**Pulse Wave Velocity (PWV) for Assessment of Arterial Stiffness Among People with Spinal Cord Injury: A Pilot Study**

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**Abstract**

**BACKGROUND:** The most significant complication and leading cause of death for people with spinal cord injury (SCI) is coronary artery disease (CAD). It has been confirmed that Aortic pulse wave velocity (PWV) is an emerging CAD predictor among able-bodied individuals. No prior study has described PWV values among people with SCI.

**PURPOSE:** To compare: Aortic (the common carotid to femoral artery) PWV, Arm (the brachial to radial artery) PWV, and Leg (the femoral to posterior tibial artery) PWV in people with SCI (SCI group) to that of age, gender, height and weight matched able-bodied controls (non-SCI group).

**METHODS:** Subjects included 12 men with SCI and nine age, gender, height and weight matched non-SCI controls. Subjects with a history of CAD or current metabolic syndrome were excluded. Aortic, Arm and Leg PWV was measured using the echo Doppler method.

**RESULTS**: Aortic PWV (Mean ± SD) in the SCI group (1274 ± 369cm/sec) was significantly higher ($P<0.05$) than in the non- SCI group (948 ± 110cm/sec). There were no significant between group differences in the mean Arm PWV (SCI: 1152 ± 193cm/sec,

non-SCI: 1237±193cm/sec) nor the mean Leg PWV (SCI: 1096±173cm/sec, non-SCI: 994±178cm/sec) values.

**CONCLUSIONS**: Aortic PWV was higher among the SCI group compared to the non-SCI group. The higher mean Aortic PWV values among the SCI group compared to the non-SCI group indicated a higher risk of CAD among SCI subjects in the absence of metabolic syndrome.

**Key Words:** Arterial stiffness; Pulse wave velocity; Spinal cord injuries; Doppler ultrasound, Coronary artery disease, Diagnosis

## Introduction

Coronary artery disease (CAD) is the most significant complication and leading cause of mortality after spinal cord injury (SCI)[1]. Individuals with chronic SCI have higher cardiovascular mortality rates and cardiovascular mortality occurs at earlier ages when compared to the able-bodied population[2-4]. Stiffening of the central or cardiothoracic arteries is a significant independent risk factor for CAD in able-bodied people[5-7]. Decreases in the elastic properties of arteries reduce their buffering capacity, leading to increased pulse pressure, aortic impedance, and left ventricular wall tension. All of which augment the workload of the heart, thereby increasing CAD risk. Several indices have been used to quantify the stiffness of the peripheral and cardiothoracic arteries. These include: 1) measuring Pulse Wave Velocity (PWV); 2) relating changes in arterial diameter to distending pressure; and 3) assessing arterial pressure wave forms. Of the above indirect methods for measuring arterial stiffness, PWV is the most widely accepted technique[8]. PWV has been a useful noninvasive measure to assess arterial stiffness and severity of CAD among able bodied people in a number of previous studies[9-11].

PWV is the velocity of the blood pressure wave as it travels a known distance between two anatomic sites within the arterial system, and is determined by the elasticity and other properties of the artery[12]. PWV values positively correlate with arterial distensibility and stiffness. Three locations or the measurement of PWV have been proposed: 1) trunk (Aortic PWV); 2) arm (Arm PWV); and 3) leg (Leg PWV). Aortic PWV is the established index for measuring arterial stiffness.

Aortic PWV values have been directly linked with cardiovascular mortality, fatal and nonfatal coronary events and fatal strokes in patients with low and high levels of traditional CAD risk factors[9-11, 13-15]. For example, Aortic PWV values ≥1300 cm/sec are a strong predictor of cardiac mortality among hypertensive patients[13]. Among people with hypertension, a 500 cm/sec increment in Aortic PWV is an independent predictor of both cardiovascular mortality (odds ratio=1.34) and all-cause mortality (odds ratio=1.51)[14]. Although Leg PWV and Arm PWV have not been studied to the same extent, it has been suggested that these peripheral PWV measures are insensitive to physical activity levels and/or aging compared to Aortic PWV in able-bodied people[16, 17].

Several CAD risk factors have been identified as determinants of PWV in the able bodied population these include: obesity[18-20], diabetes[21, 22], hypercholesterolaemia[23] and hypertension[9, 24], poor cardiorespiratory fitness[25, 26], and low physical activity[27]. These same CAD risk factors are common among people with SCI[28-32]. In addition, people with SCI above the splachnic outflow (T6) have autonomic dysfunction, which may contribute to disordered cardiac regulation and abnormalities of the vascular system. Thus, it was hypothesized that people with chronic SCI will have an increased risk of adverse vascular health and increased arterial stiffness as measured by PWV.

The purpose of this study was to compare Aortic PWV in people with chronic SCI (SCI group) to that of age, gender, height and weight matched able-bodied controls (non-SCI group), and to compare Arm PWV and Leg PWV in these same groups to determine if differences exist in the values obtained.

**Methods**

*Subjects*: The SCI group was comprised of fifteen subjects with SCI (C3-T10, AIS A, B and C). The non- SCI group was comprised of 11 age, height and weight matched sedentary

able bodied controls). SCI subjects were recruited via a poster campaign from Toronto Rehab's Lyndhurst Centre. Non-SCI subjects were recruited from the staff and friends of the authors affiliated with the Lyndhurst Centre. Subjects did not participate in any regular exercise or endurance-type wheelchair exercise beyond their normal activities of daily living for 6 months prior to enrolment. All subjects were nonsmokers for at least 1 year prior to the study. No subjects reported a prior history of CAD, pulmonary disease, diabetes mellitus or metabolic syndrome. The subject's current medications were recorded. No subjects were taking medications known to interfere with the cardiovascular system.

A 12 lead EKG, was done to screen for arrhythmia, or prior myocardial infarction. The subjects' fasting serum blood sugar, glycosylated hemoglobin (HbA1C), Total Cholesterol (TC), high-density lipoproteins (HDL), low-density lipoproteins (LDL), Triglycerides (TG), C-Reactive Protein (CRP) and Apolipoprotein (A and B) levels, resting blood pressure (BP) and waist circumference were measured to screen for metabolic syndrome. Heart rate and BP were recorded from the right antecubital fossa using a stethoscope and hand-held dynamometer. Metabolic syndrome was defined as per the

American Heart Association Guidelines as ≥ 3 or more of the following criteria: abdominal obesity (waist circumference ≥102 cm for men); dyslipidemia (TC/HDL >4 or LDL >2.5); glucose intolerance (fasting blood sugar >7 mmol/L); elevated CRP (>3mg/dl); or hypertension (BP >140/90 mmHg).

Fifteen SCI subjects and 11 non-SCI subjects were screened for enrolment in the study. Five subjects' data were excluded from the analysis; three subjects due to having metabolic syndrome; one subject for an arrhythmia (atrial fibrillation) which interfered with PWV measurement; and one subject for an incomplete assessment. In total, 12 SCI subjects and 9 Non-SCI subjects were included in the study. The study protocol was approved by the Toronto Rehab Research Ethics Board.

*Pulse wave velocity (PWV):* PWV was measured from the foot, blood flow waves were recorded at two points along the path of the arterial pulse wave. PWV was calculated from the measured wave latency and the distance traveled between the two arterial recording sites (Figure1)[10, 15, 17, 33, 34]. Two identical transcutaneous Doppler flowmeters (Smartdop50, Hadeco, Inc., Kanagawa, Japan) were used to obtain the PWV values at three

locations: (1) between the carotid and the femoral arteries (Aortic PWV); (2) between the femoral and posterior tibial arteries (Leg PWV); and, (3) between the brachial and radial arteries (Arm PWV) (Figure1B). Distance traveled by the pulse was measured over the surface of the body with a tape measure as the distance (D) between recording sites (cm). A minimum of 20 sequentially recorded wave forms were analyzed and averaged. All PWV data were obtained by two trained technicians between 10AM and 1PM to avoid circadian changes in PWV values. Measurement of PWV was conducted after abstinence from caffeine and an overnight fast of at least 8 hours. Flow measurements were obtained sequentially in the arm, aorta and then the leg over a 40 minute time period. Arterial pulse waves were digitized for off-line analysis with signal-processing software (Chart 5.5.5, AD Instruments, Australia). PWV was determined over the three arterial segments as PWV= D/ $\Delta t$ (cm/sec), where $\Delta t$ was determined from time delay between the proximal and the distal foot of the wave form (Figure1A). The foot of the wave was identified as the commencement of the sharp systolic upstroke. All analyses were performed by a trained technician blinded to the subject's group assignment (SCI or non-SCI).

The test-retest variability of PWV measures in our laboratory was established by sequential measurement of 9 able-bodied males (21 ~ 39 yrs) on two separate days. The intraclass correlation coefficients for test-retest reliability were 0.730 ~ 0.972 for each PWV value. The mean PWV combined for the three sites was 1095 ±238 versus 1057 ±210 cm/sec for trial 1 versus trial 2 (not significant).

*Statistical Analysis:* The statistical analyses were performed using StatView (Version 5.0) software. Demographic, anthropometric and PWV data are expressed as mean±SDs. The SCI and non-SCI subjects were compared by the nonparametric Mann-Whitney U-test because of the small group size. A two sided *P* value of <0.05 was considered significant.

**Results**

There were no significant differences between groups in the baseline demographic or anthropometric parameters including: age, height, weight, heart rate and BP (Table1). The mean duration of injury of the SCI subjects was 20 ±13 years (Mean±SD). Mean Aortic PWV in the SCI group (1274±369cm/sec) were significantly higher (P<0.05) than that of the non-SCI group (948±110cm/sec) (Figure 2a). There were no statistically significant

differences between the SCI group and the non- SCI group (Figure 2b, c) in either Arm PWV (SCI: 1152±193cm/sec, non-SCI: 1237±193cm/sec) (P=0.434) nor Leg PWV (SCI: 1096±173cm/sec non-SCI: 994±178cm/sec) values (P=0.145).

**Discussion**

Aortic PWV values in the SCI group were higher than those of able-bodied controls (non-SCI group), while there were no significant differences between the SCI and non-SCI groups in Arm PWV and Leg PWV values. Recently reported Aortic PWV values in healthy able-bodied individuals 24 to 62 years of age ranged from 600 to 1000 cm/s[8]. Among hypertensive subjects, Aortic PWV values ranged from 1100 to 1500cm/s[8]. Arm and Leg PWV values in healthy able-bodied individuals ranged from 840 to 1200 cm/s and from 920 to 1050 cm/s, respectively[8]. The values for PWV documented herein are comparable with those previously reported for able-bodied individuals within the same age range.

Additionally, we found that Aortic PWV values among healthy SCI subjects were higher than the age-matched non-SCI subjects. Aortic PWV values in the SCI group were

equally high compared to the values (>1300 cm/sec) associated with an increased risk of developing CAD in Blacher's report[13]. These results suggest that the SCI group has a high risk of CAD. Screening protocols to diagnose and prevent CAD related mortality are urgently needed.

In contrast to the Aortic PWV result, there were no significant differences between the SCI group and non-SCI group in either Arm PWV or Leg PWV. These results concur with a prior study reporting that Aortic PWV is sensitive to daily activity and aging while Leg and Arm PWV values are not[16, 17]. This sensitivity of the PWV of central versus peripheral arteries may be related to their distinct roles in hemodynamic regulation. Compared with the central arteries whose cushioning function damps fluctuations in flow, the peripheral arteries do not exhibit the same extent of pulsatile changes in diameter[35] and, as such, may not undergo the adaptations leading to a loss of elasticity. However, previous studies, which investigated femoral arterial stiffness by augmentation index (AI)[36] and arterial compliance[37, 38] among people with SCI, demonstrated that stiffness of femoral artery in people with SCI was higher than that of able-bodied people. The reasons for this

discrepancy is not clear, however, the use of the PWV methodology as opposed to the AI and arterial compliance to assess arterial stiffness may in part explain the discrepancy between our finding of normal leg PWV values and prior publications reporting elevated femoral AI and decreased arterial compliance values among patients with SCI. The PWV method measures pulse wave latency over the femoral and posterior tibial arteries as opposed the femoral artery alone when assessing the AI or arterial compliance.

Although there are no prior studies determining PWV in people with SCI, three previous studies investigated arterial stiffness using other measures of arterial stiffness: the AI[36] and arterial compliance[37, 38] and compared them to those of able-bodied controls. de Groot et al. and Schmidt-Trucksass et al. (2000) reported that arterial compliance of the superficial femoral and carotid artery were significantly lower in subjects with SCI compared to non-SCI subjects[37, 38]. Wecht et al. (2004) reported that arterial stiffness evaluated by AI was high in a group of people with paraplegia compared with an able-bodied group[36]. Moreover, premature and advanced coronary atherosclerosis was found in persons with SCI compared with able-bodied people using electron beam

tomography[39]. Our observation of increased aortic arterial stiffness supports prior reports of premature CAD in the SCI population.

Mechanisms which may potentially account for higher Aortic PWV among SCI subjects include: 1) structural changes in the vessel as a result of long-term sympathectomy and increased collagen content in the vascular wall[40]; or, 2) functional changes in the endothelium due to decreased regional blood flow. Decreased regional blood flow as a result of inactivity impedes endothelium function and subsequently inhibits nitric oxide production, which is a mediator of endothelium dilatation[38]. Although the relative importance of structural and functional changes in vascular tone is unknown, these may relate to both disordered cardiac regulation and inactive lifestyles after SCI. The mechanism(s) which account for the current results are unknown.

The current study has limitations that require caution when interpreting and generalizing the findings reported herein. Firstly, the reliability of PWV values for people with SCI has not been reported. Secondly, this pilot study had a small sample size. Thirdly, adjustments for confounding variables including: the subject's injury level, duration of

injury and physical activity levels, which impact CAD risk, were not done. Future studies may want to use validated measures such as the PARA-SCI[31] to quantify activity and explore the relationship between PWV and activity. Lastly, it is uncertain if the high PWV values reported reflect the presence of CAD among the subjects' in this study. Further investigations with larger representative samples of SCI subjects are needed to determine the relationship between the increased arterial stiffness and the development/onset of atherosclerotic and asymptomatic CAD among people with SCI.

To our knowledge, this is the first study describing Aortic PWV in people with SCI. High Aortic PWV values were found in subjects with SCI compared to age, gender, height, and weight matched able-bodied subjects indicating a higher risk of CAD among SCI subjects. Arm PWV and Leg PWV were found to be insensitive to the differences between the two groups. PWV is potentially a good screening test to assess CAD risk among people with chronic SCI. However, this pilot study merely measured PWV among people with SCI. Further study is needed to confirm the reproducibility of PWV measures among people with SCI. Further study of the PWV method's reliability, validity and responsiveness while

considering the potential confounding effects of: age, duration of injury, impairment and physical activity on PWV among subject's with SCI are needed.

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**References**


**1.** Garshick E, Kelley A, Cohen SA, et al. A prospective assessment of mortality in chronic spinal cord injury. *Spinal Cord.* Jul 2005;43(7):408-416.

**2.** DeVivo MJ, Shewchuk RM, Stover SL, Black KJ, Go BK. A cross-sectional study of the relationship between age and current health status for persons with spinal cord injuries. *Paraplegia.* Dec 1992;30(12):820-827.

**3.** DeVivo MJ, Ivie CS, 3rd. Life expectancy of ventilator-dependent persons with spinal cord injuries. *Chest.* Jul 1995;108(1):226-232.

**4.** Whiteneck GG, Charlifue SW, Frankel HL, et al. Mortality, morbidity, and psychosocial outcomes of persons spinal cord injured more than 20 years ago. *Paraplegia.* Sep 1992;30(9):617-630.

**5.** Bots ML, Evans GW, Riley WA, Grobbee DE. Carotid intima-media thickness measurements in intervention studies: design options, progression rates, and sample size considerations: a point of view. *Stroke.* Dec 2003;34(12):2985-2994.

**6.** Glasser SP, Arnett DK, McVeigh GE, et al. Vascular compliance and cardiovascular disease: a risk factor or a marker? *Am J Hypertens.* Oct 1997;10(10 Pt 1):1175-1189.

**7.** Safar ME, Levy BI, Struijker-Boudier H. Current perspectives on arterial stiffness and pulse pressure in hypertension and cardiovascular diseases. *Circulation.* Jun 10 2003;107(22):2864-2869.

**8.** O'Rourke MF, Staessen JA, Vlachopoulos C, Duprez D, Plante GE. Clinical applications of arterial stiffness; definitions and reference values. *Am J Hypertens.* May 2002;15(5):426-444.

**9.** Benetos A, Adamopoulos C, Bureau JM, et al. Determinants of accelerated progression of arterial stiffness in normotensive subjects and in treated hypertensive subjects over a 6-year period. *Circulation.* Mar 12 2002;105(10):1202-1207.

**10.** Boutouyrie P, Tropeano AI, Asmar R, et al. Aortic stiffness is an independent predictor of primary coronary events in hypertensive patients: a longitudinal study. *Hypertension.* Jan 2002;39(1):10-15.

**11.** Taquet A, Bonithon-Kopp C, Simon A, et al. Relations of cardiovascular risk factors to aortic pulse wave velocity in asymptomatic middle-aged women. *Eur J Epidemiol.* May 1993;9(3):298-306.

**12.** Oliver JJ, Webb DJ. Noninvasive assessment of arterial stiffness and risk of atherosclerotic events. *Arterioscler Thromb Vasc Biol.* Apr 1 2003;23(4):554-566.

**13.** Blacher J, Asmar R, Djane S, London GM, Safar ME. Aortic pulse wave velocity as a marker of cardiovascular risk in hypertensive patients. *Hypertension.* May 1999;33(5):1111-1117.

**14.** Laurent S, Boutouyrie P, Asmar R, et al. Aortic stiffness is an independent predictor of all-cause and cardiovascular mortality in hypertensive patients. *Hypertension.* May 2001;37(5):1236-1241.

**15.** Sutton-Tyrrell K, Najjar SS, Boudreau RM, et al. Elevated aortic pulse wave velocity, a marker of arterial stiffness, predicts cardiovascular events in well-functioning older adults. *Circulation.* Jun 28 2005;111(25):3384-3390.

**16.** Hayashi K, Sugawara J, Komine H, Maeda S, Yokoi T. Effects of aerobic exercise training on the stiffness of central and peripheral arteries in middle-aged sedentary men. *Jpn J Physiol.* Aug 2005;55(4):235-239.

**17.** Tanaka H, DeSouza CA, Seals DR. Absence of age-related increase in central arterial stiffness in physically active women. *Arterioscler Thromb Vasc Biol.* Jan 1998;18(1):127-132.

**18.** Sutton-Tyrrell K, Newman A, Simonsick EM, et al. Aortic stiffness is associated with visceral adiposity in older adults enrolled in the study of health, aging, and body composition. *Hypertension.* Sep 2001;38(3):429-433.

**19.** Czernichow S, Bertrais S, Oppert JM, et al. Body composition and fat repartition in relation to structure and function of large arteries in middle-aged adults (the SU.VI.MAX study). *Int J Obes (Lond).* Jul 2005;29(7):826-832.

**20.** Ferreira I, Snijder MB, Twisk JW, et al. Central fat mass versus peripheral fat and lean mass: opposite (adverse versus favorable) associations with arterial stiffness? The Amsterdam Growth and Health Longitudinal Study. *J Clin Endocrinol Metab.* Jun 2004;89(6):2632-2639.

**21.** Christensen T, Neubauer B. Arterial wall stiffness in insulin-dependent diabetes mellitus. An in vivo study. *Acta Radiol.* Mar-Apr 1987;28(2):207-208.

**22.** Oxlund H, Rasmussen LM, Andreassen TT, Heickendorff L. Increased aortic stiffness in patients with type 1 (insulin-dependent) diabetes mellitus. *Diabetologia.* Oct 1989;32(10):748-752.

**23.** Lehmann ED, Watts GF, Fatemi-Langroudi B, Gosling RG. Aortic compliance in young patients with heterozygous familial hypercholesterolaemia. *Clin Sci (Lond).* Dec 1992;83(6):717-721.

**24.** Simon AC, Levenson J, Bouthier J, Safar ME, Avolio AP. Evidence of early degenerative changes in large arteries in human essential hypertension. *Hypertension.* Sep-Oct 1985;7(5):675-680.

**25.** Vaitkevicius PV, Fleg JL, Engel JH, et al. Effects of age and aerobic capacity on arterial stiffness in healthy adults. *Circulation.* Oct 1993;88(4 Pt 1):1456-1462.

**26.** Boreham CA, Ferreira I, Twisk JW, Gallagher AM, Savage MJ, Murray LJ. Cardiorespiratory fitness, physical activity, and arterial stiffness: the Northern Ireland Young Hearts Project. *Hypertension.* Nov 2004;44(5):721-726.

**27.** Mackey RH, Sutton-Tyrrell K, Vaitkevicius PV, et al. Correlates of aortic stiffness in elderly individuals: a subgroup of the Cardiovascular Health Study. *Am J Hypertens.* Jan 2002;15(1 Pt 1):16-23.

**28.** Demirel S, Demirel G, Tukek T, Erk O, Yilmaz H. Risk factors for coronary heart disease in patients with spinal cord injury in Turkey. *Spinal Cord.* Mar 2001;39(3):134-138.

**29.** Lee MY, Myers J, Hayes A, et al. C-reactive protein, metabolic syndrome, and insulin resistance in individuals with spinal cord injury. *J Spinal Cord Med.* 2005;28(1):20-25.

**30.** Lee MY, Myers J, Abella J, Froelicher VF, Perkash I, Kiratli BJ. Homocysteine and hypertension in persons with spinal cord injury. *Spinal Cord.* Aug 2006;44(8):474-479.

**31.** Ginis KA, Latimer AE, Hicks AL, Craven BC. Development and evaluation of an activity measure for people with spinal cord injury. *Med Sci Sports Exerc.* Jul

2005;37(7):1099-1111.

**32.** Noreau L, Shephard RJ. Spinal cord injury, exercise and quality of life. *Sports Med.* Oct 1995;20(4):226-250.

**33.** Avolio AP, Chen SG, Wang RP, Zhang CL, Li MF, O'Rourke MF. Effects of aging on changing arterial compliance and left ventricular load in a northern Chinese urban community. *Circulation.* Jul 1983;68(1):50-58.

**34.** Avolio AP, Deng FQ, Li WQ, et al. Effects of aging on arterial distensibility in populations with high and low prevalence of hypertension: comparison between urban and rural communities in China. *Circulation.* Feb 1985;71(2):202-210.

**35.** Boutouyrie P, Laurent S, Benetos A, Girerd XJ, Hoeks AP, Safar ME. Opposing effects of ageing on distal and proximal large arteries in hypertensives. *J Hypertens Suppl.* Aug 1992;10(6):S87-91.

**36.** Wecht JM, Weir JP, DeMeersman RE, Spungen AM, Bauman WA. Arterial stiffness in persons with paraplegia. *J Spinal Cord Med.* 2004;27(3):255-259.

**37.** de Groot P, Crozier J, Rakobowchuk M, Hopman M, MacDonald M. Electrical stimulation alters FMD and arterial compliance in extremely inactive legs. *Med Sci Sports Exerc.* Aug 2005;37(8):1356-1364.

**38.** Schmidt-Trucksass A, Schmid A, Brunner C, et al. Arterial properties of the carotid and femoral artery in endurance-trained and paraplegic subjects. *J Appl Physiol.* Nov 2000;89(5):1956-1963.

**39.** Zeilig G, Dolev M, Weingarden H, Blumen N, Shemesh Y, Ohry A. Long-term morbidity and mortality after spinal cord injury: 50 years of follow-up. *Spinal Cord.* Sep 2000;38(9):563-566.

**40.** Fronek K, Bloor CM, Amiel D, Chvapil M. Effect of long-term sympathectomy on the arterial wall in rabbits and rats. *Exp Mol Pathol.* Jun 1978;28(3):279-289.

**Figure legends**

**Figure1. A**: Determination of time delay of waveform **B**: Scheme of Pulse wave velocity (PWV) measurement. Δt: Time delay of waveform; CA: Carotid artery; FA: Femoral artery; BA: Brachial artery; RA: Rradial artery; PTA: Posterior tibial artery; D Aortic: Distance of between CA and FA; D Arm: Distance of between BA and RA; D Leg: Distance of between FA and PTA

**Figure 2.** Pulse wave velocity (PWV) in subjects with spinal cord injury (SCI) and able bodied controls (non-SCI).

**Table1**

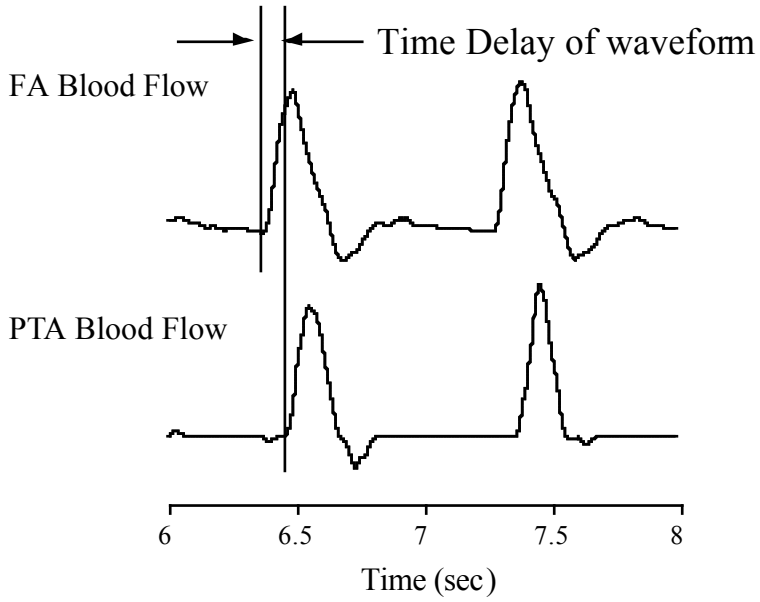
Table 1 Subject characteristics

| | SCI group | non-SCI group |
|---|---|---|
| N | 12 | 9 |
| Age (yr) | 45.9 ± 7.8 | 44.1 ± 10.9 |
| Height (cm) | 177.6 ± 7.0 | 174.5 ± 8.2 |
| Weight (kg) | 81.1 ± 20.6 | 73.7 ± 11.5 |
| Body mass index ($m^2$/kg) | 25.5 ± 5.7 | 24.1 ± 1.9 |
| Systolic blood pressure (mm Hg) | 121.0 ± 9.5 | 116.2 ± 11.4 |
| Diastolic blood pressure (mm Hg) | 74.8 ± 9.3 | 71.2 ± 6.5 |
| Heart rate (beats/min) | 64.3 ± 10.5 | 65.4 ± 8.0 |

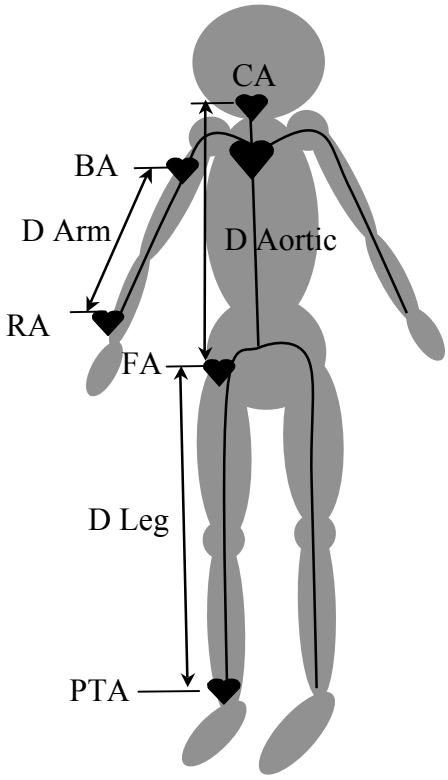
Data are means±SD

Figure1

**A**




**B**



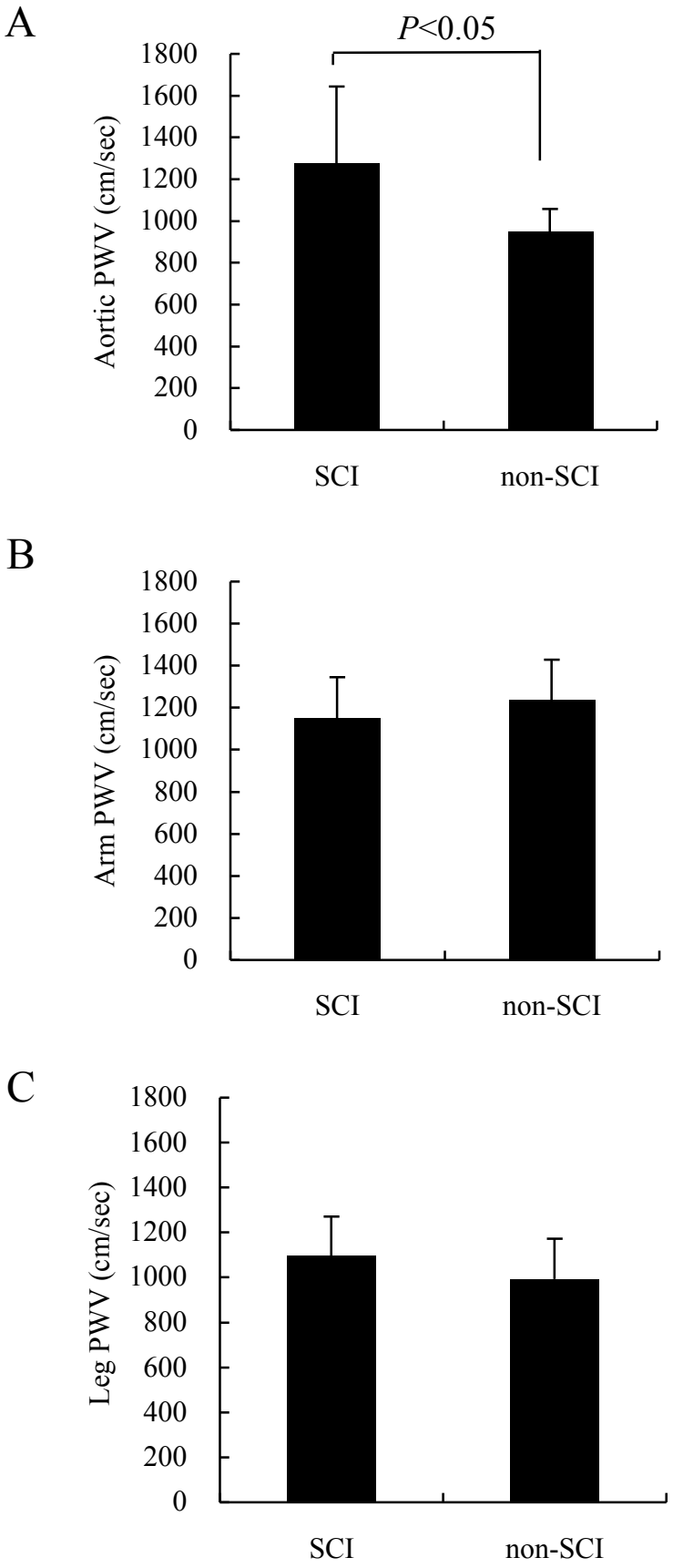

• Calculation of PVW

(1) Aortic PWV (cm/sec)=
D Aortic / TD between CA and FA

(2) Arm PWV (cm/sec) =
D Arm / TD between BA and RA

(3) Leg PWV (cm/sec) =
D Leg / TD between FA and PTA

**Figure2**


A
P<0.05
Aortic PWV (cm/sec)
1800
1600
1400
1200
1000
800
600
400
200
0
SCI
non-SCI
B
Arm PWV (cm/sec)
1800
1600
1400
1200
1000
800
600
400
200
0
SCI
non-SCI
C
Leg PWV (cm/sec)
1800
1600
1400
1200
1000
800
600
400
200
0
SCI
non-SCI