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Hayleigh Elizabeth Raiff

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# **The Acute Effects of Aerobic and Resistance Exercise on Cardiovascular Function and Arterial Stiffness**



Honors Thesis

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Department: Health and Sport Science

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## Abstract

The cardiovascular system changes acutely to the stresses of exercise to support the increased metabolic demand of the working tissues. This is accomplished through the augmentation of several parameters including heart rate, blood pressure, and vascular tone such as arterial stiffness. Exercise training has been shown to elicit changes in arterial stiffness but the acute effects of exercise on arterial stiffness have not been thoroughly studied. The current study examined the acute effects of no (control), aerobic (30 minutes of cycling at ~70% maximum heart rate), and resistance exercise (30 minutes, 3 sets of 10 repetitions for 6 exercises) on arterial stiffness in healthy males (n=11) utilizing measures of carotid-femoral pulse wave velocity and pulse wave analysis at rest and during recovery for 60 minutes. The exercise sessions utilized were consistent with American College of Sports Medicine guidelines for exercise in healthy individuals. Carotid-femoral pulse wave velocity demonstrated no significant change from resting values throughout recovery for any of the activities ( $\sim 9 \text{ m}\cdot\text{s}^{-1}$ ). Systemic arterial stiffness values (corrected to a heart rate of 75 bpm) were significantly higher post-resistance exercise than the control and aerobic exercise activities initially ( $34.2 \pm 10.3\%$  vs.  $14.2 \pm 10.9\%$  and  $3.2 \pm 12.7\%$ ,  $p < 0.05$ ) and remained statistically higher throughout recovery. These results indicate that resistance exercise alone resulted in an increase in systemic arterial stiffness that lasted for at least 60 minutes. In contrast, neither aerobic or resistance activity elicited a change in regional arterial stiffness. Further studies may clarify the time course and mechanisms for changes in arterial stiffness following acute and chronic exercise of various modalities and intensities.

## Acknowledgements

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## INTRODUCTION

The human cardiovascular system dynamically responds to the demands of the body elicited by changes in environment, activity, or internal conditions. These responses occur through the augmentation of several parameters, including heart rate and several measures of pressure within the cardiovascular system<sup>1</sup>. A common stress placed on the cardiovascular system is that elicited through physical activity and exercise. Exercise is a stress placed upon the body that requires extensive changes to the functioning of the cardiovascular system as well as other bodily systems to fulfill the metabolic demands of active tissues and sustain activity<sup>1</sup>.

During moderate intensity exercise, the cardiovascular system changes due to an increase in sympathetic nervous system activity and inhibition of parasympathetic activity<sup>2</sup>. Upon exercise initiation, heart rate and cardiac output increase proportionally with exercise intensity until maximal intensity<sup>2</sup>. The increase in cardiac output is also supported by constriction of venous vasculature and active muscles pumping blood back toward the heart. Dynamic exercise results in an increase in systolic blood pressure and mean arterial pressure, while diastolic blood pressure has been shown to decrease with increasing exercise intensity<sup>1</sup>. This is caused by the pressure the cardiac output associated with exercise puts on the constricted vessels in non-exercising muscles<sup>3</sup>. However, this increase in systolic blood pressure is buffered by the vasodilation in active muscles and results in a minimal change in diastolic blood pressure<sup>3</sup>.

Though the changes on the cardiovascular system during exercise are physiologically predictable, different types of exercise elicit different effects. Endurance exercise has been reported to exhibit a linear relationship between exercise intensity and systolic blood pressure changes, while diastolic blood pressure has been shown to be constant or slightly decrease by 10 to 20 mmHg<sup>3</sup>. Resistance exercise has been shown to elicit more extreme increases in systolic and diastolic blood pressure than endurance exercise due to sympathetic vasoconstriction in non-exercising vascular beds, compression of vessels in exercising muscles, and the Valsalva manoeuvre<sup>3</sup>. Unlike the linear and constant change during blood pressures observed in endurance

exercise, changes in blood pressure during resistance exercise are oscillatory and depend on the phase of the exercise. Blood pressure is maximized during the concentric lifting phase, declines (often below resting values) upon completion of the lift, and increases again during the eccentric lowering phase of the exercise<sup>4</sup>. In addition, the type of resistance exercise has been shown to cause different cardiovascular effects. Arm exercises are associated with ten percent greater increase in arterial pressure than seen with exercises that target lower extremity muscle groups<sup>3</sup>.

After completion of exercise, the cardiovascular system must again make changes to maintain homeostasis. Upon cessation of endurance exercise, a blood pressure decrease is often observed due to the pooling of blood in dilated vascular beds. The hypotensive effect of resistance exercise is often more pronounced than that observed in endurance exercise<sup>3</sup>. However, baroreceptor stimulation and the resulting baroreflex normally return blood pressure homeostasis within ten minutes post exercise (Figure 1)<sup>3</sup>. During this post-exercise hypotensive period, systemic and regional peripheral resistance have been shown to decrease, even in the vasculature of non-exercising muscles<sup>5</sup>. This post exercise hypotension is typical following most exercise modes. Studies observing the duration of post-exercise hypotension have produced confounding results in which some studies document a return to baseline values within an hour of recovery while others show a perpetuating effect for several hours<sup>6,7,8</sup>. This may suggest that there is an oscillatory pattern of blood pressure return after exercise. A study conducted by Pescatello et al observed an oscillatory pattern in systolic blood pressure over a twelve hour post exercise period when blood pressure measures were taken every 30 minutes<sup>9</sup>. MacDonald et al. states that a long duration, controlled study needs to be completed to assess the time course of post-exercise hypotension<sup>3</sup>. Studying the time course of post-exercise hypotension could inform the mechanism causing hypertension and further inform the exercise prescription of those at risk or suffering

from this condition<sup>3</sup>.

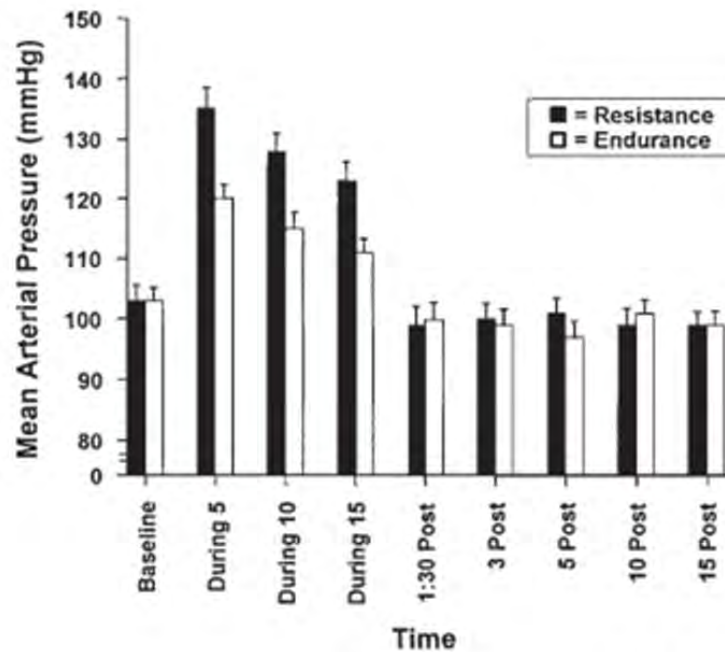


Figure 1: Mean blood pressure responses to endurance exercise (cycling at 65%  $\text{VO}_2$  Peak) and resistance exercise (unilateral leg press at 65% 1 RM)<sup>3</sup>.

In addition to augmentation of blood pressure, there is a decrease in heart rate upon completion of exercise<sup>2</sup>. Cardiodeceleration after exercise, also known as heart rate recovery, is affected by several neurological and physiological influences<sup>2</sup>. Initial decrease in heart rate after exercise is due to the termination of the exercise stimulus from the cerebral cortex. However, slower factors that may contribute to the post-exercise cardioacceleration effect include changes in stimuli to the metabaroreceptors as the body works to eliminate the metabolites, catecholamines, and excess body heat that are produced through exercise<sup>2</sup>. Additionally, the main mechanism of cardioacceleration is parasympathetic activation upon cessation of exercise followed by sympathetic withdrawal. During recovery from moderate to heavy exercise, heart rate has been shown to remain elevated above resting levels for up to sixty minutes<sup>10, 11</sup>. This sustained elevation suggests that sympathetic activity remains influential on heart rate after 60 minutes of recovery<sup>10</sup>.

Another parameter that responds to changes in cardiovascular function is arterial stiffness. Arterial stiffness is a measure that assesses the structural integrity of the artery. It collectively describes the compliance, elasticity, and distensibility of the arterial system<sup>12</sup>. The arterial response to exerted pressures is affected by many factors including the vascular structure, neurological factors, and pathophysiological processes<sup>12</sup>. The location of arteries has a profound influence on arterial composition and, therefore, how a given artery responds to the pressure exerted from the heart. Proximal arteries are more elastic, reflected by the lower blood pressure values, whereas distal arteries are stiffer, displaying higher blood pressure values<sup>13</sup>. An increase in arterial stiffness has been associated with increased central pulse pressure and increased systolic blood pressure<sup>12</sup>. Arterial stiffness increases due to the loss of the elastic fibers and laminae of the arteries which are replaced with collagen fibers and ground substance that can be associated with calcium deposition<sup>14</sup>. While these changes can occur naturally with age, there are other factors that may expedite the process of stiffening the arteries. Several studies have reported that genetics contribute to arterial stiffness independently from blood pressure<sup>14</sup>. Other pathophysiological processes have been found to increase the rate of arterial stiffening, including hypertension, diabetes mellitus type I and II, and renal diseases while tobacco use and dyslipidemia have been hypothesized to contribute to stiffening, the connection between these factors remain unclear<sup>15</sup>. The degree of arterial stiffness over time and between conditions has been a developing area of research and is clinically relevant as it not only reflects the chronic health of the arteries, but may also limit the arteries' responses to stressors such as exercise. Studies have also reported that an increase in arterial stiffness was strongly associated with atherosclerosis<sup>16</sup> and serves as an indicator for cardiovascular disease (CVD) in the clinical setting and screening for risk assessment<sup>17</sup>.

Arterial stiffness can be assessed regionally or systemically. Measuring regional arterial stiffness allows for the assessment of structural integrity at a particular arterial site whereas evaluating systemic arterial stiffness assesses the ability of the entire arterial system to respond to



the pressures caused by the cardiac cycle<sup>12</sup>. Arterial stiffness can be assessed using several methods including angiography, echocardiography, ultrasound, and magnetic resonance imaging<sup>17</sup>. However, these methods are often expensive and cannot be used practically to study acute effects of various stressors on arterial stiffness. Therefore, indirect methods of measuring arterial stiffness have been developed for use in the clinical and research setting. These measures include observation of pulse wave velocity (PWV) and pulse wave analysis (PWA) as measured by applanation tonometry.

Pulse wave velocity (PWV) is a cost effective method of measuring regional arterial stiffness<sup>12</sup>. Obtaining PWV measurements involves measuring a pulse wave at two peripheral sites, commonly the carotid and femoral arteries, via a tonometric device<sup>12</sup>. The measurement of PWV at the carotid and femoral artery is known as carotid-femoral pulse wave velocity, cfPWV, and is the current gold standard for assessing arterial stiffness, particularly the stiffness of the aorta<sup>13, 18</sup>. The cfPWV can be calculated using several devices including the SphygmCor XCEL system (Atcor, Australia) with these devices recording the pulse transit time and the distance between the two recording sites<sup>19</sup>. The more elastic an artery is, the lower the PWV value. Due to the nature of the less elastic periphery, PWV tends to be faster in peripheral arteries than in centrally located arteries. For example, in a normotensive individual, PWV is 4-5 m/s for the ascending aorta and 8-9 m/s for the peripheral femoral artery<sup>13</sup>. An increase in arterial stiffness results in an increase in PWV the artery as the reflected pressure wave associated with the heart contraction reaches the aortic valve sooner than normal<sup>20</sup>. Subsequently, an increase in systolic pressure is needed to overcome the pressure caused by this premature wave in order to deliver the blood pumped in the next cardiac cycle<sup>20</sup>. While peripheral arterial stiffness can be determined via PWV, PWA can assess systemic arterial stiffness via applanation tonometry<sup>12</sup>. The process of PWA involves analyzing the aortic pulse pressure waveform using a tonometric device that flattens the artery to approximate aortic pressure. The aortic pressure waveform presents information about the overall integrity of the arterial system (Figure 2). The parameters assessed

by PWA include augmentation pressure, augmentation index, and augmentation index at a heart rate of seventy-five beats per minute. Augmentation pressure (AP) is a measure of aortic systolic pressure caused by return of the reflected pulse waves at the central aorta<sup>21</sup>. Augmentation index (AIx) is the augmentation pressure expressed as a percentage of central pulse pressure. It is a measure that accounts for aortic wave reflection and represents systemic arterial stiffness<sup>21</sup>. The AIx increases as mean arterial pressure increases but decreases with an increase in body height and heart rate<sup>12</sup>. An increase in heart rate of ten beats per minute can elicit a four percent reduction in AIx<sup>22</sup> and, therefore AIx should be normalized for with heart rate of 75 beats per minute (AIx@75)<sup>12</sup>. The SphygmoCor XCEL system decreases AIx by 4.8% for every increase of 10 beats per minute to produce AIx@75 values<sup>12</sup>. Though PWV and AIx are related, they are not interchangeable in that vasoactive drugs can affect AI by affecting the pressure of reflected waves while not influencing PWV<sup>23</sup>. The AIx is therefore influenced by the integrity of smaller arteries, while PWV is determined only by condition of the aorta<sup>12</sup>.

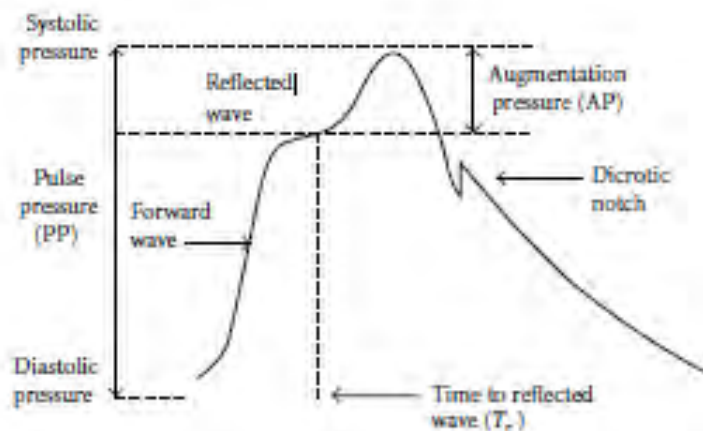


Figure 2: Aortic pulse pressure waveform<sup>12</sup>

The PWV and PWA are measures that can be taken at rest to assess arterial functioning. However, these measures can also be observed before and after a set of conditions to assess arterial changes caused by the given condition. For example, PWV and PWA can be measured pre and post exercise to determine a change in arterial stiffness associated with the exercise.

Exercise training effects on arterial stiffness have been investigated in several studies. Aerobic exercise has been shown to prevent or even reverse arterial stiffness in participants. In an eight week study in which young sedentary male participants completed aerobic exercise bouts at 70% of estimated maximal heart rate, there was an associated 5% decrease in resting cfPWV<sup>24</sup>.

Another study conducted on a population of pre-hypertensive and hypertensive participants that completed four weeks of aerobic exercise training at 65% of their predicted maximal heart rate demonstrated an 8% decrease in cfPWV<sup>25</sup>. Resistance exercise training has also been shown to affect arterial stiffness, though the effect appears to be greatly influenced by exercise intensity. For example, high intensity resistance exercise training of 16 weeks demonstrated an increase in resting arterial stiffness<sup>26</sup>. After a 24 week deconditioning period, the elevated arterial stiffness parameters of the participants in the study returned to baseline values<sup>26</sup> which further supports that the observed increase in arterial stiffness in the study was elicited by the high intensity resistance exercise. In contrast, moderate intensity resistance exercise training programs have resulted in smaller increases of arterial stiffness with values not significantly different from pre-training values<sup>27, 28</sup>. Therefore, while the effects of resistance exercise training on arterial stiffness have been investigated, studies have yielded intensity-dependent results.

The chronic training effects of exercise on arterial stiffness have been thoroughly investigated, yet the acute effects of exercise have not been explored as extensively. Preliminary research suggests that aerobic exercise leads to an increase in arterial stiffness. Doonan et al. investigated cfPWV during a fifteen minute recovery period after aerobic activity to exhaustion and concluded that cfPWV increased significantly after two minutes recovery in male subjects, indicating an increase in arterial stiffness<sup>19</sup>. A significant increase in AIX@75 after five minutes of recovery was also observed in men however, this difference was lost when the results were adjusted for age and body mass index<sup>19</sup>. The acute effects of resistance exercise have been studied as well. Low intensity resistance exercise has been reported to acutely increase arterial compliance and decrease arterial stiffness at 30 and 60 minutes after exercise when assessed by

ultrasound and applanation tonometry<sup>29</sup>. In contrast, high intensity resistance exercise was shown to acutely increase arterial stiffness. A study by DeVan et al. observed that arterial stiffness as measured by ultrasound and applanation tonometry increased after high intensity resistance exercise, but that this elevated effect lasted for less than sixty 60 minutes<sup>30</sup>. Collectively these results indicate variable recovery responses for arterial stiffness that were dependent on exercise modality and intensity.

While the effects of various aerobic and resistance exercises on arterial stiffness have been previously studied, there has not been an investigation about the acute effects of exercise consistent with American College of Sports Medicine (ACSM) recommendations for exercise in healthy individuals<sup>31</sup>. The aim of this study was to investigate the acute effects of aerobic and resistance exercise consistent with ASCM recommendations on arterial stiffness post-exercise. It was hypothesized that resistance exercise would elicit a greater and longer increase in arterial stiffness compared with aerobic exercise.

## **MATERIALS AND METHODS**

### **Participants:**

Participants were recruited via local advertisement and word of mouth at James Cook University. Participants were healthy males between the ages of 18 and 45. Exclusion criteria were: previously diagnosed cardiovascular disease (CVD), possessing more than two CVD risk factors as determined by a pre-participation questionnaire, resting hypertension, prescription medication use, and smoking. If the participant was deemed eligible, they signed a pre-participation informed consent approved by James Cook University and University of Dayton Institutional Review Boards. Eleven individuals volunteered for participation in this study with a one hundred percent retention rate. Participants completed a familiarization session followed by three experimental sessions consisting of a single bout of no (control) exercise, aerobic exercise, or resistance exercise. The experimental sessions were completed in a randomized order (computer generated) and there was at least 72 hours between experimental sessions. The participants were pre-prandial during each of the sessions and were instructed to not eat or consume beverages except water after midnight the night before the session<sup>12</sup>. They were instructed to not use caffeine or alcohol or participate in exercise for at least 24 hours before each session.

### **Familiarization:**

Eligible participants completed a familiarization session to determine the intensities to be used in the experimental sessions. Anthropometric data was obtained for each participant with height measured using a mounted stadiometer. Mass, percent body

fat, and body mass index were determined using a bioelectrical impedance analysis scale (Tanita Corporation of America, Inc., Arlington Heights, Illinois, USA). The participant was then fitted with a Polar RS800 heart rate monitor (POLAR Electro, Kempele, Finland). The chest strap was placed so that the sensor was placed securely in the midline of the body just below the pectoral muscles. The participant then lay supine on a table and the researcher performed a round of measures that were to be taken throughout the remaining sessions for resting values. The order of these measures were the recording of: brachial blood pressure from the left arm, pulse wave velocity (PWV) assess from the right carotid and femoral arteries, and pulse wave analysis (PWA) assessed from the right arm. After completing a cycle of measurements and confirming normotensive results at rest, the participant completed an aerobic bout of exercise on a cycle ergometer (828E, Monark, Varberg, Sweden). The participant's age predicted max heart rate was determined using the equation of Inbar<sup>32</sup>. The target heart rate for the aerobic exercise session was determined to be 70-75% of the age predicted heart rate maximum. The participant pedaled at 65-70 revolutions per minute (rpm) with the resistance of the cycle ergometer adjusted to maintain a heart rate within the determined range. The workload identified was noted for use during the aerobic session.

After 10 minutes of recovery, the workloads for the resistance session were determined. The exercises performed were hack squats, chest press, leg curl, prone row, shoulder press, and bicep curl. The chest press, leg curl, and shoulder press were conducted using a Nautilus® multigym apparatus (Nautilus International, Independence, Virginia, USA). The chest press, prone row, and bicep curl were performed using free weights. The participant completed a warm up set of 10 repetitions at 50% of the

perceived/estimated 10 repetition maximum (10-RM) for each exercise. The participants' 10-RM was then determined for each exercise by progressively increasing the weight until the participant could just achieve 10 repetitions and perceived an exertion level of more than 18. Workloads for the experimental session were set at 90% of the 10-RM level for the hack squat and prone row, and 80% of the 10-RM for the bicep curl. For the exercises using the Nautilus® multigym equipment, the chosen resistance level was two weight plates lower (i.e. 9.1 kg or 20 lbs.) than the 10-RM weight. After the familiarization session was completed, the participant was scheduled to complete the first of 3 experimental sessions, with each sessions consisting of initial rest, experimental activity, and recovery.

### **1. Initial Rest:**

The participant would put on the Polar RS800 heart rate monitor upon entering the lab and lie supine on the table for twenty minutes. During this time, lights were dimmed and the participant monitored. After 20 minutes of rest, venous blood samples were taken for the analysis of blood biomarkers of vascular function. The samples were centrifuged and stored for further analysis in conjunction with the Vascular Biology Unit at James Cook University. After the blood draw, peripheral blood pressure was taken in the left arm using the Welch Allyn Connex® ProBP™ 3400 digital blood pressure device. Pulse wave velocity (PWV) and pulse wave analysis (PWA) measures were then taken. All resting values were recorded in duplicate and averaged.

## **2. Experimental Activity:**

After the initial rest component, the participant completed one of the following 30-minute activities.

### **A. Control:**

During the thirty minute control activity, the participant sat in a chair while maintaining good posture with both feet on the floor and the left arm lay on a table at heart level. Blood pressure was measured every five minutes using a Welch Allyn Connex® ProBP™ 3400 digital blood pressure device over the left brachial artery with heart rate and rating of perceived exertion (RPE) recorded every minute.

### **B. Aerobic Exercise:**

The aerobic exercise activity involved the participant pedaling on a Monark cycle ergometer at 65-70 revolutions per minute for 30 minutes. The resistance of the cycle ergometer was set to the workload determined in the familiarization session. Rating of perceived exertion (RPE) and heart rate were recorded every minute while blood pressure was recorded using the Welch Allyn Connex® ProBP™ 3400 digital blood pressure device or manually via a stethoscope and sphygmomanometer.

### **C. Resistance Exercise:**

The resistance exercise component involved the participant completing three sets of ten repetitions of the six exercises at the workloads determined in the familiarization session. A minute of rest was allowed between each set and exercise. Heart rate and RPE



were recorded after every set while blood pressure was monitored using the digital blood pressure device after the third set of every exercise. The resistance exercise session was completed in approximately 30 minutes.

### **3. Recovery:**

After the completion of the activity, the participant returned immediately to the table to lie in the supine position. Blood samples were again taken, centrifuged, and stored for later assessment of blood biomarkers of vascular function. In a similar order to the resting measures, single recordings of brachial blood pressure, PWV, and PWA were obtained at 10, 20, 30, 40, 50 and 60 minutes of recovery.

## **Procedures**

The variables observed in this study were obtained using the following methods.

### **1. Measurement of Brachial Blood Pressure:**

While in the supine position, a Welch Allyn Connex® ProBP™ 3400 digital blood pressure device was placed at the level of the brachial artery on the participant's left arm. The automated device provided heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure readings.

### **2. Measurement of Pulse Wave Velocity:**

The participant remained in the supine position with their right carotid artery located (i.e. the participant tilted their head back and slightly to the left). The location of

the strongest carotid pulse was marked. The participants femoral pulse was then located in the right leg by finding the inguinal fold and palpating laterally until the femoral pulse was felt. The SphygmoCor thigh cuff was placed 15 cm below the location of the femoral pulse as measured via tape measure. The distance from the carotid pulse to the femoral cuff was measured and entered into the recording SphygmoCor system (SphygmoCor XCEL, Atcor, Australia). The SphygmoCor probe was placed on the pre-determined carotid pulse location to obtain a waveform using the SphygmoCor software and PWV was recorded from twelve consistent waveforms. At this point, the femoral cuff inflated and slowly deflated to measure the heart rate and pulse transit time from the carotid to femoral artery. SphygmoCor XCEL system provided a quality control check for all assessments. The SphygmoCor XCEL system has been reported to be reliable and valid for the assessment of PWV<sup>33</sup>.

### **3. Measurement of Pulse Wave Analysis:**

A cuff was placed at the level of the brachial artery of the participant's right arm while they remained in the supine position. Upon completion of the PWV measurement, the measurement of PWA was then initiated and automatically assessed by the SphygmoCor XCEL system by inflating and slowly deflating the brachial cuff. The parameters measured during the PWA assessment were heart rate, brachial systolic blood pressure, brachial diastolic blood pressure, central systolic blood pressure, central diastolic blood pressure, mean arterial pressure, pulse pressure (PP), augmentation pressure (AP), augmentation index (AIx), and augmentation index corrected for a heart

rate of 75 bpm (AIx@75). The SphygmoCor XCEL system has been reported to be reliable and valid for the assessment of PWA<sup>33</sup>.

### **Data Analysis**

Data was analyzed using Microsoft Excel and IBM SPSS Statistics software. The data was analyzed using a two-way ANOVA to determine the change in values over time (e.g. rest vs. recovery) and between activities (i.e. control, aerobic, and resistance). A post-hoc Tukey test was also completed to confirm statistical differences in means with the alpha level set at 0.05.

## RESULTS

. Participant characteristics are available in Table 1 (Appendix 1A). The testing environment was thermoneutral for all experimental sessions with a mean temperature of  $30 \pm 0.2$  C and a humidity of  $64.2 \pm 5.5\%$ .

### **Cardiovascular Responses During Activities:**

Cardiovascular responses elicited by each activity are displayed in Table 2 (Appendix 1B). Mean heart rate during aerobic and resistance exercise was significantly higher than the control session and the resistance exercise heart rate was significantly higher than aerobic exercise (Table 2). The mean heart rate elicited by the aerobic exercise was 70.6% of the age predicted heart rate maximum while the resistance exercise resulted in a mean heart rate representing 79.7% of the age predicted heart rate maximum. The aerobic exercise activity yielded a higher mean exercise systolic blood pressure than both the control and resistance exercise activities (Table 2). Resistance exercise resulted in a significantly lower diastolic pressure response than the other two activities. Mean arterial pressure was significantly higher for aerobic exercise compared with both the control and resistance activities (Table 2).

### **Cardiovascular Responses during Recovery:**

Table 3 (Appendix 1C) displays the hemodynamic changes during recovery from the three activities. During post exercise recovery, heart rates at each time point from were significantly greater for resistance exercise compared to both the aerobic and control activities (Table 3). Similarly, the heart rate at each time point of recovery from

aerobic exercise was significantly higher than the corresponding values for the control activity. There was no significant change in peripheral systolic blood pressure during recovery with similar values across the three exposure activities (Table 3). Central aortic systolic blood pressure was similar over time and between activities except at 50 minutes of recovery where both aerobic and resistance exercise values were significantly lower than control values (Table 3). Peripheral diastolic blood pressure after resistance exercise was significantly lower than aerobic and control activities at 10 minutes of recovery and continued to be lower than the control values throughout the remainder of recovery (Table 3). Central diastolic pressure after resistance exercise was significantly lower than control values throughout post exercise recovery and were significantly lower than resting values at 10, 30, and 50 minutes of recovery (Table 3).

Table 4 displays the PWV and PWA arterial stiffness variables at rest and throughout recovery for each activity. The cfPWV values were similar over time and between activities except at 30 minutes of recovery where the resistance exercise value was slightly higher than the aerobic value (Table 4). Augmentation pressure and augmentation index for resistance exercise remained elevated above control and aerobic activity values throughout recovery (Table 4) except at 50 minutes where it was only significantly higher than the aerobic activity. The  $AIx@75$  following resistance exercise was significantly higher than aerobic and control activity values throughout recovery and remained significantly higher than resting resistance values (Table 4). Figure 1 depicts  $AIx@75$  values for each exposure throughout recovery (Appendix 2A).

## DISCUSSION

To my knowledge, this is the first study to observe the acute effects of aerobic and resistance exercise in line with ACSM recommendations utilizing PWV and PWA arterial stiffness parameters. Measures of PWV and PWA revealed that none of the exercise activities were associated with a significant change in regional arterial stiffness (cfPWV) but that the resistance exercise increased systemic arterial stiffness (PWA). The increase in AP, AIx, and AIx@75 suggest that the resistance exercise acutely increased arterial stiffness throughout the recovery period. A previous study utilizing a similar resistance protocol discovered that arterial stiffness parameters returned to resting values after less than an hour of recovery<sup>30</sup>. However, in the current study the PWA values were significantly different than resting values at the end of the recovery, suggesting that 60 minutes was not sufficient for the arteries to recovery from the resistance session. The exact time for systemic arterial stiffness values to return to resulting values post-resistance exercise is not known but the timing of the experimental sessions provides some insight. Each participant had at least 72 hours between experimental sessions and all participants had returned to resting PWA measurements when they returned for their subsequent session. Therefore, it can be inferred that the recovery time for PWA values is between one and 72 hours. Further study is needed to determine the exact kinetics of return that could help determine the normal recovery time for acute arterial stiffness changes elicited by resistance exercise. Subsequent studies of chronic exercise training could elaborate on these results to determine the potential long-term changes in arterial stiffness.

In contrast to resistance exercise, aerobic exercise did not display an increase in arterial stiffness either via PWV or PWA in the current study. A similar study conducted by Doonan et al. elicited an increase in cfPWV after 10 minutes of aerobic exercise by utilizing a Bruce protocol to elicit exhaustion<sup>19</sup>. The current study elicited an exercise response consistent with ACSM recommendations (~70% of age predicted HRmax) but did not yield an increase in cfPWV. This difference in results may indicate an exercise intensity influence, with exercise at very high levels (e.g. maximum or exhaustion) needed for acute changes in regional stiffness. It is also feasible that the time of the recovery assessment was too long with any potential change in cfPWV missed (<10 minutes post-exercise). The measurement of PWV and PWA at a time point sooner than 10 minutes of recovery could identify whether aerobic exercise exposure caused an acute change in arterial stiffness.

An unexpected result from the study was a change in AIx and AIx@75 during recovery from the control exposure. The recovery values at each time point were significantly lower than resting values. This was an unexpected change because the participant's position was the only change (i.e. supine to seated upright) during the control exposure. Studies have shown that recovery position can have an effect on arterial resistance. When body position at rest is changed from upright to supine, cardiac sympathetic nerve activity decreases and vagus nerve activity increases<sup>34</sup>. However, this would lead to an initial increase in arterial stiffness as opposed to the decrease that was observed. Therefore, the change in body position from the seated control session to the supine recovery session should not have caused the associated decrease in PWA parameters. Further study is needed to determine the cause of these unexpected results.

This study provides baseline measures of arterial stiffness assessed by PWV and PWA and could direct continued study in this area. Exercise exposures utilizing different aerobic exercise modalities and resistance exercise routines could be implemented to observe if the modality of exercise affects the cardiovascular parameters observed in the current study. For example, a treadmill exercise exposure within ACSM guidelines could be implemented and the elicited cardiovascular effects could be compared to those observed after completion of an aerobic exposure on a Monark cycle ergometer. Similarly, different resistance exercise routines could be implemented focusing on multijoint versus single joint motions, bodyweight versus weight-stack exercises, upper extremity versus lower extremity exercises, etc. to observe if different resistance routines produce varying cardiovascular effects. The current study identified that the recovery for systemic arterial stiffness values was between one hour and 72 hours with future studies possibly examining the time course and, when these values return to rest. This could give information about how long the acute effect of resistance exercise lasts after exercise cessation and whether chronic exercise exposures results in alterations in the time course. Another line of continued study that could result from the current study is determining how vasculature affected by pathophysiological processes responds to the stresses of exercise. The current study was conducted utilizing healthy participants without cardiovascular diseases. Future studies could examine similar measures in those with known cardiovascular diseases, such as peripheral artery disease (PAD) and abdominal aortic aneurysm (AAA). However, further study is needed to determine the effects of various exercise exposures on healthy vasculature before it can be implemented in a diseased population. The modality, intensity, and duration of exercise that allows for



the positive health gains of exercise while minimizing the negative vascular effects needs to be identified before it can be safely tested in those with cardiovascular conditions and diseases.

Limitations of the study include its small sample size. While the same size was similar to other studies, a larger sample size could clarify the results obtained. The varying degree of resistance exercise experience by participants may have also affected the results of the study. While all participants had some experience with resistance exercise, some were avid resistance trainers. Increased resistance experience could have affected for m and allowed them to implement different techniques, such as the Valsalva maneuver, which has been shown to increase peripheral venous pressure and total peripheral resistance during exercise and after completion of the exercise bout<sup>35</sup>. This could have affected the PWA measures obtained. A final limiting factor of the study was it was not possible to measure arterial stiffness parameters with the SphygmoCor XCEL system while the participant was exercising<sup>36</sup>. These measures could only be taken at rest and during the post-exercise recovery. Therefore, the study was only able to analyze the change in arterial stiffness parameters pre and post exercise, while the effects during exercise remain unknown. A different system of measurement would need to be utilized to observed changes in arterial stiffness during exercise. To my knowledge, there is not currently a system that can measure arterial stiffness while the participant is completing activity.

## **CONCLUSION**

The results of this study demonstrate that the resistance exercise activity elicited a significant increase in systemic arterial stiffness as measured by AP, AIx, and AIx@75 throughout a 60 minute recovery period that was greater than control and aerobic exercise activities. None of the three exercise activities altered regional (aortic) arterial stiffness, as measured by cfPWV. Further studies may determine the time course and mechanisms for acute systemic arterial stiffness changes following resistance exercise.

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## APPENDIX 1

## A.

**Table 1:** Participant characteristics (n=11)

	Mean $\pm$ SD
Age (yrs)	30.3 $\pm$ 7.4
Height (cm)	176.5 $\pm$ 4.8
Mass (kg)	82.8 $\pm$ 11.0
Body fat (%)	20.0 $\pm$ 6.7
BMI ( $kg \cdot m^{-2}$ )	26.6 $\pm$ 3.5

## B.

**Table 2:** Cardiovascular and perceptual responses during no (C), aerobic (A) and resistance (R) exercise

	C	A	R
Exercise HR (bpm)	64.5 $\pm$ 6.4	130.7 $\pm$ 3.4 <sup>†</sup>	147.5 $\pm$ 1.6 <sup>†‡</sup>
Exercise HR (%HRmax)	34.9 $\pm$ 3.5	70.6 $\pm$ 0.9 <sup>†</sup>	79.7 $\pm$ 0.1 <sup>†‡</sup>
Exercise RPE	6.0 $\pm$ 0.0	12.9 $\pm$ 1.1 <sup>†</sup>	17.6 $\pm$ 1.1 <sup>†‡</sup>
Exercise SBP (mmHg)	117.6 $\pm$ 11.0	160.3 $\pm$ 10.1 <sup>†</sup>	129.8 $\pm$ 10.8 <sup>†‡</sup>
Exercise DBP (mmHg)	74.3 $\pm$ 7.8	76.7 $\pm$ 9.2	68.4 $\pm$ 4.3 <sup>†‡</sup>
Exercise MAP (mmHg)	88.8 $\pm$ 8.5	104.6 $\pm$ 8.1 <sup>†</sup>	88.9 $\pm$ 6.0

<sup>†</sup>p<0.05 vs C; <sup>‡</sup>p<0.05 vs A

C.

Table 3: Hemodynamic responses prior to (Rest) and following no (C), aerobic (A), and resistance (R) exercise

	Rest	Post-exercise					
		10 mins	20 mins	30 mins	40 mins	50 mins	60 mins
<b>HR (bpm)</b>	C	59.3 ± 6.8	57.9 ± 8.5	55.1 ± 6.4	56.1 ± 7.4	55.3 ± 10.7	53.7 ± 4.2
	A	57.4 ± 6.3	73.5 ± 6.7 <sup>†‡</sup>	67.9 ± 8.9 <sup>†‡</sup>	65.5 ± 6.9 <sup>†‡</sup>	66.5 ± 7.8 <sup>†‡</sup>	65.2 ± 7.2 <sup>†‡</sup>
	R	58.0 ± 7.0	91.6 ± 8.2 <sup>†‡</sup>	87.1 ± 7.8 <sup>†‡</sup>	85.5 ± 6.8 <sup>†‡</sup>	77.6 ± 9.7 <sup>†‡</sup>	76.9 ± 9.6 <sup>†‡</sup>
<b>Peripheral SBP (mmHg)</b>	C	126.1 ± 7.7	126.1 ± 6.2	127.0 ± 8.1	126.6 ± 6.0	125.8 ± 6.5	128.2 ± 9.5
	A	125.0 ± 8.3	128.0 ± 8.7	125.7 ± 9.6	124.5 ± 9.9	122.8 ± 11.2	122.7 ± 7.3
	R	123.2 ± 8.4	128.5 ± 12.7	125.9 ± 12.2	122.5 ± 8.6	121.9 ± 7.6	119.2 ± 7.4
<b>Peripheral MAP (mmHg)</b>	C	85.7 ± 8.6	88.7 ± 5.9	86.9 ± 7.1	87.4 ± 6.9	87.0 ± 5.7	89.3 ± 7.1
	A	84.4 ± 6.3	89.4 ± 6.7	86.1 ± 5.2	86.9 ± 7.7	85.4 ± 5.7	85.7 ± 8.4
	R	84.4 ± 6.8	83.0 ± 7.2	81.5 ± 6.8	81.8 ± 7.7	82.0 ± 7.7	80.2 ± 5.0
<b>Peripheral DBP (mmHg)</b>	C	71.1 ± 7.4	71.8 ± 7.3	73.2 ± 6.4	70.4 ± 10.3	74.9 ± 6.7	75.7 ± 7.6
	A	72.5 ± 8.8	73.0 ± 8.7	71.1 ± 7.1	70.0 ± 9.3	69.8 ± 9.2	70.1 ± 9.9
	R	70.4 ± 9.9	63.7 ± 9.3 <sup>†‡</sup>	65.8 ± 6.7 <sup>†</sup>	65.3 ± 8.1 <sup>†</sup>	64.5 ± 9.8 <sup>†‡</sup>	64.3 ± 7.9 <sup>†‡</sup>
<b>Central Diastolic Pressure (mmHg)</b>	C	72.5 ± 7.3	72.9 ± 6.8	74.1 ± 6.2	74.7 ± 5.6	75.9 ± 6.9	77.1 ± 7.8
	A	73.6 ± 8.5	73.6 ± 8.9	72.7 ± 7.1	71.4 ± 8.9	70.8 ± 8.9	71.5 ± 9.2 <sup>†</sup>
	R	71.7 ± 9.9	65.9 ± 10.2 <sup>†‡</sup>	68.2 ± 6.6 <sup>†</sup>	67.6 ± 7.8 <sup>†</sup>	65.9 ± 10.2 <sup>†‡</sup>	65.2 ± 8.0 <sup>†‡</sup>
<b>Central MAP (mmHg)</b>	C	87.2 ± 7.8	86.3 ± 6.4	87.5 ± 6.8	87.5 ± 6.5	88.2 ± 7.0	90.2 ± 8.2
	A	86.2 ± 10.0	89.9 ± 9.0	87.8 ± 7.3	85.6 ± 9.6	85.2 ± 9.5	84.9 ± 9.1
	R	85.5 ± 9.5	86.5 ± 12.2	86.3 ± 8.9	83.8 ± 8.8	82.4 ± 10.6 <sup>†</sup>	81.3 ± 8.5 <sup>†</sup>
<b>Central Aortic Systolic Pressure (mmHg)</b>	C	112.8 ± 8.4	111.6 ± 7.4	112.2 ± 7.2	112.7 ± 7.5	112.5 ± 8.1	115.2 ± 10.1
	A	111.8 ± 10.1	112.5 ± 9.5	110.6 ± 9.9	109.0 ± 10.2	108.2 ± 11.7	107.8 ± 8.6 <sup>†</sup>
	R	110.9 ± 9.3	113.3 ± 11.7	111.7 ± 10.3	108.5 ± 7.9	107.2 ± 8.2	104.5 ± 7.9 <sup>†‡</sup>
<b>Pulse pressure (mmHg)</b>	C	40.4 ± 4.9	38.7 ± 6.4	38.1 ± 5.8	38.0 ± 4.0	36.5 ± 5.1	38.1 ± 7.6
	A	38.2 ± 6.4	37.9 ± 8.1	37.9 ± 9.0	37.6 ± 8.0	37.4 ± 7.3	36.4 ± 6.6
	R	39.1 ± 6.2	47.4 ± 7.4 <sup>†‡</sup>	43.5 ± 7.3	40.8 ± 5.5	41.3 ± 6.4	39.3 ± 5.7 <sup>†‡</sup>

<sup>†</sup>p<0.05 vs. Rest; <sup>‡</sup>p<0.05 vs. 10 mins; <sup>†‡</sup>p<0.05 vs. 20 mins; <sup>†‡‡</sup>p<0.05 vs. 30 mins; <sup>†‡‡‡</sup>p<0.05 vs. 40 mins; <sup>†‡‡‡‡</sup>p<0.05 vs. 50 mins; <sup>†‡‡‡‡‡</sup>p<0.05 vs. 60 mins; <sup>†‡‡‡‡‡‡</sup>p<0.05 vs. A; <sup>†‡‡‡‡‡‡‡</sup>p<0.05 vs. R.



D.

Table 4: Arterial stiffness responses prior to (Rest) and following no (C), aerobic (A), and resistance (R) exercise

	Arterial stiffness parameter	Rest	Post-exercise					
			10 mins	20 mins	30 mins	40 mins	50 mins	60 mins
Pulse Wave Velocity	C	8.8 ± 1.8	9.3 ± 1.3	9.3 ± 1.5	9.4 ± 1.4	9.4 ± 1.5	9.4 ± 1.4	9.4 ± 1.4
	A	9.3 ± 1.2	9.4 ± 1.1	9.1 ± 1.1	8.3 ± 2.7	9.1 ± 1.1	9.1 ± 1.1	9.2 ± 1.2
	R	9.3 ± 1.5	9.7 ± 1.2	9.8 ± 1.3	9.4 ± 1.1 <sup>‡</sup>	9.3 ± 1.5	9.0 ± 1.3	9.1 ± 1.3
Pulse Wave Analysis	Augmentation Index							
	C	23.0 ± 12.9	14.1 ± 12.4 <sup>‡</sup>	13.5 ± 10.7 <sup>‡</sup>	16.4 ± 11.9	14.1 ± 11.9 <sup>‡</sup>	16.3 ± 13.2	13.9 ± 12.0 <sup>‡</sup>
	A	16.7 ± 14.3	18.1 ± 10.8	15.7 ± 12.3	12.6 ± 13.3	13.5 ± 13.2	12.3 ± 12.1	13.9 ± 13.6
	R	20.0 ± 13.7	27.8 ± 10.0 <sup>‡‡</sup>	25.9 ± 10.0 <sup>‡‡</sup>	25.4 ± 13.6 <sup>†‡</sup>	25.4 ± 8.8 <sup>†‡</sup>	21.0 ± 12.6 <sup>‡</sup>	22.5 ± 13.4 <sup>†‡</sup>

<sup>†</sup>p<0.05 vs. Rest; <sup>‡</sup>p<0.05 vs. 10 mins; <sup>‡‡</sup>p<0.05 vs. 20 mins; <sup>†‡</sup>p<0.05 vs. 30 mins; <sup>‡</sup>p<0.05 vs C; <sup>†‡</sup>p<0.05 vs A; <sup>†</sup>p<0.05, <sup>‡‡</sup>p<0.01.

## APPENDIX 2

A.

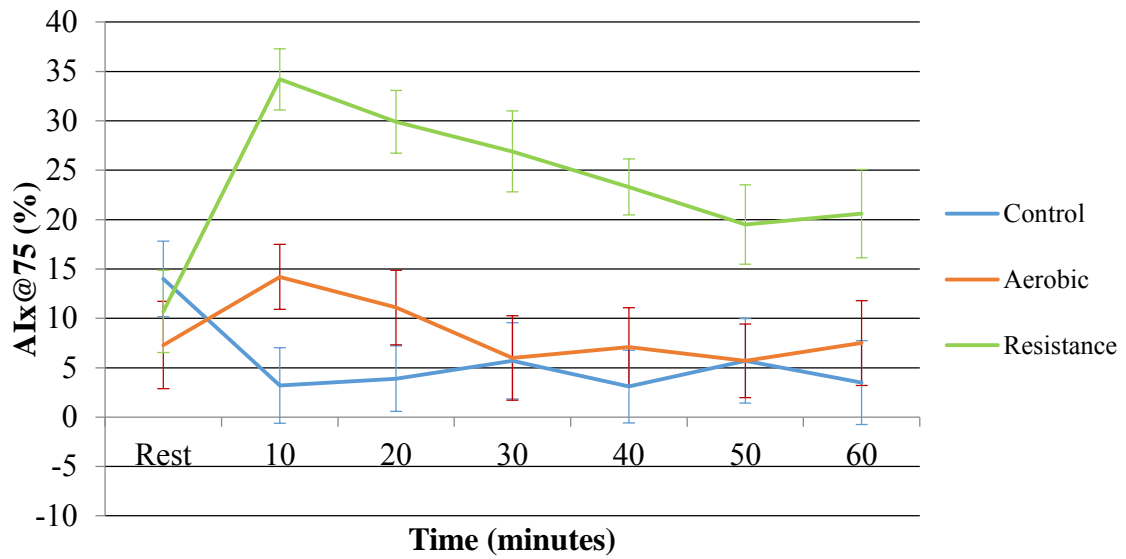


Figure 1: Change in AIx@75 for control, aerobic, and resistance experimental exposures at rest and throughout post-exercise recovery. Change in mean s.e.