Cardiac Autonomic Control and Muscle Sympathetic Nerve Activity during Dynamic Exercise

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Abstract We compared sympathetic outflow to the heart and skeletal muscle (MSNA) during dynamic exercise to test whether their mechanisms of control were the same. The sympathetic component to the heart was evaluated by heart rate variability analysis of the power spectrum. MSNA was recorded from the median nerve during graded leg cycling lasting 16 min at loads of 20, 40, 60, and 75% of maximal oxygen uptake ($\dot{V}O_{2\max}$) in the sitting position. The R–R interval and heart rate variability as well as low ($P_l$) and high ($P_h$) power frequency decreased with increasing exercise intensity while no significant change was observed in total power ($P_t$). The indicator of the cardiac sympathetic component, $P_l/P_h$, and the parasympathetic component, $P_h/P_t$, increased and decreased relative to exercise intensities, respectively. MSNA, represented as burst frequency (BF), was suppressed by 21.4% at 20% $\dot{V}O_{2\max}$, and thereafter BF increased with the exercise intensity by 23.5% and by 79.4% at 60 and 75% $\dot{V}O_{2\max}$, respectively, compared to the baseline level. There was a close positive and negative correlation between changes in BF and those in $P_l/P_h$ ($r = 0.593, p < 0.002$) and $P_h/P_t$ ($r = -0.681, p < 0.0001$), respectively. These results indicate acceleration of the sympathetic component of heart rate and increase in sympathetic outflow to the skeletal muscle during graded exercise. However, the exact control mechanisms of these sympathetic responses to graded exercise in two different organs remain unclear.

Key words: sympathetic nerve activity, parasympathetic nerve activity, heart rate, metaboreflex.

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961
Recent microneurographic studies have demonstrated that human muscle sympathetic nerve activity increases during isometric handgrip exercise [1–3], as well as dynamic arm [4] and leg cycling [5], is associated with elevated blood pressure. However, dissociation between the MSNA response and heart rate (HR) response during muscular exercise was reported [6], and this control mechanism is not well understood.

Elevated MSNA activity and blood pressure persist during ischemia following exercise, and these observations have been attributed mainly to muscle metaboreceptors [1, 7–9]. Furthermore, the magnitude of the response is coupled with the concomitant decrease in muscle pH [9] and the magnitude could be diminished by inhibition of lactate production by treatment with dichloroacetate [10]. These metabolic changes activate group III and IV afferent nerves [11, 12]. However, in conscious human subjects, somatosympathetic stimulation induced by post-muscle contraction ischemia did not maintain a heart rate above the resting level, while MSNA remained at an elevated level, indicating a difference between MSNA and HR control during muscle contraction [1, 6, 13].

Experiments in animals with sciatic and aortic nerve denervation showed that increased heart rate or cardiac sympathetic nerve activity (CSNA) could be produced by stimulation of thin afferent group IV nerves [14]. However, only small increases could be obtained with stimulation of GIII, whose afferent traffic could also impinge on MSNA neurons and enhance MSNA during muscular exercise [15].

Recent studies of heart rate variability have shown spectral analysis to be a good tool for assessing these sympathetic and parasympathetic components of the autonomic nervous system [16, 17]. These components during dynamic exercise have shown that the response patterns of sympathetic and parasympathetic systems to dynamic exercise are similar to those obtained by pharmacological blockade of either or both CSNA and parasympathetic nerve activity (PSNA) [18, 19].

The purpose of the present study was to test whether the sympathetic outflow to the heart assessed by spectral analysis and to the skeletal muscle was the same during graded cycling. We assessed the relative components of the sympathetic and parasympathetic systems of heart rate response during various exercise intensities of light to moderate cycling. Regression analysis of this sympathetic responsiveness showed a close positive correlation between MSNA changes and the sympathetic component of rising heart rate, while these were inversely related to the parasympathetic component.

MATERIALS AND METHODS

Subjects. Seven male students participated in the study. They did not participate in any regular training program but were relatively active and in good health. Their mean age, height, weight, maximal oxygen uptake (\(V_{O_{2,max}}\)), and lactate threshold were 24.5 (3.3, SD) years, 170 (0.5) cm, 70 (9) kg, 40 (6)
ml/min/kg, and 58% $\dot{V}_{O_2, max}$ (6, $n=4$), respectively. Each subject was given a detailed explanation of the experimental objectives, protocol, and possible discomfort and risks, and they gave their written informed consent before participation in the experiments. This study was approved by the Institutional Human Subject Protection Committee, Toyota Technological Institute.

**Procedure and experimental protocol.** Each subject performed a progressive exercise protocol on an electrically braked bicycle ergometer in the sitting position (Aerobike 800, Combi, Tokyo, Japan). The load was increased by 20 W/min until the subject could no longer maintain the pedaling frequency (60–70 rpm). $\dot{V}_{O_2, max}$ was determined by the Douglas bag method. Oxygen and carbon dioxide contents of expired gas were analyzed by an expired-gas monitor (1H2A, San-Ei, Tokyo Japan), and pulmonary ventilation was determined by a wet-gas meter (NWK-10A, Shinagawa Seiki, Tokyo, Japan). Simultaneously, in four of seven subjects arterialized blood was drawn from a finger tip at rest and during exercise every minute and was analyzed for lactate by an electrode-enzymatic method (YSI 1500L, Japan Scientific Instruments, Tokyo, Japan). The lactate threshold was estimated from the nonlinear increase in blood lactate concentration versus % $\dot{V}_{O_2, max}$ by multiregression analysis [20].

On the second day, each subject performed four different loads of bicycle exercise in the sitting position with the upper limbs free from supporting his body for 16 min (Fig. 1). The crank level of the pedals was set 10 cm lower than the hip joints. Exercise loads were 20% (I), 40% (II), 60% (III), and 75% (IV) of

![Image of experimental position of graded cycling](image)

**Fig. 1.** Experimental position of graded cycling. MSNA, muscle sympathetic nerve activity; ECG, electrocardiogram; BP, blood pressure.
\( V_{\text{O}_2, \text{max}} \), which were estimated from the curve of oxygen uptake versus exercise load (in W) determined from the exercise test conducted on the previous day.

In the second test, electrocardiogram (ECG) and MSNA were recorded continuously during graded exercise. Blood pressure determined by finger arterial blood pressure (Finapres 2300, Ohmeda, USA) was also measured throughout exercise. Systolic and diastolic blood pressure were determined as the means of 10 heartbeats every minute. Mean blood pressure (MBP) was calculated as one-third of the pulse pressure plus the diastolic pressure. On the 3rd day, each subject performed the same exercise protocol to determine oxygen consumption, and measurements were performed during the last minute of each load of graded exercise.

**Sympathetic nerve recording.** Multunit muscle sympathetic nerve discharge was recorded by inserting a tungsten microelectrode into the right median nerve at the elbow. Identification of MSNA was based on the criteria described previously [21]: 1) spontaneous burst discharges with a rhythm the same as that of the heartbeat; 2) burst discharges activated during baroreceptor unloading with the Valsalva maneuver; and 3) burst rhythms that did not change with sensory stimuli such as loud noise or electrical stimulation of the nerve trunk. The neurogram was fed to a differential amplifier, amplified $1.0-1.5 \times 10^5$ times through a band-pass filter (500-3,000 Hz), and stored on FM magnetic tape (DTR-36, Kyowa, Tokyo, Japan) for later analysis. The neurogram was fed to a full-wave-rectified and capacitance-integrated circuit (time constant 0.1 or 0.05 s) and recorded on a pen recorder. The paper speed was set at 5-25 mm/s, as appropriate for identification by inspection of the integrated neurogram tracing and represented as burst frequency (BF) (bursts/min). In 7 of 14 tests in 10 volunteers, MSNA was recorded throughout the cycling exercise, but the other 7 tests failed to record MSNA completely during exercise because of displacement of the electrode position from the muscle sympathetic nerve with movement of the arm or the body. Thus, the former seven tests were analyzed.

**Spectral analysis.** HR was measured continuously from surface electrocardiogram using standard bipolar leads with an ECG (AT-601G, Nihon Kohden), and stored on magnetic tape with neurograms. After experiments, the analog output of the ECG was differentiated, and the resultant QRS spikes triggered an analog circuit to generate a train of rectangular impulses. The impulse train was processed on a real-time basis with a personal computer (PC-9801 Vm2, NEC) at a sampling frequency of 1,000 Hz and stored sequentially for data analysis. A 3-min HR at rest before exercise and the last 3-min HR data during cycling at four different intensities were subjected separately to spectral analysis. There was a small number of abnormal R–R intervals caused by body movements during exercise. These abnormal intervals were corrected by either omitting (for those <300 ms) or inserting beats (for those with double or triple lengths of the proceeding intervals) [18]. Thereafter, unequal R–R intervals were aligned sequentially to obtain equal spectral samples. Spectral analysis of the HRV was
performed according to the autoregressive model [22, 23]. From the spectrum, the integrated powers in 0.1–0.15 Hz ($P_l$) and 0.15 Hz ($P_h$) were calculated. Sympathetic and parasympathetic activities on the sinoatrial node were evaluated by $P_l/P_h$ and $P_h/P_0$, respectively [17, 22], where $P_0$ was the total spectral power.

**Statistics.** A repeated-measures one-way analysis of variance was used to test for exercise intensity effects with respect to the resting control. Post hoc analysis was performed with Student’s $t$-test. Statistical significance was established with values of $p < 0.05$. The relationship between the change in oxygen uptake, burst rate, and HRVs was examined by linear regression analysis.

**RESULTS**

**Physiological response to graded exercise**

Mean values of physiological measurements at rest and during graded cycling are summarized in Table 1. The mean values of oxygen uptake, ventilation, HR, and MBP in the control rest period (0.271±0.027 l/min, 10.3 l/min, 67.7±1.9 beats/min, and 82.6±4.1 mmHg) were increased by 97, 103, 11, and 13%, respectively, at the lowest intensity of exercise (stage I). This was followed by a linear increase up to the highest intensity of exercise (stage IV).

**Heart rate variability changes during exercise**

The R–R interval, which averaged 892±29 (±SE) ms at rest, was shortened slightly (797±21) at 20% $V_{O_2, max}$ and decreased to 517±8.9 ms at 40% $V_{O_2, max}$, reaching 390±6 ms at the highest intensity (75% $V_{O_2, max}$). The decrease in the R–

| Table 1. Physiological measurements at rest and during graded exercise. |
|-------------|-------------|-----|-----|-----|-----|
|             | At rest     | I   | II  | III | IV  |
| Oxygen uptake (l/min) | 0.271±0.017 | 0.534±0.042* | 1.224±0.107* | 1.780±0.142* | 2.220±0.153* |
| % $V_{O_2, max}$ (%) | —           | 18.7±1.2 | 42.9±2.1 | 62.1±2.9 | 77.1±1.3 |
| Ventilation (l/min)    | 10.0±1.0    | 20.3±2.5* | 31.7±1.7* | 47.0±3.4* | 65.2±5.4* |
| Heart rate (beats/min) | 67.7±1.9    | 75.3±1.8* | 105.0±1.6* | 128.8±8.1* | 154.1±2.9* |
| Mean blood pressure (mmHg) | 82.6±3.3    | 93.7±4.7* | 109.0±4.4* | 126.0±5.6* | 134.6±5.8* |
| MSNA burst frequency (bursts/min) | 33.1±1.4  | 26.0±1.8* | 34.5±2.5 | 40.9±2.9* | 59.4±4.4* |

% $V_{O_2, max}$, % of maximal oxygen uptake; MSNA, muscle sympathetic nerve activity.

*Significantly different from control rest period ($p < 0.05$).

Vol. 45, No. 6, 1995
R interval and increase in HR were relative to the exercise intensities (Figs. 2 and 3). Likewise, the coefficient of variation of the R–R intervals (CV_R: standard deviation of consecutive R–R intervals for 3 min/mean R–R interval at the given period×100) as an index of HR variability decreased progressively with increasing exercise intensity (Fig. 4). The decreases from control values at rest were significant except at 20% $V_O_{2\text{max}}$. The decreased heart rate variability changes reflected decreases in HRV spectra in the high and low frequency power spectral components in relation to exercise intensity (Table 2 and Fig. 5A–C), but no significant changes in total power were observed. The ratio of $P_i/P_h$, used as a selective index of cardiac sympathetic activity ($i_{CSNA}$), and the ratio of $P_0/P_i$ used as

![Graph](image)

Fig. 2. Heart rate and mean voltage neurogram before and during graded cycling. The baseline of the mean voltage neurogram varied sometimes with mixing afferent and efferent nerve activities evoked with body movements during exercise. HR, heart rate; MSNA, muscle sympathetic nerve activity; bpm, beats per minute.

![Graph](image)

Fig. 3. Changes in R–R interval and heart rate at rest and during graded exercise. Vertical and horizontal bars of each plot indicate standard errors of means. $V_O_{2\text{max}}$, maximal oxygen uptake. Values are means and SE. *Significantly different from control rest period ($p<0.05$).
an index of parasympathetic activity (\(i_{PCNA}\)), were plotted against \(\% \dot{V}_O_{2\,max}\) (Fig. 6A and B). The \(i_{CNA}\) increased with increasing exercise intensity, although there were large inter-individual deviations at higher exercise intensities. In contrast, \(i_{PSNA}\) decreased with increasing exercise intensity.

**MSNA response to exercise**

MSNA burst frequency, which averaged 33.1 ± 1.4 bursts/min at rest, decreased significantly by 21.5% from the control value during exercise at 20% \(\dot{V}_O_{2\,max}\). There was no significant difference (4.2%) from control at 40% \(\dot{V}_O_{2\,max}\). At 60 and
Fig. 5. Changes in low (A) and high (B) frequency power and total power (C) at rest and during graded exercise. C, control rest; \( P_L \) low frequency power; \( P_H \), high frequency power; \( P_t \), total power. \( \dot{V}_{O_2\text{max}} \), maximal oxygen uptake. Values are means and SE. *Significantly different from control rest period \( (p < 0.05) \).

75% \( \dot{V}_{O_2\text{max}} \), the increases in burst frequencies were 28.5 and 79.4%, respectively (Table 1 and Fig. 7), and these were significantly different from the control values \( (p < 0.05) \).

Relationship between MSNA and HRVs

The relationship between the MSNA and HR responses is plotted as individual data points in Fig. 8. There was a high correlation coefficient between changes in...
Fig. 6. Changes in indices of sympathetic and parasympathetic components of the sinoatrial node of the heart at rest and during graded exercise. $P_s/P_h$, index of sympathetic component; $P_n/P_h$, index of parasympathetic component. Other explanations are the same as in Fig. 5.

Fig. 7. Changes in burst frequency at rest and during graded exercise. Other explanations are the same as in Fig. 3.
Fig. 8. Relationship between changes in burst frequency and heart rate in pooled data during graded exercise.

Fig. 9. Relationship between changes in burst frequency and index of sympathetic (A) and parasympathetic (B) components of the heart rate changes in pooled data during exercise.
MSNA BF and HR, despite their quite different responses to the graded cycling. The relationship between MSNA and HRV analysis of spectra in pooled data is shown in Fig. 9A and B. The ratios of $P_d/P_h$ and of $P_h/P_l$ were positively and inversely related to burst frequency changes during graded cycling, respectively. The correlation coefficients between changes in burst frequency and heart rate (changes in HR), $P_d/P_h$ and $P_h/P_l$ were 0.823 ($p < 0.001$), 0.593 ($p < 0.002$), and $-0.681$ ($p < 0.001$), respectively, indicating a relatively low coefficient between changes in BF and $P_d/P_h$ compared to those in HR and $P_h/P_l$.

DISCUSSION

We have demonstrated that the low power spectra over high power spectra areas ($P_d/P_h$) calculated from heart rate variability analysis (representative of cardiac sympathetic contribution as well as heart rate), increased with the dynamic exercise intensity, and showed a close correlation between sympathetic outflow bursts and the quiescent skeletal muscles. High power spectra over total power of heart rate variability ($P_h/P_l$) (i.e. parasympathetic nerve activity) decreased progressively with increasing exercise intensity and showed an inverse correlation with the MSNA BF changes during graded exercise.

The MSNA response during graded cycling between 20 to 75% of $\dot{V}_{O_2, max}$ increased with exercise intensity and was significantly higher at 60 and 75% $\dot{V}_{O_2, max}$ compared to the control value, despite MSNA suppression at a load of 20% (Fig. 7). This was in agreement with the previous reports that the magnitude of MSNA is correlated with the isometric handgrip force exerted [2, 3]. Victor et al. [9] showed that the initiation of MSNA increased during sustained and rhythmic handgrip exercise coupled with lowering of muscle pH as an index of glycolysis, such as lactate production rather than the cellular concentration of inorganic phosphate, and ADP production as an index of oxidative phosphorylation. Furthermore decreases in blood perfusion by applying a cuff to the upper arm during arm cranking at a constant work rate enhanced the MSNA response [4]. In this experiment, although we did not determine the blood lactate concentration, but measured oxygen uptake as an indicator of metabolic rate during graded cycling, four of seven subjects showed an average lactate threshold of 58% of $\dot{V}_{O_2, max}$ (see METHODS). Therefore, the significant increase in MSNA at work intensities above 60% of $\dot{V}_{O_2, max}$ may have been, at least in part, elicited reflexively by anaerobic metabolism in the contracting leg muscles, stimulating muscle metabo-

receptors, as the metabolite lactic acid could stimulate group III and IV muscle afferents [11, 12]. The effect of this muscle mechanoreceptor reflex on the MSNA response to dynamic exercise would be little because of the delayed increase in MSNA from the onset of the muscle contraction, while an instantaneous increase was observed instantly in the skin sympathetic nerve activity [1, 13].

The MSNA suppression observed here during very light dynamic exercise differed from the results of previous studies on the MSNA response to isometric
exercise [1–3]. No MSNA suppression was observed previously during isometric exercise [2, 3]. Since MSNA neurons in the medulla oblongata receive various excitatory and inhibitory input from central and peripheral structures [24], the sympathetic efferent activity may be determined by the results of competition of these input. In the resting sitting posture, MSNA is already activated by arterial and cardiopulmonary unloading produced by hemodynamic changes with gravitational force pulling blood to the lower part of the body [25]. With dynamic muscle contraction, however, the muscle pumping action and increased respiration could enhance venous return, and might stimulate cardiopulmonary baroreceptors to increase inhibitory input to the MSNA neuron [26]. However, very light dynamic exercise might not increase input from the metaboreceptors in the working muscles, whereas mechanoreceptors that have little effect on MSNA response might also be stimulated (see above).

In animal experiments, muscle contraction was shown to evoke elevation of blood pressure, while only small increases in heart rate induced reflexively via group III and IV muscle afferents were demonstrated by Coote et al. [8] and McClosky and Mitchell [27]. Subsequently, Sato et al. [14] demonstrated that cardiac sympathetic nerves in the rat could be evoked directly by stimulating group IV fibers originated from the skeletal muscle receptors. Recently, O'Leary [28] reported that muscle metaboreceptor stimulation by reduced perfusion could increase both arterial blood pressure and heart rate reflexively in conscious dogs during treadmill walking. However, during postexercise ischemia HR decayed whereas higher arterial blood pressure remained, but this response was abolished by muscarinic blocker treatment suggesting metaboreflex increase in HR could be inhibited by baroreflex vagal mechanisms [28, 29].

The previous suggestions [17] that $P_{H/P_X}$ might reflect the sympathetic component of the sinoatrial node in the heart and that $P_{H/P_I}$ is an indicator of parasympathetic activity are in agreement with the results of many investigators who have performed various manipulations such as pharmacological investigations in animals [16, 30] and humans [31], postural changes [32], and exercise [18, 19, 23, 33].

Although there are various techniques for estimating HRV spectra such as the FFT method [17, 33], the autoregressive method [22, 23], and the coarse-graining method [19], none of these has been confirmed as a reliable standard for quantitative assessment of cardiac autonomic nervous activities. We used the autoregressive method because the data length was too short to obtain improved resolution of HRV spectra by coarse-graining analysis. Yamamoto et al. [19] and Nakamura et al. [18] found that marked reductions in parasympathetic parameters ($P_{H}$ or $P_{H/P_I}$) occurred at mild exercise intensity using coarse-graining spectral analysis. However, no significant changes were observed in the sympathetic parameter $P_{H/P_X}$ during mild intensity, whereas a significant increase occurred above the resting level at a higher exercise intensity; i.e. 110% of ventilatory threshold and 60% $V_{O_2,max}$. These observations are in agreement with our autoregressive results that a greater decrease occurs in $i_{PSNA}$ ($P_{H/P_I}$) during light and mild exercise ($>40\% V_{O_2,max}$) and

*Japanese Journal of Physiology*
no significant change occurs in $i_{CSNA}$ ($P_i/P_h$) at 60% $V_{O_2,\text{max}}$ (Figs. 6A, B and 9A, B). However, Arai et al. [33] found no increase in the sympathetic component ($P_i/P_h$) during incremental exercise until exhaustion, despite evidence of a profound reduction in the parasympathetic component ($P_h$). Kamath et al. [23] also reported no changes in $P_i$ or $P_h$ during steady-state exercise at 50% $V_{O_2,\text{max}}$. The differences in findings are probably due to several factors: the method of spectral estimation, indicators used to evaluate parasympathetic and sympathetic components of the sinoatrial node of the heart, exercise pattern, and the length of data acquisition. $P_i/P_h$ increased and $P_h/P_i$ decreased as a function of the exercise intensity between light and moderate exercise ($>75\% V_{O_2,\text{max}}$), showing a pattern of change very similar to those reported in previous studies conducted using the pharmacological agents atropine and propranolol [34, 35]. These studies demonstrated that the relative contribution of the sympathetic and parasympathetic systems to heart rate control during exercise differed from each other in relation to exercise intensity; sympathetic activity increased progressively, while parasympathetic activity decreased gradually in relation to overall dynamic exercise intensity from low to maximum.

There is an interesting relationship between changes in HRV and MSNA response during graded exercise. The point at which $P_i/P_h$ increased and the rate at which the decrease in $P_h/P_i$ was reduced during graded exercise corresponded very closely to the point when MSNA BF reached the resting level during exercise, i.e. 100% of resting burst frequency or less, which was almost 40% $V_{O_2,\text{max}}$ or a heart rate of 110 beats/min (Fig. 9A and B). Thereafter, an appreciable increase occurred in $P_i/P_h$ and very little reduction in $P_h/P_i$ occurred, while MSNA was still increasing. Since MSNA could affect muscle vascular tone in both the active and non-active regions [36], the enhancement of MSNA itself at higher exercise work rates might modulate the blood pressure fluctuation-induced low frequency power of HRV. More recently, Matsukawa et al. [37] reported that both cardiac and renal sympathetic nerve activity in cats could be activated by not only muscle metaboreceptors stimulated by muscle contraction but also by mechanoreceptors stimulated by muscle stretching. Thus, there is an additional possibility of mechanoreflex-induced enhancement of either cardiac or muscle sympathetic nerve activity with increases in muscle force exerted to pedals during graded cycling [38]. In contrast, the primary reduction in $i_{PSNA}$ at lower exercise intensity may be due to central command [15, 39], and the volley could withdraw parasympathetic activity [40, 41].

Although there was a significant correlation between the burst frequency change and $P_i/P_h$ change, large inter-individual differences in the $P_i/P_h$ component were observed when BF increased above the resting value (Fig. 9A). Recently, Wallin et al. [42] suggested that sympathetic outflow to the skeletal muscle and heart during static handgrip and mental stress have a common pathway, but the balance was different between these maneuvers. The second point was a difference in the baroreflex effects on either heart rate controls and MSNA response, as shown
in post-exercise ischemia [27, 29]. Thirdly, since increased respiratory rate and tidal volume affect the HRV power spectrum, we did not control respiratory movement because voluntary respiration itself strongly modulates sympathetic outflow to the skeletal muscle [43, 44]. Thus, the distinguished increase in ventilation (Table 1) during graded exercise might partly modulate HRV response. Fourthly, the exaggerated increases in venous return with dynamic exercise may modulate heart rate variability independently from the neural effects, as with the Starling effect [45]. Finally, other factors are involved in the low frequency power spectrum of HRV in which not only sympathetic activity but also parasympathetic components are involved [31] as well as humoral and thermal changes [16]. Thus, there is a considerable number of factors involved in heart rate variability which must be characterized further to improve the rationale of power spectral calculation.

Saul et al. [46] found no relationship between the low power component of heart rate variability and MSNA burst frequency of the peroneal nerve during blood pressure elevation perturbation induced by vasoactive drugs at rest. However, they showed a weak but significant correlation between MSNA burst rate and low and high power frequency during blood pressure reduction. In the present study, however, close correlations between MSNA BF and $P_d/P_h$ and $P_h/P_i$ were observed during graded exercise despite a concomitant rise in blood pressure (Table 1 and Fig. 9). This was different from the results of our study, which suggested that the afferent input from the active skeletal muscle to the cardiovascular center of the heart and the skeletal muscle overwhelm the arterial baroreflex inhibition with exercise, especially at higher exercise intensities. Furthermore, we analyzed HRV and showed that heart rate increased up to an average of 154 (range 140 to 170) beats/min, while Saul et al. [46] performed HRV analysis at heart rates ranging between only 56 and 89 beats/min. Thus, the parasympathetic component in the heart rate change under these conditions might be greater than the sympathetic component [1], suggesting that during exercise not only baroreflex resetting and parasympathetic withdrawal but also sympathetic activity play an important role in accelerating the heart rate above 100 beats/min.

In summary, to test whether the sympathetic outflow to the heart and to the skeletal muscle was the same during graded cycling, we estimated cardiac sympathetic and parasympathetic components using spectral analysis of heart rate variability and recorded MSNA simultaneously by a microneurographic method. There was a close relationship between MSNA the and cardiac sympathetic index, $P_d/P_h$, during graded cycling, whereas there was an inverse relationship to the parasympathetic index, $P_h/P_i$. These results indicate acceleration of the sympathetic component of heart rate and increase in sympathetic outflow to the skeletal muscle during graded cycling. However, the exact control mechanisms of these sympathetic response to graded exercise in two different organs still remain unclear.

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*Japanese Journal of Physiology*


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