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The augmentation index (AIx), a marker of wave reflection, decreases following acute leg exercise. Passive vibration (PV) causes local vasodilation that may reduce AIx. This study investigated the effects of acute PV on wave reflection and aortic hemodynamics. In a crossover fashion 20 (M = 9, F = 11) healthy young (22±3 year) participants were randomized to 10 min PV or no vibration control (CON) trials. Subjects rested in the supine position with their legs over a vibration platform for the entire session. Radial waveforms were obtained by applanation tonometry before and after 3 min (Post-3) and 30 min (Post-30) of PV (~5.37 G) or CON. No change in parameters was found at Post-3. We found significant time-by-trial interactions (Po0.01) at Post-30 for augmented pressure, AIx and second systolic peak pressure (P2), such that these parameters significantly (P<0.05) decreased (-2.3±3.0 mm Hg, -7.2±6.9% and -1.5±3.5 mm Hg, respectively) after PV but not after CON. These findings suggest that acute PV applied to the legs decreases AIx owing to a decrease in wave reflection magnitude (P2). Further research is warranted to evaluate the potential clinical application of PV in populations at an increased cardiovascular risk who are unable to perform conventional exercise.

Keywords

passive vibration; hemodynamics; wave reflection magnitude; augmentation index

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Introduction

Indices of pulse wave analysis, including aortic blood pressure (BP) and augmentation index (AIx), are more sensitive predictors of cardiovascular events as well as anti-hypertensive therapeutic efficacy than brachial BP.[1–5] The AIx, a marker of wave reflection, is influenced by both the magnitude and the timing of the pressure wave reflected from peripheral sites to the aorta.[6] Accordingly, increased AIx has been associated with high rates of cardiovascular morbidity and mortality,[5,7] due to an increased left ventricular afterload.[8]

Previous research has demonstrated that the AIx reduces following an acute bout of cycling exercise[9,10] and low-intensity leg resistance exercise in healthy young individuals.[11] However, the use of traditional exercise in frail individuals or in individuals with physical disabilities, such as bedridden patients, may not be feasible. Alternatively, whole-body vibration (WBV) exercise has been proposed as a new and effective method to improve cardiovascular function in young as well as elderly populations.[12–14] WBV is performed using static or dynamic exercises on a vibrating platform, which evokes reflexive muscle contractions and ultimately a training effect.[15,16] Acute squat exercise with WBV has been shown to decrease AIx mainly owing to changes in the amplitude but not the timing of the reflected wave.[17] Similarly, passive vibration (PV), defined as exposure of the limbs to continuous vibration without performing voluntary muscle contractions, has been shown to increase arm as well as leg skin blood flow in both healthy individuals and type 2 diabetes patients.[18–20] Together these studies suggest that acute vibration per se evokes local vasodilation of peripheral arteries, which may decrease AIx and ultimately reduce left ventricular afterload. PV has a potential clinical application as a therapeutic adjuvant, particularly for individuals with disabilities who are unable to embrace regular exercise training. However, the effects of PV on wave reflection and aortic hemodynamics have not been evaluated.

Therefore, the aim of the present study was to investigate the effects of a PV session on wave reflection and aortic hemodynamics. We hypothesized that acute exposure of PV on the legs would decrease AIx driven by a reduction in the amplitude of the reflected wave.

Materials and methods

Subjects

A total of 20 (M = 9, F = 11) apparently healthy participants between the ages of 18 and 27 years volunteered for this study. All subjects were nonsmokers and either sedentary or moderately physically active (≤ h of regular exercise per week). Participants were normotensives (systolic BP (SBP) <140 mm Hg and diastolic BP (DBP) <90 mm Hg), free of cardiovascular and metabolic/endocrine diseases as evaluated by health history, and not taking medications or nutritional supplements. Female participants were tested in the early follicular phase of the menstrual cycle to avoid potential variations in pressure wave morphology.[21,22] This study was approved by the institutional review board of the Florida State University. All subjects were aware of the experimental procedures and gave written consent before data collection.

Study design

Anthropometrics and health history were assessed during the initial visit. In a crossover randomized fashion, subjects were evaluated on 2 days separated by ~48 h, during which they completed either the PV or the no-vibration control (CON) trial. The experiments were conducted in the afternoon after at least 4 h postprandial in a quiet temperature controlled room (23 ºC) and at the same time of the day (±2 h) in order to

minimize potential diurnal variations in vascular reactivity. Participants abstained from caffeine and alcohol for 12 h and avoided intense exercise 48 h before testing. After 10 min of rest, pulse wave parameters were collected at baseline, 3–5 min (Post-3) and 30–32 min (Post30) after 10 min of PV or CON trial.

Experimental and vibration protocol

Subjects were instructed to assume the supine position on an inflatable mattress with their legs over the vibration platform for the entire session. The direct exposure to vibration included the inferior third of the gluteal region to the inferior third of the lower leg with the feet outside the vibrating platform. After the baseline measurements, the participants were exposed to 10 min of PV or CON. The vibration frequency and peak-to-peak displacement were set at 25 Hz and 2.0 mm, respectively, which correspond to a peak-to-peak acceleration of ~5.37 G. The selected vibration protocol was similar to that used in previous studies that have showed significant vascular changes after WBV exercise and PV, respectively.[13,14,18] Subsequently, subjects remained in the supine position for 32 min of recovery period.

Pulse wave analysis

Brachial BP was recorded using an automated oscillometric device (HEM-705CP; Omron Healthcare, Vernon Hill, IL, USA). SBP and DBP were used to calibrate radial waveforms obtained from a 10-s epoch using a high-fidelity tonometer (SPT-301B; Millar Instruments, Houston, TX, USA).[23,24] Pulse pressure was calculated as the difference between SBP and DBP. Aortic BP waveforms were derived using a generalized validated transfer function (SphygmoCor, AtCor Medical, Sydney, Australia). The aortic BP wave is composed of a forward wave (P1), caused by stroke-volume ejection, and a reflected wave (P2) that returns to the aorta from peripheral sites.[25] The AIx was defined as the augmented pressure (AP = P2 – P1) expressed as a percentage of the aortic pulse pressure. AIx was normalized to a heart rate (HR) of 75 beats min^{-1} (AIx@75) as it is influenced by HR.[26] 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.[25] AIx and Tr have been used as markers of wave reflection and aortic stiffness, respectively.[27] HR was obtained from the time between pulse waveforms. The average of two measurements of brachial BP and high-quality (operator index ≥85%) aortic hemodynamics was used in the analysis. In our laboratory, the intraclass correlation coefficients for resting aortic SBP, aortic DBP, AIx and Tr taken on two separate days are 0.97, 0.97, 0.95 and 0.97, respectively.

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. Body mass index was calculated as kg m^{-2}.

Statistical analysis

Data are presented as mean±s.d., unless otherwise specified. Student's *t*-test was used to determine possible differences between trials at baseline in all dependent variables. Differences in mean values for each variable between groups were compared by a 2 x 3 ANOVA with repeated measures (trial (CON vs PV) by time (baseline, Post-3 and Post-30)), followed by Fisher's LSD test for pairwise comparisons. Univariate associations were analyzed with Pearson's correlation coefficients. An a *priori-α* level of <0.05 was considered to be significant. SPSS version 18.0 (SPSS Inc, Chicago, IL, USA) was used for all analyses.

Results

Subject characteristics and cardiovascular parameters

Age, height, weight and body mass index were 22.3±13.8 years, 1.66±0.18 m, 64.6±15.2 kg and 23.0±2.7 kg m^{-2}, respectively. The peripheral and central hemodynamic parameters at baseline and after the trials are shown in Table 1. There were no significant differences in baseline cardiovascular parameters between the trials. HR, brachial BP and aortic BP were unaffected by PV and CON.

Table 1 Cardiovascular parameters at baseline and after PV

Variable	*Baseline*	*Post-3*	*Post-30*
HR (beats min⁻¹)			
CON	66 ± 9	64 ± 9	65 ± 9
PV	66 ± 8	64 ± 8	63 ± 9
BSBP (mm Hg)			
CON	112 ± 9	113 ± 9	113 ± 9
PV	112 ± 9	114 ± 9	114 ± 9
BDBP (mm Hg)			
CON	64 ± 7	62 ± 6	65 ± 6
PV	63 ± 6	64 ± 6	65 ± 6
BPP (mm Hg)			
CON	48 ± 7	48 ± 7	48 ± 7
PV	49 ± 8	50 ± 8	49 ± 8
ASBP (mm Hg)			
CON	94 ± 7	94 ± 6	94 ± 7
PV	95 ± 7	95 ± 8	94 ± 7
ADBP (mm Hg)			
CON	65 ± 7	63 ± 7	66 ± 6
PV	64 ± 5	65 ± 6	66 ± 6
APP (mm Hg)			
CON	29 ± 1	31 ± 1	29 ± 1
PV	31 ± 1	30 ± 1	29 ± 1

Abbreviations: A, aortic; ADBP, aortic diastolic blood pressure; APP, aortic pulse pressure; ASBP, aortic systolic blood pressure; B, brachial; BDBP, brachial diastolic blood pressure; BPP, brachial pulse pressure; BSBP, brachial systolic blood pressure; CON, no vibration; DBP, diastolic blood pressure; HR, heart rate; PP, pulse pressure; PV, passive vibration; SBP, systolic blood pressure.
Data are means ± s.d.

Table 2 Aortic hemodynamics at baseline and after passive vibration

Variable	*Baseline*	*Post-3*	*Post-30*
AP (mm Hg)			
CON	0.6 ± 3.6	0.9 ± 4.5	0.9 ± 3.3
PV	0.4 ± 4.3	−0.4 ± 3.9	−1.9 ± 3.7*,**
AIx@75 (%)			
CON	−2.8 ± 13.3	−3.7 ± 13.5	−2.0 ± 11.9
PV	−3.5 ± 13.2	−7.7 ± 12.5	−11.8 ± 12.3*,**
Tr (ms)			
CON	152 ± 17	158 ± 7	150 ± 4
PV	157 ± 21	157 ± 5	149 ± 4
ED (ms)			
CON	320 ± 4	321 ± 25	323 ± 15
PV	321 ± 6	326 ± 24	326 ± 17
P1 (mm Hg)			
CON	92 ± 7	92 ± 7	93 ± 7
PV	93 ± 6	94 ± 7	94 ± 7

Abbreviations: AP, augmentation pressure; AIx@75, augmentation index adjusted for a HR of 75 beats min^{-1} CON, no vibration; ED, systolic ejection duration; P1, pressure of the first systolic peak; PV, passive vibration; Tr, transit time of the reflected pressure wave.
Data are means ± s.d.
*$P<0.01$, significant time effect.
**$P<0.01$, significant time-by-trial interaction.

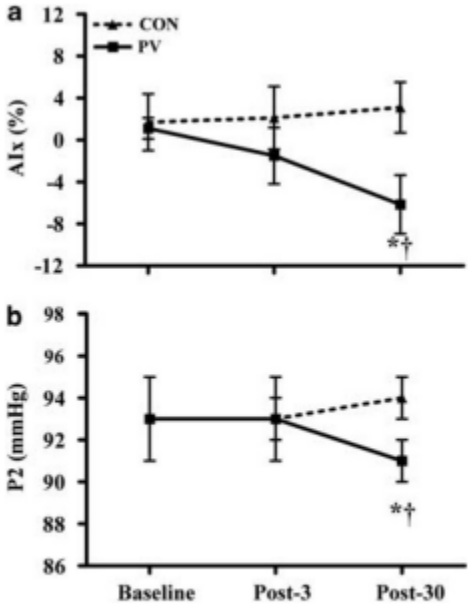

Figure 1 Changes in wave reflection at baseline and after 10 min of PV and CON trials. (**a**) AIx. (**b**) P2. Data are means ± s.e. *$P<0.01$, significant time effect; †$P<0.01$, significant time-by-trial interaction.

Pulse wave analysis

Wave reflection parameters are shown in Table 2 and Figure 1. There were significant ($P < 0.01$) trial-by-time interactions for AIx (Figure 1a), AIx@75 (Table 2), AP (Table 2) and P2 (Figure 1b). There were significant reductions ($P<0.01$) in AIx (-7.2±6.9%), AIx@75 (-8.4±7.1%), AP (-2.3±3.0 mm Hg) and P2 (-1.5±3.5 mm Hg) at Post-30 for the PV trial, while no changes from baseline were observed after the CON trial. The reductions in AIx and AIx@75 from baseline to Post-30 in the PV trial were associated with the change in P2 ($r = 0.67$, $P<0.01$ and $r =0.90$, $P<0.001$; respectively).

Discussion

We examined the impact of PV applied to the lower extremities on aortic hemodynamics. The major findings of the present study are that PV reduced AIx at Post-30, which was mediated by a reduction in the amplitude (P2) but not the HR or Tr. These findings suggest that 10 min of PV acutely decreases wave reflection and left ventricular afterload.

Previous research has demonstrated that the AIx reduces after a maximal bout of cycling (~5%) and low-intensity leg resistance exercise (-6%).[9–11] Similarly, 10 sets of static squat exercise with addition of WBV (10 min intermittent exposure to vibration) decreases AIx (~4%).[17] The post-exercise AIx reduction has been attributed to arteriolar and muscular artery vasodilation in the exercised limbs.[9,10] Indeed, repeated muscle contractions performed during physical exercise have been shown to increase the metabolic demand, leading to the production of metabolites that modulate local vascular resistance.[28] In the present study, vascular alterations were induced by means of PV without voluntary muscle contractions. We noted a 7.1% reduction in AIx that is comparable to those reported by others after single bouts of exercise longer than 10 min. As the AIx is inversely related to HR and is influenced by Tr,[26,29] it can be argued that the post-exercise reduction in AIx may be partially mediated by changes in these factors. However, as opposed to previous studies that have shown reductions in the AIx along with increases in HR 30 min after exercise,[9–11] HR and Tr did not change in response to PV, and hence, they did not influence the AIx response. As the changes in P2 and AIx were correlated and Tr was unaffected after PV, our data suggest that the post-vibration AIx response could be mainly driven by a reduction in vascular smooth muscle tone in the legs.[30,31]

The aortic pressure pulse wave is a composite of an early forward wave (P1) and a reflected wave traveling from the periphery back to the aorta (P2).[6] In cardiac patients with increased AIx, aortic SBP is determined by P2 whereas peripheral SBP is more dependent on P1.[31] Thus, the reduction in aortic P2 after administration of a vasodilator drug causes a decrease in aortic SBP.[31] In the present study, although PV evoked significant decreases in AIx and AP (-2.3 mm Hg), both aortic and brachial SBP as well as P1 did not change. As our subjects were healthy young adults and P1 was higher than P2 after PV, the aortic and brachial SBP were determined by P1. Therefore, the decrease in P2 did not affect SBP after PV.

The potential mechanisms that may explain the decrease in wave reflection in response to PV are unclear. Nevertheless, increased muscular activity and vascular shear stress in the exposed limbs may be implicated. Previous studies have shown increased reflexive muscular contractions in the limbs exposed to low-frequency vibration with exercise.[15,16] Recently, Herrero et al.[32] demonstrated that PV increased leg muscle activity and blood flow in individuals with paraplegia. These studies suggest that vascular changes associated with the vibration may be partially owing to an increased metabolic demand in the exposed muscles.[33] In addition, it appears that PV increases local blood flow by increasing endothelial nitric oxide production,[20] while reducing the circulating levels of the vasoconstrictor endothelin-1.[34] Similarly, vasodilation of peripheral arteries by exercise[9] and vasoactive drugs[35] can significantly influence wave reflection by decreasing the tone of small muscular arteries. Reduction of the amplitude of the reflected wave causes a decrease in AP and AIx, leading to a reduction in left ventricular afterload.[6,36–38]

The principal limitations of the present study are the lack of direct measurement of arterial vasodilation and vasoactive substances. However, AIx and P2 changes after acute exercise and vasoactive drug infusion are due to vasodilation of muscular arteries.[9,35] An additional limitation of the present study is that our measurements were limited to 32 min after the end of the PV, and hence we cannot determine the precise duration of the decrease in AIx. However, it appears that the peak reduction in AIx (~5%), which occurs 15–30 min after the end of an acute bout of cycling, is no longer apparent at 60 min after the exercise.[9] In addition, Otsuki et al.[13] noted that brachial-to-ankle pulse wave velocity, which includes leg arterial stiffness, remained reduced 40 min after 10 min of intermittent static exercise with WBV. Therefore, based on the magnitude of the AIx response after PV (-7.1%), we speculate that this effect may persist between 40 and 60 min after the end of the PV session. The present study examined arterial responses in young healthy individuals, and thereby the results may not be generalized to other populations.

In conclusion, these findings suggest that PV applied to the lower extremities decreases AIx, which is mediated via a reduction in the magnitude of the reflected wave. Prospective research is warranted to evaluate the potential long-term therapeutic effects of PV in vascular function in individuals with physical disabilities who are unable to perform conventional exercise.

What is known about this topic

- Acute PV increases local vasodilation.
- WBV with exercise decreases AIx

What this study adds

- Acute PV decreases AIx due to a decrease in the magnitude of the reflected wave.

- The changes in wave reflection with PV are independent of changes in HR and Tr.

Conflict of interest

The authors declare no conflict of interest.

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