable data points, three participants had to be excluded from this variable (N= 15) (Figure 15).

Figure 15. Augmentation Index



Augmentation Index values before and after exercise. [#] Indicates a condition main effect ($p<0.05$). [@] Indicates a significant condition*time interaction ($p<0.05$). [A] Represents significant time difference ($P<0.05$) from pre. [B] Represents significant time difference ($P<0.05$) from post. [C] Represents significant time difference ($P<0.05$) from post 10 min. [D] Represents significant time difference ($P<0.05$) from post 20 min. [E] Represents significant time difference from post 40 min. Values reported as Mean ± SE.

CHAPTER V

DISCUSSION

The present investigation is the first to examine the effects of different aerobic exercise durations on arterial elasticity in male participants as measured by pulse wave analysis, and pulse wave velocity. This investigation uncovered three main findings. Firstly, this investigation illustrated significant changes for condition suggesting that the aerobic exercise duration of 60 min instigates a larger increase in small arterial elasticity than the 30 min condition. Secondly, the current study showed that a 60 min bout of aerobic exercise caused a greater decrease in AIx over the course of recovery than either the 30 min or 45 min conditions. Thirdly, a 60 min bout of moderate intensity aerobic exercise was demonstrated to significantly lower systemic vascular resistance 10 min post exercise in comparison to the 30 min condition. These findings are significant as they validate the problems presented in chapters I and II that no study has evaluated the effects of different durations of aerobic exercise on arterial compliance in male participants.

Hemodynamic Response

No significant condition changes were found for SBP, DBP, MAP, PP, CET, CO by this study as a consequence of the various aerobic exercise durations. However, a number of time main effects were seen in these measures post exercise. Both SBP and DBP were demonstrated to increase immediately post exercise as a result of all three conditions before beginning to decline gradually during the recovery period. In a study by Brownley et al. (1996), aerobic exercise was shown to decrease blood pressure following aerobic exercise in hypertensive individuals, and found no reduction in blood pressure amongst a group of normotensive subjects.

This finding was also consistent with other studies that have demonstrated reduction in blood pressure in hypertensive subjects, while finding no attenuation in blood pressure in normotensive individuals (Quinn, 2000; Wallace et al., 1997; and Hara et al., 1995) indicating that the physical condition and health status affect the hemodynamic response to aerobic exercise. It is important to note that the participants of the present study were all normotensive, active males. It is therefore possible that significant changes in hemodynamic response were blunted due to health status of the participants. Also, the current study only monitored the hemodynamic response for 40 min into recovery, which differs from the 24-hour blood pressure observation employed in the study by Brownley et al. (1996). Further reductions in SBP and DBP may have occurred as sympathetic activation withdraws and parasympathetic activation increased beyond 40 min post exercise.

No condition main effect was seen in MAP. However, MAP was demonstrated to be elevated immediately post exercise before returning to baseline levels by 40 min post exercise. In a study by Pescaletto et al. (1991), blood pressure was monitored hourly for 13 hour following a 30 min bout of cycling exercise at both 40% and 70% of their respective VO2max. In normotensive participants, post exercise MAP levels remained similar to that of baseline measurements. However, measurements of MAP were conducted in hour-long intervals and not immediately following exercise. In the present study, pulse pressure was shown to increase from pre to post exercise with no differences between the duration conditions. There is a lack of information regarding the effects of different durations of aerobic exercise on PP in the literature.

Heart rate was shown to be elevated as a result of all three aerobic exercise conditions. Increased heart rate in response to exercise is a result of an increase in sympathetic activity and a simultaneous decrease in parasympathetic activity (Pierpont et al., 2000). This is consistent with

a study that found a proportional relationship between exercise duration and plasma norepinephrine (Leuenberger et al., 1993). The HR response to aerobic exercise was shown to be significantly greater after exercise following the 60 min condition than the 30 min condition. This may indicate that a greater response of sympathetic activity (greater blood concentrations of epinephrine and norepinephrine) occurred due to longer durations of aerobic exercise. In a study of hormonal and metabolic response to different aerobic exercise intensities, sympathetic and metabolic response to exercise was shown to be directly proportional to the intensity of the exercise (Duester et al., 1989; Terziotti et al., 2001). The increased heart rate following the 60 min condition suggests that prolonged exercise durations increase sympathetic and metabolic stress similar to greater intensities of aerobic exercise. Douglas et al. (2005) attributed the primary cause of body water loss to sweat loss resulting from prolonged or strenuous physical activity. It might be possible that sweat loss over the course of the 60 min condition might have lead to a decrease in plasma volume. Exercise induced reductions in plasma volume cause hormone concentrations in the plasma to increase. Despite all conditions being performed at the same intensity, lower blood volume following the 60 min condition might have resulted in a higher concentration of plasma catecholamines, thereby amplifying the magnitude of the effect of the hormone epinephrine (Kargotich et al., 1997).

Stroke volume was demonstrated to show a greater decrease following the 60 min of aerobic exercise in contrast to the 30 min condition. Stroke volume has been shown to decrease following moderate to high and moderate intensity aerobic exercise, and is associated with increases in heart rate (Gonzalez-Alonso et al., 2000; Fritzsche et al., 1999). As heart rate increases in response to exercise, the less time is available for diastolic filling to occur, thereby causing decreases in end diastolic volume and stroke volume. This is consistent with the present

study showing that post exercise cardiac ejection time also significantly decreased. However, there was no significant difference between the different conditions of aerobic exercise durations for CET.

All three conditions of moderate intensity aerobic exercise employed by the current study elicited a decrease in systemic vascular resistance. This is consistent with a study by Forjaz et al. (2004) where systemic vascular resistance was shown to decline following low to moderate intensities of aerobic exercise for 45 min on a cycle ergometer. The present experiment demonstrated a significant decrease in systemic vascular resistance occurred as a result of the 60 min of aerobic exercise in comparison to the 30 min condition. The decrease in SVR resulting from prolonged aerobic exercise is in line with a study by Halliwill et al. (2000) in which a 60 min aerobic exercise at 60% VO2peak yielded decreased SVR. In a study by Stamler (1994), systemic vascular resistance was attenuated by endothelium-derived nitric oxide (NO), a recognized vasodilatory biochemical responsible for the regulation of vascular tone. The possible increase in NO concentration following aerobic exercise would be consistent with a study by Goto et al. (2003), which reported in increase in NO following moderate intensity aerobic exercise. It might be possible that a 60 min bout of aerobic exercise effectively provokes the release of greater concentrations of NO in contrast to lower durations of aerobic exercise.

No differences in total vascular impedance were observed as a result of the various durations of aerobic exercise used in the present study. Vascular impedance is defined as the opposition to blood flow in a pulsatile system (Morgan and Hosking, 2006). Increased large arterial compliance reduces vascular impedance (Bénétos et al., 1997). The lack in the response of total vascular impedance observed in the present study might be a consequence of the lack in response of large arterial compliance to the different durations of aerobic exercise.

Arterial Elasticity

The hypothesis that greater increases to arterial elasticity would result from 30 min bout of aerobic exercise condition was not supported by the results of the present study. It was demonstrated that the 60 min duration of aerobic exercise, resulted in significant improvements in small arterial compliance. This is in contrast to a study that found an increased arterial compliance response following a 30 min bout of cycling exercise at 65% VO2max (Kingwell et al., 1997). No significant changes occurred in large arterial elasticity as a result of the different aerobic exercise durations assessed in the present study. This is consistent with a study by Nickel (2011) in which a 30 min bout of moderate intensity aerobic exercise on a cycle ergometer failed to elicit changes in large arterial elasticity. The inconsistency between the effects of aerobic exercise on large arterial compliance between the studies by Nickel (2011) and Kingwell (1997) might stem from differences in the samples employed. In the study by Kingwell (1997) the mean sample age was 24 years; in contrast participants in the Nickel (2011) study were required to be over the age of 60. A training effect of aerobic exercise might be necessary to provoke changes large arterial compliance. In a training study of an older population, a 30 min bout of cycling exercise at 65% VO2max three times a week for four weeks was shown to increase resting arterial compliance (Cameron and Dart, 1994). However, no change in large arterial compliance has been exhibited following an 8-week aerobic exercise training protocol in patients with isolated systolic hypertension (Ferrier et al., 2000). Because the sample used in the present study consisted of young normotensive males and assessment of arterial elasticity was measured following a single bout of aerobic exercise, large arterial compliance might react differently to such a protocol in the present population.

A single 60 min bout of moderate intensity aerobic exercise was demonstrated by this study to significantly increase small arterial compliance. Modification of small arterial compliance might be a result of structural or functional changes associated with endothelial dysfunction (Ross, 1993). An increase in NO in response to prolonged aerobic exercise might have been responsible for causing increased small arterial elasticity. It has been shown that the inhibition of endothelial NO selectively attenuates small arterial compliance in contrast to large arterial compliance in human subjects (Gilani et al., 2000). A more prolonged or intense bout of aerobic exercise might be necessary to provoke more pronounced effect on small artery compliance. Aerobic exercise training might effectively elicit benefits to small arterial compliance. Regular participation in aerobic exercise was shown to be associated with reduced medium and large arterial stiffness in male participants of different age groups (Tanaka et al, 2000; Vaitkevicius et al., 1993). In the study conducted by Tanaka (2000), participants performed 25-30 min of aerobic exercise at 60 % of their individually determined heart rate maximum.

The various durations of aerobic exercise employed by the present study were shown to not have an effect on either central and peripheral pulse wave velocity. These findings are in agreement with McClean et al. (2011) who demonstrated no change in PWV following a 60 min bout of aerobic exercise at 60% HRmax on a cycle ergometer. However, this is in contrast to a study by Kingwell et al, (1997) which demonstrated a reduction in cPWV and pPWV occurred following a 30 min bout of moderate intensity cycling protocol. Similarly, Naka et al. (2003) showed that a maximal treadmill exercise bout reduced pPWV about 23% below baseline level 10 min after exercise. Phillips et al. (2012) demonstrated a significant decrease in PWV was seen following participation in a prolonged bout of aerobic exercise such as an ultramarathon

event. A more prolonged bout of aerobic exercise may therefore be necessary to elicit an effect of PWV. Pulse wave analysis is a measure of pulse transit times through large arteries (Kingwell et al., 1997). PWV can be viewed as a measure of LAC (Kingwell et al., 1997; Naka et al., 2003). The lack of any effect on PWV by the different durations of aerobic exercise parallels the absence in change seen in LAC as a result of aerobic exercise in the present study.

AIx was shown to attenuate over the course of post exercise recovery resulting from the 60 min condition. This is consistent with a study by Heffernan et al. (2007) where after increasing immediately following exercise, AIx was shown to reduce by 20 and 30 min post exercise. A 20 min bout of moderate intensity aerobic exercise on a cycle ergometer was not shown to change AIx. However, the 20 min duration might have not been sufficient in eliciting physiological response. Vasoactive drugs, such as NO, affect AIx and therefore vessel diameter and small artery compliance are modified in the presence of elevated NO concentrations (Oliver et al, 2003). Reductions seen in AIx might indicate an increase in vascular tone caused by an influx in NO concentration.

Conclusions

The purposes of this study were to 1) examine the acute effects of different aerobic exercise durations on large and small arterial compliance, 2) to examine the acute effects of different aerobic exercise durations on central pulse wave velocity (cPWV), peripheral pulse wave velocity (pPWV), and augmentation index (AIx), and 3) to examine the acute effects of aerobic exercise durations on hemodynamics by measuring blood pressure, mean arterial pressure (MAP), Pulse Pressure (PP), heart rate (HR), cardiac output (CO), stroke volume (SV) systemic vascular resistance (SVR) and total vascular impedance (TVI). The research questions asked were:

1) What changes in large and small arterial compliance would be induced as a result of different aerobic exercise durations?
2) What changes in central pulse wave velocity (cPWV), peripheral pulse wave velocity (pPWV) and augmentation index (AIx) would be induced as a result of different aerobic exercise durations?
3) What changes in systolic and diastolic blood pressure, mean arterial pressure (MAP), cardiac output (CO), cardiac ejection time (CET) stroke volume (SV), systemic vascular resistance (SVR) and total vascular impedance (TVI) would be induced as a result of different durations of aerobic exercise durations?

Research Hypothesis 1: Aerobic exercise duration of 30 min would yield greater increases in large and small arterial elasticity, in contrast to the longer exercise durations of 45 min and 60 min.

The results of the present study did not support this hypothesis. No significant changes in large arterial elasticity occurred following 30 min, 45 min, or 60 min of aerobic exercise. In contrast to the hypothesis an acute bout of aerobic exercise significantly elicited an increase in small arterial elasticity occurred following the 60 min bout of aerobic exercise 10 min post exercise.

Research Hypothesis 2: Aerobic exercise duration of 30 min would yield greater decreases in cPWV, pPWV, and AIx in contrast to the longer exercise durations of 45 min and 60 min.

The evidence gathered in the present study did not support the hypotheses that cPWV and pPWV would show a greater decrease as a result of the 30 min condition in comparison to the longer durations of 45 min and 60 min. No significant changes were found for either cPWV or pPWV between the three conditions. However, AIx was demonstrated to increase immediately post exercise before decreasing below baseline over the course of recovery following the 60 min aerobic exercise condition, suggesting a decrease in arterial stiffness occurred.

Research Hypothesis 3: Differences in hemodynamics including systolic (SBP) and diastolic blood pressure (DBP), mean arterial pressure (MAP), cardiac ejection time (CET), cardiac output (CO), and stroke volume (SV), systemic vascular resistance (SVR), and total vascular impedance (TVI) would be induced as a result of different durations of aerobic exercise.

No significant changes were seen between the condition s for SBP, DBP, MAP, CO, or TVI. The hypothesis that HR would show greatest increase as a result of the 60 min bout of aerobic exercise was supported by the results. Both SV and SVR were shown to significantly decrease following the 60 min bout of aerobic exercise in comparison to the 30 min aerobic exercise condition.

This is the first study to investigate the effects of different duration of moderate intensity aerobic exercise on large and small arterial compliance, cPWV, pPWV, and AIx, using both CR-2000 and SphygmoCor instruments. This investigation revealed that aerobic exercise duration of 60 min provoked increases to small arterial elasticity in contrast to the 30 min condition. Secondly, this investigation showed that duration of aerobic exercise of 60 min caused a greater decrease in AIx than the 30 min condition. Thirdly, the current study demonstrated that an acute bout of moderate intensity exercise lasting 60 min provoked greater increases in SVR than either 30 or 45 min bouts which suggests a relaxation of vascular tone occurred following longer bouts of aerobic exercise.

We conclude that a long duration (~60 min or more) of moderate intensity aerobic exercise might be necessary to improve arterial elasticity. The results of the present study might prove to be valuable in exercise prescription for individuals afflicted with or at risk for cardiovascular disease. Future studies maybe needed to investigate the effects of various durations of aerobic in combination with various intensities on indices of arterial compliance.

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APPENDICES

Appendix A. Recruitment Flyer
Appendix B. Informed Consent
Appendix C. PAR-Q
Appendix D. IRB Approval Letter
Appendix E. Data Collection Sheets

Appendix A: Recruitment Flyer

UT BROWNSVILLE

PARTICIPANTS NEEDED

CARDIOVASCULAR HEALTH RESEARCH

MALES BETWEEN THE AGES OF 18 AND 40 YEARS OLD

You are invited to participate in a research study at the Health and Human Performance Department at the University of Texas at Brownsville. The purpose of the study is to assess the acute effects of moderate intensity aerobic exercise durations on large and small arterial stiffness, hemodynamics, and vascular resistance. Total time required for completion of the study is 4 visits for a total of about 6 hours.

PLEASE CONTACT:

Joe-Angel Lopez

Tel: 956 579 1678

E-mail: joeangel.lopez@utb.edu

Dr. Murat Karabulut

Tel: 956 882 7236

Email: murat.karabulut@utb.edu

Appendix B. Informed Consent

The University of Texas at Brownsville
Institutional Review Board
Informed Consent to Participate in a Research Study

Project Title: **The Acute Effects of Aerobic Exercise Durations on Arterial Compliance in Recreationally Active Males**

Principal Investigator: Joe-Angel Lopez, Graduate Assistant, Dr. Murat Karabulut
Department: Health and Human Performance

You are being asked to volunteer for this research study. This study is being conducted at the Exercise Physiology laboratory in the Biomedical Research 2. You were selected as a possible participant because of your inquiry into the study.

Please read this form and ask any questions that you may have before agreeing to take part in this study.

Purpose of the Research Study

The purposes of this study are to 1) investigate the acute effects of different aerobic exercise durations on large and small arterial elasticity, and 2) to examine how various aerobic exercise durations elicit changes in hemodynamics by measuring resting heart rate (RHR), blood pressure, mean arterial pressure (MAP), cardiac output (CO), stroke volume (SV), systemic vascular resistance (SVR) and total vascular impedance (TVI) in recreationally active male subjects.

Number of Participants

40 recreationally active male participants will take part in this study.

Procedures

If you agree to be in this study, you will be asked to do the following:

a. You will be required to visit the research labs in the Department of Health and Human Performance on 4 separate days for a total time commitment of about 6 hours.

b. On the first visit (about 60 min), you will be required to read and sign an informed consent, PAR-Q and health status questionnaire before any testing takes place. Participants that answer yes to any PAR-Q question, or have blood pressure higher than 140/90 mmHg will be excluded from this study.

You will then have your height, weight, blood pressure and resting heart rate measured. Weight and body fat percentage will be measured using the Tanita Body Composition Analyzer. Once

body anthropometric and body composition measurements have been taking, you will perform a VO2 Max test on a treadmill. Testing consists of running on a treadmill, with increasing speeds and incline, until exhaustion. You will be fitted with a mask for the metabolic cart, gas exchange, blood pressure and heart rate will be monitored continuously by metabolic cart machine while performing VO2 Max testing and endurance exercise on a treadmill. After completing all the necessary paperwork, descriptive measures, and VO2 Max testing, participants will return on a different day and start the testing sessions with a randomly selected condition.

c. On the next 3 visits (each visit separated by at least 48 hours), participants will perform endurance exercise at a randomly selected condition. Conditions will consist of running on a treadmill for 30 min, 45 min and 60 min. Participants will perform endurance exercise on a treadmill 60-65% VO2 Max. Following aerobic exercise, participants' large and small arterial compliance, RHR, SBP, DBP, MAP, CO, SV, SVR and TVI will be assessed via PWA at 10 min, 20 min, 30 min, and 40 min post-exercise.

Length of Participation

You will be required to visit the research labs in the Department of Health and Human Performance on four separate days for a total time commitment of approximately 6 hours.

This study has the following risks:

The study has the following risks:

You understand there are minimal risks to healthy individuals when performing any of the requirements for this project. However, even though these standard protocols have been approved at numerous other institutions and will be performed by qualified and trained personnel, You should be aware of the following:

Benefits of being in the study are

The benefits to participation are: You can receive information about your anthropometric measures such as height, weight, resting BP and HR. Also, you will obtain information about your cardiovascular health when performing endurance exercise, and arterial health from Pulse Wave Analysis assessment.

Injury

In case of injury or illness resulting from this study, emergency medical services will be contacted. However, you or your insurance company may be expected to pay the usual charge from this treatment. The University of Texas at Brownsville has set no funds to compensate you in the event of injury.

Confidentiality

In published reports, there will be no information included that will make it possible to identify you without your permission. Research records will be stored securely and only approved researchers will have access to the records.

There are organizations that may inspect and/or copy your research records for quality assurance and data analysis. These organizations include Dr. Murat Karabulut and the UTB Institutional Review Board.

Costs

There is no cost for participation.

Compensation

You will not be reimbursed for you time and participation in this study.

Rights

Refusal to participate will involve no penalty or loss of benefits to which you are otherwise entitled. You can discontinue participation at any time without penalty or loss of benefits to which you are otherwise entitled.

Voluntary Nature of the Study

Participation in this study is voluntary. If you decline to participate, you will not be penalized or lose benefits or services unrelated to the study. If you decide to participate, you may decline to answer any question and may choose to withdraw at any time.

Waivers of Elements of Confidentiality

Your name will not be linked with your responses unless you specifically agree to be identified. Please select one of the following options

_____ I consent to being quoted directly.

_____ I do not consent to being quoted directly.

Contacts and Questions

If you have concerns or complaints about the research, the researcher(s) conducting this study can be contacted at the Department of Health and Human Performance: Joe-Angel Lopez, B.S., (956)579-1678, joeangel.lopez10@utb.edu OR Dr. Murat Karabulut, Ph.D., The University of Texas at Brownsville, (956)882-7236, Murat.Karabulut@utb.edu. You are encouraged to contact the researcher if you have any questions. If you have any questions about the right of research subjects, contact the Chairman of the UTB IRB - Human Subjects or the Office of Research at UTB (956) 882-7731.

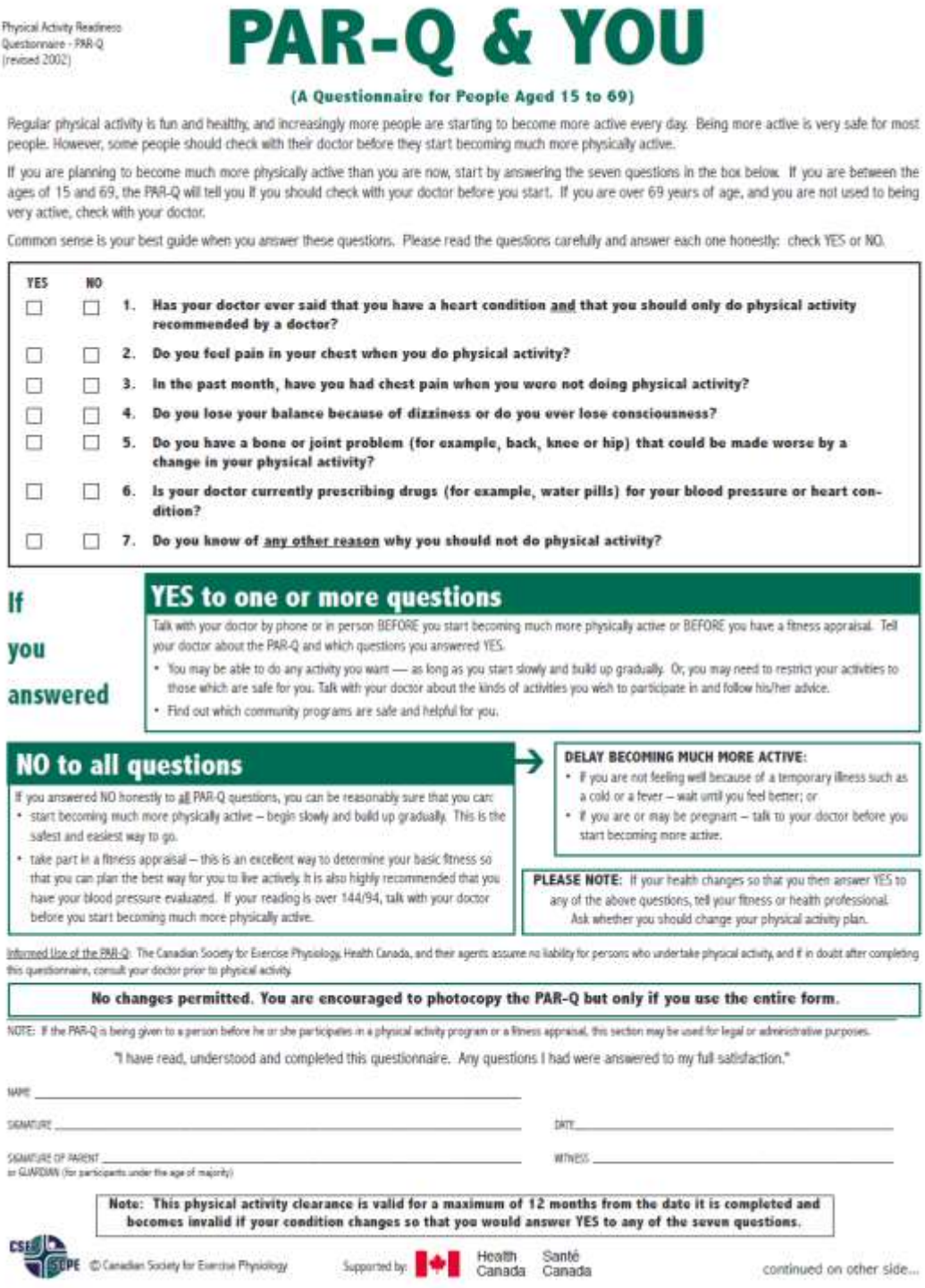
You are voluntarily making a decision whether or not to participate. Your signature indicates that, having read and understood the information provided above, you have decided to participate. You will be given a copy of this information to keep for your records. If you are not given a copy of this consent form, please request one.

Statement of Consent

I have read the above information. I have asked questions and have received satisfactory answers. I consent to participate in the study.

Signature Date

Appendix C. PAR-Q



Physical Activity Readiness Questionnaire - PAR-Q (revised 2002)

PAR-Q & YOU

(A Questionnaire for People Aged 15 to 69)

Regular physical activity is fun and healthy, and increasingly more people are starting to become more active every day. Being more active is very safe for most people. However, some people should check with their doctor before they start becoming much more physically active.

If you are planning to become much more physically active than you are now, start by answering the seven questions in the box below. If you are between the ages of 15 and 69, the PAR-Q will tell you if you should check with your doctor before you start. If you are over 69 years of age, and you are not used to being very active, check with your doctor.

Common sense is your best guide when you answer these questions. Please read the questions carefully and answer each one honestly: check YES or NO.

YES	NO		
☐	☐	1.	**Has your doctor ever said that you have a heart condition and that you should only do physical activity recommended by a doctor?**
☐	☐	2.	**Do you feel pain in your chest when you do physical activity?**
☐	☐	3.	**In the past month, have you had chest pain when you were not doing physical activity?**
☐	☐	4.	**Do you lose your balance because of dizziness or do you ever lose consciousness?**
☐	☐	5.	**Do you have a bone or joint problem (for example, back, knee or hip) that could be made worse by a change in your physical activity?**
☐	☐	6.	**Is your doctor currently prescribing drugs (for example, water pills) for your blood pressure or heart condition?**
☐	☐	7.	**Do you know of any other reason why you should not do physical activity?**

If you answered

YES to one or more questions

Talk with your doctor by phone or in person BEFORE you start becoming much more physically active or BEFORE you have a fitness appraisal. Tell your doctor about the PAR-Q and which questions you answered YES.

- You may be able to do any activity you want — as long as you start slowly and build up gradually. Or, you may need to restrict your activities to those which are safe for you. Talk with your doctor about the kinds of activities you wish to participate in and follow his/her advice.
- Find out which community programs are safe and helpful for you.

NO to all questions

If you answered NO honestly to all PAR-Q questions, you can be reasonably sure that you can:

- start becoming much more physically active – begin slowly and build up gradually. This is the safest and easiest way to go.
- take part in a fitness appraisal – this is an excellent way to determine your basic fitness so that you can plan the best way for you to live actively. It is also highly recommended that you have your blood pressure evaluated. If your reading is over 144/94, talk with your doctor before you start becoming much more physically active.

DELAY BECOMING MUCH MORE ACTIVE:

- If you are not feeling well because of a temporary illness such as a cold or a fever – wait until you feel better; or
- If you are or may be pregnant – talk to your doctor before you start becoming more active.

PLEASE NOTE: If your health changes so that you then answer YES to any of the above questions, tell your fitness or health professional. Ask whether you should change your physical activity plan.

Informed Use of the PAR-Q: The Canadian Society for Exercise Physiology, Health Canada, and their agents assume no liability for persons who undertake physical activity, and if in doubt after completing this questionnaire, consult your doctor prior to physical activity.

No changes permitted. You are encouraged to photocopy the PAR-Q but only if you use the entire form.

NOTE: If the PAR-Q is being given to a person before he or she participates in a physical activity program or a fitness appraisal, this section may be used for legal or administrative purposes.

"I have read, understood and completed this questionnaire. Any questions I had were answered to my full satisfaction."

NAME _____

SIGNATURE _____ DATE _____

SIGNATURE OF PARENT or GUARDIAN (for participants under the age of majority) _____ WITNESS _____

Note: This physical activity clearance is valid for a maximum of 12 months from the date it is completed and becomes invalid if your condition changes so that you would answer YES to any of the seven questions.

Supported by: Health Canada Santé Canada

continued on other side...

Appendix D. IRB Approval Letter

Research Integrity and Compliance
The University of Texas at Brownsville

Matthew Johnson, Ph.D.
IRB Chair

April 23, 2014

Dr. Murat Kasabalut
The University of Texas at Brownsville
One West University Blvd.
Brownsville, Texas 78520
RE: IRB-HS Approval

Study Title: "The Acute Effects of Aerobic Exercise Durations on Arterial Compliance in Recreationally Active Males"

Protocol #: 2014-049-IRB

Dear Dr. Kasabalut,

In accordance with Federal Regulations for review of research protocols, the Institutional Review Board -Human Subjects of The University of Texas at Brownsville has reviewed your study as requested.

Approval Type:
D Full Board Review
[S] Designated Member Review
D Continuing Review
D Change request/Modification/Amendment
D Exempt Category
[S] Expedited Category 4

Approval Period:

Start Date: April 23, 2014

End Date: April 22, 2015

The IRB-HS grants its approval for this project contingent on compliance with the following items. You may make as many copies of the stamped consent form as are necessary for your activity. All consent forms MUST bear the UTB IRB stamp indicating approval.

Responsibilities of the Principal Investigator also include:

- Inform the IRB-HS in writing immediately of any emergent problems or proposed changes.
- Do not proceed with the research until any problems have been resolved and the IRB-HS have reviewed and approved any changes.
- Report any significant findings that become known in the course of the research that might affect the willingness of the subjects to take part.
- Protect the confidentiality of all personally identifiable information collected.
- Submit for review and approval by the IRB-HS all modifications to the protocol or consent form(s) prior to implementation of any change(s).
- Submit an activity/progress report regarding research activities to the IRB-HS on no less than an annual basis or as directed by the IRB-HS through the Continuing Review Form.
- Notify the IRB-HS when study has been completed through submission of a Project Completion Report.

Should you have any questions or need any further information concerning this document please feel free to contact me at (956) 882-8888 or via email at Matthew.Johnson@utb.edu.

Sincerely yours,

Matthew Johnson, Ph.D.

Matthew Johnson, Ph.D.
IRB-Chair

Appendix E. Data Collection Sheets

HDI/*PulseWave*™ CR-2000

Research CardioVascular Profile Report

Research Subject ID:

Research Subject Name:

Date:
Time:
Age:
Gender:
Height:
Weight:
BSArea:
Body Mass Index:

Average Blood Pressure Waveform

PARAMETER	RESEARCH SUBJECT VALUE
SYSTOLIC BLOOD PRESSURE	
DIASTOLIC BLOOD PRESSURE	
MEAN ARTERIAL BLOOD PRESSURE	
PULSE PRESSURE	
PULSE RATE (beats/min)	
ESTIMATED CARDIAC EJECTION TIME (msec)	
ESTIMATED STROKE VOLUME (ml/beat)	
ESTIMATED STROKE VOLUME INDEX ($ml/beat/m^2$)	
ESTIMATED CARDIAC OUTPUT (L/min)	
ESTIMATED CARDIAC INDEX ($L/min/m^2$)	
LARGE ARTERY ELASTICITY INDEX (ml/mmHg x 10) (Capacitive Arterial Compliance)	
SMALL ARTERY ELASTICITY INDEX (ml/mmHg x 100) (Oscillatory or Reflective Arterial Compliance)	
SYSTEMIC VASCULAR RESISTANCE ($dyne \cdot sec \cdot cm^{-5}$)	
TOTAL VASCULAR IMPEDANCE ($dyne \cdot sec \cdot cm^{-5}$)	

Eagan, MN 55121 +1-651-687-9999 Toll-Free: 1-888-*PulseWave* (785-7392)

Form: 00017-001 (Rev. A / 08.Oct. 99) *"For Research Purposes Only"*