

ORIGINAL ARTICLE

Impact of a motivated performance task on autonomic and hemodynamic cardiovascular reactivity

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ABSTRACT

Motivated performance (MP) tasks include mental stressors characterized by a high degree of motivation, individual engagement, and sympathetic overstimulation. It is therefore important to document the independent influence of motivation apart from engagement on markers of cardiovascular autonomic modulation, including vasomotor tone (low-frequency systolic blood pressure, LFSBP), blood pressure homeostasis (baroreflex sensitivity, BRS), and myocardial oxygen consumption (rate pressure product, RPP). Accordingly, an arithmetic task (AT) was used to manipulate motivation to evaluate its impact on cardiovascular reactivity. Forty-two young adults ($M_{\text{age}} = 20.21$ years, $SD = 2.09$) qualified for the study. After a 10-min resting period, electrocardiogram and finger beat-to-beat blood pressure were recorded at three distinct 5-min stages: baseline (BASE), AT, and recovery (REC). Prior to AT initiation, participants were randomized into two groups based on directions stating that the AT task was either designed to be entertaining and fun (low MP, LMP) or a test diagnostic of one's intelligence (high MP, HMP). Independent of task engagement ratings, motivation to complete the AT task as well as solution success was significantly greater in the HMP than the LMP condition. Regarding physiological parameters, two (LMP vs. HMP) \times three (BASE, AT, REC) repeated measures ANOVAs revealed no significant baseline differences but a significant higher order interaction indicating that in comparison to LMP, individuals in the HMP condition had significantly higher vasomotor tone and myocardial oxygen consumption but not BRS. Greater motivation during a performance task may provide the substrate for the development of adverse cardiovascular events by increasing sympathetic activity and ultimately increasing myocardial oxygen demand which could lead to acute coronary syndromes.

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Introduction

Mental stressors have long been implicated in the development of acute coronary syndromes and adverse cardiovascular outcomes (Strike et al., 2006; Vlastelica, 2008). Although no single precipitant accounts for acute myocardial ischemia, prior studies have demonstrated that activation of the sympathetic nervous system as well as the inability to provide adequate coronary blood supply in response to increases in myocardial oxygen demand are common events in the development of cardiovascular complications (Krantz & Burg, 2014; Minakuchi et al., 2013). Interestingly, not all individuals are equally susceptible to mental stress-induced myocardial ischemia as it can be argued that genetic and chronic disease states may play a critical role in the development of mental stress-induced myocardial ischemia (Krantz & Burg, 2014). Nevertheless, the relative intensity of the mental stressor, and more importantly, the extent to which any given task is subjectively self-relevant, such as a motivated performance (MP) task, may also be implicated (Krantz & Burg, 2014). Seery (2011) defines motivated performance tasks as situations in “which an individual must actively perform instrumental responses to reach a goal that is self-relevant or important in some way”. Test-taking, public speaking, and social

interactions serve as such examples. In fact, MP tasks are characterized by a high degree of individual engagement and motivation (Seery, 2011), and therefore may provide the substrate for the development of adverse cardiovascular events by increasing sympathetic activity, decreasing vagal tone, and ultimately increasing myocardial oxygen demand.

Previous research examining the effects of MP on cardiovascular hemodynamic regulation has most commonly assessed cardiac output, total peripheral resistance (Lackner et al., 2015; Seery et al., 2010; Watkins et al., 2003), and autonomic modulation via indices of heart rate variability (HRV; Croizet et al., 2004). Although the cardiovascular responses associated with MP have been previously documented, lacking are studies assessing the impact of MP tasks on autonomic modulation of the vascular system as well as the feedback loop accountable for the regulation of blood pressure, namely the baroreflex. This is worth noting because a clearer understanding of the underlying mechanisms accountable for cardiovascular responses during a MP task may shed light on the specific triggers for developing acute coronary syndromes and cardiovascular complications.

Furthermore, in the field of MP it is important to have evaluations that separate task engagement from the level of

motivation to invest effort during a performance task (Lackner et al., 2015). For example, in interpreting correlational findings relating goal orientation, cardiac response, and arithmetic task (AT) performance, Lackner et al. (2015) suggest that motivation, more so than engagement, is “apparently the dimension affecting the experience of an individual performance situation and related behavior most”. Thus an experimental evaluation separating task engagement from performance motivation can provide a more comprehensive analysis of the casual mechanisms affecting cardiac responses during a MP task.

The present study therefore used an AT to experimentally evaluate the causal influence of motivation on cardiac changes during MP as well as expand the understanding of cardiovascular functioning pertaining to MP-induced sympathetic stimulation by reporting on indices of myocardial oxygen consumption via rate pressure product (RPP) (Gobel et al., 1978) and markers of vascular autonomic modulation; namely low-frequency systolic blood pressure (LFSBP) and baroreflex sensitivity (BRS); surrogates of vasomotor tone and blood pressure homeostasis (Malliani et al., 1991), respectively. It was hypothesized that relative to a manipulation inducing lower performance motivation, higher performance motivation would evoke greater increases in sympathetic vasomotor tone and myocardial oxygen consumption, independent of task engagement.

Methods

Subjects and study design

Forty-two apparently healthy young adults ($M_{\text{age}} = 20.21$ years, $SD = 2.09$; 82% Caucasian) qualified for study inclusion and signed an informed consent as approved by the Florida State University Institutional Review Board. Participants were excluded from study participation if they smoked, exercised regularly (>3 h per week), were hypertensive, had chronic diseases, or were taking beta blockers, antidepressants, or stimulants. Female participants were tested in the early follicular phase of the menstrual cycle in order to avoid potential variations in pressure wave morphology and cardiovascular functioning (Adkisson et al., 2010).

Data collection was conducted in the morning, after at least an 8-h postprandial period, in a quiet, dimly lit, temperature-controlled room ($23 \pm 1^\circ\text{C}$) at the same time of the day (± 2 h) in order to minimize potential diurnal variations in vascular reactivity. Participants were asked to abstain from caffeine, alcohol, and strenuous physical activity for at least 24 h before the assessment period. After a 10-min resting period in a seated position, continuous electrocardiogram (ECG, Lead II) and beat-to-beat blood pressure, via finger plethysmography, were recorded at three distinct stages for 5 min each: baseline (BASE), an AT, and a recovery (REC) period. In order to avoid the potential influence of forced breathing on cardiovascular autonomic modulation, participants were asked to breathe spontaneously (see Sanchez-Gonzalez et al., 2013 for similar methodology). However, breathing rate was not quantified. After the baseline measurements were concluded, and prior to the initiation of the AT, participants were randomized into two AT groups based on directions designed to manipulate motivation of performance. Groups were equally distributed

at $n = 21$ (11 males) per group. Participants were given directions stating that the AT task was derived either from a (1) common entertainment booklet designed to be completed for fun (low MP, LMP) or (2) from an IQ test that was diagnostic of one’s intelligence (high MP, HMP). Participants were not told they would receive feedback based on their performance. Following task completion, participants were debriefed and thanked. During post-test debriefing, all participants confirmed having no prior experience or exposure to the task descriptions and study methodology.

Motivated performance instructions

Directions for the motivation manipulation were created to increase personal involvement in task performance via self-relevancy of the task outcome (see Gendolla & Richter, 2010 for a review concerning self-involvement manipulations). Pilot testing of manipulation directions revealed that HMP directions significantly increased participants’ belief in motivation to complete a task, $t(119) = 6.89$, $p < 0.001$. Using an online survey, 121 students ($M_{\text{age}} = 21.22$ years, $SD = 2.98$; 68% Caucasian, 62% Female) responded to the items: “How motivated would you be to complete a task that was diagnostic of your intelligence” ($M = 3.54$, $SD = 1.46$) and “How motivated would you be to complete a task that was designed to be completed for fun” ($M = 1.98$, $SD = 1.01$). Responses ranged from 1 = *not at all motivating* to 5 = *extremely motivating*. Mean item responses did not significantly differ in regard to ethnicity or gender ($p > 0.05$).

Arithmetic task

The AT comprised a 5-min serial subtraction task (i.e. serially subtracting 7 from a four digit number: $2407 - 7 = 2400$, $2400 - 7 = 2393$, etc., see May et al., 2015). As a manipulation check, participants were asked to self-report task engagement and motivation for task completion at the end of the physiological measurements (i.e. How engaged in the task were you, 1 = *not at all* to 9 = *very much*; How motivated were you to do the task, 1 = *not at all* to 9 = *very much*).

Cardiovascular measurements

Hemodynamics

From the recorded finger blood pressure (BP) signal (NIBP-100 Biopac Inc., Goleta, CA), the following parameters were measured: heart rate (HR), systolic BP (SBP), diastolic BP (DBP), mean blood pressure ($MBP = 1/3SBP + 2/3DBP$), pulse pressure ($PP = SBP - DBP$), and rate pressure product ($RPP = HR \times SBP$), a surrogate of myocardial oxygen demand (Gobel et al., 1978). The finger plethysmography method has been validated and shown to provide accurate measurement of BP changes when compared with intra-arterial assessments (Jellema et al., 1999).

Heart rate variability

Electrocardiograms were recorded using a single lead (similar to Lead II) tracing at a sampling rate of 256 Hz (MP-150

Biopac Inc., Goleta, CA). The ECG tracings were inspected for artifacts and any premature beats, and all QRS complexes were used to calculate the time intervals between consecutive R waves (i.e. R-R intervals) and were automatically detected using commercially available software (WinCPRS, Turku, Finland). Only RRs corresponding to cardiac cycles of sinus node origin were used to calculate the following HRV parameters: percentage of adjacent R-R intervals that differ by a length of time exceeding the experimental threshold of 50 ms (pNN50), the root mean square of successive R-R differences (RMSSD), the power of low frequency (LF; 0.04–0.15 Hz) by means of Fast Fourier transformation (Pagani et al., 1986), and the power of high frequency (HF; 0.15–0.4 Hz) by means of Fast Fourier transformation (Pagani et al., 1986), and the ratio of LF to HF power (LF/HF).

The indices of pNN50 and RMSSD serve as vagal indices and indicators of parasympathetic activity (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). The HF power is a marker of cardiac parasympathetic (PNS) activity (Pagani et al., 1986) while the LF component of HRV is mediated by both sympathetic (SNS) and PNS activities (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996) and depends partially on baroreflex function (Rahman et al., 2011). The use of absolute units (ms^2) for HF and LF may be obtained in proportion to the total power (TP) minus the very low frequency (VLF) and expressed in normalized units (nu). Normalization (nu) was used to exclude the influence of other fractal components and to control for the changes in TP during stress (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). Since there is structural algebraic redundancy inherent in the normalized spectral HRV measures with respect to each other ($\text{LFnu} = 1 - \text{HFnu}$), and also with respect to the LF/HF ratio, here we report LFnu to denote cardiac sympathovagal tone (Burr, 2007; Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996).

Blood pressure variability

The SBP time series was resampled at 5 Hz and the continuous data passed through a low pass impulse response filter with a cutoff frequency of 0.5 Hz. The data were then subjected to Fast Fourier transform algorithms using a Hanning spectral window and subsequently smoothed using a triangular averaging function to produce a spectrum. The power was calculated by measuring the area under the peak of the power spectra density curve. Power spectra within the 0.04–0.15 Hz range were defined as low-frequency band of SBP (LFSBP) and taken as an estimate of sympathetic vasomotor modulation and increased sympathetic drive (Malliani et al., 1991; Parati et al., 2013).

Baroreflex sensitivity

BRS was calculated from the ECG and beat-by-beat BP recordings with the use of the cross-correlation method, which is a

time-domain sequential method for baroreflex function based on spontaneous SBP and R-R variability changes (Westerhof et al., 2004).

Statistics

Shapiro–Wilk normality tests were used to examine absolute values for tested variables. Results indicated TP, LF, HF, and R-R to be non-normally distributed, thus a logarithmic (Ln) transformation was used. All other tested variables were normally distributed ($p > 0.05$) and therefore parametric statistics were performed. Independent sample *t*-test comparisons of LMP vs. HMP groups were used to evaluate differences in demographics, baseline cardiovascular parameters, self-reported post task motivation ratings as well as AT completion attempts and accuracy scores. A one-sample *t*-test evaluated task engagement values of each condition from a null value of 4.5 (scale mean, $\text{SD} = 1.0$). A modified calculation of Cohen's *d*, Hedges's *g*, was reported for all *t*-test comparisons. Hedges's *g* produces a standardized index of a mean difference often used between two samples or between two measurement points. Hedges's *g* calculation included standardizing (i.e. dividing) the mean difference by the pooled sample standard deviation (instead of using the population standard deviation as in the Cohen's *d* calculation). This calculation protects against overinflating effect size estimates for smaller samples sizes (Fritz et al., 2012). Thus a $g = 0.5$ indicates that the mean difference is half a standard deviation. As with Cohen's *d*, convention indicates $g = 0.2$ as a small effect, $g = 0.5$ as a medium effect, and $g = 0.8$ as a large effect (Cohen, 1992).

To investigate cardiovascular and autonomic variable differences, a 2 (group: LMP vs. HMP) \times 3 (time: BASE, AT, and REC) repeated measures analysis of variance (ANOVA) was conducted. ANOVA analyses were followed by univariate analysis and Fisher's LSD pairwise comparisons for post hoc investigations to detect within-trial differences across time. Partial η^2 was reported for ANOVA comparisons. Partial η^2 describes the proportion of variability associated with an effect when the variability associated with all other effects has been removed (see Fritz et al., 2012). Therefore, it is the ratio of the sum of squares for the variable under analysis (i.e. systematic variance of the factor) divided by the total of that sum of squares and the sum of squares of the relevant error term (i.e. systematic variance of the factor plus error variance). A partial $\eta^2 = 0.5$ would indicate that the independent variable accounted for 50% of the variability in the dependent variable scores. Partial η^2 conventions indicate 0.01 to be a small effect, 0.06 a medium effect and 0.14 a large effect (Cohen, 1992). An *a priori* α level of $p < 0.05$ was considered to be significant. SPSS version 20.0 (SPSS Inc., Chicago, IL) was used for all analyses. Data are reported as mean \pm SD unless otherwise specified.

Results

Demographics and results for motivation and engagement are shown in Table 1. Independent sample *t*-test comparisons of LMP vs. HMP groups self-reported post task motivation ratings

Table 1. Between-group demographics and arithmetic task performance comparisons.

Variable	HMP (<i>n</i> = 21; males = 11)	LMP (<i>n</i> = 21; males = 11)	Group comparison (<i>t</i> -test)		
			<i>t</i>	<i>p</i>	<i>g</i>
Height (m)	1.74 ± 0.90	1.76 ± 0.90	0.79	0.434	0.273
Weight (kg)	70.7 ± 13.3	71.2 ± 17.1	0.09	0.929	0.033
Body mass index (kg/m ²)	23.18 ± 2.89	22.44 ± 3.76	0.65	0.519	0.221
Age (years)	23.31 ± 2.82	24.27 ± 3.47	0.88	0.384	0.304
Motivation	6.41 ± 0.96	4.85 ± 1.18	4.71	<0.001	1.450
Attempts	19.55 ± 7.69	15.25 ± 5.10	2.11	0.041	0.659
Errors	1.31 ± 1.14	3.57 ± 0.84	8.53	<0.001	2.257
Engagement	5.90 ± 1.26	5.95 ± 1.22	0.13	0.988	0.031

Data are mean ± SD.

HMP: high motivated performance; LMP: low motivated performance.

as well as AT completion attempts and accuracy scores indicated significantly greater motivation, solution attempts and accuracy in the HMP group. Task engagement ratings did not significantly differ between groups as indicated by an independent sample *t*-test. Both groups did significantly differ (significantly greater) in engagement values from a null value of 4.5 (mean of engagement scale) via one-sample *t*-tests; $t(20) = 5.10$, $p < 0.001$, $d = 1.675$ for the HMP group and $t(20) = 5.35$, $p < 0.001$, $d = 1.729$ for the LMP group. Two (LMP vs. HMP) \times three (BASE, AT, REC) repeated measures ANOVAs indicated that (i) there were no significant baseline differences between conditions regarding any physiological measurement ($F_s < 1$) and (ii) there was a significant higher order interaction for HR, LnR-R, RMSSD, pNN50, LnTP, LnLF, LnHF, LFnu, SBP, DBP, MBP, RPP, and LFSBP but not for PP or BRS (see Table 2 and Figure 1). Follow-up contrasts (see Table 3) indicated that in comparison to LMP, individuals who were assigned to the HMP condition had significantly lower LnR-R, pNN50, RMSSD, LnTP, and LnHF and higher blood pressure values (SBP, DBP, MBP), LnLF, LFnu, RPP, and LFSBP during the AT. Contrasts at REC indicated that HR was significantly lower for the HMP condition than the LMP condition.

Discussion

We evaluated the impact of a MP task on hemodynamics, cardiac autonomic modulation, sympathetic vasomotor tone, myocardial oxygen consumption, and baroreflex function. The main findings of the present experiment are that independent of task engagement, increases in MP lead to an increase in arithmetic solution success, autonomic and cardiovascular reactivity, sympathetic vasomotor modulation, and myocardial oxygen consumption to a greater extent than less MP. Strikingly, the baroreflex function remained unchanged during both performance tasks suggesting that this feedback mechanism was unaltered. These findings indicate that a subjectively self-relevant mental stressor, in this particular case a more motivating performance task, can influence processes that may ultimately be implicated in the development of adverse cardiovascular events by increasing vasomotor tone and, in turn, increasing myocardial work and myocardial oxygen consumption. The findings of the present study regarding vasomotor tone, BRS, and myocardial oxygen consumption are novel for MP adding to the current notion that a self-relevant task triggers pronounced cardiovascular reactivity.

Consistent with prior research, the results of the present study demonstrated that MP can impact cardiac autonomic modulation and hemodynamics to an extent comparable to the effects produced by negative emotions (Carney et al., 2004; Seery et al., 2010; Seery, 2011). Psychological load, in particular greater MP, may evoke dramatic effects on cardiovascular physiology. For instance, various kinds of emotional excitation (i.e. anxiety, depression, psychological stress, etc.) are known to be associated with increased sympathetic activity characterized by decreased HRV, which may lead to angina and myocardial ischemia in susceptible patients (Strike et al., 2006; Vlastelica, 2008). The neurocardiological mechanisms accountable for the MP induced sympathetic stimulation have been proposed to be associated with higher order thinking and stimulation of the prefrontal cortex, leading to stimulation of the vasomotor center in the medulla oblongata (Esler et al., 1989; Fauvel et al., 2000). Consistent with this notion and prior studies showing stress-induced adrenergic cardiovascular responses (Carter et al., 2005; Esler et al., 1989), we observed increased cardiovascular reactivity as suggested by higher HR and BP during the AT in the more MP condition. In fact, a prior study emphasized the role of sympathetic induced vasoconstriction, demonstrated here as an increase in LFSBP, in the pressure response at the onset of a stressing stimulation (Fauvel et al., 2000). Therefore, life events and tasks carrying high psychological loads, which are inherent to HMP tasks, may potentially have deleterious effects on cardiac health.

In the present study, we did not find any significant changes in baroreflex between baseline and the stress task in either the LMP or the HMP conditions. The results of the present study share some similarities with those of Fauvel et al. (2000) who reported that mental stress-induced an increase in blood pressure, although the stress response was not related to BRS at rest. This is important to mention as the changes in BP in the HMP condition were unlikely to be associated with resting BRS levels. Conversely, in the same study, Fauvel et al. (2000) found a small but significant decrease in BRS during stress, although they used a different stress task from the one used in the present study (series of 4-color words vs. arithmetic subtraction) and had a greater sample size ($n = 280$ vs. $n = 42$). Additionally, Steptoe and Sawada (1989) documented that BRS was reduced significantly during mental arithmetic (~ 3 ms/mmHg). Similarly, Durocher et al. (2011) reported that a 5-min serial subtraction task attenuated BRS. They also highlighted the possibility that during AT, attenuated sympathetic

Table 2. Between-group comparisons of hemodynamic and cardiovascular autonomic responses to arithmetic task.

Variable	HMP (<i>n</i> = 21)			LMP (<i>n</i> = 21)			2 × 3 interaction		
	BASE (M ± SD)	AT (M ± SD)	REC (M ± SD)	BASE (M ± SD)	AT (M ± SD)	REC (M ± SD)	<i>F</i> (2, 82)	<i>p</i>	Partial η^2
HR (bpm)	75.16 ± 7.98	82.32 ± 8.49*	70.81 ± 8.44#*	76.43 ± 7.16	79.49 ± 9.27	75.66 ± 7.02	12.72	<0.001	0.237
Ln R-R	6.67 ± 0.15	6.44 ± 0.14#	6.69 ± 0.13	6.71 ± 0.15	6.68 ± 0.15	6.73 ± 0.18	41.59	<0.001	0.667
RMSSD (ms ²)	48.52 ± 19.61	31.24 ± 14.80#*	50.57 ± 19.70	48.75 ± 15.27	39.37 ± 10.06*	49.31 ± 11.37	3.92	0.028	0.164
pNN50 (ms ²)	27.35 ± 11.18	18.57 ± 14.24#*	28.65 ± 12.39	27.80 ± 12.41	26.41 ± 11.22	28.15 ± 11.84	3.49	0.039	0.078
LnTP	8.09 ± 0.91	7.42 ± 0.84#*	7.99 ± 0.75	8.07 ± 0.91	7.90 ± 0.73	7.94 ± 0.75	3.62	0.036	0.153
LnLF	6.81 ± 0.80	7.49 ± 0.86#*	6.23 ± 0.95*	6.91 ± 0.84	7.00 ± 0.85	6.43 ± 0.97*	6.40	0.004	0.242
LnHF	6.26 ± 0.54	5.57 ± 0.87#*	6.08 ± 0.74	6.38 ± 0.77	6.23 ± 0.95	6.36 ± 0.93	4.37	0.019	0.179
LF (nu)	0.61 ± 0.17	0.69 ± 0.16#*	0.57 ± 0.18	0.61 ± 0.18	0.57 ± 0.17	0.58 ± 0.17	4.18	0.019	0.093
SBP (mmHg)	117.59 ± 11.35	128.14 ± 14.54#*	119.52 ± 12.35	117.15 ± 11.23	118.77 ± 10.74	116.34 ± 10.03	15.91	<0.001	0.280
DBP (mmHg)	71.72 ± 7.31	79.31 ± 5.74#*	73.35 ± 5.74	70.82 ± 3.90	73.16 ± 5.48	70.07 ± 5.79	11.53	<0.001	0.220
MBP (mmHg)	87.49 ± 12.27	95.59 ± 7.64#*	88.74 ± 7.67	86.90 ± 8.20	88.67 ± 5.74	85.50 ± 6.15	6.76	0.006	0.142
PP (mmHg)	48.53 ± 21.78	53.18 ± 30.66	53.57 ± 24.48	44.37 ± 13.80	45.83 ± 14.41	47.10 ± 14.66	1.27	0.284	0.031
RPP (×100 units)	88.47 ± 13.56	100.56 ± 13.78#*	84.71 ± 13.83	89.49 ± 12.44	94.30 ± 13.18	87.97 ± 10.78	24.60	<0.001	0.375
LFSBP (mmHg ²)	5.61 ± 5.54	8.41 ± 9.12#*	7.79 ± 8.10	5.32 ± 4.47	3.56 ± 4.16	5.96 ± 6.24	3.96	0.024	0.088
BRS (ms/mmHg)	11.26 ± 8.00	11.47 ± 10.21	11.90 ± 9.56	9.94 ± 6.36	10.15 ± 7.59	9.73 ± 5.70	0.11	0.877	0.003

Data are mean ± SD.

BASE: baseline; AT: arithmetic task; REC: recovery; HMP: high motivated performance; LMP: low motivated performance; HR: heart rate; LnR-R: R-R interval; RMSSD: root mean square of successive R-R differences; pNN50: percentage of adjacent R-R intervals that differ by a length of time exceeding the experimental threshold; LnTP: total power; LF: low-frequency power of the heart rate variability; HF: high-frequency power of the heart rate variability; SBP: systolic blood pressure; DBP: diastolic blood pressure; MBP: mean blood pressure; PP: pulse pressure; RPP: rate pressure product; LFSBP: low-frequency power of the systolic blood pressure variability; BRS: baroreflex sensitivity.

#*p* < 0.05 different from LMP interaction. **p* < 0.05 different from BASE.

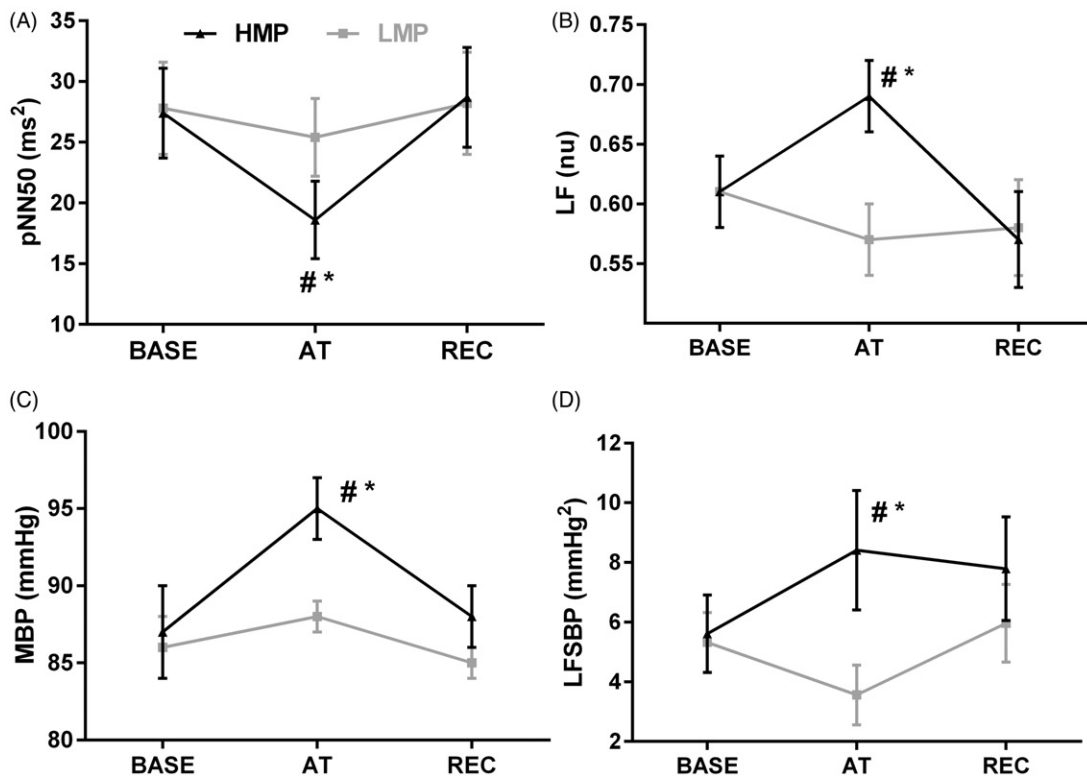


Figure 1. Cardiovascular and autonomic modulation responses to motivated performance task. Data are mean ± SEM. BASE: baseline; AT: arithmetic task; REC: recovery; HMP: high motivated performance; LMP: low motivated performance; (A) pNN50: percentage of adjacent R-R intervals that differ by a length of time exceeding the experimental threshold; (B) LF: low-frequency power of the heart rate variability; (C) MBP: mean blood pressure; (D) LFSBP: low-frequency power of the systolic blood pressure variability. #*p* < 0.05 different from LMP interaction. **p* < 0.05 different from BASE

BRS might contribute to the sympatho-excitation and the pressor response.

It is worth noting that the specific AT used in prior studies were slightly different (i.e. time of task, subtraction directions) from the one used in the present study. We speculate that the low stress nature of the AT used in the present study in addition to the short time used (5 min) was not sufficient to

significantly impact the baroreflex despite significant changes in HR, BP and RPP. In addition, the participants of the present study were young healthy individuals which may explain the lack of a significant BRS decrease using the sequential method of analysis in response to the AT.

BRS has been associated with exaggerated cardiovascular responses to mental stress and aging (Lipman et al., 2002;

Table 3. Between-group contrasts on hemodynamic and cardiovascular autonomic responses to arithmetic task.

Variable	HMP (<i>n</i> = 21) vs. LMP (<i>n</i> = 21)								
	BASE			AT			REC		
	<i>F</i>	<i>p</i>	Partial η^2	<i>F</i>	<i>p</i>	Partial η^2	<i>F</i>	<i>p</i>	Partial η^2
HR (bpm)	0.30	0.587	0.007	1.09	0.303	0.026	4.17	0.048	0.092
Ln R-R	0.51	0.481	0.012	28.15	<0.001	0.407	0.94	0.339	0.022
RMSSD (ms ²)	0.00	0.967	0.000	4.47	0.041	0.098	0.07	0.798	0.002
pNN50 (ms ²)	0.01	0.939	0.000	4.09	0.049	0.091	0.01	0.935	0.000
LnTP	0.00	0.965	0.000	4.10	0.049	0.091	0.05	0.825	0.001
LnLF	0.17	0.681	0.004	4.34	0.044	0.095	0.46	0.501	0.011
LnHF	0.32	0.577	0.008	6.77	0.013	0.142	1.18	0.285	0.028
LF (nu)	0.00	0.965	0.000	6.14	0.017	0.130	0.04	0.837	0.001
SBP (mmHg)	0.02	0.898	0.000	5.73	0.021	0.123	0.85	0.362	0.020
DBP (mmHg)	0.45	0.508	0.011	12.90	0.001	0.239	2.95	0.093	0.067
MBP (mmHg)	0.03	0.854	0.001	12.20	0.001	0.229	2.32	0.135	0.054
PP (mmHg)	0.55	0.464	0.013	0.99	0.326	0.024	1.08	0.305	0.026
RPP ($\times 100$ units)	0.07	0.795	0.002	5.95	0.019	0.127	0.72	0.400	0.017
LFSBP (mmHg ²)	0.03	0.856	0.001	4.94	0.032	0.107	0.68	0.414	0.016
BRS (ms/mmHg)	0.35	0.555	0.009	0.23	0.635	0.006	0.81	0.374	0.019

BASE: baseline; AT: arithmetic task; REC: recovery; HMP: high motivated performance; LMP: low motivated performance; HR: heart rate; LnR-R: R-R interval; RMSSD: root mean square of successive R-R differences; pNN50 percentage of adjacent R-R intervals that differ by a length of time exceeding the experimental threshold; LnTP: total power; LF: low-frequency power of the heart rate variability; HF: high-frequency power of the heart rate variability; SBP: systolic blood pressure; DBP: diastolic blood pressure; MBP: mean blood pressure; PP: pulse pressure; RPP: rate pressure product; LFSBP: low-frequency power of the systolic blood pressure variability; BRS: baroreflex sensitivity.

Moore et al., 2015). It can be argued that the differences found in cardiovascular responses in the present study could be influenced, at least in part, by between subject differences in BRS. Although possible, this is highly unlikely owing to the non-significant differences in BRS between the HMP and LMP groups in both baseline and AT conditions. Continued research into MP and BRS appears necessary as Reyes del Paso et al. (2004) reported that the BRS decreased in response to an AT. However, they also reported that visual attention, which is considered an external attention condition, exhibited a slight increase in BRS.

A small sample size and the inability to recruit an older population are two of the limitations of this study. However, we did observe significant CV alterations in the face of high motivational performance. Hence, using MP for testing cardiovascular responses to stress could be an alternative to traditional stress tests using physical manipulations (i.e. treadmill, stationary bike) as it appears to be independent of the influence of the baroreflex which is strongly associated with aging and cardiac reactivity (Lipman et al., 2002).

Another study limitation is that only healthy subjects were used and hence results of this study cannot be generalized to clinical populations. However, the existence of diseases or medicated subjects may have complicated our findings and their interpretations leading us to choose more restrictive study eligibility criteria. Furthermore, although this MP manipulation was sufficient to produce significant differences in behavioral outcomes (motivation, attempts, and errors) as well as cardiovascular reactivity, it was not strong enough to produce a clear vagal rebound effect (i.e. an increase in parasympathetic activity above resting levels following an acute stressor; Arai et al., 1989; May et al., 2014).

Looking ahead, examination of possible affective mediators (i.e. anxiety or depressive symptoms) or qualitative differences in motivation (i.e. intrinsic vs. extrinsic motivational differences; Ryan & Deci, 2000) in future research may provide additional insight into boundary conditions and underlying

mechanisms which influence the motivational manipulation utilized in this study. In the current study, as depression and anxiety symptoms were not measured, the small subgroup sample size might have produced an uneven distribution of vulnerability to abnormal stress responses despite randomization efforts. Finally, this study focused only on hemodynamic measurement, prospective studies examining brain imaging and vascular autonomic functioning during HMP tasks in both healthy subjects and patients suffering from increased CV appears warranted.

Conclusion

In conclusion, a self-relevant MP task or HMP is associated with increased vasomotor tone, along with exaggerated vascular sympathetic activity in young healthy adults. This in turn can cause noticeable effects on cardiovascular physiology, including increased myocardial oxygen consumption which could lead to acute coronary syndromes especially in populations at increased cardiovascular risk. This research focused on mechanistic components responsible for heart rate (cardiac) and blood pressure regulation by investigating markers of myocardial oxygen consumption via RPP and cardiovascular autonomic function via LFSBP (vascular) and BRS which have not been previously investigated in the context of MP tasks.

Disclosure statement

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