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# Isometric handgrip training reduces arterial pressure at rest without changes in sympathetic nerve activity

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**Ray, Chester A., and Dario I. Carrasco.** Isometric handgrip training reduces arterial pressure at rest without changes in sympathetic nerve activity. *Am J Physiol Heart Circ Physiol* 279: H245–H249, 2000.—The purpose of this study was to determine whether isometric handgrip (IHG) training reduces arterial pressure and whether reductions in muscle sympathetic nerve activity (MSNA) mediate this drop in arterial pressure. Normotensive subjects were assigned to training ( $n = 9$ ), sham training ( $n = 7$ ), or control ( $n = 8$ ) groups. The training protocol consisted of four 3-min bouts of IHG exercise at 30% of maximal voluntary contraction (MVC) separated by 5-min rest periods. Training was performed four times per week for 5 wk. Subjects' resting arterial pressure and heart rate were measured three times on 3 consecutive days before and after training, with resting MSNA (peroneal nerve) recorded on the third day. Additionally, subjects performed IHG exercise at 30% of MVC to fatigue followed by muscle ischemia. In the trained group, resting diastolic ( $67 \pm 1$  to  $62 \pm 1$  mmHg) and mean arterial pressure ( $86 \pm 1$  to  $82 \pm 1$  mmHg) significantly decreased, whereas systolic arterial pressure ( $116 \pm 3$  to  $113 \pm 2$  mmHg), heart rate ( $67 \pm 4$  to  $66 \pm 4$  beats/min), and MSNA ( $14 \pm 2$  to  $15 \pm 2$  bursts/min) did not significantly change following training. MSNA and cardiovascular responses to exercise and postexercise muscle ischemia were unchanged by training. There were no significant changes in any variables for the sham training and control groups. The results indicate that IHG training is an effective nonpharmacological intervention in lowering arterial pressure.

physical training; isometric exercise; muscle sympathetic nerve activity

CHRONIC PARTICIPATION IN RESISTIVE exercise was once thought to elicit elevated resting arterial pressure by causing vascular hypertrophy and increased vascular resistance due to large increases in arterial pressure elicited by the isometric or resistive exercise. However, studies have shown this is not the case. Reductions in arterial pressure have been reported following isometric or resistive exercise training such as isometric handgrip (IHG) exercise or weightlifting (11–13, 18, 31). Furthermore, epidemiological data indicate that regular exposure to isometric activity at the workplace

lowers the incidence of hypertension (4). These limited findings suggest that isometric exercise training may become an important part of a nonpharmacological intervention to prevent and combat hypertension. However, only a few studies have examined the efficacy of purely isometric training on reducing arterial pressure. Moreover, the mechanism(s) responsible for the reduction in arterial pressure has not been investigated.

One mechanism that may lower arterial pressure with exercise training is a reduction in sympathetic nerve activity. Currently, no studies have examined the effect of isometric training on sympathetic nerve activity; therefore, the purpose of the present study was to examine 1) whether IHG training lowers arterial pressure at rest and 2) whether possible reductions in arterial pressure are associated with reductions in efferent muscle sympathetic nerve activity (MSNA).

## METHODS

**Subjects.** We studied 17 healthy, normotensive, untrained volunteers (11 females, 13 males) 19–35 yr of age. Written informed consent was obtained from all subjects, and the study was approved by the Institutional Review Board of the University of Georgia.

**Experimental design.** MSNA, heart rate, and mean arterial pressure (MAP) were measured at rest, during fatiguing IHG exercise at 30% of maximum voluntary contraction (MVC), and during 2 min of posthandgrip muscle ischemia before and after 5 wk of IHG training.

**Training protocol.** Nine subjects were trained using unilateral IHG of the dominant arm for a total of 5 wk. Each subject attended four training sessions per week. During each session, subjects performed four 3-min bouts of IHG at 30% MVC while sitting with the working arm extended toward the front. Each bout was separated by a 5-min rest period. Before every training session, each subject's MVC value was determined as the highest value obtained on three attempts, separated by 1 min of rest. The training protocol was adapted from an earlier study that demonstrated significant reductions in arterial pressure at rest (31). Eight subjects did not train and served as experimental time controls. The control subjects were periodically brought back into the laboratory (2 or 3 times per week) during the 5-wk period. An

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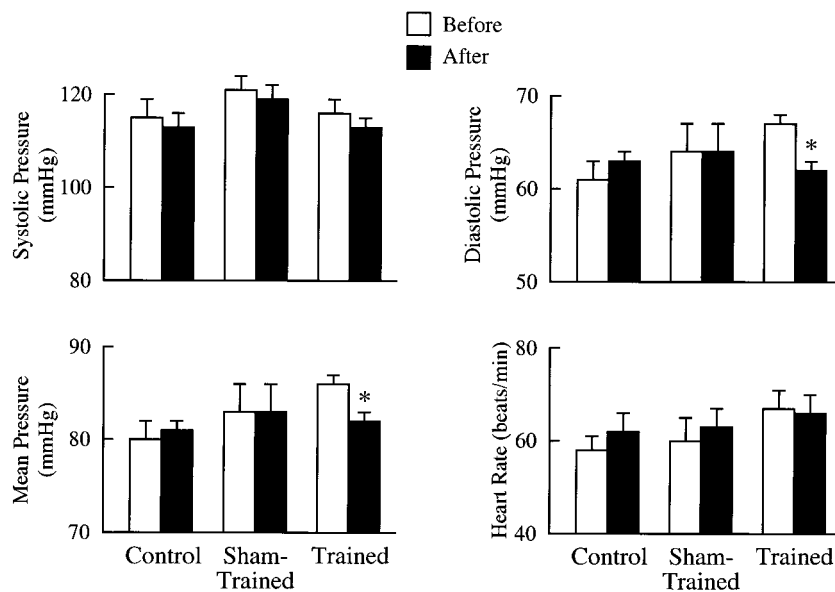


Fig. 1. Arterial pressure and heart rate at rest before and after 5 wk of isometric forearm training. Training elicited reductions in diastolic and mean arterial pressure, but no changes were observed in the control and sham-trained groups. \*Significantly different from before training;  $P < 0.05$ .

additional seven subjects served as sham-trained controls. The sham-trained subjects replicated the training program of the trained group with the exception that they held the handgrip dynamometer but generated no force during the exercise bouts.

**Testing protocol.** On 3 consecutive days, the subjects' resting arterial pressure and heart rate were measured before and after training. Measurements were taken three times after 15 min of rest in the supine position, and each measurement was separated by 5 min. These measurements were obtained at the same time of day for all subjects. The testing was conducted in a quiet and dimly lit room. On the third day after the measurements of resting arterial pressure and heart rate were obtained, the subjects in the trained and time-control groups were instrumented for measurement of resting MSNA (via the peroneal nerve). Ten minutes after a satisfactory nerve recording was obtained, MSNA, arterial pressure, and heart rate were recorded for 10 min from subjects in the supine posture. Because MSNA adaptations to isometric training during exercise had not been examined, after data were collected at rest the subjects then performed IHG exercise at 30% MVC to fatigue followed by postexercise muscle ischemia. A direct-reading digital meter connected directly to the dynamometer provided visual feedback of the subjects' force production, which assisted in maintaining the desired force output. Fatigue was defined as the point when the desired force production was not maintained for 5 consecutive seconds. Postexercise muscle ischemia was elicited by inflating a pneumatic cuff on the subject's exercising arm to suprasystolic levels (220 mmHg) 5 s before the end of IHG exercise. Verbal encouragement was given to all subjects during the IHG testing. Because the training protocol did not significantly alter MVC ( $\leq 5\%$ ), the same absolute workload was used for testing before and after training.

**Measurements.** Multifiber recordings of MSNA were made with a tungsten microelectrode inserted into the peroneal nerve. A reference electrode was placed subcutaneously 2–3 cm from the recording electrode. Criteria for an acceptable site for the MSNA recording were presented previously (30). The nerve signals were amplified, filtered with a bandwidth of 700–2,000 Hz, and passed through a resistance-capacitance integrating network with a time constant of 0.1 s to obtain a mean voltage display of the nerve activity.

Resting arterial pressure was measured using an automatic arterial pressure device (Accutorr 3, Datascope, Montvale, NJ). Continuous measurements of arterial pressure and heart rate during the exercise protocol were made using a Finapres arterial pressure monitoring unit (Ohmeda, Englewood, CO). Finapres measurements were taken to detect changes in arterial pressure and heart rate elicited by IHG and postexercise muscle ischemia.

All data, except arterial pressure and heart rate at rest, were collected online (MacLab 8e, ADInstruments, Milford, MA) with a Macintosh computer (Quadra 840AV). The mean voltage neurograms, arterial pressure tracings, and biotach outputs were routed to an online computer for monitoring and data collection purposes throughout the study.

**Data analysis.** The mean values for the three arterial pressure and heart rate measurements, taken while subjects were at rest before and after training over the 3 days of data collection period, were calculated. The second minute of IHG was used as a common nonfatiguing exercise endpoint, and the last 30 s of exercise were used for the fatigue data. Sympathetic bursts were identified by inspection of the mean voltage neurogram. MSNA was expressed in burst frequency (bursts/min) and percent change in total activity (sum of the area of each burst per min) from baseline. All variables were analyzed using a repeated-measures ANOVA (SuperAnova, Abacus Concepts, Berkeley, CA). All values are expressed as means  $\pm$  SE. Significance was considered at  $P < 0.05$ .

## RESULTS

The data for all variables for subjects at rest are presented in Figs. 1 and 2. Five weeks of IHG training resulted in a reduction in mean ( $86 \pm 1$  to  $82 \pm 1$  mmHg) and diastolic arterial pressure ( $67 \pm 1$  to  $62 \pm 1$  mmHg) for subjects at rest (Fig. 1). There were no significant changes in systolic arterial pressure ( $116 \pm 3$  to  $113 \pm 2$  mmHg), heart rate ( $67 \pm 4$  to  $66 \pm 4$  beats/min), and MSNA ( $14 \pm 2$  to  $15 \pm 2$  bursts/min) for subjects at rest after the 5-wk training period. There were no significant changes for subjects at rest in any of the variables for the sham-trained and control groups (Figs. 1 and 2).

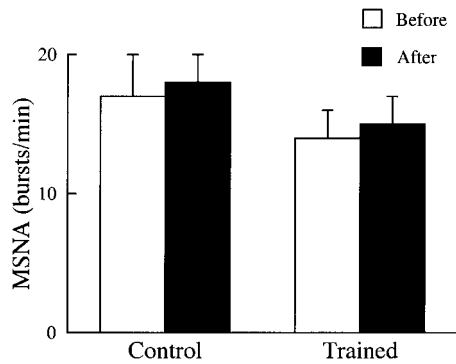


Fig. 2. Muscle sympathetic nerve activity (MSNA) at rest before and after 5 wk of isometric forearm training. Training elicited no changes in MSNA.

Data for all variables during the second minute of IHG exercise and fatigue and during posthandgrip muscle ischemia are presented in Fig. 3. IHG exercise elicited significant increases in heart rate, mean arterial pressure, and MSNA for both the control and trained groups (Fig. 3). During posthandgrip muscle ischemia, both MSNA and mean arterial pressure remained significantly elevated above baseline, whereas heart rate returned to baseline (Fig. 3). These responses during IHG exercise both at the second minute and fatigue and during posthandgrip muscle ischemia were not altered in the trained or control group. Training did not significantly change the time to fatigue for either group ( $4.1 \pm 0.4$  to  $4.4 \pm 0.4$  min and  $4.0 \pm 0.3$  to  $4.4 \pm 0.3$  min for the trained and control groups, respectively).

#### DISCUSSION

Hypertension is a serious health problem that is estimated to affect ~50 million Americans (5). Hypertension increases the risk of coronary heart disease, stroke, and kidney disease (15). Physical inactivity has been shown to be associated with hypertension in epidemiology studies (3, 20); thus physical activity has been recommended in the prevention and treatment of hypertension (1, 9, 14, 16). However, Arroll and Beaglehole (2) stated that the quality of the literature remains poor and that major design flaws are prevalent (e.g., no control group). Moreover, in studies that report a reduction in resting arterial pressure, the mechanism responsible for this reduction is equivocal.

The current study shows that 5 wk of unilateral IHG training elicits reductions in diastolic and mean arterial pressure at rest. Although the reported reduction in diastolic arterial pressure appears modest (5 mmHg), recent studies indicate that small reductions in diastolic arterial pressure in the population would have significant health benefits. A 2-mmHg drop in diastolic arterial pressure would lead to a 17% decrease in hypertension as well as a 6% reduction in coronary heart disease and a 15% reduction in stroke-related events (7). A 5- to 6-mmHg reduction in diastolic arterial pressure decreased coronary heart disease and stroke incidents by 16% and 38%, respectively

(7). Thus the 5-mmHg reduction reported in this study would have an important impact on these cardiovascular-related illnesses. Furthermore, our results support the concept that isometric training is an effective modality in the prevention of hypertension. Buck and Donner (4) found that jobs with regular exposure to moderate to high isometric activity lower the 5-yr hypertension incident rate by 29%.

The reduction in resting arterial pressure by IHG training supports the earlier reports by Kiveloff and Huber (18) and Wiley et al. (31). Unlike the current study, these studies used hypertensive patients and subjects whose arterial pressure was on the high range of normal (134/86), respectively. It is interesting to note that the reduction in diastolic arterial pressure

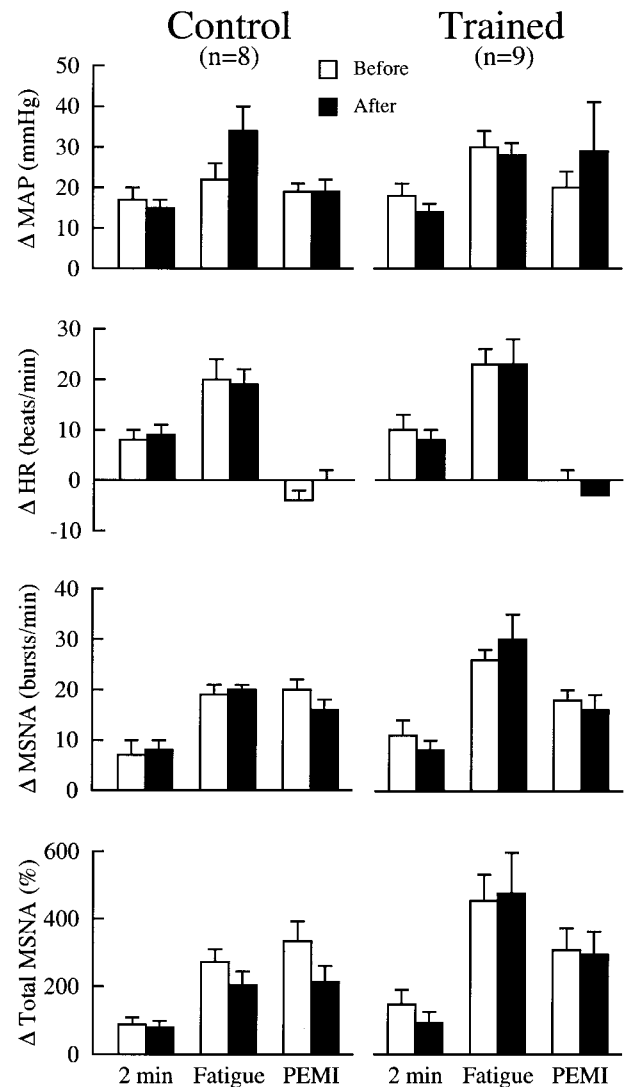


Fig. 3. Physiological responses to isometric handgrip during the second minute of exercise (2 min), fatigue, and postexercise muscle ischemia (PEMI) before and after isometric forearm training. Isometric handgrip elicited significant increases in all variables, and postexercise muscle ischemia elicited increases in mean arterial pressure ( $\Delta$ MAP) and MSNA ( $\Delta$ MSNA) for both groups. Responses to isometric handgrip and postexercise muscle ischemia were not significantly altered by training. HR, heart rate.

but not systolic arterial pressure in the current study is similar to reductions in two studies in which resistive weight training was used (12, 13). These specific and similar reductions in diastolic arterial pressure occurred in three distinctly different subject populations (younger and older subjects and borderline hypertensive subjects). Thus diastolic arterial pressure appears to be effectively lowered by isometric and resistive training in a large segment of the population.

What mechanism could be responsible for the reduction in arterial pressure observed with isometric forearm training? The lack of change in MSNA after training indicates that the lower arterial pressure was not related to a reduction in central sympathetic outflow. The failure of MSNA to change at rest with isometric training is also typically observed with either forearm or leg dynamic exercise training (23–26, 29) with one exception (10). Therefore, reductions in sympathetic outflow to skeletal muscle do not appear to be a prerequisite to lower arterial pressure in humans. However, it cannot be excluded that sympathetic outflow to other vascular beds (e.g., visceral regions) may have been reduced and may have contributed to the reduction in arterial pressure at rest. The data also indicate that altered muscle reflexes and greater sensitivity of the baroreflexes did not participate in the reduction of arterial pressure. Because diastolic and not systolic arterial pressure was reduced, this finding suggests that changes in central hemodynamics were not responsible for the changes in diastolic and mean arterial pressure. The lack of a change in heart rate also supports this notion.

Another possible mechanism for the reduction in arterial pressure is peripheral vascular adaptations. Because isometric exercise elicits marked increases in MSNA and norepinephrine release, vascular sensitivity to norepinephrine may be decreased with isometric training (17). However, the finding that arterial pressure responses to IHG exercise were not attenuated after training with comparable increases in MSNA argues against a change in vascular sensitivity to norepinephrine. Isometric exercise may improve endothelial function. The increased exposure of shear stress on the vessels throughout the entire body by the pressor response during IHG exercise may upregulate the production of nitric oxide synthase and increase the release of endothelium-derived nitric oxide (8, 19, 28). This is a potentially important effect, because essential hypertension is associated with an impairment of endothelium-derived vasodilation related to nitric oxide production (6, 21, 22).

An interesting aspect of this study was that reductions in arterial pressure after training were not associated with any observed sympathetic and cardiovascular adaptations to exercise of the trained limb. During IHG exercise and posthandgrip muscle ischemia, MSNA, arterial pressure, and heart rate responses were not changed after forearm training. These findings therefore indicate that resting and exercise adaptations to these variables do not necessarily parallel one another. Moreover, these findings are in

contrast to dynamic forearm training. Previous short-term training studies using dynamic forearm training have found attenuation in MSNA responses to both isometric (27) and dynamic handgrip (26) exercises. Sinoway et al. (26) found a corresponding attenuation in arterial pressure during dynamic handgrip exercise after training. Likewise, dynamic one-legged training has been shown to attenuate MSNA and arterial pressure responses to one-legged exercise (23). Therefore, when the results of the present study are viewed in relation to prior studies, it would appear that dynamic exercise training reduces arterial pressure responses to exercise; isometric exercise training, on the other hand, is an effective modality to reduce arterial pressure at rest but not during exercise.

It is possible that isometric training in hypertensive patients may be associated with greater reductions in arterial pressure and possible reductions in MSNA (18, 31). This may be more readily observed because of higher initial levels for both factors. Future studies with hypertensive patients will allow us to answer this important question. Moreover, studies examining different intensities, durations, and frequencies of isometric forearm training will help determine the most effective training paradigm to lower resting arterial pressure in both normotensive and hypertensive subjects. Finally, future studies that specifically examine the peripheral vasculature will help identify whether IHG training elicits vascular adaptations throughout the body. Of particular importance is possible improvement of endothelial vasodilator function.

In summary, IHG training is effective in lowering arterial pressure in normotensive subjects. Isometric training may be an effective nonpharmacological intervention in the prevention and treatment of hypertension. The reduction in arterial pressure is not related to changes in MSNA but is likely due to peripheral vascular adaptations. Notable aspects of isometric forearm exercise are that it can be performed quickly, easily, and in any location. These attributes may increase patient compliance to prescribed training interventions and thereby enhance the probability of positive clinical outcomes.

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