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Original Article

Effects of resistance training on baroreflex sensitivity function in healthy males

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Abstract. [Purpose] This experimental study examined the effects of resistance exercises with three intensities on baroreflex sensitivity (BRS) in healthy males. [Participants and Methods] This study enrolled 27 sedentary healthy males with the following demographic characteristics: mean age, 19.9 years; body mass, 63.4 kg; height, 171.7 cm; and body mass index, 21.5 kg/m^2. The participants performed 20 alternating knee extensions while sitting, and each excursion comprised 5-seconds contraction and rest periods with 20%, 50% and 80% loads of one repetition maximum. The main outcome measures examined in this study were autonomic nerve activities and BRS during the low-intensity resistance exercise utilizing cardiographic and hemodynamic impedance parameters for cardiac function. While measuring the spectral powers of high-frequency (HF) and low frequency (LF) regions, a continuous RR series of the heart rate was calculated. Moreover, the LF/HF ratio of the RR interval variability power and the HF normalized unit (HFnu) were calculated as parameters for sympathetic and parasympathetic nerve activities. [Results] After the low-intensity resistance training, a significant decrease in the LF/HF ratio associated with significant increases in HFnu and BRS were observed. [Conclusion] Low-intensity resistance training may enhance BRS function in healthy males.
Key words: Baroreflex sensitivity, Resistance training, Knee extension

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INTRODUCTION

Recently, 20% of elderly patients with heart failure experienced sarcopenia and decreased muscle strength[1)]. To prevent such conditions, enhancing cardiorespiratory and muscle endurance is important. Aerobic exercises and resistance training (RT) have been introduced to increase muscle strength and mass. RT also improves cardiac autonomic control in diseased individuals[2)]. Regarding safety concerns, the recommended load for RT has been set at 30%–40% one-repetition maximum (1RM) for the upper extremities and 40%–60% 1RM for the lower extremities. However, RT should be performed for three sets with an intensity of ≥65% 1RM, two to three times per week, to achieve exercise effects, such as muscular hypertrophy and skeletal muscle strength increase. RT with an intensity of 40%–60% 1RM frequently fails to achieve muscular hypertrophy. RT with an intensity of 80% 1RM is effective in patients with heart failure[3)]. However, high-intensity muscle training reportedly increases arterial stiffness[4)]. Given that high-intensity exercise may cause a remarkable elevation in blood pressure and impair locomotory organs and the circulatory system in older adults, it is not a suitable training method for patients with heart failure.

To ensure a balance between blood distribution to each organ in the body and maintenance of blood pressure, changes in peripheral vascular resistance—as per the demand from each organ—and blood pressure should be regulated through the baroreflex sensitivity (BRS) function. In healthy individuals, BRS decreases during exercise because peripheral organs

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require more blood[5]. Age and gender are associated with decreased BRS at rest and during exercise[6]. Vascular stiffening, increased levels of oxidative stress and decreased cardiac cholinergic responsiveness with age may be associated with impaired BRS functions, such as increased levels of blood pressure variability, impairment in the ability to respond to acute challenges in maintaining blood pressure (e.g., orthostatic hypotension) and increased risk of sudden cardiac death[7].

Recently in Western countries, baroreflex activation therapy has been initiated in patients with heart failure to improve BRS, autonomic nervous system and cardiac functions as well as exercise tolerance[8, 9].

Studies on changes in BRS during exercise are sporadic in Japan and overseas. Furthermore, few studies have evaluated changes in BRS during RT with different intensities and; therefore, leave unanswered questions. In the precedent study[10], I examined the change of BRS during RT with a single load in healthy males. As BRS improved with RT, a single bout of low-intensity resistance exercise, this study involved healthy males who underwent RT of the quadriceps femoris muscle in the sitting position with three intensities and aimed to determine a suitable exercise intensity for RT that could facilitate improvement in the BRS function. Additionally, the fact that the improvement of the BRS function clarifies the influence on autonomic nerve function and hemodynamics change has been included. Since changes in BRS after exercise remain unclear, clarifying the exercise intensity level that improves the BRS function after exercise is clinically significant.

PARTICIPANTS AND METHODS

This preliminary study included healthy males who were non-smokers and who underwent RT to the quadriceps femoris in the sitting position. The study complies with the ethical standards of the Declaration of Helsinki. Ethical approval was obtained from the Ethics Committee (approval number: 2017-0042) of Bunkyo Gakuin University, and each participant provided informed written consent.

The 27 healthy males enrolled in this study had a mean age of 19.9 years, body weight of 63.4 kg, height of 171.7 cm and body mass index of 21.5 kg/m^2. The participants did not present presented with any cardiorespiratory disease or orthopedic conditions in their lower limbs.

The measurement of the knee joint extension muscular strength in the sitting position was conducted as per the precedent study[10, 11] (Fig. 1). The maximum strength of the right and left quadriceps femoris was defined as 1RM, which was derived from the maximum number of repetitions with 60%–75% of the load[12]. The strength of the right and left quadriceps femoris was measured individually, and the stronger side was used as the leg to be measured for the study. The intensity of exercise was determined based on the low-intensity load being 20% 1RM, medium-intensity load 50% 1RM and high-intensity load 80% 1RM.

With a metronome, RT was started with the right knee in 90° flexion and then extended to 0° flexion, which took 5 s. Immediately after returning to the initial position, RT was repeated on the left knee and then alternatively performed 20 times. Each exercise with each intensity was randomly performed, and the interval between measurements was ≥72 h.

The hemodynamics and function of the autonomic nerves were measured using impedance cardiography, specifically using the Cardiac Function Measurement Taskforce Monitor (TFM-3040; CNSystems Co., Ltd., Graz, Austria)[10]. For autonomic nerve activity, LF/HF as an indicator of sympathetic nerve activity. HF was then corrected by very low frequency (VLF) and total frequency (TF), from which an HF normalized unit (HFnu)=HF/{TF−VLF} ×100 was derived and used as an indicator of parasympathetic nerve activity.

The haemodynamic parameters of heart rate, systolic blood pressure, diastolic blood pressure, cardiac output, total peripheral resistance (TPR), LF/HF, HFnu and BRS were measured with the patient in the sitting position during the resting and RT phases.

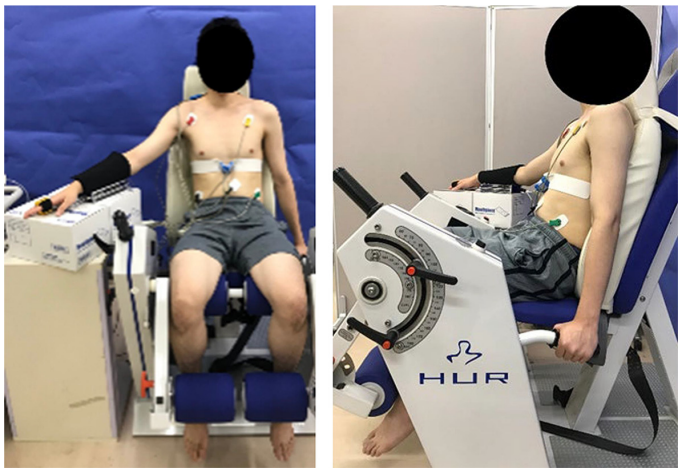

Fig. 1. Position of instruments and participants for impedance cardiography.

Using G*Power, the sample size was calculated to be adequate at 28.64. Although the necessary sample size was 29, the total number of study participants was 27; however, it was our understanding that this number was relatively adequate.

The average of each haemodynamic parameter was calculated during the resting and RT phases. The Wilcoxon signed-rank test was performed to compare the mean haemodynamic parameters. Also, the mean haemodynamic values before and after RT were compared using the Shapiro–Wilk test, which was also used to determine the normality of distribution of continuous variables, followed by the paired t-test. P-values of less than 0.05 were considered as statistically significant. Next, Spearman's rank correlation coefficient was used to evaluate the relationship between BRS and haemodynamic parameters. All statistical analyses were performed using Statistical Package for the Social Sciences, version 23.0 (IBM Corp., Armonk, NY, USA).

RESULTS

LF/HF after RT was significantly lower than LF/HF before RT at the three intensities (20% 1RM, p=0.002; 50% 1RM, p=0.0001; 80% 1RM, p=0.001). HFnu after RT was significantly higher than HFnu before RT at the three intensities (20% 1RM, p=0.002; 50% 1RM, p=0.0001; 80% 1RM, p=0.001). In addition, BRS after RT was significantly higher than BRS before RT at 20% 1RM (p=0.046). TPR after RT was significantly lower than TPR before RT at the three intensities (20% 1RM, p=0.033; 50% 1RM, p=0.0001; 80% 1RM, p=0.0001) (Table 1).

Cardiac output after RT was significantly higher than cardiac output before RT at the three intensities (20% 1RM, p=0.0001; 50% 1RM, p=0.0001; 80% 1RM, p=0.0001). The heart rate after RT was significantly higher than the heart rate before RT at the three intensities (20% 1RM, p=0.01; 50% 1RM, p=0.0001; 80% 1RM, p=0.0001). Systolic blood pressure after RT was significantly higher than systolic blood pressure before RT at 80% 1RM (p=0.0001) (Table 2).

BRS was correlated with HFnu (r=0.592; p=0.001), LF/HF (r=−0.571; p=0.002) and heart rate (r=−0.584; p=0.001) (Table 3).

DISCUSSION

During exercise, shear stress takes place in the blood vessel for local vascular adjustment, which triggers the secretion of substances from the vascular endothelial cells associated with the dilatation and constriction of blood vessels.

Following RT, a decreased sympathetic modulation is observed, which indicates that without the overloading of cardiac function, this exercise beneficially affects vascular adjustment, in consequence promoting endothelial nitric oxide bioavailability and suppressing the vascular adjustment of the sympathetic nerves[13]. Subsequently, this can potentially lead to parasympathetic nerve activation and reduction of the TPR. Although autonomic nerve and TPR have not been examined, a previous study reported that vasodilatation was provided after low strength RT[11].

The mechanism of RT increase due to BRS remains unclear. However, the functions of the left ventricle and BRS improve after RT in diabetic rats[14]. Exercise intensity is an important determinant of post-exercise carotid artery constriction[15], which influences the sympathetic nervous system via BRS modulation by TPR[15, 16]. Therefore, the results of this study, namely, the decrease in TPR and sympathetic nerve activity after RT, substantiate the cause of the increase in the BRS function.

Table 1. Changes in sympathetic nerve activity, parasympathetic nerve activity, baroreceptor sensitivity and total peripheral resistance during resistance training

		BRS [beats]	LF/HF [1]	Hfnu [%]	TPR [dyne*s/cm^5]
20%	Rest	16.4 ± 8.2	2.8 ± 2.4	34.9 ± 13.1	1,257.9 ± 199.0
	Exercise	10.3 ± 3.8	2.0 ± 1.2	43.9 ± 16.4	1,013.4 ± 157.2
	Post	17.4 ± 6.9*	1.7 ± 0.9*	44.8 ± 17.4*	1,214.1 ± 221.5*
50%	Rest	17.8 ± 9.6	2.7 ± 2.8	36.6 ± 15.4	1,306.9 ± 183.6
	Exercise	8.0 ± 3.3	1.7 ± 0.8	44.4 ± 12.2	983.9 ± 170.4
	Post	17.9 ± 8.7	1.7 ± 1.5*	49.8 ± 17.0*	1,123.6 ± 210.4*
80%	Rest	16.9 ± 6.9	2.4 ± 1.7	35.6 ± 16.3	1,271.4 ± 175.7
	Exercise	6.9 ± 4.7	1.7 ± 0.9	43.9 ± 15.7	943.6 ± 169.6
	Post	21.3 ± 13.3	1.6 ± 1.5*	52.6 ± 16.8*	1,059.3 ± 214.7*

Mean ± standard deviation.
*p<0.05 (rest vs. post).
20%: low-intensity load was 20% 1RM; 50%: medium-intensity load was 50% 1RM; 80%: high-intensity load was 80% 1RM; BRS: Baroreflex sensitivity; LF/HF: Frequency components of the heart rate changes were classified into 0–0.04 Hz as very-low frequency (VLF), 0.04–0.15 Hz as low frequency (LF), and 0.15–0.4 Hz as high-frequency (HF), determining LF/HF as an indicator of sympathetic nerve activity; HFnu: HF normalized unit (HFnu)=HF / {TF−VLF} ×100 was derived and used as an indicator of parasympathetic nerve activity; TPR: total peripheral resistance; Rest: the mean of rest 5 minutes; Exercise: during 20 alternating knee extension; Post: the mean of 5 minutes after the exercise.

Table 2. Changes in hemodynamics during resistance training

		CO [l/min]	SBP [mmHg]	DBP [mmHg]	HR [bpm]
20%	Rest	5.6 ± 0.7	118.6 ± 9.7	71.1 ± 7.9	70.8 ± 6.7
	Exercise	7.8 ± 1.1	131.3 ± 11.0	78.5 ± 9.3	93.2 ± 8.1
	Post	6.0 ± 0.8*	120.5 ± 11.6	72.0 ± 8.2	72.0 ± 7.4*
50%	Rest	5.5 ± 0.7	120.5 ± 8.4	72.5 ± 6.1	70.6 ± 10.5
	Exercise	8.7 ± 1.1	140.7 ± 17.5	86.0 ± 13.4	106.3 ± 11.5
	Post	6.6 ± 0.9*	124.9 ± 14.3	70.5 ± 8.4	75.4 ± 12.1*
80%	Rest	5.7 ± 0.6	120.7 ± 10.8	72.5 ± 7.8	70.0 ± 10.4
	Exercise	9.3 ± 1.0	137.3 ± 22.5	90.3 ± 15.8	111.9 ± 10.6
	Post	7.3 ± 1.2*	129.7 ± 15.0*	72.9 ± 10.8	75.8 ± 10.4*

Mean ± standard deviation.
*p<0.05 (rest vs. post).
20%: Low-intensity load was 20% 1RM; 50%: Medium-intensity load was 50% 1RM; 80%: High-intensity load was 80% 1RM; CO: cardiac output; SBP: systolic blood pressure; DBP: diastolic blood pressure; HR: heart rate; Rest: the mean of rest five minutes; Exercise: during 20 alternating knee extension; Post: the mean of five minutes after the exercise.

Table 3. Correlation between BRS and each parameter during resistance training

	r
LF/HF [1]	−0.571*
Hfnu [%]	0.592*
TPR [dyne*s/cm^5]	0.01
CO [l/min]	0.065
SBP [mmHg]	0.226
DBP [mmHg]	0.048
HR [bpm]	−0.584*

Mean ± standard deviation.
*p<0.05 (Correlation with parameter and BRS where a change was accepted before and after exercise).
LF/HF: Frequency components of the heart rate changes were classified into 0–0.04 Hz as very-low frequency (VLF), 0.04–0.15 Hz as low frequency (LF), and 0.15–0.4 Hz as high-frequency (HF), determining LF/HF as an indicator of sympathetic nerve activity; HFnu: HF normalized unit (HFnu)=HF / {TF−VLF} ×100 was derived and used as an indicator of parasympathetic nerve activity; TPR: total peripheral resistance; CO: cardiac output; SBP: systolic blood pressure; DBP: diastolic blood pressure; HR: heart rate.

High-intensity RT greatly attenuates sympathetic nerve activity[17]. High-intensity RT is not as safe as aerobic exercise from the viewpoint of post-exercise cardiovascular autonomic nerve modulation because ultra-high-intensity RT in contrast to low-intensity RT decreases BRS[18].

In addition, Low-intensity RT decreases sympathetic nerve activity after exercise. Impaired BRS function is associated with sympathetic tone and respiratory muscle fatigue due to insufficient circulation to the respiratory muscle, which decreases the capacity to exercise in patients with heart failure[5]. The activation of sympathetic nerves increases adrenergic drive that produces a cycle leading to the deterioration of clinical status[5]. Muscle metabolism causes metaboreflex of the sympathetic nerve activity during exercise, resulting in accentuated coronary vasoconstriction. This decreases oxygen supply to the myocardium, thereby limiting the heightening of cardiac function, leading to difficulty in exercise continuation[5]. The target population of this study is healthy males; however, sympathetic nerve activity may be to decrease after RT in patients with heart failure.

Table 3 shows a positive correlation between BRS after RT and parasympathetic nerve activity; however, it indicates a negative correlation between heart rate, sympathetic nerve activity and BRS. As the BRS function increases, heart rate decreases disproportionately. In other words, parasympathetic nervous system activation after Low-intensity RT is associated with improvement in the BRS function. This study compared only the mean values of BRS 5 min after exercise among the three groups. Thus, we did not use an indicator that shows the degree of reduction in heart rate, such as T30, resulting in a lack of results supported by a clinically objective index. This study suggests that heart rate recovery after exercise is an indicator of the improvement in the BRS function following Low-intensity RT.

This study had some limitations. This study involved healthy males with only one intervention. Therefore, to establish Low-intensity RT as a clinically feasible exercise therapy, mid- and long-term interventions with actual patients is needed. In addition, assessing resistance of the peripheral vessels should be performed. This study was centred on an indirect evaluation based on central hemodynamics. Finally, the small sample size of the study required further study with more samples.

In low-, medium-, and high-intensity sitting knee extension resistance exercises, low-intensity knee extension resistance exercises showed improvement in BRS function after exercise. In addition, a correlation was observed between improvement of BRS function, inhibition of sympathetic nerve activity, and improvement of parasympathetic nerve function. Furthermore, the fact that improvement of BRS function leads to improvement of autonomic nervous function has been confirmed, and the examination of participants who desire improvement of autonomic nervous function, such as patients with heart disease, is a participant for further research in the future.

In conclusion, low-intensity resistance exercise decreases sympathetic nerve activity and increases parasympathetic nerve activity, suggesting improved BRS function.

Furthermore, the degree of heart rate recovery may aid in predicting improvements in the BRS function.

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Conflict of interest

The author has no conflict of interests to declare.

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REFERENCES

1) Lena A, Anker MS, Springer J: Muscle wasting and sarcopenia in heart failure-the current state of science. Int J Mol Sci, 2020, 21: 6549–6567. [Medline] [CrossRef]
2) Bhati P, Moiz JA, Menon GR, et al.: Does resistance training modulate cardiac autonomic control? A systematic review and meta-analysis. Clin Auton Res, 2019, 29: 75–103. [Medline] [CrossRef]
3) Hornikx M, Buys R, Cornelissen V, et al.: Effectiveness of high intensity interval training supplemented with peripheral and inspiratory resistance training in chronic heart failure: a pilot study. Acta Cardiol, 2020, 75: 339–347. [Medline] [CrossRef]
4) Miyachi M, Kawano H, Sugawara J, et al.: Unfavorable effects of resistance training on central arterial compliance: a randomized intervention study. Circulation, 2004, 110: 2858–2863. [Medline] [CrossRef]
5) Michelini LC, O'Leary DS, Raven PB, et al.: Neural control of circulation and exercise: a translational approach disclosing interactions between central command, arterial baroreflex, and muscle metaboreflex. Am J Physiol Heart Circ Physiol, 2015, 309: H381–H392. [Medline] [CrossRef]
6) Fisher JP, Kim A, Hartwich D, et al.: New insights into the effects of age and sex on arterial baroreflex function at rest and during dynamic exercise in humans. Auton Neurosci, 2012, 172: 13–22. [Medline] [CrossRef]
7) Monahan KD: Effect of aging on baroreflex function in humans. Am J Physiol Regul Integr Comp Physiol, 2007, 293: R3–R12. [Medline] [CrossRef]
8) Schmidt R, Rodrigues CG, Schmidt KH, et al.: Safety and efficacy of baroreflex activation therapy for heart failure with reduced ejection fraction: a rapid systematic review. ESC Heart Fail, 2020, 7: 3–14. [Medline] [CrossRef]
9) Zile MR, Lindenfeld J, Weaver FA, et al.: Baroreflex activation therapy in patients with heart failure with reduced ejection fraction. J Am Coll Cardiol, 2020, 76: 1–13. [Medline] [CrossRef]
10) Shobo A: Does a single bout of low-intensity resistance exercise change baroreflex sensitivity? Int J Physiother, 2021, 8: 117–120. [CrossRef]
11) Kagita M, Obana T, Kishi T, et al.: Vasodilatory response to knee extension with different loads. Rigakuryoho Kagaku, 2018, 33: 1–5 (in Japanese). [CrossRef]
12) Arena R, Myers J, Forman DE, et al.: Should high-intensity-aerobic interval training become the clinical standard in heart failure? Heart Fail Rev, 2013, 18: 95–105. [Medline] [CrossRef]
13) Mostarda CT, Rodrigues B, de Moraes OA, et al.: Low intensity resistance training improves systolic function and cardiovascular autonomic control in diabetic rats. J Diabetes Complications, 2014, 28: 273–278. [Medline] [CrossRef]
14) Oliveira R, Barker AR, Debras F, et al.: Mechanisms of blood pressure control following acute exercise in adolescents: effects of exercise intensity on haemodynamics and baroreflex sensitivity. Exp Physiol, 2018, 103: 1056–1066. [Medline] [CrossRef]
15) Kim A, Deo SH, Vianna LC, et al.: Sex differences in carotid baroreflex control of arterial blood pressure in humans: relative contribution of cardiac output and total vascular conductance. Am J Physiol Heart Circ Physiol, 2011, 301: H2454–H2465. [Medline] [CrossRef]
16) Ogoh S, Fadel PJ, Monteiro F, et al.: Haemodynamic changes during neck pressure and suction in seated and supine positions. J Physiol, 2002, 540: 707–716. [Medline] [CrossRef]
17) Gava NS, Véras-Silva AS, Negrão CE, et al.: Low-intensity exercise training attenuates cardiac beta-adrenergic tone during exercise in spontaneously hypertensive rats. Hypertension, 1995, 26: 1129–1133. [Medline] [CrossRef]
18) Niemelä TH, Kiviniemi AM, Hautala AJ, et al.: Recovery pattern of baroreflex sensitivity after exercise. Med Sci Sports Exerc, 2008, 40: 864–870. [Medline] [CrossRef]