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Circulatory and Central Nervous System Responses to Different Types of Mental Stress

Xinxin LIU[1]*, Koichi IWANAGA[2] and Shigeki KODA[1]

[1]National Institute of Occupational Safety and Health, 6–21–1 Nagao, Tama-ku, Kawasaki 214-8585, Japan
[2]Graduate School of Engineering, Chiba University, 1–33 Yayoi-cho, Inage-ku, Chiba 263-8522, Japan

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Abstract: The purpose of the present study was to compare the physiological responses to different types of mental stress encountered in the workplace. Circulatory and central nervous system responses were examined in 8 healthy males by exposing them to 20-min of white noise (80 dB(A)) and 20-min of computer-based mental arithmetic tasks as models of vascular and cardiac stress, respectively. The results indicated that both cardiac and vascular stresses increased blood pressure and showed a cumulative effect as exposure period was extended. Heart rate and prefrontal oxygenated hemoglobin levels (measured by NIRS) increased in the face of cardiac stress but were not clearly altered by vascular stress and indicated that cardiac stress higher cardiac response and requires more oxygen supply to the brain. As the central nervous system responded, an event-related potential P300 component was elicited by an auditory oddball task presented before and after each stress. The P300 amplitude increased for both stresses. However, P300 latency increased in response to cardiac stress but decreased with vascular stress in the left prefrontal. Thus, the circulatory and central nervous system responses to cardiac stress and to vascular stress may have different underlying mechanisms, and measuring physiological indices appears to be an effective method by which to evaluate the influence of mental stress.

Key words: Mental stress, Blood pressure, Event-related potential

Introduction

As a result of technological developments, physical stress has been remarkably reduced in the workplace, while mental stress has increased. Previous studies have reported that prolonged exposure to mental stresses can increase the risk of circulatory disease and affect cognitive functioning[1–4]. It is important to evaluate the physiological influences of mental stress to reduce the risk of stress-related disorders.

It has been hypothesized that persons at risk for stress-related circulatory disorders can be identified from their chronic exaggerated blood pressure response and their underlying hemodynamic to the increased blood pressure[5]. Rose *et al.*[6] reported that circulatory reactivity to work-related mental stress, especially an increase in systolic blood pressure, may be a long-term predictor of incident hypertension. It is known that blood pressure (mean arterial pressure: MAP) was elevated by cardiac output (CO) and/or total peripheral resistance (TPR), and the relation among these three indices can be described as $MAP = CO \times TPR$. The underlying hemodynamic in increasing blood pressure was also considered to be a risk factor, and previous studies have suggested that vascular reactivity has been associated with a high risk for hypertension[7, 8].

In addition, to examine the influence of mental stress on cognitive function, the P300 component of the event-related potential (ERP) usually is used. P300 can be elicited by an oddball task, which consisted of standard (a low-frequency sound) and target (a high-frequency sound) stimuli, and the participants were instructed to press a button as quickly as possible once they heard the randomly sounded target tone. A positive peak that occurs 250–500 ms after the target tone is defined

*To whom correspondence should be addressed.
E-mail: liu@h.jniosh.go.jp

as P300. P300 is considered to reflect cognitive neuroelectric phenomena; its amplitude is considered to reflect the amount of cognitive resource allocation and the process efficiency, and its latency is considered to reflect the time needed for evaluation of stimuli[9]. P300 can be used to examine the cognitive function in the workplace; Chiovenda *et al*.[10] used P300 to examine the influence of traffic noise on traffic police officers. The results showed a wider P300 amplitude reduction in traffic police officers than in controls under noisy conditions, suggesting that occupational noise exposure has chronic effects on cognitive functioning and underlying cognitive processing. Gomes *et al*.[4] reported that P300 latency was significantly longer and the amplitude significantly lower in workers employed as aircraft technicians than in the control group. P300 latency also increased due to mental fatigue and light drowsiness[11, 12].

Various mental stresses are encountered in the workplace, including stress caused by instinctive sensations (such as stress caused by noise exposure). Another type of mental stress is caused by mental work; this is peculiar to human beings and is caused by recognizing and processing advanced information (such as stress caused by computer-based work). These two types of stress have apparently different influences on brain information handling and underlying hemodynamic: the former is nonlinguistic and intuitive, primarily causing vascular activation to increase blood pressure (vascular stress), while the latter is linguistic and logical, primarily inducing cardiac activation to increase blood pressure (cardiac stress)[13–17]. Frequently, mental stresses that occur in the workplace are related to one another. We believe that physiological factors affected by each type of stress must be evaluated before the effects of combined stresses are discussed.

Manuk *et al*.[5] reported that differences in cardiovascular reactivity to stress can be reliably detected using experimental methods and that persons at risk for stress-related disorders can be identified from their exaggerated cardiovascular response profiles. A variety of experimental models of mental stress have been proposed and employed in the laboratory to examine the physiological responses caused by mental stress. The mental arithmetic (MA) task primarily causes a beta-adrenergic activation of the sympathetic nervous system characterized by an increase in CO, and it should be considered a cardiac stress[16, 18]. White noise (WN) exposure primarily causes an alpha-adrenergic activation of the sympathetic nervous system characterized by an increase in TPR and it should be considered a vascular stress[15, 17]. A previous study[2] reported that increased blood pressure was generally maintained throughout a 28-min MA task. It was demonstrated that CO rose during the first half of the task but returned to baseline levels during the last quarter of the task. Moreover, TPR increased as the task progressed. However, whether the underlying hemodynamic was changed by vascular stress was not discussed. In addition, whether the central nervous system responses to the two different types of stress were different is not clear.

In the present study, the goal is to compare the circulatory and central nervous system responses to cardiac and vascular stresses and discuss the effectiveness of physiological indices to evaluate the influences caused by different types of mental stress.

Methods

Participants

Eight healthy males participated in this study. The age, weight, height, and BMI of the participants were 23 ± 2 yr, 60 ± 6 kg, 170 ± 5 cm, and 21 ± 2, respectively. Participants were requested to refrain from exercise and alcohol intake on the night prior to the experiment and were prohibited from drinking caffeinated beverages or smoking during the 2-h period immediately preceding the experiment. This study was approved by the Ethics Committee of the Graduate School of Engineering at Chiba University. After the details of the study were explained, the participants were asked to sign written consent forms for their participation in the study.

Stresses

The mental addition task (MA) represented a computer-based mental stress. Two random two-digit numbers were displayed on a computer screen for 3 s. The subjects were instructed to add them together in their heads and type the result (a two-digit number) into the computer within 1.5 s using a 10-key pad. The results were evaluated electronically, and the Japanese word for "correct" or "wrong" was displayed on the screen for one second, after which a new trial started.

The white noise (WN) was produced by software commercially available from Macromedia, Inc. at a sound pressure level of 80 dB(A) and was provided through earphones. The participants were told to stay quiet and relax physically during the white-noise exposure periods.

Protocol and parameters

The participants performed the MA task and were exposed to the WN on the same day with a 10-min rest period between sessions. The participants were asked to be quiet for at least 30 min after entering the laboratory and before the recording sessions began. The

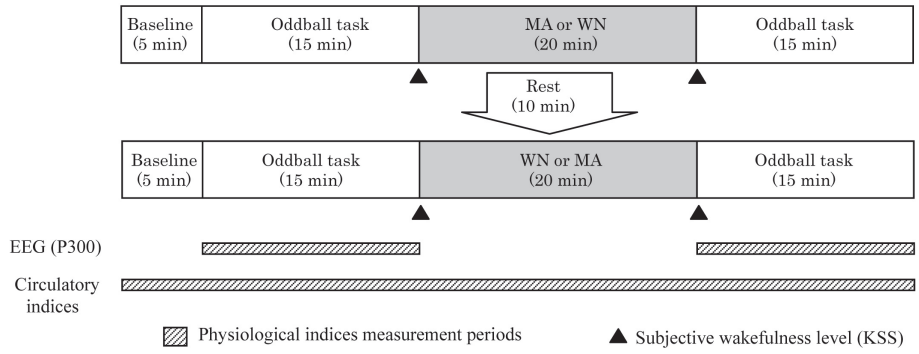

Fig. 1. Protocol of the experiment. Grey areas show the task (or exposure) period.
MA: mental arithmetic task; WN: white noise exposure.

experimental protocol consisted of a 5-min rest period (baseline), a 15-min odd ball task, a 20-min MA task (or WN exposure), and another 15-min oddball task (Fig. 1).

Electroencephalogram (EEG) and electrooculogram (EOG) activity was recorded before and after the MA (or WN) (Fig. 1). An oddball task, which was presented during the EEG recorded period to obtain P300, consisted of standard (1,000 Hz, 80%) and target (2,000 Hz, 20%) audio stimuli, generated by computer software commercially available from Macromedia, Inc. With an inter-stimulus interval of 3 s, the participants were instructed to press a button as quickly as possible once they heard the randomly sounded target tone. EEG was recorded at the Fz, Cz, Pz, F7, and F8 electrode sides of the international 10–20 system. The band-pass filter was set at 1.0–35 Hz (EEG100C, BIOPAC System, Inc.). EOG was recorded at the same time with EEG and electrodes were placed at the outer canthus and supraorbital to the left eye. The band-pass filter was set at 0.05–35 Hz. (EOG100C, BIOPAC System, Inc.).

Circulatory responses were recorded continuously throughout the experimental periods (Fig. 1). Systolic and diastolic blood pressure (SBP and DBP), mean arterial pressure (MAP), heart rate (HR), cardiac output (CO), stroke volume (SV), and total peripheral resistance (TPR) were measured using a noninvasive continuous blood pressure monitor (Portapress Model-2, Photal, Inc.). Left and right oxygenated hemoglobin concentration (O_2Hb, set at F7 and F8) were also measured continuously throughout the experimental session (NIRS-300, Hamamatsu Photonics, Inc.).

Before and after the MA task (or WN exposure), the subjective wakefulness level was evaluated using the Kwansei Gakuin Sleepiness Scale (KSS) questionnaire. This is a scale developed for the Japanese that is based on the Stanford Sleepiness Scale (SSS) devised by Hoddes in 1972. It consists of 22 items. The mean core to 0 indicates a high wakefulness level and a score close to 7 indicates a low wakefulness level[19].

Data analyses

The EEG was digitized at 1 ms/point for 600 ms, with a pre-stimulus baseline of 100 ms. The P300 amplitude was measured relative to the pre-stimulus baseline and was defined as the largest positive-going peal occurring after the N100-P200-N200 complex, within a latency window between 250 ms and 500 ms. At least 30 artifact-free target presentations were obtained for each frequency session. The measurement period included pre-stress and post-stress that was measured before and after each stress, respectively.

The mean for the cardiovascular indices and O_2Hb were calculated for every minute of all experimental periods and then the respective mean baseline value was subtracted from these means to determine the response tendency. The stress period was divided into the first half of the stress period (Fir: 1–10 min) and the latter half of the stress period (Lat: 11–20 min). Changes (Δ) calculated by subtracting the respective baseline values from the average value for the task (or exposure) period were used to conduct the statistical analysis.

Two-way repeated ANOVAs (stress type × measurement period) were conducted, and measures of effect size (partial η^2) and power were also reported. Paired *t*-tests were conducted as sub-analyses when the interaction was significant and to compare the baselines before exposure to the stresses. The level of significance was set at $p<0.05$. Statistical analysis was carried out using the IBM SPSS Statistics 19 (IBM Corp.).

Results

Circulatory responses

There were no significant differences between the baseline values before MA and WN (Table 1). The main effects of the measurement period were significant for ΔSBP ($F(1, 7)=21.57$, $p<0.01$, partial $\eta^2=0.76$, power=0.98), ΔDBP ($F(1, 7)=17.21$, $p<0.01$, partial $\eta^2=0.71$, power=0.94), and ΔMAP ($F(1, 7)=33.81$,

Table 1. Baseline values of cardiovascular indices before mental arithmetic (MA) task and white noise (WN) exposure (n=8)

	MA task (Baseline)		WN exposure (Baseline)		*t*	*p*
	mean	SD	mean	SD		
SBP (mmHg)	110.36	6.86	113.87	7.78	–1.39	0.21
DBP (mmHg)	68.60	4.31	69.92	5.90	–0.72	0.49
MAP (mmHg)	86.23	4.75	88.57	5.78	–1.12	0.30
HR (bpm)	65.31	5.62	65.02	6.18	0.19	0.86
SV (ml)	78.16	7.99	82.85	14.22	–1.26	0.25
CO (l/min)	5.07	0.61	5.33	0.76	–1.20	0.27
TPR (MU)	1.04	0.12	1.02	0.17	0.36	0.73

MA: mental arithmetic task; WN: white noise exposure. SBP: systolic blood pressure; DBP: diastolic blood pressure; MAP: mean arterial pressure; HR: heart rate; CO: cardiac output; SV: stroke volume; TPR: total peripheral resistance.

Table 2. Changes (Δ) of the circulatory responses during the first half (Fir) and the latter half (Lat) of the stress periods (n=8)

	MA Task		WN exposure	
	Fir	Lat	Fir	Lat
ΔSBP (mmHg)[a]	10.58 (5.79)	12.82 (5.26)**	6.36 (6.13)	8.53 (6.25)**
ΔDBP (mmHg)[a]	7.99 (3.87)	8.76 (4.21)**	4.14 (5.11)	5.77 (4.38)**
ΔMAP (mmHg)[a]	9.06 (4.08)	10.6 (4.19)**	4.85 (5.75)	6.70 (5.27)**
ΔHR (bpm)[b]	2.57 (3.16)*	1.83 (2.61)*	–0.47 (1.69)	–0.24 (1.90)
ΔSV (ml)	–1.35 (5.94)	1.02 (7.06)	–0.01 (3.59)	–1.15 (4.95)
ΔCO (l/min)	0.14 (0.42)	0.24 (0.48)	–0.04 (0.26)	–0.10 (0.32)
ΔTPR (MU)	0.09 (0.11)	0.09 (0.11)	0.06 (0.10)	0.09 (0.08)
ΔO_2Hb-L (μmol)[c]	3.31 (1.94)	3.24 (1.90)	1.14 (1.33)	1.36 (1.27)
ΔO_2Hb-R (μmol)[c]	2.56 (2.06)	2.25 (2.37)	0.55 (1.29)	0.74 (2.27)

Values are mean (SD). MA: mental arithmetic task; WN: white noise exposure. SBP: systolic blood pressure; DBP: diastolic blood pressure; MAP: mean arterial pressure; HR: heart rate; CO: cardiac output; SV: stroke volume; TPR: total peripheral resistance; O_2Hb: oxygenated hemoglobin concentration, L: left side; R: right side. **p<0.01; *p<0.05.

[a]The latter half parts were significantly higher than that of the first half parts (p<0.01).

[b]The changes to MA task were significantly higher than that of WN exposure (p<0.05).

[c]The changes to MA task were higher than that of WN exposure (p<0.1).

p<0.01, partial η^2=0.83, power=0.99); the latter halves were significantly higher than those of the first halves (Table 2), and the interactions between factors were not significant. In addition, the main effect of stress type for ΔHR was significant, ΔHR during MA was significantly higher than that which occurred during WN exposure (F(1, 7)=9.53, p<0.05, partial η^2=0.58, power=0.75), and the interaction between factors was not significant. The change tendencies for blood pressure and heart rate responses during stress periods are shown in Fig. 2. No significant results were found for ΔSV, ΔCO and ΔTPR.

The change tendencies for O_2Hb during stress periods are shown in Fig. 3. The left (L) and right (R) prefrontal ΔO_2Hb clearly and stably increased during the MA task but did not clearly change during WN exposure. The results of ANOVA showed that the ΔO_2Hb during the MA task was higher than that of the WN exposure on both sides (p<0.1, Table 2), but the main effects and the interaction between them were not significant. There was no significant difference between the first half and the latter half for both stresses.

Central nervous system responses

The results of the P300 amplitude and latency are shown in Table 3. At F7, the P300 amplitudes after stress were higher than those that occurred before exposure to the stresses (p<0.1, Table 3).

For the P300 latency, the main effect of the measurement period was significant at Fz, the P300 latency was significantly lower after stress exposure (F(1, 6)=9.57, p<0.05, partial η^2=0.62, power=0.73). The interaction was significant at F7 (F(1, 6)=23.17, p<0.01, partial η^2=0.79, power=0.98). The sub-analyses showed that

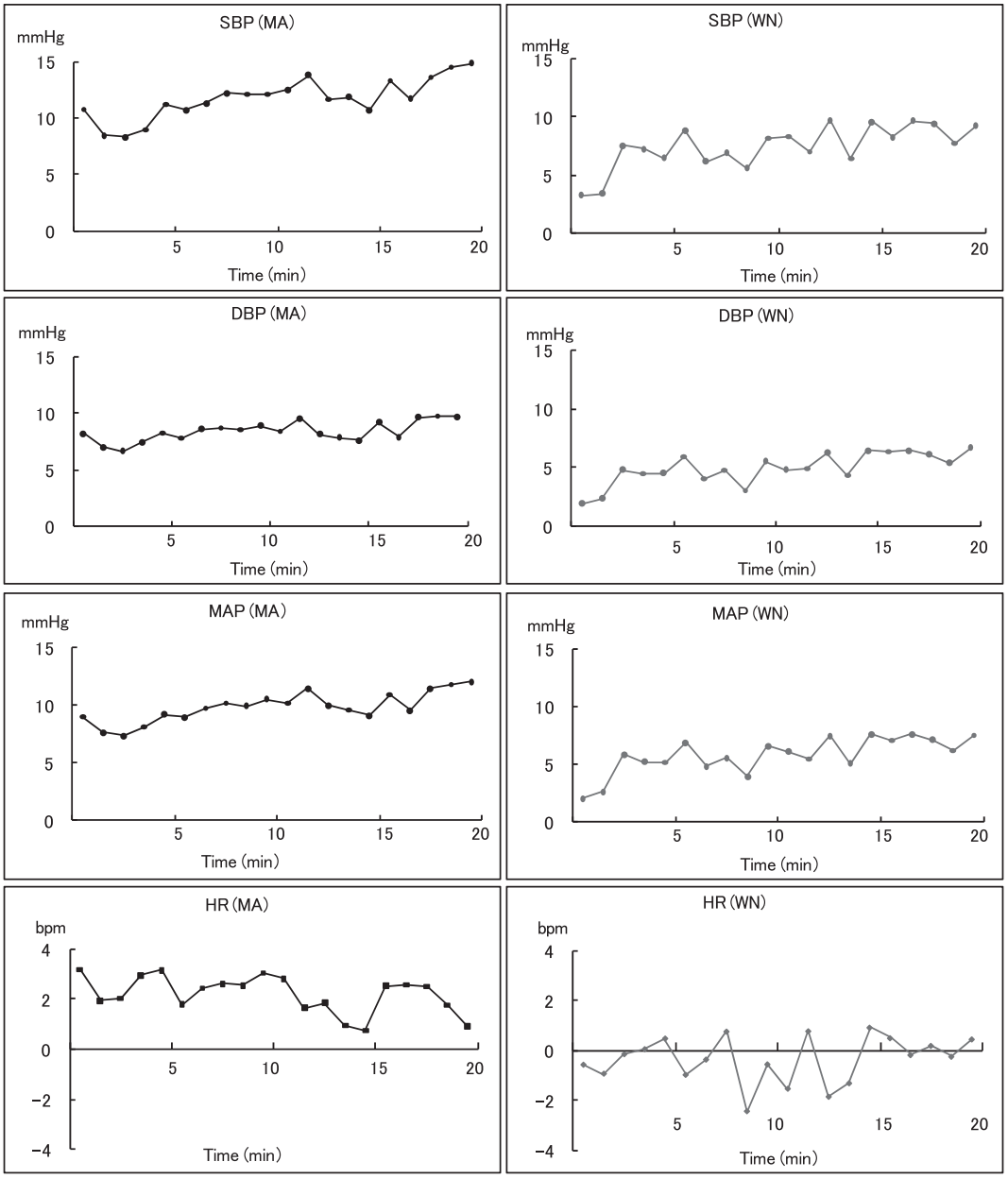

Fig. 2. The change tendency in systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), and heart rate (HR) from baseline during stress periods (n=8).
MA: mental arithmetic task; WN: white noise exposure.

P300 latency at F7 significantly decreased with WN exposure (t=3.32, p<0.05, Fig. 4).

Subjective wakefulness level

The KSS core was not significantly different before the MA task and before the WN exposure. The results of ANOVA showed a significant interaction between the stress type factor and the measurement period factor (F(1, 6)=12.91, p<0.05, partial η^2=0.68, power=0.85). The sub-analyses showed that the KSS score after the MA task was significantly lower than before the task (t=3.73, p<0.01), but there was no significant difference before and after WN exposure (Fig. 5).

Discussion

The present study showed that blood pressure had a cumulative increase with both types of stress, and the underlying hemodynamic to increased blood pressure did not change with the extension of exposure. The results differ from an aforementioned study[2], which reported that, to enhance blood pressure, the underly-

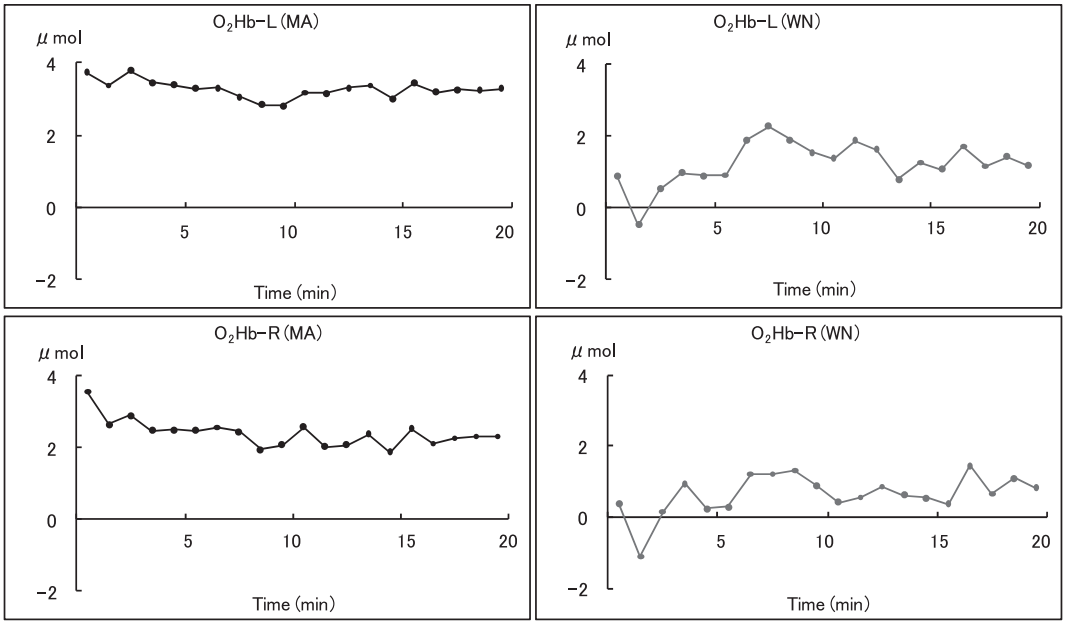

Fig. 3. Changes in oxygenated hemoglobin concentration (O_2Hb) from baseline during stress periods (n=8).
L: left side; R: right side; MA: mental arithmetic task; WN: white noise exposure.

Table 3. The P300 amplitude and the latency before (Pre) and after (Post) stress periods (n=7)

	MA Task		WN exposure	
	Pre	Post	Pre	Post
Amplitude (μv)				
Fz	6.08 (3.56)	7.57 (2.66)	6.33 (7.33)	8.56 (2.23)
Cz	10.34 (3.80)	11.50 (3.73)	10.96 (8.42)	13.25 (4.12)
Pz	11.67 (3.46)	11.68 (3.53)	12.28 (7.08)	13.33 (4.27)
F7[a]	2.71 (3.96)	4.69 (2.79)	2.95 (4.69)	5.19 (2.38)
F8	3.49 (3.10)	5.14 (2.02)	3.57 (6.19)	5.96 (2.06)
Latency (ms)				
Fz[b]	340.86 (16.81)	330.29 (9.76)*	335.43 (13.60)	327.71 (20.54)*
Cz	335.14 (26.85)	331.14 (26.53)	329.71 (22.04)	319.14 (27.27)
Pz	331.43 (30.04)	339.71 (35.73)	327.43 (24.19)	323.71 (24.86)
F7[c]	333.14 (31.24)	338.57 (31.11)	333.71 (23.54)	299.43 (34.05) *
F8	336.00 (36.24)	331.29 (24.25)	330.29 (31.63)	320.86 (26.85)

Values are mean (SD). MA: mental arithmetic task; WN: white noise exposure. *p<0.05.
[a]The amplitude after stresses were higher than that of before the stresses (p<0.1).
[b]The latency after stresses were significantly higher than that of before the stresses (p<0.05).
[c]The interaction was significant (p<0.01). The P300 latency significantly decreased by WN exposure (p<0.05).

ing hemodynamic changed during a 28-min MA task. The reason can be attributed partly due to the different presentations of the tasks: the mental task was in an auditory medium, and the participants were required to say the answer aloud; this method caused a sustained increase in HR while SV decreased continuously. The present study, however, displayed the MA task on a computer screen, and the participants typed in the response using a 10-key pad. We think that the intervention of language may partly influence the cardiac response pattern. It is known that MA tasks primarily cause cardiac activation to increase blood pressure but WN exposure primarily causes vascular activation to increase blood pressure[13–17]. In the present study, HR increased during the MA task but did not show a clear change in response to WN exposure, although increased blood pressure was not significantly different between the two types of stress. These results suggested that the

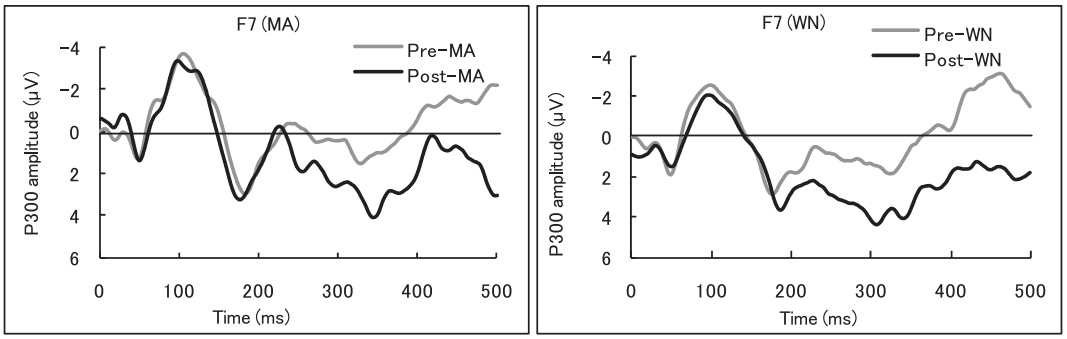

Fig. 4. P300 component elicited by audio target stimuli before (Pre) and after (Post) stresses.
MA: mental arithmetic task; WN: white noise exposure.

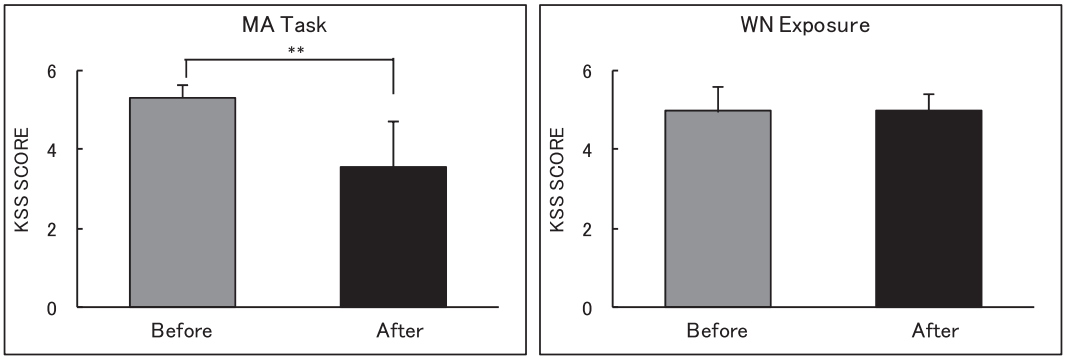

Fig. 5. Subjective wakefulness level evaluation (KSS SCORE) before and after stresses.
MA: mental arithmetic task; WN: white noise exposure. **: $p<0.01$.

underlying hemodynamic to increase blood pressure is different between the two types of stress and that prolonged exposure to these types of stress may cause a cumulative cardiovascular load.

An 18-yr follow-up study[20] reported that blood pressure showed good reproducibility to a MA task (a cardiac stress) and a cold pressure test (a vascular stress) and that people who showed exaggerated responses to stresses may have a higher risk for cardiovascular disease. The means of reducing cardiovascular responses to stresses may be a link to reducing stress-related disorders. In our previous study[15], participants were presented with four 5-min MA tasks separated by 3-min break periods and were exposed to WN with the same protocol on another day. The results indicated that the increase in blood pressure remained at the same level during the MA task periods but showed an accumulation effect in the blood pressure response during WN exposure. In addition, blood pressure increased during the MA task periods but decreased immediately during the 3-min break after each task period, although their values did not return to the baseline. On the contrary, the effect of the breaks was not clear with respect to blood pressure during WN exposure. In the present study, we presented MA or WN for a continuous 20-min period without breaks and the results showed that the elevated blood pressure increased continuously throughout the exposure period. The difference between the two studies suggests that breaks from a continuous cardiac stress may avoid further increases in blood pressure. We believe that adequate breaks during mental work, which primarily caused cardiac responses, may mitigate blood pressure increases, and that the timing of breaks should differ according to the type of stress.

The P300 amplitude is believed to be related to the amount of cognitive resources available for the evaluation of stimuli and allocation of attention, and P300 latency is thought to be related to the time required to categorize and evaluate stimuli[11, 21, 22]. Previous studies have reported that P300 amplitude and latency influenced by arousal and increase in P300 latency suggest mental fatigue[11, 23, 24]. In the present study, the MA task increased the P300 amplitude at F7. The MA task is considered an active stress, and the participants required more cognitive resources to maintain their work performance, resulting in mental fatigue characterized by increases in P300 amplitude. The change in prefrontal O_2Hb and the subjective wakefulness level

(KSS) showed that the MA task require a greater oxygen supply to the brain and that subjective wakefulness increased from stress exposure. On the contrary, WN exposure increased P300 amplitude but decreased P300 latency at F7. Some previous studies have suggested that the frontal EEG (especially at F7 and F8) predicted affective responses[25–28]. WN exposure is considered a positive stress, and the participants did not require cognitive resources to perform any task; however, with the extension of stress exposure, affective responses (such as unhappiness) may be elicited and result in an increase in amplitude but a decrease in latency. In addition, the change in prefrontal O_2Hb and subjective wakefulness level did not change clearly during WN exposure. The results of the present study suggest that the central nervous system responses to different types of mental stress may have different mechanisms, especially in the left prefrontal, and the measurement of P300 is effective in evaluating the influence on cognitive function caused by various stresses in the workplace.

There are several limitations that should be acknowledged in this study. First, the sample size was small (n=8), although the statistical analyses showed a suitable effect size and power. It is necessary to add more participants in order to verify the underlying mechanism of responses. Second, we discussed two types of stress in the present study, but it is not enough because the stresses are frequently related to each other when they occur in workplaces and the combined stresses should also be discussed. Thus, further investigations are needed including a larger sample size and more stresses in the future.

Conclusion

Different types of stress caused the circulatory and central nervous system responses. Cardiac stress caused a cumulative effect on blood pressure as the exposure period is extended, and participants require more cognitive resources and a greater oxygen supply to the brain. Further, central nervous fatigue may be elicited by performing the mental work. Vascular stress, however, showed a cumulative effect on blood pressure but may have little influence on the central nervous system during acute and short-term exposure. We believe physiological indices offer an effective means of evaluating the influences of various stresses encountered in the workplace, and the reduction of harm influence may differ according to the type of stress.

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