Effect of Maximal Intermittent Handgrip Training on Muscle Sympathetic Nerve Activity

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Aim: This study tested the hypothesis that maximal intensity conditioning does not alter increased muscle sympathetic nerve activity (MSNA) induced by central command at the same workload using 15-s strenuous intermittent handgrip contractions to evaluate training effects. Methods: Eleven males participated in this study. Handgrip contractions with 75% maximal voluntary contraction (MVC) were performed to evaluate the training effects on the MSNA responses before and after maximal intermittent handgrip training. The subjects trained three times a day, 4 days per week, for 4 weeks repeating maximal 10-s handgrip exercises 10 times during each session. The MSNA was recorded from the left tibial nerve using a microneurography technique. MSNA, heart rate and blood pressure data during a 15-s contraction were divided into two segments to compare the responses in each portion. The non-dominant hand was trained and the dominant hand used as a control. **Results:** The MSNA responses induced by central command during a 15-s intermittent contraction increased during the first bout from the baseline through five repetitions, but the enhancement in the MSNA did not change after the training program. Conditioning did not alter the maximal muscle force and heart rate, but did alter the blood pressure response. **Conclusion:** We found no change in the MSNA increment under the same exercise load with small training effects. The exercise scheme with 75% MVC intermittent strenuous exercise increased the MSNA response with central command involvement, suggesting that 15-s strenuous intermittent contractions were appropriate for evaluating MSNA training effects.

Keywords: MSNA; central command; strength training; intermittent handgrips

I INTRODUCTION

Muscle sympathetic nerve activity (MSNA) is augmented during exercise. Several factors are involved in the increase in MSNA during contraction, including central command and peripheral reflexes such as the metaboreflex and mechanoreflex, and MSNA should change with chronic exercise adaptation. It is generally accepted that the increase in MSNA mediated by the metaboreflex can be attenuated during exercise after conditioning ²⁷⁾. The mechanoreflex activated by generated muscle tension induces increased MSNA ⁹. The reflex is believed to be sensitized by metabolites that accumulate as exercise continues ^{9, 15)}. Sinoway et al. ²⁶⁾ reported that the increase in MSNA caused by mechanoreflex sensitization was reduced during non-fatiguing rhythmic forearm exercise after conditioning. Therefore, training attenuates exercise-induced augmentation of MSNA mediated by two peripheral reflexes.

Central command augments MSNA during a contraction, especially at the onset of a strenuous contraction²⁹⁾, whereas central command does not affect the MSNA response during moderate-intensity exercise²⁸⁾. It is not known whether conditioning affects the central command involvement in

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the MSNA responses. We observed exponential increases during continuous static handgrip contraction until exhaustion, which led us to speculate that central command plays a substantial role in enhancing MSNA when voluntary effort reaches the maximum level²²⁾. Because muscle force, initially at 25% of the maximum voluntary contraction (MVC), was maintained until exhaustion, the input from the mechanoreflex was thought to remain constant. Although the influence of the metaboreflex on the increase in MSNA was enhanced, an additional factor is needed to produce the exponential increase in MSNA during strenuous exercise. It was postulated that central command plays an important role in mediating increased MSNA when a near-maximal effort is performed.

We reported previously that maximal intermittent handgrip training augmented the increase in MSNA during continuous, fatiguing static handgrip contraction, and muscle strength and exhaustion time increased ¹⁹. However, there was no MSNA response during maximal rhythmic contraction following the same training protocol ¹⁹. Consequently, as rhythmic contraction at the onset, even at maximal strength, was thought to be too brief to consistently result in pronounced MSNA, we postulated that continuous nearmaximal voluntary effort is necessary to augment the MSNA increase.

Previously, we observed that MSNA increased constantly during continuous short (15-s duration) maximal handgrip contractions, indicating that central command contributes to the increase in MSNA not only at the onset of exercise but also during short continuous exercise⁸⁾. Augmented MSNA increases were seen during each exercise bout, although no effects of the metaboreflex and mechanoreflex were expected because the duration was too short for metabolites to accumulate and the muscle force was reduced, despite sustained maximal effort. We postulated that, unlike the MSNA responses to the metaboreflex, which decrease at the same work intensity²⁷⁾, those induced by central command may remain unchanged unless training enhances muscle strength¹⁸⁾.

Therefore, this study examined the hypothesis that no change in the MSNA increment would occur for exercise at the same intensity using 15-s strenuous intermittent handgrip contractions to evaluate training effects after maximal intermittent handgrip training over a 4-week period using short, repeated handgrip exercises.

II METHODS

1 Subjects

Eleven males (one left handed) participated in this study after providing informed consent. This study was approved by the Human Ethics Committee of Toyota Technological Institute. All subjects were healthy and had an average age of 22.7 \pm 2.7 years, height of 173.9 \pm 4.6 cm, and weight of 63.9 \pm 7.2 kg. The subjects were introduced to the routine 1 day before the experiment to familiarize then with the test protocol and devices.

2 Handgrip exercise and training

The subjects were asked to perform five 15-s handgrip contractions at 75% maximal voluntary effort using the nondominant hand to examine the contribution of central command to MSNA enhancement. Each subject performed the static handgrip exercise in the supine position after a 15-min rest in the supine position. The subjects started and stopped each contraction in response to an order from an experimenter issued in time with a metronome.

The maximal static handgrip was determined as the highest value of three 3-s attempts over a 3-min period in the standing position. The handgrip test was performed to evaluate the effects of training on the cardiovascular response at pre- and post-training sessions. The work performance of the handgrip was calculated by integrating the force curve.

The maximal handgrip training consisted of 10 contractions of 10-s each separated by 10-s rests. The subjects performed three sets per day, 4 days per week for 4 weeks. After training, five repetitions of a 15-s contraction were performed to evaluate training effects. The training paradigm required maximal effort with the non-dominant hand. The non-dominant hand was used to avoid unexpected negative effects, such as augmented blood pressure (BP) increments during exercise and to maximize training effects because the initial performance level was generally lower than in the dominant hand.

3 Measurements

1) Muscle sympathetic nerve recording

MSNA was determined for the left tibial nerve. A tungsten microelectrode with a shaft diameter of 0.1 mm was inserted at the popliteal fossa²⁰⁾. The nerve spikes, amplified by a fac-

tor of 100,000 were filtered at 500–3000 Hz, rectified, and integrated with a 0.1-s time constant to obtain the mean voltage. Muscle nerve activity was identified using the MSNA burst rhythm corresponding to the heart-beat cycle. The subjects performed the Valsalva maneuver to evaluate the MSNA outflow, and arousal stimuli were used to ensure signals from MSNA. The burst amplitude of the original mean voltage neurogram signal was normalized (100 arbitrary units) using the highest MSNA burst throughout the experiment setting⁸⁾. One experimenter monitored the MSNA burst frequency of all data. The burst numbers were expressed as the MSNA burst frequency (BF), and the area under each burst was referred to as total MSNA (TSNA). The subjects were allowed to breathe normally and instructed not to hold their breath or contract extra muscles, especially the legs.

2) Heart rate, blood pressure, and handgrip force

Heart rate (HR) was determined using the electrocardiogram R–R interval with bipolar electrodes positioned on the chest (AB 118, Nihon Kohden, Tokyo). Blood pressure (BP) was recorded with a Finapres blood pressure monitor (Ohmeda, Englewood, CO). A finger cuff was attached to the middle finger of the non-exercising hand. The handgrip force was displayed on a monitor placed above the bed. The subjects were able to perform 75% MVC with a strain gauge attached to a hand dynamometer (TKK 5710b, Takei, Tokyo) by looking at the force generated.

All signals were digitized at 200 Hz using an analogdigital converter (MP 100, BIOPAC Systems, Goleta, CA) and stored for later analysis on a personal computer with an online data-acquisition program, which also calculated the mean arterial pressure. Data at rest were averaged over 3 min before the experiment as the baseline. The subjects were placed in the supine position and rested while they were connected to all of the devices before experiments started.

4 Statistics

Each cardiovascular value was evaluated using two-way analysis of variance (ANOVA) for training effects as the main effect and the interaction with the contraction order (Prism 5, Graph Pad Software, San Diego, CA). Values are expressed as the means \pm standard error (SE), and P < 0.05 was determined a significant difference.

III RESULTS

Figure 1 shows an example of MSNA, handgrip performance, and respiration for one subject. MSNA, BF, and TSNA in both the training and control hands over a 15-s contraction increased from baseline (all P < 0.0001, Fig. 2). In each contraction, BF and TSNA were greater during the last 7-s portion than during the first 7-s for both hands (all P < 0.0001) (Fig. 3).



Fig. 1 The original records for one subject. Traces from top to bottom: muscle sympathetic nerve activity during the 15-s intermittent contraction, handgrip force, respiratory flow changes (upward inhalation, downward exhalation).

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Fig. 2 The MSNA responses during a 15-s intermittent static handgrip exercise. The upper and lower graphs show the burst frequency and total activity, respectively. The left and right graphs indicate the training and control hands, respectively. * P < 0.05, ** P < 0.001, and *** P < 0.0001: significant differences compared with baseline.



Fig. 3 The MSNA responses divided into the early and late portions of the 15-s contraction. The upper and lower graphs show the burst frequency and total activity, respectively. The left and right graphs indicate the training and control hands, respectively. * P < 0.05: significant difference between early and late portions of the contraction.

After conditioning, the handgrip force in the training hand did not change from before (45.3 ± 6.5 kg) to after ($46.1 \pm$ 8.3 kg) training (P = 0.55) and did not change in the control hand (48.5 ± 8.0 kg before and 47.4 ± 7.8 kg after training) (P = 0.44). The MSNA increases during the 15-s contraction (BF: P = 0.76 and TSNA: P = 0.11, Fig. 4) in the training hand did not differ after conditioning. Additionally, no training effect was observed in the two portions of the exercise for BF and TSNA in each contraction (BF: P = 0.94 early portion and P = 0.95 late portion, Fig. 5; TSNA: P = 0.47 early portion and P = 0.09 late portion, Fig. 6).

The baseline HR and BP did not differ change during training (HR: P = 0.87 and BP P = 0.19). The BP responses in the training hand increased throughout the contraction, whereas that in the control hand decreased (P < 0.05 and P < 0.05, respectively, Table 1). However, the HR during the 15-s contraction showed no training effect in either the non-dominant training hand or dominant control hand (P = 0.51 and P = 0.19, respectively, Table 1), and the HR responses in each portion of the exercise did not change after condition-



Fig. 4 The MSNA responses during a 15-s intermittent static handgrip exercise before and after maximal handgrip training. The upper and lower graphs show the burst frequency and total activity, respectively.



Fig. 5 MSNA BF responses in the early and late portions of a 15-s contraction in the training hand before and after conditioning. The upper and lower graphs show the burst frequency.



Fig. 6 TSNA response in the early and late portions of a 15-s contraction in the training hand before and after conditioning. The upper and lower graphs show the total activity.

ing in either hand (P = 0.77 early and P = 0.99 late portion, Table 2). BP in the early portion did not change after training (P = 0.25, Table 2), but it was greater in the late portion after training (P < 0.01, Table 2).

		Ν		Base	One	Two	Three	Four	Five	p-value
Heart Rate (beats·min ⁻¹)	Training Hand	10	B-T	62.3	81.7	81.5	81.5	82.5	84.0	p=0.51
		10	A-T	62.0	82.1	82.4	81.8	84.6	84.3	
	Control Hand	10	B-T	62.3	84.9	82.2	85.1	84.3	85.9	p=0.19
		10	A-T	62.0	82.5	82.5	82.1	83.1	84.1	
Mean Blood Pressure (mmHg)	Training Hand	9	B-T	80.5	81.7	88.7	87.3	85.7	83.3	(increased)
		9	A-T	76.0	86.7	90.7	92.7	95.5	94.8	p<0.05
	Control Hand	9	B-T	80.5	89.5	97.2	98.9	102.3	100.1	(decreased)
		9	A-T	76.0	85.7	92.0	94.4	95.1	96.4	p<0.05

Table 1. Heart rate and mean blood pressure before and after training during 15-s contraction.

B-T and A-T indicate before training and after training, respectively. N: number of subjects.

Table 2.	Heart rate and	mean blood	pressure in the Ea	lv and Late	portions in t	training hand	and before and	1 after training.
					p			"

		Ν		Base	One	Two	Three	Four	Five	p-value
Heart Rate (beats·min ⁻¹)	B-T	10	Early	62.3	78.1	78.7	78.1	80.1	81.1	p=0.77
	A-T	10	Early	62.0	77.4	80.1	78.4	80.9	81.3	
	B-T	10	Late	62.3	86.8	84.8	84.9	85.2	88.5	
	A-T	10	Late	62.0	86.4	84.5	84.6	87.7	87.1	p-0.99
Mean Blood Pressure (mmHg)	B-T	9	Early	80.5	79.5	88.0	87.4	84.3	81.8	p=0.25
	A-T	9	Early	76.0	82.9	85.9	89.2	91.2	90.9	
	B-T	9	Late	80.5	84.3	89.2	87.0	87.0	84.6	- p<0.01
	A-T	9	Late	76.0	90.8	95.4	95.7	99.5	98.5	

B-T and A-T indicate before training and after training, respectively. N: number of subjects. In the late portion after training mean blood pressure was greater than the early portion (p < 0.0001).

IV DISCUSSION

During 15-s handgrip at 75% MVC, MSNA, BF, and TSNA increased from baseline in the first through fifth bouts. Central command is thought to contribute to MSNA augmentation during short contractions, whereas increased strenuous voluntary effort is required to sustain constant muscle force. No change was observed in the MSNA response induced by central command in the non-dominant hand during the intermittent 15-s handgrip after the 4-week period of repeated maximal contractions, but BP increased at the same work load with no change in muscle strength. We considered two hypotheses to account for the results. First, it is possible that 75% MVC intermittent contractions were not adequate for evaluating MSNA changes, and second, it may be that maximal intensity exercise was insufficient to affect changes in MSNA responses.

1 Training effects on MSNA

Previously, we observed exponential increases in MSNA in the fatigued stage during static handgrip at 20% MVC²²⁾, leading us to postulate that greater enhancements in MSNA were elicited by both peripheral reflexes and central command contributions. We applied this finding to investigate the training effects of central command involvement in MSNA responses and found that MSNA increases were enhanced without metaboreflex influences during moderate static contraction to exhaustion, although the time to fatigue was extended, and the maximal handgrip force increased after maximal intermittent handgrip training¹⁹. We postulated that voluntary fatiguing efforts would produce maximal force generation at the final stage, when central command might be involved in the augmented MSNA responses.

The same previous study tested the MSNA response to maximal rhythmic contraction as an evaluation exercise, and no change in the MSNA response was seen ¹⁹). Because maximal intermittent contraction did not consistently induce MSNA increments ²⁹, no training effect of MSNA enhancement on central command involvement appeared during each contraction. Therefore, we assumed that to evaluate the role of central command in MSNA augmentation, strenuous effort must be continued so as to enhance the MSNA increment during the evaluation exercises.

We demonstrated that central command played an important role in enhancing the MSNA response during 15-s handgrip intermittent contraction with maximal voluntary effort, whereas the muscle force declined over 10 repeated exercise bouts⁸⁾. The intermittent contraction removed the influence of the MSNA increments caused by fatigue as a mental stress, inducing a rise in MSNA¹²⁾. This approach avoided the influence of the metaboreflex induced by accumulated metabolites.

Although a previous study indicated that central command enhanced the MSNA increases during 15-s intermittent maximal contractions, contractions performed at 75% intensity were needed to show enhanced MSNA increases caused by central command. Therefore, compared with the previous exercise paradigm in which the generating force might decline⁸, the subjects' efforts increased in this study so as to sustain the 75% voluntary force, thereby maintaining constant input from the mechanoreflex. When analyzing the cardiovascular data, we divided each 15-s bout into two portions to examine the enhanced contribution of central command to the values. The MSNA increases during the early portion should result mainly from central command²⁹ (Fig. 3), although mechanoreflex involvement was not ruled out because it begins within 4~6 s⁹. We found that the late part of the MSNA response increased more than the early portion despite the steady muscle force (Fig. 1), indicating that central command continued to increase with the augmented voluntary effort.

The MSNA enhancement during contraction was not necessarily stimulated by the mechanoreflex because the mechanoreflex involvement is associated with muscle force production ^{4, 6, 10}. Therefore, the exercise paradigm was considered to be adequate for evaluating the effects of central command on the MSNA responses during training. We believe that the MSNA increases were enhanced by training effects, resulting in an increase in muscle strength as long as continuous maximal voluntary effort was performed during the 15-s handgrip, similar to the final stage of a fatiguing contraction. However, the results indicated that no training influence on augmentation of the MSNA increases initiated by central command was obtained, suggesting that the voluntary effort was insufficient to stimulate further MSNA increments, as 15 s may be an insufficient duration to sustain a maximal voluntary effort.

Although no training effect on an augmented MSNA response was observed, we believe that the training protocol was adequate, as subjects in a previous study, who trained using the same training paradigm, increased their maximal force ¹⁹⁾. One of the reasons for the difference in results, including the lack of change in muscle strength, may be a difference in the study subjects. Although we followed the previous training protocol, in which the subjects trained at their own pace, the subjects in this study trained with less focus than the former subjects did.

In terms of a training effect on the MSNA changes, there is agreement that MSNA augmentation by the metaboreflex might be attenuated after moderate-intensity conditioning due to changes in the resistance to metabolites, as endurance training improved the resistance to hydrogen ions, one of the by-products of exercising muscle ^{1, 26, 27)}. These studies indicated that conditioning improved the MSNA response to the metaboreflex at the same workload.

However, we believe that moderate workloads were not suitable when the change in the MSNA increment induced by central command with strenuous effort was assessed,

and thus maximal intensity training might not influence the metaboreflex. Although no MSNA increment under the influence of central command was found with training in this study, the changes in BP showed that the training affected the subjects, although it did not change muscle strength. We think that the MSNA responses at the same workload were not affected after training because central command involvement in the cardiovascular response was enhanced, with an increase in muscle strength¹⁸⁾. Previously, we found that the increase in MSNA declined after 4 weeks of detraining ¹⁹. Therefore, in this study, the training was sustained at the initial level without a reduction in the physical properties of the subjects, indicating that the increase in MSNA induced by central command did not change at the same workload. However, it was also possible that the training load was insufficient, as the MSNA responses in the control subjects during training and detraining did not differ ¹⁹).

Metabolites sensitize the mechanoreflex ^{9, 15)}, although we do not know how the mechanoreflex itself affects the MSNA response after conditioning. Mechanically sensitized MSNA responses were reportedly attenuated during non-fatiguing rhythmic contractions after conditioning²⁶, but it was not clear how intermittent maximal intensity training affects such sensitization. The possibility of enhanced sensitivity was unlikely after 10-s intermittent handgrip training when the evaluation exercise was performed with unchanged mechanoreflex input⁸⁾. We attempted to minimize the influence of the metaboreflex and the effect of change in the mechanoreflex on MSNA increases by applying a short strenuous contraction in both the training and evaluation schemes. Consequently, appropriate training paradigms and exercise schemes must be chosen to evaluate the training influence on the MSNA response due to central command.

One major concern in our studies is the influence of respiration. However, Debeck et al. ⁵⁾ found no difference between the effects of spontaneous breathing and of controlled breathing at a constant tempo on the MSNA increase. We asked our subjects to avoid the Valsalva maneuver and not to hyperventilate because both actions affect the MSNA response during contraction¹¹⁾.

2 HR and BP response

We did not find effects of training on HR and BP at rest or during contraction, except that BP in the training hand increased during contraction after conditioning (Table 1). These results countered the results of Ray and Carrasco¹⁷), who found that resting BP and HR, but not MSNA, decreased following handgrip conditioning. Our inconsistent results suggest that the training load we used was insufficient to exhibit a training effect on HR.

BP increased in the training hand, unlike the control hand, during the 15-s contraction after conditioning due to a greater increase in the late portion of the contraction (Table 2). Glannattsaio et al.⁷⁾ demonstrated that BP in weightlifters was greater than that in sedentary subjects, and the peripheral vessel walls in weightlifters were thicker than those in their counterparts. Our results suggest that this training regimen had similar effects on the peripheral blood vessels (Table 2). However, we postulate that the central contribution to the BP change occurred after strenuous handgrip training, as BP changed in both the training and control hands (Table 1).

The non-dominant hand was used as the training limb because we expected that the training effects might be greater than in the dominant hand²⁷⁾. Greater training effects should be obtained when the initial fitness level is relatively moderate. However, there was no difference in the MSNA response during the 15-s contraction between the training and control hands either before or after conditioning, probably because the training hand demonstrated no training effect.

Although an electromyogram (EMG) was not used to visualize the increase in muscle activity with continuous voluntary effort, we previously reported increased EMG during static handgrip at 30% MVC while subjects watched an oscilloscope displaying their steady muscle force ²¹. It was thought that muscle activity reflected by the EMG increased gradually to continue generating a constant force ^{23, 24}. Since our subjects were asked to perform at 75% MVC, they tried to maintain the force at a level as much as possible. Although additional effort might have approached the maximum level, we believe that they maintained 75% MVC, as shown in Fig 1. However, one subject did not maintain constant force, and his data were removed from the analysis.

In conclusion, we found that the MSNA response during a 15-s handgrip contraction did not change after handgrip training involving three sets of 10 10-s maximal contractions a day 4 days a week for 4 weeks. The MSNA response induced by central command did not change under the same training load, although the training load, frequency, and duration may have been insufficient to induce a change in the study subjects. We postulate that the appropriate training scheme and training evaluation exercise are important to observe the effects of training on the MSNA response, and 15-s strenuous intermittent contraction was considered adequate for evaluating the MSNA responses induced by central command.

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筋交感神経活動に及ぼす間欠的最大握力トレーニングの効果

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Abstract

【目的】 本研究の目的はセントラルコマンドによる運動時の筋交感神経活動亢進はトレーニングの影 響を受けないという仮説の検証である.

【方法】 11名の健康な成人男性を対象とし、10秒間の最大努力ハンドグリップを10回繰り返すセッ ト運動を一日3回,週4日,4週間実施した.トレーニング側は非利き手とし、利き手は対照とした. 筋交感神経活動に対する最大努力ハンドグリップ運動の効果は15秒間のハンドグリップ運動とし、 最大握力(MVC)の75%張力発揮を5回繰り返す運動テストと、その際の筋交感神経活動反応を観 察した.筋交感神経活動は膝窩部脛骨神経より微小神経電図法により導出した.交感神経活動および 心循環反応は15秒間の運動の前半と後半に分割し解析した.

【結果】 間欠運動の1回目から運動開始と同時にセントラルコマンド性の筋交感神経活動の増加が 認められ、5回を通して同程度の反応が認められた. 間欠運動時の筋交感神経活動反応はトレーニン グ前後において差は認められなかった. 運動時の血圧反応はトレーニング後に高まる傾向を示したが、 心拍反応の差は認められなかった. トレーニング後の最大握力の増加量には有意差は認められなかった.

【結論】 筋収縮時のセントラルコマンド性筋交感神経活動亢進が最大努力ハンドグリップトレーニ ングの影響を受けないことを間欠的な高い筋収縮運動で確認した.この結果は,運動時の筋交感神 経活動亢進に対するセントラルコマンドの影響に関するトレーニング効果の評価法として間欠的な 75%MVC 筋収縮運動が有用であることを示唆する.

キーワード:筋交感神経活動;セントラルコマンド;筋力トレーニング;間欠ハンドグリップ運動