ASSESSMENT OF ARTERIAL HEALTH IN ADOLESCENTS WITH CP

BASELINE ASSESSMENT OF ARTERIAL STRUCTURE AND FUNCTION IN ADOLESCENTS WITH CEREBRAL PALSY

By

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ABSTRACT

Functional limitations place youth with cerebral palsy (CP) at an increased risk of physical inactivity and cardiovascular disease. The structure and function of the cardiovascular system of these adolescents has not been previously investigated. In the current cross-sectional study, endothelial function was assessed using flow-mediated dilation (FMD) in eleven adolescents with CP (age 13.2 ± 2.1 y) and compared to eleven healthy, age-and gender-matched control participants (12.4 ± 2.3 y). All participants with CP were ambulatory or ambulatory with assistive devices (lower leg brace) and classified as levels I-II according to the Gross Motor Function Classification System (GMFCS). Baseline arterial stiffness was examined through assessment of central and peripheral pulse wave velocity (cPWV, pPWV,) as well as carotid distensibility, a direct measure of central artery stiffness. A combination of B-mode ultrasound imaging and applanation tonometry was used to calculate carotid distensibility. Carotid intima-media thickness (IMT), a measure of vascular structure, was also quantified using B-mode ultrasound images and a semi-automated edge detection software program. cPWV was calculated using the distance (carotid to femoral via the subtraction method) and time delay between ventricular depolarization and the foot of the femoral waveform. pPWV was calculated from the femoral to dorsalis pedis artery using the distance between each site and time delay between the arrival of the foot of each corresponding waveform. Physical activity (PA) levels were assessed using a 7-day recall questionnaire. Anthropometric measurements as well as measures of resting systolic, diastolic and mean arterial blood pressures were similar in both groups. There were no group differences ($p>0.05$) in

absolute, relative or normalized FMD responses. Both groups also had similar values of carotid IMT as well as all measures of arterial stiffness including carotid distensibility, cPWV and pPWV ($p>0.05$). No group differences were found in the amount of time spent in light and moderate intensity PA; however, the control group participated in a significantly greater amount of vigorous intensity PA (CON: 196 ± 174 min. vs. CP: 38 ± 80 min). Pearson correlation coefficients with all participants revealed a significant positive relationship between age and cPWV ($r=0.485$ $p=0.026$) and negative relationship with carotid compliance ($r=-0.436$, $p=0.048$). These findings indicate that the arterial structure and function of youth with CP (GMFCS level I-II), examined in this study are not different from a healthy control group. Future research should include youth with CP of GMFCS levels III-V to gain further insight into the potential consequences of severe mobility impairments and functional limitations on levels of habitual PA and arterial health in this young, clinical population.

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LIST OF ALL ABBREVIATIONS AND SYMBOLS

APHV	Age at peak height velocity
BMI	Body mass index
CP	Cerebral Palsy
CVD	Cardiovascular disease
FMD	Flow-mediated dilation
GMFCS	Gross Motor Function Classification System
GMFCS-E&R	Gross Motor Function Classification System – Expanded and Revised
IMT	Intima-media thickness
PA	Physical activity
PWV	Pulse wave velocity
PHV	Peak height velocity
TPHV	Time to peak height velocity
WC	Waist circumference
WHR	Waist-to-height ratio

CHAPTER 1

Literature Review

1. Human arterial structure and function

1.1 Anatomy and physiology of the arterial tree

Arteries are muscular structures that consist of an outer, middle, and inner layer. These layers work together in the expansion and contraction of the artery to absorb each heartbeat and maintain optimal circulation. From deep to superficial, these layers include the tunicas intima, media and adventitia [1] (Fig. 1). The tunica intima consists of the vascular endothelium, a single layer of cells, as well as a thin layer of elastin and collagen fibers that anchor it to the internal elastic lamina. The internal elastic lamina is the structure that distinguishes the tunicas intima and media. Mechanical properties of the vessel are determined by the formation of the tunica media, the major component of the arterial wall. The media is made up of layers comprised of fibrous structures, running circularly or in a tight helix, with smooth muscle running between these layers. The tunica adventitia is the outermost layer, containing collagen and some elastin, that merge with the surrounding connective tissue, consisting of nerves, small blood vessels and fibroblasts. The adventitia is demarcated by the outer elastic lamina [1] (Fig. 1).

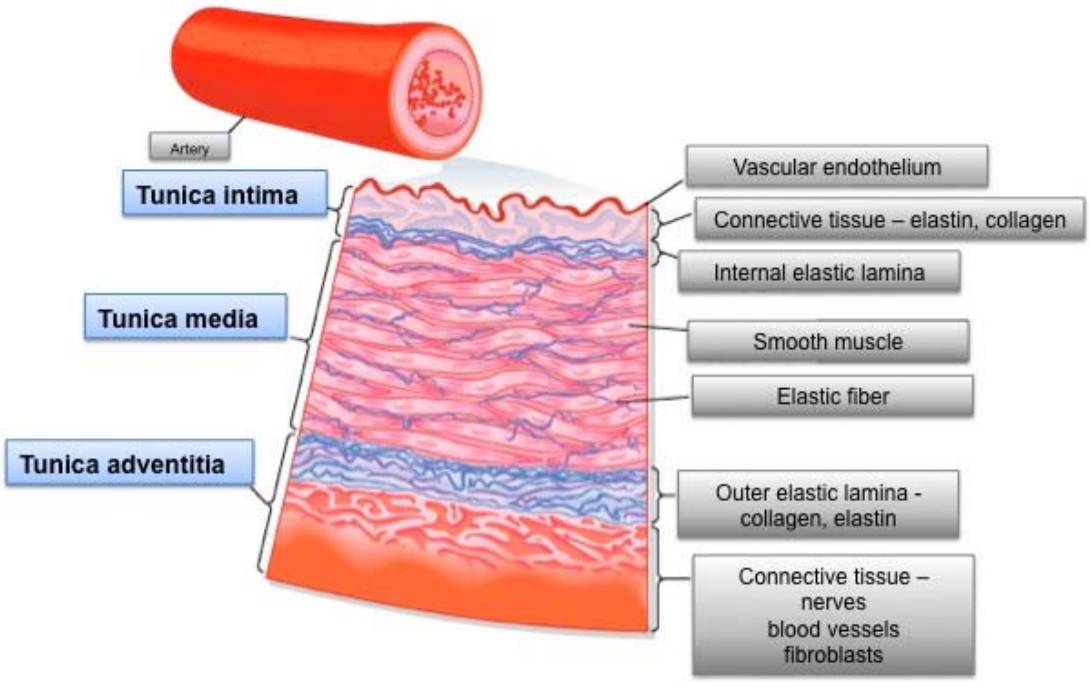

Figure 1. Anatomy of the artery consisting of the three layers, the tunicas intima, media and adventitia and their constituents. (Adapted from Encyclopedia Britannica, 2008, Britannica.com)

1.2 Arterial Stiffness

Arterial stiffness describes the rigidity in the vessel wall and is associated with adverse cardiovascular outcomes in healthy and hypertensive adults [2, 3]. Key factors associated with vessel stiffness include the arterial wall composition, smooth muscle tone, mean arterial pressure (MAP) and age.

1.2.1 Arterial wall composition

Elastin and collagen are the predominant elastic materials of the arterial wall, representing roughly 50% of the dry weight. The majority of the total weight of the artery wall is comprised of water and the remainder of dry weight consists of smooth

muscle and non-fibrous matrix. Collagen contributes to the fibrous connective tissue, lending tensile strength to vessel walls, enabling them to withstand the pressure of blood within them without rupturing [4]. Elastin is a highly stretchable extracellular protein whose fibers contribute to the elastic connective tissue, enabling blood vessels to expand or contract as the pressure of blood within them changes [4]. Central and peripheral arteries differ in their composition as a shift from primarily elastic (as seen in the aorta) to collagen (as seen in the periphery) occurs throughout the arterial tree, reflective of the function of each arterial segment. Although the elastin, smooth muscle, and collagen interlock to form a heterogeneous media, these materials function as a homogenous material. The aortic medial structure, a lamellar unit consisting of all three of the elastic materials, is responsible for the viscoelastic properties of the elastic arteries and contributes to its many static and dynamic mechanical features [1].

1.2.2 Smooth muscle tone

Smooth muscle is major constituent of the arterial wall, responsible for vasomotor tone, a key modulator of vascular resistance. Smooth muscle cells are oriented in a predominantly circumferential orientation, with their contraction and relaxation also acting in this direction, resulting in vasoconstriction and vasodilation, accordingly [4]. Smooth muscle tone is dependent on a variety of factors including local metabolic activity and extrinsic sympathetic input [4].

Metabolic activity

Vascular smooth muscle cells are sensitive to the changes in concentrations of local chemical substances in the extracellular fluid. These changes occur as a result of the

metabolic activity required to sustain steady state in local tissues. When the metabolic rate in a tissue increases such that the rate of O_2 consumption and CO_2 production rise, the metabolic demand must be met by increases in O_2 supply and thus, increases in blood flow. This metabolic shift results in an increase in circulating vasodilator substances (eg; CO_2, lactic acid, adenosine, K+, H+), which cause the smooth muscle to relax, alleviating the ischemic conditions. Evidently, 'active hyperemia' occurs as an increase in metabolic activity causes the vascular resistance in the tissue to drop (vasodilation) and an increase in local blood flow to occur. Similarly, 'reactive hyperemia' occurs as a result of changes in blood flow that stimulate changes in metabolite concentrations [4]. For example, if blood flow to a tissue is occluded or reduced below adequate levels, the rates of O_2 consumption and CO_2 production exceed the rates of delivery and removal; therefore, vasodilation is induced, reducing vascular resistance and increasing blood flow to the tissue in need. Although different causes, both conditions result in increases in vasorelaxation as a result of increases in metabolite concentrations.

Sympathetic control

The sympathetic nervous system innervates smooth muscle, which has both α- and β-adrenergic receptors. At rest, continuous low frequency stimulation of the sympathetic fibers results in partial constriction of the smooth muscle, maintaining vasomotor tone [4]. Vasonconstriction occurs as a result of increased sympathetic nerve activity, causing norepinephrine to bind to α adrenergic receptors, resulting in smooth muscle contraction, increasing total peripheral resistance and mean arterial pressure (MAP). β adrenergic receptors are most commonly found on the arteriolar smooth muscle of skeletal and

cardiac muscle and act as a binding site for epinephrine released from the adrenal medulla. With increases in sympathetic nerve activity, epinephrine can bind to both α and β_2 receptors, resulting in vasoconstriction and vasodilation, respectively. Although contradictory effects, the number of α receptors out number the number of β_2 receptors, promoting the binding of epinephrine to α receptors in high concentrations, resulting in constriction of the smooth muscle. This effect is most profound in cardiac and skeletal muscle in which vasodilation occurs in response to increasing concentrations of epinephrine during a fight-or-flight response [4].

1.2.3 Mean arterial pressure

The pressure applied on the arterial wall plays a key role in arterial stiffness. The association between prolonged hypertension and the rearrangement of arterial wall material has been put forth, with qualitative or quantitative changes in arterial components resulting in mechanical adaptation of the arterial wall [5]. It has been suggested that with increases in intra-arterial pressure, such as those experienced during smooth muscle contraction, the smooth muscle transfers its stress from elastin to collagen, resulting in an increase in stiffness. Similarly, during smooth muscle relaxation, the stress is transferred to the elastic lamellae, which decreases arterial stiffness [1].

1.2.4 Age

Stiffness of the large central elastic arteries (such as the aorta and carotid artery) has been shown to increase with age; however, less substantial increases have been seen on the distal, muscular arteries of the periphery [6]. Central arterial stiffness can increase to the same, or greater level to that of the peripheral arteries both with age, as well as with

cardiovascular disease [7-9]. The exact mechanism associated with age-related increases in arterial stiffness have not been identified; however, changes in the composition of the arterial wall, growth factors and vascular smooth muscle cell hypertrophy are believed to be related [10, 11]. Specifically, thinning, splitting and fraying of the elastic fibers and laminae is often observed as the orderly arrangement of these components is gradually lost over time and total amounts of collagenous (rigid) material increases [12].

1.3 Central and peripheral arterial stiffness

Large, central arteries (such as the ascending aorta, and the common carotid arteries) are composed of higher amounts of elastin to collagen and include limited numbers of smooth muscle cells. The elasticity of these arteries allows for cushioning of the high pressures exerted by the contraction of the heart during systole [1]. The primary function of these large arteries is to serve as buffering reservoirs that passively expand in the face of increasing pressures. The diameter of the arteries decreases towards the periphery of the vascular tree, as these segments become more muscular and less elastic. These subsequent increases in stiffness are reflective of the shift in function from cushioning to conduit, which occurs from the heart towards the periphery [1, 13]. Therefore, in healthy subjects, peripheral arteries are stiffer than central arteries resulting in pressure amplification, a phenomena describing the increase in amplitude of the pressure wave seen in the peripheral versus central arteries [13].

1.3.1 Pathophysiology of arterial stiffness

The elastic properties of the visco-elastic tube of the artery allow travel of a forward pressure wave (anterograde blood flow) while numerous bifurcations and high levels of

resistance also generate reflected waves (retrograde blood flow). In a healthy vessel, the naturally occurring level of resistance results in reflected waves generated in diastole, which can be visualized as a secondary fluctuation in the pressure waveform [1]. With advancing age, as well as in hypertensive individuals, increases in stiffness of the central elastic arteries results in an increase in transmission of both forward and retrograde waves. This causes the reflected wave to arrive earlier in the central aorta, with a greater amplitude and duration, augmenting pressure in late systole [1]. This premature return of reflected waves in late systole results in an increase in central pulse pressure (PP) as well as systolic blood pressure (BP). In turn, this increases the load on the left ventricle, increasing myocardial oxygen demand, and evidently decreases stroke output [1]. Increases in systolic pressure causes an increase in arterial wall circumferential stress, promoting local fatigue as well as the development of atherosclerosis [1]. Evidently, arterial stiffness is also associated with left ventricular hypertrophy [14, 15], a known risk factor for coronary events in normotensive and hypertensive patients [1, 15].

1.4 Cardiovascular disease

Cardiovascular disease (CVD) is the leading cause of death in North America and includes a vast array of conditions such as atherosclerosis, coronary heart disease and stroke [16]. Physical inactivity is a major, controllable risk factor for CVD. Physical inactivity has been shown to have a strong correlation with an elevated susceptibility to CVD and CVD-related death [17]. Physical inactivity is a significant precursor to CVD-related death with moderate levels of fitness proving to have protective effects against the influence of traditional risk factors on mortality [17].

1.5 Measures of arterial stiffness

Measurement of arterial stiffness enables assessment of arterial dysfunction, which may precede clinical, structural changes such as those seen through assessment of arterial wall thickness. Noninvasive measures of arterial stiffness include pulse wave velocity (PWV) and arterial compliance and distensibility [1, 13]. These measures have been shown to be clinically useful, noninvasive and easily obtainable in younger populations [7, 18].

1.5.1 Pulse wave velocity

Pulse wave velocity (PWV) describes the speed at which a pressure wave (generated by ventricular contraction) travels along an arterial segment and is positively related to arterial wall stiffness [19]. PWV is a sensitive marker of arterial wall stiffness reflective of the elastic properties of the vessel wall, and is a subsequent marker of cardiovascular risk [2, 19]. An increase in PWV is associated with increased risk of coronary artery disease, stroke and cardiovascular death in elderly populations [3, 20-22]. Aortic PWV is also a significant independent predictor of cardiovascular mortality in the general population, patients with end-stage renal disease [23], hypertensive patients [2] and has been shown to be a strong predictor of stroke death in hypertensive patients [3].

PWV is positively correlated with body mass index (BMI), waist circumference (WC) and percentage body fat and negatively correlated with cardiorespiratory fitness and physical activity (PA) levels in children [24]. Favourable changes in risk factor profile and obesity status from childhood to adulthood are associated with lower values of

PWV in adulthood [25] indicating a place for lifestyle modifications in prevention of atherosclerosis at the earliest stage possible.

1.5.2 Measurement of PWV

Both central and peripheral PWV (cPWV, pPWV) can be calculated using the formula [19]:

Equation 1 $$PWV = \frac{D}{\Delta t}$$

where D is the distance between measurement sites (m), and Δt is the pulse transit time (sec.). Pulse transit time (PTT) is calculated as the time delay between the arrival of the 'foot' of the waveform at the proximal location and the arrival of the foot of the wave at the distal location. The foot of the pressure waveform is defined as, *the point, at end diastole when the steep rise of the wavefront begins* [1]. This identification point is used because it maintains its identity most consistently throughout the propagated wave and is the least affected by wave reflections; therefore, the velocity of this point is considered characteristic of the entire wave and may be tracked throughout the arterial tree [1].

The foot of the waveform can be identified using a variety of techniques such as the maximum of the second derivative of the pressure wave [13], or through the use of a Bandpass filter [26] (Fig. 2). The sharp inflection point of a pressure waveform is comprised of high frequency harmonics [1]; therefore, when applying a digital filter with a low and high frequency cut off of 5 and 30Hz, respectively, the low frequencies and high frequency noise is eliminated. In turn, this allows for the identification of the foot of the waveform as the minimum point of the digitally filtered signal [26] (Fig. 2).

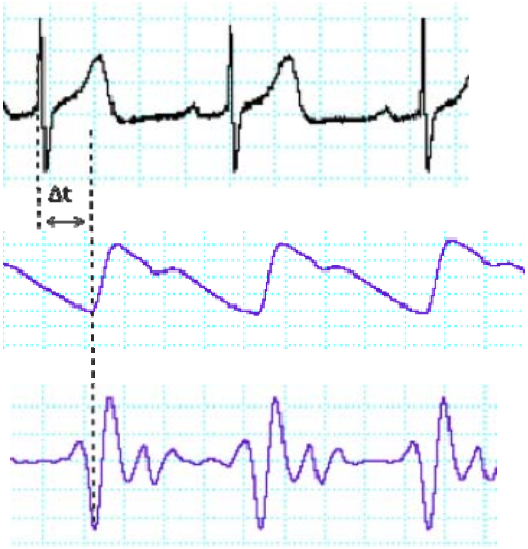

Figure 2. Calculating PTT from the depolarization of the ECG to the minimum point of the Bandpass filtered carotid waveform. The same procedure is done to calculate the PTT between the ECG signal and the femoral signal, which is also used in calculations of PTT for cPWV.

The subtraction method is used when calculating cPWV, which accounts for the pulse simultaneously travelling to both the carotid and femoral arteries [27]. When a direct distance is measured (carotid – femoral), the calculated PWV is an overestimate to that found using invasive measurements [1]. The most accurate agreement with invasive measurements is the method of subtracting the carotid to suprasternal notch distance from the suprasternal notch to femoral distance [28]. The PTT between ventricular depolarization (R spike of ECG signal) and the common carotid artery (Fig. 2) is subtracted from the time delay between ventricular depolarization and the arrival of the foot of the femoral artery pulse wave. Peripheral PTT is determined as the time delay between the arrival of the femoral artery pulse wave and the dorsalis pedis artery pulse wave [29]. Anthropometric measuring tape is used to measure the distances between sites

(sternal notch to the placement of each PPG sensor) along the surface of the body. Similarly to PTT, cPWV path length (distance) is calculated by subtracting the surface distance between the sternal notch and the carotid PPG placement from that of the sternal notch and the femoral PPG placement.

Although both cPWV and pPWV are commonly reported, only cPWV has been a consistent predictor of clinical outcomes and cardiovascular risk. Carotid-femoral PWV (cPWV) is considered the 'gold-standard' measurement of arterial stiffness because of its inclusion of the aortic track. By including the aorta and its first branches in the measurement of PWV, the segments that most closely experience the high pressures exerted by the left ventricle are assessed. Therefore, it has been suggested that these branches are responsible for the majority of pathophysiological effects of arterial stiffness [13]. However, the impact of increased stiffness of the peripheral arteries on cardiovascular health must not be overlooked. It has been suggested that stiffening of distal segments of the arterial tree may result in premature return of the peripheral pulse wave at the level of the ascending aorta, thereby increasing cardiac afterload during the systolic phase of the cardiac cycle [30].

1.5.3 Arterial compliance and distensibility

Arterial compliance is the ability of the blood vessel to expand with a given change in pressure [19] and is influenced by the interaction of arterial mechanical properties and vessel geometry [31]. Compliance is modulated by endothelial function, blood vessel wall structure, and muscle tone and is expected to relate to body size [31]. Similarly, arterial distensibility is also a direct measure of arterial stiffness, but is

independent of body size and quantifies the relative change in diameter with respect to a pressure increment [19]. Measures of compliance and distensibility may show functional changes that precede detectable structural disease of the arterial wall [32].

Throughout the lifespan, habitual PA has been shown to positively influence arterial distensibility [24, 33]. Age-related decreases in arterial distensibility and increases in stiffness have been reported [33]. Importantly, increased levels of PA may delay the age-dependent loss of arterial distensibility, with greater benefits occuring in proportion to the amount and/or intensity of exercise [33-35].

1.5.4 Measurement of carotid compliance and distensibility

Local arterial stiffness of superficial arteries is often assessed at the common carotid artery, which is convenient to access and is representative of the changes occurring at the level of the aorta [7]. Direct measurement of carotid compliance or distensibility can be made using a combination of B-mode ultrasound imaging and applanation tonometry. Ultrasound images provide a means of assessing the change in lumen size from diastole to systole by calculating the increase in diameter of the vessel during systole. These measurements are dependent on the local distending pressure driving this change in volume. Estimates of local BP are often obtained at the contralateral carotid artery using a hand held tonometer [36, 37]. This device is held over the point of greatest pulsation while continuous BP waveforms are collected, providing an estimate of the blood pressure within the common carotid artery. Carotid tonometry has been validated against invasive measurements [38], and under optimal conditions, the pressure waveforms measured noninvasively are virtually identical to those recorded invasively [39].

When calculating carotid compliance or distensibility, it is important that the same investigator analyze each ultrasound image to ensure reliability and consistency. Using consecutive heart cycles, minimum, mean, and maximum carotid artery diameters are determined from the ultrasound video clips and the following equation is used to calculate distensibility [19]:

Equation 2 $$Distensibility = \frac{\Pi\left(\frac{d_{\max}}{2}\right)^2 - \Pi\left(\frac{d_{\min}}{2}\right)^2}{\Pi\left(\frac{d_{\min}}{2}\right)^2 \times PP}$$

where d_{max} is the maximum diameter, d_{min} is the minimum diameter, and PP is the carotid pulse pressure. PP is responsible for the change in artery cross sectional area (CSA) and is the difference between the diastolic BP (DBP) and systolic BP (SBP).

Similarly, compliance can be determined using the equation [19]:

Equation 3 $$Compliance = \frac{\Pi\left(\frac{d_{\max}}{2}\right)^2 - \Pi\left(\frac{d_{\min}}{2}\right)^2}{PP}$$

1.6 Measure of arterial structure

1.6.1 Carotid intima-media thickness

Carotid intima-media thickness (IMT) is an index of arterial structure, which describes the thickness of the artery walls [19]. An increase in IMT is associated with cardiovascular risk factors and predictive of coronary artery disease in older adults [40]. Carotid IMT is also associated with prevalent stroke, peripheral vascular disease as well as an increased risk of myocardial infarction and stroke in older adults; this association persists after adjustment for known cardiovascular risk factors [18]. Measurements of

carotid IMT have been shown to be just as strong, if not more powerful than traditional risk factors at predicting coronary events in older adults [18].

Low levels of PA in childhood (3-18 years) are directly, inversely associated with accelerated IMT progression in adulthood [41]. Exposure to cardiovascular risk factors during childhood predicts increases in IMT two decades later [40, 42], with increasing atherosclerosis progression rate in adulthood in children with risk factors for CVD, including low levels of physical activity [41]. Increased central arterial stiffness as well as carotid IMT have been observed in children with cardiovascular disease risk factors such as familial hypercholesterolemia [43], type I diabetes [44, 45] and obesity [46, 47].

1.6.2 Measurement of carotid IMT

Measurements of carotid IMT are attained from ultrasound images of the superficial common carotid artery. Far-wall IMT is calculated from the proximal aspect of the intima to the proximal interface of the adventitia at end diastole.

1.7 Measure of arterial function

1.7.1 Vascular endothelium

The vascular endothelium is a large, multifunctional paracrine organ comprised of highly specialized endothelial cells that line the interior surface of the entire cardiovascular system [48, 49]. The endothelium is a source of secretions that regulate vessel diameter, cell proliferation, platelets and leukocyte interaction as well as thrombus formation and can be regarded as the primary governor of body homeostasis [49, 50]. Specifically, the vascular endothelium regulates vascular tone and maintains systemic vascular homeostasis in the face of stressors [48, 51]. Numerous vasoactive substances

are released from the endothelium such as nitric oxide (NO), a primary regulator of vasodilation [52]. The integrity of this single layer of cells is crucial for the maintenance of arterial health and prevention of hypertension and atherosclerotic development [48, 51].

1.7.2 Endothelial dysfunction

Endothelial dysfunction refers to alterations of endothelial physiology such as a reduction of the bioavailability of vasodilators, in particular NO, accompanied by an increase in endothelium-derived contracting factors [48]. This imbalance results in an impairment of endothelium-dependent vasodilation and a decreased ability of endothelial cells to respond to a variety of external and internal stimuli [48]. Endothelial dysfunction is considered an early and integral manifestation of atherosclerotic disease [48], which can be evident in the first decade of life [22].

It has been well documented that youth with traditional risk factors such as, cigarette smoking, hyperlipidemia, diabetes and hypertension [53], obesity [54, 55] and familial hypercholesterolemia [56] exhibit impaired endothelial-dependant function in comparison to their healthy controls. This dysfunction can also occur in young, symptom free subjects and precede structural, anatomical evidence of atherosclerosis [57].

1.7.3 The flow-mediated dilation assessment

Flow-mediated vasodilatation (FMD), a non-invasive tool to assess endothelial-dependent function, was developed in 1992 by Celermajer *et al.* [56]. This assessment involves occlusion-induced hyperemia as a stimulus to provoke the release of NO and cause subsequent vasodilation [51, 58]. Joannides *et al*. (1995) have shown that NO is

essential for flow-mediated dilatation of human radial arteries in vivo and under specific conditions, the FMD test is sufficient to elicit an NO dependent FMD response. This is a representation of endothelium-derived NO bioavailability, indicative of potential endothelial dysfunction, the earliest identifiable precursor to atherosclerotic cardiovascular disease [59, 60]. This vasodilator response to increased conduit arterial flow can be imaged using ultrasonic assessment and quantified as an index of vasomotor function.

Peripherally measured endothelial function in the brachial artery has been shown to have prognostic value for the development of cardiovascular disease and correlates with invasively assessed endothelial function of the coronary arteries [61]. In adults, endothelial dysfunction is strongly associated with the existence and severity of coronary endothelial dysfunction with a negative correlation shown between %FMD in the brachial artery and the number and severity of significantly diseased coronary vessels [62]. FMD assessment is established as a reliable measurement and has been shown to be the most reproducible and least variable of the techniques used in the measurement of endothelial function in children and adults [63, 64].

Although developed in 1992 [56], many adaptations, revisions and attempts to standardize the FMD procedure have been made over the years. A recent tutorial published by Harris, 2010 [58] outlines recommended guidelines for the performance and interpretation of an FMD; however, procedures continue to vary within laboratories due to equipment limitations, software requirements and experimenter expertise. Nevertheless, the primary outcome of this functional test remains the same: to stimulate

and quantify the NO-induced vasodilatory response to increased conduit arterial flow.

1.7.4 Measurement of endothelial function

In order to create the flow stimulus, the forearm cuff is typically inflated to a pressure of at least 50mmHg suprasystolic, to ensure arterial inflow occlusion and ischemia of downstream vessels / tissue [51]. A standardized pressure of 200mmHg is commonly used with the cuff positioned distally to the area of FMD measurement. An occlusion time of 5 minutes in the absent of ischemic handgrip exercise has been shown to elicit an NO dependent response [65-67]. Variations to the nature of this stimulus will result in varying shear stress profiles and resultant FMD responses [65, 66, 68, 69].

Upon completion of the 5 min occlusion, the cuff is deflated and ischemia-induced reactive hyperaemia occurs, resulting in a high-flow state through the conduit artery [51]. To capture this response, blood velocity signals and ultrasound images of the brachial artery are collected for 3 min. post occlusion. Analysis involves quantifying the lumen diameter of end-diastolic frames (as depicted using the R Spike of the ECG wave) of both baseline and post occlusion images. Frames at end-diastole are used in order to eliminate the influence of potential differences in arterial compliance between subjects [50]. Peak FMD expressed as a % change (%FMD) is taken as an index of endothelial function [70] and represents the % change in diameter relative to that of the baseline image [50]. Absolute FMD (mm) and relative FMD (%FMD) are calculated as follows [61]:

Equation 4 $$AbsoluteFMD = PeakDiameter(mm) - BaselineDiameter(mm)$$

Equation 5 $$\%FMD = \left(\frac{AbsoluteFMD}{BaselineDiameter} \right) \times 100\%$$

It is speculated that the magnitude of the stimulus is proportional to the FMD

response as smaller arteries appear to dilate relatively more than do larger arties[61] [56]; therefore this is acknowledged by normalizing the peak FMD response to the shear rate stimulus [71]. The increase in shear stress has been established as the primary physiological stimulus for the endothelium to elicit an NO-dependent FMD response in large human conduit arteries [66, 72-74]. In order to quantify the magnitude of the shear stimulus, the area under the curve (AUC) method is used. Ideally, this method utilizes the Duplex mode to obtain both blood velocity and diameter measurements throughout the entire post-occlusion time frame in order to examine the shear rate stimulus over time [58]. Shear rate is calculated as follows [75]:

Equation 6 $$ShearRate = 8 \times \left(\frac{Velocity}{Diameter} \right)$$

where velocity represents the mean blood flow velocity, and diameter is the internal artery diameter. This also provides the opportunity to determine the true peak diameter, and the time it took to reach this peak. The shear rate AUC up until the max dilation of the vessel (diameter, mm) is then divided into the %FMD to calculate the normalized FMD response [71]:

Equation 7 $$NormalizedFMD = \frac{\%FMD}{SR_{AUC}}$$

where %FMD is the relative FMD previously calculated and SR_{AUC} is the total shear rate until peak vasodilation. The validity of the normalization of endothelial dependent FMD response to the shear stimulus has been directly confirmed [74]. The total shear AUC until peak vasodilation has been shown to yield the strongest relationship with %FMD in both young and old age groups compared to normalizing to peak shear or

the entire shear rate curve [58, 71]

In theory, although this methodology is relatively straightforward and noninvasive, limitations to this procedure vary, and encompass both technical and interpretive challenges. This procedure is largely dependent on the quality of the ultrasound images (skilled investigator) and appropriate equipment and analysis software. Overall, peripherally measured endothelial function in the brachial artery has prognostic value for the development of cardiovascular disease and correlates with invasively assessed endothelial function of the coronary arteries [61].

Endothelial function testing is a non-invasive technique that allows reproducible testing of vascular function without associated patient risks. Endothelial dysfunction is associated with long-term CVD clinical outcome in both healthy, and clinical populations [50]. Thus, this test can be used as a reliable noninvasive estimate of the capacity of human endothelial cells to release NO in response to this physiological stimulus as well as an estimate of endothelial dysfunction in diseased states [66].

1.8 Interventions

Endothelial function can be modified by appropriate interventions, highlighting the importance of early identification of at-risk pediatric patients. Significant improvements in the FMD response have been seen with exercise training, dietary intervention or vitamin and folic acid supplementation to the FMD response in children with risk factors such as type I diabetes and familial hyperlipidemia. Although a variety of short-term lifestyle interventions involving dietary modifications, exercise training or a combination of both have been shown to improve arterial health [76-79], any

improvements appear to be rapidly reversed with cessation of the interventions, highlighting the need for lifestyle modifications rather than acute interventions for the maintenance of arterial health from childhood into adulthood.

1.9 Interrelationship between non-invasive measures of arterial health

It has been determined that non-invasive measurements of atherosclerosis such as FMD of the brachial artery, carotid IMT and PWV are related and their combination is of clinical significance [70]. In an adult population, age, FMD, IMT and PWV (brachial-ankle) were significantly correlated, suggesting a strong relationship between these measures and a relevant clinical tool in predicting the presence of atherosclerotic disease when used in combination [70].

PART II

1. CEREBRAL PALSY

1.8 Definition

Over the years, many attempts have been made to establish an all-encompassing definition of Cerebral Palsy (CP). A classic and still widely cited definition states that CP is, 'a disorder of movement and posture due to a defect or lesion of the immature brain' [80]. However, this definition appears to be limited in its exclusive focus on motor deficits and disregards additional impairments that often accompany CP such as sensory, cognitive, and behavioural deficits. A recent, and generally accepted definition by Rosenbaum and colleagues states that [81]:

> *Cerebral palsy (CP) describes a group of permanent disorders of the development of movement and posture, causing activity limitation, that are attributed to non-progressive disturbances that occurred in the developing fetal or infant brain. The motor disorders of cerebral palsy are often accompanied by disturbances of*

> *sensation, perception, cognition, communication, and behaviour, by epilepsy, and by secondary musculoskeletal problems.*

CP is difficult to diagnose as the term is not an etiologic diagnosis, but a descriptive term that encompasses a wide range of clinical presentations [80]. CP can be classified as a disorder of posture and movement, with evidence of motor impairment seen in the first 18 months of life [80]. Children with CP often experience some type of movement disorder classified as spastic, dyskinetic or ataxic [82]. CP can further be classified by limb distribution (unilateral or bilateral), level of self-mobility (according to the Gross Motor Function Classification System), and level of manual ability (using the Manual Ability Classification System) [83]

1.9 The Gross Motor Function Classification System (GMFCS)

A wide variety of classification systems have been employed to assist in the description of CP on the basis of various domains of disability. The International Classification of Functioning, Disability, and Health (ICF), was published in 2001 by the World Health Organization and describes disability as dysfunction at one or more of 3 levels. These levels include impairment of body structures or functions, limitations in activities, and restriction of participation [84]. The importance of evaluating the functional consequences has been well recognized and objective, functional scales have been developed on this basis.

The Gross Motor Function Classification System (GMFCS) is a five-level classification system for individuals up to the age of 12 years old, based on functional mobility and activity limitation. Level distinction is based on functional limitations, the need for mobility devices (hand-held and wheeled) and the quality of movement, with

emphasis on the distinctions between levels being meaningful in daily life [85]. Limitations to the GMFCS included the pre-adolescent age range (only up to 12 years) and the basis of classification in each level on the individual's best capability rather than their everyday performance, which may be a more accurate representation of their functional limitations.

Evidently, the expanded and revised GMFCS (GMFCS –E & R) was designed to attenuate the minor limitations to the GMFCS and include individuals up to 18 years of age. The GMFCS – E &R incorporates aspects of the ICF framework and addresses the qualitative limitations that may affect gross motor performance including environmental and personal factors [86]. This system bases its classification of the individuals on their typical performance, in home, school and community settings, rather than just their best performance, or what they are known to be capable of. Gross motor function is classified on a 5-point ordinal scale based on skills provided for 5 separate age groups (Appendix A).

1.10 Physical activity and CP

Youth with CP are shown to be less physically active than their typically developing peers [87-90]. An inverse relationship is seen between functional limitations and social participation for both adolescents and adults with CP [90]. Physical inactivity places these individuals at a greater risk of developing a variety of secondary health complications [91], but is also a major controllable risk factor for cardiovascular disease (CVD) [92, 93], which is the leading cause of death in North America [94]

For children with CP, an individual's movement, manual and intellectual abilities are influential predictors of their extent of physical independence; however, these factors are not predictive of other domains of their activities and participation [95]. Motor capacity (what a child can do in a standardized environment), motor capability (what a child can do in a daily environment), and motor performance (what a child does do in a daily environment) [96, 97] in youth with CP are highly correlated [97]. However, the largest gap between these constructs exists between capacity and performance as a host of environmental factors play a role in the actual participation of activities in the individuals' daily life [98, 99]. This is of particular interest when implementing lifestyle modification and PA interventions in this group. The specific personal and environmental factors for each child must be considered when assessing what the child can do in a controlled, standardized environment and what they can actually do in their own daily environment [97]. Taking these differences in performance into account may assist in optimizing the intervention.

Multiple child and family determinants influence the intensity of participation of children with CP [100]. As higher functioning youth with CP increase in age, their levels of dependence decreases [95], allowing for an increase in choice in their daily lives and potential to participate in PA at their own discretion. Social integration is a complex area for these youth and is comprised of a variety of factors with only 36% of the variance explained by factors related to their condition, such as movement ability and intellectual delay [101]. At this point in time, it is therefore of key importance to encourage and

communicate the value of establishing and maintaining appropriate levels of PA in adolescence into adulthood.

It is well established that cardiovascular risk factors are identifiable in childhood and are predictive of future cardiovascular risk (Berenson, 2002); therefore, it is of great importance to examine the cardiovascular health, specifically, indices of vascular structure and function in young adolescents in a clinical population. Previous findings have suggested that the prevention of vascular aging and exposure to cardiovascular risk factors may be most effective when initiated in childhood or adolescence [42]; thus, justifying the evaluation of cardiovascular risk factors in these adolescents and providing a rationale for both prevention and intervention at a young age. Andersen *et al*., 2006 [102] used Actigraph accelerometers to record physical activity in 9 and 15 year-old children, but separated PA levels into five quintiles. It was found that children who engaged in levels of PA falling in the first to third quintiles (i.e. lower levels of PA) had significantly higher risk of CVD than those in the fourth and fifth quintiles [102]. This may therefore be a more effective method of evaluating the relationship between CVD risk and levels of physical activity.

A chronological and biological age-related decline in PA has been shown in both genders, raising concern for maintenance of habitual PA throughout adolescence for any population [103]. Functional abilities also appear to decrease with age in individuals with CP, further compounding the concern for physical inactivity in this group. Adults with CP have stated that their current gross motor function according to the GMFCS has decreased by one ore more levels compared with their function during childhood [104]. It

is therefore important to attenuate any physical inactivity-related cardiovascular sequelae that these individuals may experience at the earliest stage possible. Identification of any compromises to the vascular health that may be further compounded by the normative aging process is of key interest.

1.11 Physical activity into adulthood

With difficulty in maintaining an active lifestyle throughout adolescence for any individual, particular care must be given to individuals with a physical disability [105]. Specifically, adolescents with CP are at a higher risk of leading sedentary lifestyles in comparison to age and sex matched controls [61, 90, 106]. The potential consequences of this sedentary behaviour on specific parameters of arterial health and CVD risk in a young population of CP patients have yet to be examined. To our knowledge, there is no assessment of the potential impacts of this chronic condition and impaired levels of habitual PA on the arterial health in adolescents with CP. Therefore, it is the purpose of this study to provide normative baseline measures of vascular function (endothelial function, PWV, and carotid distensibility) and structure (carotid IMT) in adolescents with CP (GMFCS levels I-II) and to make comparisons to a control group of typically developing peers.

1.12 References

1. Nichols, W.W. and M.F. O'Rourke, *McDonald's Blood Flow in Arteries: Theoretical, Experimental and Clinical Principles. 5th ed.* 2005: A Hodder Arnold Publication.
2. Laurent, S., et al., *Aortic stiffness is an independent predictor of all-cause and cardiovascular mortality in hypertensive patients.* Hypertension, 2001. **37**(5): p. 1236-41.
3. Laurent, S., et al., *Aortic stiffness is an independent predictor of fatal stroke in essential hypertension.* Stroke, 2003. **34**(5): p. 1203-6.
4. Germann, W.J. and C.L. Stanfield, *Principles of Human Physiology*. 2nd ed. The Cardiovascular System: Blood Vessels, Blood Flow, and Blood Pressure, ed. B. Yien. 2005, San Francisco: Pearson, Benjamin Cummings.
5. Laurent, S., P. Boutouyrie, and P. Lacolley, *Structural and genetic bases of arterial stiffness.* Hypertension, 2005. **45**(6): p. 1050-5.
6. Benetos, A., et al., *Arterial alterations with aging and high blood pressure. A noninvasive study of carotid and femoral arteries.* Arterioscler Thromb, 1993. **13**(1): p. 90-7.
7. Fernhall, B. and S. Agiovlasitis, *Arterial function in youth: window into cardiovascular risk.* J Appl Physiol, 2008. **105**(1): p. 325-33.
8. Vaitkevicius, P.V., et al., *Effects of age and aerobic capacity on arterial stiffness in healthy adults.* Circulation, 1993. **88**(4 Pt 1): p. 1456-62.
9. Tanaka, H., C.A. DeSouza, and D.R. Seals, *Absence of age-related increase in central arterial stiffness in physically active women.* Arterioscler Thromb Vasc Biol, 1998. **18**(1): p. 127-32.
10. Lakatta, E.G., *Arterial and cardiac aging: major shareholders in cardiovascular disease enterprises: Part III: cellular and molecular clues to heart and arterial aging.* Circulation, 2003. **107**(3): p. 490-7.
11. Nosaka, T., et al., *Influence of regular exercise on age-related changes in arterial elasticity: mechanistic insights from wall compositions in rat aorta.* Can J Appl Physiol, 2003. **28**(2): p. 204-12.
12. Lakatta, E.G., et al., *Human aging: changes in structure and function.* J Am Coll Cardiol, 1987. **10**(2 Suppl A): p. 42A-47A.
13. Laurent, S., et al., *Expert consensus document on arterial stiffness: methodological issues and clinical applications.* Eur Heart J, 2006. **27**(21): p. 2588-605.
14. O'Rourke, M.F., W.W. Nichols, and M.E. Safar, *Pulse waveform analysis and arterial stiffness: realism can replace evangelism and scepticism.* J Hypertens, 2004. **22**(8): p. 1633-4; author reply 1634.
15. Boutouyrie, P., et al., *Common carotid artery stiffness and patterns of left ventricular hypertrophy in hypertensive patients.* Hypertension, 1995. **25**(4 Pt 1): p. 651-9.

16. Lloyd-Jones, D., et al., *Heart disease and stroke statistics--2009 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee.* Circulation, 2009. **119**(3): p. 480-6.
17. Blair, S.N., et al., *Influences of cardiorespiratory fitness and other precursors on cardiovascular disease and all-cause mortality in men and women.* JAMA, 1996. **276**(3): p. 205-10.
18. O'Leary, D.H., et al., *Carotid-artery intima and media thickness as a risk factor for myocardial infarction and stroke in older adults. Cardiovascular Health Study Collaborative Research Group.* N Engl J Med, 1999. **340**(1): p. 14-22.
19. O'Rourke, M.F., et al., *Clinical applications of arterial stiffness; definitions and reference values.* Am J Hypertens, 2002. **15**(5): p. 426-44.
20. Boutouyrie, P., et al., *Aortic stiffness is an independent predictor of primary coronary events in hypertensive patients: a longitudinal study.* Hypertension, 2002. **39**(1): p. 10-5.
21. Sutton-Tyrrell, K., et al., *Elevated aortic pulse wave velocity, a marker of arterial stiffness, predicts cardiovascular events in well-functioning older adults.* Circulation, 2005. **111**(25): p. 3384-90.
22. Stary, H.C., *Evolution and progression of atherosclerotic lesions in coronary arteries of children and young adults.* Arteriosclerosis, 1989. **9**(1 Suppl): p. I19-32.
23. Pannier, B., et al., *Stiffness of capacitive and conduit arteries: prognostic significance for end-stage renal disease patients.* Hypertension, 2005. **45**(4): p. 592-6.
24. Sakuragi, S., et al., *Influence of adiposity and physical activity on arterial stiffness in healthy children: the lifestyle of our kids study.* Hypertension, 2009. **53**(4): p. 611-6.
25. Aatola, H., et al., *Lifetime risk factors and arterial pulse wave velocity in adulthood: the cardiovascular risk in young Finns study.* Hypertension, 2010. **55**(3): p. 806-11.
26. Munakata, M., et al., *Utility of automated brachial ankle pulse wave velocity measurements in hypertensive patients.* Am J Hypertens, 2003. **16**(8): p. 653-7.
27. Vermeersch, S.J., et al., *Distance measurements for the assessment of carotid to femoral pulse wave velocity.* J Hypertens, 2009. **27**(12): p. 2377-85.
28. Weber, T., et al., *Noninvasive determination of carotid-femoral pulse wave velocity depends critically on assessment of travel distance: a comparison with invasive measurement.* J Hypertens, 2009. **27**(8): p. 1624-30.
29. Rakobowchuk, M., et al., *Effect of acute sprint interval exercise on central and peripheral artery distensibility in young healthy males.* Eur J Appl Physiol, 2009. **105**(5): p. 787-95.
30. Nichols, W.W., *Clinical measurement of arterial stiffness obtained from noninvasive pressure waveforms.* Am J Hypertens, 2005. **18**(1 Pt 2): p. 3S-10S.
31. Ahimastos, A.A., et al., *Gender differences in large artery stiffness pre- and post puberty.* J Clin Endocrinol Metab, 2003. **88**(11): p. 5375-80.

32. Berenson, G.S., *Childhood risk factors predict adult risk associated with subclinical cardiovascular disease. The Bogalusa Heart Study.* Am J Cardiol, 2002. **90**(10C): p. 3L-7L.
33. Seals, D.R., et al., *Habitual exercise and arterial aging.* J Appl Physiol, 2008. **105**(4): p. 1323-32.
34. Kakiyama, T., M. Matsuda, and S. Koseki, *Effect of physical activity on the distensibility of the aortic wall in healthy males.* Angiology, 1998. **49**(9): p. 749-57.
35. Seals, D.R., et al., *Habitual exercise and vascular ageing.* J Physiol, 2009. **587**(Pt 23): p. 5541-9.
36. Aggoun, Y., I. Szezepanski, and D. Bonnet, *Noninvasive assessment of arterial stiffness and risk of atherosclerotic events in children.* Pediatr Res, 2005. **58**(2): p. 173-8.
37. Oliver, J.J. and D.J. Webb, *Noninvasive assessment of arterial stiffness and risk of atherosclerotic events.* Arterioscler Thromb Vasc Biol, 2003. **23**(4): p. 554-66.
38. Van Bortel, L.M., et al., *Non-invasive assessment of local arterial pulse pressure: comparison of applanation tonometry and echo-tracking.* J Hypertens, 2001. **19**(6): p. 1037-44.
39. Davies, J.I., et al., *Peripheral blood pressure measurement is as good as applanation tonometry at predicting ascending aortic blood pressure.* J Hypertens, 2003. **21**(3): p. 571-6.
40. Davis, P.H., et al., *Carotid intimal-medial thickness is related to cardiovascular risk factors measured from childhood through middle age: The Muscatine Study.* Circulation, 2001. **104**(23): p. 2815-9.
41. Juonala, M., et al., *Life-time risk factors and progression of carotid atherosclerosis in young adults: the Cardiovascular Risk in Young Finns study.* Eur Heart J, 2010. **31**(14): p. 1745-51.
42. Raitakari, O.T., et al., *Cardiovascular risk factors in childhood and carotid artery intima-media thickness in adulthood: the Cardiovascular Risk in Young Finns Study.* JAMA, 2003. **290**(17): p. 2277-83.
43. Virkola, K., et al., *Cholesterol and carotid artery wall in children and adolescents with familial hypercholesterolaemia: a controlled study by ultrasound.* Acta Paediatr, 1997. **86**(11): p. 1203-7.
44. Heilman, K., et al., *Arterial stiffness, carotid artery intima-media thickness and plasma myeloperoxidase level in children with type 1 diabetes.* Diabetes Res Clin Pract, 2009. **84**(2): p. 168-73.
45. Jarvisalo, M.J., et al., *Endothelial dysfunction and increased arterial intima-media thickness in children with type 1 diabetes.* Circulation, 2004. **109**(14): p. 1750-5.
46. Iannuzzi, A., et al., *Increased carotid intima-media thickness and stiffness in obese children.* Diabetes Care, 2004. **27**(10): p. 2506-8.
47. Zebekakis, P.E., et al., *Obesity is associated with increased arterial stiffness from adolescence until old age.* J Hypertens, 2005. **23**(10): p. 1839-46.
48. Vita, J.A. and J.F. Keaney, Jr., *Endothelial function: a barometer for cardiovascular risk?* Circulation, 2002. **106**(6): p. 640-2.

49. Simionescu, M. and F. Antohe, *Functional ultrastructure of the vascular endothelium: changes in various pathologies.* Handb Exp Pharmacol, 2006(176 Pt 1): p. 41-69.
50. Shechter, M., et al., *The association of endothelial dysfunction and cardiovascular events in healthy subjects and patients with cardiovascular disease.* Isr Med Assoc J, 2007. **9**(4): p. 271-6.
51. Corretti, M.C., et al., *Guidelines for the ultrasound assessment of endothelial-dependent flow-mediated vasodilation of the brachial artery: a report of the International Brachial Artery Reactivity Task Force.* J Am Coll Cardiol, 2002. **39**(2): p. 257-65.
52. Singel, D.J. and J.S. Stamler, *Chemical physiology of blood flow regulation by red blood cells: the role of nitric oxide and S-nitrosohemoglobin.* Annu Rev Physiol, 2005. **67**: p. 99-145.
53. Celermajer, D.S. and J.G. Ayer, *Childhood risk factors for adult cardiovascular disease and primary prevention in childhood.* Heart, 2006. **92**(11): p. 1701-6.
54. Tounian, P., et al., *Presence of increased stiffness of the common carotid artery and endothelial dysfunction in severely obese children: a prospective study.* Lancet, 2001. **358**(9291): p. 1400-4.
55. Gielen, S. and R. Hambrecht, *The childhood obesity epidemic: impact on endothelial function.* Circulation, 2004. **109**(16): p. 1911-3.
56. Celermajer, D.S., et al., *Non-invasive detection of endothelial dysfunction in children and adults at risk of atherosclerosis.* Lancet, 1992. **340**(8828): p. 1111-5.
57. Celermajer, D.S., et al., *Endothelium-dependent dilation in the systemic arteries of asymptomatic subjects relates to coronary risk factors and their interaction.* J Am Coll Cardiol, 1994. **24**(6): p. 1468-74.
58. Harris, R.A., et al., *Ultrasound assessment of flow-mediated dilation.* Hypertension, 2010. **55**(5): p. 1075-85.
59. Green, D., *Point: Flow-mediated dilation does reflect nitric oxide-mediated endothelial function.* J Appl Physiol, 2005. **99**(3): p. 1233-4; discussion 1237-8.
60. Ross, R., *Atherosclerosis--an inflammatory disease.* N Engl J Med, 1999. **340**(2): p. 115-26.
61. Anderson, T.J., et al., *Close relation of endothelial function in the human coronary and peripheral circulations.* J Am Coll Cardiol, 1995. **26**(5): p. 1235-41.
62. Abdessalem, S., et al., *[Assessment of endothelial function reliably predicts the existence and the severity of coronary disease].* Tunis Med, 2009. **87**(12): p. 843-50.
63. Donald, A.E., et al., *Non-invasive assessment of endothelial function: which technique?* J Am Coll Cardiol, 2006. **48**(9): p. 1846-50.
64. Uehata, A., et al., *Noninvasive assessment of endothelium-dependent flow-mediated dilation of the brachial artery.* Vasc Med, 1997. **2**(2): p. 87-92.
65. Mullen, M.J., et al., *Heterogenous nature of flow-mediated dilatation in human conduit arteries in vivo: relevance to endothelial dysfunction in hypercholesterolemia.* Circ Res, 2001. **88**(2): p. 145-51.

66. Joannides, R., et al., *Nitric oxide is responsible for flow-dependent dilatation of human peripheral conduit arteries in vivo.* Circulation, 1995. **91**(5): p. 1314-9.
67. Doshi, S.N., et al., *Flow-mediated dilatation following wrist and upper arm occlusion in humans: the contribution of nitric oxide.* Clin Sci (Lond), 2001. **101**(6): p. 629-35.
68. Leeson, P., et al., *Non-invasive measurement of endothelial function: effect on brachial artery dilatation of graded endothelial dependent and independent stimuli.* Heart, 1997. **78**(1): p. 22-7.
69. Betik, A.C., V.B. Luckham, and R.L. Hughson, *Flow-mediated dilation in human brachial artery after different circulatory occlusion conditions.* Am J Physiol Heart Circ Physiol, 2004. **286**(1): p. H442-8.
70. Kobayashi, K., et al., *Interrelationship between non-invasive measurements of atherosclerosis: flow-mediated dilation of brachial artery, carotid intima-media thickness and pulse wave velocity.* Atherosclerosis, 2004. **173**(1): p. 13-8.
71. Pyke, K.E. and M.E. Tschakovsky, *Peak vs. total reactive hyperemia: which determines the magnitude of flow-mediated dilation?* J Appl Physiol, 2007. **102**(4): p. 1510-9.
72. Koller, A., D. Sun, and G. Kaley, *Role of shear stress and endothelial prostaglandins in flow- and viscosity-induced dilation of arterioles in vitro.* Circ Res, 1993. **72**(6): p. 1276-84.
73. Pyke, K.E. and M.E. Tschakovsky, *The relationship between shear stress and flow-mediated dilatation: implications for the assessment of endothelial function.* J Physiol, 2005. **568**(Pt 2): p. 357-69.
74. Pyke, K.E., E.M. Dwyer, and M.E. Tschakovsky, *Impact of controlling shear rate on flow-mediated dilation responses in the brachial artery of humans.* J Appl Physiol, 2004. **97**(2): p. 499-508.
75. Parker, B.A., T.L. Trehearn, and J.R. Meendering, *Pick your Poiseuille: normalizing the shear stimulus in studies of flow-mediated dilation.* J Appl Physiol, 2009. **107**(4): p. 1357-9.
76. Watts, K., et al., *Exercise training normalizes vascular dysfunction and improves central adiposity in obese adolescents.* J Am Coll Cardiol, 2004. **43**(10): p. 1823-7.
77. Watts, K., et al., *Effects of exercise training on vascular function in obese children.* J Pediatr, 2004. **144**(5): p. 620-5.
78. Kelishadi, R., et al., *Association of changes in oxidative and proinflammatory states with changes in vascular function after a lifestyle modification trial among obese children.* Clin Chem, 2008. **54**(1): p. 147-53.
79. Kelly, A.S., et al., *Inflammation, insulin, and endothelial function in overweight children and adolescents: the role of exercise.* J Pediatr, 2004. **145**(6): p. 731-6.
80. Bax, M., et al., *Proposed definition and classification of cerebral palsy, April 2005.* Dev Med Child Neurol, 2005. **47**(8): p. 571-6.
81. Rosenbaum, P., et al., *A report: the definition and classification of cerebral palsy April 2006.* Dev Med Child Neurol Suppl, 2007. **109**: p. 8-14.

82. Delgado, M.R. and A.L. Albright, *Movement disorders in children: definitions, classifications, and grading systems.* J Child Neurol, 2003. **18 Suppl 1**: p. S1-8.
83. Gorter, J.W., *Rehabilitative therapies for the child with cerebral palsy: focus on family, function and fitness.* Minerva Pediatr, 2009. **61**(4): p. 425-40.
84. Rethlefsen, S.A., D.D. Ryan, and R.M. Kay, *Classification systems in cerebral palsy.* Orthop Clin North Am, 2010. **41**(4): p. 457-67.
85. Palisano, R., et al., *Development and reliability of a system to classify gross motor function in children with cerebral palsy.* Dev Med Child Neurol, 1997. **39**(4): p. 214-23.
86. Palisano, R., et al., *GMFCS-E&R Gross Motor Function Classification System expanded and revised.* 2007, CanChild Centre for Childhood Disability Research: Ontario (Canada).
87. Bjornson, K.F., et al., *Ambulatory physical activity performance in youth with cerebral palsy and youth who are developing typically.* Phys Ther, 2007. **87**(3): p. 248-57.
88. van Eck, M., et al., *Physical activity level and related factors in adolescents with cerebral palsy.* Pediatr Exerc Sci, 2008. **20**(1): p. 95-106.
89. Maher, C.A., et al., *Physical and sedentary activity in adolescents with cerebral palsy.* Dev Med Child Neurol, 2007. **49**(6): p. 450-7.
90. Schenker, R., W. Coster, and S. Parush, *Participation and activity performance of students with cerebral palsy within the school environment.* Disabil Rehabil, 2005. **27**(10): p. 539-52.
91. LaMonte, M.J., S.N. Blair, and T.S. Church, *Physical activity and diabetes prevention.* J Appl Physiol, 2005. **99**(3): p. 1205-13.
92. Blair, S.N., et al., *Changes in physical fitness and all-cause mortality. A prospective study of healthy and unhealthy men.* JAMA, 1995. **273**(14): p. 1093-8.
93. Manson, J.E., et al., *A prospective study of walking as compared with vigorous exercise in the prevention of coronary heart disease in women.* N Engl J Med, 1999. **341**(9): p. 650-8.
94. Lloyd-Jones, D., et al., *Heart disease and stroke statistics--2009 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee.* Circulation, 2009. **119**(3): p. e21-181.
95. Morris, C., et al., *Do the abilities of children with cerebral palsy explain their activities and participation?* Dev Med Child Neurol, 2006. **48**(12): p. 954-61.
96. Smits, D.W., et al., *Relationship between gross motor capacity and daily-life mobility in children with cerebral palsy.* Dev Med Child Neurol, 2010. **52**(3): p. e60-6.
97. Holsbeeke, L., et al., *Capacity, capability, and performance: different constructs or three of a kind?* Arch Phys Med Rehabil, 2009. **90**(5): p. 849-55.
98. Tieman, B.L., et al., *Gross motor capability and performance of mobility in children with cerebral palsy: a comparison across home, school, and outdoors/community settings.* Phys Ther, 2004. **84**(5): p. 419-29.
99. Ostensjo, S., et al., *Assessment of everyday functioning in young children with disabilities: an ICF-based analysis of concepts and content of the Pediatric*

Evaluation of Disability Inventory (PEDI). Disabil Rehabil, 2006. **28**(8): p. 489-504.
100. Palisano, R.J., et al., *Determinants of intensity of participation in leisure and recreational activities by children with cerebral palsy.* Dev Med Child Neurol, 2011. **53**(2): p. 142-9.
101. Verschuren, O., et al., *Exercise training program in children and adolescents with cerebral palsy: a randomized controlled trial.* Arch Pediatr Adolesc Med, 2007. **161**(11): p. 1075-81.
102. Andersen, L.B., et al., *Physical activity and clustered cardiovascular risk in children: a cross-sectional study (The European Youth Heart Study).* Lancet, 2006. **368**(9532): p. 299-304.
103. Thompson, A.M., et al., *Comparison of physical activity in male and female children: does maturation matter?* Med Sci Sports Exerc, 2003. **35**(10): p. 1684-90.
104. Reiner, A.M. and H.K. Bjarnason, *Growing up with CP: the effect of physical maturity on individuals with cerebral palsy.* Dev Med Child Neurol, 1999. **41(suppl 80)**(11).
105. Hallum, A., *Disability and the transition to adulthood: issues for the disabled child, the family, and the pediatrician.* Curr Probl Pediatr, 1995. **25**(1): p. 12-50.
106. Maltais, D.B., et al., *Physical activity level is associated with the O2 cost of walking in cerebral palsy.* Med Sci Sports Exerc, 2005. **37**(3): p. 347-53.

CHAPTER 2

BASELINE ASSESSMENT OF ARTERIAL STRUCTURE AND FUNCTION IN ADOLESCENTS WITH CEREBRAL PALSY

2.1 INTRODUCTION

Cerebral Palsy (CP) can be classified as a disorder of posture and movement with evidence of motor impairment seen in the first 18 months of life [1, 2]. CP is a non-progressive, chronic condition caused by a disturbance in the developing fetal or infant brain [1, 2], resulting in a wide variety of developmental difficulties. By definition, this condition manifests as a disorder in the development of gross motor function [3], affecting functional mobility over a lifespan [4]. An inverse relationship is often seen between functional limitations and social participation for both adolescents and adults with CP [5]. As such, youth with CP are less physically active than their typically developing peers [5-8]. Physical inactivity places young individuals with CP at a greater risk of developing a variety of secondary health complications [9], and is also a major controllable risk factor for cardiovascular disease (CVD) [10, 11].

It has been suggested that one mechanism by which physical activity (PA) exerts its protective effect on cardiovascular health is through positive effects on the endothelium [12], a single layer of cells responsible for the vasodilator response to increased conduit arterial flow. The flow-mediated dilation (FMD) assessment has been shown to be the most reproducible and least variable of the techniques used to measure endothelial function in children [13]. Endothelial dysfunction is considered an early and integral manifestation of atherosclerotic disease [14], which can be evident in the first

decade of life [15]. A strong relationship between low levels of PA and endothelial dysfunction has been well documented in children [16, 17], potentially predisposing individuals with CP to an increased risk of dysfunction. Endothelial function is an indicator of pre-clinical vascular disease and for youth with CP, may act as a marker of early changes in vessel function, indicative of future atherosclerotic risk [14].

Increased arterial stiffness is one of the key factors associated with CVD and to our knowledge, has yet to be assessed in this population. Pulse wave velocity (PWV) is a sensitive marker of arterial wall stiffness and subsequent marker of cardiovascular risk [18, 19]. In children, PWV is positively correlated with body mass index (BMI), waist circumference (WC) and percentage body fat, and negatively correlated with cardiorespiratory fitness and levels of PA [20]. Carotid artery distensibility and carotid artery intima-media thickness (IMT) are two additional indices of arterial health and their role in the development of CVD is widely accepted [21-23]. Increases in central arterial stiffness and carotid IMT have been observed in children with CVD risk factors such as familial hypercholesterolemia [24], type I diabetes [25, 26] and obesity [27, 28].

Strong relationships between cardiovascular risk factors identified in childhood and adolescence and the progression of atherosclerosis in adulthood are emerging [29-31]. Importantly, consistent, positive effects of habitual PA on vessel health have been demonstrated [20, 32]. Habitual PA from a young age has been shown to positively attenuate the progression of atherosclerosis in adulthood [33]. Low levels of PA in childhood (3-18 years) are directly, and inversely associated with accelerated IMT progression in adulthood [33]. Specifically, increased exposure to CVD risk factors in

childhood have been shown to predict increases in IMT up to two decades later [29, 31]. It has been suggested that atherosclerosis prevention by means of lifestyle modification could be effective when initiated in childhood [33]. Measuring indices of arterial stiffness and endothelial function are therefore important in this young, clinical population in order to identify potential compromises to their vascular health and attenuate future CVD risk at the earliest stage possible.

It is well recognized that the declines in PA in youth with CP are often dependent on their functional capabilities [8]; however, a variety of factors influence social participation, patterns of sedentary behaviour and activity levels across all levels of the Expanded and Revised Gross Motor Function Classification System (GMFCS-E&R) [34]. The GMFCS-E&R is a simple method used to classify children with CP (infant-18 years) based on their functional ability and limitations in performing meaningful activities in daily life [35]. Even in the highest functioning classifications of individuals with CP (levels I and II), a gap between capability and performance exists [36], which can be seen in school-age children as young as 5-9 years old [37].

Adolescents with CP are at a higher risk of leading sedentary lifestyles in comparison to age and sex matched controls [5, 38, 39]. The potential consequences of this sedentary behaviour on specific parameters of vascular health and CVD risk in a young population of individuals with CP have yet to be examined. To our knowledge, there is no assessment of the potential impacts of this physical disability and impaired levels of habitual PA on the vessel health in youth with CP. Therefore, the purpose of this study is to provide normative baseline measures of vascular function (endothelial

function, PWV, and carotid distensibility) and structure (carotid IMT) in adolescents with GMFCS levels I-II CP and make comparisons with their typically developing peers. We hypothesize that youth with CP (GMFCS levels I-II) will have decreased levels of PA and decreased endothelial function and increased stiffness compared to an age- and gender-matched control sample.

2.2 METHODS

2.2.1 *Participants*

Twenty-two adolescents (9-16 yrs) were recruited from the Hamilton and surrounding area. Eleven individuals with CP (8 boys) with mean ± SD age of 13.2 ± 2.1 yr were recruited from the Spasticity Clinic and Teen Transition clinic at McMaster Children's Hospital with approval from the Hamilton Health Sciences and McMaster University Faculty of Health Sciences Research Ethics Board. Subjects were chronological age- and gender-matched to a healthy control group with a mean age of 12.4 ± 2.3 yr. Control subjects were studied without specific exclusion criteria and all were healthy, with no known cardiovascular or metabolic conditions. Inclusion criteria for the CP group included a classification of either a level I or II (GMFCS) [35], identified by therapists or a physician at the spasticity clinic at McMaster Children's Hospital. All CP subjects were ambulatory without assistance or ambulatory with lower leg braces (level I n=7, level II n=4). Experimental procedures were explained to participants and their guardians prior to obtaining written and verbal informed consent / assent from the parent/guardian and participant respectively.

2.2.2 *Study design*

This study employed a cross-sectional design to characterize the differences in specific measures of vascular structure and function between children with CP and healthy controls. All measures were noninvasive, and took place in a quiet, temperature-controlled room (23º ± 1ºC) with the participant in a supine position. All subjects were instructed to abstain from vigorous PA 24 hours prior, and were tested 4 hours post-prandial with specific attention paid to caffeine and Vitamin C consumption in the previous 12 hours [40].

2.2.3 *The Exercise Questionnaire, anthropometric and resting hemodynamic measures*

Habitual PA patterns were assessed using the Exercise Questionnaire adopted from Brunton et al., used in a longitudinal study describing exercise participation of adolescents with CP [41]. This recall questionnaire was developed based on a design by Sallis *et al*. (1985) [42], and provides information regarding the frequency, duration and intensity of PA performed in the previous week. Participants completed the questionnaire with the assistance of the parent or guardian and were asked to 1) identify which activity they completed in the previous week, 2) indicate the number of times they performed each activity, 3) indicate the time spent performing each bout of the activity, and 4) classify the intensity of each activity based on a descriptive choice of either “light”, “medium” or “hard” (Appendix B). The questionnaire provided the participant with an adjusted list of activities to choose from based on the Previous Day Physical Activity Record [43]. The adjusted list of activities was developed through consultation with experts in the field and pilot testing confirmed content validity in this clinical population

[41]. Reliability of the Exercise Questionnaire has yet to be confirmed; however, the current study employed this questionnaire in order to provide a tool that was specific to youth with CP and easily transferable to a group of control subjects.

Anthropometric measurements

Sitting and standing height (cm), were measured to the nearest mm without shoes and in light clothing. Body mass was measured to the nearest 0.1kg using a digital scale, and body mass index (BMI) was calculated. Waist circumference (WC), an index of total abdominal fat, was measured 4 cm above the umbilicus at the end of a normal expiration [44] and the average of the measurements was reported. Two measurements were taken for each variable with a third required if a differences greater than 4mm for height and WC, and 0.4kg for weight [45, 46]. For height and weight, the average of the two measurements was reported, and the median value was reported if three measurements were obtained [47]. Waist-to-height ratio (WHR) was calculated as the WC divided by the height (cm).

As a marker of biological maturity, each individuals' age at peak height velocity (APHV) was calculated using a gender specific equation taking into account specific anthropometric variables [48]. This method has been shown to be accurate within an error of ± 1 year and is deemed sufficient for maturational classification in an adolescent population [48]. The APHV is used as an accurate representation of the maximum growth during adolescence [48] and is a valid tool when calculated using standard anthropometric variables such as chronological age, stature, sitting height, and body mass [46]. Time from peak height velocity (TPHV), an indicator of biological age was

calculated by subtracting the chronological age at time of testing from the predicted chronological age at PHV (http://taurus.usask.ca/growthutility/phv_ui.cfm?type=1) [48]

Resting heart rate and blood pressure

Testing sessions began with 10 min. of supine rest to ensure representative resting measurements prior to the commencement of the vascular assessment [49]. Continuous heart rate via a single-lead electrocardiograph and brachial blood pressure (BP) measurements via an automated applanation tonometer with oscillometric cuff calibration (model CBM-7000; Colin Medical Instruments, San Antonio, TX) were collected. All signals (including those described below) were acquired simultaneously using a commercially available data acquisition system (Powerlab model ML795, ADInstruments, Colorado Springs, USA) and software program (Labchart 7; ADInstruments Inc., Colorado Springs, CO, USA) [40, 50]. At the end of the vascular assessment, four measurements of seated brachial artery systolic (SBP), diastolic (DBP), pulse (PP) and mean arterial (MAP) pressures were obtained using an automated sphygmomanometer (Dinamap Pro 100, Critikon LCC, Tampa, Fla.). The first measurement was used for calibration purposes only and the average of the preceding three measures were reported [51].

2.2.4 *Vascular assessment*

Pulse Wave Velocity

Baseline measurements of pulse wave velocity (PWV) were acquired through electrocardiography and photoplethysmography. Both central and peripheral PWV were determined from 20 continuous heart cycles using the equation [19]:

Equation 1 $$PWV = \frac{D}{\Delta t}$$

where D is the distance between measurement sites, and Δt is the pulse transit time. Pulse transit time (PTT) is the calculated difference between the arrival time of the "foot" of the pressure waveform at the proximal location and that at the distal location. Arterial waveforms at the common carotid, femoral and dorsalis pedis arteries were collected using photoplethysmograph (PPG) sensors (IR Plethysmograph; Model MLT1020PPG; ADInstruments, Colorado Springs) on the right side of the body. PPG signals were band-pass filtered (5-30Hz) to remove variations in pressure that were not indicative of physiological changes. The lower (≤ 5Hz) and higher frequencies (≥ 30Hz) were removed in order to assist in the detection of the foot of each waveform. The foot of each waveform is identified as the minimum value of the digitally filtered signal [52] corresponds to the end of diastole, when the steep rise in the wave begins and appears as a sharp inflection of the original signal [53].

Central PTT was determined using the subtraction method [54]. The pulse transit time between ventricular depolarization (R spike of ECG signal) and the common carotid artery was subtracted from the time delay between ventricular depolarization and the arrival of the foot of the femoral artery pulse wave [55]. Similarly, central PWV path length was calculated by subtracting the surface distance between the sternal notch and the carotid PPG placement from that of the sternal notch and the femoral PPG placement. Peripheral pulse transit time was determined as the time delay between the arrival of the femoral artery pulse wave and the dorsalis pedis artery pulse wave [40], with the path

length measured as the distance between these two sites. Anthropometric measuring tape was used to measure the straight-line distances between skin sites (sternal notch to the placement of each PPG sensor) along the surface of the body.

Carotid Distensibility and intima-media thickness

Direct measurements of carotid distensibility were acquired using a combination of high-resolution, two-dimensional, B-mode ultrasound images (System FiVe; GE Medical Systems, Horten, Norway) and applanation tonometry (model SPT-301; Millar Instruments, Houston, TX, USA). With the subject in a supine position, the ultrasound probe was applied longitudinally to the surface of the skin and digital images of the left common carotid artery were collected at a frame rate of 11 frames/second. At the same time, continuous pressure waveforms (representative of carotid artery blood pressure) were collected at the right common carotid artery using a hand-held tonometer (model SPT-301; Millar Instruments, Houston, TX, USA). The tonometer was positioned over the point of greatest pulsation and held in a fixed position for ten consecutive heart cycles. Adjustments were made so that optimal ultrasound images and pressure waveforms were obtained before the 10 beat segment of data was saved for later off-line analysis.

Applanation tonometry is sensitive to hold down pressure and generates relative pressure values [40]. Thus, absolute carotid artery systolic blood pressures were calculated by calibrating the relative values (acquired using applanation tonometry) to the calibrated brachial artery blood pressures acquired using the automated applanation tonometer and oscillometric cuff device (Colin model, CMB-7000). The latter combines

blood pressures from a brachial cuff and a wrist sensor to determine beat-to-beat brachial blood pressure [40, 50]. When an individual is in a supine position, the diastolic and mean arterial pressures are assumed to be consistent in all conduit arteries [56]; therefore, the diastolic and mean arterial brachial blood pressures were equated to the minimum and mean carotid artery blood pressures obtained at the carotid artery. Using this relationship, the carotid artery systolic blood pressure was then extrapolated from the pressure waveforms [57, 58].

Ultrasound images were stored offline in Digital Image and Communications in Medicine (DICOM) format for later analysis using a semi-automated edge tracking system [AMS (Artery Measurement System) Image and Data Analysis. Tomas Gustavsson, gustav@alumni.chalmers.se]. This software was used to detect vessel diameters within specific regions of interest (ROI) according to contrasting brightness intensities between the walls and lumen of the vessel [59]. The investigator is able to scan each frame to ensure proper placement of the measurement markers and manually adjust the placement of markers if needed. In each frame, carotid artery (minimum, mean and maximum) lumen diameters were calculated from roughly 100 measurement markers along the vessel wall within the ROI, for a total of 110 000 measures in a 10 heart cycle data sample. Distensibility was calculated using the equation [19]:

Equation 2 $$Distensibility = \frac{\Pi\left(\frac{d_{\max}}{2}\right)^2 - \Pi\left(\frac{d_{\min}}{2}\right)^2}{\Pi\left(\frac{d_{\max}}{2}\right)^2 \times PP}$$

where d_{max} is the maximum diameter, d_{min} is the minimum diameter, and PP is carotid pulse pressure, the change in pressure from DBP and SBP. The mean carotid diameter was calculated using the average of all diameters acquired throughout the ten heart cycles. The same software program and ultrasound images were used to calculate carotid intima-media thickness (IMT). Far-wall IMT was calculated from the proximal aspect of the intima to the proximal interface of the adventitia using the average of the ten frames corresponding to end-diastole (as depicted by the R spike on the ECG).

Flow-mediated dilation assessment

With the participant in the supine position, the left arm was positioned (roughly 80º from the torso) and immobilized so that an optimal image of the brachial artery could be obtained in a comfortable position [60]. A sphygmomanometric cuff was placed on the forearm, below the medial epicondyle [13] and remained deflated while baseline data were collected. B-mode ultrasound images of the left brachial artery were collected through two-dimensional grayscale ultrasound imaging using a 10MHz linear array probe (System FiVe; GE Medical Systems, Horten, Norway). All images were acquired at a frame rate of 11 frames per second and stored offline for later analysis. A baseline, longitudinal image of the brachial artery (3 consecutive cardiac cycles) was acquired ~5-10cm above the antecubital fossa by a single ultrasonographer. Visualization of both near and far wall lumen-to-vessel wall boundaries were obtained and images (depth and gain settings) were optimized according to clarity of the walls and contrast with the lumen of the vessel. Baseline measurements of blood velocity using pulsed wave Doppler ultrasound were collected for 1 min. Although ideal for imaging, a 90º angle of

insonation provides a Doppler velocity of 0 and is therefore adjusted in order to attain an optimal velocity profile (rather than angle) as angles ≤70º are still highly valued and accepted in the literature [49]. To ensure a more inclusive depiction of laminar blood flow [49], an intensity weighted sample volume (versus peak envelope) was attained. The gate width was therefore adjusted accordingly, to encompass the entire lumen while minimizing wall noise of the vessel.

To create the flow stimulus, the forearm cuff was instantaneously inflated to a standardized, supra-systolic pressure of 200mmHg to ensure arterial inflow occlusion and ischemia of downstream vessels and tissue [61]. An occlusion time of 5 min. with the cuff positioned distally to the area of FMD measurement has been shown to elicit an NO-dependent response [62-64] and variations to the nature of this stimulus will result in varying shear stress profiles and FMD responses [62, 63, 65, 66]. An appropriate cuff size was used to ensure complete occlusion and to minimize any discomfort associated with the sudden 'jerk' or potential movement of the cuff during inflation. The participant was also familiarized with the entire FMD protocol and provided with ample warning of the timing of cuff inflation and deflation.

Prior to cuff deflation, B-mode ultrasound images (of 3 consecutive heart cycles) were obtained at the 4 min. occlusion time point. The cuff was instantaneously deflated after 5 min. of occlusion, thereby inducing a high-flow state through the brachial artery. To capture this response, the first 30 sec. of hyperemic blood velocity signals were collected using pulsed-wave Doppler ultrasound, held at the same location of the B-mode longitudinal images and baseline velocity measures. The forward and reverse frequency

signals were processed by an external spectral analysis system (Neurovision 500M, Multigon Ind; Yonkers NY) and an intensity-weighted calculated mean was output into a data acquisition system (Powerlab model ML795). B-mode ultrasound images of the brachial artery were stored every 15 sec. from 30 sec. up to 3 min. post cuff deflation.

End-diastolic frames were extracted from each sequence of images using a DICOM editing software program (Sante DICOM Editor 3.1.13, Santesoft, Athens, Greece). The aforementioned software program (AMS) was used to detect the vessel diameters within a specific ROI for the three end-diastolic frames at each time point. The peak dilation of the vessel was established as the single largest diameter (mm) that occurred at a specific time point measured from 30 sec. to 3 min. after cuff release. From this data, the absolute FMD (mm) and relative FMD (%FMD) were calculated as follows [38]:

Equation 3 $$AbsoluteFMD = PeakDiameter(mm) - BaselineDiameter(mm)$$

Equation 4 $$\%FMD = \left(\frac{AbsoluteFMD}{BaselineDiameter} \right) \times 100\%$$

The magnitude of the shear stimulus is proportional to the FMD response [38, 67] and was quantified as the area under the curve (AUC) of the shear rate stimulus [49, 68]. The following equation was used to calculate shear rate (SR) for each participant [69]:

Equation 5 $$ShearRate = 8 \times \left(\frac{Velocity}{Diameter} \right)$$

where velocity represents the mean blood flow velocity of the velocity profile of the first 30 sec. post cuff release and the baseline brachial diameter (mm) is used for the internal artery diameter value. The area under the curve of the shear rate was calculated from the mean of the first point, using the trapezoid rule to obtain the area under the entire curve

(GraphPad Prism version 4.00 for Windows, GraphPad Software, San Diego California USA, www.graphpad.com). Relative FMD (%FMD) was normalized to the area under the entire SR curve and reported as $\%FMD/SR_{AUC}$.

Equation 6 $$NormalizedFMD = \left(\frac{\%FMD}{SR_{AUC}} \right)$$

This method of analysis provides values of absolute maximum dilation (mm), time to reach peak dilation (sec.) and a raw calculation of the SR stimulus (SR_{AUC}).

Reproducibility of Measures

The techniques used in the measurement of vascular structure and function (carotid distensibility, carotid IMT and PWV) have previously been employed in our laboratory in children and adolescents (2-16 years) [70] as well as adult populations [40, 50], and have demonstrated acceptable reliability. In the current study, day-to-day reliability of the FMD assessment (a measure that has yet to be tested in our laboratory in this population) was evaluated using coefficients of variation (CV), Pearson correlation coefficients (r) and paired sample t-tests. A subset of the study sample consented to return for a second visit (control n=8, CP n=4) at which time the FMD assessment was repeated.

2.2.5 *Statistical Analysis*

Statistical analyses were performed using SPSS Statistics, version 19.0 (SPSS, Inc., Chicago, IL). Results are expressed as the mean ± SD. Data distribution was initially examined for normality using the Shapiro-Wilk's test and homogeneity of variance using Levene's Test. Independent t-tests were used to compare baseline data between males

and females within each group; where no gender differences existed, males and females were pooled within groups for subsequent analysis. Independent t-tests were used to compare group differences in all vascular indices, anthropometric measures and levels of PA. Analyses of vascular indices were also completed with chronological age as a covariate.

The *light, medium* or *hard* intensities identified on the Exercise Questionnaire [41] were equated to *light*, *moderate* and *vigorous* intensities in order to make comparisons with the Canadian Physical Activity Guidelines. The time spent in moderate and vigorous intensity physical activity were summed for each participant. The percentages of subjects in each group meeting the daily recommendations of 60 minutes of moderate- to vigorous-intensity physical activity (420 min./ week) (http://www.csep.ca/english/view.asp?x=804, Canadian Society for Exercise Physiology, 2011) were reported.

Pearson's product-moment correlation coefficients between age, sex, BMI, physical activity, FMD, carotid distensibility, carotid IMT and PWV were determined. In addition, the effect of maturation was also assessed by determining the correlation between TPHV and each dependent variable; if a significant relationship existed, maturation was then considered a confounding variable. As previously described, the reliability of a subset of FMD data were assessed by calculating CV and Pearson correlations as well as paired samples t-tests between each day. Each CV was calculated using Microsoft Excel, 2008 (Version 12.2.9; Microsoft Corporation, Washington, DC, USA).

2.3 RESULTS

2.3.1 *Group Differences*

There were no gender differences within either group for any dependent variable and therefore males and females were pooled for subsequent analysis. Characteristics of the study population are described in Table 1. The control and CP group were of similar age, height, weight, WC, WHR, and BMI. There were no group differences in resting seated brachial systolic blood pressure, diastolic blood pressure, mean arterial pressure, or resting supine heart rate.

Outcomes of the flow-mediated dilation (FMD) assessment are reported in Table 2. It must be noted that one CP subject was removed from all FMD analysis due to inadequate ultrasound image quality of post-occlusion data. One control subject was also identified as an outlier (via box plot and a response greater than 2 SD above the mean) and removed from the analysis. Thus, all statistical analysis of endothelial function (Table 2) were performed with an n=10 in each group, with the exception of the pre-occlusion brachial diameters (n=11) as all pre-occlusion data remained acceptable for analysis. There were no differences between groups ($p>0.05$) in pre-occlusion brachial diameter (mm) or peak diameter (mm) reached during reactive hyperemia (Table 2). Trends for significantly greater absolute FMD and %FMD response in the CP group were seen; however, these were no longer apparent when the %FMD was normalized to the SR stimulus. There were no differences in the SR stimulus or time taken to reach peak diameter between groups (Table 2).

There were no differences in baseline measures of carotid distensibility, carotid IMT or baseline carotid diameter between groups (Table 3). One control subject was a significant outlier and removed from analysis of distensibility (CON, n=10) and one CP subject could not be included in the analysis of IMT due to insufficient clarity of the far wall IMT for proper identification (CP, n=10). No differences were seen in central or peripheral PWV or PTT between groups (Table 4). One individual from the control group could not be included in the analysis due to an arrhythmia that did not permit appropriate analysis of the PWV data (CON, n=10).

The total number of minutes/week spent at each intensity of PA is reported in Table 5. There were no group differences in the total number of minutes spent in light and moderate PA. The control group reported more weekly vigorous PA than the CP group (Fig. 1) with over 60% of individuals in the CP group reporting 0 minutes of total time spent performing vigorous physical activity in the previous week. Only 36% of the control and 27% of the CP group were meeting the Canadian Physical Activity Guidelines of 60 minutes of moderate-to vigorous-intensity PA per day. Furthermore, when total PA time/week was calculated (combining each intensity of PA), there were no significant differences between groups (CON: 4840 vs. CP: 4260 min/week) (Table 5, Fig.1 B).

2.3.2 *Pearson correlation coefficients*

As no significant differences existed between groups on any of the primary outcome variables or anthropometric measures, the control and CP groups were pooled for further analysis. Pearson correlation coefficients were used to assess the relationships

between the dependent variables (%FMD/SR_{AUC}, cPWV, pPWV, carotid IMT and carotid distensibility), anthropometric measures, and PA levels.

Pearson correlation coefficients revealed a moderate, negative correlation between %FMD and baseline brachial diameter (r=-0.508, p=0.022) (Fig. 2, A); however, when the FMD response was normalized to the SR stimulus the relationship was no longer significant (Fig. 2, B). No other significant correlations were seen for %FMD or %FMD/SR_{AUC} and any of the following variables.

A negative correlation was observed between carotid PP and carotid distensibility (r=-0.628, p=0.002) and carotid compliance (r=-0.615, p=0.003) (Fig. 3, A, B). A significant, negative correlation also existed between chronological age and compliance (r=-0.436, p=0.048) (Fig. 4, A). Central pulse wave velocity was positively correlated with chronological age (r=0.485 p=0.026) (Fig. 4, B) and carotid PP (r=0.559, p=0.008) (Fig. 5, A). A negative correlation was found between cPWV and BMI (r=-0.442, p=0.05) and WHR (r=-0.482, p=0.027) (Fig. 5, B, C). No further correlations between PA (light, moderate or vigorous intensity) and any of the dependent or independent variables were observed.

Other significant relationships

A positive correlation was observed between BMI and baseline carotid diameter (r=0.438, p=0.042) (Fig. 6, A) as well as a trend toward significance between BMI and baseline brachial diameter (r=0.411, p=0.064). Similarly, a positive relationship existed between BMI and the maximum diameter reached during reactive hyperemia (r=0.467, p=0.038) (Fig. 6, B). BMI and BMI percentile were also significantly, positively

correlated with SBP (r=0.489, p=0.021 and r=0.469, p=0.028 accordingly). Finally, trends for significance were seen between brachial PP and BMI (r=0.386, p=0.076) and BMI percentile (r=0.412, p=0.057).

ANCOVA – Age as a covariate

As a result of the significant relationships found between age and indices of vascular health (cPWV and compliance), a univariate analysis of variance was performed using age as a covariate. When making this adjustment, there was a trend for a significant group difference in cPWV (p=0.079), with the CP group having higher values than the controls. However, all other outcomes between groups remained unchanged and although related to certain indices of vascular health, age does not appear to be a contributing factor to the aforementioned reported results.

2.3.3 *Reliability of the FMD assessment*

FMD reliability was assessed using data from 12 repeat visits (CON, n=8, CP, n=4). Pearson correlations, paired-sample t-tests and coefficient of variations (CVs) were calculated to compare the reliability of $\%FMD/SR_{AUC}$ between visits. When comparing the group means between visits, a trend for a positive correlation was found (p=0.066). Paired-sample t-tests (and repeated measures ANOVA) revealed no significant difference ($p>0.05$) in $\%FMD/SR_{AUC}$ between visit 1 and visit 2, and the average coefficient of variation between visits was 46%.

2.4 DISCUSSION

Over time, decreased levels of PA are generally associated with impairments of vascular function and structure and increased cardiovascular risk. This becomes

particularly important when PA levels are limited by unmodifiable physical factors, such as the motor impairments associated with CP. Thus, it is important to conduct early vascular assessments in this at-risk population in order to determine any potential CV risk factors and the timing of manifestation. The primary findings of this study indicate that arterial function and structure in adolescents with CP (GMFCS level I –II) are not different from a healthy control group despite individuals with CP spending significantly less time performing vigorous PA in comparison to their typically developing peers. The groups were matched for age and gender with no significant differences observed in anthropometric measures as well as resting hemodynamic profiles (SBP, DBP, MAP, HR) (Table 1). There were no significant gender differences in the measured anthropometric variables in either group.

During adolescence, there is a large variance in somatic and biological growth between individuals of the same chronological age [71-73]. In the current study, biological maturity was assessed using calculations of PHV, a commonly used maturational benchmark in longitudinal studies of adolescence [74]. Both groups were similar in their chronological and biological age (as estimated through TPHV calculations) and there were no differences in their APHV (Table 1**)**. This indicates the groups were well matched in terms of maturation. Within each group, males had greater APHV values than females, suggesting that males take longer to reach this maturational benchmark, regardless of group (CON: ♂13.4 ± 0.7 vs. ♀ 12.2 ± 0.9 yrs. and CP: ♂ 14.5 ± 1.0 vs. ♀ 12.7 ± 1.4yrs). These values are representative of the average APHV for males and females (♂ 13.4 ± 1.0 yrs and ♀ 11.8 ± 0.9 yrs) [75] and represent a typically

developing adolescent population. Furthermore, both groups appeared healthy with respect to the number of individuals in each group possessing traditional cardiovascular and metabolic risk factors; specifically, elevated age- and gender-specific percentiles of SBP, DBP, BMI, WC.

This cross-sectional study is the first to characterize indices of vascular health in higher functioning youth with CP (13.2 ± 2.1 yrs) and make comparisons to a group of typically developing peers (12.4 ± 2.3 yrs). Children harbouring classic CV risk factors, including physical inactivity have been shown to exhibit impairments in vascular function and structure early in life and have an increased risk of premature atherosclerosis in adulthood [33]. The predictive power of persistent PA at a young age must not be underestimated [76]. It has been shown that levels of both PA and inactivity track significantly from adolescence (9 to 18 yrs) to young adulthood placing inactive children at an increased risk of becoming physically sedentary adults [76]. With evidence of physical inactivity being a significant precursor to CVD related death, and only moderate levels of fitness proving to have protective effects against the influence of traditional risk factors on mortality [78], the value of well established, healthy patterns of habitual PA in pediatric practice must not be overlooked. In a group of youth that may have increased susceptibility to physical inactivity, identifying any early alterations in vascular function and structure may assist in identifying preclinical vascular disease, allowing for intervention at the earliest stage possible.

In the current study, no significant differences were found between absolute changes of FMD (mm), relative changes in FMD (%FMD) or normalized values of FMD

(%FMD/SR_{AUC}) between the control and CP group. Similar baseline brachial vascular dimensions and resting hemodynamic profiles were also seen between groups thereby allowing for accurate comparisons of endothelial function independent of confounding baseline variables. No significant differences existed in the average SR stimulus between groups indicating similar ischemic-induced reactive hyperemia responses. There was also no significant correlation between chronological age or biological age and %FMD/SR_{AUC}, potentially indicating little room for differences in such a small age range.

No significant relationships were seen between %FMD/SR_{AUC} and any index of body composition including BMI, BMI percentile, WC or WHR. This is in line with previous studies that showed no relationship between FMD and body composition variables in men (16-49 years) but saw modest relationships between FMD and measures of segmental fat mass as measured by dual-energy X-ray absorptiometry (DEXA) scans in children aged 10 -11 years [79]. Differences with previous literature may simply be a function of the accuracy and sensitivity of different body composition techniques. The weak correlations observed in the current study between FMD and body composition raise interest for scaling techniques of FMD for body composition in children, however no standardized scaling approach has been adopted for youth at this point [79].

We noted a positive correlation between BMI and baseline carotid diameter ($p=0.042$), indicating a potential effect of body size on vessel diameter. This was supported by a trend toward significance between BMI and baseline brachial diameter ($p=0.064$). Correspondingly, a significant relationship was also found between BMI and maximum diameter reached during reactive hyperemia ($p=0.038$), which may also be a

function of body size as these individuals have larger vessels at baseline. These relationships do not infer relationships between body composition and endothelial function, as BMI, an insufficient diagnostic tool of level of adiposity, was calculated as modest predictor of body fatness [80] rather than a direct measurement, such as DEXA. No significant relationships were found between any anthropometric variable assessing body composition and FMD outcomes. Therefore, the above relationships may be independent of body composition and vessel function, and merely reflect the fact that individuals of greater mass may have larger vessels at rest, allowing them to reach a significantly greater absolute peak diameter in response to a shear stress.

In this study, the primary risk factor (for future cardiovascular health) of interest was level of PA, as measured using the Exercise Questionnaire [41]. No significant differences in levels of light or moderate intensity PA between groups were found. Interestingly, less than 50% of subjects in each group met the Canadian Physical Activity Guidelines (CON: 36%, CP: 27%). This raises concern for the endothelial health of both groups as the benefits of regular PA in youth are widely accepted. In a previous study assessing the relationship between habitual PA (as measured using the double labeled water approach) and brachial FMD in 5-10 year old children, a significant correlation was found ($r=0.39$, $p=0.007$), highlighting PA as the most influential variable in predicting the FMD response [12]. Similarly, these findings were extended when Hopkins *et al.*, 2009 demonstrated a significant correlation between levels of PA, cardiorespiratory fitness, anthropometric measurements and the normalized FMD response in 10-11 year old children [81]. This group reported that physical fitness, as assessed using an incremental

discontinuous treadmill-based exercise test, and levels of PA, as measured using Actigraph accelerometers, were lowest in the lowest %FMD and %FMD/SR_{AUC} tertile. These relationships between fitness, PA and FMD response were significant, and it was concluded that PA measurements were the best predictors of endothelial (dys)function in this young group [81]. These data support the concept that PA exerts its protective effect on CV health via the endothelium and draws attention to the role of lifestyle modifications; specifically increases in levels of habitual PA in pediatric practice. Therefore, a shift in the focus of prevention toward increasing PA time and intensity as a means of reducing CV risk in school aged children has been suggested.

Although time spent in light to moderate PA was similar between groups, a significantly greater amount of time was spent engaging in vigorous intensity PA in the control versus the CP group. Despite the higher amount of time spent in vigorous PA in the control group in this study, there were no group differences between any of the measured indices of vascular health, specifically endothelial function. It has been suggested that the strongest relationships between exercise interventions and enhanced endothelial function exist in groups with relatively impaired FMD *a priori* with the tightest correlations between PA and FMD response existing in the lowest tertiles of endothelial function [81]. Considering this, there is no reason to believe that the control subjects have experienced vascular dysfunction and would therefore be positively influenced by this increase in vigorous activity in comparison to the seemingly healthy, CP group. Levels of vigorous intensity PA in the control group may reflect acute bouts of

high intensity activity over the previous week rather than a continuous stimulus for endothelial adaptations.

Although no group differences were seen in any of the reported FMD measures (absolute, relative or normalized), the current study provides strong evidence for the use of this widely applicable, non-invasive method for testing endothelial function *in vivo*, in both healthy children and a clinical, pediatric population. In both populations, the test was well tolerated and widely applicable to all children within each group with no apparent discomfort or technical limitations. In a small number of instances, minor adaptations to the procedure were required for individuals in the CP group. These included steadying the child's hand or assisting the child in out stretching their arm in order to minimize aberrant, spastic movement, which proved to benefit the comfort of the participant as well as the quality of data collected. Nevertheless, this study emphasizes the feasibility of performing the FMD assessment in this population and encourages its use in future, longitudinal studies of adolescents with CP in all GMFCS classifications.

No significant differences between groups were found in cPWV or pPWV. These values were comparable to a previous study assessing PWV in a slightly younger group of healthy children (10.1 ± 0.3yrs) who showed very similar cPWV values (4.2 ± 0.4 m/s) [20] to those in both groups in the current study (Table 4). This indicates preserved arterial stiffness at this time point for both the control and CP group. Similarities in PWV between groups in this study may be reflective of similar levels of low intensity PA, as indicated by the same amount of time spent in light and moderate intensity PA as well as the same total time spent performing PA a week (Table 5, Fig. 1, A, B**)**. It has been

suggested that PTTs be reported rather than PWVs due to the potential error associated with measuring distances between sites in overweight or obese individuals. This in turn, may result in a systematic bias between associations of central adiposity and PWV [20]. Therefore, when reporting cPTT and pPTT, no significant group differences were seen (Table 5**)**; similarly, there were no correlations between BMI or WC and any parameter of PWV. When controlling for WC and other variables associated with changes in central adiposity (WHR, BMI) the above results did not change, indicating the calculations of PWV were not influenced by differing waist circumferences or body shapes.

In this entire sample, a strong, positive relationship was found between chronological age and cPWV and a negative correlation between age and compliance. These relationships are in agreement with the well-documented, age-associated increases in arterial wall stiffness in children reported as increases in cPWV from childhood through puberty [58, 82]. This increase in arterial wall stiffness with age is typically observed in males and females with no significant sex differences [82]. This association continues into adulthood in healthy men and women, independent of age-related increase in arterial blood pressure [83, 84]. Aerobic fitness has been shown to mitigate the stiffening of the arterial tree that accompanies normative aging in adults [84]. The exact mechanism associated with increasing arterial stiffness with age has yet to be identified. Changes in the composition of the arterial wall, growth factors and vascular smooth muscle cell hypertrophy are believed to be related [85, 86].

An increase in body size is accompanied by increases in arterial size and correlates with increases in arterial compliance. The current study did not show increases

in baseline diameter of the carotid artery with age, but did show significant positive correlations with BMI, BMI percentile and WC, (p=0.042, p=0.0035 and p=0.047s). This finding is likely a reflection of overall changes in body size similar to those associated with growth. However, the effects of age on intrinsic properties of the arterial wall counterbalance the effect of growth [87] as evidenced in this study by the positive association between chronological age and cPWV and the negative relationship between chronological age and compliance.

No group differences were found between carotid distensibility or carotid IMT. Throughout the lifespan, habitual PA has been shown to positively influence arterial distensibility [20, 32]. Age-related decreases in arterial distensibility and increases in stiffness have been reported [32]; however, increased levels of PA have been suggested to delay the age-dependent loss of arterial distensibility, in proportion to the amount and/or intensity of exercise [32, 88, 89]. Although there was no difference in distensibility between the CP and control group at this time, sufficient rationale is provided for this clinical group of adolescents to increase their levels of high intensity PA at an early stage and maintain these behaviours into adulthood in attempt to mitigate thee normative age-related changes.

Carotid IMT measurements were also similar between groups and were comparable to other control groups used in previous studies [27, 90]. Iannuzzi and colleagues 2004 [27] characterized the differences in carotid IMT between obese children and age-matched control subjects (6-14 years) and showed a significantly greater IMT in the obese group in comparison to the healthy controls (0.55 ± 0.08mm vs. 0.49 ±

0.09mm). The carotid IMT of the obese children was approximately 24% and 25% greater than the carotid IMT of the CP and control group in the current study, suggesting healthy vascular structure in both groups in the current study.

The negative correlation observed between carotid PP and carotid compliance ($r=-0.615$, $p=0.003$) and carotid PP and distensibility ($r=-0.628$, $p=0.002$) is suggestive of a relationship between the pressure exerted on the walls of the vessel and its mechanical properties. These findings correspond to those seen in a pediatric population with end stage renal disease (ESRD) a "nonclassic" cardiovascular risk factor [91] as well as children classified as severely obese [17]. In both studies, these children exhibit alterations in carotid artery mechanical properties characterized by decreased compliance and distensibility with increased incremental elastic modulus (the theoretical pressure increment required for 100% stretch from resting diameter) [19] when compared to a healthy control group. Significantly higher wall stress as indicated by an increased PP were seen in both groups with this increase also associated with an increased PWV in the ESRD group. These findings are in line with the current study that found a positive correlation between carotid PP and cPWV ($r=0.559$, $p=0.008$) supporting the idea that an increase in the pressure experienced by central vessels is directly related to the mechanical properties of the arterial wall as reflected by the stiffness of that vessel.

Furthermore, Aggoun and colleagues showed that increases in PP at the carotid site also correlated positively with the carotid IMT ($r=0.63$, $p<0.01$) indicating negative structural adaptations to increasing wall stress on the intima-media interface. However, this may take a longer time to adapt such structural changes, as similar relationships

between carotid PP and carotid IMT were not seen in the control or CP group in the current study. Both studies also found lower endothelium-dependent function in their specified populations in comparison to healthy controls, indicating a potential relationship between functional measures of vascular health independent of mechanical changes in arterial stiffness. Regardless, the aforementioned findings of Aggoun *et al*., 2000 and Tounian *et al.,* 2001 strongly support the association between increasing wall stress and changes in the mechanical properties of the vessel wall as reflected by declines in carotid compliance, distensibility and increases in cPWV in children.

In the current study, only 27% of participants in the CP group were meeting the Canadian Physical Activity Guidelines of 60 minutes of moderate-to vigorous-intensity PA each day. This is similar to the findings of Brunton and colleagues, 2010 who also showed that both genders were participating in some activity, but not enough to meet Canadian guidelines [41]. This raises concern for these individuals as levels of both physical activity and inactivity track significantly from adolescence (12 to 18 yrs) to young adulthood [77] placing hypoactive children at an increased risk of becoming physically sedentary adults [76].

A study assessing walking performance activity as measured by a 5-day sample of StepWatch monitor data confirmed that children who were developing typically would be more active on a daily basis than youth with CP. However, this appeared to be a function of mobility capabilities as the highest functioning youth (GMFCS levels I) appeared to be just as active as their typically developing peers [6]. Daily walking activity may be classified as light to moderate PA in our sample and has been shown to decrease as

functional level decreases [6]. Since our sample were still very mobile and highly functioning, a group difference in light and moderate PA was not seen. As functional level decreases (level II and III), decreased variability of walking intensity and time spent at high intensity PA occurs [6]; accordingly, this may account for our differences in time spent in high intensity PA between our control and CP group.

Individuals in the CP group spent significantly less time participating in vigorous PA in comparison to their typically developing peers**.** Although ambulatory, these children may not be participating in activities of high enough intensity to reap the benefits associated with habitual vigorous PA. This presents an opportunity for clinicians and therapists to establish appropriate intervention programs and design health promotion initiatives that focus on the intensity, in addition to the volume, of PA. A shift in focus from improving the ability to walk or perform functional activities, often the primary therapeutic goals for children with CP [92], may be required to establish more vigorous PA patterns in the already highly mobile group of individuals with CP.

With normative aging in healthy youth, van de Laar *et al*., 2010, report a shift in increasingly more time spent in light-to-moderate levels of PA beginning in adolescence, with less time spent in vigorous habitual PA. The latter has specifically identified vigorous-intensity PA as the PA modality that has the greatest beneficial impacts on carotid arterial stiffness as well as on a number of CVD risk factors [93]. Furthermore, maintenance of relatively higher levels of vigorous-intensity activities between adolescence and young adulthood is associated with lower levels of arterial stiffness later in life. This is highlighted by the fact that even modest increases in PA are associated

with the prevention of arterial stiffness in early adulthood [93, 94]. In an attempt to attenuate the age-associated increases in arterial stiffness and critical decline in habitual vigorous-intensity PA during adolescence, one should promote high intensity PA as a tool to prevent detrimental CV outcomes later in life.

Several studies have confirmed the belief that boys are more active than girls [95] with age-associated declines in PA levels occurring in both genders [96-98]. Neither of these findings was supported in the current study. No significant difference in levels of PA were found between genders and no significant relationships were found between chronological age and light, moderate or vigorous intensity PA for the entire sample ($p=0.248$, $p=0.461$, $p=0.228$). Furthermore, no significant differences between groups were found when total PA time (including each intensity of PA) was calculated, and when pooled, total time was not related to chronological or biological age. A larger sample size with increased numbers of PA reports may be needed to highlight this association. There is potential for error in the PA questionnaire at is it may only provide a snapshot in time, which may not accurately reflect overall habitual PA patterns for each individual. Also, the overall minimal activity levels seen in each participant may have been be too small to deduce significant relationships.

There was however, a significant negative correlation between total amount of PA (min./week) and WC over the entire sample. This indicates a relationship between levels of truncal fat, which includes the type of body fat related to metabolic cardiovascular risk factors, and physical inactivity [99]. Therefore, highlighting additional consequences of physical inactivity and its role as a contributor to CVD.

As previously mentioned, an inherent error of using the 7-day recall questionnaire is the reliability and validity of the tool. It is well recognized that limitations exist when using a self-reported questionnaire. Our findings of no significant relationship between the total amount of PA and arterial compliance in 9-16 year old children are contrary to our hypothesis, but similar to those found by Reed and colleagues [100]. This group also used a 7-day recall questionnaire and found no significant relationship between the total amount of PA and arterial compliance in a group of 9-11 year old children. It is unknown whether the discrepancies in the current results and those previously shown actually represent a weak relationship between vascular health and PA levels in our sample, or are in part, a result of inaccuracies of the mode of PA measurement. This may be attributed to the inherent difficulty that comes with using a questionnaire in young populations. Ideally, accelerometer data should be collected in this population as it has been shown to be a feasible method of acquiring PA data in this young, clinical population (unpublished results). However, this was not possible in the current study.

2.4.1 *Limitations*

Although the FMD methodology used in the current study is relatively straightforward and noninvasive, limitations to the procedure vary, and encompass both technical and interpretive challenges. Data are largely dependent on the quality of the ultrasound images, requiring a skilled investigator, appropriate equipment, and analysis software. The current results are limited to highly functioning, ambulatory (with / without a lower leg brace) individuals with CP and their typically developing peers. It is difficult to say if these results are applicable to prepubertal or postpubertal individuals as

it can be assumed a mixed sample was represented. In this investigation, we did not control for or assess diet, vitamin ingestion or blood-borne CVD markers and therefore we cannot account for the contribution of these factors in any changes in vascular function.

2.4.2 *Future Directions*

There were no correlations seen between levels of PA (at light, moderate or vigorous intensity) and FMD outcome in either group, indicating a disconnect between levels of PA and endothelial function in this cohort of children. This may be due to the fact that at such a young age, the minimal stimulus of low levels of PA in both groups may be insufficient to elicit changes in the vasculature or establish a relationship with measures of endothelial function. However, long term preservation of minimal levels of low intensity PA may have deleterious consequences on vascular function in both groups later in life and warrants further, longitudinal investigation. Similarly, the assessment of individuals with CP, classified as levels III-V GMFCS may also be of interest as inclusion of individuals with a lower functional capacity may highlight group differences. The use of an objective measure of PA performance in addition to the Exercise Questionnaire is also of key importance.

2.4.3 *Conclusion*

Although no differences in vascular structure or function between youth (9-16 years) with CP and typically developing peers were observed in the current study, the establishment of techniques to assess arterial health in youth with CP is critically important for determining future CV risk in this clinical population. This study confirms

the feasibility of the use of these vascular assessment techniques in this population and presents potential for future, longitudinal assessments of individuals with CP across all levels of GMFCS classification. Each measurement of cardiovascular health was well tolerated and widely accepted by both participant and parent/guardian. These data illustrate that youth with CP maintain levels of lower intensity PA similar to their healthy counterparts and do not illustrate impairments in vascular function or structure. However, the consequences of significantly decreased amounts of time spent in vigorous PA for this group, at this time and potentially into adulthood, remain unknown. It may be of interest to assess whether vessel health is compromised in youth with decreased levels of functioning such as those in GMFCS levels III-V. Identifying these parameters may act as a tool for risk stratification in this population, thereby permitting identification of children who would benefit most from intensified physical activity and/or exercise interventions.

2.5 References

1. Bax, M., et al., *Proposed definition and classification of cerebral palsy, April 2005.* Dev Med Child Neurol, 2005. **47**(8): p. 571-6.
2. Rosenbaum, P., et al., *A report: the definition and classification of cerebral palsy April 2006.* Dev Med Child Neurol Suppl, 2007. **109**: p. 8-14.
3. Rosenbaum, P.L., et al., *Prognosis for gross motor function in cerebral palsy: creation of motor development curves.* JAMA, 2002. **288**(11): p. 1357-63.
4. Gorter, J.W., *Rehabilitative therapies for the child with cerebral palsy: focus on family, function and fitness.* Minerva Pediatr, 2009. **61**(4): p. 425-40.
5. Schenker, R., W. Coster, and S. Parush, *Participation and activity performance of students with cerebral palsy within the school environment.* Disabil Rehabil, 2005. **27**(10): p. 539-52.
6. Bjornson, K.F., et al., *Ambulatory physical activity performance in youth with cerebral palsy and youth who are developing typically.* Phys Ther, 2007. **87**(3): p. 248-57.
7. van Eck, M., et al., *Physical activity level and related factors in adolescents with cerebral palsy.* Pediatr Exerc Sci, 2008. **20**(1): p. 95-106.
8. Maher, C.A., et al., *Physical and sedentary activity in adolescents with cerebral palsy.* Dev Med Child Neurol, 2007. **49**(6): p. 450-7.
9. LaMonte, M.J., S.N. Blair, and T.S. Church, *Physical activity and diabetes prevention.* J Appl Physiol, 2005. **99**(3): p. 1205-13.
10. Blair, S.N., et al., *Changes in physical fitness and all-cause mortality. A prospective study of healthy and unhealthy men.* JAMA, 1995. **273**(14): p. 1093-8.
11. Manson, J.E., et al., *A prospective study of walking as compared with vigorous exercise in the prevention of coronary heart disease in women.* N Engl J Med, 1999. **341**(9): p. 650-8.
12. Abbott, R.A., M.A. Harkness, and P.S. Davies, *Correlation of habitual physical activity levels with flow-mediated dilation of the brachial artery in 5-10 year old children.* Atherosclerosis, 2002. **160**(1): p. 233-9.
13. Donald, A.E., et al., *Non-invasive assessment of endothelial function: which technique?* J Am Coll Cardiol, 2006. **48**(9): p. 1846-50.
14. Vita, J.A. and J.F. Keaney, Jr., *Endothelial function: a barometer for cardiovascular risk?* Circulation, 2002. **106**(6): p. 640-2.
15. Stary, H.C., *Evolution and progression of atherosclerotic lesions in coronary arteries of children and young adults.* Arteriosclerosis, 1989. **9**(1 Suppl): p. I19-32.
16. Pahkala, K., et al., *Vascular endothelial function and leisure-time physical activity in adolescents.* Circulation, 2008. **118**(23): p. 2353-9.
17. Tounian, P., et al., *Presence of increased stiffness of the common carotid artery and endothelial dysfunction in severely obese children: a prospective study.* Lancet, 2001. **358**(9291): p. 1400-4.

18. Laurent, S., et al., *Aortic stiffness is an independent predictor of all-cause and cardiovascular mortality in hypertensive patients.* Hypertension, 2001. **37**(5): p. 1236-41.
19. O'Rourke, M.F., et al., *Clinical applications of arterial stiffness; definitions and reference values.* Am J Hypertens, 2002. **15**(5): p. 426-44.
20. Sakuragi, S., et al., *Influence of adiposity and physical activity on arterial stiffness in healthy children: the lifestyle of our kids study.* Hypertension, 2009. **53**(4): p. 611-6.
21. Salonen, J.T. and R. Salonen, *Ultrasonographically assessed carotid morphology and the risk of coronary heart disease.* Arteriosclero Thromb, 1991. **11**(5): p. 1245-1249.
22. O'Leary, D.H., et al., *Carotid-artery intima and media thickness as a risk factor for myocardial infarction and stroke in older adults.* N Engl J Med, 1999. **340**(1): p. 14-22.
23. Tsivgoulis, G., et al., *Common carotid arterial stiffness and the risk of ischaemic stroke.* Eur J Neurol, 2006. **13**(5): p. 475-481.
24. Virkola, K., et al., *Cholesterol and carotid artery wall in children and adolescents with familial hypercholesterolaemia: a controlled study by ultrasound.* Acta Paediatr, 1997. **86**(11): p. 1203-7.
25. Heilman, K., et al., *Arterial stiffness, carotid artery intima-media thickness and plasma myeloperoxidase level in children with type 1 diabetes.* Diabetes Res Clin Pract, 2009. **84**(2): p. 168-73.
26. Jarvisalo, M.J., et al., *Endothelial dysfunction and increased arterial intima-media thickness in children with type 1 diabetes.* Circulation, 2004. **109**(14): p. 1750-5.
27. Iannuzzi, A., et al., *Increased carotid intima-media thickness and stiffness in obese children.* Diabetes Care, 2004. **27**(10): p. 2506-8.
28. Zebekakis, P.E., et al., *Obesity is associated with increased arterial stiffness from adolescence until old age.* J Hypertens, 2005. **23**(10): p. 1839-46.
29. Raitakari, O.T., et al., *Cardiovascular risk factors in childhood and carotid artery intima-media thickness in adulthood: the Cardiovascular Risk in Young Finns Study.* JAMA, 2003. **290**(17): p. 2277-83.
30. Juonala, M., et al., *Risk factors identified in childhood and decreased carotid artery elasticity in adulthood: the Cardiovascular Risk in Young Finns Study.* Circulation, 2005. **112**(10): p. 1486-93.
31. Davis, P.H., et al., *Carotid intimal-medial thickness is related to cardiovascular risk factors measured from childhood through middle age: The Muscatine Study.* Circulation, 2001. **104**(23): p. 2815-9.
32. Seals, D.R., et al., *Habitual exercise and arterial aging.* J Appl Physiol, 2008. **105**(4): p. 1323-32.
33. Juonala, M., et al., *Life-time risk factors and progression of carotid atherosclerosis in young adults: the Cardiovascular Risk in Young Finns study.* Eur Heart J, 2010. **31**(14): p. 1745-51.

34. Palisano, R., et al., *GMFCS-E&R Gross Motor Function Classification System expanded and revised.* 2007, CanChild Centre for Childhood Disability Research: Ontario (Canada).
35. Palisano, R., et al., *Development and reliability of a system to classify gross motor function in children with cerebral palsy.* Dev Med Child Neurol, 1997. **39**(4): p. 214-23.
36. Palisano, R.J., W.P. Copeland, and B.E. Galuppi, *Performance of physical activities by adolescents with cerebral palsy.* Phys Ther, 2007. **87**(1): p. 77-87.
37. Smits, D.W., et al., *Development of daily activities in school-age children with cerebral palsy.* Res Dev Disabil, 2011. **32**(1): p. 222-34.
38. Anderson, T.J., et al., *Close relation of endothelial function in the human coronary and peripheral circulations.* J Am Coll Cardiol, 1995. **26**(5): p. 1235-41.
39. Maltais, D.B., et al., *Physical activity level is associated with the O2 cost of walking in cerebral palsy.* Med Sci Sports Exerc, 2005. **37**(3): p. 347-53.
40. Rakobowchuk, M., et al., *Effect of acute sprint interval exercise on central and peripheral artery distensibility in young healthy males.* Eur J Appl Physiol, 2009. **105**(5): p. 787-95.
41. Brunton, L.K. and D.J. Bartlett, *Description of exercise participation of adolescents with cerebral palsy across a 4-year period.* Pediatr Phys Ther, 2010. **22**(2): p. 180-7.
42. Sallis, J.F., et al., *Physical activity assessment methodology in the Five-City Project.* Am J Epidemiol, 1985. **121**(1): p. 91-106.
43. Weston, A.T., R. Petosa, and R.R. Pate, *Validation of an instrument for measurement of physical activity in youth.* Med Sci Sports Exerc, 1997. **29**(1): p. 138-43.
44. Rudolf, M.C., J. Walker, and T.J. Cole, *What is the best way to measure waist circumference?* Int J Pediatr Obes, 2007. **2**(1): p. 58-61.
45. Bailey, D.A., *The Saskatchewan Pediatric Bone Mineral Accrual Study: bone mineral acquisition during the growing years.* Int J Sports Med, 1997. **18 Suppl 3**: p. S191-4.
46. Sherar, L.B., et al., *Prediction of adult height using maturity-based cumulative height velocity curves.* J Pediatr, 2005. **147**(4): p. 508-14.
47. Ross, W. and M.-J. MJ, *Kinanthropometry*, in *Physiological Testing of the High-Performance Athlete*. 1991, Human Kinetics Books: Champaign, Illinois. p. 223-308.
48. Mirwald, R.L., et al., *An assessment of maturity from anthropometric measurements.* Med Sci Sports Exerc, 2002. **34**(4): p. 689-94.
49. Harris, R.A., et al., *Ultrasound assessment of flow-mediated dilation.* Hypertension, 2010. **55**(5): p. 1075-85.
50. Rakobowchuk, M., et al., *Sprint interval and traditional endurance training induce similar improvements in peripheral arterial stiffness and flow-mediated dilation in healthy humans.* Am J Physiol Regul Integr Comp Physiol, 2008. **295**(1): p. R236-42.

51. Pickering, T.G., et al., *Recommendations for blood pressure measurement in humans: an AHA scientific statement from the Council on High Blood Pressure Research Professional and Public Education Subcommittee.* J Clin Hypertens (Greenwich), 2005. **7**(2): p. 102-9.
52. Munakata, R., et al., *Total synthesis of (+)-macquarimicin A.* J Am Chem Soc, 2003. **125**(48): p. 14722-3.
53. Nichols, W.W. and M.F. O'Rourke, *McDonald's Blood Flow in Arteries: Theoretical, Experimental and Clinical Principles. 5th ed.* 2005: A Hodder Arnold Publication.
54. Weber, T., et al., *Noninvasive determination of carotid-femoral pulse wave velocity depends critically on assessment of travel distance: a comparison with invasive measurement.* J Hypertens, 2009. **27**(8): p. 1624-30.
55. Laurent, S., et al., *Expert consensus document on arterial stiffness: methodological issues and clinical applications.* Eur Heart J, 2006. **27**(21): p. 2588-605.
56. Nichols, W.W., et al., *McDonald's Blood Flow in Arteries: Theoretic, Experimental, and Clinical Principles*. 1998, London: Oxford University Press.
57. Kelly, R. and D. Fitchett, *Noninvasive determination of aortic input impedance and external left ventricular power output: a validation and repeatability study of a new technique.* J. Am. Coll. Cardiol., 1992. **20**(4): p. 952-963.
58. Fernhall, B. and S. Agiovlasitis, *Arterial function in youth: window into cardiovascular risk.* J Appl Physiol, 2008. **105**(1): p. 325-33.
59. Woodman, R.J., et al., *Improved analysis of brachial artery ultrasound using a novel edge-detection software system.* J Appl Physiol, 2001. **91**(2): p. 929-37.
60. Atkinson, G., et al., *Is the ratio of flow-mediated dilation and shear rate a statistically sound approach to normalization in cross-sectional studies on endothelial function?* J Appl Physiol, 2009. **107**(6): p. 1893-9.
61. Corretti, M.C., et al., *Guidelines for the ultrasound assessment of endothelial-dependent flow-mediated vasodilation of the brachial artery: a report of the International Brachial Artery Reactivity Task Force.* J Am Coll Cardiol, 2002. **39**(2): p. 257-65.
62. Mullen, M.J., et al., *Heterogenous nature of flow-mediated dilatation in human conduit arteries in vivo: relevance to endothelial dysfunction in hypercholesterolemia.* Circ Res, 2001. **88**(2): p. 145-51.
63. Joannides, R., et al., *Nitric oxide is responsible for flow-dependent dilatation of human peripheral conduit arteries in vivo.* Circulation, 1995. **91**(5): p. 1314-9.
64. Doshi, S.N., et al., *Flow-mediated dilatation following wrist and upper arm occlusion in humans: the contribution of nitric oxide.* Clin Sci (Lond), 2001. **101**(6): p. 629-35.
65. Leeson, P., et al., *Non-invasive measurement of endothelial function: effect on brachial artery dilatation of graded endothelial dependent and independent stimuli.* Heart, 1997. **78**(1): p. 22-7.

66. Betik, A.C., V.B. Luckham, and R.L. Hughson, *Flow-mediated dilation in human brachial artery after different circulatory occlusion conditions.* Am J Physiol Heart Circ Physiol, 2004. **286**(1): p. H442-8.
67. Celermajer, D.S., et al., *Non-invasive detection of endothelial dysfunction in children and adults at risk of atherosclerosis.* Lancet, 1992. **340**(8828): p. 1111-5.
68. Pyke, K.E. and M.E. Tschakovsky, *Peak vs. total reactive hyperemia: which determines the magnitude of flow-mediated dilation?* J Appl Physiol, 2007. **102**(4): p. 1510-9.
69. Parker, B.A., T.L. Trehearn, and J.R. Meendering, *Pick your Poiseuille: normalizing the shear stimulus in studies of flow-mediated dilation.* J Appl Physiol, 2009. **107**(4): p. 1357-9.
70. Currie, K.D., et al., *Noninvasive measures of vascular health are reliable in preschool-aged children.* Appl Physiol Nutr Metab, 2010. **35**(4): p. 512-7.
71. Iuliano-Burns, S., R.L. Mirwald, and D.A. Bailey, *Timing and magnitude of peak height velocity and peak tissue velocities for early, average, and late maturing boys and girls.* Am J Hum Biol, 2001. **13**(1): p. 1-8.
72. Marshall, W.A. and J.M. Tanner, *Variations in the pattern of pubertal changes in boys.* Arch Dis Child, 1970. **45**(239): p. 13-23.
73. Marshall, W.A. and J.M. Tanner, *Variations in pattern of pubertal changes in girls.* Arch Dis Child, 1969. **44**(235): p. 291-303.
74. Malina, R.M., *Growth, Maturation, and Physical Activity*. 1991, Champaign, IL: Human Kinetics.
75. Malina, R.M., C. Bouchard, and O. Bar-Or, *Body dimensions and composition relative to peak height velocity*, in *Growth, maturation and physical activity*. 2004, Human Kinetics.
76. Telama, R., et al., *Physical activity from childhood to adulthood: a 21-year tracking study.* Am J Prev Med, 2005. **28**(3): p. 267-73.
77. Raitakari, O.T., et al., *Effects of persistent physical activity and inactivity on coronary risk factors in children and young adults. The Cardiovascular Risk in Young Finns Study.* Am J Epidemiol, 1994. **140**(3): p. 195-205.
78. Blair, S.N., et al., *Influences of cardiorespiratory fitness and other precursors on cardiovascular disease and all-cause mortality in men and women.* JAMA, 1996. **276**(3): p. 205-10.
79. Hopkins, N.D., et al., *Does brachial artery flow-mediated dilation scale to anthropometric characteristics?* Eur J Appl Physiol, 2010. **110**(1): p. 171-6.
80. Freedman, D.S. and B. Sherry, *The validity of BMI as an indicator of body fatness and risk among children.* Pediatrics, 2009. **124 Suppl 1**: p. S23-34.
81. Hopkins, N.D., et al., *Relationships between measures of fitness, physical activity, body composition and vascular function in children.* Atherosclerosis, 2009. **204**(1): p. 244-9.
82. Lenard, Z., et al., *Maturation of cardiovagal autonomic function from childhood to young adult age.* Circulation, 2004. **110**(16): p. 2307-12.

83. Tanaka, H., C.A. DeSouza, and D.R. Seals, *Absence of age-related increase in central arterial stiffness in physically active women.* Arterioscler Thromb Vasc Biol, 1998. **18**(1): p. 127-32.
84. Vaitkevicius, P.V., et al., *Effects of age and aerobic capacity on arterial stiffness in healthy adults.* Circulation, 1993. **88**(4 Pt 1): p. 1456-62.
85. Lakatta, E.G., *Arterial and cardiac aging: major shareholders in cardiovascular disease enterprises: Part III: cellular and molecular clues to heart and arterial aging.* Circulation, 2003. **107**(3): p. 490-7.
86. Nosaka, T., et al., *Influence of regular exercise on age-related changes in arterial elasticity: mechanistic insights from wall compositions in rat aorta.* Can J Appl Physiol, 2003. **28**(2): p. 204-12.
87. Senzaki, H., et al., *Age-associated changes in arterial elastic properties in children.* Eur J Pediatr, 2002. **161**(10): p. 547-51.
88. Kakiyama, T., M. Matsuda, and S. Koseki, *Effect of physical activity on the distensibility of the aortic wall in healthy males.* Angiology, 1998. **49**(9): p. 749-57.
89. Seals, D.R., et al., *Habitual exercise and vascular ageing.* J Physiol, 2009. **587**(Pt 23): p. 5541-9.
90. Hacihamdioglu, B., et al., *Abdominal obesity is an independent risk factor for increased carotid intima- media thickness in obese children.* Turk J Pediatr, 2011. **53**(1): p. 48-54.
91. Aggoun, Y., et al., *[Cardiovascular impact of end-stage renal insufficiency in children undergoing hemodialysis].* Arch Mal Coeur Vaiss, 2000. **93**(8): p. 1009-13.
92. Verschuren, O., et al., *Exercise training program in children and adolescents with cerebral palsy: a randomized controlled trial.* Arch Pediatr Adolesc Med, 2007. **161**(11): p. 1075-81.
93. van de Laar, R.J., et al., *Lifetime vigorous but not light-to-moderate habitual physical activity impacts favorably on carotid stiffness in young adults: the amsterdam growth and health longitudinal study.* Hypertension, 2010. **55**(1): p. 33-9.
94. van de Laar, R.J., et al., *Habitual physical activity and peripheral arterial compliance in young adults: the Amsterdam growth and health longitudinal study.* Am J Hypertens, 2011. **24**(2): p. 200-8.
95. Riddoch, C., et al., *Long term health implications of fitness and physical activity patterns.* Arch Dis Child, 1991. **66**(12): p. 1426-33.
96. Kimm, S.Y., et al., *Longitudinal changes in physical activity in a biracial cohort during adolescence.* Med Sci Sports Exerc, 2000. **32**(8): p. 1445-54.
97. Trost, S.G., et al., *Age and gender differences in objectively measured physical activity in youth.* Med Sci Sports Exerc, 2002. **34**(2): p. 350-5.
98. Sallis, J.F., et al., *Predictors of change in children's physical activity over 20 months. Variations by gender and level of adiposity.* Am J Prev Med, 1999. **16**(3): p. 222-9.

99. Markus, R.A., et al., *Influence of lifestyle modification on atherosclerotic progression determined by ultrasonographic change in the common carotid intima-media thickness.* Am J Clin Nutr, 1997. **65**(4): p. 1000-4.
100. Reed, K.E., et al., *Arterial compliance in young children: the role of aerobic fitness.* Eur J Cardiovasc Prev Rehabil, 2005. **12**(5): p. 492-7.

Table 1
Subject Characteristics

	Control (n=11)	CP (n=11)	P Value
Age, yrs	12.4 ± 2.3	13.2 ± 2.1	0.458
Height, m	1.6 ± 0.1	1.5 ± 0.1	0.169
Weight, kg	49.3 ± 14.2	41.4 ± 8.4	0.129
APHV	13.08 ± 0.9	14.02 ± 1.3	0.062
TPHV, yrs	-0.66 ± 2.1	-0.86 ± 1.7	0.809
WC, cm	69.8 ± 8.8	67.3 ± 7.2	0.478
WHR	0.44 ± 0.05	0.45 ± 0.06	0.750
BMI, kg/m^2	19.5 ± 3.7	18.4 ± 3.2	0.474
BMI Percentile	57 ± 31	38 ± 33	0.178
Resting HR, bpm	68 ± 10	74 ± 13	0.278
Resting Systolic BP, mmHg	113 ± 8	106 ± 12	0.164
Resting Diastolic BP, mmHg	65 ± 5	62 ± 6	0.169
Resting MAP, mmHg	84 ± 3	81 ± 6	0.152

Values are represented as means ± SD. APHV, Age at peak height velocity; TPHV, Time to peak height velocity; WC, waist circumference; WHR, waist-to-height ratio; BMI, body mass index; HR, heart rate; BPM, beats per minute; BP, blood pressure; MAP, mean arterial pressure.

Table 2
Group comparisons of brachial vascular dimensions and FMD response

	Control (n=10)	CP (n=10)	P Value
Pre-occlusion diameter, mm	3.20 ± 0.37	3.08 ± 0.48	0.803
RH peak diameter, mm	3.40 ± 0.39	3.48 ± 0.38	0.815
Absolute FMD, mm	0.19 ± 0.11	0.33 ± 0.21	0.075
Relative FMD (%FMD)	6.1 ± 3.6	11.1 ± 7.8	0.080
Normalized (%FMD/SR_{AUC})	0.0027 ± 0.0015	0.0046 ± 0.0033	0.126
Mean SR	530 ± 250	544 ± 198	0.788
Time to peak, s	110 ± 45	102 ± 46	0.826
PP, mmHg	48 ± 10	45 ± 13	0.523

Values are represented as means ± SD. RH, reactive hyperemia; FMD, flow mediated dilation; SR, Shear rate; SR_{AUC}, Shear rate area under the curve.
Note: n=11 in both groups for baseline brachial diameter.

Table 3
Group comparisons of carotid vascular dimensions and carotid distensibility

	Control (n=11)	CP (n=11)	P Value
Baseline diameter, mm	5.73 ± 0.29	5.63 ± 0.74	0.690
IMT, mm	0.41 ± 0.03	0.42 ± 0.04	0.576
Wall/lumen ratio	0.072 ± 0.007	0.077 ± 0.012	0.832
Distensibility, $mmHg^{-1}$	0.008 ± 0.002	0.008 ± 0.002	0.474
Compliance, mm^2/ mmHg	0.19 ± 0.03	0.17 ± 0.06	0.376
PP (mmHg)	36 ± 10	42 ± 11	0.208

Values are represented as means ± SD. Compliance, Distensibility: Control, n=10; IMT, Intima-media thickness and wall/lumen ratio: CP, n=10; PP, Pulse pressure.

Table 4
Group comparisons of PTT and PWV

	Control (n=10)	CP (n=11)	P Value
Central PTT	0.103 ± 0.032	0.089 ± 0.013	0.454
Central PWV (m/s)	4.1 ± 0.9	4.3 ± 0.6	0.977
Peripheral PTT	0.108 ± 0.012	0.111 ± 0.028	0.768
Peripheral PWV (m/s)	7.6 ± 1.1	7.1± 1.7	0.450

Values are represented as means ± SD. PTT, pulse transit time; PWV, pulse wave velocity.

Table 5
Group comparisons of weekly physical activity

	Control (n=11)	CP (n=11)	P Value
Light, min.	85 ± 90	92 ± 116	0.887
Medium, min.	159 ± 141	258 ± 242	0.254
Hard, min.	196 ± 174*	38 ± 80	0.016
Total, min.	440 ± 231	387 ± 308	0.655
Meeting guidelines, %	36	27	-

Values are represented as means ± SD. * indicates $p<0.05$

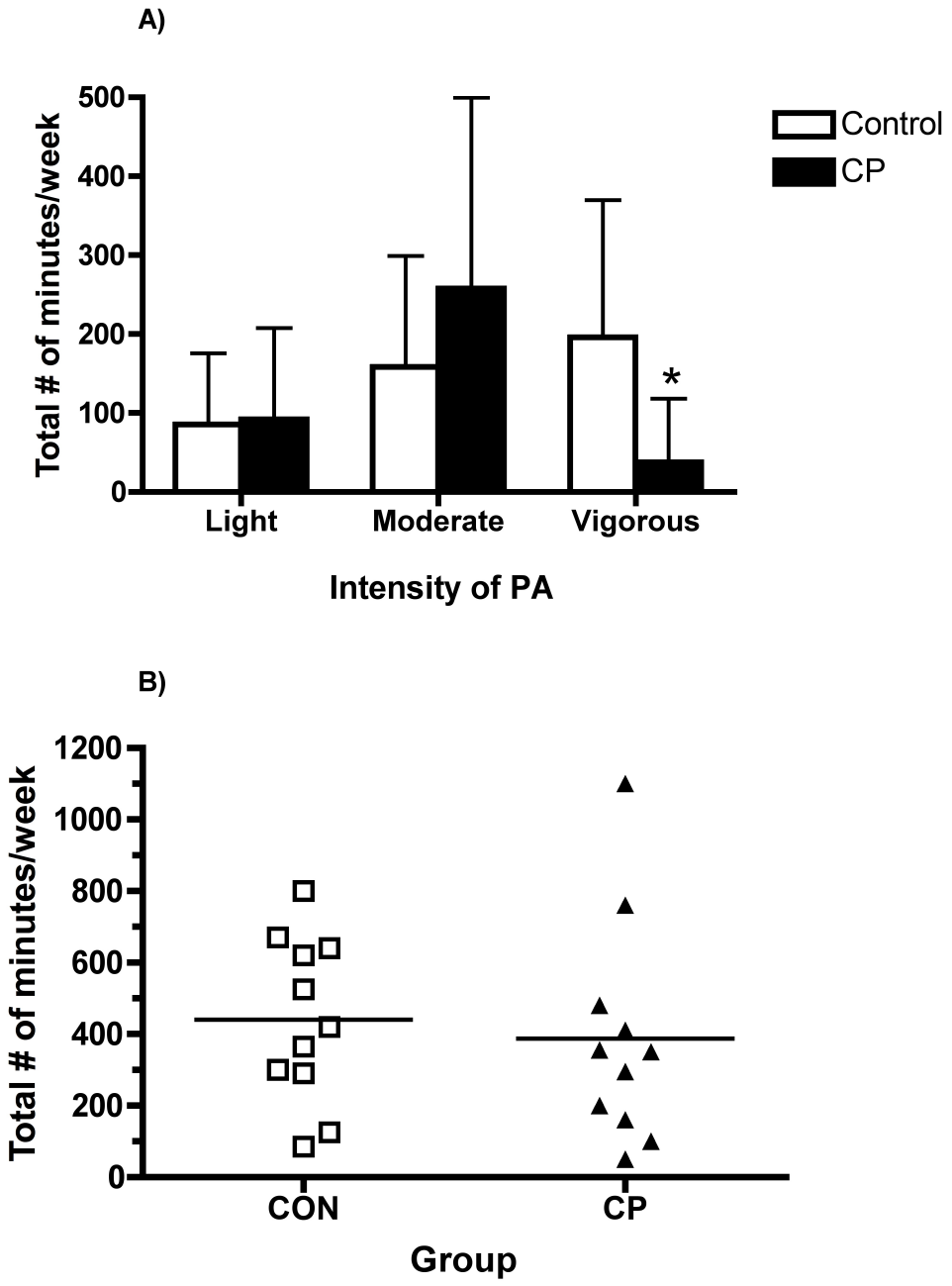

Figure 1. A) Group comparisons of weekly PA according to intensity B) Group comparisons of total PA (summation of all three intensities) performed in one week; n=11 in each group

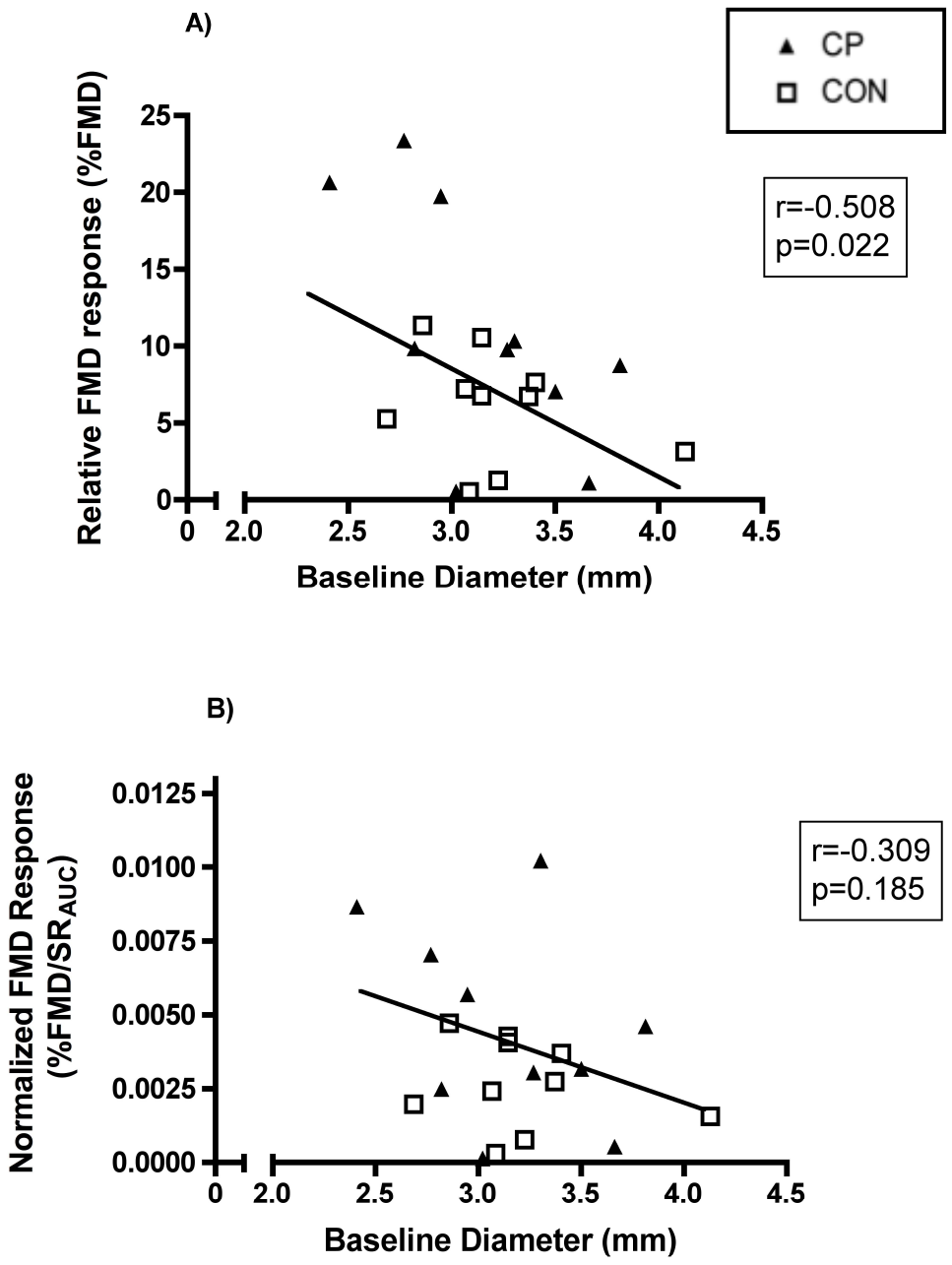

Figure 2. Relationship between baseline brachial diameter (mm) and A) the relative FMD response (%FMD) and B) the normalized FMD response (%FMD/SR_{AUC}) n=10 in both groups for A) and B)

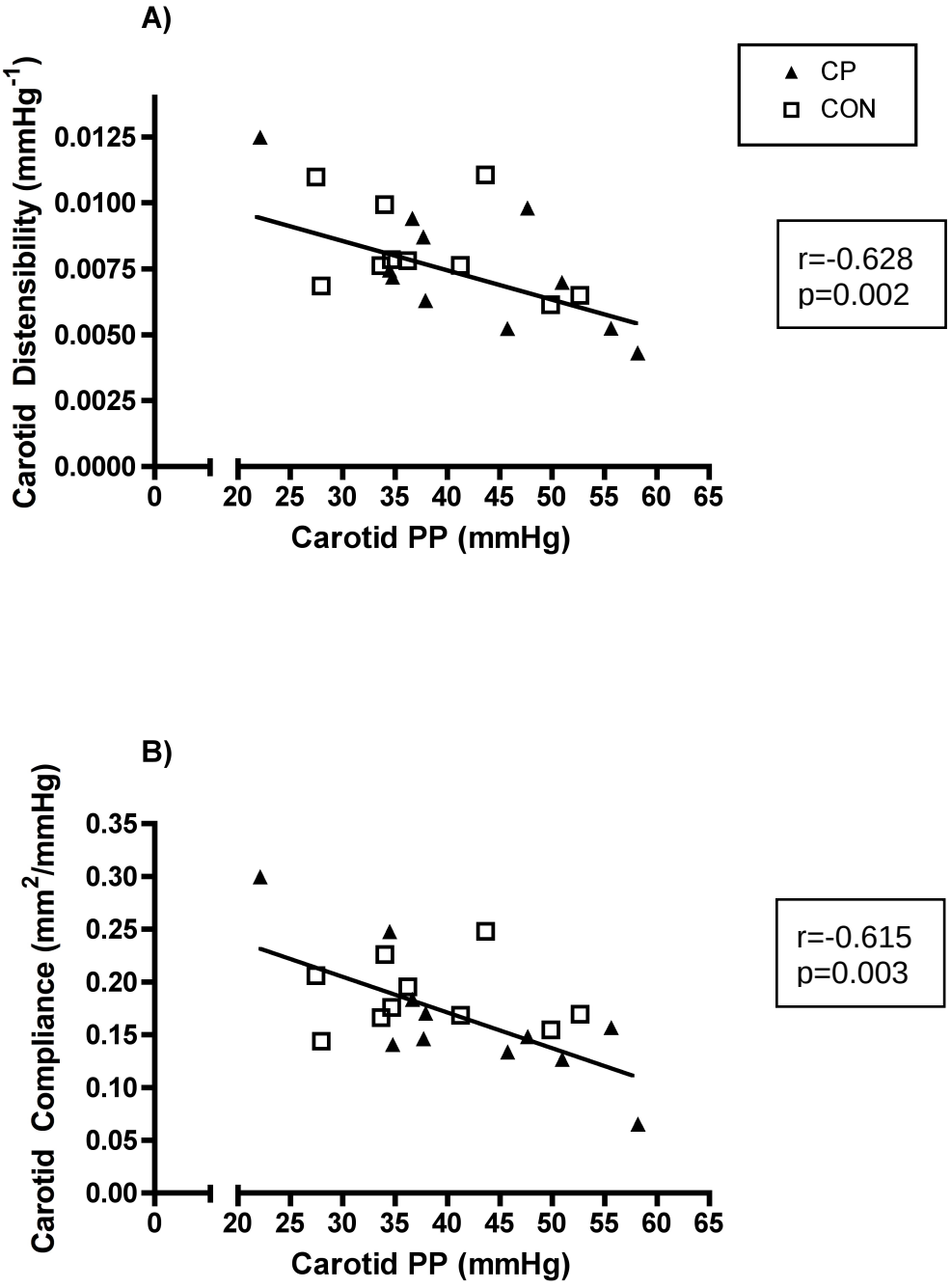

Figure 3. Relationship between carotid pulse pressure (PP) and A) Carotid distensibility and B) Carotid compliance, CON: n=10 for A) and B)

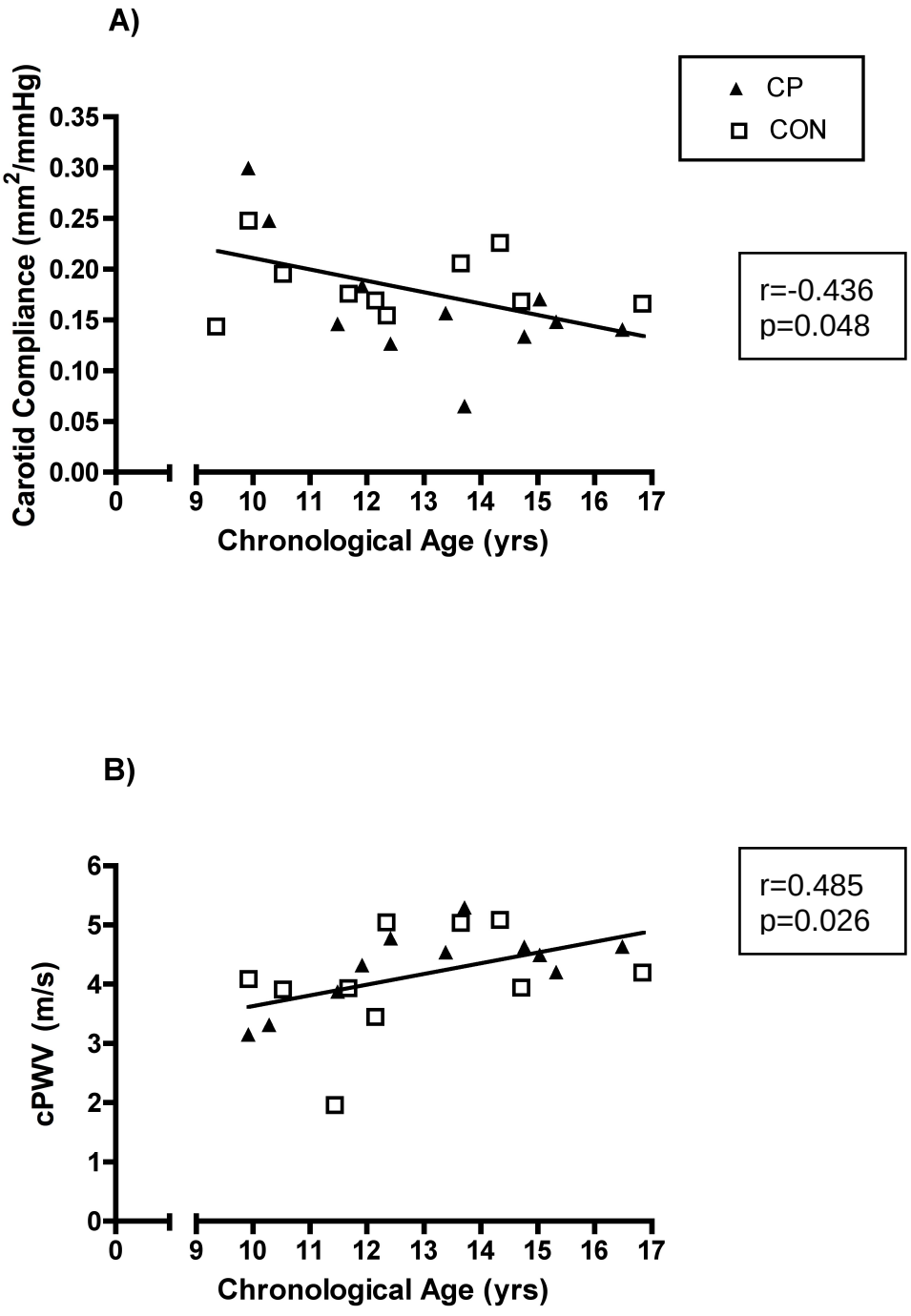

Figure 4. Relationship between chronological age and A) Carotid compliance and B) cPWV, central pulse wave velocity, CON: n=10 for A) and B)

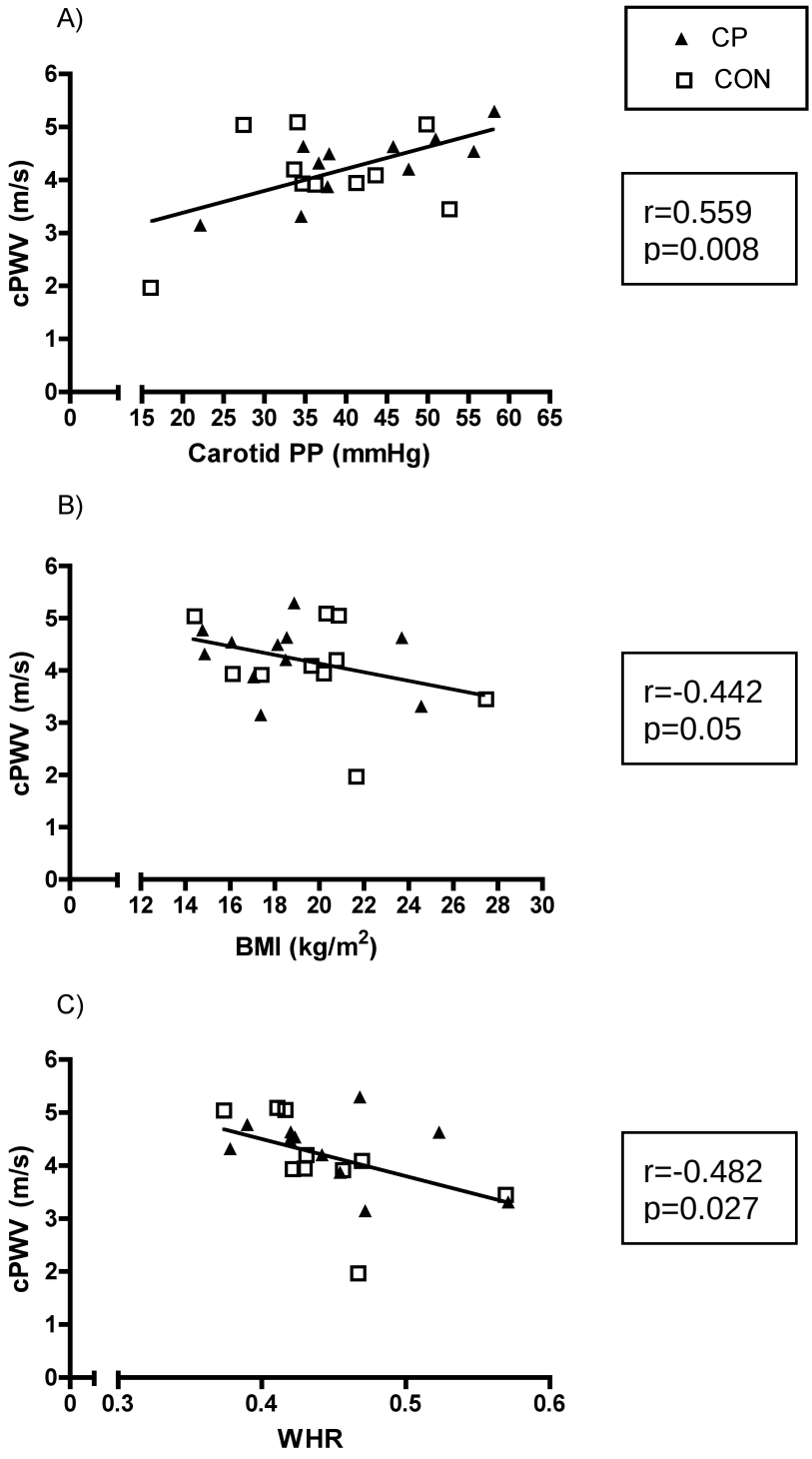

Figure 5. Relationships between central pulse wave velocity (cPWV, CON: n=10) and A) Carotid pulse pressure, B) BMI, body mass index, and C) WHR, waist-to-height ratio

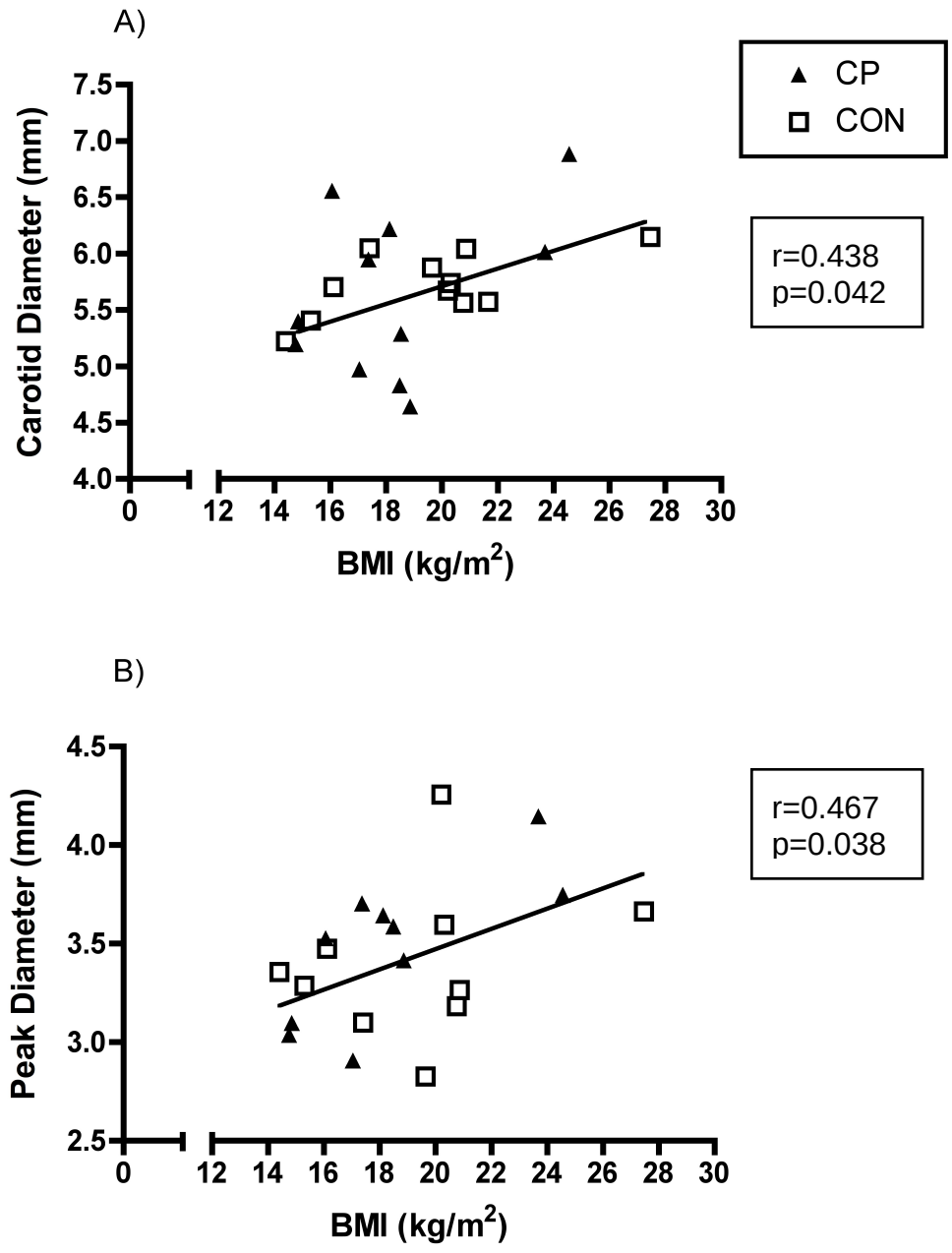

Figure 6. Relationship between body mass index (BMI) and A) Carotid diameter and B) Peak diameter obtained during the FMD assessment, n=10 in both groups for B)

APPENDIX A – Example of GMFCS-E&R Scale for children 6-12 years old

GMFCS Level I
Children walk at home, school, outdoors and in the community. Children are able to walk up and down curbs without physical assistance and stairs without the use of a railing. Children perform gross motor skills such as running and jumping but speed, balance, and coordination are limited. Children may participate in physical activities and sports depending on personal choices and environmental factors
GMFCS Level II
Children walk in most settings. Children may experience difficulty walking long distances and balancing on uneven terrain, inclines, in crowded areas, confined spaces or when carrying objects. Children walk up and down stairs holding onto a railing or with physical assistance if there is no railing. Outdoors and in the community, children may walk with physical assistance, a hand-held mobility device, or use wheeled mobility when traveling long distances. Children have at best only minimal ability to perform gross motor skill such as running and jumping. Limitations in performance of gross motor skills may necessitate adaptations to enable participation in physical activities and sports.
GMFCS Level III
Children walk using a hand-held mobility device in most indoor setting. When seated, children may require a seat belt for pelvic alignment and balance. Sit-to-stand and floor-to-stand transfers require physical assistance of a person or support surface. When traveling long distances, children use some form of wheeled mobility. Children may walk up and down stairs holding onto a railing with supervision or physical assistance. Limitations in walking may necessitate adaptations to enable participation in physical activities and sports including self-propelling a manual wheelchair or powered mobility.
GMFCS Level IV
Children use methods of mobility that require physical assistance or powered mobility in most settings. Children require adaptive seating for trunk and pelvic control and physical assistance for most transfers. At home, children use floor mobility (roll, creep, or crawl), walk short distances with physical assistance, or use powered mobility. When positioned, children may use a body support walker at home or school. At school, outdoors, and in the community, children are transported in a manual wheelchair or use powered mobility. Limitations in mobility necessitate adaptations to enable participation in physical activities and sports, including physical assistance and/or powered mobility.
GMFCS Level V
Children are transported in a manual wheelchair in all settings. Children are limited in their ability to maintain antigravity head and trunk postures and control arm and leg movements. Assistive technology is used to improve head alignment, seating, standing, and and/or mobility but limitations are not fully compensated by equipment. Transfers require complete physical assistance of an adult. At home, children may move short distances on the floor or may be carried by an adult. Children may achieve self-mobility using powered mobility with extensive adaptations for seating and control access. Limitations in mobility necessitate adaptations to enable participation in physical activities and sports including physical assistance and using powered mobility.

GMFCS descriptors from Palisano et al., 2007

Exercise Questionnaire

The chart on the next page asks about the exercises your child did last week. By "exercise", we mean activities that involve stretching, strengthening, or physical effort. Activities that involve effort or exertion cause the following things: 1) the heart works harder and faster, 2) breathing is deeper, and 3) the body perspires or sweats.

The 1st column in the chart asks you to think about and circle the number for the **exercises your child did over the past week**. There is some space for you to write in the other "sports", or "exercises" that you did.

The 2nd column in the chart asks you to write-in the **number of different times** in the past week your child did each of the exercises listed. If your child didn't do an exercise at all, just leave the space blank.

The 3rd column in the chart asks you to write-in the **average amount of time (in minutes)** your child spent doing each of the exercises listed, each time. You won't need to put anything in this column for the exercises you did not do at all last week.

The 4th column in the chart asks you to write-in **how hard your child worked on average** when he or she did each exercise last week. Again, you won't need to put anything in this column for the exercises your child did not do at all last week.

When you are thinking about how hard your child worked, please choose either **light**, **medium**, or **hard** according to the descriptions below:

Light	normal heart rate and breathing, no sweating
Medium	some increase in heart rate and breathing
Hard	heart working hard, breathing very deep, sweating

Exercise Questionnaire

Study ID: ___ ___ ___ **Date Completed: _____**

	Activity	**How many times did your child do the activity last week?** (write "zero" if he or she didn't do the activity at all last week)	**Each time your child did the activity, how much time did he or she spend doing it?** (in minutes)	**How hard did your child work at the activity?** (light, medium, hard)
1	Walk / run			
2	Wheel in a manual wheelchair			
3	Cycle			
4	Dance			
5	Swim / aquatics			
6	Basketball			
7	Hockey / sledge hockey			
8	Baseball / T ball			
9	Soccer			
10	Volleyball			
11	Football			
12	Bowling / Boccia ball			
13	Horseback riding			
14	Canoeing / kayaking			
15	Sailing			
16	Golf			
17	Skating / Skiing			

18	Rollerblading			
19	Martial Arts / wrestling / yoga / gymnastics			
20	Strength training/weight lifting			
21	Stretching exercises			
	Other activities (please list them)			
22	a.			
	b.			
	c.			
	d.			
	e.			

Light normal heart rate and breathing, not sweating
Medium some increase in heart rate and breathing
Hard heart working hard, breathing very deep, sweating

McMaster University Health Sciences

Hamilton Health Sciences
EXEMPLARY CARE • ACADEMIC EXCELLENCE

PARTICIPANT ASSENT FORM

Title of Study: *Baseline Assessment of Arterial Structure and Function in Adolescents with Cerebral Palsy*

Principal Investigator: *Brian W. Timmons (PhD), Children's Exercise & Nutrition Centre*

Co-Investigators: *Audra Martin, Department of Kinesiology, McMaster University*
Jan Willem Gorter (MD), Department of Pediatrics, McMaster University
Maureen MacDonald (PhD), Department of Kinesiology, McMaster University

Funding Source: *Natural Sciences and Engineering Research Council of Canada*

Why are we doing this study?
A research study is a way to learn more about people. We are doing a research study for youth with cerebral palsy (CP) to learn more about your health. There are things we can measure that give us clues about our health when we grow up. But these things have not been measured in youth with CP before. In kids without CP, these things are related to how much physical activity they do, so we want to know if these things are related to physical activity in youth with CP.

Why am I being asked to be in the study?
We are inviting you to be in this study because you are an adolescent with CP and because you agreed to participate in a physical activity study for Dr. Gorter. In that study, Dr. Timmons was responsible for the physical activity information and that little pager-like device that you wore around your waist and on your wrist.

What if I have questions?

You can ask questions if do you do not understand any part of this study. If you have questions later that you don't think of now, you can talk to Audra Martin again or ask your mom or dad to call Audra Martin or Dr. Timmons.

If I am in the study what will happen to me?

If you decide that you want to be in this study, you will come to McMaster University for one visit. At this visit, we will take pictures of the main blood vessel in your neck and arm. This means that you have to lie on a bed for about 20 min for the pictures to be taken. We will also place little sensors (small squares that sit on top of the skin and do not hurt) on your groin and top of foot to collect signals that show up on a computer screen. Measurements using a regular tape measure will be taken from the top of your chest to each location we had the sensors on.

Another test involves pictures of the main blood vessel in your arm. A blood pressure cuff (just like they use at the doctors) will be placed around your forearm, just below your elbow, and instantaneously inflated. We will leave the cuff blown up around your arm for five minutes. Your hand may tingle slightly, just like when it falls asleep (pins and needles) but should not hurt or cause discomfort. The cuff is deflated (air is let out) and we will continue taking pictures of your artery. Again, all you have to do is lie comfortably on the bed while we perform the tests.

We will then measure your height (or arm span) and weight and your waist size. We will take this information and compare it to your physical activity information from the study with Dr. Gorter.

Will I be hurt if I am in the study?

There is no reason why you should be hurt in this study. The machine that takes the picture of your neck and arm is the same machine that doctors use to take pictures of babies before they are born, called an ultrasound. The only discomfort you may experience might occur during the FMD test. During this test, we only let a little bit of blood flow to your hand for five minutes. This may feel similar to when your hand or foot fall asleep and you get a tingly sensation. No permanent damage or pain will result from this and any discomfort will go away as soon as the five minutes are over.

Will the study help me?

This study may not help you directly, but we will learn about how being active is related to some of these things that give us clues about our health when we grow up. We will make a report about this study and give it to you when the study is over. What we find in this study will help us make a study that will answer important questions about physical activity and health in youth with CP. You may be helping other youth with CP in the future by providing more information about what goes on in your body right now.

Do I have to be in this study?

You don't have to be in this study, if you don't want to be. If you decide that you don't want to be in the study after we begin, that's OK too. Nobody will be angry or upset. It is your choice. We are discussing the study with your parents and you should talk to them too. If you don't want to continue at any time throughout the study, just tell the researcher working with you or your parent who can then tell us, and we will stop right away.

What happens after the study?

When we are finished this study we will write a report about what was learned. This report will not include your name or that you were in the study.

Assent:

If you decide you want to be in this study, please print/write your name. If you decide that you don't want to be in the study, even if you have started in the study, then all you have to do is tell Audra that you don't want to be in the study anymore.

I,_____ (Print your name) would like to be in this research study.

_____ (Date of assent)

_____ (Name of person who obtained assent)

_____ (Signature of person who obtained assent and Date)

_____ (Local Principal Investigator name)

_____ (LPI signature and Date)

McMaster University Health Sciences

Hamilton Health Sciences
EXEMPLARY CARE • ACADEMIC EXCELLENCE

PARENT INFORMATION AND CONSENT FORM

Title of Study: *Baseline Assessment of Arterial Structure and Function in Adolescents with Cerebral Palsy*

Principal Investigator: *Brian W. Timmons (PhD), Children's Exercise & Nutrition Centre*

Co-Investigators: *Audra Martin, Department of Kinesiology, McMaster University*

Jan Willem Gorter (MD), Department of Pediatrics, McMaster University

Maureen MacDonald (PhD), Department of Kinesiology, McMaster University

Funding Source: *Natural Sciences and Engineering Research Council of Canada*

INTRODUCTION

Your child is being invited to participate in a research study conducted by Dr. Brian Timmons because they previously participated in a physical activity study with Dr. Jan Willem Gorter and because they are an adolescent with cerebral palsy (CP). In that study, Dr. Timmons was responsible for the physical activity data and that little pager-like device that your child wore around their waist and on their wrist. In order to decide whether or not you want your child to be a part of this research study, you should understand what is involved and the potential risks and benefits. This form gives detailed information about the study, which will be discussed with you. Once you understand the study, you will be asked to sign this form if you wish to participate. Feel free to discuss it with your family and take your time to make your decision.

WHY IS THIS RESEARCH BEING DONE?

We believe that exercise and physical activity are good for adolescents regardless of medical history. For youth with a disability that might lower their physical activity levels, it is important to understand the effects on other aspects of health. We are interested in learning more about traditional risk factors with a specific focus on arterial structure and function, for later life health in adolescents with CP. By understanding these factors and how they relate to physical activity

level, we will then be able to make informed recommendations on how much physical activity might be appropriate to improve later life health.

WHAT IS THE PURPOSE OF THIS STUDY?

This is an observational study designed to establish normative, baseline values of vascular measures that (to our knowledge) have not been previously determined in this clinical population. The main purpose of this study is to measure aspects of health that are related to well-being later in life. We want to see if things like blood pressure, how well the blood vessels work, and body composition are related to physical activity in adolescents with CP.

WHAT WILL MY CHILD'S RESPONSIBILITIES BE IF THEY TAKE PART IN THE STUDY?

If you and your child volunteer to participate in this study, we will ask you to do the following things:

- Make 1 visit to McMaster University and the Department of Kinesiology.

- At this visit, we will have your child complete a physical activity questionnaire and we will measure the health of the main blood vessels in your child's neck and arm, including blood pressure. These vascular health tests require your child to lie on a bed for about 20 min while the measurements are taken. We will then measure their height (or arm span), weight and their waist size.

- Because you and your child already participated in the physical activity study with Dr. Gorter (accelerometers), we will link information obtained during this single visit with your physical activity data that we have on file.

- If willing, a second visit to repeat the FMD assessment (only about 15 min) will be scheduled at your convenience. This is to assess the reliability of this assessment in adolescents, in our laboratory.

WHAT ARE THE POSSIBLE RISKS AND DISCOMFORTS?

There are no unusual risks associated with your child's participation in this study. To measure blood vessels, we will use the same ultrasound machine that doctors use to take a picture of a baby during a pregnancy. The only discomfort your child may experience is during the FMD test at which time, blood flow to the hand is

occluded for five minutes using a blood pressure cuff on the forearm. Your child may feel a tingling sensation or no feeling at all during these five minutes, a sensation similar to when your hand or foot fall asleep. No permanent damage or pain will result from this and any discomfort will go away as soon as the five minutes are over.

Any information collected from you and your child will become anonymous to ensure confidentiality.

HOW MANY PEOPLE WILL BE IN THIS STUDY?

We are asking all of the adolescents who were recruited in the early physical activity study with Dr. Gorter and those who already completed the pilot study to participate. This should be about 10 adolescents with CP and 10 control adolescents for a total of 20 participants. If your child did not participate in the pilot study (required one visit to the vascular dynamics lab already) you may be asked if you would like to return for a second visit to the lab to repeat the FMD assessment. Your participation in all aspects of the study is voluntary. If you choose to stop participation, this decision will in no way affect your normal clinical care at the McMaster Children's Hospital.

WHAT ARE THE POSSIBLE BENEFITS FOR MY CHILD AND/OR FOR SOCIETY?

We cannot promise any personal benefits to you or your child from their participation in this study. You will learn about the vascular health of your child, their blood pressure and their body composition. These aspects of health are important and can be improved with physical activity. By participating in this study, you will be helping us build a bigger study to definitively answer questions about how physical activity can improve health for youth with CP.

WHAT INFORMATION WILL BE KEPT PRIVATE?

All of your information will be stored in filing cabinets under the supervision of Dr. Brian Timmons for 10 years at the Children's Exercise & Nutrition Centre. We will supervise access to your child's information by other people in our group, only if necessary. Your child will be assigned a subject number, and this number will be used to identify them. Records identifying your child will be kept confidential. If the results of the study are published, their identity will remain confidential.

CAN PARTICIPATION IN THE STUDY END EARLY?

If your child volunteers to be in this study, you or your child may withdraw at any time with no prejudice. The investigator may withdraw your child from this research if circumstances arise which warrant doing so. In no way, will early withdraw affect the clinical care your child receives from the McMaster Children's Hospital.

WILL MY CHILD BE PAID TO PARTICIPATE IN THIS STUDY?

Your child will be paid ten dollars to participate in this study and we will reimburse you for any parking expenses.

IF I HAVE ANY QUESTIONS OR PROBLEMS, WHOM CAN I CALL?

If you have any questions about the research now or later, you can contact Dr. Timmons at 905-521-2100, ext 77218 or ext 77615 or timmonbw@mcmaster.ca or Audra Martin at 905-525-9140 ext 24694 or ext 27037 martiaa2@mcmaster.ca

If you have any questions regarding your rights as a research participant, you may contact the Office of the Chair of the Hamilton Health Sciences/Faculty of Health Sciences Research Ethics Board at 905-521-2100, ext. 42013.

CONSENT STATEMENT

I have read the preceding information thoroughly. I have had the opportunity to ask questions, and all of my questions have been answered to my satisfaction and to the satisfaction of my child. I agree to allow my child to participate in this study entitled: *"Baseline Assessment of Arterial Structure and Function in Adolescents with Cerebral Palsy".* I understand that I will receive a signed copy of this form.

_____
Name of Participant (child's name)

_____
Printed Name of Legally Authorized Representative

_____ _____
____
Signature of Legally Authorized Representative Date

Consent form administered and explained in person by:

_____
Printed Name of Person Obtaining Consent

_____
_____
Signature of Person Obtaining Consent Date

SIGNATURE OF INVESTIGATOR:

In my judgement, the participant is voluntarily and knowingly giving informed consent and possesses the legal capacity to give informed consent for their child to participate in this research study.

_____
Printed Name and title

_____ _____

Signature of Investigator Date

McMaster University
Department of Kinesiology 1280 Main Street West, Hamilton, ON, Canada, L8S 4K1
Phone: 905.525.9140
Fax: 905.523.6011
http://kinlabserver.mcmaster.ca

Baseline Assessment of Vascular Structure and Function in Adolescents with Cerebral Palsy.

PARTICIPANT INSTRUCTION SHEET

Please follow these instructions before each visit:

- ☐ No exercise or intense physical activity within 24 hours of your visit
- ☐ No alcohol or caffeine 12 hours before your visit
- ☐ No food or drink 4 hours before your visit
  (water consumption is allowed)
- ☐ No vasoactive medications (i.e. nitroglycerin) on the day of your visit
- ☐ No medications or vitamins NOT prescribed by your doctor (ex. Tylenol, Advil, allergy medicine, Vitamin C etc.) on the day of your visit
- ☐ Please bring exercise clothing including loose fitting shorts – we will need to access your femoral artery in your upper groin
- ☐ Please bring your current medications and/or a list of your current medications

McMaster University
Department of Kinesiology 1280 Main Street West, Hamilton, ON, Canada, L8S 4K1
Phone: 905.525.9140
Fax: 905.523.6011
http://kinlabserver.mcmaster.ca

Baseline Assessment of Vascular Structure and Function in Adolescents with Cerebral Palsy

Participant Information

Participant Name: _____ Age: _____

Date of Birth (dd/mm/yyyy): _____

Contact Information:
Phone (day): _____ Phone (evening): _____

Mailing Address: _____
_____

Emergency Contact Information:
Name: _____ Relationship: _____

Phone (day): _____ Phone (evening): _____

Address: _____
_____
_____

Current Prescribed Medications:
_____
_____
_____

Current O.T.C Medications and / or Vitamins:
_____
_____
_____

Allergies:
_____

Date of last Botox injection (if applicable): _____

McMaster University
Department of Kinesiology 1280 Main Street West, Hamilton, ON, Canada, L8S 4K1
Phone: 905.525.9140
Fax: 905.523.6011
http://kinlabserver.mcmaster.ca

Food / Drink / Caffeine consumption in the last 4 hours:

_____
_____
_____
_____
_____
_____

PA in the last 24 hrs:

_____
_____

Additional Information (history of cardiovascular events, complications, etc.):

_____
_____
_____
_____

APPENDIX G - Raw Data

Table 1: Anthropometric Data

Gender, GMFCS	ID	Exact Age (yrs)	Stool Ht (cm)	Recorded seated Ht (cm)	Seated Ht (cm)	Standing Ht (cm)	Weight (kg)	BMI	BMI %ile	WC	WHR
M,2	CP1	9.91	46	119.1	73.1	137.6	32.9	17.4	64	64.9	0.472
M,1	CP2	15.32	46	130.6	84.6	158.1	46.2	18.5	26	69.8	0.441
M,2	CP3	13.71	46	115.6	69.6	138.9	36.4	18.9	48	65	0.468
M,2	CP4	14.76	46	126	80	157	58.4	23.7	87	82.1	0.523
M,1	CP5	12.41	46	123.4	77.4	148.6	32.6	14.8	2	57.9	0.390
M,1	CP6	15.03	46	126	80.00	164.8	49.2	18.1	23	69.1	0.419
M,2	CP7	10.28	46	119.3	73.30	137.3	46.3	24.6	97	78.4	0.571
M,1	CP8	13.38	46	119.9	73.90	147.8	35.1	16.1	8	62.5	0.423
F,1	CP9	11.91	46	131.2	85.20	164.9	40.4	14.9	5	62.3	0.378
F,1	CP10	16.48	46	125	79.00	155.3	44.7	18.5	20	65.2	0.420
F,1	CP11	11.48	46	117.9	71.90	138.5	32.7	17.0	38	62.9	0.454
Ave:		**13.15**	**46.0**	**123.09**	**77.09**	**149.89**	**41.36**	**18.40**	**38.0**	**67.2**	**0.45**
SD:		**2.14**	**0.00**	**5.16**	**5.16**	**10.78**	**8.36**	**3.18**	**32.6**	**7.23**	**0.06**
M	CON1	11.67	46	121.5	75.50	147.6	35.1	16.1	22	62.2	0.421
M	CON2	12.34	46	134.2	88.20	174.2	63.3	20.9	82	72.5	0.416
M	CON3	13.64	46	126	80.00	172.4	42.8	14.4	1	64.4	0.374
M	CON4	14.7	46	136.3	90.30	171.6	59.5	20.2	58	73.7	0.429
M	CON5	9.35	46	114.3	68.30	133.8	27.4	15.3	26	58.1	0.434
M	CON6	12.14	46	131.2	85.20	160.9	71.1	27.5	97	91.6	0.569
M	CON7	14.33	46	134.5	88.50	170	58.7	20.3	63	69.8	0.411
M	CON8	9.92	46	119	73.00	140	38.5	19.6	87	65.7	0.469
F	CON9	11.43	46	127.2	81.2	155.1	52.1	21.7	86	72.4	0.467
F	CON10	16.83	46	134.6	88.6	168.3	58.8	20.8	49	72.5	0.431
F	CON11	10.52	46	115.6	69.6	141.6	34.9	17.4	54	64.6	0.456
Ave:		**12.44**	**46**	**126.76**	**80.76**	**157.77**	**49.29**	**19.47**	**56.8**	**69.8**	**0.44**
SD:		**2.25**	**0**	**8.10**	**8.10**	**14.86**	**14.18**	**3.64**	**30.7**	**8.83**	**0.05**

Table 2: Hemodynamic profiles, and peak height velocities

ID	SBP	DBP	MAP	Resting HR	APHV	TPHV (yrs before/ after)
CP1	112.3	56	81.7	61.626235	13.1	-3.2
CP2	111	63.7	83.7	56.84030329	14.7	0.6
CP3	96	65.5	78.5	76.14698	15.7	-2
CP4	130.3	68.7	91	73.9029425	14.9	-0.1
CP5	83	66.3	87.3	74.6824375	14.2	-1.8
CP6	111.3	65.7	84	90.808495	15.2	-0.2
CP7	101	62.3	78.7	84.7282325	13.1	-2.8
CP8	109.3	59	81.7	79.68989395	15.1	-1.7
CP9	103	56.7	77.3	60.660875	11.4	0.5
CP10	105	65.3	81.3	59.799285	14.1	2.4
CP11	108.7812725	50.740655	67.2916925	93.9270875	12.7	-1.2
Ave:	**106.453**	**61.813**	**81.136**	**73.892**	**14.018**	**-0.864**
SD:	**11.677**	**5.502**	**6.083**	**12.881**	**1.302**	**1.669**
CON1	106.7	65.7	83	62.08836	14	-2.3
CON2	122	69	88.7	60.4443575	12.6	-0.3
CON3	111	68.3	85.3	69.9196725	14.5	-0.9
CON4	117.3	64.7	85.7	54.2544425	13.6	1.1
CON5	104	69.3	83.3	86	13.3	-4
CON6	121	65.7	87.7	84.1033675	12.6	-0.5
CON7	126.3	55	85.7	64.176865	13.6	0.7
CON8	108.7	66.7	84	71.1915275	13.1	-3.2
CON9	105.3	57.3	77.7	73.2634375	11.4	0
CON10	108.7	66	84	62.2489175	13.1	3.7
CON11	107	64	81	64.9243575	12.1	-1.6
Ave:	**112.545**	**64.700**	**84.191**	**68.420**	**13.082**	**-0.664**
SD:	**7.714**	**4.579**	**3.050**	**9.803**	**0.877**	**2.140**

Table 3: Brachial vascular dimensions and FMD response

Gender, GMFCS	ID	Baseline Brachial Diameter (mm)	Absolute	Relative	Normalized	MaxRHED LD	Ave Shear Rate	Time at Peak
M,2	CP1	3.66333	0.04067	1.11010	0.00054	3.704	470.75	75
M,1	CP2	3.26767	0.31933	9.77252	0.00305	3.587	565.49	45
M,2	CP3	2.76867	0.64733	23.38069	0.00704	3.416	418.78	180
M,2	CP4	3.81233	0.33367	8.75230	0.00461	4.146	379.10	60
M,1	CP5	3.02033	0.01667	0.55182	0.00013	3.037	712.45	135
M,1	CP6	3.30267	0.34133	10.33508	0.01022	3.644	493.24	120
M,2	CP7	3.50000	0.24700	7.05714	0.00317	3.747	387.70	105
M,1	CP8	2.94600	0.58200	19.75560	0.00569	3.528	1022.59	105
F,1	CP9	2.82000	0.27800	9.85816	0.00249	3.098	577.90	150
F,1	CP10	2.37800						
F,1	CP11	2.41033	0.49767	20.64721	0.00866	2.908	410.65	45
Ave:		**3.08085**	**0.33037**	**11.11226**	**0.00456**	**3.4815**	**543.86**	**102**
SD:		**0.47593**	**0.20657**	**7.84352**	**0.00334**	**0.3770**	**197.78**	**46**
M	CON1	3.14367	0.33233	10.57152	0.00428	3.476	527.53	120
M	CON2	3.22500	0.04100	1.27132	0.00077	3.266	383.88	180
M	CON3	3.14400	0.21300	6.77481	0.00407	3.357	512.48	45
M	CON4	4.12800	0.13000	3.14922	0.00157	4.258	305.69	150
M	CON5	3.06500	0.22200	7.24307	0.00242	3.287	559.74	90
M	CON6	3.40333	0.26067	7.65916	0.00370	3.664	567.31	120
M	CON7	3.37100	0.22700	6.73391	0.00275	3.598	524.98	75
M	CON8	2.68600	0.14200	5.28667	0.00198	2.828	1186.96	90
F	CON9	3.04400						
F	CON10	2.85933	0.32467	11.35463	0.00472	3.184	398.51	60
F	CON11	3.08433	0.01667	0.54037	0.00031	3.101	336.11	165
Ave:		**3.19579**	**0.19093**	**6.05847**	**0.00266**	**3.4019**	**530.32**	**110**
SD:		**0.37077**	**0.10773**	**3.58666**	**0.00152**	**0.3868**	**249.61**	**45**

Table 4: Carotid artery dimensions, distensibility, compliance and IMT values

Gender, GMFCS	ID	Compliance	Distensibility	IMTf Mean	Wall/Lumen Ratio	Mean Carotid Diameter (mm)	Carotid PP
M,2	CP1	0.29972	0.01249	0.42580	0.07162	5.94568	22.14302
M,1	CP2	0.14842	0.00981	0.40970	0.08484	4.82923	47.66781
M,2	CP3	0.06532	0.00431	0.43090	0.09278	4.64441	58.18537
M,2	CP4	0.13371	0.00523	0.38990	0.06483	6.01453	45.75242
M,1	CP5	0.12662	0.00699	0.49100	0.09450	5.19562	50.96250
M,1	CP6	0.17060	0.00629	0.38980	0.06269	6.21834	37.93701
M,2	CP7	0.24778	0.00745			6.88343	34.49301
M,1	CP8	0.15678	0.00525	0.45570	0.06950	6.55661	55.64066
F,1	CP9	0.18364	0.00941	0.46933	0.08692	5.39956	36.65875
F,1	CP10	0.14081	0.00719	0.37390	0.07071	5.28800	34.76927
F,1	CP11	0.14628	0.00871	0.37667	0.07576	4.97193	37.72544
Ave:		**0.16542**	**0.00756**	**0.42127**	**0.07741**	**5.63158**	**41.99411**
SD:		**0.06249**	**0.00240**	**0.04057**	**0.01151**	**0.73673**	**10.68088**
M	CON1	0.17610	0.00784	0.45963	0.08057	5.70468	34.64816
M	CON2	0.15487	0.00614	0.40670	0.06729	6.04403	49.85659
M	CON3	0.20596	0.01098	0.43170	0.08266	5.22248	27.44650
M	CON4	0.16833	0.00764	0.37700	0.06645	5.67341	41.24305
M	CON5	0.14398	0.00685	0.38167	0.07058	5.40756	27.95604
M	CON6	0.16960	0.00649	0.38850	0.06319	6.14781	52.65831
M	CON7	0.22610	0.00994	0.38800	0.06761	5.73895	34.02295
M	CON8	0.24795	0.01107	0.47600	0.08098	5.87783	43.61945
F	CON9			0.44340	0.07955	5.57358	16.05406
F	CON10	0.16628	0.00762	0.39560	0.07107	5.56605	33.64877
F	CON11	0.19567	0.00781	0.38280	0.06327	6.05006	36.21288
Ave:		**0.18548**	**0.00824**	**0.41191**	**0.07211**	**5.72786**	**36.12425**
SD:		**0.03289**	**0.00179**	**0.03487**	**0.00744**	**0.28535**	**10.49713**

Table 5: Pulse wave velocity and pulse transit times

Gender, GMFCS	ID	Exact Age (yrs)	Central PTT	Central PWV	Peripheral PTT	Peripheral PWV
M,2	CP1	9.91	0.1143	3.1507	0.1197	5.7308
M,1	CP2	15.32	0.0891	4.2079	0.1493	5.8024
M,2	CP3	13.71	0.0698	5.2971	0.1027	6.4720
M,2	CP4	14.76	0.0875	4.6302	0.0972	8.0299
M,1	CP5	12.41	0.0764	4.7759	0.1256	5.7747
M,1	CP6	15.03	0.0879	4.4956	0.1000	8.5002
M,2	CP7	10.28	0.1070	3.3173	0.0756	8.7342
M,1	CP8	13.38	0.0783	4.5424	0.0987	7.3949
F,1	CP9	11.91	0.0937	4.3215	0.1424	5.7243
F,1	CP10	16.48	0.0854	4.6388	0.1455	5.6412
F,1	CP11	11.48	0.0942	3.8766	0.0637	10.7527
Ave:		**13.15181818**	**0.0894**	**4.2958**	**0.1109**	**7.1416**
SD:		**2.142413677**	**0.0129**	**0.6337**	**0.0282**	**1.6922**
M	CON1	11.67	0.0927	3.9364	0.0845	8.8226
M	CON2	12.34	0.0831	5.0543	0.1099	8.3227
M	CON3	13.64	0.0775	5.0416	0.1092	8.4738
M	CON4	14.7	0.1079	3.9497	0.1114	7.7555
M	CON5	9.35				
M	CON6	12.14	0.1202	3.4519	0.0899	9.0089
M	CON7	14.33	0.0855	5.0912	0.1174	7.1590
M	CON8	9.92	0.0832	4.0924	0.1139	6.1909
F	CON9	11.43	0.1835	1.9668	0.1218	6.3975
F	CON10	16.83	0.1108	4.2034	0.1047	8.0778
F	CON11	10.52	0.0868	3.9163	0.1187	6.0686
Ave:		**12.44272727**	**0.1031**	**4.0704**	**0.1081**	**7.6277**
SD:		**2.251435502**	**0.0316**	**0.9331**	**0.0122**	**1.1046**

Table 6: Physical activity

Gender, GMFCS	ID	LIGHT	MED.	HARD	mod/vig in 1 week	mod/vig each day	420 min ? y/n	TOTAL / week	GMFCS L I	GMFCS L II
M,2	CP1	75	220	0	220	31.429	n	295	50	295
M,1	CP2	35	15	0	15	2.143	n	50	350	100
M,2	CP3	0	100	0	100	14.286	n	100	355	160
M,2	CP4	150	10	0	10	1.429	n	160	1100	480
M,1	CP5	60	230	60	290	41.429	n	350	200	
M,1	CP6	160	150	45	195	27.857	n	355	760	
M,2	CP7	0	480	0	480	68.571	y	480	410	
M,1	CP8	400	660	40	700	100.000	y	1100		
F,1	CP9	60	140	0	140	20.000	n	200		
F,1	CP10	70	690	0	690	98.571	y	760		
F,1	CP11	0	140	270	410	58.571	n	410		
Ave:		**91.82**	**257.73**	**37.73**	**295.455**	**42.208**		**387.27**	**460.71**	**258.75**
SD:		**115.89**	**241.82**	**80.29**	**245.931**	**35.133**	27%	**308.22**	**356.12**	**168.54**
M	CON 1	120	285	120	405	57.857	n	525		
M	CON 2	0	180	240	420	60.000	y	420		
M	CON 3	0	280	360	640	91.429	y	640		
M	CON 4	135	5	480	485	69.286	n	620		
M	CON 5	140	420	240	660	94.286	y	800		
M	CON 6	85	0	0	0	0.000	n	85		
M	CON 7	300	0	0	0	0.000	n	300		
M	CON 8	0	225	445	670	95.714	y	670		
F	CON 9	75	95	120	215	30.714	n	290		
F	CON 10	80	45	0	45	6.429	n	125		
F	CON 11	5	210	150	360	51.429	n	365		
Ave:		**85.455**	**158.64**	**195.91**	**354.55**	**50.649**		**440.00**		
SD:		**90.096**	**140.50**	**173.88**	**257.85**	**36.836**	36%	**231.41**		

Table 7: FMD reliability data – visit 1 and 2

VISIT 1				VISIT 2			
Participant ID	Absolute	Relative	Normalized		Absolute	Relative	Normalized
6CP	0.3337	8.7523	0.0046	100CP	0.3290	9.1951	0.0031
14CON	0.2220	7.2431	0.0024	101CON	0.3683	11.2893	0.0072
9CON	0.3323	10.5715	0.0043	102CON	0.6280	20.4161	0.0056
7CP	0.2780	9.8582	0.0025	103CP	0.2977	9.5610	0.0037
13CON	0.3247	11.3546	0.0047	104CON	0.4197	14.2340	0.0060
11CON	0.2130	6.7748	0.0041	105CON	0.4700	16.2237	0.0069
17CON	0.0167	0.5404	0.0003	106CON	0.2340	7.2943	0.0041
18CP	0.3413	10.3351	0.0102	107CP	0.3103	9.4739	0.0104
19CON	0.2607	7.6592	0.0037	108CON	0.1517	4.4577	0.0016
20CON	0.2270	6.7339	0.0027	109CON	0.4460	15.1907	0.0037
8CP	0.0167	0.5518	0.0001	110CP	0.3387	11.2352	0.0061
22CON	0.1420	5.2867	0.0020	111CON	0.4273	13.6849	0.0015
Ave:	**0.2257**	**7.1385**	**0.0035**		**0.3684**	**11.8547**	**0.0050**
SD:	**0.1147**	**3.5765**	**0.0026**		**0.1227**	**4.3353**	**0.0026**

Table 8: Coefficient of Variation calculations

	CV's for each Pair:		
6CP	0.3337	8.7523	0.0046
100CP	0.3290	9.1951	0.0031
AVE:	**0.3313**	**8.9737**	**0.0038**
SD:	**0.0033**	**0.3131**	**0.0011**
CV:	0.9959	3.4891	28.3838
14CON	0.2220	7.2431	0.0024
101CON	0.3683	11.2893	0.0072
AVE:	**0.2952**	**9.2662**	**0.0048**
SD:	**0.1035**	**2.8611**	**0.0034**
CV:	35.0559	30.8772	70.6276
9CON	0.3323	10.5715	0.0043
102CON	0.6280	20.4161	0.0056
AVE:	**0.4802**	**15.4938**	**0.0049**
SD:	**0.2091**	**6.9612**	**0.0009**
CV:	43.5407	44.9288	18.8228
7CP	0.2780	9.8582	0.0025
103CP	0.2977	9.5610	0.0037
AVE:	**0.2878**	**9.7096**	**0.0031**
SD:	**0.0139**	**0.2101**	**0.0009**
CV:	4.8314	2.1639	28.5080

13CON	0.3247	11.3546	0.0047
104CON	0.4197	14.2340	0.0060
AVE:	**0.3722**	**12.7943**	**0.0054**
SD:	**0.0672**	**2.0360**	**0.0009**
CV:	18.0497	15.9137	17.4372
11CON	0.2130	6.7748	0.0041
105CON	0.4700	16.2237	0.0069
AVE:	**0.3415**	**11.4992**	**0.0055**
SD:	**0.1817**	**6.6814**	**0.0020**
CV:	53.2142	58.1026	36.8846
17CON	0.0167	0.5404	0.0003
106CON	0.2340	7.2943	0.0041
AVE:	**0.1253**	**3.9173**	**0.0022**
SD:	**0.1537**	**4.7757**	**0.0027**
CV:	122.6153	121.9133	121.2427
18CP	0.3413	10.3351	0.0102
107CP	0.3103	9.4739	0.0104
AVE:	**0.3258**	**9.9045**	**0.0103**
SD:	**0.0219**	**0.6089**	**0.0001**
CV:	6.7275	6.1482	0.9105
19CON	0.2607	7.6592	0.0037
108CON	0.1517	4.4577	0.0016
AVE:	**0.2062**	**6.0584**	**0.0026**
SD:	**0.0771**	**2.2638**	**0.0015**
CV:	37.3846	37.3653	56.2553
20CON	0.2270	6.7339	0.0027
109CON	0.4460	15.1907	0.0037
AVE:	**0.3365**	**10.9623**	**0.0032**
SD:	**0.1549**	**5.9799**	**0.0007**
CV:	46.0197	54.5494	21.6209
8CP	0.0167	0.5518	0.0001
110CP	0.3387	11.2352	0.0061
AVE:	**0.1777**	**5.8935**	**0.0031**
SD:	**0.2277**	**7.5543**	**0.0043**
CV:	128.1548	128.1799	135.3708
22CON	0.1420	5.2867	0.0020
111CON	0.4273	13.6849	0.0015
AVE:	**0.2847**	**9.4858**	**0.0017**
SD:	**0.2018**	**5.9384**	**0.0003**

CV:	70.8763	62.6035	18.5698

Table 9: Averaging all FMD CVs from each pair

Average CVs		
Absolute	**Relative**	**Normalized**
0.995925044	3.489054737	28.38380923
35.05588672	30.87719575	70.62764065
43.54069524	44.92879263	18.82279468
4.831418655	2.163853644	28.50804926
18.04974766	15.91365141	17.43715542
53.21418529	58.1026037	36.88463656
122.6153248	121.9133015	121.2427153
6.727460936	6.148213935	0.91050996
37.38462691	37.36529833	56.2553276
46.01972811	54.54940493	21.62092736
128.1548125	128.1799332	135.3708469
70.8762769	62.60354515	18.56978547
47.28884073	**47.18623741**	**46.21951653**

*Note: Grey boxes = insufficient data for analysis or significant outlier and removed

```
GET
  FILE='/Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP IN, r
DATASET NAME DataSet1 WINDOW=FRONT.

SAVE OUTFILE='/Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BI
    'IN, WL ratio, PP_FINAL_June15.sav'
  /COMPRESSED.
SAVE OUTFILE='/Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BI
    'IN, WL ratio, PP_FINAL_June15.sav'
 /COMPRESSED.
EXAMINE VARIABLES=Gender AGE SeatedHt StandingHt Weight BMI BMIPercentile Waist SBP DBP MAP HR
MediumPA HardPA WLRatio BrachialPP CarotidPP BY Group
  /PLOT BOXPLOT STEMLEAF HISTOGRAM NPPLOT
  /COMPARE GROUPS
  /STATISTICS DESCRIPTIVES
  /CINTERVAL 95
  /MISSING LISTWISE
  /NOTOTAL.


T-TEST GROUPS=Group(0 1)
  /MISSING=ANALYSIS
  /VARIABLES=AGE SeatedHt StandingHt Weight BMI BMIPercentile Waist SBP DBP MAP HR cPTT cPWV pI
WLRatio BrachialPP CarotidPP
  /CRITERIA=CI(.95).
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T-Test

Notes

Output Created		**15-Jun-2011 13:27:25**
Comments		
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	Active Dataset	**DataSet1**
	Filter	**<none>**
	Weight	**<none>**
	Split File	**<none>**
	N of Rows in Working Data File	**26**
Missing Value Handling	**Definition of Missing**	**User defined missing values are treated as missing.**
	Cases Used	**Statistics for each analysis are based on the cases with no missing or out-of-range data for any variable in the analysis.**
Syntax		**T-TEST GROUPS=Group(0 1) /MISSING=ANALYSIS /VARIABLES=AGE SeatedHt StandingHt Weight BMI BMIPercentile Waist SBP DBP MAP HR cPTT cPWV pPTT pPWV Compliance Distensibility SI IMTf Absolute Relative Normalized CarDiam BrachialDiam RHMax AveSR PeakTime WHR GMFCS APHV TPHV LightPA MediumPA HardPA WLRatio BrachialPP CarotidPP /CRITERIA=CI(.95).**
Resources	**Processor Time**	**00 00:00:00.026**
	Elapsed Time	**00 00:00:00.000**

[DataSet1] /Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP II
, WL ratio, PP_FINAL_June15.sav

Group Statistics

	Group	N	Mean	Std. Deviation	Std. Error Mean
AGE	CP	11	13.1518	2.14241	.64596
	CON	11	12.4427	2.25144	.67883
SeatedHt	CP	11	77.0909	5.16090	1.55607
	CON	11	80.7591	8.09928	2.44203
StandingHt	CP	11	149.8864	10.76773	3.24659
	CON	11	157.7727	14.85888	4.48012
Weight	CP	11	41.3545	8.35959	2.52051
	CON	11	49.2909	14.17473	4.27384
BMI	CP	11	18.4091	3.16779	.95513
	CON	11	19.4727	3.65023	1.10059
BMIPercentile	CP	11	38.0000	32.61288	9.83315
	CON	11	56.8182	30.65556	9.24300
Waist	CP	11	67.28	7.232	2.180
	CON	11	69.77	8.829	2.662
SBP	CP	11	106.4528	11.67750	3.52090
	CON	11	112.5455	7.71406	2.32588
DBP	CP	11	61.8128	5.50152	1.65877
	CON	11	64.7000	4.57865	1.38051
MAP	CP	11	81.1356	6.08272	1.83401
	CON	11	84.1909	3.05007	.91963
HR	CP	11	73.8921	12.88099	3.88376
	CON	11	68.4444	9.81730	2.96003
cPTT	CP	11	.0894	.01291	.00389
	CON	10	.1031	.03157	.00998
cPWV	CP	11	4.2958	.63373	.19108
	CON	10	4.0704	.93306	.29506
pPTT	CP	11	.1109	.02819	.00850
	CON	10	.1081	.01219	.00385
pPWV	CP	11	7.1416	1.69222	.51022
	CON	10	7.6277	1.10462	.34931
Compliance	CP	11	.1654	.06249	.01884
	CON	10	.1855	.03289	.01040
Distensibility	CP	11	.0076	.00240	.00072
	CON	10	.0082	.00179	.00057
SI	CP	11	4.1622	1.24914	.37663
	CON	11	3.4200	1.04119	.31393
IMTf	CP	10	.4213	.04057	.01283
	CON	11	.4119	.03487	.01051
Absolute	CP	10	.3304	.20657	.06532
	CON	10	.1909	.10773	.03407
Relative	CP	10	11.1221	7.84352	2.48034
	CON	10	6.0585	3.58666	1.13420
Normalized	CP	10	.0046	.00334	.00106

Group Statistics

	Group	N	Mean	Std. Deviation	Std. Error Mean
Normalized	CON	10	.0027	.00152	.00048
CarDiam	CP	11	5.6316	.73673	.22213
	CON	11	5.7279	.28535	.08604
BrachialDiam	CP	10	3.1511	.43738	.13831
	CON	11	3.1958	.37077	.11179
RHMax	CP	10	3.4815	.37699	.11922
	CON	10	3.4019	.38681	.12232
AveSR	CP	10	543.8641	197.78114	62.54389
	CON	10	530.3180	249.61187	78.93420
PeakTime	CP	10	102.0000	45.71652	14.45683
	CON	10	109.5000	45.30453	14.32655
WHR	CP	11	.4508	.05681	.01713
	CON	11	.4434	.04985	.01503
GMFCS	CP	11	1.3636	.50452	.15212
	CON	0[a]	.	.	.
APHV	CP	11	14.0182	1.30217	.39262
	CON	11	13.0818	.87729	.26451
TPHV	CP	11	-.8636	1.66929	.50331
	CON	11	-.6636	2.13975	.64516
LightPA	CP	11	91.8182	115.89376	34.94328
	CON	11	85.4545	90.09591	27.16494
MediumPA	CP	11	257.7273	241.81981	72.91141
	CON	11	158.6364	140.50073	42.36256
HardPA	CP	11	37.7273	80.29208	24.20897
	CON	11	195.9091	173.87953	52.42665
WLRatio	CP	11	.0704	.02577	.00777
	CON	11	.0721	.00744	.00224
BrachialPP	CP	11	44.6401	12.98550	3.91528
	CON	11	47.8455	9.92546	2.99264
CarotidPP	CP	11	41.9941	10.68088	3.22041
	CON	11	36.1242	10.49713	3.16500

a. t cannot be computed because at least one of the groups is empty.

Independent Samples Test

		Levene's Test for Equality of Variances	
		F	Sig.
AGE	Equal variances assumed	.000	.989
	Equal variances not assumed		
SeatedHt	Equal variances assumed	3.309	.084
	Equal variances not assumed		
StandingHt	Equal variances assumed	2.401	.137
	Equal variances not assumed		
Weight	Equal variances assumed	6.266	.021
	Equal variances not assumed		
BMI	Equal variances assumed	.231	.636
	Equal variances not assumed		
BMIPercentile	Equal variances assumed	.084	.775
	Equal variances not assumed		
Waist	Equal variances assumed	.085	.774
	Equal variances not assumed		
SBP	Equal variances assumed	.293	.594
	Equal variances not assumed		
DBP	Equal variances assumed	1.032	.322
	Equal variances not assumed		
MAP	Equal variances assumed	1.849	.189
	Equal variances not assumed		
HR	Equal variances assumed	.907	.352
	Equal variances not assumed		
cPTT	Equal variances assumed	3.278	.086
	Equal variances not assumed		
cPWV	Equal variances assumed	.402	.534
	Equal variances not assumed		

Independent Samples Test

		t-test for Equality of Means				
		t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference
AGE	Equal variances assumed	.757	20	.458	.70909	.93706
	Equal variances not assumed	.757	19.951	.458	.70909	.93706
SeatedHt	Equal variances assumed	-1.267	20	.220	-3.66818	2.89566
	Equal variances not assumed	-1.267	16.971	.222	-3.66818	2.89566
StandingHt	Equal variances assumed	-1.425	20	.169	-7.88636	5.53280
	Equal variances not assumed	-1.425	18.233	.171	-7.88636	5.53280
Weight	Equal variances assumed	-1.600	20	.125	-7.93636	4.96172
	Equal variances not assumed	-1.600	16.205	.129	-7.93636	4.96172
BMI	Equal variances assumed	-.730	20	.474	-1.06364	1.45724
	Equal variances not assumed	-.730	19.611	.474	-1.06364	1.45724
BMIPercentile	Equal variances assumed	-1.394	20	.178	-18.81818	13.49533
	Equal variances not assumed	-1.394	19.924	.179	-18.81818	13.49533
Waist	Equal variances assumed	-.724	20	.478	-2.491	3.441
	Equal variances not assumed	-.724	19.253	.478	-2.491	3.441
SBP	Equal variances assumed	-1.444	20	.164	-6.09261	4.21977
	Equal variances not assumed	-1.444	17.332	.167	-6.09261	4.21977
DBP	Equal variances assumed	-1.338	20	.196	-2.88721	2.15809
	Equal variances not assumed	-1.338	19.362	.196	-2.88721	2.15809
MAP	Equal variances assumed	-1.489	20	.152	-3.05530	2.05166
	Equal variances not assumed	-1.489	14.730	.158	-3.05530	2.05166
HR	Equal variances assumed	1.116	20	.278	5.44770	4.88317
	Equal variances not assumed	1.116	18.687	.279	5.44770	4.88317
cPTT	Equal variances assumed	-1.325	19	.201	-.01370	.01034
	Equal variances not assumed	-1.279	11.701	.226	-.01370	.01072
cPWV	Equal variances assumed	.653	19	.521	.22541	.34508
	Equal variances not assumed	.641	15.654	.531	.22541	.35152

Independent Samples Test

		t-test for Equality of Means	
		95% Confidence Interval of the Difference	
		Lower	Upper
AGE	Equal variances assumed	-1.24558	2.66376
	Equal variances not assumed	-1.24589	2.66407
SeatedHt	Equal variances assumed	-9.70842	2.37206
	Equal variances not assumed	-9.77828	2.44191
StandingHt	Equal variances assumed	-19.42758	3.65485
	Equal variances not assumed	-19.49972	3.72699
Weight	Equal variances assumed	-18.28634	2.41361
	Equal variances not assumed	-18.44392	2.57119
BMI	Equal variances assumed	-4.10339	1.97612
	Equal variances not assumed	-4.10726	1.97999
BMIPercentile	Equal variances assumed	-46.96895	9.33259
	Equal variances not assumed	-46.97585	9.33948
Waist	Equal variances assumed	-9.669	4.687
	Equal variances not assumed	-9.686	4.705
SBP	Equal variances assumed	-14.89489	2.70967
	Equal variances not assumed	-14.98259	2.79736
DBP	Equal variances assumed	-7.38890	1.61448
	Equal variances not assumed	-7.39844	1.62401
MAP	Equal variances assumed	-7.33499	1.22439
	Equal variances not assumed	-7.43531	1.32471
HR	Equal variances assumed	-4.73842	15.63382
	Equal variances not assumed	-4.78452	15.67991
cPTT	Equal variances assumed	-.03534	.00794
	Equal variances not assumed	-.03711	.00971
cPWV	Equal variances assumed	-.49686	.94768
	Equal variances not assumed	-.52113	.97195

Independent Samples Test

		Levene's Test for Equality of Variances	
		F	Sig.
pPTT	Equal variances assumed	7.857	.011
	Equal variances not assumed		
pPWV	Equal variances assumed	2.302	.146
	Equal variances not assumed		
Compliance	Equal variances assumed	1.374	.256
	Equal variances not assumed		
Distensibility	Equal variances assumed	.578	.457
	Equal variances not assumed		
SI	Equal variances assumed	.293	.594
	Equal variances not assumed		
IMTf	Equal variances assumed	.210	.652
	Equal variances not assumed		
Absolute	Equal variances assumed	1.913	.184
	Equal variances not assumed		
Relative	Equal variances assumed	4.395	.050
	Equal variances not assumed		
Normalized	Equal variances assumed	5.520	.030
	Equal variances not assumed		
CarDiam	Equal variances assumed	13.569	.001
	Equal variances not assumed		
BrachialDiam	Equal variances assumed	1.111	.305
	Equal variances not assumed		
RHMax	Equal variances assumed	.022	.883
	Equal variances not assumed		
AveSR	Equal variances assumed	.003	.958
	Equal variances not assumed		

Independent Samples Test

		t-test for Equality of Means				
		t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference
pPTT	Equal variances assumed	.290	19	.775	.00280	.00966
	Equal variances not assumed	.300	13.883	.768	.00280	.00933
pPWV	Equal variances assumed	-.771	19	.450	-.48615	.63093
	Equal variances not assumed	-.786	17.339	.442	-.48615	.61834
Compliance	Equal variances assumed	-.906	19	.376	-.02006	.02214
	Equal variances not assumed	-.932	15.433	.366	-.02006	.02152
Distensibility	Equal variances assumed	-.730	19	.474	-.00068	.00093
	Equal variances not assumed	-.740	18.356	.468	-.00068	.00092
SI	Equal variances assumed	1.514	20	.146	.74221	.49031
	Equal variances not assumed	1.514	19.372	.146	.74221	.49031
IMTf	Equal variances assumed	.569	19	.576	.00936	.01646
	Equal variances not assumed	.564	17.888	.580	.00936	.01659
Absolute	Equal variances assumed	1.893	18	.075	.13943	.07367
	Equal variances not assumed	1.893	13.558	.080	.13943	.07367
Relative	Equal variances assumed	1.857	18	.080	5.06359	2.72736
	Equal variances not assumed	1.857	12.606	.087	5.06359	2.72736
Normalized	Equal variances assumed	1.641	18	.118	.00191	.00116
	Equal variances not assumed	1.641	12.557	.126	.00191	.00116
CarDiam	Equal variances assumed	-.404	20	.690	-.09628	.23821
	Equal variances not assumed	-.404	12.934	.693	-.09628	.23821
BrachialDiam	Equal variances assumed	-.253	19	.803	-.04465	.17639
	Equal variances not assumed	-.251	17.773	.805	-.04465	.17784
RHMax	Equal variances assumed	.466	18	.647	.07960	.17081
	Equal variances not assumed	.466	17.988	.647	.07960	.17081
AveSR	Equal variances assumed	.135	18	.894	13.54608	100.70922
	Equal variances not assumed	.135	17.106	.895	13.54608	100.70922

Independent Samples Test

		t-test for Equality of Means	
		95% Confidence Interval of the Difference	
		Lower	Upper
pPTT	Equal variances assumed	-.01741	.02302
	Equal variances not assumed	-.01723	.02284
pPWV	Equal variances assumed	-1.80670	.83440
	Equal variances not assumed	-1.78879	.81650
Compliance	Equal variances assumed	-.06640	.02628
	Equal variances not assumed	-.06582	.02570
Distensibility	Equal variances assumed	-.00263	.00127
	Equal variances not assumed	-.00261	.00125
SI	Equal variances assumed	-.28056	1.76497
	Equal variances not assumed	-.28269	1.76710
IMTf	Equal variances assumed	-.02510	.04382
	Equal variances not assumed	-.02550	.04423
Absolute	Equal variances assumed	-.01535	.29422
	Equal variances not assumed	-.01907	.29793
Relative	Equal variances assumed	-.66638	10.79357
	Equal variances not assumed	-.84728	10.97447
Normalized	Equal variances assumed	-.00053	.00434
	Equal variances not assumed	-.00061	.00442
CarDiam	Equal variances assumed	-.59319	.40062
	Equal variances not assumed	-.61118	.41861
BrachialDiam	Equal variances assumed	-.41383	.32453
	Equal variances not assumed	-.41862	.32932
RHMax	Equal variances assumed	-.27925	.43845
	Equal variances not assumed	-.27927	.43847
AveSR	Equal variances assumed	-198.03613	225.12830
	Equal variances not assumed	-198.83170	225.92386

Independent Samples Test

		Levene's Test for Equality of Variances	
		F	Sig.
PeakTime	Equal variances assumed	.007	.932
	Equal variances not assumed		
WHR	Equal variances assumed	.313	.582
	Equal variances not assumed		
APHV	Equal variances assumed	2.160	.157
	Equal variances not assumed		
TPHV	Equal variances assumed	.192	.666
	Equal variances not assumed		
LightPA	Equal variances assumed	.237	.631
	Equal variances not assumed		
MediumPA	Equal variances assumed	2.720	.115
	Equal variances not assumed		
HardPA	Equal variances assumed	8.441	.009
	Equal variances not assumed		
WLRatio	Equal variances assumed	2.121	.161
	Equal variances not assumed		
BrachialPP	Equal variances assumed	.502	.487
	Equal variances not assumed		
CarotidPP	Equal variances assumed	.139	.713
	Equal variances not assumed		

Independent Samples Test

		t-test for Equality of Means				
		t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference
PeakTime	Equal variances assumed	-.368	18	.717	-7.50000	20.35313
	Equal variances not assumed	-.368	17.999	.717	-7.50000	20.35313
WHR	Equal variances assumed	.323	20	.750	.00736	.02279
	Equal variances not assumed	.323	19.668	.750	.00736	.02279
APHV	Equal variances assumed	1.978	20	.062	.93636	.47341
	Equal variances not assumed	1.978	17.527	.064	.93636	.47341
TPHV	Equal variances assumed	-.244	20	.809	-.20000	.81826
	Equal variances not assumed	-.244	18.882	.810	-.20000	.81826
LightPA	Equal variances assumed	.144	20	.887	6.36364	44.26022
	Equal variances not assumed	.144	18.853	.887	6.36364	44.26022
MediumPA	Equal variances assumed	1.175	20	.254	99.09091	84.32474
	Equal variances not assumed	1.175	16.061	.257	99.09091	84.32474
HardPA	Equal variances assumed	-2.739	20	.013	-158.18182	57.74624
	Equal variances not assumed	-2.739	14.079	.016	-158.18182	57.74624
WLRatio	Equal variances assumed	-.215	20	.832	-.00174	.00809
	Equal variances not assumed	-.215	11.656	.834	-.00174	.00809
BrachialPP	Equal variances assumed	-.650	20	.523	-3.20540	4.92801
	Equal variances not assumed	-.650	18.711	.523	-3.20540	4.92801
CarotidPP	Equal variances assumed	1.300	20	.208	5.86986	4.51534
	Equal variances not assumed	1.300	19.994	.208	5.86986	4.51534

Independent Samples Test

		t-test for Equality of Means	
		95% Confidence Interval of the Difference	
		Lower	Upper
PeakTime	Equal variances assumed	-50.26034	35.26034
	Equal variances not assumed	-50.26060	35.26060
WHR	Equal variances assumed	-.04018	.05489
	Equal variances not assumed	-.04023	.05495
APHV	Equal variances assumed	-.05115	1.92388
	Equal variances not assumed	-.06016	1.93289
TPHV	Equal variances assumed	-1.90687	1.50687
	Equal variances not assumed	-1.91337	1.51337
LightPA	Equal variances assumed	-85.96156	98.68883
	Equal variances not assumed	-86.32284	99.05011
MediumPA	Equal variances assumed	-76.80741	274.98923
	Equal variances not assumed	-79.61451	277.79633
HardPA	Equal variances assumed	-278.63836	-37.72527
	Equal variances not assumed	-281.96990	-34.39373
WLRatio	Equal variances assumed	-.01861	.01513
	Equal variances not assumed	-.01942	.01594
BrachialPP	Equal variances assumed	-13.48504	7.07425
	Equal variances not assumed	-13.53062	7.11983
CarotidPP	Equal variances assumed	-3.54896	15.28869
	Equal variances not assumed	-3.54915	15.28888

```
CORRELATIONS
  /VARIABLES=AGE SeatedHt StandingHt Weight BMI BMIPercentile Waist SBP DBP MAP HR cPTT cPWV pl
WLRatio BrachialPP CarotidPP
  /PRINT=TWOTAIL NOSIG
  /MISSING=PAIRWISE.
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Correlations

Notes

Output Created		15-Jun-2011 14:27:44
Comments		
Input	Data	/Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP IN, WL ratio, PP_FINAL_June15.sav
	Active Dataset	DataSet1
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	26
Missing Value Handling	Definition of Missing	User-defined missing values are treated as missing.
	Cases Used	Statistics for each pair of variables are based on all the cases with valid data for that pair.
Syntax		CORRELATIONS /VARIABLES=AGE SeatedHt StandingHt Weight BMI BMIPercentile Waist SBP DBP MAP HR cPTT cPWV pPTT pPWV Compliance Distensibility SI IMTf Absolute Relative Normalized CarDiam BrachialDiam RHMax AveSR PeakTime WHR GMFCS APHV TPHV LightPA MediumPA HardPA WLRatio BrachialPP CarotidPP /PRINT=TWOTAIL NOSIG /MISSING=PAIRWISE.
Resources	Processor Time	00 00:00:00.106
	Elapsed Time	00 00:00:00.000

[DataSet1] /Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP II
, WL ratio, PP_FINAL_June15.sav

Correlations

		AGE	SeatedHt	StandingHt	Weight	BMI	BMIPercentile	Waist
AGE	Pearson Correlation	1	.592**	.644**	.475*	.105	-.254	.211
	Sig. (2-tailed)		.004	.001	.026	.642	.254	.345
	N	22	22	22	22	22	22	22
SeatedHt	Pearson Correlation	.592**	1	.918**	.817**	.339	.119	.476*
	Sig. (2-tailed)	.004		.000	.000	.123	.599	.025
	N	22	22	22	22	22	22	22
StandingHt	Pearson Correlation	.644**	.918**	1	.740**	.160	-.062	.353
	Sig. (2-tailed)	.001	.000		.000	.478	.785	.107
	N	22	22	22	22	22	22	22
Weight	Pearson Correlation	.475*	.817**	.740**	1	.775**	.553**	.854**
	Sig. (2-tailed)	.026	.000	.000		.000	.008	.000
	N	22	22	22	22	22	22	22
BMI	Pearson Correlation	.105	.339	.160	.775**	1	.866**	.944**
	Sig. (2-tailed)	.642	.123	.478	.000		.000	.000
	N	22	22	22	22	22	22	22
BMIPercentile	Pearson Correlation	-.254	.119	-.062	.553**	.866**	1	.738**
	Sig. (2-tailed)	.254	.599	.785	.008	.000		.000
	N	22	22	22	22	22	22	22
Waist	Pearson Correlation	.211	.476*	.353	.854**	.944**	.738**	1
	Sig. (2-tailed)	.345	.025	.107	.000	.000	.000	
	N	22	22	22	22	22	22	22
SBP	Pearson Correlation	.240	.486*	.490*	.666**	.489*	.469*	.600**
	Sig. (2-tailed)	.283	.022	.021	.001	.021	.028	.003
	N	22	22	22	22	22	22	22
DBP	Pearson Correlation	.182	.034	.165	.184	.108	.013	.156
	Sig. (2-tailed)	.416	.879	.464	.414	.633	.954	.489
	N	22	22	22	22	22	22	22
MAP	Pearson Correlation	.286	.433*	.474*	.499*	.264	.162	.367
	Sig. (2-tailed)	.197	.044	.026	.018	.235	.473	.093
	N	22	22	22	22	22	22	22
HR	Pearson Correlation	-.307	-.497*	-.432*	-.197	.116	.063	.069
	Sig. (2-tailed)	.165	.019	.045	.379	.608	.782	.760
	N	22	22	22	22	22	22	22
cPTT	Pearson Correlation	-.210	.219	.033	.323	.435*	.464*	.399
	Sig. (2-tailed)	.361	.340	.886	.153	.049	.034	.073
	N	21	21	21	21	21	21	21
cPWV	Pearson Correlation	.485*	.108	.318	-.025	-.338	-.442*	-.282
	Sig. (2-tailed)	.026	.640	.160	.913	.134	.045	.216
	N	21	21	21	21	21	21	21
pPTT	Pearson Correlation	.269	.310	.284	-.036	-.303	-.307	-.277
	Sig. (2-tailed)	.239	.171	.212	.877	.182	.176	.224
	N	21	21	21	21	21	21	21

Correlations

		SBP	DBP	MAP	HR	cPTT	cPWV	pPTT	pPWV
AGE	Pearson Correlation	.240	.182	.286	-.307	-.210	.485*	.269	.006
	Sig. (2-tailed)	.283	.416	.197	.165	.361	.026	.239	.981
	N	22	22	22	22	21	21	21	21
SeatedHt	Pearson Correlation	.486*	.034	.433*	-.497*	.219	.108	.310	.067
	Sig. (2-tailed)	.022	.879	.044	.019	.340	.640	.171	.772
	N	22	22	22	22	21	21	21	21
StandingHt	Pearson Correlation	.490*	.165	.474*	-.432*	.033	.318	.284	.130
	Sig. (2-tailed)	.021	.464	.026	.045	.886	.160	.212	.573
	N	22	22	22	22	21	21	21	21
Weight	Pearson Correlation	.666**	.184	.499*	-.197	.323	-.025	-.036	.290
	Sig. (2-tailed)	.001	.414	.018	.379	.153	.913	.877	.202
	N	22	22	22	22	21	21	21	21
BMI	Pearson Correlation	.489*	.108	.264	.116	.435*	-.338	-.303	.285
	Sig. (2-tailed)	.021	.633	.235	.608	.049	.134	.182	.211
	N	22	22	22	22	21	21	21	21
BMIPercentile	Pearson Correlation	.469*	.013	.162	.063	.464*	-.442*	-.307	.175
	Sig. (2-tailed)	.028	.954	.473	.782	.034	.045	.176	.448
	N	22	22	22	22	21	21	21	21
Waist	Pearson Correlation	.600**	.156	.367	.069	.399	-.282	-.277	.362
	Sig. (2-tailed)	.003	.489	.093	.760	.073	.216	.224	.106
	N	22	22	22	22	21	21	21	21
SBP	Pearson Correlation	1	-.003	.402	-.167	.073	.054	-.146	.350
	Sig. (2-tailed)		.989	.063	.457	.753	.815	.528	.120
	N	22	22	22	22	21	21	21	21
DBP	Pearson Correlation	-.003	1	.699**	-.096	-.319	.379	.074	-.015
	Sig. (2-tailed)	.989		.000	.672	.159	.091	.751	.948
	N	22	22	22	22	21	21	21	21
MAP	Pearson Correlation	.402	.699**	1	-.320	-.196	.340	.223	-.129
	Sig. (2-tailed)	.063	.000		.146	.395	.131	.331	.578
	N	22	22	22	22	21	21	21	21
HR	Pearson Correlation	-.167	-.096	-.320	1	.028	-.122	-.679**	.543*
	Sig. (2-tailed)	.457	.672	.146		.906	.597	.001	.011
	N	22	22	22	22	21	21	21	21
cPTT	Pearson Correlation	.073	-.319	-.196	.028	1	-.887**	-.018	.018
	Sig. (2-tailed)	.753	.159	.395	.906		.000	.938	.937
	N	21	21	21	21	21	21	21	21
cPWV	Pearson Correlation	.054	.379	.340	-.122	-.887**	1	.124	-.014
	Sig. (2-tailed)	.815	.091	.131	.597	.000		.593	.953
	N	21	21	21	21	21	21	21	21
pPTT	Pearson Correlation	-.146	.074	.223	-.679**	-.018	.124	1	-.870**
	Sig. (2-tailed)	.528	.751	.331	.001	.938	.593		.000
	N	21	21	21	21	21	21	21	21

Correlations

		Compliance	Distensibility	SI	IMTf	Absolute	Relative	Normalized
AGE	Pearson Correlation	-.436*	-.268	.207	-.330	.345	.269	.387
	Sig. (2-tailed)	.048	.239	.355	.144	.136	.252	.092
	N	21	21	22	21	20	20	20
SeatedHt	Pearson Correlation	-.008	.065	-.273	-.178	-.194	-.252	-.133
	Sig. (2-tailed)	.974	.781	.218	.440	.412	.284	.575
	N	21	21	22	21	20	20	20
StandingHt	Pearson Correlation	-.071	.012	-.166	-.185	-.152	-.211	-.008
	Sig. (2-tailed)	.760	.958	.459	.423	.523	.372	.974
	N	21	21	22	21	20	20	20
Weight	Pearson Correlation	-.017	-.195	-.061	-.397	-.110	-.198	-.008
	Sig. (2-tailed)	.941	.397	.788	.075	.644	.403	.973
	N	21	21	22	21	20	20	20
BMI	Pearson Correlation	.080	-.273	.034	-.395	.000	-.081	.015
	Sig. (2-tailed)	.729	.231	.880	.076	.999	.735	.951
	N	21	21	22	21	20	20	20
BMIPercentile	Pearson Correlation	.322	-.044	-.115	-.270	-.218	-.251	-.200
	Sig. (2-tailed)	.155	.850	.610	.236	.355	.285	.399
	N	21	21	22	21	20	20	20
Waist	Pearson Correlation	.072	-.229	.047	-.408	-.010	-.111	.051
	Sig. (2-tailed)	.755	.317	.835	.066	.967	.642	.831
	N	21	21	22	21	20	20	20
SBP	Pearson Correlation	.221	.068	.027	-.539*	-.045	-.124	.046
	Sig. (2-tailed)	.335	.770	.905	.012	.851	.602	.846
	N	21	21	22	21	20	20	20
DBP	Pearson Correlation	-.321	-.386	.436*	-.035	-.228	-.304	-.163
	Sig. (2-tailed)	.156	.084	.042	.881	.333	.192	.493
	N	21	21	22	21	20	20	20
MAP	Pearson Correlation	.001	-.154	.204	-.010	-.436	-.557*	-.395
	Sig. (2-tailed)	.996	.504	.362	.964	.054	.011	.085
	N	21	21	22	21	20	20	20
HR	Pearson Correlation	-.160	-.350	.294	-.090	.418	.434	.612**
	Sig. (2-tailed)	.488	.119	.185	.700	.067	.056	.004
	N	21	21	22	21	20	20	20
cPTT	Pearson Correlation	.449*	.230	-.545*	-.058	-.225	-.246	-.143
	Sig. (2-tailed)	.047	.328	.011	.809	.355	.309	.560
	N	20	20	21	20	19	19	19
cPWV	Pearson Correlation	-.562**	-.330	.559**	-.029	.227	.219	.169
	Sig. (2-tailed)	.010	.156	.008	.902	.350	.368	.490
	N	20	20	21	20	19	19	19
pPTT	Pearson Correlation	-.008	.300	-.344	.178	-.397	-.407	-.535*
	Sig. (2-tailed)	.974	.200	.126	.453	.092	.083	.018
	N	20	20	21	20	19	19	19

Correlations

		CarDiam	BrachialDiam	RHMax	AveSR	PeakTime	WHR	GMFCS
AGE	Pearson Correlation	-.248	.201	.370	-.229	-.179	-.273	-.365
	Sig. (2-tailed)	.267	.383	.108	.332	.450	.219	.269
	N	22	21	20	20	20	22	11
SeatedHt	Pearson Correlation	.002	.382	.329	-.227	-.014	-.204	-.475
	Sig. (2-tailed)	.994	.088	.157	.337	.955	.363	.140
	N	22	21	20	20	20	22	11
StandingHt	Pearson Correlation	-.011	.319	.276	-.234	.022	-.381	-.529
	Sig. (2-tailed)	.961	.158	.239	.321	.927	.080	.094
	N	22	21	20	20	20	22	11
Weight	Pearson Correlation	.276	.482*	.484*	-.308	.037	.299	.203
	Sig. (2-tailed)	.214	.027	.031	.186	.878	.177	.548
	N	22	21	20	20	20	22	11
BMI	Pearson Correlation	.438*	.411	.467*	-.227	.015	.816**	.686*
	Sig. (2-tailed)	.042	.064	.038	.336	.950	.000	.020
	N	22	21	20	20	20	22	11
BMIPercentile	Pearson Correlation	.451*	.342	.303	-.174	.066	.782**	.875**
	Sig. (2-tailed)	.035	.129	.194	.463	.783	.000	.000
	N	22	21	20	20	20	22	11
Waist	Pearson Correlation	.428*	.520*	.569**	-.291	-.046	.728**	.583
	Sig. (2-tailed)	.047	.016	.009	.213	.849	.000	.060
	N	22	21	20	20	20	22	11
SBP	Pearson Correlation	.334	.500*	.514*	-.208	-.262	.228	.234
	Sig. (2-tailed)	.129	.021	.020	.378	.264	.308	.489
	N	22	21	20	20	20	22	11
DBP	Pearson Correlation	.037	.225	.122	-.007	.224	.024	.189
	Sig. (2-tailed)	.871	.327	.607	.976	.342	.914	.578
	N	22	21	20	20	20	22	11
MAP	Pearson Correlation	.266	.581**	.421	.063	.081	-.001	.175
	Sig. (2-tailed)	.232	.006	.065	.792	.734	.996	.608
	N	22	21	20	20	20	22	11
HR	Pearson Correlation	.196	-.318	-.145	.137	-.147	.381	.013
	Sig. (2-tailed)	.382	.160	.543	.564	.536	.080	.970
	N	22	21	20	20	20	22	11
cPTT	Pearson Correlation	.154	.165	.346	-.320	-.175	.348	.321
	Sig. (2-tailed)	.506	.487	.147	.181	.475	.123	.336
	N	21	20	19	19	19	21	11
cPWV	Pearson Correlation	-.276	-.121	-.140	.067	.164	-.482*	-.246
	Sig. (2-tailed)	.225	.612	.567	.784	.501	.027	.465
	N	21	20	19	19	19	21	11
pPTT	Pearson Correlation	-.365	.072	-.094	.167	.114	-.467*	-.342
	Sig. (2-tailed)	.104	.761	.703	.494	.643	.033	.303
	N	21	20	19	19	19	21	11

Correlations

		APHV	TPHV	LightPA	MediumPA	HardPA	WLRatio
AGE	Pearson Correlation	.507	.841**	.257	-.166	-.268	.166
	Sig. (2-tailed)	.016	.000	.248	.461	.228	.460
	N	22	22	22	22	22	22
SeatedHt	Pearson Correlation	-.152	.780**	.158	-.500*	-.036	.053
	Sig. (2-tailed)	.499	.000	.483	.018	.873	.814
	N	22	22	22	22	22	22
StandingHt	Pearson Correlation	-.008	.748**	.166	-.398	.058	.124
	Sig. (2-tailed)	.972	.000	.461	.067	.798	.581
	N	22	22	22	22	22	22
Weight	Pearson Correlation	-.124	.626**	.094	-.501*	-.089	-.260
	Sig. (2-tailed)	.582	.002	.678	.018	.692	.243
	N	22	22	22	22	22	22
BMI	Pearson Correlation	-.164	.226	-.057	-.336	-.210	-.547**
	Sig. (2-tailed)	.466	.313	.802	.126	.349	.008
	N	22	22	22	22	22	22
BMIPercentile	Pearson Correlation	-.356	-.071	-.190	-.351	.036	-.498*
	Sig. (2-tailed)	.104	.754	.397	.109	.874	.018
	N	22	22	22	22	22	22
Waist	Pearson Correlation	-.112	.316	-.020	-.421	-.195	-.486*
	Sig. (2-tailed)	.619	.152	.929	.051	.384	.022
	N	22	22	22	22	22	22
SBP	Pearson Correlation	-.023	.292	.318	-.381	.081	-.203
	Sig. (2-tailed)	.920	.187	.149	.080	.719	.366
	N	22	22	22	22	22	22
DBP	Pearson Correlation	.365	-.022	-.205	.065	.197	.008
	Sig. (2-tailed)	.095	.922	.360	.774	.380	.971
	N	22	22	22	22	22	22
MAP	Pearson Correlation	.304	.136	.238	-.188	-.003	.013
	Sig. (2-tailed)	.169	.545	.286	.402	.989	.953
	N	22	22	22	22	22	22
HR	Pearson Correlation	.128	-.436*	.068	.161	-.046	-.273
	Sig. (2-tailed)	.570	.042	.764	.475	.838	.219
	N	22	22	22	22	22	22
cPTT	Pearson Correlation	-.594**	.157	-.054	-.238	-.048	-.165
	Sig. (2-tailed)	.005	.498	.818	.300	.836	.476
	N	21	21	21	21	21	21
cPWV	Pearson Correlation	.601**	.159	.145	.065	.000	.285
	Sig. (2-tailed)	.004	.490	.530	.778	.998	.210
	N	21	21	21	21	21	21
pPTT	Pearson Correlation	-.079	.365	-.039	.003	-.145	.440*
	Sig. (2-tailed)	.735	.103	.867	.989	.532	.046
	N	21	21	21	21	21	21

Correlations

		BrachialPP	CarotidPP
AGE	Pearson Correlation	.131	.249
	Sig. (2-tailed)	.561	.264
	N	22	22
SeatedHt	Pearson Correlation	.417	.046
	Sig. (2-tailed)	.053	.840
	N	22	22
StandingHt	Pearson Correlation	.362	.036
	Sig. (2-tailed)	.098	.874
	N	22	22
Weight	Pearson Correlation	.510*	.142
	Sig. (2-tailed)	.015	.528
	N	22	22
BMI	Pearson Correlation	.386	.156
	Sig. (2-tailed)	.076	.488
	N	22	22
BMIPercentile	Pearson Correlation	.412	-.028
	Sig. (2-tailed)	.057	.902
	N	22	22
Waist	Pearson Correlation	.463*	.167
	Sig. (2-tailed)	.030	.458
	N	22	22
SBP	Pearson Correlation	.892**	-.007
	Sig. (2-tailed)	.000	.976
	N	22	22
DBP	Pearson Correlation	-.455*	.289
	Sig. (2-tailed)	.033	.193
	N	22	22
MAP	Pearson Correlation	.042	.268
	Sig. (2-tailed)	.852	.227
	N	22	22
HR	Pearson Correlation	-.105	.098
	Sig. (2-tailed)	.641	.665
	N	22	22
cPTT	Pearson Correlation	.209	-.593**
	Sig. (2-tailed)	.363	.005
	N	21	21
cPWV	Pearson Correlation	-.120	.559**
	Sig. (2-tailed)	.603	.008
	N	21	21
pPTT	Pearson Correlation	-.165	-.104
	Sig. (2-tailed)	.474	.654
	N	21	21

Correlations

		AGE	SeatedHt	StandingHt	Weight	BMI	BMIPercentile	Waist
pPWV	Pearson Correlation	.006	.067	.130	.290	.285	.175	.362
	Sig. (2-tailed)	.981	.772	.573	.202	.211	.448	.106
	N	21	21	21	21	21	21	21
Compliance	Pearson Correlation	-.436*	-.008	-.071	-.017	.080	.322	.072
	Sig. (2-tailed)	.048	.974	.760	.941	.729	.155	.755
	N	21	21	21	21	21	21	21
Distensibility	Pearson Correlation	-.268	.065	.012	-.195	-.273	-.044	-.229
	Sig. (2-tailed)	.239	.781	.958	.397	.231	.850	.317
	N	21	21	21	21	21	21	21
SI	Pearson Correlation	.207	-.273	-.166	-.061	.034	-.115	.047
	Sig. (2-tailed)	.355	.218	.459	.788	.880	.610	.835
	N	22	22	22	22	22	22	22
IMTf	Pearson Correlation	-.330	-.178	-.185	-.397	-.395	-.270	-.408
	Sig. (2-tailed)	.144	.440	.423	.075	.076	.236	.066
	N	21	21	21	21	21	21	21
Absolute	Pearson Correlation	.345	-.194	-.152	-.110	.000	-.218	-.010
	Sig. (2-tailed)	.136	.412	.523	.644	.999	.355	.967
	N	20	20	20	20	20	20	20
Relative	Pearson Correlation	.269	-.252	-.211	-.198	-.081	-.251	-.111
	Sig. (2-tailed)	.252	.284	.372	.403	.735	.285	.642
	N	20	20	20	20	20	20	20
Normalized	Pearson Correlation	.387	-.133	-.008	-.008	.015	-.200	.051
	Sig. (2-tailed)	.092	.575	.974	.973	.951	.399	.831
	N	20	20	20	20	20	20	20
CarDiam	Pearson Correlation	-.248	.002	-.011	.276	.438*	.451*	.428*
	Sig. (2-tailed)	.267	.994	.961	.214	.042	.035	.047
	N	22	22	22	22	22	22	22
BrachialDiam	Pearson Correlation	.201	.382	.319	.482*	.411	.342	.520*
	Sig. (2-tailed)	.383	.088	.158	.027	.064	.129	.016
	N	21	21	21	21	21	21	21
RHMax	Pearson Correlation	.370	.329	.276	.484*	.467*	.303	.569**
	Sig. (2-tailed)	.108	.157	.239	.031	.038	.194	.009
	N	20	20	20	20	20	20	20
AveSR	Pearson Correlation	-.229	-.227	-.234	-.308	-.227	-.174	-.291
	Sig. (2-tailed)	.332	.337	.321	.186	.336	.463	.213
	N	20	20	20	20	20	20	20
PeakTime	Pearson Correlation	-.179	-.014	.022	.037	.015	.066	-.046
	Sig. (2-tailed)	.450	.955	.927	.878	.950	.783	.849
	N	20	20	20	20	20	20	20
WHR	Pearson Correlation	-.273	-.204	-.381	.299	.816**	.782**	.728**
	Sig. (2-tailed)	.219	.363	.080	.177	.000	.000	.000
	N	22	22	22	22	22	22	22

Correlations

		SBP	DBP	MAP	HR	cPTT	cPWV	pPTT	pPWV
pPWV	Pearson Correlation	.350	-.015	-.129	.543*	.018	-.014	-.870**	1
	Sig. (2-tailed)	.120	.948	.578	.011	.937	.953	.000	
	N	21	21	21	21	21	21	21	21
Compliance	Pearson Correlation	.221	-.321	.001	-.160	.449*	-.562**	-.008	-.070
	Sig. (2-tailed)	.335	.156	.996	.488	.047	.010	.974	.769
	N	21	21	21	21	20	20	20	20
Distensibility	Pearson Correlation	.068	-.386	-.154	-.350	.230	-.330	.300	-.231
	Sig. (2-tailed)	.770	.084	.504	.119	.328	.156	.200	.327
	N	21	21	21	21	20	20	20	20
SI	Pearson Correlation	.027	.436*	.204	.294	-.545*	.559**	-.344	.247
	Sig. (2-tailed)	.905	.042	.362	.185	.011	.008	.126	.280
	N	22	22	22	22	21	21	21	21
IMTf	Pearson Correlation	-.539*	-.035	-.010	-.090	-.058	-.029	.178	-.378
	Sig. (2-tailed)	.012	.881	.964	.700	.809	.902	.453	.100
	N	21	21	21	21	20	20	20	20
Absolute	Pearson Correlation	-.045	-.228	-.436	.418	-.225	.227	-.397	.327
	Sig. (2-tailed)	.851	.333	.054	.067	.355	.350	.092	.172
	N	20	20	20	20	19	19	19	19
Relative	Pearson Correlation	-.124	-.304	-.557*	.434	-.246	.219	-.407	.327
	Sig. (2-tailed)	.602	.192	.011	.056	.309	.368	.083	.172
	N	20	20	20	20	19	19	19	19
Normalized	Pearson Correlation	.046	-.163	-.395	.612**	-.143	.169	-.535*	.564*
	Sig. (2-tailed)	.846	.493	.085	.004	.560	.490	.018	.012
	N	20	20	20	20	19	19	19	19
CarDiam	Pearson Correlation	.334	.037	.266	.196	.154	-.276	-.365	.237
	Sig. (2-tailed)	.129	.871	.232	.382	.506	.225	.104	.301
	N	22	22	22	22	21	21	21	21
BrachialDiam	Pearson Correlation	.500*	.225	.581**	-.318	.165	-.121	.072	-.007
	Sig. (2-tailed)	.021	.327	.006	.160	.487	.612	.761	.976
	N	21	21	21	21	20	20	20	20
RHMax	Pearson Correlation	.514*	.122	.421	-.145	.346	-.140	-.094	.131
	Sig. (2-tailed)	.020	.607	.065	.543	.147	.567	.703	.594
	N	20	20	20	20	19	19	19	19
AveSR	Pearson Correlation	-.208	-.007	.063	.137	-.320	.067	.167	-.307
	Sig. (2-tailed)	.378	.976	.792	.564	.181	.784	.494	.201
	N	20	20	20	20	19	19	19	19
PeakTime	Pearson Correlation	-.262	.224	.081	-.147	-.175	.164	.114	-.243
	Sig. (2-tailed)	.264	.342	.734	.536	.475	.501	.643	.317
	N	20	20	20	20	19	19	19	19
WHR	Pearson Correlation	.228	.024	-.001	.381	.348	-.482*	-.467*	.260
	Sig. (2-tailed)	.308	.914	.996	.080	.123	.027	.033	.254
	N	22	22	22	22	21	21	21	21

Correlations

		Compliance	Distensibility	SI	IMTf	Absolute	Relative	Normalized
pPWV	Pearson Correlation	-.070	-.231	.247	-.378	.327	.327	.564*
	Sig. (2-tailed)	.769	.327	.280	.100	.172	.172	.012
	N	20	20	21	20	19	19	19
Compliance	Pearson Correlation	1	.759**	-.724**	.120	-.512*	-.510*	-.376
	Sig. (2-tailed)		.000	.000	.613	.021	.021	.102
	N	21	21	21	20	20	20	20
Distensibility	Pearson Correlation	.759**	1	-.867**	.185	-.437	-.381	-.355
	Sig. (2-tailed)	.000		.000	.434	.054	.097	.124
	N	21	21	21	20	20	20	20
SI	Pearson Correlation	-.724**	-.867**	1	-.216	.499*	.447*	.361
	Sig. (2-tailed)	.000	.000		.347	.025	.048	.118
	N	21	21	22	21	20	20	20
IMTf	Pearson Correlation	.120	.185	-.216	1	-.027	-.001	-.224
	Sig. (2-tailed)	.613	.434	.347		.912	.997	.356
	N	20	20	21	21	19	19	19
Absolute	Pearson Correlation	-.512*	-.437	.499*	-.027	1	.982**	.812**
	Sig. (2-tailed)	.021	.054	.025	.912		.000	.000
	N	20	20	20	19	20	20	20
Relative	Pearson Correlation	-.510*	-.381	.447*	-.001	.982**	1	.799**
	Sig. (2-tailed)	.021	.097	.048	.997	.000		.000
	N	20	20	20	19	20	20	20
Normalized	Pearson Correlation	-.376	-.355	.361	-.224	.812**	.799**	1
	Sig. (2-tailed)	.102	.124	.118	.356	.000	.000	
	N	20	20	20	19	20	20	20
CarDiam	Pearson Correlation	.513*	-.139	.001	-.048	-.210	-.290	-.097
	Sig. (2-tailed)	.017	.549	.997	.836	.373	.215	.683
	N	21	21	22	21	20	20	20
BrachialDiam	Pearson Correlation	.253	-.017	.071	-.367	-.378	-.508*	-.309
	Sig. (2-tailed)	.281	.945	.761	.111	.100	.022	.185
	N	20	20	21	20	20	20	20
RHMax	Pearson Correlation	.033	-.223	.277	-.403	.062	-.087	.048
	Sig. (2-tailed)	.892	.344	.237	.087	.796	.716	.839
	N	20	20	20	19	20	20	20
AveSR	Pearson Correlation	.156	.169	-.201	.665**	.094	.108	-.049
	Sig. (2-tailed)	.511	.475	.395	.002	.695	.652	.836
	N	20	20	20	19	20	20	20
PeakTime	Pearson Correlation	-.283	-.478*	.273	.170	-.157	-.142	-.228
	Sig. (2-tailed)	.227	.033	.245	.486	.508	.551	.335
	N	20	20	20	19	20	20	20
WHR	Pearson Correlation	.159	-.211	.152	-.297	.095	.040	.052
	Sig. (2-tailed)	.492	.358	.498	.190	.691	.868	.827
	N	21	21	22	21	20	20	20

Correlations

		CarDiam	BrachialDiam	RHMax	AveSR	PeakTime	WHR	GMFCS
pPWV	Pearson Correlation	.237	-.007	.131	-.307	-.243	.260	.047
	Sig. (2-tailed)	.301	.976	.594	.201	.317	.254	.891
	N	21	20	19	19	19	21	11
Compliance	Pearson Correlation	.513*	.253	.033	.156	-.283	.159	.269
	Sig. (2-tailed)	.017	.281	.892	.511	.227	.492	.424
	N	21	20	20	20	20	21	11
Distensibility	Pearson Correlation	-.139	-.017	-.223	.169	-.478*	-.211	-.062
	Sig. (2-tailed)	.549	.945	.344	.475	.033	.358	.857
	N	21	20	20	20	20	21	11
SI	Pearson Correlation	.001	.071	.277	-.201	.273	.152	.370
	Sig. (2-tailed)	.997	.761	.237	.395	.245	.498	.263
	N	22	21	20	20	20	22	11
IMTf	Pearson Correlation	-.048	-.367	-.403	.665**	.170	-.297	-.098
	Sig. (2-tailed)	.836	.111	.087	.002	.486	.190	.789
	N	21	20	19	19	19	21	10
Absolute	Pearson Correlation	-.210	-.378	.062	.094	-.157	.095	-.055
	Sig. (2-tailed)	.373	.100	.796	.695	.508	.691	.880
	N	20	20	20	20	20	20	10
Relative	Pearson Correlation	-.290	-.508*	-.087	.108	-.142	.040	-.115
	Sig. (2-tailed)	.215	.022	.716	.652	.551	.868	.752
	N	20	20	20	20	20	20	10
Normalized	Pearson Correlation	-.097	-.309	.048	-.049	-.228	.052	-.186
	Sig. (2-tailed)	.683	.185	.839	.836	.335	.827	.607
	N	20	20	20	20	20	20	10
CarDiam	Pearson Correlation	1	.402	.332	.117	.129	.447*	.259
	Sig. (2-tailed)		.071	.153	.622	.587	.037	.442
	N	22	21	20	20	20	22	11
BrachialDiam	Pearson Correlation	.402	1	.901**	-.401	.032	.285	.561
	Sig. (2-tailed)	.071		.000	.080	.892	.211	.092
	N	21	21	20	20	20	21	10
RHMax	Pearson Correlation	.332	.901**	1	-.388	-.039	.360	.620
	Sig. (2-tailed)	.153	.000		.091	.871	.119	.056
	N	20	20	20	20	20	20	10
AveSR	Pearson Correlation	.117	-.401	-.388	1	-.125	-.130	-.565
	Sig. (2-tailed)	.622	.080	.091		.600	.584	.089
	N	20	20	20	20	20	20	10
PeakTime	Pearson Correlation	.129	.032	-.039	-.125	1	-.064	.056
	Sig. (2-tailed)	.587	.892	.871	.600		.788	.877
	N	20	20	20	20	20	20	10
WHR	Pearson Correlation	.447*	.285	.360	-.130	-.064	1	.804**
	Sig. (2-tailed)	.037	.211	.119	.584	.788		.003
	N	22	21	20	20	20	22	11

Correlations

		APHV	TPHV	LightPA	MediumPA	HardPA	WLRatio
pPWV	Pearson Correlation	.037	-.017	.024	-.116	.226	-.369
	Sig. (2-tailed)	.872	.942	.918	.617	.324	.100
	N	21	21	21	21	21	21
Compliance	Pearson Correlation	-.434*	-.253	-.013	.064	.124	-.405
	Sig. (2-tailed)	.049	.269	.956	.782	.593	.068
	N	21	21	21	21	21	21
Distensibility	Pearson Correlation	-.375	-.095	-.230	-.126	.259	.130
	Sig. (2-tailed)	.094	.681	.316	.585	.258	.574
	N	21	21	21	21	21	21
SI	Pearson Correlation	.577**	-.120	.173	.188	-.195	-.043
	Sig. (2-tailed)	.005	.596	.442	.401	.384	.848
	N	22	22	22	22	22	22
IMTf	Pearson Correlation	-.007	-.376	-.040	.159	.000	.721**
	Sig. (2-tailed)	.976	.093	.865	.491	.998	.000
	N	21	21	21	21	21	21
Absolute	Pearson Correlation	.506*	.089	.292	.110	-.293	.101
	Sig. (2-tailed)	.023	.710	.211	.643	.210	.673
	N	20	20	20	20	20	20
Relative	Pearson Correlation	.418	.054	.206	.120	-.227	.164
	Sig. (2-tailed)	.067	.820	.383	.613	.336	.488
	N	20	20	20	20	20	20
Normalized	Pearson Correlation	.483*	.149	.178	-.015	-.167	.000
	Sig. (2-tailed)	.031	.531	.452	.951	.480	1.000
	N	20	20	20	20	20	20
CarDiam	Pearson Correlation	-.146	-.193	.352	.262	-.097	-.744**
	Sig. (2-tailed)	.518	.389	.108	.239	.668	.000
	N	22	22	22	22	22	22
BrachialDiam	Pearson Correlation	.152	.131	.228	-.219	-.030	-.406
	Sig. (2-tailed)	.510	.570	.319	.340	.898	.068
	N	21	21	21	21	21	21
RHMax	Pearson Correlation	.381	.194	.382	-.197	-.170	-.386
	Sig. (2-tailed)	.098	.412	.097	.405	.474	.093
	N	20	20	20	20	20	20
AveSR	Pearson Correlation	.129	-.345	.293	.427	.150	.275
	Sig. (2-tailed)	.588	.136	.209	.061	.528	.240
	N	20	20	20	20	20	20
PeakTime	Pearson Correlation	-.156	-.111	-.120	.040	.022	.025
	Sig. (2-tailed)	.513	.641	.614	.869	.927	.916
	N	20	20	20	20	20	20
WHR	Pearson Correlation	-.111	-.244	-.164	-.111	-.228	-.611**
	Sig. (2-tailed)	.623	.274	.466	.624	.308	.003
	N	22	22	22	22	22	22

Correlations

		BrachialPP	CarotidPP
pPWV	Pearson Correlation	.324	.047
	Sig. (2-tailed)	.152	.840
	N	21	21
Compliance	Pearson Correlation	.337	-.615**
	Sig. (2-tailed)	.136	.003
	N	21	21
Distensibility	Pearson Correlation	.229	-.628**
	Sig. (2-tailed)	.318	.002
	N	21	21
SI	Pearson Correlation	-.173	.575**
	Sig. (2-tailed)	.441	.005
	N	22	22
IMTf	Pearson Correlation	-.461*	.124
	Sig. (2-tailed)	.035	.592
	N	21	21
Absolute	Pearson Correlation	.060	.369
	Sig. (2-tailed)	.800	.109
	N	20	20
Relative	Pearson Correlation	.023	.348
	Sig. (2-tailed)	.922	.133
	N	20	20
Normalized	Pearson Correlation	.113	.147
	Sig. (2-tailed)	.636	.536
	N	20	20
CarDiam	Pearson Correlation	.281	-.019
	Sig. (2-tailed)	.205	.935
	N	22	22
BrachialDiam	Pearson Correlation	.344	-.083
	Sig. (2-tailed)	.126	.722
	N	21	21
RHMax	Pearson Correlation	.403	.026
	Sig. (2-tailed)	.078	.914
	N	20	20
AveSR	Pearson Correlation	-.182	.284
	Sig. (2-tailed)	.442	.225
	N	20	20
PeakTime	Pearson Correlation	-.332	.418
	Sig. (2-tailed)	.153	.067
	N	20	20
WHR	Pearson Correlation	.192	.118
	Sig. (2-tailed)	.392	.602
	N	22	22

Correlations

		AGE	SeatedHt	StandingHt	Weight	BMI	BMIPercentile	Waist
GMFCS	Pearson Correlation	-.365	-.475	-.529	.203	.686	.875**	.583
	Sig. (2-tailed)	.269	.140	.094	.548	.020	.000	.060
	N	11	11	11	11	11	11	11
APHV	Pearson Correlation	.507*	-.152	-.008	-.124	-.164	-.356	-.112
	Sig. (2-tailed)	.016	.499	.972	.582	.466	.104	.619
	N	22	22	22	22	22	22	22
TPHV	Pearson Correlation	.841**	.780**	.748**	.626**	.226	-.071	.316
	Sig. (2-tailed)	.000	.000	.000	.002	.313	.754	.152
	N	22	22	22	22	22	22	22
LightPA	Pearson Correlation	.257	.158	.166	.094	-.057	-.190	-.020
	Sig. (2-tailed)	.248	.483	.461	.678	.802	.397	.929
	N	22	22	22	22	22	22	22
MediumPA	Pearson Correlation	-.166	-.500*	-.398	-.501*	-.336	-.351	-.421
	Sig. (2-tailed)	.461	.018	.067	.018	.126	.109	.051
	N	22	22	22	22	22	22	22
HardPA	Pearson Correlation	-.268	-.036	.058	-.089	-.210	.036	-.195
	Sig. (2-tailed)	.228	.873	.798	.692	.349	.874	.384
	N	22	22	22	22	22	22	22
WLRatio	Pearson Correlation	.166	.053	.124	-.260	-.547**	-.498*	-.486*
	Sig. (2-tailed)	.460	.814	.581	.243	.008	.018	.022
	N	22	22	22	22	22	22	22
BrachialPP	Pearson Correlation	.131	.417	.362	.510*	.386	.412	.463*
	Sig. (2-tailed)	.561	.053	.098	.015	.076	.057	.030
	N	22	22	22	22	22	22	22
CarotidPP	Pearson Correlation	.249	.046	.036	.142	.156	-.028	.167
	Sig. (2-tailed)	.264	.840	.874	.528	.488	.902	.458
	N	22	22	22	22	22	22	22

Correlations

		SBP	DBP	MAP	HR	cPTT	cPWV	pPTT	pPWV
GMFCS	Pearson Correlation	.234	.189	.175	.013	.321	-.246	-.342	.047
	Sig. (2-tailed)	.489	.578	.608	.970	.336	.465	.303	.891
	N	11	11	11	11	11	11	11	11
APHV	Pearson Correlation	-.023	.365	.304	.128	-.594**	.601**	-.079	.037
	Sig. (2-tailed)	.920	.095	.169	.570	.005	.004	.735	.872
	N	22	22	22	22	21	21	21	21
TPHV	Pearson Correlation	.292	-.022	.136	-.436*	.157	.159	.365	-.017
	Sig. (2-tailed)	.187	.922	.545	.042	.498	.490	.103	.942
	N	22	22	22	22	21	21	21	21
LightPA	Pearson Correlation	.318	-.205	.238	.068	-.054	.145	-.039	.024
	Sig. (2-tailed)	.149	.360	.286	.764	.818	.530	.867	.918
	N	22	22	22	22	21	21	21	21
MediumPA	Pearson Correlation	-.381	.065	-.188	.161	-.238	.065	.003	-.116
	Sig. (2-tailed)	.080	.774	.402	.475	.300	.778	.989	.617
	N	22	22	22	22	21	21	21	21
HardPA	Pearson Correlation	.081	.197	-.003	-.046	-.048	.000	-.145	.226
	Sig. (2-tailed)	.719	.380	.989	.838	.836	.998	.532	.324
	N	22	22	22	22	21	21	21	21
WLRatio	Pearson Correlation	-.203	.008	.013	-.273	-.165	.285	.440*	-.369
	Sig. (2-tailed)	.366	.971	.953	.219	.476	.210	.046	.100
	N	22	22	22	22	21	21	21	21
BrachialPP	Pearson Correlation	.892**	-.455*	.042	-.105	.209	-.120	-.165	.324
	Sig. (2-tailed)	.000	.033	.852	.641	.363	.603	.474	.152
	N	22	22	22	22	21	21	21	21
CarotidPP	Pearson Correlation	-.007	.289	.268	.098	-.593**	.559**	-.104	.047
	Sig. (2-tailed)	.976	.193	.227	.665	.005	.008	.654	.840
	N	22	22	22	22	21	21	21	21

Correlations

		Compliance	Distensibility	SI	IMTf	Absolute	Relative	Normalized
GMFCS	Pearson Correlation	.269	-.062	.370	-.098	-.055	-.115	-.186
	Sig. (2-tailed)	.424	.857	.263	.789	.880	.752	.607
	N	11	11	11	10	10	10	10
APHV	Pearson Correlation	-.434*	-.375	.577**	-.007	.506*	.418	.483*
	Sig. (2-tailed)	.049	.094	.005	.976	.023	.067	.031
	N	21	21	22	21	20	20	20
TPHV	Pearson Correlation	-.253	-.095	-.120	-.376	.089	.054	.149
	Sig. (2-tailed)	.269	.681	.596	.093	.710	.820	.531
	N	21	21	22	21	20	20	20
LightPA	Pearson Correlation	-.013	-.230	.173	-.040	.292	.206	.178
	Sig. (2-tailed)	.956	.316	.442	.865	.211	.383	.452
	N	21	21	22	21	20	20	20
MediumPA	Pearson Correlation	.064	-.126	.188	.159	.110	.120	-.015
	Sig. (2-tailed)	.782	.585	.401	.491	.643	.613	.951
	N	21	21	22	21	20	20	20
HardPA	Pearson Correlation	.124	.259	-.195	.000	-.293	-.227	-.167
	Sig. (2-tailed)	.593	.258	.384	.998	.210	.336	.480
	N	21	21	22	21	20	20	20
WLRatio	Pearson Correlation	-.405	.130	-.043	.721**	.101	.164	.000
	Sig. (2-tailed)	.068	.574	.848	.000	.673	.488	1.000
	N	21	21	22	21	20	20	20
BrachialPP	Pearson Correlation	.337	.229	-.173	-.461*	.060	.023	.113
	Sig. (2-tailed)	.136	.318	.441	.035	.800	.922	.636
	N	21	21	22	21	20	20	20
CarotidPP	Pearson Correlation	-.615**	-.628**	.575**	.124	.369	.348	.147
	Sig. (2-tailed)	.003	.002	.005	.592	.109	.133	.536
	N	21	21	22	21	20	20	20

Correlations

		CarDiam	BrachialDiam	RHMax	AveSR	PeakTime	WHR	GMFCS
GMFCS	Pearson Correlation	.259	.561	.620	-.565	.056	.804**	1
	Sig. (2-tailed)	.442	.092	.056	.089	.877	.003	
	N	11	10	10	10	10	11	11
APHV	Pearson Correlation	-.146	.152	.381	.129	-.156	-.111	.111
	Sig. (2-tailed)	.518	.510	.098	.588	.513	.623	.746
	N	22	21	20	20	20	22	11
TPHV	Pearson Correlation	-.193	.131	.194	-.345	-.111	-.244	-.552
	Sig. (2-tailed)	.389	.570	.412	.136	.641	.274	.079
	N	22	21	20	20	20	22	11
LightPA	Pearson Correlation	.352	.228	.382	.293	-.120	-.164	-.243
	Sig. (2-tailed)	.108	.319	.097	.209	.614	.466	.471
	N	22	21	20	20	20	22	11
MediumPA	Pearson Correlation	.262	-.219	-.197	.427	.040	-.111	-.181
	Sig. (2-tailed)	.239	.340	.405	.061	.869	.624	.594
	N	22	21	20	20	20	22	11
HardPA	Pearson Correlation	-.097	-.030	-.170	.150	.022	-.228	-.373
	Sig. (2-tailed)	.668	.898	.474	.528	.927	.308	.259
	N	22	21	20	20	20	22	11
WLRatio	Pearson Correlation	-.744**	-.406	-.386	.275	.025	-.611**	-.402
	Sig. (2-tailed)	.000	.068	.093	.240	.916	.003	.220
	N	22	21	20	20	20	22	11
BrachialPP	Pearson Correlation	.281	.344	.403	-.182	-.332	.192	.130
	Sig. (2-tailed)	.205	.126	.078	.442	.153	.392	.702
	N	22	21	20	20	20	22	11
CarotidPP	Pearson Correlation	-.019	-.083	.026	.284	.418	.118	-.137
	Sig. (2-tailed)	.935	.722	.914	.225	.067	.602	.687
	N	22	21	20	20	20	22	11

Correlations

		APHV	TPHV	LightPA	MediumPA	HardPA	WLRatio
GMFCS	Pearson Correlation	.111	-.552	-.243	-.181	-.373	-.402
	Sig. (2-tailed)	.746	.079	.471	.594	.259	.220
	N	11	11	11	11	11	11
APHV	Pearson Correlation	1	-.040	.316	.161	-.155	.133
	Sig. (2-tailed)		.860	.152	.475	.491	.556
	N	22	22	22	22	22	22
TPHV	Pearson Correlation	-.040	1	.100	-.290	-.215	.106
	Sig. (2-tailed)	.860		.656	.191	.336	.639
	N	22	22	22	22	22	22
LightPA	Pearson Correlation	.316	.100	1	.180	-.238	-.027
	Sig. (2-tailed)	.152	.656		.422	.287	.906
	N	22	22	22	22	22	22
MediumPA	Pearson Correlation	.161	-.290	.180	1	-.040	-.262
	Sig. (2-tailed)	.475	.191	.422		.860	.240
	N	22	22	22	22	22	22
HardPA	Pearson Correlation	-.155	-.215	-.238	-.040	1	.135
	Sig. (2-tailed)	.491	.336	.287	.860		.551
	N	22	22	22	22	22	22
WLRatio	Pearson Correlation	.133	.106	-.027	-.262	.135	1
	Sig. (2-tailed)	.556	.639	.906	.240	.551	
	N	22	22	22	22	22	22
BrachialPP	Pearson Correlation	-.186	.270	.376	-.368	-.017	-.184
	Sig. (2-tailed)	.408	.224	.085	.092	.941	.412
	N	22	22	22	22	22	22
CarotidPP	Pearson Correlation	.469	-.006	.107	-.066	-.112	.140
	Sig. (2-tailed)	.028	.981	.637	.769	.619	.534
	N	22	22	22	22	22	22

Correlations

		BrachialPP	CarotidPP
GMFCS	Pearson Correlation	.130	-.137
	Sig. (2-tailed)	.702	.687
	N	11	11
APHV	Pearson Correlation	-.186	.469*
	Sig. (2-tailed)	.408	.028
	N	22	22
TPHV	Pearson Correlation	.270	-.006
	Sig. (2-tailed)	.224	.981
	N	22	22
LightPA	Pearson Correlation	.376	.107
	Sig. (2-tailed)	.085	.637
	N	22	22
MediumPA	Pearson Correlation	-.368	-.066
	Sig. (2-tailed)	.092	.769
	N	22	22
HardPA	Pearson Correlation	-.017	-.112
	Sig. (2-tailed)	.941	.619
	N	22	22
WLRatio	Pearson Correlation	-.184	.140
	Sig. (2-tailed)	.412	.534
	N	22	22
BrachialPP	Pearson Correlation	1	-.136
	Sig. (2-tailed)		.545
	N	22	22
CarotidPP	Pearson Correlation	-.136	1
	Sig. (2-tailed)	.545	
	N	22	22

**. Correlation is significant at the 0.01 level (2-tailed).
*. Correlation is significant at the 0.05 level (2-tailed).

```
EXAMINE VARIABLES=Gender AGE SeatedHt StandingHt Weight BMI BMIPercentile Waist SBP DBP MAP HR
MediumPA HardPA WLRatio BrachialPP CarotidPP BY Group
  /PLOT BOXPLOT STEMLEAF HISTOGRAM NPPLOT
  /COMPARE GROUPS
  /STATISTICS DESCRIPTIVES
  /CINTERVAL 95
  /MISSING LISTWISE
  /NOTOTAL.


SAVE OUTFILE='/Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine Bl
    'IN, WL ratio, PP_FINAL_June15.sav'
 /COMPRESSED.
EXAMINE VARIABLES=Gender AGE SeatedHt StandingHt Weight BMI BMIPercentile Waist SBP DBP MAP HR
MediumPA HardPA WLRatio BrachialPP CarotidPP
```

```
  /ID=Group
  /PLOT BOXPLOT STEMLEAF HISTOGRAM NPPLOT
  /COMPARE GROUPS
  /STATISTICS DESCRIPTIVES
  /CINTERVAL 95
  /MISSING LISTWISE
  /NOTOTAL.


EXAMINE VARIABLES=AGE SeatedHt StandingHt Weight BMI BMIPercentile Waist SBP DBP MAP HR cPTT c]
WLRatio BrachialPP CarotidPP BY Group
  /PLOT BOXPLOT STEMLEAF HISTOGRAM NPPLOT
  /COMPARE GROUPS
  /STATISTICS DESCRIPTIVES
  /CINTERVAL 95
  /MISSING LISTWISE
  /NOTOTAL.
```

Explore

Notes

Output Created		**15-Jun-2011 15:01:57**
Comments		
Input	**Data**	**/Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP IN, WL ratio, PP_FINAL_June15.sav**
	Active Dataset	**DataSet1**
	Filter	**<none>**
	Weight	**<none>**
	Split File	**<none>**
	N of Rows in Working Data File	**26**
Missing Value Handling	**Definition of Missing**	**User-defined missing values for dependent variables are treated as missing.**
	Cases Used	**Statistics are based on cases with no missing values for any dependent variable or factor used.**
Syntax		**EXAMINE VARIABLES=AGE SeatedHt StandingHt Weight BMI BMIPercentile Waist SBP DBP MAP HR cPTT cPWV pPTT pPWV Compliance Distensibility SI IMTf Absolute Relative Normalized CarDiam BrachialDiam RHMax AveSR PeakTime WHR APHV TPHV LightPA MediumPA HardPA WLRatio BrachialPP CarotidPP BY Group /PLOT BOXPLOT STEMLEAF HISTOGRAM NPPLOT /COMPARE GROUPS /STATISTICS DESCRIPTIVES /CINTERVAL 95 /MISSING LISTWISE /NOTOTAL.**
Resources	**Processor Time**	**00 00:02:10.691**
	Elapsed Time	**00 00:02:16.000**

```
[DataSet1] /Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP II
, WL ratio, PP_FINAL_June15.sav
```

```
SAVE OUTFILE='/Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BI
    'IN, WL ratio, PP_FINAL_June15.sav'
 /COMPRESSED.
T-TEST GROUPS=Group(0 1)
  /MISSING=ANALYSIS
  /VARIABLES=BrachialDiam
  /CRITERIA=CI(.95).
```

T-Test

Notes

Output Created		16-Jun-2011 13:46:06
Comments		
Input	Data	/Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP IN, WL ratio, PP_FINAL_June15.sav
	Active Dataset	DataSet1
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	26
Missing Value Handling	Definition of Missing	User defined missing values are treated as missing.
	Cases Used	Statistics for each analysis are based on the cases with no missing or out-of-range data for any variable in the analysis.
Syntax		T-TEST GROUPS=Group(0 1) /MISSING=ANALYSIS /VARIABLES=BrachialDiam /CRITERIA=CI(.95).
Resources	Processor Time	00 00:00:00.016
	Elapsed Time	00 00:00:00.000

```
[DataSet1] /Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP IN
, WL ratio, PP_FINAL_June15.sav
```

Group Statistics

	Group	N	Mean	Std. Deviation	Std. Error Mean
BrachialDiam	CP	11	3.0808	.47593	.14350
	CON	11	3.1958	.37077	.11179

Independent Samples Test

		Levene's Test for Equality of Variances	
		F	Sig.
BrachialDiam	Equal variances assumed	1.759	.200
	Equal variances not assumed		

Independent Samples Test

		t-test for Equality of Means				
		t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference
BrachialDiam	Equal variances assumed	-.632	20	.535	-.11494	.18190
	Equal variances not assumed	-.632	18.871	.535	-.11494	.18190

Independent Samples Test

		t-test for Equality of Means	
		95% Confidence Interval of the Difference	
		Lower	Upper
BrachialDiam	Equal variances assumed	-.49438	.26450
	Equal variances not assumed	-.49584	.26597

```
GET
  FILE='/Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP IN, W
DATASET NAME DataSet1 WINDOW=FRONT.
T-TEST GROUPS=Group(0 1)
  /MISSING=ANALYSIS
  /VARIABLES=TotalPA
  /CRITERIA=CI(.95).
```

T-Test

Notes

Output Created		10-Jul-2011 15:44:47
Comments		
Input	Data	/Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP IN, WL ratio, PP_FINAL_June27.sav
	Active Dataset	DataSet1
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	26
Missing Value Handling	Definition of Missing	User defined missing values are treated as missing.
	Cases Used	Statistics for each analysis are based on the cases with no missing or out-of-range data for any variable in the analysis.
Syntax		T-TEST GROUPS=Group(0 1) /MISSING=ANALYSIS /VARIABLES=TotalPA /CRITERIA=CI(.95).
Resources	Processor Time	00 00:00:00.029
	Elapsed Time	00 00:00:00.000

```
[DataSet1] /Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP IN
, WL ratio, PP_FINAL_June27.sav
```

Group Statistics

	Group	N	Mean	Std. Deviation	Std. Error Mean
TotalPA	CP	11	387.2727	308.21554	92.93048
	CON	11	440.0000	231.40873	69.77236

Independent Samples Test

		Levene's Test for Equality of Variances	
		F	Sig.
TotalPA	Equal variances assumed	.139	.713
	Equal variances not assumed		

Independent Samples Test

		t-test for Equality of Means				
		t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference
TotalPA	Equal variances assumed	-.454	20	.655	-52.72727	116.20781
	Equal variances not assumed	-.454	18.555	.655	-52.72727	116.20781

Independent Samples Test

		t-test for Equality of Means	
		95% Confidence Interval of the Difference	
		Lower	Upper
TotalPA	Equal variances assumed	-295.13253	189.67798
	Equal variances not assumed	-296.34801	190.89347

Partial Corr

Notes

Output Created		19-Jun-2011 11:12:46
Comments		
Input	Data	/Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP IN, WL ratio, PP_FINAL_June15.sav
	Active Dataset	DataSet7
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	26
Missing Value Handling	Definition of Missing	User defined missing values are treated as missing.
	Cases Used	Statistics are based on cases with no missing data for any variable listed.
Syntax		PARTIAL CORR /VARIABLES=cPWV pPWV Compliance Distensibility IMTf Absolute Relative Normalized LightPA MediumPA HardPA BMI BMIPercentile WHR TPHV BY AGE /SIGNIFICANCE=TWOTAIL /STATISTICS=DESCRIPTIVES /MISSING=LISTWISE.
Resources	Processor Time	00 00:00:00.021
	Elapsed Time	00 00:00:00.000

[DataSet7] /Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP IN, WL ratio, PP_FINAL_June15.sav

Descriptive Statistics

	Mean	Std. Deviation	N
cPWV	4.3353	.58573	18
pPWV	7.4479	1.43386	18
Compliance	.1746	.05056	18
Distensibility	.0080	.00226	18
IMTf	.4192	.03722	18
Absolute	.2636	.18533	18
Relative	8.7503	6.83003	18
Normalized	.0037	.00285	18
LightPA	92.5000	109.65521	18
MediumPA	160.8333	159.43143	18
HardPA	122.7778	164.72992	18
BMI	18.7000	3.30490	18
BMIPercentile	45.2222	31.37732	18
WHR	.4414	.04862	18
TPHV	-.6889	1.69007	18
AGE	13.0000	1.95482	18

Correlations

Control Variables			cPWV	pPWV	Compliance	Distensibility	IMTf	Absolute
AGE	cPWV	Correlation	1.000	-.142	-.367	-.257	.376	.097
		Significance (2-tailed)	.	.588	.147	.318	.137	.712
		df	0	15	15	15	15	15
	pPWV	Correlation	-.142	1.000	-.143	-.213	-.452	.298
		Significance (2-tailed)	.588	.	.583	.411	.069	.245
		df	15	0	15	15	15	15
	Compliance	Correlation	-.367	-.143	1.000	.788	-.140	-.463
		Significance (2-tailed)	.147	.583	.	.000	.593	.061
		df	15	15	0	15	15	15
	Distensibility	Correlation	-.257	-.213	.788	1.000	.014	-.364
		Significance (2-tailed)	.318	.411	.000	.	.958	.151
		df	15	15	15	0	15	15
	IMTf	Correlation	.376	-.452	-.140	.014	1.000	.111
		Significance (2-tailed)	.137	.069	.593	.958	.	.673
		df	15	15	15	15	0	15
	Absolute	Correlation	.097	.298	-.463	-.364	.111	1.000
		Significance (2-tailed)	.712	.245	.061	.151	.673	.
		df	15	15	15	15	15	0
	Relative	Correlation	.114	.316	-.482	-.332	.097	.987
		Significance (2-tailed)	.663	.217	.050	.193	.713	.000
		df	15	15	15	15	15	15
	Normalized	Correlation	.001	.570	-.282	-.274	-.133	.782
		Significance (2-tailed)	.997	.017	.273	.288	.612	.000
		df	15	15	15	15	15	15
	LightPA	Correlation	-.029	-.006	.252	-.139	.108	.209
		Significance (2-tailed)	.913	.982	.328	.596	.679	.421
		df	15	15	15	15	15	15
	MediumPA	Correlation	.248	.001	-.051	-.204	.491	.326
		Significance (2-tailed)	.338	.998	.845	.432	.046	.202
		df	15	15	15	15	15	15
	HardPA	Correlation	.080	.298	.100	.200	-.114	-.229
		Significance (2-tailed)	.761	.245	.702	.442	.663	.376
		df	15	15	15	15	15	15
	BMI	Correlation	-.307	.242	.008	-.260	-.515	-.094
		Significance (2-tailed)	.231	.349	.976	.313	.034	.721
		df	15	15	15	15	15	15
	BMIPercentile	Correlation	-.206	.149	.136	-.129	-.563	-.197
		Significance (2-tailed)	.428	.567	.602	.621	.019	.449
		df	15	15	15	15	15	15
	WHR	Correlation	-.444	.208	-.126	-.315	-.480	.190
		Significance (2-tailed)	.074	.422	.629	.218	.051	.464
		df	15	15	15	15	15	15

Correlations

Control Variables			Relative	Normalized	LightPA	MediumPA	HardPA	BMI
AGE	cPWV	Correlation	.114	.001	-.029	.248	.080	-.307
		Significance (2-tailed)	.663	.997	.913	.338	.761	.231
		df	15	15	15	15	15	15
	pPWV	Correlation	.316	.570	-.006	.001	.298	.242
		Significance (2-tailed)	.217	.017	.982	.998	.245	.349
		df	15	15	15	15	15	15
	Compliance	Correlation	-.482	-.282	.252	-.051	.100	.008
		Significance (2-tailed)	.050	.273	.328	.845	.702	.976
		df	15	15	15	15	15	15
	Distensibility	Correlation	-.332	-.274	-.139	-.204	.200	-.260
		Significance (2-tailed)	.193	.288	.596	.432	.442	.313
		df	15	15	15	15	15	15
	IMTf	Correlation	.097	-.133	.108	.491	-.114	-.515
		Significance (2-tailed)	.713	.612	.679	.046	.663	.034
		df	15	15	15	15	15	15
	Absolute	Correlation	.987	.782	.209	.326	-.229	-.094
		Significance (2-tailed)	.000	.000	.421	.202	.376	.721
		df	15	15	15	15	15	15
	Relative	Correlation	1.000	.783	.132	.309	-.182	-.146
		Significance (2-tailed)	.	.000	.614	.228	.483	.576
		df	0	15	15	15	15	15
	Normalized	Correlation	.783	1.000	.072	.202	-.076	-.085
		Significance (2-tailed)	.000	.	.783	.436	.771	.745
		df	15	0	15	15	15	15
	LightPA	Correlation	.132	.072	1.000	.509	-.260	-.020
		Significance (2-tailed)	.614	.783	.	.037	.314	.941
		df	15	15	0	15	15	15
	MediumPA	Correlation	.309	.202	.509	1.000	.007	-.552
		Significance (2-tailed)	.228	.436	.037	.	.978	.022
		df	15	15	15	0	15	15
	HardPA	Correlation	-.182	-.076	-.260	.007	1.000	-.083
		Significance (2-tailed)	.483	.771	.314	.978	.	.751
		df	15	15	15	15	0	15
	BMI	Correlation	-.146	-.085	-.020	-.552	-.083	1.000
		Significance (2-tailed)	.576	.745	.941	.022	.751	.
		df	15	15	15	15	15	0
	BMIPercentile	Correlation	-.229	-.174	-.082	-.578	.068	.920
		Significance (2-tailed)	.378	.505	.755	.015	.795	.000
		df	15	15	15	15	15	15
	WHR	Correlation	.125	.152	-.025	-.422	-.232	.863
		Significance (2-tailed)	.632	.560	.923	.092	.371	.000
		df	15	15	15	15	15	15

Correlations

Control Variables			BMIPercentile	WHR	TPHV
AGE	cPWV	Correlation	-.206	-.444	-.333
		Significance (2-tailed)	.428	.074	.191
		df	15	15	15
	pPWV	Correlation	.149	.208	.088
		Significance (2-tailed)	.567	.422	.738
		df	15	15	15
	Compliance	Correlation	.136	-.126	.256
		Significance (2-tailed)	.602	.629	.322
		df	15	15	15
	Distensibility	Correlation	-.129	-.315	.251
		Significance (2-tailed)	.621	.218	.331
		df	15	15	15
	IMTf	Correlation	-.563	-.480	-.338
		Significance (2-tailed)	.019	.051	.185
		df	15	15	15
	Absolute	Correlation	-.197	.190	-.391
		Significance (2-tailed)	.449	.464	.120
		df	15	15	15
	Relative	Correlation	-.229	.125	-.327
		Significance (2-tailed)	.378	.632	.200
		df	15	15	15
	Normalized	Correlation	-.174	.152	-.353
		Significance (2-tailed)	.505	.560	.165
		df	15	15	15
	LightPA	Correlation	-.082	-.025	-.203
		Significance (2-tailed)	.755	.923	.435
		df	15	15	15
	MediumPA	Correlation	-.578	-.422	-.438
		Significance (2-tailed)	.015	.092	.079
		df	15	15	15
	HardPA	Correlation	.068	-.232	.051
		Significance (2-tailed)	.795	.371	.846
		df	15	15	15
	BMI	Correlation	.920	.863	.227
		Significance (2-tailed)	.000	.000	.380
		df	15	15	15
	BMIPercentile	Correlation	1.000	.752	.213
		Significance (2-tailed)	.	.001	.411
		df	0	15	15
	WHR	Correlation	.752	1.000	-.059
		Significance (2-tailed)	.001	.	.822
		df	15	0	15

Correlations

Control Variables			cPWV	pPWV	Compliance	Distensibility	IMTf	Absolute
AGE	TPHV	Correlation	-.333	.088	.256	.251	-.338	-.391
		Significance (2-tailed)	.191	.738	.322	.331	.185	.120
		df	15	15	15	15	15	15

Correlations

Control Variables			Relative	Normalized	LightPA	MediumPA	HardPA	BMI
AGE	TPHV	Correlation	-.327	-.353	-.203	-.438	.051	.227
		Significance (2-tailed)	.200	.165	.435	.079	.846	.380
		df	15	15	15	15	15	15

Correlations

Control Variables			BMIPercentile	WHR	TPHV
AGE	TPHV	Correlation	.213	-.059	1.000
		Significance (2-tailed)	.411	.822	.
		df	15	15	0

```
UNIANOVA cPWV BY Group WITH AGE
  /CONTRAST(Group)=Simple
  /METHOD=SSTYPE(3)
  /INTERCEPT=INCLUDE
  /EMMEANS=TABLES(Group) WITH(AGE=MEAN) COMPARE ADJ(BONFERRONI)
  /PRINT=DESCRIPTIVE
  /CRITERIA=ALPHA(.05)
  /DESIGN=AGE Group.
```

Univariate Analysis of Variance

Notes

Output Created		19-Jun-2011 11:33:48
Comments		
Input	Data	/Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP IN, WL ratio, PP_FINAL_June15.sav
	Active Dataset	DataSet7
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	26
Missing Value Handling	Definition of Missing	User-defined missing values are treated as missing.
	Cases Used	Statistics are based on all cases with valid data for all variables in the model.
Syntax		UNIANOVA cPWV BY Group WITH AGE /CONTRAST(Group)=Simple /METHOD=SSTYPE(3) /INTERCEPT=INCLUDE /EMMEANS=TABLES(Group) WITH(AGE=MEAN) COMPARE ADJ (BONFERRONI) /PRINT=DESCRIPTIVE /CRITERIA=ALPHA(.05) /DESIGN=AGE Group.
Resources	Processor Time	00 00:00:00.021
	Elapsed Time	00 00:00:00.000

[DataSet7] /Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP IN, WL ratio, PP_FINAL_June15.sav

Between-Subjects Factors

		Value Label	N
Group	.00	OP	11
	1.00	CON	10

Descriptive Statistics

Dependent Variable:cPWV

Group	Mean	Std. Deviation	N
OP	4.2958	.63373	11
CON	4.0704	.93306	10
Total	4.1885	.77838	21

Tests of Between-Subjects Effects

Dependent Variable:cPWV

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	2.977[a]	2	1.489	2.932	.079
Intercept	1.778	1	1.778	3.502	.078
AGE	2.711	1	2.711	5.339	.033
Group	.124	1	.124	.244	.628
Error	9.140	18	.508		
Total	380.529	21			
Corrected Total	12.118	20			

a. R Squared = .246 (Adjusted R Squared = .162)

```
UNIANOVA cPWV BY Group WITH AGE
  /METHOD=SSTYPE(3)
  /INTERCEPT=INCLUDE
  /EMMEANS=TABLES(Group) WITH(AGE=MEAN) COMPARE ADJ(BONFERRONI)
  /PRINT=DESCRIPTIVE
  /CRITERIA=ALPHA(.05)
  /DESIGN=AGE Group.
```

Univariate Analysis of Variance

Notes

Output Created		19-Jun-2011 11:40:20
Comments		
Input	Data	/Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP IN, WL ratio, PP_FINAL_June15.sav
	Active Dataset	DataSet7
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	26
Missing Value Handling	Definition of Missing	User-defined missing values are treated as missing.
	Cases Used	Statistics are based on all cases with valid data for all variables in the model.
Syntax		UNIANOVA cPWV BY Group WITH AGE /METHOD=SSTYPE(3) /INTERCEPT=INCLUDE /EMMEANS=TABLES(Group) WITH(AGE=MEAN) COMPARE ADJ (BONFERRONI) /PRINT=DESCRIPTIVE /CRITERIA=ALPHA(.05) /DESIGN=AGE Group.
Resources	Processor Time	00 00:00:00.012
	Elapsed Time	00 00:00:00.000

[DataSet7] /Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP II
, WL ratio, PP_FINAL_June15.sav

Between-Subjects Factors

		Value Label	N
Group	.00	CP	11
	1.00	CON	10

Descriptive Statistics

Dependent Variable:cPWV

Group	Mean	Std. Deviation	N
CP	4.2958	.63373	11
CON	4.0704	.93306	10
Total	4.1885	.77838	21

Tests of Between-Subjects Effects

Dependent Variable:cPWV

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	2.977[a]	2	1.489	2.932	.079
Intercept	1.778	1	1.778	3.502	.078
AGE	2.711	1	2.711	5.339	.033
Group	.124	1	.124	.244	.628
Error	9.140	18	.508		
Total	380.529	21			
Corrected Total	12.118	20			

a. R Squared = .246 (Adjusted R Squared = .162)

```
UNIANOVA Compliance BY Group WITH AGE
  /METHOD=SSTYPE(3)
  /INTERCEPT=INCLUDE
  /EMMEANS=TABLES(Group) WITH(AGE=MEAN) COMPARE ADJ(BONFERRONI)
  /PRINT=DESCRIPTIVE
  /CRITERIA=ALPHA(.05)
  /DESIGN=AGE Group.
```

Univariate Analysis of Variance

Notes

Output Created		19-Jun-2011 11:43:40
Comments		
Input	Data	/Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP IN, WL ratio, PP_FINAL_June15.sav
	Active Dataset	DataSet7
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	26
Missing Value Handling	Definition of Missing	User-defined missing values are treated as missing.
	Cases Used	Statistics are based on all cases with valid data for all variables in the model.
Syntax		UNIANOVA Compliance BY Group WITH AGE /METHOD=SSTYPE(3) /INTERCEPT=INCLUDE /EMMEANS=TABLES(Group) WITH(AGE=MEAN) COMPARE ADJ (BONFERRONI) /PRINT=DESCRIPTIVE /CRITERIA=ALPHA(.05) /DESIGN=AGE Group.
Resources	Processor Time	00 00:00:00.011
	Elapsed Time	00 00:00:00.000

[DataSet7] /Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP IN, WL ratio, PP_FINAL_June15.sav

Between-Subjects Factors

		Value Label	N
Group	.00	CP	11
	1.00	CON	10

Descriptive Statistics

Dependent Variable:Compliance

Group	Mean	Std. Deviation	N
CP	.1654	.06249	11
CON	.1855	.03289	10
Total	.1750	.05044	21

Tests of Between-Subjects Effects

Dependent Variable:Compliance

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	.011[a]	2	.005	2.404	.119
Intercept	.050	1	.050	22.327	.000
AGE	.009	1	.009	3.862	.065
Group	.001	1	.001	.469	.502
Error	.040	18	.002		
Total	.694	21			
Corrected Total	.051	20			

a. R Squared = .211 (Adjusted R Squared = .123)

Estimated Marginal Means

Group

Estimates

Dependent Variable:Compliance

Group	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
CP	.168[a]	.014	.138	.198
CON	.182[a]	.015	.151	.214

a. Covariates appearing in the model are evaluated at the following values: AGE = 12.8624.

Pairwise Comparisons

Dependent Variable:Compliance

(I) Group	(J) Group	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
CP	CON	-.014	.021	.502	-.058	.030
CON	CP	.014	.021	.502	-.030	.058

Based on estimated marginal means

a. Adjustment for multiple comparisons: Bonferroni.

Univariate Tests

Dependent Variable:Compliance

	Sum of Squares	df	Mean Square	F	Sig.
Contrast	.001	1	.001	.469	.502
Error	.040	18	.002		

The F tests the effect of Group. This test is based on the linearly independent pairwise comparisons among the estimated marginal means.

```
UNIANOVA Distensibility BY Group WITH AGE
  /METHOD=SSTYPE(3)
  /INTERCEPT=INCLUDE
  /EMMEANS=TABLES(Group) WITH(AGE=MEAN) COMPARE ADJ(BONFERRONI)
```

```
  /PRINT=DESCRIPTIVE
  /CRITERIA=ALPHA(.05)
  /DESIGN=AGE Group.
```

Univariate Analysis of Variance

Notes

Output Created		19-Jun-2011 11:46:12
Comments		
Input	Data	/Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP IN, WL ratio, PP_FINAL_June15.sav
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	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	26
Missing Value Handling	Definition of Missing	User-defined missing values are treated as missing.
	Cases Used	Statistics are based on all cases with valid data for all variables in the model.
Syntax		UNIANOVA Distensibility BY Group WITH AGE /METHOD=SSTYPE(3) /INTERCEPT=INCLUDE /EMMEANS=TABLES(Group) WITH(AGE=MEAN) COMPARE ADJ (BONFERRONI) /PRINT=DESCRIPTIVE /CRITERIA=ALPHA(.05) /DESIGN=AGE Group.
Resources	Processor Time	00 00:00:00.013
	Elapsed Time	00 00:00:00.000

```
[DataSet7] /Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP IN
, WL ratio, PP_FINAL_June15.sav
```

Between-Subjects Factors

		Value Label	N
Group	.00	CP	11
	1.00	CON	10

Descriptive Statistics

Dependent Variable:Distensibility

Group	Mean	Std. Deviation	N
CP	.0076	.00240	11
CON	.0082	.00179	10
Total	.0079	.00211	21

Tests of Between-Subjects Effects

Dependent Variable:Distensibility

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	7.876E-6[a]	2	3.938E-6	.875	.434
Intercept	6.769E-5	1	6.769E-5	15.045	.001
AGE	5.451E-6	1	5.451E-6	1.212	.286
Group	1.470E-6	1	1.470E-6	.327	.575
Error	8.099E-5	18	4.499E-6		
Total	.001	21			
Corrected Total	8.886E-5	20			

a. R Squared = .089 (Adjusted R Squared = -.013)

Estimated Marginal Means

Group

Estimates

Dependent Variable:Distensibility

Group	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
CP	.008[a]	.001	.006	.009
CON	.008[a]	.001	.007	.010

a. Covariates appearing in the model are evaluated at the following values: AGE = 12.8624.

Pairwise Comparisons

Dependent Variable:Distensibility

(I) Group	(J) Group	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
CP	CON	-.001	.001	.575	-.003	.001
CON	CP	.001	.001	.575	-.001	.003

Based on estimated marginal means

a. Adjustment for multiple comparisons: Bonferroni.

Univariate Tests

Dependent Variable:Distensibility

	Sum of Squares	df	Mean Square	F	Sig.
Contrast	1.470E-6	1	1.470E-6	.327	.575
Error	8.099E-5	18	4.499E-6		

The F tests the effect of Group. This test is based on the linearly independent pairwise comparisons among the estimated marginal means.

```
UNIANOVA Normalized BY Group WITH AGE
  /METHOD=SSTYPE(3)
  /INTERCEPT=INCLUDE
  /EMMEANS=TABLES(Group) WITH(AGE=MEAN) COMPARE ADJ(BONFERRONI)
```

```
  /PRINT=DESCRIPTIVE
  /CRITERIA=ALPHA(.05)
  /DESIGN=AGE Group.
```

Univariate Analysis of Variance

Notes

Output Created		19-Jun-2011 11:50:50
Comments		
Input	Data	/Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP IN, WL ratio, PP_FINAL_June15.sav
	Active Dataset	DataSet7
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	26
Missing Value Handling	Definition of Missing	User-defined missing values are treated as missing.
	Cases Used	Statistics are based on all cases with valid data for all variables in the model.
Syntax		UNIANOVA Normalized BY Group WITH AGE /METHOD=SSTYPE(3) /INTERCEPT=INCLUDE /EMMEANS=TABLES(Group) WITH(AGE=MEAN) COMPARE ADJ (BONFERRONI) /PRINT=DESCRIPTIVE /CRITERIA=ALPHA(.05) /DESIGN=AGE Group.
Resources	Processor Time	00 00:00:00.012
	Elapsed Time	00 00:00:00.000

```
[DataSet7] /Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP IN
, WL ratio, PP_FINAL_June15.sav
```

Between-Subjects Factors

		Value Label	N
Group	.00	CP	10
	1.00	CON	10

Descriptive Statistics

Dependent Variable:Normalized

Group	Mean	Std. Deviation	N
CP	.0046	.00334	10
CON	.0027	.00152	10
Total	.0036	.00271	20

Tests of Between-Subjects Effects

Dependent Variable:Normalized

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	3.656E-5[a]	2	1.828E-5	3.020	.075
Intercept	2.798E-6	1	2.798E-6	.462	.506
AGE	1.841E-5	1	1.841E-5	3.042	.099
Group	1.570E-5	1	1.570E-5	2.593	.126
Error	.000	17	6.053E-6		
Total	.000	20			
Corrected Total	.000	19			

a. R Squared = .262 (Adjusted R Squared = .175)

Estimated Marginal Means

Group

Estimates

Dependent Variable:Normalized

Group	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
CP	.004[a]	.001	.003	.006
CON	.003[a]	.001	.001	.004

a. Covariates appearing in the model are evaluated at the following values: AGE = 12.6815.

Pairwise Comparisons

Dependent Variable:Normalized

(I) Group	(J) Group	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
CP	CON	.002	.001	.126	-.001	.004
CON	CP	-.002	.001	.126	-.004	.001

Based on estimated marginal means

a. Adjustment for multiple comparisons: Bonferroni.

Univariate Tests

Dependent Variable:Normalized

	Sum of Squares	df	Mean Square	F	Sig.
Contrast	1.570E-5	1	1.570E-5	2.593	.126
Error	.000	17	6.053E-6		

The F tests the effect of Group. This test is based on the linearly independent pairwise comparisons among the estimated marginal means.

Partial Corr

Notes

Output Created		19-Jun-2011 11:55:31
Comments		
Input	Data	/Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP IN, WL ratio, PP_FINAL_June15.sav
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	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	26
Missing Value Handling	Definition of Missing	User defined missing values are treated as missing.
	Cases Used	Statistics are based on cases with no missing data for any variable listed.
Syntax		PARTIAL CORR /VARIABLES=cPWV pPWV Compliance Distensibility IMTf Absolute Relative Normalized LightPA MediumPA HardPA BMI WHR TPHV AGE BY BMIPercentile /SIGNIFICANCE=TWOTAIL /STATISTICS=DESCRIPTIVES /MISSING=LISTWISE.
Resources	Processor Time	00 00:00:00.030
	Elapsed Time	00 00:00:00.000

[DataSet7] /Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP IN, WL ratio, PP_FINAL_June15.sav

Descriptive Statistics

	Mean	Std. Deviation	N
cPWV	4.3353	.58573	18
pPWV	7.4479	1.43386	18
Compliance	.1746	.05056	18
Distensibility	.0080	.00226	18
IMTf	.4192	.03722	18
Absolute	.2636	.18533	18
Relative	8.7503	6.83003	18
Normalized	.0037	.00285	18
LightPA	92.5000	109.65521	18
MediumPA	160.8333	159.43143	18
HardPA	122.7778	164.72992	18
BMI	18.7000	3.30490	18
WHR	.4414	.04862	18
TPHV	-.6889	1.69007	18
AGE	13.0000	1.95482	18
BMIPercentile	45.2222	31.37732	18

Correlations

Control Variables			cPWV	pPWV	Compliance	Distensibility	IMTf
BMIPercentile	cPWV	Correlation	1.000	-.021	-.466	-.402	.047
		Significance (2-tailed)	.	.937	.059	.110	.859
		df	0	15	15	15	15
	pPWV	Correlation	-.021	1.000	-.235	-.256	-.482
		Significance (2-tailed)	.937	.	.363	.321	.050
		df	15	0	15	15	15
	Compliance	Correlation	-.466	-.235	1.000	.848	.164
		Significance (2-tailed)	.059	.363	.	.000	.530
		df	15	15	0	15	15
	Distensibility	Correlation	-.402	-.256	.848	1.000	.136
		Significance (2-tailed)	.110	.321	.000	.	.602
		df	15	15	15	0	15
	IMTf	Correlation	.047	-.482	.164	.136	1.000
		Significance (2-tailed)	.859	.050	.530	.602	.
		df	15	15	15	15	0
	Absolute	Correlation	.188	.380	-.529	-.478	-.173
		Significance (2-tailed)	.469	.133	.029	.052	.507
		df	15	15	15	15	15
	Relative	Correlation	.158	.393	-.512	-.426	-.153
		Significance (2-tailed)	.544	.118	.036	.088	.557
		df	15	15	15	15	15
	Normalized	Correlation	.116	.631	-.381	-.399	-.411
		Significance (2-tailed)	.656	.007	.131	.112	.101
		df	15	15	15	15	15
	LightPA	Correlation	.085	.068	.091	-.251	-.096
		Significance (2-tailed)	.745	.794	.727	.332	.715
		df	15	15	15	15	15
	MediumPA	Correlation	-.071	-.007	.242	-.091	.433
		Significance (2-tailed)	.787	.980	.349	.729	.083
		df	15	15	15	15	15
	HardPA	Correlation	-.010	.230	.184	.279	.043
		Significance (2-tailed)	.969	.374	.480	.278	.871
		df	15	15	15	15	15
	BMI	Correlation	.074	.331	-.499	-.506	-.342
		Significance (2-tailed)	.779	.195	.042	.038	.179
		df	15	15	15	15	15
	WHR	Correlation	-.428	.135	-.295	-.291	-.068
		Significance (2-tailed)	.087	.604	.250	.257	.795
		df	15	15	15	15	15
	TPHV	Correlation	.166	.196	-.233	-.152	-.547
		Significance (2-tailed)	.524	.451	.369	.560	.023
		df	15	15	15	15	15

Correlations

Control Variables			Absolute	Relative	Normalized	LightPA	MediumPA	HardPA
BMIPercentile	cPWV	Correlation	.188	.158	.116	.085	-.071	-.010
		Significance (2-tailed)	.469	.544	.656	.745	.787	.969
		df	15	15	15	15	15	15
	pPWV	Correlation	.380	.393	.631	.068	-.007	.230
		Significance (2-tailed)	.133	.118	.007	.794	.980	.374
		df	15	15	15	15	15	15
	Compliance	Correlation	-.529	-.512	-.381	.091	.242	.184
		Significance (2-tailed)	.029	.036	.131	.727	.349	.480
		df	15	15	15	15	15	15
	Distensibility	Correlation	-.478	-.426	-.399	-.251	-.091	.279
		Significance (2-tailed)	.052	.088	.112	.332	.729	.278
		df	15	15	15	15	15	15
	IMTf	Correlation	-.173	-.153	-.411	-.096	.433	.043
		Significance (2-tailed)	.507	.557	.101	.715	.083	.871
		df	15	15	15	15	15	15
	Absolute	Correlation	1.000	.982	.802	.282	.051	-.281
		Significance (2-tailed)	.	.000	.000	.273	.847	.274
		df	0	15	15	15	15	15
	Relative	Correlation	.982	1.000	.788	.181	.072	-.218
		Significance (2-tailed)	.000	.	.000	.487	.783	.401
		df	15	0	15	15	15	15
	Normalized	Correlation	.802	.788	1.000	.165	-.074	-.144
		Significance (2-tailed)	.000	.000	.	.528	.777	.580
		df	15	15	0	15	15	15
	LightPA	Correlation	.282	.181	.165	1.000	.318	-.309
		Significance (2-tailed)	.273	.487	.528	.	.213	.227
		df	15	15	15	0	15	15
	MediumPA	Correlation	.051	.072	-.074	.318	1.000	.164
		Significance (2-tailed)	.847	.783	.777	.213	.	.530
		df	15	15	15	15	0	15
	HardPA	Correlation	-.281	-.218	-.144	-.309	.164	1.000
		Significance (2-tailed)	.274	.401	.580	.227	.530	.
		df	15	15	15	15	15	0
	BMI	Correlation	.388	.280	.378	.310	-.375	-.425
		Significance (2-tailed)	.124	.276	.135	.225	.138	.089
		df	15	15	15	15	15	15
	WHR	Correlation	.477	.439	.389	.039	.043	-.407
		Significance (2-tailed)	.053	.078	.122	.883	.871	.105
		df	15	15	15	15	15	15
	TPHV	Correlation	.074	.024	.113	.145	-.599	-.169
		Significance (2-tailed)	.778	.927	.664	.580	.011	.517
		df	15	15	15	15	15	15

Correlations

Control Variables			BMI	WHR	TPHV	AGE
BMIPercentile	cPWV	Correlation	.074	-.428	.166	.406
		Significance (2-tailed)	.779	.087	.524	.106
		df	15	15	15	15
	pPWV	Correlation	.331	.135	.196	.201
		Significance (2-tailed)	.195	.604	.451	.439
		df	15	15	15	15
	Compliance	Correlation	-.499	-.295	-.233	-.440
		Significance (2-tailed)	.042	.250	.369	.077
		df	15	15	15	15
	Distensibility	Correlation	-.506	-.291	-.152	-.381
		Significance (2-tailed)	.038	.257	.560	.131
		df	15	15	15	15
	IMTf	Correlation	-.342	-.068	-.547	-.508
		Significance (2-tailed)	.179	.795	.023	.038
		df	15	15	15	15
	Absolute	Correlation	.388	.477	.074	.340
		Significance (2-tailed)	.124	.053	.778	.182
		df	15	15	15	15
	Relative	Correlation	.280	.439	.024	.236
		Significance (2-tailed)	.276	.078	.927	.362
		df	15	15	15	15
	Normalized	Correlation	.378	.389	.113	.362
		Significance (2-tailed)	.135	.122	.664	.153
		df	15	15	15	15
	LightPA	Correlation	.310	.039	.145	.310
		Significance (2-tailed)	.225	.883	.580	.226
		df	15	15	15	15
	MediumPA	Correlation	-.375	.043	-.599	-.490
		Significance (2-tailed)	.138	.871	.011	.046
		df	15	15	15	15
	HardPA	Correlation	-.425	-.407	-.169	-.235
		Significance (2-tailed)	.089	.105	.517	.364
		df	15	15	15	15
	BMI	Correlation	1.000	.455	.587	.682
		Significance (2-tailed)	.	.067	.013	.003
		df	0	15	15	15
	WHR	Correlation	.455	1.000	-.235	-.044
		Significance (2-tailed)	.067	.	.363	.867
		df	15	0	15	15
	TPHV	Correlation	.587	-.235	1.000	.809
		Significance (2-tailed)	.013	.363	.	.000
		df	15	15	0	15

Correlations

Control Variables			cPWV	pPWV	Compliance	Distensibility	IMTf
BMIPercentile	AGE	Correlation	.406	.201	-.440	-.381	-.508
		Significance (2-tailed)	.106	.439	.077	.131	.038
		df	15	15	15	15	15

Correlations

Control Variables			Absolute	Relative	Normalized	LightPA	MediumPA	HardPA
BMIPercentile	AGE	Correlation	.340	.236	.362	.310	-.490	-.235
		Significance (2-tailed)	.182	.362	.153	.226	.046	.364
		df	15	15	15	15	15	15

Correlations

Control Variables			BMI	WHR	TPHV	AGE
BMIPercentile	AGE	Correlation	.682	-.044	.809	1.000
		Significance (2-tailed)	.003	.867	.000	.
		df	15	15	15	0

```
UNIANOVA cPWV BY Group WITH BMIPercentile
  /METHOD=SSTYPE(3)
  /INTERCEPT=INCLUDE
  /PRINT=DESCRIPTIVE
  /CRITERIA=ALPHA(.05)
  /DESIGN=BMIPercentile Group.
```

Univariate Analysis of Variance

Notes

Output Created		19-Jun-2011 11:57:05
Comments		
Input	Data	/Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP IN, WL ratio, PP_FINAL_June15.sav
	Active Dataset	DataSet7
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	26
Missing Value Handling	Definition of Missing	User-defined missing values are treated as missing.
	Cases Used	Statistics are based on all cases with valid data for all variables in the model.
Syntax		UNIANOVA cPWV BY Group WITH BMIPercentile /METHOD=SSTYPE(3) /INTERCEPT=INCLUDE /PRINT=DESCRIPTIVE /CRITERIA=ALPHA(.05) /DESIGN=BMIPercentile Group.
Resources	Processor Time	00 00:00:00.010
	Elapsed Time	00 00:00:00.000

```
[DataSet7] /Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP II
, WL ratio, PP_FINAL_June15.sav
```

Between-Subjects Factors

		Value Label	N
Group	.00	CP	11
	1.00	CON	10

Descriptive Statistics

Dependent Variable:cPWV

Group	Mean	Std. Deviation	N
CP	4.2958	.63373	11
CON	4.0704	.93306	10
Total	4.1885	.77838	21

Tests of Between-Subjects Effects

Dependent Variable:cPWV

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	2.363[a]	2	1.181	2.180	.142
Intercept	126.881	1	126.881	234.124	.000
BMIPercentile	2.097	1	2.097	3.869	.065
Group	.000	1	.000	.000	.989
Error	9.755	18	.542		
Total	380.529	21			
Corrected Total	12.118	20			

a. R Squared = .195 (Adjusted R Squared = .106)

```
CORRELATIONS
  /VARIABLES=BMIPercentile cPWV
  /PRINT=TWOTAIL NOSIG
  /MISSING=PAIRWISE.
```

Correlations

Notes

Output Created		19-Jun-2011 11:57:46
Comments		
Input	Data	/Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP IN, WL ratio, PP_FINAL_June15.sav
	Active Dataset	DataSet7
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	26
Missing Value Handling	Definition of Missing	User-defined missing values are treated as missing.
	Cases Used	Statistics for each pair of variables are based on all the cases with valid data for that pair.
Syntax		CORRELATIONS /VARIABLES=BMIPercentile cPWV /PRINT=TWOTAIL NOSIG /MISSING=PAIRWISE.
Resources	Processor Time	00 00:00:00.004
	Elapsed Time	00 00:00:00.000

```
[DataSet7] /Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP IN
, WL ratio, PP_FINAL_June15.sav
```

Correlations

		BMIPercentile	cPWV
BMIPercentile	Pearson Correlation	1	-.442*
	Sig. (2-tailed)		.045
	N	22	21
cPWV	Pearson Correlation	-.442*	1
	Sig. (2-tailed)	.045	
	N	21	21

*. Correlation is significant at the 0.05 level (2-tailed).

```
UNIANOVA cPWV BY Group WITH HardPA
  /METHOD=SSTYPE(3)
  /INTERCEPT=INCLUDE
  /PRINT=DESCRIPTIVE
  /CRITERIA=ALPHA(.05)
  /DESIGN=HardPA Group.
```

Univariate Analysis of Variance

Notes

Output Created		19-Jun-2011 12:00:52
Comments		
Input	Data	/Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP IN, WL ratio, PP_FINAL_June15.sav
	Active Dataset	DataSet7
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	26
Missing Value Handling	Definition of Missing	User-defined missing values are treated as missing.
	Cases Used	Statistics are based on all cases with valid data for all variables in the model.
Syntax		UNIANOVA cPWV BY Group WITH HardPA /METHOD=SSTYPE(3) /INTERCEPT=INCLUDE /PRINT=DESCRIPTIVE /CRITERIA=ALPHA(.05) /DESIGN=HardPA Group.
Resources	Processor Time	00 00:00:00.011
	Elapsed Time	00 00:00:00.000

[DataSet7] /Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP IN, WL ratio, PP_FINAL_June15.sav

Between-Subjects Factors

		Value Label	N
Group	.00	OP	11
	1.00	CON	10

Descriptive Statistics

Dependent Variable:cPWV

Group	Mean	Std. Deviation	N
OP	4.2958	.63373	11
CON	4.0704	.93306	10
Total	4.1885	.77838	21

Tests of Between-Subjects Effects

Dependent Variable:cPWV

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	.355[a]	2	.178	.272	.765
Intercept	203.310	1	203.310	311.132	.000
HardPA	.089	1	.089	.137	.716
Group	.355	1	.355	.544	.470
Error	11.762	18	.653		
Total	380.529	21			
Corrected Total	12.118	20			

a. R Squared = .029 (Adjusted R Squared = -.079)

```
UNIANOVA Distensibility BY Group WITH HardPA
  /METHOD=SSTYPE(3)
  /INTERCEPT=INCLUDE
  /PRINT=DESCRIPTIVE
  /CRITERIA=ALPHA(.05)
  /DESIGN=HardPA Group.
```

Univariate Analysis of Variance

Notes

Output Created		19-Jun-2011 12:03:15
Comments		
Input	Data	/Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP IN, WL ratio, PP_FINAL_June15.sav
	Active Dataset	DataSet7
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	26
Missing Value Handling	Definition of Missing	User-defined missing values are treated as missing.
	Cases Used	Statistics are based on all cases with valid data for all variables in the model.
Syntax		UNIANOVA Distensibility BY Group WITH HardPA /METHOD=SSTYPE(3) /INTERCEPT=INCLUDE /PRINT=DESCRIPTIVE /CRITERIA=ALPHA(.05) /DESIGN=HardPA Group.
Resources	Processor Time	00 00:00:00.012
	Elapsed Time	00 00:00:00.000

```
[DataSet7] /Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP IN
, WL ratio, PP_FINAL_June15.sav
```

Between-Subjects Factors

		Value Label	N
Group	.00	CP	11
	1.00	CON	10

Descriptive Statistics

Dependent Variable:Distensibility

Group	Mean	Std. Deviation	N
CP	.0076	.00240	11
CON	.0082	.00179	10
Total	.0079	.00211	21

Tests of Between-Subjects Effects

Dependent Variable:Distensibility

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	6.033E-6[a]	2	3.017E-6	.656	.531
Intercept	.001	1	.001	139.344	.000
HardPA	3.609E-6	1	3.609E-6	.784	.388
Group	9.083E-8	1	9.083E-8	.020	.890
Error	8.283E-5	18	4.602E-6		
Total	.001	21			
Corrected Total	8.886E-5	20			

a. R Squared = .068 (Adjusted R Squared = -.036)

```
UNIANOVA cPWV BY Group WITH WHR
  /METHOD=SSTYPE(3)
  /INTERCEPT=INCLUDE
  /EMMEANS=TABLES(Group) WITH(WHR=MEAN) COMPARE ADJ(BONFERRONI)
  /PRINT=DESCRIPTIVE
  /CRITERIA=ALPHA(.05)
  /DESIGN=WHR Group.
```

Univariate Analysis of Variance

Notes

Output Created		19-Jun-2011 12:13:39
Comments		
Input	Data	/Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP IN, WL ratio, PP_FINAL_June15.sav
	Active Dataset	DataSet7
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	26
Missing Value Handling	Definition of Missing	User-defined missing values are treated as missing.
	Cases Used	Statistics are based on all cases with valid data for all variables in the model.
Syntax		UNIANOVA cPWV BY Group WITH WHR /METHOD=SSTYPE(3) /INTERCEPT=INCLUDE /EMMEANS=TABLES(Group) WITH(WHR=MEAN) COMPARE ADJ (BONFERRONI) /PRINT=DESCRIPTIVE /CRITERIA=ALPHA(.05) /DESIGN=WHR Group.
Resources	Processor Time	00 00:00:00.012
	Elapsed Time	00 00:00:00.000

[DataSet7] /Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP IN, WL ratio, PP_FINAL_June15.sav

Between-Subjects Factors

		Value Label	N
Group	.00	CP	11
	1.00	CON	10

Descriptive Statistics

Dependent Variable:cPWV

Group	Mean	Std. Deviation	N
CP	4.2958	.63373	11
CON	4.0704	.93306	10
Total	4.1885	.77838	21

Tests of Between-Subjects Effects

Dependent Variable:cPWV

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	3.206[a]	2	1.603	3.238	.063
Intercept	15.366	1	15.366	31.036	.000
WHR	2.940	1	2.940	5.938	.025
Group	.385	1	.385	.778	.389
Error	8.912	18	.495		
Total	380.529	21			
Corrected Total	12.118	20			

a. R Squared = .265 (Adjusted R Squared = .183)

Estimated Marginal Means

Group

Estimates

Dependent Variable:cPWV

Group	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
CP	4.318[a]	.212	3.872	4.764
CON	4.046[a]	.223	3.578	4.514

a. Covariates appearing in the model are evaluated at the following values: WHR = .4477.

Pairwise Comparisons

Dependent Variable:cPWV

(I) Group	(J) Group	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
CP	CON	.272	.308	.389	-.376	.919
CON	CP	-.272	.308	.389	-.919	.376

Based on estimated marginal means

a. Adjustment for multiple comparisons: Bonferroni.

Univariate Tests

Dependent Variable:cPWV

	Sum of Squares	df	Mean Square	F	Sig.
Contrast	.385	1	.385	.778	.389
Error	8.912	18	.495		

The F tests the effect of Group. This test is based on the linearly independent pairwise comparisons among the estimated marginal means.

```
NEW FILE.
DATASET NAME DataSet4 WINDOW=FRONT.
DATASET ACTIVATE DataSet3.
T-TEST GROUPS=Visit(1 2)
  /MISSING=ANALYSIS
  /VARIABLES=Absolute Relative Normalized
  /CRITERIA=CI(.95).
```

T-Test

Notes

Output Created		17-Jun-2011 17:15:30
Comments		
Input	Active Dataset	DataSet3
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	24
Missing Value Handling	Definition of Missing	User defined missing values are treated as missing.
	Cases Used	Statistics for each analysis are based on the cases with no missing or out-of-range data for any variable in the analysis.
Syntax		T-TEST GROUPS=Visit(1 2) /MISSING=ANALYSIS /VARIABLES=Absolute Relative Normalized /CRITERIA=CI(.95).
Resources	Processor Time	00 00:00:00.007
	Elapsed Time	00 00:00:00.000

[DataSet3]

Group Statistics

	Visit	N	Mean	Std. Deviation	Std. Error Mean
Absolute	Visit 1	12	.2257	.11470	.03311
	Visit 2	12	.3684	.12273	.03543
Relative	Visit 1	12	7.1385	3.57653	1.03246
	Visit 2	12	11.8547	4.33532	1.25150
Normalized	Visit 1	12	.0035	.00262	.00076
	Visit 2	12	.0050	.00255	.00074

Independent Samples Test

		Levene's Test for Equality of Variances		t-test for Equality of Means			
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference
Absolute	Equal variances assumed	.029	.866	-2.943	22	.008	-.14272
	Equal variances not assumed			-2.943	21.900	.008	-.14272
Relative	Equal variances assumed	.643	.431	-2.907	22	.008	-4.71621
	Equal variances not assumed			-2.907	21.233	.008	-4.71621
Normalized	Equal variances assumed	.148	.704	-1.451	22	.161	-.00153
	Equal variances not assumed			-1.451	21.986	.161	-.00153

Independent Samples Test

		t-test for Equality of Means		
			95% Confidence Interval of the Difference	
		Std. Error Difference	Lower	Upper
Absolute	Equal variances assumed	.04849	-.24329	-.04215
	Equal variances not assumed	.04849	-.24332	-.04213
Relative	Equal variances assumed	1.62241	-8.08088	-1.35154
	Equal variances not assumed	1.62241	-8.08794	-1.34448
Normalized	Equal variances assumed	.00106	-.00372	.00066
	Equal variances not assumed	.00106	-.00372	.00066

```
CORRELATIONS
  /VARIABLES=Absolute1 Absolute2
  /PRINT=TWOTAIL NOSIG
  /STATISTICS XPROD
  /MISSING=PAIRWISE.
```

Correlations

Notes

Output Created		17-Jun-2011 17:17:58
Comments		
Input	Active Dataset	DataSet4
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	12
Missing Value Handling	Definition of Missing	User-defined missing values are treated as missing.
	Cases Used	Statistics for each pair of variables are based on all the cases with valid data for that pair.
Syntax		CORRELATIONS /VARIABLES=Absolute1 Absolute2 /PRINT=TWOTAIL NOSIG /STATISTICS XPROD /MISSING=PAIRWISE.
Resources	Processor Time	00 00:00:00.009
	Elapsed Time	00 00:00:00.000

[DataSet4]

Correlations

		Absolute1	Absolute2
Absolute1	Pearson Correlation	1	.250
	Sig. (2-tailed)		.433
	Sum of Squares and Cross-products	.145	.039
	Covariance	.013	.004
	N	12	12
Absolute2	Pearson Correlation	.250	1
	Sig. (2-tailed)	.433	
	Sum of Squares and Cross-products	.039	.166
	Covariance	.004	.015
	N	12	12

```
CORRELATIONS
  /VARIABLES=Relative1 Relative2
  /PRINT=TWOTAIL NOSIG
  /STATISTICS XPROD
  /MISSING=PAIRWISE.
```

Correlations

Notes

Output Created		17-Jun-2011 17:19:00
Comments		
Input	Active Dataset	DataSet4
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	12
Missing Value Handling	Definition of Missing	User-defined missing values are treated as missing.
	Cases Used	Statistics for each pair of variables are based on all the cases with valid data for that pair.
Syntax		CORRELATIONS /VARIABLES=Relative1 Relative2 /PRINT=TWOTAIL NOSIG /STATISTICS XPROD /MISSING=PAIRWISE.
Resources	Processor Time	00 00:00:00.007
	Elapsed Time	00 00:00:00.000

[DataSet4]

Correlations

		Relative1	Relative2
Relative1	Pearson Correlation	1	.265
	Sig. (2-tailed)		.405
	Sum of Squares and Cross-products	140.707	45.215
	Covariance	12.792	4.110
	N	12	12
Relative2	Pearson Correlation	.265	1
	Sig. (2-tailed)	.405	
	Sum of Squares and Cross-products	45.215	206.745
	Covariance	4.110	18.795
	N	12	12

```
CORRELATIONS
  /VARIABLES=Normalized1 Normalized2
  /PRINT=TWOTAIL NOSIG
  /STATISTICS XPROD
  /MISSING=PAIRWISE.
```

Correlations

Notes

Output Created		17-Jun-2011 17:19:29
Comments		
Input	Active Dataset	DataSet4
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	12
Missing Value Handling	Definition of Missing	User-defined missing values are treated as missing.
	Cases Used	Statistics for each pair of variables are based on all the cases with valid data for that pair.
Syntax		CORRELATIONS /VARIABLES=Normalized1 Normalized2 /PRINT=TWOTAIL NOSIG /STATISTICS XPROD /MISSING=PAIRWISE.
Resources	Processor Time	00 00:00:00.003
	Elapsed Time	00 00:00:00.000

[DataSet4]

Correlations

		Normalized1	Normalized2
Normalized1	Pearson Correlation	1	.547
	Sig. (2-tailed)		.066
	Sum of Squares and Cross-products	.000	.000
	Covariance	.000	.000
	N	12	12
Normalized2	Pearson Correlation	.547	1
	Sig. (2-tailed)	.066	
	Sum of Squares and Cross-products	.000	.000
	Covariance	.000	.000
	N	12	12

```
SAVE OUTFILE='/Users/audramartin/Desktop/MSc Thesis STATS/Reliability, 1 row for each '+
    'subject_June17.sav'
  /COMPRESSED.
DATASET ACTIVATE DataSet1.
DATASET CLOSE DataSet4.
DATASET ACTIVATE DataSet3.

SAVE OUTFILE='/Users/audramartin/Desktop/MSc Thesis STATS/Reliability, stacked groups_June17.sa
  /COMPRESSED.
DATASET ACTIVATE DataSet1.
DATASET CLOSE DataSet3.
GET
  FILE='/Users/audramartin/Desktop/MSc Thesis STATS/Reliability, 1 row for each subject_June17
DATASET NAME DataSet5 WINDOW=FRONT.
T-TEST PAIRS=Absolute1 Relative1 Normalized1 WITH Absolute2 Relative2 Normalized2 (PAIRED)
  /CRITERIA=CI(.9500)
```

```
/MISSING=ANALYSIS.
```

T-Test

Notes

Output Created		18-Jun-2011 19:01:37
Comments		
Input	Data	/Users/audramartin/Desktop/MSc Thesis STATS/Reliability, 1 row for each subject_June17.sav
	Active Dataset	DataSet5
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	12
Missing Value Handling	Definition of Missing	User defined missing values are treated as missing.
	Cases Used	Statistics for each analysis are based on the cases with no missing or out-of-range data for any variable in the analysis.
Syntax		T-TEST PAIRS=Absolute1 Relative1 Normalized1 WITH Absolute2 Relative2 Normalized2 (PAIRED) /CRITERIA=CI(.9500) /MISSING=ANALYSIS.
Resources	Processor Time	00 00:00:00.006
	Elapsed Time	00 00:00:00.000

```
[DataSet5] /Users/audramartin/Desktop/MSc Thesis STATS/Reliability, 1 row for each subject_June
17.sav
```

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Absolute1	.2257	12	.11470	.03311
	Absolute2	.3684	12	.12273	.03543
Pair 2	Relative1	7.1385	12	3.57653	1.03246
	Relative2	11.8547	12	4.33532	1.25150
Pair 3	Normalized1	.0035	12	.00262	.00076
	Normalized2	.0050	12	.00255	.00074

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	Absolute1 & Absolute2	12	.250	.433
Pair 2	Relative1 & Relative2	12	.265	.405
Pair 3	Normalized1 & Normalized2	12	.547	.066

Paired Samples Test

		Paired Differences				
					95% Confidence Interval of the Difference	
		Mean	Std. Deviation	Std. Error Mean	Lower	Upper
Pair 1	Absolute1 - Absolute2	-.14272	.14554	.04202	-.23520	-.05025
Pair 2	Relative1 - Relative2	-4.71621	4.83380	1.39540	-7.78746	-1.64496
Pair 3	Normalized1 - Normalized2	-.00153	.00246	.00071	-.00310	.00003

Paired Samples Test

		t	df	Sig. (2-tailed)
Pair 1	Absolute1 - Absolute2	-3.397	11	.006
Pair 2	Relative1 - Relative2	-3.380	11	.006
Pair 3	Normalized1 - Normalized2	-2.154	11	.054

```
GET
  FILE='/Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP IN, r
DATASET NAME DataSet6 WINDOW=FRONT.
DATASET ACTIVATE DataSet1.
DATASET CLOSE DataSet6.

SAVE OUTFILE='/Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-5COAL PWV '+
    'removed_FINAL_June15.sav'
  /COMPRESSED.
DATASET ACTIVATE DataSet5.
DATASET CLOSE DataSet1.
GET
  FILE='/Users/audramartin/Desktop/MSc Thesis STATS/MSc Stats-Outliers REMOVED, supine BP IN, r
DATASET NAME DataSet7 WINDOW=FRONT.
DATASET ACTIVATE DataSet5.
GLM Normalized1 Normalized2
  /WSFACTOR=Normalized 2 Polynomial
  /METHOD=SSTYPE(3)
  /CRITERIA=ALPHA(.05)
  /WSDESIGN=Normalized.
```

General Linear Model

Notes

Output Created		19-Jun-2011 17:53:06
Comments		
Input	Data	/Users/audramartin/Desktop/MSc Thesis STATS/Reliability, 1 row for each subject_June17.sav
	Active Dataset	DataSet5
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	12
Missing Value Handling	Definition of Missing	User-defined missing values are treated as missing.
	Cases Used	Statistics are based on all cases with valid data for all variables in the model.
Syntax		GLM Normalized1 Normalized2 /WSFACTOR=Normalized 2 Polynomial /METHOD=SSTYPE(3) /CRITERIA=ALPHA(.05) /WSDESIGN=Normalized.
Resources	Processor Time	00 00:00:00.026
	Elapsed Time	00 00:00:00.000

[DataSet5] /Users/audramartin/Desktop/MSc Thesis STATS/Reliability, 1 row for each subject_June17.sav

Within-Subjects Factors

Measure:MEASURE_1

Normalized	Dependent Variable
1	Normalized1
2	Normalized2

Multivariate Tests[b]

Effect		Value	F	Hypothesis df	Error df	Sig.
Normalized	Pillai's Trace	.297	4.640[a]	1.000	11.000	.054
	Wilks' Lambda	.703	4.640[a]	1.000	11.000	.054
	Hotelling's Trace	.422	4.640[a]	1.000	11.000	.054
	Roy's Largest Root	.422	4.640[a]	1.000	11.000	.054

a. Exact statistic
b. Design: Intercept
Within Subjects Design: Normalized

Mauchly's Test of Sphericity[b]

Measure:MEASURE_1

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[a]		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Normalized	1.000	.000	0	.	1.000	1.000	1.000

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.
b. Design: Intercept
Within Subjects Design: Normalized

Tests of Within-Subjects Effects

Measure:MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Normalized	Sphericity Assumed	1.407E-5	1	1.407E-5	4.640	.054
	Greenhouse-Geisser	1.407E-5	1.000	1.407E-5	4.640	.054
	Huynh-Feldt	1.407E-5	1.000	1.407E-5	4.640	.054
	Lower-bound	1.407E-5	1.000	1.407E-5	4.640	.054
Error(Normalized)	Sphericity Assumed	3.337E-5	11	3.033E-6		
	Greenhouse-Geisser	3.337E-5	11.000	3.033E-6		
	Huynh-Feldt	3.337E-5	11.000	3.033E-6		
	Lower-bound	3.337E-5	11.000	3.033E-6		

Tests of Within-Subjects Contrasts

Measure:MEASURE_1

Source	Normalized	Type III Sum of Squares	df	Mean Square	F	Sig.
Normalized	Linear	1.407E-5	1	1.407E-5	4.640	.054
Error(Normalized)	Linear	3.337E-5	11	3.033E-6		

Tests of Between-Subjects Effects

Measure:MEASURE_1
Transformed Variable:Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	.000	1	.000	41.690	.000
Error	.000	11	1.034E-5		

```
GLM Relative1 Relative2
  /WSFACTOR=Relative 2 Polynomial
  /METHOD=SSTYPE(3)
  /CRITERIA=ALPHA(.05)
  /WSDESIGN=Relative.
```

General Linear Model

Notes

Output Created		19-Jun-2011 17:56:42
Comments		
Input	Data	/Users/audramartin/Desktop/MSc Thesis STATS/Reliability, 1 row for each subject_June17.sav
	Active Dataset	DataSet5
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	12
Missing Value Handling	Definition of Missing	User-defined missing values are treated as missing.
	Cases Used	Statistics are based on all cases with valid data for all variables in the model.
Syntax		GLM Relative1 Relative2 /WSFACTOR=Relative 2 Polynomial /METHOD=SSTYPE(3) /CRITERIA=ALPHA(.05) /WSDESIGN=Relative.
Resources	Processor Time	00 00:00:00.010
	Elapsed Time	00 00:00:00.000

```
[DataSet5] /Users/audramartin/Desktop/MSc Thesis STATS/Reliability, 1 row for each subject_June
17.sav
```

Within-Subjects Factors

Measure:MEASURE_1

Relative	Dependent Variable
1	Relative1
2	Relative2

Multivariate Tests[b]

Effect		Value	F	Hypothesis df	Error df	Sig.
Relative	Pillai's Trace	.509	11.423[a]	1.000	11.000	.006
	Wilks' Lambda	.491	11.423[a]	1.000	11.000	.006
	Hotelling's Trace	1.038	11.423[a]	1.000	11.000	.006
	Roy's Largest Root	1.038	11.423[a]	1.000	11.000	.006

a. Exact statistic
b. Design: Intercept
Within Subjects Design: Relative

Mauchly's Test of Sphericity[b]

Measure:MEASURE_1

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[a]		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
Relative	1.000	.000	0	.	1.000	1.000	1.000

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.
b. Design: Intercept
Within Subjects Design: Relative

Tests of Within-Subjects Effects

Measure:MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Relative	Sphericity Assumed	133.456	1	133.456	11.423	.006
	Greenhouse-Geisser	133.456	1.000	133.456	11.423	.006
	Huynh-Feldt	133.456	1.000	133.456	11.423	.006
	Lower-bound	133.456	1.000	133.456	11.423	.006
Error(Relative)	Sphericity Assumed	128.511	11	11.683		
	Greenhouse-Geisser	128.511	11.000	11.683		
	Huynh-Feldt	128.511	11.000	11.683		
	Lower-bound	128.511	11.000	11.683		

Tests of Within-Subjects Contrasts

Measure:MEASURE_1

Source	Relative	Type III Sum of Squares	df	Mean Square	F	Sig.
Relative	Linear	133.456	1	133.456	11.423	.006
Error(Relative)	Linear	128.511	11	11.683		

Tests of Between-Subjects Effects

Measure:MEASURE_1
Transformed Variable:Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	2164.432	1	2164.432	108.745	.000
Error	218.941	11	19.904		