Regular endurance exercise in young men increases arterial baroreflex sensitivity through neural alteration of baroreflex arc

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Submitted 5 November 2008; accepted in final form 16 March 2009

Regular endurance exercise in young men increases arterial baroreflex sensitivity through neural alteration of baroreflex arc. J Appl Physiol 106: 1499–1505, 2009. First published March 19, 2009; doi:10.1152/japplphysiol.91447.2008.—Endurance exercise training increases arterial baroreflex sensitivity that corresponds to alteration in vessel wall compliance of the carotid artery in elderly men. Here, we examined whether regular endurance exercise increases arterial baroreflex sensitivity through neural alteration of the baroreflex arc in young men. We assessed arterial baroreflex sensitivity in eight sedentary men (age 24 ± 1 yr) and nine men trained in endurance exercise (age 23 ± 1 yr) during phase IV of the Valsalva maneuver [systolic arterial blood pressure (SAP)–R-R interval relationship]. Arterial baroreflex sensitivity was further analyzed by dividing the mechanical component [SAP–end-systolic carotid lumen diameter relationship (ultrasonography)] and the neural component (end-systolic carotid lumen diameter–R-R interval relationship). Carotid arterial compliance was determined using B-mode ultrasound and arterial applanation tonometry on the common carotid artery. Arterial baroreflex sensitivity and its neural component were greater in the exercise-trained group (P < 0.05). In contrast, carotid arterial compliance and the mechanical component of arterial baroreflex sensitivity did not differ between groups. These results suggest that regular endurance exercise in young men increases arterial baroreflex sensitivity through changes in the neural component of the baroreflex arc and not through alterations in vessel wall compliance of the carotid artery.

aerobic exercise; exercise training; Valsalva maneuver; ultrasound imaging; arterial stiffness

THE ARTERIAL BAROREFLEX is a negative-feedback control mechanism that helps maintain constant arterial blood pressure. Decreased arterial baroreflex sensitivity (BRS) is associated with impaired regulation of arterial blood pressure (29) and with a risk for life-threatening arrhythmias (2, 4). Thus factors that affect BRS and the mechanisms underlying these influences are of physiological and clinical interest.

Endurance exercise training favorably increases BRS (6, 12, 13, 15, 17, 20–23, 27). There are at least two possible mechanisms responsible for the exercise-induced increase in BRS. One is alterations in vessel wall compliance of the carotid artery, because stretch-sensitive baroreceptors are embedded in the carotid artery wall. Indeed, Seals and colleagues have demonstrated that regular endurance exercise inhibits the age-associated decrease in carotid arterial compliance (21, 23, 31), and identified a positive relationship between exercise-induced changes in carotid arterial compliance and BRS (21, 23). The other possible mechanism is neural alterations in the baroreflex arc. We previously demonstrated that the central characteristics of the arterial baroreflex dynamically change from moment to moment during exercise (14). Additionally, exercise training in rats attenuated the sympathoexcitation evoked by a GABA agonist injection into the nucleus tractus solitarius (NTS), which is a site constituting the baroreflex arc in the brain (24). These findings indicate that exercise training as well as acute exercise changes BRS through central alterations in the baroreflex arc. However, studies of elderly men showed that the neural alterations of arterial baroreflex are inconsistent. Hunt et al. (11) reported that the neural BRS estimated by carotid arterial diameter and the R-R interval was higher in trained elderly men than in sedentary peers, whereas Monahan et al. (23) found no differences in the neural BRS between the two populations. The neural alteration might be hard to be find in elderly men, because the exercise-induced increase in BRS in the elderly mostly depends on the changes in carotid arterial compliance that decreases with aging (28), despite neural alterations of the baroreflex arc (11).

The neural alterations of the baroreflex arc should be clarified in young men, because exercise training also induces an increase in BRS in this population (6, 13, 20) but does not alter their carotid arterial compliance (9, 18, 31). We hypothesized that regular endurance exercise in young men increases BRS through alterations in the neural component of the baroreflex arc rather than via an increase in carotid arterial compliance. We therefore investigated BRS (systolic arterial blood pressure and R-R interval relationship) and carotid arterial compliance in sedentary and trained young men in the present study. BRS was further assessed by being divided into the mechanical component that reflects the transduction of arterial blood pressure into carotid diameter changes and the neural component that reflects the transduction of carotid diameter changes into R-R interval responses.

METHODS

Subjects

We studied 17 healthy male volunteers between the ages of 19 and 27 yr old. Volunteers were divided into two groups, sedentary (n = 8) and endurance trained (n = 9), on the basis of their exercise habits. The sedentary men had not regularly exercised for at least 2 years. The trained men had run for 8.0 ± 1.1 min/day, 5.2 ± 0.1 days/wk for 6.6 ± 1.3 yr. All participants were normotensive, nonobese, nonsmokers, taking no medications, and free of overt cardiovascular diseases as assessed by medical history. The purpose and procedures of this study were explained to the subjects, and written informed consent to participate was obtained. The Ethics Committee of the Institute for Human Science and Biomedical Engineering of the National Institute of Advanced Industrial Science and Technology reviewed and approved the study protocols.
**Experimental Protocol**

All experiments proceeded in the morning after a 12-h overnight fast. Subjects abstained from alcohol and caffeine for at least 12 h before the experiment. The trained group did not exercise for a minimum of 16 h before starting the experiment to avoid the potential acute effects of exercise. Measurements were taken in a quiet, temperature-controlled room (24–26°C). After 20 min of supine rest, three-lead electrocardiogram (ECG), radial arterial blood pressure, left carotid arterial blood pressure, and ultrasound images of the right carotid artery were obtained in the same position to determine carotid arterial compliance. Thereafter, BRS was determined using the Valsalva maneuver as previously described (30). The subjects maintained an expiratory mouth pressure of 40 mmHg for 15 s by blowing through a short tube. Mouth pressure was measured with a pressure transducer connected, and the pressure values were displayed to provide visual feedback to the subjects. Immediately thereafter, subjects inspired normally, avoiding excessively deep respiration. All subjects performed 5–15 Valsalva maneuvers at 3- to 5-min intervals during which heart rate (HR) and arterial blood pressure returned to the baseline levels. After assessing BRS, maximal oxygen consumption (VO$_{2\text{max}}$) was measured during incremental cycle ergometer exercise. Plasma nitrite/nitrate and endothelin-1 levels were measured in venous blood samples obtained in the morning after a 12-h overnight fast within 1 wk of testing sessions.

**Measurements**

**Arterial blood pressure and R-R interval.** Beat-by-beat radial arterial blood pressure was continuously measured by applanation tonometry (JENTOW 7700, Colin Medical Technology, Komaki, Japan). This equipment can calibrate radial arterial blood pressure with brachial arterial blood pressure obtained from a conventional pressure cuff wrapped around the upper arm. Beat-by-beat left carotid arterial blood pressure was noninvasively measured by applanation tonometry with a high-fidelity strain-gauge transducer (TCB-600, Millar Instruments, Houston, TX). Because the baseline level of carotid arterial blood pressure is subject to hold-down force, the pressure signal obtained by tonometry was calibrated by equating the carotid mean arterial and diastolic blood pressure to the radial artery value (1). The R-R interval and HR were continuously measured by ECG. Radial and carotid arterial blood pressure, ECG, and mouth pressure during the Valsalva maneuver were simultaneously recorded on an eight-channel pen-writing recorder (8K23, GE Marquette Medical Systems, Tokyo, Japan) and stored in a computer via an analog-to-digital converter (PowerLab16/30, ADInstruments, Sydney, Australia) at a sampling frequency of 1 kHz for offline analysis.

**Carotid ultrasonography.** Longitudinal B-mode images of the right common carotid artery were obtained using ultrasound equipment (SonoSite 180PLUS, SonoSite, Bothell, WA) with a high-resolution linear-array transducer (10 MHz) placed at 1 to 2 cm proximal to the carotid bulb, with an ~90° angle to the vessel so that the near and far wall interfaces were clearly discernible. The ultrasound images were recorded on digital videotapes for offline analysis.

**Blood samples.** Plasma nitrite/nitrate and endothelin-1 might affect arterial compliance (25). We measured levels of these factors using the Greiss assay and radioimmunoassay, respectively, in plasma obtained from venous blood samples that were centrifuged at 3,000 rpm for 15 min and then stored at ~80°C.

**VO$_{2\text{max}}$.** To assess aerobic fitness, VO$_{2\text{max}}$ was measured during incremental cycle ergometer exercise with online computer-assisted circuit spirometry (AE280S; Minato Medical Science, Osaka, Japan). All subjects underwent an incremental cycle exercise test (after 3 min at 10 W, with 20-W increases every 1 min) until volitional exhaustion. VO$_{2\text{max}}$ was defined at the highest value recorded during the test. HR and the rating of perceived exertion (Borg scale) were measured throughout the exercise, and total exercise duration required to reach exhaustion was recorded.

**Data Analysis**

**Assessment of baroreflex sensitivity.** BRS was estimated as the R-R interval corresponding to radial systolic arterial blood pressure (SAP) during phase IV of the Valsalva maneuver. The SAP values were linearly regressed against corresponding (lag 1) R-R intervals on a beat-to-beat basis from the point at which the R-R interval began to lengthen, and continued to the point of maximal SAP elevation. The slope of the R-R interval and the SAP relationship was used as a measure of BRS if the correlation coefficient exceeded $r = 0.80$, because of excellent correlation between this procedure and the Oxford technique (26). BRS was further assessed by being divided into mechanical and neural components. The mechanical component of arterial baroreflex sensitivity was determined by the end-systolic carotid lumen diameter corresponding to radial SAP during phase IV of the Valsalva maneuver. The neural component of arterial baroreflex sensitivity was determined by the R-R interval, which corresponds to the end-systolic carotid lumen diameter. End-systolic carotid lumen diameter was analyzed using B-mode ultrasound images as described in the analysis of carotid arterial compliance. In our laboratory, the day-to-day coefficients of variation in a pilot study on five subjects were 7.4 ± 0.6%, 9.2 ± 0.3%, and 7.7 ± 0.4%, for BRS, the mechanical component of BRS, and the neural component of BRS, respectively.

**Carotid arterial compliance.** Carotid arterial compliance was determined using a combination of ultrasound imaging of the common carotid artery and simultaneous applanation tonometry of the carotid or radial arteries. Ultrasound images recorded on digital videotapes were stored in a computer at 30 Hz and analyzed using image-analysis software (ImageJ 1.32J, National Institutes of Health, Bethesda, MD). One investigator who was blinded to the assignment groups of the participants performed all image analyses. Carotid arterial lumen diameter was determined as the distance between the vessel far-wall boundary corresponding to the interface between the lumen and intima. Near-wall boundary was defined as the interface of the adventitia and media, at minimal diastolic relaxation and at maximal systolic expansion of the vessel. Arterial lumen diameter at minimal diastolic relaxation and maximal systolic expansion of the vessel was measured at three points per video frame and then averaged. Each parameter was averaged over 10–15 continuous beats and statistically analyzed. Arterial compliance was obtained using the equation:

$$\frac{(CSAs - CSAd)}{\Delta P}$$

where CSAs and CSAd are cross-sectional areas at the maximal systolic expansion and at the minimal diastolic relaxation of the carotid artery, and $\Delta P$ is the carotid or radial arterial pulse pressure. In addition to arterial compliance, the $\beta$-stiffness index was analyzed using the equation:

$$\ln(cSAP/cDAP) \times \frac{D}{\Delta D}$$

where cSAP and cDAP are systolic and diastolic carotid or radial arterial blood pressure, $D$ is end-diastolic carotid lumen diameter, and $\Delta D$ is the change in carotid lumen diameter from end diastole to peak systole (10). The $\beta$-stiffness index provided an index of arterial compliance adjusted for distending pressure (10). To examine the relation to the mechanical component of baroreflex function based on the radial arterial pressure, carotid compliance was calculated via the radial pressure as well as carotid pressure.

**Statistical Analysis**

We compared data from the sedentary and trained groups using unpaired t-tests. The level of statistical significance was defined as $P < 0.05$ in all cases. Data in the text, table, and figures are expressed as means ± SE.
RESULTS

Cardiovascular Baseline Values and Physical Characteristics

Table 1 shows the physical characteristics of the participants and the baseline cardiovascular values at rest. There were no group differences in age, height, body mass, and ratio (%) of body fat between the two groups, whereas VO_{2max} was higher and HR was lower in the trained group (P < 0.05). Arterial blood pressure measured at the radial artery, carotid lumen diameter, and venous levels of nitrite/nitrate and endothelin-1 were not significantly different between groups.

Arterial Baroreflex Sensitivity

Figure 1 shows typical responses of radial arterial blood pressure and ECG during the Valsalva maneuver. Arterial blood pressure increased and the R-R interval lengthened during phase IV of the Valsalva maneuver, which occurs after mouth pressure is released (Fig. 1). Figure 2A shows typical linear regression between radial SAP and the R-R interval during phase IV of the Valsalva maneuver. The slope of the regression line, which was determined as BRS, was steeper in the trained subject. Figure 3A shows that the average slope of the regression (average BRS) was greater in the trained than in the sedentary group (18.9 ± 2.1 vs. 12.1 ± 1.3 mmHg; P < 0.05). The SAP at the start of phase IV of the Valsalva maneuver (minimal SAP) and the increase in SAP during phase IV did not differ between the sedentary and trained groups (minimal SAP, 112 ± 4 vs. 111 ± 2 mmHg; increase in SAP, 26 ± 3 vs. 19 ± 2 mmHg in the sedentary and the trained group, respectively). Acceptable data (e.g., correlation coefficient between the R-R interval and the SAP ≥ 0.8) were acquired with 7.9 ± 1.2 and 5.9 ± 1.2 trials in each sedentary and trained individual.

Mechanical and Neural Components of Arterial Baroreflex Sensitivity

Figure 2B shows linear regression between the SAP and the carotid lumen diameter during phase IV of the Valsalva maneuver obtained from one sedentary and one trained subject derived from trials by the same subjects shown in Fig. 2A. The slope of the regression line (the mechanical component of BRS) was comparable between sedentary and trained subjects. As shown in Fig. 3B, there was no significant difference in mean slopes of regression line between groups (sedentary 0.0255 ± 0.0039 vs. trained 0.0305 ± 0.0039 mmHg/mmHg). Figure 2C shows linear regression between carotid lumen diameter and R-R interval in the same sedentary and trained individuals derived from the trials shown in Fig. 2, A and B. The slope of the regression line, the index of the neural component of BRS, was steeper in the trained than in the sedentary participant. As shown in Fig. 3C, the average slope of the regression was greater in the trained than in the sedentary group (654 ± 64 vs. 460 ± 48 ms/mmHg, P < 0.05). The neural and mechanical components of arterial baroreflex sensitivity were assessed only when clear longitudinal B-mode images of the common carotid artery were obtained during phase IV of the Valsalva maneuver (data from 3.6 ± 0.6 and 3.6 ± 1.0 trials per subject analyzed in 7 of 8 of the sedentary group and in 8 of 9 subjects of the trained group, respectively). We reanalyzed BRS in the same trials used to determine the mechanical and neural components of BRS and reconfirmed that arterial baroreflex was more sensitive in the trained than in the sedentary group (19.8 ± 1.8 vs. 12.2 ± 1.4 ms/mmHg, P < 0.05).

BRS was also assessed using carotid arterial blood pressure to examine the transduction of carotid arterial (but not peripheral) blood pressure, into a change in carotid diameter although the data were only preliminary. This was because we could only record carotid arterial blood pressure in limited trials of seven participants in each of the sedentary and trained groups (1.7 ± 0.4 and 1.6 ± 0.4 trials per person, respectively). BRS analyzed using carotid arterial blood pressure was similar to that determined with radial arterial blood pressure; BRS and its neural component were decreased in the sedentary, compared with the trained group, whereas the mechanical component of BRS did not differ between the groups (BRS, 12.9 ± 1.3 vs. 21.6 ± 2.5 ms/mmHg, P < 0.05; neural component, 423 ± 56 vs. 654 ± 51 ms/mm, P < 0.05; mechanical component, 0.0294 ± 0.0036 vs. 0.0322 ± 0.0041 mm/mmHg in sedentary and the trained groups, respectively).

Table 1. Subject characteristics

<table>
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<tr>
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<th>Sedentary</th>
<th>Trained</th>
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<td>No. of subjects, n</td>
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<td>9</td>
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<td>Age, yr</td>
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<td>Height, cm</td>
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<td>Body mass, kg</td>
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<td>Body fat, %</td>
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<td>VO_{2max}, ml·kg^{-1}·min^{-1}</td>
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<td>HR, beats/min</td>
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<td>DAP, mmHg</td>
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<tr>
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<tr>
<td>ET-1, pg/ml</td>
<td>3.4 ± 0.3</td>
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</table>

Values are means ± SE. VO_{2max}, maximal oxygen consumption; HR, heart rate; SAP, systolic arterial blood pressure; MAP, mean arterial blood pressure; DAP, diastolic arterial blood pressure; NOx, nitrite/nitrate; ET-1, endothelin-1.

*Significant difference (P < 0.05) between sedentary and trained groups.
Carotid Arterial Compliance

Figure 4A shows that carotid arterial compliance calculated with carotid arterial pressure did not differ between the trained and sedentary groups (1.44 ± 0.12 vs. 1.38 ± 0.12 mm²/mmHg × 10⁻¹). As shown in Fig. 4B, the β-stiffness index also did not differ between the trained and sedentary groups (5.5 ± 0.4 vs. 5.4 ± 0.5 µ). Carotid arterial compliance and β-stiffness index calculated with radial arterial blood pressure did not also differ between the trained and sedentary group (carotid arterial compliance, 1.11 ± 0.08 vs. 1.02 ± 0.09 mm²/mmHg × 10⁻¹; β-stiffness index, 6.6 ± 0.4 vs. 6.8 ± 0.6 µ). The correlation between carotid arterial compliance or β-stiffness index calculated with carotid arterial blood pressure and that with radial arterial blood pressure were excellent and significant (carotid arterial compliance, r = 0.95, P < 0.05; β-stiffness index, r = 0.96, P < 0.05).

Figure 5 shows the relationship between carotid arterial compliance and BRS across the sedentary and the trained groups. BRS was related to carotid arterial compliance (r = 0.48, P < 0.05). The relationship was rather distributed upward (toward higher BRS) in the trained group compared with the sedentary group.

DISCUSSION

We examined whether regular endurance exercise in young men increases BRS due to alterations in the neural component of the baroreflex arc rather than an increase in carotid arterial compliance. The novel findings of the present study were as follows. BRS assessed by radial SAP and corresponding R-R interval was significantly greater in the trained than in the sedentary age-matched young men and carotid arterial compliance did not differ between the two groups. Second, the neural component of BRS, determined by the carotid lumen diameter and corresponding R-R interval, was greater in the trained than...
in the sedentary young men. Third, the mechanical component of BRS, determined by radial SAP and corresponding carotid lumen diameter, did not differ between the two groups. These data suggest that regular endurance exercise in young men increases BRS through alterations in the neural component of the baroreflex arc and not to changes in vessel wall compliance of the carotid artery.

Effects of Exercise on Arterial Baroreflex Sensitivity

Regular endurance exercise increased BRS in young men in the present study and in elderly men in other studies, but the underlying mechanisms seem to differ. The increase in carotid artery compliance due to endurance exercise has explained much of the exercise-associated increase in BRS in the elderly (21–23), despite neural alterations of the baroreflex arc (11). Low baseline carotid arterial compliance associated with aging should increase with endurance exercise training, thus improving BRS. Consequently, neural alterations, if any, of the baroreflex arc might be masked. In contrast, the exercise-induced increase in BRS in young men in the present study was not due to change in carotid artery compliance, but to the neural alteration. If baseline carotid arterial compliance is high in young men, exercise would be unlikely to further increase carotid arterial compliance. Thus BRS would increase depending on changes in the neural alteration. In support of this notion, endothelium-dependent vasodilation, which is associated with carotid arterial compliance (5), is augmented by endurance exercise in elderly men but not in young men or women (7). Collectively, regular endurance exercise in young men with compliant carotid arteries increases BRS depending on changes in the neural component of the baroreflex arc, whereas that in elderly men with stiffer carotid arteries increases mostly depending on alterations in carotid arterial compliance.

Although carotid arterial compliance was not changed by regular endurance exercise, we found proportionality between carotid arterial compliance and BRS across the sedentary and trained groups (Fig. 5). This suggests that BRS corresponds to individual carotid arterial compliance, irrespective of physical activity. This proportional relationship is in agreement with previous findings in young healthy humans (3). Interestingly, the relationship between carotid arterial compliance and BRS in the trained group in the present study was distributed upward (toward higher BRS) compared with that in the sedentary group. This upward distribution reflects the exercise-induced increase in BRS without changing carotid arterial compliance. Baroreflex afferents that correspond to carotid arterial compliance might interact with exercise-induced factors to increase BRS at the neural component of the baroreflex arc and thus alter efferent autonomic outflow to the heart. We postulate that BRS changes correspond to the wall compliance of the carotid artery even though regular endurance exercise does not alter such compliance.

The precise mechanisms through which regular endurance exercise augmented the neural component of the BRS in the present study remain elusive. Changes in the neural component would include alterations in baroreceptor sensitivity, central integration of baroreceptor input, efferent autonomic outflow, and sinoatrial node responsiveness. A recent study has shown that exercise training in rats attenuates sympathoexcitation through alterations in the GABAergic neurotransmission at the level of the NTS, which is a site constituting the baroreflex arc in the brain (24). Although BRS assessed during the Valsalva phase IV is predominantly mediated by vagal nerve (19), changes in neural transmission in the brain might also contribute to the neural alteration of arterial baroreflex in young men.

Fig. 4. Carotid arterial compliance (A) and β-stiffness index (B) in sedentary and exercise-trained groups.

Fig. 5. Relationship between carotid arterial compliance and arterial baroreflex sensitivity. ○, Sedentary individual; ●, exercise-trained individual.
Clinical Significance

BRS decreases with advancing age even in young healthy population (16). Because reduced BRS is associated with impaired regulation of arterial blood pressure (29) and a risk of life-threatening arrhythmias (2, 4), the present finding of increased BRS by exercise has important clinical implications. Furthermore, the present finding of the neural alteration in baroreflex extends the clinical relevance. Since physical activity induces neurological adaptations much sooner than structural adaptations (8), an increase in BRS caused by neural alterations might be accomplished by short-term or low-intensity exercise training.

Limitations

Several potential limitations in this study should be discussed. First, we did not assess diameter and arterial blood pressure changes in the aorta, at which the arterial baroreceptors are located. Since a recent study reported that endurance exercise in young men augmented aortic or systemic arterial compliance (25), it remains a possibility that the increased aortic compliance might contribute to the exercise-induced increase in BRS. Second, BRS estimated with radial blood pressure might not reflect blood pressure of the barosensory blood vessel. However, BRS estimated with carotid arterial blood pressure was similar to that determined with radial blood pressure. Additionally, the neural BRS is not affected by arterial pulse wave transmission. Furthermore, carotid arterial compliance estimated with carotid arterial blood pressure did not differ between the two groups. We therefore believe that radial arterial blood pressure did not substantially influence our results or their implications. Third, our study was cross sectional. Thus physiological differences other than aerobic fitness might have influenced BRS. However, cross-sectional studies can more readily evaluate the long-term effects of exercise than longitudinal studies. Indeed, the trained men in our study had exercised for 6.6 ± 1.3 yr. Finally, the Valsalva maneuver used to estimate BRS is a complex stimulus for cardiovascular reflexes (30). For example, acute baroreflex resetting may have occurred (30). A difference in resetting between the trained and sedentary groups could have influenced our results, although we are not aware of any evidence to support this notion.

In conclusion, regular endurance exercise in young men increases BRS through alterations in the neural component of the baroreflex arc, which encompasses the baroreflex afferent and efferent pathways, and central integration of the baroreceptor input.

ACKNOWLEDGMENTS

We are very grateful to Dr. Kanji Matsukawa for reviewing the manuscript and for providing excellent constructive advice regarding the study.

GRANTS

This study was supported by Grants-in-Aid for Scientific Research (B), Young Scientists (B) from the Japan Society for the Promotion of Science (JSPS), and JSPS Postdoctoral Fellowships for Research.

REFERENCES


