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ORIGINAL ARTICLE

Acute mental stress-caused arterial stiffening can be counteracted by brief aerobic exercise

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Abstract

Purpose Acute mental stress (MS) causes an elevation in pulse wave velocity (PWV), an index of arterial stiffness. In contrast, aerobic exercise acutely decreases arterial stiffness, even in the short term. The present study aimed to examine whether acute MS-caused arterial stiffening can be counteracted by brief aerobic exercise.

Methods Thirteen young healthy men (mean age, 20 ± 1 years) participated in two randomized experimental visits where they were subjected to acute MS followed by seated rest (RE) or cycling exercise (EX) trials. Following a 5-min MS task, the participants in the RE trial rested on a chair for 10 min (from 10 to 20 min after the cessation of the task), whereas those in the EX trial cycled at 35% of heart rate reserve for the same duration. Heart-brachial PWV (hbPWV), brachial-ankle PWV (baPWV), heart-ankle PWV (haPWV), and the cardio-ankle vascular index (CAVI) were simultaneously measured at baseline and 5, 30, and 45 min after the task.

Results Both trials caused significant elevations ($P < 0.05$) in hbPWV, haPWV, and CAVI at 5 min after the task; subsequently, this persisted until 45 min after the task in the RE trial, whereas the elevations in the EX trial were eliminated. In the RE trial, baPWV significantly increased ($P < 0.05$) at 30 and 45 min after the task, whereas such an increase was not observed in the EX trial.

Conclusion The findings of the present study reveal that brief aerobic exercise counteracts arterial stiffening caused by acute MS.

Keywords Arterial stiffness · Pulse wave velocity · Cardio-ankle vascular index · Cycling exercise · Endothelial function

Abbreviations

ANOVA	Analysis of variance
baPWV	Brachial-ankle pulse wave velocity
BP	Blood pressure
CAVI	Cardio-ankle vascular index
cfPWV	Carotid-femoral pulse wave velocity
ECG	Electrocardiogram
EX	Acute mental stress followed by cycling exercise
haPWV	Heart-ankle pulse wave velocity
hbPWV	Heart-brachial pulse wave velocity
HR	Heart rate
HRR	Heart rate reserve
MAP	Mean arterial pressure
MS	Mental stress
NS	Not significant
PWV	Pulse wave velocity
RE	Acute mental stress followed by seated rest

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Introduction

Arterial stiffening is an independent risk factor for cardiovascular disease (Mitchell et al. 2010; van Sloten et al. 2014), and pulse wave velocity (PWV) is an established

index of arterial stiffness. Mental stress (MS), which is often experienced in daily life, is known to impact arterial stiffness. Observational cross-sectional studies demonstrated that chronic MS is positively associated with arterial stiffness (Kaewboonchoo et al. 2018; Utsugi et al. 2009). In addition, arterial stiffness is transiently affected by an acute brief episode of MS. Vlachopoulos et al. (2006) reported that acute MS, induced by mental arithmetic, elicited an elevation in carotid-femoral PWV (cfPWV), an index of aortic stiffness, and this negative vascular effect persisted for 60 min. Recently, we reported that the acute MS-caused elevation in PWV was seen in various arterial segments, which was sustained for at least 30 min (Kume et al. 2020). These findings indicate that acute MS evokes a systemic and persistent arterial stiffening. An exaggerated elevation in blood pressure (BP) during acute MS and a delayed BP recovery after the stress are associated with an increased risk of cardiovascular disease (Chida and Steptoe 2010; Matthews et al. 2004), suggesting that both acute and chronic cardiovascular responses to MS are probably relevant. Thus, repeated exposure to increased arterial stiffness, due to acute MS in daily life, may contribute to persistent arterial stiffening. For maintenance of vascular health, it is important to devise an effective strategy to counteract the detrimental vascular effects of daily stress.

The physiological mechanisms underpinning the increase in arterial stiffness after acute MS are poorly understood. However, we and others (Kume et al. 2020; Vlachopoulos et al. 2006) have speculated that an impairment of endothelial function resulting from acute MS (Ghiadoni et al. 2000; Sales et al. 2014; Spieker et al. 2002) is key, because arterial stiffness is largely regulated by endothelial function (McEniery et al. 2006). Sales et al. (2014) reported that a 50-min exercise session after acute MS prevented endothelial dysfunction, which intrinsically occurred at 90 min after the stress. Such an exercise approach is tempting from the viewpoint of maintenance of vascular health. However, whether exercise can counteract the increased arterial stiffness after acute MS remains unclear to date. Low-to-moderate-intensity aerobic exercise is known to acutely decrease arterial stiffness measures (Kingwell et al. 1997; Okamoto et al. 2018; Wang et al. 2014; Zhou et al. 2015). Interestingly, this decrease is even induced by exercise for short-term (10–15 min) (Wang et al. 2014; Zhou et al. 2015). Therefore, brief aerobic exercise may be effective to offset arterial stiffening resulting from acute MS, and if so, it could potentially be applied as a feasible means to protect the vasculatures from daily stress.

Accordingly, the present study aimed to examine the effect of brief aerobic exercise on arterial stiffening caused by acute MS. Specifically, arterial stiffness was assessed before and after acute MS where 10 min of low-intensity cycling exercise was incorporated after the stress, and the values were then compared with those where the exercise was not performed after the stress. We hypothesized that acute MS-caused arterial stiffening would be counteracted by brief aerobic exercise.

Methods

Participants

Thirteen young healthy men participated in the study. Physical characteristics were as follows: age 20 ± 1 years; height 169 ± 6 cm; and body mass 65 ± 6 kg (means ± standard deviation). The sample size was determined by power calculations using $G \times$ Power 3 and assumed that arterial stiffness would change by 6% based on our and other previous findings (Kume et al. 2020; Nishiwaki et al. 2020; Vlachopoulos et al. 2006). To detect the differences with 80% power and a two-tailed α of 5%, the experiment required a minimum of 13 participants. None of the participants were smokers or taking medication. The purpose, experimental procedure, and risks connected to the study were fully explained to all participants, who provided written informed consent. The study was approved by the Ethics Committee of Okinawa University (#2019-01) and was conducted in accordance with the guidelines of the Declaration of Helsinki.

Experimental procedures

The participants visited the laboratory three times throughout the experimental period. During the first visit, the participants were familiarised with the experimental apparatus. Subsequently, following a 5-min resting heart rate (HR) measurement while seated on a chair, they engaged in a submaximal incremental exercise test using an electromagnetically braked cycle ergometer (Aerobike 75XLIII; Combi Wellness, Tokyo, Japan) where cadence was maintained at 60 rpm. During the exercise test, the HR was recorded to obtain workload-HR data. The resting HR value and workload-HR data were used to determine the exercise workload for subsequent experimental visit.

The schematic representation of the experimental protocol of this study is presented in Fig. 1. The order of the experimental visits was randomly administered, and consecutive visits were separated by approximately one week. During the two experimental visits, the participants were subjected to acute MS followed by seated rest (RE) or cycling exercise (EX) trials. All experiments were conducted in a quiet air-conditioned room (23–25 ℃). For each participant, the experiments were conducted at the same time of the day to avoid any potential diurnal effects. Furthermore, the participants were asked to refrain from doing strenuous

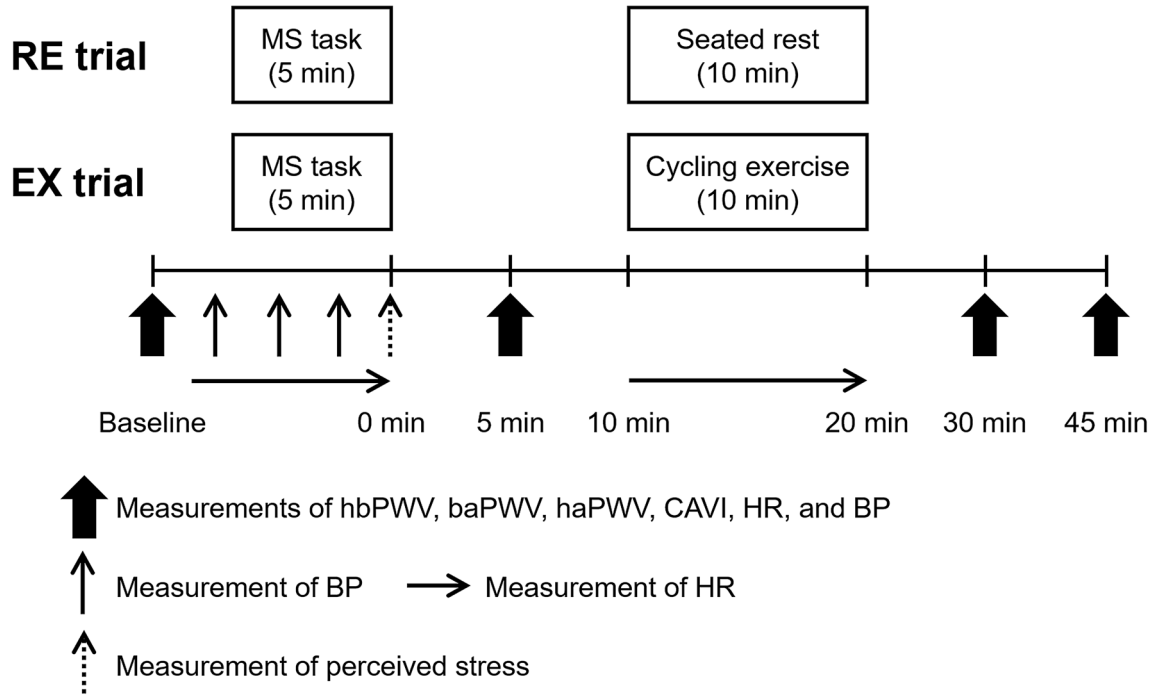

Fig. 1 Experimental protocol. RE and EX, acute mental stress followed by seated rest and cycling exercise, respectively; *MS* mental stress, *hbPWV* heart-brachial pulse wave velocity, *baPWV* brachial-ankle pulse wave velocity, *haPWV* heart-ankle pulse wave velocity, *CAVI* cardio-ankle vascular index, *HR* heart rate, *BP* blood pressure

exercise, consuming alcohol (≥ 24 h) and caffeine (≥ 12 h) and, eating (≥ 3 h) before the trials.

After resting in the supine position for at least 15 min, baseline measures of arterial stiffness and hemodynamic variables were obtained. Subsequently, the participants performed a 5-min MS task and a post-task phase was set to 45 min. The participants in the RE trial rested on a chair for 10 min (from 10 to 20 min after the cessation of the task), whereas those in the EX trial cycled at 35% of HR reserve (HRR) for the same duration. This is considered as light-intensity aerobic exercise (Garber et al. 2011) and was chosen because of the potential high applicability to daily life. The exercise was started at the predetermined workload while monitoring HR using a three-lead electrocardiogram (ECG) (413; Intercross, Tokyo, Japan), and the workload was adjusted manually, if necessary, to maintain the targeted HR value (Wang et al. 2014). The participants were kept in the supine position throughout the task and post-task, other than the seated rest or exercise period.

Mental arithmetic was used as the MS task according to our previous study (Kume et al. 2020). Specifically, each participant was asked to serially subtract 13 from a 3-digit number (close to 1000), as quickly and accurately as possible for a period of 5 min. During the task, the participants were intentionally frustrated by being asked to perform faster and by being corrected immediately if wrong answers were provided. A metronome was played loudly for additional distraction. When the number was < 13 (the answer was not allowed to go below 0), the participants restarted the task using the original 3-digit number.

Measurements

Each variable was measured as shown in Fig. 1 and all measurements were performed by the same investigator. In addition to the baseline measures, PWVs, HR, systolic and diastolic BP, and mean arterial pressure (MAP) were measured at 5, 30, and 45 min after the MS task using a vascular testing system (VaSera VS-1500AN; Fukuda Denshi, Tokyo, Japan). For the measurements, BP cuffs were wrapped around both upper arms and ankles. A phonocardiograph and ECG electrodes were also placed on the pectoral region and on both wrists, respectively. ECG, heart sounds, and arterial pressure waveforms at the brachial and posterior-tibial arteries were simultaneously recorded. We assessed heart-brachial PWV (hbPWV), brachial-ankle PWV (baPWV), and heart-ankle PWV (haPWV) as index of arterial stiffness, according to our previous study (Kume et al. 2020). Specifically, hbPWV, baPWV, and haPWV were calculated from each arterial path length, along with the time intervals between the second heart sound and the dicrotic notch on the brachial arterial pressure waveform, between the foot of the brachial arterial pressure waveform and the foot of the posterior-tibial arterial waveform, and the sum

of these time intervals (Nishiwaki et al. 2017; Sugawara et al. 2019; Tomoto et al. 2017). Furthermore, the cardio-ankle vascular index (CAVI) was automatically calculated. In our laboratory, the day-to-day coefficients of variation for hbPWV, baPWV, haPWV, and CAVI were 2.3 ± 2.1%, 1.6 ± 1.1%, 1.6 ± 1.4%, and 2.7 ± 2.4%, respectively.

Before and during the MS task, systolic and diastolic BP, and MAP were measured using an automated sphygmomanometer (Tango+; SunTech Medical Instruments, North Carolina, USA). The BP measurement was carried out twice (approximately 2 and 4 min after the beginning of the task) during the task, and the average value was calculated. HR was also measured continuously using the same ECG system. The HR data at the same time as the BP measurement were averaged. Further, immediately after the MS task, the participants were asked to rate their perceived stress during the task using a standard five-point scale of 0 (not stressful), 1 (somewhat stressful), 2 (stressful), 3 (very stressful), and 4 (very, very stressful) (Callister et al. 1992).

HR during the 10-min cycling exercise in the EX trial was recorded continuously using the same device; it was also obtained during the seated rest in the RE trial. The HR data during last 5 min in each trial were averaged because of the unstable states at the beginning of exercise in the EX trial.

Statistics

Data are expressed as means ± standard deviation. Perceived stress level during the MS task between the trials were compared using a paired Student's *t* test. Two-way (time × trial) repeated-measures analysis of variance (ANOVA) with Bonferroni-corrected post-hoc testing was performed for arterial stiffness measures and hemodynamic variables. The significance was considered at P values < 0.05. The statistical analyses were conducted using SPSS version 23.0 (IBM SPSS Japan, Tokyo, Japan).

Results

HR and BP measures significantly increased in response to the MS task in both trials, with no significant difference between the trials (Table 1). We found no significant difference in perceived stress level (RE trial: 2.8 ± 0.8 vs. EX trial: 2.9 ± 0.9) between the trials.

HR during the seated rest in the RE trial was 64 ± 7 bpm. On the other hand, the value during the cycling exercise in the EX trial was 114 ± 5 bpm, which corresponded to the participants' 35.4 ± 1.7% of HRR.

Arterial stiffness measures before and after the MS task are illustrated in Fig. 2. Both trials caused significant elevations in hbPWV, haPWV, and CAVI at 5 min after the task. Subsequently, the elevations persisted until 45 min after the task in the RE trial. However, those in the EX trial decreased towards baseline levels; CAVI value at 45 min after the task was significantly below baseline. At 30 and 45 min after the task, these variables were significantly lower in the EX trial than those in the RE trial. baPWV significantly increased at 30 and 45 min after the task in the RE trial, whereas that in the EX trial exhibited no significant change over time. At 30 and 45 min after the task, baPWV showed significantly lower values in the EX trial than that in the RE trial.

HR and BP before and after the MS task are presented in Table 2. At 30 and 45 min after the task, HR was significantly higher in the EX trial than that in the RE trial. Systolic BP significantly elevated at 30 min after the task in the EX trial, whereas that in the RE trial showed no significant change across the measurement time points. There was no

Table 1 Hemodynamic variables before and during the MS task

	Trial	Before	Task	ANOVA
HR (bpm)	RE	56 ± 3	73 ± 6*	Time: $F = 69.4$, $P < 0.05$; Trial: $F = 0.0$, NS
	EX	57 ± 3	72 ± 9†	Interaction: $F = 0.5$, NS
Systolic BP (mmHg)	RE	112 ± 7	128 ± 7*	Time: $F = 75.1$, $P < 0.05$; Trial: $F = 0.9$, NS
	EX	111 ± 7	126 ± 9†	Interaction: $F = 0.1$, NS
Diastolic BP (mmHg)	RE	58 ± 5	75 ± 8*	Time: $F = 122.2$, $P < 0.05$; Trial: $F = 0.7$, NS
	EX	58 ± 4	73 ± 5†	Interaction: $F = 2.0$, NS
MAP (mmHg)	RE	76 ± 5	92 ± 6*	Time: $F = 118.0$, $P < 0.05$; Trial: $F = 1.3$, NS
	EX	76 ± 4	91 ± 6†	Interaction: $F = 1.8$, NS

Data are expressed as means ± standard deviation

MS mental stress, *HR* heart rate, *BP* blood pressure, *MAP* mean arterial pressure, *RE and EX* acute mental stress followed by seated rest and cycling exercise, respectively, *ANOVA* analysis of variance, *NS* not significant

*$P < 0.05$ vs. baseline in the RE trial

†$P < 0.05$ vs. baseline in the EX trial

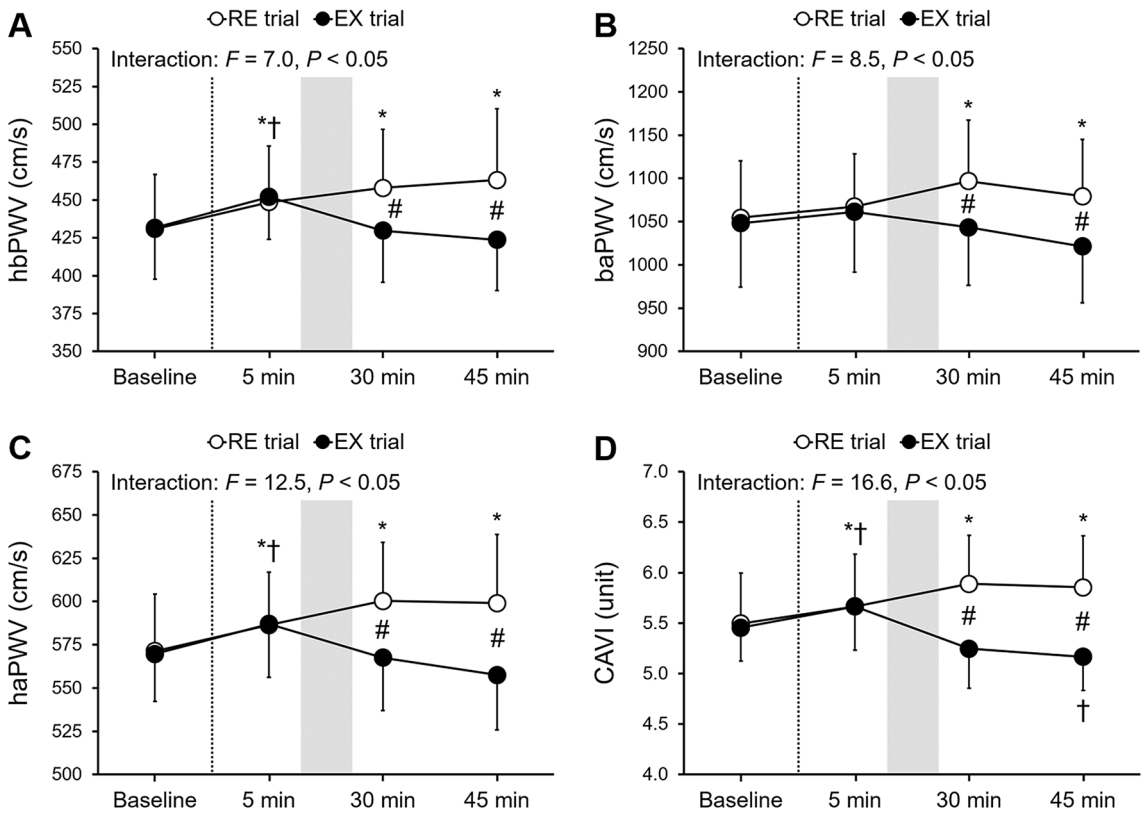

Fig. 2 Measurements of hbPWV (**a**), baPWV (**b**), haPWV (**c**), and CAVI (**d**) before and after the MS task. The dashed line indicates the time point of the MS task. The shaded box indicates the duration of seated rest or cycling exercise. *RE* and *EX* acute mental stress followed by seated rest and cycling exercise, respectively, *hbPWV* heart-brachial pulse wave velocity, *baPWV* brachial-ankle pulse wave velocity, *haPWV* heart-ankle pulse wave velocity, *CAVI* cardio-ankle vascular index, *MS* mental stress. $^{*}P<0.05$ vs. baseline in the RE trial. $^{\dagger}P<0.05$ vs. baseline in the EX trial. $^{\#}P<0.05$ vs. between the trials. Data are expressed as mean ± standard deviation

Table 2 Hemodynamic variables before and after the MS task

	Trial	Baseline	5 min	30 min	45 min	ANOVA
HR (bpm)	RE	56 ± 5	57 ± 7	55 ± 7	54 ± 5	Time: $F=1.5$, NS; Trial: $F=12.4$, $P<0.05$
	EX	56 ± 6	57 ± 6	61 ± 7$^{\#}$	60 ± 8$^{\#}$	Interaction: $F=5.3$, $P<0.05$
Systolic BP (mmHg)	RE	121 ± 6	124 ± 6	123 ± 8	120 ± 9	Time: $F=4.1$, $P<0.05$; Trial: $F=0.4$, NS
	EX	120 ± 6	122 ± 7	126 ± 9†	122 ± 10	Interaction: $F=3.2$, $P<0.05$
Diastolic BP (mmHg)	RE	67 ± 4	69 ± 4	69 ± 5	71 ± 5	Time: $F=2.6$, NS; Trial: $F=0.1$, NS
	EX	67 ± 5	69 ± 6	69 ± 6	68 ± 6	Interaction: $F=2.0$, NS
MAP (mmHg)	RE	85 ± 4	87 ± 4	87 ± 6	87 ± 6	Time: $F=3.2$, $P<0.05$; Trial: $F=0.2$, NS
	EX	85 ± 5	87 ± 5	88 ± 6	86 ± 7	Interaction: $F=1.6$, NS

Data are expressed as means ± standard deviation

MS mental stress, *HR* heart rate, *BP* blood pressure, *MAP* mean arterial pressure, *RE and EX* acute mental stress followed by seated rest and cycling exercise, respectively, *ANOVA* analysis of variance, *NS* not significant

$^{\#}P<0.05$ vs. the RE trial

$^{\dagger}P<0.05$ vs. baseline in the EX trial

significant change across the measurement time points in diastolic BP and MAP in either trial.

Discussion

The main finding of the present study is that a sustained elevation in hbPWV, baPWV, haPWV, and CAVI resulting from acute MS was found when the participants remained rested after the stress, but the elevation was eliminated completely by performing 10 min of cycling exercise. The present study demonstrates for the first time that acute MS-caused arterial stiffening can be counteracted by brief aerobic exercise.

Elevations in HR and BP are well known as typical cardiovascular responses during acute MS (Carter et al. 2005; Hayashi et al. 2006; Kuipers et al. 2008; Kume et al. 2020). In this study, HR and BP markedly increased during the MS task and the responses did not differ between the trials. Additionally, no difference between the trials was found in perceived stress rating. Therefore, it is reasonable to consider that our participants perceived the stressor equally on physiological and psychological levels in both trials.

In this study, we assessed hbPWV, baPWV, haPWV, and CAVI as an index of arterial stiffness, similar to our recent study (Kume et al. 2020). hbPWV reflects stiffness from the heart to the brachial artery, which can serve as a marker of proximal aortic stiffness (Sugawara et al. 2019). baPWV reflects stiffness of the abdominal aorta and the leg arteries. haPWV reflects stiffness from the aorta to the ankle and CAVI is a parameter of haPWV adjusted by BP (Shirai et al. 2011). In the present study, acute MS caused an elevation in hbPWV, haPWV, and CAVI at 5 min after the stress; this persisted until 45 min after the stress when the participants remained rested (the RE trial), which is in line with previous studies showing a sustained increase in arterial stiffness after acute MS (Kume et al. 2020; Vlachopoulos et al. 2006). For baPWV, however, no elevation was found at 5 min after acute MS. In this regard, a relatively minor increase in baPWV, compared with PWVs in other arterial segments, after acute MS has been found previously (Kume et al. 2020). The vasodilatory response and increases in blood flow in limb vasculatures are elicited during acute MS at the same time as the vasoconstriction in the visceral arteries (Carter et al. 2005; Hayashi et al. 2006; Kuipers et al. 2008). Thus, it could be speculated that acute MS induced the vasodilation and the accompanied increases in blood flow and shear stress in lower-limb vasculatures, which mitigated arterial stiffening in the leg segment (Heffernan et al. 2007); thereby, the increase in baPWV, which has a high rate of measurement area in the lower extremities, should have been blunted. In this study, it is possible that similar physiological responses acted more strongly, which might inhibit the increase in baPWV. On the other hand, baPWV was consistently elevated 30 min after acute MS in the RE trial; this may indicate that the acute MS-derived stiffening effect in the leg arteries overrode the postulated inhibition.

In previous studies, CAVI exhibited a transient decrease after low-to-moderate-intensity aerobic (cycling) exercise, even in the short-term (10–15 min) (Wang et al. 2014; Zhou et al. 2015). Based on this information, the present study used 10 min of low-intensity cycling exercise to counteract the increase in arterial stiffness resulting from acute MS. As a result, the exercise completely offset the elevation in hbPWV, baPWV, haPWV, and CAVI in the EX trial. This finding demonstrates that brief aerobic exercise has a counteracting effect on arterial stiffness caused by acute MS. Importantly, the exercise effect was seen in all arterial segments measured in this study. Although exercise intensity and duration were different from our study, Kingwell et al. (1997) reported a decrease in cfPWV and leg (e.g., femoral-ankle) PWV after 30 min of cycling at 65% of maximal oxygen uptake; another study wherein the participants were subjected to 30 min of walking observed a similar result (Okamoto et al. 2018). These studies suggest that lower-limb exercises could decrease both central and peripheral arterial stiffness. Therefore, it is plausible that the current observations were due to a systemic modulation of arterial stiffness by cycling exercise.

The precise mechanisms by which the counteracting effect of brief aerobic exercise on the acute MS-caused arterial stiffening are unknown. One possibility is a restoration of endothelial function. Acute MS causes impaired endothelial function (Ghiadoni et al. 2000; Sales et al. 2014; Spieker et al. 2002), which is considered as a key mechanism of the increase in arterial stiffness after acute MS (Kume et al. 2020; Vlachopoulos et al. 2006), because endothelial function is an important determinant of arterial stiffness (McEniery et al. 2006). It has been suggested that the synthesis of nitric oxide, an endothelial-derived vasodilatory factor, would be augmented even from the short-term exercise (Goto et al. 2010). Indeed, impaired endothelial function resulting from prolonged sitting and resistance exercise have been shown to be rescued by subsequent 10-min bouts of walking and cycling, respectively (Morishima et al. 2019; Restaino et al. 2015). Furthermore, improvements in endothelial function by acute bouts of aerobic exercise are not only be observed in active regions but also non-active regions (Morishima et al. 2019; Restaino et al. 2015; Totosy de Zepetnek et al. 2015), implying the presence of a systemic effect; this aspect is supported by a longitudinal study (Tinken et al. 2008). Taken together, we speculate that the restoration of endothelial function at a systemic level by aerobic exercise would result in the observed offset of arterial stiffness in various segments. Additional assessment of endothelial function should be conducted to verify this

possibility in future studies. In addition, some studies have reported that mirthful laughter induced by comical movies leads to a temporal amelioration of arterial stiffness or endothelial function (Miller et al. 2006; Sugawara et al. 2010; Vlachopoulos et al. 2009). Thus, beneficial mood alterations after exercise (Brellenthin et al. 2017) could be proposed as an additional possibility explaining our observations, although this was not assessed in the present study.

It has been suggested that both acute and chronic cardiovascular responses to MS are probably relevant (Chida and Steptoe 2010; Matthews et al. 2004). Therefore, it can be speculated that repeated daily exposure to increased arterial stiffness due to acute MS may contribute to persistent arterial stiffening. This study reveals that acute MS-caused arterial stiffening can be counteracted by performing aerobic exercise for only 10 min thereafter. This finding may be helpful in the establishment of an effective strategy to counteract the adverse vascular effects of daily stress. In terms of practical application, because the present findings were obtained in a laboratory setting using a cycle ergometer, further investigations using practical exercises that are more easily accessible in real-life, such as walking (Restaino et al. 2015) and stair climbing-descending (Takaishi et al. 2012), are warranted. We believe that performing frequent, brief exercises daily to offset acute MS-caused arterial stiffening would be beneficial to maintain vascular health.

The present study has the following limitations. First, only young healthy men were included. There are sex-based differences in the vascular response to acute bouts of MS or exercise (Morishima et al. 2020; Yang et al. 2013); thus, it is necessary to investigate whether our findings can be extrapolated to women. In addition, older individuals and hypertensive patients should also be examined in future studies. Second, cfPWV, the standard technique to assess aortic stiffness, was not measured. Thus, caution should be applied when discussing central arterial stiffness, although we showed changes in hbPWV that are a useful marker of proximal aortic stiffness (Sugawara et al. 2019).

Conclusion

The present study showed that a sustained elevation in hbPWV, baPWV, haPWV, and CAVI resulting from acute MS was offset by a subsequent 10 min bout of cycling exercise. This study provides the first evidence that brief aerobic exercise has a counteracting effect on arterial stiffening caused by acute MS.

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Author contributions DK conceived and designed the study. DK performed the experiments. DK analyzed the data. All authors interpreted the results of the experiments. DK and MN drafted the manuscript. DK edited and revised the manuscript. All authors approved the final version of manuscript.

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Compliance with ethical standards

Conflict of interest The authors have no conflict of interest to declare.

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