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RESEARCH ARTICLE

Acute cardiovascular responses to a single bout of high intensity inspiratory muscle strength training in healthy young adults

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Abstract

High intensity, low volume inspiratory muscle strength training (IMST) has favorable effects on casual systolic blood pressure and systemic vascular resistance. However, the acute effects of IMST on heart rate (HR), blood pressure (BP), and sympathetic regulation of vascular resistance and the trajectory of post exercise recovery are not known. We recruited 14 young adults (7 women/7 men, age: 22 ± 2 years) to perform a single bout of high intensity IMST (inspiratory resistance set at 75% of maximal inspiratory pressure) importantly, female and male subjects were matched in regard to the target inspiratory pressure and target inspiratory muscle work per breath. We recorded HR, beat-to-beat changes in BP and postganglionic, muscle sympathetic nerve activities (MSNA) continuously throughout baseline, a single bout of IMST (comprising five sets of 6 inspiratory efforts) and in recovery. We show that one bout of IMST does not effect a change in BP, however, it effects a significant increase in HR (68.4 ± 11.7 beats/min versus 85.4 ± 13.6 beats/min; $P < 0.001$) and a significant decline in MSNA (6.8 ± 1.1 bursts/15 s bin; $P < 0.001$ versus 3.6 ± 0.6 bursts/15 s bin) relative to baseline. Remarkably, among men MSNA rebounded to baseline levels within the first minute of recovery, however, in women, MSNA suppression persisted for 5 min. We show that in healthy young adults, high intensity, low volume respiratory training results in the acute suppression of MSNA. Importantly, MSNA suppression is of greater magnitude and longer duration in women than in men.

NEW & NOTEWORTHY Previous studies show 6 weeks of high intensity, low volume inspiratory muscle strength training (IMST) lowers blood pressure (BP) and systemic vascular resistance in young adults. However, the acute response to IMST is unknown. We characterized BP, heart rate, and sympathetic nervous activity (SNA) in healthy young adults at baseline, during IMST, and in recovery. There was no acute effect of IMST on BP, however, there was significant IMST-related suppression of SNA that was of greater magnitude in women than men.

exercise; sympathetic activation; respiratory training

INTRODUCTION

Despite abundant evidence that aerobic exercise lowers blood pressure and improves cardiovascular health, an estimated 60–80% of adults do not adhere to guidelines for minimum physical activity (150 min/week of moderate intensity aerobic exercise or 75 min/week of vigorous intensity aerobic exercise) (1, 2). The most often cited reason for not exercising is a "lack of time" (3).

Beginning in 2013, we modified a respiratory strength training protocol originally devised for use among ventilator-dependent patients (4, 5). In its original format, this respiratory strength training protocol known as inspiratory muscle strength training (or IMST) followed a traditional exercise format requiring 20–30 min training/day or ~150 min/week (6–11). In our hands, IMST comprises 30 (5 sets of 6 breaths) inspiratory efforts against a resistance (75% of the individual's maximum inspiratory pressure; PI_{max}). Training is performed 5 min each day, 5 days/week for a total weekly training time of ~30 min. In this high intensity, low volume format, IMST is considered somewhat akin to high intensity interval training or HIIT (3, 12, 13).

We have shown that young healthy adults who complete 6 weeks of this respiratory training regimen exhibit significant improvements in blood pressure, autonomic balance, and systemic vascular resistance (14, 15), with similar outcomes in select patient populations (16, 17).

Despite these encouraging outcomes and burgeoning interest in HIIT-type protocols (18, 19), the acute and post-exercise responses to IMST have yet to be characterized. Characterizing the cardiac and autonomic responses to an acute bout of IMST is a necessary first step toward understanding the intermediate and longer term (4–6 weeks) health-related physiologic adaptations. Accordingly, we obtained continuous recordings of heart rate, blood pressure, and sympathetic nervous system activity (via microneurography) in women and men at baseline, during high-intensity IMST and immediately post-training, testing the hypothesis that a single bout of high-intensity IMST will effect a short-term suppression of

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Table 1. *Group averages for anthropomorphic measures for female) and male subject participants*

Measure	Female		Male	
	Mean	SD	Mean	SD
Age, years	21.3	2.4	22.1	0.9
Height, cm	171.7*	6.3	183.1	10.4
Weight, kg	68.0	13.4	78.6	10.5
BMI	22.9	3.3	23.4	2.3
HR, beats/min	75.2	7.7	73.6	15.1
SBP, mmHg	114.0	9.1	114.0	4.7
DBP, mmHg	71.9	8.7	70.8	4.7
$FEV_{1.0}$, L	3.6	0.5	4.2	0.7

Values are means ± SD; n = 7 female and n = 7 male participants.*Significant difference ($P < 0.05$). BMI, body mass index; DBP, diastolic blood pressure; FEV, force expiratory volume; HR, heart rate, SBP, systolic blood pressure.

muscle sympathetic nerve activity (MSNA) in young healthy adults.

METHODS

Ethical Approval and Human Subjects

We recruited 14 healthy adults (7 women, 7 men; ages 18–30 years) from the student population at The University of Arizona (see Table 1). All were casual exercisers (i.e., ~30 min of exercise 3–5 days/week) (20), non-smokers, non-obese, normotensive, and free from overt cardiovascular disease. After obtaining written consent, all subjects underwent screening assessments of pulmonary function. All studies were performed in The University of Arizona's Human Neurophysiology Laboratory following 4 h of fasting and 12 h free from caffeine and exercise. Experimental procedures were approved by the University of Arizona Human Subjects Protection Program, and in accordance with the Declaration of Helsinki. Complete datasets were obtained for all subjects with one exception. In a male subject, MSNA data was not obtained during minutes 4 and 5 of recovery.

Maximum Inspiratory Pressure

While seated in a dental chair, subjects were coached to generate a maximum inspiratory pressure (PI_{max}) from residual lung volume via a bespoke resistance device comprising a mouthpiece attached to a non-rebreathing valve (2600 series; Hans Rudolph, Shawnee, KS) fitted with a flow limitation end cap on the inhalation port providing a constant, near-maximal resistance. Inspiratory pressure was detected via a tube attached to the non-rebreathing valve and coupled to a pressure transducer (Omegadyne Inc., Stamford, CT). PI_{max} was determined for each subject as the average of at least three maximum inspirations against resistance that were within 5% of each other. For all subjects, 75% PI_{max} defined the individual's target training pressure (see general procedures below).

Blood Pressure and Heart Rate

Beat-to-beat changes in blood pressure were monitored via an automated finger cuff pressure transducer (500 Hz sampling rate; ccNexfin; Bmeye, Amsterdam, The Netherlands) on the non-dominant hand. Continuous lead-II ECG (1000 Hz sampling rate; band pass filters 0.3–1.0 KHz) was sampled using surface electrodes (Kendall 133 foam electrodes; Covidien, Mansfield, MA) and recorded online (LabChart 8.0, ADInstruments. Colorado Springs, CO).

Muscle Sympathetic Nerve Activity

Concurrent with ECG recordings, sympathetic nerve traffic was recorded from the common peroneal nerve via tungsten microelectrode (200 μm: 25-40 mm, impedance: 5 MΩ) (FHC, Bowdoin, ME) inserted percutaneously immediately posterior to the fibular head. Subjects rested semi-upright with the right knee and foot supported by positioning pillows (VersaForm, Performance Health, Warrenville, IL). Microelectrode placement was confirmed via electrical stimulation (0.02 mA, 1 Hz) as described previously (21). A second microelectrode was inserted just below the skin surface ~1.0 cm from the first served as a reference electrode. Electrode position in muscle fascicles was confirmed by pulse synchronous bursts of activity, elicitation of afferent nerve activity by mild muscle stretch, and absence of response to light stroking of the skin or with startle response to loud noises (21). The recorded signal was amplified (gain 2×10^4), bandpass filtered (500-2.0 kHz) using a pre-amplifier (NeuroAmp Ex; ADInstruments, Colorado Springs, CO), and signals were full wave rectified (0.1 s moving window), sampled at 10 kHz. The resulting signal was monitored using a computer-based data acquisition and analysis system (LabChart 8.0 software, ADInstruments, Colorado Springs, CO) and loudspeaker throughout the experiment.

General Experimental Protocol

With the resistance device coupled to a pneumotachometer (PNT series 4183; Hans Rudolph) to record expiratory airflow, subjects were coached to inspire to their PI_{max} and to sustain inspiratory effort against the resistance for 1–2 s before exhaling to end-expiratory volume. Accordingly, the breathing rate for all subjects was set at 12 breath cycles per minute (14, 15). After a period of rest breathing, subjects completed a single bout of IMST comprising 5 sets of 6 inspiratory efforts with a 60–90 s rest between sets (see Fig. 1). Subjects were provided audio and visual cues to guide them in attaining correct breath timing and their inspiratory target pressure (75% of PI_{max}). Following the fifth and final set, subjects were allowed to recover (see recovery). End tidal CO_2 levels were monitored via a CO_2 analyzer (model 17515, Vacumetrics, Inc., Ventura, CA) and supplemental CO_2 was titrated as needed to prevent hypocapnia.

Figure 1. Schematic of experimental protocol. Baseline: subjects sat quietly for 5 min. IMST sets 1-5: subjects performed 5 sets of 6 breaths against an maximum inspiratory resistance (75% PI_{max}) interspersed with ~60 s rest. Recovery: subjects sat quietly for 5 min.

Table 2. *Birth control and menstrual phase status for female participants*

Subject	Birth Control	Menstrual Phase	MSNA Response (% Baseline)
1	No	Follicular	−67.2%
2	Yes	Follicular	−23.2%
3	Yes	Follicular	−46.5%
4	No	Follicular	−33.7%
5	No	Luteal	−52.0%
6	Yes	Luteal	−61.4%
7	No	Luteal	−94.9%

MSNA, muscle sympathetic nerve activities.

Data Analysis

Beat-to-beat measures of systolic blood pressure, diastolic blood pressure, mean arterial pressure, instantaneous heart rate, respiratory muscle work and muscle sympathetic nerve activity were averaged in each of the following experimental segments: baseline, IMST (sets 1–5), and recovery. For baseline, data were averaged over the entire 5-min segment. For IMST (sets 1–5), data were averaged separately for each set (corresponding to 6 breaths against the resistance) and for each of the intervening rest periods (corresponding to the 1 min of rest between each IMST set). In recovery, data were averaged each minute for 5 min. Data in IMST and recovery were expressed as a percentage of baseline (% baseline).

Respiratory work (mmHg/s) was calculated as the area beneath the inspiratory pressure waveform and respiratory volume (L), determined by calculating the area under the expiratory portion of the flow waveform.

Negative deflecting cardiac-related sympathetic bursts were identified using unprocessed and root mean squared MSNA signals. Bursts that exceeded a predetermined threshold were marked and counted. Baseline MSNA was averaged over the entire 5-min segment. In view of the abbreviated training window (<1.0 min), MSNA burst frequency (bursts/minute) was expressed as number of bursts per 15-s bin and weighted averages used to quantify MSNA when experimental segments exceeded 15 s (e.g., baseline, IMST, and recovery). Likewise, MSNA burst incidence (bursts/100 heart beats) was calculated over successive IMST bouts as follows:

$$\frac{\text{Total number of MSNA bursts}}{\text{Total training time (s)}} \times \frac{60 \text{ seconds}}{\text{Average HR during IMST}} \times 100$$

Estimates of MSNA burst frequency and incidence recorded during IMST (sets and intervening rests) and in each minute of recovery subsequently were expressed as a percentage of baseline.

Statistical Analyses

Differences in anthropomorphic data between the sexes were assessed using independent sample t tests with significance set at $P < 0.05$. A mixed model with fixed effects was used to assess the main effects of IMST on each parameter (heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and MSNA) in each experimental segment (IMST sets, intervening rests, and recovery) and in which subjects served a random factor. A 95% confidence interval (CI) was used to delineate significant differences between each experimental segment and baseline. Significant differences between IMST sets, intervening rests, and recovery were determined by pairwise comparisons. Post hoc mixed models with fixed effects were used to test the effects of IMST on select experimental time points (i.e., IMST set 5 and recovery minute 5) and the effect of sex. Significant differences in effects (time or sex) were set at $P < 0.05$ and significance between experimental time points (IMST set 5 and recovery minute 5) and baseline were determined using 95% CIs. Sex differences at specific time points in recovery (minutes 1–5) were evaluated via pairwise contrasts with significance corrected to account for multiple comparisons ($P < 0.01$). All data were normalized to baseline. Accordingly, experimental time points were significantly different from baseline if the 95% CI excluded zero.

RESULTS

Subject Characteristics

Female and male subject participants were matched for age (P = 0.386), weight (P = 0.125), body mass index (P = 0.751), resting heart rate (P = 0.987), casual systolic blood pressure (P = 0.946), and casual diastolic blood pressure (P = 0.792). Female subjects were shorter than their male counterparts (P = 0.028). Details of menstrual phase and birth control status are presented in Table 2. At the time of assessment, 3 women were in the luteal phase and 4 in the follicular phase. Three of seven women were taking prescription oral contraceptives (combination progestin/estradiol).

Respiratory Parameters

Maximal inspiratory pressures (F: −79.9 mmHg; M: −78.3; P = 0.879) and target inspiratory pressures (75% PI_{max}) (F: −58.6 mmHg; M: −58.7 mmHg; P = 0.991) were the same for women and men. Furthermore, both sexes performed the same inspiratory work per breath (F: 84.1 mmHg·s; M: 85.40 mmHg·s; P = 0.918) and generated comparable expiratory volumes (F: 0.649 L; M: 0.771 L; P = 0.189) (see Table 3) during IMST.

Table 3. *Group averages for PI_{max}, target training pressure (75% PI_{max}), inspiratory work per breath, and expiratory flow obtained from 30 IMST breaths for female and male subjects*

Measure	Female		Male	
	Mean	SD	Mean	SD
Max inspiratory pressure, PI_{max}, mmHg	−79.9	17.7	−78.3	21.7
Target pressure, 75% PI_{max}, mmHg	−58.6	13.0	−58.7	16.3
Inspiratory work, mmHg·s	84.1	22.2	85.0	25.9
Expiratory volume, L	0.649	0.1	0.771	0.2

Values are means ± SD; n = 7 female and n = 7 male participants. BMI, body mass index; DBP, diastolic blood pressure; HR, heart rate; PI_{max}, maximum inspiratory pressure; SBP, systolic blood pressure.

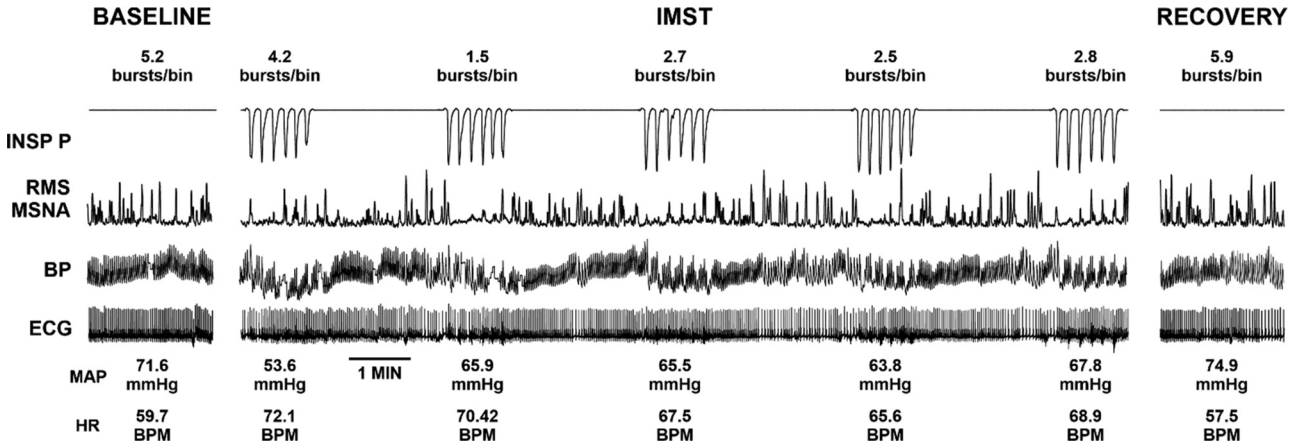

Figure 2. Representative recording of inspiratory pressure (INSP P), postganglionic, multiunit muscle sympathetic nerve activity (RMS MSNA), blood pressure (BP), and electrocardiogram (ECG) signals recorded from a subject at baseline, during IMST, and in recovery. Burst frequency counts (in bursts/15 s bin), mean arterial pressure (MAP), and heart rate (HR) are provided for baseline, each of the IMST sets and in recovery. BPM, beats/min; IMST, inspiratory muscle strength training; MSNA, muscle sympathetic nerve activities.

Cardiovascular Responses to IMST

A representative continuous recording obtained at baseline, and throughout IMST and recovery in one subject is presented in Fig. 2. As shown, in each of the 5 IMST sets, the subject generated large, negative intrathoracic pressures (approximately −66.5 mmHg) with resultant increases in heart rate and BP fluctuations. Note that the suppression of

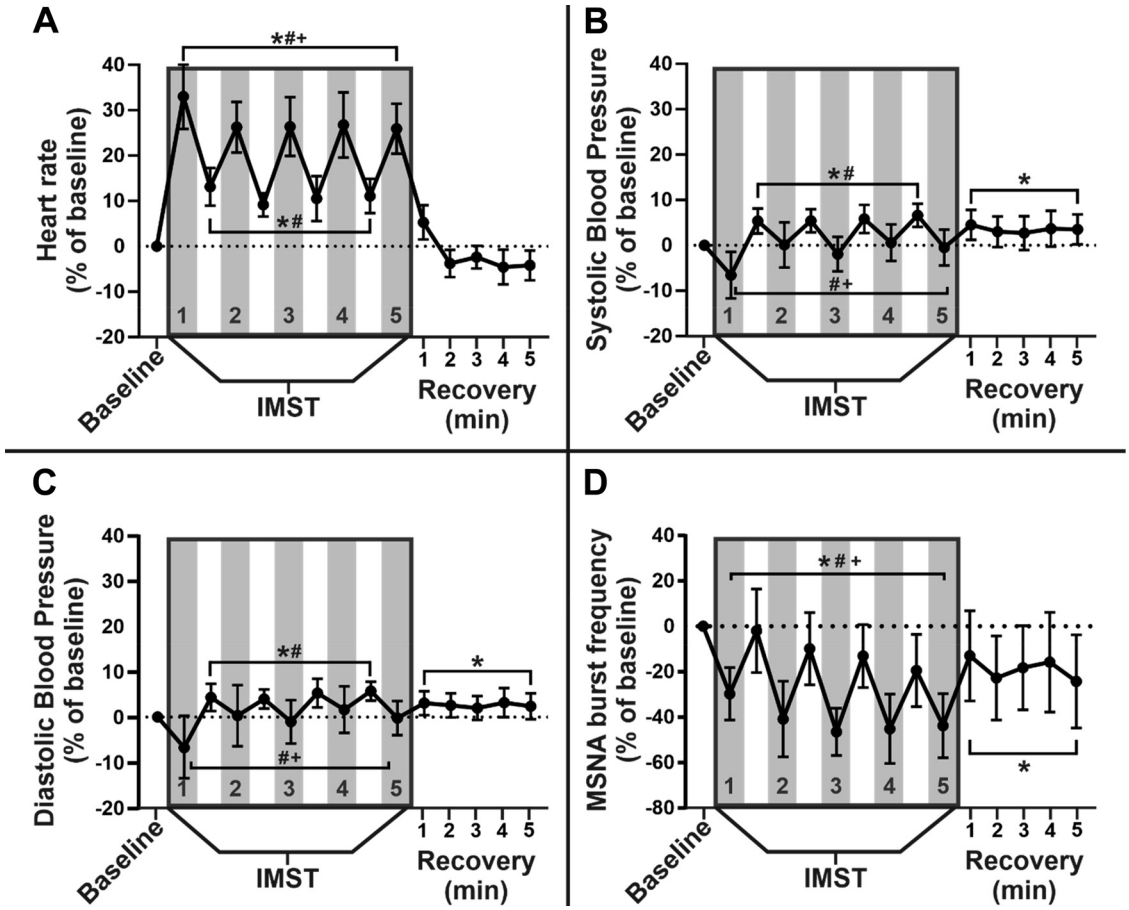

Figure 3. Group averages (±95% CI) for (*A*) heart rate; (*B*) systolic blood pressure and (*C*) diastolic blood pressure; and (*D*) MSNA burst frequency expressed as percentage of baseline (% baseline). For IMST, subjects performed five sets of six inspiratory efforts against a resistance (75% PI_{max}) (gray columns) interspersed with ~1 min of resistance-free, rest breathing (white columns). Following IMST, subjects sat quietly for 5 min of recovery. *Significant difference from baseline (95% CI excludes zero); #significant difference from recovery ($P < 0.05$); +Significant difference between IMST sets and intervening rests ($P < 0.05$). CI, confidence interval; IMST, inspiratory muscle strength training; PI_{max}, maximum inspiratory resistance.

sympathetic activity coincides with the transition into each IMST set. For this subject in recovery, MSNA, blood pressure, and heart rate all approximated baseline levels.

Figure 3 shows averaged outcomes (% baseline) for HR, SBP, DBP, and MSNA during each of the experimental segments (IMST sets, intervening rests, and recovery). During each IMST set, there were significant increases in heart rate (26.3 ± 1.48% [95% CI 23.2, 29.4]) and significant declines in MSNA (−41.2 ± 5.42% [95% CI -52.5, 29.8]). In the intervening rest periods, heart rate declined but remained elevated relative to baseline (11.3 ± 1.5% [95% CI 8.1, 14.5]). Subjects also exhibited modest increases in SBP (5.8 ± 1.3% [95% CI 3.1, 8.5]) and DBP (4.8 ± 1.2% [95% CI 2.3, 7.2]), whereas MSNA trended toward baseline values (−11.1 ± 5.6% [95% CI −22.7, 0.53]). In recovery, heart rate returned to baseline values [95% CI −4.6, 1.7], whereas SBP and DBP remained slightly elevated above baseline (SBP 95% CI 0.5, 5.9; DBP 95% CI 0.01, 4.8). Conversely, MSNA suppression persisted throughout recovery (−18.8 ± 5.4% [95% CI −30.8, −7.4]).

Figure 4 depicts results for HR, SBP, and DBP at the terminus of the IMST training bout. By IMST set 5, average heart rate was ~25% higher (25.2 ± 2.1% [95% CI 20.8, 29.7]) but quickly returned to baseline frequency by recovery minute 5 (−4.1 ± 2.2% [95% CI −8.7, 0.5]). In contrast, there was no consistent effect of IMST on blood pressure, and SBP and DBP were not different at training end (i.e., IMST set 5) and recovery (minute 5) (SBP: 3.5 ± 1.5%, P = 0.154; DBP: 2.4 ± 1.3%, P = 0.473).

Figure 5 shows results for MSNA burst frequency (bursts/15 s bin) and estimated burst incidence (bursts/100 heart beats) during IMST. Burst frequency was ~45% lower during IMST than at baseline (−43.8 ± 7.5% [95% CI −59.3, −28.2]) and on average, suppression persisted throughout recovery (−23.3 ± 7.8% [95% CI −39.5, −7.2]). Burst incidence followed the same trend, with burst incidence rates ~60% lower during IMST (−60.2 ± 15.9% [95% CI −73.4, −47.0]) and continued suppression throughout recovery (−17.1 ± 29.8% [95% CI −30.3, −3.9]).

A more fine-grained analysis of MSNA as a function of sex revealed comparable burst frequencies for women and men at baseline (F: 14.1 ± 2.3 versus M: 18.7 ± 2.7 bursts/min) that progressively declined during each IMST set, attaining a nadir in set 5 (F: −54.1 ± 8.9%; M: −33.3 ± 8.4%, P = 0.328). Importantly, trajectories in recovery differed between women versus men (Fig. 6). In men, MSNA burst frequency rebounded to baseline by recovery minute 1, whereas among women suppression was of greater magnitude (minutes 1–3; $P < 0.009$) and persisted throughout recovery. It should be noted that at the same time, heart rates were lower in women than in men (P = 0.0298), however, SBP and DBP were not different between the two groups (P = 0.269 and P = 0.393). Despite lower heart rates, MSNA burst incidence was lower among women than men (F: −35.5 ± 7.8%; M: 1.3 ± 10.2%, P = 0.0148) and also slower to return to baseline.

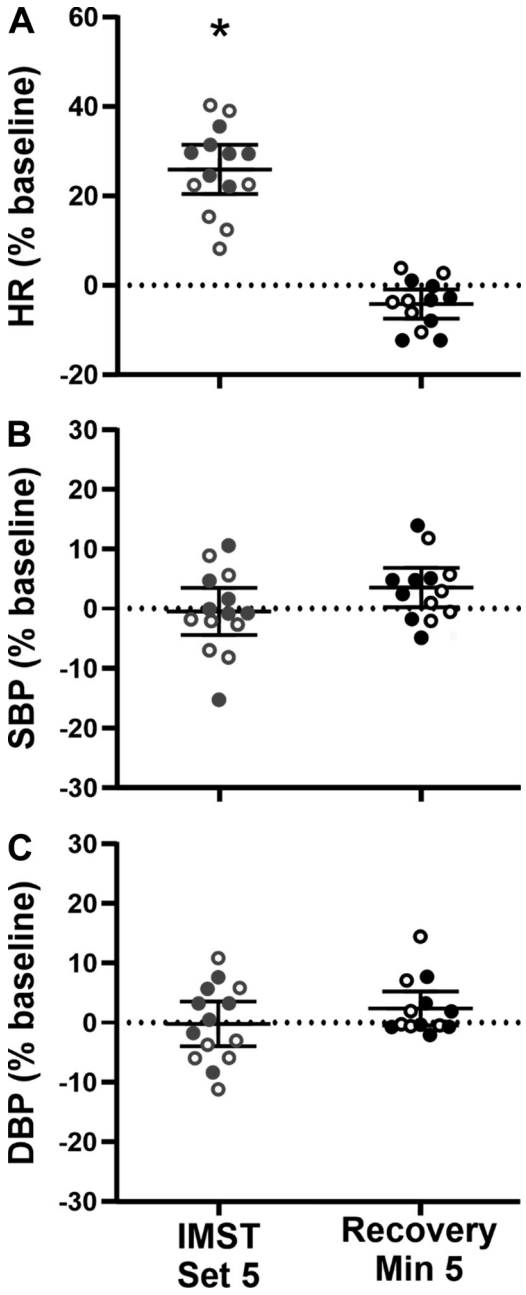

Figure 4. Group averages (±95% CI) for (*A*) heart rate (HR); (*B*) systolic blood pressure (SBP); and (*C*) diastolic blood pressure (DBP) (% baseline) during IMST Set 5 (gray symbols) and recovery minute 5 (black symbols) for female (solid symbols) and male (open symbols) subjects. *Significantly different from baseline (95% CI excludes zero). IMST, inspiratory muscle strength training.

DISCUSSION

Our objective in the current study was to characterize the acute effects of a single bout of high intensity (75% of PI_{max}) low volume (30 breaths) IMST on key cardiovascular parameters in young healthy adults. The primary novel finding(s) of the study are that IMST results in acute increases in heart rate (~25%) and the simultaneous acute suppression of MSNA (~40%) that reaches a nadir in the fifth and final training set. Second, in recovery, IMST-related suppression of MSNA is of greater magnitude and longer duration in women. Among men, sympathetic neural activity quickly rebounds and returns to baseline within the first minute of recovery.

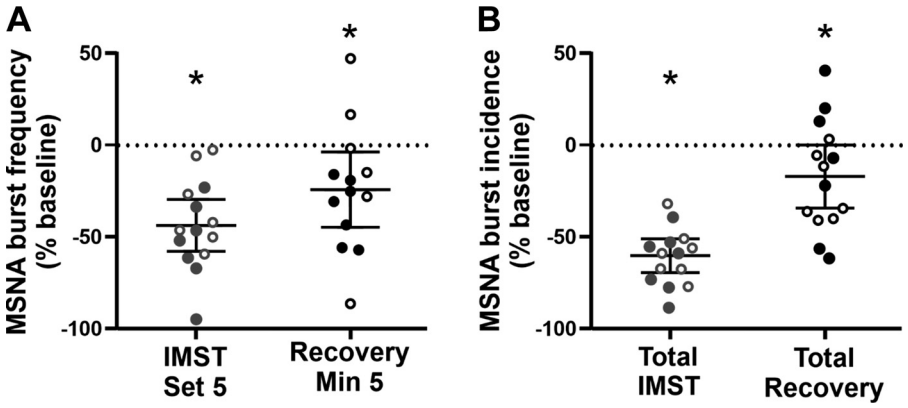

Figure 5. *A*: group averages (±95% CI) for MSNA burst frequency (% baseline) at IMST set 5 (gray symbols) and at recovery minute 5 (black symbols) for female (solid symbols) and male (open symbols) subjects. *B*: calculated MSNA burst incidence (% baseline) over the 5 discontinuous IMST sets and over the entire 5-min recovery period for female (solid symbols) and male (open symbols) subjects. *Significantly different from baseline (95% CI excludes zero). IMST, inspiratory muscle strength training; MSNA, muscle sympathetic nerve activities.

IMST and the Regulation of Heart Rate

In response to IMST, we report a transient increase in average HR (68.4 ± 11.7 beats/min versus 85.4 ± 13.6 beats/min) that returns to baseline values after cessation of training. The present findings are similar to those reported in other nonfatiguing inspiratory resistive breathing protocols, and show both low (≤10% PI_{max}) and high (≥70% PI_{max}) workloads elicit comparable increases in heart rate (22–24).

The magnitude of HR change during IMST was not significantly different between female and male subjects (F: 28.9 ± 4.4% versus M: 22.9 ± 12.6%; P = 0.257, main effect for sex). To our knowledge, little has been reported on sex-specific differences in HR responses to *non-fatiguing* respiratory muscle work. However, our findings are in line with evidence from aerobic exercise, where high intensity interval/circuit training (25) and incremental exercise protocols (26, 27) elicit similar HR increases between women and men, especially when workload is matched at the same percentage of subject VO_{2max}.

IMST and the Regulation of Blood Pressure

An extensive literature documents the acute effects of resistive breathing on blood pressure and reveals effects that are distinct for low versus high resistance breathing. Inspiratory efforts against low resistance (≤10% PI_{max}) have been used to clinical advantage to effect acute increases in stroke volume, cardiac output, and SBP (22–24), whereas moderate (i.e., 60% PI_{max}) or higher (i.e., 90% PI_{max}) resistances yield different outcomes (28, 29).

Importantly, the current study comprising equal numbers of women and men matched in regard to target inspiratory pressure and inspiratory muscle work provides valuable new insight into the effects of the stimulus (IMST) on regulation of blood pressure. Specifically, where previous studies have documented sex differences in arterial baroreflex function during dynamic exercise (30, 31), we report BP responses to each training set that are similar for both groups (SBP, p = 0.803; DBP, P = 0.283). In view of the similarity of the BP response to IMST and the transient (~30 s) nature of the respiratory stimulus (32), we think it unlikely that IMST-related suppression of MSNA has its origins in the arterial baroreflex regulation of sympathetic nervous system activity.

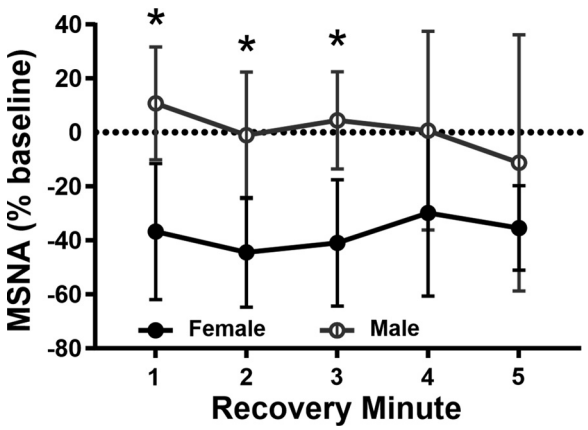

Figure 6. Group averages (±95%CI) for MSNA burst frequencies (% baseline) for men (open symbols) and women (filled symbols) in recovery minutes 1-5. In men, MSNA rebounded to baseline by minute 1. *Significantly different from females (P < 0.05). MSNA, muscle sympathetic nerve activities.

IMST and Suppression of Muscle Sympathetic Nerve Activity

Others previously have documented within-breath inspiratory-related inhibition of sympathetic outflow in the context of loaded and unloaded respiratory tasks (29, 33). Importantly, a key distinction between this earlier work and the current study lies in the magnitude of the inspiratory pressure generated. Specifically, the inspiratory pressures reported by St Croix et al. (29, 33), encompass −1.0 and −13.0 mmHg as compared to −60 and −100 mmHg generated by the subjects in the current study. Thus, it is the much larger (~4-fold greater) inspiratory pressures generated in the context of high intensity low volume IMST that distinguishes the current protocol from previous studies of this type and which is the unique stimulus that modulates MSNA.

In recent studies, we attributed IMST-related reductions in BP and systemic vascular resistance to cardiopulmonary receptor inhibition of (central) sympathetic outflow secondary to repeated large (negative) intrathoracic pressures driving respiratory pump-mediated venous return (14, 15). The current finding of MSNA suppression coincident with the initiation of the inspiratory efforts and therefore, in time with the respiratory pump-induced

augmentation of venous return, provides further strong support for this hypothesis.

IMST and Vascular Endothelial Function

Inspiratory efforts against a significant resistance also contribute to transient increases in cardiac output (20–40%) (34) presumably with downstream effects on regional and/or systemic blood flow and increases in shear stress sensed by endothelial cells. Although we did not attempt an assessment of flow-mediated arterial dilatation (FMD) in the acute context, de novo findings obtained in older men and women (70 years) following 6 weeks high intensity, low volume IMST show significant improvements in FMD and NO production with reductions in superoxide production (35, 36). In light of the current findings, an assessment of vascular endothelial function in young women and young men during IMST is warranted.

Experimental Considerations: Strengths and Limitations

Female and male participants were matched for inspiratory work. To our knowledge only one other study has attempted a similar level of matching (37). Thus, the majority have evaluated sex differences in the cardiovascular response to inspiratory resistance/work in women and men matched for age and force expiratory volume (FEV) 1.0 (%) (38–40). In matching across sex for absolute inspiratory muscle work and anthropomorphic data (save height), the current study provides a unique opportunity to assess the effects of IMST on cardiovascular parameters independent of differences in size/muscle mass.

Available published data for the effects of menstrual phase (41–43), birth control (44), and stressors (45) on sympathetic nervous activity in young women remain equivocal. Although the current findings indicate a comparable blunting of MSNA for subjects in the follicular versus luteal phases (Table 2), the data set is too small to draw a definitive conclusion. Additional studies that entail a larger female cohort and that control for menstrual phase and/or hormonal birth control are indicated.

Conclusions

Traditional aerobic exercise has well-documented and favorable effects on blood pressure and cardiovascular health and yet 60–70% of adults fail to meet the minimum weekly requirement for physical activity. We show here that a novel training protocol, referred to as IMST, results in acute increases in heart rate and acute suppression of sympathetic nervous outflow. Among young women and men, IMST-related reductions in sympathetic nervous system activation are comparable in magnitude, however, suppression is of greater magnitude and longer lasting in women than in men. Our results provide new insight into the acute effects of IMST, which when repeated daily over the intermediate and longer term can modify cardiovascular health via reductions in BP, systemic vascular resistance, and sympathetic nervous outflow.

ACKNOWLEDGMENTS

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

C.M.D. and E.F.B. conceived and designed research; C.M.D., D.R.D., S.M.S., and E.F.B., performed experiments; C.M.D., D.R.D., S.M.S., and E.F.B., analyzed data; C.M.D., D.R.D., S.M.S., and E.F.B., interpreted results of experiments; C.M.D. and E.F.B., prepared figures; C.M.D. and E.F.B., drafted manuscript; C.M.D. and E.F.B., edited and revised manuscript; C.M.D., D.R.D., S.M.S., and E.F.B., approved final version of manuscript.

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