EFFECTS OF 8-WEEKS OF ISOMETRIC HANDGRIP TRAINING ON RESTING ARTERIAL PRESSURE

EFFECTS OF 8-WEEKS OF ISOMETRIC HANDGRIP TRAINING ON RESTING ARTERIAL PRESSURE

By

PHILIP J. MILLAR, B.Sc.

A Thesis

Submitted to the School of Graduate Studies

in Partial Fulfillment of the Requirements

for the Degree

Master of Science

McMaster University

MASTER OF SCIENCE (2006)
(Kinesiology)

McMaster University
Hamilton, Ontario

TITLE: Effects of 8-weeks of Isometric Handgrip Training on Resting Arterial Pressure

AUTHOR: Philip J. Millar, B.Sc. (McMaster University)

SUPERVIOR: Dr. Neil McCartney, Ph.D.

SUPERVISORY COMMITTEE: Dr. Maureen MacDonald, Ph.D.
Dr. Steven Bray, Ph.D.
Dr. Markad Kamath, Ph.D.

NUMBER OF PAGES: 103, CIII

ABSTRACT

Recent evidence has demonstrated that isometric handgrip training may improve resting arterial blood pressure. The current study evaluated the ability of simple, spring handgrips to reduce resting arterial blood pressure in normotensive participants using an 8-week randomized controlled design.

Forty-nine (age: 66 ± 1) participants, 28 female and 21 males were recruited. All participants completed 5 pre-training sessions, used to familiarize and assess baseline blood pressure, heart rate and maximal hand strength. Maximal hand strength was assessed by three bilateral, maximal contractions with a hand dynamometer. Blood pressure and heart rate were assessed with an automated acquisition system. Participants were stratified to control and intervention groups based on baseline age and blood pressure by matched-pair randomization.

Participants in the training condition (n = 25) completed 8 weeks of thrice weekly handgrip training at approximately 30% of their baseline maximal hand strength using a spring handgrip. Seated blood pressure and heart rate were assessed prior to each training session following 10 minutes of isolated rest. Training included 4, 2-minute contractions separated by 2 minutes of rest and completed bilaterally. Control participants (n = 24) completed weekly-seated measurements of blood pressure and heart rate following 10 minutes of isolated rest. Following 8 weeks of intervention all participants completed 3 sessions of post-training measurements.

In trained participants, resting systolic and diastolic pressure decreased significantly from pre- to post- (SBP: 122 ± 3 mmHg to 112 ± 3 mmHg, DBP: 70 ± 1

mmHg to 67 ± 1 mmHg) while heart rate remained unaltered. Pulse pressure was significantly reduced with handgrip training, from 52 ± 3 mmHg to 45 ± 3 mmHg. Control participants demonstrated no changes in blood pressure, heart rate and pulse pressure, over the course of the study.

In conclusion, spring handgrip training results in significant decreases in systolic, diastolic and pulse pressure. The mechanisms behind these improvements remain unknown and require further investigation.

ACKNOWLEDGEMENTS

I would like to thank my committee members, Neil McCartney, Maureen MacDonald and Steve Bray, all of whom helped me achieve this goal.

To my supervisor, Neil McCartney, thank you for the opportunity to work and learn from you. I appreciate the space and flexibility I have been given to study areas of personal interest. The fishing slide shows always seemed to be at the right time and helped alleviate stress and let me refocus on my objectives. The past 2 years has been an incredible experience, and has only motivated me to strive for further success.

I would also like to extend my gratitude to Maureen MacDonald for allowing me to enter her lab and be a part of a great group of individuals. I would like to acknowledge the time she devoted to me so that I could be here today. I appreciate this investment on your part.

Last but not least, Steve Bray, who always had an open ear when I needed to vent or ask questions. Even with all of your own students, you always seemed to find time to talk or check up on things. You provided the encouragement needed to stay focused.

Thank you to my classmates, who helped guide me through the last 2 years. There was always someone there to discuss potential problems or just chat. I feel lucky to be surrounded by such an intelligent and easy-going group of students that undoubtedly made things easier for me.

I would also like to thank my parents and family who through it all provided the support necessary to continue. Through the qualities learned from them, I have been able to achieve these goals. I would like to thank these influential people in my life; Mom, Dad, Dave, Anne, AJ & DC, GPA and GMA, for any achievements are surely a reflection of your hard work and dedication.

I would also like to thank Laura, who through it all was by my side. You were the daily confidence booster I needed to get through the last few months. The sacrifices made to achieve this goal are truly recognized by you.

Table of Contents

List of Figures **pg.**

List of Tables **pg.**

List of Abbreviations

Advancement of Medical Instrumentation	AAMI
Angiotensin Converting Enzyme	ACE
Antidiuretic Hormone	ADH
Arterial Blood Pressure	ABP
Atrial Natriuretic Peptide	ANP
Autonomic Nervous System	ANS
British Hypertension Society	BHS
Cardiac Output	CO
Cardiovascular Disease	CVD
Diastolic Blood Pressure	DBP
European Society of Hypertension	ESH
Heart Rate	HR
Hypertension	HTN
International Society of Hypertension	ISH
Joint National Committee	JNC
Maximal Voluntary Contraction	MVC
Mean Arterial Pressure	MAP
Millimeters of Mercury	mmHg
Muscle Sympathetic Nerve Activity	MSNA
Post Exercise Hypotension	PEH
Pounds of Force	lbs
Pulse Pressure	PP
Reactive Oxygen Species	ROS
Stroke Volume	SV
Systolic Blood Pressure	SBP
Total Peripheral Resistance	TPR
World Health Organization	WHO

1.0 REVIEW OF LITERATURE

1.1 Introduction

Cardiovascular disease (CVD) was estimated to be responsible for the deaths of 75,000 Canadians in 2003 (Statistics Canada, 2005). CVD affects the heart and blood vessels, which distribute essential nutrients throughout the body. It is estimated that 80% of Canadians have at least one CVD risk factor while 11% have greater than three (Wielgosz et al., 2003).

One risk factor for CVD is hypertension (HTN). HTN is the chronic elevation of systolic and/or diastolic blood pressure above normal resting values. The percentage of individuals affected by hypertension worldwide is estimated to be approaching 30%, or close to 1 billion (Chobanian et al., 2003; Mackay & Mensah, 2004). While the prevalence rates for HTN are approaching 1 in 3 in the general population, the incidence rates for individuals above the age of fifty-five are as high as 90% (Vasan et al., 2002). HTN is especially dangerous due to its lack of symptoms and visible signs, and approximately 30% of individuals are unaware they possess HTN (Chobanian et al., 2003).

Currently, lifestyle modifications and pharmacological agents are used to control HTN (Fagard, 2005). While current treatments are effective, they leave many individuals at increased risk, as 47% of medicated hypertensives are estimated to have above normal blood pressure (Hajjer & Kotchen, 2003). In recent years, studies of isometric handgrip training have shown this type of exercise is associated with reductions in resting arterial

blood pressure (ABP) (Peters et al., 2005; Ray & Carrasco, 2000; Taylor et al., 2003; Wiley et al., 1992). The mechanisms responsible for these reductions are yet to be determined.

1.2 Summary and Purpose

The effects of CVD and hypertension on the global healthcare system are profound and costly. Isometric handgrip training has shown promise as a technique for reducing resting arterial blood pressure (Peters et al., 2005; Ray & Carrasco, 2000; Taylor et al., 2003; Wiley et al., 1992). Most of the previous isometric handgrip training studies were conducted using an expensive, programmable digital handgrip device. The main purpose of this investigation was to determine if handgrip training using an inexpensive, spring handgrip device would result in reductions in resting arterial blood pressure.

1.3 Blood Pressure

1.3.1 Definition

Blood pressure is defined as "a measure of pressure or force of the blood against the walls of the blood vessels" (Seeley, Stephens & Tate, 2000; Wilmore & Costill, 1999). It is measured in millimeters of mercury (mmHg), which is the force/pressure required to raise a column of mercury an equal number of millimeters. Blood pressure is represented by two values; systolic pressure and diastolic pressure, corresponding to the pressure/force exerted on the blood vessels during the contraction and rest phases of the

heart, respectively. An example of this would be a blood pressure of 120 mmHg / 80 mmHg, in which 120 mmHg represents systolic or maximal pressure and 80 mmHg, represents diastolic or the lowest arterial pressure.

Measurements derived from systolic and diastolic pressures include pulse pressure and mean arterial pressure. Pulse pressure (PP) is the difference between systolic and diastolic pressure. Mean arterial pressure (MAP) is the principal measure in blood pressure regulation and is the average pressure (systolic and diastolic) in the aorta. The calculation for MAP takes in to account the proportional time of systole versus diastole. Common values for MAP are approximately 90-100 mmHg (Guyton et al., 1996).

Two main factors that determine blood pressure are the amount of blood being pumped from the heart (flow) and the opposing resistance in the vessel to the flow. These two factors are termed cardiac output (CO) and total peripheral resistance (TPR), respectively. CO is defined as the amount of blood pumped by the left ventricle per minute and is the product of heart rate (HR) and stroke volume (SV). TPR is the total resistance of all the body's vessels to blood flow. Resistance, under normal circumstances is mostly governed by the manipulation of a vessel's cross-sectional area. An example of the association between CO and TPR is the vasodilation of a vessel resulting in an increased diameter; this effect would cause a dramatic decrease in resistance and an increase in flow.

The ability of the vessel wall to expand and recoil passively (Δ volume) in response to changes in pressure is termed the compliance of the vessel. Vessel radius has

an impact on resistance and flow and therefore blood pressure. High compliance values are indicative of large increases in vessel volume for minimal increases in corresponding blood pressure. Vascular compliance tends to decrease with age as vascular elastic properties decrease and atherosclerosis progresses. Other factors influencing vessel state include nervous and metabolic mechanisms responsible for vasodilation and constriction.

The control of blood pressure involves a variety of mechanisms allowing for acute and long-term regulation. The redundancy of control factors allows for the maintenance of MAP during rest and peak stress.

1.3.2 Blood Pressure Values and Regulation

1.3.2.1 Normal Values

Blood pressure is influenced by multiple factors at any given time causing the standard deviation of repeated measures to be high. The "normal" adult resting blood pressure is approximately 120 mmHg systolic and 80 mmHg diastolic, as determined by the Joint National Committee on prevention, detection, evaluation, and treatment of high blood pressure (7^{th} report) and British Hypertension Society (Chobanian et al., 2003; Williams et al., 2004).

Systolic blood pressure (SBP) increases with age. Age associated increases in SBP have been linked to alterations in the vessels, such as stiffening of the arteries, and not due to changes in functional demand (Asmar et al., 1995; Avolio et al., 1985). In the absence of overt disease diastolic blood pressure (DBP) tends to increase until approximately 60 years of age, thereafter it appears to remain stable (Divine, 2006).

Blood pressure varies throughout the body and typical values in select vessels can differ due to numerous disease processes. In healthy individuals, right and left brachial artery pressures would be expected to be within 10 mmHg of each other. It is normal for the right arm to be slightly lower due to its distance from the heart. Blood pressures in the lower extremities when measured in the supine condition are normally ± 10% of values found in the upper body (Aronow, 2005; Wills, 2000). The effects of hydrostatic pressure on blood pressure can lead to variability of measurements. The position of the upper arm above or below the right atrium can cause alterations in blood pressure greater then 10 mmHg (Pickering et al., 2005). Similarly, changes in body position, from supine to upright can cause further inconsistency in blood pressure measurements. In certain conditions such as peripheral arterial disease or aortic coarctation large differences may exist between selected vessels.

Similar to variations in blood pressure within the body, there exist marked cyclic fluctuations in resting blood pressure during the day and year. Typically, resting blood pressure decreases during the night concomitant with a down regulation in sympathetic nervous activity (Chobanian et al., 2003). Upon waking, individuals commonly undergo a morning surge in ABP of 25-35 mmHg which is dependent on initial resting pressure, age and daily temperature (Modesti et al., 2006). This surge in blood pressure is associated with increased risk of cardiovascular events in the morning (Muller et al., 1989). In addition, this morning surge is usually largest on Monday mornings versus other days of the week due to psychological stressors (Murakami et al., 2004).

Akin to these acute changes are the cyclic fluctuations caused by climate and in particular outdoor temperature. It has been demonstrated that blood pressure tends to undergo a reduction (~ 5-10 mmHg) during warmer summer months (Brennan et al., 1982; Goodwin et al., 2001; Sega et al., 1998; Woodhouse et al., 1993). More recently, daily temperatures have been shown to exhibit the same effect. Modesti and colleagues (2006) found cold daily temperatures were linked to increased systolic daily pressure while hot nightly temperatures were associated with increased systolic night time pressure (Modesti et al., 2006). All of these temperature related fluctuations are increasingly more profound with advancing age and evident in medicated hypertensives (Goodwin et al., 2001; Modesti et al., 2006). Temperature appears to be a factor in observed increases in cardiovascular mortality and morbidity during the winter months (Donaldson et al., 1997; van Rossum et al., 2001). These acute and cyclic changes make it essential to utilize a control group when longitudinally examining resting arterial blood pressure, to account for these confounding factors.

1.3.3 Regulation

The human body is able to withstand many environmental and physical stressors. Blood pressure changes as the result of exercise, temperature variations or emergency situations are regulated by a variety of unconscious systems. Blood pressure changes can be regulated locally or by the central cardiovascular centres in the brain.

The autonomic nervous system (ANS) can stimulate involuntary responses to smooth muscle, cardiac muscle and numerous glands. The ANS is divided into the

parasympathetic and sympathetic divisions. These opposing divisions regulate rest and exercise functions, respectively. The innervation of vascular smooth muscle and cardiac muscle provide an avenue through which the nervous system can affect blood pressure through changes in heart rate, cardiac contractility and vessel diameter. These changes are regulated by the nervous system through the cardioregulatory and vasomotor centres located in the medulla oblongata (Seeley, Stephens & Tate, 2000). Parasympathetic stimulation leads to local vasodilation of vasculature and decreased heart rate primarily from the medulla and through the vagus nerve, while sympathetic activation causes vasoconstriction and an increased heart rate through the innervation of a wide network of sympathetic nervous system neurons. Parasympathetic and sympathetic systems can be regulated through either increased activation or decreased stimulation to maintain a consistent MAP.

The vasomotor centre is found in the reticular formation of the brain and is responsible for vascular tone via its efferent connections with smooth muscle cells (Brubaker, Kaminsky & Whaley, 2002). Smooth muscle is located in the tunica media of the vessel (Seeley, Stephens & Tate, 2000). Under resting conditions smooth muscle elicits a relatively constant tension, termed smooth muscle tone or vasomotor tone via the translation of afferent impulses from various sensors located throughout the vasculature which maintains MAP relatively constant.

The cardioregulatory centre is located in the medulla oblongata and exhibits control over both the sympathetic and parasympathetic modulation of heart rate and cardiac contractility. This is accomplished directly through the innervation of the cardiac

nerves and vagus nerve. Indirectly, sympathetic nervous system stimulated release of norepinephrine and epinephrine increases heart rate and contractility. Conversely, parasympathetic release of acetylcholine leads to decreased heart rate (Seeley, Stephens & Tate, 2000). These mechanisms allow for the rapid adjustment of blood pressure.

In addition to nervous system regulation of blood pressure there exist both short and long-term alternative mechanisms in the maintenance of adequate pressure and flow values. These regulatory mechanisms operate by changing either peripheral resistance or cardiac output in an attempt to maintain sufficient ABP. Normal blood pressure regulation is contingent upon the healthy status of the required pathways and tissues.

1.3.2.1 Short Term Regulation

The basis of short-term blood pressure regulation is to elicit a quick response to changes in pressure in order to maintain homeostatic values. There are four major pathways under neural control and maintenance by which blood pressure can be quickly adjusted.

The first mechanism for short-term blood pressure regulation involves the baroreceptors. Baroreceptors are mechanical stretch sensors located in the walls of atria, venae cava, aortic arch and the carotid sinuses. Baroreceptors detect alterations in vessel diameter caused by blood pressure fluctuations, typically between the blood pressure ranges of 60 to 180 mmHg (Kirchheim, 1976). Deformation of these mechanoreceptors results in the initiation of neural signals to the cardioregulatory and vasomotor centres in the medulla that activate the carotid sinus and aortic arch reflexes. In response to an

increase in MAP, baroreceptors send afferent signals to the nucleus tractus solitarius, located in the dorsal medulla. The medullary response is the inhibition of medulla related vasoconstriction and activation of the vagus nerve. The combined effects of decreased medullary vasoconstriction and vagal vasodilation lead to a reduction in TPR. At the same time, parasympathetic stimulation by the vagus nerve results in decreased heart rate and contractility, causing a reduction in CO. The end product of these responses is the reduction of MAP. Lohmeier and colleagues (2004) have demonstrated that carotid sinus stimulation significantly reduced blood pressure during angiotensin II induced hypertension (Lohmeier et al., 2005; Lohmeier et al., 2004). Similarly, reduced MAP causes a reduction in baroreceptor stimulation and results in increased sympathetic nervous activity and parasympathetic withdrawal. This manifests as increased heart rate, contractility and vasoconstriction of the vasculature, resulting in the restoration of MAP. Baroreceptors respond almost instantaneously to alterations in ABP and are responsible for the maintenance of ABP during postural changes.

The second mechanism regulating short-term BP is the adrenal medullary pathway, which is active during increased sympathetic activation. Sympathetic nervous system outflow stimulates the adrenal medulla via direct innervation, to release epinephrine and norepinephrine into the blood stream. These hormones result in short-term increases in heart rate and stroke volume while constricting peripheral vessels and dilating cardiac vessels. The result of this pathway is a systemic increase in MAP.

A third short term regulatory mechanism of blood pressure is the chemoreceptor reflex. Chemoreceptors are located in the carotid and aortic bodies and are sensitive to

changes in oxygen, carbon dioxide and pH concentrations (Guyton et al., 1996). Chemoreceptors are only activated during periods of strenuous exercise or medical emergencies (Guyton et al., 1996). The chemoreceptors are influential during periods of low blood pressure when oxygen levels may be diminished due to decreased pulmonary flow. The result of this detection by the chemoreceptors is vasoconstriction of the peripheral blood vessels, leading to increased systemic resistance and pulmonary flow in an effort to stabilize oxygen levels.

The final mechanism of short term ABP regulation is the central nervous system ischemic response of the medulla oblongata. This response is caused by a lack of blood flow to the medulla oblongata causing increased concentrations of carbon dioxide and hydrogen ions. The result is potent sympathetic vasoconstriction of the peripheral vessels, in some cases to the point of occlusion. This stimulation causes a dramatic increase in systemic arterial pressure and increased blood flow to the medulla in order to clear the high ion concentrations. This pathway is only initiated once pressure falls below 50-60 mmHg and blood flow to the brain is near a detrimental level (Seeley, Stephens & Tate, 2000).

In cases of HTN, these regulatory mechanisms may be altered or damaged. Adaptations to baroreceptor sensitivity and sympathetic nervous activity are commonly observed in hypertensive individuals (Bakris & Mensah, 2002; Julius & Schork, 1978). These changes can cause permanent alterations to the regulatory pathways leading to chronic elevation of resting ABP.

1.3.3.2 Long Term Regulation

The long-term regulation of resting blood pressure is accomplished through the interaction of several systems via hormonal and sympathetic stimulation. These systems include: the renin-angiotensin-aldosterone system, antidiuretic hormone, and the atrial natriuretic system.

The renal system is the starting point for the renin-angiotensin-aldosterone mechanism. During reductions in MAP, renal stimulation from sympathetic nerves causes renin to be released into the blood stream. Renin converts angiotensin, an inactivated circulating hormone, to angiotensin I. In the lungs, angiotensin I is thereby converted to angiotensin II by angiotensin converting enzyme (ACE). ACE is primarily produced in the lungs and found in the pulmonary capillaries, a smaller amount of ACE is produced locally by vascular endothelial cells (Bakris & Mensah, 2002; Guyton et al., 1996). Angiotensin II is a potent acute vasoconstrictor. Long-term exposure to Angiotensin II is also responsible for the stimulated release of aldosterone from the adrenal cortex, which triggers the kidneys to increase water reabsorption and decrease urine production. Angiotensin II formation also stimulates antidiuretic hormone (ADH) secretion. The result of these two hormones is the increase or maintenance in MAP, and is crucial in conditions of distress.

Another mechanism for long-term regulation is antidiuretic hormone (ADH), which is released upon detection of low plasma solute concentrations or blood pressure by the hypothalamus (Guyton et al., 1996). The hypothalamus in turn stimulates the posterior pituitary to secrete ADH. Circulating ADH results in vasoconstriction and

water reabsorption in the kidneys in an attempt to reestablish normal MAP values (Bakris & Mensah, 2002).

A third hormonal blood pressure regulatory pathway originates from atrial cardiac muscle cells, which release atrial natriuretic peptide (ANP) upon stimulation by stretch receptors, as with increased venous return. ANP acts on the kidneys to increase or decrease sodium and water concentrations in conjunction with altered MAP values.

It is evident that the human body possesses multiple regulatory measures for maintaining long-term MAP. It is crucial that during periods of exertion or emergency that the body is able to both act in the short and long-term to prevent the deleterious effects of chronically low or high blood pressure.

1.3.4 Measurement of Blood Pressure

Methods of blood pressure measurements are outlined in the criteria established by the Joint National Committee seventh report (JNC7). These guidelines state that all patients undergo at least 5 minutes of seated rest prior to any measurements. During this time patients should not speak and both feet should be on the floor. Following this period of relaxation all measurements should be made with the arm at or near heart level to ensure an accurate representation of blood pressure relative to the heart. With supine measurements, a similar rest period should be observed with the measured limb(s) relaxed at the patient's side. All measurements should use an appropriate sized cuff to avoid unreliable readings (Chobanian et al., 2003).

The measurement of blood pressure can be affected by many factors; one common condition known to affect this measurement is "white coat hypertension" (Angeli et al., 2005). This condition is usually observed in a clinician's office and results in an abnormal elevation of resting blood pressure. This elevation can be caused by the up-regulation of stress hormones brought about by the location and/or purpose of the visit. Clinical diagnosis of hypertension should only follow repeated measurements over an extended period of time (3-6 months) to accurately detail each individual's typical resting blood pressure (Table 1. Diagnostic criteria).

1.3.4.1 Manual Measurement

Arterial blood pressure (ABP) can be measured manually with the use of a sphygmomanometer and stethoscope. This method is commonly referred to as the auscultatory method as it uses auditory signals to determine ABP. A sphygmomanometer is a visual display representing the pressure in millimeters of mercury and can be electronic or mercury based. The sphygmomanometer is attached to the blood pressure cuff to provide pressure readings as the cuff is inflated.

To measure BP the cuff is placed over the upper arm of the individual, at least 1 inch above the antecubital space (Brubaker, Kaminsky & Whaley, 2002). A stethoscope is placed over the brachial artery of the same arm distal to the cuff. The cuff is inflated until the blood flow thru the brachial artery is completely occluded as determined by the absence of sound with the stethoscope. A typical inflation pressure is approximately 200mmHg. Finally, the pressure is progressively lowered, when blood flow returns to the

brachial artery the corresponding pressure is deemed the systolic pressure. During this time the flow will be heard as turbulent, once the pressure is sufficiently lowered to normal levels the pressure at which the turbulent sounds cease is deemed the diastolic pressure. This method of detection utilizes Korotkoff sounds or the sounds heard through the stethoscope. These sounds correspond to the pressure within an artery as described by the Russian physician Nikolai S. Korotkoff (1874-1920) (Seeley, Stephens & Tate, 2000). The 1st Korotkoff sound occurs during the contraction of the ventricles as the mitral and tricuspid valves (atrioventricular valves) close and are an indication of systolic blood pressure. The 2nd Korotkoff sound corresponds to the closure of the aortic and pulmonary valves (Brubaker, Kaminsky & Whaley, 2002). This method has the potential for human error but is consistently demonstrated to be reliable among experienced clinicians.

1.3.4.2 Automated Measurement

There are many different commercial brands of automated ABP acquisition systems. Typically, these systems use one of two detection methods, oscillometry or auscultation for detection. Automated systems provide quick measurements of ABP with no prior knowledge required for detection. These devices have been shown to provide conflicting ABP values and may not be representative of true ABP in every situation (Bailey & Bauer, 1993).

The oscillometry and auscultation methods both utilize a cuff to occlude blood flow. The oscillometry method also makes use of a pressure transducer within the

occlusion cuff. As the cuff is deflated the transducer records the oscillations in pressure, the monitoring system subsequently smoothes these data and employs arithmetic calculations to determine accurate pressure oscillations. The auscultation method again makes use of an occlusion cuff with the addition of a stethoscope. This automatic method is closely related to the manual method as blood pressure is determined from the detection of the Korotkoff sounds.

1.3.4.3 Other Techniques – Invasive methods

Invasive ABP assessment is usually accomplished by inserting a catheter with a pressure transducer in the artery of interest. Catheterization produces the most accurate pressure values but also involves the greatest risk to the subject. One caveat to this measurement is that values tend to be higher and more accurate then those determined via non-invasive methods (Beers & Berkow, 1999). This method is not commonly utilized in surgical procedures and is mainly a research tool.

1.3.4.4 Validation of Blood Pressure Measurement

Currently three main bodies, the Association for the Advancement of Medical Instrumentation (AAMI), the British Hypertension Society (BHS), and the European Society of Hypertension (ESH) are producing standards to test the accuracy of automated blood pressure monitors. Though the use of automated blood pressure acquisition devices is widespread, the validity of the results obtained with these devices has not been firmly established. The conditions provided by the AAMI, BHS and ESH provide the

criteria whereby systems may be developed to ensure accurate readings within a target population. The ESH method is the most recent and takes the AAMI and BHS methods into account. This validation procedure is completed in a 3-step process whereby measurements are taken manually and by the device in question in an alternating fashion for a given number of measurements. Validity is assessed based upon the difference between the manual and automated measured readings (O'Brien et al., 2002). An example of a validity report is available in appendix D.

1.4 Hypertension

1.4.1 Types/Causes/Epidemiology

Hypertension is estimated to affect nearly 1 billion people worldwide (Chobanian et al., 2003). Though the cause of hypertension is debated, most experts believe the root is multifactorial. Approximately 85-95% of individuals with HTN are considered to be essential or primary hypertensives, or those whom HTN is caused by unidentified or debated factors. The remaining 10-15% are considered to have secondary hypertension (Beers & Berkow, 1999; Chobanian et al., 2003; Divine, 2006), which results from an underlying condition such as sleep apnea, renal disease, fibromuscular dysplasia, adrenal hyperfunctioning or coarctation of the aorta.

Primary hypertension currently exists in two documented forms; the first being isolated hypertension or a tendency for pressure to be elevated in either the systolic or diastolic phases. This condition is diagnosed by repeated measures with systolic values > 140 mmHg or diastolic values > 90 mmHg. In past years these numbers have been

revisited and values > 130 mmHg systolic or > 90 mmHg diastolic are now considered adequate diagnostic criteria (Chobanian et al., 2003). The second form of essential hypertension involves both diastolic and systolic pressures that are chronically elevated above recommended values. Table 1 provides blood pressure classifications for adult diagnosis of hypertension (Chobanian et al., 2003; Divine, 2006).

According to this classification scheme individuals displaying blood pressure values of 120-130 mmHg systolic and 80-90 mmHg diastolic are termed to have 'Prehypertension' (Chobanian et al., 2003). It is estimated that individuals with prehypertension have a 90% chance of developing hypertension within their lifetime (Chobanian et al., 2003; Vasan et al., 2002).

Table 1. Classification of Blood Pressure for Adults.

Blood Pressure Classification	SBP (mmHg)	DBP (mmHg)
Normal	<120	and <80
Pre-hypertension	120-139	or 80-89
Stage 1 Hypertension	140-159	or 90-99
Stage 2 Hypertension	>160	or >100

SBP, Systolic blood pressure; DBP, Diastolic blood pressure

(Chobanian et al., 2003)

Some of the predisposing factors that have been linked to hypertension include: age, race, genetic background, environment and lifestyle. Hypertension prevalence increases markedly past 40-50 years of age (Chobanian et al., 2003, Vasan et al., 2002). Similarly, it is well established that race differences impact the prevalence of developing hypertension. Currently, a disproportionate number of African American adults possess

hypertension when compared to Caucasian and Mexican adults (Bakris & Mensah, 2002; Bosworth et al., 2006; Wong et al., 2002). This trend is most likely a combination of genetic and environmental factors (Bakris & Mensah, 2002; Bosworth et al., 2006). This genetic predisposition to hypertension is further impacted by the fact that hypertensive individuals have increased susceptibility to environmental stressors (Bosworth et al., 2006). The likelihood of developing hypertension is far greater if an individual has an affected first degree relative (Naber & Siffert, 2004; Staessen et al., 2003). The genetic component is believed to account for approximately 30-60% of the variability in hypertensives (Bakris & Mensah, 2002; Staessen et al., 2003).

Theories describing the cause of hypertension involve changes to cellular sodium transport, sympathetic nervous system stimulation, the renin-angiotensin-aldosterone system and/or potential vasodilator deficiencies (Beers & Berkow, 1999). Increased vasoconstriction via altered sympathetic activity or renal functioning has been identified in hypertensive individuals (Bakris & Mensah, 2002; Lohmeier, 2001; Mancia et al., 1999). Similarly, maladaptive baroreceptor functioning has been demonstrated in hypertensives, possibly leading to improper regulation of renal sympathetic activity (Lohmeier, 2001). Hypertension can result in changes to the vasculature through endothelial damage, remodeling or plaque formation. These vascular changes can lead to increases in TPR as the result of decreased arterial compliance (Bakris & Mensah, 2002). The cause of hypertension is difficult to ascertain, especially considering the majority of these adaptations may be due to the chronically elevated blood pressure and not solely the result of one root cause or deficiency. The most probable model is the mosaic theory,

which outlines that numerous accumulative factors result in the observed elevation of resting arterial blood pressure (Chobanian et al., 2003).

1.4.2 Risks of Untreated Hypertension

It is currently estimated that 30% of individuals with hypertensive are unaware of their condition (Beers & Berkow, 1999). This large proportion of individuals combined with those not responding to treatments present a large group susceptible to the increased mortality and morbidity associated with hypertension. Hypertension has been associated with 4 main disease states and drastically increases the risk of all-cause mortality. The 4 conditions in question are: heart disease, stroke, retinopathy and renal disease/failure (Brubaker, Kaminsky & Whaley, 2002).

As outlined in the introduction, cardiovascular disease is the leading cause of worldwide mortality. Hypertension is a major risk factor in the progression of CVD as increases in resting blood pressure by approximately 20/10 mmHg are associated with a doubling of cardiovascular risk (Chobanian et al., 2003). Damage done to the vessels in the brain or retina can lead to stroke or vision change/blindness, respectively. With respect to kidney function, high blood pressure is the leading cause of permanent kidney failure.

Hypertension has also been linked to the acceleration of atherosclerosis through increased arterial stiffness (Safar et al., 2003; Tomiyama et al., 2004; Wilmore & Costill, 1999). In a recent study, accelerated arterial stiffness was observed in high-normal (>130/85 mmHg) male subjects and blood pressure values above (140/90 mmHg) did not

demonstrate further increases in indices of stiffness (Tomiyama et al., 2006). The change in arterial stiffness has been directly related to a greater risk of cardiovascular events (Laurent et al., 2001).

Hypertension has also been linked to increases in left ventricular mass through thickening of the left ventricular wall, which reduces valuable cavity space. Left ventricular hypertrophy is a commonly used measure of hypertension severity (Susimoto et al., 1991). In severe cases, left ventricular hypertrophy will cause a reduction in stroke volume necessitating increased force of contraction and eventually leading to heart failure. In some individuals, hypertension proceeds rapidly causing a hypertensive crisis as tissue and vessels undergo damage from this dramatic increase in blood pressure.

All of the negative effects caused by untreated or uncontrolled hypertension are even more alarming as most of these conditions further increases in blood pressure and underscores the necessity for effective and safe blood pressure treatment(s).

1.5 Effects of Aerobic Training

The Joint National Committee on Detection, Evaluation and Treatment of High Blood Pressure (JNC), World Health Organization (WHO) and International Society of Hypertension (ISH) all recommend physical activity as a modifiable method to reduce elevated blood pressure. They advocate both aerobic and resistance training components, with the main focus on aerobic training (Chobanian et al., 2003).

The ability of chronic aerobic exercise to have a positive effect on resting blood pressure is well documented. In 1961, The Framingham Heart study demonstrated that

physical activity reduced the risk of heart disease (Kannel et al., 1961). Since then, numerous studies have shown that habitual exercise can decrease systolic and diastolic blood pressure over time (Braith et al., 1994; Jennings et al., 1986; Paffenbarger et al., 1983). Typically, research on physical activity and blood pressure has used epidemiological methods to investigate a large number of participants (Paffenbarger et al., 1983). More recently, longitudinal training studies have shown smaller reductions in ABP (Braith et al., 1994; Jennings et al., 1986). A meta-analysis of 44 randomized control trials using aerobic exercise interventions displayed overall reduction of 3.4 mmHg SBP and 2.4 mmHg DBP, respectively (Fagard, 2005). Larger reductions in ABP for individuals with higher initial values were also noted. Reductions of 7.4 mmHg SBP and 5.8 mmHg DBP were observed in a meta-analysis of aerobic exercise interventions among hypertensive subjects (SBP >140 mmHg, DBP >90 mmHg) (Fagard, 2001). Hypertensive individuals appear to achieve greater blood pressure reductions in response to habitual aerobic exercise. Inherently, these individuals do possess higher ABP and therefore should have a greater potential for improvement (Birkenhager, Reid & Bulpitt, 2000).

The reductions in blood pressure observed through aerobic exercise are likely an alteration of many pathways. Potential pathways affected with aerobic training include reductions in sympathetic activity (Pescatello et al., 2004), changes to the renin-angiotensin-aldosterone system (Chobanian et al., 2003), and improvements in endothelial function (Wilmore & Costill, 1999).

1.6 Effects of Resistance Training

Resistance exercise is characterized by fluctuations in dynamic and static contractions. The ability of chronic resistance exercise to reduce resting blood pressure is currently disputed due to an inability by the literature to produce consistent and repeatable results. In some cases an effective strength training routine has been demonstrated to improve and control blood pressure (Katz & Wilson, 1992; Stone et al., 1991). In contrast, numerous studies have not been able to reproduce any effects of resistance training on reducing blood pressure (Blumenthal, Siegel & Appelbaum, 1991; Cononie et al., 1991; Dunstan et al., 1998; Harris & Holly, 1987). In 2000, Kelley and Kelley examined eleven resistance-training studies and observed a reduction of 3 mmHg in both systolic and diastolic pressure (Kelley & Kelley, 2000). Of note, one problem with this meta-analysis is the fact that 57% of the subjects in those studies were described as "active", which may have led to lower-than-expected results. In contrast, Van Hoof and colleagues (1996) conducted a 16-week randomized control trial of 30 sedentary men which observed no significant alterations in blood pressure and heart rate (Van Hoof et al., 1996). While the actual reduction(s) may be modest they could potentially translate into a large reduction in CVD risk.

Resistance training studies have demonstrated the ability to increase muscle cross-sectional area, which is associated with increased muscle capillary density (Wilmore & Costill, 1999). The increase in vascular bed cross-sectional area results in decreased TPR (Wilmore & Costill, 1999). A decrease in TPR may be achieved through reducing the release and effects of vasoconstrictive stress hormones (Wilmore & Costill,

1999) and/or improvements in endothelial function (Rakobowchuk et al., 2005). The effects of resistance training on arterial compliance are still not determined. A randomized intervention study involving 28 males demonstrated a reduction in carotid arterial compliance following 4 months of resistance training (Miyachi et al., 2004). More recently, carotid cross-sectional compliance remained unchanged after 12 weeks of resistance training (Rakobowchuk et al., 2005). It may be that arterial compliance might only be altered following a long-term resistance program (>12 weeks).

The JNC recommends resistance training should be done as part of a regular exercise routine in conjunction with aerobic exercise to combat hypertension (Chobanian et al., 2003). They stress resistance training must be conducted properly to ensure maximum benefits. Currently, further research needs to be undertaken before the true benefits of resistance exercise for HTN are understood. At the present time, resistance training is only recommended as a supplement to aerobic exercise and not a modality to undertake by itself.

1.7 Effects of Isometric Training

Isometric or static exercise is defined as a muscle contraction in which the length of the muscle does not change but the tension produced increases (Seeley, Stephens & Tate, 2000). This form of exercise was widely popular during the nineteenth and early twentieth centuries. Early interest focused on the differences between isometric (static) and resistance training. One key difference is the ability of isometric exercise to occlude blood flow at low intensity levels (~20% MVC), thus initiating the metaboreflex in an

attempt to restore blood flow (Barcroft & Millen, 1939; Mark et al., 1985). The blood pressure and heart rate responses to isometric exercise are influenced by the force of the contraction (Seals, 1993), the size of the contracting muscle (Mitchell et al., 1980) and the length of time contracted (MacDougall et al., 1985). In contrast to resistance training, isometric training elicits a pressure load on the heart.

Two early studies examining the effects of isometric exercise on the incidence of hypertension demonstrated lower hypertension rates for individuals performing occupational isometric contractions (Buck & Donner, 1985; Kiveloff & Huber, 1971). These studies were conducted in both medicated and unmedicated individuals of varying resting arterial pressures and were predictive of the potential ability of handgrip training to reduce resting arterial blood pressure and/or lower hypertension incidence (Buck & Donner, 1985; Kiveloff & Huber, 1971).

More recently, Wiley and colleagues (1992) used isometric training to demonstrate 13 mmHg and 15 mmHg reductions in systolic and diastolic pressure, respectively (Wiley et al., 1992). Participants completed four, 2-minute contractions separated by 3-minute rest periods at 30% MVC, $3d.wk^{-1}$ for 8-weeks. In an accompanying study, Wiley and colleagues (1992) trained participants with four 45-second contractions separated by 1-minute rest periods at 50% MVC. These participants trained $5d.wk^{-1}$ for 5-weeks. The results demonstrated 9.5 and 8.9 mmHg reductions in resting systolic and diastolic pressure, respectively (Wiley et al., 1992).

An investigation to determine if possible changes in sympathetic nerve stimulation were responsible for the observed reductions in resting blood pressure was

conducted by Ray and Carrasco (2000). Normotensive participants trained 4 times per week for 5 weeks. They completed 4 contractions of 3-minutes each at 30% MVC, with each contraction separated by 5-minute rest periods. The training produced a 5 mmHg reduction in diastolic pressure and a 4 mmHg reduction in mean arterial pressure (Ray & Carrasco, 2000). The extent of sympathetic stimulation was estimated by efferent muscle sympathetic nerve activity (MSNA) and no significant changes were observed with training (Ray & Carrasco, 2000).

More recently, isometric handgrip training in hypertensive participants produced reductions in systolic and mean arterial pressures, decreased sympathetic stimulation and increased vagal modulation (Taylor et al., 2003). This protocol mimicked that of Wiley and colleagues (1992) and produced a SBP reduction of 19 mmHg, an 11 mmHg reduction in mean arterial pressure and a 7 mmHg decrease in DBP. Of note, the control group in this study demonstrated significant reductions in SBP and MAP of 8 mmHg and 5 mmHg, respectively (Taylor et al., 2003). In a recent study, 10 pre-hypertensive participants achieved an average reduction of 13 mmHg in systolic pressure following 6 weeks of $3d.wk^{-1}$ at 50% of MVC isometric handgrip training (Peters et al., 2005). These blood pressure alterations were combined with reductions in exercise-induced reactive oxygen species.

In contrast to these findings, 8 weeks of isometric handgrip training produced no significant changes in resting blood pressure in antihypertensive medicated participants (Levy et al., 2005). They did, however, observe trends towards decreases in systolic blood pressure (Levy et al., 2005). The contrasting results of these studies may serve as

an example how investigations on blood pressure must possess the utmost controls to limit potentially confounding variables, such as familiarization, measurement error, and collection times.

1.8 Isometric Handgrip Training

1.8.1 Mechanisms for Reductions in Arterial Blood Pressure

The proposed mechanisms behind the reductions in resting arterial blood pressure with isometric handgrip training include alterations to the sympathetic nervous system, endothelial function, oxidative stress, arterial compliance and/or the baroreceptors. To date conclusive examination of these potential mechanisms has not been completed.

The findings of Ray and Carrasco (2000), which revealed no significant changes in sympathetic stimulation, led them to theorize that handgrip exercise may have caused acute increases in MSNA and norepinephrine that blunted vascular sensitivity to norepinephrine (Kingwell et al., 1996; Ray & Carrasco, 2000). This is unlikely the cause, as participants did not demonstrate any attenuation in the arterial pressure response or MSNA with acute handgrip exercise, post-training. Contrary to previous work, the inability of handgrip training to reduce systolic blood pressure led Ray and Carrasco (2000) to propose that reductions occur through changes in the peripheral vascular system. They hypothesized that improvements in endothelial function resulted in increased release of nitric oxide, a potent vasodilator that is impaired in hypertensive individuals. Taylor and colleagues (2003) displayed similar heart rate variability data as previous endurance training studies (Pagani et al., 1988). The handgrip training resulted

in a decrease in sympathetic and increase in parasympathetic modulation as assessed by blood pressure variability (Taylor et al., 2003). This change in autonomic modulation may have altered the sympathovagal balance and led to the changes in resting blood pressure demonstrated with training.

A second potential mechanism that may account for the improvement in ABP with handgrip training is alterations in endothelial function. Improved local endothelial function has recently been demonstrated in medicated hypertensives with bi-lateral isometric handgrip training (McGowan et al., 2004). Sinoway and colleagues (1987) have shown that 4-weeks of handgrip training leads to an increased vasodilatory response and maximal blood flow in the localized tissues. In conjunction with these increases, studies have observed decreases in peripheral resistance (Sinoway et al., 1987; Yasuda & Miyamura, 1983). No changes in peripheral resistance were observed in the participants untrained control arms. In a similar study, normotensive subjects displayed no changes in endothelial function or vascular remodeling with 8 weeks of unilateral handgrip training (McGowan et al., 2005, McGowan et al., 2006). This evidence suggests endothelial function is an unlikely mechanism, as observed changes in endothelial function were only local to the trained limb and not found systemically.

Another possible mechanism for the reduction in ABP is through alterations in oxidative stress induced by handgrip training. Peters et al (2005) observed a large reduction of oxygen centred radicals (-266%), in combination with increased whole body glutathione (+61%) in hypertensive individuals following 6 weeks of handgrip training (Peters et al., 2005). The effects of handgrip training were also hypothesized to be

caused by a reduced stimulation of chemoreceptors in the muscle (Somers et al., 1992). This theory was developed from results describing an isometric training induced attenuation of decreasing pH (Kent-Braun et al., 1990). These results support the theory that isometric training produces an attenuated oxidative stress response to exercise and at rest. This attenuation in oxidative stress could lead to an improvement of systemic endothelial function.

Alterations in arterial compliance have been identified as a potential mechanism for reduced ABP. Increased arterial compliance could reduce TPR leading to the observed improvements in ABP. Previously, increased arterial compliance has been associated with decreases in pulse pressure (Franklin et al., 1997). Recently, aerobic and handgrip training have demonstrated no alterations in aortic or carotid stiffness respectively (Ferrier et al., 2001; Levy et al., 2005). In the case of handgrip training, changes in pulse pressure may be caused by the alterations in ABP or cardiac output and not vascular changes.

Another potential mechanism for the attenuation in resting blood pressure is through the alteration of the baroreceptors. Pagani and colleagues (1988) demonstrated potential baroreceptor resetting with endurance training in mild hypertensives (Pagani et al., 1988). Similarly, the decrease in cardiovagal baroreflex sensitivity associated with age has been attenuated with aerobic exercise (Monahan et al., 2001). The baroreceptors may also exhibit increased sensitivity due to the changes in endothelial function and/or arterial compliance (Kingwell et al., 1995; Monahan et al., 2001). This may also explain

the altered sympathetic activity due to decreased stimulation by the autonomic nervous system.

To date the exact mechanism(s) for the observed reductions in resting arterial blood pressure have not been conclusively identified. Further research examining potential alterations in sympathetic activity, endothelial function, reactive oxygen species, arterial compliance and/or baroreceptor functioning need to be conducted to correctly understand the observed improvements with handgrip training.

1.9 Hypothesis

Although the mechanism(s) behind the reduction in resting arterial pressure with handgrip training remains in question, the current literature provides numerous examples of this positive association (Peters et al., 2005; Ray & Carrasco, 2000; Taylor et al., 2003; Wiley et al., 1992). One major limitation of the literature to date is the over reliance on expensive, digital devices for handgrip training. Similarly, these studies all utilized rigid training protocols intended for the digital handgrips. These limitations restricted individuals who were unable to obtain or purchase digital handgrips from potentially benefiting from handgrip training. With these limitations in mind, the main hypothesis for this investigation is that 8 weeks of isometric handgrip training, utilizing inexpensive, spring-loaded trainers will result in significant reductions in resting arterial pressure. Secondary to the main hypothesis, this investigation sought to establish that rigid workload requirements of 30% MVC are no more effective than workloads approximating 20-40% MVC in eliciting similar benefits.

2.0 Methods

2.1 Subjects

2.1.1 Participant Demographics

The sample population was derived from the Centre for Health Promotion and Rehabilitation at McMaster University, Hamilton, Canada. All were patients of the MacSenior or MacTurtle exercise programs. Participants were recruited with the aid of posters approved by the McMaster Research Ethics Board (MREB). Subjects were informed of the purpose, procedures, time requirements and benefits upon request. If individuals still showed interest in participation, a visit was scheduled to obtain informed consent. Participant demographics are outlined in Table 4 in section 3.1, Subject Characteristics.

2.1.2 Inclusion Criteria

The sample population consisted of males and females of any age, although exercise program requirements provided subjects above fifty years of age. The MacSenior exercise program has a minimum age of 60, while the MacTurtle program requires that patients have undergone a cardiac event (e.g. myocardial infarction, coronary bypass, angina). These programs are offered to spouses of patients even if they do not meet all of the normal requirements to join.

2.1.3 Exclusion Criteria

Participants were primarily excluded if they were currently taking any antihypertensive medications. Secondary exclusion criteria included diabetes, congestive heart failure, smoking or individual's currently undergoing hormone replacement therapy. Smoking was defined as having smoked a cigarette within 5 years of the study's commencement. Subjects who possessed chronic physical limitations with respect to hand strength or gripping ability (e.g. arthritis) were also excluded. Participants unwilling to abstain from alcohol and caffeine twelve hours prior to each session were also excluded from participating. Only subjects able to commit to the full study length were accepted.

2.1.4 Patient Health Information

All participants fitting the described inclusion/exclusion criteria provided further information from a participant health questionnaire and previous medical documentation. Specifically, this information provided full names, age, current prescribed medications and past medical surgeries and procedures. Participant health questionnaires were completed prior to the study's inception and ensured investigators that all inclusion/exclusion criteria were truthfully met. Information required as part of their participation in the MacSenior/MacTurtle exercise programs provided current documentation from their physician about their medical histories. This information was not fully collected until the completion of the study. Inclusive patient demographics are provided in section 3.1, subject characteristics.

2.2 Design and Intervention

2.2.1 Design

This 10-week study was a stratified matched-pair design, with subjects being stratified according to resting arterial pressure and age into an intervention or control group. All data were collected between February 2006 and May 2006. Patient demographics were collected prior to and following the study's completion. Health demographics were obtained with consent from exercise stress tests and medical records held by the Centre for Health Promotion and Rehabilitation, McMaster University. All testing was completed at the Centre for Health Promotion and Rehabilitation, McMaster University, prior to participation in their twice-weekly exercise programs (MacSenior or MacTurtle). All participants completed 2 familiarization visits prior to testing to adequately demonstrate required procedures. Participants completed 1 week each of pre- and post- measures in addition to the 8 weeks of training. Pre- and post- measures were done over 3 sessions, during approximately 1 week. Participants were asked to maintain consistent scheduled appointments (± 1 hour) to limit changes in resting arterial pressure due to daily fluctuations. Dietary intake information was collected during pre- and post-sessions, with emphasis placed on quantity. Subjects were asked to avoid vigorous exercise for a 24-hour period prior to all sessions.

2.2.2 Intervention

Participants completed 3 visits prior to and following the intervention period to assess pre- and post- resting arterial pressure. Participants in the training group

completed 8-weeks of thrice-weekly handgrip training. Training was completed with the aid of a spring handgrip device (Bodyflux Corp., Richmond Hill, Canada), available in multiple resistance tensions. Handgrip strength was established prior to training, with each individual's training resistance corresponding to 30-50% of their maximum voluntary contraction (MVC). Each individual was provided his/her own handgrip and stop clock to complete all training. Maximum hand strength was determined in each individual with the aid of a hand force transducer (ADInstruments Inc., Colorado Springs, CO, USA).

Subjects were required to complete two sessions of training per week at the Centre for Health Promotion and Rehabilitation and one session per week at home. Resting blood pressure was measured during each of the two visits to the Centre per week. Participants unable to fulfill these weekly requirements due to personal or business commitments were permitted to continue training 3 times per week at home or their current location. Figure 1 illustrates the design of the training protocol.

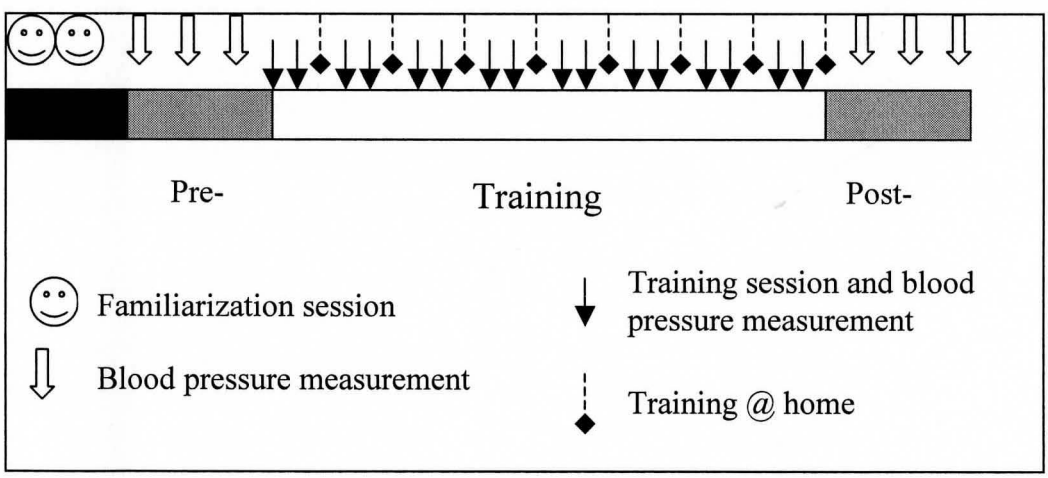

Figure 1. Schematic outlining the training protocol design.

2.2.3 Control

The remaining study participants were allotted to the control group. These control participants did the same three pre- and post-training visits as the intervention group, and were required to complete one visit per week to the Centre for Health Promotion and Rehabilitation, for resting arterial pressure measurements. These participants continued their twice weekly exercise (MacSenior/MacTurtle) and therefore participant interaction with the author was similar to that of the exercise group. This group was used to monitor fluctuations in blood pressure over the study period, in those with similar characteristics as the training group. Participants unable to attend a weekly visit due to absence or travel were either rescheduled or brought in the following week for an extra visit. Figure 2 illustrates the design of the control protocol.

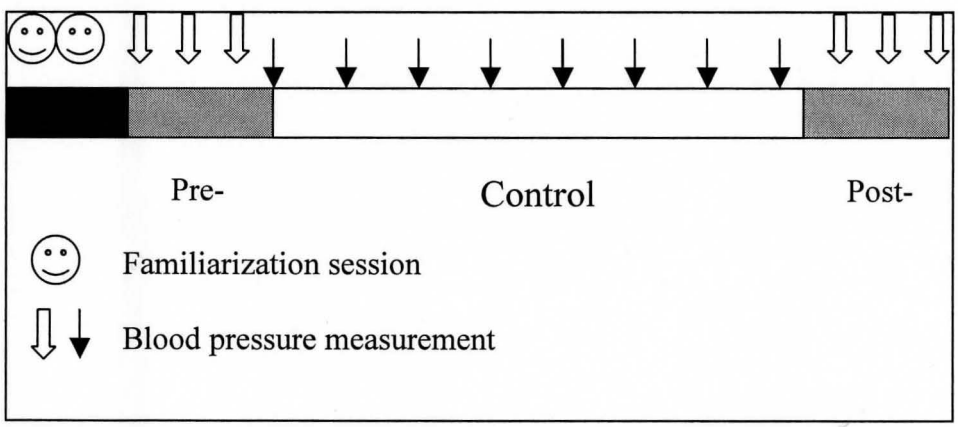

Figure 2. Schematic outlining the control protocol design.

2.3 Testing Protocol

All visits for training participants contained measurements of resting arterial pressure and completion of handgrip exercise while each at home session included only

the training protocol. During each visit to McMaster University training subjects underwent the protocol for measurement of resting arterial pressure followed by 5-10 minutes of a general warm-up (treadmill/bike/arm cycle). At this time participants completed their training protocol as outlined below (Figure 1, Table 3).

Control participants were required to complete 1 visit per week for the measurement of resting arterial pressure (Figure 2). The measurement protocol is outlined in the following section.

2.3.1 Resting Arterial Blood Pressure Measurement

Control and intervention participants were subjected to 52 and 88 measures of resting arterial pressure, respectively, over the study period, 4 measures during each visit. For measurement of resting arterial pressure, subjects were directed to a quiet solitary, temperature-controlled room where they rested for 10 minutes in a seated position. Subjects sat upright in their chair with all restrictive clothing removed and were instructed to abstain from crossing their legs or feet. Meditation was permitted. Each subject was accurately fitted for the appropriate cuff size, with this cuff being maintained over all measures. The cuff was placed approximately 2-3 cm proximal to the cubital fossa (PAHI, 2003). Following this period 4 arterial measures were taken over each subject's left brachial artery with the aid of a calibrated Dinamap automatic acquisition system (Pro 100 V2, Critikon Corp., Tampa, FL, USA). Each measurement was separated by 30 seconds. During measurement each subject's left arm was passively held

at heart level. All resting arterial pressure measurements were taken by the same individual and Dinamap system to ensure consistency.

Determination of each sessions resting arterial pressure was done with the omission of the first measurement and the average of the remaining three. Pre- and post-training resting arterial pressures were thus compiled by the mean of nine values each, three per visit.

2.3.2 Maximum Hand Strength

Following the third pre-training and the first post-training visits maximum hand strength was obtained from all subjects. This was accomplished through the use of a hand dynamometer (MLT003/D, ADInstruments Inc., Colorado Springs, CO, USA), Powerlab acquisition system (Model ML825, ADInstruments Inc., Colorado Springs, CO, USA) and Chart 5 software (ADInstruments Inc., Colorado Springs, CO, USA). The hand dynamometer produced outputs in millivolts (mV) which were converted to a measure of force in Newtons (Conversion: 2.3702mV/V/100N). The Newton unit describes the net force required to move an object (Net Force = Mass x Acceleration). These values were converted to pounds of force with a conversion equation of 1 Newton = 0.2248 pounds of force.

Demonstrations of the dynamometer were completed just prior to testing. Subjects completed 3 maximal contractions with the dynamometer in each hand in a standing position. All subjects began contractions in their right hand, following acceptable warm-up and practice contractions. The dynamometer was equipped with a

foam grip to provide comfort. The investigator advised the subject when to contract and relax. Contractions were held for approximately 2-3 seconds with 3-5 second rest periods provided between contractions. Figure 6 (Appendix D) depicts a series of contractions typically observed in all participants.

Maximum values were obtained from each contraction. Calculation of 30% and 50% of each contraction was completed following testing in the pre-training visit (Table 4). The 30-50% of maximum was used to determine the model of handgrip and therefore the resistance tension each subject in the intervention group would utilize for training.

2.3.3 Isometric Hand Training

Training was accomplished with the aid of spring handgrip devices (Bodyflux Corp., Richmond Hill, ON, Canada) which were available in 3 different spring lengths, 4 mm, 4.5 mm, 5 mm which corresponded to the green, red and blue gripper handles. These spring lengths were measured with the aid of a load cell to establish the pounds of force required to half and fully close the device. Table 2 outlines the measured force required for each of the 3 handgrip models. Each subject completed 24 training sessions. Prior to each exercise session, participants had resting arterial blood pressure measured as previously described, followed by a general warm-up and/or stretching of their hands and fingers. Subjects were permitted to wear gloves or wrap the handle in a foam pad to alleviate any uncomfortable sensations caused by the plastic handle.

Table 2. Handgrip Tensions.

Hand Grip Colour	Spring Length (millimeters)	½ Closure (Pounds of Force)	Full Closure (Pounds of Force)
Green	4 mm	15	24
Red	4.5 mm	20	33
Blue	5 mm	31	51

With respect to the handgrip, individuals were instructed to maintain a 1.5 inch gap in handles and maintain this distance for each contraction. This distance corresponded to closure of the handgrip half-way and these instructions were emphasized during training with the aid of visual markers. Various handgrips were re-examined post-training to assess possible reductions in tensions caused by habitual training.

The training protocol consisted of four 2-minute isometric contractions completed equally with both hands. Contractions alternated between hands (i.e. right → left → right → left) with a 1-minute rest period between each squeeze. Therefore, the total training time was 12-minutes, with 8-minutes of contraction. This training protocol was the same as previous handgrip training studies (Taylor et al., 2003; Wiley et al., 1992). All participants completed the first contraction with their dominant hand. All training sessions at McMaster University (2x/wk) were completed with the aid of a study investigator or trained volunteer to ensure accurate completion of these protocols. All participants were provided written outlines of the protocol. Table 3 outlines the protocol and corresponding times.

Table 3. Isometric handgrip training protocol.

Step	Description
1	2-minute isometric contraction with dominant hand
2	1-minute of rest
3	2-minute isometric contraction with non-dominant hand
4	1-minute of rest
5	2-minute isometric contraction with dominant hand
6	1-minute of rest
7	2-minute isometric contraction with non-dominant hand
8	1-minute of rest

2.4 Statistical Analysis

Participant data were examined pre- to post- using a two-factor (Group x Time) analysis of variance (ANOVA) with repeated measures on the second factor. Analyses were completed for pre- to post- resting arterial pressures, pulse pressure and heart rate (Statistica 5.1, Statsoft Inc., Tulsa, OK, USA). All significant differences were further analyzed using Tukey post hoc procedures. Individual and group reproducibility was determined by coefficients of variation (CV) and intra-class correlation coefficients (ICC). Statistical significance was demonstrated by an alpha level of < 0.05. All data are represented as the mean ± the standard error of the mean (SEM), unless otherwise stated.

3.0 Results

3.1 Subject Characteristics

Fifty-three participants enrolled in the study and 49 completed study protocols. The main reason for dropout was "lack of available time". In total, 25 individuals participated in the isometric handgrip training program with the remaining 24 making up the control group. The average age of this sample was 66.4 ± 6.2 years, ranging from 47 to 80 years. The sample consisted of 21 male and 28 females. Further information regarding age, gender, baseline blood pressure and baseline hand strength are described in Table 4. Of note 48 participants (n = 24 and n = 24) completed pre-/post- measurements, as 1 female training participant was unable to complete the post-training testing sessions. See Appendix A for complete participant demographics.

Table 4. Baseline Participant Characteristics.

	Exercise	**Control**	**Total**
Number of Subjects (*n*)	25	24	49
Female	11	17	28
Male	14	7	21
Age (years)	66 ± 1	67 ± 2	66 ± 1
SBP (mmHg)	122 ± 3	117 ± 3	119 ± 2
DBP (mmHg)	70 ± 1	68 ± 2	69 ± 1
Pulse Pressure (mmHg)	52 ± 3	50 ± 2	51 ± 2
R-max (Lbs)	60.3 ± 3.9	44.5 ± 3.9	53.3 ± 3.0
L-max (Lbs)	60.6 ± 4.2	45.3 ± 3.6	53.6 ± 3.0

All values expressed as means ± SEM; mmHg, millimeters of mercury; R-max, Right hand maximal hand strength, n=46; L-max, Left hand maximal hand strength, n=46; Lbs, Pounds of force

3.2 Effects of Training on Cardiovascular Measures

3.2.1 Systolic Blood Pressure

Handgrip training resulted in decreases in resting systolic blood pressure in the training group while systolic blood pressure was unaltered in the control group (Table 5, figure 3).

Table 5. Effects of Training on Systolic Blood Pressure.

	Pre-	Post-	Change (±)
SBP			
Training	122 ± 3	112 ± 3	-10 ± 3
Control	117 ± 3	118 ± 3	1 ± 3

All measures in mmHg

3.2.2 Diastolic Blood Pressure

Handgrip training resulted in decreases in resting diastolic blood pressure in the training group while diastolic blood pressure was unaltered in the control group (Table 6, figure 4).

Table 6. Effects of Training on Diastolic Blood Pressure.

	Pre-	Post-	Change (±)
DBP			
Training	70 ± 1	67 ± 1	-3 ± 1
Control	68 ± 2	68 ± 1	0 ± 1

All measures in mmHg

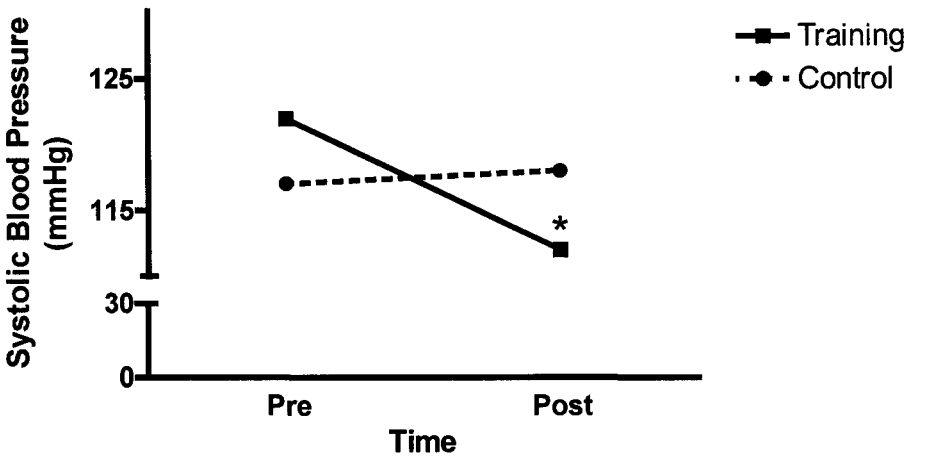

Figure 3. Effects of Isometric Handgrip Training on Systolic Blood Pressure . Data presented as mean only for clarity purposes ; *, significantly different from Pre - (Interaction effect, $p<0.01$)

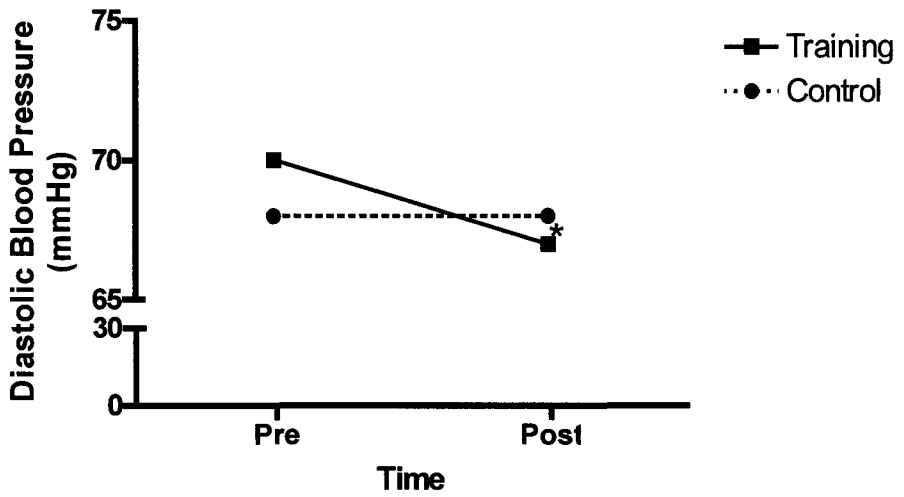

Figure 4. Effects of Isometric Handgrip Training on Diastolic Blood Pressure . Data presented as mean only for clarity purposes ; *, significantly different from Pre - (Interaction effect, $p<0.05$)

3.2.3 Pulse Pressure

Isometric handgrip training resulted in a decrease in pulse pressure in the training group while pulse pressure was unaltered in the control group (Table 7, figure 5).

Table 7. Effects of Training on Pulse Pressure.

	Pre-	**Post-**	**Change (±)**
PP			
Training	52 ± 3	45 ± 3	-7 ± 3
Control	50 ± 2	50 ± 2	0 ± 2

All measures in mmHg

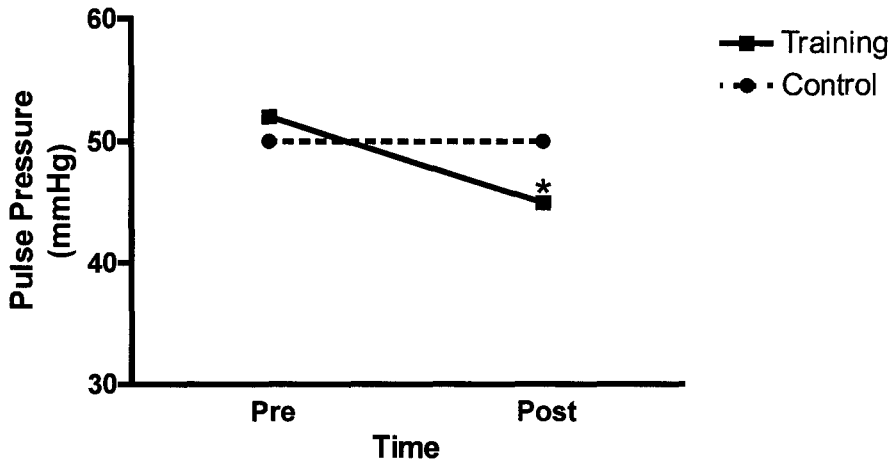

Figure 5. Effects of Isometric Handgrip Training on Pulse Pressure.
Data presented as mean only for clarity purposes; *, significantly different from Pre - (Interaction effect, $p<0.01$)

3.2.4 Heart Rate

Heart rate was unaltered over the 8-weeks of the study in either the training or control groups (Table 8).

Table 8. Effects of Training on Heart Rate.

	Pre-	Post-	Change (±)
HR			
Training	69 ± 2	70 ± 2	0 ± 1
Control	71 ± 2	72 ± 2	1 ± 1

All measures in beats per minute

3.3 Effects of Training on Handgrip Resistance

The manual spring-loaded handgrips were assessed with a load cell before and after the 8 week training intervention to determine if 8 weeks of use would result in alterations in mechanical spring resistance. There was no variation in resistance as a result of the 8 week protocol. See Appendix D for pre- and post- measured handgrip resistance.

3.4 Reproducibility of Cardiovascular Measures

The reproducibility of systolic blood pressure was determined using the coefficient of variation (CV) and intra-class correlation coefficients (ICC). Handgrip training resulted in a decrease in the CV of systolic blood pressure measurements in the training group (3.0 % ± 0.3 to 2.5% ± 0.2). Diastolic blood pressure and heart rate showed no significant changes in the training or control groups (Table 9). Intraclass

correlation coefficients were calculated for all participants ($n = 49$) and for the control and intervention groups, individually. In terms of all participants, SBP reproducibility was highest following the 8-week protocol (0.91 vs. 0.81) regardless of intervention participation. The intraclass correlation coefficient for DBP in all participants remained consistent in the pre- and post- measurement sessions (0.80 vs. 0.77). For all intraclass correlation coefficients refer to Table 10.

Table 9. Effects of Training on the Variation of Cardiovascular Measurements.

CV (%)		
	Pre-	**Post-**
SBP		
Training	3.4 ± 0.3	2.5 ± 0.2
Control	3.0 ± 0.2	3.3 ± 0.2
DBP		
Training	2.3 ± 0.3	2.4 ± 0.3
Control	2.3 ± 0.2	2.8 ± 0.2
HR		
Training	2.6 ± 0.5	3.1 ± 0.9
Control	2.5 ± 0.2	2.4 ± 0.2

SBP, Systolic blood pressure; DBP, Diastolic blood pressure; HR, heart rate

Table 10. Intraclass Correlation Coefficients for Pre- and Post- Resting Arterial Measurements.

	Pre- Measurements	**Post- Measurements**
SBP		
All Participants	0.81	0.91
Training	0.87	0.92
Control	0.74	0.91
DBP		
All Participants	0.80	0.77
Training	0.80	0.75
Control	0.80	0.79

SBP, Systolic blood pressure; DBP, Diastolic blood pressure; Pre- and Post-, 3 measurements sessions each; All participants, $n = 49$; Training participants, $n = 25$, Control participants, $n = 24$.

4.0 DISCUSSION

In accordance with our primary hypothesis, 8-weeks of isometric handgrip training resulted in an overall reduction in resting ABP. As will be discussed below, these findings are comparable to those observed in previous reported studies (Peters et al., 2005; Taylor et al., 2003; Wiley et al., 1992). The secondary hypothesis was also supported by the data, as individuals training at approximately 30% of pre-training maximum hand strength (MVC) produced similar reductions in ABP as those seen in studies where individuals trained at exactly 30% of MVC for each training session.

4.1 Effects of Isometric Handgrip Training on Resting Arterial Pressure

The current study resulted in significant reductions in systolic and diastolic pressure after 8 weeks of handgrip training with a spring handgrip device. Resting systolic and diastolic blood pressure decreased by an average of 10 and 3 mmHg, respectively, in the training group. The observed attenuations in resting ABP are associated with a 20% reduction in cardiovascular disease risk (Chobanian et al., 2003) and can have a significant impact on participant health, as it may prevent the administration of pharmacological medications for HTN by their clinicians. These results are slightly smaller in magnitude than those shown in previous studies which reported reductions in SBP between 12-19 mmHg (Peters et al., 2005; Taylor et al., 2003; Wiley et al., 1992) and DBP of 5 mmHg (Ray & Carrasco, 2000). Nevertheless, the present study included only normotensive participants whereas these other studies included

hypertensive individuals. It would seem reasonable to assume that the magnitude of reduction in resting ABP would be positively associated with initial resting levels.

In total, 24 out of 25 training participants observed reductions in SBP. This occurred even though all participants were habitually active and completing twice weekly aerobic and resistance training sessions in a fitness setting throughout the study. It would seem that handgrip training may have significant 'added value' potential in the treatment of hypertension, as recent reviews of endurance training and dietary modification have described reductions in SBP of 3.4 and 5.9 mmHg and DBP of 2.4 and 4.2 mmHg (Fagard, 2005) and a meta-analysis of resistance training interventions demonstrated modest reductions of 3 mmHg for both SBP and DBP (Kelley & Kelley, 2000).

As mentioned, observed reductions with the spring handgrip device are comparable to those utilizing the programmed, digital handgrip apparatus (McGowan et al., 2006; Taylor et al., 2003; Wiley et al., 1992) and to date exceed the reductions demonstrated by dietary modification, endurance training and resistance training. Thus, the effectiveness of spring handgrip training in comparison to alternative modalities demonstrates a significant tool in reducing resting ABP.

4.2 Prospective Mechanisms for the Reduction in Resting Arterial Pressure

The mechanism(s) behind the improvements in resting arterial blood pressure were not examined in this study but previous work has outlined several possibilities. Alterations in the modulation of the heart and ABP by the autonomic system may help to explain the observed results. Taylor and colleagues (2003) demonstrated reduced SBP

variability and improved heart rate variability as assessed by power spectral analysis. They suggested an alteration in sympathovagal balance, as determined by the decreased low frequency/high frequency ratio, as a potential mechanism for the 19 mmHg reduction in SBP (Taylor et al., 2003). The training participants demonstrated a significant decrease in low frequency area that is associated with sympathetic modulation (Taylor et al., 2003). Previously, an elevated low frequency component has been associated with hypertension (Pagani et al., 1988) and increased all-cause mortality (Bigger et al., 1992). Similarly, rhythmic handgripping at 30% MVC revealed an attenuation of muscle sympathetic nerve activity (MSNA) in young healthy participants (Sinoway et al., 1996; Somers et al., 1992). In contrast, Ray and Carrasco (2000) measured muscle sympathetic nerve activity (MSNA) in normotensive participants and found no alterations following training despite an observed 5 mmHg reduction in DBP (Ray & Carrasco, 2000). In contrast to Taylor et al (2003), Ray and Carrasco (2000) studied only young, normotensive participants. The possibility of training induced adaptations to the autonomic nervous system needs to be further studied in different populations

A second potential mechanism is the ability of handgrip training to improve endothelial function. The endothelium is essential for modulating vascular tone through the release of nitric oxide, and improvements in endothelial function would provide the vasculature with a greater ability to dilate in response to increased flow. McGowan and colleagues (2004) reported improved local endothelial function in medicated hypertensives following bilateral isometric handgrip training (McGowan et al., 2004). Conversely, a similar study showed no improvements in endothelial function in

normotensive participants undergoing unilateral handgrip training (McGowan et al., 2005). It is well recognized that hypertensive individuals suffer from endothelial dysfunction and more recently, exercise (aerobic or isometric) has demonstrated the potential to improve endothelial dependent dilation (Higashi et al., 1999a, b; McGowan et al., 2004; Taddei et al., 2006). It is thought that these local improvements are caused by an increase in vascular sheer stress during exercise (Ciampi et al., 2003). To date, benefits appear to be greatest in the hypertensive population, most likely because they generally possess the most dysfunction. A second potential cause for improvements in endothelial function and/or ABP is through decreases in oxidative stress. A recent study demonstrated a reduced production of reactive oxygen species (ROS) (-266%) and increased antioxidant production (+61%) following 6 weeks of handgrip training (Peters et al., 2005). Reactive oxygen species are thought to damage the endothelium, increasing the likelihood of dysfunction. Elevated ROS are also associated with impaired ventricular function in hypertensives (Higashi et al., 1999a; Vukasovic et al., 2005). This attenuation in exercise induced oxidative stress may help explain the improvements in endothelial function and/or ABP.

The potential alteration in arterial compliance with handgrip training is an additional mechanism that may explain the observed reduction in resting ABP. Previous research has demonstrated a link between pulse pressure and arterial compliance (Dart & Kingwell, 2001; Franklin et al., 1997). Franklin and colleagues (1997) observed that increases in PP with age were associated with decreases in arterial compliance (Franklin et al., 1997). Similarly, age related increases in PP have been associated with reduced

elasticity of the major vessels, particularly aortic stiffness (Avolio, Jones & Tafazzoli-Shadpour, 1998). The observed reduction in PP may be the result of decreases in ABP, cardiac output or increased arterial compliance (Dart & Kingwell, 2001). Previously, digital handgrip (Cardiogrip Corp., Boise, Idaho, USA) training utilizing an identical training protocol produced no variations in cardiac output (Levy et al., 2005) in medicated participants, who also demonstrated no alteration in resting arterial blood pressure. Levy et al (2005) also documented no change in aortic distensibility or stiffness (Levy et al., 2005). In the case of reductions in PP, increased arterial compliance may be the potential adaptation responsible for the improvement in ABP.

Another potential pathway for alteration is through the modulation of the baroreceptors. If training elicits a new "set point" for blood pressure or improved sensitivity, the baroreceptors could limit sympathetic outflow to skeletal muscle (Joyner, 2006). In previous work, activation of the carotid baroreceptors elicited marked reductions in blood pressure with no changes in heart rate (Epstein et al., 1969), observations that are similar to the results of the present study. The relationship between aortic stiffness and baroreflex functioning suggests a potential link between arterial compliance and baroreceptor reflex activity (Kingwell et al., 1995; Monahan et al., 2001). In contrast, the link between reduced vascular stiffness and baroreceptor function has also produced insignificant results (Hunt, Farquhar, & Taylor, 2001). Hunt and colleagues (2001) described a neural component for control of baroreflex gain, as reduced arterial stiffness with age did not adequately explain the gains in older, physically active males (Hunt, Farquhar, & Taylor, 2001). This finding is similar to that

of Osborn and colleagues (2005) who concluded that a neural set point exists for long term MAP with acute modulation accomplished by the baroreflexes (Osborn, Jacob, & Guzman, 2005). Therefore, any potential improvement in arterial compliance may be evidence for an improvement in acute baroreceptor sensitivity. The fact that participants were already physically active at least twice per week (aerobic + resistance), may point to the ability of handgrip training to elicit further adaptations beyond conventional endurance training alterations. These additional adaptations may be from increased baroreceptor sensitivity and/or neural adaptations.

4.3 Effects of Isometric Handgrip Training on Pulse Pressure

The 7 mmHg reduction in pulse pressure (PP) in the training group has interesting implications. An increased PP in older individuals is associated with a greater risk of mortality from cardiovascular disease. (Lee, Rosner & Weiss, 1999). Increased PP is also associated with endothelial damage and atherosclerosis (Dart & Kingwell, 2001). Moreover, PP has been related to increased carotid intima-media thickness and left ventricular mass index (Khattar et al., 1997), both of which are commonly elevated in hypertensive individuals. As mentioned previously, PP is closely associated with arterial compliance (Dart & Kingwell, 2001; Franklin et al., 1997) and alterations in ABP, cardiac output and arterial compliance can influence PP (Dart & Kingwell, 2001). In the present study, if cardiac output had decreased with training, then the observed reduction in PP could be attributed to a reduced SV. Nevertheless, it is established that chronic exercise training reduces arterial blood pressure primarily via a reduction of TPR and not

decreases in cardiac output (Pescatello et al., 2004). Therefore, in line with the results of Levy and colleagues (2005) it is unlikely that cardiac output was affected by handgrip training in the present study. If this is true, the reduction in PP could have resulted from increases in arterial compliance. It may be that a reduction in ABP is of itself an adequate stimulus for change in arterial compliance. This potential adaptation is speculative as cardiac output, stroke volume and arterial compliance were not measured in this study.

4.4 Effects of Isometric Handgrip Training on Resting Heart Rate

The current investigation demonstrated no significant changes in resting heart rate over the study duration in both the training and control groups (Section 3.4.4, Table 9). These results are in accordance with previous studies (Ray & Carrasco, 2000; Taylor et al., 2003; Wiley et al., 1992) of isometric handgrip training. The ability of the training stimulus to cause reductions in arterial blood pressure is apparent but this stimulus may not be appropriate to produce alterations in heart rate. This is not surprising as resting heart rate is primarily regulated via the parasympathetic system and previous studies of isometric handgrip training have demonstrated adaptations to the sympathetic system (Sinoway et al., 1996; Somers et al., 1992). These adaptations would have limited influence on resting heart rate.

4.5 Reproducibility of Cardiovascular Measures

The measurement of blood pressure is subject to influence by many environmental, physiological and psychological factors. To demonstrate reliability of our measurements, coefficients of variation (CV) were completed for resting SBP, DBP and HR, pre- and post-training (Appendix C). The CV measures the dispersion of measurements as a ± percentage of the mean. Intraclass correlation coefficients (ICC) were also completed for SBP and DBP, pre- and post-training. The ICC assessed the correlation within the pre- and post-testing sessions, to ensure measurements were reproducible over time. The CV and ICC were calculated for all participants ($n = 49$) and for the training ($n = 25$) and control ($n = 24$) groups independently. The mean CV for all measurements within the training and control group was <5% while the ICC was >0.74 (Section 3.5, Table 10 and Table 11). Interestingly, SBP in training participants experienced a decrease in CV and increase in ICC over the 8-week protocol that could represent enhanced familiarization and potentially, improved blood pressure regulation. It is likely that greater participant familiarization accounted for this change, as the control group also demonstrated enhanced SBP correlation in post-training measurements, following the 8-week protocol (Table 11). The ICC for DBP was consistent during the pre- and post- measurement sessions for all participants, 0.80 and 0.77, respectively. The ICC values for SBP and DBP coincide with the results for similar short-term ABP measurements (0.70-0.94) (van de Borne et al., 1997). The results of the CV and ICC analyses ensure that the collected baseline and post-training ABP measurements were statistically reproducible.

4.6 Study Benefits

4.6.1 Methods

In comparison to the literature, this investigation sought to improve upon previous methodology by utilizing a matched-pair design and large sample size. The use of a matched-pair design enabled us to pair participants according to age and resting arterial pressure. This pairing helped reduce the variability between the control and training groups. Unlike previous studies, the utilization of this method would provide a more uniform stratification of participants ensuring that both groups had similar characteristics, important to the primary outcome. With this in mind, the differences in pre-MVC between the control and training groups are irrelevant as the primary outcome is the effect of training on resting arterial pressure and not strength. A second advantage of this protocol was the large sample size (n = 49) which afforded this investigation sufficient power to detail an effect for changes that previous studies may have found to be not significant. This is significant as any reduction in resting arterial pressure coincides with health benefits. The purposeful alterations in methodology undoubtedly aided in producing clear and concise data.

A third difference in the methods is the omission of maximal contractions prior to each training session. The omission of these maximal contractions casts doubt on the potential explanation that reductions in resting arterial pressure are the result of repeated maximal contractions and not the handgrip training protocol. In the context of our current knowledge on mechanisms, the removal of a potential hypothesis is a noteworthy result.

4.6.2 Participants

An important finding of this investigation was that handgrip training resulted in a reduction in resting arterial pressure in participants who already perform weekly aerobic and resistance training exercise. This is potentially important as it may elucidate that the mechanisms behind these reductions may be different in handgrip training compared to aerobic training. An equally important aspect of the results is the status of the initial resting values of the participants. These individuals began training with normal resting arterial pressure (~120/70 mmHg) and training produced reductions. This suggests the improvements in resting arterial pressure are not necessarily linked to improvements in dysfunctional pathways (e.g., arterial compliance) seen in hypertensive individuals.

4.6.3 Practicality

Another compelling implication for the observed reductions in resting arterial pressure is the corresponding reductions in cardiovascular disease risk and all-cause mortality incurred through training. Any reduction in arterial pressure can have an effect on risk reduction but in the case of hypertensive individuals these reductions may prevent the need for antihypertensive medications which are commonly associated with negative side effects. The ability of handgrip training to provide improvements in multiple health outcomes with a minimal investment of time further underscores the benefits of this training modality.

4.7 Study Limitations

In an attempt to prevent confounding factors, cardiovascular measures were taken according to protocols outlined by the Joint National Committee's seventh report (JNC 7) (Chobanian et al., 2003). Nevertheless, failure to measure cardiac output, stroke volume, muscle sympathetic nerve activity, or other potentially adapted pathways prevented us from identifying the mechanisms for the reductions in resting arterial blood pressure.

4.8 Future Research

The ability of handgrip training to reduce resting arterial blood pressure has been confirmed in the present study. Future research should attempt to identify and understand the mechanisms behind the observed improvements.

As described, this training modality has been conducted previously on normotensive and medicated hypertensive individuals. One population that has still not been studied is unmedicated hypertensives. The effect of handgrip training on these individuals warrants examination to determine the full effectiveness of the intervention as a potential treatment in hypertension. A long term randomized control trial needs to examine the viability of this training as a chronic treatment or prevention method in comparison with pharmacological and lifestyle treatments.

Acutely, the effects of handgrip training need to be examined 0-24 hours following a handgrip exercise. The use of an ambulatory blood pressure acquisition system would allow for the determination and/or classification of a post-exercise hypotension effect.

Increasing research will hopefully provide the answers to the current questions and allow for a new effective treatment of hypertension.

4.9 Summary and Conclusion

In agreement with our main hypothesis, eight weeks of bilateral spring handgrip training produced significant reductions in resting arterial blood pressure. The average reduction of systolic and diastolic blood pressure by 10 and 3 mmHg translates to an approximate 20% reduction in risk for cardiovascular disease (Chobanian et al., 2003). Furthermore, handgrip training produced a significant reduction in resting pulse pressure of 7 mmHg. Conversely, the control group did not demonstrate any alterations in resting arterial blood pressure or pulse pressure over time. In line with previous studies, training and control groups did not demonstrate any alterations in resting heart rate (Ray & Carrasco, 2000; Taylor et al., 2003; Wiley et al., 1992).

The ability of this study to demonstrate similar results to previous handgrip studies will hopefully stimulate further investigation into the mechanism(s) responsible for the improvements in resting arterial blood pressure. The original studies utilizing the CardioGrip® hand trainer (CardioGrip Corp., Boise, Idaho, USA) produced viable results but the cost of the device may make it impracticable for widespread public prescription. The spring handgrip trainers now provide a secondary device available to individuals at risk for or demonstrating hypertension. These results also hint at the possibility that other devices such as stress/tennis balls or other objects may be used to produce similar results.

Though the mechanisms behind the observed attenuation of resting arterial blood pressure with training are undetermined, the positive results are evidence for the success of this training modality. The simple use and cost effectiveness of the spring handgrip in combination with these positive results will hopefully lead to handgrip training within rehabilitation centres focused on risk reduction.

5.0 References

Listed in alphabetical order

Angeli, F., Verdecchia, P., Gattobigio, R., Sardone, M. & Reboldi, M. (2005). White-coat hypertension in adults. *Blood Pressure Monitoring*, 10(6), 301-305.

Aronow, W.S. (2005). Management of peripheral arterial disease. *Cardiology in Review*, 13, 61-68.

Asmar, R., Benetos, A., Topouchian, J., Laurent, P., Pannier, B., Brisac, A.M., et al. (1995). Assessment of arterial distensibility by automatic pulse wave velocity measurement: validation and clinical application studies. *Hypertension*, 26, 485–490.

Avolio, A.P., Deng, F.Q., Li, W.Q., Luo, Y.F., Huang, Z.D., Xing, L.F., et al. (1985). Effects of aging on arterial distensibility in populations with high and low prevalence of hypertension: comparison between urban and rural communities in China. *Circulation*, 71, 202–210.

Avolio, A., Jones, D., & Tafazzoli-Shadpour, M. (1998). Quantification of alterations in structure and function of elastin in the arterial media. *Hypertension*, 32, 170 –175.

Bailey, R.H. & Bauer, J.H. (1993). Ambulatory blood pressure measurement. *Arch Intern Med*, 153, 2741.

Bakris, G.L. & Mensah, G.A. (2002). Pathogenesis and clinical physiology of hypertension. *Cardiol Clin*, 20, 195-206.

Barcroft, H. & Millen, J.L.E. (1939). The blood flow through muscle during sustained contraction. *J Physiol*, 97, 17-31.

Beers, M.H. & Berkow, R. (1999). *The Merck manual of diagnosis and therapy*. Ch. 16. Whitehouse Station, NJ, USA: Merck Research Laboratories

Bigger, J.T. Jr., Fleiss, J.L., Steinman, R.C., Rolnitzky, L.M., Kleiger, R.E. & Rottman, J.N. (1992). Frequency domain measures of heart period variability and mortality after myocardial infarction. *Circulation,* 85, 164–71.

Birkenhager, W.H., Reid, J.L. & Bulpitt, C.J. (2000). *Handbook of Hypertension*, Vol 20: Epidemiology of Hypertension. pp 191-211. Amsterdam: Elsevier.

Blumenthal, J.A., Siegel, W.C. & Appelbaum, M. (1991). Failure of exercise to reduce blood pressure in patients with mild hypertension. *JAMA,* 266, 2098–2104.

Bosworth, H.B., Dudley, T., Olsen, M.K., Voils, C.I., Powers, B., Goldstein, M.K., et al. (2006). Racial differences in blood pressure control: Potential explanatory factors. *Am J Med*, 119, 9-15.

Braith, R.W., Pollock, M.L., Lowenthal, D.T., Graves, J.E. & Limacher, M.C. (1994). Moderate-and high intensity exercise lowers blood pressure in normotensive subjects 60 to 79 years of age. *Am J Cardiol*, 73, 1124-1128.

Brennan, P.J., Greenberg, G., Mial, W.E. & Thompson, S.G. (1982). Seasonal variations in arterial blood pressure. *BMJ*, 285, 919-923.

Brubaker, P.H., Kaminsky, L.A. & Whaley, M.H. (2002). *Coronary artery disease: essentials of prevention and rehabilitation programs*. Ch 1, 2, 3, 4. Champaign, IL, USA: Human Kinetics.

Buck, C. & Donner, A.P. (1985). Isometric occupational exercise and the incidence of hypertension. *J Occup Med*, 27, 370-372.

Chobanian, A.V., Bakris, G.L., Black, H.R., Cushman, W.C., Green, L.A., Izzo, Jr, J.L., et al. (2003). Seventh report of the joint national committee on prevention, detection, evaluation, and treatment of high blood pressure. *Hypertension*, 42, 1206-1252.

Ciampi, Q., Betocchi, S., Violante, A., Lombardi, R., Losi, M.A., Storto, G., et al. (2003). Hemodynamic effects of isometric exercise in hypertrophic cardiomyopathy: comparison with normal subjects. J Nucl Cardiol, 10, 154–60.

Cononie, C.C., Graves, J.E., Pollock, M., Pollock, L., Phillips, M.I., Sumners, C. & Hagberg, J. M. (1991). Effect of exercise training on blood pressure in 70- to 79-yr-old men and women. *Med. Sci. Sports Exercise,* 23, 505–511.

Dart, A.M. & Kingwell, B.A. (2001). Pulse Pressure: A review of mechanisms and clinical relevance. *JACC*, 37(4), 975-984.

Divine, J.G. (2006). *Action plan for high blood pressure* (American College of Sports Medicine): Ch 1,2,4,5. Champaign, IL, USA.: Human Kinetics.

Donaldson, G.C., Robinson, D., Allaway, S.L. (1997). An analysis of arterial disease mortality and BUPA health screening data in men, in relation to outdoor temperature. *Clin Sci* (Lond), 92, 261-268.

Dunstan, D.W., Puddey, I.B., Beilin, L.J., Burke, V., Morton, A.R. & Stanton, K.G. (1998). Effects of a short-term circuit weight training program on glycaemic control in NIDDM. *Diabetes Res. Clin. Pract,* 40, 53–61.

Epstein, S.E., Beiser, G.D., Goldtein, R.E., Redwood, D., Rosing, D.R., Glick, G., et al. (1969). Treatment of angina pectoris by electrical stimulation of the carotid-sinus nerves. *N Eng J Med*, 280, 971-978.

Fagard, R.H. (2001). Exercise charateristics and the blood pressure response to dynamic physical training. *Med Sci Sports Exerc*, 33(6), 484-492.

Fagard, R.H. (2005). Effects of exercise, diet and their combination on blood pressure. *J Hum Hypertens*, 19, S20-S24.

Ferrier, K.E., Waddell, T.K., Gatzka, C.D., Cameron, J.D., Dart, A.M. & Kingwell, B.A. (2001). Aerobic exercise training does not modify large-artery compliance in isolated systolic hypertension. *Hypertension*, 38, 222-226.

Franklin, S., Gustin, W, IV., Wong, N., Larson, MG., Weber, MA., Kannel, WB., et al. (1997). Hemodynamic patterns of age-related changes in blood pressure: the Framingham heart study. *Circulation*, 96, 308-315.

Goodwin, J., Pearce, V.R., Taylor, R.S., Read, K.L. & Powers, S.J. (2001). Seasonal cold and circadian changes in blood pressure and physical activity in young and elderly people. *Age Ageing*, 30, 311-317.

Guyton, A.C. & Hall, J.E. (1996). *Textbook of medical physiology*. (9^{th} ed.) Philadelphia: W.B. Sanders.

Hajjar, I. & Kotchen, T.A. (2003). Trends in prevalence, awareness, treatment, and control of hypertension in the United States, 1988–2000. *JAMA*, 290, 199 –206.

Harris, K.A. & Holly, R.G. (1987). Physiological responses to circuit weight training in borderline hypertensive subjects. *Med. Sci. Sports Exercise,* 19, 246–252.

Higashi, Y., Sasaki, S., Kurisu, S., Yoshimizu, A., Sasaki, N., Matsuura, H., et al. (1999a). Regular aerobic exercise augments endothelium-dependent vascular relaxation in normotensive as well as hypertensive subjects: role of endothelium-derived nitric oxide. *Circulation*, 100, 1194-1202.

Higashi, Y., Sasaki, S., Sasaki, N., Nakagawa, K., Ueda, T., Yoshimizu, A., et al. (1999b). Daily aerobic exercise improves reactive hyperemia in patients with essential hypertension. *Hypertension*, 33, 591-597.

Hunt, B.E., Farquhar, W.B., & Taylor, J.A. (2001). Does reduced vascular stiffening fully explain preserved cardiovagal baroreflex function in older, physically active men? *Circulation,* 103, 2424-2427.

Jennings, G., Nelson, L., Nestel, P., Esler, M., Korner, P, Burton, D., et al. (1986). The effects of changes in physical activity on major cardiovascular risk factors, hemodynamics, sympathetic function, and glucose utilization in man: a controlled study of four levels of activity. *Circulation*, 73, 30–40.

Joyner, M.J. 2005. Baroreceptor function during exercise: resetting the record. *Exp Physiol*, 91(1), 27-36.

Julius, S. & Schork, M.A. (1978). Predictors of hypertension. *Ann NY Acad Sci*, 304, 38-58.

Kannel, W.B., Dawber, T.R., Kagan, A., Revotskien, N. & Stokes, J. 3rd. (1961). Factors of risk in the development of coronary heart disease--six year follow-up experience. The Framingham Study. Ann Intern Med, 55, 33-50.

Katz, J. & Wilson, B.R.A. (1992). The effects of a six-week, low intensity Nautilus circuit training program on resting blood pressure in females. *J Sports Med Phys Fitness*, 32, 299 –302.

Kelley, G.A. & Kelley, K.S. (2000). Progressive resistance exercise and resting blood pressure: A meta-analysis of randomized controlled trials. *Hypertension,* 35, 838-843.

Kent-Braun, J.A., McCully, K.K. & Chance, B. (1990). Metabolic effects of training in humans: a ^{31}P-MRS study. *J Appl Physiol*, 69, 1165-1170.

Khattar, R.S., Acharya, D.U., Kinsey, C., Senior, R., & Lahiri, A. (1997). Longitudinal association of ambulatory pulse pressure with left ventricular mass and vascular hypertrophy in essential hypertension. *J Hypertens*, 15, 737– 743.

Kingwell, B.A., Cameron, J.D., Gillies, K.J., Jennings, G.L., & Dart, A.M. (1995). Arterial compliance may influence baroreceptor function in athletes and hypertensives. *Am J Physiol*, 268, 411-418.

Kingwell, B.A., Tran, B., Cameron, J.D., Jennings, G.L. & Dart, A.M. (1996). Enhanced vasodilation to Ach in athletes is associated with lower plasma cholesterol. *Am J Physiol Heart Circ Physiol*, 270, H2008-H2013.

Kirchheim, H.R. (1976). Systemic arterial baroreceptor reflexes. *Physiol Rev*, 56, 100-177.

Kiveloff, B. & Huber, O. (1971). Brief maximal isometric exercise in hypertension. *J Am Geriatr Soc*, 19, 1006-1012.

Laurent, S., Boutouyrie, P., Asmar, R., Gautier, I., Laloux, B., Guize, L., et al. (2001). Aortic stiffness is an independent predictor of all-cause mortality and cardiovascular mortality in hypertensive patients. *Hypertension*, 37, 1236-1241.

Lee, M.L., Rosner, B.A. & Weiss, S.T. (1999). Relationship of blood pressure to cardiovascular death: the effects of pulse pressure in the elderly. *Ann Epidemiol*, 9, 101–107.

Levy, A.S., McGowan, C.L., Rakobowchuk, M., McCartney, N & MacDonald, M.J. Effects of 8-week isometric handgrip exercise on aortic distensibility and central cardiovascular responses. Ref Type: Unpublished work. 2005.

Lohmeier, T.E. (2001). The sympathetic nervous system and long-term blood pressure regulation. *Am J Hyperens*, 14, 147S-154S.

Lohmeier, T.E., Dwyer, T.M., Hildebrandt, D.A., Irwin, E.D., Rossing, M.A., Serdar, M.J., et al. (2005). Influence of prolonged baroreflex activation on arterial pressure in angiotensin hypertension. *Hypertension*, 46, 1194-1200.

Lohmeier, T.E., Irwin, E.D., Rossing, M.A., Serdar, M.J. & Kieval, R.S. (2004). Prolonged activation of the baroreflex produces sustained hypotension. *Hypertension*, 43, 306-311.

MacDougall, J.D., Tuxen, D., Dale, D., Moroz, J. & Sutton, J. (1985). Arterial blood pressure response to heavy resistance exercise. *J Appl Physiol*, 58, 785-790

Mackay, J. & Mensah, G. (2004). *The atlas of heart disease and stroke*. Ch. 1, 5. Switzerland: The World Health Organization.

Mancia, G., Grassi, G., Giannattasio, C. & Seravalle, G. (1999). Sympathetic activation in the pathogenesis of hypertension and progression of organ damage. *Hypertension*, 34, 724-728.

Mark, A.L., Victor, R.G., Nerhed, C. & Wallin, B.G. (1985). Microneurographic studies of the mechanisms of sympathetic nerve responses to static exercise in humans. *Circ Res*, 57, 461-469.

McGowan, C.L., Levy, A.S., McCartney, N. & MacDonald, M.J. Unilateral isometric handgrip training reduces blood pressure but does not improve brachial artery flow-mediated dilation in persons with normal blood pressure.
Ref type: Unpublished work. 2005.

McGowan, C.L., Levy, A.S., Millar, P.J., Guzman, J.C., Morillo, C.A., McCartney, N., et al. (2006). Acute vascular responses to isometric handgrip (IHG) exercise and the effects of training in persons medicated for hypertension. *Am J Physiol Heart Circ Physiol.* [Epub April 28]

McGowan, C.L., Visocchi, A., Faulkner, M., Rakobowchuk, M., McCartney, N. & MacDonald, M.J. (2004). Isometric handgrip training improves blood pressure and endothelial function in persons medicated for hypertension (Abstract). *The Physiologist*, 47, 285.

Mitchell, J.H., Payne, F.C., Saltin, B. & Schibye, B. (1980). The role of muscle mass in the cardiovascular response to static contractions. *J Physiol* (Lond), 309, 45-54.

Miyachi, M., Kawano, H., Sugawara, J., Takahasi, K., Hayashi, K., Yamazaki, K., et al. (2004). Unfavorable effects of resistance training on central arterial compliance: a randomized intervention study. *Cirulation*, 110, 2858-2863.

Modesti, P.A., Morabito, M., Bertolozzi, I., Massetti, L., Panci, G., Lumachi, C., et al. (2006). Weather-related changes in 24 hour blood pressure profile: Effect of age and implications for hypertension management. *Hypertension*, 47, 155-161.

Monahan, K.D., Tanaka, H., Dinenno, F.A., Handly, B. & Seals, D.R. (2001). Central arterial compliance is associated with age-and habitual exercise-related differences in cardiovagal baroreflex sensitivity. *Circulation*, 104, 1627-1632.

Muller, J.E., Tofler, G.H. & Stone, P.T. (1989). Circadian variation and triggers of onset of acute cardiovascular disease. *Circulation*, 79, 733-743.

Murakami, S., Otsuka, K., Kubo, Y., Shinagawa, M., Yamanaka, T., Ohkawa, S., et al. (2004). Repeated ambulatory monitoring reveal a Monday morning urge in blood pressure in a community-dwelling population. *Am J Hypertens*, 17, 1179-1183.

Naber, C.K. & Siffert, W. (2004). Genetics of human arterial hypertension. *Minerva Med*, 95, 347-356.

O'Brien, E., Pickering, T., Asmar, R., Myers, M., Parati, G., Staessen, J., et al. (2002). Working group on blood pressure monitoring of the European society of hypertension international protocol for validation of blood pressure measuring devices in adults. *Blood Pressure Monitoring*, 7, 3-17.

Osborn, J.W., Jacob, F. & Guzman, P. (2005). A neural set point for the long-term control of arterial pressure: beyond the arterial baroreceptor reflex. *Am J Physiol Regul Integr Comp Physiol*, 288, R846–R855.

Paffenbarger, R.S. Jr., Wing, A.L., Hyde, R.T. & Jung, D.L. (1983). Physical activity and incidence of hypertension in college alumni. *Am J Epidemiol*, 117, 245-257.

Pagani, M., Somers, V., Furlan, R., Dell'Orto, S., Conway, J., Baselli, G., et al. (1988). Changes in autonomic regulation induced by physical training in mild hypertension. *Hypertension*, 12(6), 600-610.

PAHI. Pan American Hypertension Initiative. (2003). Working meeting on blood pressure measurement: suggestions for measuring blood pressure to use in population surveys. *Pan Am J Public Health*, 14(5), 300-302.

Pescatello, L.S. (2005). Exercise and hypertension: recent advances in exercise prescription. *Curr Hypertens Rep*, 7(4), 281-286.

Pescatello, L.S., Franklin, B.A., Fagard, R., Farquhar, W.B., Kelley, G.A., Ray, C.A., et al. (2004). American College of Sports Medicine position stand. Exercise and hypertension. *Med Sci Sports Exerc*, 36, 533-553.

Peters, P.G., Alessio, H.M., Hagerman, A.E., Aston, T., Nagy, S. & Wiley, R.L. (2005). Short-term isometric exercise reduces systolic blood pressure in hypertensive adults: Possible role of reactive oxygen species (R1). *Int J Cardiol*. [Epub ahead of print].

Pickering, T.G. (2005). New guidelines on diet and blood pressure. *Hypertension*, 47, 135-136.

Rakobowchuk, M., McGowan, C.L., de Groot, P.C., Hartman, J.W., Phillips, S.M. & MacDonald, M.J. (2005). Endothelial function of young healthy males following whole body resistance training. *J Appl Physiol*, 98(6), 2185-2190.

Ray, C.A. & Carasco, D.I. (2000). Isometric handgrip training reduces arterial pressure at rest without changes in sympathetic nerve activity. *Am J Physiol Heart Circ Physiol*, 279, H245-H249.

Safar, M.E., Levy, B.I. & Struijker-Boudier, H. (2003). Current perspectives on arterial stiffness and pulse pressures in hypertension and cardiovascular diseases. *Circulation*, 107, 2864-2869.

Seals, D.R. (1993). Influence of force on muscle and skin sympathetic nerve activity during sustained isometric contractions in humans. *J Physiol* (Lond), 462, 147-159.

Seeley, R.R., Stephens, T.D. & Tate, P. (2000). *Anatomy & Physiology* (5th ed). Ch 16. Toronto, Canada.: McGraw-Hill.

Sega, R., Cesana, G., Bombelli, M., Grassi, G., Stella, M.L., Zanchetti, A., et al. (1998). Seasonal variations in home and ambulatory blood pressure in the PAMELA population. Presione Arteriose Monitorate e Loro Associazioni. *J Hypertens*, 16, 1585-1592.

Sinoway, L., Shenberger, J., Leaman, G., Zelis, R., Gray, K., Baily, R., et al. (1996). Forearm training attenuates sympathetic responses to prolonged rhythmic forearm exercise. *J Appl Physiol*, 81, 1778-1784.

Sinoway, L.I., Shenberger, J., Wilson, J., McLaughlin, D., Musch, T. & Zelis, R. (1987). A 30-day forearm work protocol increases maximal forearm blood flow. *J Appl Physiol*, 62(3), 1063-1067.

Somers, V.K., Leo, K.C., Shields, R., Clary, M. & Mark, A.L. (1992). Forearm endurance training attenuated sympathetic nerve response to isometric handgrip in normal humans. *J Appl Physiol*, 72(3), 1039-1043.

Staessen, J.A., Wang, J.G., Bianchi, G. & Birkenhager, W.H. (2003). Essential hypertension. *Lancet*, 361, 1629-1641.

Statistics Canada. (2005). Death Rate, 2003. Ottawa, Canada: Minister of Industry. Retrieved April 16, 2006 from http://www.statcan.ca/english/freepub/84F0209XIE/2003000/t001_en.htm
Ref Type: Internet Communication

Stillwell, S.B. & Randall, E.M. (1994). *Cardiovascular Care* (2nd ed). St. Louis, MO, USA: Mosby.

Stone, M.H., Fleck, S.J., Triplett, N.T. & Kraemer, W.J. (1991). Health and performance related potential of resistance training. *Sports Med,* 11, 210–231.

Taddei, S., Virdis, A., Ghiadoni, L., Versari, D., & Salvetti, A. (2006). Endothelium, aging, and hypertension. *Curr Hypertens Rep*, 8(1), 84-89.

Taylor, A.C., McCartney, N., Kamath, M.V. & Wiley, R.L. (2003). Isometric training lowers resting blood pressure and modulates autonomic control. *Med Sci Sports Exerc*, 35(2), 251-256.

Tomiyama, H., Arai, T., Koji, Y., Yambe, M., Motobe, K., Zaydun, G., et al. (2004). The age-related increase in arterial stiffness is augmented in phases according to the severity of hypertension. *Hypertension Res*, 27, 465-470.

Tomiyama, H., Hashimoto, H., Hirayama, Y., Yambe, M., Yamada, J., Koji, Y., et al. (2006). Synergistic acceleration of arterial stiffness in the presence of raised blood pressure and raised plasma glucose. *Hypertension*, 47, 180-188

van de Borne, P., Montano, N., Zimmerman, B., Pagani, M., & Somers, V.K. (1997). Relationship between repeated measures of hemodynamics, muscle sympathetic nerve activity, and their spectral oscillations. Circulation, 96(12), 4326-4332.

Van Hoof, R., Macor, F., Lijnen, P., Staessen, J., Thijs, L., Vanhees, L., et al. (1996). Effect of strength training on blood pressure measured in various conditions in sedentary men. *Int J Sports Med*, 17(6), 415-422.

van Rossum, C.T., Shipley, M.J., Hemingway, H., Grobbee, D.E., Mackenbach, J.P. & Marmot, M.G. (2001). Seasonal variation in cause-specific mortality: are there high risk groups? 25 year follow-up of civil servants from the first Whitehall study. *Int J Epidemiol*, 30, 1109-1116.

Vasan, R.S., Beiser, A., Seshadri, S., Larson, M.G., Kannel, W.B., D'Agostino, R.B., et al. (2002). Residual lifetime risk for developing hypertension in middle-aged women and men: The Framingham heart study. *JAMA*, 287, 1003-1010.

Vukasovic, J.L., Moraga, F., Diaz-Araya, G., Turner, E., Chiong, M., Uriarte, P., et al. (2005). Oxidative stress in pericardial fluid and plasma and its association with ventricular function. Int J Cardiol, 101, 197–201.

Wielgosz, A., Arango, M., Johansen, H., Mao, Y., Nair, C., Paradis, G., et al. (2003). The growing burden of heart disease and stroke in Canada. Heart and Stroke foundation of Canada.

Wiley, R.L., Dunn, C.L., Cox, R.H., Hueppchen, N.A. & Scott, M.S. (1992). Isometric exercise training lowers resting blood pressure. *Med Sci Sports Exerc*, 24(7), 749-754.

Williams, B., Poulter, N.R., Brown, M.J., Davis, M., McInnes, G.T., Potter, J.P., et al. (2004). The BHS Guidelines Working Party Guidelines for Management of Hypertension: Report of the Fourth Working Party of the British Hypertension Society, 2004 - BHS IV. *Journal of Human Hypertension*, 18, 139-185.

Wills, S.H. (2000). Calculating the ankle-brachial index. Advances in Skin & Wound Care, 13(2), 86.

Wilmore, J.H. & Costill, D.L. (1999). *Physiology of sport and exercise* (2nd ed). Ch 5, 6, 7, 17, 19, 21. Champaign, IL, USA.: Human Kinetics

Wong, M.D., Shapiro, M.F., Boscardin, W.J. & Ettner, S.L. (2002). Contribution of major diseases to disparities in mortality. *N Engl J Med*, 347(20), 1585-1592

Woodhouse, P.R., Khaw, K.T. & Plummer, M. (1993). Seasonal variation of blood pressure and its relationship to ambient temperature in an elderly population. *J Hypertens*, 11, 1267-1274.

Yasuda, Y. & Miyamura, M. (1983). Cross transfer effect of muscular training on blood flow in the ipsilateral and contralateral forearm. *Eur J Appl Physiol Occup Physiol*, 51, 321-329.

Appendices

Appendix A – Study Data

Participant Characteristics

Control Subjects	Sex	Age	Months in Exercise Program	Baseline SBP	Baseline DBP
BA	F	62	10	144	87
JiA	M	62	28	109	73
JoA	M	74	7	135	82
AB	F	78	70	103	52
DB	F	76	18	123	71
NB	F	47	4	115	69
RD	M	69	12	116	70
BE	M	62	15	109	69
BG	M	63	15	108	67
GG	F	57	15	105	64
AH	F	73	72	129	69
OH	F	68	4	134	64
UH	F	67	72	125	66
BK	M	68	30	103	66
JK	F	65	30	120	74
NK	F	69	13	104	68
MM	F	68	23	118	65
DoP	F	63	9	90	52
BP	F	70	30	122	65
DP	F	80	20	106	62
RS	F	62	5	104	64
BS	M	74	39	143	71
MS	F	70	18	132	60
ST	F	62	12	117	72
Mean		**67**	**24**	**117**	**68**
SEM		**1.47**	**4.19**	**2.84**	**1.56**

Sex: M, Male; F, Female
Age: Represented in years
Months in exercise program, membership in MacTurtle or MacSenior
SBP, systolic blood pressure; DBP, diastolic blood pressure (SBP and DBP represented in mmHg); SEM, standard error of the mean

Participant Characteristics

Training Subjects	Sex	Age	Months in Exercise Program	Baseline SBP	Baseline DBP
AA	F	58	20	113	75
SA	F	63	5	109	64
JB	F	72	26	151	64
FC	F	69	55	140	81
JC	M	69	55	106	66
BoE	M	67	3	132	69
AE	M	70	76	105	64
BF	M	63	25	135	71
JF	F	68	20	129	62
VI	M	66	10	115	68
RJ	F	59	36	96	63
TJ	M	63	36	135	73
LK	M	72	16	113	64
AL	M	73	96	129	67
JL	F	63	39	125	69
BM	M	69	54	125	76
JM	M	62	36	116	78
SS	F	53	16	113	74
JS	M	66	28	134	78
LS	F	61	23	100	57
BW	M	73	17	106	65
DW	M	73	72	140	83
RW	M	64	7	122	68
CW	F	63	7	128	71
PV	F	67	53	122	77
Mean		**66**	**33**	**122**	**70**
SEM		**1.03**	**4.86**	**2.79**	**1.32**

Sex: M, Male; F, Female
Age: Represented in years
SBP, systolic blood pressure; DBP, diastolic blood pressure (SBP and DBP represented in mmHg); SEM, standard error of the mean

Baseline systolic blood pressure data

Session 1

Control	Session 1				Training	Session 1			
Subjects	Pre1	Pre1	Pre1	Mean1	Subjects	Pre1	Pre1	Pre1	Mean1
BA	136	133	128	**132**	AA	117	107	109	**111**
JiA	103	116	112	**110**	SA	120	112	116	**116**
JoA	124	119	120	**121**	JB	152	156	151	**153**
AB	101	104	96	**100**	FC	144	140	142	**142**
DB	125	124	125	**125**	JC	103	106	103	**104**
NB	113	118	111	**114**	BoE	134	127	134	**132**
RD	117	118	110	**115**	AE	96	104	106	**102**
BE	106	116	115	**112**	BF	134	133	139	**135**
BG	112	111	105	**109**	JF	129	132	130	**130**
GG	110	103	100	**104**	VI	113	113	112	**113**
AH	119	117	125	**120**	RJ	102	99	88	**96**
OH	139	130	142	**137**	TJ	136	126	134	**132**
UH	115	118	120	**118**	LK	120	119	113	**117**
BK	108	109	112	**110**	AL	122	120	120	**121**
JK	121	121	118	**120**	JL	118	128	120	**122**
NK	100	95	99	**98**	BM	123	118	122	**121**
MM	122	116	123	**120**	JM	118	120	127	**122**
DoP	91	83	86	**87**	SS	115	118	109	**114**
BP	130	120	128	**126**	JS	129	139	129	**132**
DP	120	118	118	**119**	LS	95	100	107	**101**
RS	109	101	103	**104**	PV	128	123	127	**126**
BS	146	153	142	**147**	BW	103	103	107	**104**
MS	136	129	122	**129**	CW	140	140	140	**140**
ST	118	118	119	**118**	DW	142	131	132	**135**
					RW	118	115	121	**118**
Mean	**118**	**116**	**116**	**116**	**Mean**	**122**	**121**	**122**	**122**
SEM	**2.74**	**2.79**	**2.75**	**2.67**	**SEM**	**3.02**	**2.86**	**2.94**	**2.86**

All data represented as mmHg
SEM, standard error of the mean

Baseline systolic blood pressure data

Session 2

Control		Session 2			Training		Session 2		
Subjects	Pre2	Pre2	Pre2	Mean2	Subjects	Pre2	Pre2	Pre2	Mean2
BA	144	148	144	**145**	AA	112	117	113	**114**
JiA	116	107	113	**112**	SA	111	98	101	**103**
JoA	139	134	138	**137**	JB	150	151	151	**151**
AB	105	106	108	**106**	FC	138	139	138	**138**
DB	127	133	125	**128**	JC	104	97	106	**102**
NB	117	120	124	**120**	BoE	140	128	133	**134**
RD	122	117	122	**120**	AE	110	107	107	**108**
BE	112	108	105	**108**	BF	134	136	134	**135**
BG	107	110	103	**107**	JF	128	133	126	**129**
GG	110	104	109	**108**	VI	121	120	124	**122**
AH	140	141	134	**138**	RJ	94	96	99	**96**
OH	131	133	125	**130**	TJ	129	138	142	**136**
UH	130	117	130	**126**	LK	110	116	116	**114**
BK	103	96	98	**99**	AL	134	140	136	**137**
JK	128	126	124	**126**	JL	134	132	119	**128**
NK	116	112	113	**114**	BM	126	128	125	**126**
MM	122	123	120	**122**	JM	116	115	119	**117**
DoP	91	91	89	**90**	SS	111	110	117	**113**
BP	123	130	117	**123**	JS	128	117	144	**130**
DP	100	93	97	**97**	LS	105	106	101	**104**
RS	106	98	105	**103**	PV	114	121	122	**119**
BS	134	151	141	**142**	BW	111	112	105	**109**
MS	132	128	137	**132**	CW	125	120	126	**124**
ST	118	118	122	**119**	DW	143	147	148	**146**
					RW	122	128	132	**127**
Mean	**120**	**119**	**118**	**119**	**Mean**	**122**	**122**	**123**	**122**
SEM	**2.81**	**3.42**	**3.02**	**3.01**	**SEM**	**2.78**	**3.06**	**3.04**	**2.88**

All data represented as mmHg
SEM, standard error of the mean

Baseline systolic blood pressure data

Session 3

Control Subjects	Session 3 Pre3	Pre3	Pre3	Mean3	Training Subjects	Session 3 Pre3	Pre3	Pre3	Mean3
BA	153	157	154	**155**	AA	113	115	118	**115**
JiA	104	107	104	**105**	SA	112	105	110	**109**
JoA	141	148	151	**147**	JB	150	151	146	**149**
AB	n/a	n/a	n/a	**n/a**	FC	142	142	132	**139**
DB	115	120	111	**115**	JC	105	114	114	**111**
NB	111	110	113	**111**	BoE	125	136	129	**130**
RD	111	116	109	**112**	AE	103	109	107	**106**
BE	105	102	108	**105**	BF	135	141	132	**136**
BG	108	109	105	**107**	JF	128	130	128	**129**
GG	104	106	100	**103**	VI	109	112	111	**111**
AH	n/a	n/a	n/a	**n/a**	RJ	95	95	98	**96**
OH	136	134	135	**135**	TJ	138	134	136	**136**
UH	137	131	126	**131**	LK	105	108	108	**107**
BK	100	98	100	**99**	AL	133	132	128	**131**
JK	117	114	112	**114**	JL	111	130	132	**124**
NK	98	96	104	**99**	BM	125	126	130	**127**
MM	111	108	113	**111**	JM	109	112	105	**109**
DoP	91	91	89	**90**	SS	113	107	113	**111**
BP	121	119	113	**118**	JS	146	134	136	**139**
DP	96	101	108	**102**	LS	93	97	93	**94**
RS	99	103	108	**103**	PV	117	123	123	**121**
BS	135	141	140	**139**	BW	107	103	106	**105**
MS	138	128	137	**134**	CW	119	123	122	**121**
ST	116	114	113	**114**	DW	140	137	141	**139**
					RW	118	118	122	**119**
Mean	**116**	**116**	**116**	**116**	**Mean**	**120**	**121**	**121**	**121**
SEM	**3.65**	**3.70**	**3.64**	**3.64**	**SEM**	**3.18**	**3.02**	**2.77**	**2.93**

All data represented as mmHg
SEM, standard error of the mean

Baseline diastolic blood pressure data

Session 1

Control		Session 1			Training		Session 1		
Subjects	Pre1	Pre1	Pre1	Mean1	Subjects	Pre1	Pre1	Pre1	Mean1
BA	87	87	84	**86**	AA	78	77	75	**77**
JiA	69	69	73	**70**	SA	65	64	60	**63**
JoA	78	74	74	**75**	JB	62	61	66	**63**
AB	47	48	47	**47**	FC	82	81	80	**81**
DB	69	67	68	**68**	JC	67	68	69	**68**
NB	73	69	68	**70**	BoE	74	73	71	**73**
RD	69	68	69	**69**	AE	63	65	64	**64**
BE	67	65	65	**66**	BF	73	71	71	**72**
BG	69	68	67	**68**	JF	64	65	65	**65**
GG	64	63	64	**64**	VI	67	67	69	**68**
AH	65	67	69	**67**	RJ	65	62	63	**63**
OH	67	67	65	**66**	TJ	76	73	73	**74**
UH	65	63	64	**64**	LK	66	65	65	**65**
BK	66	67	68	**67**	AL	65	64	67	**65**
JK	74	75	71	**73**	JL	67	69	67	**68**
NK	67	67	68	**67**	BM	73	74	75	**74**
MM	66	65	65	**65**	JM	81	81	79	**80**
DoP	50	51	52	**51**	SS	77	79	76	**77**
BP	69	66	66	**67**	JS	81	78	86	**82**
DP	68	67	67	**67**	LS	57	59	56	**57**
RS	65	65	62	**64**	PV	82	80	80	**81**
BS	72	61	71	**68**	BW	63	64	62	**63**
MS	61	55	59	**58**	CW	75	74	79	**76**
ST	72	74	74	**73**	DW	79	80	76	**78**
					RW	69	69	67	**68**
Mean	**67**	**66**	**67**	**67**	**Mean**	**71**	**71**	**70**	**71**
SEM	**1.60**	**1.59**	**1.48**	**1.53**	**SEM**	**1.46**	**1.38**	**1.46**	**1.42**

All data represented as mmHg
SEM, standard error of the mean

Baseline diastolic blood pressure data

Session 2

Control		Session 2			Training		Session 2		
Subjects	Pre2	Pre2	Pre2	Mean2	Subjects	Pre2	Pre2	Pre2	Mean2
BA	90	90	89	**90**	AA	80	80	77	**79**
JiA	75	74	74	**74**	SA	65	62	63	**63**
JoA	85	83	83	**84**	JB	67	63	64	**65**
AB	57	58	57	**57**	FC	80	77	78	**78**
DB	75	77	75	**76**	JC	65	62	64	**64**
NB	69	70	70	**70**	BoE	66	67	70	**68**
RD	72	71	71	**71**	AE	64	65	65	**65**
BE	73	81	69	**74**	BF	74	71	71	**72**
BG	67	67	67	**67**	JF	59	60	60	**60**
GG	63	60	64	**62**	VI	68	74	67	**70**
AH	68	72	73	**71**	RJ	64	62	62	**63**
OH	60	64	61	**62**	TJ	73	71	74	**73**
UH	66	64	66	**65**	LK	65	65	62	**64**
BK	66	65	65	**65**	AL	67	69	70	**69**
JK	74	75	72	**74**	JL	71	67	65	**68**
NK	67	69	67	**68**	BM	76	76	78	**77**
MM	65	68	64	**66**	JM	81	79	83	**81**
DoP	55	52	50	**52**	SS	74	74	73	**74**
BP	65	64	65	**65**	JS	70	83	71	**75**
DP	58	60	62	**60**	LS	59	58	60	**59**
RS	64	65	67	**65**	PV	79	77	74	**77**
BS	70	78	76	**75**	BW	66	65	65	**65**
MS	63	57	56	**59**	CW	69	67	69	**68**
ST	74	71	71	**72**	DW	86	84	87	**86**
					RW	68	69	69	**69**
Mean	**68**	**69**	**68**	**69**	**Mean**	**70**	**70**	**70**	**70**
SEM	**1.67**	**1.82**	**1.71**	**1.72**	**SEM**	**1.40**	**1.47**	**1.42**	**1.39**

All data represented as mmHg
SEM, standard error of the mean

Baseline diastolic blood pressure data

Session 3

Control		Session 3			Training		Session 3		
Subjects	Pre3	Pre3	Pre3	Mean3	Subjects	Pre3	Pre3	Pre3	Mean3
BA	84	86	84	**85**	AA	69	69	69	**69**
JiA	74	72	74	**73**	SA	63	65	65	**64**
JoA	84	94	87	**88**	JB	65	64	66	**65**
AB	n/a	n/a	n/a	**n/a**	FC	85	83	81	**83**
DB	69	69	69	**69**	JC	67	67	67	**67**
NB	71	68	67	**69**	BoE	65	70	69	**68**
RD	68	69	69	**69**	AE	64	65	62	**64**
BE	65	67	67	**66**	BF	69	69	72	**70**
BG	67	67	67	**67**	JF	61	62	64	**62**
GG	69	67	65	**67**	VI	67	68	69	**68**
AH	n/a	n/a	n/a	**n/a**	RJ	63	65	63	**64**
OH	67	66	62	**65**	TJ	72	74	75	**74**
UH	71	67	66	**68**	LK	61	61	62	**61**
BK	65	66	65	**65**	AL	65	67	67	**66**
JK	74	76	75	**75**	JL	69	72	76	**72**
NK	67	67	74	**69**	BM	77	77	76	**77**
MM	63	64	63	**63**	JM	75	73	74	**74**
DoP	52	54	54	**53**	SS	71	69	71	**70**
BP	63	62	62	**63**	JS	71	74	86	**77**
DP	58	58	61	**59**	LS	54	51	56	**54**
RS	63	64	65	**64**	PV	74	73	73	**73**
BS	69	68	70	**69**	BW	67	68	67	**67**
MS	63	62	63	**63**	CW	71	65	67	**68**
ST	71	72	72	**72**	DW	86	85	84	**85**
					RW	66	68	69	**68**
Mean	**68**	**68**	**68**	**68**	**Mean**	**69**	**69**	**70**	**69**
SEM	**1.54**	**1.81**	**1.58**	**1.61**	**SEM**	**1.42**	**1.38**	**1.41**	**1.36**

All data represented as mmHg
SEM, standard error of the mean

Baseline pulse pressure data

Calculated from mean SBP and DBP per session

Control		Session			Training		Session		
Subjects	Pre1	Pre2	Pre3	Mean	Subjects	Pre1	Pre2	Pre3	Mean
BA	46	56	70	**57**	AA	34	35	46	**39**
JiA	40	38	32	**36**	SA	53	40	45	**46**
JoA	46	53	58	**52**	JB	90	86	84	**87**
AB	53	49	n/a	**51**	FC	61	60	56	**59**
DB	57	53	46	**52**	JC	36	39	44	**40**
NB	44	51	43	**46**	BoE	59	66	62	**62**
RD	46	49	43	**46**	AE	38	43	43	**41**
BE	47	34	39	**40**	BF	64	63	66	**64**
BG	41	40	40	**40**	JF	66	69	66	**67**
GG	41	45	36	**41**	VI	45	52	43	**47**
AH	53	67	n/a	**60**	RJ	33	34	32	**33**
OH	71	68	70	**70**	TJ	58	64	62	**61**
UH	54	60	63	**59**	LK	52	50	46	**49**
BK	43	34	34	**37**	AL	55	68	65	**63**
JK	47	52	39	**46**	JL	54	61	52	**56**
NK	31	46	30	**36**	BM	47	50	50	**49**
MM	55	56	47	**53**	JM	41	36	35	**37**
DoP	36	38	37	**37**	SS	37	39	41	**39**
BP	59	59	55	**58**	JS	51	55	62	**56**
DP	51	37	43	**44**	LS	43	45	41	**43**
RS	40	38	39	**39**	PV	45	42	48	**45**
BS	79	67	70	**72**	BW	41	44	38	**41**
MS	71	74	72	**72**	CW	64	55	54	**58**
ST	45	47	43	**45**	DW	57	60	54	**57**
					RW	50	59	52	**53**
Mean	**50**	**50**	**48**	**50**	**Mean**	**51**	**53**	**51**	**52**
SEM	**2.33**	**2.34**	**2.89**	**2.30**	**SEM**	**2.56**	**2.61**	**2.38**	**2.43**

All data represented as mmHg
SEM, standard error of the mean

Baseline heart rate data

Session 1

Control Subjects	Session 1 Pre1	Pre1	Pre1	Mean1	Training Subjects	Session 1 Pre1	Pre1	Pre1	Mean1
BA	68	68	72	**69**	AA	72	68	70	**70**
JiA	72	76	72	**73**	SA	75	74	72	**74**
JoA	68	65	63	**65**	JB	78	76	82	**79**
AB	66	62	62	**63**	FC	63	62	63	**63**
DB	90	86	86	**87**	JC	62	56	66	**61**
NB	86	83	83	**84**	BoE	56	58	60	**58**
RD	68	74	74	**72**	AE	76	74	74	**75**
BE	69	64	65	**66**	BF	65	68	67	**67**
BG	48	51	50	**50**	JF	69	70	65	**68**
GG	73	72	71	**72**	VI	62	61	61	**61**
AH	54	55	55	**55**	RJ	80	78	76	**78**
OH	94	91	91	**92**	TJ	69	70	74	**71**
UH	68	69	70	**69**	LK	51	51	52	**51**
BK	70	71	73	**71**	AL	79	79	80	**79**
JK	78	78	80	**79**	JL	61	66	64	**64**
NK	89	86	88	**87**	BM	56	58	58	**57**
MM	93	88	89	**90**	JM	70	73	75	**73**
DoP	53	57	59	**56**	SS	83	82	82	**82**
BP	69	66	71	**69**	JS	64	68	58	**63**
DP	74	74	74	**74**	LS	77	77	73	**76**
RS	59	62	60	**60**	PV	75	73	74	**74**
BS	69	67	67	**68**	BW	74	70	71	**72**
MS	76	74	76	**75**	CW	64	63	64	**64**
ST	72	74	74	**73**	DW	65	66	63	**65**
					RW	44	45	45	**45**
Mean	**72**	**71**	**72**	**72**	**Mean**	**68**	**67**	**68**	**68**
SEM	**2.46**	**2.15**	**2.20**	**2.24**	**SEM**	**1.93**	**1.81**	**1.83**	**1.83**

All data represented as beats per minute (bpm)
SEM, standard error of the mean

Baseline heart rate data

Session 2

Control Subjects	Pre2	Session 2 Pre2	Pre2	Mean2	Training Subjects	Pre2	Session 2 Pre2	Pre2	Mean2
BA	70	72	72	**71**	AA	68	68	69	**68**
JiA	76	73	76	**75**	SA	80	79	80	**80**
JoA	64	61	61	**62**	JB	83	81	82	**82**
AB	63	61	59	**61**	FC	71	71	72	**71**
DB	77	80	80	**79**	JC	66	65	66	**66**
NB	80	80	89	**83**	BoE	65	67	67	**66**
RD	77	77	77	**77**	AE	78	76	75	**76**
BE	63	67	62	**64**	BF	67	68	67	**67**
BG	50	50	52	**51**	JF	74	75	72	**74**
GG	70	72	70	**71**	VI	61	60	60	**60**
AH	50	51	51	**51**	RJ	80	76	76	**77**
OH	97	98	97	**97**	TJ	76	74	76	**75**
UH	58	61	62	**60**	LK	55	57	57	**56**
BK	79	81	79	**80**	AL	78	77	76	**77**
JK	76	78	77	**77**	JL	67	66	72	**68**
NK	86	88	86	**87**	BM	59	61	56	**59**
MM	86	91	86	**88**	JM	73	76	80	**76**
DoP	62	61	61	**61**	SS	80	82	86	**83**
BP	70	67	68	**68**	JS	62	86	66	**71**
DP	72	75	74	**74**	LS	82	80	81	**81**
RS	68	72	76	**72**	PV	77	76	76	**76**
BS	57	61	60	**59**	BW	77	76	79	**77**
MS	74	76	76	**75**	CW	61	61	62	**61**
ST	64	64	64	**64**	DW	66	66	66	**66**
					RW	45	45	45	**45**
Mean	**70**	**72**	**71**	**71**	**Mean**	**70**	**71**	**71**	**70**
SEM	**2.32**	**2.42**	**2.41**	**2.36**	**SEM**	**1.90**	**1.87**	**1.91**	**1.83**

All data represented as beats per minute (bpm)
SEM, standard error of the mean

Baseline heart rate data

Session 3

Control Subjects	Pre3	Session 3 Pre3	Pre3	Mean3	Training Subjects	Pre3	Session 3 Pre3	Pre3	Mean3
BA	67	67	69	**68**	AA	71	73	74	**73**
JiA	74	72	76	**74**	SA	86	86	83	**85**
JoA	70	72	65	**69**	JB	86	86	85	**86**
AB	n/a	n/a	n/a	**n/a**	FC	71	72	68	**70**
DB	80	78	80	**79**	JC	63	67	59	**63**
NB	81	76	86	**81**	BoE	68	68	65	**67**
RD	76	80	76	**77**	AE	74	73	73	**73**
BE	62	64	63	**64**	BF	66	70	72	**69**
BG	56	55	56	**56**	JF	70	69	69	**69**
GG	74	70	69	**71**	VI	65	67	67	**66**
AH	n/a	n/a	n/a	**n/a**	RJ	72	71	76	**73**
OH	88	84	85	**86**	TJ	72	72	72	**72**
UH	58	57	57	**57**	LK	59	60	56	**58**
BK	73	72	74	**73**	AL	83	82	81	**82**
JK	80	82	80	**81**	JL	66	67	66	**66**
NK	74	76	82	**77**	BM	68	70	69	**69**
MM	74	76	76	**75**	JM	68	74	73	**72**
DoP	56	57	56	**56**	SS	78	81	80	**80**
BP	68	67	70	**68**	JS	66	66	84	**72**
DP	80	79	80	**80**	LS	70	68	70	**69**
RS	70	72	80	**74**	PV	80	83	81	**81**
BS	65	66	62	**64**	BW	84	86	84	**85**
MS	80	76	79	**78**	CW	62	63	62	**62**
ST	59	59	61	**60**	DW	62	64	62	**63**
					RW	48	48	49	**48**
Mean	**71**	**71**	**72**	**71**	**Mean**	**70**	**71**	**71**	**71**
SEM	**1.91**	**1.79**	**2.04**	**1.87**	**SEM**	**1.79**	**1.79**	**1.88**	**1.79**

All data represented as beats per minute (bpm)
SEM, standard error of the mean

Post training systolic blood pressure data

Session 1

Control Subjects	Pre1	Session 1 Pre1	Pre1	Mean1	Training Subjects	Pre1	Session 1 Pre1	Pre1	Mean1
BA	127	125	126	**126**	AA	100	102	100	**101**
JiA	107	106	111	**108**	SA	100	95	102	**99**
JoA	128	130	127	**128**	JB	143	140	145	**143**
AB	103	111	107	**107**	FC	n/a	n/a	n/a	**n/a**
DB	129	116	116	**120**	JC	92	99	99	**97**
NB	116	119	120	**118**	BoE	120	121	116	**119**
RD	107	110	111	**109**	AE	107	95	102	**101**
BE	106	110	110	**109**	BF	133	131	132	**132**
BG	114	108	103	**108**	JF	125	114	118	**119**
GG	98	99	95	**97**	VI	100	100	105	**102**
AH	137	141	147	**142**	RJ	93	98	91	**94**
OH	126	129	131	**129**	TJ	119	121	113	**118**
UH	136	121	125	**127**	LK	108	101	104	**104**
BK	100	102	105	**102**	AL	134	134	139	**136**
JK	136	125	127	**129**	JL	109	109	110	**109**
NK	n/a	n/a	n/a	**n/a**	BM	120	117	124	**120**
MM	109	106	112	**109**	JM	112	108	111	**110**
DoP	94	93	89	**92**	SS	105	103	106	**105**
BP	134	135	123	**131**	JS	111	116	117	**115**
DP	96	100	104	**100**	LS	94	96	93	**94**
RS	112	95	101	**103**	PV	n/a	n/a	n/a	**n/a**
BS	147	141	133	**140**	BW	98	99	102	**100**
MS	136	142	135	**138**	CW	119	118	115	**117**
ST	120	121	122	**121**	DW	125	115	119	**120**
					RW	122	113	119	**118**
Mean	**118**	**117**	**117**	**117**	**Mean**	**113**	**111**	**112**	**112**
SEM	**3.25**	**3.12**	**2.94**	**3.01**	**SEM**	**2.93**	**2.70**	**2.85**	**2.78**

All data represented as mmHg
SEM, standard error of the mean

Post training systolic blood pressure data

Session 2

Control Subjects	Session 2 Pre2	Pre2	Pre2	Mean2	Training Subjects	Session 2 Pre2	Pre2	Pre2	Mean2
BA	126	123	127	**125**	AA	107	108	102	**106**
JiA	114	116	121	**117**	SA	101	100	98	**100**
JoA	131	131	131	**131**	JB	147	149	145	**147**
AB	106	95	107	**103**	FC	119	116	117	**117**
DB	115	117	132	**121**	JC	100	96	100	**99**
NB	118	121	133	**124**	BoE	126	126	125	**126**
RD	110	112	106	**109**	AE	103	97	99	**100**
BE	112	121	119	**117**	BF	126	127	123	**125**
BG	112	113	112	**112**	JF	118	119	117	**118**
GG	103	99	107	**103**	VI	108	106	103	**106**
AH	137	137	135	**136**	RJ	94	95	97	**95**
OH	124	126	133	**128**	TJ	122	125	126	**124**
UH	135	131	125	**130**	LK	114	111	113	**113**
BK	100	99	100	**100**	AL	138	143	134	**138**
JK	138	132	133	**134**	JL	116	113	108	**112**
NK	108	109	110	**109**	BM	125	118	125	**123**
MM	112	114	109	**112**	JM	117	113	113	**114**
DoP	90	90	95	**92**	SS	103	104	104	**104**
BP	114	119	111	**115**	JS	106	115	126	**116**
DP	102	103	108	**104**	LS	81	86	87	**85**
RS	109	106	109	**108**	PV	n/a	n/a	n/a	**n/a**
BS	153	158	159	**157**	BW	100	101	102	**101**
MS	133	131	133	**132**	CW	108	111	112	**110**
ST	110	112	113	**112**	DW	127	122	117	**122**
					RW	118	118	123	**120**
Mean	**117**	**117**	**120**	**118**	**Mean**	**114**	**113**	**113**	**113**
SEM	**2.99**	**3.12**	**3.01**	**2.96**	**SEM**	**2.96**	**3.00**	**2.79**	**2.84**

All data represented as mmHg
SEM, standard error of the mean

Post training systolic blood pressure data

Session 3

Control Subjects	Session 3 Pre3	Pre3	Pre3	Mean3	Training Subjects	Session 3 Pre3	Pre3	Pre3	Mean3
BA	126	121	121	**123**	AA	107	107	107	**107**
JiA	116	107	111	**111**	SA	93	109	100	**101**
JoA	142	141	130	**138**	JB	145	147	144	**145**
AB	108	112	108	**109**	FC	104	104	100	**103**
DB	125	118	123	**122**	JC	101	105	100	**102**
NB	n/a	n/a	n/a	**n/a**	BoE	114	120	115	**116**
RD	114	110	108	**111**	AE	100	105	105	**103**
BE	111	110	113	**111**	BF	129	131	130	**130**
BG	118	112	117	**116**	JF	128	129	121	**126**
GG	102	101	97	**100**	VI	102	102	101	**102**
AH	n/a	n/a	n/a	**n/a**	RJ	96	87	89	**91**
OH	129	124	124	**126**	TJ	124	130	124	**126**
UH	135	131	128	**131**	LK	113	108	109	**110**
BK	106	100	105	**104**	AL	134	134	133	**134**
JK	126	127	122	**125**	JL	119	116	115	**117**
NK	112	115	110	**112**	BM	121	120	120	**120**
MM	110	114	115	**113**	JM	104	111	102	**106**
DoP	89	84	88	**87**	SS	108	105	105	**106**
BP	115	125	115	**118**	JS	n/a	n/a	n/a	**n/a**
DP	94	99	108	**100**	LS	82	86	90	**86**
RS	107	104	108	**106**	PV	n/a	n/a	n/a	**n/a**
BS	150	143	154	**149**	BW	95	96	99	**97**
MS	135	130	141	**135**	CW	111	108	108	**109**
ST	n/a	n/a	n/a	**n/a**	DW	126	126	126	**126**
					RW	n/a	n/a	n/a	**n/a**
Mean	**118**	**116**	**116**	**117**	**Mean**	**112**	**113**	**111**	**112**
SEM	**3.34**	**3.16**	**3.15**	**3.15**	**SEM**	**3.27**	**3.27**	**3.03**	**3.13**

All data represented as mmHg
SEM, standard error of the mean

Post training diastolic blood pressure data

Session 1

Control Subjects	Pre1	Session 1 Pre1	Pre1	Mean1	Training Subjects	Pre1	Session 1 Pre1	Pre1	Mean1
BA	84	86	88	**86**	AA	73	72	72	**72**
JiA	74	77	78	**76**	SA	65	67	64	**65**
JoA	79	76	79	**78**	JB	63	64	67	**65**
AB	58	59	58	**58**	FC	n/a	n/a	n/a	**n/a**
DB	73	71	72	**72**	JC	63	64	63	**63**
NB	69	69	67	**68**	BoE	66	69	68	**68**
RD	67	67	65	**66**	AE	63	60	60	**61**
BE	67	66	66	**66**	BF	67	71	71	**70**
BG	65	67	67	**66**	JF	58	60	63	**60**
GG	67	67	65	**66**	VI	64	63	65	**64**
AH	76	72	76	**75**	RJ	65	65	60	**63**
OH	65	58	60	**61**	TJ	69	69	68	**69**
UH	64	67	66	**66**	LK	64	61	63	**63**
BK	64	64	61	**63**	AL	67	69	67	**68**
JK	76	75	76	**76**	JL	65	64	66	**65**
NK	n/a	n/a	n/a	**n/a**	BM	74	75	75	**75**
MM	65	63	60	**63**	JM	78	80	80	**79**
DoP	52	53	53	**53**	SS	74	74	74	**74**
BP	65	65	68	**66**	JS	65	74	75	**71**
DP	59	61	60	**60**	LS	61	58	56	**58**
RS	62	72	60	**65**	PV	n/a	n/a	n/a	**n/a**
BS	69	66	68	**68**	BW	63	64	63	**63**
MS	67	62	65	**65**	CW	74	69	73	**72**
ST	74	71	70	**72**	DW	76	74	73	**74**
					RW	65	67	66	**66**
Mean	**68**	**68**	**67**	**68**	**Mean**	**67**	**68**	**67**	**67**
SEM	**1.51**	**1.49**	**1.68**	**1.51**	**SEM**	**1.10**	**1.18**	**1.22**	**1.21**

All data represented as mmHg
SEM, standard error of the mean

Post training diastolic blood pressure data

Session 2

Control		Session 2			Training		Session 2		
Subjects	Pre2	Pre2	Pre2	Mean2	Subjects	Pre2	Pre2	Pre2	Mean2
BA	74	78	86	**79**	AA	76	76	74	**75**
JiA	71	73	73	**72**	SA	67	65	62	**65**
JoA	85	81	81	**82**	JB	66	64	65	**65**
AB	59	56	55	**56**	FC	67	68	67	**67**
DB	69	69	66	**68**	JC	64	63	64	**64**
NB	69	78	75	**74**	BoE	70	68	69	**69**
RD	67	69	66	**67**	AE	60	58	59	**59**
BE	65	67	65	**66**	BF	72	70	73	**72**
BG	67	68	67	**67**	JF	61	63	64	**63**
GG	68	67	67	**67**	VI	65	65	67	**66**
AH	66	69	70	**68**	RJ	64	65	65	**65**
OH	57	56	62	**58**	TJ	70	74	69	**71**
UH	59	65	67	**64**	LK	65	67	67	**66**
BK	65	63	64	**64**	AL	67	66	67	**67**
JK	81	81	77	**80**	JL	67	66	69	**67**
NK	67	67	65	**66**	BM	75	67	74	**72**
MM	65	63	65	**64**	JM	76	74	76	**75**
DoP	53	56	53	**54**	SS	74	72	72	**73**
BP	66	71	66	**68**	JS	67	66	77	**70**
DP	63	64	64	**64**	LS	53	52	54	**53**
RS	67	65	63	**65**	PV	n/a	n/a	n/a	**n/a**
BS	79	75	71	**75**	BW	65	65	64	**65**
MS	61	64	60	**62**	CW	65	65	60	**63**
ST	69	67	67	**68**	DW	77	78	76	**77**
					RW	65	67	67	**66**
Mean	**67**	**68**	**67**	**67**	**Mean**	**67**	**67**	**68**	**67**
SEM	**1.49**	**1.45**	**1.50**	**1.42**	**SEM**	**1.15**	**1.13**	**1.18**	**1.09**

All data represented as mmHg
SEM, standard error of the mean

Post training diastolic blood pressure data

Session 3

Control Subjects	Pre3	Session 3 Pre3	Pre3	Mean3	Training Subjects	Pre3	Session 3 Pre3	Pre3	Mean3
BA	76	77	72	**75**	AA	78	78	79	**78**
JiA	73	69	69	**70**	SA	59	61	65	**62**
JoA	81	83	84	**83**	JB	67	65	65	**66**
AB	63	66	64	**64**	FC	64	64	65	**64**
DB	72	73	73	**73**	JC	71	68	73	**71**
NB	n/a	n/a	n/a	**n/a**	BoE	65	65	65	**65**
RD	67	67	65	**66**	AE	61	63	63	**62**
BE	67	69	75	**70**	BF	70	68	68	**69**
BG	70	69	71	**70**	JF	66	62	64	**64**
GG	65	64	63	**64**	VI	64	64	63	**64**
AH	n/a	n/a	n/a	**n/a**	RJ	64	60	62	**62**
OH	55	56	55	**55**	TJ	69	67	71	**69**
UH	67	66	67	**67**	LK	65	63	65	**64**
BK	67	66	67	**67**	AL	69	68	68	**68**
JK	78	72	79	**76**	JL	67	67	67	**67**
NK	73	74	68	**72**	BM	76	71	69	**72**
MM	61	58	59	**59**	JM	76	75	74	**75**
DoP	52	51	53	**52**	SS	73	71	69	**71**
BP	65	65	65	**65**	JS	n/a	n/a	n/a	**n/a**
DP	55	58	57	**57**	LS	47	45	51	**48**
RS	65	64	58	**62**	PV	n/a	n/a	n/a	**n/a**
BS	74	74	72	**73**	BW	63	63	64	**63**
MS	65	66	62	**64**	CW	61	67	65	**64**
ST	n/a	n/a	n/a	**n/a**	DW	77	77	75	**76**
					RW	n/a	n/a	n/a	**n/a**
Mean	**67**	**67**	**67**	**67**	**Mean**	**67**	**66**	**67**	**67**
SEM	**1.64**	**1.61**	**1.72**	**1.63**	**SEM**	**1.50**	**1.45**	**1.21**	**1.34**

All data represented as mmHg
SEM, standard error of the mean

Post training pulse pressure data

Calculated from mean SBP and DBP per session

Control		Session			Training		Session		
Subjects	Pre1	Pre2	Pre3	Mean	Subjects	Pre1	Pre2	Pre3	Mean
BA	40	46	48	**45**	AA	28	30	29	**29**
JiA	32	45	41	**39**	SA	34	35	39	**36**
JoA	50	49	55	**51**	JB	78	82	80	**80**
AB	49	46	45	**47**	FC	n/a	50	38	**44**
DB	48	53	49	**50**	JC	33	35	31	**33**
NB	50	50	n/a	**50**	BoE	51	57	51	**53**
RD	43	42	44	**43**	AE	40	41	41	**41**
BE	42	52	41	**45**	BF	62	54	61	**59**
BG	42	45	46	**44**	JF	59	55	62	**59**
GG	31	36	36	**34**	VI	38	40	38	**39**
AH	67	68	n/a	**68**	RJ	31	31	29	**30**
OH	68	69	70	**69**	TJ	49	53	57	**53**
UH	62	67	65	**64**	LK	42	46	46	**45**
BK	39	36	37	**37**	AL	68	72	65	**68**
JK	54	55	49	**52**	JL	44	45	50	**46**
NK	n/a	43	41	**42**	BM	46	51	48	**48**
MM	46	47	54	**49**	JM	31	39	31	**34**
DoP	39	38	35	**37**	SS	31	31	35	**32**
BP	65	47	53	**55**	JS	43	46	n/a	**45**
DP	40	41	44	**41**	LS	36	32	38	**35**
RS	38	43	44	**42**	PV	45	42	48	**45**
BS	73	82	76	**77**	BW	36	36	33	**35**
MS	73	71	71	**72**	CW	45	47	45	**46**
ST	49	44	n/a	**47**	DW	45	45	50	**47**
					RW	52	53	n/a	**53**
Mean	**50**	**51**	**50**	**50**	**Mean**	**44**	**46**	**45**	**45**
SEM	**2.64**	**2.48**	**2.57**	**2.41**	**SEM**	**2.55**	**2.50**	**2.73**	**2.45**

All data represented as mmHg
SEM, standard error of the mean

Post training heart rate data

Session 1

Control Subjects	Pre1	Session 1 Pre1	Pre1	Mean1	Training Subjects	Pre1	Session 1 Pre1	Pre1	Mean1
BA	77	78	76	**77**	AA	72	71	70	**71**
JiA	74	76	76	**75**	SA	89	83	84	**85**
JoA	66	62	64	**64**	JB	79	79	77	**78**
AB	n/a	65	62	**64**	FC	n/a	n/a	n/a	**n/a**
DB	80	81	82	**81**	JC	77	74	74	**75**
NB	88	83	83	**85**	BoE	62	64	62	**63**
RD	74	72	74	**73**	AE	78	81	80	**80**
BE	67	67	65	**66**	BF	64	66	65	**65**
BG	50	51	51	**51**	JF	67	68	70	**68**
GG	76	77	75	**76**	VI	65	63	63	**64**
AH	51	52	55	**53**	RJ	79	80	76	**78**
OH	88	90	90	**89**	TJ	72	80	81	**78**
UH	64	64	64	**64**	LK	67	64	70	**67**
BK	74	72	72	**73**	AL	78	76	76	**77**
JK	80	77	80	**79**	JL	55	52	52	**53**
NK	n/a	n/a	n/a	**n/a**	BM	66	65	64	**65**
MM	85	77	81	**81**	JM	79	78	81	**79**
DoP	56	57	56	**56**	SS	79	75	76	**77**
BP	66	68	68	**67**	JS	58	85	76	**73**
DP	80	80	80	**80**	LS	74	72	70	**72**
RS	74	75	68	**72**	PV	n/a	n/a	n/a	**n/a**
BS	64	65	67	**65**	BW	73	72	73	**73**
MS	75	74	76	**75**	CW	62	62	64	**63**
ST	62	61	59	**61**	DW	67	67	67	**67**
					RW	45	45	46	**45**
Mean	**71**	**71**	**71**	**71**	**Mean**	**70**	**71**	**70**	**70**
SEM	**2.30**	**2.07**	**2.11**	**2.10**	**SEM**	**2.05**	**2.05**	**1.92**	**1.92**

All data represented as beats per minute (bpm)
SEM, standard error of the mean

Post training heart rate data

Session 2

Control		Session 2			Training		Session 2		
Subjects	Pre2	Pre2	Pre2	Mean2	Subjects	Pre2	Pre2	Pre2	Mean2
BA	80	80	81	**80**	AA	71	72	73	**72**
JiA	69	69	70	**69**	SA	76	78	78	**77**
JoA	61	60	63	**61**	JB	74	76	78	**76**
AB	63	63	65	**64**	FC	74	75	75	**75**
DB	81	82	84	**82**	JC	64	64	64	**64**
NB	106	105	102	**104**	BoE	59	59	57	**58**
RD	75	80	76	**77**	AE	73	74	72	**73**
BE	68	70	66	**68**	BF	63	66	66	**65**
BG	47	47	49	**48**	JF	66	68	67	**67**
GG	78	77	77	**77**	VI	74	72	68	**71**
AH	50	51	50	**50**	RJ	78	75	76	**76**
OH	87	86	89	**87**	TJ	63	69	66	**66**
UH	66	65	72	**68**	LK	60	65	61	**62**
BK	71	73	72	**72**	AL	81	81	80	**81**
JK	77	75	75	**76**	JL	58	57	56	**57**
NK	97	95	90	**94**	BM	55	56	61	**57**
MM	70	67	64	**67**	JM	62	67	67	**65**
DoP	56	54	55	**55**	SS	75	72	70	**72**
BP	69	71	73	**71**	JS	75	45	78	**66**
DP	85	80	80	**82**	LS	75	75	74	**75**
RS	63	64	62	**63**	PV	n/a	n/a	n/a	**n/a**
BS	71	68	66	**68**	BW	77	75	76	**76**
MS	72	69	71	**71**	CW	73	70	72	**72**
ST	62	68	68	**66**	DW	72	74	73	**73**
					RW	45	44	44	**44**
Mean	**72**	**72**	**72**	**72**	**Mean**	**68**	**68**	**69**	**68**
SEM	**2.76**	**2.67**	**2.53**	**2.62**	**SEM**	**1.80**	**1.97**	**1.75**	**1.72**

All data represented as beats per minute (bpm)
SEM, standard error of the mean

Post training heart rate data

Session 3

Control Subjects	Session 3 Pre3	Pre3	Pre3	Mean3	Training Subjects	Session 3 Pre3	Pre3	Pre3	Mean3
BA	85	85	83	**84**	AA	78	80	80	**79**
JiA	72	72	72	**72**	SA	88	89	95	**91**
JoA	62	64	64	**63**	JB	82	83	82	**82**
AB	n/a	n/a	70	**70**	FC	72	71	72	**72**
DB	80	80	82	**81**	JC	55	51	54	**53**
NB	n/a	n/a	n/a	**n/a**	BoE	60	62	62	**61**
RD	78	79	77	**78**	AE	74	75	72	**74**
BE	62	64	64	**63**	BF	65	65	66	**65**
BG	50	52	53	**52**	JF	67	66	67	**67**
GG	80	82	80	**81**	VI	62	57	56	**58**
AH	n/a	n/a	n/a	**n/a**	RJ	82	80	83	**82**
OH	86	84	85	**85**	TJ	71	76	77	**75**
UH	62	62	58	**61**	LK	65	64	66	**65**
BK	71	71	74	**72**	AL	76	76	74	**75**
JK	76	74	75	**75**	JL	60	63	62	**62**
NK	87	87	82	**85**	BM	57	58	57	**57**
MM	72	79	73	**75**	JM	81	82	81	**81**
DoP	56	56	57	**56**	SS	77	76	79	**77**
BP	72	73	67	**71**	JS	n/a	n/a	n/a	**n/a**
DP	76	78	77	**77**	LS	72	76	69	**72**
RS	70	76	83	**76**	PV	n/a	n/a	n/a	**n/a**
BS	67	68	67	**67**	BW	68	70	74	**71**
MS	69	70	72	**70**	CW	64	66	65	**65**
ST	n/a	n/a	n/a	**n/a**	DW	68	71	72	**70**
					RW	n/a	n/a	n/a	**n/a**
Mean	**72**	**73**	**72**	**72**	**Mean**	**70**	**71**	**71**	**71**
SEM	**2.21**	**2.15**	**2.01**	**2.02**	**SEM**	**1.90**	**2.03**	**2.14**	**2.02**

All data represented as beats per minute (bpm)
SEM, standard error of the mean

Appendix B – Analysis of Variance Summary Tables

Seated Data – Repeated measures ANOVA (Pre-Post)

Systolic blood pressure

Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
Group (G)	1	2.7710	46	364.4489	0.00760	0.930894
Time (T)	**1**	**455.2281**	**46**	**22.4190**	**20.30544**	**0.000045**
G x T	**1**	**533.3137**	**46**	**22.4190**	**23.78844**	**0.000013**

Marked Effects at p<0.05

Post Hoc – Tukey's HSD for Group x Time

Group		{1}	{2}	{3}	{4}
1	Pre {1}		**0.000171**	**0.012936**	**0.025694**
1	Post {2}	**0.000171**		**0.006815**	**0.003258**
2	Pre {3}	**0.012936**	**0.006815**		0.993651
2	Post {4}	**0.025694**	**0.003258**	0.993651	

Marked Effects at p<0.05

Diastolic blood pressure

Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
Group (G)	1	15.50434	46	81.08303	0.191216	0.663953
Time (T)	**1**	**40.87260**	**46**	**5.36421**	**7.619499**	**0.008264**
G x T	**1**	**35.69720**	**46**	**5.36421**	**6.654698**	**0.013150**

Marked Effects at p<0.05

Post Hoc – Tukey's HSD for Group x Time

Group		{1}	{2}	{3}	{4}
1	Pre {1}		**0.002601**	**0.020471**	**0.014646**
1	Post {2}	**0.002601**		0.876406	0.924589
2	Pre {3}	**0.020471**	0.876406		0.999317
2	Post {4}	**0.014646**	0.924589	0.999317	

Marked Effects at p<0.05

Pulse Pressure

Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
Group (G)	1	31.3845	46	275.1199	0.11408	0.737087
Time (T)	**1**	**223.2905**	**46**	**10.5296**	**21.20607**	**0.000033**
G x T	**1**	**293.0558**	**46**	**10.5296**	**27.83174**	**0.000003**

Marked Effects at p<0.05

Post Hoc – Tukey's HSD for Group x Time

Group		{1}	{2}	{3}	{4}
1	Pre {1}		**0.000171**	0.071758	0.190198
1	Post {2}	**0.000171**		**0.000428**	**0.000220**
2	Pre {3}	0.071758	**0.000428**		0.964458
2	Post {4}	0.190198	**0.000220**	0.964458	

Marked Effects at p<0.05

Heart Rate

Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
Group (G)	1	81.49377	46	178.2219	0.457260	0.502293
Time (T)	1	6.82133	46	10.8361	0.629503	0.431611
G x T	1	0.67503	46	10.8361	0.062294	0.804017

Marked Effects at p<0.05

Appendix C – Measures of Reproducibility

Coefficients of Variation (CV)
Baseline Measurements
Control Group

Subject	SBP	DBP	HR	Mean
BA	2.0	1.3	2.2	1.9
JiA	3.9	1.9	2.7	2.8
JoA	2.5	3.4	4.0	3.3
AB	2.7	1.1	3.5	2.4
DB	2.5	1.0	2.1	1.9
NB	2.5	2.5	4.8	3.3
RD	3.1	0.8	2.6	2.2
BE	3.7	3.9	3.2	3.6
BG	2.9	0.5	2.1	1.8
GG	3.6	2.4	2.2	2.8
AH	3.1	3.4	1.1	2.5
OH	2.8	3.1	1.6	2.5
UH	4.1	2.4	2.0	2.8
BK	2.2	1.1	1.7	1.7
JK	1.7	2.1	1.4	1.7
NK	2.9	2.8	2.8	2.8
MM	2.2	1.7	2.6	2.2
DoP	2.4	3.0	2.5	2.6
BP	4.3	1.5	2.7	2.8
DP	3.5	2.4	0.9	2.3
RS	4.2	2.2	5.1	3.8
BS	4.0	5.3	2.8	4.1
MS	4.3	4.2	1.9	3.5
ST	1.3	1.6	1.2	1.3
Mean	**3.0**	**2.3**	**2.5**	**2.6**
SEM	**0.18**	**0.24**	**0.22**	**0.15**

All data, excluding SEM represented as percent (%)
SEM, standard error of the mean

Coefficients of Variation (CV)

Baseline Measurements
Training Group

Subject	SBP	DBP	HR	Mean
AA	3.1	1.4	1.9	2.1
SA	4.4	2.8	1.6	3.0
JB	1.3	3.0	1.9	2.1
FC	2.0	1.9	1.6	1.8
JC	3.7	1.3	5.1	3.4
BoE	4.0	3.0	2.6	3.2
AE	3.2	1.6	1.4	2.1
BF	2.2	2.2	2.5	2.3
JF	1.6	1.4	2.3	1.8
VI	1.2	2.9	1.2	1.8
RJ	4.0	2.0	3.1	3.0
TJ	3.5	2.2	1.8	2.5
LK	2.6	1.5	2.2	2.1
AL	1.7	2.1	1.1	1.6
JL	6.7	3.7	3.2	4.5
BM	1.8	1.2	2.6	1.9
JM	3.0	1.8	4.2	3.0
SS	3.5	1.5	2.1	2.4
JS	6.5	8.3	13.5	9.4
LS	3.7	3.0	2.0	2.9
PV	2.9	1.8	1.3	2.0
BW	2.6	1.1	2.1	1.9
CW	1.4	3.2	0.9	1.9
DW	2.6	1.9	1.4	2.0
RW	2.8	1.6	0.8	1.7
Mean	**3.4**	**2.3**	**2.6**	**2.6**
SEM	**0.28**	**0.29**	**0.50**	**0.31**

All data, excluding SEM represented as percent (%)
SEM, standard error of the mean

Coefficients of Variation (CV)

Post Measurements
Control Group

Subject	SBP	DBP	HR	Mean
BA	1.6	4.5	1.1	2.4
JiA	3.2	2.5	0.8	2.2
JoA	2.0	2.3	2.5	2.3
AB	4.1	2.3	2.6	3.0
DB	5.6	1.6	1.5	2.9
NB	4.1	3.9	2.7	3.6
RD	2.5	1.9	2.1	2.2
BE	2.5	2.8	2.2	2.5
BG	2.8	1.3	2.2	2.1
GG	2.9	1.4	1.2	1.8
AH	2.2	3.1	2.5	2.6
OH	2.7	4.2	1.4	2.7
UH	4.2	3.2	3.1	3.5
BK	2.0	1.7	1.8	1.9
JK	3.0	2.9	1.7	2.5
NK	1.6	3.1	3.6	2.8
MM	2.4	2.8	4.8	3.4
DoP	3.0	2.1	1.3	2.1
BP	4.5	2.3	3.0	3.3
DP	4.7	1.8	1.6	2.7
RS	4.0	6.4	5.1	5.2
BS	3.6	3.1	2.3	3.0
MS	2.6	3.5	1.9	2.7
ST	1.1	2.3	3.9	2.4
Mean	**3.0**	**2.8**	**2.4**	**2.7**
SEM	**0.23**	**0.23**	**0.23**	**0.15**

All data, excluding SEM represented as percent (%)
SEM, standard error of the mean

Coefficients of Variation (CV)

Post Measurements
Training Group

Subject	SBP	DBP	HR	Mean
AA	1.4	1.0	1.4	1.3
SA	4.4	3.7	3.1	3.8
JB	1.4	2.2	1.6	1.7
FC	1.8	0.9	0.8	1.1
JC	3.0	1.8	2.1	2.3
BoE	1.8	1.2	1.9	1.7
AE	3.9	2.1	1.8	2.6
BF	1.1	2.4	1.7	1.7
JF	3.0	3.2	1.5	2.6
VI	1.9	1.4	3.9	2.4
RJ	3.6	2.9	2.2	2.9
TJ	2.7	2.5	5.1	3.4
LK	2.4	2.0	3.4	2.6
AL	1.9	1.1	1.3	1.4
JL	2.0	1.3	2.5	1.9
BM	2.2	3.9	2.7	3.0
JM	2.8	1.4	2.4	2.2
SS	1.2	1.5	2.7	1.8
JS	5.7	8.2	23.2	12.4
LS	3.4	4.2	2.8	3.5
PV	n/a	n/a	n/a	n/a
BW	1.7	0.9	2.1	1.6
CW	1.7	4.3	1.8	2.6
DW	2.8	1.6	1.4	1.9
RW	3.1	1.6	1.3	2.0
Mean	**2.5**	**2.4**	**3.1**	**2.7**
SEM	**0.23**	**0.33**	**0.89**	**0.45**

All data, excluding SEM represented as percent (%)
SEM, standard error of the mean

Intraclass Correlation Coefficients (ICC)

Baseline systolic blood pressure measurements

Group	1-2	2-3	1-3	Mean
All	0.84	0.92	0.67	0.81
Control	0.80	0.90	0.53	0.74
Training	0.88	0.94	0.79	0.87
Mean	**0.84**	**0.92**	**0.66**	**0.81**
SEM	**0.02**	**0.01**	**0.08**	**0.04**

Represented as r; SEM, standard error of the mean

Baseline diastolic blood pressure measurements

Group	1-2	2-3	1-3	Mean
All	0.83	0.89	0.68	0.80
Control	0.85	0.90	0.72	0.82
Training	0.87	0.87	0.64	0.80
Mean	**0.85**	**0.89**	**0.68**	**0.81**
SEM	**0.01**	**0.01**	**0.02**	**0.01**

Represented as r; SEM, standard error of the mean

Post training systolic blood pressure measurements

Group	1-2	2-3	1-3	Mean
All	0.91	0.93	0.88	0.91
Control	0.89	0.95	0.87	0.91
Training	0.95	0.93	0.87	0.92
Mean	**0.92**	**0.94**	**0.87**	**0.91**
SEM	**0.02**	**0.01**	**0.003**	**0.003**

Represented as r; SEM, standard error of the mean

Post training diastolic blood pressure measurements

Group	1-2	2-3	1-3	Mean
All	0.85	0.86	0.59	0.77
Control	0.86	0.85	0.65	0.79
Training	0.85	0.89	0.50	0.75
Mean	**0.85**	**0.87**	**0.58**	**0.77**
SEM	**0.003**	**0.01**	**0.04**	**0.01**

Represented as r; SEM, standard error of the mean

Appendix D – Figures and Illustrations

Table 11. Typical ESH validation procedure for automated blood pressure acquisition devices. This device reaches the third phase (2.2) before failing to meet requirements for approved validity.

Phase 1		within 5 mmHg	within 10 mmHg	within 15 mmHg	Recommendation		
Required	One of	25	35	40			
Achieved	SBP	22	35	43	Continue		
	DBP	35	42	44	Continue		
Phase 2.1		**within 5 mmHg**	**within 10 mmHg**	**within 15 mmHg**	**Recommendation**	**Mean difference**	**Standard deviation**
Required	Two of	65	80	95			
	All of	60	75	90			
Achieved	SBP	52	79	90	Fail	3.4 mmHg	8.4 mmHg
	DBP	77	90	94	Pass	-0.6 mmHg	6.9 mmHg
Phase 2.2		**2/3 within 5 mmHg**	**0/3 within 5 mmHg**		**Recommendation**		
Required		⩾22	⩽3				
Achieved	SBP	17	4		Fail		
	DBP	28	2		Pass		

The device passes for diastolic blood pressure (DBP, but fails for systolic blood pressure (SBP), thereby failing overall.

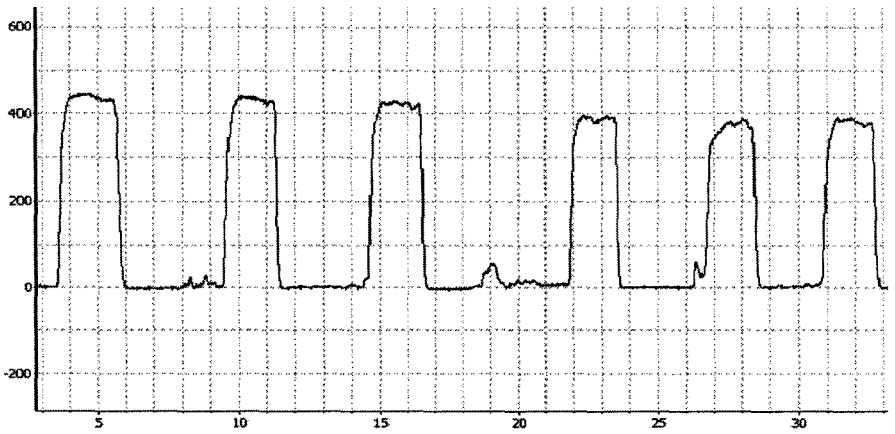

Figure 6. Typical hand dynamometer output used to determine maximal hand strength. Values expressed in Newtons (Y-axis) and seconds (X-axis).

Table 12. Pre- and post- training analysis of handgrip resistance.

	Pre-	Post-
Handgripper	**Resistance Range**	**Resistance Range**
Green (Easy)	16-24	16-23
Red (Medium)	20-33	22-32
Blue (Hard)	31-51	30-44