Large elastic arteries in the central region (e.g., aorta) and midsized muscular arteries (e.g., femoral artery) have two functions, i.e., as a low-resistance conduit and as a cushion (or buffer) of flow pulsations at its input [1]. An artery with lower stiffness may have a higher buffering capacity and can efficiently absorb the energy during the systolic component of pulsatile blood flow and reduce the energy loss by making the blood flow smooth. During exercise, it may be favorable to increase the arterial buffering capacity by a decrease of arterial stiffness, because blood flow should be markedly increased to meet oxygen demand in active muscle. Kingwell et al. [2] demonstrated that a 30-min bout of moderate cycling using both legs decreased pulse wave velocities (PWV, an index of arterial stiffness) from the aorta to the femoral artery and from the femoral to the dorsalis pedis arteries 30 min after the exercise. Arterial stiffness is determined by both the properties of the arterial wall matrix and the vascular smooth muscle tone. Such acute change in arterial stiffness is probably mediated by an alteration of vascular muscle tone. Although the previous study [2] assumed the possible influence of systemic (e.g., sympathetic nervous activity, circulating hormones) and regional (e.g., endothelial-derived vasoactive substances, exercised muscle-derived metabolites) factors in altering the muscle tone, the dominant factor has not been clarified.

We hypothesized that the exercise-induced decrease in arterial stiffness, at least in midsized muscular arteries, would be mainly caused by exercise-related regional factors. [The Japanese Journal of Physiology 53: 239–241, 2003]

Abstract: We examined the effect of low-intensity single-leg exercise (20 or 30 watt, 5 min) on pulse wave velocity from the femoral to the ankle arteries in 18 young men. After the exercise, the velocity significantly decreased in the exercised leg, but not in the counterpart, suggesting that the decrease in arterial stiffness in the exercised leg was induced mainly by exercise-related regional factors. [The Japanese Journal of Physiology 53: 239–241, 2003]

Key words: arterial stiffness, pulse wave velocity, exercise.
30 W workload, using a cycle ergometer (232C-EX, Combi Co., Tokyo, Japan). The f-a PWV in each leg was measured just before and 2 min after the exercise by the automatic PWV measurement system (form-PWV/ABI, Colin Co., Aich, Japan). The PWV was determined from the distance between the two recording sites of arterial pressure pulse wave and the delay time of wave travel. The pressure waveforms were simultaneously recorded for 8 s with an applanation tonometry probe at the right common femoral artery and with oscillometric sensor cuffs at the ankle arteries. The delay times between the start of the sharp systolic upstroke of the femoral and ankle arterial pulse waves were automatically determined with an algorithm included in form-PWV/ABI (Fig. 1). The distance between the placement point of the applanation tonometry sensor on the femoral artery and the top of the medial malleolus was measured in duplicate, and the mean value was calculated. The systolic, diastolic, and mean blood pressures and the heart rate were measured before, during (4 min after the start of exercise), and 2 min after the exercise with a finger cuff using Portapres 2.0 (TNO-Biomedical Instrumentation; Amsterdam, The Netherlands).

The results were analyzed by ANOVA with repeated measures. In regard to significant $F$ values, the Student-Newman-Keuls post hoc test was used to identify significant differences among mean values. Statistical significance was set at $p < 0.05$ for all comparisons. Values are reported as means±SE.

## Results and Discussion

All measurements are shown in Table 1. The systolic and mean blood pressures significantly increased during the exercise and returned to the preexercise levels (baseline) 2 min after the exercise. The diastolic blood pressure increased slightly but not significantly ($p = 0.124$) during the exercise. The heart rate significantly increased. It decreased almost to the baseline level 2 min after the exercise, but was slightly and significantly higher than that of the baseline. The f-a PWV significantly decreased in the exercised leg, but not in the counterpart.

The PWV depends not only on the wall stiffness (elasticity), but also on the blood density and the ratio of wall thickness to the cross-sectional area [1]. Moreover, the PWV is increased significantly when the heart rate is more than 120 bpm [3]. Therefore we measured f-a PWV after a low-intensity, short-duration exercise in the present study to minimize these factors, and thereby the heart rate returned almost to the baseline level 2 min after the exercise. Possible dehydration to increase the blood density and a possible increase in local body temperature (i.e., in the exercised leg) to expand the arterial cross-sectional area might be negligible with a low-intensity, short-dura-

![Fig. 1. Typical changes in arterial pressure wave transitions before and after a low-intensity single-leg exercise. Arterial pressure waveforms were recorded at the right common femoral artery (FA) and ankle arteries (AA) in the exercised legs.](image)

### Table 1. Changes in heart rate, blood pressure, and pulse wave velocity.

<table>
<thead>
<tr>
<th></th>
<th>Before exercise</th>
<th>During exercise</th>
<th>After exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate (bpm)</td>
<td>54 ± 2</td>
<td>82 ± 2*</td>
<td>58 ± 2*</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>117 ± 2</td>
<td>132 ± 3*</td>
<td>119 ± 2</td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>70 ± 1</td>
<td>74 ± 3</td>
<td>70 ± 1</td>
</tr>
<tr>
<td>Mean blood pressure (mmHg)</td>
<td>87 ± 1</td>
<td>96 ± 3*</td>
<td>87 ± 1</td>
</tr>
<tr>
<td>Pulse wave velocity (m/s)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exercised leg</td>
<td>9.1 ± 0.3</td>
<td>8.4 ± 0.3*</td>
<td>9.2 ± 0.2</td>
</tr>
<tr>
<td>Nonexercised leg</td>
<td>9.0 ± 0.2</td>
<td></td>
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</table>

* $p < 0.05$, vs. before exercise; † $p <$, vs. nonexercised leg.
tion exercise, though the influence of these factors could not be completely excluded. Therefore it is considered that the exercise-induced decrease in the f-a PWV in the exercised leg may mainly reflect the decrease in the wall stiffness.

Kingwell et al. [2] showed that the PWV of both the aorta and the femoral arteries in young men significantly decreased 30 min after moderate intensity exercise (30 min, 65% VO₂max). They proposed that some regional factors (increases in temperature, exercising muscle-derived metabolites, e.g., adenosine, potassium, and flow-mediated vasodilators, e.g., nitric oxide: NO) might contribute to vasodilation and could account for the decrease in peripheral and central arterial stiffness. In the present study, postexercise PWV changes in the peripheral arteries were observed only in the exercised leg. To minimize the effect of some systemic factors, which could affect the PWV (e.g., changes in heart rate, body temperature, sympathetic nervous activity, and circulatory hormones), we conducted a low-intensity and short-duration exercise and measured the f-a PWV after the exercise. The findings may strongly suggest that the changes in arterial stiffness were induced mainly by regional factors.

Vascular smooth muscle tone is regulated via various vasoactive substances synthesized by vascular endothelial cells, e.g., NO, prostacyclin, and endothelial-derived hyperpolarizing factor (EDHF). In particular, the production of NO, which is a potent endothelial-dependent vasodilator and, moreover, which reduces the vasoconstrictor response to α-adrenergic receptor stimulation [4], is increased with increased blood flow and cyclic wall stress associated with pulsatile blood flow and acute exercise [5]. Even during a low-intensity exercise, the blood flow to active muscles may increase, whereas the blood flow to inactive muscles might not. A recent study [6] showed that a lower leg exercise at a high intensity increased the blood flow in the forearm and that a systemic inhibition of NO synthase (the infusion of N⁰-monomethyl-L-arginine) inhibited the forearm blood flow increase, suggesting an increased production of NO in the resting forearm vessels. Therefore the high-intensity single-leg exercise may decrease the arterial stiffness in the nonexercised leg. In the present study, it is possible that the exercise-induced release of NO with an increased blood flow might have occurred in the exercised leg, but might not have occurred, or at least it would have been smaller, in the counterpart. Thus the exercise-induced release of NO might be a possible factor explaining the difference between the postexercise changes in the arterial stiffness in the legs. Our data, however, cannot specify which factor decreases the peripheral conduit arterial stiffness with exercise. This is a limitation of the present study.

In summary, our findings suggest that the acute decrease in stiffness in the midsize conduit artery with a low-intensity, short-duration exercise may be predominantly mediated by regional factors rather than by systemic factors. Further studies (e.g., using vasoactive substance inhibitors with a higher intensity exercise and in a central artery) are needed to investigate the mechanism and implication of the change in the conduit arterial stiffness.

REFERENCES