Hemodynamics and Arterial Properties in Response to Mental Stress in Individuals with Mild Hypertension

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Objective: The role of tonic sympathetic stimulation on the properties of large arteries is largely unknown. The purpose of this study was to determine the effect of mental stress on hemodynamics and arterial properties in mild hypertensives. **Method:** Twenty-three subjects with mild hypertension and 19 age-matched normotensives were compared to examine changes in hemodynamics and central arterial wave reflection before, during, and after mental stress. **Results:** The results demonstrate an acute effect of mental stress on blood pressure, heart rate, and arterial compliance. The static component (MBP) and the pulsatile (PP) component of arterial pressure increased significantly during mental stress and returned to baseline within a few minutes. Mild hypertensives did not have an increased response to mental stress. For both groups, an increase in HR and a consequent rise in CO were responsible for the increase in BP in response to mental stress. Compared with baseline, both groups demonstrated a decrease in arterial compliance during stress. Mental stress did not induce a significant change in total peripheral vascular resistance nor did it affect central arterial wave reflection in both groups. Individuals with mild hypertension demonstrated higher PP ($p < .001$), lower arterial compliance ($p < .01$), and higher AI ($p < .05$) than those with normal BP. **Conclusions:** Hemodynamic and arterial responses to mental stress in individuals with normal BP and mild hypertension were similar. Several parameters, however, were different in basal state. These differences (ie, higher PP, lower compliance, and higher AI in the mild hypertensive group) could be due to the chronic effect of sympathetic stimulation on central arteries. **Key words:** augmentation index, arterial properties, mild hypertension, hemodynamics, sympathetic nervous system

AI = augmentation index; **BP** = blood pressure; **CO** = cardiac output; **DBP** = diastolic blood pressure; **HR** = heart rate; **MBP** = mean blood pressure; **PP** = pulse pressure; **SBP** = systolic blood pressure; **SCWT** = Stroop Color and Word Test; **SV** = stroke volume; **TPR** = total peripheral resistance.

INTRODUCTION

Researchers have recently gained a greater understanding of the role played by the mechanical properties of the large central arteries in the pathogenesis of increased BP (1, 2). The aorta and large arteries buffer the pressure oscillations that result from intermittent ventricular ejection of blood, transforming intermittent flow into a steady flow of blood in the periphery. These vessels expand in systole to accommodate a portion of the SV and rebound in diastole to facilitate forward blood flow. Pressure waves are reflected back from the periphery of the circulation and summate with the forward wave, producing the characteristic aortic pressure waveform. With optimal timing of the ventricular-vascular coupling, the reflected waves return to the aorta during diastole and the systolic pressure is not affected. Because distensibility of the large arteries decreases with age and/or hypertension, the transmission velocity and amplitude of both the forward and reflected waves increase, causing the reflected wave to arrive earlier in the central aorta and augmenting pressure in late systole. This disproportionate increase in SBP increases PP, also known as the pulsatile component of arterial pressure.

Pulse wave analysis uses a validated generated transfer function to synthesize the aortic pressure wave and aortic SBP and PP from the radial pressure wave (2). This method also allows researchers and clinicians to analyze the timing of wave reflection by calculating AI, a measure of wave reflection in the aorta. AI is inversely related to aortic distensibility. Pulse wave analysis is thus a useful method for studying the effects of sympathetic stimulation on the large arteries.

The role of tonic sympathetic stimulation on the properties of large arteries is still unclear. Pharmacologically induced increases in adrenergic tone have been shown to decrease distensibility of in situ isolated carotid arteries in rats (3). Conversely, carotid and femoral arterial distensibility increased and elastic modulus (ie, stiffness) decreased in sympathectomized rats (4). In addition, Failla et al. (5) reported that distensibility of medium-sized and large arteries in humans increased after ipsilateral anesthesia of the brachial plexus. Taken together, these data indicate that sympathetic tone restrains large artery distensibility. However, Sonesson et al. (6) demonstrated that sympathetic activation produced by lower body negative pressure did not change compliance, distensibility indices, or elastic modulus of the abdominal aorta in humans. Thus, additional research is needed to better understand the role of tonic sympathetic stimulation on the physical properties of the large arteries.

Several researchers have used laboratory stressors as a means to activate sympathetic tone in studies of stress reactivity. Lafleche et al. (7) documented central arterial responses to the cold pressor test, a classic test of sympathetic activation causing arteriolar vasoconstriction. Studies that examine the effect of mental stress-induced sympathetic activation on large-artery properties, however, have been lacking.

The purpose of this study was to examine hemodynamic and arterial responses to mental stress in individuals with untreated mild hypertension. The authors intended to study the ensuing BP and other physiological responses to stress in the hypertensive sample, including both high normal and mild hypertensives, to learn about the presumed stress-disease pathway. The results from this study may shed light on the role of tonic sympathetic stimulation in the pathogenesis of hypertension.

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METHODS

Participants

The study was approved by the institutional review board. All participants gave informed consent. Subjects selected for participation in the study were healthy volunteers who could be classified as either normotensive or mild hypertensive. The mild hypertensive group included individuals qualified the high normal or stage I hypertension based on guidelines drawn from the sixth report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (8). Participants were recruited via public BP screening and via direct invitation to study participants through wide dissemination of flyers advertising the study. Those passing the initial screen were invited to undergo a detailed eligibility evaluation. Subjects were excluded from the study if they were taking anitihypertensive medications or if their medical history demonstrated evidence of significant cardiovascular, renal, metabolic, neurological, or psychiatric disease.

Participants were classified as mild hypertensive if both screening SBP measurements—which were obtained on two separate occasions—were within the range of 130 to 159 mm Hg and/or both DBP measurements were within the range of 85 to 99 mm Hg. Subjects were classified as normotensive if both screening SBP measurements were below 130 mm Hg and the DBP measurements were below 85 mm Hg.

Mental Stress

The mental stress instrument used in the study was the SCWT. The SCWT is composed of a color-name reading task, a color naming task, and an interference task. Various studies have demonstrated an association between the SCWT and significant increases in respiratory rate, plasma and urinary adrenaline, electrodermal activity (9), CO (10), HR (9, 10), and BP (10, 11). These findings validate the efficacy of the SCWT in inducing sympathetic responses.

Blood Pressure

BP was measured continuously and noninvasively with the use of radial artery tonometry (Model 7000, Colin, San Antonio, TX) coupled with a computerized data acquisition system (WINDAQ, Dataq Instruments, Akron, OH). Radial artery tonometry provides beat-to-beat BP values and a high-fidelity arterial pressure waveform and has been shown to offer a reliable trend indicator of pressure changes (12). This instrument satisfied the Association for the Advancement of Medical Instrumentation standards for mean SBP and DBP and only minimally exceeded the allowed standard deviations (13). The tonometric sensor contains an array of pressure transducers. When a pneumatic pump and bellows press the transducer array against the skin and tissue above the artery, the sensor records the pressure wave. The radial tonometric equipment also includes an oscillometric cuff and a pressure transducer with electronic processing capability, which can be used to determine brachial SBP and DBP and MBP. These pressure values were used to calibrate radial sensor output.

Augmentation Index

Kelly et al. (14) initially defined AI as the ratio of augmentation pressure to PP expressed as a percentage. Augmentation pressure is defined as the difference in pressure between the early and late systolic shoulders of central aortic pressure waveforms. In this study the central aortic wave was derived from a radial pressure wave recorded using an applanation tonometer (Millar Pressure Tonometer, Millar Instruments) and a pulse wave analysis system with a generalized transfer function (SCOR-Px/P, SphygmoCor pulse wave analysis system, AtCor Medical). This device automates the assessment of AI, expressed as a percentage, using the formula $AI = 100 \times (P_s - P_i)/(P_s - P_d)$, where P_i is the inflection point, P_s is the peak systolic pressure, and P_d is the minimum DBP (Figure 1). The pulse wave analysis system allows for adjustment of the effect of HR on wave reflection. Adjusted values of AI with HR at 75 bpm were used in the data analysis.

Previous studies (15–17) have reported excellent reproducibility for AI measurements with a between-visit correlation coefficient of 0.98, standard deviation of within-observer measurement difference of 1.3% to 5.37%, and standard deviation of between-observer measurement differences of 3.80%. In terms of validity of this approach, aortic pressure waves derived from the radial pressure waves showed agreement with the measured aortic pressure waves with respect to systolic, diastolic, pulse, and mean pressures (18). Differences between recorded and derived aortic pressures were within specified limits of the Association of the Advancement of Medical Instrumentation SP10 criteria.

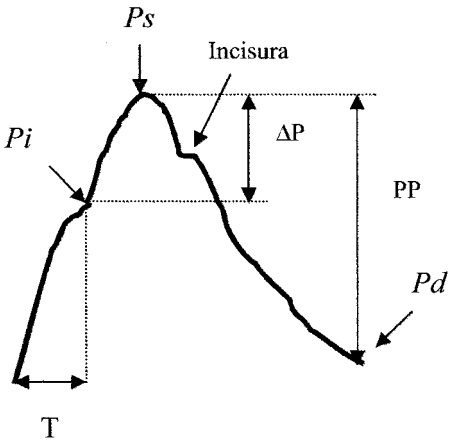

Fig. 1. Calculation of AI. AI is calculated as the difference between P_s and P_i (ΔP), expressed as a percentage of the difference between P_s and P_d (PP). T is the time between the foot of the wave and the inflection point, which provides a measure of the travel time of the pressure wave to and from the major reflection site.

HR, SV, and TPR

The procedure for impedance instrumentation and signal acquisition was based on published guidelines by Sherwood et al. (19). Briefly, HR and SV were measured using the Minnesota impedance cardiograph (MIC model 304B, Surcom, Minneapolis, MN). Four band electrodes were placed around the participant's neck and chest. The upper and lower voltage electrodes were placed around the base of the neck and around the thorax at the level of the xiphisternal junction. The outer current electrodes were placed at a distance of 3-cm separation from the inner electrodes. The Impedance Cardiogram computer (model 7000, Surcom) was used to calculate and record data. SV was calculated for each impedance wave, and three to four waves were averaged to obtain one value for each data collection point. HR (in bpm) was calculated from the R-R interval on the EKG waveform. TPR was estimated using the formula $TPR = MBP/CO \times 80$ (dyn·s·cm^{-5}). CO (in l/min) was calculated using the formula $CO = (SV \times HR)/1000$.

The validity of the impedance-derived measurements has been well documented through comparison with reference measures. A close agreement between impedance-derived CO and CO measured by the thermodilution method in humans has been reported in absolute values ($r = 0.85$) and in percent change ($r = 0.87$) (20). A high correlation ($r = 0.82$) has also been shown between impedance-derived SV and SV measured by nuclear ventriculography in humans (21). Mehlsen et al. (22) have provided evidence that supports outstanding reproducibility for impedance-derived measurements with low measurement differences on the same day (standard deviation of the differences in CO = 0.12 l/min) and on different days (standard deviation of the differences in CO = 0.45 l/min) and low intraobserver variability (standard deviation of differences in CO = 0.12 l/min).

Arterial Compliance

Arterial compliance was estimated using the ratio of SV to PP. Aortic PP was calculated by subtracting aortic DBP from aortic SBP. The SV/PP ratio has been demonstrated to be a valid estimate of total arterial compliance (23–25).

Study Protocol

BP and HR measurements were taken with an oscillometric BP monitor (SpaceLab, Redmond, WA) at 2-minute intervals after each participant had

been seated quietly for 5 minutes. Measurements were taken until two BP measurements were found to be within 5 mm Hg of each other. The mean of those two readings and the corresponding HR measurements were defined as clinic BP and HR.

Before the beginning of laboratory testing, participants acclimatized to the laboratory environment by relaxing in a seated position with their eyes open for a 10- to 15-minute adaptation and resting period. The testing phase consisted of three 6-minute periods during which BP waves were continuously recorded using beat-to-beat radial tonometry. The first 6 minutes constituted a resting period and was used to establish baseline measurements. During the next 6-minute period, participants received a brief introduction to and performed the SCWT. The final 6 minutes served as a recovery period. The mean BP was calculated for each of the three periods. Three pairs of cardiac impedance and AI measurements were taken during each period of the testing phase and averaged for the analysis. The investigator ensured that each impedance measurement was obtained simultaneously with an AI measurement.

Statistical Analysis

Data were analyzed with the use of statistical analysis software (version 11.0, SPSS, Chicago, IL). Results are expressed as means ± SD. The clinical characteristics of both groups were compared using a t test for independent samples. The changes in BP, hemodynamics, and arterial properties throughout the SCWT were analyzed using multivariate repeated-measures analysis of variance. A p value $<.05$ was considered significant.

RESULTS

Participants included 17 men and 25 women aged 20-64 years (mean = 42.7 years, SD = 11.9). The normotensive group consisted of 19 participants (11 women), and 23 participants (14 women) comprised the mildly hypertensive group. Table 1 shows the clinical characteristics of both groups of participants. Although height and age did not differ between the groups, body mass index was significantly higher in the mild hypertensive group. BP and HR measured with an oscillometric monitor also differed significantly between the groups, as expected.

Table 2 shows the changes in mean values of BP, hemodynamics, and arterial properties throughout the mental stress protocol. All of the BP variables (SBP, DBP, MBP, and PP) were significantly higher in the mildly hypertensive group than in the normotensive group (group factor: $p < .001$). In addition, HR, TPR, and adjusted AI (AI adjusted for HR at 75 bpm) were significantly higher and compliance was significantly lower in participants with mild hypertension than in those with normotension (Table 2).

TABLE 1. Clinical Characteristics of Participants by Group

Variables	Mild Hypertensive (N = 23)	Normotensive (N = 19)
Height (cm)	171.7 ± 10.5	171.7 ± 8.34
BMI (kg · m^{-2})[a]	30.7 ± 6.9*	24.1 ± 3.60
Age (year)	44.4 ± 10.9	40.5 ± 12.9
Clinic SBP (mm Hg)[b]	142.6 ± 7.4*	117.3 ± 9.2
Clinic DBP (mm Hg)[b]	88.2 ± 9.3*	69.6 ± 6.7
Clinic HR (bpm)[b]	79.1 ± 11.1*	67.7 ± 9.5

Values are mean ± SD.
N = number of participants.
[a] BMI: body mass index.
[b] Clinic blood pressure and heart rate were measured with an oscillometric BP monitor.
* $p < .01$, versus normotensive controls, by t-test.

Mean SBP, DBP, MAP, and PP increased significantly (time factor: $p < .001$) in response to the SCWT for both groups. The two study groups demonstrated similar patterns of change in all variables of BP (ie, no group and time interaction). Figure 2 shows the change in MBP, and Figure 3 shows the change in PP throughout the mental stress protocol.

During the SCWT, HR and CO rose significantly from baseline for both groups, but TPR did not. Neither the pattern of changes in HR nor the pattern of changes in CO differed between groups (ie, no group and time interaction). Figure 4 shows the changes in HR during the mental stress protocol. The SCWT did not induce significant changes in AI (ie, no time effect on AI adjusted for HR at 75 bpm). Arterial compliance, however, decreased significantly (time factor: $p < .05$) in response to the SCWT. Figure 5 shows the changes in arterial compliance during the mental stress protocol. In both groups, PP amplification, calculated from the ratio of radial PP to aortic PP, increased during the SCWT and returned to baseline during the recovery period (time factor: $p < .01$). Figure 6 shows the changes in the ratio of radial PP to aortic PP throughout the mental stress protocol. Again, the two study groups did not differ in the pattern of changes in PP amplification.

DISCUSSION

This study demonstrated an acute effect of mental stress on BP, HR, and arterial compliance (calculated as the SV/PP ratio). Both the static component (ie, MBP) and pulsatile component (ie, PP) of BP markedly increased in response to mental stress and returned to baseline within a few minutes after exposure to the stressor. The increase in PP in response to mental stress in the absence of a SV change reflects a decrease in vascular compliance. Mental stress did not induce a significant change in vascular resistance nor did it affect central arterial wave reflection in individuals with mild hypertension or in those with normal BP. One potential limitation of these findings is that the validity of using the SphygmoCor transfer function to derive aortic waveforms during sympathetic stimulation remains to be tested as the algorithm has previously been validated only in baseline conditions.

Hypertension is known to decrease central arterial distensibility and systemic compliance (26, 27). In this study, individuals with mild hypertension demonstrated higher PP, lower arterial compliance, and higher AI than those with normal BP. These findings suggest that decreases in the elastic properties of central arteries and in arterial compliance are likely to show up at an early stage in the development of hypertension.

Central arterial stiffness causing an earlier return of reflection waves and a higher aortic PP leads to a decrease in or disappearance of PP amplification. A previous study (7) demonstrated that arterial wave reflection increased and PP amplification disappeared in response to the cold pressor test in both individuals with borderline hypertension and those with normotension. In the current study, however, PP amplification

TABLE 2. Changes in Blood Pressure, Hemodynamics, and Arterial Properties During Mental Stress Protocol

Variables	Mild Hypertensive			Normotensive			Repeated-Measures ANOVA		
	Baseline	SCWT	Recovery	Baseline	SCWT	Recovery	Group	Time	Interaction
SBP (mm Hg)	133.7 (12.3)	144.2 (13.0)	138.1 (11.5)	105.0 (11.9)	117.1 (13.6)	106.2 (13.6)	<0.001	<0.001	NS
DBP (mm Hg)	72.6 (10.4)	78.5 (11.0)	75.5 (10.5)	56.6 (10.0)	63.9 (11.5)	57.0 (12.0)	<0.001	<0.001	NS
MBP (mm Hg)	93.4 (10.7)	101.2 (11.0)	96.5 (10.1)	72.8 (9.5)	82.0 (11.4)	73.6 (12.2)	<0.001	<0.001	NS
PP (mm Hg)	61.1 (10.0)	65.7 (12.7)	62.5 (10.0)	48.4 (9.4)	53.3 (9.5)	49.5 (9.1)	<0.001	<0.001	NS
HR (bpm)	74.9 (12.0)	82.2 (12.7)	73.5 (9.9)	65.6 (9.9)	79.4 (12.2)	66.8 (9.2)	<0.01	<0.001	NS
SV (ml)	62.8 (34.2)	60.9 (27.8)	62.7 (31.8)	74.3 (22.2)	71.9 (28.6)	74.7 (21.9)	NS	NS	NS
CO (l)	4.5 (1.9)	4.9 (2.1)	4.4 (1.8)	4.8 (1.2)	5.5 (1.9)	4.8 (1.1)	NS	<0.001	NS
TPR (dyn · s · cm^{-5})	2020.2 (937.6)	1926.9 (751.8)	2063.5 (882.4)	1298.5 (387.9)	1306.4 (452.6)	1275.6 (377.1)	0.001	NS	NS
PP_r/PP_{ao}[a]	1.37 (0.23)	1.42 (0.2)	1.38 (0.17)	1.42 (0.23)	1.47 (0.26)	1.40 (0.22)	NS	<0.01	NS
AI @HR75 (%)[b]	21 (12)	23 (12)	21 (11)	13 (19)	13 (20)	12 (18)	0.05	NS	NS
Compliance (ml · mm Hg^{-1})	1.45 (0.90)	1.36 (0.76)	1.44 (0.93)	2.25 (0.85)	2.01 (0.79)	2.18 (0.82)	<0.01	<0.05	NS

Values are expressed as means (SD).
NS = not significant.
[a] PP_r: radial pulse pressure, PP_{ao}: aortic pulse pressure.
[b] AI @HR75: AI adjusted for HR at 75 bpm.

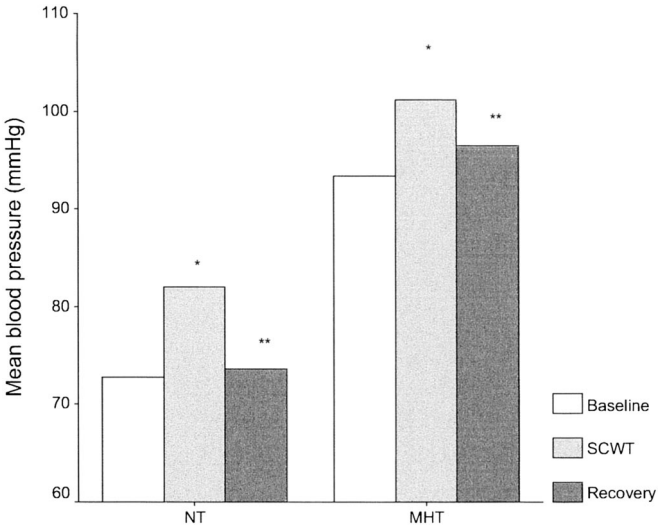

Fig. 2. Changes in MBP in response to mental stress. NT: normotensives, MHT: mild hypertensives. *$p < .001$ vs. baseline, **$p < .001$ vs. SCWT.

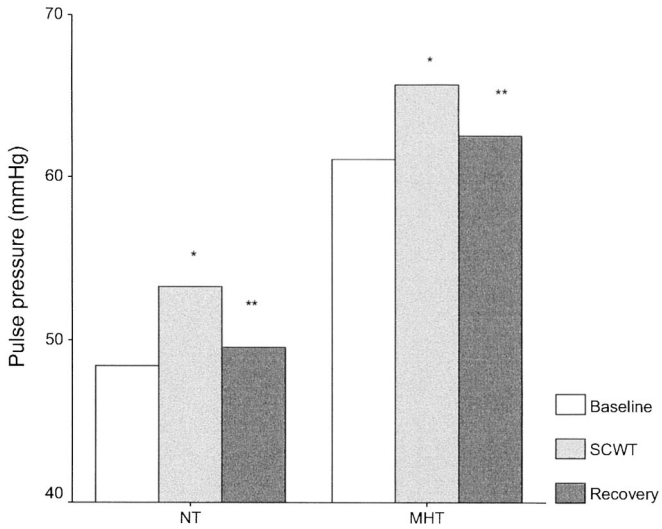

Fig. 3. Changes in PP in response to mental stress. *$p < .001$ vs. baseline; **$p < .001$ vs. SCWT.

did not decrease in response to mental stress; in fact, the ratio of radial to aortic PP increased in both groups of participants. This discrepancy can be explained by the different mechanisms through which the cold pressor test and the SCWT increase BP. The early return of the reflection wave (and thus higher AI) and the higher central arterial PP seen with the cold pressor test occur as a result of vasoconstriction moving the arteriolar site of reflection closer to the heart. In contrast, though HR increased markedly in response to the SCWT, there was no change in peripheral vascular resistance. Consequently, AI, an indicator of central arterial reflection, did not change throughout the mental stress protocol in the present study. One might argue, however, that the lack of observed change in peripheral vascular resistance does not preclude a certain degree of vasoconstriction in some regional territories, such as the splanchnic territory.

This study demonstrated that mental stress induced a de-

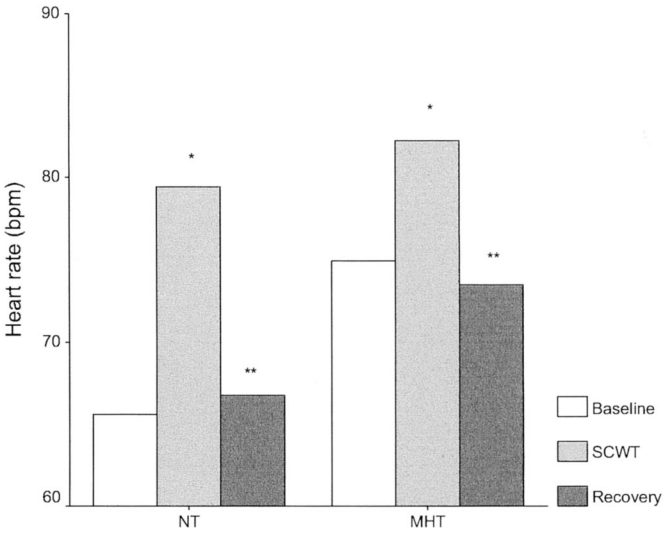

Fig. 4. Changes in HR in response to mental stress. *$p < .001$ vs. baseline; **$p < .001$ vs. SCWT.

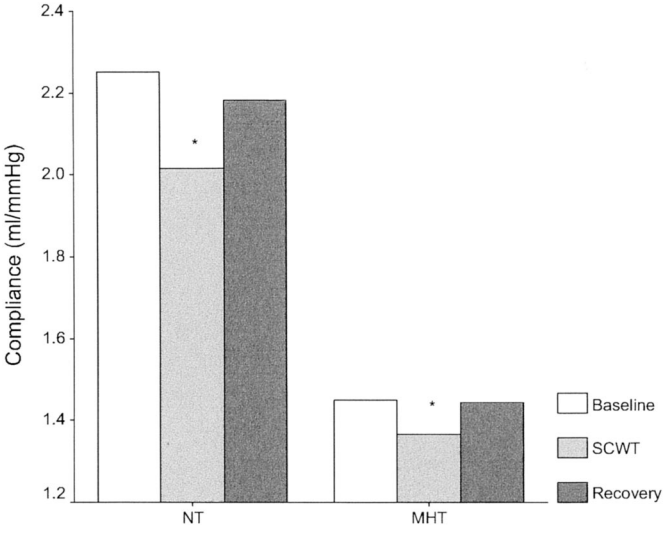

Fig. 5. Changes in arterial compliance in response to mental stress. *$p < .05$ vs. baseline.

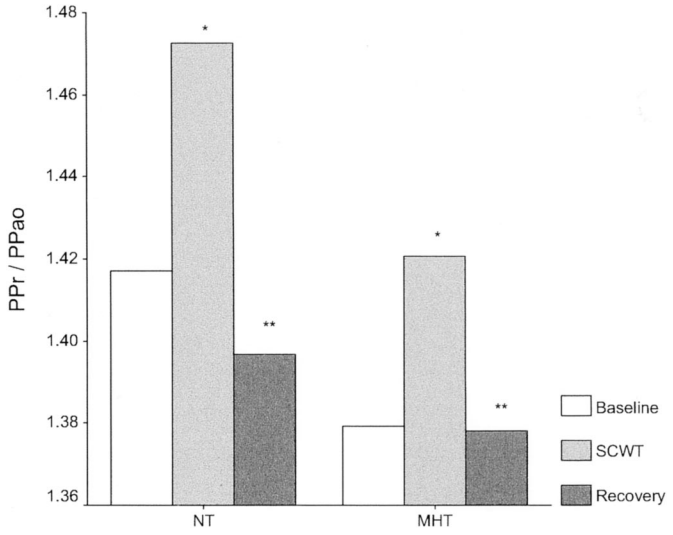

Fig. 6. Changes in PP amplification in response to mental stress. P_{Pr}/PP_{ao}: ratio of radial to aortic PP. *$p < .05$ vs. baseline; **$p < .05$ vs. SCWT.

cline in SV/PP but did not induce significant changes in AI. One would expect both indices reflect changes in arterial compliance with mental stress. There are two possible explanations for this discrepancy. First, mental stress mainly induced a change in HR but not peripheral vascular resistance. AI is primarily determined by the amplitude and timing of waves reflected from the lower body (28) and is directly related to aortic pulse wave velocity, a well-established measure of aortic distensibility (27). As aortic distensibility decreases, pulse wave velocity increases, causing the reflected wave to arrive during ventricular systole and thereby increasing systolic pressure and, thus, AI. However, evidence suggests that the distance of the wave travels from the reflection site to the aorta is as important as arterial distensibility for the determination of AI (28). Because this study demonstrated that mental stress induces an increase in HR but not TPR, it is not surprising to see no change in AI with mental stress. Second, the SV to PP ratio estimates overall vascular compliance. This index is probably more related to capacitive compliance than reflective compliance. It is likely that acute mental stress only affects peripheral arterial compliance but not properties of the central artery. Most importantly, the magnitude and pattern of change in both arterial compliance and AI during the stress protocol were similar between groups. This leads us to speculate that the putative effect of acute mental stress on central arterial properties is unlikely to mark a pathway for the development of hypertension.

Several studies have demonstrated that the initial hemodynamic abnormality in the early phase of hypertension is normal vascular resistance with an elevated CO and HR (29, 30), associated with markers of increased sympathetic and decreased parasympathetic tone of central origin (30). In the present study, the experimental group consisted of individuals presumably in an early phase of hypertension. As such, they would be expected to show a hyperkinetic state (ie, increased CO and HR). Instead, the majority of individuals in the hypertensive group exhibited a normokinetic state. Of the 23 subjects of that group, 19 had a cardiac index that was less than the mean plus 1 SD of the normotensive group's cardiac index and only three had a cardiac index above that level. A significantly higher TPR was also observed in the mild hypertensive group, suggesting that individuals in the early phase of hypertension are likely to manifest a vascular profile. These observations combined with the results that individuals in the early stage of hypertension did not have an increased response to mental stress support the structural adaptation model of hypertension development advocated by Folkow (31).

Various studies (32, 33) have suggested that individuals with mild hypertension display exaggerated BP reactivity to stress compared with normotensives. In this study, the mag-

nitude and pattern of changes in BP, HR, and TPR demonstrated by individuals with mild hypertension were similar to those demonstrated by individuals with normal BP. These findings suggest that mental stress does not abnormally increase sympathetic nervous activity in individuals with mild hypertension as compared with those with normal BP. These findings also support the concept that factors other than basal BP level may mediate the individual differences in laboratory stress reactivity (34).

Certain limitations of the present study may preclude a definitive conclusion about whether reactivity is increased in persons with mild hypertension. First, the current study sample had a mean age of 44 with a wide age range. Increased sympathetic reactivity to stress may be restricted to young persons with hypertension (35). Second, the mild hypertensive group included two categories of the JNC VI classification: high normal BP and stage I hypertension. Circulatory patterns thus may not be uniform among all study participants. The high normal group may include true borderline neurogenic hypertensives whereas the stage I hypertensive group may not. However, further analysis revealed that there were no differences in hemodynamics and arterial properties in response to mental stress when these two categories of mild hypertensive individuals were analyzed separately (data not shown). Also, as stated earlier the majority of individuals in the hypertensive group exhibited a normokinetic state. This uneven distribution makes the comparison of the two subgroups (hyperkinetic vs. normokinetic) impossible. Third, there was a significant difference in body mass index between the two study groups, with the mild hypertensive group exhibiting a higher body mass index. However, because obese individuals are known to have higher sympathetic activity than individuals of normal weight, if this difference were to affect the results at all, it would have been to skew the data in support of the hypothesis. It seems clear, then, that this difference did not have an effect on the findings. Finally, men and women may respond differently to stress. In the present study, resting HR, TPR, and AI were significantly higher in women than in men (data not shown). However, there were no differences in hemodynamic and arterial responses to mental stress when men and women were analyzed separately.

In terms of hemodynamic patterns underlying BP responses to metal stress, HR was the only factor that changed significantly across the three experimental periods. In both the mild hypertensive group and the normotensive group, the SCWT induced a significant increase in HR but not SV or TPR. Not surprisingly, the pattern of change and the magnitude of increases in HR were not significantly different between the mild hypertensive group and the normotensive group. As discussed earlier, the majority of the individuals in the hypertensive group exhibited a normokinetic state. A decreased responsiveness of the heart to β-adrenergic stimulation has been suggested to explain the transition from a hyperkinetic state to a normokinetic state in borderline hypertension (36). A reduced cardiac responsiveness may account for the lack of difference in HR reactivity and recovery between groups. Consistent with this line of thinking, there was no difference in BP reactivity and recovery between groups. In fact the percent increases in MAP, SBP, and DBP were smaller in the mild hypertensive group than in the normotensive group (13% vs. 9%, 12% vs. 8%, and 13% vs. 8%, respectively). Similar findings have been reported by a previous study in which the relatively smaller increase in BP in the hypertensive group was attributed to a reduced SV responsiveness related to left ventricular hypertrophy (37).

A reduced cardiac responsiveness must be accompanied by a vascular hyperresponsiveness in order to preserve the BP response to stress, assuming that BP response is obligatory (36). A hypertrophy-related vascular hyperresponsiveness and a preserved BP response to stress have been well documented in subjects with established hypertension (36). Yet, in the present study, mild hypertensives had an elevated vascular resistance, a decreased central arterial compliance, a preserved BP response to stress and a tendency toward reduced cardiac responsiveness without evidence of vascular hyperresponsiveness to stress. Taken together, these findings lead us to speculate that withdrawal of the vagal tone is the most likely mechanism underlying the BP response to mental stress in mild hypertension.

CONCLUSIONS

Hemodynamic and arterial responses to mental stress in individuals with normal BP and mild hypertension were similar. Several parameters, however, were different in basal state. These differences (ie, higher PP, lower compliance, and higher AI in the mild hypertensive group) could be due to the chronic effect of sympathetic stimulation on central arteries. On the basis of these results, we speculate that the putative effect of acute mental stress on central arterial properties is unlikely to mark a pathway for the development of hypertension.

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