Sympathetic neural responses to mental stress: responders, nonresponders and sex differences

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Carter JR, Ray CA. Sympathetic neural responses to mental stress: responders, nonresponders and sex differences. Am J Physiol Heart Circ Physiol 296: H847–H853, 2009. First published January 23, 2009; doi:10.1152/ajpheart.01234.2008.—Mental stress consistently increases heart rate (HR) and blood pressure (BP) in humans, despite inconsistent sympathetic neural responses that include increases, decreases, or no change in muscle sympathetic nerve activity (MSNA). The purpose of the present study was to examine associations between MSNA, BP, and HR responses to mental stress. Leg MSNA, BP, HR, and perceived stress levels were recorded during 3–5 min of mental arithmetic in 82 subjects (53 men and 29 women). MSNA, BP, and HR similarly in positive responders (Δ−6 ± 1 bursts/min; n = 10), negative responders (Δ−3 ± 3 bursts/min; n = 9), and nonresponders (n = 33). Mental stress increased MSNA in positive responders (Δ6 ± 1 bursts/min), decreased MSNA in negative responders (Δ−6 ± 3 bursts/min), and did not change MSNA in nonresponders (Δ1 ± 1 bursts/min). Mental stress increased mean BP and HR similarly in positive responders (Δ15 ± 1 mmHg and Δ16 ± 1 beats/min; P < 0.001), nonresponders (Δ15 ± 1 mmHg and Δ19 ± 2 beats/min; P < 0.001), and negative responders (Δ12 ± 2 mmHg and Δ19 ± 3 beats/min; P < 0.001). Perceived stress levels and sex distributions were similar across responders and nonresponders; thus, perceived stress and sex do not appear to influence MSNA during mental stress. However, men demonstrated higher increases of mean BP during mental stress when compared with women (Δ16 ± 1 vs. Δ12 ± 1 mmHg; P < 0.05), despite no differences in MSNA responses. In conclusion, our results demonstrate marked differences in MSNA responses to mental stress and a disassociation between MSNA and BP responses to mental stress, suggesting complex patterns of vascular responsiveness during mental stress.

autonomic nervous system; blood pressure; mental arithmetic; muscle sympathetic nerve activity; cardiovascular control

SYMPATHETIC NEURAL REACTIVITY is routinely assessed in healthy and diseased patients. Common techniques used to activate sympathetic neural outflow include mental stress, isometric handgrip, cold pressor test, hypoxia, Valsalva’s maneuver, head-up tilt, and lower body negative pressure. Although most of these techniques consistently elicit a neurally mediated sympaethoexcitation, the sympaethoexcitation induced by mental stress is remarkably inconsistent. Mental stress has been reported to increase (2, 3, 7, 12, 13, 18, 20, 26, 27, 33), decrease (14, 24), and not change (10, 11, 34) muscle sympathetic nerve activity (MSNA).

In contrast to the inconsistent MSNA response, mental stress elicits prompt and consistent increases in arterial blood pressure and heart rate. However, the relations between MSNA, blood pressure, and heart rate, during mental stress have not been explored adequately. Specifically, no studies have attempted to determine if the pressor response and tachycardia during mental stress are associated with the MSNA responsiveness. In other words, do individuals with marked increases in MSNA during mental stress have a more dramatic rise in arterial blood pressure and heart rate?

An explanation for the highly variable MSNA response to mental stress is still lacking. Callister et al. (7) suggested the perception of stress may modulate the MSNA response, but recent evidence from our laboratory challenges this concept (10). We recently demonstrated no correlation between the self-reported perceived stress level and MSNA responsiveness, but this study did not have a wide range of MSNA responsiveness to mental stress (10). It is possible that a larger data set that includes both MSNA responders and nonresponders would be more revealing. Finally, only one study has probed for potential sex differences in the MSNA responsiveness to mental stress (19).

Therefore, the primary purpose of this study was to comprehensively examine the relations between MSNA, arterial blood pressure, and heart rate during mental stress. Based on the literature, we classified subjects as positive MSNA responders, negative MSNA responders, and nonresponders. We hypothesized that the hypertension and tachycardia associated with mental stress would be more dramatic in positive MSNA responders. A secondary purpose of this study was to reexamine the relationship between perceived stress and MSNA during mental stress. Based on previous evidence (7), we hypothesized perceived stress levels would be higher in positive MSNA responders. Finally, a tertiary purpose of this study was to explore for sex differences. Based on previous evidence (19), we did not anticipate any sex differences.

METHODS

Subjects. Eighty-two volunteers (53 men and 29 women; age 18–31 yr) were studied. All subjects were healthy, nonsmoking adults with no history of autonomic dysfunction or cardiovascular disease. Subjects arrived at the laboratory after abstaining from caffeine and exercise for at least 12 h. Data for this study were collected from previous experiments that measured MSNA responses during mental stress (10–13, 21, and unpublished data). Subject characteristics are provided in Tables 1 and 2. The experimental protocols were consistent with the principles of the Declaration of Helsinki, and were approved by the Institutional Review Boards of Michigan Technological University and Pennsylvania State University. All subjects provided written informed consent before the study.

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Table 1. Baseline subject characteristics: responders vs. nonresponders

<table>
<thead>
<tr>
<th>Variable</th>
<th>POS (n = 40)</th>
<th>NON (n = 33)</th>
<th>NEG (n = 9)</th>
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<td>Sex, M/F</td>
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<td>Heart rate, beats/min</td>
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<td>69 ± 2</td>
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<td>MSNA, bursts/min</td>
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<td>13 ± 1</td>
<td>15 ± 2</td>
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</tbody>
</table>

Values are means ± SE; n, no. of subjects. M, males; F, females; MSNA, muscle sympathetic nerve activity; BP, blood pressure; POS, positive MSNA response to mental stress (≥3 bursts/min); NON, no MSNA response to mental stress (range: <3 bursts/min to >3 bursts/min); NEG, negative MSNA response to mental stress (≥3 bursts/min). *P < 0.05 vs. corresponding values.

Experimental design. Subjects were divided into groups based upon their MSNA responsiveness to mental stress. Based on evidence that mental stress increases, decreases, and does not change MSNA, we categorized subjects into the following three groups: 1) positive responders (increased MSNA by ≥3 bursts/min during mental stress; n = 40), 2) negative responders (decreased MSNA by ≥3 bursts/min during mental stress; n = 9), and 3) nonresponders (failed to increase or decrease MSNA by 3 bursts/min during mental stress; n = 33). The cut-off value of 3 bursts/min was chosen because this represents the typical increase of MSNA during mental stress in studies reporting a significant sympathoexcitation during mental stress (2, 3, 7, 12, 13, 18, 20, 26, 27, 33). However, data were also analyzed in a continuum using correlation analysis to ensure our arbitrary MSNA cut-off points were appropriate. In addition to examining responders versus nonresponders, data were also examined for sex differences.

Data collected for the present study came from various studies. Specifically, 40 subjects came from cross-sectional studies that examined the interactions and/or relationships between mental stress and other sympathoexcitatory maneuvers, such as vestibular activation (13), negative pictures (10), head-up tilt (21), and head-down tilt (unpublished observation). To limit the potential influence of one protocol on another, adequate rest intervals were dispersed between protocols to ensure all recorded variables returned to baseline values. For each study (10, 13, 21), baseline measurements between the various protocols were similar. The remaining 42 subjects came from a study examining arm and leg MSNA during mental stress (11) and longitudinal studies that examined the influence of menstrual phase (12), exercise training (unpublished observations), and nutritional supplementation (unpublished observations) on MSNA during mental stress. For the longitudinal studies, only preintervention data were examined.

Mental stress. Mental arithmetic was used to elicit mental stress in all 82 subjects. Briefly, subjects continuously subtracted the number 6 or 7 from a two- or three-digit number. Subjects answered verbally and were encouraged by an investigator to subtract as quickly as possible. An investigator provided a new number from which to subtract every 5–10 s. Mental arithmetic was performed for either 3 min (n = 32) or 5 min (n = 50). The length of the mental stress trial did not elicit different responses; thus, data were pooled to provide 40 positive responders, 9 negative responders, and 33 nonresponders. All subjects rated their perceived stress during the mental arithmetic protocol using the following standard five-point scale: 0, not stressful; 1, somewhat stressful; 2, stressful; 3, very stressful; and 4, very, very stressful (7).

The authors for the current study have previously published together on MSNA responses to mental stress (9, 11, 13, 21), and the two laboratories utilized the same mental arithmetic procedures for all studies included in the current data set. Analysis of MSNA by multiple individuals can introduce error, but in this case the two individuals (Carter and one laboratory technician) analyzing the neural recordings for the 82 subjects in the present study were both trained by the same investigator (Ray). Thus we are confident that our MSNA analysis is consistent and appropriate for the current study.

Microneurography. Microneurographic recordings of MSNA were recorded through the use of microneurography of the peroneal nerve, as previously described (32). The nerve signal was amplified (80,000 gain), band-pass filtered (700–2,000 Hz), and integrated at a time constant of 0.1 s to obtain a mean voltage display of nerve activity.

Blood pressure and heart rate. Resting arterial blood pressures were recorded using an automated sphygmonanometer (Omron HEM-907XL, Omron Health Care or Dinamap XL, Johnson & Johnson). The average of the three blood pressure readings was calculated to provide a resting value. Beat-to-beat arterial blood pressure was measured continuously throughout the experiment using a Finapres or Finometer (Finapres Medical Systems). Heart rate was recorded via a three-lead electrocardiogram. Arterial blood pressures are expressed as systolic, diastolic (DAP), and mean (MAP) arterial pressures.

Data analysis. Data were imported and analyzed in commercially available software programs (WinCPRS, Absolute Aliens or Peaks; ADInstruments). Muscle sympathetic bursts were detected on the basis of amplitude using a signal-to-noise ratio of 3:1, within a 0.5-s search window centered on the 1.3-s expected burst peak latency from the previous R-wave. Potential bursts were displayed and edited by an investigator. MSNA was expressed as burst frequency (bursts/min) and total activity (total number of bursts multiplied by an averaged normalized burst area; expressed as %change).

Statistical analysis. Baseline subject characteristics and resting values were analyzed using a one-way ANOVA (positive responders vs. negative responders vs. nonresponders) or a t-test (males vs. females). A repeated-measures ANOVA was used to determine the effects of our intervention (i.e., mental stress). Pearson correlations were used to probe for relationships between perceived stress, MSNA, heart rate, and arterial blood pressure responses to mental stress. Means were considered significantly different when P < 0.05. Results are expressed as means ± SE.

RESULTS

Responders vs. nonresponders. Baseline subject characteristics for the positive responder, nonresponder, and negative responder groups are shown in Table 1. Baseline variables were not significantly different across groups, except for age, which was slightly older in the negative responders.

Figure 1 demonstrates that mental stress increased MSNA in positive responders (Δ6 ± 1 bursts/min and Δ167 ± 37%; P < 0.001), decreased MSNA in negative responders (Δ−6 ± 1 bursts/min and Δ−38 ± 8%; P < 0.01), and did not change

Table 2. Baseline subject characteristics: males vs. females

<table>
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<tr>
<th>Variable</th>
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<th>Females (n = 29)</th>
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<td>Height, cm</td>
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<td>Body mass, kg</td>
<td>84 ± 2</td>
<td>64 ± 2*</td>
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<tr>
<td>Body mass index, kg/m²</td>
<td>26 ± 1</td>
<td>23 ± 1*</td>
</tr>
<tr>
<td>Systolic BP, mmHg</td>
<td>123 ± 2</td>
<td>111 ± 3*</td>
</tr>
<tr>
<td>Diastolic BP, mmHg</td>
<td>68 ± 2</td>
<td>66 ± 1</td>
</tr>
<tr>
<td>Mean BP, mmHg</td>
<td>87 ± 2</td>
<td>81 ± 2*</td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>68 ± 2</td>
<td>67 ± 2</td>
</tr>
<tr>
<td>MSNA, bursts/min</td>
<td>13 ± 1</td>
<td>10 ± 1</td>
</tr>
</tbody>
</table>

Values are means ± SE; n, no. of subjects. *P < 0.05 vs. males.
MSNA in nonresponders ($\Delta 0 \pm 1$ bursts/min and $\Delta 17 \pm 8\%$).
In contrast to the variable neural responses, mental stress increased MAP and heart rate similarly in positive responders ($\Delta 15 \pm 1$ mmHg and $\Delta 16 \pm 1$ beats/min; $P < 0.001$), negative responders ($\Delta 12 \pm 2$ mmHg and $\Delta 19 \pm 3$ beats/min; $P < 0.001$), and nonresponders ($\Delta 15 \pm 1$ mmHg and $\Delta 19 \pm 2$ beats/min; $P < 0.001$). Self-reported perceived stress levels were similar in positive responders (2.6 ± 0.1 arbitrary units (AU)), nonresponders (2.7 ± 0.1 AU), and negative responders (2.9 ± 0.3 AU; Fig. 1).

Figures 2 and 3 demonstrate that, when all data were analyzed in a continuum (i.e., not classified based on MSNA responsiveness; $n = 82$), findings were similar to Fig. 1. Changes in MSNA during mental stress were not correlated to changes in MAP (burst frequency: $r = -0.01$, $P = 0.90$; total activity, $r = 0.02$, $P = 0.87$). Because of the strong relationship between MSNA and DAP (32), we also examined for correlations between these two variables. Figure 2 demonstrates that changes in MSNA during mental stress were not correlated to changes in DAP (burst frequency: $r = -0.03$, $P = 0.79$; total activity, $r = 0.01$, $P = 0.93$). Figure 3 demonstrates that changes in MSNA were not correlated to perceived stress levels (burst frequency: $r = 0.10$, $P = 0.38$; total activity: $r = -0.07$, $P = 0.55$). Furthermore, changes in heart rate during mental stress were not correlated to changes

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**Fig. 1.** Changes in muscle sympathetic nerve activity (MSNA), mean arterial pressure (MAP), heart rate (HR), and perceived stress levels during mental stress in positive MSNA responders (RES), nonresponders (NON), and negative MSNA responders (NEG). Mental stress elicited similar increases in MAP and HR in all three groups despite significantly different MSNA responses. Perceived stress levels were not different across groups. *$P < 0.001$ vs. nonresponders.

**Fig. 2.** Correlations of MSNA and diastolic arterial pressure (DAP) during mental stress. Changes in MSNA were not correlated to changes in DAP.
in MSNA (burst frequency: \( r = -0.05, P = 0.75 \); total activity: \( r = -0.08, P = 0.49 \)) or perceived stress (\( r = 0.04, P = 0.75 \)), and changes in MAP were not correlated to perceived stress (\( r = -0.14, P = 0.20 \)).

**Sex differences.** Although sex distributions appeared similar in the responder and nonresponder groups (Table 1), we divided our subjects into males and females (disregarding our initial classification of responders and nonresponders) to probe for potential sex differences. Table 2 reports baseline subject characteristics for males and females. Baseline blood pressure and body mass index were higher in males, but resting MSNA and heart rate were similar between males and females.

Figure 4 shows that males demonstrated higher increases in MAP during mental stress compared with females (\( \Delta 16 \pm 1 \) vs. \( \Delta 12 \pm 1 \) mmHg; \( P < 0.05 \)), despite reporting a lower perceived stress score (2.5 ± 0.1 vs. 2.9 ± 0.1 AU; \( P < 0.05 \)). The MAP sex difference remained when responses were grouped by MSNA responsiveness (time × sex interaction, \( P < 0.03 \); time × sex × group interaction, \( P > 0.64 \)). Specifically, males demonstrated augmented MAP responses to mental stress in positive MSNA responders (\( \Delta 16 \pm 1 \) vs. \( \Delta 13 \pm 2 \) mmHg), nonresponders (\( \Delta 16 \pm 2 \) vs. \( \Delta 12 \pm 2 \) mmHg), and negative MSNA responders (\( \Delta 16 \pm 3 \) vs. \( \Delta 8 \pm 4 \) mmHg) compared with females. Changes in MSNA (\( \Delta 2 \pm 1 \)...
vs. $\Delta 3 \pm 1$ bursts/min) and HR ($\Delta 17 \pm 1$ vs. $\Delta 18 \pm 1$ beats/min) during mental stress were not different in males and females.

**DISCUSSION**

Sympathetic neural and cardiovascular responses to mental stress have been studied extensively, but a systematic investigation of the relations between MSNA, blood pressure, and heart rate during mental stress has been lacking. This is surprising because mental stress appears to elicit rather consistent increases in blood pressure and heart rate despite highly variable sympathetic neural outflow. The present study reports three new findings. First, the level of MSNA responsiveness (i.e., positive responders, negative responders, or nonresponders) does not influence blood pressure and heart rate responses to mental stress. Second, the perception of stress does not appear to influence MSNA responsiveness. Third, although sex does not influence MSNA responses to mental stress, our results demonstrate sex differences in blood pressure responses and perceived stress ratings during mental stress. These findings help clarify the relations between MSNA, blood pressure, and heart rate during mental stress. The apparent dissociation of MSNA and blood pressure during mental stress suggests complex patterns of vascular responsiveness.

It is well documented that arterial and cardiopulmonary baroreflexes modulate the control of MSNA at rest and during baroreceptor loading or unloading (6, 32, 35). Specifically, reductions of blood pressure increase MSNA, whereas elevations of blood pressure reduce MSNA. This classic negative feedback system allows for beat-to-beat control of arterial blood pressure, but certain conditions (i.e., exercise and stress) challenge this biological control system. For example, exercise produces concurrent increases in blood pressure and MSNA (23, 30). The ability to increase MSNA during elevations in blood pressure has been explained by a resetting of the baroreflexes (i.e., an upward and rightward shift in the arterial baroreflex curve) (28, 29). A similar resetting of the baroreflex has been proposed to explain concurrent increases in blood pressure and MSNA during mental stress. However, unlike exercise, mental stress has a highly variable MSNA response. The physiological significance of this highly variable MSNA response to mental stress has not been clarified.

The present study aimed to determine if the highly variable MSNA response to mental stress influences arterial blood pressure and heart rate responses. Our results demonstrate that blood pressure and heart rate responses to mental stress are not influenced by the level of MSNA. We observed similar increases of blood pressure and heart rate in positive MSNA responders, negative MSNA responders, and nonresponders. Furthermore, changes in MSNA were not correlated to blood pressure or heart rate. The dissociation of MSNA and blood pressure suggests that mental stress may elicit complex patterns of vascular control. For instance, the remarkably similar increases in blood pressure during mental stress indicate that positive responders may experience more vasodilation during mental stress, or perhaps the nonresponders and negative responders experience more vasoconstriction. The present study did not include measurements of blood flow; thus, we cannot determine which vascular bed(s) may be modulated differently in responders and nonresponders. However, it is important to note that forearm vasodilation during mental stress is consistent and well-documented (4, 5, 16, 31). The mechanisms responsible for the stress-induced forearm vasodilation remain unresolved, but studies have reported that nitric oxide (8, 15), circulating epinephrine (22), and MSNA (16) may contribute to this response, but the role of MSNA in stress-induced forearm vasodilation has recently been questioned (11). Furthermore, it has been reported that children with at least one hypertensive parent had significantly increased forearm bloodflow responses to mental stress compared with age-matched children with no family history of hypertension, yet the increases in arterial pressure associated with the mental stress were similar between groups (1). This finding (1) suggests genetic differences may contribute to disassociations of vascular and pressor responses during mental stress. It would be of interest to determine if the amount of forearm vasodilation differs in positive MSNA responders, negative MSNA responders, and nonresponders. Based on our results demonstrating no difference in blood pressure responses, it could be hypothesized that positive MSNA responders experience greater forearm vascular conductance during mental stress compared with negative responders or nonresponders.

It has been reported that MSNA responses to mental stress are governed primarily by the perception of stress. Callister et al. (7) examined MSNA during a graded mental stress trial that included six levels of increasing difficulty. It was determined that MSNA decreased during low levels of difficulty and perceived stress, but increased linearly during higher levels of difficulty and perceived stress. Therefore, it has been argued that the highly variable MSNA response to mental stress may be the result of the level of perceived stress. Recently, we challenged this concept by demonstrating no correlation between perceived stress levels and changes in MSNA during mental stress triggered by mental arithmetic and emotional stress triggered by negative pictures (10).

The present study provides strong and persuasive evidence that MSNA is not primarily governed by the perception of stress. The present study demonstrates consistent levels of perceived stress in positive MSNA responders, negative MSNA responders, and nonresponders. Moreover, changes in MSNA were not correlated to perceived stress levels. We utilized the same perceived stress scale as Callister et al. (7), and the highest levels of perceived stress reported by Callister et al. (7) are consistent with ratings of perceived stress reported in the present study. Taken together with our previous findings (10), we conclude that the perception of stress does not appear to modulate MSNA during mental stress.

Finally, the present study had a statistically robust number of men and women, allowing us to examine sex differences. Only one other study has adequately probed for autonomic sex differences during mental stress. Jones et al. (19) reported no differences in MSNA, blood pressure, or heart rate responses to isometric handgrip, cold pressor test, and mental arithmetic in men and women. Our MSNA and heart rate responses to mental stress are in agreement with Jones et al. (19), but our blood pressure data are not. The present study demonstrates an attenuated pressor response in women compared with men. The differences between the present study and Jones et al. (19) are not entirely clear but may be because of differences in...
experimental protocol. Jones et al. (19) tested the autonomic responses in a nonrandomized manner, with the mental arithmetic always being performed last. This may have resulted in a reduced excitation over time, an idea supported by the lower-than-average increases in blood pressure and heart rate during mental stress (19). In fact, the increases in blood pressure and heart rate reported by Jones et al. (19) appear to be less than half of the values reported in the present study.

What is perhaps more surprising is that the augmented pressor response in men was associated with a significantly reduced perceived stress level compared with the women. In other words, despite perceiving the mental arithmetic trial as more stressful than their male counterparts, women had a significantly reduced hypertensive response to mental stress. Moreover, the differences in arterial blood pressure responses were not associated with differences in MSNA. Thus our sex data further support the idea that perceived stress does not modulate autonomic responses to mental stress and that there is a dissociation of MSNA and blood pressure during mental stress. Potential mechanisms underlying the sex-dependent blood pressure responses to mental stress remain unclear, but previous evidence suggests that it is unlikely related to endogenous levels of sex steroids (12) or differences in stress-induced forearm vasodilation (17).

In conclusion, the present study provides the first comprehensive examination of the relations between perceived stress, MSNA, blood pressure, and heart rate during mental stress. The classification of subjects into either responders and nonresponders, or men and women, indicates a dissociation of MSNA and blood pressure during mental stress. This dissociation suggests complex patterns of vascular responsiveness to mental stress, and the clinical implications of such responses are unclear, particularly in patients at increased risk for cardiovascular complications. For instance, it has been reported that the augmentations of forearm blood flow during mental stress are blunted in patients with heart failure compared with control subjects (25). Furthermore, patients with heart failure have greater resting renal vasoconstriction compared with control subjects, and mental stress intensifies this renal vasoconstriction (25). Future studies examining the complex patterns of vascular control during mental stress, and the potential mechanisms responsible, may lend valuable insight into understanding the role psychological stress plays in the development of hypertension, heart failure, and other cardiovascular diseases.

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GRANTS

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