Acute Effect of Interval Walking on Arterial Stiffness in Healthy Young Adults

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ABSTRACT
The purpose of this study was to determine the acute effects of interval walking (IW) on arterial stiffness. The participants in this study were 14 healthy men and women (age 27.5 ± 3.8 years). Carotid-femoral pulse wave velocity (cfPWV) was measured using an automatic oscillometric device at 30 min before (baseline) and at 30 and 60 min after walking. Participants repeated five sets of 3-min walks at 30% and 70% of maximum aerobic capacity for a total of 6 min per set in the IW trial. The participants also walked for 30 min at 50% (moderate intensity) of maximum aerobic capacity in a continuous walking (CW) trial. cfPWV was significantly decreased from baseline at 30 min (P = 0.02) after the IW trial, and this reduction in cfPWV persisted for 60 min (P = 0.01). In contrast, cfPWV was significantly decreased from baseline at 30 min (P = 0.03) after the CW trial, but the reduction did not persist for 60 min. Moreover, changes in cfPWV in the IW trial after 30 and 60 min were significantly lower than in the CW trial (P < 0.05). These results suggest that IW acutely reduces central arterial stiffness more than CW in healthy young adults.

Introduction
Increasing central arterial stiffness assessed by pulse wave velocity (PWV) is associated with increased risk for a first cardiovascular event [18]. It is well known that arterial stiffness increases with aging among adults [31]. However, it has been reported that central arterial stiffness also increases with age throughout childhood to adolescence [14, 26]. Therefore, the suppression of increases in central arterial stiffness from youth is important.

Several studies indicate that acute interval and/or accumulated aerobic exercise reduces systemic and peripheral arterial stiffness [32, 38, 39]. Tordi et al. [32] reported that six sets of 4-min cycling at 65% and 1-min cycling at 85% of predicted maximum heart rate (HR) decreased peripheral arterial stiffness in healthy young men. Mahmud and Feely [15] reported that 30 bouts of 30-s cycling at 100% of maximum aerobic capacity with a 30-s interval at 100% of maximum aerobic work between bouts decreased peripheral arterial stiffness, but not central arterial stiffness in healthy young men. Siassos et al. [27] reported that arterial wave reflections were significantly improved after moderate-intensity continuous cycling at 50% of maximum aerobic work, while there was no significant change after high-intensity interval cycling (30 bouts of 30-s cycling with a 30-s interval) at 100% of maximum aerobic work. Wilkinson et al. [36] reported that 4 x 4 min interval training at 90–95% HRmax that was performed with 3 min of active recovery periods at 70% HRmax between each interval decreased pulse wave reflection compared to moderate continuous exercise. In contrast, Zheng et al. [38] reported that two sets of 15-min cycling at 50% HR reserve (HRR) with a 20-min interval between sets decreased systemic arterial stiffness in healthy young men. Zhou et al. [39] reported that accumulated exercise in three sets of 10-min cycling at 50% HRR with a 20-min interval between sets decreased systemic arterial stiffness more than one bout of 30-min cycling. Wang et al. [35] reported that two sets of 15-min cycling at 35% HRR with a 20-min interval between sets decreased systemic arterial stiff-
ness for a longer duration compared to continuous exercise in healthy young men. Taken together, these past studies indicate that acute interval and/or accumulated aerobic exercise decrease systemic and/or peripheral arterial stiffness. However, these studies used cycling as part of exercise training programs at exercise facilities to examine changes in arterial stiffness after interval and/or accumulated aerobic exercise. Moreover, the rest periods between bouts of exercise increase the duration until accumulated exercise is completed. Therefore, a more efficient strategy than conventional exercise programs is needed for improving cardiovascular health. One possible strategy is walking.

Walking with repeated periods of fast and slow walking speeds is called interval walking (IW). IW comprises walking at ≥ 70% peak aerobic capacity for 3 min followed by 3 min of walking at ≤ 40% peak aerobic capacity; therefore, IW may be considered moderate-intensity exercise, which reduces blood pressure (BP) and/or lifestyle-related risk factors in middle-aged and older adults [16,17,19,21]. Because moderate-intensity aerobic exercise reduces central arterial stiffness, IW might reduce central arterial stiffness and have important clinical implications for improvement and maintenance of cardiovascular health. Moreover, since walking is generally a normal part of an individual’s day and does not require expensive exercise equipment, IW should be simple to include among activities of daily living. Therefore, many people can likely use IW as a form of exercise. However, the effect of IW on arterial stiffness is unknown.

The present study investigates whether IW reduces arterial stiffness in healthy young adults. We hypothesized that IW would reduce central arterial stiffness in healthy young adults.

Methods

Subjects

The participants in this study were 14 healthy, non-obese men (n = 5) and women (n = 9) who were not actively involved in regular physical exercise. All participants had optimal BP (systolic BP [SBP]/diastolic BP [DBP] < 120/80 mmHg). Table 1 shows the physical characteristics of the participants. Based on the guidelines of the International Society of Hypertension, optimal BP was determined as SBP/DBP < 120/80 mmHg [3]. Participants were excluded from the study if they were obese, smoked, had cardiovascular disease, diabetes, menstrual irregularity, amenorrhea, or were taking anti-hyperlipidemic, anti-hypertensive, anti-hyperglycemic medications or oral contraceptives. The medical history of each participant was confirmed by the authors in the screening before the experiment. Some of the participants had engaged in regular exercise in the past. However, participants had not engaged in regular physical activity for the previous 2 years. Moreover, none of the participants were currently performing any type of physical activity, including either resistance or aerobic exercises. Physical activity was assessed using the short form of the International Physical Activity Questionnaire (IPAQ). The physical characteristics and physical activity of participants were measured by the same investigator, who was blinded to the group assignment of the participants. Written, informed consent was obtained from all participants after they were given a complete explanation about the purpose of the study and the experimental procedures. The Ethics Committee of Nippon Sport Science University approved this study, which proceeded in accordance with the guidelines for experimental studies involving human participants published by our Institutional Review Board. The study conformed to the principles outlined in the ethical standards of the International Journal of Sports Medicine [9]. This study was conducted in accordance with the Declaration of Helsinki.

Measurement

Prior to the experiments, participants rested in a supine position for 20 min at a constant room temperature (23–25 °C) in a quiet room. After the 20-min rest, carotid-femoral PWV (cfPWV), femoral-ankle PWV (faPWV), carotid and brachial BP, carotid augmentation index (AIx) and HR were simultaneously measured using a vascular testing device (form PWV/ABI; Omron-Colin Co., Ltd., Tokyo, Japan) in the same environment used for the rest period. These were measured with established measurement methods [28]. Electrocardiograms were recorded using electrodes placed on the left and right wrists, heart sounds were monitored using a microphone placed on the sternum, and arterial pressure waveform was recorded using sensory cuffs that were wrapped around both brachia and the ankles. All measurement and recording devices were attached while participants were in the supine position.

The applanation tonometers were subsequently attached in order of the femoral arteries and carotid arteries. cfPWV, faPWV, carotid and brachial BP, AIx and HR were obtained before, and at 30 and 60 min after the IW and continuous walking (CW) trials. To avoid potential diurnal variation, the participants were tested at the same time of the day throughout the study period. The participants abstained from intense physical activity within 48 h of the study. Moreover, participants abstained from caffeine for at least 4 h and fasted for at least 4 h. Since this study employed adult women as participants, we considered the possibility that menstrual cycles would affect changes in PWV. Therefore, all female participants were studied during the early follicular phase of the cycle at intervals of about 1 month to avoid any hormonal influences. All parameters were measured by the same investigator, who was blinded to the group assignment of the participants.

Body composition

Body composition was determined using bioelectric impedance (Inbody, Biospace Co. Ltd., Seoul, Korea).

Table 1

<table>
<thead>
<tr>
<th>Physical characteristics of subject (n = 14)</th>
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<tbody>
<tr>
<td>Age (years)</td>
</tr>
<tr>
<td>Height (cm)</td>
</tr>
<tr>
<td>Weight (kg)</td>
</tr>
<tr>
<td>Body fat (%)</td>
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<tr>
<td>BMI (kg/m²)</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
</tr>
<tr>
<td>Systolic BP (mmHg)</td>
</tr>
<tr>
<td>Diastolic BP (mmHg)</td>
</tr>
<tr>
<td>VO₂max (mL/kg/min)</td>
</tr>
</tbody>
</table>

Values are means ± SE; BMI: body mass index, BP: blood pressure.
Pulse wave velocity

cfPWV and faPWV, which reflect aortic and leg arterial stiffness, respectively, were measured as previously described [23, 37]. Carotid and femoral artery pressure waveforms were obtained for 30 s using arterial applanation tonometry incorporating form PWV/ABI micro piezo resistive transducers (Omron-Colin Co., Ltd.), which comprise 15 aligned pressure-sensitive elements that can identify carotid and femoral pulse traces, arranged side-by-side and attached to the left common carotid and femoral arteries, respectively. A vascular testing device (form PWV/ABI; Omron-Colin Co., Ltd.) was then used to simultaneously measure electrocardiograms, bilateral brachial and ankle BPs, and carotid and femoral arterial pulse waves. cfPWV and faPWV were calculated by dividing the distance between the two arterial recording sites by the transit time, which was determined based on the time delay between the proximal and distal “foot” waveforms. faPWV was calculated based on the transit time between the femoral artery site and the ankle site. Next, time delays were obtained from between the right brachial and post-tibial arteries, the carotid and femoral arteries (Tcf), and the femoral and post-tibial arteries. A nonelastic tape measure was then used to make duplicate random zero length measurements over the body surface to determine the path length from the carotid to the femoral artery (Dcf). cfPWV was then calculated using the following equation:

$$\text{cfPWV} = \frac{D_{cf}}{T_{cf}}$$

Carotid AIx

Carotid AIx was measured simultaneously with PWV, BP and HR. Carotid arterial pressure waveforms were recorded from the common carotid artery using an applanation tonometry sensor (form PWV/ABI; Omron-Colin Co., Ltd.). A neck collar device was used to secure and stabilize the applanation tonometry sensor (form PWV/ABI; Omron-Colin Co., Ltd.). The carotid AIx was calculated as the pressure wave above the systolic shoulder divided by the pulse pressure, i.e.:

$$\text{AIx} (\%) = \left( \frac{\Delta P}{PP} \right) \times 100,$$

where $\Delta P$ is the pressure difference between the peak systolic pressure and an early inflection point that indicates the beginning upstroke of the reflected pressure wave, and PP is the pulse pressure.

The systolic foot and shoulder of carotid arterial pressure waveforms were automatically detected using algorithms of the measurement device based on band-pass filtering (5–30 Hz) and fourth-order derivatives, respectively [28, 29]. AIx was adjusted for a heart rate of 75 bpm (AIx@75).

Carotid and brachial BP and HR

After resting in the supine position for at least 20 min, carotid BP was determined using an applanation tonometer (form PWV/ABI, Omron–Colin Co. Ltd.) secured to the left common carotid artery by a neck collar, and was calibrated by equating its mean arterial and diastolic BP values to those of the brachial artery. Normal resting levels of BP and HR at rest in the supine position were measured in triplicate using an automated oscillometric device (form PWV/ABI, Omron-Colin Co. Ltd.) over the brachial and dorsalis pedis arteries. The pressure signal obtained using a volume-plethysmographic apparatus was calibrated by equating SBP and DBP values and then used to calculate mean arterial pressure.

Maximum aerobic capacity

$\dot{V}_O_2$\text{max} was assessed breath-by-breath during an incremental walking exercise test (Bruce protocol [2]). $\dot{V}_O_2$\text{max} was determined 1 week before the IW or CW trials using a motor-driven treadmill (BiOMILL BM-1200, S&ME, Inc., Tokyo, Japan). Volumes of O$_2$ and CO$_2$ were continuously measured by open-circuit spirometry and analyzed using a metabolic measurement cart (AE-100; Minato Medical Science Co., Ltd., Kyoto, Japan). The Bruce protocol included 3-min stages, with the first starting at a 10% gradient and a speed of 2.7 km/h. At the end of each stage the gradient increased by 2% and speed was increased to 4.0, 5.5, 6.8, 8.0, and 8.8 km/h during subsequent stages. $\dot{V}_O_2$\text{max} was identified as the average of the two highest 15-s $\dot{V}_O_2$ values occurring in the last 30 s of incremental exercise. All participants exhibited a leveling off of variables, displaying a phenomenon represented by a plateau in $\dot{V}_O_2$. HR was monitored using electrocardiography (Life Scope (BSM-2400; Nihon Kohden, Co., Ltd., Tokyo, Japan). The criterion used to assess $\dot{V}_O_2$\text{max} included a respiratory exchange ratio of 1.10, HR > 90% of age-predicted HR maximum (220 – age), and a plateau (150 ml·min$^{-1}$ increase) in $\dot{V}_O_2$\text{max} despite a further increase in velocity. The maximum respiratory exchange ratio was 1.18 ± 0.4, the percent of predicted maximum HR was 93 ± 3%, and the $\dot{V}_O_2$ plateau was 36.3 ± 3.8 ml/kg/min.

Walking

The IW or CW trials were performed using the same motor-driven treadmill (BIOMILL BM-1200, S&ME, Inc). The IW trial consisted of five sets of 3-min walks at 30% (slow walking) and 70% (fast walking) of $\dot{V}_O_2$\text{max} for a total of 6 min per set. The CW trial consisted of a 30-min walk at 50% (moderate walking) of $\dot{V}_O_2$\text{max}. The IW trial was performed based on methods used in previous studies [16, 17, 19]. Briefly, the IW trial consisted of 3 min of low-intensity walking at 30% of $\dot{V}_O_2$\text{max} followed by 3 min of high-intensity walking at 70% of $\dot{V}_O_2$\text{max}. These changes in walking intensity were repeated for a total IW trial time of 30 min. The 30-min CW trial was performed by maintaining 50% of $\dot{V}_O_2$\text{max}. A metabolic measurement cart (AE-100; Minato Medical Science Co., Ltd.) was used to monitor oxygen uptake (i.e., exercise intensity) during the IW and CW trials. If required, the walking speed was manually adjusted to maintain the target exercise intensity. The IW and CW trials were conducted in a random order on separate days at intervals of approximately 1 month. HR and ratings of perceived exertion data were obtained during the 30-min IW and CW trials. HR during the IW and CW trials was monitored using electrocardiography (Life Scope BSM-2400; Nihon Kohden, Co., Ltd.). Percent HR values for the IW trial were 49 ± 5% at 30% of $\dot{V}_O_2$\text{max} (slow walking) and 63 ± 3% at 70% of $\dot{V}_O_2$\text{max} (fast walking). Percent HR values for the CW trial were 55 ± 1% at 50% of $\dot{V}_O_2$\text{max} (moderate walking). The average walking speeds for the IW trial were 3.4 (2.8–5.0) km/h at 30% of $\dot{V}_O_2$\text{max} (slow walking) and 7.1 (6.4–8.5) km/h at 70% of $\dot{V}_O_2$\text{max} (fast walking). The average walking speed for the CW trial was 5.7 (5.0–6.8) km/h at 50% of $\dot{V}_O_2$\text{max} (moderate walking). Walking speed (exercise intensity) during the IW and CW trials was determined based on $\dot{V}_O_2$\text{max}.
Physiology & Biochemistry

**Results**

Fig. 1 shows a comparison of the average oxygen uptake and HR between the IW and CW trials. There were no significant differences between the IW and CW trials for the average oxygen uptake and HR. However, the average oxygen uptake and HR during fast walking (at 70% of VO₂max) were significantly higher than those during slow (at 30% of VO₂max) and moderate walking (at 50% of VO₂max) (P<0.001). In addition, the average oxygen uptake and HR during moderate walking (at 50% of VO₂max) were significantly higher than those during slow walking (at 30% of VO₂max) (P<0.001).

Fig. 2 shows the changes in cfPWV before and at 30 and 60 min after the IW and CW trials. No significant differences were seen in cfPWV between the two trials at baseline. cfPWV was significantly decreased from baseline at 30 min (P=0.01) after the IW trial and the decrease persisted for 60 min (P=0.02). In contrast, cfPWV was significantly decreased from baseline at 30 min (P=0.03) after the CW trial, but the decrease did not persist for 60 min. Although cfPWV in both trials was not significantly different after 30 min (P=0.06), cfPWV in the IW trial after 60 min was significantly lower than in the CW trial (P=0.01).

cfPWV was decreased from baseline by 12±9% (Δ86±63 cm/s) and 13±10% (Δ92±77 cm/s) at 30 and 60 min after the IW trial. On the other hand, cfPWV was decreased from baseline by 6±8% (Δ45±55 cm/s) and 5±7% (Δ38±54 cm/s) at 30 and 60 min after the CW trial. There were significant differences in percent changes in cfPWV between the IW and CW trials after 30 (P=0.04) and 60 min (P=0.02).

Table 2 shows the changes in faPWV, carotid and brachial BP, carotid AIx and HR before and at 30 and 60 min after the IW and CW trials. faPWV was significantly decreased from baseline at 30 min (IW: P=0.02, CW: P=0.04) after both trials, and the decrease persisted for 60 min (IW: P=0.01, CW: P=0.03). Carotid AIx was significantly decreased from baseline at 30 min (P=0.003) after the IW trial and the decrease persisted for 60 min (P=0.007). In contrast, carotid AIx was significantly decreased from baseline at 30 min (P=0.01) after the CW trial, but the decrease did not persist for 60 min. No significant differences were observed in carotid and brachial SBP, brachial DBP, mean arterial pressure (MAP), pulse pressure (PP) and HR from baseline to any time point after the IW and CW trials.

**Discussion**

The key finding of this study was that although central and peripheral arterial stiffness was significantly reduced after both IW and CW, central arterial stiffness remained lower than baseline until 60 min after IW and the magnitude of the decreases in central arterial stiffness from IW was greater than that from CW. These results suggest that walking with repeated slow speed and fast speed
Table 2 Changes in faPWV, carotid and brachial BP, carotid Alx and HR.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Before</th>
<th>30 min after</th>
<th>60 min after</th>
</tr>
</thead>
<tbody>
<tr>
<td>faPWV (cm/sec)</td>
<td>853 ± 25</td>
<td>797 ± 26 *</td>
<td>767 ± 24 *, †</td>
</tr>
<tr>
<td>Brachial SBP (mmHg)</td>
<td>860 ± 19</td>
<td>810 ± 21 *</td>
<td>795 ± 21 *</td>
</tr>
<tr>
<td>IW</td>
<td>108 ± 2</td>
<td>107 ± 2</td>
<td>108 ± 2</td>
</tr>
<tr>
<td>CW</td>
<td>108 ± 3</td>
<td>107 ± 2</td>
<td>109 ± 2</td>
</tr>
<tr>
<td>Brachial MAP (mmHg)</td>
<td>63 ± 2</td>
<td>64 ± 1</td>
<td>65 ± 2</td>
</tr>
<tr>
<td>IW</td>
<td>45 ± 2</td>
<td>43 ± 2</td>
<td>44 ± 1</td>
</tr>
<tr>
<td>CW</td>
<td>46 ± 2</td>
<td>44 ± 1</td>
<td>43 ± 2</td>
</tr>
<tr>
<td>Carotid SBP (mmHg)</td>
<td>114 ± 2</td>
<td>113 ± 3</td>
<td>111 ± 3</td>
</tr>
<tr>
<td>IW</td>
<td>114 ± 2</td>
<td>114 ± 2</td>
<td>111 ± 2</td>
</tr>
<tr>
<td>Carotid Alx@75 (%)</td>
<td>4.7 ± 3.1</td>
<td>-17.5 ± 3.8 *</td>
<td>-14.9 ± 2.0 *, †</td>
</tr>
<tr>
<td>CW</td>
<td>4.4 ± 3.4</td>
<td>-11.5 ± 2.8 *</td>
<td>-2.6 ± 2.2</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>56 ± 1</td>
<td>59 ± 2</td>
<td>58 ± 2</td>
</tr>
<tr>
<td>IW</td>
<td>58 ± 1</td>
<td>57 ± 2</td>
<td>58 ± 1</td>
</tr>
</tbody>
</table>

Values are means ± SE, IW: interval walking, CW: continuous walking, faPWV: femoral-ankle pulse wave velocity, SBP: systolic blood pressure, MAP: mean arterial pressure, DBP: diastolic blood pressure, PP: pulse pressure, Alx@75: augmentation index adjusted for 75 beats/min, HR: heart rate; *: significantly (P < 0.05) different from baseline. †: significantly (P < 0.05) different from the CW.

maintained decreases in central arterial stiffness a for longer duration than walking at a constant speed.

Some studies reported the effects of accumulated exercise on arterial stiffness. Zheng et al. [38] reported that accumulated exercise (2 × 15-min cycling) with a 20-min interval between sets reduces systemic arterial stiffness compared to continuous exercise (30-min cycling) in healthy young men. Zhou et al. [39] reported that accumulated exercise (3 × 10-min cycling) with a 10-min interval between sets can lead to greater reduction in systemic arterial stiffness than continuous exercise (30-min cycling) in healthy young men. Thus, accumulated exercise including a rest between exercise sets reduces systemic arterial stiffness as compared with continuous exercise [38, 39]. Tordi et al. [32] reported that interval cycling exercise consisting of six consecutive periods of 5 min of a base of 4-min duration (approximately 65 % of the predicted maximum HR) and a peak of 1-min duration (approximately 85 % of the predicted maximum HR) reduces leg arterial stiffness more than continuous exercise in healthy young adults. In contrast, Nichols et al. [22] reported that three sets of 20-s cycling at maximum exercise intensity with a 2-min interval between sets improved endothelial function, whereas the central arterial stiffness was not significantly affected. In addition, Siasos et al. [33] reported that moderate-intensity continuous exercise and high-intensity interval exercise significantly improved the flow-mediated dilation of brachial artery and that femoral dorsalis pedis PWV was improved after both exercises, but not central arterial stiffness in healthy young men. Hasegawa et al. [10] reported that sprint interval cycling exercise of four standard Wingate protocols interspersed with recovery intervals of 4.5 min of low cadence (50 rpm) cycling against a resistance of 40 W decreased peripheral arterial stiffness, whereas central arterial stiffness did not change from baseline resting levels at 30 and 60 min after exercise in healthy young adults. Thus, interval exercise at an intensity that is too high might not have a favorable effect on central arterial stiffness. However, we found that IW reduces peripheral arterial stiffness as well as central arterial stiffness. These results suggest that exercise like IW without rest might be effective for reducing arterial stiffness similar to previous studies [38, 39].

The potential mechanism by which IW reduces central arterial stiffness might be associated with the changes in exercise intensity during IW. Acute moderate-intensity aerobic exercise has been shown to increase blood flow and significantly improve vascular endothelial function in healthy adults [1, 7, 13, 24, 25, 33]. Increased arterial flow [1, 7, 13] elicits significant vasodilation [4, 5], which is regulated by vascular endothelial function. The rapid increase in blood flow that takes place during exercise generates powerful shear stress on the walls of blood vessels [34]. Enhanced expression of endothelial nitric oxide (NO) synthase by vascular endothelial cells in response to the shear stress leads to increased production of NO, a molecule that plays an important role in vasodilation [38]. Thus, shear stress on endothelial cells is a potent stimulus for NO production. It is expected that the blood flow will increase during IW when the pace is switched from slow walking (low intensity) to fast walking (high intensity). It has been suggested that the higher cardiac output as a result of the increase in HR during exercise induces a greater arterial dilation because of the elevated blood flow [11]. In fact, the HR during fast walking in this study was significantly higher than that during slow and moderate walking. The repeated elevations in shear stress during exercise might decrease the stiffness of large elastic arteries, thereby increasing the elasticity of the arterial wall and decreasing vascular tone [8]. It has been suggested that the reduction in central arterial stiffness by interval exercise is caused by an increase in aortic NO bioavailability [10]. That is, higher exercise intensities applied in intermittent bouts of IW might be effective in decreasing PWV compared to CW.

It is known that aerobic exercise acutely reduces Alx [12]. Consistent with previous findings, Alx was reduced at 30 and 60 min after IW trials [12, 20]. The reduction in Alx is associated with an increase in peripheral vasodilatation [15], caused by a decrease in the wave reflection at medium-sized muscular arteries [36]. Siasos et al. [27] reported that although Alx was improved after moderate-intensity continuous cycling at 50 % of maximum aerobic work, 30 bouts of 30-s cycling at 100 % of maximum aerobic work with a 30-s interval had no significant impact on Alx. In contrast, Compton et al. [6] reported that fast walking at 125 steps/min increased mean blood flow and the shear rate in the superficial femoral artery compared to slow walking at 80 steps/min, and significant dilation in the femoral artery was observed at 30 and 60 min after walking. Therefore, it is possible to consider that decreased Alx at 30 and
60 min after the IW trial may be caused by peripheral vasodilation. Moreover, the results of measurements of arterial stiffness obtained from noninvasive pressure waveforms suggested that the decrease in aortic stiffness is associated with a reduction in Aix [22]. Thus, our findings suggest that changes in arterial stiffness after IW primarily resulted from changes in arterial distension.

The present findings have potential clinical implications. While exercise acutely reduces cardiovascular risk factors, transient reductions in these risk factors may not affect the response to subsequent bouts of exercise. Thompson et al. [30] reported, however, that residual effects of acute exercise may accrue in a cumulative manner. In the present study, arterial stiffness decreased at 30 min after IW compared with baseline, and persisted for 60 min, and that reduction was greater than the reduction after CW. Thus, because repeated acute reductions in arterial stiffness may decrease baseline levels of arterial stiffness, the acute reduction of arterial stiffness due to IW might benefit cardiovascular health. Moreover, because IW does not include rest during continuous exercise, it can be done in a short time compared to accumulated exercise that includes a rest period. The present results require prospective confirmation in an intervention study.

In conclusion, one bout of IW caused a greater acute decrease in central arterial stiffness for longer duration than CW in healthy young adults. These results suggest that IW may be effective as an exercise prescription for maintenance of cardiovascular health.

Conflict of Interest

The authors declare no conflict of interest.

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