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ORIGINAL ARTICLE

Whole-body vibration attenuates the increase in leg arterial stiffness and aortic systolic blood pressure during post-exercise muscle ischemia

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Abstract Exercise with whole-body vibration (WBV) decreases brachial-ankle pulse wave velocity (baPWV), a marker of systemic arterial stiffness. To examine the effect of WBV on arterial responses, 12 young men underwent three experimental trials: (1) no-exercise control (CON), (2) static squat with WBV, and (3) static squat without WBV (no-WBV). Bilateral baPWV and femoral-ankle PWV (faPWV), carotid-femoral PWV (cfPWV), augmentation index (AIx), first (P1) and second (P2) systolic peaks, aortic systolic blood pressure (aSBP), and heart rate (HR) were assessed at rest, during 4-min post-exercise muscle ischemia (PEMI) on the left thigh, and 4-min recovery. During PEMI, right faPWV increased ($P < 0.05$) after no-WBV and did not change after CON and WBV. Right baPWV, P2, and aSBP increased ($P < 0.05$) after both exercise trials, but the increase was lower ($P < 0.05$) after WBV than no-WBV. The increases in cfPWV ($P < 0.05$), AIx ($P < 0.05$), P1 ($P < 0.01$), and HR ($P < 0.05$) were similar in both trials during PEMI. During recovery, right faPWV and baPWV remained similar than rest after WBV and CON, but remained elevated ($P < 0.05$) after no-WBV. Aortic SBP, P1, and P2 remained elevated ($P < 0.05$) in both exercise trials during recovery, but the levels were lower ($P < 0.05$) than PEMI. Left faPWV and baPWV were reduced ($P < 0.05$) from rest in the three trials. CfPWV, AIx, and HR returned to resting levels in both exercises. WBV prevents the increases in faPWV and attenuates the increase in baPWV and aSBP induced by post-static squat muscle ischemia due to an attenuated P2 response.

Keywords Aortic hemodynamics · Pulse wave velocity · Wave reflection · Static exercise

Introduction

The vascular response to static exercise includes increased aortic stiffness (pulse wave velocity, PWV) (Geleris et al. 2004), which leads to increases in aortic systolic blood pressure (BP) and wave reflection (augmentation index, AIx) (Figueroa et al. 2010a; Murakami 2002), indicating increased left ventricular after load. Whole-body resistance exercise has acutely increased carotid stiffness and systolic BP (SBP) (DeVan et al. 2005). Conversely, the local decrease in PWV is pronounced in the exercised limb after acute leg static and resistance exercise (Davies et al. 2007; Heffernan et al. 2006). This reduction in leg PWV after exercise may be associated with decreases in SBP (Kingwell et al. 1997) and AIx (Figueroa and Vicil 2010; Heffernan et al. 2007b; Munir et al. 2008).

Exercise with whole-body vibration (WBV) is a new training modality that has been shown to increase muscle strength and mass (Delecluse et al. 2003; Machado et al. 2009), which can be of clinical importance in individuals who cannot perform high-intensity and prolonged traditional exercise. However, the acute vascular responses to static exercise with WBV are presently unclear. It has been shown that static squat with WBV acutely decreases brachial to ankle PWV (baPWV) (Otsuki et al. 2008), a

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marker of systemic arterial stiffness (Yamashina et al. 2002). However, the effect of exercise with WBV on the main components (aortic and leg PWV) of baPWV has not been investigated. A predominant effect of exercise with WBV on leg PWV (Figueroa et al. 2010b) would be consistent with the increased leg muscle blood flow noted during and immediately after static squat with WBV (Kerschan-Schindl et al. 2001; Lythgo et al. 2009).

Since PWV cannot be measured during WBV, muscle metaboreflex activation during post-exercise muscle ischemia (PEMI) can be used as a means to evaluate arterial responses following isometric leg exercise (Davies et al. 2007). Metaboreflex activation by trapped metabolites using mechanical arterial occlusion in the exercised limb, maintains exercise-induced increases in BP and systemic sympathetic-mediated vasoconstriction (Hansen et al. 1994; Rowell and O'Leary 1990). It has been shown that these vasodilatory metabolites can suppress the increase in PWV in a non-occluded leg during PEMI (Davies et al. 2007), a phenomenon termed functional sympatholysis (Remensnyder et al. 1962). Previous studies have reported that metaboreflex activation increases aortic SBP and AIx after static handgrip exercise (Figueroa et al. 2010a). However, whether WBV can suppress or attenuate sympathetic mediated increases in PWV, aortic BP, and wave reflection is unknown.

The purpose of the present study was to examine the effect of WBV on arterial stiffness and aortic hemodynamics during PEMI following a bout of static squat. We hypothesized that WBV would attenuate the increases in baPWV, aortic BP, and wave reflection due to a decrease in leg PWV.

Methods

Subjects

Twelve apparently healthy men (23 ± 1 years) were studied. Participants were normotensives, nonsmokers, noncompetitive athletes (≤3 h of exercise per week), free of overt chronic diseases as evaluated by a medical health history questionnaire, and not on medications or taking nutritional supplements. All subjects signed an informed consent prior to data acquisition. This study was approved by the Institutional Review Board of The Florida State University and conformed to the Declaration of Helsinki.

Study design

In a randomized fashion, subjects were evaluated on 3 days separated by at least 72 h. The experiments were conducted in the morning after at least 3 h postprandial in a quiet temperature-controlled room (23°C) and at the same time of the day (±1 h) in order to minimize potential diurnal variations. Participants abstained from caffeine and alcohol for 12 h and avoided intense exercise 48 h prior to testing. After 20 min of rest, baseline hemodynamics and arterial function parameters were collected. Immediately after the cessation of exercise or the no-exercise control (CON) period in the seated position, subjects reassumed the supine position for PEMI and recovery. Post-exercise cardiovascular parameters were collected during the last 2 min of PEMI and recovery.

Experimental protocol

Subjects performed 4 min of bilateral static squat exercise with a knee angle of 120° with WBV or without WBV (no-WBV) on the vibration platform (pro5 AIRdaptive Power Plate, Badhoevedorp, The Netherlands). In the WBV trial, subjects were exposed to vertical vibration with a frequency and peak-to-peak displacement of 30 Hz and 1 mm, respectively, which provides a peak-to-peak acceleration of ~4.16 G. Ten seconds before the end of the exercise or CON, a rapid pneumatic cuff around the left thigh was rapidly inflated (E20, Hokanson, Belleveue, WA) to 250 mmHg for 4 min to isolate the muscle metaboreflex (Iellamo et al. 1999a, b). After completion of the exercise or CON period, subjects lay down on a bed located next to the vibrating machine. After the release of the occlusion, subjects remained supine for 4 min of recovery.

Pulse wave velocity, brachial blood pressure, and heart rate

After 20 min of supine rest, brachial BP and aortic PWV were measured using an automatic device (VP-2000, Omron Healthcare Inc., Vernon Hills, IL). BP cuffs were wrapped around both arms (brachial artery) and ankles (posterior tibial artery), and 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 the measurements of aortic and leg arterial stiffness, respectively (Laurent et al. 2006). BP 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 ECG. The distance between carotid and femoral artery was measured with a nonelastic tape measure as a straight line, while the distance between sampling points of baPWV and faPWV was calculated automatically according to the height of the subjects (Yamashina et al. 2002). The path length from the

suprasternal notch to the femoral artery was automatically calculated as 0.564 × height (cm) − 18.4. PWV was calculated as the distance between two sampling sites divided by the transit time. Heart rate (HR) was obtained from the ECG. Two measurements were collected at each time point and averaged.

Pulse wave analysis

Brachial SBP and diastolic BP (DBP) were used to calibrate radial waveforms, which were obtained from a 10 s epoch using a high-fidelity tonometer (SPT-301B; Millar Instruments, Houston, TX). Aortic BP waveforms were derived using a validated generalized transfer function (SphygmoCor, AtCor Medical, Sydney, Australia) (O'Rourke et al. 2001; Wilkinson et al. 2002). The aortic BP wave is composed of a forward wave, caused by stroke volume ejection, and a reflected wave that returns to the aorta from peripheral sites (Nichols and Singh 2002). Augmentation pressure (AP) is the difference between the second (P2) and first (P1) systolic peaks. The AIx was defined as the AP expressed as a percentage of the aortic pulse pressure (PP). AIx was normalized to a HR of 75 beats/min (AIx@75) because it is influenced by HR (Wilkinson et al. 2000). Transit time of the reflected wave (Tr) indicates the round-trip travel of the forward wave to the peripheral reflecting sites and back to the aorta (Nichols and Singh 2002). Systolic ejection duration (ED) was determined from the inflection point to the incisura of the dicrotic notch (O'Rourke et al. 2001). The average of two measurements of brachial BP and high-quality (operator index ≥ 80%) aortic hemodynamics was used in the analysis.

Statistical analysis

One-way ANOVA was used to identify possible differences at baseline among the trials. Two-way ANOVA with repeated measures [trial (CON, no-WBV, and WBV) × time (baseline, PEMI, and recovery)] was used to compare the effects of static exercise with and without WBV on hemodynamic and arterial responses during PEMI and recovery. If a significant trial by time interaction was detected, Tukey's test was used for post hoc comparisons. Pearson's correlation coefficient analysis was performed between significant parameters during PEMI and recovery. Data are reported in mean ± SE. Statistical analyses were performed using SPSS 17.0 (SPSS Inc., Chicago, IL, USA). Based on the results of a previous study (Otsuki et al. 2008), we estimated that a sample size of eight subjects would have a 80% power to detect a 3% difference in PWV.

Results

Height, weight, and body mass index were 1.78 ± 0.02 m, 81.4 ± 2.9 kg, and 25.2 ± 0.8 kg/m^2, respectively. All the cardiovascular parameters at baseline were similar among the trials (Tables 1, 2; Fig. 1).

Table 1 Arterial stiffness and wave reflection responses after control, static squat with and without whole-body vibration ($n = 12$)

	Baseline	PEMI	Recovery
Left faPWV (m/sec)			
CON	8.8 ± 0.3	–	7.9 ± 0.2[a]
No-WBV	8.9 ± 0.2	–	7.2 ± 0.2[a]
WBV	9.1 ± 0.3	–	7.4 ± 0.3[a]
CfPWV (m/sec)			
CON	8.2 ± 0.3	8.7 ± 0.3	8.6 ± 0.4
No-WBV	8.4 ± 0.4	10.1 ± 0.4[a, c]	8.6 ± 0.4
WBV	8.0 ± 0.3	10.8 ± 0.6[a, c]	8.8 ± 0.3
Left baPWV (m/sec)			
CON	11.7 ± 0.4	–	10.5 ± 0.5[a]
No-WBV	12.0 ± 0.5	–	10.2 ± 0.3[a]
WBV	12.1 ± 0.3	–	10.5 ± 0.4[a]
AIx (%)			
CON	0 ± 1	1 ± 1	1 ± 1
No-WBV	0 ± 1	5 ± 2[a, c]	2 ± 2
WBV	0 ± 1	2 ± 2[a]	0 ± 2
AIx @75 (%)			
CON	−2 ± 1	0 ± 1	−2 ± 1
No-WBV	−2 ± 1	4 ± 1[a, c]	−1 ± 1
WBV	−2 ± 1	2 ± 1[a]	−2 ± 1
Tr (msec)			
CON	173 ± 9	173 ± 6	175 ± 7
No-WBV	174 ± 9	143 ± 4[b, c]	162 ± 6
WBV	178 ± 7	152 ± 4[b, c]	162 ± 8
P1 (mmHg)			
CON	99 ± 2	104 ± 3	101 ± 2
No-WBV	100 ± 2	125 ± 3[b, c]	107 ± 3[a, d]
WBV	101 ± 2	121 ± 3[b, c]	106 ± 3[a, d]

Data are means ± SE

CON control, *WBV* exercise with whole-body vibration, *no-WBV* exercise without WBV, *PEMI* post-exercise muscle ischemia, left (occluded leg), *PWV* pulse wave velocity, *faPWV* femoral-ankle PWV, *cfPWV* carotid-femoral PWV, *baPWV* brachial-ankle PWV, *AIx* augmentation index, *AIx @75* AIx adjusted for 75 beats/min, *Tr* reflection time, *P1* first systolic peak

[a] $P < 0.05$, different than baseline

[b] $P < 0.01$, different than baseline

[c] $P < 0.05$, different than CON

[d] $P < 0.05$, different than PEMI

Table 2 Peripheral and central blood pressure responses after control and static squat with and without whole-body vibration ($n = 12$)

	Baseline	PEMI	Recovery
Brachial SBP (mmHg)			
CON	120 ± 2	126 ± 2	122 ± 3
No-WBV	122 ± 2	154 ± 4[b, c]	131 ± 3[a, c, d]
WBV	123 ± 2	150 ± 4[b, c]	128 ± 3
Brachial DBP (mmHg)			
CON	63 ± 2	69 ± 2	65 ± 2
No-WBV	63 ± 2	84 ± 2[b, c]	69 ± 2
WBV	66 ± 3	83 ± 4[b, c]	69 ± 2
Brachial MAP (mmHg)			
CON	87 ± 4	93 ± 4	88 ± 2
No-WBV	85 ± 3	109 ± 3[b, c]	92 ± 2
WBV	89 ± 4	111 ± 4[b, c]	93 ± 2
Brachial PP (mmHg)			
CON	57 ± 2	56 ± 3	55 ± 2
No-WBV	59 ± 2	71 ± 4[b, c]	63 ± 2
WBV	89 ± 4	67 ± 2[b, c]	59 ± 2
Aortic DBP (mmHg)			
CON	68 ± 3	73 ± 2	70 ± 2
No-WBV	65 ± 2	88 ± 3[b, c]	71 ± 2
WBV	67 ± 3	84 ± 3[b, c]	71 ± 2
Aortic MAP (mmHg)			
CON	80 ± 4	86 ± 4	80 ± 2
No-WBV	80 ± 3	107 ± 2[b, c]	87 ± 2
WBV	82 ± 3	104 ± 3[b, c]	88 ± 2
Aortic PP (mmHg)			
CON	32 ± 2	33 ± 2	32 ± 2
No-WBV	36 ± 3	44 ± 2[b, c]	39 ± 2
WBV	32 ± 3	37 ± 3[b, c]	37 ± 2
HR (beats/min)			
CON	60 ± 3	62 ± 6	60 ± 5
No-WBV	63 ± 3	77 ± 6[a, c]	68 ± 5
WBV	64 ± 4	75 ± 5[a, c]	64 ± 4
ED (ms)			
CON	326 ± 8	321 ± 9	328 ± 9
No-WBV	326 ± 8	311 ± 9[a c]	328 ± 9
WBV	326 ± 8	311 ± 8[a c]	330 ± 8

Data are means ± SE

CON control, *WBV* exercise with whole-body vibration, *no-WBV* exercise without WBV, *PEMI* post-exercise muscle ischemia, *B* brachial, *A* aortic, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *MAP* mean arterial pressure, *PP* pulse pressure, *HR* heart rate, *ED* systolic ejection duration

[a] $P < 0.05$, different than baseline

[b] $P < 0.01$, different than baseline

[c] $P < 0.05$, different than CON

[d] $P < 0.05$, different than PEMI

Responses during PEMI

PWV and wave reflection are shown in Table 1. Brachial and aortic BPs, HR, and ED are shown in Table 2. All cardiovascular parameters in the CON trial and right (non-occluded leg) faPWV in the WBV trial were not significantly different than baseline. Compared to baseline and the WBV trial, right faPWV increased ($P < 0.05$) in the no-WBV trial. The increases in cfPWV ($P < 0.05$), P1 ($P < 0.01$), P2 ($P < 0.01$), brachial BPs ($P < 0.01$), and aortic BPs ($P < 0.01$) as well as the decreases in Tr ($P < 0.01$) and ED ($P < 0.05$) in both exercise trials were significantly different than baseline and the CON trial. Right baPWV, AIx, and AIx@75 increased ($P < 0.05$) in both exercise trials compared to baseline, but only the responses in the no-WBV trial were higher than the CON trial. The increases in right faPWV and baPWV, P2, and aortic SBP were greater ($P < 0.05$) in the no-WBV than WBV trial (Fig. 1). There was a significant correlation between the increase in aortic SBP and the increases in P2 ($r = 0.98$, $P < 0.001$), right faPWV ($r = 0.61$, $P < 0.05$), and right baPWV ($r = 0.63$, $P < 0.05$) as well as between the increases in HR and cfPWV ($r = 0.67$, $P < 0.05$) in the WBV trial.

Responses during recovery from PEMI

Left (occluded leg) faPWV and baPWV were reduced ($P < 0.05$) similarly in the three trials compared with baseline. Right faPWV and baPWV, cfPWV, AIx, AIx@75, Tr, brachial BPs, aortic DBP, aortic MAP, aortic PP, HR, and ED in the CON and WBV trial were no different ($P > 0.05$) than baseline. In the no-WBV trial, right faPWV and baPWV were increased ($P < 0.05$) compared with baseline, while brachial SBP was increased ($P < 0.05$) compared with baseline and the CON trial. In both exercise trials, P1 and P2 were increased ($P < 0.05$) compared with baseline, while aortic SBP was increased ($P < 0.05$) compared with the CON trial and baseline. Right faPWV, right baPWV, and brachial SBP in the no-WBV trial and P1, P2, aortic SBP in both exercise trials were significantly lower ($P < 0.05$) during the recovery compared with PEMI. The decreases in left faPWV and left baPWV were correlated in the CON ($r = 0.91$, $P < 0.001$), no-WBV ($r = 0.87$, $P < 0.001$), and WBV ($r = 0.87$, $P < 0.001$) trials. The increase in right faPWV was correlated with the increases in aortic SBP ($r = 0.87$, $P < 0.001$) and P2 ($r = 0.62$, $P < 0.05$) in the no-WBV trial.

Discussion

The major findings of the present study are that WBV prevented the increase in faPWV and attenuated the increases in baPWV, the magnitude of the reflected wave (P2), and aortic SBP induced by muscle metaboreflex activation after static squat exercise. These findings suggest

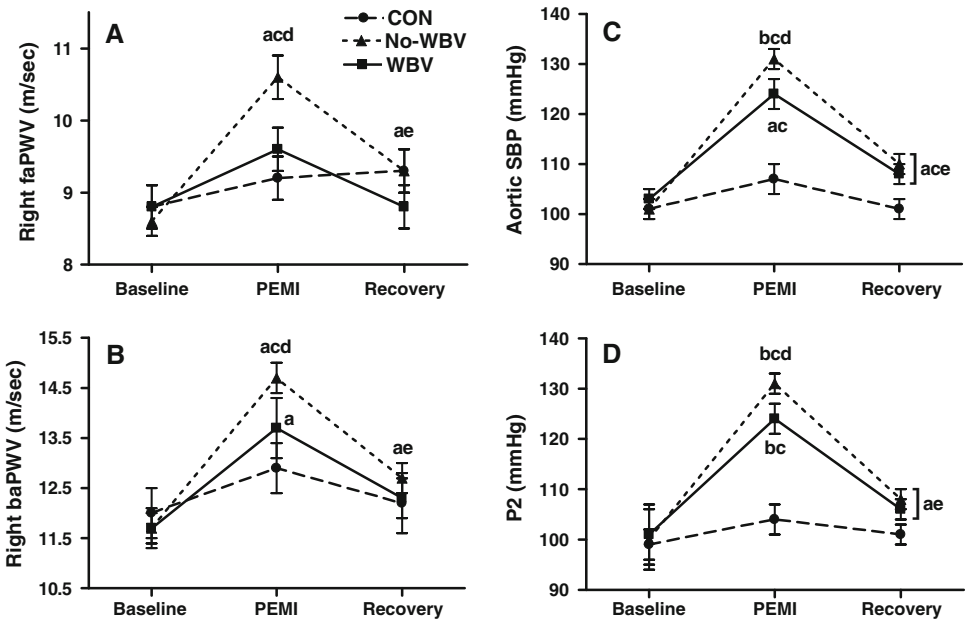

Fig. 1 Changes in leg arterial stiffness (**a**), systemic arterial stiffness (**b**), aortic systolic blood pressure (ASBP, **c**), and magnitude of the reflected wave (**d**) after static squat with and without whole-body vibration. *faPWV* femoral-ankle pulse wave velocity of the non-occluded leg (*right*); *baPWV* right brachial-ankle pulse wave velocity; P2, second systolic peak pressure. [a]$P < 0.05$, [b]$P < 0.01$, different than baseline. [c]$P < 0.05$, different than CON, [d]$P < 0.05$, different than WBV, [e]$P < 0.05$, different than PEMI

that WBV attenuates the increase in left ventricular load during PEMI through decreases in leg arterial stiffness and wave reflection.

Without PEMI, PWV decreases in the exercised leg after a bout of resistance (Heffernan et al. 2006), static (Davies et al. 2007), and endurance exercise (Kingwell et al. 1997; Sugawara et al. 2003) due to local vasodilation induced by muscle metabolites. During PEMI of the forearm, previous work suggests that local vasodilation can override the metaboreflex-induced vasoconstriction and prevent the increase in leg PWV after static calf exercise (Davies et al. 2007). Our finding that metaboreflex activation increased faPWV in the non-occluded leg during PEMI of the exercised thigh muscles after the no-WBV trial seems to contradict the previous study. However, greater muscle mass size (legs vs. forearm), greater exercise intensity (body weight vs. 40% of maximal voluntary contraction), and longer exercise and PEMI duration (4 vs. 2 min) used in the present study than in the previous study (Davies et al. 2007) may explain the apparent discrepancy. It is known that the magnitude of the cardiovascular responses is associated to the muscle mass and exercise intensity (Fisher et al. 2010; Freund et al. 1978). In the present study, WBV suppressed the increase in faPWV induced by PEMI after static squat with no-WBV. This finding suggests that functional sympatholysis may have occurred in the leg during PEMI following WBV. Our observation is consistent with a previous result in our laboratory showing that faPWV was reduced 5 min after an acute bout of static squat with WBV when the metaboreflex is not evoked (Figueroa et al. 2010b). Moreover, an increased leg muscle blood flow has been reported during and immediately after acute squat with WBV (Kerschan-Schindl et al. 2001; Lythgo et al. 2009). It appears that WBV counteracted the metaboreflex-induced increase in faPWV via the predominant effect of local vasodilatory factors (Davies et al. 2007).

Our findings are consistent with previous results showing increased central (carotid and aortic) PWV during conditions characterized by increased sympathetic activation such as static exercise, cold stress, and infusion of catecholamines (Geleris et al. 2004; Lafleche et al. 1998; Wilkinson et al. 2001). During PEMI, we observed an increase in baPWV, which includes aorta and peripheral (arm and leg) arterial stiffness (Yamashina et al. 2002). Since aortic PWV is the main component of baPWV (Sugawara et al. 2005), the absence of vasodilation in peripheral and central arteries likely explains the increase in baPWV after static squat. Despite the suppression of the faPWV response in the non-occluded leg, WBV did not affect the increases in cfPWV. It is likely that a reduced vibration transmission to the trunk and upper limbs by the squat position (Pel et al. 2009) and vibration intensities equal or higher than 30 Hz (Rubin et al. 2003) and, hence, no vasodilation in these arterial segments, may have influenced this finding. Conversely, the decrease in right faPWV would have attenuated the increase in baPWV in the WBV trial during PEMI.

In the present study, brachial SBP increased similarly in both exercise trials during PEMI. Previous work has shown that brachial SBP is less sensitive than central SBP (DeVan et al. 2005) to assess vascular responses after acute exercise. In addition, peripheral SBP underestimates the effect of peripheral vasodilation on aortic SBP (Takazawa et al. 1995). In the present study, aortic SBP was able to reveal differences between the trials. The increased systemic

arterial stiffness, including faPWV, would have evoked a faster return of the reflected wave in late systole and its fusion with the incident wave increased aortic SBP and, hence, the AIx (Nichols and Singh 2002; Wilkinson et al. 2001) after static squat. Although, it has been shown that bilateral femoral artery occlusion increases wave reflection (Latham et al. 1985), we noted that unilateral femoral artery occlusion did not affect wave reflection and aortic BP in the CON trial. This observation indicates that the increase in wave reflection during PEMI after both no-WBV and WBV trials was induced by metaboreflex activation. A novel finding of the present study is that the aortic SBP response was attenuated by WBV. Although the increase in AIx@75 and reduction in Tr tended to be smaller after the WBV than the no-WBV trial, we noted that WBV attenuated the increase in P2, an effect that has been attributed to a reduced vascular tone of small muscular arteries (Hashimoto et al. 2008; Kelly et al. 2001). This finding is consistent with a previous report showing that the magnitude of the reflected wave from the leg muscular arteries decreases after exercise (Munir et al. 2008). Since maximal SBP corresponds to P2 in the ascending aorta (Takazawa et al. 1995), the lower aortic SBP response during PEMI after WBV was likely due to the attenuated increase in P2, which was associated with the suppressed arterial stiffness in the vibrated leg.

HR recovery during PEMI of the forearm muscles is due to reactivation of the cardiovagal baroreflex by the distending effect of an elevated SBP and the loss of inhibitory signals from the central command (Figueroa et al. 2010a; Iellamo et al. 1999b; Nishiyasu et al. 1994). In line with results of the present study, previous work has shown that HR is maintained elevated during PEMI of the leg muscles (Crisafulli et al. 2006; Tokizawa et al. 2006). Apparently, higher exercise intensity mainly determines the increase in HR during PEMI due to an enhanced cardiac sympathetic activity induced by a strong muscle metaboreflex activation (Fisher et al. 2010). An increased cfPWV may reduce arterial wall deformation in response to elevated SBP, leading to reduced cardiovagal baroreflex activation after acute exercise (Heffernan et al. 2007a). We observed that the changes in cfPWV and HR were positively correlated during PEMI. Thus, the increased cfPWV may have contributed to the elevated HR during PEMI most likely due to reduced baroreflex reactivation and increased cardiac sympathetic activity.

Femoral artery dilation is predominantly mediated by nitric oxide (NO) after the release of arterial occlusion (Kooijman et al. 2008). During the recovery from PEMI, faPWV and baPWV of the occluded side (left) were reduced in all the trials. Conversely, peripheral and systemic arterial stiffness of the non-occluded side (right) remained elevated in the no-WBV trial, while right arterial stiffness was maintained at baseline levels in the WBV and CON trials. These data indicate that the effect of reactive hyperemia was localized to the left side of the body in all trials. Besides local exercise-related vasodilatory factors (Davies et al. 2007), the unaffected faPWV in the vibrated non-occluded leg during PEMI and recovery may have involved endothelial factors evoked by the mechanical effect of vibration (Nakamura et al. 1996). While cfPWV, AIx, Tr, HR, and ED returned to resting levels, P1, P2, and aortic SBP were partially recovered in both exercise trials, suggesting that the influence of the metaboreflex persisted on peripheral arteries. In contrast, brachial SBP remained significantly elevated only after the no-WBV trial. Thus, it appears that WBV attenuated the residual influence of the metaboreflex on peripheral arterial stiffness and SBP during the recovery form PEMI.

The major limitations of the present study were the lack of measurement of arterial vasodilation, autonomic function, and vasodilatory substances in blood or muscle. However, PWV and wave reflection have been suggested to reflect the effect of exercise on vascular tone (Kingwell et al. 1997; Munir et al. 2008). Although we did not directly measure autonomic function, it is well known that PEMI increases sympathetic activity (Fisher et al. 2010; Hansen et al. 1994; Iellamo et al. 1999a) that explains the cardiovascular responses observed in our study. Although autonomic cardiovascular responses to PEMI are fully recovered within 4 min after PEMI (Iellamo et al. 1999a, b), the lack of further post measures (10–15 min) would be considered a limitation of the present study. We examined arterial responses in young men and the results may not be generalized to other populations. Because exercise with WBV has the potential to prevent the age-related decrease in muscle mass and strength (Machado et al. 2009), future research is needed to evaluate the cardiovascular responses to this exercise mode in older individuals.

In conclusion, PEMI evoked increases in systemic arterial stiffness and hemodynamics following 4 min of static squat. WBV prevents the increase in leg arterial stiffness and attenuates the increases in systemic arterial stiffness, the magnitude of the reflected wave, and aortic SBP during PEMI.

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Conflict of interest The authors declare no conflict of interest.

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