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Original Research

Hemodynamic and Autonomic Modulation in Response to Additive Sympathetic Stressors in Young, Healthy Individuals

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Abstract

International Journal of Exercise Science 18(6): 1047-1060, 2025. https://doi.org/10.70252/WWDK4926 Isometric handgrip (IHG) coupled with post-exercise muscular ischemia (PEMI) and the cold pressor test (CPT) have been demonstrated to increase measures of hemodynamics and to reduce vagal tone. However, little is known about how acute resistance exercise (RE) alters these responses. The purpose was to evaluate an acute bout of RE in conjunction with a single- or dual-stressor task on hemodynamics and autonomic modulation in resistance-trained individuals. Ten resistance-trained individuals (Mean ± SD; Age: 23 ± 3 years) completed a single- (SS: IHG + PEMI only) or dual-stressor (DS: IHG + PEMI + CPT) task condition. Before and after the acute RE variables were monitored during five minutes of rest (REST), two minutes of IHG, three minutes of PEMI with or without concurrent CPT (STRESS). Hemodynamics included heart rate (HR), mean arterial pressure (MAP), cardiac output (Q,). stroke volume (SV) and total peripheral resistance (TPR), while autonomic data were measured via heart rate variability and heart rate complexity. There were no significant (p > 0.05) three-way interactions for HR, Q, or TPR. However, there was a significant three-way interaction (p = 0.007) for SV such that SV was significantly increased during STRESS compared to REST in the SS condition but did not change in the DS condition. There were no significant (p > 0.05) interactions for measures of autonomic modulation. These data suggest that young, resistance-trained individuals have a significant cardiac sympathetic reserve and thus a large capacity to handle multiple stressors following acute RE.

Keywords: Vagal, heart rate variability, cold pressor test, isometric handgrip

Introduction

Isometric hand grip (IHG), post-exercise muscular ischemia (PEMI), and the cold pressor test (CPT) acutely alter hemodynamics and autonomic modulation.[1-3] Accordingly, these tests have been utilized as physiological stressors to evaluate hemodynamic responses and alterations in

autonomic modulation in healthy individuals[4-8] as well as clinical populations.[9,10] Short duration (2-3min) IHG and CPT have been demonstrated to augment hemodynamics such as heart rate (HR), mean arterial pressure (MAP), cardiac output (Q), stroke volume (SV) and total peripheral resistance (TPR), while also transiently inducing vagal withdrawal.[1,3]

Acute resistance exercise (RE) has also been demonstrated to acutely alter hemodynamics and autonomic modulation in resistance-trained men and women.[11] Indeed, multiple investigations have demonstrated that acute RE can induce a transient period of vagal withdrawal that may persist between 30- and 75-minutes following the acute RE.[12-14] Therefore, additive sympathetic stimuli such as IHG, PEMI, CPT, and acute RE may all augment hemodynamics such as MAP, Q, SV, and TPR and can also induce short-term vagal withdrawal as well. Work by Kalfon et al[6] reported that PEMI + CPT resulted in a significantly increased HR, as well MAP, when compared to PEMI alone in healthy young, sedentary overweight or obese men. However, knowledge is limited regarding the effect of additive sympathetic stimuli in conjunction with acute RE on hemodynamics or autonomic modulation in young, healthy individuals that are lacking co-morbidities that further alter hemodynamics and autonomic modulation.

One study has utilized additive sympathetic stimuli in young, healthy, individuals.[5] Fu et al[5] evaluated responses in hemodynamics, primarily HR, MAP, SV, and TPR in 12 young, healthy individuals to head-up tilt alone or in conjunction with a CPT. Heart rate, MAP, SV, and TPR were all significantly augmented with head-up tilt and were further augmented with the addition of the CPT indicating the ability to further vasoconstrict under additional autonomic stress in young, healthy individuals.[5] Thus, further work on additive sympathetic stimuli is necessary to elucidate these responses in young, healthy individuals at rest and following acute RE.

As vagal modulation is attenuated following an acute RE, it can be postulated that individuals may have reduced capacity to maintain autonomic modulation in the face of additive sympathetic stimuli during this transient period of vagal withdrawal. It is key to understand the effects of additive sympathetic stimuli in conjunction with acute RE on hemodynamics and autonomic modulation as a well-established relationship exists between periods of vagal withdrawal and increased risk for sudden cardiac death.[15,16] While reduced autonomic modulation in response to additive sympathetic stimuli may not manifest itself as sudden cardiac death in young, healthy individuals, exploratory data in this population are necessary prior to investigating responses to high levels of physiological stress in clinical populations or older adults. Therefore, the purpose of the present study was to examine changes in hemodynamics and autonomic modulation during a single (SS: IHG + PEMI) or dual (DS: IHG + PEMI and the CPT) stressor task conditions both prior to and following acute upper-body RE in healthy, resistance-trained men and women. We hypothesized that significant alterations in hemodynamics (primarily increases in HR, MAP, Q, SV, and TPR) and reductions in vagal modulation at REST would occur following the acute RE for both conditions. Additionally, we hypothesized that individuals would display significantly attenuated hemodynamic and autonomic modulation responses to the PEMI + CPT after the acute RE when compared to before the acute RE.

Methods

Participants

Ten resistance-trained men (*n*=8) and women (*n*=2) between the ages of 18-30, free of cardiovascular, metabolic, or renal disease, and hypertension, volunteered to participate in the present investigation. Statistical power was calculated in G*Power version 3.1.9.7 (Dusseldorf, Netherlands) post hoc using the smallest observed main effect of time effect sizes (η_p^2 = 0.416 and η_p^2 = 0.392) for the dependent variables HR and the natural logarithm (ln) of RMSSD, respectively. A sample size of four was determined to be sufficient to observe responses in hemodynamics and autonomic modulation based on a power of 80% and a Cohen's d of 1.1. Descriptive characteristics of participants are presented in Table 1. Participants were required to have been resistance training for a minimum of six months consecutively. Exclusion criteria entailed any physical limitations or orthopedic injury that could prevent safe completion of the acute RE, current hypertension (systolic BP ≥ 130 or a diastolic BP ≥ 80 mmHg), or ingestion of any medication or supplements that could alter cardiovascular responses. Women were tested during the follicular phase (day 1-9) of their menstrual cycle. Participants provided their written informed consent after being briefed on the study procedures. This study was approved by the Kent State University Institutional Review Board prior to data collection and followed the Declaration of Helsinki.

Protocol

Experimental Overview.

To investigate the effect of the SS and DS conditions on hemodynamics and autonomic modulation in resistance-trained adults, a randomized, within subjects' design was implemented. Participants reported to the Cardiovascular Dynamics Laboratory for a total of four visits. During visit one, participants were informed of all experimental procedures, provided their written informed consent, and verified their eligibility to participate by completing the Physical Activity Readiness Questionnaire (PAR-Q), and a brief medical history form and were then assessed for anthropometrics. During visit two, participants were assessed for their one repetition maximum (1RM) on the bench press and biceps curl exercises. Finally, visits three and four were identical experimental trials where participants completed the SS or DS conditions in a randomized order. All participants were fitted with a three-lead electrocardiogram (ECG) and a finger photoplethysmography cuff which sampled continuously during data collection. During experimental trials, participants rested in a seated position for 10 minutes prior to competing five minutes of resting data collection, two minutes of isometric handgrip at 30% of their maximal voluntary contraction (MVC), and three minutes of PEMI or PEMI and concomitant CPT prior to and immediately following an acute RE. Visits three and four were performed at the same time of day (± 1 hr) and were separated by at least seven days and no more than 14 days.

Anthropometrics.

During their first visit to the laboratory, participants' height and weight were measured with a Healthometer 500KL specialty scale (McCook, IL, USA). Additionally, participant body composition including percent body fat, lean body mass, and fat mass was assessed through standing bioelectrical impedance analysis (InBody 570, InBody Co., Seoul, South Korea).

One-repetition maximum testing.

After assessment of anthropometrics, 1RM on the barbell bench press and biceps curl exercises was verified for each participant according to guidelines by the National Strength and Conditioning Association.[17] The 1RM process began with a warm-up of five minutes of pedaling on a cycle ergometer (Schwinn Airdyne, Vancouver, WA) at a self-selected pace, followed by sets of five to ten repetitions, three to five repetitions, and one to three repetitions interspersed by two minutes of rest on both the bench press and biceps curl. Next, participants were allotted a maximum of five attempts to establish their 1RM, with each attempt followed by three to five minutes of rest. Intensities increased by five to 10 percent following each successful attempt. One repetition-maximum was defined as the maximal load that participants could complete a repetition of both exercises while maintaining appropriate exercise technique.

Experimental Protocol

During each experimental visit, participants reported to the laboratory having abstained from caffeine, alcohol, and strenuous exercise for 24 hours, and having fasted for a minimum of three hours. After arriving at the laboratory, participants were fitted with a three-lead ECG and a finger photoplethysmography cuff (Finapres Medical Systems, Enschede, Netherlands) that was placed on the middle finger of the left hand. Additionally, an automated brachial blood pressure (BP) cuff (SphygmoCor, AtCor Medical, Naperville, IL) and an additional rapidly inflatable cuff (Hokanson E20, Hokanson inc., Bellevue, WA) were placed on the right arm of the participant for the PEMI. Participants then were assessed for their maximal voluntary contraction (MVC) on the IHG with three attempts. The maximal voluntary contraction was recorded as the average of all three attempts. Next, participants rested in a seated position for ten minutes. Following this rest period, participants' brachial BP (SphygmoCor, AtCor Medical, Naperville, IL) was assessed in duplicate with no more than five mmHg separating the repeated systolic and diastolic pressures and one minute separating measurements. A third BP reading was assessed only if deemed necessary.

After assessment of BP, participants completed either the SS or DS task condition (Figure 1). Both conditions consisted of identical experimental procedures regarding cardiovascular assessment and exercise protocols with the exception of the stress administered (Figure 1). Continuous ECG and finger photoplethysmography sampling began with five minutes of seated rest where participants performed paced breathing at 12 breaths per minute. Next, participants performed two minutes of IHG at 30% of MVC. Finally, following IHG, participants were exposed to three minutes of STRESS which varied based on condition. During the SS task condition, participants were exposed only to PEMI, with the cuff rapidly inflated to 220 mmHg, one second prior to the termination of IHG for a duration of three minutes. During the DS task

condition, participants were exposed to PEMI with a concomitant CPT, specifically three minutes of cold-water foot immersion in water between one and three degrees Celsius.

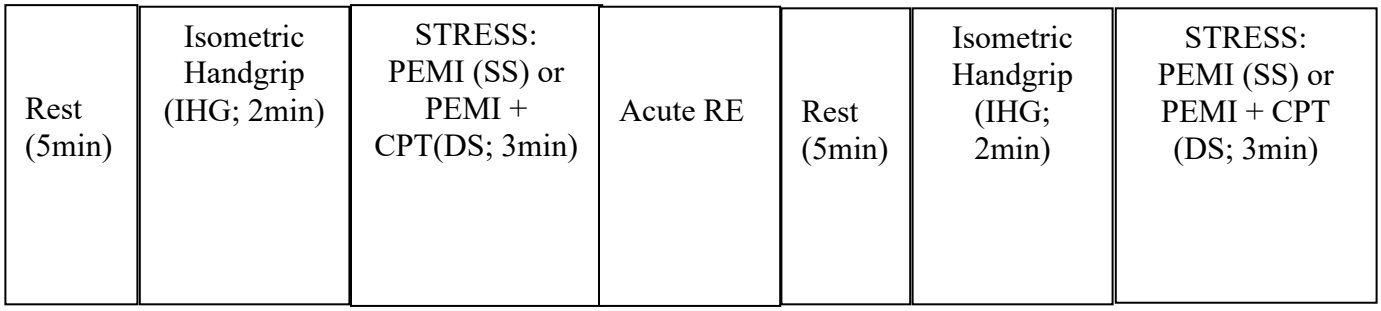

Figure 1. Timeline of acute cardiovascular assessment (CPT, cold pressor test; DS, dual stressor; PEMI, post-exercise muscle ischemia; SS, single stressor).

With the termination of this protocol, participants then performed acute upper-body RE. Prior to the acute RE all participants completed a five-minute warm-up on a cycle ergometer. Following the warm-up the acute RE consisted of four sets of five repetitions at 80% 1RM on the bench press and four sets of 10 repetitions at 70% 1RM on the biceps curl based on previously published standards.[18] This protocol has been demonstrated to significantly alter hemodynamics. Two minutes of rest was given between sets and exercises. With the completion of the acute RE, participants immediately repeated the same experimental protocol as before the acute RE.

Hemodynamic and HRV Data Analysis.

Hemodynamic measures included HR, MAP, Q, SV, and TPR. These measures were derived from beat-to-beat blood pressure recordings from finger photoplethysmography cuff at a sampling frequency of 500Hz using the Modelflow technique. This technique has been shown to be reliable and valid.[19] Heart rate variability (HRV) data were derived from 3-lead ECG data that was acquired using PowerLab data acquisition tools and LabChart software at a sampling rate of 1000Hz (AD Instruments, Colorado Springs, CO, USA). Time and frequency domain metrics of HRV were calculated using WinCPRS (Absolute Aliens Oy, Turku, Finland). Prior to being imported into WinCPRS, all ECG data were evaluated for artifact or ectopic beats. Time and frequency domain measurements of HRV were based upon distances in time between successive R-R intervals over the duration of each individual time point according to guidelines set by the Task force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology.[20] The root mean square of successive differences (RMSSD) in R-R intervals was used to represent vagal modulation.[20] After applying a fast Fourier transform to the array of successive R-R interval differences, the signal was transposed to the frequency domain.[20] The high-frequency (HF) component (0.15-0.4 Hz) represented vagal modulation, while the low-frequency (LF) component (0.04-0.15 Hz) indicated both vagal and sympathetic activity.[20] The total power (TP is noted as the sum of HF and LF components) was used as a metric of overall autonomic activity.[20] The ratio between LF and HF has been demonstrated to

reflect sympathovagal dominance.[20] Based on data collected in five individuals at rest not affiliated with this study demonstrated an ICC of HRV of 0.86 for ln HF, and 0.83 for the LF:HF ratio.

Additionally, heart rate complexity (HRC) was assessed using methods first employed by Richman and Moorman.[21] Sample entropy (SampEn) is the primary metric for HRC and provides information about the predictability of the successive differences in R-R intervals. Sample entropy typically ranges from 0 to 2. Values closer to 2 indicate a less predictable signal, and in turn, greater vagal modulation, while values closer to 0 are indicative of a much more predictable signal and less vagal modulation.[21]

Statistical Analysis

A Shapiro-Wilk test was used to assess data normality and the ln of data that were not normally distributed was calculated accordingly. If the data were not normally distributed, then they were converted to ln. A Two (condition: SS and DS) x Two (acute RE: Before and After acute RE) x Three (time: REST, IHG, and STRESS) three-way analysis of variance (ANOVA) was implemented to assess the effect of condition (SS, DS) across the repeated measures of condition, acute RE and time for measures of hemodynamics (HR, MAP, Q, SV, TPR) and HRV measured via ln RMSSD, ln LF, ln HF, ln TP, ln LF/HF, SampEn. Paired samples t-tests with a Benjamini-Hochberg correction factor were utilized to evaluate specific differences within conditions and across the acute RE and time points if the ANOVA was significant. Effect size for each ANOVA was presented as partial eta squared (η_p^2) and categorized as small (0.20-0.49), moderate (0.50-0.79), or large (≥ 0.80).[22] All data analyses were completed using IBM SPSS Version 27 (Armonk, NY, USA). Data are presented as mean ± standard deviation except Figures 2 and 3 where the data are presented as mean ± standard error of the mean.

Results

Participant characteristics are presented in Table 1.

Table 1. Participant descriptive statistics.

Participant Characteristics (N = 10; Men: n=8; Women, n=2))	
Age (yr)	23 ± 3
Height (m)	1.7 ± 0.8
Weight (kg)	79.4 ± 12.6
Body Fat (%)	21.6 ± 7.6
Bench Press 1RM (kg)	82.4 ± 24.7
Biceps Curl 1RM (kg)	41.1 ± 9.9

Data are presented mean ± standard deviation. 1RM: one-repetition maximum.

Hemodynamics

Measures of hemodynamics are presented in Table 2 as well as Figures 2 and 3**.** Mean, and individual responses, for HR (Figure 2) and MAP (Figure 3). There were no significant three-way interactions for HR, Q, or TPR. There was a significant three-way interaction ($F_{[2,18]}$ = 6.629,

p = 0.007, η_p^2 = 0.424) for SV such that SV was significantly increased during STRESS compared to REST in the SS condition but did not change in the DS condition. A condition x time interaction ($F_{[2,18]}$ = 6.223, p = 0.009, η_p^2 = 0.409) was observed for HR such that HR was augmented during REST and IHG compared to STRESS in the SS condition, and augmented from REST to IHG in the DS condition and elevated above all time points After RE compared to Before RE. A condition x time interaction ($F_{[2,18]}$ = 36.286, p < 0.001, η_p^2 = 0.801) was observed for MAP such that MAP was significantly augmented during STRESS in the SS condition compared to the DS condition. Moreover, MAP was augmented during both IHG and STRESS compared to REST in both the SS and DS conditions, with MAP being further increased from IHG to STRESS in the DS condition. A condition x time interaction ($F_{[2,18]}$ = 9.077, p = 0.002, η_p^2 = 0.502) and a main effect of acute RE ($F_{[1,9]}$ = 28.592, p < 0.001, η_p^2 = 0.761) were noted for Q such that Q was attenuated during STRESS compared to IHG in the SS condition but augmented during STRESS compared to REST and IHG in the DS condition. Furthermore, Q was elevated at all time points After RE compared to Before RE regardless of condition. Main effects of acute RE ($F_{[1,9]}$ = 11.225, p = 0.009, η_p^2 = 0.555) and time ($F_{[2,18]}$ = 11.601, p < 0.001, η_p^2 = 0.563) were present for TPR such that TPR was augmented at REST, IHG, and STRESS Before RE compared to REST, IHG, and Stress After RE. TPR was also significantly increased from REST to IHG, REST to STRESS, and IHG to STRESS both Before and After RE.

Table 2. Mean responses in hemodynamics to the single- and dual-stressor task conditions both before and after resistance exercise (N = 10).

	BEFORE RE			AFTER RE		
	REST	IHG	STRESS	REST	IHG	STRESS
Q (L/min)						
SS	5.4 ± 0.3	5.8 ± 0.30	5.5 ± 0.3	6.2 ± 0.3 ¥	6.8 ± 0.4 ¥	6.1 ± 0.04 ¥#
DS	5.6 ± 0.3	5.8 ± 0.04	6.0 ±0.04 *#	6.2 ± 0.03 ¥	7.0 ± 0.5 *¥	7.0 ± 0.5 *¥
SV (ml/beat)						
SS	72.8 ± 3.1	74.1 ± 3.9	79.4 ± 3.2 *	70.3 ± 2.3	73.4 ± 3.3	73.7 ± 2.9 *
DS	75.2 ± 4.0	76.0 ± 5.0	77.3 ± 5.0	70.7 ± 3.5	75.4 ±4.9	78.0 ± 5.0
TPR (mmHg/L/min)						
SS	0.91 ± 0.05	1.00 ± 0.06 *	1.02 ± 0.06	0.84 ± 0.03 ¥	0.91 ± 0.05 ¥	0.97 ± 0.05 ¥#
DS	0.92 ± 0.06	1.04 ± 0.07 *	1.20 ± 0.13 *#	0.82 ± 0.05 ¥	0.87 ± 0.08 *¥	0.97 ± 0.10 *¥#

DS, Dual-Stressor Task Condition; IHG, Isometric Handgrip; Q, Cardiac Output; SS, Single-Stressor Task Condition; TPR, Total Peripheral Resistance; SV, Stroke Volume. Data are presented mean ± standard deviation. * p ≤ 0.05, significantly different from REST, # p ≤ 0.05, significantly different from IHG, ¥ p ≤ 0.05 significantly different from BEFORE RE.

Autonomic Modulation

Autonomic modulation measures are presented in Table 3. There were no significant three-way interactions for measures of autonomic modulation. Main effects of time ($F_{[2,18]}$ = 5.806, p = 0.011,

η_p^2 = 0.392) and RE Time ($F_{[1,9]}$ = 18.513, p = 0.002, η_p^2 = 0.673) were noted for ln RMSSD such that ln RMSSD was attenuated during IHG compared to REST regardless of condition, and during all After RE time points compared to Before RE. A main effect of time ($F_{[2,18]}$ = 6.736, p = 0.007, η_p^2 = 0.428) was observed for ln LF as ln LF was augmented during STRESS compared to IHG across both conditions and was elevated above REST both Before and After RE. Main effects of acute RE ($F_{[1,9]}$ = 18.173, p = 0.002, η_p^2 = 0.669) and time ($F_{[2,18]}$ = 8.737, p = 0.002, η_p^2 = 0.493) were exhibited for ln HF such that ln HF reduced during REST and STRESS compared to IHG both After RE compared to Before RE. In additional all times After RE were elevated compared to Before RE. Furthermore, main effects of acute RE ($F_{[1,9]}$ = 13.724, p = 0.005, η_p^2 = 0.604) and time ($F_{[2,18]}$ = 8.603, p = 0.002, η_p^2 = 0.489) and were noted for ln TP such that ln TP was attenuated

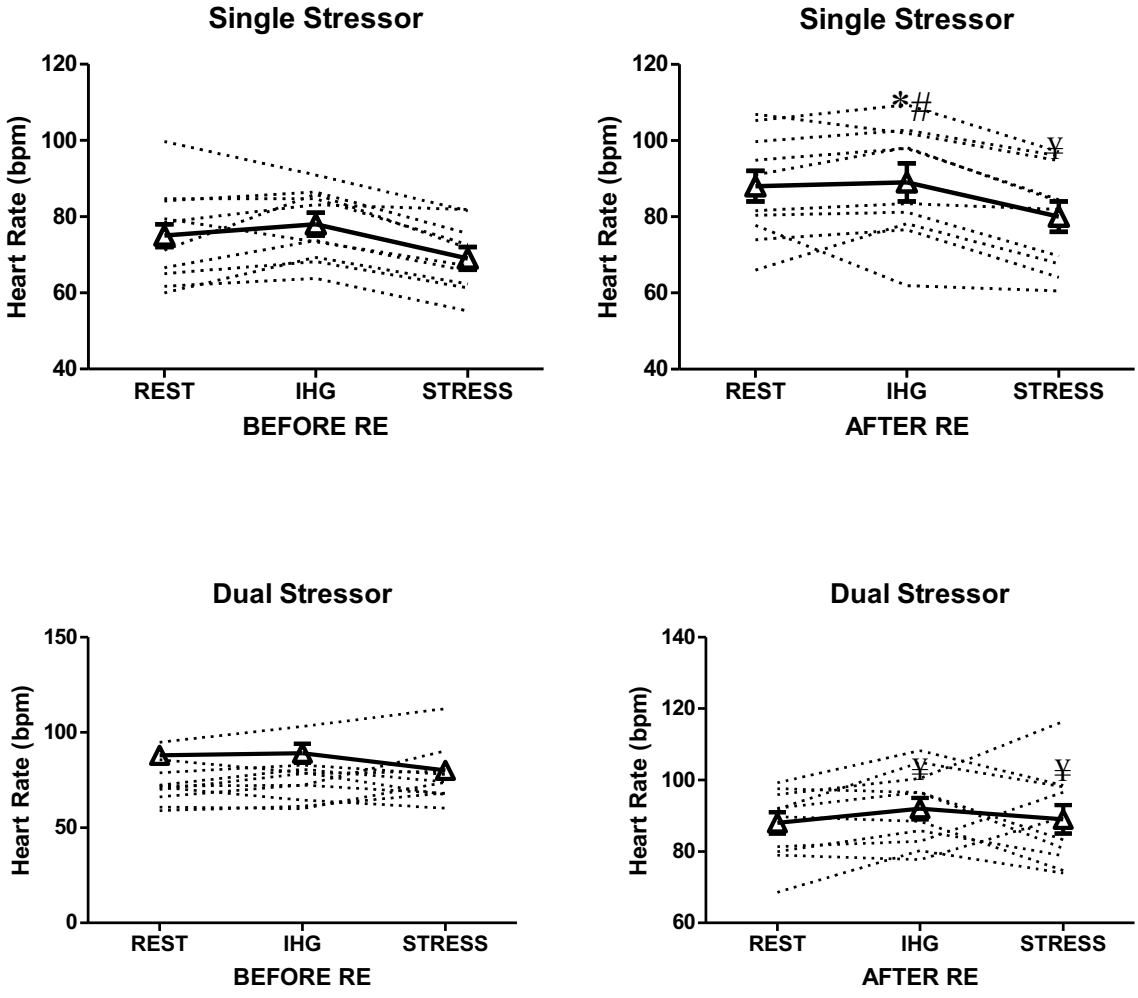

Figure 2. Individual and mean alterations in heart rate during rest, isometric handgrip (IHG), and exposure to the single- and dual-stressor task conditions both before and after resistance exercise (N = 10). Data are presented mean ± standard deviation. * p ≤ 0.05, significantly different from REST, # p ≤ 0.05, significantly different from IHG, ¥ p ≤ 0.05 significantly different from BEFORE RE.

during IHG compared to Rest and STRESS Before RE and IHG compared to STRESS After RE, while also being significantly reduced at all After RE time points compared to Before RE. Main effects of acute RE ($F_{[1,9]} = 19.186$, $p = 0.002$, $\eta_p^2 = 0.681$) and time ($F_{[2,18]} = 10.506$, $p = < 0.001$, $\eta_p^2 = 0.539$) were also observed for ln LF/HF as well such that ln LF/HF was significantly increased during IHG and STRESS compared to REST across both conditions and augmented at all After RE time points compared to Before RE. Finally, a main effect of acute RE ($F_{[1,9]} = 29.986$, $p < 0.001$, $\eta_p^2 = 0.771$) was observed for SampEn, such that SampEn was significantly reduced After RE time points compared to Before RE as well for all time points.

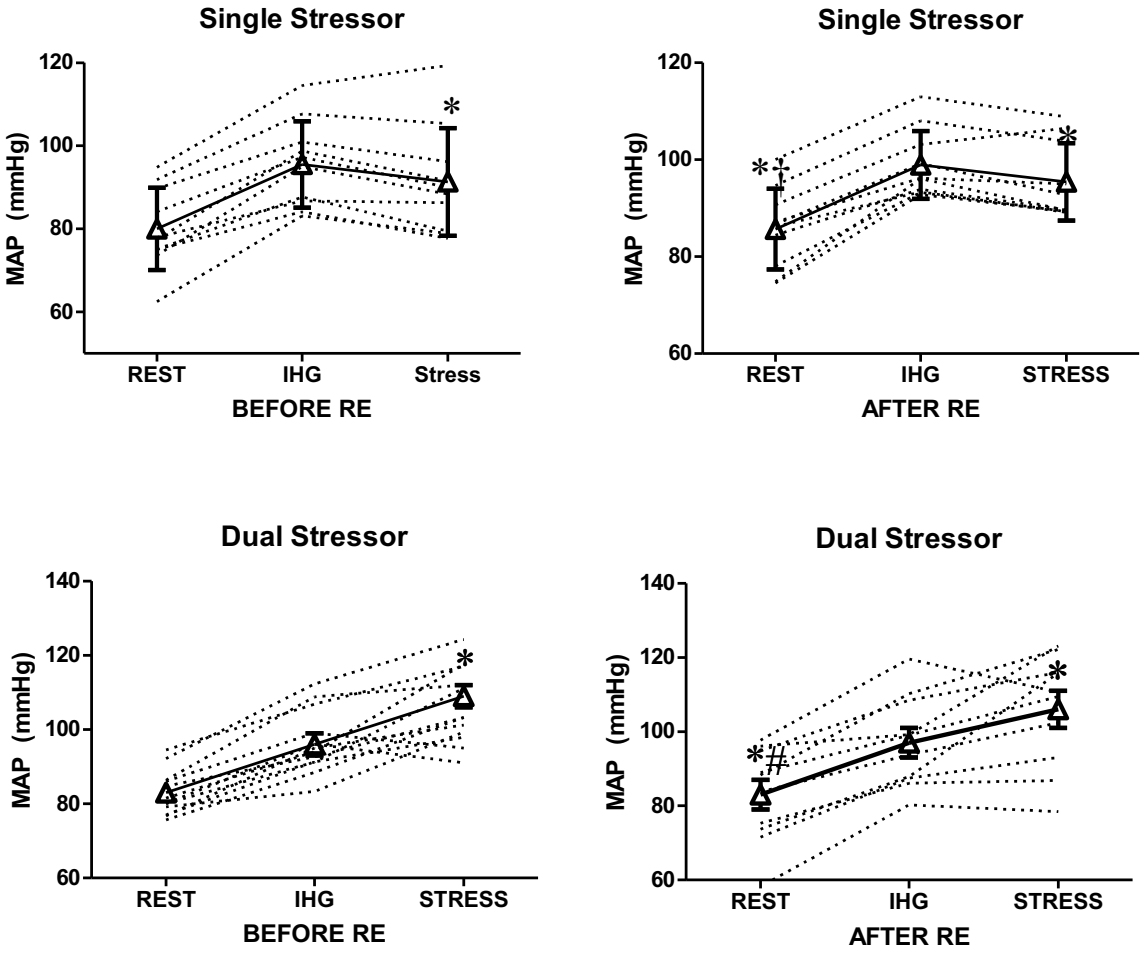

Figure 3. Individual and mean alterations in mean arterial pressure (MAP) during rest, isometric handgrip (IHG), and exposure to the single- and dual-stressor task conditions both before and after resistance exercise (N = 10). Data are presented mean ± standard deviation. * p ≤ 0.05, significantly different from REST, # p ≤ 0.05, significantly different from IHG, ¥ p ≤ 0.05 significantly different from BEFORE RE, † p ≤ 0.05 significantly different than Dual Stressor.

Table 3. Measures of autonomic modulation during rest, isometric handgrip, and exposure to the single- and dual-stressor task conditions both before and after resistance exercise (N = 10).

	BEFORE RE			AFTER RE		
	REST	IHG	STRESS	REST	IHG	STRESS
ln RMSSD (ln ms^2)						
SS	3.7 ± 0.7	3.4 ± 0.5 *	3.9 ± 0.4 #	2.9 ± 0.6 ¥	2.8 ± 0.6 ¥	3.2 ± 0.5 ¥
DS	3.7 ± 0.5	3.5 ± 0.5 *	3.7 ± 0.6 *	2.9 ± 0.06 ¥	2.9 ± 0.7 ¥	3.2 ± 0.5 ¥
ln LF (ln ms^2)						
SS	6.7 ± 0.9	6.5 ± 0.8	7.1 ± 0.9 *#	5.8 ± 1.3 ¥	6.1 ± 1.1	6.8 ± 1.1 *#
DS	6.3 ± 1.0	6.6 ± 0.9	7.0 ± 0.8 *#	5.9 ± 1.5 ¥	6.1 ± 1.1	6.7 ± 1.0 *#
ln HF (ln ms^2)						
SS	6.6 ± 1.2	5.7 ± 0.6 *	6.5 ± 0.7	5.1 ± 1.2 ¥	4.3 ± 1.2 *¥	5.3 ± 1.2 ¥
DS	6.6 ± 0.8	5.8 ± 0.7 *	6.2 ± 1.2	5.4 ± 1.0 ¥	4.3 ± 1.1 *¥	5.2 ± 1.0 ¥
ln TP (ln ms^2)						
SS	8.0 ± 0.8	7.4 ± 0.7 *	8.2 ± 0.7 #	7.0 ± 1.2 ¥	6.7 ± 0.9 ¥	7.6 ± 1.0 #¥
DS	7.9 ± 0.7	7.5 ± 0.8 *	8.0 ± 0.9 #	7.0 ± 1.2 ¥	6.8 ± 1.0 ¥	7.4 ± 1.0 #¥
ln LF/HF ratio						
SS	4.7 ± 1.2	5.5 ± 0.7 *	5.2 ± 0.6 *	5.3 ± 1.1 ¥	6.4 ± 1.0 *¥	6.2 ± 0.7 *¥
DS	4.3 ± 0.9	5.4 ± 1.1 *	5.4 ± 1.1 *	5.1 ± 0.9 ¥	6.4 ± 0.6 *¥	6.1 ± 0.6 *¥
SampEn						
SS	1.3 ± 0.3	1.2 ± 0.3	1.5 ± 0.3	1.1 ± 0.3 ¥	1.0 ± 0.3 ¥	1.2 ± 0.3 ¥
DS	1.4 ± 0.2	1.4 ± 0.4	1.2 ± 0.2	1.1 ± 0.2 ¥	1.1 ± 0.1 ¥	1.0 ± 0.2 ¥

IHG, Isometric Handgrip; ln, natural log; STRESS, exposure to single- and dual-stressor tasks; RE, resistance exercise; RMSSD, Root Mean Square of Successive Differences in R-R Intervals; SampEn, Sample Entropy. TP, Total Power. Data are presented mean ± standard deviation. * $p \leq 0.05$, significantly different from REST, # $p \leq 0.05$, significantly different from IHG, ¥ $p \leq 0.05$ significantly different from BEFORE RE.

Discussion

The present study sought to evaluate how additive sympathetic stimuli, using SS or DS task conditions, alter hemodynamics and autonomic modulation before and after acute RE in young, healthy, resistance-trained participants. The data demonstrated that resistance-trained individuals had the ability to adapt to additive sympathetic stimuli following acute RE in terms of hemodynamics and autonomic modulation. Both conditions resulted in similar responses in HR, Q, SV, and TPR as well as autonomic modulation across time. However, activation of the muscle metaboreflex combined with the CPT (DS condition) amplified MAP both Before and After RE to a greater extent than the SS condition.

The present study demonstrated that resistance-trained individuals had significant alterations in hemodynamics in response to SS and DS conditions, both before and after RE. Before RE for the SS condition, there were significant increases in MAP and TPR in response to the IHG along with significant decreases in HR, MAP, and SV, in response to the PEMI. These data demonstrated that after RE increases in HR and MAP in response to the DS condition were able to further increase compared to Before RE and REST. Kalfon et al[6] (2015) reported that in obese young men there was a significant increase in MAP in response to PEMI + CPT compared to PEMI alone. However, in the present study the participants were young, healthy normal weight individuals compared to overweight/obese individuals in the work by Kalfon et al.[6] Despite this distinction, the responses between the two studies were similar for MAP. Fu et al[5] reported significant increases in MAP in response to head up tilt (HUT) in combination with the CPT in young, healthy individuals. Both of these studies are in agreement with the present study. While it has been demonstrated that the CPT alone increased SBP, DBP and MAP in young, healthy individuals[8,23], the addition of acute RE had little effect on hemodynamics. It has been demonstrated that the CPT increased pain perception, and thus in turn increased sympathetic activity, resulting in increased HR and SBP. However, it has also been demonstrated that acute RE may result in nociception, thereby decreasing the pain experienced with the CPT and thus decreasing cardiovascular reactivity to the cold stressor. Collectively, it is clear that additive sympathetic stimuli when applied to young, healthy individuals results in a robust capacity to increase hemodynamics.

It was hypothesized that both conditions would result in significant decreases in vagal modulation and the data demonstrated this hypothesis to be correct. Before RE, the SS and DS conditions both resulted in significant decreases in ln RMSSD and ln HF, each of which are measures of vagal modulation. Contrary to the hypothesis, SampEn, another measure of vagal modulation, was not reduced by either the SS or DS condition, only acute RE. The differences noted in measures of vagally mediated HRV to HRC are of interest. It has been suggested that HRC is a more sensitive measure of vagal modulation.[24] Notably, there was no change in SampEn in response to SS or DS conditions After RE. However, this may be due to the fact that vagal modulation was significantly decreased in response to acute RE and had not recovered, however this is speculation. Regardless, the data demonstrated that autonomic modulation in young, healthy resistance-trained individuals in response to additive sympathetic stimuli is robust.

Before RE there was a significant increase in LF/HF from IHG to STRESS for both conditions, demonstrating that each PEMI was able to further augment sympathovagal dominance in young, healthy, resistance-trained individuals. After RE, there was a further significant increase in sympathovagal dominance for both conditions. These data suggest that additive sympathetic stimuli were able to further augment sympathovagal dominance in young, health, resistance-trained individuals.

This present study is not without limitations. First, the sample size was small; however based on the *a priori* power equation, there was enough power to detect alterations in hemodynamics and autonomic modulation in response to the stimuli. While the participants were young, healthy individuals they were not assessed for autonomic dysfunction as exclusionary criteria. The individuals in the present study were resistance trained; thus these data cannot be applied to sedentary or aerobically-trained individuals as the responses may be divergent. HRV is primarily focused on vagal mediation and measures of sympathetic activity are not truly possible.

The data from the present study demonstrated that resistance-trained individuals are able to adapt to additive sympathetic stimuli in terms of hemodynamic and autonomic modulation. While the responses in MAP were divergent between the conditions, these responses were expected due to the increased sympathetic stimuli mediated by the CPT. Future studies should consider measurement of sympathetic activity in addition to measures of vagal activity with additive sympathetic stressors.

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