Isometric Training Lowers Resting Blood Pressure and Modulates Autonomic Control

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ABSTRACT

TAYLOR, A. C., N. MCCARTNEY, M. V. KAMATH, and R. L. WILEY. Isometric Training Lowers Resting Blood Pressure and Modulates Autonomic Control. *Med. Sci. Sports Exerc.*, Vol. 35, No. 2, pp. 251–256, 2003. **Purpose:** This study examined the effects of isometric handgrip training on resting arterial blood pressure, heart rate variability, and blood pressure variability in older adults with hypertension. **Methods:** Nine subjects performed four 2-min isometric handgrip contractions at 30% of the maximum voluntary contraction force, 3 $d \cdot wk^{-1}$ for 10 wk, and eight subjects served as controls. **Results:** After training, there was a significant reduction in resting systolic pressure and mean arterial pressure. In addition, power spectral analysis of heart rate variability demonstrated that the low frequency: high frequency area ratio tended to decrease. **Conclusions:** It is concluded that isometric training at a moderate intensity elicits a hypotensive response and a simultaneous increase in vagal modulation in older adults with hypertension. **Key Words:** HEART RATE VARIABILITY, BLOOD PRESSURE VARIABILITY, OLDER ADULTS, HANDGRIP EXERCISE

Power spectral analysis (PSA) of heart rate variability (HRV) has been used by researchers and clinicians as a noninvasive tool for the assessment of cardiac autonomic function in healthy and diseased individuals (1,11). This method has been used extensively to assess the cardiovascular responses to exercise (12). One of the potential benefits of measuring HRV in the frequency domain is the ability to identify frequency specific oscillations in heart rate signals that can be related to distinct physiological mechanisms and thereby provide an estimation of neurocardiac regulation. It is believed that a high-frequency peak (HF) around 0.15–0.4 Hz corresponds to parasympathetically mediated respiratory sinus arrhythmia and a low-frequency peak (LF) oscillating between 0.04 and 0.15 Hz is believed to reflect sympathetic modulation of cardiac function. The LF/HF ratio thus provides a noninvasive measure of sympathovagal balance.

It is generally accepted that regular endurance exercise can effectively attenuate resting arterial blood pressure (8,9). There is limited evidence that resistance training may also lower blood pressure, but the published results are equivocal, with some studies showing decreases (10) whereas others have shown no change (3). Most recently, isometric training has reportedly lowered blood pressure in short-term studies (21). However, there are few published investigations, and this form of exercise training has not yet been adopted for individuals with hypertension.

The acute circulatory responses to rhythmic and sustained isometric exercise in normal subjects have been investigated by several researchers (15,19,21). The rate of rise in blood pressure is proportional to the relative intensity of contraction as expressed by the percentage of the maximum voluntary contraction force (MVC) and the duration of the contraction (14), two factors that can be manipulated to minimize the pressor response. In previous work, it was demonstrated that blood pressure elevation during the final 30 s of 2-min isometric handgrip contractions at 30% of MVC was modest, approximately 16–17 mm Hg for both systolic and diastolic pressure (21). Thus, the lower magnitude of the pressor response during moderate-intensity isometric contractions interspersed with periods of rest avoids the risk of large acute rises in blood pressure yet may be an effective stimulus to elicit reductions in resting blood pressure over days and weeks (21).

At this time, only three studies have examined the blood pressure responses to isometric training (5,13,21). In an early investigation, Kiveloff and Huber (13) reported that training with whole-body isometric contractions elicited a hypotensive effect. In support of this finding, Buck and Donner (5) reported that the incidence of hypertension was inversely related to the quantity of isometric exercise associated with various occupations. The most recent evidence to suggest that isometric exercise can lower resting blood pressure was provided by Wiley and colleagues (21). In the first of their two studies, subjects with high-normal resting diastolic pressure did four 2-min handgrip contractions at 30% MVC, interspersed with 3-min rest intervals, on 3 $d \cdot wk^{-1}$ for 8 wk. Training resulted in a significant decline in both systolic and diastolic resting pressure, with no

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TABLE 1. Subject characteristics for training and control group.

Demographic	Training ($N = 9$)	Control ($N = 8$)
Age (yr)	69.3 ± 6.0	64.2 ± 5.5
Gender	5 male, 4 female	5 male, 3 female
Height (cm)	162 ± 15.0	162 ± 9.0
Weight (kg)	77.5 ± 15.8	88.9 ± 9.6
Resting SBP (mm Hg)	156.0 ± 9.4	152.0 ± 7.8
Resting DBP (mm Hg)	82.3 ± 9.3	87.1 ± 10.8
Exercise program participation (yr)	4.0 ± 2.9	2.0 ± 1.4
Medication (yr)	9.4 ± 11.8	9.2 ± 10.6

Data are given ± SD.

change in matched controls. In a second study, subjects with borderline hypertension did four 45-s contractions at 50% MVC with 1-min rest intervals between contractions, 5 $d \cdot wk^{-1}$ for 5 wk. Training was associated with a significant reduction in resting systolic and diastolic pressure, but with cessation of training, blood pressure returned to its pretraining value over a similar time course.

Potential mechanisms associated with the attenuated blood pressure response after isometric training have been proposed but not thoroughly investigated (21). These include modulation of the autonomic nervous system that could result in alterations in cardiac output and/or total peripheral resistance (TPR), or other changes. The purpose of the present study was to examine the effects of isometric handgrip training on resting blood pressure in older adults with isolated systolic hypertension and to determine whether alterations in autonomic balance may be a contributing factor.

METHODS

Subjects. Seventeen hypertensive men and women (10 male, 7 female) aged 60–80 yr ($\bar{x} = 67.5$ yr) volunteered to participate in this investigation (Table 1) and were randomized to either an isometric training group ($N = 9$) or reserved as controls ($N = 8$). Subjects were in a good general state of health and had a seated resting blood pressure of ≥ 140 mm Hg for systolic pressure and/or ≥ 85 mm Hg for diastolic pressure. All subjects were recruited from the McMaster University Seniors' Exercise and Wellness Program. The investigation was approved by the President's Committee on the Ethics of Research in Human Subjects, McMaster University, and subjects gave written informed consent to participate. At the start of the study, mean participation in the Seniors' Exercise and Wellness Program was 4.0 ± 2.9 yr in the isometric training group and 2.0 ± 1.4 yr in the controls, and 75% of the subjects had been taking medication to control blood pressure for 9.4 ± 11.8 and 9.2 ± 10 yr, respectively. In the experimental group, four were taking ACE inhibitors, two took β-blockers, and one each took a calcium channel blocker or a diuretic. Among controls, three were taking ACE inhibitors, and one each were treated with a diuretic or calcium channel blocker.

Experimental protocol. Isometric handgrip training (IHG) was performed 3 $d \cdot wk^{-1}$ for 10 wk. Training consisted of four 2-min isometric contractions at 30% MVC, using alternate hands with a programmed handgrip dynamometer (IBX H-101, MD Systems, Inc., Westerville, OH). Subjects had a 1-min rest period between each contraction. Subjects registered their maximum force with each hand on the dynamometer, which then gave visual targets representing 30% of their MVC, and visual and audible signals for proper timing of exercise contractions and rest periods. It also provided an accurate score of subjects' tracking of the targets. This protocol was chosen based on a previous study (21) in which IHG contractions held to submaximal levels elicited a hypotensive training response.

On the third training day of each week, subjects came into the laboratory and sat quietly in a relaxed state for at least 10 min before seated resting blood pressure measurements were taken. Blood pressure was measured in the left arm of each subject three times, with 1 min separating each reading, and the average of the three values was determined. Blood pressure was measured indirectly by auscultation using a standard sphygmomanometer. For the entire duration of the study, blood pressure measurement was taken by only one experimenter. In addition, at baseline and week 10, heart rate, respiration, and blood pressure were recorded in the supine position for 10 min to assess autonomic modulation of HRV and blood pressure variability (BPV) by using PSA, and then in the standing position to determine that the subjects had a normal orthostatic response as indicated by an increase in the LF:HF ratio.

Signal processing. The skin was prepared in six locations on the chest for a bipolar lead II placement. In addition to the ECG electrodes, three more electrodes were attached to the chest to simultaneously record the respiratory signal by plethysmography. Previous observation of the effects of respiratory sinus arrhythmia (RSA) on HRV made it essential for respiratory frequency to be monitored simultaneously with heart rate (4). We have noted previously in our laboratory that if the breathing frequency is below 0.12 Hz (~7.2 $breaths \cdot min^{-1}$), there is a fusion of LF and HF peaks due to entrainment, making their separate identification impossible. Consequently, we studied each power spectra individually and there was no fusion of peaks, likely because all of our subjects had breathing frequencies above 0.12 Hz. Continuous blood pressure recordings were also obtained from the subject's middle finger of the nondominant hand by photoplethysmography with a Finapres blood pressure monitor (Ohmeda 2300, Madison, WI).

Each of the ECG, blood pressure, and respiration signals were sampled at 500 Hz using a 12 bit analog-to-digital converter (CODAS, DATAQ Inc., Akron, OH). The signals were continuously displayed on a IBM laptop computer using WINDAQ data acquisition software. Adjustments were made to the sensitivity of the ECG signal before the recording to maximize the resolution of the successive R-R intervals. Three channels were displayed simultaneously, and the data were saved on the computer's hard drive.

Data analysis. Power spectral analysis of HRV was performed on all Windaq raw data files. Files were backed up on zip disk and transferred to a Pentium III computer for PSA of HRV by using MATLAB software. Each of the ECG files recorded during the supine position and the stand-

ing position for each subject (at baseline and week 10) were analyzed using HRV power spectral computational software. An RR-interval tachogram was found from the continuous ECG data by using a QRS detection algorithm, then the RR tachogram was inspected for ectopic beats. If any ectopic beats were present, they were corrected using a linear interpolation algorithm (11). Those files that contained several ectopic beats were eliminated from the analysis, unless a sufficiently long portion (3-min minimum) of the ECG recording was ectopic free and could be analyzed in isolation. A total of two subjects from the control group were excluded from statistical analysis because of excessive ectopic beats (> 5 min^{-1}).

Systolic and diastolic blood pressure values were identified for each beat and an equally sampled time series at two samples/second was constructed for each variable. A 10th-order autoregressive (AR) model was fitted to the time series data and an AR power spectra was computed. Data lengths of a minimum of 128 s were used for computation. After identification of low- and high-frequency peaks for each power spectra, the power under each frequency band was integrated, and a ratio of these powers (LF:HF ratio) was computed for each condition.

Statistical analysis. The data were analyzed using a two-factor (group $\times$ time) ANOVA with repeated measures, with a Tukey A *post hoc* procedure to evaluate specific differences between means. An alpha level of <0.05 was considered to be statistically significant. All descriptive data in the text and Table 1 are presented as means $\pm$ SD, whereas the figures are $\pm$ SE for the sake of clarity.

RESULTS

Effects of Exercise Training on Resting Blood Pressure

Systolic blood pressure. There was a significant main effect for time ($P < 0.001$) as well as a significant group $\times$ time interaction ($P < 0.005$). Systolic blood pressure (SBP) decreased in the training group (156 $\pm$ 9.4 mm Hg to 137 $\pm$ 7.8 mm Hg) versus the control group (152 $\pm$ 7.8 mm Hg to 144 $\pm$ 11.8 mm Hg) ($P < 0.0005$) (Fig. 1). *Post hoc* analysis showed a difference between groups at week 8 and a difference from week 1 for the training group at weeks 6, 7, 8, 10, and 11.

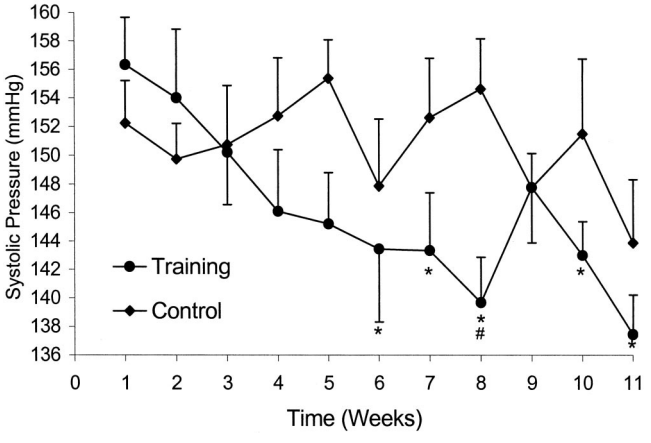

FIGURE 1—Systolic blood pressure responses (means ± SE) by isometric exercise trained ($N = 9$) group and control ($N = 8$) group. Week 1 points represent initial measurements; 2–11 the 10 wk of training; * weekly averages significantly different from week 1; # significant differences between groups ($P < 0.05$).

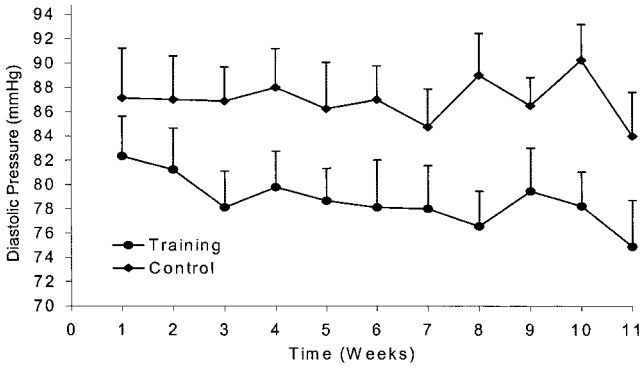

FIGURE 2—Diastolic blood pressure responses (means ± SE) by isometric exercise trained ($N = 9$) group and control ($N = 8$) group. Time scale is the same as Figure 1.

Diastolic blood pressure. As demonstrated in Figure 2, diastolic blood pressure (DBP) showed a downward trend in the training group (82 $\pm$ 9.3 mm Hg to 75 $\pm$ 10.9 mm Hg) compared with the control group (87 $\pm$ 10.8 mm Hg to 84 $\pm$ 9.6 mm Hg), but the difference was not statistically significant.

Mean arterial pressure (MAP). Analysis of MAP revealed a main effect for time ($P < 0.009$) and a significant group $\times$ time interaction ($P < 0.025$). The attenuated MAP response can be seen in Figure 3, with the training group showing an 11 mm Hg reduction (107 $\pm$ 8.53 mm Hg to 96 $\pm$ 8.7 mm Hg) versus a 5 mm Hg reduction in the control group (109 $\pm$ 9.1 mm Hg to 104 $\pm$ 9.3 mm Hg). *Post hoc* analysis confirmed differences between groups at weeks 5, 8, and 10. As well, a significant difference from week 1 for the training group was observed at weeks 8 and 11.

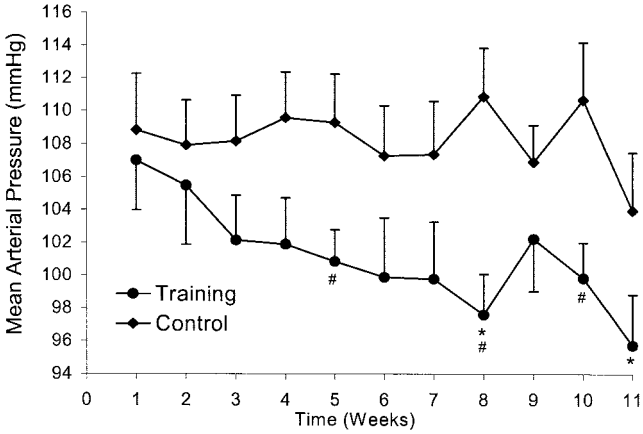

FIGURE 3—Mean arterial blood pressure responses by isometric exercise trained ($N = 9$) group and control ($N = 8$) group. Time scale and significance symbols are the same as previous figures.

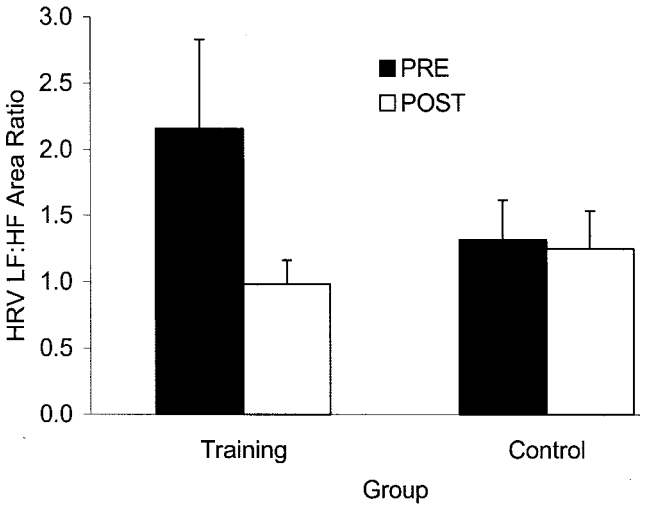

FIGURE 4—Low-frequency/high-frequency area ratio of heart rate variability during supine rest for isometric exercise trained ($N = 9$) group and control ($N = 6$) group. Values are means (± SE) for weeks 0 and 10.

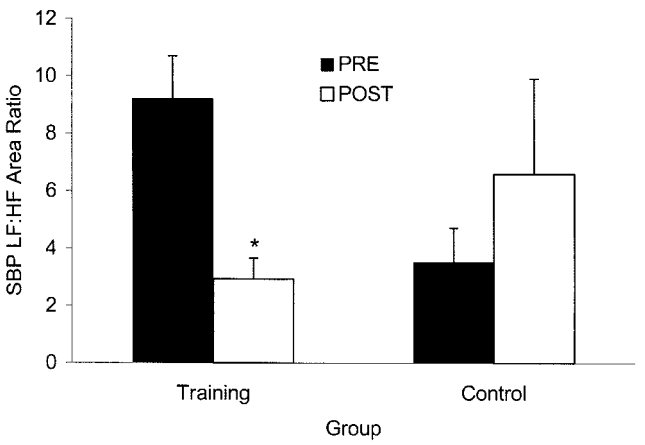

FIGURE 5—Low-frequency/high-frequency area ratio of systolic blood pressure variability during supine rest for isometric exercise trained ($N = 9$) group and control ($N = 6$) group. Values are means (± SE) for weeks 0 and 10; * significant differences in LF/HF area ratios pretraining versus posttraining.

Effect of Exercise on Heart Rate and HRV

Heart rate. The training group showed no change in resting heart rate after training (70 ± 14.2 to 68 ± 12.1 beats·min^{-1}), whereas there was a rise in resting heart rate in the control group (70 ± 8.5 to 76 ± 15.3 beats·min^{-1}; $P < 0.05$).

HRV. Analysis of LF area revealed a main effect for time ($P < 0.05$) and a nonsignificant reduction for the training group (6150 ± 1002 (beats·min^{-1})^2 to 5009 ± 1507 (beats·min^{-1})^2) versus the control group (5797 ± 731 (beats·min^{-1})^2 to 5253 ± 1195 (beats·min^{-1})^2). Analysis of HF area demonstrated a significant group × time interaction ($P < 0.002$). The training group showed an increase in HF area after training (4958 ± 1689 (beats·min^{-1})^2 to 5775 ± 1661 (beats·min^{-1})^2), whereas the control group showed a change in the opposite direction (5230 ± 1736 (beats·min^{-1})^2 to 4849 ± 1342 (beats·min^{-1})^2). Although there was a trend toward a reduction in the LF:HF area ratio, it did not reach statistical significance (training = 2.2 ± 1.9 to 1.0 ± 0.5; control = 1.3 ± 0.7 to 1.3 ± 0.6; $P = 0.13$) (Fig. 4).

Effect of Exercise Training on Blood Pressure Variability

Based on the BPV data, analysis of SBP revealed a main effect for time for both LF area ($P < 0.006$) and HF area ($P < 0.04$). In addition, a significant group × time interaction was observed for LF area ($P < 0.02$), HF area ($P < 0.009$), and LF:HF area ratio ($P < 0.01$). LF area decreased significantly in the training group 221.1 ± 12.9 (mm Hg)^2 to 157.7 ± 36.4 (mm Hg)^2 with only a slight change in the control group from 176.1 ± 69.3 (mm Hg)^2 to 169.6 ± 63.7 (mm Hg)^2. As well, there was a significant increase in HF area for the training group from 33.6 ± 12.0 (mm Hg)^2 to 83.1 ± 25.2 (mm Hg)^2, whereas the control group showed only a modest reduction from 92.1 ± 58.4 (mm Hg)^2 to 85.4 ± 60.1 (mm Hg)^2. These changes led to a corresponding decrease in LF:HF area ratio for the training group (9.2 ± 4.22 to 2.9 ± 2.1), whereas the control group showed a change in the opposite direction (3.5 ± 2.4 to 6.6 ± 6.4) (Fig. 5).

Analysis of DBP values showed a main effect for time for LF area ($P < 0.04$) and HF area ($P < 0.05$). There were no further main effects or interactions for the rest of the DBP data.

DISCUSSION

Effects of training on resting blood pressure. It is very significant that this study showed an attenuation of resting blood pressure in these older individuals, most of whom were already managed using antihypertensive medications.

The changes in SBP and DBP (decreases of 19 and 7 mm Hg, respectively) in this study confirmed the previous report by Wiley et al. (21) which showed that brief repeated submaximal isometric contractions over several weeks elicited a hypotensive effect in hypertensive subjects (reductions of 13 and 15 mm Hg in SBP and DBP). Because both studies used similar methodologies, we are able to make direct comparisons between our results and those obtained by Wiley et al. (21).

In addition to the present work and that by Wiley et al. (21), only two other studies have investigated the effects of isometric training on resting blood pressure in hypertension. These include a study of Kiveloff and Huber (13) that examined whole-body isometric exercise on resting blood pressure and a study by Buck and Donner (5) that compared components of occupational isometric effort with the incidence of hypertension. Neither study reported negative physiological effects as a consequence of isometric exercise in their subject populations. However, neither study used a standardized method to quantify the evoked isometric effort,

nor did they propose any logical physiological mechanisms contributing to the observed blood pressure reduction.

Effects of training on heart rate variability. PSA of HRV is used to describe indices of autonomic modulation. Although the LF:HF ratio has been used as a measure of sympathovagal balance in human and animal studies, there are some limitations. Although the high-frequency power is mediated by vagal inflow to the sinoatrial node, the power within the low-frequency peak arises from predominantly sympathetic contributions. Separation of sympathetic and vagal influences contributing to the power within the low-frequency area has not been successful thus far (2). Therefore, the LF:HF ratio is used to compare two conditions within the same subjects and study the changes within the sympathovagal influences contributing to the autonomic modulation of the heart rate signal.

In hypertension, HRV power spectra have consistently shown an elevated LF component and a blunted or nonexistent HF component (16) suggesting an elevated sympathetic modulation is associated with this disorder. The data from the present study are in agreement with these observations, as the LF area invariably showed more power than the HF area before training. The trend to a decrease in LF:HF area ratio following the isometric training intervention suggested a concurrent reduction in sympathetic modulation and an increase in vagal modulation.

It has been shown that endurance trained athletes tend to have a larger vagal component and a smaller sympathetic component at rest compared with their untrained counterparts (7). A change in autonomic regulation after physical training in sedentary individuals has also been demonstrated by Pagani et al. (16) who explored the relationship between physical training and HRV in 11 subjects with mild hypertension. Subjects underwent a 6-month training program that consisted of jogging for 20 min at least 5 d a week in addition to a daily routine of calisthenics. They reported that the training intervention produced a training bradycardia, a decrease in the LF component and an increase in the HF component. To our knowledge the present study is the first to demonstrate similar effects of isometric training on HRV in individuals with elevated systolic pressures

Effects of training on blood pressure variability. The LF and HF autospectrum components of systolic arterial pressure variability are similar to those observed for HRV (16). An increase in arterial pressure variability has been demonstrated in patients with hypertension and an elevated LF component has been documented during daytime recordings in ambulatory patients (16). The greater LF component in hypertension can be explained by higher sympathetic activity. In this study, we were able to confirm an elevated LF component of arterial pressure with a correspondingly small HF component as is characteristically seen in hypertensive patients. Before the exercise intervention, the power of the LF area for the training group was much greater than the power of the HF area but after training there was a significant decrease in LF area and a significant increase in HF area resulting in a reduced LF:HF area ratio. Taken together, the findings of this study suggest that training with brief, repeated bouts of isometric handgrip exercise leads to decreased sympathetic, and enhanced parasympathetic modulation of both heart rate and blood pressure.

Mechanisms associated with an attenuated blood pressure response. It was suggested previously that changes in sympathetic neural influences on total vascular resistance might act as a sufficient stimulus to produce a decline in blood pressure after isometric handgrip training (21). While the present study does not reveal the precise mechanisms responsible for these changes the data suggest that the attenuated blood pressure response was at least in part mediated by alterations in autonomic nervous system activity. Previous investigators have proposed alternative mechanisms such as decreased muscle sympathetic nerve activity (17,19), increased muscle blood flow (18) and baroreceptor resetting (16).

Following 6 wk of isometric handgrip training, Somers et al. (19) showed attenuation in sympathetic nerve activity in their subjects as measured by microneurography. The authors proposed that the decrease in sympathetic nerve activity was probably secondary to a reduction in muscle chemoreceptor stimulation. Surprisingly, a reduction in blood pressure did not accompany the decrease in sympathetic nerve activity and the investigators speculated that the method of blood pressure measurement may have contributed to their nonsignificant finding. Alternatively, they proposed that vasoconstriction in other vascular beds (e.g. mesenteric and renal) might override any blood pressure reductions resulting from the decrease in sympathetic nerve activity in skeletal muscle. In the present study we did not measure muscle sympathetic nerve activity so we are unable to offer any further insights.

In a recent investigation, Sinoway et al. (17) reported a reduction in muscle sympathetic nerve activity that was accompanied by a decrease in lactate production during forearm exercise after training. They suggested that venous lactate served as a useful marker of metabolic by-product production during exercise. Perhaps the reduction in sympathetic nerve activity resulted from a decrease in metabolite accumulation following training as was suggested by Mostoufi-Moab et al. (15). If this is the case, endurance forearm training might have the potential to decrease anaerobic metabolism and increase aerobic metabolism during exercise (19). Other investigators have suggested that the measurement of muscle sympathetic nerve activity can be used as an indirect index of chemosensitive muscle afferent activation (20). This is because there is little or no increase in sympathetic nerve activation during handgrip work until the chemoreceptors are stimulated by a decrease in muscle pH and other metabolites.

Another physiological adaptation documented following training is an increase in blood flow to the exercising muscle. It is uncertain as to whether the increased flow is the result of reduced sympathetic vasoconstrictor influences (6) and/or the result of increased intrinsic vasodilatory capacity (18). Sinoway et al. (18) reported that after 4 wk of handgrip exercise, a localized training induced increase in forearm blood flow occurred that was associated with an increase in

vascular vasodilatory capacity. The increase in blood flow resulted from a decrease in minimal peripheral resistance. This adaptation could possibly explain the attenuated blood pressure response seen in our investigation.

The present study explored the relationship between brief bouts of isometric handgrip training and subsequent changes in autonomic modulation of heart rate and blood pressure. We showed that training attenuated the resting arterial pressure and was associated with a corresponding change in sympathovagal balance. We conclude that isometric handgrip training at a modest intensity could be a useful adjunct to the pharmacological treatment of hypertension.

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REFERENCES

1. Akselrod, S. Spectral analysis of fluctuations in cardiovascular parameters: a quantitative tool for the investigation of autonomic control. *Trends Pharmacol. Sci.* 9:6–9, 1988.
2. Akselrod, S., D. Gordon, F. A. Ubel, D. C. Shannon, A. C. Barger, and R. J. Cohen. Power spectrum analysis of heart rate fluctuations: a quantitative probe of beat to beat cardiovascular control. *Science* 213:220–222, 1981.
3. Blumenthal, J. A., W. C. Siegel, and A. Appelbaum. Failure to reduce blood pressure in patients with mild hypertension. *JAMA* 266:2098–2101, 1991.
4. Brown, T. E., L. A. Beightol, J. Koh, and D. L. Eckberg. Important influence of respiration on human RR-interval power spectra is largely ignored. *J. Appl. Physiol.* 75:2310–2317, 1993.
5. Buck, C., and A. Donner. Isometric occupational exercise and the incidence of hypertension. *J. Occup. Med.* 27:370–372, 1985.
6. Clausen, J. P., K. Klausen, B. Rasmussen, and J. Trapjensen. Central and peripheral circulatory changes after training of the arms or legs. *Am. J. Physiol.* 225:675–682, 1973.
7. Dixon, E. M., M. V. Kamath, N. Mccartney, and E. L. Fallen. Neural regulation of HRV in endurance athletes and sedentary controls. *Cardiovasc. Res.* 26:713–719, 1992.
8. Frick, M. H., A. Konttinen, and S. Sarajas. Effects of physical training on circulation at rest and during exercise. *Am. J. Cardiol.* 12:142–150, 1963.
9. Hagberg, J., D. Goldring, A. Ehsani, et al. Effect of exercise training on the blood pressure and hemodynamic features of hypertensive adolescents. *Am. J. Cardiol.* 52:763–768, 1983.
10. Harris, K. A., and R. G. Holly. Physiological responses to circuit weight training in borderline hypertensive subjects. *Med. Sci. Sports Exerc.* 19:246–253, 1987.
11. Kamath, M. V., and E. L. Fallen. Power spectral analysis of heart rate variability: a noninvasive signature of cardiac autonomic function. *CRC Biomed. Eng.* 21:245–311, 1993.
12. Kamath, M. V., E. L. Fallen, and R. Mckelvie. Effects of steady state exercise on the power spectrum of heart rate variability. *Med. Sci. Sports Exerc.* 23:428–434, 1991.
13. Kiveloff, B., and O. Huber. Brief maximal isometric exercise in hypertension. *J. Am. Geriatr. Soc.* 9:1006–1012, 1971.
14. Macdougall, J. D., D. Tuxen, D. Dale, J. Moroz, and J. Sutton. Arterial blood pressure response to heavy resistance exercise. *J. Appl. Physiol.* 58:785–790, 1985.
15. Mostoufi-Moab, S., E. Widmaier, J. Cornett, K. Gray, and L. Sinoway. Forearm training reduces the exercise pressor reflex during ischemic rhythmic handgrip. *J. Appl. Physiol.* 84:277–283, 1998.
16. Pagani, M., V. Somers, R. Furlan, et al. Changes in autonomic regulation induced by physical training in mild hypertension. *Hypertension* 12:600–610, 1988.
17. Sinoway, L., J. Shenberger, L. Gretchen, et al. Forearm training attenuates sympathetic responses to prolonged rhythmic forearm exercise. *J. Appl. Physiol.* 81:1778–1784, 1996.
18. Sinoway, L., J. Shenberger, J. Wilson, S. Mclaughlin, T. Musch, and R. Zelis. A 30-day forearm work protocol increases maximal forearm blood flow. *J. Appl. Physiol.* 62:1063–1067, 1987.
19. Somers, V., K. Leo, R. Shields, M. Clary, and A. Mark. Forearm endurance training attenuates sympathetic nerve response to isometric handgrip in normal humans. *J. Appl. Physiol.* 72:1039–1043, 1992.
20. Victor, R. G., L. A. Bertocci, S. L. Pryor, and R. L. Nunnally. Sympathetic nerve discharge is coupled to muscle cell pH during exercise in humans. *J. Clin. Invest.* 82:1301–1305, 1988.
21. Wiley, R., C. Dunn, R. Cox, N. Hueppchen, and M. Scott. Isometric exercise training lowers resting blood pressure. *Med. Sci. Sports Exerc.* 24:749–754, 1992.