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ORIGINAL ARTICLE

Acute passive vibration reduces arterial stiffness and aortic wave reflection in stroke survivors

Andrew P. Koutnik · Alexei Wong · Roy Kalfon · Takudzwa A. Madzima · Arturo Figueroa

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Abstract
Purpose Impaired leg arterial stiffness (pulse wave velocity, PWV) and vasodilatory function are found after stroke. Acute passive vibration (PV) decreases leg PWV (legPWV) and pressure wave reflection (aortic augmentation index, aAIx) in healthy men. Our objective was to evaluate the effects of acute PV on aAIx and PWV in the paretic and non-paretic sides in stroke survivors.
Methods Eleven stroke survivors (4 females) were randomized to either no-PV (control) or PV (25 Hz and 2 mm amplitude) trials on two separated visits. Following 20 min of supine rest with legs on a vibration platform, blood pressure, PWV, and aAIx were gathered before 10 continuous minutes of control or PV. Measurements were repeated at post-5, post-15, and post-30 min after control or PV.
Results LegPWV and brachial-ankle PWV (baPWV, systemic stiffness) in paretic and non-paretic sides along with aAIx were significantly ($P < 0.05$) decreased from baseline at post-5 min after PV compared with control. At post-15 min, paretic and non-paretic legPWV remained significantly lower than baseline, but only non-paretic legPWV was different from control. We noted correlations between changes in paretic legPWV and changes in paretic baPWV ($r = 0.47$, $P = 0.028$) and aAIx ($r = 0.51$, $P = 0.017$) at post-5 min.
Conclusions Acute PV applied to the legs of stroke survivors reduces systemic arterial stiffness and aortic wave reflection due to a reduction in leg arterial stiffness, which last longer in the non-paretic than in the paretic leg.

Keywords Pulse wave velocity · Aortic augmentation index · Stroke · Hemiparesis

Abbreviations

PWV	Pulse wave velocity
baPWV	Brachial-ankle pulse wave velocity
aorticPWV	Aortic pulse wave velocity
legPWV	Leg pulse wave velocity
aAIx	Aortic augmentation index
AIx	Augmentation index
WBVE	Whole-body vibration exercise
PV	Passive vibration
BP	Blood pressure
DBP	Diastolic blood pressure
Control	No passive vibration
HR	Heart rate
ECG	Electrocardiogram
SBP	Systolic blood pressure
PP	Pulse pressure
P1	Aortic pressure wave composed of a forward wave
P2	Aortic pressure wave composed of a reflected wave
aAIx@75	AIx normalized to a heart rate of 75 bpm
Tr	Time of reflection
BMI	Body mass index

Introduction

Stroke is currently the fourth leading cause of death and primary cause of serious long-term disability in the United

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A. P. Koutnik · A. Wong · R. Kalfon · T. A. Madzima · A. Figueroa (✉)
Department of Nutrition, Food and Exercise Sciences, The Florida State University, 120 Convocation Way, Tallahassee, FL 32306, USA
e-mail: afiguero@fsu.edu

States (Towfighi and Saver 2011). Independent predictors of stroke are increased central (aortic) arterial stiffness (pulse wave velocity, PWV) (Mattace-Raso et al. 2006) and brachial-ankle PWV (baPWV) (Han et al. 2012), which is mainly composed of aortic PWV (aorticPWV) and leg PWV (legPWV) (Yamashina et al. 2002; Sugawara et al. 2005). BaPWV is a marker of cerebral arterial damage (Kim et al. 2008, 2011). A marked increase in legPWV in the paretic limb that occurs after a cerebrovascular accident (Okabe et al. 2004) may contribute to the increase in baPWV (Sugawara et al. 2005). Arterial stiffening increases wave reflection from peripheral to central arteries leading to an increase in aortic augmentation index (aAIx), a marker of left ventricular afterload (London 1997). An increased aAIx may account for a high risk of stroke (Ding et al. 2012) and predict a poor outcome in stroke survivors (Soiza et al. 2010).

Although, there is evidence that legPWV and aAIx can be reduced with acute aerobic (Kingwell et al. 1997) and resistance (Figueroa and Vicil 2011) exercise, stroke survivors are severely deconditioned and experience post-stroke fatigue with conventional exercises at moderate intensities (Gordon et al. 2004). Recently, whole-body vibration exercise (WBVE), which evokes reflexive muscle contractions through the "tonic-vibration reflex," has shown to reduce baPWV, legPWV, and aAIx after a single session in healthy young men (Otsuki et al. 2008; Figueroa et al. 2011). Alternatively, passive vibration (PV) exposes the legs to continuous vibration without performing voluntary muscle contractions (Herrero et al. 2011; Sanchez-Gonzalez et al. 2012; Wong et al. 2012). To date, only two studies have examined arterial responses to a session of PV and found decreases in baPWV, legPWV, and aAIx after 10 min of exposure to continuous PV in young healthy men (Sanchez-Gonzalez et al. 2012; Wong et al. 2012). Recently, Herrero et al. (2011) reported increases in neuromuscular activation and leg blood flow in the paralyzed limbs of individuals with spinal cord injury after a session of PV, suggesting that PV may reduce legPWV and aAIx in post-stroke survivors.

Therefore, the aims of this study were to examine the acute effects of PV on PWV and aAIx in stroke patients and to compare the baPWV and legPWV responses in the paretic and non-paretic sides. We hypothesize that PV will decrease aAIx and the reduction in baPWV and legPWV would be greater in magnitude and duration in the non-paretic as compared to the paretic sides.

Methods

Participants

Eleven (4 women) stroke survivors (age 42–74 years) with at least 4 months after their cerebrovascular event (ischemic $n = 6$) participated in this study. Participants were recruited from Tallahassee, FL and the surrounding areas through self-enrolled stroke and aphasia community support groups. All participants did not smoke or exercise more than 60 min per week. Only those participants with prehypertension or stage-1 hypertension [systolic blood pressure (SBP)/diastolic blood pressure (DBP): 121–159/81–99 mmHg] were included. Participant's medications are listed in Table 1. They were on stable dosages for at least 4 months prior to the study. This study was approved by the institutional review board of the Florida State University. All participants were aware of the experimental procedures and gave written consent to participate before data collection.

Table 1 Medications

Medication classifications	$n = 11$
Salicylates	7
Calcium channel blocker	6
HMG-CoA reductase inhibitors	6
Proton pump inhibitors	6
ACE inhibitors	5
Angiotensin II receptor blocker	5
NSAID	1
Beta blockers	1

HMG-CoA 3-hydroxy-3-methylglutaryl coenzyme A, *ACE* angiotensin converting enzyme, *NSAID* nonsteroidal anti-inflammatory drug

Study design

Participants were randomized to either the PV or no-PV (control) trials performed on 2 days separated by a minimum of 48 h in a crossover design. The experiments were conducted in a quiet, temperature controlled room (23 ± 1 °C) and at the same time of the day (8–10 a.m.) in order to minimize potential diurnal variations in vascular reactivity. Participants abstained from food for at least 8 h, any supplements or medications that may affect cardiovascular measures for 24 h, caffeine and alcohol for 12 h, and moderate to intense exercise or physical activity for 48 h before testing. The only premenopausal woman was examined during the early (days 3 and 5, considering day 0 as the first day of bleeding) follicular phase of the menstrual cycle to avoid effects of endogenous estrogens on arterial function (Adkisson et al. 2010).

Experimental protocol

Participants were assisted to adopt the supine position on a padded mattress with both legs over the vibration platform.

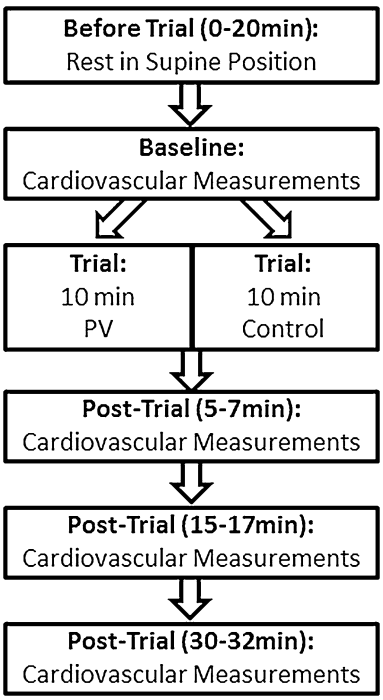

Fig. 1 Experimental protocol. *PV* passive vibration; *Control* no-passive vibration; *Post-Trial* time after passive vibration or no-passive vibration trial was completed; *Cardiovascular Measurement* measurements of brachial-ankle pulse wave velocity, carotid-femoral pulse wave velocity, femoral-ankle pulse wave velocity, heart rate, aortic augmentation index, aortic augmentation index normalized to a HR of 75 bpm, time of reflection, systolic blood pressure, diastolic blood pressure, and mean arterial pressure

The area directly exposed to vibration was from the lower gluteal region to above the malleoli, with both feet placed out of the platform (pro5 AIRdaptive; Power Plate, Northbrook, IL, USA). Participants remained in this position for 20 min of rest, followed by baseline measurements of PWV, brachial blood pressure (BP), heart rate (HR), and pulse wave analysis. Subsequently, participants completed a continuous 10-min PV or control session. The intensity of the vibration was set at a 25 Hz frequency and 2 mm amplitude (~5.37 G). For the post-PV or post-control period, participants stayed in the same position while PWV, brachial BP, HR, and pulse wave analysis measurements were gathered at 5–7 min (post-5), 15–17 min (post-15), and 30–32 min (post-30) marks. This protocol (Fig. 1) was previously used in healthy young men (Sanchez-Gonzalez et al. 2012; Wong et al. 2012).

Pulse wave velocity, brachial blood pressure, and heart rate

HR, brachial BP, and PWV were measured using an automatic device (VP-1000, Omron Healthcare Inc., Vernon Hills, IL, USA). BP cuffs were wrapped around both arms (brachial artery) and ankles (posterior tibial artery), and two tonometers were placed over the carotid and femoral arteries to obtain PWV measurements from three arterial segments: baPWV, carotid-femoral PWV (aorticPWV) and femoral-ankle PWV (legPWV), which are considered measurements of systemic, central and peripheral arterial stiffness, respectively (Laurent et al. 2006). Pressure waveforms from carotid and femoral arteries (via tonometers) and from brachial and posterior tibial arteries (via cuffs) were collected for 10 s. The transient time was calculated automatically by relating the feet of the waveforms to the R-wave of the electrocardiogram (ECG). The distance between carotid and femoral artery was measured with a non-elastic tape measure as a straight line, while the distance between sampling points of baPWV and legPWV were calculated automatically according to the height of the participants (Yamashina et al. 2002). PWV was calculated as the distance between two sampling sites divided by the transit time. HR was obtained from the ECG. The intraclass correlation coefficient for legPWV and baPWV calculated on two separate days in our laboratory is 0.95.

Pulse wave analysis

SBP and DBP were measured in duplicate using an automated monitor (HEM-705CP; Omron Healthcare, Vernon Hill, IL, USA) and the average was used in subsequent analysis. Pulse wave analysis indexes were assessed using radial applanation tonometry with a high-fidelity transducer (Millar Instruments, Houston, TX, USA). The average of two measurements with an operator index of at least 85 % was accepted (O'Rourke et al. 2001; Wilkinson et al. 2002). The aortic pressure waveform is a composite of the forward traveling wave (P1) produced by the stroke volume and a reflective wave (P2) which travels from the peripheral arteries back towards the aorta (Nichols and Singh 2002). The time that it takes for the pressure wave to travel from the aortic value to the reflection sites and back to the aorta is measured as the time of reflection (Tr), an estimate of aortic stiffness (McEniery et al. 2005). Aortic AIx, a measure of wave reflection (McEniery et al. 2005), was calculated as the ratio of the difference between P2 and P1 to the aortic pulse pressure (SBP–DBP) and expressed as a percentage. Due to the influence of HR on aAIx, we also used aAIx adjusted to 75 bpm (aAIx@75) (Wilkinson et al. 2000). The intraclass correlations for pulse wave analysis measurements taken over two different days in our laboratory are >0.90.

Anthropometrics and body composition

Height and weight were measured using a stadiometer and scale with measures rounded to the nearest 0.5 cm and 0.1 kg, respectively. Body mass index (BMI) was

calculated as weight in kg divided by height in square meter. Participants who could not voluntarily stand, height was determined with a tape measure while lying. Arm and leg lean mass were determined with a whole-body dual-energy X-ray absorptiometry scan (DPX-IQ; GE Lunar, Madison, WI, USA).

Statistical analysis

Normal distribution was evaluated using the Shapiro–Wilk test. Student's *t* test was used to determine possible differences in all dependent variables at rest or at baseline between trials. Differences in mean values for each variable were compared by a 2 × 4 analysis of variance with repeated measures [trial (control vs PV) by time (baseline, post-5, post-15, and post-30)], followed by Fisher's LSD test for pairwise comparisons. Post hoc comparisons were made with paired *t* tests to detect within-trial differences across time. Pearson's correlations were used to analyze the relationship between changes in selected variables. An a priori-α level of <0.05 was considered to be significant. SPSS version 20.0 (SPSS Inc, Chicago, IL, USA) was used for all analyses. Results are reported as mean ± SE.

Results

Participant characteristics and cardiovascular parameters

Age, height, weight, BMI, and lean mass are reported in Table 2. Arm and leg lean mass were not different in the paretic and non-paretic sides. The peripheral and central hemodynamics parameters at baseline and after control and PV are shown in Table 3 and Fig. 2. There were no significant differences in all cardiovascular parameters between the trials at baseline.

There were significant trial-by-time interactions for paretic legPWV ($P = 0.004$), non-paretic legPWV ($P = 0.015$), paretic baPWV ($P = 0.007$), and non-paretic baPWV ($P = 0.007$). At post-5 min, the decrease in paretic legPWV (−94.0 ± 26.0 cm/s, $P = 0.005$), non-paretic legPWV (−81.9 ± 18.1 cm/s, $P = 0.001$), paretic baPWV (−76.6 ± 24.0 cm/s, $P = 0.01$), and non-paretic baPWV (−115.7 ± 22.0 cm/s, $P = 0.0001$) were significantly different from baseline after PV compared with control. At post-15 min, paretic legPWV (−59.2 ± 21.6 cm/s, $P = 0.021$) and non-paretic legPWV (−70.3 ± 14.9 cm/s, $P = 0.001$) remained significantly different from baseline, but only non-paretic legPWV ($P = 0.02$) was significantly different from control.

There were significant trial-by-time interactions for aAIx ($P = 0.001$) and aAIx@75 ($P = 0.005$) indicating that the decreases in aAIx (−3.4 ± 0.65 %, $P = 0.001$) and aAIx@75 (−3.6 ± 1.1, $P = 0.009$) were significantly different from baseline after PV compared with control

Table 2 Subject characteristics ($n = 11$)

Age (years)	62 ± 3
Height (m)	1.73 ± 0.03
Weight (kg)	82.9 ± 4.6
BMI (kg/m^2)	27.7 ± 1.4
Paretic arm lean mass (kg)	2.69 ± 0.28
Non-paretic arm lean mass (kg)	3.05 ± 0.27
Paretic leg lean mass (kg)	8.28 ± 0.63
Non-paretic leg lean mass (kg)	8.61 ± 0.56

Values are mean ± SE

BMI body mass index

Table 3 Aortic pulse wave velocity, aortic augmentation index, time of reflection, heart rate, and blood pressures before and after control and passive vibration trials

Variable	Baseline	Post-5	Post-15	Post-30
AorticPWV (cm/s)				
Control	1321 ± 113	1378 ± 121	1336 ± 67	1373 ± 90
PV	1359 ± 112	1315 ± 118	1354 ± 110	1306 ± 119
Aortic AIx (%)				
Control	27.4 ± 4	28.2 ± 3.8	28.6 ± 3.9	28.7 ± 3.9
PV	28.9 ± 3.4	26.2 ± 3.4*,†	28.7 ± 3.4	30.3 ± 3.5
Aortic AIx@75 (%)				
Control	21.5 ± 3.6	21.8 ± 3.6	21.7 ± 3.6	22.1 ± 3.6
PV	23.6 ± 3.3	20.4 ± 3.6*,‡	22.1 ± 3.6	23.5 ± 3.4
Tr (ms)				
Control	152 ± 6	142 ± 3	142 ± 2	141 ± 2
PV	144 ± 3	145 ± 5	143 ± 3	142 ± 4
HR (bpm)				
Control	63 ± 4	62 ± 3	62 ± 3	62 ± 4
PV	64 ± 4	63 ± 3	61 ± 3	61 ± 3
Brachial SBP (mmHg)				
Control	134 ± 7	137 ± 7	132 ± 6	136 ± 6
PV	137 ± 7	141 ± 7	140 ± 7	140 ± 6
Brachial DBP (mmHg)				
Control	82 ± 5	84 ± 4	82 ± 5	82 ± 5
PV	83 ± 3	85 ± 4	88 ± 4	84 ± 3
Brachial MAP (mmHg)				
Control	105 ± 6	105 ± 5	103 ± 6	106 ± 6
PV	107 ± 6	108 ± 6	110 ± 5	110 ± 5

Values are mean ± SE

PV passive vibration, *aorticPWV* aortic pulse wave velocity, *aAIx* aortic augmentation index, *aAIx@75* aortic augmentation index adjusted for a heart rate of 75 beats/min, *Tr* transit time of the reflected pressure wave, *HR* heart rate, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *MAP* mean arterial pressure

* $P < 0.05$ vs. baseline. † $P < 0.05$, ‡ $P < 0.01$ vs. control

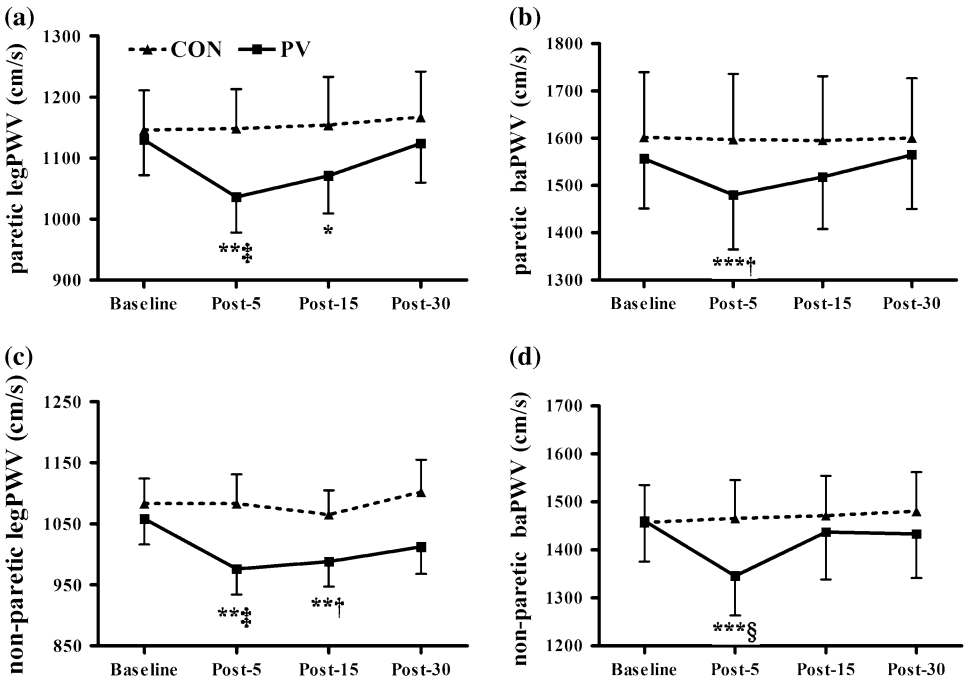

Fig. 2 Brachial-ankle and leg pulse wave velocities in paretic and non-paretic sides. *CON* control, *PV* passive vibration, *paretic legPWV* paretic leg pulse wave velocity, *non-paretic legPWV* non-paretic leg pulse wave velocity, *paretic baPWV* paretic brachial-ankle pulse wave velocity, *non-paretic baPWV* non-paretic brachial-ankle pulse wave velocity. Values are mean ± SE. $^{*}P < 0.05$, $^{**}P < 0.01$, $^{***}P < 0.001$ vs. baseline. $^{\dagger}P < 0.05$, $^{\ddagger}P < 0.01$, $^{\S}P < 0.001$ vs. control

at post-5 min. AorticPWV, Tr, HR and BP were not significantly affected by PV and control. There were significant correlations between changes in paretic legPWV and paretic baPWV ($r = 0.47$, $P = 0.028$) and between changes in paretic legPWV and aAIx ($r = 0.51$, $P = 0.017$) at post-5 min.

Discussion

The major findings of this study are that a 10 min session of PV decreased bilateral legPWV and baPWV concurrently with a reduction in aAIx at post-5 min, but only the non-paretic legPWV remained reduced at post-15 min as compared to the control trial. Acute PV did not affect HR, brachial BP, and aorticPWV in stroke survivors.

LegPWV decreased similarly in the non-paretic (6.6 %) and paretic leg (5.3 %) at post-5 min as compared to the control trial, suggesting that peripheral arterial responses are not influenced by neuromuscular impairment during early recovery from PV in stroke survivors. This reduction in paretic legPWV was short-lasting and did not remain significantly reduced at post-15 min as compared to control. Although the decrease in non-paretic legPWV persisted lower than baseline and control until post-15 min, this response was transitory as compared to previous findings in healthy men (Wong et al. 2012). Evidence suggests that a 10 min session of either continuous PV or intermittent (10 sets of 1 min) WBVE decreases legPWV within the 30 min post-vibration in young men (Figueroa et al. 2011; Wong et al. 2012). These reductions in legPWV post-vibration are consistent with the impact of acute low-intensity leg resistance and endurance exercise in young healthy individuals (Sugawara et al. 2005; Heffernan et al. 2006). Of note, reductions in legPWV after WBVE and resistance exercise occur concomitantly with increases in HR and SBP (Figueroa et al. 2011; Heffernan et al. 2006). Consistent with the present findings, HR and brachial BP were not affected following PV (Wong et al. 2012), suggesting an attenuated cardiovascular stress. Consequently, PV appears to be effective to acutely decrease legPWV in stroke survivors, but more research is needed to elucidate the chronic effects of PV on legPWV.

BaPWV is mainly composed of aorticPWV (~58 %) and legPWV (~23 %) (Sugawara et al. 2005); hence, changes in either component are likely to influence baPWV. We found that both paretic baPWV (~77 cm/s) and non-paretic baPWV (~116 cm/s) were decreased 5 min after PV. While the effect of PV on legPWV persisted up to 15 min, baPWV returned to baseline. We have previously demonstrated that baPWV is less sensitive than legPWV to the acute effects of PV and WBVE in young healthy men (Figueroa et al. 2011; Wong et al. 2012). The reductions in baPWV observed at post-5 min in stroke survivors are similar to those reported following acute PV (Figueroa et al. 2011) and WBVE (Otsuki et al. 2008) in healthy men. This response may be clinically important because increased baPWV is associated with cerebral atherosclerosis (Kim et al. 2011) and arterial stiffness (Kim et al. 2008), as well as ischemic stroke risk (Han et al. 2012). The longer duration and higher reduction in legPWV than baPWV could be attributed to the local impact of vibration on leg arteries

with no vibration propagation to the aorta (Figueroa et al. 2011; Wong et al. 2012). Since aorticPWV did not change, the acute decrease in baPWV is likely attributed to the change in legPWV, which is consistent with previous findings in young healthy men using the same protocol (Wong et al. 2012).

Aortic AIx was decreased by 3.4 % at post-5 min. The transient increase in leg blood flow observed 1 min post-PV in individuals with paralysis due to spinal cord injury (Herrero et al. 2011) may support our current finding of reduced aAIx in a population also characterized by paralysis and increased PWV and aAIx (Okabe et al. 2004; Soiza et al. 2010). Our data showed that the reduction in legPWV was correlated with the decrease in aAIx, suggesting that vibration-induced leg artery vasodilation (Herrero et al. 2011) attenuated wave reflection to the aorta via reduced leg arterial stiffness (Wong et al. 2012). It is important to note that a 10 min session of either PV or WBVE does not reduce aAIx at post-5 min in healthy men (Figueroa et al. 2011; Sanchez-Gonzalez et al. 2012), indicating that peripheral vasodilation does not occur during the early recovery from vibration in individuals with normal leg blood flow. Consistent with our data, type-2 diabetics experience a paradoxical decrease in aAIx immediately after exercise, which may suggest a compensatory mechanism to sustain a favorable ventricular-arterial coupling (Sacre et al. 2012).

Aortic AIx recovered at post-15 in post-stroke survivors. In contrast, previous work reported a decrease in aAIx (−7.2 %) at 30 min post-PV in healthy men (Sanchez-Gonzalez et al. 2012). In the current and previous study (Sanchez-Gonzalez et al. 2012), the reductions in aAIx occurred with no increase in HR, suggesting that the negative influence of increased HR on aAIx reduction (Wilkinson et al. 2000) did not contribute to the response. The absence of a reduction in aAIx at post-15 min would be partially explained by chronically increased legPWV (Okabe et al. 2004) and impaired leg vasodilatory capacity in stroke survivors (Ivey et al. 2004, 2010). In individuals with high cardiovascular risk, increased legPWV limits local blood flow (Suzuki et al. 2001). Ivey et al. (2004, 2010) reported a 28–35 % reduction in endothelium-dependent blood flow reactivity in the paretic as compared to the non-paretic leg in post-stroke survivors. It has been suggested that stroke-related loss of leg muscle mass may contribute to the increased legPWV (Okabe et al. 2004). In our study, although legPWV and lean mass tended to be smaller in the paretic than in the non-paretic leg, the differences were not statistically significant.

The exact mechanisms by which PV decreases legPWV and aAIx are unclear. However, we know that reflexive muscle contractions have been seen in the limbs exposed to low-frequency vibration with (Roelants et al. 2006) and without (Herrero et al. 2011) exercise, which evokes peripheral arterial vasodilation through the release of contraction-related vasodilatory substances (Kingwell et al. 1997; Figueroa et al. 2011; Heffernan et al. 2006) and nitric oxide (Maloney-Hinds et al. 2008). Nitric oxide donor infusion (Schmitt et al. 2005) and exercise (Figueroa and Vicil 2011) can decrease local PWV and aAIx. Taken together, it can be suggested that PV evokes vasodilation on arteries exposed to vibration resulting in reduction of legPWV, which consequently induces reductions in baPWV and aAIx.

The principal limitations of the present study are the relative small sample size and the wide range of time after the cerebrovascular event. In addition, because we did not take direct measures of arterial vasodilation and vasoactive substances, we are unable to determine if nitric oxide is the main substance behind vasodilation-induced changes in PWV and aAIx. The present study examined arterial responses in stroke survivors, and thereby, the results cannot be generalized to other populations at increased cardiovascular risk with or without paralysis.

This study provides the first evidence of the beneficial effects of PV on arterial stiffness and aortic wave reflection in stroke survivors. We showed that the magnitude of the changes in systemic and peripheral PWV following PV is similar in both paretic and non-paretic sides, but the reduction in legPWV is maintained for a longer time in the non-paretic leg than in the paretic leg. However, the effects of PV were transient and prospective research is needed to evaluate the long-term effects of PV on arterial function in stroke survivors.

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Conflict of interest The authors declare no conflict of interest.

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