



Original Article

Does short-term whole-body vibration training affect arterial stiffness in chronic stroke? A preliminary study

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Abstract. [Purpose] Previous studies have shown that stroke is associated with increased arterial stiffness that can be diminished by a program of physical activity. A novel exercise intervention, whole-body vibration (WBV), is reported to significantly improve arterial stiffness in healthy men and older sedentary adults. However, little is known about its efficacy in reducing arterial stiffness in chronic stroke. [Subjects and Methods] Six participants with chronic stroke were randomly assigned to 4 weeks of WBV training or control followed by cross-over after a 2-week washout period. WBV intervention consisted of 3 sessions of 5 min intermittent WBV per week for 4 weeks. Arterial stiffness (carotid arterial stiffness, pulse wave velocity [PWV], pulse and wave analysis [PWA]) were measured before/after each intervention. [Results] No significant improvements were reported with respect to carotid arterial stiffness, PWV, and PWA between WBV and control. However, carotid arterial stiffness showed a decrease over time following WBV compared to control, but this was not significant. [Conclusion] Three days/week for 4 weeks of WBV seems too short to elicit appropriate changes in arterial stiffness in chronic stroke. However, no adverse effects were reported, indicating that WBV is a safe and acceptable exercise modality for people with chronic stroke.

Key words: Arterial stiffness, Pulse wave velocity, Augmentation index

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INTRODUCTION

Post-stroke survivors face the problem of being predisposed to a sedentary lifestyle that restricts their ability to undertake physical activity resulting in further deconditioning, which can significantly increase stroke reoccurrence and cardiovascular disease¹⁾. To overcome the potential for cardiovascular disease, physical activity and exercise programs are often implemented. However, for stroke survivors there are numerous challenges to undertaking an exercise regime, such as time, convenience, compliance, ambulation, and postural control. Thus, traditional exercise may not be always feasible. Alternatively, whole-body vibration (WBV) is seen as an attractive and efficient treatment modality to complement other forms of exercise to improve muscle function, force, and power in healthy, compromised, and sporting individuals²⁾.

WBV is a rhythmic activity where body-weight static and/or dynamic exercises are performed on an oscillating plate that elicits rapid stretch-reflexes³⁾ involving the tonic vibration reflex⁴⁾, which causes changes in muscle fiber length⁵⁾. Consequently, small changes in oxygen uptake occur, suggesting that muscle energy exists⁶⁾, that may benefit cardiovascular indices. Acute WBV has been shown to significantly augment leg blood flow⁷⁾, although heart rate (HR) and brachial blood pressure (BP) have been reported to cause only small increases⁸⁾ or no changes^{7, 9)}.

Increased brachial-ankle pulse wave velocity (PWV) and arterial stiffness (PWV) are independent predictors of stroke. Other indices of pulse wave analysis of aortic blood pressure (BP) and augmentation index (AIx) are regarded to be more sensitive predictors of cardiovascular events than brachial BP¹⁰⁾. AIx is considered an indicator of left ventricular afterload

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and can increase due to arterial stiffening where wave reflection from peripheral to central arteries rise. It has been reported that following an acute intermittent WBV protocol, brachial-ankle PWV, leg PWV, and AIX were significantly reduced in healthy young men^{9, 11}. Similarly, 10 min of continuous passive vibration, which was defined by lying supine and placing both legs on a vibrating platform, decreased brachial-ankle PWV, leg PWV, and AIX in healthy participants¹⁰. In stroke survivors, leg PWV and brachial-ankle PWV were significantly reduced by acute passive vibration (10 min), but it had no effect on HR, brachial BP, and aortic PWV¹².

Aerobic exercise has the capability to improve arterial stiffness and cardiovascular risk factors¹³. Thirty min (2×/week, 6 months) of moderate-high intensity aerobic exercise reduces BP and arterial stiffness¹³. However, as previously stated, traditional exercise such as aerobic conditioning may not be appropriate for certain patients. Thus, WBV may provide an alternative to traditional exercise as it provides an easy and efficient modality, as shown by its improving of health parameters in less time per session compared to resistance training¹¹. Acute WBV has been shown to decrease arterial stiffness in healthy individuals^{9, 11}. Similarly, long-term (3 months) WBV training has been reported to reduce arterial stiffness in middle-aged/older adults¹⁴. WBV is similar to mechanical compression, which produces oscillations to elicit muscle contractions¹⁵ and causes arterial vasodilation¹⁶. The propagation of the vibration stimulus has the ability to transmit mechanical energy to the upper-body and head¹⁷ that can enhance upper-body muscular performance¹⁸. Thus, WBV may reduce arterial stiffness through the propagation of mechanical stimuli capable of stimulating lower and upper-body arteries. However, to our knowledge, no study has investigated the efficacy of short-term WBV training in post-stroke individuals. Therefore, the aim of this study was to examine the effects of short-term WBV training on carotid arterial stiffness in chronic stroke survivors. We hypothesized that 4 weeks of WBV training would significantly reduce arterial stiffness.

SUBJECTS AND METHODS

Subjects

Four sedentary men (mean and standard deviation [SD], age 50.5±14.5 years; body mass 106.6±25.9 kg; height 179.3±3.8 cm) and two sedentary women (age 39±2 years; body mass 86.5±15.5 kg; height 168.0±4.0 cm) volunteered to participate in the study. The inclusion criteria required that the participants be aged between 35–65 years and have suffered a clinically diagnosed stroke between 6 months to 5 years prior to the commencement of the study. The type of stroke (ischemic $n = 1$; hemorrhagic $n = 4$; transient ischemic attack $n = 1$, its locality, left hemisphere $n = 2$; right hemisphere $n = 4$) and extent of disability were classified by the modified Rankin Scale (range 1–4). The participants had to meet the requirement never having undertaken WBV and were classified as sedentary according to the ACSM guidelines¹⁹. Five participants were involved in weekly, organized activities such as bocce, aqua jogging, and physiotherapy, but these activities did not exceed the aforementioned parameters of a sedentary lifestyle. Exclusion criteria included uncontrolled hypertension or hypotension, bone tumors, herniated discs, and recent fractures (<6 months). The study was approved by the University Human Ethics Committee, and written informed consent was obtained from all participants.

Methods

Prior to testing, every participant completed a familiarization session that included WBV, sphygmoCor, and ultrasound. After baseline measurement, participants were randomly assigned to 4 weeks of WBV training or control followed by cross-over after a 2-week washout period. During the control and washout periods, the participants were instructed to continue their normal daily activities. The WBV intervention consisted of 3 sessions of 5 min intermittent WBV per week for 4 weeks, with at least 24 hours of rest between each WBV session. Pre- and post- (4 weeks) WBV and control measurements of arterial stiffness and cardiovascular indices were performed for all participants. Prior to testing, participants refrained from consuming caffeine during the preceding 12 hours, and refrained from taking drugs with known vascular effects. Participants rested in the supine position for 20 min prior to testing and to account for daily biorhythms, all testing was conducted at the same time each day. During the course of the study, participants were informed not to engage in any additional physical activity above what they were currently performing (Table 1).

WBV was performed on a commercial machine (Galileo Sport, Novotec, Pforzheim, Germany), which had a motorized teeterboard that produced side-alternating vertical sinusoidal vibrations of up to 30 Hz, and maximum peak-to-peak displacement ($p-p$) of 12 mm. With this WBV machine, $p-p$ was dependent on the participant's foot position. The further the feet were from the central oscillating axis the larger the $p-p$, and vice versa. Thus, a single-axis accelerometer (iMEMS®, ADXL250, Analog Devices, Norwood, MA, USA) was fixed to the edge of the vibrating platform to measure the $p-p$ and determine the three different foot positions (FtP 1–3). To guarantee the location and identification of the different displacements, longitudinal strips of reflective adhesive tape were applied to the WBV plate. This allowed participants to place their second toe and heel midpoint in line with the tape. This helped keep the feet in the correct position during each session. The researcher constantly checked each participant's foot positioning and corrected any deviations. Over the 12 sessions, participants placed their feet in three different positions: FtP 1 = 2.1 mm $p-p$; FtP 2 = 4.3 mm $p-p$; and FtP 3 = 6.5 mm $p-p$.

Participants were asked to maintain a static squat stance with 70° knee flexion attained using a manual goniometer. Seventy-degree knee flexion was selected based on evidence that beyond 30° of static squatting maximizes leg extensor activation²⁰ and reduces the negative side effects of vibration being transferred through to the head¹⁷. With shoes on,

participants were instructed to place their feet in the protocol-defined positions, maintain an upright torso with their eyes and head facing forward, and evenly distribute their body weight through the mid-foot of both feet. They were allowed to use the support bar if required.

To date, there has been no scientific test of a short-term WBV training protocol for stroke. A four-week training protocol was implemented, which was based on earlier work that reported improved muscle force output, functional ability, and general well-being in people with multiple sclerosis²¹. In the current study, the vibration parameters of vibration frequency (Hz), $p-p$ (mm), peak acceleration (ms^{-2}), and session duration (min) were periodized into four blocks (Table 1). Each block consisted of three WBV sessions where the vibration frequency was systematically increased by 2 Hz (22 Hz, 24 Hz, 26 Hz) and $p-p$ was increased at the commencement of a new block of training.

The duration remained the same during the first three blocks; five sets of 1-min bouts of WBV with 1-min rests between each. The intermittent nature and volume of WBV exposure was selected on the basis of the Mason, Cochrane, Denny, Firth, Stannard²²) aforementioned study, which reported an improvement in mobility parameters in multiple sclerosis. This is a common approach to increase muscular power in healthy individuals²³). To maximize the associated benefits of overload, the fourth block of training (session 10–12) was set at 26 Hz, 6.5 mm $p-p$, and seven sets of 1-min exposures with 1-min rests (Table 1). The vibration frequency was selected from previous research that showed 25–26 Hz to enhance muscular performance²⁴). The vibration duration of 1-min exposure with 1-min rest was prescribed based on previous research demonstrating positive reductions in arterial stiffness^{9, 11}).

PWA was performed using a SphygmoCor device (AtCor Medical, Sydney, Australia), which is a non-invasive tool used to assess arterial stiffness, central blood pressures, and autonomic function. Brachial BP was recorded using an automated oscillometric device (Nissei DS-157; Mentone Educational Centre, Carnegie, Victoria, Australia). Radial artery waveforms were non-invasively recorded with a high-fidelity micromanometer (SPC-301; Millar Instruments, TX, USA) from the wrist of the non-affected stroke arm using applanation tonometry. These waveforms were calibrated against brachial systolic blood pressure (SBP) and diastolic blood pressure (DBP) from comparable hemodynamic properties of the upper limb arteries. A corresponding aortic pressure waveform was generated using a validated transfer function²⁵), from which central SBP (cSBP), central DBP (cDBP), central pulse pressure (cPP), and AIx was calculated using the integrated software (SCOR Px 7.1, AtCor Medical, Sydney, Australia). AIx was normalized to a heart rate (HR) of 75 beats/min (AIx@75). HR was measured from the time between pulse waveforms, and participants remained in the supine position for all PWA measurements. If the first two consecutive AIx@75 values differed by more than 4% or blood pressures of greater than 5 mmHg, a third measurement was taken and the mean of the closest two values was recorded. Data were collected directly via a personal computer (Toshiba, Intel® Core™ i5, Windows 7 operating system).

PWV was measured using the SphygmoCor device. The pulse pressure wave of the common carotid and radial arteries were recorded non-invasively using applanation tonometry as described above. The recordings were gated using the integrated electrocardiogram, with the velocity of the pulse wave being calculated using the integrated software (SCOR Px 7.1, AtCor Medical, Sydney, Australia). Participants remained in the supine position for BP measurements that were obtained prior to PWV measurements. Prior to electrode placement, any excess hair was removed with a razor and the area cleaned with alcohol wipes. Surface pre-gelled Ag-AgCl electrodes (Ambu, Ballerup, Denmark) were placed on the participant's chest: 1 cm below the suprasternal notch on the sternum, 1 cm above the xiphoid process on the sternum, and 3 cm above the left iliac crest. Distal and proximal measurements were then measured and recorded for each participant. The measurements of PWV and carotid-to-radial pulse transit time standard deviation (PTT SD) were used for subsequent analysis.

Table 1. WBV training protocol

Session	Vibration frequency (Hz)	Peak-to-peak displacement (mm)	Peak acceleration (ms^{-2})	Vibration duration (min)
1	22	2.1	29.8	5 × 1
2	24	2.1	30.2	5 × 1
3	26	2.1	34.1	5 × 1
4	22	4.3	48.8	5 × 1
5	24	4.3	52.6	5 × 1
6	26	4.3	60.7	5 × 1
7	22	6.5	67.3	5 × 1
8	24	6.5	74.5	5 × 1
9	26	6.5	86.8	5 × 1
10	26	6.5	86.8	7 × 1
11	26	6.5	86.8	7 × 1
12	26	6.5	86.8	7 × 1

The common carotid artery was imaged non-invasively using commercial B-mode ultrasound (Sonoite Micromaxx) equipped with a 6–13 MHz linear array transducer. Ultrasound was applied to find local arterial stiffness (β), where $\beta = \ln(\text{SBP}/\text{DBP})/(\Delta D/Dd)$ and is defined by the arterial diameter over the cardiac cycle relative to change in blood pressure²⁶. Blood pressure of the common carotid artery was recorded using applanation tonometry as described above. Blood pressure at the brachial artery, systolic blood pressure (SBP), diastolic blood pressure (DBP), and pulse pressure (PP) were sampled from four 6-second video recordings. For each participant, the probe was placed on the common carotid artery opposite to that of the stroke-affected side at a perpendicular (90°) angle to the vessel, 1 to 2 cm below bifurcation. Ultrasound global and probe-dependent settings were all standardized. Participants held their breath during each of the four 6-second video recordings with the common carotid artery diameters extending across the entire imaging plane. The four 6-second diameter measurements of systolic diameter (Dsys), diastolic diameter (Ddia), and the difference between systolic diameter and diastolic diameter (Dist) were collected and averaged. Additionally, distensibility coefficient (DC) and compliance coefficient (CC) measurements were obtained via ultrasound; where DC is the relative rise of arterial cross-sectional area for a given increase in pressure, and CC is the absolute rise in cross-sectional area for a given increase in arterial pressure, and assumes that the length of the vessel is unaltered by the pulse wave.

A repeated measures Analysis of Variance (ANOVA) was performed to determine differences in dependent variables versus time (time [pre-WBV, post-WBV, pre-control, post-control] x intervention). Data gathered was analyzed using SPSS software (version 21, SPSS Inc., Chicago, IL, USA). The level of statistical significance was set at $p < 0.05$ and all values are expressed as mean±standard deviation (SD).

RESULTS

All six participants completed the required 12 sessions of WBV and four weeks of control, on average it took 4.5±0.5 weeks (mean±SD) to complete 12 WBV sessions. Following a one-month follow-up, no participant suffered any further strokes, symptoms of stroke, or any other complications associated with WBV training.

There was no significant interaction effect of intervention and time, and no main effect of intervention or time for any of the PWA measurements (Table 2). However, a decrease in AIx@75 was displayed for WBV and the control illustrated an increase over time but this time effect was not significant ($p = 0.225$). There was a significant time effect post intervention for systolic ($p = 0.039$) and diastolic diameter ($p = 0.040$) (Table 2) but there was no interaction or intervention effect.

Table 2 illustrates the interaction effect of PWV over time, demonstrating a decrease over time for both WBV and control but these measurements were not significant ($p = 0.474$). Furthermore, there was no significant difference ($p = 0.299$) in

Table 2. PWV, PWA, blood pressure, and carotid artery diameter values for pre and post WBV and control

	WBV		Control	
	Pre	Post	Pre	Post
PWV (m/s)	7.0±2.1	6.6±1.8	6.7±1.3	6.5±0.7
Carotid to radial PTT SD (%)	6.5±3.2	6.9±1.9	6.8±2.6	5.5±2.8
Arterial stiffness (Beta)	12.1±2.5	10.2±2.7	10.0±3.1	11.6±2.7
DC (10-3 /kPa)	19.6±3.2	22.0 ±6.5	23.1±6.4	20.0±6.2
CC (mm ² /kPa)	0.8±0.2	1.0±0.3	1.0±0.3	0.9±0.3
AIx@75	18.1±5.0	17.5±5.9	14.2±6.8	18.3±5.8
cSBP (mmHg)	110.8±9.0	112.5±12.7	111.9±13.3	110.3±11.7
cDBP (mmHg)	76.4±10.0	77.9±8.6	77.9±10.0	76.6±10.1
cPP (mmHg)	34.3±3.7	34.6±7.5	34.0±6.7	33.8±6.4
HR (beats/min)	60.3±9.8	63.3±11.6	62.0±12.5	64.3±13.7
SBP (mmHg)	120.2±10.1	123.5±14.5	122.8±15.4	120.8±14.1
DBP (mmHg)	75.7±9.7	77.0±9.7	77.3±9.4	76.0±9.8
PP (mmHg)	44.5±6.4	46.5±9.8	45.5±8.7	44.8±8.2
Dsys (mm) *	7.6±0.9	7.8±0.8	7.7±0.6	7.8±0.7
Ddia (mm) *	7.3±0.9	7.5±0.8	7.3±0.7	7.5±0.6
Dist (mm)	0.3±0.1	0.4±0.1	0.4±0.1	0.3±0.1

PWV: pulse wave velocity; Carotid to radial pulse transit time standard deviation (PTT SD); DC: distensibility coefficient; CC: compliance coefficient; AIx@75: augmentation index normalized HR 75 bpm; cSBP: central systolic blood pressure; cDBP: central diastolic blood pressure; cPP: central pulse pressure; HR: heart rate; SBP: systolic blood pressure; DBP: diastolic blood pressure; PP: pulse pressure (SBP-DBP); Dsys: systolic diameter; Ddia: diastolic diameter; Dist: difference between Dsys and Ddia

* Significant time effect post intervention for Dsys ($p = 0.039$) and Ddia ($p = 0.040$)

carotid to radial pulse transit time standard deviation between WBV and control (Table 2).

Arterial stiffness (Table 2) decreased over time for WBV. In contrast, arterial stiffness increased over time in the control group, but the interaction effect was not significant ($p = 0.166$). For the distensibility coefficient (DC) (Table 2) there was no significant interaction effect ($p = 0.124$) or main effect between the WBV and control groups ($p = 0.431$). Similarly, there was no significant interaction effect ($p = 0.237$) or main effect for compliance coefficient (CC) (Table 2) between the WBV and control groups ($p = 0.496$).

DISCUSSION

The primary aim of this study was to assess the efficacy of short-term WBV training on indices of arterial stiffness (carotid arterial stiffness, augmentation index, pulse wave velocity) in individuals who have experienced chronic stroke. The results of the current WBV training protocol did not produce any significant positive or negative effects on central BPs, AIx@75, HR, PWV, carotid arterial stiffness (β), DC, and CC. These results are supported by recent research, which found no significant changes in blood pressure (SBP and DBP) following 3 months of WBV in middle-aged and older adults¹⁴). However, some studies of acute WBV have shown significant changes in BP. For example, Figueroa, et al.¹¹) and Rittweger, Beller, Felsenberg²⁷) reported significant increases in SBP after WBV with body-weight squats, where values returned to baseline 15 min post intervention, with Rittweger et al.²⁷) showing a significant decrease in DBP after WBV compared to control (bicycle ergometry). Additionally, an acute WBV study by Otsuki et al.⁹) found no significant changes in blood pressure (SBP, DBP, and PP) after 60 min of WBV or control (no WBV). In contrast, short-term WBV (6 weeks) showed a significant decrease in SBP 3 min after WBV when compared to control (no exercise)²⁸). However, the discrepancies between the results of Gil²⁸) and the present study may be attributed to exercise protocols and the participants' health conditions (overweight and obese women vs. chronic stroke).

In the present study, AIx@75 appeared to decrease post-WBV ($0.7 \pm 3.1\%$) and increase post-control ($4.3 \pm 4.8\%$). A non-significant decrease in AIx post-WBV has been reported by Figueroa, et al.¹¹). The authors reported that AIx decreased significantly during 15 and 30 min of recovery following an acute WBV bout involving a static squat compared to a significant increase in AIx during post-recovery from no-WBV (static squat only). According to the authors, the decrease in AIx wave magnitude following WBV was unlikely to be influenced by HR, but rather a result of vasodilation in the peripheral arteries¹¹), which may explain the decrease in central AIx seen in the present study.

Previously, Otsuki, et al.⁹) reported that following acute intermittent WBV (10×60 s, interspersed with 60 s rest) performed by healthy men, brachial-ankle PWV decreased during recovery at 20 and 40 min and returned to baseline 60 min after compared to no WBV (control), indicating that WBV acutely decreased arterial stiffness. In a similar study, the vibration protocol was modified slightly to include a higher frequency (40 Hz) that acquired PWV from carotid-femoral, brachial-ankle, and femoral-ankle sites¹¹). Femoral-ankle PWV decreased significantly at 5 min in both the WBV and without WBV (control) groups, where it continued to remain low during the 30-min recovery post-WBV period and the control returned to baseline. However, there were no significant changes in carotid-femoral PWV or brachial-ankle PWV after either trial. It should be noted that brachial-ankle PWV involves both central and peripheral arterial stiffness, where aortic and leg PWV are the main independent correlates, explaining 58% and 23% of the total variance in brachial-ankle PWV respectively²⁹), and as a result any changes in this measure of arterial stiffness should be interpreted carefully⁹).

The theory of a local effect may help to explain the current findings. If WBV generates a local effect on PWV, it would elicit changes in leg PWV rather than aortic or carotid-radial PWV. This has been demonstrated by changes in femoral-ankle PWV¹¹) and brachial-ankle PWV⁹), where brachial-ankle PWV was independently correlated with leg PWV²⁹). The changes seen by Otsuki et al.⁹) may represent changes in both aortic and leg arterial stiffness. However, Otsuki et al.⁹) did not measure aortic and leg PWV separately, and it cannot be determined whether leg PWV alone contributed to the improvement. Likewise, Figueroa et al.¹¹) did not display any significant changes in brachial-ankle PWV, and it is therefore possible that a local effect could explain the insignificant findings of the present study.

The findings of the current study revealed no significant change in carotid arterial stiffness (β), DC, or CC. Although, a significant time effect was evident for both diastolic diameter ($p = 0.040$) and systolic diameter (0.039), these significant time effects did not show any significance for the relative stroke change in diameter (Dist). A previous study reported that low-intensity resistance exercise in healthy individuals³⁰) acutely increases carotid arterial compliance, systolic and diastolic diameters, and decreased β stiffness index 30 and 60 min post-exercise. However, HR and carotid and brachial BPs showed no significant changes. The differences between the previous and the present study may be attributed to the duration of intervention and consequently when assessments took place after intervention (acute vs. short-term), mode of exercise (bench press vs. WBV), and participant characteristics (healthy individuals compared to chronic stroke).

The aim of this study was to recruit eight participants to undergo a trial based on previous work that studied six weeks of WBV in overweight/obese women that improved brachial-ankle PWV by 8%³¹) and achieved a power of 0.89 with $\alpha = 0.05$. However, the research was conducted in an area with a small stroke community, where only six participants volunteered for the study. Thus, the non-significant findings of the current study were possibly due to the small sample size. Furthermore, a longer duration of WBV may have elicited the desired changes. This assumption is supported by Gil²⁸), who suggested that it may take longer than 6 weeks to detect changes in PWV.

In conclusion, while there were no significant findings, the present study did identify a non-significant decrease in carotid arterial stiffness post-WBV compared to pre-WBV, while control decreased between pre and post. However, no adverse effects were reported, indicating that WBV is a safe exercise modality for chronic stroke patients.

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