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Original Article

Supine effect of passive cycling movement induces vagal withdrawal

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Abstract. [Purpose] The purpose of this study was to examine changes in vagal tone during passive exercise while supine. [Subjects and Methods] Eleven healthy males lay supine for 5 min and then performed passive cycling for 10 min using a passive cycling machine. The lower legs moved through a range of motion defined by 90° and 180° knee joint angles at 60 rpm. Respiratory rates were maintained at 0.25 Hz to elicit respiratory sinus arrhythmia. Heart rate variability was analyzed using the time domain analysis, as the root mean squared standard differences between adjacent R-R intervals (rMSSD), and spectrum domain analysis of the high frequency (HF) component. [Results] Compared to rest, passive cycling decreased rMSSD (rest, 66.6 ± 92.6 ms; passive exercise, 53.5 ± 32.5 ms). However, no significant changes in HR or HF were observed (rest, 68.2 ± 6.9 bpm, 65.6 ± 12.0 n.u.; passive exercise, 70.2 ± 7.2 bpm, 67.9 ± 10.0 n.u.). [Conclusion] These results suggest that passive exercise decreases rMMSD through supine-stimulated mechanoreceptors with no effect on HR or HF. Therefore, rMSSD is not affected by hydrostatic pressure during passive cycling in the supine position.
Key words: Passive cycling movement, Supine position, Vagal tone

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INTRODUCTION

Stimulation of mechanoreceptors during passive cycling induces increases in heart rate (HR) and stroke volume (SV) in humans[1, 2)]. These changes occur in passive exercise, but moderate intensity knee extension exercises do not induce sympathetic nerve activity[3)]. Moreover, sympathetic nerve activation is unlikely to occur during passive cycling, particularly at HR < 100 bpm[4)]. According to this paradigm, the neural components most likely to be responsible for increases in HR during passive cycling are afferent feedback pathways from group III mechanoreceptors and subsequent vagal withdrawal[5)]. Passive exercise-induced changes in autonomic function are termed the mechanoreflex[6)].

A previous study[7)] suggested that the mechanoreflex contributes less to regulation of the circulatory system during exercise than other functions, such as the central command, baroreflex, and metaboreflex. However, another study reported that the mechanoreflex is occurred with overactivation of sympathetic nerves, leading to increased heart rate and systolic blood pressure in patients with heart failure compared with that in controls[8)]. In addition, elderly individuals have significantly smaller changes in HR and cardiac output (CO) in response to passive exercise than young individuals[5)], demonstrating that passive exercise-induced physiological responses are affected by aging. Thus, mechanoreflex is involved in the regulation of autonomic function in heart failure[8)], muscle atrophy[9)], and peripheral arterial disease[10)]. Therefore, the evaluation of autonomic function via the mechanoreflex has clinical importance.

Previous studies have demonstrated that passive cycling induced decreases vagal tone using passive exercise models performed in the upright seated position[1, 2)]. Postural changes affect hydrostatic pressure associated with physiological responses[11–13)]. However, the effect of passive exercise in the supine position on vagal tone remains unknown.

This study investigated passive cycling movement in the supine position induces decreasing vagal tone using heart rate variability.

SUBJECTS AND METHODS

Eleven healthy males (mean age, 23.0 ± 2.0 years; height, 170.4 ± 6.1 cm; weight, 63.1 ± 7.8 kg) participated in this study. The subjects did not perform exercise for the past 24 h before, or consumed caffeine fort 12 h before the measurements. This study conformed to the Declaration of Helsinki, and informed consent was obtained from all participants in accordance with the protocol of the Ethics Committee of the Seirei Christopher University (13060).

Subjects lay supine for 5 min. One minute prior to the start of passive cycling for 10 min, their ankles were placed

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on the pedals and set in the straps of passive cycling movement device (Room March Pro, Yuubun Ltd., Japan). The lower legs moved through a range of motion defined of 90° and 180° knee joint angles at 60 rpm. Prior to the start of passive exercise and throughout the protocol, subjects were encouraged to remain passive and resist any urge to assist with leg movement.

Electrocardiography (ECG) was performed using one lead in a standard CM5 configuration, with three silver chloride monitoring electrodes placed on the chest. ECG traces were recorded at a sampling frequency of 1,000 Hz (Powerlab and software Chart5, AD instruments, Australia). Respiratory rates were monitered by an expired gas analyzer (AE-300S, MINATO, Japan) and maintained throughout the protocol at 0.25 Hz, with 2 s exhalation and 2 s inhalation, to elicit the effect of respiratory sinus arrhythmia (RSA)[14].

Heart rate variability was analyzed as root mean squared standard differences between adjacent R-R intervals (rMSSD) using measures in the time domain and spectrum domain analysis of the high frequency (HF) components, both of which provided information regarding parasympathetic heart modulation[15].

All data are presented as the mean ± standard deviation over the average data over the 5-min rest periods and 10-min passive exercise periods. rMSSD, HF, and HR were compared between rest and passive exercise using the paired t-test. The significance of all statistical tests was accepted at values of $p < 0.05$. All data are presented as mean ± standard deviation.

RESULTS

All data are shown in Table 1. Passive exercise induced significant decreases in rMSSD (rest, 66.6 ± 92.6 ms; passive exercise, 53.5 ± 32.5 ms, $p < 0.05$). No significant difference in HF or HR were observed during passive exercise compared with rest (their respective values; rest, 68.2 ± 6.9 bpm, 65.6 ± 12.0 n.u.; passive exercise, 70.2 ± 7.2 bpm, 67.9 ± 10.0 n.u., $p \geq 0.05$).

Table 1. HR, rMSSD and HF during rest and passive cycling movement

	Rest	Passive cycling movement
HR (bpm)	68.2±6.9	70.2±7.2
rMSSD (ms)	66.6±38.0	53.5±32.5*
HF (n.u.)	65.6±12.0	67.9±10.0

Values are mean ± SD
Significantly dirrerent from rest: * $p < 0.05$

DISCUSSION

The present findings demonstrate that passive exercise induces decreases in rMSSD, which is an indicator of vagal tone, despite having no effect on HR or HF.

The passive exercise model used in this study allows assessment of the mechanoreflex that is independent of the effects of metaboreflex and central command[5], and does not involve muscle contractions, as confirmed by the lack of changes in electromyography signals compared with rest[2].

Of the three mechanoreceptor subtypes known to exist, i.e., those responsive to muscle contraction, those responsive to stretch, and those responsive to both mechanical stimuli[16], passive limb movements are considered to predominantly stimulate stretch-responsive mechanoreceptors.

Activation of the mechanoreflex mediates cardio acceleration by reducing the excitability of the cardiac vagal motoneuron pool[17–18]. Furthermore, electrically evoked static contraction and passive stretch mechanically distort type III muscle afferents and reflexively reset the baroreflex neural arc to higher sympathetic nerve activity[19], resulting in increased cardiac[17] and renal[18] sympathetic traffic before the activation of the metaboreflex[20]. Thus, the activation of the muscle mechanoreflex mediates vagal inhibition and sympatho excitation in the absence of a central command and the muscle metaboreflex. This shifting of sympatho vagal balance is not counteracted by the baroreflex because neural input from the muscle mechanoreflex resets the baroreflex operating point to a higher operating pressure[19, 21] in a manner similar to that of the central command[22].

This study found no change in HR in response to passive cycling when supine. A previous study[23] demonstrated that SV and mean arterial pressure are unchanged in the supine position. Therefore, CO is likely solely driven by transient increases in HR because the contribution of the muscle pump is reduced and the baroreflex is not invoked while supine. The lack of change in SV in the supine position can be explained by a greater central blood volume, central venous pressure, and left ventricular end-diastolic volume[24–27].

Subjects' respiratory rates were controlled because RSA is known to affect the HF components that the decrease in the respiratory rate increases the HF component without changing the mean cardiac vagal tone[28]. Therefore, no significant changes in HF components were observed in response to passive exercise in the supine position. Moreover, passive cycling induced decreases in rMSSD because of vagal withdrawal in the supine position. These results from a model of passive exercise in the supine position suggest rMSSD is a more highly sensitive indicator of cardiac vagal tone than the HF component during very low intensity exercise, such as passive cycling movement.

In conclusion, passive cycling in the supine position induced decreases in vagal tone with no effect on the HF components or HR. These findings indicate rMSSD is a more sensitive indicator of the mechanoreflex during passive cycling than the measurements of the HF components.

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