Combined aerobic and resistance training and vascular function: effect of aerobic exercise before and after resistance training

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Combined aerobic exercise training combined with resistance training (RT) might prevent the deterioration of vascular function. However, how aerobic exercise performed before or after a bout of RT affects vascular function is unknown. The present study investigates the effect of aerobic exercise before and after RT on vascular function. Thirty-three young, healthy subjects were randomly assigned to groups that ran before RT (BRT: 4 male, 7 female), ran after RT (ART: 4 male, 7 female), or remained sedentary (SED: 3 male, 8 female). The BRT and ART groups performed RT at 80% of one repetition maximum and ran at 60% of the targeted heart rate twice each week for 8 wk. Both brachial-ankle pulse wave velocity (baPWV) and flow-mediated dilation (FMD) after combined training in the BRT group did not change from baseline. In contrast, baPWV after combined training in the ART group reduced from baseline (from 1,025 ± 43 to 910 ± 33 cm/s, P < 0.01). Moreover, brachial artery FMD after combined training in the ART group increased from baseline (from 7.3 ± 0.8 to 9.6 ± 0.8%, P < 0.01). Brachial artery diameter, mean blood velocity, and blood flow in the BRT and ART groups after combined training increased from baseline (P < 0.05, P < 0.01, and P < 0.001, respectively). These values returned to the baseline during the detraining period. These values did not change in the SED group. These results suggest that although vascular function is not improved by aerobic exercise before RT, performing aerobic exercise thereafter can prevent the deteriorating of vascular function.

Combined aerobic exercise training combined with resistance training improved endothelium-dependent nitric oxide (NO)-mediated vascular function in both conduit and resistance vessels. Therefore, combined training can improve vascular function. However, aerobic exercise was performed after RT in almost all previous studies. Therefore, whether aerobic exercise performed before RT rather than after RT as previously examined could improve vascular function is unknown.

Beneficial training associated changes in blood pressure (21, 27). In addition, aerobic exercise intervention improves impaired endothelial function, and the induced increase in blood flow velocity elicits endothelial shear stress (4, 13). On the other hand, high-intensity resistance exercise promotes acute increases in blood pressure to a much larger degree than aerobic exercise (19). Furthermore, resistance exercise has been shown to cause decrease central arterial compliance (8). One bout of intense aerobic exercise also has been shown to decrease arterial compliance acutely. Because resistance exercise-stimulated increases in blood pressure might cause vascular function to deteriorate, aerobic exercise performed before resistance exercise might not favorably affect vascular function to the same degree as when the aerobic stimulus occurs after the blood pressure-elevating resistance exercise bout. That is, although blood pressure and arterial stiffness that has been increased by RT can be decreased by subsequent aerobic exercise, these effects might not be positively influenced when the aerobic exercise takes place before resistance exercise. The present study investigates the training effect of the influence of timing of aerobic exercise with respect to resistance exercise on vascular function in healthy young adults. We postulated that aerobic exercise before RT does not positively affect vascular function.

METHODS

Subjects

Participants in this study were 33 healthy nonsmoking males and females who were not actively involved in regular physical exercise (11 male, 22 female; age 18.6 ± 0.1 yr, mean ± SE). Health examinations revealed no abnormal findings, and blood pressure levels were within normal range. Health examination was performed as follows: anamnesis, blood pressure, dipstick test, electrocardiogram, and chest X-ray. Blood examination was not performed. Anamnesis was performed to confirm the current health condition, and a dipstick test was performed to indicate renal function. The daily rhythms (sleeping hours, bedtime, and wake-up time) of the subjects were nearly constant. Although some subjects who had an exercise habit in the past were included, most of the subjects had not exercised

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for more than 1 yr and had not engaged in RT. Although we did not measure aerobic fitness in this study, all participants could continuously run for 20 min at an intensity of 60% of the targeted heart rate. Subjects were randomly assigned to groups that ran before RT (BRT; \( n = 11 \): 4 male, 7 female), ran after RT (ART; \( n = 11 \): 4 male, 7 female), or remained sedentary (SED; \( n = 11 \): 3 male, 8 female). Table 1 shows physical characteristics of the subjects. We did not include a group that performed only RT because the primary focus of this study was to determine the effects of aerobic exercise before and after RT. Experiments were carried out under the approval of the Ethical Committee of the Kinki Welfare University, and all subjects provided written informed consent.

**Study Design**

Training involved a combination of treadmill running and RT performed on weight-stack machines and proceeded twice weekly between 2:00 and 6:00 PM. Subjects in the SED group were instructed not to alter their normal activity levels throughout the study period.

**Aerobic Training**

The exercise severities for running were set at 60% of the targeted heart rate using a heart rate monitor (Polar RS100; Canon Trading, Tokyo, Japan). The targeted heart rates were calculated using the following formula according to the Carbonen method (15): targeted heart rate = \([\text{maximal heart rate} \times (220 - \text{age}) - \text{resting heart rate}] \times 0.6\) (exercise intensity 60%) + resting heart rate. The BRT group ran before RT for 20 min. The ART group ran after RT for 20 min.

**Resistance Training**

RT was conducted two times (Tuesday and Friday) weekly for 8 wk. The RT program consisted of chest press, arm curl, seated row, shoulder press, leg curl, leg press, and abdominal sit-up (sit up). One repetition maximal (1RM) in all exercises was measured with the exception of abdominal bent. Because of the potential risks involved in 1RM testing, this test was not performed in the SED. The load was set to 80% of 1RM. Training was conducted as 5 sets of 8–10 repetitions with an interset rest period of 2 min. The 1RM was measured in all subjects again after 4 wk from the start of training, and the load was adjusted based on a new 1RM for subjects whose 1RM had increased. Subjects performed the resistance training with 1-s eccentric (lowering phase) and 1-s concentric (lifting phase) muscle actions. Subjects repeated the actions at approximately constant velocity and frequency with a metronome. Subjects were instructed in the correct lifting technique and in how to prevent the Valsalva maneuver.

**Measurements**

The BRT, ART, and SED groups were studied before (baseline) and after training (8 wk, completion of training) and after detraining (4 wk, completion of detraining).

**Measurement of brachial-ankle pulse wave velocity.** After the subject had rested in a supine position for at least 10 min, brachial-ankle pulse wave velocity (baPWV) was measured using an automatic oscillometric device (form PWV/ABI; Omron-Colin, Komaki, Japan). This device records the PWV, blood pressure, an electrocardiogram, and heart sounds from and arterial blood pressure at both the left and right brachia and ankles. The device has four cuffs that are used simultaneously. It was reported that baPWV may provide information qualitatively similar to that derived from central arterial stiffness (35). baPWV was measured in subjects at rest in the supine position with sensory cuffs wrapped around both brachials and ankles. Before measurement, subjects abstained from caffeine and fasted for at least 4 h. Left and right baPWV were measured using the oscillometric method. Pulse volume records of bilateral brachial and ankle arteries were monitored by a continuous deflation of the cuffs. The distance between sampling points of baPWV was calculated automatically according to the height of the subject. baPWV was calculated by dividing the distances (\(D\)) from the aorta to the cuff on the left (right) brachial [Lb: Lb = 0.2195 \times \text{height of the subject (cm)} - 2.0734] and to that on the left (right) ankle [La: La = 0.8129 \times \text{height of the subject (cm)} + 12.328] by each pulse transit time (t). That is, baPWV = \(D/(Lb - La/t)\).

The sample acquisition frequency for PWV was set at 1,200 Hz, and the sampling time was 10 s with automatic gain analysis and quality adjustment. Because all of the participants were right-handed, we used the left baPWV value in this study. In our laboratory, the day-to-day reproducibility of the measurements for baPWV was 5.3%. We confirmed the reliability of PWV beforehand by measuring blood pressure at the upper arm during rest using the oscillometric method and comparing this value to the upper arm blood pressure determined 10 min later. The coefficients of variation (CV) for baPWV were 4.8, 5.1, and 4.8% for before training, after training and after detraining, respectively.

**Measurement of brachial artery hemodynamics.** Measurement of brachial artery diameter and brachial artery mean blood velocity (MBV) were performed while subjects were supine in a quiet room. All of the measurements were performed at a constant room temperature (23–25°C). In all the studies, brachial artery diameter and brachial MBV were obtained after at least a 10-min rest. All image analyses were performed by the same investigator, who was blinded to the group assignment of subjects. The brachial artery diameter and brachial MBV were imaged using a B-mode ultrasonography and a 7.5-MHz convex probe (Sonosite 180PLUS; Washington, DC) at a location 3–7 cm above the antecubital fossa. Ultrasound images were recorded on a personal computer, and brachial artery diameter measurements were analyzed using computerized image-analysis software (Scion Image Beta 4.02).

Brachial artery diameter was measured from longitudinal images with the lumen-intima interface visualized on both (anterior and posterior) walls. Brachial artery diameter was assessed using automatic edge detection. The CV for brachial artery diameter were 6.2, 6.9, and 6.2% for before training, after training, and after detraining, respectively. Because brachial artery diameter was measured twice, the CV was calculated from the difference of the measurement values at both times.

Pulsed Doppler measurements for measuring brachial MBV were performed with the sample volume placed in midartery. Brachial
MBV measurements were performed with the insonation angle <60° and were corrected for the insonation angle. The position of the transducer was marked to ensure the same position of the transducer for all measurements. Hyperemic flow velocity was determined during the first 10- to 15-s arterial pulses after cuff deflation to establish the magnitude of the hyperemic response.

Brachial artery blood flow (BF) and hyperemic BF were calculated as follows: brachial BF = MBV × circular area × (6 × 10^4), and hyperemic BF = hyperemic blood velocity × circular area × (6 × 10^4). The constant 6 × 10^4 is the conversion factor from meters per second to liters per minute. Moreover, brachial vascular conductance (VC) and resistance (VR) were calculated as follows (1, 23): brachial VC = brachial BF/brachial mean pressure, and brachial VR = brachial mean pressure/brachial BF. The brachial mean pressure was determined using the value provided by the baPWV measurement (1, 23).

Brachial artery flow-mediated dilation (FMD) was assessed noninvasively as described by Corretti et al. (6). Once the basal measurements were obtained, arterial occlusion was created by inflating a cuff placed on the forearm to 240 mmHg for 5 min. After 5 min of inflation, the cuff was deflated, producing a brief high-flow state resulting in artery dilatation due to increased shear stress. For the FMD scans, brachial artery diameter and brachial BF measurements were taken between 50 and 70 s after cuff deflation, and the maximal diameter was taken. The increase in BF after the release of the cuff was expressed as the percent change from the baseline flow. FMD was calculated as the percent increase in brachial artery diameter from the resting state (DB, for basal diameter) to maximal dilatation (DM, for maximal diameter); %FMD = (DM − DB)/DB × 100.

The CV for FMD were 6.9, 7.1, and 6.8% for before training, after training, and after detraining, respectively. To most effectively evaluate the stimulus-response relationship between shear rate and vasodilation, FMD was normalized to shear rate (32) and calculated as follows (30): normalized FMD = %diameter/shear rate area under the curve (MBV/diameter).

Statistical Analysis

Data are means ± SE. The training period on physiological variables (baPWV, %FMD) was analyzed via two-way (group × period) repeated-measures ANOVA. The Newman-Keuls method was used for multiple post hoc comparisons. Findings were considered significant at P < 0.05.

RESULTS

Changes in Body Composition

In all groups, there were no changes in body mass and body mass index throughout the intervention periods. However, body fat in both the BRT and ART groups significantly decreased (P < 0.05) (Table 1).

Changes in 1RM

All the subjects in both the BRT and ART groups completed the training program. Increases of 1RM in the BRT group were observed in the shoulder press, seated row (P < 0.05), arm curl, leg press (P < 0.01), and leg curl (P < 0.001). Percent increases for each of the exercises were 13% in shoulder press, 15% in seated row, 33% in arm curl, 16% in leg press, and 16% in leg curl. There were no significant differences in the chest press (percent increases were 18%). On the other hand, increases of 1RM in the ART group were observed in the shoulder press, leg press (P < 0.01), chest press, seated row, arm curl, and leg curl (P < 0.001). Percent increases for each of the exercises were 17% in shoulder press, 39% in leg press, 20% in chest press, 16% in seated row, 52% in arm curl, and 29% in leg curl.

Changes in baPWV

Figure 1, top, shows changes in baPWV before and after training and after detraining. There were no significant differences in the baseline baPWV among the three groups. baPWV after combined training in the BRT group did not change from baseline. In contrast, baPWV after combined training in the ART group significantly reduced from baseline (P < 0.01). After the detraining period, baPWV returned to the baseline level in the ART group. Moreover, baPWV significantly reduced after combined training in the ART group compared with the BRT group (P < 0.01). In addition, baPWV significantly reduced after combined training in the ART group compared with the SED group (P < 0.05). baPWV in the SED group did not differ before and after training and after detraining. There were no significant differences in baPWV after detraining among the three groups.
Changes in Brachial Artery FMD and Normalized FMD

Figure 1, middle and bottom, shows changes in brachial artery FMD and normalized FMD before and after training and after detraining. There were no significant differences in the baseline brachial artery FMD and normalized FMD among the three groups. Brachial artery FMD and normalized FMD after combined training in the BRT group did not change from baseline. In contrast, brachial artery FMD and normalized FMD after combined training in the ART group significantly increased from baseline \( (P < 0.01) \). After the detraining period, brachial artery FMD and normalized FMD returned to the baseline level in the ART group. Moreover, brachial artery FMD and normalized FMD significantly increased after combined training in the ART group compared with the BRT group \( (P < 0.01) \). In addition, brachial artery FMD and normalized FMD significantly increased after combined training in the ART group compared with the SED group \( (P < 0.05) \). Brachial artery FMD and normalized FMD in the SED group did not differ before and after training and after detraining. There were no significant differences in brachial artery FMD and normalized FMD after detraining among the three groups.

Changes in Brachial Blood Pressure and Heart Rate

Table 2 shows changes in brachial blood pressure and heart rate before and after training and after detraining. There were no significant differences in brachial blood pressure and heart rate before and after training and after detraining among the three groups.

Changes in Brachial Artery Hemodynamics

Table 3 shows changes in brachial artery hemodynamics before and after training and after detraining. There were no significant differences in the baseline hemodynamics among the three groups. However, brachial artery diameter, MBV, hyperemic BV, BF, and hyperemic BF after combined training in the BRT group significantly increased from baseline \( (P < 0.05) \). On the other hand, brachial artery diameter, MBV, hyperemic BV, BF, and hyperemic BF after combined training in the ART group significantly increased from baseline \( (P < 0.01) \). Moreover, brachial artery diameter, MBV, hyperemic BV, BF, and hyperemic BF significantly increased after combined training in the BRT group compared with the SED group \( (P < 0.01, P < 0.05) \). Furthermore, brachial artery diameter, MBV, hyperemic BV, BF and hyperemic BF significantly increased after combined training in the ART group compared with the SED group \( (P < 0.01, P < 0.05) \). Brachial VC after combined training in the BRT and ART groups significantly increased from baseline \( (P < 0.001) \). Brachial VR after combined training in the BRT and ART groups significantly reduced from baseline \( (P < 0.001) \). After the detraining period, hemodynamics returned to the baseline level in the BRT and ART groups. In addition, brachial VR significantly reduced after combined training in the BRT and ART groups compared with the SED group \( (P < 0.001) \). Differences in the SED group brachial artery hemodynamics did not differ before and after training and after detraining. There were no significant differences in the hemodynamics after detraining among the three groups.

Table 2. Changes in brachial blood pressure and heart rate before and after training and after detraining

<table>
<thead>
<tr>
<th>Variables</th>
<th>Before Training (Baseline)</th>
<th>After Training</th>
<th>After Detraining</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic pressure, mmHg</td>
<td>113.9±3.1</td>
<td>112.5±2.1</td>
<td>112.4±2.6</td>
</tr>
<tr>
<td>SED</td>
<td>113.6±3.4</td>
<td>111.2±3.0</td>
<td>108.9±3.5</td>
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<tr>
<td>BRT</td>
<td>113.5±4.3</td>
<td>110.1±3.7</td>
<td>110.7±3.7</td>
</tr>
<tr>
<td>ART</td>
<td>63.3±2.1</td>
<td>61.5±1.4</td>
<td>63.1±1.7</td>
</tr>
<tr>
<td>Diastolic pressure, mmHg</td>
<td>62.5±2.2</td>
<td>60.5±2.0</td>
<td>59.8±2.1</td>
</tr>
<tr>
<td>SED</td>
<td>64.5±1.9</td>
<td>59.3±2.5</td>
<td>60.3±2.9</td>
</tr>
<tr>
<td>BRT</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ART</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean pressure, mmHg</td>
<td>83.0±2.3</td>
<td>78.5±0.8</td>
<td>80.9±2.0</td>
</tr>
<tr>
<td>SED</td>
<td>82.7±2.9</td>
<td>79.1±2.7</td>
<td>80.8±2.9</td>
</tr>
<tr>
<td>BRT</td>
<td>83.2±2.8</td>
<td>78.6±2.9</td>
<td>81.0±2.5</td>
</tr>
<tr>
<td>ART</td>
<td>50.6±2.7</td>
<td>51.0±1.3</td>
<td>49.3±1.9</td>
</tr>
<tr>
<td>Pulse pressure, mmHg</td>
<td>51.2±1.9</td>
<td>50.6±1.6</td>
<td>49.1±2.2</td>
</tr>
<tr>
<td>SED</td>
<td>48.9±3.0</td>
<td>50.8±2.0</td>
<td>50.5±2.3</td>
</tr>
<tr>
<td>BRT</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ART</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>68.7±2.1</td>
<td>68.6±2.7</td>
<td>68.3±2.3</td>
</tr>
<tr>
<td>SED</td>
<td>66.3±2.2</td>
<td>63.8±3.9</td>
<td>64.8±3.6</td>
</tr>
<tr>
<td>BRT</td>
<td>66.5±1.9</td>
<td>65.4±1.9</td>
<td>66.5±2.4</td>
</tr>
<tr>
<td>ART</td>
<td></td>
<td></td>
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</table>

Values are means ± SE.

Table 3. Changes in brachial artery hemodynamics before and after training and after detraining

<table>
<thead>
<tr>
<th>Variables</th>
<th>Before Training (Baseline)</th>
<th>After Training</th>
<th>After Detraining</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brachial artery diameter, mm</td>
<td>3.9±0.2</td>
<td>4.0±0.2</td>
<td>3.9±0.2</td>
</tr>
<tr>
<td>SED</td>
<td>4.0±0.3</td>
<td>4.3±0.3&lt;sup&gt;ad&lt;/sup&gt;</td>
<td>4.0±0.3</td>
</tr>
<tr>
<td>BRT</td>
<td>4.0±0.3</td>
<td>4.3±0.3&lt;sup&gt;ad&lt;/sup&gt;</td>
<td>4.0±0.3</td>
</tr>
<tr>
<td>ART</td>
<td>4.21±0.17</td>
<td>4.11±0.22</td>
<td>4.14±0.20</td>
</tr>
<tr>
<td>Brachial artery mean blood velocity, cm/s</td>
<td>21.07±0.25</td>
<td>20.57±0.31</td>
<td>20.68±0.28</td>
</tr>
<tr>
<td>SED</td>
<td>30.8±3.0</td>
<td>30.4±3.6</td>
<td>30.3±3.3</td>
</tr>
<tr>
<td>BRT</td>
<td>31.7±5.8</td>
<td>39.9±6.2&lt;sup&gt;be&lt;/sup&gt;</td>
<td>31.8±5.7</td>
</tr>
<tr>
<td>ART</td>
<td>32.5±4.6</td>
<td>40.7±5.9&lt;sup&gt;be&lt;/sup&gt;</td>
<td>31.8±4.3</td>
</tr>
<tr>
<td>Brachial artery hyperemic blood flow, ml/min</td>
<td>177.8±5.5</td>
<td>176.2±6.7</td>
<td>174.7±6.0</td>
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<tr>
<td>SED</td>
<td>183.9±9.6</td>
<td>226.7±10.5&lt;sup&gt;be&lt;/sup&gt;</td>
<td>184.3±9.3</td>
</tr>
<tr>
<td>BRT</td>
<td>187.2±7.8</td>
<td>243.3±10.2&lt;sup&gt;be&lt;/sup&gt;</td>
<td>183.7±7.1</td>
</tr>
<tr>
<td>Vascular conductance, ml·min&lt;sup&gt;-1&lt;/sup&gt;·mmHg&lt;sup&gt;-1&lt;/sup&gt;</td>
<td>0.37±0.04</td>
<td>0.39±0.05</td>
<td>0.38±0.04</td>
</tr>
<tr>
<td>SED</td>
<td>0.38±0.06</td>
<td>0.51±0.07&lt;sup&gt;ef&lt;/sup&gt;</td>
<td>0.40±0.06</td>
</tr>
<tr>
<td>BRT</td>
<td>0.40±0.07</td>
<td>0.52±0.07&lt;sup&gt;ef&lt;/sup&gt;</td>
<td>0.39±0.05</td>
</tr>
<tr>
<td>ART</td>
<td>2.71±0.30</td>
<td>2.66±0.32</td>
<td>2.69±0.30</td>
</tr>
<tr>
<td>Vascular resistance, mmHg·ml&lt;sup&gt;-1&lt;/sup&gt;·min&lt;sup&gt;-1&lt;/sup&gt;</td>
<td>2.66±0.44</td>
<td>2.01±0.28&lt;sup&gt;ef&lt;/sup&gt;</td>
<td>2.59±0.42</td>
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<tr>
<td>SED</td>
<td>2.60±0.48</td>
<td>1.96±0.33&lt;sup&gt;ef&lt;/sup&gt;</td>
<td>2.58±0.35</td>
</tr>
</tbody>
</table>

Values are means ± SE. *P < 0.05; *P < 0.01; †P < 0.001 vs. baseline. <sup>ad</sup>P < 0.05; *P < 0.01; †P < 0.001 vs. SED group.
DISCUSSION

This is the first study to investigate the influence of the timing of aerobic exercise in relation to resistance exercise on vascular function in a training program. The key novel findings are that arterial stiffness did not change in the BRT group but were significantly reduced in the ART group. Moreover, FMD in the ART group significantly increased. These findings suggest that the aerobic exercise before RT does not favorably affect vascular function and do not support the notion that aerobic exercise always exerts a beneficial effect on vascular function. Nevertheless, the present findings have potentially important clinical implications.

A previous study indicated that simultaneous endurance and resistance training may negate potentially negative effects of arterial stiffening observed with some strength training programs (5). Moreover, aerobic exercise after RT might prevent the stiffening of carotid arteries associated with RT in healthy young men (16). However, the impact of aerobic exercise performed before resistance exercise is not understood. Consistent with previous results, the present study demonstrates that 8 wk of aerobic exercise training performed after resistance exercise resulted in reduced arterial stiffness in healthy young adults. However, when the format of the training program was altered so that aerobic exercise was performed before resistance exercise, no alterations in arterial stiffness were observed. Our findings therefore suggest that aerobic exercise before RT does not promote arterial flexibility. Green et al. (13) indicated that aerobic combined with resistance training improves endothelium-dependent NO-mediated endothelial function. The ability of a conduit artery to accommodate changes in BF and shear stress by increasing internal diameter was termed FMD. The present study found that aerobic exercise after RT significantly increased the FMD in young healthy adults, which supported the results of others. In contrast, aerobic exercise before RT did not change FMD. Thus, although aerobic exercise before RT improves vascular function, such favorable effect might be neutralized by RT. Although significant changes in FMD and PWV in the ART group might be explained by greater cardiovascular benefits, we did not measure aerobic fitness, which is an important limitation of the present study.

We could not define why aerobic exercise before RT did not improve vascular function. Intense RT is a powerful stimulator of sympathetic nervous system activity, which might intensify vasoconstriction through a sympathetic adrenergic vasoconstrictor effect (1, 29, 31). In addition, RT might increase arterial stiffness because of powerful sympathetic vasoconstrictive effects as well as through effects on arterial walls (29), since vascular function is closely associated with the sympathetic nervous system (37). Arterial blood pressure during systole exerts a powerful sympathetic vasoconstrictor effect (1, 29, 31). In addition, RT might increase arterial stiffness because of powerful sympathetic vasoconstrictive effects as well as through effects on arterial walls (29), since vascular function is closely associated with the sympathetic nervous system (37). Arterial blood pressure during high-intensity resistance exercise can increase to 320/250 mmHg (19). Frequent increases in blood pressure reduce arterial elasticity (18) and levels of elastin and increase those of collagen. In addition, Ebenbichler et al. (10) indicated that the endothelial function of body builders taking anabolic steroids and performing isometric exercise is impaired. Thus RT might adversely affect vascular function. Whereas RT promotes, aerobic exercise suppresses increases in blood pressure (21, 27). Aerobic exercise before RT might suppress a subsequent increase in blood pressure induced by RT. That is, although vascular function is not improved by aerobic exercise before RT, deterioration caused by RT could be prevented by performing aerobic exercise thereafter. Accordingly, the present findings demonstrate that the favorable effects of aerobic exercise are negated by subsequent RT, whereas the unfavorable effects of RT are counteracted by subsequent aerobic exercise. However, the precise mechanisms responsible for the changes in vascular function induced by aerobic exercise before RT remain to be determined, and the present findings require confirmation.

Some investigators (32, 33) have reported that RT does not confer unfavorable effects on vascular function, whereas others (2, 7, 8, 10, 22, 26) have shown the opposite. Whether or not RT hardens arteries has been controversial since Bertovic et al. (2) reported that RT increases arterial stiffness. Therefore, the effects of RT on vascular function remain a matter of controversy. Further investigations must assess the beneficial effects of resistance training on vascular function.

Both the BRT and ART groups had a larger brachial arterial diameter than the SED group in the present study. Dinello et al. (9) observed expansion of the femoral arterial lumen diameter in previously sedentary middle-aged and elderly men after 3 mo of aerobic exercise intervention (primarily walking). Moreover, other studies reported that RT enlarges brachial and femoral artery diameter (23, 32). Thus both aerobic exercise and RT increase brachial or femoral arterial diameter. These results suggest that both types of training cause enlargement at the level of the major conduit arteries. The present findings extend these results from endurance-trained individuals to those who perform RT. Arterial expansion seems to relate to structural remodeling or reduced vascular smooth muscular tone and helps to decrease peripheral arterial stiffness. The beneficial effects of an exercise program on vascular function probably relate to the effect of increased flow on the vascular endothelium. Shear stress plays an important role in changes in vascular endothelial function induced by aerobic exercise (25). However, the present study found that aerobic exercise after, but not before, RT improved NO-mediated vascular function. These results are similar to those of an intervention study using RT (32). The improved vascular function in the ART group suggests that enhanced vascular function at the arteriolar level contributes to the increased brachial artery diameter.

Moreover, blood pressure and heart rate in this study remained unchanged among the three groups. Although we cannot exclude the effects of sympathetic nervous tone after resistance training (20), all participants in this study were normotensive. We therefore considered that alterations in vascular function in the ART group primarily resulted from changes in arterial distension. In addition, brachial MBV, hyperemic BV, BF, hyperemic BF, VC, and VR in the BRT and ART groups significantly changed from baseline. Short-term RT increases basal femoral BF and VC in healthy middle-aged and older adults (1). On the other hand, a previous study indicated that calf VR is reduced after aerobic exercise (14). Anton et al. (1) suggested that RT affects basal limb perfusion through a mechanism underlying its effects on glucose uptake. Thus hemodynamic improvement induced by aerobic and resistance training is important. That is, although aerobic exercise before and after RT can improve hemodynamics, aerobic exercise before RT does not seem to improve vascular function. However, the physiological mechanisms underlying the
changed hemodynamics in RT remain obscure. Further studies are required to determine the physiological mechanisms underlying the effects of RT on arterial hemodynamics.

The value of 1RM in the ART group was significantly increased compared with the BRT group. Previous study has found that the growth hormone (GH) response to resistance exercise is attenuated by prior endurance exercise (12). Therefore, aerobic exercise after RT might intensify the training effect. However, Kawano et al. (16) reported that the strength gains were consistently smaller in a group that performed combined training compared with a group that had performed only high-intensity RT, especially in the lower limbs. Accordingly, combined training may favorably affect vascular function but suppress increases in muscular strength. Moreover, the beneficial effects of an exercise program on vascular function may relate to the effect of increased muscle strength. GH indicates marked activation of the endothelium-dependent component of vasodilation, and the vascular effects of GH are linked to the activity of the NO pathway (24). That is, because aerobic exercise before RT reduces GH response, it may not have contributed to the improvement of the vascular function, although GH response was not measured. Furthermore, because aerobic exercise before and after RT may change metabolism, it may affect the improvement of vascular function.

In conclusion, the present findings indicate that aerobic exercise after, but not before, RT improves vascular function. That RT might favorably influence leg perfusion and hemodynamics suggests that not only quantitative but also qualitative changes in skeletal muscle and/or alterations in non-skeletal muscle components induced by resistance training are responsible for the absence of age-related reductions in basal leg blood flow in resistance-trained men (23). Although we investigated the arms, our findings support those of the previous study. However, although aerobic exercise after RT improved vascular function in the present study, aerobic exercise before RT did not. Thus we speculate that although habitual RT promotes an increase in blood flow through an impact on skeletal muscle mass, it does not improve vascular function. Further studies are warranted to determine the effects of resistance exercise on arterial hemodynamics and vascular function.

REFERENCES

27. Paffenbarger RS, Hyde RT, Wing AL, Lee IM, Jung DL, Kampert JB. AHA Science Advisory. Resistance exercise in individuals with and without cardiovascular disease: benefits, rationale, safety, and prescription: an advisory from the Committee on Exercise, Rehabilitation, and...


