SHORT COMMUNICATION

Interactive effects between isometric exercise and mental stress on the vascular responses in glabrous and nonglabrous skin

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Abstract Cutaneous vascular responses to mental arithmetic (MA) and handgrip exercise (HG) were studied independently and combined at different local skin temperatures ($T_{\rm loc}$). MA and HG induced (P < 0.05) vasoconstrictor responses in glabrous and nonglabrous skin at a higher level of $T_{\rm loc}$, resulting in a nonadditive effect of these two stresses.

Keywords Skin blood flow · Local warming · Local cooling

Introduction

In everyday life and work, we frequently experience a combination of physical and mental stress. These stresses influence vasomotor control in various organs including viscera and muscle via the sympathetic nervous system [1–3]. Vasomotion in the skin is one of the targets for these stimuli. That is, skin vasomotor control is influenced by some non-thermal factors including mental excitation and mechanochemical stimuli during exercise. Although the skin vascular responses to either physical or mental stress alone have been extensively examined, information regarding skin vascular control during concurrent combination of physical

and mental stress is limited. It is unknown whether these two stressors independently or synergistically elicit skin vascular responses. To further understand the impact of non-thermal factors on peripheral vasomotion, the interaction of these stresses in skin vasomotor control should be examined.

Skin vascular responses to physical and mental stress depend on temperature conditions [4–6]. Elam and Wallin [6] reported that in warm conditions with high baseline blood flow, mental stress led to vasoconstriction in the hands and feet, while in cool conditions with low baseline blood flow, it led to vasodilatation. Thus, when the interactive effects of physical and mental stress in the vasomotor controlling systems in human skin are examined, we should consider the pre-stress baseline levels in skin blood flow (SkBF) that change with temperature. Furthermore, skin vascular responses to physical and mental stress are not always similar between glabrous regions, such as the palm and sole, and nonglabrous regions, such as the forearm and dorsal hand [7–11].

In the present study, therefore, we examined the vascular responses in glabrous and nonglabrous skin to mental or physical stress and combined stresses at different levels of SkBF. We hypothesized that combined mental and physical stresses produce a synergistic interaction in the vascular responses at any level of SkBF.

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Methods

Subjects

Nine male volunteers participated in the experiments. Their average age was 22 ± 0 (SE) years, average weight was 66 ± 3 kg, and average height was 171 ± 2 cm. The



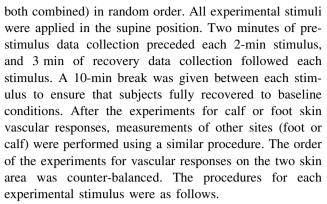
maximal voluntary handgrip strength was 50.4 ± 2.6 kg. All subjects were healthy nonsmokers with no history of cardiovascular disease. The Institutional Review Board of the University of Yamaguchi approved the experimental protocol and written informed consent was obtained after a thorough explanation of the present study, including its purpose and risks.

Measurements

SkBF was monitored continuously with laser-Doppler flow meters (ALF21, Advance, Tokyo, Japan). Three blood flow probes were applied to the left calf or left foot, and the distance between probes was approximately 5 cm in each region. The local temperature (T_{loc}) of the 6.3 cm² area surrounding the site of SkBF measurement was controlled by a metal sleeve for the flow probe that had both a heating element and a Peltier cooling element. As an index of core temperature, esophageal temperature (T_{es}) was measured with a polyethylene-sealed thermocouple swallowed via the mouth to 36–42 cm down the esophagus. The $T_{\rm es}$ probe was located at the highest temperature. Skin temperature $(T_{\rm sk})$ was measured by copper-constantan thermocouples placed at four skin sites (chest, upper arm, thigh, calf) using surgical tape and the mean $T_{\rm sk}$ was calculated using the weighting factors of Ramanathan [12]. Heart rate was determined by electrocardiogram (CM5 lead) using a telemetric device (Bioview 1000, NEC, Tokyo, Japan). Mean arterial pressure (MAP) was measured continuously by arterial tonometry (JENTOW-7700, Colin, Komaki, Japan) on the left radial artery.

Experimental procedures and protocols

The subjects arrived in the laboratory after having abstained from caffeine and alcohol for at least 1 day and from food for at least 2 h. All experiments were conducted in an environmental chamber maintained at an ambient temperature of 28°C and a humidity of 50%. After entering the chamber, each subject rested in the supine position for ~ 60 min. During this period, the subjects performed maximum voluntary handgrip contractions (MVC) using their dominant hand (i.e. right hand) and were then equipped with sensors and probes. T_{loc} at three sites was maintained at 34°C. After baseline measurements were obtained for the last 5 min while the subject rested under normothermic conditions, T_{loc} was simultaneously changed to 29°C (cooling) at one site, 39°C (warming) at another site, or 34°C (neutral, not changed) at the remaining site. This phase of experiments was continued for 30-35 min to obtain steady SkBF levels. The subjects then underwent each experimental stimulus (static handgrip exercise (HG), mental arithmetic (MA) or



For exercise stimulus, subjects performed 2 min of static HG at 30% MVC. Since short-term static exercise does not change $T_{\rm es}$ or mean $T_{\rm sk}$, the effect of thermal factors for modulating skin vasomotion can be minimized. An electronic handgrip dynamometer (EG-220, Sakai Medical, Tokyo, Japan) provided grip-force input to our data acquisition system. The subject controlled handgrip strength within 1 kg of their target level by watching the device monitor. All subjects used their right hands for the static HG. For mental stimulus, subjects performed 2 min of MA aloud, consisting of subtraction of two-digit integers from four-digit integers and addition of two-digit integers and four-digit integers. The substraction and addition questions were mutually set every 12 s. Subjects were made aware of any mistakes by a buzzer. For combined stimuli of exercise and MA, subjects simultaneously performed both the challenges described above. MA started as soon as the static handgrip force stabilized at a subject's target level.

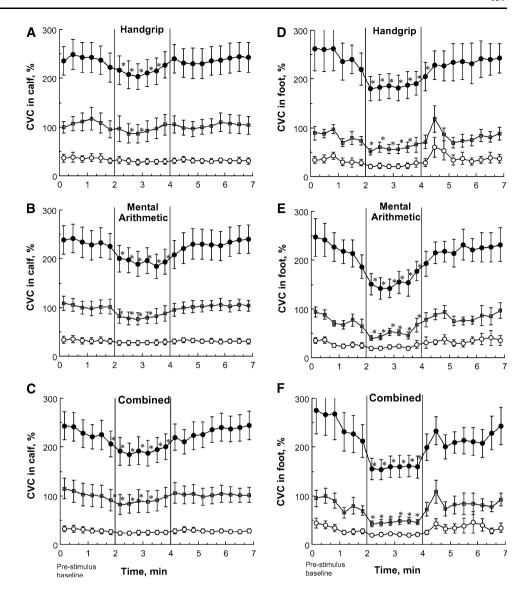
Data acquisition and analysis

The measured variables were recorded by a data acquisition system (PowerLab/16sp, ADInstruments, Colorado Springs, CO, USA) every 1 s and averaged every 20 s. Cutaneous vascular conductance (CVC) was calculated from the ratio of SkBF to MAP. Data averaged over the 5 min just before the beginning of local warming and cooling were used as baseline control values. Changes in CVC were also expressed as percent changes from the prestimulus baseline values before the experimental stimuli (first minute of pre-stimulus data).

Effects of experimental stimuli and time on changes on each variable were evaluated using two-way repeated measures ANOVA, followed by Contrasts when a significant difference was detected. Effects of $T_{\rm loc}$ or experimental stimuli on changes in CVC were evaluated using one-way ANOVA followed by Fisher's PLSD tests. Paired t tests were used to compare the changes in CVC in the calf and foot. Statistical significance was set at P < 0.05. Data are presented as means \pm SE.



Fig. 1 Changes in cutaneous vascular conductance (CVC) in the calf $(\mathbf{a}-\mathbf{c})$ and foot $(\mathbf{d}-\mathbf{f})$ during 2 min of physical or mental stress and the two combined stresses. The CVC were expressed as a percentage from the baseline values before the beginning of local warming and cooling. Open circles, squares, and closed circles show data in the 29, 34 and 39°C conditions, respectively. *P < 0.05 versus pre-stimulus baseline (two-way ANOVA and Contrast)



Results

Figure 1 shows the CVC responses in the calf and foot to HG, MA and combined stimuli. Local warming significantly increased baseline CVC in the calf and foot and local cooling significantly decreased them. The baseline CVC values in each T_{loc} did not differ among the experimental conditions, and the average values were 239 \pm 1% in calf and 256 \pm 6% in foot at 39°C, 106 \pm 1% in calf and 90 \pm 2% in foot at 34°C, and 30 \pm 1% in calf and $36 \pm 1\%$ in foot at 29°C. Local warming and cooling did not change MAP, $T_{\rm es}$ and $T_{\rm sk}$. The three experimental stimuli decreased (P < 0.01, two-way ANOVA) CVC in the calf and foot at 39°C and 34°C, but not at 24°C for T_{loc} . $T_{\rm es}$ and $T_{\rm sk}$ did not change during each stimulus. Each stimulus increased (P < 0.05, two-way ANOVA) MAP from baseline levels (the average value; $75.7 \pm$ 1.5 mmHg). The sum ($\Delta 17.2 \pm 1.2$ mmHg) of increases in MAP during HG ($\Delta 13.0 \pm 1.1$ mmHg) and MA ($\Delta 4.2 \pm 0.9$ mmHg) was greater (P < 0.01, one-way ANOVA) than that during the combined stimulus ($\Delta 11.5 \pm 1.0$ mmHg).

The reductions in CVC to the three experimental stimuli are presented in Fig. 2 as the changes from the pre-stimulus baseline value. In all experimental conditions, the reductions in CVC in the calf and foot increased (P < 0.01, one-way ANOVA) with increasing $T_{\rm loc}$. However, when CVC was expressed as a percentage change from the pre-stimulus baseline value (Fig. 3), the reductions in CVC during each stimulus did not differ (P > 0.32, one-way ANOVA) among the temperature conditions. Regardless of CVC expressions, the reductions in CVC in each temperature condition did not differ (P > 0.41, one-way ANOVA) among the experimental conditions (Figs. 2 and 3). In both expressions for CVC (Figs. 2 and 3), the sum of CVC reductions during HG and MA were greater (P < 0.05,



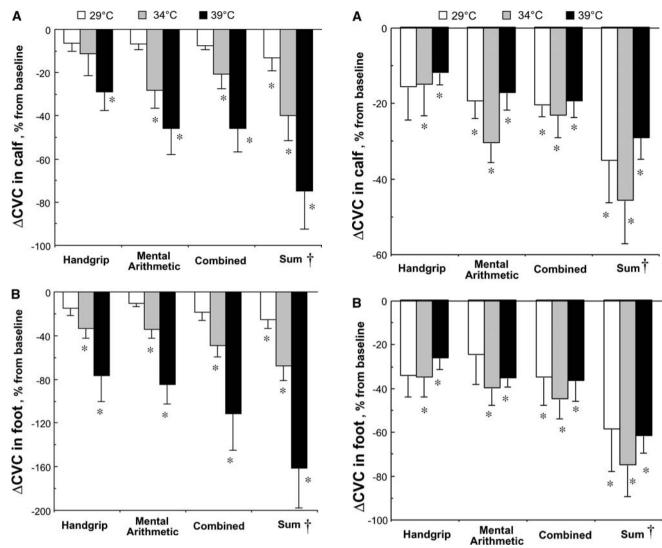


Fig. 2 Delta changes in CVC in the calf (a) and foot (b) for the three stimuli and the sum of the handgrip and mental arithmetic stimuli. The CVC were expressed as a percentage from the baseline values before the beginning of local warming and cooling. *P < 0.05 versus pre-stimulus baseline (one-way ANOVA). $^{\dagger}P < 0.05$ versus combined (one-way ANOVA)

one-way ANOVA) than those during the combined stimulus. Additionally, the reductions in CVC in the foot during experimental stimuli were greater (P < 0.05) than those in the calf for each temperature condition (Figs. 2 and 3).

Discussion

The major findings of this study are as follow. First, the reductions in CVC in the calf and foot during HG, MA, and the combination of both were not different among the stimuli within the local temperature range of 29–39°C. Second, the sum of reductions in CVC during HG and MA was greater than that during the combination stimulus.

Fig. 3 Delta changes in CVC in the calf (a) and foot (b) for the three stimuli and the sum of the handgrip and mental arithmetic stimuli. The CVC were expressed as a percentage from the pre-stimulus baseline values. *P < 0.05 versus pre-stimulus baseline (one-way ANOVA). †P < 0.05 versus combined (one-way ANOVA)

These findings suggest that physical and mental stress do not interact in an additive manner in generating vasoconstrictor responses in glabrous and nonglabrous skin.

Skin vascular response during physical exercise depends on thermal factors (e.g. core and skin temperatures) and non-thermal factors (e.g. central command, afferent signals from muscle mechanoreceptors and metaboreceptors) associated with muscle work [13–15]. In this study, since $T_{\rm es}$ and mean $T_{\rm sk}$ were not altered by each stimulus, and $T_{\rm loc}$ at the measuring sites of SkBF was maintained during stimuli, the exercise-induced vasoconstrictor response was due to non-thermal factors caused by the static HG. A greater vasoconstrictor response in foot skin to exercise than in calf skin was consistent with previous reports [7, 11]. Since pharmacological blockade of adrenergic



nerves achieved by iontophoretically applying bretylium tosylate to glabrous skin diminished the vasoconstrictor response at the onset of cycle exercise [9], the vasoconstriction must originate from noradrenergic sympathetic activation. Saad et al. [7] suggested that the marked constriction in anastomotic vessels to sympathetic nerve activity is responsible for the greater vasoconstrictor response in glabrous skin during isometric exercise. Moreover, the difference in the magnitude of vasoconstrictor responses between foot and calf skin is, partly at least, explained by the difference in the baseline SkBF at the two sites.

Regarding the vascular response to mental stress, Halliwill et al. [2] reported that forearm blood flow increased with decreasing muscle sympathetic nerve activity in radial nerves during a Stroop word-colour conflict test in normothermia. They also observed a significant increase in forearm SkBF during mental stress. However, MA or arousal stimuli increased sympathetic nerve activity in supraorbital nerve followed by increases in SkBF and sweating activity in the forehead during normothermia and body heating [16]. In the present study, we found that CVC in the calf and foot decreased during MA at 34 and 39°C. The conflict results in the vascular responses during mental stress may be due to the regional differences and the different mental tests. In addition to the differences in experimental conditions, our results show that the skin vascular responses during physical and mental stress are altered by the T_{loc} because no significant vasoconstrictor responses were observed at the sites at 29°C (Fig. 2). Previous reports [6, 17] showed that a number of stimuli containing mental stress led to skin vasoconstriction in glabrous sites in the hands and feet if the subjects were warm (skin temperature $> 30^{\circ}$ C). If the subjects were cold (skin temperature < 25°C), the same stimuli evoked vasodilation. Our results are consistent with the findings obtained in warm conditions in previous reports [6, 17]. We observed in an other study that subjects with a lower foot temperature (< 29°C) showed skin vasodilation in the foot during HG or while doing MA [unpublished data]. Therefore, more severe local cooling ($T_{loc} < 29$ °C) markedly decreases baseline SkBF and may induce skin vasodilation. During combined stress, signals transmitted from the cortex in response to mental stress interact with other central and peripheral signals associated with exercise at the brainstem level. Furthermore, skin vascular responses were influenced by T_{loc} , suggesting that the vascular responses to mental and physical stress may also be modified at the peripheral level, but the detailed mechanisms remain unclear.

The role of baseline SkBF should be considered when evaluating cutaneous vasoconstrictor responses to physiological stress such as skin cooling [18, 19]. In this study, local heating increased baseline SkBF by 240–260% and

local cooling decreased it by 60–70%. When CVC was expressed as a percentage change from the pre-stimulus baseline value (Fig. 3), the skin vasoconstrictor responses did not differ among the local temperature conditions. These findings suggest that the magnitude of skin vasoconstrictor responses to exercise and/or mental stress depends on the baseline SkBF in both glabrous and nonglabrous sites. The reduction in baseline SkBF may act to diminish a significant vasoconstrictor response to exercise and mental stress in human skin.

In conclusion, the present results indicate that HG and MA stimuli do not interact in an additive manner in vasomotor control in glabrous and nonglabrous skin. The skin vascular responses to these stimuli are dependent on the local skin temperature.

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