Effect of Upper Body Aerobic Exercise on Arterial Stiffness in Older Adults

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The authors evaluated the effects of acute arm-cycling exercise on arterial stiffness of the brachial artery (BA: working limb) and posterior tibial artery (PTA: nonworking limb) in healthy older participants. Eleven participants were tested to evaluate BA and PTA stiffness. Blood pressure (BP), heart rate (HR), and arterial stiffness indices of the BA and PTA measured by Doppler ultrasound were determined before and 10 min after graded arm-cycling exercise to volitional fatigue on 2 separate days. After the exercise, although BA diameter, brachial systolic BP, pulse pressure, and HR increased significantly (all \( p < .05 \)), arterial stiffness indices of the BA remained unchanged. Similarly, arterial stiffness indices of the PTA remained unchanged after the exercise, whereas HR increased significantly \( (p < .05) \). These results show that acute arm-cycling exercise failed to modify arterial stiffness of the BA and PTA, suggesting that it has no systemic effect on arterial stiffness in healthy older adults.

Keywords: aging, arm-cycling exercise, Doppler ultrasound, blood pressure

Aging is associated with increased arterial stiffness, a condition regarded as a possible mechanism of initiation or progression of cardiovascular disease such as left-ventricular hypertrophy and heart failure by limiting the ability of arteries to expand and recoil during cardiac pulsation and relaxation (Arnett, Evans, & Riley, 1994). Although regular aerobic exercise training has been shown to be associated with reduced development of metabolic risk factors in older adults (Petrella, Lattanzio, Demeray, Varallo, & Blore, 2005), the results of recent studies also indicate that aerobic exercise can prevent and reverse the age-associated increase in arterial stiffness in middle-aged and older participants (Tanaka et al., 2000; Vaitkevicius et al., 1993).

Reduced arterial stiffness has been observed after a single bout of acute aerobic exercise (cycling and treadmill) in young participants (Kingwell, Berry, Cameron, Jennings, & Dart, 1997; Naka et al., 2003). The reduction in arterial stiffness was observed in sites that were not primary working muscles (the aorta to femoral artery and the brachial to radial artery), as well as primary working muscles (the
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femoral to dorsalis-pedis artery). Although the results of recent studies indicate a regional effect of a single bout of lower body aerobic exercise on arterial stiffness in young adults (Heffernan, Jae, Echols, Lepine, & Fernhall, 2007; Sugawara et al., 2004), the effect of aerobic exercise on arterial stiffness is generally regarded as systemic in nature. Moreover, this systemic effect is considered one of the possible mechanisms of lower central artery stiffness associated with vigorous aerobic exercise training (Tanaka et al., 2000; Vaitkevicius et al., 1993). Given that a 12-week arm-cycling exercise-training program had a systemic effect on maximal and submaximal exercise capacity in older adults (Pogliaghi, Terziotti, Cevese, Balestrieri, & Schena, 2006), the interesting question now arises, Is the effect of a single bout of arm-cycling exercise on arterial stiffness also systemic in older participants? This may have important implications in terms of cardiovascular health for older adults who would like to have a mode of aerobic exercise other than lower body aerobic exercise such as walking and cycling because of lower limb impairment.

Accordingly, the purpose of this study was to evaluate the effects of acute maximal upper body (arm-cycling) aerobic exercise on arterial stiffness of the brachial (working limb) and posterior tibial (nonworking limb) arteries in older participants. We hypothesized that acute arm-cycling exercise would reduce arterial stiffness of both the brachial and posterior tibial arteries and that arm-cycling exercise would exert a systemic effect on arterial stiffness in older participants.

Methods

Participants

Eleven older adults (6 men and 5 women, 71.0 ± 4.4 years) who were free from overt coronary artery disease as assessed by medical history participated in the study. Two participants took antihypertensive medications (1 took an angiotensin-converting enzyme inhibitor, and the other, an angiotensin-receptor blocker) and another 2 took a cholesterol-lowering medication (atorvastatin). Their blood pressures and cholesterol levels had been well controlled for at least the past 12 months by the same drug regimens. Participants were either sedentary or recreationally active. Before participating in the study, all participants gave written informed consent, and all procedures were approved by the institutional review board at the University of Western Ontario. Sample size was calculated a priori based on a previously published study (Gokce et al., 2002), with a mean brachial artery diameter difference of 0.33 mm, SD of 0.25, α level of .05, and power of 80% (two-sided tests). Nine participants would be needed after consideration for loss to follow-up.

Study Design

Participants were tested on two different days (1 week apart). The first was to evaluate arterial stiffness of an upper limb and the other was to evaluate arterial stiffness of a lower limb. Testing sessions were randomized. Each test was conducted in a temperature-controlled room at the same time of day for each participant. Before being tested, participants fasted for at least 4 hr, abstained from caffeine,
and did not exercise 24 hr before the testing sessions. They were asked to rest quietly in a supine position for a minimum of 10 min. Blood pressure (BP) and arterial stiffness indices of either the brachial artery or the posterior tibial artery (preexercise measurements) were obtained while participants were in the supine position. After the preexercise measurements, participants performed graded arm-cycling exercise to volitional fatigue. On completion of the exercise, they returned to the supine position and the same measurements were repeated (postexercise measurements) 10 min after the exercise. The timing of the postexercise measurements was selected based on the study by Naka et al. (2003).

**Resting BP**

Brachial artery BP (systolic blood pressure [SBP], diastolic blood pressure [DBP], and pulse pressure [PP]) and heart rate (HR) were measured in the supine position with an automated oscillometric device (HDI/PulseWave CR-2000 research cardiovascular system, Hypertension Diagnostic Inc., Eagan, MN). After a 5-min rest, three BP measurements were taken, with a 2-min interval between them. The first BP measurement was discarded, and the subsequent two BP measurements were used for analysis. Posterior tibial artery SBP was also measured in the supine position with a handheld Doppler probe (Nicolet Elite, VIASYS Healthcare Inc., Madison, WI). After suprasystolically inflating the BP cuff placed proximally to the measurement site and deflating it slowly, the posterior tibial artery SBP was determined at the point when the Doppler signal reappeared. Brachial artery DBP was used as the posterior tibial artery DBP, assuming that DBP of large and medium-sized arteries changes little (Nichols & O’Rourke, 1998). Accordingly, posterior tibial artery PP was the difference between posterior tibial artery SBP and brachial artery DBP.

**Measurement of Arterial Stiffness**

Arterial stiffness indices in the brachial and posterior tibial arteries were measured using a 10-MHz linear-array transducer attached to a high-resolution ultrasound machine (VingMed System 5, GE Ultrasound A/S, Horton, Norway). All scans were performed by the same investigator under similar conditions. The longitudinal B-mode brachial internal diameter on the right arm 3–5 cm proximal to the antecubital fossa was imaged with simultaneous BP measurements of the left brachial artery. The longitudinal two-dimensional posterior tibial internal diameter on the right leg was imaged with simultaneous BP measurements of the left posterior tibial artery. In each image, the largest diameter, strong wall signals and longitudinal section of the artery were searched. Images were recorded to S-VHS tape for later off-line analysis. HR was continuously monitored with a three-lead electrocardiogram. The images of the brachial and posterior tibial arteries were analyzed from the stored images using calipers with a resolution of 0.03 mm. All images were analyzed by the same investigator. Relative change in diameter of the brachial and posterior tibial arteries during a cardiac cycle (strain) was calculated as \((D_s - D_d)/D_d\), where \(D_s\) and \(D_d\) are diameters at systole and diastole, respectively. To characterize arterial stiffness, arterial distensibility (elastic response of the artery as a hollow structure; Laurent et al., 1994) and \(\beta\)-stiffness index (stiff-
ness of the arterial wall independent of distending pressure; Hirai, Sasayama, Kawasaki, & Yagi, 1989) of the brachial and posterior tibial arteries were calculated using the equations \((D_s^2 - D_d^2)/PP \times D_d^2\) and \(\ln(SBP/DBP)/[(D_s - D_d)/D_d]\), respectively, where \(D_s^2\) and \(D_d^2\) are the square of diameter at systole and diastole, respectively. In our laboratory, the day-to-day coefficients of variation for two trials were \(1.7\% \pm 1.2\%\) and \(2.2\% \pm 1.3\%\) for the brachial artery diastolic diameter and the posterior tibial artery diastolic diameter, respectively.

**Arm-Cycling Exercise**

Participants performed graded arm-cycling exercise on a custom-built upper body ergometer while sitting in a chair. To elicit participants' maximal effort, work rate was increased every minute by 10 W for men and 3 W for women until participants reached volitional fatigue. The crank rate was set by a tachometer at 60 rpm. HR (Polar Vantage T31, Polar Electro Oy, Finland) and oxygen consumption (K4b\(^2\), COSMED USA Inc., Chicago, IL) were monitored continuously during the exercise. The traditional criteria for maximal oxygen consumption are not met during arm-cycling exercise because of localized fatigue (Franklin, 1989), so the criteria for terminating arm-cycling exercise included any of the ACSM guidelines for stopping an exercise test (American College of Sports Medicine, 2000). In this study, we considered the highest attained oxygen consumption value peak oxygen consumption.

**Statistical Analysis**

All data were reported as \(M \pm SD\), unless otherwise stated. Data were analyzed using paired-samples \(t\) tests. Because arterial stiffness is influenced by changes in mean arterial pressure, analysis of covariance (ANCOVA) was performed with mean arterial pressure as a covariate. Significance was set at \(p < .05\) for all comparisons.

**Results**

All participants completed the study. Table 1 shows their characteristics. There were no differences between the two testing days in either peak oxygen consumption or peak power output reached by participants.

**Brachial Artery Stiffness Indices and BP**

Brachial artery systolic (4.18 \(\pm\) 1.01 vs. 3.82 \(\pm\) 0.95 mm) and diastolic (4.11 \(\pm\) 1.01 vs. 3.77 \(\pm\) 0.95 mm) diameter significantly increased after the arm-cycling exercise compared with baseline (both \(p < .05\)), whereas strain did not (Table 2). Brachial artery SBP increased significantly after the exercise (136.5 \(\pm\) 15.3 vs. 124.1 \(\pm\) 13.2 mm Hg, \(p < .05\)), whereas brachial artery DBP remained unchanged. Consequently, brachial PP increased significantly after the exercise (65.3 \(\pm\) 9.3 vs. 54.1 \(\pm\) 8.2 mm Hg, \(p < .05\)). HR increased significantly after the exercise (75.4 \(\pm\) 14.1 vs. 69.5 \(\pm\) 12.2 beats/min, \(p < .05\)). Arm-cycling exercise had no effect on arterial stiffness indices of the brachial artery (Figure 1; arterial distensibility,
### Table 1  Participants’ Baseline Characteristics, $N = 11$

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>$M \pm SD$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>71.0 ± 4.4</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>170.9 ± 8.2</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>77.7 ± 15.5</td>
</tr>
<tr>
<td>Body-mass index (kg/m²)</td>
<td>26.3 ± 3.1</td>
</tr>
<tr>
<td>Ankle-brachial index (AU)</td>
<td>1.18 ± 0.09</td>
</tr>
<tr>
<td>Peak oxygen consumption (ml · kg⁻¹ · min⁻¹)</td>
<td></td>
</tr>
<tr>
<td>Day 1</td>
<td>11.2 ± 4.8</td>
</tr>
<tr>
<td>Day 2</td>
<td>10.4 ± 4.9</td>
</tr>
<tr>
<td>Peak power output (W)</td>
<td></td>
</tr>
<tr>
<td>Day 1</td>
<td>55.2 ± 27.7</td>
</tr>
<tr>
<td>Day 2</td>
<td>53.4 ± 27.7</td>
</tr>
</tbody>
</table>

*Note. AU = arbitrary unit.*

### Table 2  Effect of Arm-Cycling Exercise on Exercise Responses of the Brachial and Posterior Tibial Arteries Before and After the Exercise ($M \pm SD$), $N = 11$

<table>
<thead>
<tr>
<th>Variable</th>
<th>Baseline</th>
<th>After exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brachial artery</td>
<td></td>
<td></td>
</tr>
<tr>
<td>systolic diameter (mm)</td>
<td>3.82 ± 0.95</td>
<td>4.18 ± 1.01*</td>
</tr>
<tr>
<td>diastolic diameter (mm)</td>
<td>3.77 ± 0.95</td>
<td>4.11 ± 1.01*</td>
</tr>
<tr>
<td>strain (%)</td>
<td>1.50 ± 0.51</td>
<td>1.71 ± 0.73</td>
</tr>
<tr>
<td>systolic blood pressure (mm Hg)</td>
<td>124.1 ± 13.2</td>
<td>136.5 ± 15.3*</td>
</tr>
<tr>
<td>diastolic blood pressure (mm Hg)</td>
<td>70.0 ± 6.7</td>
<td>71.1 ± 7.9</td>
</tr>
<tr>
<td>mean arterial pressure (mm Hg)</td>
<td>88.0 ± 8.5</td>
<td>92.9 ± 10.0*</td>
</tr>
<tr>
<td>pulse pressure (mm Hg)</td>
<td>54.1 ± 8.2</td>
<td>65.3 ± 9.3*</td>
</tr>
<tr>
<td>heart rate (beats/min)</td>
<td>69.5 ± 12.2</td>
<td>75.4 ± 14.1*</td>
</tr>
<tr>
<td>Posterior tibial artery</td>
<td></td>
<td></td>
</tr>
<tr>
<td>systolic diameter (mm)</td>
<td>2.36 ± 0.46</td>
<td>2.40 ± 0.49</td>
</tr>
<tr>
<td>diastolic diameter (mm)</td>
<td>2.31 ± 0.47</td>
<td>2.34 ± 0.50</td>
</tr>
<tr>
<td>strain (%)</td>
<td>2.13 ± 0.57</td>
<td>2.38 ± 0.73</td>
</tr>
<tr>
<td>systolic blood pressure (mm Hg)</td>
<td>146.9 ± 17.6</td>
<td>155.7 ± 18.0</td>
</tr>
<tr>
<td>diastolic blood pressure (mm Hg)</td>
<td>68.7 ± 8.8</td>
<td>70.7 ± 7.6</td>
</tr>
<tr>
<td>pulse pressure (mm Hg)</td>
<td>78.2 ± 16.2</td>
<td>85.1 ± 15.1</td>
</tr>
<tr>
<td>heart rate (beats/min)</td>
<td>65.1 ± 10.4</td>
<td>73.1 ± 12.4*</td>
</tr>
</tbody>
</table>

*p < .05 vs. baseline.
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0.045 ± 0.004 vs. 0.044 ± 0.004 L/mm Hg × 10⁻²; β-stiffness index, 42.6 ± 4.7 vs. 44.2 ± 3.4 arbitrary units.

**Figure 1** — Effect of arm-cycling exercise on arterial distensibility (upper) and β-stiffness index (lower) of the brachial artery in older participants, \( M ± SE \).

Posterior Tibial Artery Stiffness Indices and BP

In contrast to the brachial artery, posterior tibial diameter did not increase after the arm-cycling exercise (Table 2). Strain of the posterior tibial artery also remained unchanged. Posterior tibial artery SBP (155.7 ± 18.0 vs. 146.9 ± 17.6 mm Hg) tended to be higher after the exercise (\( p > .05 \)), while posterior tibial DBP and PP did not change. HR increased significantly after the exercise (73.1 ± 12.4 vs. 65.1 ± 10.4 beats/min, \( p < .05 \)). Arm-cycling exercise had no effect on arterial stiffness indices of the posterior tibial artery (Figure 2; arterial distensibility, 0.047 ± 0.005 vs. 0.045 ± 0.003 L/mm Hg × 10⁻²; β-stiffness index, 40.5 ± 4.6 vs. 39.7 ± 3.8 arbitrary units.)
This study was designed to evaluate the effect of acute arm-cycling exercise on arterial stiffness of the brachial and posterior tibial arteries in older adults and to determine whether upper body exercise, similar to lower body exercise, would have a systemic effect on arterial stiffness. In contrast to previous studies that demonstrated the systemic effect of acute lower body exercise (cycling and treadmill exercise) on arterial stiffness in young participants (Kingwell et al., 1997; Naka et al., 2003), the results of this study showed that, in older adults, arterial stiffness of the brachial and posterior tibial arteries remained unchanged after acute arm-cycling exercise and that upper body exercise did not appear to have a systemic effect on arterial stiffness in older adults.

Arterial Stiffness of the Brachial Artery (Working Limb)

In this study, participants performed acute arm-cycling exercise to volitional fatigue. Blood flow to the working limb increased markedly during exercise. In turn, this enhanced blood flow may have increased shear stress, leading to increased nitric oxide and other metabolites produced from vascular endothelium that modulates arterial distensibility (Wilkinson et al., 2002). Indeed, brachial blood flow in our participants was increased by about 64% 10 min after the arm-
cycling exercise (data not shown). The enhanced blood flow and shear stress increase brachial artery diameter, as was observed in this study. However, the increase in diameter could stretch arterial-wall material and transfer stress from distensible elastin fibers to less distensible collagen fibers, leading to the movement toward a stiffer portion of the diameter–pressure curve (Safar & London, 1994). Thus, although enhanced endothelial function leads to vasodilation, vasodilation by itself and/or with increased BP as observed in this study could be responsible for the lack of effect on arterial stiffness of the working limb (brachial artery). Alternatively, increased oxidative stress associated with aging (Hamilton, Brosnan, McIntyre, Graham, & Dominiczak, 2001) and/or with maximal exercise (Silvestro et al., 2002) may decrease nitric oxide availability in older adults. Endothelial function is known to decline with aging (Taddei et al., 1995), so increased oxidative stress may, in part, account for the unchanged arterial stiffness of the brachial artery after the arm-cycling exercise in this study.

**Arterial Stiffness of the Posterior Tibial Artery (Nonworking Limb)**

Contrary to the brachial artery, neither posterior tibial artery diameter nor its BP increased significantly 10 min after arm-cycling exercise in this study. Not only does blood flow to working limbs increase during exercise, but also blood flow to nonworking limbs increases markedly during incremental exercise in healthy young participants (Tanaka et al., 2006), suggesting that conduit arteries of non-working limbs may be receiving sufficient stimuli from increased blood flow and shear stress. This increase may result in the augmented endothelial function via increased nitric oxide availability as previously discussed and lead to vasodilation in those arteries. Pharmacologically induced vasodilation in the leg has been shown to be preserved with aging, which suggests preserved endothelial function in the leg compared with reduced endothelial function in the arm (Newcomer, Leuenberger, Hogeman, & Proctor, 2005). This finding would also indicate that endothelial function might not be a determining factor for arterial stiffness of the lower limbs in healthy older adults.

The question is raised as to what might be responsible for the discrepancy between previous studies (Kingwell et al., 1997; Naka et al., 2003) and this study. We can only speculate on the mechanisms by which acute arm-cycling exercise failed to modify arterial stiffness indices of the nonworking limb in older participants. Arterial stiffness of peripheral muscular arteries appears less closely related to aging (van der Heijden-Spek et al., 2000). Although blood flow in working limbs increases during exercise, leg blood flow during knee-extension exercise has been shown to be less in older than in young participants (Donato et al., 2006). It is possible, therefore, that nonworking lower limbs of older participants may not be receiving sufficient stimuli from blood flow and shear stress during acute arm-cycling exercise compared with younger participants. Another possibility is that $\alpha_1$-adrenergic receptor sensitivity and/or $\alpha_1$-adrenergic receptor numbers may be increased in the lower leg (Pawelczyk & Levine, 2002). These changes coupled with the age-associated increase in sympathetic nerve activity are thought to reflect the chronically increased hydrostatic pressure in the legs relative to the arms and may play an important regulatory role to prevent blood pooling while
standing, especially in older adults. Consistent with these possibilities and the results of this study, arterial distensibility and blood flow of the posterior tibial artery did not change with the increase in distending pressure (Eiken & Kölegård, 2004).

**Study Limitations**

There are several limitations of this study that need to be mentioned. First, we did not include young participants as a control group, so it is unclear whether acute arm-cycling exercise would have a systemic effect on arterial stiffness in young individuals. This issue needs to be addressed in a future study. Second, we did not assess blood chemistries such as glucose levels and lipid profiles. If these levels were elevated, there might have been an effect on our results, although our participants were free from overt cardiovascular disease. Third, because we did not have time-controlled data in this study, it could be argued that the increase in brachial artery diameter observed after the exercise was not an effect of exercise but of the periodical 3–4% diameter oscillations of muscular arteries (Hayoz et al., 1993). This is unlikely, however, because the diameter change in the brachial artery we observed was more than twofold greater than that observed previously (Hayoz et al.). Thus, we believe that the diameter change was a true effect of the exercise. Fourth, we assessed arterial stiffness indices only once after the arm-cycling exercise (10 min after the exercise). Although previous studies have shown reduced arterial stiffness immediately after aerobic exercise in young adults (Naka et al., 2003; Sugawara et al., 2004), this does not preclude the possibility that changes in arterial stiffness occurred at a later time point (e.g., 20 min) in our older participants. Future study examining the time course of change in arterial stiffness will shed light on this issue. Fifth, although all images were analyzed by the same investigator so that we were able to minimize intraobserver variability, we were not completely free from bias in that the investigator was not blinded to the time of the measurement. Finally, we did not examine an effect of short-term or long-term arm-cycling exercise training on arterial stiffness in older participants, so it is unclear whether there is an effect of chronic arm-cycling exercise training on arterial stiffness in this population. Future study will be warranted to address this issue.

**Conclusions**

In conclusion, the results of this study show that acute arm-cycling exercise failed to modify arterial stiffness of the brachial artery or the posterior tibial artery. Furthermore, in contrast to previous acute lower body exercise studies in young participants (Kingwell et al., 1997; Naka et al., 2003), acute upper body exercise had no systemic effect on arterial stiffness in older healthy participants. This might be because of the reduced stimuli from blood flow and shear stress in the legs during arm-cycling exercise and the increased $\alpha_1$-adrenergic receptor sensitivity and/or its receptor numbers in older adults. To determine whether short-term and long-term arm-cycling exercise training modify arterial stiffness systemically in older adults will require further research.
Acknowledgment

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References


