Effects of Vigorous Resistance Training On Cardiovagal Baroreflex Sensitivity: A Cross-Sectional Study

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Abstract

Background: Blunted cardiovagal baroreflex sensitivity (BRS) is correlated with elevation of central arterial stiffness and/or reduction of arterial compliance with aging. Resistance-trained who habitually perform vigorous resistance training (RT) may blunt cardiovagal BRS due to an increase in central arterial stiffness and a decrease in arterial compliance. Therefore, the purpose of this study was to examine the effect of vigorous RT on cardiovagal BRS in a cross-sectional study to compare resistance-trained with age-matched non-resistance-trained.

Methods: This cross-sectional study included resistance-trained men (resistance-trained group: n = 20) and age-matched non-resistance-trained men (control group: n = 20). The β-stiffness index and arterial compliance were assessed at the right carotid artery, and the cardiovagal BRS was estimated by the slope of the R-R interval and systolic blood pressure during phase IV of the Valsalva maneuver.

Results: The β-Stiffness index was significantly higher in the resistance-trained group than in the control group (4.4 ± 0.2 vs. 5.9 ± 0.3 a.u., $P < 0.01$). In contrast, the resistance-trained group had significantly lower arterial compliance and cardiovagal BRS than the control group (0.20 ± 0.01 vs. 0.15 ± 0.01 mm^2/mmHg and 12.9 ± 1.2 vs. 9.0 ± 0.5 ms/mmHg, respectively, $P < 0.01$ for both measurements). Moreover, cardiovagal BRS was inversely and positively correlated with the β-stiffness index (combined: r = - 0.59, $P < 0.01$; control: r = - 0.56, $P < 0.01$; resistance-trained: r = - 0.50, $P < 0.05$) and arterial compliance (combined: r = 0.64; control: r = 0.61; resistance-trained: r = 0.55, all $P < 0.01$), respectively.

Conclusion: Resistance-trained group was higher arterial stiffness and lower compliance and blunter cardiovagal BRS compared with control group. These results suggest that vigorous RT blunts cardiovagal BRS.

Trial registration: University hospital Medical Information Network (UMIN) in Japan, UMIN000038116. Registered on September 27, 2019.

Key Points

- Blunted cardiovagal baroreflex sensitivity (BRS) is correlated with elevation of central arterial stiffness and/or reduction of arterial compliance with aging.
- Although vigorous resistance training (RT) increases central arterial stiffness and decreases arterial compliance, it is unclear effect of these adaptations on cardiovagal baroreflex sensitivity.
- Vigorous RT should be performed carefully because there is a risk of blunting cardiovagal BRS.

Introduction

The arterial baroreflex greatly contributes to beat-to-beat control of arterial blood pressure (ABP) through the autonomic nervous system. The cardiovagal baroreflex sensitivity (BRS) is blunted by ageing [1-4]. Blunted cardiovagal BRS is a risk factor for life-threatening arrhythmias and is a predictor of sudden

cardiac death [5,6]. The cardiovagal BRS is correlated with age-related changes in central arterial mechanical properties such as stiffness and compliance where the baroreceptors are located (the carotid artery and the aortic arch) [2,3,7].

Recently, several health organizations have recommended resistance training (RT) for muscular hypertrophy and maximizing strength [8,9]. Several studies have reported that RT increases and decreases central arterial stiffness and compliance in healthy individuals, respectively [10-13]. On the other hand, García-Mateo et al. [14] have reported that RT, of at least four weeks duration and two days per week frequency, does not alter central arterial stiffness and compliance. However, resistance-trained individuals who habitually perform vigorous RT (high intensity and training frequency) such as bodybuilders, hammer throwers and weight-lifters, almost certainly have higher central arterial stiffness and lower arterial compliance than age-matched sedentary individuals [15-20]. Vigorous RT may blunt cardiovagal BRS through central arterial adaptation. It is necessary to investigate the potential problem of the central arterial adaptation with vigorous RT.

The purpose of this study was to examine the effect of increased central arterial stiffness and decreased arterial compliance caused by vigorous RT on cardiovagal BRS. To investigate this purpose, we designed a cross-sectional study in which central arterial stiffness, compliance, and cardiovagal BRS, were compared between resistance-trained and age-matched non-resistance-trained individuals.

Methods

Subjects

Forty healthy men (resistance-trained group; n = 20 and control group; n = 20) were recruited to participate in this study. The subjects in the resistance-trained group had been performing vigorous RT for > 2 years, > 5 days/week. None of the subjects in the control group regularly engaged in RT, and performed other exercise training < 3 days/ week. All of the subjects were normotensive (< 140/90 mmHg), had no history of cardiovascular diseases, diabetes, smoking, and were not taking medications such as anabolic steroids.

All of the subjects provided written informed consent to participate prior to the start of the study. All of the procedures and risks of this study were reviewed and approved by the Human Research Committee of Waseda University (approval No. 2019-102). The study adhered to the principles of the Declaration of Helsinki.

Protocol

The studies were performed following a 3-hour fast. Subjects were required to avoid caffeine intake for at least 12 hours and alcohol intake for at least 24 hours before participating in the study. The subjects were evaluated 24 hours after their last exercise session to avoid the acute effects of exercise.

All subjects underwent measurement of haemodynamic and carotid arterial variables, and cardiovagal BRS in a temperature- and humidity-controlled environment (Temperature: 22.0 ± 0.1 °C, Humidity: 50.0 ± 0.3 %) after 15 min in a resting supine position.

Body composition

Body composition was measured using bioelectrical impedance analysis (InBody 720, InBody Japan Inc., Tokyo, Japan) with the subject in the upright position.

Muscular strength

Handgrip strength was measured using a grip dynamometer (Grip-D, Takei Scientific Instruments Co., Ltd., Japan) as an index of muscular strength with the subject in the standing position. The subjects were instructed to stand and extend their arms by their sides during a hand grip execution and to grip the dynamometer with full effort for three seconds. The values (kg) were calculated as the average of two trials.

Central arterial stiffness and compliance

The β-stiffness index and arterial compliance of the carotid artery were measured as an index of central arterial stiffness and compliance, respectively. Both the β-stiffness index and arterial compliance were measured in the right carotid artery using a combination of a brightness mode ultrasonography system for the carotid artery diameter, and applanation tonometry for the carotid BP. The carotid artery diameter was obtained 1.0 – 2.0 cm proximal to the carotid bifurcation using an ultrasonography system equipped with a 10-MHz linear transducer (LOGIQ-e, GE Medical Systems, Japan). The diameter was recorded over ten cardiac cycles with the brightness mode in the longitudinal section. The images obtained were analyzed using image analysis software (ImageJ, NIH, USA), and these images were used to analyze the systolic diameter (sD) and diastolic diameter (dD).

The carotid pressure waveform was obtained in the right carotid artery. The obtained pressure waveforms were converted from a pencil-type probe incorporating a high-fidelity strain-gauge transducer (SPT-301, Millar Instruments, TX, USA) at a sampling rate of 1000 Hz through an analogue/digital converter (PowerLab/16SP, AD Instruments, Australia) and recorded in a device connected to a personal computer (Macbook, Apple, USA). Then, the obtained data were analyzed using an analysis software (LabChart5, AD Instruments, Australia). The carotid arterial pressure was calibrated by equating the carotid diastolic blood pressure (DBP) and mean arterial pressure (MAP) to the brachial artery value [21]. The β-stiffness index and arterial compliance were calculated as follows [10,11,17,18]:

$$\beta - \text{stiffness index} = \frac{\ln(\text{carotid systolic blood pressure [SBP]/DBP})}{(\text{sD} - \text{dD})/\text{dD}}$$

$$\text{Arterial compliance} = \frac{(\text{sD} - \text{dD})/\text{dD}}{2(\text{carotid SBP} - \text{DBP})}\pi\text{dD}^2$$

Carotid arterial intima-media thickness

The carotid arterial intima-media thickness (CA IMT) was measured 1.0 – 2.0 cm proximal to the carotid bifurcation with an ultrasonography system equipped with a 10-MHz linear transducer (LOGIQ-e, GE Medical Systems, Japan). The obtained images were analyzed using image analysis software (ImageJ, NIH, USA). At least ten CA IMT measurements were taken, and the mean value was used for analysis [10,11,17,18].

Haemodynamics

HR and beat-to-beat ABP were acquired using a three-lead ECG (BSM-2401, NIHON KOHDEN, Japan) and finger photoplethysmography (Finapres Medical Systems, Amsterdam, The Netherlands), respectively. The photoplethysmograph was attached to the middle finger of the right hand. Additionally, stroke volume (SV) was calculated from the obtained ABP waveform using the Modelflow method [22,23], which incorporates age, height and weight, and simulates aortic flow waveforms from an arterial pressure signal using a nonlinear three-element model of the aortic input impedance (Beatscope, version 1.1, Finapres Medical Systems). CO and TPR were then calculated as SV × HR and MAP / CO, respectively.

Cardiovagal baroreflex sensitivity

The cardiovagal BRS was estimated with the Valsalva manoeuvre test that evaluated the slope of the R-R interval and systolic blood pressure (SBP) during phase IV of the Valsalva manoeuvre [2-4,24]. The subjects maintained an expiratory mouth pressure of 40 mmHg for 15 seconds by blowing through a short tube connected pressure gauge after deep inspiration. The pressure values were displayed to provide visual feedback to the subjects. Immediately after, subjects were instructed to maintain normal respiration, and to avoid deep respiration. The beat-to-beat R-R interval and SBP were obtained from a three-lead electrocardiogram (ECG) and a finger photoplethysmograph, respectively. The waveform of the two variables were digitally converted at a sampling rate of 1000 Hz through an analogue/digital converter and recorded in a device connected to a personal computer, and analyzed with analysis software. Furthermore, the two variables were linearly regressed from the point at which the R-R interval began to lengthen, and continued to the point of maximal SBP elevation (Fig. 1a). Then, the slope of linear correlation between the R-R interval and the SBP was assessed as a cardiovagal BRS, which was determined if the r value was > 0.8 as previously described (Fig. 1b) [25].

Statistical analysis

All values were presented as mean ± standard error of the mean (SEM). Statistical analyses were performed using statistical analysis software (SPSS version 26.0 for Mac, IBM, Japan). The mean differences in the two groups were examined using the Student's unpaired *t* test. Pearson correlations were used to assess the relationship between cardiovagal BRS and β-stiffness index, and between cardiovagal BRS and arterial compliance. In all of the analyses, the level of significance for all comparisons was set at $P < 0.05$.

Results

Subjects' characteristics

Table 1 shows the characteristics of the subjects. Body weight, body mass index, lean body mass, and hand grip strength were significantly higher in the resistance-trained group than in the control group ($P < 0.05$). However, no significant differences were observed in the other subject characteristics between the two groups.

Haemodynamics and carotid arterial variables

Table 2 shows haemodynamics and carotid arterial variables. The brachial DBP was significantly lower in the resistance-trained group ($P < 0.05$). Brachial and carotid pulse pressure (PP) were significantly higher in the resistance-trained group than in the control group (both, $P < 0.05$). However, no significant differences were observed in other haemodynamic and carotid arterial variables between the two groups at rest.

Central arterial stiffness and compliance, cardiovagal baroreflex sensitivity

The β-Stiffness index was significantly higher in the resistance-trained group than in the control group ($P < 0.01$, Fig. 2a). By contrast, the resistance-trained group demonstrated significantly lower arterial compliance and cardiovagal BRS than the control group ($P < 0.01$, Fig. 2b and Fig. 3, respectively).

Relationship between cardiovagal baroreflex sensitivity and central arterial mechanical properties

The associations between cardiovagal BRS and β-stiffness index, and between cardiovagal BRS and arterial compliance are shown in Fig. 4 (a) and (b), respectively. A negative correlation was found between cardiovagal BRS and the β-stiffness index (Combined: r = - 0.59, $P < 0.01$; Control: r = - 0.56, $P < 0.01$; Resistance-trained: r = - 0.50, $P < 0.05$), and a positive correlation was found between cardiovagal BRS and arterial compliance (Combined: r = 0.64, $P < 0.01$; Control: r = 0.61, $P < 0.01$; Resistance-trained: r = 0.55, $P < 0.01$).

Discussion

The present study showed that the resistance-trained group demonstrated a higher β-stiffness index, lower arterial compliance, and lower cardiovagal BRS compared with the control group. In addition, cardiovagal BRS was inversely and positively correlated with the β-stiffness index and arterial compliance, respectively. These findings suggest that vigorous RT may blunt cardiovagal BRS through central arterial mechanical properties.

Central arterial adaptation with vigorous resistance training

The β-stiffness index was significantly higher in the resistance-trained group than in the control group. On the other hand, the arterial compliance in the resistance-trained group was significantly lower than that in the control group. Several studies have shown that resistance-trained individuals have higher central arterial stiffness and lower compliance than age-matched sedentary individuals [15-20]. Several factors increase central arterial stiffness and decrease compliance such as impaired vascular endothelial function and increased sympathetic vasoconstrictor tone.

Otsuki et al. [20] have suggested that higher central arterial stiffness and lower arterial compliance in RT athletes are associated with a higher plasma endothelin-1 (ET-1) concentration than in the age-matched sedentary individuals and endurance athletes. ET-1 is produced by vascular endothelial cells and is a strong vasoconstrictor [31]. A study has reported that pulse wave velocity, as an index of central arterial stiffness, is increased by the intraarterial infusion of ET-1 [32]. These findings suggest that ET-1 may contribute to the central arterial adaptation associated with vigorous RT.

Elevation of muscle sympathetic nerve activity (MSNA) causes vasoconstriction [33], resulting in increasing arterial stiffness [29]. MSNA and central arterial stiffness are higher in resistance-trained subjects than in endurance-trained subjects, and MSNA is positively correlated with central arterial stiffness [29]. Moreover, norepinephrine (NE) concentration in the blood and central arterial stiffness are increased with upper limb RT, but not with lower limb RT according to Okamoto et al. [12]. The study has also shown a positive correlation between the changes in NE concentration in the blood and central arterial stiffness. These results indicate that changes in sympathetic nerve activity are associated with central arterial stiffness following RT.

Pressor response during acute resistance exercise may also be associated with the elevation of central arterial stiffness and reduction of compliance. RT may alter the arterial load-bearing properties, and thereby cause arterial stiffening, because acute resistance exercise dramatically elevates BP as high as 320/250 mmHg in resistance-trained individuals [34]. Ozaki et al. [13] have demonstrated that carotid arterial compliance is decreased in the high-intensity RT group, and the changes are correlated with SBP elevations during acute exercise sessions.

However, it is unclear whether the effects of these factors on higher central arterial stiffness and lower arterial compliance were found in the present study. Also, evidence is limited regarding the mechanisms underlying the central arterial adaptation with vigorous RT. Thus, further studies are needed to investigate the precise mechanism of increased central arterial stiffness and/or decreased arterial compliance with vigorous RT.

Relationship between cardiovagal baroreflex sensitivity and central arterial mechanical properties

The present study found a negative correlation between cardiovagal BRS and β-stiffness index, and a positive correlation between cardiovagal BRS and arterial compliance. These findings may support previous studies, which report that cardiovagal BRS is correlated with alteration of central arterial stiffness and compliance [2,3,7]. A previous animal study has found that low intensity resistance training

improves cardiovagal BRS [35]. Okamoto et al. [36] have reported that low-intensity resistance training decreases arterial stiffness. In other words, the different result with previous study may be influenced by training intensity.

Arterial baroreceptors such as the aortic arch and carotid sinus constantly monitor ABP and the information is fed back to the medulla oblongata through the baroreceptive afferents nerve [37]. The ABP is then modified by an alteration of CO and peripheral vascular resistance via the autonomic nervous system [37]. The arterial baroreflex is associated with central arterial mechanical properties such as stiffness and compliance [2,3,7], because the arterial baroreceptors primarily sense the deformation of the arterial wall rather than intraarterial pressure changes [38]. A previous study has found that alteration of the arterial diameter is related to neural firing rather than ABP [39]. Based on these results, deformation of the arterial wall may be inhibited in resistance-trained individuals compared with age-matched sedentary individuals.

The cardiovagal BRS is determined by not only by the central arterial mechanical properties but also by the neural component [40]. In an animal study, exercise training has been confirmed to attenuate sympathoexcitation through alterations in the GABAergic neurotransmission at the level of the nucleus tractus solitarius [40]. The neural transmission in the brain might have affected the cardiovagal BRS, which was assessed during phase IV of the Valsalva manoeuvre similar to the present study. Even so, the present study did not assess the neural component. Therefore, further studies should be conducted to investigate the effects of vigorous RT on the arterial baroreceptor-related neural component.

Study Limitation

There are several potential limitations in the present study. First, we recruited only men in both groups. A previous study has shown that RT increases central arterial stiffness in women [16]. Thus, it is necessary to conduct a similar study with women. Second, the subjects in the present study were all young. Several studies have shown that central arterial mechanical properties and cardiovagal BRS are impaired by advancing age [2,3,7]. The interaction between age and vigorous RT on cardiovagal BRS should be examined in future studies. Finally, the present study was designed as a cross-sectional study that compared resistance-trained individuals and non-resistance-trained individuals. Therefore, this study's findings should be confirmed with an intervention study.

Conclusions

β-Stiffness index was significantly higher in the resistance-trained group than in the control group. By contrast, the arterial compliance and cardiovagal BRS in the resistance-trained group were significantly lower than those in the control group. Moreover, a negative correlation was found between cardiovagal BRS and β-stiffness index, and a positive correlation was found between cardiovagal BRS and arterial compliance. Considering these results, central arterial adaptation with vigorous RT may blunt cardiovagal BRS.

Abbreviations

ABP: Arterial blood pressure, BRS: Baroreflex sensitivity, CA: Carotid artery, CO: Cardiac output, DBP: Diastolic blood pressure, dD: Diastolic diameter, ECG: Electrocardiogram, ET-1: Endothelin-1, HR: Heart rate, IMT: intima-media thickness, MAP: Mean arterial pressure, MSNA: Muscle sympathetic nerve activity, PP: Pulse pressure, RT: Resistance training, SBP: Systolic blood pressure, sD: Systolic diameter, SV: Stroke volume, TPR: Total peripheral resistance.

Declarations

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Availability of Data and Materials

The data used to support the findings of this study are available from the corresponding author upon request.

Ethics Approval and Consent to Participate

All of the procedures and risks of this study were reviewed and approved by the Human Research Committee of Waseda University (approval No. 2019-102). The study adhered to the principles of the Declaration of Helsinki. It was registered at UMIN (UMIN000038116).

Authors' Contributions

N.N. and I.M. conceived and designed research; N.N performed experiments; N.N. analyzed data; N.N. and I.M. interpreted results of experiments; N.N. prepared figures; N.N. drafted manuscript;

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Consent for Publication

N/A

Competing interests

The authors, Nobuhiro Nakamura and Isao Muraoka, have no competing interests to declare.

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Tables

Table 1 Subjects' characteristics in control (n = 20) and resistance-trained groups (n = 20).

	Control (n = 20)	Resistance-Trained (n = 20)
Age (y)	25 ± 1	22 ± 1
Height (cm)	171.8 ± 1.2	172.7 ± 1.6
Body weight (kg)	70.0 ± 2.0	79.7 ± 2.1*
Body fat (%)	19.2 ± 1.1	17.4 ± 0.9
Body mass index (kg/m^2)	24 ± 1	27 ± 1*
Lean body mass (kg)	56.2 ± 1.1	65.7 ± 1.6*
Handgrip (kg)	41 ± 1	48 ± 1*

Values are mean ± SEM. *P < 0.05 vs. control group.

Table 2 Haemodynamics and carotid arterial variables in control (n = 20) and resistance-trained groups (n = 20).

	Control (n = 20)	Resistance-Trained (n = 20)
Brachial SBP (mmHg)	110 ± 2	110 ± 1
Brachial DBP (mmHg)	61 ± 1	57 ± 1*
Brachial MAP (mmHg)	79 ± 1	78 ± 1
Brachial PP (mmHg)	49 ± 1	53 ± 1*
Carotid SBP (mmHg)	101 ± 2	104 ± 2
Carotid PP (mmHg)	40 ± 1	47 ± 2*
CA diameter (mm)	6.0 ± 0.1	6.1 ± 0.1
CA IMT (mm)	0.43 ± 0.01	0.43 ± 0.01
HR (bpm)	56 ± 2	58 ± 2
SV (ml)	96 ± 3	94 ± 4
CO (l/min)	5.4 ± 0.2	5.5 ± 0.3
TPR (mmHg/l/min)	15.1 ± 0.6	14.9 ± 0.8

Values are mean ± SEM. SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; PP, pulse pressure; CA, carotid artery; IMT, intima-media thickness; HR, heart rate; SV, stroke volume; CO, cardiac output; TPR, total peripheral resistance. *$P < 0.05$ vs. control group.

Figures

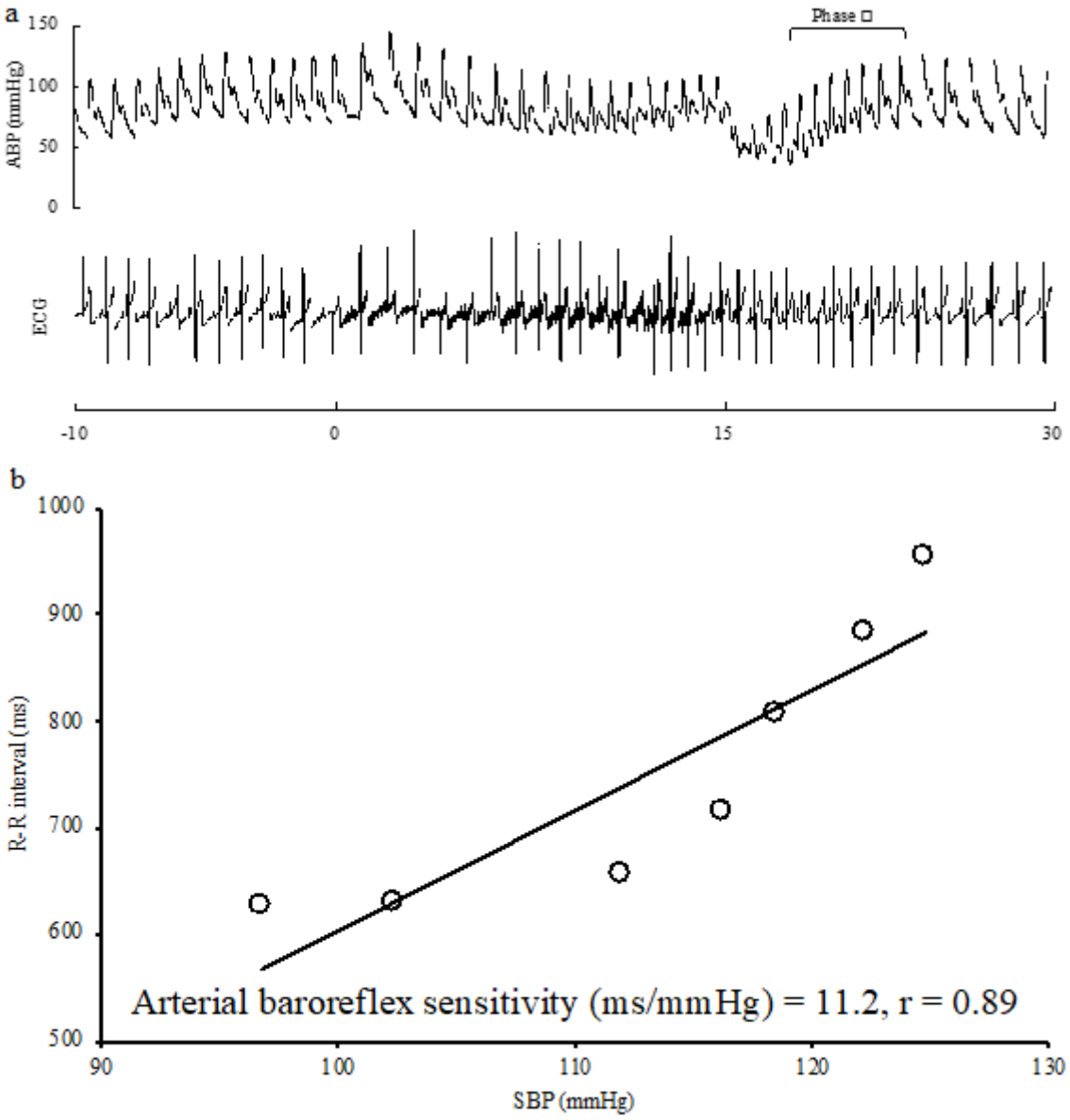


Figure 1

Typical responses of ABP and ECG during the Valsalva manoeuvre. The ABP is increased and the R-R interval is lengthened during phase IV of the Valsalva manoeuvre (a). (b) shows a typical linear regression result between SBP and R-R interval during phase IV of the Valsalva manoeuvre, which occurs after releasing mouth pressure (40mm Hg). ABP, arterial blood pressure; ECG, electrocardiogram; SBP, systolic blood pressure.

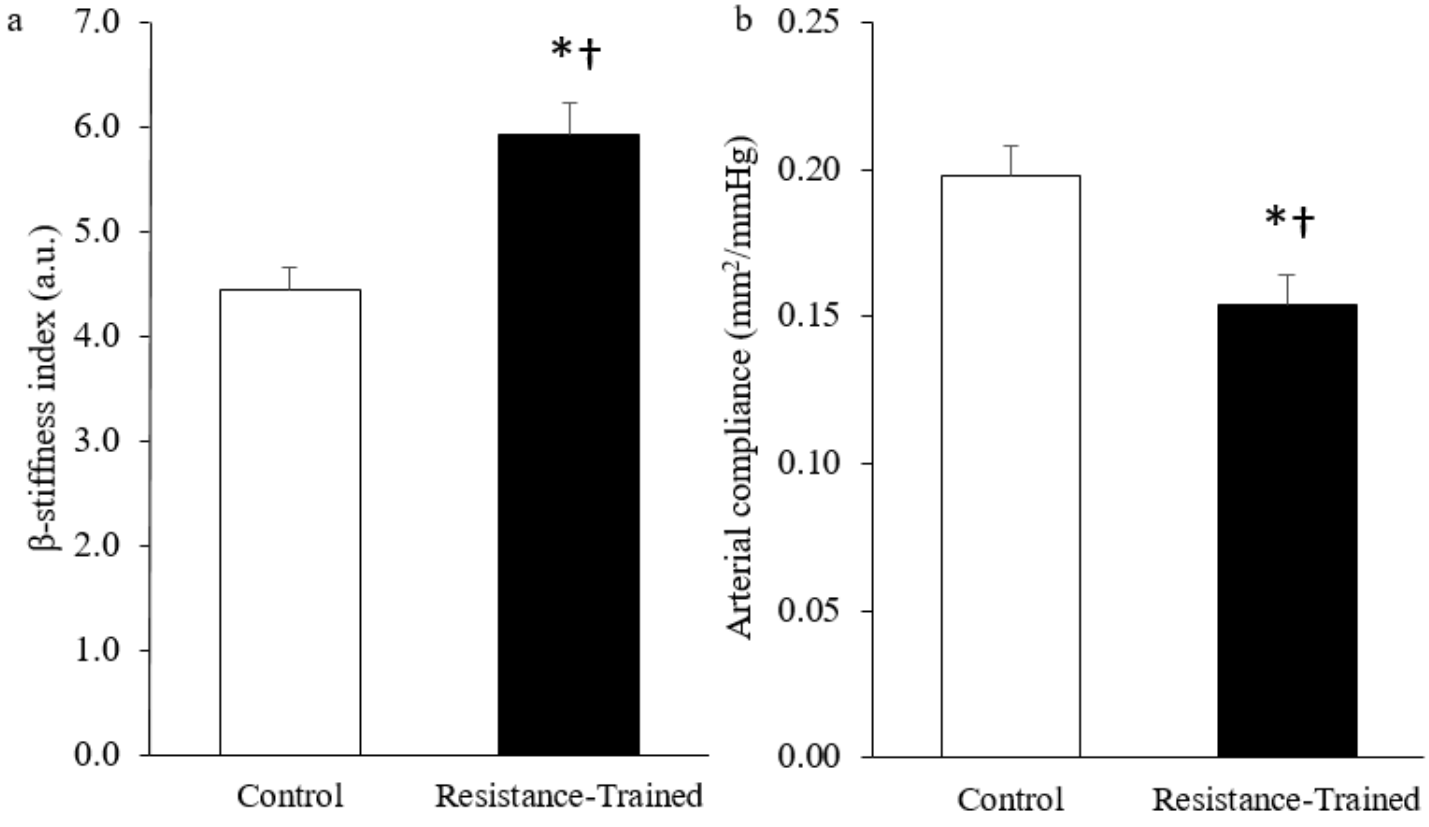


Figure 2

β-stiffness index (a) and arterial compliance (b) in control (n = 20) and resistance-trained groups (n = 20). Values are mean ± SEM. *P < 0.05 and †P < 0.01 vs. control group.

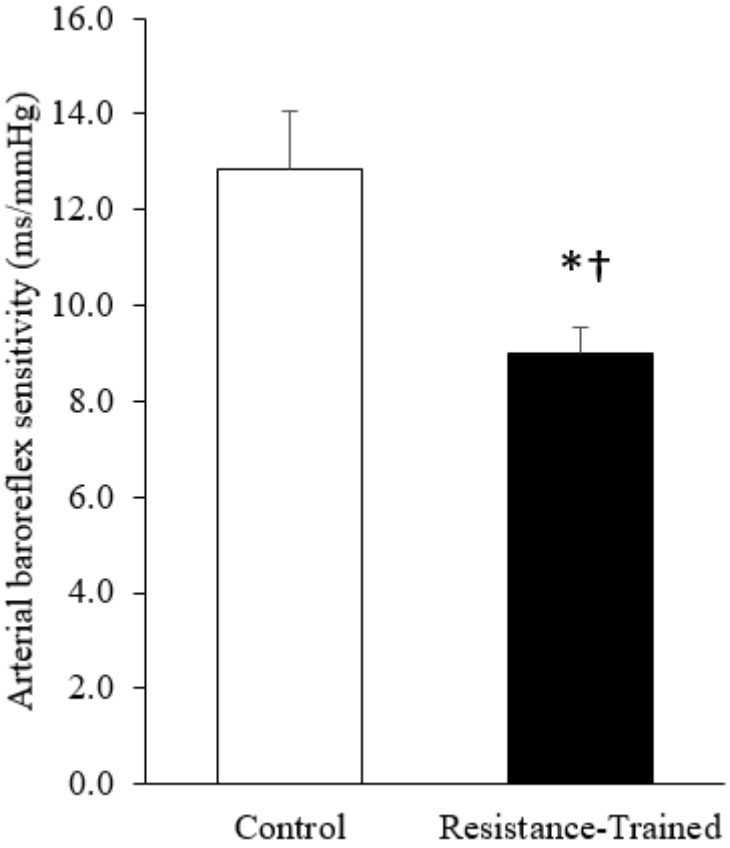
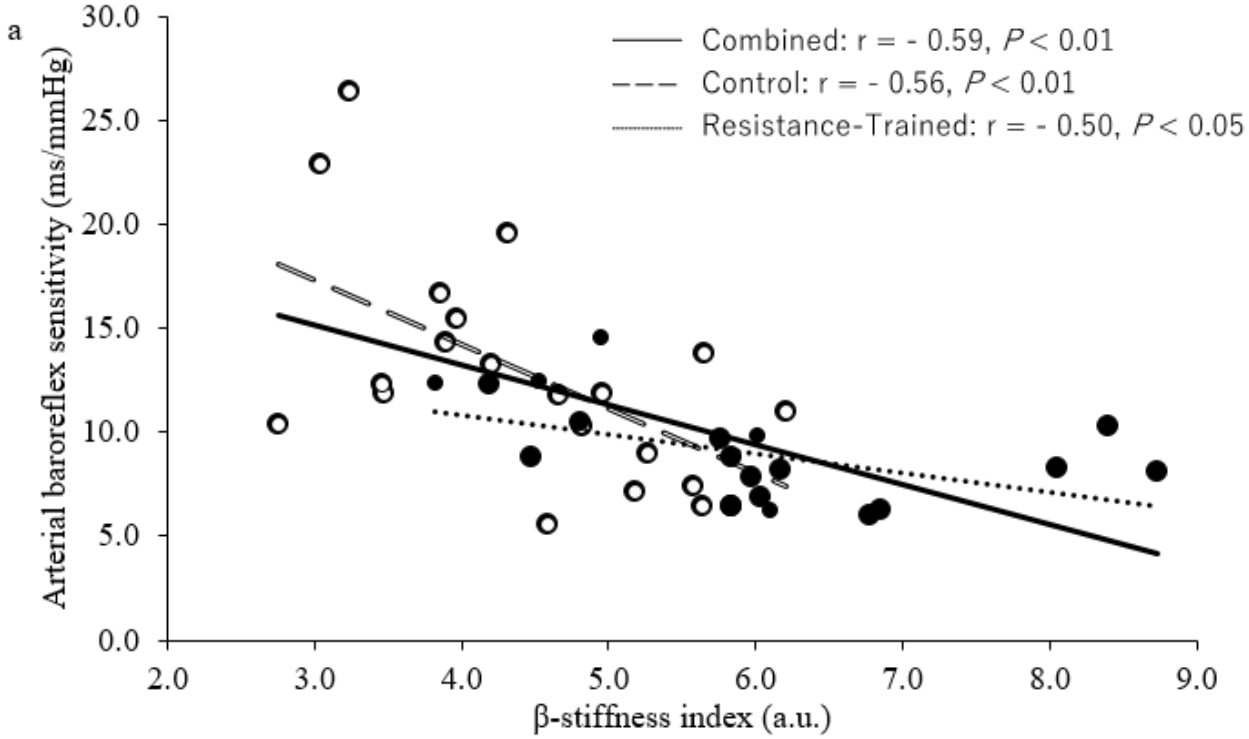


Figure 3

Cardiovagal baroreflex sensitivity is assessed by the SBP and R-R interval slope during the Valsalva manoeuver phase IV, in control and resistance-trained groups (n = 20/group). Values are mean ± SEM. SBP, systolic blood pressure. *P < 0.05 and †P < 0.01 vs. control group.



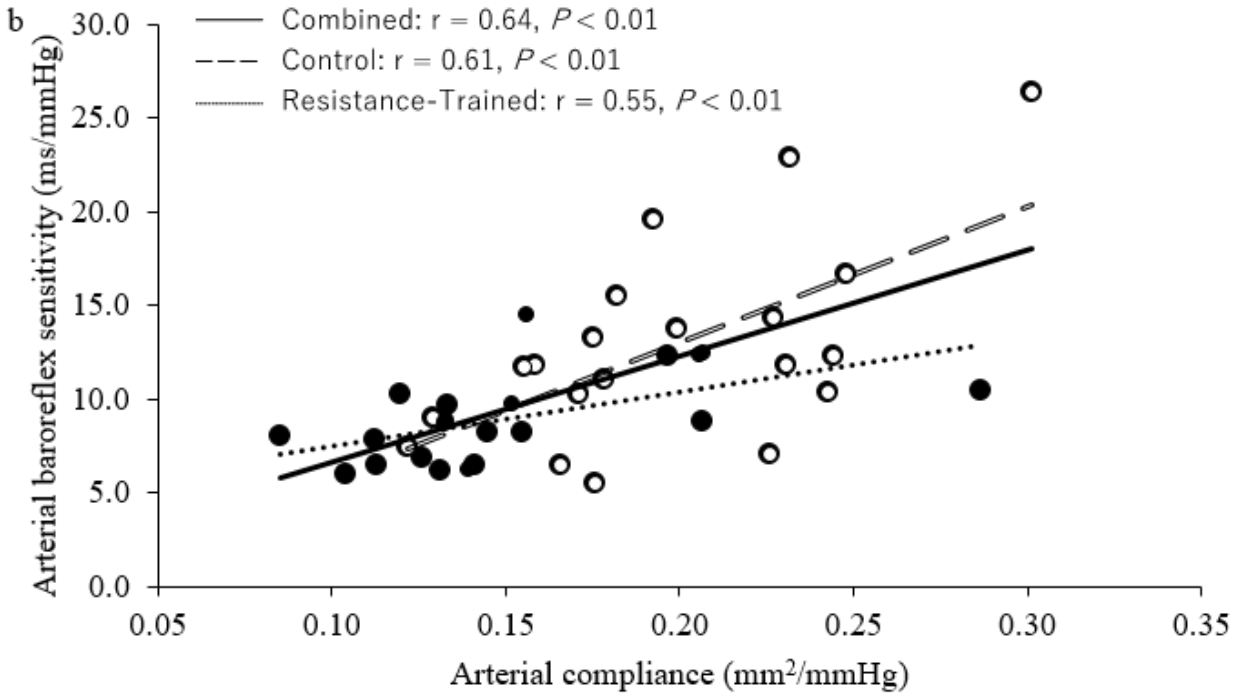


Figure 4

Linear regression between cardiovagal baroreflex sensitivity and β-stiffness index (a), arterial compliance (b). Open circle (○): control subjects (n = 20), close circle (●): resistance-trained subjects (n = 20).