Arterial stiffness acutely decreases after whole-body vibration in humans

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Abstract

Background: Increased arterial stiffness is a well-established cardiovascular risk factor. Mechanical stimuli to artery, such as compression, elicit vasodilation and acutely decrease arterial stiffness. As whole-body vibration (WBV)-induced oscillation is propagated at least to lumbar spine, WBV mechanically stimulates abdominal and leg arteries and may decrease arterial stiffness. WBV is feasible in vulnerable and immobilized humans. Therefore, it is worthwhile to explore the possibility of WBV as a valuable adjunct to exercise training.

Aim: The aim of this study was to investigate the acute effects of WBV on arterial stiffness.

Methods: Ten healthy men performed WBV and control (CON) trials on separate days. The WBV session consisted of 10 sets of vibration (frequency, 26 Hz) for 60 s with an inter-set rest period of 60 s. Subjects maintained a static squat position with knees bent on a platform. In the CON trial, WBV stimulation was not imposed. Blood pressure, heart rate and brachial-ankle pulse wave velocity (baPWV), an index of arterial stiffness, were measured before and 20, 40 and 60 min after both trials.

Results and conclusion: Heart rate and blood pressure did not change from baseline after both trials. Although baPWV did not change in the CON trial (baseline vs. after 20, 40 and 60 min; 1144 ± 35 vs. 1164 ± 41, 1142 ± 39, and 1148 ± 34 cm s^{-1}), baPWV decreased 20 and 40 min after the WBV trial and recovered to baseline 60 min after the trial (1137 ± 28 vs. 1107 ± 30, 1108 ± 28, and 1128 ± 25 cm s^{-1}). These results suggest that WBV acutely decreases arterial stiffness.

Keywords arterial stiffness, blood pressure, heart rate, pulse wave velocity, whole-body vibration.

Whole-body vibration (WBV), which is applied to humans via a vibrating platform, has been investigated in the field of sports, space travelling, rehabilitations and treatments of osteoporosis. During WBV training, subjects stand on a platform maintaining a static position or performing dynamic exercise and are exposed to vibration stimuli. WBV is feasible not only in healthy humans but also in vulnerable populations such as elderly nursing home residents (Bautmans *et al.* 2005) and immobilized patients, such as those with osteogenesis imperfecta (Semler *et al.* 2007). Therefore, it is interesting to explore the possibility of WBV as an adjunct to exercise training. Previous studies have investigated whether WBV improves muscular strength and bone mineral density (Delecluse *et al.* 2003, Rubin *et al.* 2004, Verschueren *et al.* 2004). Also, the effects

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of WBV on cardiorespiratory function may be of interest (Rittweger *et al.* 2001, Yamada *et al.* 2005). Rittweger *et al.* (2001) have reported that WBV treatment acutely increased oxygen uptake. Additionally, Yamada *et al.* (2005) have demonstrated that blood volume in the vastus lateralis acutely increased after a WBV session. It may be possible that WBV is beneficial not only to the skeletal system and musculature but also to the cardiovascular system.

Increased arterial stiffness is an independent risk factor for the development of atherosclerosis and cardiovascular disease (Blacher *et al.* 1999, Laurent *et al.* 2001). Humans who performed aerobic exercise training on a regular basis demonstrate lower levels of arterial stiffness in comparison with sedentary peers (Cameron & Dart 1994, Kingwell *et al.* 1995, Schmidt-Trucksass *et al.* 2000, Tanaka *et al.* 2000, Otsuki *et al.* 2007a,b). Additionally, a single session of aerobic exercise training acutely decreases arterial stiffness (Kingwell *et al.* 1997, Heffernan *et al.* 2007a). Local exercise session also acutely reduces arterial stiffness (Sugawara *et al.* 2004, Heffernan *et al.* 2006). Although the mechanisms underlying the exercise-induced acute reduction of arterial tone are unclear, it may be associated with mechanical stimulus. Muscle contractions physically compress the arterial wall. It has been well known that mechanical stimuli to arteries elicit vasodilation via arterial endothelial function (Chen *et al.* 2002, Clifford *et al.* 2006). Additionally, Heffernan *et al.* (2007b) have reported that external mechanical compression of leg reduced regional arterial stiffness. As WBV-induced oscillation is propagated at least to the lumbar spine (Rubin *et al.* 2003), it is reasonable to consider that WBV mechanically stimulates abdominal and leg arteries. Taken together, WBV may reduce arterial tone and decrease arterial stiffness via mechanical stimuli to arteries.

The purpose of this study was to investigate the effects of WBV on arterial stiffness. The hypothesis of the present study was that WBV acutely reduces arterial stiffness. To test our hypothesis, subjects underwent WBV and sham control (CON) trials on separate days in randomized order and we measured brachial-ankle pulse wave velocity (baPWV), an index of arterial stiffness (Sugawara *et al.* 2005, Iemitsu *et al.* 2006). Also, haemodynamic measures such as heart rate (HR) and blood pressure were examined before and after these trials.

Methods

Subjects

Ten healthy young men (age 26.6 ± 1.9 years, height 173 ± 2 cm, weight 72.8 ± 3.9 kg) volunteered to participate in this study. As one subject could not return to the laboratory on the second day of testing, the number of subjects in the CON trial was nine. All subjects were free of signs, symptoms and history of any overt chronic disease. None of the participants had a history of smoking, and none were currently taking any medications. All subjects provided written informed consent before inclusion in the study. The experimental protocol was approved by the Review Board on Human Experiments, Kyoto Prefectural University of Medicine. This study conformed to the principles outlined in the Helsinki Declaration.

Brachial-ankle pulse wave velocity

Before all measurements, subjects refrained from intense physical activity (exercise) for 24 h and caffeine consumption for 4 h to avoid immediate (acute) effects. After a resting period of at least 20 min in a quiet and temperature-controlled room (25 °C), baPWV was measured as previously described, with minor modifications (formPWV/ABI; Omron Colin, Tokyo, Japan) (Sugawara *et al.* 2005, Iemitsu *et al.* 2006). Briefly, brachial and post-tibial artery pressure waveforms were simultaneously obtained in duplicate by the cuffs connected to a plethysmographic sensor and an oscillometric pressure sensor. The pulse wave-travelled distance from the heart to the brachial recording site (Distance A) and that from the heart to the post-tibial recording site (Distance B) were estimated from the height of subjects according to previous studies (Sugawara *et al.* 2005, Iemitsu *et al.* 2006). Time from when pulse waves reach the brachial recording site to when those reach the post-tibial recording site (T) was determined from the time delay between the brachial and post-tibial 'foot' waveforms. The foot of the wave was identified as the commencement of the sharp systolic upstroke, which was automatically detected. baPWV was calculated as the difference between Distance A and B divided by T. At the time of waveform recording, brachial arterial systolic and diastolic blood pressure (SBP and DBP respectively) and HR were also measured using oscillometry and ECG (formPWV/ABI; Omron Colin). The pressure signal obtained by plethysmography was calibrated by equating SBP and DBP, and was used to calculate mean blood pressure (MBP). These measurements were performed before and 20, 40 and 60 min after WBV/CON sessions. In our laboratory, the day-to-day coefficient of variation for baPWV at rest was 2.2 ± 1.3%.

Whole-body vibration

Two testing trials, WBV and CON (a static squat position without WBV stimulation), were performed on separate days in randomized order. The WBV and CON

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procedures were performed according to previous studies (Bosco *et al.* 2000, Goto & Takamatsu 2005), with minor modifications. In the WBV trial, subjects maintained a static squat position on the platform with a knee angle of 120° (180° at full extension) and were exposed to a WBV stimulus by using a special device producing vertical sinusoidal vibrations (Power Plate; Power Plate, London, UK). The vibration frequency and amplitude were set at 26 Hz and 2–4 mm respectively. The stimulation protocol consisted of 10 sets of vibrations for 60 s with inter-set rest periods of 60 s. During the rest periods, subjects rested on chairs. In the CON trial, they completed the same protocol as in the WBV trial, but WBV stimulation was not imposed.

Statistical analysis

Data are expressed as mean ± SE. Statistical analysis was carried out using repeated-measures two-way ANOVA followed by Fisher's PLSD test for multiple comparisons. $P < 0.05$ was accepted as significant.

Results

Table 1 summarizes blood pressure and HR before and after the CON and WBV sessions. There were no interactions (sessions × time) in SBP ($F = 1.42$), MBP ($F = 1.43$), DBP ($F = 0.57$) and pulse pressure ($F = 0.52$). Again, we did not find an interaction between sessions and time in HR ($F = 0.36$).

Brachial-ankle pulse wave velocity before and after both trials is demonstrated in Figure 1. Repeated-measures two-way ANOVA revealed an interaction between trials and time ($F = 2.86$). In multiple comparisons, baPWV was lower 20 and 40 min after the WBV session compared with the baseline, while there were no differences in baPWV before and after CON trials.

Discussion

The present study investigated baPWV following a WBV session. We demonstrated for the first time that baPWV acutely decreased 20 and 40 min after WBV stimuli, although there were no differences in blood pressure and HR between before and after the trials. The decreased baPWV returned to resting levels within 1 h of WBV cessation. These results suggest that WBV acutely decreases arterial stiffness.

Stiffness of the central elastic arteries, but not of the peripheral muscular arteries, is an independent cardiovascular risk factor. Although baPWV which involves both central and peripheral arterial stiffness is associated with cardiovascular risk (Yamashina *et al.* 2003, Imanishi *et al.* 2004), we should carefully interpret the changes in this measure of arterial stiffness. Recently, Sugawara *et al.* (2005) have demonstrated that aortic and leg pulse wave velocity were the primary independent correlates of baPWV, explaining 58% and 23% of the total variance in baPWV respectively. It is likely that aerobic exercise session at middle intensity (cycling exercise, 65% maximal oxygen uptake for 30 min) acutely reduces systemic arterial stiffness (Kingwell *et al.* 1997, Heffernan *et al.* 2007a) and that regional exercise (single-leg leg press or single-leg cycling) affects only the regional artery (Sugawara *et al.* 2004, Heffernan *et al.* 2006). As transmissibility of WBV of 26 Hz

Table 1 Blood pressure and heart rate before and after control and whole-body vibration (WBV) sessions

	Baseline	Post 20 min	Post 40 min	Post 60 min
Systolic blood pressure (mmHg)				
Control	125 ± 2	122 ± 2	123 ± 2	124 ± 3
WBV	121 ± 2	121 ± 3	118 ± 3	122 ± 2
Mean blood pressure (mmHg)				
Control	92 ± 2	91 ± 2	91 ± 2	91 ± 3
WBV	88 ± 1	89 ± 2	86 ± 2	90 ± 2
Diastolic blood pressure (mmHg)				
Control	74 ± 2	73 ± 2	73 ± 2	73 ± 2
WBV	70 ± 2	70 ± 2	68 ± 1	71 ± 2
Pulse pressure (mmHg)				
Control	51 ± 2	50 ± 1	51 ± 1	50 ± 1
WBV	51 ± 2	51 ± 2	50 ± 2	51 ± 2
Heart rate (beats min^{-1})				
Control	58 ± 2	58 ± 2	58 ± 2	59 ± 2
WBV	57 ± 2	57 ± 2	55 ± 2	57 ± 2

Values are mean ± SE.

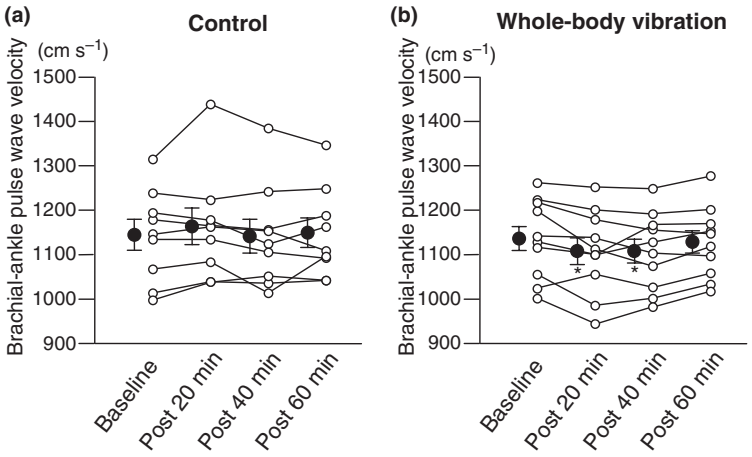

Figure 1 Brachial-ankle pulse wave velocity (baPWV), an index of arterial stiffness, before and 20, 40 and 60 min after control (a) and whole-body vibration (WBV, b) sessions. Open circles are individual values and closed circles are mean ± SE. $*P < 0.05$ vs. baseline. The interaction (sessions × time) was identified in baPWV ($F = 2.86$, $P < 0.05$). Control trial did not change baPWV, but baPWV decreased 20 and 40 min after WBV session and recovered to the baseline 60 min after the cessation of WBV stimuli.

at the lumbar spine during bent knee posture is 70–80% and greater in comparison with that at the hip (Rubin *et al.* 2003), it would be reasonable to consider that WBV-induced oscillation was propagated to the abdominal aorta. Although we could not separately measure aortic and leg arterial stiffness, the observed changes in baPWV may reflect the changes in both aortic and leg arterial stiffness.

Is WBV-related acute reduction of arterial stiffness a beneficial response? The purpose of the present study was only to determine whether WBV affects arterial stiffness and we cannot currently answer this question. However, we have some possible explanations for this issue. First, acute reductions in arterial stiffness may decrease exposure to cardiovascular risk. The Bogalusa Heart Study reported that carotid artery intima-media thickness was associated with the cumulative burden of cardiovascular risk factors (Li *et al.* 2003). Whereas the effect of a single WBV session is slight, it may be possible that long-term treatment with WBV is favourable for the cardiovascular system. Second, repetition of acute reduction in arterial stiffness may decrease baseline levels of arterial stiffness. Baseline arterial stiffness is decreased by aerobic exercise training (i.e. repetition of aerobic exercise) (Cameron & Dart 1994, Kingwell *et al.* 1995, Schmidt-Trucksass *et al.* 2000, Tanaka *et al.* 2000, Otsuki *et al.* 2007a,b) and increased by whole-body strength exercise training (Bertovic *et al.* 1999, Miyachi *et al.* 2004, Kawano *et al.* 2006, Otsuki *et al.* 2007a,b). Additionally, some study groups have reported that arterial stiffness acutely decreases after a session of aerobic exercise (Kingwell *et al.* 1997, Heffernan *et al.* 2007a) and acutely increases after a whole-body strengthening exercise session (Devan *et al.* 2005, Heffernan *et al.* 2007a). These previous studies suggest that acute and chronic responses of arterial stiffness to exercise can be common, although there are some conflicting reports. As arterial stiffness acutely decreased after the WBV trial, repetition of WBV stimulus could reduce arterial stiffness at rest. However, these are only possibilities and prospective intervention studies are needed to discuss these ideas. This study is an initial step to elucidate the effects of WBV on arterial stiffness.

Aerobic exercise training has been efficacious in the primary prevention of arterial stiffening. However, some humans cannot perform the recommended amount of aerobic exercise at adequate intensity because of physical or psychological factors. It would be beneficial to develop an adjunctive to exercise training for this population. Interestingly, even in WBV applied during standing posture, Rubin *et al.* (2004) have revealed that it could inhibit bone loss. Also, WBV has been reported to be feasible even in elderly nursing home residents (Bautmans *et al.* 2005) and immobilized patients with osteogenesis imperfecta (Semler *et al.* 2007). In this study, we demonstrated that arterial stiffness acutely decreased following a WBV session in young humans. For clinical applications of WBV, further studies including older or unhealthy humans are needed and the contraindications, such as prostheses and epilepsy, should be well examined. However, this study demonstrated the possibility of WBV as an adjunctive to exercise training. It may be worthwhile to further investigate WBV for humans who cannot sufficiently perform aerobic exercise training.

The mechanisms responsible for the reduction in arterial stiffness after a WBV session are unclear. However, it is reasonable to hypothesize that the acute reductions of arterial stiffness is associated with the arterial functional changes (i.e. relaxation of arterial smooth muscle cells) but not with the vascular organic changes. One of the possible explanations may be vascular endothelial function. Vascular endothelial cells play an important role in the regulation of vascular activity by producing vasoactive substances, such as nitric oxide. Previous studies have demonstrated that pharmacological inhibition of nitric oxide synthase

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increased arterial stiffness in humans, suggesting that nitric oxide participates in the regulation of arterial stiffness (Kinlay *et al.* 2001, Wilkinson *et al.* 2002, Sugawara *et al.* 2004). Awolesi *et al.* (1995) have reported that mechanical stretch of aortic endothelial cell increased endothelial nitric oxide synthase expression *in vitro*. Additionally, mechanical stimuli such as compression induced vasodilation in intact arteries but the dilation was attenuated after the removal of endothelium (Clifford *et al.* 2006). Also *in vivo*, vasodilation was elicited by mechanical stimuli and the administration of nitric oxide synthase blocker reduced this dilation (Chen *et al.* 2002). The mechanical influences of WBV on artery may be related to endothelial function and to the acute decreases in arterial stiffness. However, it would be difficult to sufficiently explain the acute reductions in arterial stiffness following WBV by only endothelial function. Other vasodilators and vasoconstrictors, sympathetic nerve system and changes in body temperature may be implicated in this phenomenon. Mechanisms underlying the WBV-associated acute reductions in arterial stiffness are requested to be comprehensively investigated.

The WBV procedure in this study was in line with previous studies investigating the acute hormonal responses to WBV (Bosco *et al.* 2000, Goto & Takamatsu 2005). We cannot compare the effects of this procedure on arterial stiffness to that of other procedures because this is the first study to examine WBV-related changes in arterial stiffness. As for muscular functions and bone density, other procedures have been tested and demonstrated to improve it (Delecluse *et al.* 2003, Rubin *et al.* 2004, Verschueren *et al.* 2004, Bautmans *et al.* 2005, Semler *et al.* 2007). Also, in arterial functions, there may be other efficient procedures.

In conclusion, arterial stiffness acutely decreases after WBV. We propose the possibility that WBV affects not only the skeletal system and musculature but also the cardiovascular system.

Conflict of interest

We have no financial or other relationships that might lead to a conflict of interest.

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