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Effects of face/head and whole body cooling during passive heat stress on human somatosensory processing

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¹Department of Health Sciences, Faculty of Human Life and Environment, Nara Women’s University, Nara, Japan; ²Graduate School of Humanities and Sciences, Nara Women’s University, Nara, Japan; and ³Department of Integrative Physiology, National Institute for Physiological Sciences, Okazaki, Japan

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Nakata H, Namba M, Kakigi R, Shibasaki M. Effects of face/head and whole body cooling during passive heat stress on human somatosensory processing. Am J Physiol Regul Integr Comp Physiol 312: R996–R1003, 2017. First published April 12, 2017; doi:10.1152/ajpregu.00039.2017.—We herein investigated the effects of face/head and whole body cooling during passive heat stress on human somatosensory processing recorded by somatosensory-evoked potentials (SEPs) at C4’ and Fz electrodes. Fourteen healthy subjects received a median nerve stimulation at the left wrist. SEPs were recorded at normothermic baseline (Rest), when esophageal temperature had increased by ~1.2°C (heat stress: HS) during passive heating, face/head cooling during passive heating (face/head cooling: FHC), and after HS (whole body cooling: WBC). The latencies and amplitudes of P14, N20, P25, N35, P45, and N60 at C4’ and P14, N18, P22, and N30 at Fz were evaluated. Latency indicated speed of the subcortical and cortical somatosensory processing, while amplitude reflected the strength of neural activity. Blood flow in the internal and common carotid arteries (ICA and CCA, respectively) and psychological comfort were recorded in each session. Increases in esophageal temperature due to HS significantly decreased the amplitude of N60, psychological comfort, and ICA blood flow in the HS session, and also shortened the latencies of SEPs (all, P < 0.05). While esophageal temperature remained elevated, FHC recovered the peak amplitude of N60, psychological comfort, and ICA blood flow toward preheat baseline levels as well as WBC. However, the latencies of SEPs did not recover in the FHC and WBC sessions. These results suggest that impaired neural activity in cortical somatosensory processing during passive HS was recovered by FHC, whereas conduction velocity in the ascending somatosensory input was accelerated by increases in body temperature.

hyperthermia; somatosensory-evoked potentials; conduction velocity; central fatigue; tactile

Somatosensory-evoked potentials (SEPs), obtained by time-locked averaging electroencephalography (EEG) with high temporal resolution, have been used to evaluate cortical and subcortical somatosensory processing. SEPs are elicited by stimulating peripheral nerves, such as the median nerve at the wrist or posterior tibial nerve at the ankle. When the median nerve is stimulated, the latencies and amplitudes of the P14, N20, P25, N35, P45, and N60 components are recorded at centrotemporal electrodes contralateral to the stimulated site. N20 is the primary response from Brodmann’s area 3b of the primary somatosensory cortex, and subsequent components recorded at ~20–60 ms are generated in areas 3b, 1, and 4 (1, 2, 13). The P14 component is recorded just before N20 and is generated from higher segments of the cervical cord (31) and at or near the foramen magnum (10). The P22 and N30 components are recorded at the frontal electrodes and are generated from the primary motor cortex, premotor area, and prefrontal cortex (9, 16, 35). Our previous study using median nerve stimulation demonstrated that the peak latencies of some SEP components were shortened, but these peak amplitudes were reduced in subjects with increases in body temperature (24). We also reported that the conduction velocity of the ascending somatosensory input was accelerated by increases in body temperature, and aerobic exercise did not alter the strength of neural activity in cortical somatosensory processing (25). However, the mechanisms underlying alterations in ascending central processing due to HS currently remain unknown.

To establish a preventable methodology for hyperthermia-induced fatigue, recent studies examined the effects of face cooling during exercise and in a hot environment. They showed that face cooling prolonged workout performance, maintained the prolactin response, and reduced the ratings of perceived exertion and thermal sensation (4, 20, 21, 32, 33). Moreover, maintained cerebral perfusion may be a key factor because cerebral perfusion and oxygenation decrease during prolonged exercise with passive HS (30), resulting in central fatigue. Passive HS decreases cerebral blood flow (6, 11, 28, 38) but increases extracranial blood flow (i.e., facial skin blood flow) for heat dissipation (6, 28). Therefore, the recovery of cerebral perfusion due to extracranial cooling may contribute to clarifying the mechanisms underlying alterations in ascending central processing during HS. To the best of our knowledge, however, the effects of face/head cooling (FHC) have not yet been investigated on cerebral perfusion. Alternatively, increases in the internal temperature have been shown to alter...
ascending central processing. On the basis of these findings, the aim of the present study was to examine the effects of FHC and whole body cooling (WBC) during passive HS by using psychological, physiological, and neuroscientific methods. We utilized SEPs as an index of neural activity in somatosensory (ascending) processing, and hypothesized that neural activity recovers during FHC and/or WBC with improvements in psychological comfort and cerebral perfusion.

**METHODS**

**Subjects.** Fourteen male subjects (mean age 21.1 yr, range 20–24 yr) participated in this study. None of subjects had a history of a neurological or psychiatric disorder. The procedures used complied with the Declaration of Helsinki regarding human experimentation, and the study was approved by the Ethics Committee of Nara Women’s University, Nara, Japan. All subjects gave their written informed consent to participate in the study.

**Procedure.** Experiments were performed in a temperature-controlled laboratory at 26°C. On arrival at the laboratory, subjects weighed themselves nude on a scale, and then only wore underwear and short pants. Each subject inserted a copper-constantan thermocouple via the nasal passage to a distance equivalent to one-fourth of the subject’s height to measure esophageal temperature. External canal temperatures were attached during the equilibration period. Subsequent, EEG electrodes were placed on the scalp and earlobes, while except for the head, face, hands, and feet. During this equilibrium period, water (25°C) was then immediately perfused through the suit to decrease mean skin temperatures. When the esophageal temperature was 0.5°C higher than the preheat stress baseline, the fourth SEP was recorded (WBC session). Face/head cooling did not continue during WBC (Fig. 1).

To record SEPs, the electric stimulus used was a constant current square-wave pulse delivered to the left median nerve at a rate of 3 Hz (22–24). The stimulus duration was 0.2 ms, and stimulus intensity was sufficient to produce a slight but definite twitch of the thumb. Subjects were instructed to keep their eyes open and look at a small fixation point positioned in front of them at a distance of ~1.5 m. Two hundred stimuli were applied in each session, and the length of the recording time was ~80 s in each session. The starting time of the session was 63.0 ± 9.8 (SD) min in HS, 76.5 ± 10.1 min in FHC, and 93.4 ± 10.4 min in WBC.

**Hemodynamic and thermoregulatory variables.** Heart rate was obtained from an electrocardiogram (Bio multin 1000, NEC, Tokyo, Japan) and intermittent arterial blood pressure by auscultation of the brachial artery via electrophymomanometry (STBP-780, Colin, Tokyo, Japan) before and after each SEP recording. Skin blood flux was measured via laser-Doppler flowmetry (Moor VMS-LDF2, Moor Instruments, UK) by using a combined temperature and eight-collecting fibers-bundled probe (VP1T/7, Moor Instruments) attached to the forehead and left forearm. Heart rate, skin blood flux, and local skin temperatures at the forehead and forearm were continuously measured and sampled at 20 Hz via a data acquisition system (MP150, BIOPAC Systems). Blood flow was measured in the left ICA and common carotid artery (CCA) by using a color-coded ultrasound system (Vivid-i; GE Healthcare, Tokyo, Japan) equipped with a 10-MHz linear transducer. ICA and CCA blood flow measurements were performed ~1.0–1.5 cm distal and proximal to the carotid bifurcation, respectively. Blood flow was measured 1 min before each session. The diameter of each vessel was measured at three points in a longitudinal section by using the brightness mode, and the Doppler velocity spectrum was subsequently identified with the pulsed wave mode. Systolic and diastolic diameters were measured in detail, and the mean diameter (cm) was then calculated in relation to the blood pressure curve: mean diameter = [(systolic diameter × 1/3)] + [(diastolic diameter × 2/3)]. The time-averaged mean flow velocity obtained in the pulsed wave mode was defined as the mean blood flow velocity (cm/s). Blood flow velocity was measured from the average of ~10–20 cardiac cycles to eliminate the effects of the breathing cycle. When making blood flow velocity measurements, care was taken to ensure that the probe position was stable, that the insonation angle did not vary (~60° in most cases), and that the sample volume was positioned in the center of the vessel and adjusted to cover the width of the vessel diameter. Blood flow was calculated by multiplying the cross-sectional area [π × (mean diameter/2)] by mean blood flow velocity.

**Fig. 1.** Schema of the experimental time course. CBF, recording for cerebral blood flow; SEPs, somatosensory-evoked potentials; T_e, esophageal temperature.
Flow velocity; blood flow = mean blood flow velocity \times area \times 60 (ml/min). External carotid artery (ECA) blood flow was estimated by subtracting blood flow in the ICA from that in the CCA. The same operator performed all blood flow measurements.

**EEG recordings.** SEPs were recorded using Ag/AgCl disk electrodes placed on the scalp at Fz, Cz, Pz, and C4’ (C4’ was 2 cm posterior to C4), according to the International 10–20 System (Fig. 2). The C4’ electrode, which was located at the contralateral hemisphere to the left hand stimulation, was used to record the neural activity of the primary somatosensory cortex. The Fz electrode was set to measure neural activities generated by the primary motor cortex, premotor area, and prefrontal cortex. The Cz and Pz electrodes were supplemental used to assess the peak amplitudes and latencies of each component at C4’ and Fz. Each electrode was referenced to linked earlobes. To eliminate eye movements or blinks exceeding 100 μV, an electrooculogram was recorded bipolarly with a pair of electrodes placed 2 cm lateral to the lateral canthus of the right eye and 2 cm above the upper edge of the right orbit. Impedance was maintained at less than 5 kΩ. Low frequency 

Temperature, hemodynamic, and skin blood velocity variables are listed in Table 1.

### RESULTS

Temperature, hemodynamic, and skin blood velocity variables were assessed using a measuring scale on the Neuropack system with visual inspection. A 60-s average was calculated for thermoregulatory variables and heart rate before and after each SEP recording. These values and mean blood pressure were averaged between before and after recordings. Thermoregulatory and hemodynamic variables were analyzed by a one-way analysis of variance (ANOVA) with repeated-measures using the within-subject factor of Session (Rest, HS, FHC, and WBC). The peaks of all recognizable components in SEPs were measured, and the peak amplitude of each component was identified immediately prior (i.e., peak-to-peak). On the basis of previous studies, we focused on the C4’ and Fz electrodes (22–24). Peak latencies at C4’ were identified in the P14, N20, P25, N35, P45, and N60 components, and peak amplitudes were measured for N20 (P14-N20), P25 (N20-P25), N35 (P25-N35), P45 (N35-P45), and N60 (P45-N60). Peak latencies at Fz were identified in the P14, N18, P22, and N30 components, and peak amplitudes were measured for N18 (P14-N18), P22 (N18-P22), and N30 (P22-N30). Data were separately submitted to a one-way ANOVA with Session as a factor. In all repeated-measures factors, we tested whether Mauchly’s sphericity assumption was violated. If the result of Mauchly’s test was significant and the assumption of sphericity was violated, the Greenhouse-Geisser adjustment was used to correct sphericity by altering the degrees of freedom with a correction coefficient epsilon. When the significant effects of Session were identified, the post hoc paired t-test was adjusted to identify specific differences between Rest (the 1st) and other sessions. Statistical tests were performed using computer software (SPSS for windows ver. 22.0, SPSS). Significance was set at $P < 0.05$.

### Table 1. Temperature, hemodynamic, and skin blood velocity variables

<table>
<thead>
<tr>
<th></th>
<th>Rest (1st session)</th>
<th>HS (2nd session)</th>
<th>FHC (3rd session)</th>
<th>WBC (4th session)</th>
</tr>
</thead>
<tbody>
<tr>
<td>$T_{es}$ °C</td>
<td>36.8 (0.2)</td>
<td>38.1 (0.2)**</td>
<td>37.9 (0.2)**</td>
<td>37.0 (0.2)**</td>
</tr>
<tr>
<td>$T_{es}$ °C</td>
<td>36.9 (0.3)</td>
<td>38.2 (0.3)**</td>
<td>37.6 (0.6)*</td>
<td>36.6 (0.6)</td>
</tr>
<tr>
<td>$T_{es}$ °C</td>
<td>34.2 (0.7)</td>
<td>39.1 (0.6)**</td>
<td>38.7 (0.5)**</td>
<td>34.0 (0.9)</td>
</tr>
<tr>
<td>Local skin temperature, °C</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Forehead</td>
<td>32.7 (1.1)</td>
<td>34.6 (0.8)**</td>
<td>31.0 (0.8)**</td>
<td>32.3 (1.4)</td>
</tr>
<tr>
<td>Forearm</td>
<td>33.1 (0.6)</td>
<td>37.2 (1.6)**</td>
<td>37.0 (1.4)**</td>
<td>32.9 (1.7)</td>
</tr>
<tr>
<td>Skin blood flux, au</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Forehead</td>
<td>43.4 (12.2)</td>
<td>135.1 (49.3)**</td>
<td>122.2 (38.9)**</td>
<td>69.6 (49.2)</td>
</tr>
<tr>
<td>Forearm</td>
<td>17.5 (14.4)</td>
<td>76.1 (29.3)**</td>
<td>72.9 (24.5)**</td>
<td>39.8 (21.4)*</td>
</tr>
<tr>
<td>HR, beats/min</td>
<td>64.9 (9.0)</td>
<td>107.4 (13.5)**</td>
<td>103.3 (11.8)**</td>
<td>70.9 (11.8)**</td>
</tr>
<tr>
<td>MAP, mmHg</td>
<td>19.8 (8.3)</td>
<td>88.2 (6.9)</td>
<td>86.9 (6.9)</td>
<td>95.6 (9.0)</td>
</tr>
</tbody>
</table>

Data were expressed as the means (SD); $n = 14$. $T_{es}$, esophageal temperature; $T_{es}$, external canal temperature; $T_{es}$, mean skin temperature; au, arbitrary units; HR, heart rate; MAP, mean arterial blood pressure; Rest, normothermic baseline; HS, heat stress; FHC, face/head cooling; WBC, whole body cooling. *$P < 0.05$, post hoc results vs. Rest. **$P < 0.01$, post hoc results vs. Rest. ***$P < 0.001$, post hoc results vs. Rest.
forearm decreased, indicating that they had returned to the pre-heating level with WBC.

**Hemodynamic variables.** ANOVAs for heart rate showed the significant main effect of Session \( F(3,36) = 331.253, P < 0.001 \). Heart rate increased with HS, and was maintained with FHC. Heart rate with WBC almost returned to the preheating level, but was still significantly higher than that at Rest. ANOVAs for mean blood pressure also showed the significant main effect of Session \( F(3,30) = 6.137, P < 0.01 \), whereas the post hoc test did not detect any significant differences between Rest and the other sessions.

ANOVAs for skin blood flux at the forehead and forearm demonstrated the significant main effects of Session \( F(3,36) = 26.771, P < 0.001 \); Greenhouse-Geisser correction: \( F(1.695,18.640) = 26.864, P < 0.001, \varepsilon = 0.565 \). The post hoc test revealed that skin blood flux at the forehead and forearm was significantly larger with HS than at Rest. In the FHC session, skin blood flux at the forehead decreased, but was significantly larger with FHC than at Rest. Skin blood flux at the forearm was also significantly larger with FHC than at Rest. In the WBC session, skin blood flux at the forehead returned to the preheating level, whereas that at the forearm was still significantly smaller than that at Rest.

**Blood flow variables.** Figure 3, A–C, shows the mean values of ICA, CCA, and ECA with SD, respectively. ANOVAs for ICA, CCA, and ECA demonstrated the significant main effects of Session [Greenhouse-Geisser correction: \( F(3,24) = 5.568, P < 0.05, \varepsilon = 0.514 \); \( F(3,24) = 37.892, P < 0.001 \); \( F(3,24) = 36.194, P < 0.001 \)]. ICA blood flow was significantly lower with HS than at Rest \( (P < 0.001) \), whereas blood flow in the CCA and ECA was significantly higher with HS than at Rest \( (P < 0.001, \text{respectively}) \). In the FHC session, blood flow in the ECA was lower with FHC than with HS, while that in the ICA returned to the preheat level \( (P > 0.05) \). No significant differences were observed in blood flow in the ICA, CCA, and ECA between at Rest and with WBC.

**VAS for psychological comfort.** Figure 3D shows VAS with SD, with the significant main effect of Session being observed \( F(3,39) = 49.975, P < 0.001 \). Post hoc tests demonstrated that psychological comfort was significantly lower with HS than at Rest \( (P < 0.001) \), and was higher with WBC than at Rest \( (P < 0.001) \).

**Peak latency of SEPs.** Figure 4 shows grand-averaged SEP waveforms at C4 for each session, and the P14, N20, P25,
N35, P45, and N60 components were examined. Figure 5 shows the grand-averaged SEP waveforms at Fz for each session, and the P14, N18, P22, and N30 components were assessed.

ANOVA for the peak latency of P14, N20, P25, and N35 at C4 showed the significant main effect of Session \[ F(3,39) = 17.197, P < 0.001; F(3,39) = 26.078, P < 0.001; F(3,39) = 3.461, P < 0.05; F(3,39) = 4.433, P < 0.01. \] The post hoc test showed that these peak latencies were significantly shorter with HS and FHC than at Rest. Moreover, latencies were significantly shorter with WBC than at Rest (Table 2).

There were no significant main effects in the peak latencies of P45 or N60 at C4 or N30 at Fz.

**Peak amplitude of SEPs.** ANOVAs for the peak amplitude of N60 at C4 showed the significant main effect of Session \[ F(3,39) = 7.047, P < 0.01; F(3,39) = 4.385, P < 0.01; F(3,39) = 6.514, P < 0.01. \] The post hoc test also showed that these peak amplitudes were significantly shorter with HS, FHC, and WBC than at Rest (Table 2).

There were no significant main effects in the peak amplitudes of N20, P25, N35, or P45 at C4 or N18, P22, or N30 at Fz (Table 3).

**DISCUSSION**

We herein demonstrated the effects of FHC and WBC during passive HS on human somatosensory processing by using SEPs. The peak latencies of some SEP components were significantly shorter with HS, FHC, and WBC than at Rest (Table 2). The peak amplitudes of N20, P25, N35, or P45 at C4 or N18, P22, or N30 at Fz (Table 3).

<table>
<thead>
<tr>
<th>Component</th>
<th>Rest (1st session)</th>
<th>HS (2nd session)</th>
<th>FHC (3rd session)</th>
<th>WBC (4th session)</th>
</tr>
</thead>
<tbody>
<tr>
<td>C4</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P14</td>
<td>14.6 (0.2)</td>
<td>13.1 (0.2)****</td>
<td>13.5 (0.2)****</td>
<td>14.2 (0.2)*</td>
</tr>
<tr>
<td>N20</td>
<td>19.4 (0.2)</td>
<td>18.1 (0.1)****</td>
<td>18.0 (0.2)****</td>
<td>18.9 (0.1)*</td>
</tr>
<tr>
<td>P25</td>
<td>24.2 (0.2)</td>
<td>23.1 (0.4)**</td>
<td>23.3 (0.3)</td>
<td>23.3 (0.3)*</td>
</tr>
<tr>
<td>N35</td>
<td>30.5 (0.8)</td>
<td>29.0 (0.7)*</td>
<td>29.9 (0.8)</td>
<td>30.7 (0.7)</td>
</tr>
<tr>
<td>P45</td>
<td>42.7 (1.1)</td>
<td>41.9 (0.9)</td>
<td>42.1 (1.1)</td>
<td>43.4 (0.9)</td>
</tr>
<tr>
<td>N60</td>
<td>57.8 (0.8)</td>
<td>56.8 (0.9)</td>
<td>57.4 (1.1)</td>
<td>58.1 (1.0)</td>
</tr>
<tr>
<td>Fz</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P14</td>
<td>14.4 (0.2)</td>
<td>12.9 (0.3)**</td>
<td>13.5 (0.2)**</td>
<td>13.6 (0.2)*</td>
</tr>
<tr>
<td>N18</td>
<td>16.7 (0.3)</td>
<td>16.0 (0.2)**</td>
<td>16.2 (0.2)*</td>
<td>16.4 (0.2)</td>
</tr>
<tr>
<td>P22</td>
<td>19.2 (0.2)</td>
<td>18.6 (0.2)**</td>
<td>18.7 (0.2)*</td>
<td>18.8 (0.2)*</td>
</tr>
<tr>
<td>N30</td>
<td>29.9 (0.9)</td>
<td>28.7 (0.8)</td>
<td>29.3 (0.8)</td>
<td>29.2 (0.8)</td>
</tr>
</tbody>
</table>

Data were expressed as the mean ± SE; \( n = 14 \). *\( P < 0.05 \), post hoc results vs. Rest. **\( P < 0.01 \), post hoc results vs. Rest. ***\( P < 0.001 \), post hoc results vs. Rest.
significantly shortened by increases in body temperature, and this acceleration was maintained during FHC. In addition, latency was still accelerated during WBC. On the other hand, the peak amplitude of N60 at C4’ decreased with increases in body temperature, but recovered with FHC and WBC.

**Effects of HS.** The peak latencies of P14, N20, P25, and N35 at C4’, and P14, N18, and P22 at Fz were shortened with increases in body temperature in the HS session (Figs. 4 and 5, and Table 2), which was consistent with our previous findings (24). The primary response of SEPs was recorded ~20 ms after stimulating the median nerve, and was referred to as the N20 component. As described in the introduction, this component is generated from Brodmann’s area 3b of the primary somatosensory cortex, and its latency has been used as an index of the conduction velocity of the peripheral somatosensory pathway from the median nerve to the primary somatosensory cortex. Thus our results indicated that the conduction velocity of the ascending somatosensory input was accelerated by increases in body temperature. The P14 component, which is recorded just before N20, is generated from higher segments of the cervical cord (31) and at or near the foramen magnum (10). P14 at C4’ and Fz was accelerated in the HS session, indicating acceleration not only in cortical processing but also in subcortical processing. In addition, the peak latencies of P25 and N35 were significantly shorter with HS than at Rest. The generator mechanisms for P25 and N35 remain unknown. A previous study using dipole modeling with magnetoencephalography identified multiple cortical areas as generators at ~20–60 ms, involving areas 3b, 1, and 4 and the posterior parietal cortex (13). The P25 component is also known to reflect different neural processing from the N20 component. N20 is generated from area 3b of the primary somatosensory cortex, whereas P25 is generated from area 1 (3, 13). In contrast, Valeriani and colleagues (36), using brain electrical source analysis, showed a common generator for the N20 and P24 components, which may represent the opposite counterparts of the primary response. P25 and N35 has been suggested to arise not only from the primary somatosensory cortex (area 1), but also from more anterior areas including area 4 (15, 18).

The peak amplitudes of N60 were significantly smaller in the HS session than in the Rest session (Fig. 4 and Table 2), which was consistent with our previous findings (24). N60 and other SEP components, including N20, P25, N35, and P45, are known to be generated from the primary somatosensory cortex. However, Barba and colleagues (7, 8), utilizing depth electrodes on epilepsy patients, reported that the fronto-central N60 response originated from not only the primary somatosensory cortex but also the supplementary motor area. On the basis of these findings, neural activities including those at the primary somatosensory cortex and supplementary motor area became impaired with HS.

Blood flow in the CCA and ECA increased with HS, whereas ICA blood flow was less with HS than at Rest, which was also consistent with previous findings (Fig. 3) (28). This result indicated that HS modified the distribution of intra- and extracranial blood flow.

**Effects of FHC.** In the FHC session, the values for peak latencies during FHC were similar to those during HS, indicating that peak latencies remained accelerated even during FHC. In other words, FHC did not affect the conduction velocity of ascending signals from the periphery to the subcortical regions and primary somatosensory cortex (Figs. 4 and 5, and Table 2).

On the other hand, the peak amplitude of N60 was recovered by FHC in the FHC session (Fig. 4 and Table 3). Since N60 is the latest component among these SEP components, the neural mechanisms involved may be more complex and higher than other earlier SEP components. For example, N60 was more easily affected by various somatosensory inputs than the other SEP components during passive HS (24), movement preparation (14), and mastication (26).

The present study attempted to clarify whether FHC recovered ICA and ECA to prebaseline levels. Consistent with our hypothesis, FHC recovered cerebral perfusion (Fig. 3). In addition, psychological comfort was also recovered by FHC (Fig. 3D). As for modulations of the peak amplitude of N60, cerebral perfusion and psychological comfort may be related to the strength of neural activity in somatosensory processing. An adequate supply of oxygen and glucose is needed for adequate brain functions. We assumed that the recovery of cerebral blood flow contributes to variations in the changes observed in the amplitude of SEPs.

Although the impedance of each electrode was verified for each session, changes in the sweat rate during the recording may affect the amplitude of SEP components. However, HS and FHC only affected the amplitude of N60 at C4’. Other SEP components at C4’ and Fz did not change throughout the experiment. Therefore, the reduction observed in the amplitude of N60 to HS was unlikely to be affected by sweat-induced shifts in potential.

**Effects of WBC.** In the WBC session, the peak amplitude of N60 recovered to the preheat level observed in the Rest session (Figs. 4 and 5, and Table 3). The results obtained for temperature variables in the WBC session also showed that body temperature and cerebral perfusion recovered to those in the Rest session (Fig. 3). However, the peak latencies of P14, N20, and P25 at C4’ and P14 and N22 at Fz were still significantly earlier in the WBC session than in the Rest session (Figs. 4 and 5, and Table 2). These results suggest the aftereffects of passive HS, even if body temperature, psychological comfort, and cerebral perfusion have sufficiently recovered to preheat stress levels; ascending somatosensory processing is still affected. In other words, abnormal ascending signals may still remain at this time. However, we did not examine how long this aftereffect on the peak latency of SEPs existed. Therefore, further studies are needed to clarify this issue.

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**Table 3. Peak amplitudes (μV) of somatosensory-evoked potentials and statistical results at C4’ and Fz**

<table>
<thead>
<tr>
<th>Session</th>
<th>C4’</th>
<th>HS (2nd session)</th>
<th>FHC (3rd session)</th>
<th>WBC (4th session)</th>
</tr>
</thead>
<tbody>
<tr>
<td>N20 (μV)</td>
<td>2.7 (0.3)</td>
<td>2.6 (0.2)</td>
<td>2.5 (0.2)</td>
<td>2.5 (0.3)</td>
</tr>
<tr>
<td>P25 (μV)</td>
<td>3.4 (0.4)</td>
<td>3.2 (0.4)</td>
<td>3.3 (0.4)</td>
<td>3.5 (0.4)</td>
</tr>
<tr>
<td>N35 (μV)</td>
<td>2.0 (0.3)</td>
<td>2.1 (0.3)</td>
<td>2.2 (0.3)</td>
<td>2.3 (0.3)</td>
</tr>
<tr>
<td>P45 (μV)</td>
<td>3.3 (0.3)</td>
<td>3.2 (0.3)</td>
<td>3.4 (0.4)</td>
<td>3.4 (0.3)</td>
</tr>
<tr>
<td>N60 (μV)</td>
<td>4.5 (0.4)</td>
<td>3.4 (0.5)**</td>
<td>4.1 (0.7)</td>
<td>4.1 (0.5)</td>
</tr>
<tr>
<td>N18 (μV)</td>
<td>1.2 (0.1)</td>
<td>1.3 (0.1)</td>
<td>1.6 (0.2)</td>
<td>1.3 (0.1)</td>
</tr>
<tr>
<td>P22 (μV)</td>
<td>1.2 (0.1)</td>
<td>1.3 (0.1)</td>
<td>1.5 (0.2)</td>
<td>1.3 (0.1)</td>
</tr>
<tr>
<td>N30 (μV)</td>
<td>3.3 (0.4)</td>
<td>3.0 (0.3)</td>
<td>3.1 (0.4)</td>
<td>3.5 (0.4)</td>
</tr>
</tbody>
</table>

Data were expressed as the means ± SE; *n* = 14. **P < 0.01, post hoc results vs. Rest.**
Limitations of the present study. Although we showed reductions and the recovery of the peak amplitude of N60 by utilizing SEPs during passive HS and FHC, it currently remains unknown whether higher cognitive functions or neural activities for other sensory modalities such as auditory and visual processing are also modulated, similar to our previous findings, because we only focused on neural activity in ascending somatosensory processing. Moreover, the present study did not directly evaluate the actual perception and cognition of the somatosensory stimulus with psychophysical tests. We examined modulations in the peak amplitude and latency of some SEP components during each session. The relationship between modulations in SEPs and actual cognitive performance need to be clarified in future studies.

Perspectives and Significance

The present study using SEPs showed the effects of FHC during passive HS on psychological comfort, cerebral perfusion, and human somatosensory processing. The results obtained appear to provide insights into the psychological, physiological, and neuroscientific mechanisms underlying hyperthermia. Our results may contribute to the development of a preventative methodology for hyperthermia in daily life and sports activities. In recent decades, a number of severe heat waves have occurred throughout the Northern Hemisphere (29). FHC may be one of the simple and effective methods for maintaining psychological comfort, cerebral perfusion, and the somatosensory system during hyperthermia. In future studies, other preventative methodologies, such as drinking, an effective cooling time period, and cooling of other body parts, need to be established.

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

H.N., R.K., and M.S. conception and design of the research; H.N., M.N., and M.S. performed experiments; H.N. and M.S. analyzed the data; H.N. and M.S. interpreted the results of the experiments; H.N. prepared figures; H.N., A.S., and M.N. drafted the manuscript.

REFERENCES


