Forearm training attenuates sympathetic responses to prolonged rhythmic forearm exercise

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Systemic exercise conditioning increases maximal oxygen consumption (19). This is due in large part to an increase in blood flow to exercising muscle (19). As mentioned above, muscle vasodilator capacity can exceed cardiac pumping capacity. Therefore, training-induced increases in skeletal muscle blood flow are likely to be associated with reduced vasoconstrictor drive directed toward skeletal muscle blood vessels (16). Additionally, it is well established that at a given level of work, constrictor activity to inactive vascular beds is reduced after conditioning. These observations are consistent with the concept that sympathetic nervous system responses to rhythmic exercise are attenuated by exercise training (11). The neural systems responsible for this postulated reduction in sympathetic drive are unclear, although altered muscle metaboreceptor responses have been implicated in the attenuated muscle sympathetic nerve responses to static exercise after forearm conditioning (26).

However, the neural systems responsible for sympathetic nerve activation during rhythmic exercise may differ from those seen during static exercise. For example, a report from our laboratory demonstrated that nonfatiguing rhythmic forearm exercise performed at 25% maximal voluntary contraction (MVC) for 30 min led to a progressive rise in sympathetic nerve traffic directed to skeletal muscle (11). This is in contrast to the sympathetic nervous system responses observed during fatiguing static forearm exercise where metabolite-sensitive afferents are the key determinants of sympathetic activation. In this report we examined whether forearm exercise training would attenuate sympathetic nervous system responses to rhythmic forearm exercise. We measured heart rate, mean arterial blood pressure (MAP), muscle sympathetic nerve activity (microneurography), plasma norepinephrine (NE), and NE spillover and clearance (tritiated NE kinetics) during nonfatiguing rhythmic forearm exercise before and after a 4-wk unilateral forearm training paradigm. Training had no effect on forearm mass, maximal voluntary contraction, or heart rate but did attenuate the increase in MAP (increase in MAP: from 15.2 ± 1.8 before training to 11.4 ± 1.4 mmHg after training; P < 0.017), muscle sympathetic nerve activity (increase in bursts: from 10.8 ± 1.4 before training to 6.2 ± 1.1 bursts/min after training; P < 0.030), and the NE spillover (increase in arterial spillover: from 1.3 ± 0.2 before training to 0.6 ± 0.2 nmol·min⁻¹·m⁻² after training, P < 0.014; increase in venous spillover: from 2.0 ± 0.6 before training to 1.0 ± 0.5 nmol·min⁻¹·m⁻² after training, P < 0.037) seen in response to exercise performed by the trained forearm. Thus forearm training reduces sympathetic responses during a nonfatiguing rhythmic handgrip paradigm that does not engage muscle metaboreceptors. We speculate that this effect is due to a conditioning-induced reduction in mechanically sensitive muscle afferent discharge.

Exercise conditioning: autonomic nervous system; muscle sympathetic nerve activity and exercise

During systemic exercise the rise in vascular conductance is tightly linked to the cardiac output. This relationship ensures that blood pressure (BP) does not rise or fall to dangerous levels during exercise (18). However, it is clear that under the appropriate experimental conditions skeletal muscle vasodilator capacity can greatly exceed maximal cardiac output (1, 15). Thus some vasoconstrictor system must be activated during exercise to oppose this potent dilator system. A number of studies have provided evidence that increased sympathetic nervous system tone plays a crucial role in constricting these dilated skeletal muscle blood vessels (6, 8, 9, 11, 22, 27).

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Forearm training attenuates sympathetic responses to prolonged rhythmic forearm exercise. J. Appl. Physiol. 81(4): 1778–1784, 1996.—We previously demonstrated that nonfatiguing rhythmic forearm exercise at 25% maximal voluntary contraction (12 2-s contractions/min) evokes sympathoexcitation without significant engagement of metabolite-sensitive muscle afferents (B. A. Batman, J. C. Hardy, U. A. Leuenberger, M. B. Smith, Q. X. Yang, and L. I. Sinoway, J. Appl. Physiol. 76: 1077–1081, 1994). This is in contrast to the sympathetic nervous system responses observed during fatiguing static forearm exercise where metabolite-sensitive afferents are the key determinants of sympathetic activation. In this report we examined whether forearm exercise training would attenuate sympathetic nervous system responses to rhythmic forearm exercise. We measured heart rate, mean arterial blood pressure (MAP), muscle sympathetic nerve activity (microneurography), plasma norepinephrine (NE), and NE spillover and clearance (tritiated NE kinetics) during nonfatiguing rhythmic forearm exercise before and after a 4-wk unilateral forearm training paradigm. Training had no effect on forearm mass, maximal voluntary contraction, or heart rate but did attenuate the increase in MAP (increase in MAP: from 15.2 ± 1.8 before training to 11.4 ± 1.4 mmHg after training; P < 0.017), muscle sympathetic nerve activity (increase in bursts: from 10.8 ± 1.4 before training to 6.2 ± 1.1 bursts/min after training; P < 0.030), and the NE spillover (increase in arterial spillover: from 1.3 ± 0.2 before training to 0.6 ± 0.2 nmol·min⁻¹·m⁻² after training, P < 0.014; increase in venous spillover: from 2.0 ± 0.6 before training to 1.0 ± 0.5 nmol·min⁻¹·m⁻² after training, P < 0.037) seen in response to exercise performed by the trained forearm. Thus forearm training reduces sympathetic responses during a nonfatiguing rhythmic handgrip paradigm that does not engage muscle metaboreceptors. We speculate that this effect is due to a conditioning-induced reduction in mechanically sensitive muscle afferent discharge.

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attenuate central command and/or muscle mechanoreceptor-mediated responses. To test this hypothesis, we measured heart rate (HR), mean arterial pressure (MAP), MSNA, plasma norepinephrine (NE) levels, and arterial and venous NE spillover during a 30-min rhythmic handgrip paradigm before and after a 4-wk period of exercise conditioning. Our results demonstrated an attenuated increase in MAP, MSNA, and measurements of NE spillover seen with handgrip exercise. Therefore, conditioning can attenuate sympathetic responses to exercise paradigms that do not engage muscle metaboreceptors. We speculate that this effect is due to mechanoreceptor desensitization.

METHODS

Thirty-two normal male subjects not on medications were studied. The mean age of the group was 26 ± 1 yr. All subjects signed informed consent before being studied. No subjects with a history of forearm training were studied.

Two separate conditioning protocols were performed in 13 individuals (25 ± 1 yr). Protocol 1 was designed to determine whether unilateral forearm exercise conditioning evoked a local or generalized training response. This was a noninvasive protocol. Protocol 2 was an invasive study performed on a separate day. In this protocol we examined neural and hemodynamic responses to 30 min of rhythmic forearm exercise. A schematic representation of the two exercise protocols is shown in Fig. 1. In 19 separate study volunteers, reproducibility data were obtained.

Protocol 1

Protocol 1 was performed by both the dominant and nondominant forearm of 13 subjects before and after unilateral conditioning. Subjects performed rhythmic forearm exercise at ~70% of the highest sustainable 3-min workload. During this protocol we measured endurance time, HR, and MAP (Dinamap Critikon, Tampa, FL). These tests were, in general, performed at the same workload by each forearm. In two subjects, large differences in MVC were noted and thus the workload used for the endurance test was different in the two forearms. However, in all cases the workload was the same for each forearm before and after conditioning. In these subjects, forearm circumferences and volumes (water-displacement method) and MVC were determined before and after forearm conditioning.

Protocol 2

In protocol 2, we elected to study only the nondominant forearm because prior studies in our laboratory suggested that MSNA responses to forearm exercise are greater during nondominant than during dominant forearm exercise (24). Thus any training-induced attenuation in sympathetic responses would be more likely to be observed with nondominant than with dominant forearm training. Rhythmic handgrip (2-s contraction/3-s release) was performed at 25% MVC. Over the 30 min, this workload causes a substantial increase in MSNA yet is rarely fatiguing in normal subjects (3).

Instrumentation for protocol 2. The subjects were placed supine on a table. HR was recorded by electrocardiograph, and respiration was recorded with a pneumograph. The following instrumentation was performed. 1) A 20-gauge angiocatheter was placed in the radial artery of the dominant arm. This catheter was used for arterial blood sampling for NE spillover and clearance and for monitoring arterial BP (Transteq disposable pressure transducer, American Edwards Laboratories, Irvine, CA). 2) A 20-gauge intravenous catheter was inserted into a left foot vein. This catheter was used for the infusion of [3H]NE. 3) A 16-gauge intravenous catheter was inserted into an antecubital vein in the dominant forearm. This catheter sampled venous NE spillover and clearance. 4) A 16-gauge intravenous catheter was inserted into an antecubital vein in the nondominant forearm. This catheter was inserted to sample forearm venous lactate. For the two antecubital catheters, great care was taken to note the exact location of catheter insertion. This was done to ensure adequate pre- and posttraining comparisons. A strain-gauge

Fig. 1. Two protocols used in 13 subjects who underwent unilateral rhythmic forearm training. MVC, maximal voluntary contraction; NE, norepinephrine; MSNA, muscle sympathetic nerve activity; n, no. of subjects.
plethysmograph was then placed on the nondominant forearm to measure the blood flow response. Finally, a peroneal nerve electrode was placed in the right leg to measure MSNA. MSNA was recorded by using tungsten electrodes and standard recording techniques as previously described (24–26, 28–30).

The protocol to be described was performed at the same workload before and after conditioning. After instrumentation the subjects rested for 30 min. A 5-min bolus of sterile pyrogen-free L-[ring-2,5,6-3H]NE (15 µCi/m2) was begun (2, 5). The [3H]NE was obtained from Du Pont-New England Nuclear (Boston, MA). This was followed by an infusion of [3H]NE (0.35 µCi·min⁻¹·m⁻²) at a rate of 0.2 ml/min. Twenty minutes into the infusion, 7–10 ml of arterial and venous blood samples for NE and [3H]NE were drawn. The forearm venous blood was taken from the nondominant forearm. Between minutes 20 and 25 of the rest period (postbolus), eight forearm blood flows were measured, and MSNA was measured continuously. At 25 min, venous and arterial blood samples were again drawn. The 20- and 25-min blood sample values were pooled.

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The subjects then began exercise at 25% MVC at a cadence of 2–5 contractions/3–5 releases, the same rate and cadence of exercise as during protocol 1. For the NE and [3H]NE measurements, samples were taken at 5 and 10 min, and the lactate sample was taken at 10 min. During this 5-min period, three forearm flows were obtained and MSNA was recorded. The 5- and 10-min blood sample values were pooled as were samples obtained during minutes 20 and 25. Three flows were obtained between minutes 20 and 25, and MSNA was recorded and a lactate sample was taken at minute 25.

Training Regimen

Subjects were given a spring-loaded variable-tension handgrip dynamometer and were instructed to exercise five times per week. The subjects exercised the nondominant forearm at the 12 contractions/min cadence beginning at 30–35% MVC until fatigue. When the subject was able to exercise at a given workload for 30 min, the workload was increased. Each person exercised in the laboratory weekly to determine the duration of exercise.

Reproducibility Studies

In 19 different subjects (27 ± 1 yr) rhythmic handgrip, as performed in protocol 2, was performed twice 30 days apart without an intervening period of handgrip conditioning. During these studies, we measured MSNA (n = 11) and venous plasma NE (n = 16, none exercised forearm).

Statistical Analysis

The resting values, peak values and Δ values (change from peak to baseline) obtained before and after conditioning were analyzed by using a Wilcoxon matched-pairs signed-ranks test. P < 0.05 was considered statistically significant. All data are expressed as means ± SE.

RESULTS

Protocol 1

The results of protocol 1 are shown in Table 1. Unilateral forearm conditioning did not change forearm size (circumference and volume). Surprisingly, MVC decreased slightly (−4%) but significantly in the untrained forearm but did not change in the trained forearm. Endurance time increased by 144% in the trained forearm and by 16% in the opposite forearm. Of note, the MAP response to exercise was attenuated only when exercise was performed by the trained forearm.

There was no effect of training on the HR response to exercise performed by either arm.

Protocol 2

Effects of training on resting values (Table 2). After training, we noted an increase in resting burst count (18 ± 3 vs. 25 ± 4 bursts/min; P < 0.05). An increase in burst count was noted in 9 of 10 subjects. The increase in MSNA was not associated with an increase in either arterial or venous NE spillover. Surprisingly, we noted a small reduction in resting arterial plasma NE. The

Table 1. Protocol 1

<table>
<thead>
<tr>
<th></th>
<th>Trained Forearm</th>
<th>Untrained Forearm</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
</tr>
</tbody>
</table>
| Forearm
circumference, cm |     |      |     |      |
| 25%            | 27.6±0.5 | 27.6±0.6 | 27.9±0.5 | 28.0±0.5 |
| 50%            | 24.5±0.5 | 25.2±0.6* | 24.5±0.5 | 24.8±0.6 |
| 75%            | 19.4±0.4 | 20.0±0.7 | 19.6±0.4 | 19.7±0.5 |
| Forearm volume, ml |     |      |     |      |
| MVC, kg         | 49±2 | 50±2 | 53±2 | 51±2* |
| Endurance time, min | 14.9±1.9 | 36.3±3.1* | 23.9±2.3 | 27.7±2.5* |
| Exercise HR, beats/min | 83±2 | 84±3 | 82±3 | 84±2 |
| Exercise MAP, mmHg | 113±2 | 105±3* | 111±1 | 107±2 |

Values are means ± SE for 13 subjects for all comparisons. Pre, beforetraining; Post, after training; MVC, maximal voluntary contraction; HR, heart rate; MAP, mean arterial pressure. In all subjects, trained forearm was the nondominant one. Forearm circulations were measured at percentages of distance from midolecranon to styloid process. Forearm volumes exclude hand volume. Endurance times were measured at same isotonic workload before and after conditioning in each forearm. Post values for HR and MAP represent values at time fatigue occurred during the pretraining experiment. *P < .05 for Pre vs. Post (Wilcoxon signed-rank test).

Table 2. Baseline data before and after forearm conditioning

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>Pretraining</th>
<th>Posttraining</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Venous lactate, mmol/l</td>
<td>12</td>
<td>0.7±0.1</td>
<td>0.7±0.1</td>
<td>NS</td>
</tr>
<tr>
<td>Forearm blood flow, ml·min⁻¹·100 ml⁻¹</td>
<td>8</td>
<td>0.4±1.1</td>
<td>5.5±2.1</td>
<td>NS</td>
</tr>
<tr>
<td>Resistance, mmHg·ml⁻¹·min⁻¹·100 ml⁻¹</td>
<td>8</td>
<td>21.3±4.3</td>
<td>25.7±4.5</td>
<td>NS</td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>13</td>
<td>62±3</td>
<td>63±2</td>
<td>NS</td>
</tr>
<tr>
<td>Mean arterial blood pressure, mmHg</td>
<td>13</td>
<td>86±3</td>
<td>83±3</td>
<td>NS</td>
</tr>
<tr>
<td>Arterial norepinephrine, pg/ml</td>
<td>7</td>
<td>279±40</td>
<td>197±25</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Venous norepinephrine, pg/ml</td>
<td>7</td>
<td>296±37</td>
<td>220±39</td>
<td>NS</td>
</tr>
<tr>
<td>Arterial norepinephrine spillover, nmol·min⁻¹·m⁻²</td>
<td>7</td>
<td>1.6±0.3</td>
<td>1.3±0.2</td>
<td>NS</td>
</tr>
<tr>
<td>Venous norepinephrine spillover, nmol·min⁻¹·m⁻²</td>
<td>7</td>
<td>3.2±0.4</td>
<td>3.0±0.6</td>
<td>NS</td>
</tr>
<tr>
<td>MSNA, bursts/min</td>
<td>10</td>
<td>17±3</td>
<td>25±4</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

Values are means ± SE; n, no. of subjects. MSNA, muscle sympathetic nerve activity; NS, not significant. P values are for pre- vs. posttraining (Wilcoxon signed-rank test).
difference between the two baselines was <100 pg/ml in five of the seven subjects.

Responses to handgrip (Figs. 2 and 3). Conditioning attenuated the increase in lactate (Δ lactate; Fig. 2A) as well as the peak lactate response (Fig. 3A). The forearm flow response to rhythmic handgrip was attenuated (Fig. 3B) and forearm vascular resistance was higher (Fig. 3C) after conditioning.

The training paradigm had no effect on the HR response to exercise (Figs. 2D and 3D) but did attenuate the MAP response to exercise (Figs. 2E and 3E).

Conditioning lowered both arterial and venous NE (Fig. 3, F and G), Δ venous NE (Fig. 2G), Δ arterial and venous NE spillover (Fig. 2, H and I), and arterial NE spillover (Fig. 3H). The NE spillover values had reached steady state by the time of the earlier (5- and 10-min) samples because the early NE spillover values were not statistically different from the late ones for either arterial or venous samples (negative sign test; for 5- to 10-min vs. 20- to 25-min sample comparisons). NE clearance was not affected by conditioning.

The MSNA response to exercise was attenuated by conditioning. This was true for both Δ burst count (Fig. 2B) and Δ percent amplitude (Fig. 2C). The peak burst count was unchanged by conditioning (Fig. 3J).

Reproducibility studies. Resting and exercise MSNA responses were not different on the 2 study days (Δ percent amplitude pretraining: 158 ± 48 arbitrary units/min, Δ percent amplitude posttraining: 134 ± 40 arbitrary units/min, NS; Δ burst pretraining: 8 ± 2 bursts/min, Δ burst posttraining: 10 ± 1 bursts/min, NS). Venous plasma NE was also similar on the 2 study days (baseline NE pretraining 256 ± 29 pg/ml, baseline NE posttraining 291 ± 55 pg/ml, NS; peak NE pretraining 278 ± 22 pg/ml, peak NE posttraining 310 ± 44 pg/ml, NS).

DISCUSSION

In this report, we demonstrated that forearm conditioning attenuated sympathetic nervous system responses during nonfatiguing rhythmic handgrip at 25% MVC. A prior report from our laboratory suggested that the previous paradigm evoked a sympathoexcitatory response without engaging muscle metaboreceptors (3). Thus the results of the present report, when viewed in conjunction with the prior study, suggest that "localized" exercise training must be capable of reducing the sympathoexcitatory effects of central command and/or muscle mechanoreceptors.

Potential Explanation for Our Results

Our report does not allow us to definitively distinguish between a primary effect of conditioning on muscle mechanoreceptors vs. one on central command. Muscle mechanoreceptor activation is related to the

Fig. 2. Change from baseline to peak value seen during exercise (Δ) for 9 measured indexes during forearm handgrip before (Pre) and after (Post) handgrip conditioning. Units of measure are as follows: lactate (A), mmol/l; bursts (B), bursts/min; % amplitude (C), arbitrary units/min; heart rate (D), beats/min; mean arterial pressure (MAP; E), mmHg; NE (F and G), pg/ml; NE spillover (NE SO; H and I), nmol·min⁻¹·m⁻². Values are means ± SE; n, no. of subjects. *Statistically significant effect of conditioning (Pre vs. Post) for Δ values, P < 0.05 (by Wilcoxon matched-pairs signed-rank test).
amount of tension generated during contraction (10, 14). Because the tension generated during exercise was unchanged after conditioning, it could be argued that the attenuated sympathetic responses were not due to reduced mechanoreceptor activation. However, prior data from animal studies suggest that the discharge properties of mechanically sensitive group III muscle afferents can also be influenced by the concentrations of by-products of metabolism such as lactic acid within the muscle interstitium during contraction (21). In this report we demonstrate reduced lactate production during exercise after training. Accordingly, we would speculate that conditioning by reducing metabolite production leads to reduced mechanoreceptor activation during exercise. This in turn is associated with a reduced level of sympathetic nervous system activation. It should also be emphasized that a prior report in cats has suggested that repeated infusions of lactic acid leads to a progressive decline in group III discharge frequency. Thus it is possible that the recurrent bouts of exercise during the 30-day period of training leads to mechanoreceptor “desensitization” (21). Parenthetically, these comments are not intended to suggest that lactate is the sole chemical determinant of mechanoreceptor activity; rather we would speculate that venous lactate serves a useful marker for metabolic by-product production during exercise.

Other laboratories using different training paradigms have suggested that a portion of the autonomic training effects seen are due to a reduction in central command (4, 13, 20). However, it is unlikely that a decrease in central command would explain the reduced sympathetic responses seen after conditioning in the present report. In addition to having a prominent effect on sympathetic tone directed to skin (31) and a less-impressive influence on MSNA (30), central command is thought to be a major determinant of HR responses during forearm exercise (30). Because HR responses during rhythmic handgrip were unaffected by training, it is unlikely that central command was

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Fig. 3. Peak values during handgrip for 10 measured indexes before (Pre) and after (Post) handgrip conditioning. Forearm blood flow (B) is in ml·min⁻¹·100 ml⁻¹ and forearm resistance (C) is in mmHg·min·100 ml·ml⁻¹; other units of measure are as in Fig. 2. *Statistically significant effect of conditioning (Pre vs. Post) for Δ values, P < 0.05 (by Wilcoxon matched-pairs signed-rank test).
attenuated. Additionally, central command activation is related to the percentage of maximal tension utilized during exercise (30), and our neural measurements were performed at the same percent MVC before and after training. Additionally, muscle mass and MVC were unchanged by training. Thus it is unlikely that central command was altered by conditioning.

In protocol 1, we observed a large increase in endurance time in the trained forearm and a small but statistically significant increase in endurance time in the untrained forearm. Other laboratories have reported cross-training effects in response to unilateral limb training (7a, 32). The mechanism for these effects is unclear, although alterations in central motor outflow are thought to play an important role. The fact that training increased "control" forearm endurance, whereas HR and BP responses were not attenuated, suggests that motor and autonomic systems were being regulated by different neural systems. This finding provides further indirect evidence that the attenuated sympathetic nervous system responses seen after conditioning were not due to a reduction in central command.

We doubt that the attenuated sympathetic responses during handgrip were due to greater baroreceptor buffering after conditioning. If this were the case, then we would have expected attenuated HR and MAP responses after conditioning by the untrained forearm in protocol 1.

An interesting finding in the present report was that we noted an increase in resting MSNA (bursts/min) after forearm conditioning. The increase in burst count was seen in 9 of 10 subjects. The mechanisms for this potential training effect remain unclear and will require further study.

Limitations of the various neural indexes. A number of different indexes of sympathetic activity were attenuated during exercise after forearm conditioning. It should be noted that each of these indexes provides slightly different information and that each has specific strengths and limitations. Accordingly, our goal was to study a number of different indexes of sympathetic outflow. We reasoned that if a number of these indexes were influenced by conditioning in a similar fashion, then the results would corroborate each other and in the process strengthen our conclusions.

In the present report, plasma NE responses to exercise were reduced after conditioning. NE concentrations reflect a balance between the catecholamine release and clearance from the circulation. Therefore, a rise in NE concentration can be partially due to a decrease in NE clearance. Accordingly, changes in NE concentration should be used cautiously as a "direct" index of sympathetic activation. It is for this reason we measured NE spillover with the tritiated NE tracer methods. These data demonstrated that the attenuated NE responses during exercise were due to a reduction in NE spillover.

In this report, both arterial and venous plasma NE were attenuated and this was due to a reduced release of NE into the circulation (reduced NE spillover). However, these findings must be viewed in light of the limitations associated with plasma NE kinetics measurements. First, NE spillover represents <20% of the NE released at the nerve terminal. Second, factors aside from changes in NE release can affect the NE spillover (7). For these reasons, changes in systemic NE spillover with training could conceivably be due to factors aside from changes in sympathetic outflow. Additionally, measurements of arterial and venous NE spillover do not allow one to evaluate the distribution of NE release in the various organs (7). This last limitation precludes a definitive assessment of the regional effects of training on the sympathetic nervous system activity.

It should also be emphasized that conclusions regarding the effects of a given intervention (or clinical state) on NE kinetics can be influenced by the plasma sampling site (venous vs. arterial). In other words, it is possible that the presumed effect of conditioning on NE spillover could be due, in a large part, to the sampling site alone (12). To exclude this possibility, we measured NE spillover in both arterial and venous plasma and we did not observe an effect of sampling site on our general conclusions. For these reasons, we believe that the attenuated increases in arterial and venous NE spillover reflect a reduction in sympathetic outflow.

The attenuated rise in MSNA is consistent with a training-induced alteration in sympathetic outflow directed to lower limb skeletal muscle. However, this index does not provide evidence regarding sympathetic tone directed to other skeletal muscle beds.

In conclusion, handgrip exercise training reduces sympathetic responses to nonfatiguing rhythmic handgrip. We speculate that this effect is due to a training-induced reduction in mechanically sensitive muscle afferent discharge.

We appreciate the technical support of Steven Prophet, Danuta Huber, Cynthia Hogeman, and Sandy Whisler. We also acknowledge the expert typing of Jennifer Stoner.

This work was supported by National Heart, Lung, and Blood Institute Grants HL-44667 (to L. Sinoway) and HL-02654 (U. Leuenberger) and by National Aeronautics and Space Administration Grant PL 57-258 (to L. Sinoway). This work was also supported by a grant from the W. W. Smith Charitable Trust (to R. Zelis). L. Sinoway has an Established Investigator Award from the American Heart Association.

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Received 4 December 1995; accepted in final form 22 May 1996.

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