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RESEARCH ARTICLE

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Vision and gaze direction modulate tactile processing in somatosensory cortex: evidence from event-related brain potentials

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Abstract Several behavioural studies have shown that directing one's gaze at a body part reduces detection speed and enhances discrimination of tactile stimuli at that location. We investigated how vision of a body part stimulated and manipulations of gaze direction affect tactile processing. Participants' gaze was directed to one of their hands, with vision of this hand either available or prevented in different experiments. They had to detect infrequent tactile targets among non-targets. Somatosensory event-related brain potentials were recorded in response to stimulation of the hand towards which gaze was directed (G+ trials) and in response to stimulation of the other hand (G– trials). When vision (V+) of the hand gaze was directed at was available (G+V+), an early positivity overlapping with the P45 and N80 component was observed for G+V+ trials relative to G–V– trials. In contrast, when the hands were occluded from view (V–), an enhanced N140 component followed by a late negativity was observed for G+V– as compared to G–V– trials. It is suggested that vision of the body part stimulated can modulate processing in primary somatosensory cortex (S1), while effects of gaze direction in the absence of vision of the body part touched are located in higher order somatosensory areas. Such effects of vision and gaze on tactile processing may be mediated by pathways from multimodal brain regions to somatosensory cortex.

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Introduction

If we expect to be touched at a certain location, we tend to look at that body part even if we cannot see the application of the tactile stimulus. One reason for this might be that we tend to rely more on visual input than on touch when localizing external events. Another reason might be that vision aids tactile perception. The act of looking at a body part comprises both adjustments of gaze direction as well as vision. Here, we defined "gaze" as the orientation of the eyes towards a specific body part, and "vision" as the actual sensory perception of this body part. Although gaze and vision usually operate in unison, this is not always the case. When directing gaze at a body part it is not guaranteed that we see the particular body part, as we may be blindfolded or the body part may be occluded from view (gaze without vision). Likewise, when a body part is reflected in a mirror, vision is present although gaze is not in fact directed to the physical location of this body part (vision without gaze). Thus, when looking at a body part where we expect to be touched, both gaze direction and vision may contribute jointly or independently to our perception of the subsequent tactile sensation.

Several behavioural studies have investigated the effect of gaze and of vision on the processing of tactile stimuli in various discrimination and detection tasks. These studies have investigated the independent or combined effects of gaze direction and vision on tactile processing. To investigate pure effects of vision, Kennett et al. (2001) examined 2-point threshold discrimination on the forearm. They found that tactile discrimination was better under conditions when participants were viewing their forearm compared to when the arm was occluded from view, and also relative to a condition

where an object was shown in the same location as the arm. These results suggest that vision of the body part touched enhances threshold discrimination of tactile stimuli (see also Press et al. 2004). In Kennett et al.'s (2001) study, vision of a body part touched was manipulated while gaze direction was held constant. In contrast, an effect of gaze direction on tactile processing was reported in a detection task by Honoré et al. (1989). In this study, participants' gaze was directed at different light sources while they were sitting in a dark room, thus the body part touched could never be seen. Tactile stimuli were detected faster when gaze was directed at a light that came from the same location as the tactile stimulation than when it came from a different location. This study, where vision was held constant while gaze direction was manipulated, suggests a pure effect of gaze direction on tactile processing in the absence of vision of the body part touched. In sum, previous behavioural studies have manipulated either gaze direction or vision separately and have shown that both affect performance in tactile tasks.

Other studies have explored possible interactions between effects of gaze direction and of vision on touch. Driver and Grossenbacher (1996) presented tactile stimuli simultaneously to both hands. Participants' task was to detect tactile target stimuli amongst non-target stimuli presented to one hand and ignore distractor stimuli presented to the other hand. Tactile distractors were presented to impair discrimination of tactile stimuli presented to the other hand. This impairment of tactile discrimination at one hand was reduced when participants gazed at that hand, compared to when their gaze was directed at the hand where distractors were presented. Such gaze-dependent improvements of tactile discrimination were found not only when vision was available, but also when participants were blindfolded. Thus, it appears that gaze direction can affect tactile processing regardless of the presence of vision. Along similar lines, Tipper et al. (1998) reported that gaze direction facilitates tactile detection both when participants viewed the hand touched and when the hand was hidden from view. In their study, participants' task was to detect tactile stimuli presented to both hands while their gaze was directed to one of their hands either with or without vision of this hand. Irrespective of the availability of vision, responses to tactile stimuli at the hand gaze was directed at were faster as compared to responses to stimuli to the other hand, again suggesting an effect of gaze direction independent of vision. In another condition, participants viewed one of their hands displayed on a monitor placed in front of them. Thus, gaze was directed at a monitor and not at the body site touched. Here, Tipper et al. (1998) found facilitated detection of tactile stimuli presented to the hand that was viewed on the monitor compared to detection latencies of stimuli at the other hand suggesting, an effect of vision that is independent of gaze direction (see also Tipper et al. 2001). In summary, it appears that both gaze direction and vision can facilitate tactile processing, and that their respective effects might be largely independent of each other.

To date, only two electrophysiological studies have investigated the effects of gaze and vision on tactile processing. Taylor-Clarke et al. (2002) manipulated vision while holding gaze direction constant. Participants viewed either their arm or a neutral object while performing a tactile near-threshold discrimination task. Somatosensory event-related potentials (SEPs) were measured in response to tactile events under conditions when either the arm or the object was viewed. An enhanced N80 component was observed contralateral to the stimulated arm in the view-arm condition, and this effect was followed by a late enhanced positivity for the view-arm relative to the view-object condition. Hesse et al. (2004) manipulated gaze direction while participants received tactile stimulation at the right wrist. Gaze was directed to a visual marker located either on the body midline or 15° to the right or left, and no differential SEP modulations were found for these different gaze direction conditions. This negative result is surprising when considering behavioural studies (Driver and Grossenbacher 1996; Honoré et al. 1989; Tipper et al. 1998), which have shown clear effects of gaze direction on performance in tactile detection and discrimination tasks. However, in contrast to these earlier studies, Hesse et al. (2004) asked participants to direct their gaze to locations in far space (80 cm from the body), and not to the body part touched.

The objective of the present ERP experiment was to further obtain electrophysiological evidence for the effects of vision and of gaze on somatosensory processing. Although effects of vision have been reported in one previous study (Taylor-Clarke et al. 2002), possible ERP effects of gaze direction towards a body part have not yet been systematically investigated at all. In the present study, we instructed participants to direct their gaze to one hand, and to detect and respond to infrequent tactile target stimuli, which were presented equiprobably to the hand where gaze was directed at and to the other hand. SEPs in response to tactile non-target stimuli presented to the left and right hand were compared as a function of gaze (gaze directed towards versus away from the current stimulus location). In experiments 1 and 2, gaze and vision were manipulated in parallel (that is, the hand gaze was directed at was also viewed). In experiment 1, hands were located to the left and right of the body midline, and participants looked at one of these hands (deviated gaze). In experiment 2, one hand was aligned with the body midline, while the other hand was located to the left or right in its own hemispace, and participants looked at the hand in front of them (straight gaze). Any differential effects of gaze on SEPs observed in these two experiments could be either due to an effect of vision, an effect of gaze, or a combination of both types of effects on somatosensory processing. In order to identify pure effects of gaze, experiment 3 was a replication of experiment 2, except that vision of the hands was now prohibited by placing them under a second tabletop.

Experiment 1

To investigate the timing and locus of any effects of vision and/or gaze direction on tactile processing, we presented mechanical tactile stimuli to the participants' right and left hand, while their gaze was directed at one of their hands. Gaze direction was manipulated by instructing participants to move their eyes to one of the hands that were located to the right and the left of the body midline.

Materials and methods

Participants

Sixteen paid volunteers participated in the experiment. Two participants had to be excluded due to excessive alpha activity, and another two participants were excluded because of eye movements. Thus, 12 participants (four females, eight males) aged 20–36 years (mean age of 26 years) remained in the sample. All participants gave written informed consent. The study was performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and was approved by the Ethics Committee, School of Psychology, Birkbeck College.

Stimuli and procedure

Participants sat in a dimly lit experimental chamber, wearing a head-mounted microphone. Tactile stimuli were presented using 12 V solenoids, driving a metal rod with a blunt conical tip to the middle segment of the index fingers, making contact with the fingers whenever a current was passed through the solenoid. The middle segment of the participants' index fingers were placed palm side down on the solenoids that were positioned 23 cm from the right and the left of the body midline and 50 cm from the participants' body. Participants' index fingers were held in place by a Velcro strap occluding the movement of the solenoids and any related artefacts (Fig. 1). A chin rest was used to hold the head midline (nose) in line with the body midline. White noise (62 dB SPL) was continuously present to mask any sounds made by the tactile stimulators. Tactile non-target stimuli consisted of one rod contacting a finger for 200 ms. Tactile target stimuli had a gap, where this contact was interrupted for 10 ms after a duration of 95 ms.

Throughout a block, participants maintained fixation on either their left or right index finger by diverting their gaze from the body midline, while keeping their head aligned with the body midline. Participants' gaze direction was monitored via a video camera throughout the experiment. Half of the participants fixated their right index finger for the first four blocks and their left index finger for the following four blocks. The other participants performed the experiment in reverse order. One block consisted of 120 trials, with ten target (gap) and 110 non-target stimuli presented randomly and equiprobably to the right and left hand. The interval between tactile stimulus presentations was 1000 ms. Participants were instructed to respond vocally (''yes'') whenever a target stimulus was detected at either the left or right index finger. Vocal reaction times were recorded by a voice key.

EEG recording and data analyses

EEG was recorded with Ag-AgCl electrodes and linked-earlobe reference from 23 scalp electrodes (midline

Fig. 1 Setup of experiment 1 showing the position of the participants' hands and the Velcro straps holding in place the index fingers on top of the solenoids. The *small inset* (*bottom left*) shows a top view of a solenoid set into a small box. The middle segment of participants' index finger was placed palm-down on the top of the solenoid

electrodes FPz, Fz, Cz, Pz, Oz, electrodes F4, F8, FC6, C4, T8, CP6, P4, P8, OR and homologous electrode sites over the left hemisphere). Horizontal EOG was recorded bipolarly from the outer canthi of both eyes. Electrode impedance was kept below 5 kΩ, amplifier bandpass was 0.1–40 Hz, and digitization rate was 200 Hz. No additional filters were applied to the EEG data, and all ERP analyses were based on these unfiltered data. Trials with eyeblinks, horizontal eye movements, or muscle artifacts as well as trials immediately following a vocal response were excluded to avoid any contamination of averaged ERPs by movement-related artifacts.

ERPs to non-target stimuli were averaged relative to a 100 ms pre-stimulus baseline, separately for all combinations of stimulus location (left versus right) and gaze direction (left versus right). ERP mean amplitudes were computed within measurement windows centred on the latency of somatosensory P45 (30–60 ms), N80 (70–95 ms), P110 (100–120 ms) and N140 (125–160 ms) components. To investigate longer-latency effects of gaze direction, mean amplitudes were also computed between 190 and 250 ms post-stimulus. Mean amplitudes were analysed with repeated measures ANOVAs. Analyses of ERP data were restricted to midline electrode Cz and electrodes contralateral to the stimulated hand (FC5/6c, C3/4c and CP5/6c). These electrodes are located close to and over somatosensory cortex, where early SEP components are maximal. Analyses included the factors gaze (stimuli presented to the hand to which gaze was currently directed and which was viewed versus to the other hand: G + V + versus G–V-), stimulus location (left versus right), and electrode (Cz, FC5/6c, C3/4c and CP5/6c). When appropriate, Greenhouse-Geisser adjustments to the degrees of freedom were performed.

The latency of vocal responses was measured with a voice key relative to the gap onset of the target stimuli (95 ms after stimulus onset), as target/non-target discriminations were only possible after this interval. For vocal responses to tactile targets, mean response times (RT) for each participant were calculated for each combination of target location and gaze direction. A repeated measures ANOVA was performed on mean RTs with the factors gaze direction and stimulus location.

Results and discussion

An effect of gaze direction on vocal response latencies to tactile stimuli was observed, with participants responding on average 21 ms faster to infrequent tactile target stimuli at the hand that was currently looked at (G + V + trials) compared to stimuli at the other hand (G-V- trials) [497 and 518 ms, respectively; $F(1,11) = 6.3$, $P < 0.03$]. There was no difference in response latencies to left (508 ms) and right (508 ms) hand stimuli [$F(1,11) < 1$], nor was there a gaze×location interaction [$F(1,11) < 1$]. Participants missed on average 4% of target stimuli and the false alarms rate was below 1%.

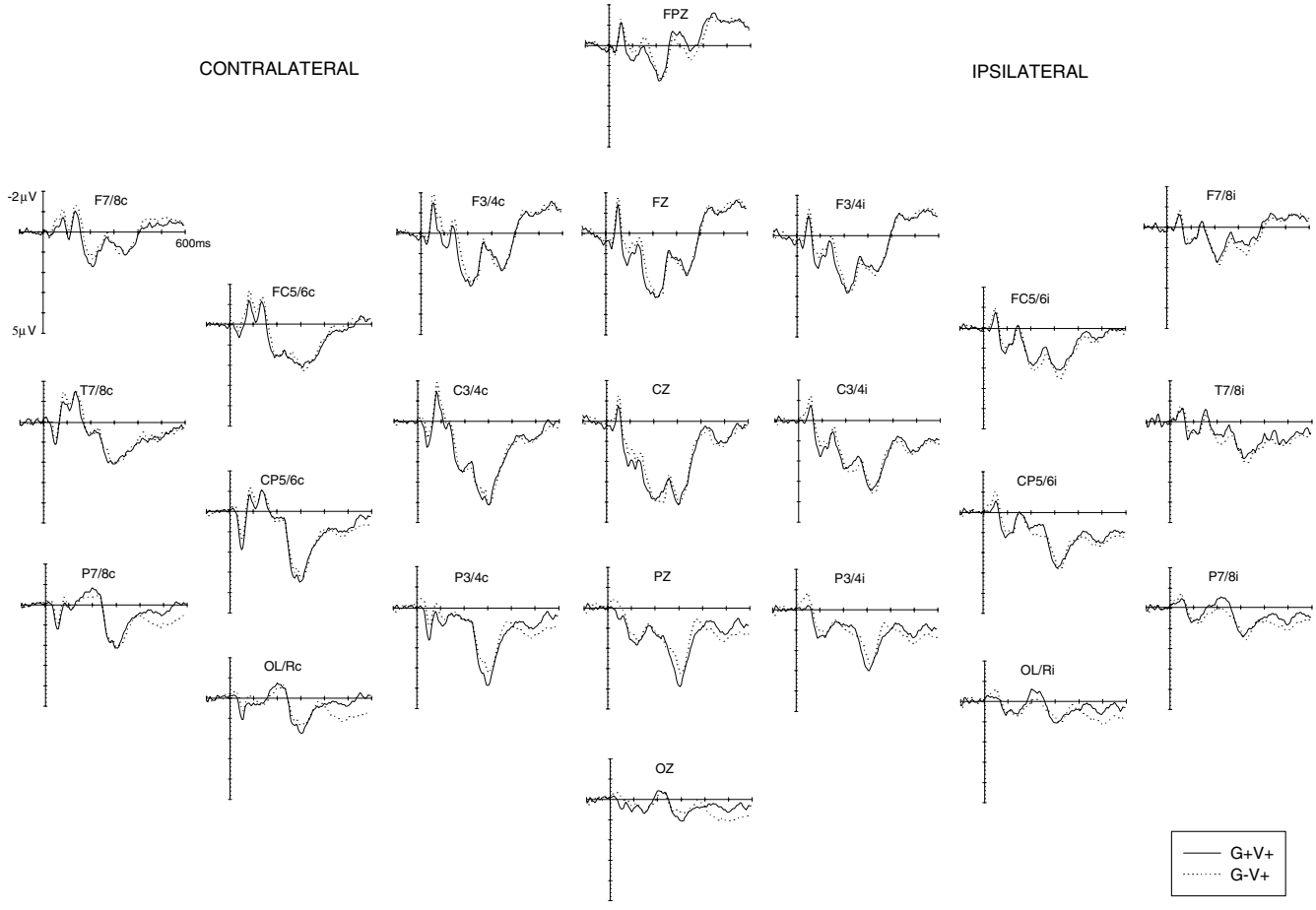
Figure 2 shows SEPs in response to tactile non-target events delivered to the hand to which gaze was directed (G + V +) and to the other hand (G-V-) as observed at all recording sites in the 600 ms interval after stimulus onset, and displayed separately for midline sites, and for electrodes ipsilateral and contralateral to the stimulated hand. Systematic effect of gaze at short latencies can be seen more clearly in Fig. 3. This figure shows SEPs recorded close to somatosensory cortex contralateral to the stimulated hand and at midline electrode Cz in the first 300 ms after stimulus onset, together with grand-averaged horizontal EOG (HEOG) waveforms, for G + V + trials (solid lines) and G-V- trials (dashed lines). An early positivity was present for tactile stimuli on G + V + trials relative to G-V- trials in the time range of the P45 [30–60 ms post-stimulus onset; main effect of gaze: $F(1,11) = 13.8$, $P < 0.01$] and the N80 component [70–95 ms post-stimulus; main effect of gaze: $F(1,11) = 5.6$, $P < 0.04$]. The effect of gaze marginally failed to reach significance in the subsequent P110 analysis window [100–120 ms post-stimulus; $F(1,11) = 4.4$, $P = 0.06$]. No reliable effects of gaze or gaze×electrode interactions were found for the N140 time range (125–160 ms post-stimulus) and for the subsequent longer-latency analysis window (190–250 ms post-stimulus).

These findings differ from the SEP effects previously reported by Taylor-Clark et al. (2002), who observed an enhanced N80 component on view-hand trials, and by Hesse et al. (2004), who found no SEP effects of gaze direction at all. One obvious difference between the current experiment 1 and these earlier studies was that here, we varied vision and gaze direction simultaneously and in parallel, whereas only vision or only gaze direction were manipulated in by Taylor-Clark et al. (2002) and Hesse et al. (2004), respectively. For example, in the Taylor-Clark et al. (2002) study, gaze was constant across conditions while vision of the body part touched was manipulated. The fact that in the present experiment 1, effects of gaze were already observed in the P45 time range may suggest that the combination of gaze and vision may effect somatosensory processing at even shorter latencies than does vision alone.

There is, however, another possible, and more trivial, account for the pattern of results observed in experiment 1. The diversion of gaze to one hand and way from the other hand might have had a systematic effect on early stages of somatosensory processing by straining the muscles of the eyes to keep fixation. Experiment 2 was conducted to evaluate any potential impact of gaze deviation as such on the pattern of results observed in the first experiment.

Experiment 2

This experiment was conducted to rule out the possibility that the gaze effects found in experiment 1 are merely due to the diversion of gaze from the body midline to-



wards one hand and away from the other hand. In experiment 2, participants looked straight ahead at the hand, which was placed on the midline in front of them. The other hand was placed next to the body in its own hemispace. Their task was to detect infrequent changes in the intensity of vibratory tactile stimuli presented to their right and left hand. This change from the ''gap-detection'' task in experiment 1 was done to increase task difficulty, in order to test whether the early SEP effects found there would generalize across variations in task demands. In all other aspects, the design and procedure was identical to experiment 1.

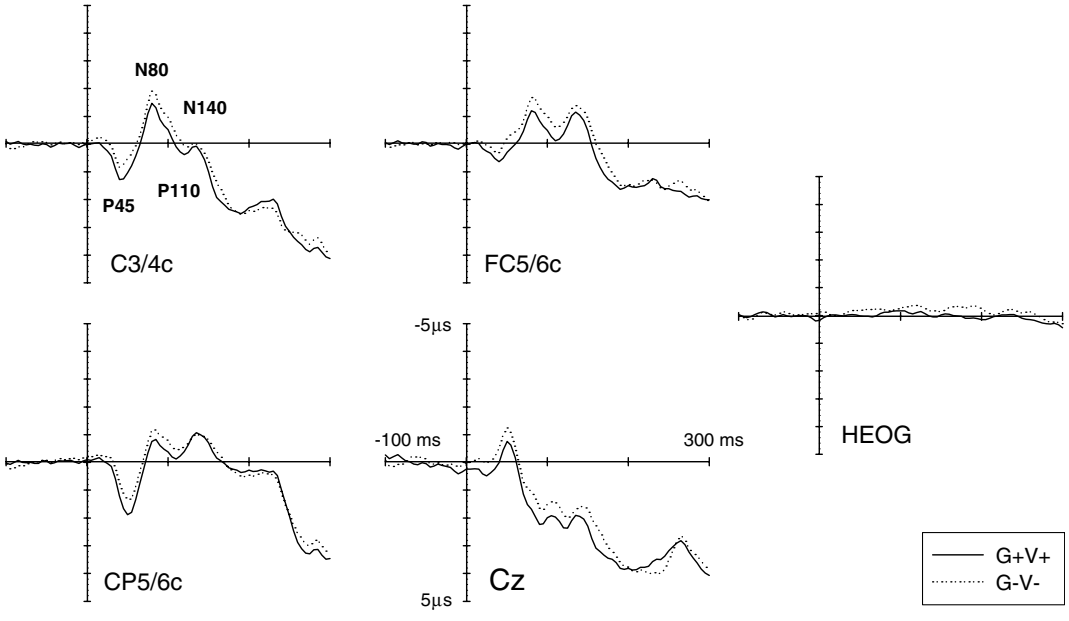


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Fig. 2 Grand-averaged somatosensory ERP (SEP) waveforms elicited in experiment 1 in the 600 ms interval following stimulus onset by tactile non-target stimuli delivered to the hand where gaze was directed at (G + V +; *solid lines*) and the other hand (G-V-; *dotted lines*). SEPs are shown for all electrode sites, separately for electrodes contralateral (*left side*) and ipsilateral (*right side*) to the stimulated hand, as well as for midline electrodes (*middle*)

Materials and methods

Participants

Thirteen paid volunteers participated in experiment 2 (two participants took part in experiment 1). One participant had to be excluded due to excessive amount of blinks and insufficient eye fixation control. Thus, 12 participants (nine females, three males) aged 19–38 years (mean age of 26 years) remained in the sample. All participants gave written informed consent.

Stimuli and procedure

The general experimental set-up and procedure was the same as in experiment 1. However, experiments differed with respect to the placement of the arms, the type of tactile stimuli used, and the tactile discrimination task used. Vibratory tactile stimuli with a constant frequency of 58.8 Hz were used. Participants' task was to respond to infrequent tactile target stimuli that were weaker in intensity compared to the stronger non-target stimuli. To create weak and strong intensity stimuli, the contact time between the rod and the skin was 2 ms (followed by a 15 ms interpulse interval) for soft vibrations, and 3 ms (followed by a 14 ms inter-pulse interval) for strong vibrations. This difference in contact time created the subjective perception of clearly distinct soft and strong stimuli. In contrast to experiment 1, where hands were placed to the left and right of the body midline, one hand was now placed on the body midline (50 cm in front of the participants' body), while the other hand was placed 36 cm to the right or left of the body midline next to the participants' body, and this hand was covered. As in experiment 1, the distance between the index fingers was 46 cm. Gaze was always directed straight ahead towards the hand placed on the body midline. In four successive blocks, the left hand was placed on the body midline, and the right hand to the right of the body midline. In the other four successive blocks, the right hand was placed on the body midline, while the left hand was located to the left of the body midline. In all other respects, procedure, EEG recording and data analyses were identical to experiment 1.

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Fig. 3 Grand-averaged SEPs and horizontal EOG waveforms elicited in the 300 ms interval following stimulus onset by tactile non-target stimuli delivered to the hand where gaze was directed at (G + V +; *solid lines*) and the other hand (G-V-; *dotted lines*) at frontocentral (*FC5*/*6c*) central (*C3*/*4c*) and centralparietal (*CP5*/*6c*) electrodes contralateral to the stimulated hand, and at midline electrode Cz in experiment 1

Results and discussion

Figure 4 shows ERPs at electrodes contralateral to the stimulated hand and at midline electrode Cz in response to tactile stimuli delivered to the hand gaze was directed at (G + V +, solid lines) and the other hand (G-V-, dashed lines). In spite of the fact that the factor gaze was now manipulated indirectly by varying hand posture, rather than directly, by varying gaze direction, as in experiment 1, effects of gaze on early SEP components (P45 and N80) were very similar to the short-latency effects found in experiment 1. An early enhanced positivity for G + V + as compared to G-V- trials was present, which overlapped with the P45 [30–60 ms post-stimulus onset; main effect of gaze: $F(1,11) = 4.9$, $P < 0.05$] and the N80 time windows [70–95 ms post-stimulus; main effect of gaze: $F(1,11) = 7.7$, $P < 0.02$]. In contrast, no effects of gaze on the subsequent P110 (100–120 ms post-stimulus) and the N140 (125–160 ms post-stimulus) components and at longer latencies [all Fs$(1,11) < 1.8$] were observed, nor were there any gaze × electrode interaction [all Fs(3,33) < 1]. This similarity of the effects of gaze direction on SEPs in the P45 and N80 latency range between in experiments 1 and 2 rules out the idea that the early SEP modulations found in experiment 1 were due to the physical consequences of the diversion of gaze direction from the body midline, but rather reflects genuine effects of gaze and/or vision on somatosensory processing.

It should be noted that this similarity also rules out another objection that might otherwise be put forward against this interpretation of the effects observed in experiment 2. One could argue that the differences between G + V + and G-V- trials obtained in experiment 2 were simply due to hand posture, with the hand stimulated on G + V + trials was always aligned with the body midline, whereas the hand stimulated on G-V- trials was always positioned to the left or right of the midline. This does however not apply to experiment 1, where the left and right hand were always placed symmetrically to the left and right of the body midline. The fact that in spite of this difference, the pattern of SEP modulations obtained in these two experiments was highly similar provides strong evidence against any account of these effects in terms of differences in hand posture or gaze deviation.

Although SEP effects of gaze were very similar in experiments 1 and 2, gaze failed to affect response latencies in experiment 2 [mean vocal response time: 636 ms; main effect of gaze: $F(1,11) = 1.1$]. This difference to experiment 1 might be related to the differences in task difficulty between these two experiments, as vocal response times were about 100 ms slower in experiment 2 than in experiment 1 (see General discussion for more details). In experiment 2, as in experiment 1, there was no significant difference in the vocal response time to

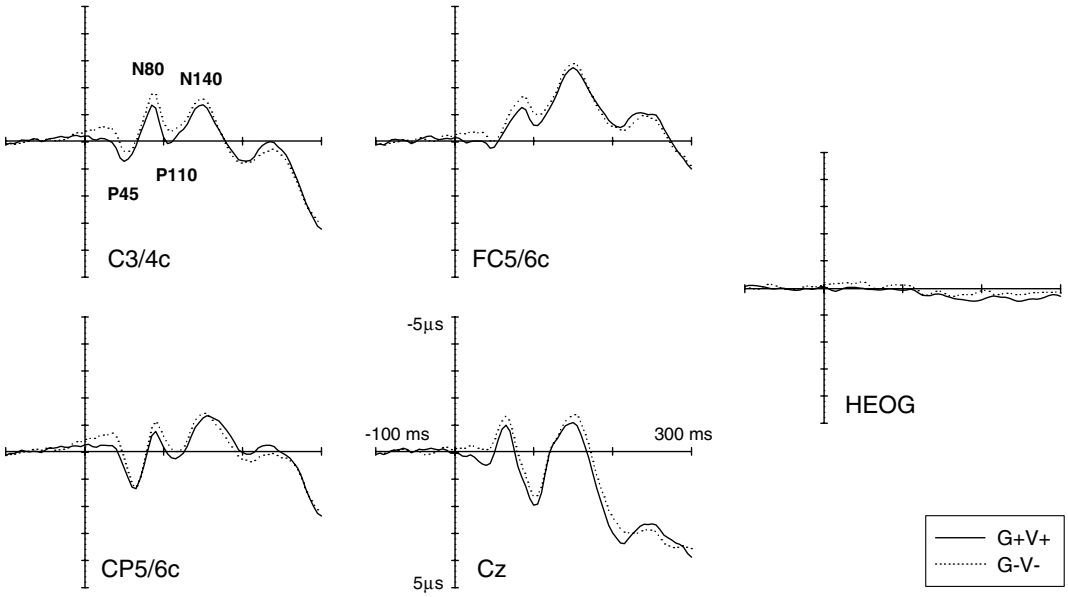

Fig. 4 Grand-averaged SEPs and horizontal EOG waveforms elicited in the 300 ms interval following stimulus onset by tactile non-target stimuli delivered to the hand where gaze was directed at (G + V +; *solid lines*) and the other hand (G-V-; *dotted lines*) at frontocentral (*FC5/6c*) central (*C3/4c*) and central–parietal (*CP5/6c*) electrodes contralateral to the stimulated hand, and at midline electrode Cz in experiment 2

tactile stimuli at the left or right hand [$F(1,11) < 1$], nor did gaze interact with location [$F(1,11) < 1$]. Participants missed on average 5% of target stimuli and the false alarm rate was below 1%.

In experiment 1 as well as in experiment 2, gaze and vision were manipulated together. It is thus not clear whether the early effects of gaze found here were exclusively due to gaze direction towards the body part touched, whether they were solely based on vision of the stimulated body part, or caused by a combination of both factors. Previous behavioural studies have shown facilitation of tactile performance under conditions when participants were directing their gaze at a body part that was touched in darkness or while they were blindfolded (Honoré et al. 1989; Driver and Grossenbacher 1996; Tipper et al. 1998). If the effects of gaze on early tactile processing found in experiments 1 and 2 were likewise exclusively due to gaze, but independent of vision, these effects should not be affected by occluding the hands from view. In contrast, if they were exclusively due to vision, and not to gaze, preventing vision of the stimulated hand should completely eliminate the SEP effects observed in experiments 1 and 2. These alternative possibilities were investigated in experiment 3.

Experiment 3

Experiment 3 was conducted to investigate whether the effects of gaze direction found in the previous two experiments were independent of vision. Participants' arms were placed in the same positions as in experiment 2, but hands and forearms were now completely hidden under a second table top. Throughout the experiment, participants were fixating a cross, which was placed on the second table top at the intersection of the table top surface with the line of gaze directed at the (now invisible) location of the stimulated finger of the hand positioned along the body midline. Like in the first two experiments, participants' task was to detect infrequent tactile targets, regardless of whether they were presented to the hand gaze was directed at (now G + V- trials), or to the other hand (G-V- trials). In all other aspects, the design and procedure was identical to experiment 2.

Materials and methods

Participants

Fourteen paid volunteers participated in experiment 3 (one participant took part in experiment 1 and two participants took part in experiment 2). Two participants had to be excluded due to excessive amounts of alpha activity. Thus 12 participants (six females, six males) aged 19–31 years (mean age of 25 years) remained in the sample. All participants gave written informed consent.

Stimuli and procedure

The experimental set-up and procedure was identical to experiment 2, except that a second table top was placed over the participants' forearms and the upper arms were covered by a piece of cloth obscuring any visual information about body posture. Participants were fixating a cross (1×1 cm) placed on the second table top 7 cm above the table surface where the arms were placed. This cross was carefully positioned at the intersection of the

second table top with the participants' line of gaze directed at the stimulated finger of the hand placed on the body midline. To achieve this, a small hole cut out of the second table top was included, through which the hand could be seen during positioning of the fixation cross. Once the fixation cross was appropriately positioned, it was closed, thus blocking the sight of the hand. EEG recording and data analyses were identical to experiments 1 and 2.

Results and discussion

Figure 5 shows SEPs at electrodes contralateral to the stimulated hand and at midline electrode Cz in response to tactile stimuli delivered to the hand where gaze was directed at (G + V-, solid lines) and the other hand (G-V-, dashed lines). The current manipulation of gaze without concurrent manipulation of vision resulted in an enhanced negativity for stimuli on G + V- trials, and, in addition, this effect occurred at substantially longer latencies than in the first two experiments, with an enhanced N140 component [125–160 ms post-stimulus; main effect of gaze: $F(1,11) = 6.8$, $P < 0.03$] followed by an enhanced negativity [190–250 ms post-stimulus; main effect of gaze: $F(1,11) = 4.9$, $P < 0.05$] for G + V- as compared with G-V- trials. In marked contrast to the first two experiments, there was no evidence whatsoever for any gaze-dependent modulations of the P45 and the N80, or the P110 component [main effect of gaze: all $F(1,11) < 1.7$]. No significant gaze by electrode site interactions were found for any of the analysis windows [all $F(3,33) < 1.9$].

These differences between the SEP effects of gaze obtained in the first two experiments (where gaze and vision were directed in unison) and in experiment 3 (where gaze was manipulated independently of vision) clearly indicate that vision of the body part stimulated has a strong impact on somatosensory processing. However, the results of experiment 3 also indicate that even in the absence of vision, gaze direction can influence the processing of tactile stimuli (see below for a more detailed discussion; see also Fig. 4).

Finally, analogous to experiment 2, but unlike experiment 1, gaze direction failed to affect vocal response latencies to tactile targets on G + V- compared to G-V- trials [$F(1,11) = 2.7$]. Mean vocal response time was 619 ms, which was similar to the response latencies observed in experiment 2, but more than 100 ms slower than vocal responses in experiment 1, again reflecting the increased demands posed by the more difficult intensity discrimination task. In experiment 3, as in the first two experiments, there was no significant difference between response times to tactile targets at the left or right hand [$F(1,11) < 1$], and participants missed on average 4% of target stimuli and the false alarm rate was below 1%.

General discussion

We investigated the effects of vision and gaze direction on tactile processing by means of measuring SEPs. Our aim was to find out whether both gaze and vision can modulate somatosensory processing, and whether their respective effects are independent from each other. Participants' task was to detect infrequent tactile targets

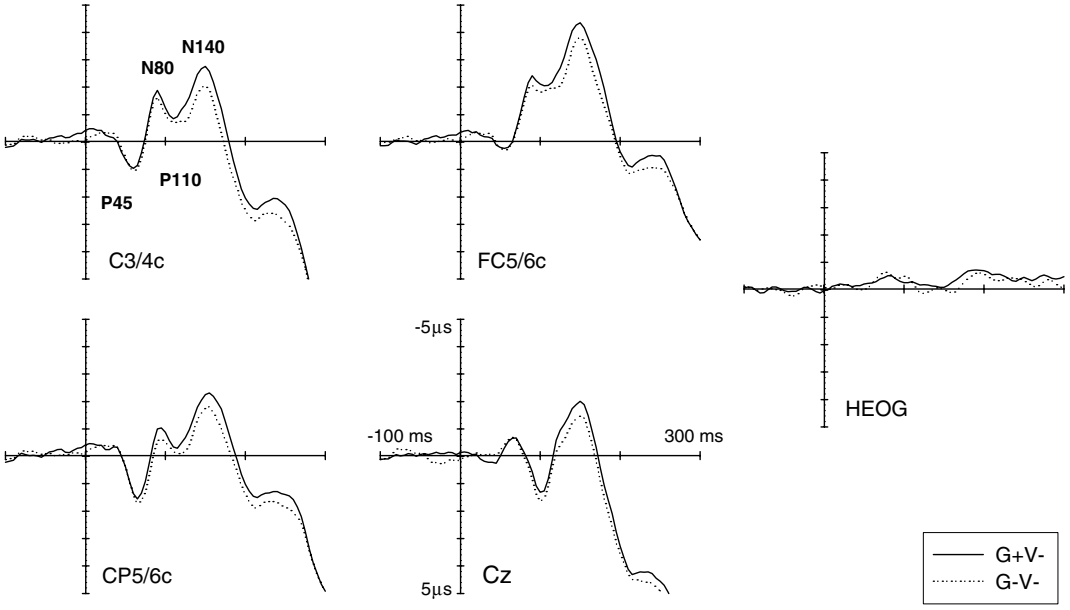

Fig. 5 Grand-averaged SEPs and horizontal EOG waveforms elicited in the 300 ms interval following stimulus onset by tactile non-target stimuli delivered to the hand where gaze was directed at (G + V-; *solid lines*) and the other hand (G-V-; *dotted lines*) under conditions when both hands were occluded from view at frontocentral (*FC5*/*6c*) central (*C3*/*4c*) and central–parietal (*CP5*/*6c*) electrodes contralateral to the stimulated hand, and at midline electrode Cz in experiment 3

at both hands while directing gaze to one of these hands. SEPs elicited by tactile stimulation of the hand located within the line of gaze (G+ trials) were compared to SEPs elicited by tactile stimulation of the other hand (G- trials).

In experiments 1 and 2, gaze and vision were manipulated in parallel (thus, the hand gaze was currently directed at was also viewed, i.e. G+V+, while the other hand was not, i.e. G-V-), by moving the eyes toward the body part touched (experiment 1), or by moving the body part into view while gaze is held straight ahead (experiment 2). Irrespective of these different posture arrangements, gaze direction resulted in the same short-latency SEP modulations. An early-sustained enhanced positivity was present on G+V+ relative to G-V- trials, and this effect overlapped with the P45 and N80 components. In experiment 3, gaze was manipulated in the same way as in experiment 2, but hands were now hidden from view. Here, a very different pattern of SEP results was obtained. Gaze now resulted in an enhanced N140 component followed by a later enhanced negativity for G+V- as compared to G-V- trials, whereas earlier SEP components (P45, N80) were entirely unaffected by the manipulation of gaze direction.

The observation that the short-latency effects of gaze on SEPs observed in experiments 1 and 2 where gaze and vision were manipulated together were absent in experiment 3 where gaze was varied in the absence of vision suggests that these effects exculsively reflect the impact of vision, but not gaze, on early stages of somatosensory processing. Comparisons of intracranial and scalp ERP recordings have suggested that somatosensory ERP activity elicited up to at least 60–90 ms after stimulus onset originate in primary S1, while later activity originates in higher order S1 (Allison et al. 1992; Frot and Mauguière 1999; Barba et al. 2002). The presence of early SEP modulations overlapping with the P45 and N80 component in experiments 1 and 2 therefore suggest that vision of the body part touched can modulate tactile processing in S1.

This conclusion may initially seem counterintuitive, since primary sensory cortices have traditionally been viewed to only process information of a particular modality. However, some evidence suggests that early unimodal processing may be influenced by information from other modalities (e.g. Sathian et al. 1997; Calvert et al. 1999; McDonald et al. 2000). It has been suggested that back-projections from multimodal areas alter operations in areas, which are traditionally thought of as unimodal (cf. Macaluso et al. 2000). Multimodal areas receive input from the different senses, and neurons in these areas are responsive to stimuli from different modalities (cf. Stein and Meredith 1993). Importantly, neurons in area 5 of the primate parietal cortex have been shown to respond to the seen position of the monkey's arm and to tactile stimulation (Graziano et al. 2000), thus combining somatosensory and visual signals in individual neurons. In addition, neurons in other somatosensory association areas, such as the caudal part of the postcentral gyrus, have also been described to integrate somatosensory and visual information (see Iwamura 2003 for review). Thus, it is entirely conceivable that vision of the body part stimulated can modulate processing within S1 via back-projections from these multimodal areas.

In the only previous ERP study investigating the impact of vision on somatosensory processing, Taylor-Clarke et al. (2002) reported enhanced negativity in the time range of the N80 component for tactile stimuli on the forearm when this arm was viewed compared to when an object was viewed. In this experiment, gaze was always directed at the arm. One possible reason as to why SEP effects of gaze in experiments 1 and 2 of the present study were observed at shorter latencies as compared to the effects reported by Taylor-Clarke et al. (2002) is that in this study, the stimulated body part was in fact not directly viewed during stimulation. In the "view arm" condition, the site of tactile stimulation was visible between trials, but vision of the arm was extinguished 50 ms before tactile stimulus onset. This absence of continuous visual stimulation during the presentation of a tactile event may be responsible for the slightly delayed effects of vision found by Taylor-Clarke et al. (2002) as compared to the present study.

It needs to be stressed that in the present experiments 1 and 2, vision and gaze direction were manipulated in parallel. Although the lack of short-latency SEP modulations in experiment 3, where gaze was varied in the absence of vision indicates that these early effects were primarily due to vision, possible contributions of gaze direction cannot be ruled out entirely. To further investigate the timing and locus of effects of vision on tactile processing independently of gaze direction, future studies will need to compare SEPs in response to tactile stimulation of a body part viewed on a monitor or mirror to SEPs elicited by stimulation of another unseen part of the body.

While experiments 1 and 2 provided evidence for modulations of early stages of somatosensory processing in S1 produced by vision, experiment 3 demonstrated that in the absence of vision, gaze direction alone can also affect the processing of tactile stimuli, albeit presumably at later somatosensory processing stages. Interestingly, the "pure" effects of gaze direction found in experiment 3 were very similar to modulations reported in tactile spatial attention tasks requiring participants to covertly attend to tactile stimuli at one hand while ignoring stimuli at another hand, with gaze at a neutral location (Michie 1984; Michie et al. 1987; García-Larrea et al. 1995; Eimer and Driver 2000; Eimer et al. 2001, 2002, 2004; Van Velzen et al. 2002; Eimer and Forster 2003a,b; Hötting et al. 2003). Under such covert tactile attention conditions, an enhanced negativity at the N140 component elicited for stimuli at the covertly attended hand is usually found. In experiment 3, the same pattern of results was observed in response to tactile stimuli at the hand gaze was currently directed

at (G + V- trials). This similarity of present and previous findings suggests the intriguing possibility that manipulating gaze direction without vision of the body part touched may activate the same mechanisms that are responsible for the covert orienting of spatial attention in touch.

The SEP modulations resulting from gaze direction in the absence of vision, as observed in experiment 3, also complement previous behavioural studies, which have demonstrated effects of gaze direction on performance in response to tactile target stimuli under conditions where vision of the body part stimulated was prevented (Honoré et al. 1989; Driver and Grossenbacher 1996; Tipper et al. 1998). It should be noted, however, that while systematic effects of gaze on SEP waveforms were observed in all three experiments of the present study, behavioural effects (i.e. faster vocal responses to tactile stimuli at the hand gaze was directed at compared to the other hand) were confined to experiment 1. There are several possible reasons for the absence of behavioural effects in experiments 2 and 3. First, only a very small number of tactile target stimuli (a total of 40 tactile targets for G + V + and G-V- trials, respectively) were included in all three experiments. This was due to the necessity to obtain a sufficient number of non-target trials for averaging and ERP analyses (which was the main focus of the present study), but inevitably resulted in a very limited number of data points per participant for the behavioural analyses. This fact alone may have been responsible for the absence of any systematic effects of gaze on vocal response times in experiments 2 and 3, where increased task difficulty resulted in an overall increase of response times by more than 100 ms relative to experiment 1. Alternatively, one might argue that if vision and gaze primarily affect early stages of somatosensory processing within 200 ms after stimulus onset (as suggested by our SEP results), but has little effect on perceptuo-motor processes at longer latencies, effects of gaze direction on response latencies will decline or disappear when an increase in overall task difficulty results in longer stimulus processing, and in a delay of response decision and execution processes. These possibilities will need to be explored in future experiments, which include a sufficiently large number of target trials.

The overall pattern of SEP results obtained in the present study provides novel evidence that manipulations of vision and gaze affect different stages of somatosensory processing. Vision appears to be able to modulate processes within S1, while gaze without vision seems to influence activity in higher order somatosensory areas (Allison et al. 1992; Frot and Mauguière 1999; Barba et al. 2002). This dissociation in the timing and, therefore, the associated locus of vision and gaze effects on somatosensory processing appears to suggest that these two factors affect the processing of tactile stimuli independently. However, this latter conclusion is not fully supported by the present data. If vision and gaze were to affect different stages of tactile processing in a strictly independent fashion, then one would expect to find both short-latency SEP effects of vision as well as longer-latency SEP modulations due to gaze direction under conditions where both vision and gaze are manipulated in parallel, as in experiments 1 and 2. However, systematic effects on longer-latency components such as the N140 were entirely absent in these experiments, suggesting that modulation of somatosensory processing due to vision of the body part stimulated prevents any further modulation of subsequent processing stages as a function of gaze direction. Again, this prediction will need to be evaluated in future experiments.

To summarize, the present experiments provided new electrophysiological evidence that early, modality-specific stages in the processing of tactile stimuli can be modulated by vision of the body part stimulated, and by manipulations of gaze direction. While vision appears to affect somatosensory processing in S1, gaze direction seems to affect subsequent stages in the processing of tactile information beyond S1. Both of these effects may be mediated by back-projections from multimodal brain regions to S1.

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