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Does a Single Bout of Low-Intensity Resistance Exercise Change Baroreflex Sensitivity?

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ABSTRACT

Background: Baroreflex sensitivity (BRS) has decreased in patients with hypertension, diabetes mellitus, and congestive heart failure. According to ATRAMI (Autonomic Tone and Reflex After Myocardial Infarction), BRS is helpful for the prediction of the sudden death of persons who have experienced myocardial infarction.

Methods: Twenty-four sedentary healthy men with a mean age of 21.0 years, mean body mass of 62.5kg, mean height of 171.1cm, and a body mass index of 21.3kg/m².

Procedure: In a single session, the participants performed a total of 20 alternating knee extensions in sitting with each excursion consisting of a five-second contraction and five-second rest period with a 20% load of one-repetition maximum. Autonomic nerve activity and BRS were measured during LRE using impedance cardiography and hemodynamic parameters for cardiac function. In addition, a continuous R-R series was taken of the heart rate with quantification of spectral powers for regions of high frequency (HF) and low frequency (LF). Also calculated were LF/HF of the R-R interval variability power ratio and the HF normalized unit (HFnu) as indicators for sympathetic and parasympathetic nerve activity.

Results: There was a significant decrease in LF/HF post-LRT ($P=0.045$) with a significant increase in HFnu and BRS ($P=0.01$ and $P=0.032$, respectively).

Conclusion: A single bout of LRE proved to enhance BRS function in healthy men.

Keywords: Baroreflex sensitivity; Low-intensity resistance exercise; Knee extension.

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INTRODUCTION

In recent years, aerobic training and more recently resistance training has attracted attention to the aged and patients with cardiac conditions. Aerobic training is already an established means of exercise therapy for patients with cardiac conditions. However, there remains ground for further investigation into resistance training. For carrying out resistance training, an acceptable level of intensity of resistance for patients with cardiac conditions is 30 to 40% of one-repetition maximum (1RM) for upper limbs and 50 to 60%1RM for lower limbs. It has been found that ultra-high-intensity resistance training is beneficial for patients with cardiac failure [1] but may cause arterial stiffness [2]. Thus, a consensus has not yet been reached about whether these intensities are appropriate for patients with cardiac conditions and whether they are acceptable as safe exercise regimes.

Contraction of muscles during resistance training may bring about varying effects on intramuscular pressure and central and peripheral circulation. Amongst neural circulatory mediating functions, baroreflex sensitivity (BRS) detects changes in blood pressure and controls cardiac output and peripheral vascular resistance via the autonomic nervous system, thereby modulating blood pressure. BRS during exercise is known to decrease with an increase in exercise intensity compared to the resting state. According to the cardiovascular trial database 'Autonomic Tone and Reflexes After Myocardial Infarction' [3], the BRS function, together with changes in heart rate, is a helpful predictor for sudden death in patients with myocardial infarction. It is known that BRS in healthy individuals during exercise at any intensity decreases due to excess blood being distributed to peripheral organs such as skeletal muscles [4]. As the exercise intensity increases, it necessitates a decrease in BRS. However, few reports are available concerning changes in BRS during resistance training at varying intensities.

In this preliminary study, the objective was to investigate whether a single session of low-intensity resistance exercise (LRE) for the quadriceps femoris of healthy young men in a sitting would show a change in their BRS response. The findings from this study could also be relevant to exercise physiology and, following further studies, be applied to patients with cardiac conditions in a rehabilitation setting.

METHODS

This single preliminary bout of LRE study involved non-smoking healthy young men who underwent exercise to the quadriceps femoris in sitting. Ethical approval was received from the Bunkyo Gakuin University Ethics Committee (2017-0042), and the participants provided informed written consent.

The participants were 24 sedentary healthy men with a mean age of 21.0 years, mean body mass of 62.5kg, mean height of 171.1cm, and a body mass index of 21.3kg/m². They presented with no cardiorespiratory disease or orthopedic condition in their lower limbs and were non-

smokers.

The participant sat on the Leg Extension / Curl unit (HUR Co., Ltd.) with the knees in a starting position of 90-degree flexion. The two arms of this unit were placed on the distal ends of the tibia (Fig. 1), and the participant held the two handles attached to the unit for LRE. Before carrying out LRE for the quadriceps femoris to 0-degree flexion, the maximum strength of the right and left quadriceps femoris was individually determined by the 1RM test [5], and the stronger limb was used for the initial hemodynamic measurement. In addition, the exercise intensity was determined according to the revised version of the Guidelines for Rehabilitation of Patients with Cardiovascular Diseases 2012 [6]. Specifically, 20%1RM, which is considered as low resistance, was employed in this study.

Using a metronome, LRE commenced with the right knee in 90-degree flexion and then, taking 5 secs, it was extended to 0-degree flexion, which was maintained for further 5 secs. Immediately after returning to the starting position, the same sequence was repeated on the left knee and then carried out alternately a total of 20 times.

Seventy-two hours were allowed between pre-and post-measurement for 1RM to allow for sufficient recovery from muscle fatigue.

Hemodynamics and response of the autonomic nerve activity were measured through impedance cardiography using the Cardiac Function Measurement Taskforce Monitor (TFM-3040; CNSystems Co., Ltd.). The TFM-3040 analyses changes in heart rate using an autoregressive method. As for autonomic nerve activity, changes in the heart rate were calculated using spectral analysis. Frequency components of the heart rate changes were classified into 0~0.04Hz as being very low frequency (VLF), 0.04~0.15Hz as low frequency (LF), and 0.15~0.4Hz as high frequency (HF), determining LF/HF as an indicator of sympathetic nerve activity. HF was then corrected by VLF and total frequency (TF), from which an HF normalized unit (HFnu)= $HF/\{TF-VLF\}\times 100$ was derived and used as an indicator of parasympathetic nerve activity.

The hemodynamic parameters of the heart rate, systolic blood pressure, stroke volume, cardiac output, total peripheral resistance (TPR), LF/HF, HFnu, and BRS were measured in the sitting position pre-and-post-LRE phases.

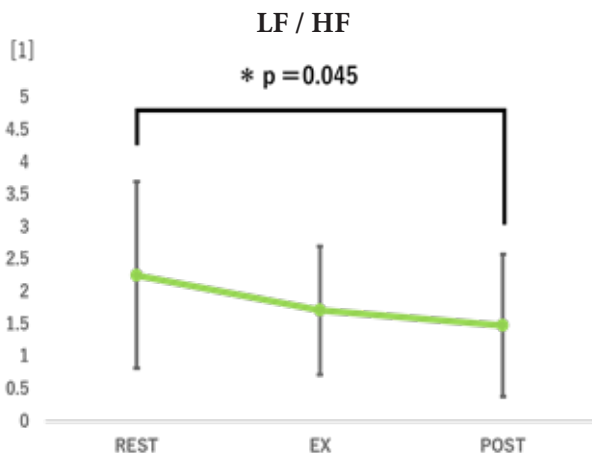
Each haemodynamic parameter was averaged during the pre-and-post-LRE phases. The Wilcoxon signed-rank test was employed for comparison between the mean parameters. Also compared were the mean pre-and-post-LRE values using the Shapiro-Wilk test, followed by the paired t-test with the level of significance set at $p<0.05$. The Statistics Package for Social Sciences version 23.0 for Windows (IBM Corp.) was employed for data analysis.

RESULTS

Post-LF/HF significantly decreased compared to pre-LF/HF ($P=0.045$) (Fig. 2), while post-HFnu significantly increased compared to pre-HFnu ($P=0.01$) (Fig. 3).

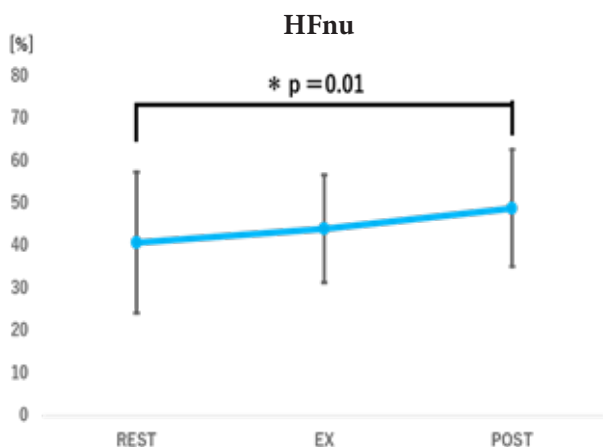
However, post-BRS significantly increased compared to pre-BRS ($P=0.032$) (Fig. 4). Post-TPR significantly decreased compared to pre-TPR ($P=0.011$) (Fig. 5).

Figure 2: Changes in sympathetic nerve activity during LRE.



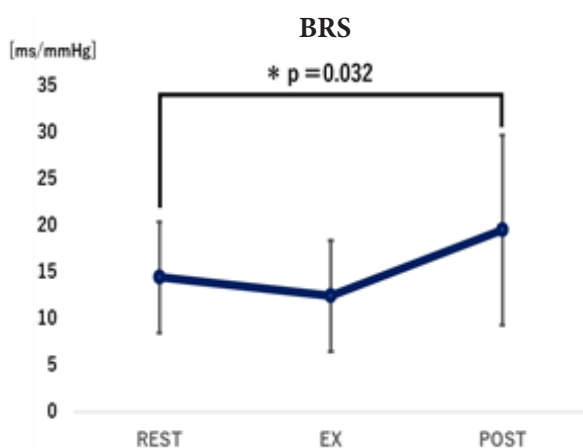
Pre: Pre-20% 1RM measurement
LRE: single bout of low-resistance exercise
Post: post-20%1RM measurement

Figure 3: Changes in parasympathetic nerve activity during LRE.



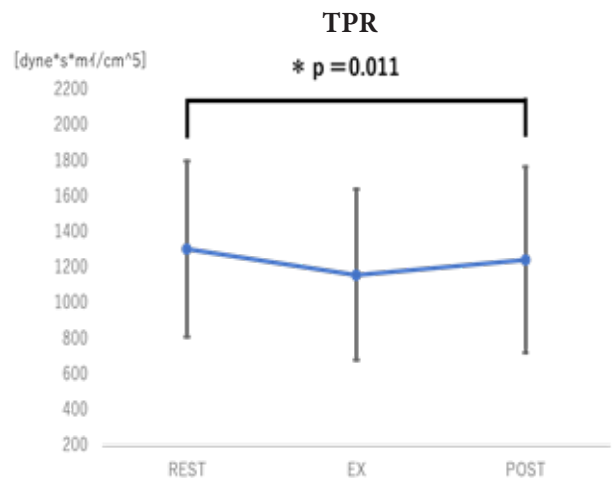
Pre: Pre-20% 1RM measurement
LRE: single bout of low-resistance exercise
Post: post-20%1RM measurement

Figure 4: Changes in baroreceptor sensitivity during LRE.



Pre: Pre-20% 1RM measurement
LRE: single bout of low-resistance exercise
Post: post-20%1RM measurement

Figure 5: Changes in total peripheral resistance during LRE.



Pre: Pre-20% 1RM measurement
LRE: single bout of low-resistance exercise
Post: post-20%1RM measurement

DISCUSSION

The findings showed that the single bout of LRE brought about a decrease in sympathetic nerve activity and TPR with an increase in parasympathetic nerve activity and BRS. Therefore, the resulting activation of the parasympathetic nerves and increase in BRS can be interpreted as being due to vasodilatation. However, the parameters for measuring vascular function such as elasticity of the arteries, were not carried out. Therefore, interpretation is limited to the observation of peripheral vascular resistance brought about by changes in the blood pressure and cardiac output.

Regarding local vascular adjustment, shear stress occurs within blood vessels during exercise, which triggers the secretion of substances from the vascular endothelial cells related to vascular dilatation and constriction. Endothelin-1, one of the vasopressor substances, is secreted into non-active muscles during exercise with its concentration is proportional to the exercise intensity [7]. In contrast, secretion of nitric oxide, one of the vasodilators, decreases within one minute [8], although there is some secretion due to an increase in the shear stress. The amount of secretion and duration of effect of other vasodilators during exercise remains unknown [9]. From these findings, the effect of vasodilator substances and Endothelin-1 due to LRE may be the reason for the increase in BRS.

Decreased sympathetic modulation post-LRE suggests that this means of exercise produces, without overloading cardiac function, a beneficial effect on vascular adjustment, thereby promoting bioavailability of endothelial nitric oxide and suppressing vascular adjustment of the sympathetic nerves [10]. This may, in turn, activate the parasympathetic nerves, thereby decreasing the TPR.

It remains unclear why BRS increases post-LRE. However,

the left ventricle and BRS function have been known to improve following LRE in diabetic rats [11]. Furthermore, exercise intensity is an essential determinant for post-exercise carotid artery constriction [12], influencing the sympathetic nervous system via BRS modulation by TPR [13,14]. Therefore, the findings from this study, namely, the post-LRE decrease in TPR and sympathetic nerve activity, substantiate the increase in the BRS activity. Furthermore, LRE has been verified to attenuate sympathetic nerve activity [15] significantly. Therefore, LRE may be as safe as aerobic exercise from the viewpoint of autonomic regulation, for it is known that heavy resistance exercise decreases BRS [16].

This study involved healthy men with one intervention. Therefore, establishing LRE as clinically feasible exercise therapy, mid-and long-term trials with actual patients will be required. In addition, assessment of the resistance of the peripheral vessels by means of their function requires to be carried out. Therefore, this study was centered on an indirect assessment based on central haemodynamics.

CONCLUSION

A single bout of LRE demonstrated decreased sympathetic nerve activity and increased parasympathetic nerve activity, suggesting improved BRS function.

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REFERENCES

- [1] Hornikx M, Buys R, Cornelissen V, Deroma M, Goetschalckx K. Effectiveness of high intensity interval training supplemented with peripheral and inspiratory resistance training in chronic heart failure: a pilot study. *Acta Cardiol* 2020; 75: 339-47.
- [2] Miyachi M, Kawano H, Sugawara J, Takahashi K, Hayashi K, Yamazaki K, et al. Unfavorable effects of resistance training on central arterial compliance: a randomized intervention study. *Circulation* 2004; 110: 2858-63.
- [3] La Rovere MT, Bigger Jr JT, Marcus FI, Mortara A, Schwartz PJ. Baroreflex sensitivity and heart-rate variability in prediction of total cardiac mortality after myocardial infarction. ATRAMI (Autonomic Tone and Reflexes After Myocardial Infarction) Investigators. *Lancet* 1998; 351: 478-84.
- [4] Michelini LC, O'Leary DS, Raven PB, Nobrega ACL. 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: 381-92.
- [5] <https://exrx.net/Calculators/OneRepMax>. (accessed 18th October 2020)
- [6] Arena R, Myers J, Forman DE, Lavie CJ, Guazzi M. Should high-intensity-aerobic interval training become the clinical standard in heart failure? *Heart Fail Rev* 2013; 18: 95-105.
- [7] Nohara T. Guidelines for rehabilitation in patients with cardiovascular disease (JCS 2012), Mendeley Data, v1; 2012. http://www.jacr.jp/pdf/JCS2012_nohara_d_2015.01.14. (accessed 28th June 2020) (in Japanese)
- [8] Saito M. *Circulation 2: human cardiovascular regulation during exercise and adaptation*. Tokyo: Nap; 2007, p. 37-43, 58. (in Japanese)
- [9] Enkhjargal B, Hashimoto M, Sakai Y, Shido O. Characterization of vasoconstrictor-induced relaxation in the cerebral basilar artery. *Eur J Pharmacol* 2010; 637: 118-23.
- [10] Macedo FN, Mesquita TRR, Melo VU, Mota MM, Silva TLTB, Santana MN. et al. Increased nitric oxide bioavailability and decreased sympathetic modulation are involved in vascular adjustments induced by low-intensity resistance training. *Front Physiol* 2016; 28: 265.
- [11] Mostarda CT, Rodrigues B, De Moraes OA, Moraes-Silva IC, Arruda PBO, Cardoso R, et al. Low intensity resistance training improves systolic function and cardiovascular autonomic control in diabetic rats. *J Diabetes Complications* 2014; 28: 273-8.
- [12] Oliveira R, Barker AR, Debras F, O'Doherty A, Williams CA. Mechanisms of blood pressure control following acute exercise in adolescents: effects of exercise intensity on haemodynamics and baroreflex sensitivity. *Exp Physiol* 2018; 103: 1056-66.
- [13] Kim A, Deo SH, Vianna LC, Balanos GM, Hartwich D, Fisher JP, 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: 2454-65.
- [14] Ogoh S, Fadel PJ, Monteiro F, Wasmund WL, Raven PB. Haemodynamic changes during neck pressure and suction in seated and supine positions. *J Physiol* 2002; 540: 707-16.
- [15] Gava NS, Vêras-Silva AS, Negrão CE, Krieger EM. Low-intensity exercise training attenuates cardiac beta-adrenergic tone during exercise in spontaneously hypertensive rats. *Hypertension* 1995; 26: 1129-33.
- [16] Niemelä TH, Kiviniemi AM, Hautala AJ, Salmi JA, Linnamo V, Tulppo MP. Recovery pattern of baroreflex sensitivity after exercise. *Med Sci Sports Exerc* 2008; 40: 864-70.

Figure legend

Figure 1: Position of instruments and participant for impedance cardiography.