Absence of gaze direction effects on EEG measures of sensorimotor function

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Abstract

Objective: Gaze direction is known to modulate the activation patterns of sensorimotor areas as seen at the single cell level and in functional magnetic resonance imaging (fMRI). To determine whether such gaze direction effects can be observed in scalp-recorded electroencephalogram (EEG) measures of sensorimotor function we investigated somatosensory evoked potentials (SEPs) and steady state movement related cortical potentials (MRPs).

Methods: In two separate experiments, SEPs were elicited by electrical stimulation of the median nerve (experiment 1) and steady state MRPs were induced by 2 Hz tapping paced by an auditory cue (experiment 2), while subjects directed their gaze 15° to the left or to the right.

Results: Gaze direction failed to produce any appreciable differences in the waveforms of the SEPs or MRPs. In particular, there was no effect on peak amplitude, peak latency and peak scalp topography measures of SEP and MRP components, or on spatial or temporal parameters of dipole models of the underlying cortical generators. Additional frequency domain analyses did not reveal reliable gaze-related changes in induced power at electrode sites overlying somatosensory and motor areas, or in coherence between pairs of parietal, central and frontal electrodes, across a broad range of frequencies.

Conclusions: EEG measures of sensorimotor function, obtained in a non-visual motor task, are insensitive to modulatory effects of gaze direction in sensorimotor areas that are observable with fMRI.

Keywords: Somatosensory evoked potentials; Movement related cortical potentials; Gaze direction; Spatial attention

1. Introduction

Various sensorimotor areas in parietal and frontal cortex show a modulation of their activity dependent on the direction of gaze, as shown in monkey (Andersen et al., 1990; Boussaoud and Bremmer, 1999; Mushiake et al., 1997), and in man (DeSouza et al., 2000). Such a modulation by gaze direction reveals an involvement of these areas in sensory-motor transformations that take the position of the eyes into account, as typically studied in visually guided pointing or reaching tasks. Gaze direction effects were recently also observed in a simple finger tapping task whose execution was not dependent on visual input (Baker et al., 1999). This investigation used functional magnetic resonance imaging (fMRI) and showed a relative increase in the extent of activation in primary motor cortex, lateral and medial premotor cortex, and parietal areas when static gaze was aligned with the moving hand compared to when gaze was directed away from the moving hand.

The aim of the present study was to determine whether gaze effects similar to those reported by Baker et al. (1999), i.e. effects obtained in an essentially non-visual task, can be observed in electroencephalogram (EEG) measures of sensorimotor function, such as movement-related potentials and somatosensory evoked potentials, or in measures of the non-phase-locked background cortical activity. Replication of the observation is relevant to the understanding of the modulatory effects of gaze on movement-related brain activity. Moreover, replication with EEG would provide an attractive method for the further investigation of such effects in humans. Finally, the simple manipulation of
gaze direction might be a useful means to emphasize contributions from non-primary motor and non-primary sensory areas to the generation of movement-related and sensory-evoked potentials, respectively.

The movement-related readiness potential \cite{(Kornhuber and Deecke, 1965)} is traditionally recorded as a slow pre-movement negative wave in tasks that involve single movements with inter-movement intervals on the order of a few seconds. Given the long inter-movement intervals we preferred steady-state movement-related potentials (ssMRPs) for this study. SsMRPs were recently proposed as an alternative index of sensorimotor function and are acquired with fast repetitive finger movements performed, like the readiness potential, in a self-paced way or paced by a metronome \cite{(Gerloff et al., 1997)}. SsMRPs are averaged time-locked to electromyogram (EMG) or tap (see Müller et al., 2000) onset. They are characterised by a prominent pre-movement negative component, generated in the primary motor and possibly lateral pre-motor cortex, and a post-movement component thought to represent re-afferent activation of the primary sensory cortex \cite{(Gerloff et al., 1998)}.

If gaze effects are not necessarily dependent on the use of visual information for guidance of movement, they might be found not only with movement-related activation of sensorimotor areas, but also with sensory activation. To probe gaze effects on sensory evoked activity, we used standard somatosensory evoked potentials (SEPs) elicited by electrical stimulation of the median nerve at the wrist. Early components of the SEP (such as the N/P20 and N/P30) are thought to originate in primary somatosensory cortex \cite{(Allison et al., 1991)}, although it has also been suggested that the N/P30 may be generated in motor areas \cite{(e.g. Waberski et al., 1999)}. Relevant here, there is evidence that the amplitudes of early and mid-latency SEP components can be modulated by \cite{(spatial) attention \cite{Desmedt and Tomberg, 1989; García-Larrea et al., 1991; Mima et al., 1998; Buchner et al., 1999; Iguchi et al., 2001)}.

Our analysis was initially focused on measures taken from evoked potentials only, but negative results for both SEPs and ssMRPs led us to extend the analysis to include source analysis, spectral analysis and coherence analysis. Whilst we did not expect to \cite{(and indeed did not)} find physiologically relevant gaze effects, the additional analyses were nevertheless carried out in order to be confident of an absence of gaze effects.

2. Methods

The effects of gaze direction on SEPs and ssMRPs were investigated in two separate experiments. Aspects of the experimental set-up, such as the gaze direction manipulation and electrode placement in the EEG recording were identical. The data analysis approach for both experiments was also essentially the same, but there were slight differences in implementation. Two subjects participated in both experiments.

2.1. Gaze direction task

Subjects sat in a chair with their head comfortably supported but immobilised by a chin rest. At 80 cm viewing distance was a white screen with three horizontally spaced markers \cite{(left, centre, right)} at eye level. When looking at the left or right markers, the gaze angle subtended 15° relative to central fixation. Eye movements and gaze direction were monitored using an infrared camera during the MRP experiment but not the SEP experiment.

2.2. EEG electrode placement and recording

EEG was recorded with Ag/AgCl electrodes from 128 scalp electrodes relative to a vertex reference. Electrodes were applied to the scalp using a carefully positioned nylon cap in accordance with the 10-5 extension of the International 10-10 electrode system \cite{(Oostenveld and Praamstra, 2001)}. EEG signals were amplified by BioSemi Active-One amplifiers with a band pass that was dependent on the sampling rate \cite{(0.16–512 Hz for SEPs and 0.16–128 Hz at 512 Hz for ssMRPs)}. EEG recordings were referenced to linked mastoids.

2.3. Electrical median nerve stimulation (experiment 1)

Ten paid volunteer adult participants \cite{(6 male and 4 female)} with a mean \cite{(± SD)} age of 46 \cite{(± 10)} years took part in the study with informed consent and local ethics committee approval. All subjects were right handed according to the Edinburgh Handedness Inventory \cite{(Oldfield, 1971)} and had normal or corrected to normal vision. Electrical median nerve stimulation was applied via surface electrodes at the wrist \cite{(intensity just above motor threshold)}). The stimulation consisted of square-wave pulses with a duration of 0.5 ms with an inter-stimulus interval of 500 ms. Median nerve stimulation was delivered in blocks of 3 min duration, during which subjects were instructed to maintain left or right gaze, in alternate blocks, for 4 blocks per condition.

2.4. Finger tapping task (experiment 2)

Twelve paid volunteer adult participants \cite{(7 male and 5 female)} with a mean \cite{(± SD)} age of 27 \cite{(± 5)} years took part in the study with informed consent and local ethics committee approval. All subjects were right handed, had normal or corrected to normal vision and good hearing. Subjects performed an auditory paced tapping task at 2 Hz with the right index finger. The right hand was concealed from view. A block consisted of 5 sets of 50 taps separated by short intervals of 5 s during which subjects could blink. The task was to maintain left or right gaze during a block,
for 10 alternating blocks per gaze direction, yielding 2500 taps for each condition. Half the subjects started in the left gaze condition and the other half in the right gaze condition. Taps were registered by a force transducer. Surface EMG recordings were taken from the ventral side (flexor muscles) and dorsal side (m. ext. indicis) of the right forearm.

2.5. SEP analysis

The continuous EEG was segmented into epochs comprising a pre and post stimulus interval (−50 to +250 ms), separately for each gaze direction. Individual epochs containing artefacts were rejected before averaging using a threshold of ±100 μV. SEP averages were band pass filtered (5–250 Hz) and were based on at least 1000 trials in each subject.

For statistical analysis of the gaze effect, the peak amplitudes and latencies of six components of the SEP waveforms, identified in the combined grand average, were compared using separate paired t tests. Amplitude and latency measures for each component were taken from an electrode pool which comprised electrode sites adjacent to and including the electrode with maximum amplitude for that component. SEP components selected for analysis were the N20 (pooled at P3, P5, P1, PPO3h, PPO7h, CPP3h, CPP5h), the N30 (Cz, C1, C2, CCP1h, CCP2h, FCC1h, FCC2h), the P45 (C1, C3, C5, FCC5h, CCP3h, CCP5h, CP3), the N65 (C1, Cz, FC1, FCz, FCC1h, FCC2h, FCC3h), the N100 (F1, Fz, FC1, FCz, FCC1h, FCC2h, FCC3h), and the N140 (P3, P5, CP3, CP5, CPP3h, CPP5h, TPP7h). To complement the analysis of component amplitudes, possible gaze effects on the spatial topography of the SEP components were investigated using analysis of variance (ANOVA) on the scalp voltage distributions at the component peak latencies (combined grand average), with factors Gaze (2) and Channel (128). Since the voltage distributions were normalised (vector norm) with respect to the average of each gaze condition as proposed by McCarthy and Wood (1985), a gaze effect would manifest itself as a significant Gaze by Channel interaction. Greenhouse-Geisser correction of the degrees of freedom was applied throughout this study, where appropriate.

2.6. SEP source analysis

The primary aim of source modelling in the present context is to provide a physiologically inspired means for data reduction to allow extraction and statistical comparison of a small number of measures, to complement the preceding SEP analysis. Source analysis on the 128 channel SEP waveforms for left and right gaze conditions from individual subjects was performed in Brain Electrical Source Analysis (BESA 2000, v.4.2.24, MEGIS Software GmbH, Munich, Germany) using the standard 4 shell ellipsoidal head model.

Following procedures from the literature (Franssen et al., 1992; Buchner et al., 1995) we initially fitted a spatiotemporal dipole model comprising a tangential cortical source (T), a radial cortical source (R) and a brain stem source (B) which accounted for SEP components in an interval from 10 to 100 ms after stimulus onset (the N140 could not be reliably localised in individual subjects). Statistical analysis of the dipole locations, orientations and (windowed) source waveforms did not yield any evidence of gaze effects. Although physiologically sound, this approach may be insensitive to a gaze effect involving relative increases in the spatial extent of cortical activation but no intensity change (Baker et al., 1999). Whilst a larger area of cortical activity might result in an increase in the depth (i.e. a decrease in eccentricity) of a cortical (regional) source, the presence of a deep brain source (B) could mask the observation of such a depth effect.

Thus, to determine whether gaze direction may have lead to a relative spatial increase in activity in sensorimotor cortical areas we adopted a less physiological approach using a single regional source fitted to the SEP waveforms over an interval from 0 to 200 ms, and focusing the statistical comparison on the depth (eccentricity) parameter of the source only (paired t test).

2.7. MRP formation and analysis

EEG recordings of the tapping task (experiment 2) were segmented into epochs with respect to force onset (−300 to +200 ms) for each gaze direction. Epochs containing eyeblink artefacts were rejected using a threshold criterion of ±100 μV and electrode Fpz as index channel. Any remaining artefacts in individual channels were rejected using the same threshold criterion. The epochs (at least 2400 per channel for each subject) were then averaged and baseline corrected to form steady-state MRP waveforms for left and right gaze conditions. Data from one subject were excluded because of absent pre-movement negative waves.

Similar to the analysis of SEP components, gaze effects on steady-state MRPs were investigated by considering the peak amplitude, peak latency and scalp distribution of four components of the MRP waveforms, identified in the combined grand average. As before, amplitude and latency measures for each component were taken from an electrode pool which comprised electrode sites adjacent to and including the electrode with maximum amplitude for that component. The MRP components selected for analysis were a broad pre-movement negative peak (preM1) at approximately −140 ms latency (pooled at C1, C3, Cz, CCP1h, CCP3h, FCC1h, FCC3h), a brief pre-movement positive deflection (preM2) at −40 ms latency (F1, Fz, FC1, FCz, FCC1h, FCC2h, FCC3h), a post-movement negative peak (postM1) at 27 ms latency (F1, F2, Fz, FCC1h, FCC2h, AFF1h, AFF2h) and a positive peak (postM2) at 150 ms latency (C1, Cz, FC1, FCz, FCC1h, FCC2h, FCC3h). The scalp voltage distributions at each component latency were normalised for each gaze condition (see Section 2.5) and
analysed using ANOVA with factors Gaze (2) and Channel (128), where gaze direction effect on MRP component topography would be indicated by a Gaze by Channel interaction.

2.8. MRP source analysis

Recent work by Gerloff et al. (1998) suggests that the main contributions to ssMRP waveforms come from cortical generators located bilaterally in central areas. Specifically, there is thought to be bilateral activation of pre-motor areas, and contralateral activation presumably in the primary motor and somatosensory areas.

Thus, MRPs in paced tapping can be adequately described by a 3 dipole source model, comprising two radial dipoles located approximately in the crown region of the pre-central gyrus, and one tangentially oriented source that accounts for primary motor and re-afferent activity in the anterior and posterior walls of the central sulcus.

Source analysis of the 128 channel MRP waveforms for left and right gaze conditions from individual subjects was carried out with the same tools as the source analysis of the SEP waveforms (see Section 2.6). The procedure for estimating the parameters of a contralateral radial source (Rc), an ipsilateral radial source (Ri) and a contralateral tangential source (Tc) over the 500 ms interval was as follows.

First, the positions of a pair of regional sources that were symmetric relative to a vertical plane of reflection along the midline were estimated, and the first orientation for both sources was fit at a peak around 140 ms prior to movement onset. The orientation tended to be radial for both sides. Next, the orientation of the second component of the contralateral source was determined at about 25 ms after tap onset and was consistently found to be tangential. The second and third components of the ipsilateral source and the third component of the contralateral source were switched off, and the orientations of the two bilateral radial components Rc and Ri as well as the contralateral tangential component Tc were optimised without orthogonality constraint. Thus, the locations of all three dipoles are specified by only one set of position coordinates, but the orientations were independent.

In addition to the evaluation of gaze effects on the spatial parameters of the model (especially eccentricity), the source waveforms of Rc, Ri and Tc were averaged over short time intervals (20 msec) and analysed for gaze effects using ANOVA with factors Gaze (2), Source (3) and Time (25).

2.9. Frequency domain analysis

The observed gaze effect in the fMRI study by Baker et al. (1999) was characterised by an increase in the spatial extent of activity in sensorimotor areas but not an increase in signal intensity. It is conceivable that these changes represent rhythmic activity that is not necessarily time-locked to the finger movements, and thus invisible to evoked potential analyses. A possible EEG correlate of such brain activity would be a modulation of frequency domain averaged (induced) power across various frequency bands at electrode sites overlying central sensorimotor areas. To investigate this possibility, the following frequency domain analysis was carried out separately on the data sets from experiments 1 and 2. Induced power and coherence analyses (Section 2.10) were performed using standard FFT and coherence methods implemented in BrainVision EEG analysis software (http://www.brainproducts.de).

EEG recordings for each gaze condition from individual subjects were segmented into stimulus (tap onset) locked intervals of 2 s duration, such that each segment contained 4 stimuli (tap onsets). Prior to determining the frequency spectrum of each segment, the data were detrended to minimise the effects of slow drifts and baseline corrected to remove the DC component. Segments containing eye blink artefacts were rejected using a threshold criterion of ±100 μV with reference to channel Fpz. Remaining artefacts in individual channels were rejected using the same threshold criterion. The power spectra of the remaining segments (approximately 150–300 per channel per subject for experiment 1, and between 350 and 600 per channel per subject for experiment 2) were computed and averaged to obtain the spectrum of induced power for left and right gaze condition. Spectral components at integer frequencies in the range of 2–30 Hz only were considered for analysis.

To focus on relevant areas, averaged power spectra were formed at three electrode pools that reflected the induced neural activity in central somatosensory and motor areas on the contralateral side (pool C3, FC3, CP3, FCC5h, FCC3h, CCP5h, CCP3h), the midline (Cz, FCz, CPz, FCC1h, FCC2h, CCP1h, CCP2h), and the ipsilateral side (C4, FC4, CP4, FCC4h, FCC6h, CCP4h, CCP6h). Gaze direction effects were investigated separately in different frequency bands using ANOVA with factors Gaze (2) and Pool (3). The frequency bands were the alpha band (8–12 Hz), and the lower and upper beta bands (13–21 and 22–30 Hz, respectively). Of interest were the main effects of Gaze and Gaze by Pool interactions.

2.10. Coherence analysis

Related to gaze dependent modulations of induced power might be changes in the degree of synchronisation of ongoing rhythmic activity within the cortical networks comprising primary sensorimotor, premotor and visuomotor areas. One aspect of synchronisation is frequency locking, which can be quantified using coherence, a measure of correlation in the frequency domain between two signals over a number of epochs. To determine whether there were any gaze related changes in functional coupling in central areas, coherence analysis was performed on electrode pairs connecting frontal, central and parietal areas within the contralateral and ipsilateral hemispheres. Selected electrodes within each of these regions were F1, F3, F5 (frontal),
C1, C3, C5 (central), and P1, P3, P5 (parietal) for the left hemisphere and their right hemisphere homologues. All possible frontal-central, frontal-parietal, and central-parietal connections were included in the analysis, pooled by region. The procedure was the same for the data sets from experiments 1 and 2, and was based on the 2 s data segmentation of the frequency domain analysis. As initial comparisons did not reveal any interhemispheric differences or interactions of hemisphere with gaze direction, we focussed the analysis on intrahemispheric gaze effects. For each hemisphere, separate ANOVAs with factors Gaze (2) and Connectivity (3) were carried out on (pooled) coherence in the alpha band and the lower and upper beta bands (see Section 2.9). Of interest were main effect of Gaze and the Gaze by Connectivity interaction.

3. Results

3.1. Analysis of SEP components

For each SEP component of interest a row of Fig. 1 shows the locations of constituent electrodes that formed the pool used for statistical analysis of gaze effects on peak latency and amplitude, the associated pooled grand average SEP waveforms (left gaze, right gaze and the difference), as well as scalp voltage distributions (left gaze, right gaze and the difference) at the corresponding peak latency, identified in the combined pooled grand average.

The lack of any appreciable difference between pooled SEP waveforms is supported by the absence of significant differences (α = 0.05) due to gaze in the peak latencies and amplitudes of the N20, N30, P45, N65, N100, and N140 components (see Table 1). The scalp voltage distributions for left and right gaze are practically indistinguishable and the left-from-right subtraction shows no systematic structure. Specifically, ANOVA of the normalised scalp voltage distributions showed no significant Gaze by Channel interaction at N20 (F(4,94,44.50) = 1.98, P = 0.10), N30 (F(5,04,45.35) = 0.75, P = 0.59), P45 (F(6,25,56.27) = 0.98, P = 0.45), N65 (F(4,30,38.67) = 1.09, P = 0.38), N100 (F(4,00,35.96) = 1.07, P = 0.39), and N140 (F(4,32,38.89) = 1.07, P = 0.39) latencies. Hence, gaze direction did not modulate SEP waveforms or spatial topography.

Fig. 1. Pooled waveforms for different SEP components with peak latency scalp voltage distributions for left gaze, right gaze, and their subtraction.
3.2. SEP source analysis

The single regional source model accounted sufficiently well for the early and medium latency SEP waveforms in all subjects for left gaze (mean RV ± SD; 11.6 ± 4.5%) and right gaze (11.7 ± 4.8%) conditions, without difference in goodness of fit between conditions. The source was consistently localised in the contralateral sensorimotor area in the vicinity of the central sulcus, with a negligible difference in location between left and right gaze conditions (mean location difference ± SD (mm); x = -0.28 ± 2.17; y = 0.00 ± 1.77; z = 0.17 ± 1.04), and no significant difference in source depth between left and right gaze conditions (mean eccentricity ± SD; left: 0.703 ± 0.031; right: 0.697 ± 0.032; t(9) = 1.5, P = 0.17). Thus there is no evidence that gaze direction altered the spatial extent of cortical activation underlying SEP waveforms.

3.3. Analysis of MRP components

Fig. 2 (top) illustrates that there were no differences between the grand average steady-state MRP waveforms for left and right gaze conditions at any of the 128 electrode sites. For each steady-state MRP component of interest, a row in Fig. 2 (bottom) shows the locations of constituent electrodes that formed the electrode pool used for statistical analysis of gaze effects on peak amplitude and latency, the associated pooled grand average MRP waveforms (left, right, and right–left), as well as scalp voltage distributions (left, right, and right–left) at the corresponding peak latency, identified in the combined pooled grand average.

There is no visible difference between pooled MRP waveforms, and this is corroborated by the absence of significant differences (α = 0.05) due to gaze direction in the peak latencies and amplitudes of the preM1, preM2, postM1 and postM2 components (see Table 2). The scalp voltage distributions for left and right gaze are virtually indistinguishable and the left-from-right subtractions show no systematic structure. Specifically, ANOVA of the normalised scalp voltage distributions showed no significant Gaze by Channel interaction at preM1 (F(5,88, 58.79) = 0.72, P = 0.63), preM2 (F(5,76, 57.56) = 0.71, P = 0.64), postM1 (F(5,03, 50.23) = 0.83, P = 0.54) and postM2 (F(4,52, 45.19) = 1.15, P = 0.35) latencies. Thus, gaze direction did not differentially affect steady-state MRPs.

3.4. MRP source analysis

The 3 dipole model accounted sufficiently well for the ssMRP waveforms in all subjects for left gaze (mean RV ± SD; 11.4 ± 5.1%) and right gaze (11.8 ± 5.4%) conditions, without difference in goodness of fit between gaze conditions. Fig. 3 shows the averaged locations, orientations and source waveforms for left and right gaze of the three dipoles that accounted for the ssMRP. The sources are located bilaterally in the central sulcus without an appreciable difference in location between gaze conditions (mean location difference ± SD (mm); x = ± 0.42 ± 1.38; y = ± 0.39 ± 1.77; z = ± 0.62 ± 1.82) and practically no difference in orientation. In particular, there was no significant difference in source depth between gaze conditions (mean eccentricity ± SD; left: ± 0.60 ± 0.071; right: ± 0.66 ± 0.067; t(10) = 0).

Comparison of the windowed source waveforms revealed no significant main effect or interaction involving Gaze (Gaze: F(1, 6) = 0.16, P = 0.71; Gaze by Source: F(1,22, 7.35) = 0.02, P = 0.92; Gaze by Time: F(3,45, 20.80) = 0.1.24, P = 0.32; Gaze by Source by Time: F(3,86, 23.15) = 1.18, P = 0.34). Thus gaze direction did not affect the spatial distribution or activation time course of cortical neural generators of the ssMRP.

3.5. Frequency domain and coherence analysis

For the comparison of ongoing cortical activity during 2 Hz electrical median nerve stimulation between gaze
conditions there were no significant main effects or interactions involving Gaze on (pooled) induced power or intrahemispheric coherence in the alpha, or the lower and upper beta bands.

Fig. 4 shows the spectra of induced power (top) during the 2 Hz finger tapping task from experiment 2, as well as scalp power distributions at selected frequencies (bottom). There was no significant main effect or interaction involving
Gaze on (pooled) induced power in the alpha band. In the lower and upper beta bands, there was also no significant main effect of Gaze, but the Gaze by Pool interactions were significant (lower beta: $F(1.30, 13.02) = 4.84, P = 0.039$; upper beta: $F(1.26, 12.63) = 4.54, P = 0.046$). As further detailed below, this appears to be due to EMG activity from frontal scalp muscles.

There were no significant main effects or interactions of Gaze in any of the frequency bands (alpha, lower beta, upper beta) in intrahemispheric cortico-cortical coherence during 2 Hz finger tapping.

### 4. Discussion

The manipulation of gaze direction failed to elicit differences in peak latency, amplitude and topographical measures of the early and medium latency SEP components, and the pre- and post-movement steady-state MRP components. There was also no reliable gaze dependent change in the depth of cortical regional sources modelling the neural generators for SEPs and MRPs in somatosensory and motor areas, or on the time course of the source waveforms underlying MRPs. This result suggests that there was no gaze dependent increase in the spatial extent of phase-locked cortical activity during median nerve stimulation or 2 Hz finger tapping. Using camera monitoring or subject reports (see also Baker et al., 1999), we could confirm that lateral gaze in the required direction was maintained during each block. Hence, the absence of a gaze effect is unlikely to be explained by a failure of subjects to perform the tasks.

As to the frequency domain analyses, the results from the analysis of induced power and coherence for both electrical median nerve stimulation and 2 Hz finger tapping.

#### Table 2

<table>
<thead>
<tr>
<th>Component</th>
<th>Peak measure</th>
<th>Left (mean ± SD)</th>
<th>Right (mean ± SD)</th>
<th>t-value (df = 10)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>PreM1</td>
<td>Latency (ms)</td>
<td>$-141.70 ± 11.37$</td>
<td>$-143.64 ± 23.14$</td>
<td>0.30</td>
<td>0.77</td>
</tr>
<tr>
<td></td>
<td>Amplitude (μV)</td>
<td>$-2.48 ± 1.14$</td>
<td>$-2.52 ± 1.14$</td>
<td>0.50</td>
<td>0.63</td>
</tr>
<tr>
<td>PreM2</td>
<td>Latency (ms)</td>
<td>$-30.89 ± 8.59$</td>
<td>$-32.49 ± 12.99$</td>
<td>0.45</td>
<td>0.66</td>
</tr>
<tr>
<td></td>
<td>Amplitude (μV)</td>
<td>$0.79 ± 0.83$</td>
<td>$0.75 ± 0.87$</td>
<td>0.57</td>
<td>0.58</td>
</tr>
<tr>
<td>PostM1</td>
<td>Latency (ms)</td>
<td>$28.58 ± 5.04$</td>
<td>$30.01 ± 7.22$</td>
<td>0.75</td>
<td>0.47</td>
</tr>
<tr>
<td></td>
<td>Amplitude (μV)</td>
<td>$-1.70 ± 1.24$</td>
<td>$-1.72 ± 1.10$</td>
<td>0.20</td>
<td>0.85</td>
</tr>
<tr>
<td>PostM2</td>
<td>Latency (ms)</td>
<td>$148.79 ± 10.44$</td>
<td>$151.28 ± 6.50$</td>
<td>0.75</td>
<td>0.47</td>
</tr>
<tr>
<td></td>
<td>Amplitude (μV)</td>
<td>$2.91 ± 1.71$</td>
<td>$2.81 ± 1.57$</td>
<td>0.74</td>
<td>0.48</td>
</tr>
</tbody>
</table>

Gaze on (pooled) induced power in the alpha band. In the lower and upper beta bands, there was also no significant main effect of Gaze, but the Gaze by Pool interactions were significant (lower beta: $F(1.30, 13.02) = 4.84, P = 0.039$; upper beta: $F(1.26, 12.63) = 4.54, P = 0.046$). As further detailed below, this appears to be due to EMG activity from frontal scalp muscles.

There were no significant main effects or interactions of Gaze in any of the frequency bands (alpha, lower beta, upper beta) in intrahemispheric cortico-cortical coherence during 2 Hz finger tapping.

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The manipulation of gaze direction failed to elicit differences in peak latency, amplitude and topographical measures of the early and medium latency SEP components, and the pre- and post-movement steady-state MRP components. There was also no reliable gaze dependent change in the depth of cortical regional sources modelling the neural generators for SEPs and MRPs in somatosensory and motor areas, or on the time course of the source waveforms underlying MRPs. This result suggests that there was no gaze dependent increase in the spatial extent of phase-locked cortical activity during median nerve stimulation or 2 Hz finger tapping. Using camera monitoring or subject reports (see also Baker et al., 1999), we could confirm that lateral gaze in the required direction was maintained during each block. Hence, the absence of a gaze effect is unlikely to be explained by a failure of subjects to perform the tasks.

As to the frequency domain analyses, the results from the analysis of induced power and coherence for both electrical median nerve stimulation and 2 Hz finger tapping.

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Fig. 3. Source waveforms and dipole source locations for the ipsilateral and contralateral cortical generators of ssMRP components, shown for left and right gaze direction conditions.
tapping do not provide compelling evidence for gaze effects on sources of activity located in contralateral central sensorimotor areas. Extrapolating from the findings by Baker et al. (1999) one might have expected to observe relative increases in spectral power at the hemisphere contralateral to the side of movement or sensory stimulation when gaze direction was towards that side, relative to the condition where gaze was incongruent with the side of movement or sensory stimulation. While Fig. 4 shows slightly higher power in the beta range with right directed gaze than with left directed gaze, this difference did not remotely approach significance and did not show the required topographic specificity for the left hemisphere.

For median nerve stimulation, there was no evidence of any gaze related change in induced power, but spectral analysis of ongoing EEG during the tapping task did show significant interactions between gaze direction and electrode pools (induced power) in the lower and upper beta bands. Visual inspection of the full scalp topography of gaze related power changes, however, revealed gaze modulations above the left and right eye, extending up to frontocentral electrodes included in lateral electrode pools. The gaze modulation at these locations consisted of an increased power at frontal sites above the abducting eye (i.e. right eye for right gaze and left eye for left gaze). The directional pattern and the topography of this modulation suggested that it might be due to contamination of the EEG by EMG from frontal scalp muscles, i.e. the m. frontalis. This contamination with EMG potentially explained the effects in induced power and was experimentally verified in one subject with needle EMG recordings from the m. frontalis. This confirmed increased EMG at the right recording site during rightward gaze relative to leftward gaze and vice versa, especially with a relatively low position of the chin rest. Interestingly, on further inspection of the data, similar gaze-related power changes with a maximum directly above the eyes were present in experiment 1, providing further support for the subjects’ compliance with the task. However, only in the case of data from experiment 2 this EMG contamination showed up in significant effects in power analyses.

Another possible effect of gaze direction on ongoing rhythmic cortical activity during median nerve stimulation and finger tapping might be a relative change in frequency locking between parietal visuomotor areas and frontocentral sensorimotor areas, particularly on the contralateral side, exhibited by changes in coherence in higher frequency
bands between frontal-parietal, central-parietal and frontal-central electrode pairs. One outcome might have been a relative increase in coherence on the contralateral side when gaze direction coincided with the side of stimulation (movement). However, neither in experiment 1 nor in experiment 2 there was evidence of significant gaze related changes in coherence in either hemisphere.

Given the close association between spatial attention and gaze direction, the absence of gaze direction effects on SEPs, in the present study, may seem at odds with earlier studies reporting spatial attention effects on SEPs (e.g. Garcia-Larrea et al., 1991; Iguchi et al., 2001). It should be noted, however, that the reported spatial attention effects involved comparisons between conditions where attention was or was not focused on the limb/body part that received the stimuli eliciting SEPs. In other words, the presumed spatial attention effects were mediated by proprioceptive body awareness. Related proprioceptive mechanisms, supporting crossmodal sensory integration, may enhance tactile acuity and amplify tactile SEPs when vision is directed at the stimulated body part (Taylor-Clarke et al., 2002). It is unlikely, however, that the frames of reference for attention involved comparisons between conditions where attention was or was not focused on the limb/body part that received the stimuli eliciting SEPs. In other words, the presumed spatial attention effects were mediated by proprioceptive body awareness correspond in a straightforward way (e.g. Flanders et al., 1992). Hence, the absence of a gaze effect on SEPs in this study need not be inconsistent with previous findings.

In summary, the present study did not find any EEG correlates of gaze-dependent activity modulation in sensorimotor areas, as was found previously with fMRI by Baker et al. (1999). This may be due to the use of a simple finger tapping task and passive sensory stimulation, which did not require visual input processing. Under similar conditions, the fMRI effects reported by Baker et al. consisted of an expansion of the active area in the examined brain regions, i.e. increases in the number of active voxels. It is conceivable that such changes are more difficult to recover from EEG than true changes in signal intensity. These results do not, therefore, disqualify EEG for the investigation of interactions between oculomotor position signals and skeletomotor control.

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