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Workplace Standing Desks and Arterial Stiffness

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WORKPLACE STANDING DESKS AND ARTERIAL STIFFNESS

By

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List of abbreviations

AIx	Aortic Augmentation Index
ATP	Adenosine Triphosphate
cfPWV	Carotid-Femoral Pulse Wave Velocity
crPWV	Carotid-Radial Pulse Wave Velocity
CVD	Cardiovascular Disease
DAP	Diastolic Arterial Pressure
ECG	Electrocardiogram
HDL	High Density Lipoprotein
LDL	Low Density Lipoprotein
LPL	Lipoprotein Lipase
lPWV	Leg Pulse Wave Velocity, or Femoral-Dorsalis Pedis PWV
MET	Metabolic Equivalent of a Task
NO	Nitric Oxide
PP	Pulse Pressure
PWA	Pulse Wave Analysis
SAP	Systolic Arterial Pressure

Abstract

Many jobs in today's society require sitting at a desk with little physical activity. Individuals who engage in ten hours of sedentary behavior per day double their CVD risk. Standing desks are thought to decrease sedentary time and improve cardiovascular health. Acute use of standing desks is shown to lower PWV. However, chronic effects remain unknown. Forty eight participants qualified as seated (19 females, 5 males: age 41 ± 2 years, BMI 25 ± 1 kg/m^2) or standing (21 females, 3 males: age 45 ± 2 years, BMI 25 ± 1 kg/m^2) groups based on habitual workplace use. Arterial stiffness was assessed as pulse wave velocity (PWV) by using applanation tonometry in conjunction with electrocardiography. No differences were detected in carotid-femoral PWV (cfPWV) between seated and standing groups ($p = 0.47$). However, age ($p < 0.01$), aerobic fitness ($p < 0.01$), and fat percentage ($p = 0.02$) classifications revealed significant differences between groups. Standing for 50% of a workday does not affect cfPWV. Although, cardiorespiratory fitness and healthy body composition are associated with less arterial stiffness.

1 Introduction

To further explore the potential relation between workplace standing desks and arterial stiffness, the introduction portion of this thesis will focus on historic vs. current employment modalities to demonstrate how this may have contributed to the increase in sedentary activity in the United States. The associated negative health consequences of sedentary behavior along with recommendations of physical activity will be discussed. Arterial stiffness, an indicator of cardiovascular health, will be introduced along with the technique used to assess arterial health and factors that can influence it. Finally, alternative workstations, namely standing desks, will be introduced to examine their effect on workers who regularly use them to potentially influence overall health and arterial stiffness.

1.1 Historic Employment vs. Today

Since the turn of the 20th century to present, employment in the United States has changed drastically. Take for example the types of jobs that founded many of the cities and villages of the Upper Peninsula of Michigan and the Midwest region. Employment consisted of occupations such as farming, logging, and mining, which were labor intensive. In the early 1900s, 38% of the labor force consisted of farmers compared to less than 3% by 2000. In contrast, the service industry grew dramatically throughout the 20th century, indicative of the growth of healthcare, education, personal services, and the business community. In 1900, 31% were employed in service compared to 78% in 1999 (Fisk, 2001). This shift of the service industry becoming the largest portion of the United States economy changed the

way Americans work every day. Those who may have worked on the farm, in the forest, or in a mine, now could find themselves at a desk sitting in front of a computer for extended portions of the day.

1.1.1 Shift from Active to Sedentary Work

Since the 1960s, the American work place has undergone a massive transformation, to primarily benefit productivity. However, this productivity may come at a cost to human health. Previously, much of the American workforce consisted of jobs in agriculture and goods producing which required significant energy expenditure from the worker. Beginning in the 1960s, new jobs entering the workforce required more sedentary activity like desk work. Nearly 50% of all jobs in 1960 required moderate intensity physical activity, decreasing to 30% by 1970 compared to a dismal 20% in today's workplace (Church et al., 2011). Additionally, jobs where the worker is sedentary, or only required to perform light physical activity, doubled from 20% to 40% between 1970 and 2000 (Brownson, Boehmer, & Luke, 2005). Several physiologists noted differences in employee health between the 1950s and 1990s (Convertino, Bloomfield, & Greenleaf, 1997; Morris, Heady, Raffle, Roberts, & Parks, 1953; Norman, 1958). Much of the classic work done in the United Kingdom is considered the advent of sedentary behavior research.

1.2 Types of Employment & Health Consequences

1.2.1 London Bus Drivers and Post Office Workers

The very beginnings of modern inactivity physiology began in the 1950s when physiologists examined the health status of a variety of occupations in the United Kingdom. Specifically, a group examined the health of London bus workers and their occupation as either the driver or the conductor. Novel observations of the time was the lack of physical activity of the bus drivers as compared to the conductors, who move about the vehicle throughout the day (Morris et al., 1953; Norman, 1958). The participants were followed longitudinally for nearly ten years, and the risk of myocardial infarction in the bus drivers was twice that of bus conductors. It was noted that this risk was apparent independent of the individual's physique (i.e. measurements of chest, waist, and hip circumference) (Morris, Kagan, Pattison, & Gardner, 1966). These findings provided some of the first clinical evidence of physical inactivity and the relation to human health. Similar associations of cardiovascular disease incidence was observed between sedentary government employees and postmen. The decreased level of coronary artery disease, when compared to seated government employees, was attributed, in part, to the increased physical activity of the postmen (Morris et al., 1953). These classic studies provide the first examples that simply moving more throughout the day can significantly benefit human health.

1.2.2 Dallas Astronaut Studies

Other classic studies with detrimental implications for sedentary behavior include a variety of bed rest studies. Bed rest studies began during the World War II and space race eras where they sought to examine the effect of prolonged hospitalization and microgravity on human physiology. The consensus on a variety of work confirmed prolonged best rest had lasting negative effects on the cardiovascular and musculoskeletal systems in addition to many other body systems (Convertino et al., 1997). One of the most striking studies involved four NASA astronauts who were enrolled in a 21-day bed rest study, where their aerobic capacity was measured before and after the bed rest intervention. The bed rest significantly reduced the astronauts aerobic capacity by an average of 26%, providing insight of the effect of microgravity and the act of doing nothing has on the body (Saltin, 1968). Another group of researchers decided to follow up on the same group of astronauts 30 and 40 years later. The group of astronauts still had higher VO_{2max} values three decades later compared to the 21-day bed rest intervention (McGuire et al., 2001). Forty years of aging produced similar decreases in VO_{2max} as did a mere 21 days of bed rest, 27% vs. 26% respectively (McGavock et al., 2009). Bed rest studies continue to provide evidence of how acute sedentary behavior alters human physiology and has potential to reveal new mechanistic insight as to why “sitting is the new smoking” (Baddeley, Sornalingam, & Cooper, 2016).

1.3 Sedentary Behavior

1.3.1 Definition and Classification of Sedentary Behavior

Sedentary behavior is an epidemic that plagues the daily lives of American citizens, which is predicted to become worse with further advancements in technology. However, what is the actual definition of sedentary behavior from a physiological perspective? Given 1 metabolic equivalent of a task (MET) is 3.5 mL/kg/min or an individual's resting basal metabolic rate, Gibbs, et al. (2015) concluded any seated activity less than 1.5 MET is classified as sedentary behavior (American College of Sports Medicine, 2013). There remains an open debate as to whether standing activities are classified as sedentary behavior (B. B. Gibbs, Hergenroeder, Katzmarzyk, Lee, & Jakicic, 2015).

1.3.2 Negative Health Outcomes

A popular buzz phrase related to sedentary behavior literature is "sitting is the new smoking" (Baddeley et al., 2016). Some may deem this an exaggeration, however many studies highlight that simply doing nothing can be as detrimental to human health as smoking. For example, a recent study on older cigarette smokers reported a hazard ratio of 2.81, or 181% more likelihood to die from a cardiovascular disease (CVD) (Taghizadeh, Vonk, & Boezen, 2016). In comparison, a 2012 review found individuals who reported long bouts of sedentary activity are associated with a 147% increased risk of CVD or cardiovascular event. Engaging in sedentary behavior also increased cardiovascular mortality by 90% (Wilmot et al., 2012). Further examination of women with CVD like coronary artery

disease and cerebrovascular events revealed a 63% increased risk when average sitting time was 10 or more hours per day (Chomistek et al., 2013). The risk of CVD is further increased when obesity is factored into physical activity status (Warren et al., 2010). There remains a need to outline specific mechanisms associated with the detrimental changes of sedentary behavior (Hamilton, Hamilton, & Zderic, 2007).

1.3.3 Physiologic Mechanisms of Sedentary Behavior

Lipoprotein Lipase

In an effort to provide mechanistic insight into the relation between sedentary behavior and a variety of CVDs, experts in the field of inactivity physiology suggested the role of lipoprotein lipase (LPL) and its regulation. In healthy individuals, the LPL enzyme is located within the vasculature where triglycerides are catabolized and shuttled into glycolytic muscle tissue for energy production (Miles et al., 2004). Research in animal models revealed a reduction in LPL activity led to an increase in the triglyceride level in circulation (Bey & Hamilton, 2003), which puts the individual at increased risk of metabolic syndrome development. This accumulation of triglycerides in the blood is also one of the hallmarks of atherosclerotic plaque formation in the arteries (Huang, 2009). Hamilton and colleagues determined that immobilization of a rat's hind limb caused decreased activity of LPL as the energy demand decreased (Zderic & Hamilton, 2006). In humans, female trained distance runners demonstrate increased LPL activity and improved triglycerides compared to controls (Podl et al., 1994). Taken

together, LPL activity is regulated by energy demand of surrounding tissue. LPL activity, and other regulators, can contribute to CVDs.

Nitric Oxide and Endothelin-1

The antagonistic regulators of blood vessel diameter, nitric oxide (NO) and endothelin-1, undergo differing gene regulation and expression during prolonged sedentary behavior. During exercise, NO is released from the endothelial cells that line the walls of arteries due to increased shear stress. Shear stress is created when arterial blood flow increases. NO, a powerful vasodilator, increases vessel diameter to accommodate the increased blood volume to be delivered to exercising muscle (Zhang et al., 2006). Exercise also results in down-regulated expression of endothelin-1, a vasoconstrictor. Numerous studies report the ability of exercise to decrease expression of endothelin-1 (Maeda et al., 2001; Maeda et al., 2003; Van Guilder, Westby, Greiner, Stauffer, & DeSouza, 2007). However, after engaging in long bouts of sedentary activity, NO expression and bioavailability remain unchanged (Donato et al., 2009; Thosar, Johnson, Johnston, & Wallace, 2012). In contrast, sedentary bouts, in conjunction with aging, can result in overexpression of endothelin-1 (Donato et al., 2009) to suggest that sedentary activity problems with blood pressure may be a result of overexpression of endothelin-1 rather than decreased NO. This overexpression of endothelin-1 can contribute to hypertension and ultimately lead to structural changes to the vasculature, making it less elastic and accepting of increases in blood volume (Marti et al., 2012).

1.3.4 Physical Activity Recommendations

Increased energy expenditure is associated with a variety of health indicators which range from reductions in blood pressure (Sriram, Hunter, Fisher, & Brock, 2014), weight management (Muller, Enderle, & Bosy-Westphal, 2016), and improvement of plasma triglycerides (Hirose et al., 2015). In principal, increased energy expenditure triggers increased energy production in the form of adenosine triphosphate (ATP). Production of ATP is primarily through glucose and fat oxidation (Rosen & Spiegelman, 2006). An increase in fat oxidation has the potential to decrease body adiposity, ultimately improving weight, body mass index (BMI), and waist circumference (Esposito et al., 2003; Kelley, Goodpaster, Wing, & Simoneau, 1999). Protein oxidation constitutes a small percentage of total energy production, typically reserved for extreme circumstances (Dickerson, Guenter, Gennarelli, Dempsey, & Mullen, 1990). Recently, the ACSM released new guidelines for maintaining fitness in normal, healthy adults. Included in the recommendation are guidelines for maintaining cardiorespiratory health by engagement in 150 minutes of moderate exercise per week, 75 minutes of vigorous exercise per week, or any combination moderate or vigorous of exercise that results in energy expenditure of 500-1000 MET minutes per week or greater (Garber et al., 2011). Moderate or vigorous physical activity will not result from working at a standing desk for set amount of time.

1.3.5 Increased Physical Activity and Positive Health Outcomes

Engagement in physical activity and exercise as recommended by the ACSM can help to produce a variety of positive health outcomes. Training induced improvements can improve oxygen delivery to the muscular tissue via increased capillary density (Mandroukas et al., 1984; Warburton, Nicol, & Bredin, 2006). In combination with improvement in muscle oxidative capacity via proliferation of type I muscle fibers (Schiaffino & Reggiani, 2011), VO_{2max} can increase (Mandroukas et al., 1984; Warburton, Gledhill, & Quinney, 2001). In addition, interventions aimed at increased physical activity, namely moderate and vigorous intensity, is able to reduce or prevent increased fat percentage in children (Ruiz et al., 2006), men (King, Haskell, Young, Oka, & Stefanick, 1995), premenopausal women (Trapp, Chisholm, Freund, & Boutcher, 2008) and postmenopausal women (Irwin et al., 2003). However, light physical activity can produce some of the same health benefits as moderate or vigorous activity.

1.3.6 Light Physical Activity and Human Health

Light physical activity is defined as any activity capable of utilizing 3.5 kilocalories per minute or an energy expenditure of 1.5 – 3.0 METs (American College of Sports Medicine, 2013; Healy et al., 2007). Common examples of light physical activity includes easy walking or biking. This physical activity category is of particular importance to older individuals and is associated with physical health (Buman et al., 2010). When compared to sedentary behavior, light physical activity revealed the ability to significantly reduce both central and brachial blood

pressures (Gerage et al., 2015). With the health benefits of light activity, there remains the question of whether standing is enough to produce an energy expenditure equivalent to at least 1.5 METs and have an impact on blood pressure and arterial stiffness (i.e. ability of arteries to expand and recoil with each cardiac cycle).

1.4 Arterial Stiffness

1.4.1 Normal Arterial Function

In young, healthy individuals, the arteries of the cardiovascular system possess a large amount of distensibility, or the ability to stretch. During systole, fresh, oxygenated blood is ejected from the left ventricle of the heart, passes through the aortic semilunar value, into the aorta. The addition of new blood volume to systemic circulation causes the aorta to stretch. The ability of the aorta to stretch inhibits excessive increases in blood pressure (London & Guerin, 1999). As the heart enters diastole, negative or decreased pressure within the ventricle causes the aortic values to close. The elastic recoil of the aorta allows for the preservation of both blood flow and diastolic pressure (Michel E Safar, 2004). In addition to blood ejection into systemic circulation, the heart contraction produces a pulse wave that travels through the vasculature. This pulse wave is also called the palpable pulse, which can be felt most commonly at the wrist or neck. Reflected pulse wave timing in reference to systole and diastole can either be beneficial or detrimental to the heart.

Pulse Wave Reflection

With each cardiac cycle, a pulse wave is sent through the vasculature during systole. This wave travels forward through the aorta. As the aorta begins to branch into smaller arteries and arterioles, the initial pulse wave sends a forward wave into the smaller arteries, but also sends a reflected wave back toward the heart (London & Guerin, 1999). The reflected waves have the potential to cause additional stress on the aorta in older individuals or individuals who have abnormally high arterial stiffness for their age. Increased arterial stiffness has the potential risk of being pathological as stiffness can affect the timing of when the reflected waves return to the aorta (Mayet & Hughes, 2003). In a young, healthy individual, the reflected wave arrives during diastole, when the reflected wave can

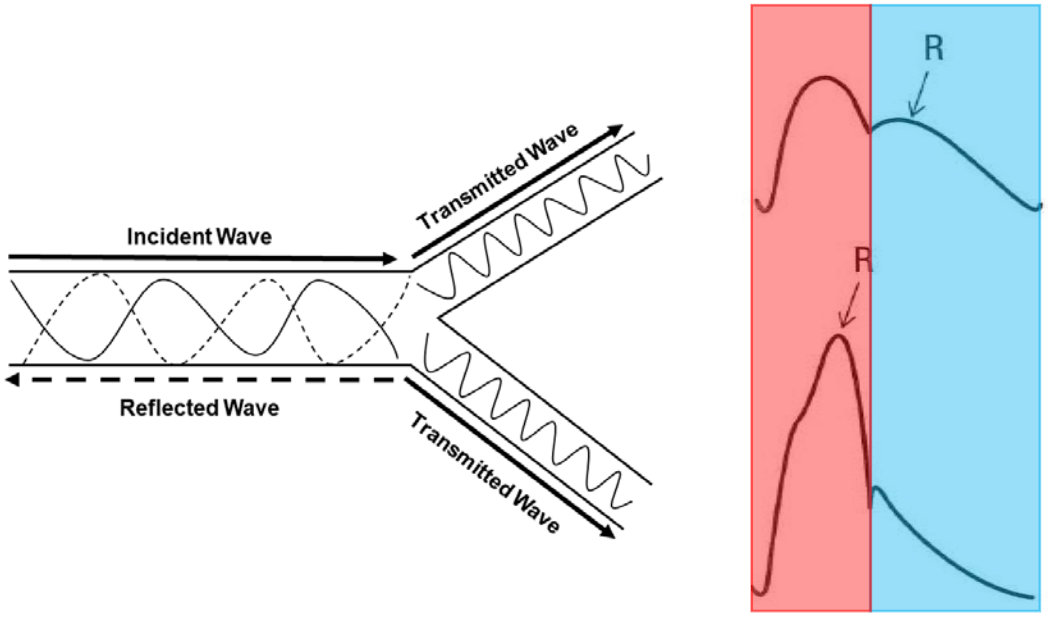

Figure 1.1. Wave reflection associated with low arterial stiffness (top BP waveform) and wave reflection associated with high arterial stiffness (bottom BP waveform). Reflected waves arise from artery branch points or areas of stiffness within the vasculature. Reflected waves during systole (e.g. bottom waveform) can place added stress on the heart.

help to further perfuse the coronary arteries to aid with oxygen delivery to the myocardium (Kelly, Daley, Avolio, & O'Rourke, 1989; London & Guerin, 1999). In an older or unhealthy individual, the reflective wave returns during systole and further increases the blood pressure in the aorta. Over a long period of time, the added stress can lead to further stiffening of the aorta and increases in the aortic systolic pressure and decreases in aortic diastolic pressure. This forces the heart to generate more and more force with each heart contraction and increased stress on the vasculature with larger changes in pulse pressure.

1.4.2 Methodological Development

While brachial blood pressure is still considered an excellent screening tool for cardiovascular diseases and serves as an accurate predictor of future cardiovascular events, blood pressure varies within the arterial division of the cardiovascular system (Carmel M. McEniery, Cockcroft, Roman, Franklin, & Wilkinson, 2014). A healthy individual's aortic systolic pressure is normally lower than the corresponding brachial blood pressure due to artery distensibility changes in periphery and vessel radius (Roman et al., 2009). However, instances arise where brachial blood pressure values are normal, or near normal, and the aortic blood pressure is comparable to the brachial blood pressure (Carmel M McEniery et al., 2008). This discrepancy may be evident from differing levels of stiffness of the large arteries elevating central blood pressure (Michel E Safar, Levy, & Struijker-Boudier, 2003). This discovery outlines the need for direct assessment of aortic blood pressure as an independent risk factor for cardiovascular disease.

A variety of techniques exist for the assessment of arterial stiffness in humans. Invasive measures include implantation of aortic catheters, which are equipped with pressure transducers to obtain measures of blood pressure at the level of the heart (Chen et al., 1998; Currie et al., 1985; Kawaguchi, Hay, Fetics, & Kass, 2003). Early work in the animal model confirmed pressure within the aorta was equivocal to pressure in the left ventricle during systole as blood is ejected into systemic circulation (Wiggers, 1928). In an effort to determine a less invasive technique of determining aortic blood pressure, radial artery catheterization can be used to generate aortic blood pressure via a generalized transfer function to generate an aortic blood pressure waveform. Actual and computer modeled aortic wave forms prove comparable and reliable (Chen et al., 1997; Pauca, O'rourke, & Kon, 2001). The new technique termed applanation tonometry, which is proven to be reliable and repeatable, is now wildly used for cardiovascular research (Crilly, Coch, Bruce, Clark, & Williams, 2007; Papaioannou et al., 2004; Wilkinson et al., 1998).

1.4.3 Applanation Tonometry

Arterial stiffness is measured through two main techniques associated with applanation tonometry. Pulse wave analysis (PWA) is a rapid recording where a tonometer records pressure waves of an artery of interest, most often the radial

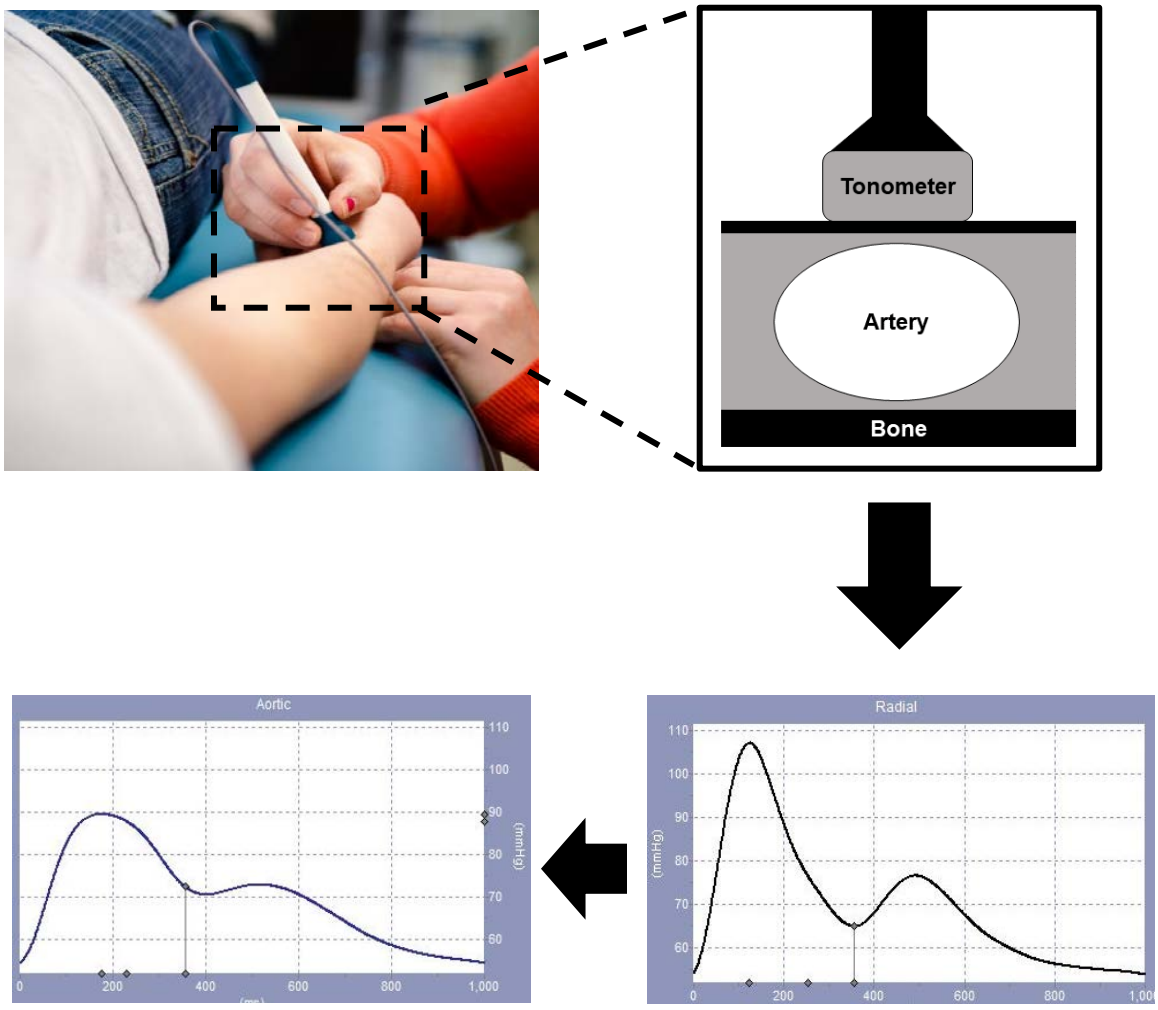

Figure 1.2. Applanation tonometry, the process of flattening out an artery of interest against a hard surface, like bone, to obtain pulse-wave recordings. A general transfer function can generate the estimated aortic blood pressure waveform.

artery. SphygmoCor computer software is used to analyze characteristics of the pulse wave. When calibrated to a brachial blood pressure, this measure can provide estimates of the blood pressure waveform in the aorta, via generalized transfer function, to generate aortic blood pressure (systolic, diastolic, mean, and pulse pressure), which are confirmed against aortic and radial catheterization (Adji, Hirata, Hoegler, & O'Rourke, 2007; Chen et al., 1997). Additionally, an aortic augmentation index is calculated from the characteristics of the pulse wave. This

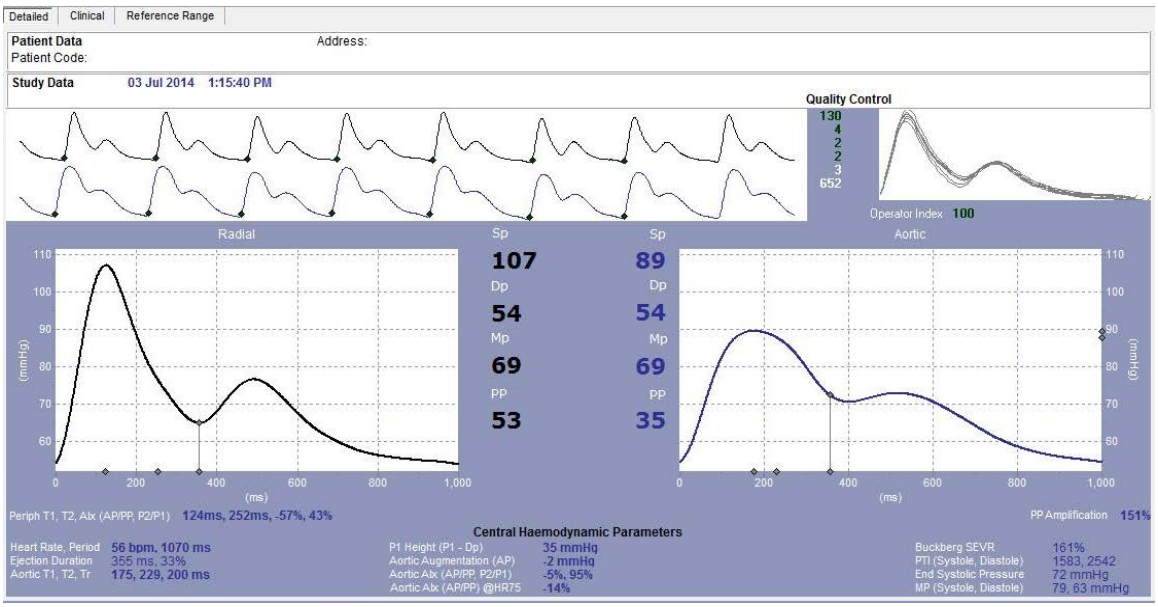

Figure 1.3. Sample pulse wave analysis (PWA) recording of the radial artery following calibration with brachial BP. Via generalized transfer function, pulse wave characteristics can estimate aortic SAP, DAP, MAP, and PP.

is defined as the quotient of the aortic augmentation pressure (i.e. aortic systolic pressure – blood pressure at inflection point, AIx) and aortic pulse pressure. This index can also be normalized to 75 heart beats, however there is debate as to when to use the normalized value vs. the non-normalized value (Stoner et al., 2014). However, AIx is recognized to be dependent on heart rate, body height, and blood ejection duration (Townsend et al., 2015).

Another technique under the classification of applanation tonometry is pulse wave velocity, where pulse wave speed can be estimated. Carotid-femoral pulse wave velocity (cfPWV) is often considered the gold standard indicator of cardiovascular health assessed with applanation tonometry, where a 1.0 m/s increase in velocity increases the risk of a cardiovascular event by 15% (Vlachopoulos, Aznaouridis, & Stefanadis, 2010). This measurement is done in

Figure 1.4. Sample pulse wave velocity (PWV) recording of cfPWV. Based on pulse wave distance, time delay of pulse creation to pulse arrival is recorded at carotid and femoral sites. A speed in meters per second is calculated.

tandem with an electrocardiogram recording. Each R wave of the ECG serves as the creation of the pulse wave. Two pulse sites are referenced where the distance from the suprasternal notch (i.e. location of the aorta) to each pulse site is measured. The tonometer is placed over the artery to examine when the pulse wave arrives. The time delay is calculated from the pulse wave creation (i.e. R wave) and the pulse wave arrival (i.e. waveform upstroke) for each cardiac cycle. The data collection software uses the distance from the aorta and time delay of pulse wave arrival to calculate the pulse wave velocity (Doupis, Papanas, Cohen, McFarlan, & Horton, 2016). A cfPWV below 10 m/s is considered to normal, whereas upwards of 10 m/s may be indicative of arterial stiffness within the vasculature (Van Bortel et al., 2012).

1.4.4 Pathological Associations with Arterial Stiffness

Arterial stiffness is associated with a variety of cardiovascular diseases including hypertension, atherosclerosis, etc. There has long been a debate on whether arterial stiffness is the precursor to hypertension or if the inverse is true. A review by Franklin addressed this concern where he suggested an interplay between hypertension and arterial stiffness (Franklin, 2005), now termed a “vicious cycle”. High arterial stiffness can induce incident hypertension (Tomiyama & Yamashina, 2012). Left untreated, rapid increases in arterial stiffening occur, which can further increase the severity of hypertension (Franklin et al., 1997). Long term stress on arterial walls triggers vascular remodeling, which is can be categorized as, hypertrophic, hypotrophic, or eutrophic, corresponding to increase, decrease, or unchanged amount of new tissue in the blood vessel (Mulvany et al., 1996; Schiffrin, 2012). Inward eutrophic and hypertrophic remodeling are common within smaller arteries undergoing the stress of hypertension (Schiffrin, 2012). Inward eutrophic remodeling leads to decreased size of the vessel ultimately leading to decreased lumen diameter. Inward hypertrophic remodeling also reduces the lumen diameter via increased lumen endothelial growth. Large artery stiffness is characterized by outward hypotrophic growth where the lumen diameter is increased (Schiffrin, 2012). Over time, the elastin within the vessels is broken down and replaced with less compliant, dense collagen (O'rourke, 1990). This phenomenon explains, in part, why stiff arteries produce increases in pulse pressure and disruptions in blood flow (Renna, de Las Heras, & Miatello, 2013). In addition, chronic stress on arterial walls can lead to inflammation (Booth et al.,

2004), where the inner layers of the vasculature are not replaced or undergo the same remodeling process as other arterial layers. Inflammation within the vasculature can lead to the buildup of LDL cholesterol, which can exacerbate the progression of atherosclerotic plaque formation (Libby, 2012).

Another pathological aspect of arterial stiffness includes the potential of end organ damage. Common examples include the kidney, brain and heart (Gary F Mitchell, 2008). As stated, the disruptions in blood flow from vascular remodeling contributes to more of a pulsatile blood flow rather than a constant flow. Decreased myocardial perfusion is evident in individuals with high arterial stiffness, where reflected pulse waves arrive to the heart during systole, rather than diastole, increasing the risk of myocardial ischemia from increased contractility and decreased oxygen availability (Kelly et al., 1989; London & Guerin, 1999). Increased pulsatility to the brain and kidney puts added stress on the microvasculature. Within the kidney, the glomerulus vasculature becomes damaged, which can allow large molecules like proteins into the urine (M. E. Safar, Nilsson, Blacher, & Mimran, 2012). Within the brain, there are associations between high arterial stiffness and beta-amyloid plaque deposition. Because of the decrease in blood vessel integrity from increases in pulsatility, the vasculature acts comparable to the kidney, allowing larger substances move across the blood brain barrier, which may contribute to the progression of Alzheimer's disease (Singer, Trollor, Baune, Sachdev, & Smith, 2014).

1.5 Factors Influencing Arterial Stiffness

1.5.1 Non-Modifiable Risk Factors

Numerous studies have reported the relationship between age and arterial stiffness (Benetos et al., 2002; Vaitkevicius et al., 1993; Wen et al., 2015). The Framingham Heart Study cohort data showed age was a strong predictor of both cfPWV and reflected wave transit time (i.e. time from reflection to arrival at the heart). PWV increased with age, whereas the reflected wave time decreased. This phenomena may contribute to arterial stiffness related SAP and pulse pressure (PP) increases. (G. F. Mitchell et al., 2004). In addition, there is data to further suggest arterial stiffness progression throughout the aging process independent of hypertension status (Vaitkevicius et al., 1993). However, interventions exist to ameliorate age related increases in arterial stiffness. Other non-modifiable risk factors include sex and ethnicity. African-American men appear to have increased central blood pressure, intima-media thickness, and carotid beta-stiffness as compared to age matched Caucasian men (Heffernan, Jae, Wilund, Woods, & Fernhall, 2008). In addition, African-American men appear to have increased baseline aortic stiffness as compared to Caucasian men (Heffernan, Jae, & Fernhall, 2007). Sex differences of arterial stiffness show women have increased arterial stiffness following menopause compared to men (Coutinho, Borlaug, Pellikka, Turner, & Kullo, 2013), consistent with increased prevalence of hypertension as women age (Oparil & Miller, 2005).

1.5.2 Modifiable Risk Factors

Interventions to improve aerobic capacity are common within the scientific community and range in focus from cognition (Colcombe & Kramer, 2003) to cardiovascular health (Warburton et al., 2006). One cross-sectional study demonstrated the ability of increased aerobic capacity to significantly lower levels of arterial stiffness when compared to sedentary controls (Vaitkevicius et al., 1993). Another modifiable factor is maintenance of a healthy body composition. Increased abdominal visceral fat in obese individuals is associated with increased arterial stiffness (Sutton-Tyrrell et al., 2001; Zebekakis et al., 2005), whereas interventions that decrease obesity also can improve arterial stiffness (Goldberg, Boaz, Matas, Goldberg, & Shargorodsky, 2009). Easy to implement interventions include increasing physical activity throughout the day, and use of an alternative workstation, which can increase energy expenditure and potentially to improve arterial stiffness (Hamasaki, Yanai, Kakei, Noda, & Ezaki, 2015).

1.6 Alternative Workstations

1.6.1 Examples of Alternative Workstations

Sedentary behavior in the workplace is a problem. A variety of new, active workstations were created and are now widely used. These new workstations range from a standing desk, to a biking workstation, to a treadmill desk. The main goal of each alternative workstation is to breakup prolonged seated periods and improve energy expenditure throughout the workday (Torbeyns, Bailey, Bos, & Meeusen, 2014). However, the implementation of new workstations created new

questions to answer. Productivity was a large concern especially when alternative desks were bought for employees rather than in the home. Is the worker as productive while standing, walking, or biking as compared to remaining seated (Karol & Robertson, 2015)? Do the alternative workstations truly provide a significant benefit to health (Torbeyns et al., 2014)? Each question was warranted to justify investment in a new workstations to ensure there would be no harm to the employee or company productivity.

1.6.2 Standing Desk

Perhaps the easiest and simplest of all active workstations, the standing desk, is one of the most popular to encourage reduced sitting time while at work. Most who readily use a standing desk report the desire to move more throughout the day, which is consistent with the principal of active workstations (Levine & Miller, 2007). In addition, there is little evidence to suggest office programs, like intermittent walking, effectively promote a reduction in sedentary time due to poor adherence (Chau et al., 2010). By providing an option of an active workstation like a standing desk, workplace sedentary time and perhaps productivity and health could improve.

1.6.3 Standing Desks and Productivity

Standing workstation productivity was one of the first questions to be posed to the scientific community. The question was of particular interest to employers, whereas an investment in a standing workstation that decreased productivity would

be irrational from a financial perspective. The most frequent test of productivity at workstations are typing performance tests. Two studies report no differences in typing performance (Drury et al., 2008; Ebara et al., 2008). Another study provided evidence of increased productivity with changes in posture like standing, but longer standing time decreased productivity, driven by employee fatigue (Hasegawa, Inoue, Tsutsue, & Kumashiro, 2001). A recent study of a Texas call center, who implemented a standing desk intervention, saw increased productivity (up to 50%) in the form of successful phone calls at months one and six (Garrett et al., 2016). In summary, the majority of research into standing desk productivity provided support for their implementation into the workplace given only long bouts of standing decreased workplace productivity. If a standing desk can both boost productivity and improve employee health, the purchase can be financially justified.

1.6.4 Standing Desks and Health

In an effort to reduce sitting time throughout the day, researchers have inferred that replacement of sitting with standing for part of the workday will positively impact human health and decrease biomarkers for certain cardiovascular or metabolic disorders. A recent study published by Winkler et al (2017), reported improvements in a variety of health variables. Standing was associated with improvements in triglycerides, HDL cholesterol, and fasting glucose via a 12- month program to promote more standing throughout the workday (Winkler et al., 2017). HDL cholesterol improvement was documented to increase as much as 4.68 mg/dL (Alkhajah et al., 2012). An increase of this

magnitude can improve HDL cholesterol from a range indicative of increased risk of heart disease (18.74 mg/dL) to an acceptable level (21.08 mg/dL) (Stone et al., 2014). There is no current evidence on standing desk ability to improve resting blood pressure, only one study reports blood pressure is increased during standing rather than sitting (Cox et al., 2011). However, there is differing literature to suggest standing is not enough to provide positive health outcomes like improvements in body composition and blood pressure (Carr, Swift, Ferrer, & Benzo, 2016). Furthermore, moderate or vigorous physical activity will not result from working at a standing desk for set amount of time. Perhaps standing throughout in the workday is enough to achieve light physical activity.

1.6.5 Standing Desks and Energy Expenditure

A recent review reported that the use of standing desks produced increases in energy expenditure ranging from 4.1 to 20.4 kcal/hr or VO_2 of 0.18 to 0.90 mL/kg/min (MacEwen, MacDonald, & Burr, 2015). Yet, an important question remains. Is the increase in energy expenditure able to produce light physical activity compared to physical activity associated with seated desks? Given 1 MET is 3.5 mL/kg/min, an average 75 kg individual would produce at most an additional ¼ of a MET by standing. However, standing at the work place puts workers at higher likelihood to walk more at work, which could, indirectly, increase energy expenditure to as much as 119 kcal/hr or 1.5 METs (Levine & Miller, 2007).

1.7 Standing Desks and Arterial Stiffness

Current research into energy expenditure of workstations employed indirect calorimetry analyses to determine specific caloric use and substrate metabolism. In one study, utilization of a standing desk increased energy expenditure by 7.5 kcal/hour when compared to a seated desk control (Roemmich, 2016). In addition, increased light physical activity in older individuals is correlated with improved carotid-femoral PWV (Yuko Gando et al., 2010), but could a standing desk be enough to promote physical and cardiovascular well-being if light physical activity is achieved?

1.7.1 Acute Effect of Standing Desk on Arterial Stiffness

Very few studies exist which examine the effect of standing desks on arterial stiffness. One study observed the effects of using a standing desk during one stimulated workday, but alternated between sitting and standing throughout the day. The study revealed acute changes in carotid-ankle PWV compared to the seated control group. However, no changes were seen in carotid-femoral PWV (Bethany Barone Gibbs et al., 2017). No current research has examined the chronic effects of standing desk use during a normal work day.

Thus, the purpose of this study is to determine the chronic effect of standing desk use on arterial stiffness vs. seated sedentary controls. We hypothesized that individuals who chronically stand at work would demonstrate lower arterial stiffness than those that chronically sit at work.

2 Methods

2.1 Participant Information

Fifty-five participants were recruited from the Michigan Tech and Houghton, Michigan community. Of the 55 participants, 50 (42 females, 8 males) were enrolled in the study. Twenty-six participants were chronic users of a seated desk (21 females, 5 males: age 42 ± 11 years, BMI 25 ± 4 kg/m^2) and 24 chronic standing desk users (21 females, 3 males: age 45 ± 12 years, BMI 25 ± 5 kg/m^2) via self-report questionnaire. Standing desk users must have used a standing desk for at least 8 weeks (desk use 2 ± 1 years, 19 ± 16 months). All participants were free of any diagnosed cardiovascular or metabolic diseases. All women were screened for particular phase of menstrual cycle in an effort to have equal groups of early follicular, mid-luteal, and post-menopausal women within the seated and stranding desk groups. This study was approved by the Michigan Technological University Institutional Review Board (M1457). All participants provided written informed consent prior to study enrollment.

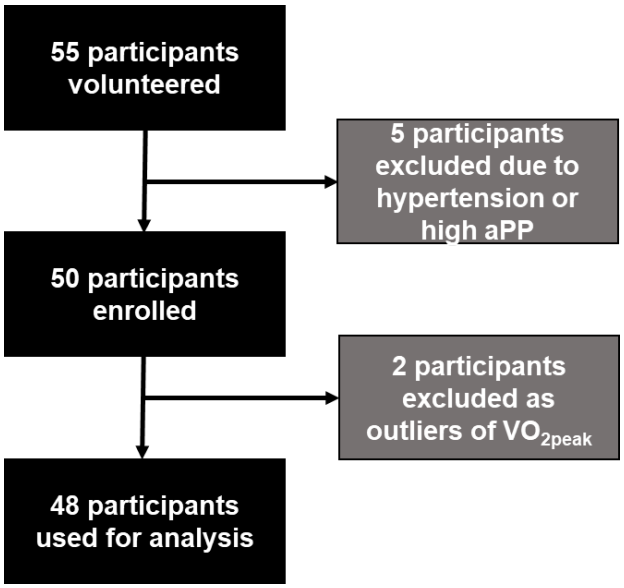

Figure 2.1. Study schematic of enrollment. Hypertension: ≥140/90 mmHg, Aortic Pulse Pressure (aPP): >50mmHg

2.2 Procedures

Participants arrived to Michigan Tech's Clinical and Applied Human Physiology Lab following a fast for at least three hours and abstaining from exercise and caffeine for at least 12 hours prior to the scheduled orientation and testing sessions. During the orientation session, participants completed an informed consent form, participant information sheet, and a Godin Leisure Time Questionnaire (i.e. quantify physical activity habits outside of normal workday). Height, body mass, and body fat percentage were recorded. Participants were instructed to lay supine and quiet on an exam table for five minutes. Up to three brachial blood pressure recordings were taken with an automated sphygmomanometer to screen for potential hypertension (i.e. ≥140/≥90 mmHg). Preliminary arterial stiffness measures were taken via a pulse wave analysis recording of the radial artery following calibration of the SphygmoCor system with the brachial blood pressure. Participants with an aortic pulse pressure of ≥50 mmHg were excluded from the study due to a potential increased risk of atherosclerotic plaque (Oliver & Webb, 2003; Roman et al., 2009). Aerobic fitness was estimated via a Rockport Walk Test on a treadmill within the lab.

Following the orientation session, participants reported back to the laboratory for their scheduled testing session. Each were instructed to lay supine and refrain from talking for five minutes on an exam table. Using an automated sphygmomanometer, three brachial blood pressures were obtained, with each measurement separated by one minute. Duplicate pulse wave analysis recordings

were collected from the radial pulse site for 10 cardiac cycles, with operator indices above 80. An operator index above 80 is considered satisfactory by the manufacturer and indicates maintenance of pulse height and limited variation of the systolic and diastolic portions of the waveform. Three electrodes were placed on the participant's chest. Two at the shoulders near the collar bone and one on the bottom portion of the rib cage on the left side of the body to capture lead II of an ECG. Three regional pulse wave velocity measures (carotid-radial, femoral-dorsalis pedis, carotid-femoral) were performed to assess arterial stiffness within the arm, leg, and central region of the body. Measures of distance (mm) were taken from the suprasternal notch (aorta location) to the pulse site of interest (carotid, radial, femoral, or dorsalis-pedis). Recordings were executed in duplicate, where recordings having a standard deviation of <10% were kept for analyses.

2.3 Measurements

2.3.1 Body Fat Percentage

Body Fat percentage was measured with a bioelectrical impedance scale (BC-418 Segmental Body Composition Analyzer; Tanita, Tokyo, Japan). Participants were instructed to remove shoes and socks and place their feet on the metal pads on the scale. Medal handles were also held at the participant's side during the recording. Each were instructed to remain still as the electromagnetic wave passed through their body. Impedance is indicative of adipose tissue given the hydrophobicity of adipose cells and the lipids they possess. Other tissues like

muscle are water rich and result in low impedance. Whole body and regional assessments of fat percentage were obtained.

2.3.2 Rockport Walk Test

To estimate aerobic fitness, a Rockport walk test was administered to estimate a VO_{2peak} score, given ease of test administration and reliability with actual VO_{2peak} via graded exercise test (Kline et al., 1987). Participants completed a physical activity readiness questionnaire (PAR-Q; Canadian Society of Exercise Physiology) prior to the test and then were instructed to perform a three minute submaximal walking warm-up at a 1% grade on the laboratory treadmill, which produces similar results to track administration (Nieman, 1999). Directly following, participants were instructed to walk one mile as fast as possible, without running, at a 1% grade. A ten second heart rate, test time, and modified Borg rating of perceived exertion were collected at conclusion of the test. Weight (lbs), age (years), sex (1=men, 0=women), time (minutes), and minute heart rate were entered into the following regression equation: VO_{2peak} = 132.853 – (0.00769 * weight) – (0.3877 * age) + (6.315 * sex) – (3.2649 * test time) – (0.1565 * heart rate) (American College of Sports Medicine, 2013). Participants were instructed to cool down ad libitum for at least two minutes.

2.3.3 Blood Pressure

Following five minutes of supine rest, a brachial blood pressure recording was taken with an automated sphygmomanometer (Omron HEM-907XL; Omron

Health Care, Kyoto, Japan). Blood pressure recordings was performed in triplicate during the testing session. The average blood pressure was used to calibrate arterial stiffness data collection software.

2.3.4 Pulse Wave Analysis

An average brachial blood pressure was entered in the SphygmoCor data collection software (SphygmoCor; AtCor Medical, Sydney, Australia) for calibration purposes. The tonometer probe was placed over the radial artery by flattening out the artery and pressing it against the carpals of the wrist. Following probe adjustment to find a strong reading and eliminate systolic and diastolic variation, the probe was kept still for approximately 10-12 seconds to capture ten radial pulse waves. This technique was done in duplicate to ensure consistent quality recordings. The software analyzed the radial pulse wave via a generalized transfer function to generate an aortic pulse wave, aortic blood pressure values, and aortic augmentation indices.

2.3.5 Pulse Wave Velocity

Three electrodes, two at the shoulder region and one on the bottom, left side of the rib cage, were placed to obtain lead II of an ECG. Measurements of straight-line distance (mm) were recorded from the suprasternal notch to two pulse sites of interest to examine carotid-radial, femoral-dorsalis pedis, and carotid-femoral pulse wave velocities. Each pulse wave reading was gated to the R-wave of the ECG to calculate the time delay between pulse creation at the heart and to arrival

at the pulse site. The change in the distance (proximal – distal in reference to aorta) is divided by the change in time delay (proximal – distal time delay) to provide a speed in meters per second. Duplicate readings with a standard deviation ≤10% were kept for data analysis.

2.4 Data and Statistical Analyses

Data were exported from the SphygmoCor system to a Microsoft excel file and then to SPSS. Initially, normality tests were conducted on the variables of age, VO_{2peak}, and fat percentage. Two participant's data were excluded from analysis due to non-normally distributed VO_{2peak} scores. Forty-eight participants, twenty-four chronic seated desk users (19 females, 5 males: age 41 ± 10 years, BMI 25 ± 4 kg/m^2) and 24 chronic standing desk users (21 females, 3 males: age 45 ± 12 years, BMI 25 ± 5 kg/m^2) were used for final analysis. Differences in age, estimated VO_{2peak}, BMI, fat percentage, blood pressure (SAP and DAP), and heart rate between seated and standing groups were assessed using independent samples t-tests. We used a median analysis to classify participants by age, aerobic fitness (VO_{2peak}), and fat percentage (i.e. young v. old, high fitness v. low fitness, etc.) for additional secondary analysis of cfPWV. Each pulse wave velocity was averaged with corresponding value in preparation for statistical analysis using commercial software (SPSS 25.0, SPSS, Chicago, IL). Results are expressed as mean ± SD (Streiner, 1996). Means were considered significantly different when $P \leq 0.05$ (i.e. two-tailed test).

2.4.1 Power Analysis

An additional analysis was performed to ensure adequately powered sample size to produce 1 m/s differences in cfPWV, where 1 m/s reductions of cfPWV may reportedly reduce CVD risk by 15% (Vlachopoulos et al., 2010). Power analysis software (G*Power 3.1.9.2, Kiel, Germany) was used to determine proper effect size for total sample power of 0.8, alpha of 0.05, and equal allocation ratio. Effect size was determined via group means and group standard deviations. A difference of 1 was selected between group means based on the findings of Vlachopoulos et al. (2010). A standard deviation of 1.2 was selected from *The Reference Values for Arterial Stiffness Collaboration* via cfPWV reference value of 40-49 year old individuals with normal blood pressure (n = 562) (Collaboration, 2010). A computed effect size of 0.8333 was achieved with a sample size of n = 48.

3 Results

3.1 Participant Characteristics

Participant demographic values between seated and standing groups are shown in Table 3.1. Age, VO_{2peak}, Godin score, height, weight, body mass index (BMI), fat percentage, systolic arterial pressure (SAP), and heart rate (HR) were all similar between seated and standing groups. However, diastolic arterial pressure (DAP) was significantly higher in the standing group.

Table 3.1. Participant Characteristics: Seated vs. Standing

Variable	Seated (*n* = 24, 19 female)	Standing (*n* = 24, 21 female)	*P* Value
Age (years)	41 ± 10	45 ± 12	0.238
$VO2_{peak}$ (mL/kg/min)	39 ± 8	34 ± 10	0.124
Godin (score)	61 ± 63	47 ± 22	0.311
Height (cm)	167 ± 8	167 ± 8	0.961
Weight (kg)	70 ± 12	71 ± 13	0.704
BMI (kg/m^2)	25 ± 4	25 ± 5	0.860
Fat Percentage	28 ± 8	30 ± 8	0.356
SAP (mmHg)	113 ± 8	115 ± 11	0.529
DAP (mmHg)	66 ± 5	71 ± 7*	0.008
HR (beats/min)	59 ± 11	63 ± 9	0.208

Values are means ± SD; *n*, number of participants; Godin, Physical Activity Questionnaire activity score; BMI, Body Mass Index; SAP, systolic arterial pressure; DAP, diastolic arterial pressure; HR, heart rate. *Significantly different from corresponding seated value, $P < 0.05$

3.2 Carotid-Femoral Pulse Wave Velocity

Figure 3.1 compares carotid-femoral pulse wave velocity (cfPWV) or arterial stiffness in the central region of the body when categorized by seated and standing. No differences were detected between seated and standing groups (p = 0.474). Figure 3.2 shows participants separated into categories of young v. old, low fitness v. high fitness, and high fat v. low fat, where a median analysis generated two groups for comparison in each respective category (i.e. age, fitness, and fat percentage). Carotid-femoral pulse wave velocity was significantly higher in older participants (Panel A) when compared to younger (p=0.002). High fitness and low fat percentage (Panel B and C, respectively) had significantly attenuated cfPWV when compared to low fitness and high fat percentage (p<0.001 and p=0.022, respectively).

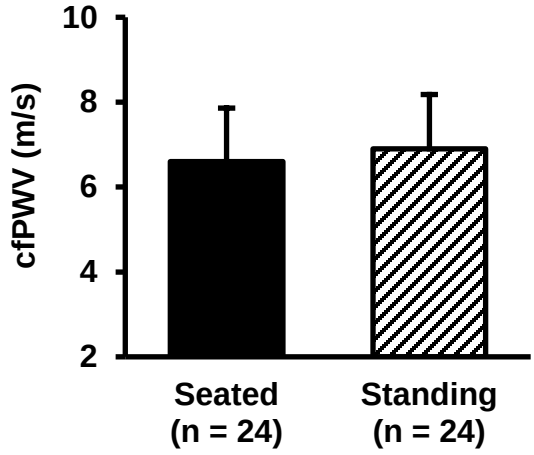

Figure 3.1 Carotid-femoral Pulse Wave Velocity (cfPWV) when classified by seated v. standing (p = 0.474). Result is mean ± SD

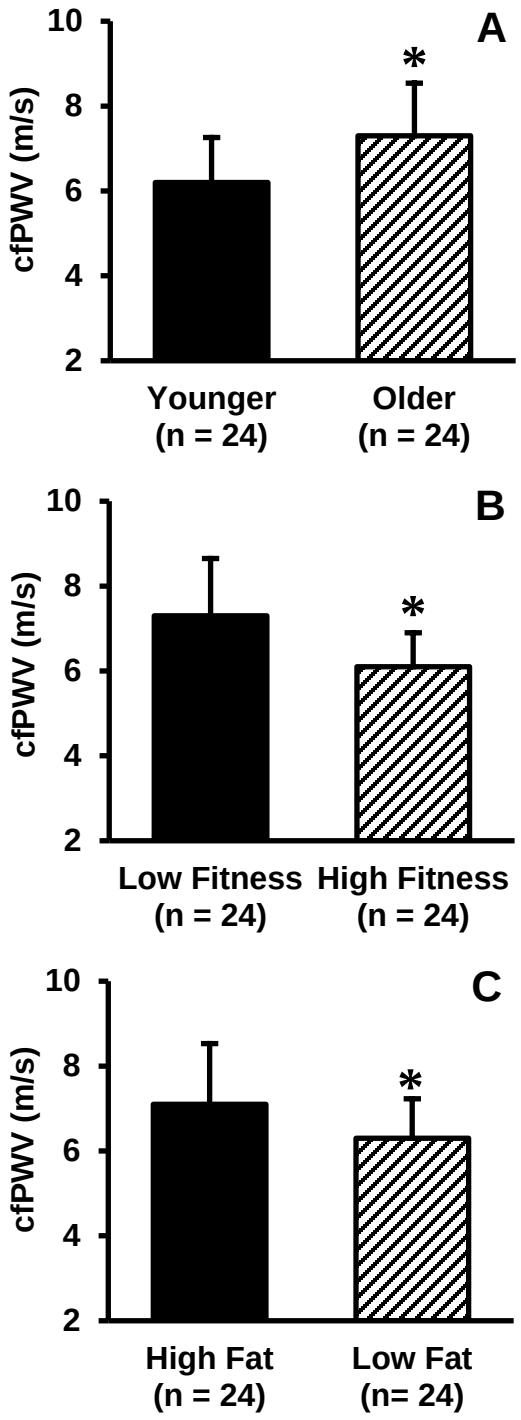

Figure 3.2 Carotid-femoral Pulse Wave Velocity (cfPWV) classified using traditional factors such as age (Panel A; median = 42.0 years, p = 0.002), fitness (Panel B; median = 36.0 mL/kg/min, p < 0.001), and fat (Panel C; median = 28.7%, p = 0.022). Results are means ± SD. *Significantly different from corresponding value, *P* < 0.05

3.3 Peripheral Pulse Wave Velocity

Figure 3.3 represents carotid-radial pulse wave velocity (panel A; crPWV) and femoral-dorsalis-pedis pulse wave velocity (panel B; lPWV). Both recordings represent arterial stiffness in the arm and leg respectively. Both analyses saw no significant difference between seated and standing groups (p = 0.133 and 0.661, respectively).

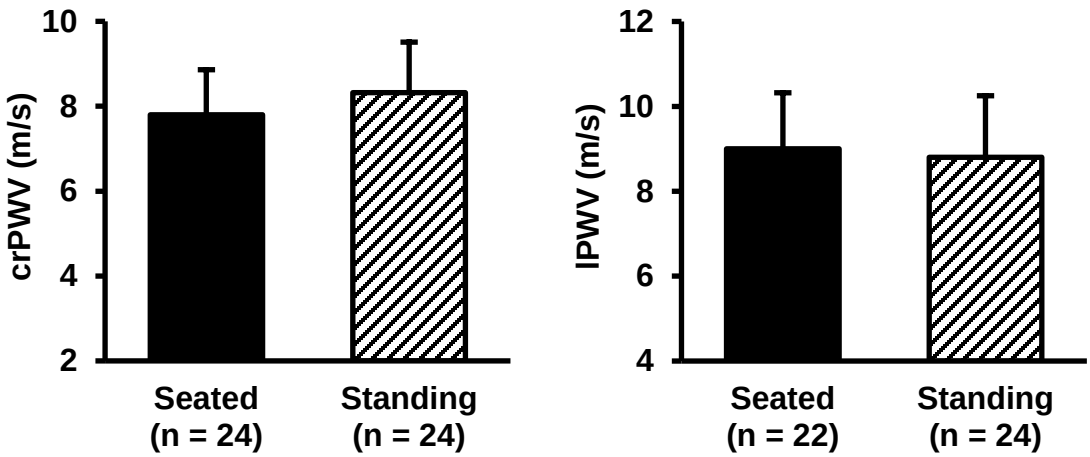

Figure 3.3 Carotid-radial pulse wave velocity (crPWV) and Leg Pulse Wave Velocity (lPWV) when classified by seated v. standing (*P* = 0.133 and 0.661) Results are means ± SD

4 Discussion

To our knowledge, this is the first study to examine chronic use of a standing desk throughout a normal workday. cfPWV, crPWV, and lPWV were not different between chronic seated and standing desk workers. Secondary analysis of traditional factors of age, aerobic fitness, and fat did produce significant differences in cfPWV between groups (i.e. younger v. older, low v. high fitness, high v. low fat) The results of this study were not in support of our hypothesis, where cfPWV of individuals who used a standing desk for at least 50% of the day was not lower as compared to seated desk controls. However, our results do confirm the influence of age, aerobic fitness, and body fat on arterial stiffness. The findings of this study further advance the field of both inactivity physiology and alternative workstations.

4.1 Carotid-Femoral Pulse Wave Velocity

As stated previously, cfPWV is considered to be the gold-standard of assessing cardiovascular health where a 1 m/s decrease is associated with a 15% reduction in CVD risk (Vlachopoulos et al., 2010). The comparison between seated and standing groups revealed little difference between cfPWV, possibly due to lack of achieving light physical activity while working at a standing desk. Light physical activity may trigger NO release (Niebauer & Cooke, 1996) and some degree of endothelin-1 suppression (Nyberg, Mortensen, & Hellsten, 2013). The relationship between light physical activity and cfPWV has been demonstrated in older individuals (i.e. 65-85 years old), but not young (van de Laar et al., 2010). There is some evidence to suggest only vigorous activity is correlated with improved

arterial stiffness in young adults, rather than habitual moderate and light physical activity (van de Laar et al., 2010). Working to reduce other CVD factors such as hypertension and dyslipidemia can also work to prevent age related increase in arterial stiffness (Benetos et al., 2002).

Arterial stiffness is known to increase with age (Benetos et al., 2002; Vaitkevicius et al., 1993; Wen et al., 2015), which is supported by our results. Over time, arteries undergo a process called remodeling where vessel wall elasticity decreases and the diameter of the vessel lumen can increase (Mulvany et al., 1996). Increases in arterial stiffness lead to increased SAP, while increases in vessel lumen diameter via outward remodeling decreases DAP (Mulvany et al., 1996; Schiffrin, 2012). The opposing changes in SAP and DAP results in increased pulse pressure (Schiffrin, 2012). Increased pulse pressure, especially aortic, is indicative of CVD like atherosclerosis and other cerebrovascular disease like an ischemic stroke due to interrupted blood flow (Townsend et al., 2015).

Many studies have noted the association between increased physical activity and decreased arterial stiffness (Vaitkevicius et al., 1993; Zieman, Melenovsky, & Kass, 2005). We believe the decreased cfPWV in those with higher aerobic fitness in the present study to be a robust finding, as fitness was based on the Rockport Walk test results independent of exercise and physical activity habits. Another possible explanation is individuals who engage in regular physical activity expose vasculature to higher levels of shear stress triggering the expression and production of eNOS and NO, respectively. This increased vessel diameter and

bioavailability of NO is associated with reduced cfPWV (Gilligan et al., 1994; Wilkinson et al., 2002).

Promotion of healthy body composition is another factor associated with arterial stiffness. Those with higher overall body fat in the present study demonstrated significantly higher cfPWV. Increased fat percentage is reportedly associated with decreased distensibility of both the aorta and femoral arteries (Ferreira et al., 2004). Additionally, increased percentage of fat/adipose tissue produces molecules like adipokines and leptin. Specifically, leptin's effect on arterial stiffness seems dose dependent with percentage body fat including both visceral and subcutaneous fat. However, adipokine levels vary independent of body fat, but are still associated with increased arterial stiffness (Windham et al., 2010). Visceral or perivascular fat's ability to release adipokines into circulation can promote vascular endothelial dysfunction, leading to arterial stiffness (Villacorta & Chang, 2015). While body composition and fat percentage are related to arterial stiffness, decreased fat percentage may not always result in improved arterial stiffness. Both amount and distribution of the adipose tissue within the body may be the larger player in arterial stiffness regulation.

4.2 Carotid-Radial and Leg Pulse Wave Velocity

Previous work on the acute effects of standing desk use show significant reductions in carotid-radial and carotid-ankle pulse wave velocities (Bethany Barone Gibbs et al., 2017). Inherently, the nature of pulse wave velocity recordings

in the periphery (i.e. arm and leg) provide an indication of the stiffness of some of the muscular arteries. Muscular arteries aid in filtering excess pulsatility throughout the cardiovascular system and various end-organs (Zarrinkoob et al., 2016). Given the similarity of both groups in the crPWV and lPWV reading, pulsatility should also be similar between groups. However, perhaps sit to stand transition, and walking more throughout the day may only impact the arterial stiffness of the periphery rather than the central region of the body. Sitting for more than three hours is shown to impair superficial femoral artery flow mediated dilation (Thosar, Bielko, Mather, Johnston, & Wallace, 2015). Chronic impairment of arterial flow can lead to decreased NO production via decreased shear stress leading to increased arterial stiffness (Wilkinson et al., 2002). Future work warrants investigation whether lPWV increases during prolonged sitting and the potential relationship to cfPWV or crPWV.

4.3 Limitations

One limitation in the present study in the lack of control of the menstrual cycle. Menstrual cycle status is reported to influence arterial stiffness. Pulse wave velocity measures are at the lowest during the mid-luteal phase (Madhura & Sandhya, 2014) following ovulation, and higher during early follicular (Ounis-Skali, Mitchell, Solomon, Solomon, & Seely, 2006). Additionally, the onset of menopause appears to accelerate the age related increases of arterial stiffness when compared to age-matched women who still possess their menstrual cycle (Moreau & Hildreth, 2014). We did record menstrual cycle status and this potential limitation

should be minimized by the similar distribution of women in each phase/status (i.e. early follicular, mid-luteal, perimenopause, and postmenopause). Distributions were 41%, 23%, 6%, and 29% in seated participants and 25%, 30%, 5%, and 40%, respectively in standing participants.

4.4 Implications

The present study did not detect any differences in PWV between seated and standing desk participants. The current study suggests standing for at least 50% of a normal workday may not be enough to produce an effect on arterial stiffness. If light physical activity is achieved while using a standing desk, this may not result in decreased arterial stiffness in younger populations. There is a suggestion that only moderate and/or vigorous physical activity can improve arterial stiffness in younger populations (van de Laar et al., 2010). However, older individuals can reduce their arterial stiffness with increased light physical activity (Y. Gando et al., 2010). In contrast to employee health, standing is reported to increase productivity (Garrett et al., 2016; Hasegawa et al., 2001). By this measure, perhaps the purchase of a standing desk can still be justified, independent of arterial stiffness improvement. On the contrary, further research into the cardiovascular health effects of standing desk use is warranted.

4.5 Future Directions

Plenty of opportunities exist in the realm of arterial stiffness and standing desk, or alternative work stations. Cross-sectional designs have inherent draw back by

comparing data across individuals rather than to the same person. Future work should initiate a standing desk intervention to examine if/when arterial stiffness is affected from standing for most of the day after controlling for physical activity minutes outside of work. Additionally, longitudinal studies should be built from initial cross-sectional designs to determine if chronic standing desk can significantly attenuate arterial stiffness during the aging process compared to the seated desk controls.

4.6 Summary

Workplace standing desks and active workstations, in general, are in popular demand in the office, where the effects on human health are largely unknown. The present study comparing chronic standing desk users and chronic standing desk users did not find any differences in cfPWV, crPWV, or lPWV. However, secondary analysis of traditional factors of age, aerobic fitness, and fat revealed significant differences between young and old, low and high aerobic fitness, and high and low fat percentage. This finding further supports arterial stiffness increases with age, and promotion of exercise and healthy body composition can work to ameliorate age related increases in arterial stiffness. Standing for at least 50% of the workday does not appear to directly influence arterial stiffness based on our initial cross-sectional analysis.

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A Appendix A – Raw Data

A.1 Participant Characteristics

Table A.1. *Subject Characteristics Raw Data*

Part. #	Age	Sex	VO_{2peak}	Godin	Height	Weight	BMI	Fat %	SAP	DAP	HR
1	25	F	45.8	52	156	83.0	34.0	36.1	108	72	64
2	54	F	35.6	96	163	64.4	24.4	24.6	126	77	67
3	48	F	28.4	37	173	68.5	23.0	24.3	119	77	64
4	38	F	39.5	21	160	56.3	21.7	30.2	112	69	57
5	34	M	50.9	44	168	73.9	26.2	39.6	122	81	62
6	44	M	41.9	51	167	81.7	29.0	32.7	120	73	60
7	47	F	24.5	62	172	72.6	24.1	33.6	134	81	59
8	56	F	26.0	203	163	57.1	21.5	25.6	107	54	61
9	50	F	37.8	26	161	58.5	22.1	33.1	107	61	54
10	32	F	42.1	25	160	74.9	29.3	37.1	96	64	77
11	56	F	34.9	39	180	71.4	22.0	34.7	106	62	50
12	22	F	47.6	18	165	96.2	35.3	44.2	105	69	66
13	25	F	57.1	21	164	56.8	20.8	18.7	107	66	51
14	41	M	35.0	54	185	103	29.8	24.0	114	72	49
15	46	F	33.8	91	180	80.5	24.7	14.8	122	70	40
16	45	M	48.2	55	167	60.8	21.6	20.5	123	67	60
17	42	F	36.3	125	167	82.4	29.3	20.7	105	71	49
18	29	F	41.9	15	158	82.4	33.2	40.5	110	61	54
19	37	M	51.8	26	169	74.9	26.1	34.8	113	74	49
20	35	F	31.4	290	158	76.1	30.7	41.6	116	64	53
21	35	F	30.9	81	179	65.7	20.5	12.3	129	78	59
22	57	F	11.2	29	171	90.4	31.2	46.2	129	78	67
23	57	F	31.0	49	167	78.9	28.1	39.6	115	75	67
24	53	F	33.4	41	162	62.3	23.6	25.0	133	79	51
25	34	F	38.5	49	162	65.5	24.8	24.8	109	68	51
26	38	F	30.8	50	170	67.1	23.2	30.2	109	69	72
27	50	F	32.7	52	162	59.6	22.6	25.4	125	68	60
28	47	F	39.6	77	175	72.4	23.6	31.2	105	60	67
29	55	F	26.3	54	154	60.3	25.5	24.8	134	65	69
30	47	F	30.6	83	162	68.5	26.3	36.6	123	73	71
31	51	F	29.2	36	175	64.3	20.9	24.6	115	65	49

32	63	F	27.2	30	178	101	32.1	29.5	112	67	64
33	36	F	36.3	21	160	51.2	20.0	19.7	115	63	75
34	35	F	19.8	39	185	83.6	24.6	21.0	111	64	41
35	39	M	51.3	12	170	65.5	22.6	29.2	102	60	58
36	63	F	40.0	42	155	47.2	19.6	24.9	103	66	59
37	56	F	16.2	40	168	63	22.4	31.1	110	62	59
38	31	F	35.1	55	184	79.1	23.3	15.0	117	61	53
39	27	M	55.7	28	157	71.7	28.9	31.8	110	68	64
40	61	F	32.1	45	166	84.6	30.6	44.4	109	69	54
41	42	F	37.7	43	175	76.5	33.7	33.7	108	66	49
42	62	F	28.7	25	160	70.1	21.8	28.2	120	74	66
43	32	F	41.9	17	167	60.4	22.6	26.9	115	65	82
44	52	F	11.3	72	160	57.8	27.4	39.9	135	86	72
45	50	F	23.5	45	158	80.8	32.1	42.5	127	88	69
46	61	F	14.9	56	161	69.5	26.7	37.8	102	64	64
47	27	F	42.7	47	173	55.1	18.5	18.5	115	72	93
48	42	F	38.2	15	162	58.5	22.1	25.9	93	60	68
49	28	M	45.1	26	173	79.3	26.6	23.9	123	74	75
50	37	F	46.3	54	169	62.2	21.8	24.8	103	69	56

Part. = Participant; Age = Years; VO2peak = mL/kg/min; Godin = Physical Activity Questionnaire; Height = cm; Weight = kg; BMI = Body Mass Index (kg/m2); SAP= Systolic Arterial Pressure (mmHg); DAP = Diastolic Arterial Pressure (mmHg); HR = Heart Rate (beats/min)

A.2 Rockport Walk Test

Table A.2. *Rockport Walk Test Raw Data*

Participant	Weight	Age	Sex Code	Time	Heart Rate	VO2peak
1	183	38	0	15.23	150	30.84
2	142	50	0	15.35	126	32.71
3	151	47	0	14.25	108	39.59
4	124	55	0	18.00	108	26.32
5	163	47	0	14.42	156	30.61
6	180	51	0	14.55	144	29.21
7	159	63	0	15.10	126	27.20
8	126	25	0	13.53	150	45.82
9	129	54	0	14.32	126	35.57
10	165	48	0	15.78	138	28.41
11	157	36	0	15.27	132	36.29
12	212	35	0	18.30	150	19.76
13	125	38	0	12.80	174	39.47
14	226	39	1	11.80	108	51.26
15	177	34	1	12.78	126	50.90
16	134	63	0	12.05	120	40.00
17	182	44	1	13.68	138	41.88
18	182	56	0	18.48	132	16.18
19	165	31	0	15.18	150	35.10
20	168	47	0	17.03	138	24.53
21	145	27	1	11.47	156	55.70
22	199	57	0	27.4	132	11.2
23	174	56	0	15.36	138	26.02
24	137	61	0	15.20	108	32.11
25	144	50	0	12.59	150	37.78
26	148	32	0	14.17	132	42.14
27	131	56	0	14.23	126	34.86
28	160	42	0	13.49	144	37.72
29	133	22	0	13.18	150	47.59
30	151	62	0	14.38	138	28.66
31	142	25	0	11.72	108	57.10
32	223	41	1	14.87	144	35.02

33	113	46	0	15.60	138	33.82
34	184	45	1	11.55	138	48.25
35	144	42	0	14.57	138	36.31
36	104	32	0	14.42	150	41.91
37	139	29	0	15.10	126	41.92
38	174	37	1	13.38	102	51.75
39	158	35	0	17.17	126	31.37
40	187	35	0	15.18	156	30.95
41	169	57	0	13.25	150	31.04
42	133	53	0	14.98	126	33.42
43	127	34	0	14.38	156	38.50
44	178	50	0	17.60	120	23.52
45	155	52	0	22.5	102	11.3
46	153	61	0	19.25	126	14.85
47	121	27	0	13.22	174	42.67
48	129	42	0	16.07	102	38.23
49	175	28	1	13.62	162	45.06
50	137	37	0	12.57	132	46.27

Weight = lbs; Age = years; Sex Code, Female = 0, Male = 1; Time = Minutes; Heart Rate = beats/min; VO_{2peak} = mL/kg/min

A.3 Pulse Wave Velocity Raw Data

Table A.3. *Pulse Wave Velocity Raw Data*

Participant	cfPWV	crPWV	lPWV
1	8.05	9.1	6.0
2	7.25	9.3	7.8
3	6.3	7.7	8.2
4	7	7.4	7.2
5	6.65	10.4	9.8
6	7	7.1	8.6
7	9.9	8.3	10.4
8	5.55	6.3	7.7
9	6.8	7.0	11.6
10	8.7	7.9	---
11	5.3	6.9	8.2
12	5.75	7.2	10.0
13	5.85	7.5	9.5
14	7	10.5	11.3
15	5.6	7.7	9.4
16	7.15	6.7	10.4
17	5.8	9.2	7.3
18	8.6	8.6	6.3
19	6.2	7.5	9.0
20	7.65	7.8	11.4
21	5.25	7.7	8.2
22	9.9	8.8	9.1
23	6.55	8.4	10.3
24	6.55	8.0	9.4
25	6.7	6.6	8.7
26	8.4	9.5	9.9
27	6.1	7.2	8.4
28	4.95	6.9	10.0
29	9.3	8.5	11.3
30	5.95	7.8	10.0
31	8.8	9.8	9.0
32	6.05	8.1	8.5

33	6	9.0	5.6
34	5.75	7.1	8.2
35	4.7	7.1	9.6
36	6.15	6.6	9.8
37	6.95	7.0	9.2
38	5.05	7.1	---
39	5.5	10.1	8.2
40	6.25	6.8	9.1
41	6.4	8.0	9.4
42	6.6	7.7	7.7
43	7.1	9.3	7.5
44	6.8	7.4	9.3
45	5.7	8.6	7.1
46	6.15	8.6	8.6
47	8.5	10.8	8.5
48	5.8	9.5	10.1

cfPWV = Carotid-Femoral PWV; crPWV = Carotid-Radial PWV; lPWV = Leg PWV; --- = Missing Data Point

B Appendix B - Statistical Analyses

Table B.1a - Descriptive Statistics for Age

	Condition	N	Mean	Std. Deviation	Std. Error Mean
age	1.00	24	41.0833	10.45037	2.13317
	2.00	24	45.0000	12.18338	2.48692

Table B.1b - Independent Samples T-Test for Age

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
age	Equal variances assumed	1.172	.285	-1.195	46	.238	-3.91667	3.27646	-10.51184	2.67851
	Equal variances not assumed			-1.195	44.958	.238	-3.91667	3.27646	-10.51597	2.68264

Table B.2a – Descriptive Statistics for VO_{2peak}

	Condition	N	Mean	Std. Deviation	Std. Error Mean
VO2max	1.00	24	38.5149	8.46315	1.72753
	2.00	24	34.2419	10.34880	2.11244

Table B.2b – Independent Samples T-Test for VO_{2peak}

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
VO2max	Equal variances assumed	.838	.365	1.566	46	.124	4.27301	2.72888	-1.21993	9.76596
	Equal variances not assumed			1.566	44.257	.125	4.27301	2.72888	-1.22578	9.77181

Table B.3a – Descriptive Statistics for Godin Leisure Time Questionnaire

	Condition	N	Mean	Std. Deviation	Std. Error Mean
Godin	1.00	24	61.3333	63.44728	12.95112
	2.00	24	47.3125	21.84372	4.45883

Table B.3b – Independent Sample T-Test for Godin Leisure Time Questionnaire

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
Godin	Equal variances assumed	4.840	.033	1.024	46	.311	14.02083	13.69718	-13.55016	41.59183
	Equal variances not assumed			1.024	28.377	.315	14.02083	13.69718	-14.01979	42.06146

Table B.4a – Descriptive Statistics for Height

	Condition	N	Mean	Std. Deviation	Std. Error Mean
height	1.00	24	167.1042	8.44931	1.72471
	2.00	24	166.9875	7.95227	1.62325

Table B.4b – Independent Sample T-Test for Height

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
height	Equal variances assumed	.027	.871	.049	46	.961	.11667	2.36845	-4.65078	4.88411
	Equal variances not assumed			.049	45.832	.961	.11667	2.36845	-4.65125	4.88458

Table B.5a – Descriptive Statistics for Weight

	Condition	N	Mean	Std. Deviation	Std. Error Mean
weight	1.00	24	69.8821	11.98086	2.44558
	2.00	24	71.2458	12.73926	2.60039

Table B.5b – Independent Sample T-Test for Weight

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
weight	Equal variances assumed	.041	.840	-.382	46	.704	-1.36375	3.56972	-8.54922	5.82172
	Equal variances not assumed			-.382	45.828	.704	-1.36375	3.56972	-8.54995	5.82245

Table B.6a – Descriptive Statistics for BMI

	Condition	N	Mean	Std. Deviation	Std. Error Mean
BMI	1.00	24	25.2583	3.98943	.81434
	2.00	24	25.4792	4.62892	.94488

Table B.6b - Independent Samples T-Test for BMI

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
BMI	Equal variances assumed	.264	.610	-.177	46	.860	-.22083	1.24737	-2.73166	2.29000
	Equal variances not assumed			-.177	45.019	.860	-.22083	1.24737	-2.73314	2.29147

Table B.7a – Descriptive Statistics for VO_{2peak}

	Condition	N	Mean	Std. Deviation	Std. Error Mean
Overall_Fat	1.00	24	27.9792	7.82715	1.59771
	2.00	24	30.1333	8.16635	1.66695

Table B.7b – Independent Samples T-Test for VO_{2peak}

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
Overall_Fat	Equal variances assumed	.348	.558	-.933	46	.356	-2.15417	2.30898	-6.80191	2.49357
	Equal variances not assumed			-.933	45.917	.356	-2.15417	2.30898	-6.80213	2.49380

Table B.8a – Descriptive Statistics for SAP

	Condition	N	Mean	Std. Deviation	Std. Error Mean
SAP	1.00	24	113.2500	8.43002	1.72077
	2.00	24	115.0000	10.55009	2.15353

Table B.8b – Independent Samples T-Test for SAP

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
SAP	Equal variances assumed	1.964	.168	-.635	46	.529	-1.75000	2.75658	-7.29871	3.79871
	Equal variances not assumed			-.635	43.864	.529	-1.75000	2.75658	-7.30601	3.80601

Table B.9a – Descriptive Statistics for DAP

	Condition	N	Mean	Std. Deviation	Std. Error Mean
DAP	1.00	24	66.3750	5.30637	1.08316
	2.00	24	71.4583	7.30681	1.49150

Table B.9b – Independent Samples T-Test for DAP

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
DAP	Equal variances assumed	2.786	.102	-2.758	46	.008	-5.08333	1.84331	-8.79373	-1.37294
	Equal variances not assumed			-2.758	41.981	.009	-5.08333	1.84331	-8.80334	-1.36333

Table B.10a – Descriptive Statistics for HR

	Condition	N	Mean	Std. Deviation	Std. Error Mean
HR	1.00	24	59.0833	10.99769	2.24489
	2.00	24	62.9583	9.25710	1.88960

Table B.10b – Independent Samples T-Test for HR

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
HR	Equal variances assumed	1.631	.208	-1.321	46	.193	-3.87500	2.93430	-9.78144	2.03144
	Equal variances not assumed			-1.321	44.699	.193	-3.87500	2.93430	-9.78609	2.03609

Table B.11a – Descriptive Statistics for cfPWV (Seated v. Standing)

	Condition	N	Mean	Std. Deviation	Std. Error Mean
aorticPWV	1.00	24	6.5896	1.25516	.25621
	2.00	24	6.8542	1.28299	.26189

Table B.11b – Independent Samples T-Test for cfPWV

		Levene's Test for Equality of Variances		t-test for Equality of Means					
								95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
aorticPWV	Equal variances assumed	.015	.902	-.722	46	.474	-.26458	.36637	-1.00205	.47289
	Equal variances not assumed			-.722	45.978	.474	-.26458	.36637	-1.00206	.47290

Table B.12a – Descriptive Statistics for crPWV (Seated v. Standing)

	Condition	N	Mean	Std. Deviation	Std. Error Mean
armPWV	1.00	24	7.8271	1.06066	.21651
	2.00	24	8.3250	1.19209	.24334

Table B.12b – Independent Samples T-Test for crPWV

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
armPWV	Equal variances assumed	.622	.434	-1.529	46	.133	-.49792	.32571	-1.15354	.15770
	Equal variances not assumed			-1.529	45.386	.133	-.49792	.32571	-1.15378	.15794

Table B.13a – Descriptive Statistics for lPWV (Seated v. Standing)

	Condition	N	Mean	Std. Deviation	Std. Error Mean
legPWV	1.00	22	9.0159	1.31515	.28039
	2.00	24	8.8354	1.44902	.29578

Table B.13b – Independent Samples T-Test for lPWV

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
legPWV	Equal variances assumed	.934	.339	.441	44	.661	.18049	.40931	-.64443	1.00541
	Equal variances not assumed			.443	43.997	.660	.18049	.40756	-.64089	1.00188

Table B.14a – Median Analysis for Age

			Statistic	Std. Error
age	Mean		43.0417	1.64569
	95% Confidence Interval for Mean	Lower Bound	39.7310	
		Upper Bound	46.3524	
	5% Trimmed Mean		43.0139	
	Median		42.0000	
	Variance		129.998	
	Std. Deviation		11.40168	
	Minimum		22.00	
	Maximum		63.00	
	Range		41.00	
	Interquartile Range		18.25	
	Skewness		.085	.343
	Kurtosis		-.994	.674

Table B.15a – Descriptive Statistics for cfPWV (Younger v. Older)

	AGE_GROUP	N	Mean	Std. Deviation	Std. Error Mean
aorticPWV	1.00	24	6.1875	1.06007	.21639
	2.00	24	7.2562	1.24029	.25317

Table B.15b – Independent Samples T-Test for cfPWV

		Levene's Test for Equality of Variances		t-test for Equality of Means					
								95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
aorticPWV	Equal variances assumed	.926	.341	-3.209	46	.002	-1.06875	.33305	-1.73914	-.39836
	Equal variances not assumed			-3.209	44.911	.002	-1.06875	.33305	-1.73958	-.39792

Table B.16a – Median Analysis for VO_{2peak}

			Statistic	Std. Error
VO2max	Mean		36.3784	1.38535
	95% Confidence Interval for Mean	Lower Bound	33.5914	
		Upper Bound	39.1653	
	5% Trimmed Mean		36.4279	
	Median		35.9500	
	Variance		92.122	
	Std. Deviation		9.59801	
	Minimum		14.85	
	Maximum		57.10	
	Range		42.25	
	Interquartile Range		11.40	
	Skewness		.024	.343
	Kurtosis		-.109	.674

Table B.17a – Descriptive Statistics for cfPWV (Low v. High Fitness)

	FIT_GROUP	N	Mean	Std. Deviation	Std. Error Mean
aorticPWV	1.00	24	7.3313	1.35477	.27654
	2.00	24	6.1125	.80478	.16427

Table B.17b – Independent Samples T-Test for cfPWV

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
aorticPWV	Equal variances assumed	8.224	.006	3.789	46	.000	1.21875	.32165	.57130	1.86620
	Equal variances not assumed			3.789	37.435	.001	1.21875	.32165	.56727	1.87023

Table B.18a – Median Analysis for Fat Percentage

			Statistic	Std. Error
Overall_Fat	Mean		29.0563	1.15290
	95% Confidence Interval for Mean	Lower Bound	26.7369	
		Upper Bound	31.3756	
	5% Trimmed Mean		29.0741	
	Median		28.7000	
	Variance		63.800	
	Std. Deviation		7.98751	
	Minimum		12.30	
	Maximum		44.40	
	Range		32.10	
	Interquartile Range		10.40	
	Skewness		.076	.343
	Kurtosis		-.609	.674

Table B.19a – Descriptive Statistics for cfPWV (Low v. High Fat Percentage)

	FAT_GROUP	N	Mean	Std. Deviation	Std. Error Mean
aorticPWV	1.00	24	6.3104	.92748	.18932
	2.00	24	7.1333	1.42986	.29187

Table B.19b – Independent Samples T-Test for cfPWV

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
aorticPWV	Equal variances assumed	5.116	.028	-2.365	46	.022	-.82292	.34789	-1.52319	-.12264
	Equal variances not assumed			-2.365	39.443	.023	-.82292	.34789	-1.52634	-.11949