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Arturo Figueroa, Florence Vicil and Marcos A Sanchez-Gonzalez

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Original Article

Acute exercise with whole-body vibration decreases wave reflection and leg arterial stiffness

Arturo Figueroa, Florence Vicil, Marcos A. Sanchez-Gonzalez

Department of Nutrition, Food and Exercise Sciences, Florida State University, Tallahassee, FL 32306, USA

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Abstract: Aim: Whole-body vibration exercise (WBV) acutely decreases brachial-ankle pulse wave velocity (baPWV), an index of systemic arterial stiffness. However, the effect of WBV on segmental PWV and aortic hemodynamics is unknown. We examined the acute effects of WBV on arterial function. Methods: Fifteen young men performed ten 1-min sets of static squat with WBV (40 Hz, 1 mm, 5.37 G) and without WBV (no-WBV). Brachial and aortic blood pressure (BP), heart rate (HR), augmentation index (Alx), baPWV, carotid-femoral PWV (cfPWV), and femoral-ankle (faPWV), were recorded before and 5, 15, and 30 min after both trials. Results: Brachial and aortic SBP ($P < 0.01$), and HR ($P < 0.01$) were increased only at 5 min after both exercise trials. Alx was elevated through the recovery after no-WBV while decreased at 15 and 30 min after WBV exercise. FaPWV was decreased ($P < 0.01$) at 5 min after both trials, but returned to baseline at 15 min after no-WBV exercise and was maintained decreased at 15 and 30 min after WBV exercise. There were no significant changes in brachial and aortic diastolic BP, cfPWV and baPWV after both trials. Conclusions: Our findings indicate that regardless of WBV, static squat causes a small transient increase in hemodynamic responses during early recovery. WBV counteracts the increase in Alx induced by static squat and reduces wave reflection magnitude through a local effect on arterial stiffness.

Keywords: Aortic hemodynamics, pulse wave velocity, wave reflection, static exercise

Introduction

Whole-body vibration (WBV) is a relatively new exercise training mode that has shown to improve muscle strength and mass [1, 2], bone mineral density [3], and glycemic control [4]. Although exercise with WBV training requires less time per session to improve health parameters than resistance exercise training [4], only a few studies have evaluated the acute cardiovascular responses to WBV. Recent studies have reported a decrease in brachial-ankle pulse wave velocity (baPWV) [5], an index of systemic arterial stiffness. Additionally, mild increases [6] or no changes [5, 7] in heart rate (HR) and brachial blood pressure (BP) have been reported after a session of static squat with WBV at a vibration frequency of 26 Hz. These data suggest that WBV appears to exert low levels of cardiovascular stress and possible reduction in systemic PWV. However, the specific effect of static squat and WBV on the main components

of baPWV (aortic PWV and leg PWV) [8] needs to be examined before recommending them to individuals with high cardiovascular risk.

Static squat acutely increases aortic SBP by ~8 mmHg and pressure wave reflection (augmentation index, Alx) by ~18.7% suggesting an increase in left ventricular afterload in young healthy adults [9]. Although static squat is the most common exercise used in WBV training, the aortic hemodynamic and regional PWV responses after an acute bout of static squat with and without WBV are unknown. Some studies have shown a decrease in leg PWV after acute single-leg static [10] and dynamic resistance exercise. Despite no change in aortic PWV, vasodilation of peripheral arteries may decrease leg PWV and the magnitude of the reflected wave and, thus, the aortic Alx after exercise [11-14]. Thus, it is possible that acute WBV would have a predominant effect on leg PWV and Alx as previously shown by an increase in leg mus-

cle blood flow during and immediately after static squat with WBV [7, 15].

The purpose of this study was to determine the effects of a single bout of static exercise with and without WBV on aortic hemodynamics, Alx, and arterial stiffness. We hypothesized that WBV would reduce aortic hemodynamic, Alx, and PWV responses compared to static squat without WBV.

Methods

Subjects

Fifteen apparently healthy men (21 ± 4 years) were recruited from the local university population. Eligibility criteria were that participants should be normotensive (BP <140/90 mm Hg), without overt chronic diseases as evaluated by health history. Participants were nonsmokers and either sedentary or moderately physically active (≤ 1.5 hours/week). Exclusion criteria were prescribed medications or supplements known to affect cardiovascular parameters and contraindications for WBV including recent fractures or wounds and gallbladder or kidney stones. This study was approved by the Institutional Review Board of The Florida State University and all subjects gave informed consent.

Study design

Measurements were performed during the morning in a quiet temperature-controlled room (23°C) and at the same time of the day (± 1 hour) to minimize potential diurnal variations. Subjects were tested in the postprandial (≥ 3 hours) condition on two separate visits at least 72 hours apart. Participants abstained from caffeine and alcohol for 12 hours and avoided intense exercise 48 hours prior to testing. After electrocardiogram (ECG), BP, and arterial tonometry instrumentation, participants rested supine for at least 20 min before a 5 min baseline period. Thereafter, subjects performed static squat with (WBV) or without WBV (no-WBV) in a randomized order. Immediately after exercise, subjects reassumed the supine position for 30 min of recovery. Post-exercise measurements were collected at 5 min, 15 min, and 30 min.

Exercise trials

During the no-WBV and WBV trials, subjects

stood on the vibration platform (Powerplate, Badhoevedorp, The Netherlands) with both knees flexed at 120° (full extension 180°) and slightly grasped the handlebar with the right hand. The vibration frequency and peak-by-peak amplitude for the WBV trial were 40 Hz and 1 mm, respectively, which corresponds to an acceleration of ~ 5.37 G. The protocol consisted of ten 1-min sets of static squat separated by 1-min inter-set rest periods in the seated position. The rationale for the number of bouts and durations was based on a previous study that has shown a decrease in baPWV after acute WBV [5].

Pulse wave velocity

PWV and brachial BP were measured using an automatic device (VP-2000, Omron Healthcare Inc., Vernon Hills, IL, USA). BP cuffs were wrapped around both arms (brachial artery) and ankles (posterior tibial artery) while two tonometers were placed over the right carotid and femoral arteries to obtain PWV measurements from three arterial segments: baPWV, carotid-femoral PWV (cfPWV), and femoral-ankle PWV (faPWV). The cfPWV and faPWV are considered measurements of aortic and leg arterial stiffness, respectively [16]. BaPWV is an index of systemic arterial stiffness, which mainly includes aortic PWV and leg PWV [8, 17]. ECG electrodes were placed on the forearms, while a heart sound microphone was placed on the left parasternal border of the second intercostal space. Transit time was automatically determined from the time delay between the feet of the pulse waves related to the R-wave of a simultaneously recorded ECG. We got the time delay between brachial and tibial arteries (Tba), carotid and femoral arteries (Tcf), and femoral and tibial arteries (Tfa). The distance from the carotid to femoral artery (Dcf) was measured with a nonelastic tape measure. The distances from the suprasternal notch to brachial arteries (Dhb), from the suprasternal notch to femoral arteries (Dhf), and from femoral to tibial arteries (Dfa) were calculated automatically according to the following formulas: $Dhb = 0.220 \times \text{height (cm)} - 2.07$, $Dhf = 0.564 \times \text{height (cm)} - 18.4$, and $Dfa = 0.249 \times \text{height (cm)} + 30.7$ [8,17]. PWV was calculated for each arterial segment using the following formulas: aortic PWV = Dcf/Tcf , faPWV = Dfa/Tfa , and baPWV = $(Dhf + Dfa - Dhb)/Tba$ [17]. Two measurements were collected at each time point and averaged. HR was

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Table 1. Peripheral and central hemodynamics before and after static squat without (no-WBV) and with whole-body vibration (WBV) trials in young men (n=15)

	Baseline	Post 5 min	Post 15 min	Post 30 min
Brachial SBP (mm Hg)				
No-WBV	127 ± 1	131 ± 2†	129 ± 1	128 ± 2
WBV	128 ± 1	130 ± 2†	126 ± 1	126 ± 2
Brachial DBP (mm Hg)				
No-WBV	70 ± 2	71 ± 2	71 ± 1	71 ± 2
WBV	71 ± 2	73 ± 2	69 ± 1	67 ± 2
Brachial MAP (mm Hg)				
No-WBV	86 ± 1	89 ± 2	89 ± 2	88 ± 2
WBV	88 ± 1	89 ± 2	85 ± 2‡	84 ± 2*‡
Aortic SBP (mm Hg)				
No-WBV	106 ± 1	110 ± 1†	109 ± 1	108 ± 2
WBV	107 ± 1	110 ± 1†	105 ± 1‡	105 ± 2
Aortic DBP (mmHg)				
No-WBV	71 ± 2	72 ± 2	73 ± 2	72 ± 2
WBV	72 ± 2	74 ± 3	70 ± 1	68 ± 2‡
Aortic MAP (mmHg)				
No-WBV	87 ± 1	89 ± 2	89 ± 1	88 ± 2
WBV	88 ± 1	89 ± 2	85 ± 1	84 ± 1‡
HR (beats/min))				
No-WBV	64 ± 3	69 ± 4†	66 ± 3	63 ± 3
WBV	64 ± 3	70 ± 4†	67 ± 4	64 ± 3

Values are mean ± SEM. SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; HR, heart rate; Significant difference from baseline at * $P < 0.05$, † $P < 0.01$; Significant difference from No-WBV at ‡ $P < 0.05$.

determined from the ECG.

Pulse wave analysis

Pulse waveforms were obtained from a 10 sec epoch using a high-fidelity tonometer (SPT-301B; Millar Instruments, Houston, TX, USA) placed on the left radial artery. Radial pressure waveforms were calibrated against brachial SBP and diastolic BP (DBP) because of comparable hemodynamic properties in the upper limb arteries [18]. Aortic pressure waveform was derived using a generalized transfer function (SphygmoCor, AtCor Medical, Sydney, Australia), which has been validated at rest [19] and during exercise [20]. The first (P1) and second (P2) systolic peaks of the aortic pressure wave are the result of a forward traveling wave produced by the stroke volume and a reflected wave returning to the ascending aorta from the peripheral arteries. The augmentation pressure (AP) is defined as the difference between P2 and P1 of the central aortic pressure wave and it can increase or decrease depending on the magnitude of P2 and P1 [11]. The Alx was defined as the AP expressed as a percentage of the aortic pulse pressure (SBP-DBP). Alx was normalized

to a HR of 75 beats/min (Alx@75) because it is influenced by HR [23]. Alx is an index of wave reflection intensity, which is affected by peripheral and aortic PWV [21]. The average of 2 measurements of brachial BP and high-quality (operator index ≥ 80%) aortic hemodynamics was used in the analysis.

Anthropometrics

Height was measured using a stadiometer to the nearest 0.5 cm and body weight was measured using a seca scale (Sunbeam Products Inc., Boca Raton, FL, USA) to the nearest 0.1 kg.

Statistical analysis

Data are presented as mean ± SEM. Student *t*-tests were used to determine possible baseline differences between trials. Differences in mean values for each variable between trials were compared by analysis of variance with repeated measures [trial (no-WBV vs. WBV) by time (baseline and 5, 15, and 30 min post-exercise)], followed by Fisher's LSD test. Appropriate post hoc analysis was used where significant main effect and interaction occurred. $P < 0.05$ was

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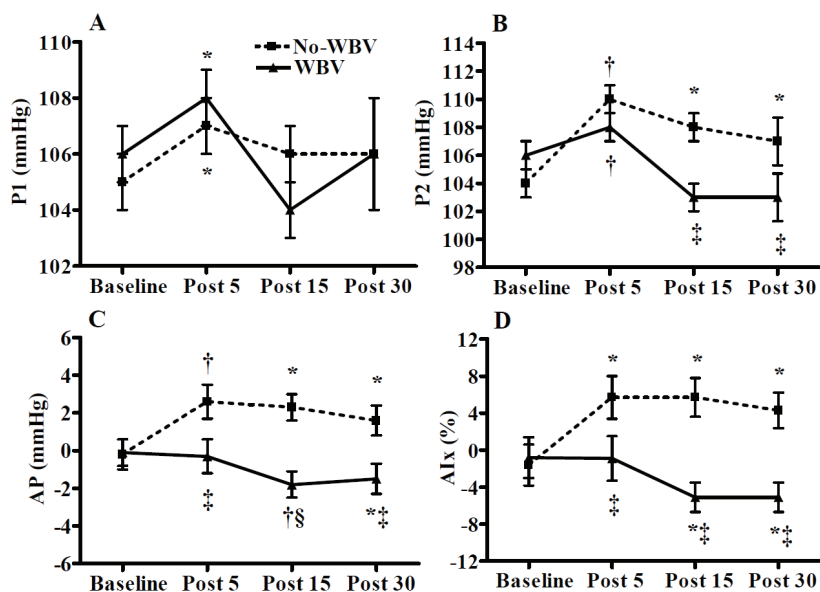


Figure 1. Aortic hemodynamic responses before (baseline) and 5, 15, and 30 min following a bout of static squat with whole-body vibration (WBV) and without WBV (no-WBV) trials in young men ($n = 15$). P1, first systolic peak pressure (A); P2, second systolic peak pressure (B); AP, augmentation pressure (C); and Alx, augmentation index (D). Values are mean \pm SEM. * $P < 0.05$ and † $P < 0.01$ vs. baseline; ‡ $P < 0.05$ and § $P < 0.01$ vs. No-WBV.

considered to be significant. SPSS version 16.0 (SPSS Inc., Chicago, IL, USA) was used for all analyses.

Results

Weight and height were 92.7 ± 3.7 kg and 1.80 ± 0.02 m, respectively. Peripheral and central hemodynamics are shown in **Table 1** and **Table 2**. Brachial and aortic SBP ($P < 0.01$), P1 ($P < 0.05$, **Figure 1**), P2 ($P < 0.01$, **Figure 1**), and HR ($P < 0.01$) were increased at 5 min after both exercise trials. All these parameters returned to baseline values at 15 min post-exercise, except P2, which remained elevated ($P < 0.05$) at 15 and 30 min after the no-WBV trial. The AP and Alx increased ($P < 0.05$) at 5, 15, and 30 min after the no-WBV trial, while they decreased ($P < 0.05$) at 15 and 30 min after the WBV trial (**Figure 1**). Changes in Alx and Alx@75 were similar. FaPWV decreased ($P < 0.01$) at 5 min after both trials, but it was maintained lower than baseline values ($P < 0.01$) at 15 and 30 min only after the WBV trial (**Figure 2**). A time-by-trial interaction ($P < 0.05$) was detected for AP, Alx, and Alx@75 at 5, 15, and 30 min post-exercise and for P2 and faPWV at 15 and 30 min post-exercise. There were no significant changes in brachial and aortic DBP, cfPWV, and baPWV after either trial.

Discussion

The main findings of the present study are that

hemodynamic parameters that influence the incident wave were transiently increased whereas faPWV was decreased at 5 min after both trials. In addition, wave reflection was increased and faPWV was not affected 15 to 30 min after no-WBV exercise while both parameters were reduced after WBV exercise.

In the present study, there were increases in SBP, HR, and P1 at 5 min after squat with and without WBV. We showed an increase in both brachial and aortic SBP (~ 4 mm Hg) during the early recovery of an acute bout of static squat exercise that was not affected by WBV. The

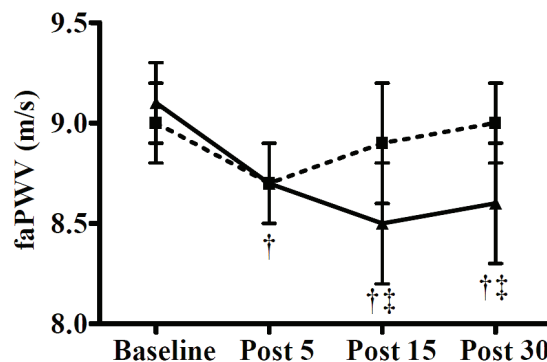


Figure 2. Femoral-ankle pulse wave velocity (faPWV) before (baseline) and 5, 15, and 30 min following a bout of static squat with whole-body vibration (WBV) and without WBV (no-WBV) trials in young men ($n = 15$). Values are mean \pm SEM. † $P < 0.01$ vs. baseline; ‡ $P < 0.05$ vs. No-WBV.

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Table 2. Wave reflection and arterial stiffness before and after static squat without (no-WBV) and with whole-body vibration (WBV) trials in young men (n= 15)

	Baseline	Post 5 min	Post 15 min	Post 30 min
Alx@75 (%)				
No-WBV	-6.9 ± 1.8	3.1 ± 2.8†	2.6 ± 2.8†	-1.1 ± 2.5*
WBV	-6.1 ± 1.7	-3.3 ± 3.0*‡	-9.1 ± 2.3*§	-9.6 ± 2.1*§
cfPWV (m/s)				
No-WBV	8.7 ± 0.3	9.1 ± 0.4	8.9 ± 0.3	8.8 ± 0.2
WBV	8.8 ± 0.3	9.1 ± 0.4	8.9 ± 0.3	8.5 ± 0.3
baPWV (m/s)				
No-WBV	12.0 ± 0.4	12.0 ± 0.3	12.0 ± 0.3	12.2 ± 0.4
WBV	12.0 ± 0.4	11.9 ± 0.3	11.8 ± 0.3	11.8 ± 0.3

Note: Values are mean ± SEM. Alx @ 75, augmentation index adjusted at HR 75; PWV, pulse wave velocity; cf, carotid-femoral; ba, brachial-ankle. Significant difference from baseline at * $P < 0.05$, † $P < 0.01$. Significant difference from No-WBV at ‡ $P < 0.05$, § $P < 0.01$.

magnitude of change in aortic SBP after our two trials was smaller than those reported during the early recovery of resistance exercise (9-12 mm Hg) [11, 22]. It is known that SBP and Alx are determined by HR, pulse wave reflection, and aortic PWV [16, 23]. Although an inverse relationship exists between HR and Alx [23], a low HR (~66-76 bpm) does not influence the increase in Alx after static handgrip [24] and squat exercise [9]. Since there was no increase in aortic PWV, the increases in aortic SBP and Alx after no-WBV squat were likely influenced by an increase in the magnitude of the reflected wave (P2), which has been attributed to peripheral vasoconstriction [25]. Although the pressures of the first (P1) and second systolic (P2) peaks increased 5 min after both trials, a significant increase in AP occurred only after no-WBV exercise resulting in an increased Alx. Conversely, the similar increase in P2 and P1 did not change the AP and, hence, the Alx after WBV. The increases in hemodynamic factors that influenced the incidence wave returned to baseline levels 15 min after static squat with and without WBV.

P2, AP, Alx and Alx@75 remained elevated 15 and 30 min after no-WBV squat exercise indicating an increased wave reflection from peripheral sites to the aorta. After isometric exercise, the acute increase in sympathetic activity evokes an increase in Alx [24], which leads to an increase in left ventricle afterload [9]. It is likely that the factors that influenced P2 and the Alx at 5 min post-exercise persisted for the remaining of the recovery only after the no-WBV trial. Conversely, WBV caused a decrease in AP and Alx after an acute bout of static squat. In agreement with

our findings, previous studies have observed reductions in Alx 15 and 30 min after maximal cycling exercise [13, 14] and low-intensity resistance exercise [11]. However, the decrease in Alx in the previous studies was likely influenced by the increased HR due to the inverse relationship between the two parameters [23]. In the present study, HR was recovered and P2 was lower after the WBV trial compared with the no-WBV trial. It is most likely that HR did not influence the decrease in wave reflection after WBV exercise. Thus, the decrease in wave reflection magnitude was due to vasodilation of peripheral arteries [14]. Our findings indicate that WBV counteracts the acute increase in Alx induced by isometric exercise [9, 24] and may prevent the increase in Alx noted after resistance exercise training [26].

Central artery stiffness is increased via structural changes in the arterial media such as increased collagen and fragmentation of elastin fibers [27]. In peripheral arteries, stiffness is influenced by acute changes in vascular tone regulated by sympathetic activity, endothelial and contraction-related factors. We previously noted that leg PWV and SBP can be increased by intense vascular sympathetic activation during muscle ischemia after no-WBV exercise [28]. The concurrent increases in PWV and SBP after a bout of exercise suggests that acute changes in PWV may be pressure-dependent [22]. In the present study, we observed a decrease in leg PWV concomitant with a small increase in SBP immediately after both exercise trials, which is consistent with previous data [29, 30]. Since BP returned to resting levels 15 min after both trials, it is likely that the decrease in leg PWV may

be due to changes in local arterial function independent of BP [10, 29, 31]. Our data support that the decrease in leg PWV influenced the decrease in Alx after WBV exercise. Previous studies have shown that the reduction in leg PWV after acute resistance [12], static [10], and endurance exercise [29, 31, 32] has been attributed to contraction-related vasodilatory factors. However, the reduction in leg PWV after no-WBV did not remain after 5 min post-exercise, suggesting the effect of other vasodilatory factors. An increased vasodilation in the inactive vibrated limb has been attributed to increased nitric oxide production [33]. Thus, the prolonged decrease in leg PWV after WBV exercise could be attributed mainly to the local effect of vibration on the leg arteries.

In contrast to our findings, Otsuki et al. [5] showed a decrease in baPWV 20 to 40 min after a session of WBV squat. Since the exercise protocols in both studies were comparable in number and duration of sets, the discrepancy in post-exercise baPWV could be due to the greater vibration frequency used in the present (40 Hz) than in the previous study (26 Hz). Vibration transmissibility to the trunk is completely abolished with frequencies greater than 30 Hz [34]. Thus, reduced vibration transmissibility to the trunk and no effect on the aorta may explain the no apparent effect of WBV on cfPWV and baPWV in the present study. However, the effect of antihypertensive treatment on peripheral arteries appears to be more related to the reduction in wave reflection magnitude rather than cfPWV [35]. Thus, the vascular effects of WBV may have relevant implications for individuals with conditions characterized by elevated Alx such as hypertension and stroke [35, 36].

We examined arterial function responses after a session of static squat with WBV at 40 Hz in young healthy men and the results may not be directly applied to different populations or WBV protocols. In addition, the vascular effects of exercise with WBV beyond 30 min of recovery were not assessed. Evaluation of lower WBV frequencies, greater exercise time, and longer recovery periods than those used in the present study is warranted in populations that would likely get more benefits from exercise with WBV such as the elderly and individuals with type 2 diabetes who have an intermediate increase in faPWV besides the preferential increase in central PWV [37].

In conclusion, a bout of static squat induced an acute increase in Alx due to increased magnitude of the reflected wave. WBV overcomes the increase in Alx during early recovery and decreases Alx during late recovery by decreasing pressure wave reflection and leg PWV.

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Conflict of interest: The authors declare no conflict of interest.

Please address correspondence to: Arturo Figueroa, MD, PhD, Department of Nutrition, Food, and Exercise Sciences, College of Human Sciences, 120 Convocation Way, Tallahassee, FL 32306-1493, USA, Tel: 850-644-8089, Fax: 850-645-5000, E-mail: afigueroa@fsu.edu

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