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Effects of Pointing Direction and Direction Predictability on Event-related Lateralisations of the EEG

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Abstract. In two experiments, we investigated hemispheric EEG differences in 9(12) healthy volunteers during pointing to lateral and central targets. The questions addressed were whether horizontal pointing direction and the predictability of pointing direction modulated hemispheric differences (event-related lateralisations of the EEG = ERLs). To vary pointing direction predictability, targets were displayed either randomly at one of nine different positions on a screen ('random') or at the same horizontal position in five subsequent trials ('sequenced') while vertical positions varied randomly. ERLs varied with pointing direction. This was true across changes in target eccentricity and pointing distance. Foci of the ERLs were in premotor, motor and parietal cortex, reflecting the critical involvement of these areas in the control of visually guided reaching. Direction predictability reduced the parietal ERL before pointing onset, probably reflecting a lesser effort in visuomotor transformation. Predictability also added an additional component to the early ERLs after target onset and increased direction effects during movement.

Introduction

To accurately reach towards a target, visual and somatosensory information have to be integrated and transformed into the appropriate motor output. It is believed that several areas of the brain are involved in the underlying neural processes in a network of fronto-parietal connections (for review, see Glickstein et al., 2000). At one 'end' of this network, posterior parietal cortex is known to be involved in the control of goal directed reaching movements, coding the location of the target in motor coordinates (for review, see Laquantini & Caminti, 1998). At the other 'end', premotor neurons receive visual input from parietal cortex and project to motor cortex.

Little is known about the temporal aspects of neural processing for reaching movements in humans. Electroencephalography (EEG) is a means of investigating cortical processes with high temporal resolution. Here, we studied hemispheric differences in EEG activity during visually triggered pointing movements in humans. To analyze EEG asymmetries (event-related lateralisations = ERLs), activity at electrode sites ipsilateral to the response or the stimulus is subtracted from that at the corresponding contralateral sites (Verleger et al., 2000; Wascher & Waschkuhn, 1996; Eimer, 1996). Contra-ipsilateral EEG asymmetries were first used to analyze movement preparation (Gratton et al., 1988; De Jong et al., 1988) and revealed the well-investigated component called LRP (lateralized readiness potential) over the contralateral motor cortex, which reflects selection of an effector prior to movement execution. Another lateralized but non-motor component is the N2pc (N2 posterior contralateral), which is thought to reflect the selection of task relevant stimuli (Luck&Hilliard, 1994a; Luck&Hilliard, 1994b). We applied this subtraction method to electrode sites distributed over the whole scalp (illustrated in Figure 1) to gain insights into the interaction of the different cortical areas involved in visuomotor coordination (Wascher & Waschkuhn, 1996).

Due to the observation of consistent differences in speed and accuracy between reaches to targets presented in the contralateral visual field and reaches to ipsilateral targets, it has been suggested that hemispherically organized neural systems are involved in the programming of visually guided movements (Fisk & Goodale, 1985). The focus of the present study is on this directional aspect of the pointing movements that is pointing to a target in the ipsilateral or contralateral hemifield with respect to the pointing arm. Data from two different experiments will be presented, in which participants pointed to a target that could appear at different positions on a screen. The horizontal position of the target determined, which one of the three types of pointing movements was required. These movements differed in pointing direction: contralateral (from a right starting position to a target on the left side of the screen or vice versa), ipsilateral (from a right starting position to a right-sided target or from a left starting position to a left-sided target), and central (from a left or right starting position to a target on the centerline of the screen). This allowed us to study effects of target position, and consequently the movement

direction, on hemispheric EEG asymmetries at different times during processing.
Additionally, we examined, whether repeating the horizontal target position, which corresponded to pointing direction, had an effect on visuomotor processing. If the target repeatedly appears in the same horizontal position, that is when a similar response is required repeatedly, the task difficulty should decrease. This should result in a reduced processing effort, which would be reflected by decreased cortical activity in the corresponding brain areas. On the other hand, predictable target position may increase visuospatial attention in the respective hemifield or enhance possible automated visuomotor activations (Eimer, 1995; Wascher & Waschkuhn, 1996), which would be reflected by increased activation in the cortical areas concerned with those computations.
Our aim was to identify components of visuomotor transformation from target onset to movement execution that are (a) reflected in contra-ipsilateral EEG asymmetries and (b) modulated by pointing direction and/or direction predictability and the cortical areas, which may be involved in those processes.

Experiment 1

In Experiment 1, we investigated whether pointing to ipsilateral, central or contralateral targets with respect to the active arm changed ERLs from target onset to movement execution and whether these effects were modulated by the predictability of pointing direction.

Methods

Subjects

Event-related potentials (ERPs) were recorded from nine right-handed healthy participants (6 females). Age ranged from 17 to 27 (average 20.7 years). Vision was normal or corrected to normal.

Apparatus

During the recording, participants were seated in a soundproof EEG cabin, shielding electromagnetic fields. Participants sat on a chair with their chin and forehead fixed in a headrest. Stimuli were presented on a 21" computer monitor that was located 47 cm in front of the participants. The center of the screen was aligned with participants' eye height. At the beginning of each trial, the hands were placed in the starting positions on a table in 27 cm distance to the participants and 17 cm distance between the left and right hand position. When in the starting positions, participants' forearms and upper arms were aligned in a 90 deg. angle. Starting positions were haptically detectable small ridges, which the index fingers rested on. A shield prevented the participants from seeing their hands in the starting positions. The hand used for pointing became visible when it approached the screen during a pointing movement. Lights in the cabin were turned off during the recording.

Stimuli

Targets were white filled circles (1cm or 0.85 deg. of visual angle) on a black background. A trial started with a fixation cross that stayed on for 200 ms in the center of the screen. After an ISI of 750 ms (+/- 250 ms) the target was presented and stayed visible for 1500 ms. The target could appear in one of nine different positions, which were spaced on the nodes of a 3*3 grid with a vertical distance of 9.78 deg. (11.5 cm) and a horizontal distance of 8.01 deg. (9.5 cm). The central target was in the center of the screen. Hence, pointing to a target contralateral to the active arm required a movement across the body center and pointing to an ipsilateral target a more or less straightforward movement.

Procedure

In one part of the experiment, targets were presented randomly in one of the nine positions ('random'). In the other part, the horizontal position of the target was the same in five subsequent trials, whereas the vertical position varied randomly ('sequenced'). Therefore, in four out of five trials in the 'sequenced' condition, the participants could predict the horizontal position of the target and the horizontal direction of the pointing movement.
Participants were instructed to keep both hands in the starting positions before and after each pointing movement with the index fingers extended. Once the target appeared, participants were to point as quickly and accurately as possible with one hand and touch the target on the screen briefly with the index finger. Participants were instructed not to slow down before touching the screen, thereby avoiding visually guided corrections of the trajectory. The movement should be completed, (i.e., the hand should be back in the starting position) when the target disappeared.
Participants performed four blocks. The 'random and sequenced' conditions were performed with the left and right hand in separate blocks. The hand to be used was changed in subsequent blocks in order to avoid tiring of the arms. The order of blocks was counterbalanced. The 'random' condition blocks consisted of 450 trials (9 positions * 50 repetitions).

In the 'sequenced' condition, the blocks consisted of 900 trials (3 horizontal positions * 5 trials in a sequence * 60 repetitions). The trajectories of the pointing movements were recorded by means of an ultrasonic tracking device (ZEBRIS system). For this purpose, a marker was fixed to the tip of the index finger. ZEBRIS data were used to compute response times, response-locked potentials and to exclude invalid trials.

Recording

EEG was recorded with Ag/AgCl electrodes from 53 scalp positions distributed over the head. An electrode attached to the tip of the nose was used as reference. Vertical EOG (vEOG) was recorded bipolarly from above and below the right eye and horizontal EOG (hEOG) from the outer canthi of both eyes. EEG and EOG were amplified and filtered by seven PSYLAB amplifiers (EEG8) with a 5.31s time constant and a 0.03 Hz - 35 Hz bandpass. EEG and EOG were digitized at 100 Hz for a period of three seconds, starting 190 ms before the fixation cross. The PC that presented the stimuli triggered ZEBRIS and EEG recordings simultaneously.

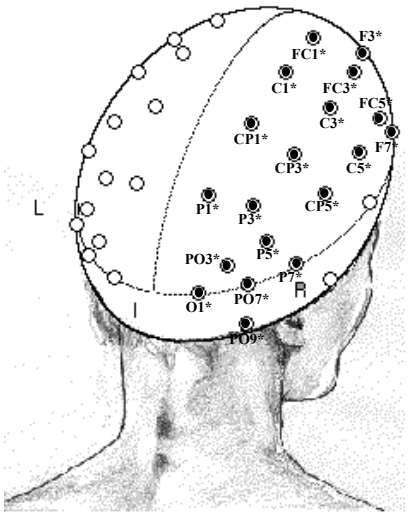

Fig. 1: Map of electrode sites, for which difference potentials were calculated. Names of sites are given only in the right hemisphere. The asterisk indicates the fact that contralateral and ipsilateral electrode sites were combined in the difference potential (i.e. P1* = P1/2). F stands for frontal, FC for fronto-central, C for central, CP for centro-parietal, P for parietal, PO for parieto-occipital and O for occipital areas.

Data Processing and Analysis

Trials with zero lines, out-of-scale values, slow drifts larger than 80 µV in the measurement and fast shifts larger than 120 µV / 500 ms were excluded from further analyses. The transmission of vEOG and hEOG into the EEG was estimated separately in areas of maximum EOG variance, and was subtracted from the EEG data.

Response time was defined as the moment when the finger was at least 20 mm away from the starting position. Only trials in which the movement started with a minimal response time of 100 ms after target onset and which met an accuracy criterion (15 mm maximal distance to the target when participants first touched the screen) were analyzed. The 3D trajectories were reconstructed, but will not be reported here.

ERL: To assess hemispheric EEG differences, difference potentials were calculated for 21 electrode pairs (see Figure 1): Activity at the electrode site ipsilateral to the moving arm was subtracted from the activity at the corresponding contralateral electrode. This was done for left arm and right arm movements separately and the two difference waves were averaged. To better render visible the effect of horizontal target position on hemispheric asymmetries, activity at electrode sites ipsilateral to the hemifield, in which the target appeared, was subtracted from the activity in the contralateral hemisphere. ERLs were averaged across trials and participants (grand mean), time locked to the onset of the target (stimulus-locked) or time locked to the start of the pointing movement (response-locked).

For statistical analysis, ANOVAs with two within-subjects factors - target predictability (levels 'sequenced' and 'random') and target position (levels 'ipsilateral', 'central', and 'contralateral' with respect to pointing arm) - were computed. F-statistics of the ANOVA were corrected by Greenhouse Geyser Epsilon. Paired two-tailed t-tests were performed to compare 'early' and 'late' trials in the 'sequenced' condition. For this purpose, we computed means of the first two trials in a sequence and the last two trials, respectively.

Results

From the time of target onset to the ongoing pointing movement, we identified different ERL components. Figure 2 shows stimulus-locked difference potentials at exemplary electrode pairs. The magnitudes of components were measured as mean amplitudes in time intervals in which maximal amplitudes were evident in the grand means. If effects of factors on the latency of maximal amplitudes were evident, peak amplitudes and latencies were measured in the corresponding time interval.

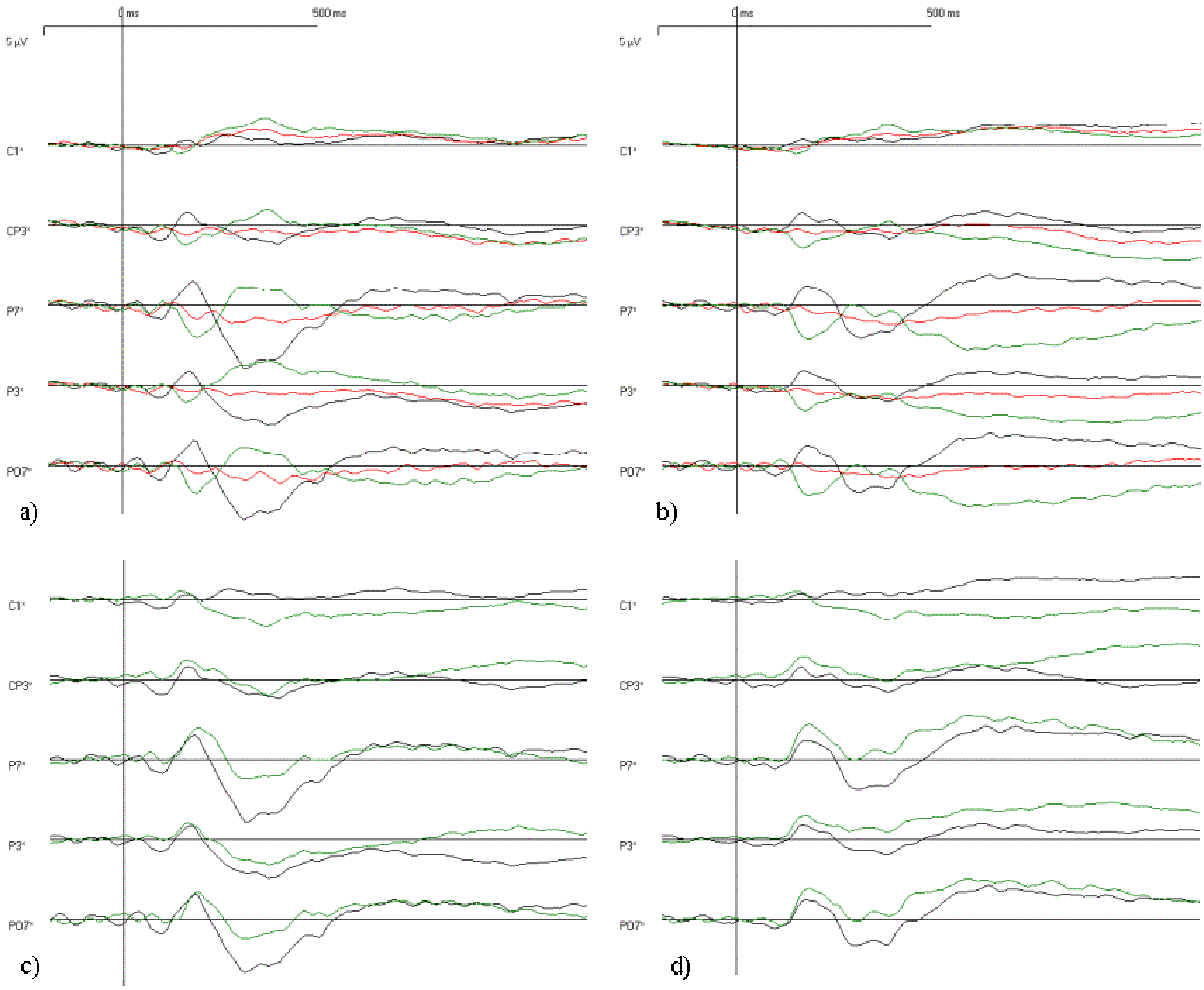

Fig. 2: Stimulus-locked difference potentials at exemplary central and parietal sites. a) and b) hemisphere contralateral to response plotted upwards, c) and d) hemisphere contralateral to target up. a) and c) 'random', b) and d) 'sequenced'. Black lines: targets ipsilateral to response side, green lines: contralateral targets, red lines: central targets. 0 ms is target onset. Note the ERL component at ca. 180 ms after target onset with higher activity in the hemisphere contralateral to the target; the ERL at around 350 ms with higher activity in the hemisphere ipsilateral to the target, when compared with central targets; and an ERL after movement onset at many sites, in which activity is higher in the hemisphere contralateral to the target.

a) In stimulus-locked averages, an early component after target onset was most prominent at lateral parieto-occipital and parieto-temporal sites (PO7/8 and P7/8 pairs) and spread up to anterior sites. Figure 3 illustrates scalp topographies of the early and intermediate ERLs. The magnitude of this ERL component was measured as the mean amplitude between 150 and 190 ms after target onset. When targets were lateralized, activity was greater in the hemisphere contralateral to the target. Though this effect of target position had greater amplitudes at lateral posterior sites, it only marginally reached significance there.

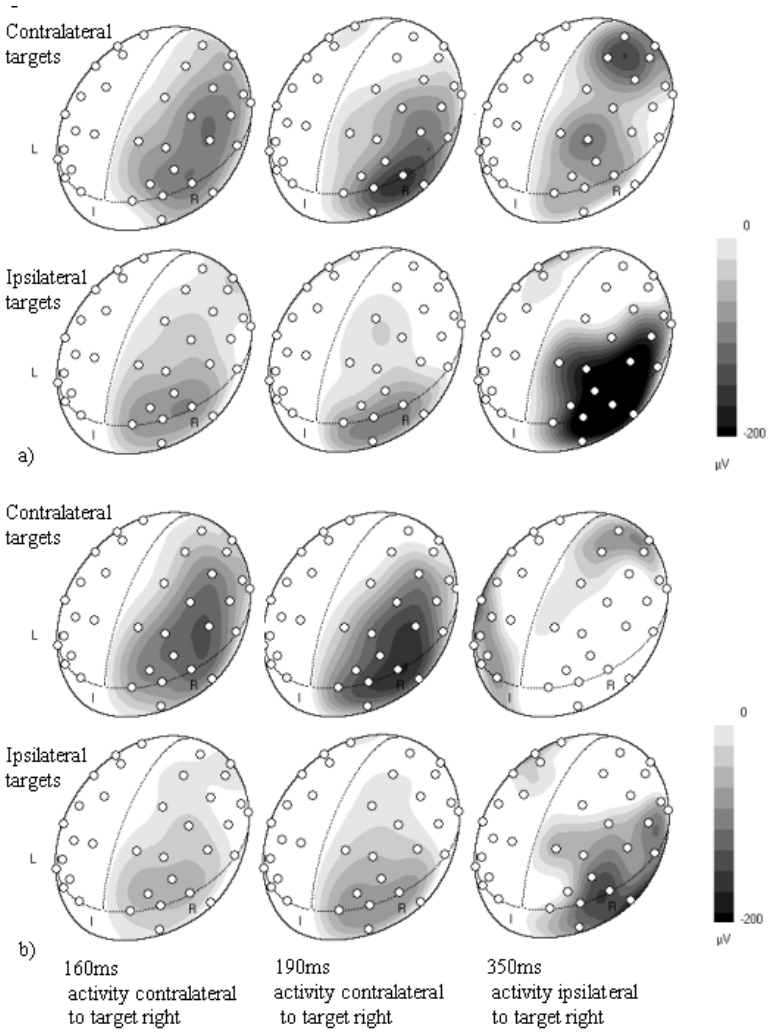

Fig. 3: Topography maps of stimulus-locked difference potentials at 160 ms, 190 ms and 350 ms. a) 'random, b) 'sequenced'. At 160 ms and 190 ms, the right hemisphere shows activity that was higher contralateral to the target and at 350 ms, activity that is higher ipsilateral to the target. Note the evidence of the early ERL at anterior sites at 160 ms when compared to 190 ms. In the 'random' condition, parietal activity at 350 ms is higher in the hemisphere ipsilateral to the target, whereas in the 'sequenced' condition, this effect was decreased for ipsilateral targets and not visible for contralateral targets. Note though, that the relative deviance from activity with central targets cannot be regarded in this kind of averages. Frontal activity at 350 ms is higher in the hemisphere contralateral to the response side.

Table 1 contains levels of significance with $p < .05$ for the discussed effects. Significant effects were found at central and centro-parietal sites (CP5/6, CP3/4, C1/2 and C3/4). Significance levels at posterior sites with $p > .05$ and $p < .07$ were: $F(2/16)=4.5$, $p=.067$ at P7/8; $F(2/16)=4.9$, $p=.057$ at P5/6; $F(2/16)=4.7$, $p=.062$ at P3/4, and $F(2/16)=5.0$, $p=.054$ at PO3/4. Remarkably, this component peaked about 15-30ms earlier at fronto-central and central (premotor and motor) sites, than at posterior (primary visual) sites ($t(8)=5.1$, $p=.001$).

The ERL did not differ in amplitude with target predictability (except for site O1/2), but tended to show a second peak around 240 ms after target onset in the 'sequenced' condition. This was measured as the mean amplitude between 220 ms and 260 ms after target onset. In the 'random' condition, a second peak was vaguely visible with contralateral stimuli, but not with ipsilateral stimuli. When 'early' and 'late' trials in a sequence were compared in the 'sequenced' condition, greater ERL amplitudes were evident for 'late' trials at lateral central sites (C5/6, C3/4). At central and parietal sites, the second peak was more prominent in 'late' trials.

Table 1
F-, t-, and p-values of effects of factors direction and predictability on mean amplitudes in experiment 1.

component	effects of factor on component	electrode sites													
		C5/6	C3/4	C1/2	CP5/6	CP3/4	CP1/2	P7/8	P5/6	P3/4	P1/2	PO9/10	PO7/8	PO3/4	O1/2
early ERL	direction		F(2,16)=6.3 p=.034		F(2,16)=5.9 p=.040	F(2,16)=6.0 p=.040									
	predictability														F(1,8)=9.9 p=.014
	interaction														
	'late' > 'early'	t(8)=3.7 p=.006	t(8)=3.1 p=.015												
	2nd peak in 'sequenced' vs. 'random'				t(8)=-3.2 p=.013; ipsi			t(8)=-3.0 p=.017; ipsi		t(8)=-3.1 p=.014;ipsi			t(8)=-2.7 p=.029; ipsi	t(8)=-2.8 p=.022; ipsi t(8)=3.1 p=.015; contra	t(8)=2.5 p=.038; contra
	2nd peak in 'late' vs. 'early'	t(8)=2.5 p=.035	t(8)=3.7 p=.006	t(8)=3.4 p=.009		t(8)=2.8 p=.024				t(8)=3.2 p=.014	t(8)=3.1 p=.016				
intermediate ERL	direction	F(2,16)=6.2 p=.028			F(2,16)=6.6 p=.025			F(2,16)=32.3 p<.001	F(2,16)=8.0 p=.016	F(2,16)=16.6 p=.002	F(2,16)=6.8 p=.026	F(2,16)=6.4 p=.025	F(2,16)=23.5 p=.001		
	predictability														
	interaction				F(2,16)=13.6 p=.004 a)			F(2,16)=7.3 p=.022 a)	F(2,16)=5.6 p=.040 a)	F(2,16)=9.7 p=.014 a)				F(2,16)=10.3 p=.011a)	F(2,16)=6.6 p=.031a)
	'early' > 'late'	t(8)=3.3 p=.012	t(8)=4.5 p=.002			t(8)=3.5 p=.008				t(8)=2.8 p=.024					
late ERL	direction		F(2,16)=4.2 p=.050		F(2,16)=9.0 p=.011			F(2,16)=7.1 p=.027				F(2,16)=14.8 p=.003	F(2,16)=5.5 p=.047		F(2,16)=7.7 p=.021
	predictability														
	interaction		F(2,16)=6.4 p=.031a		F(2,16)=10.3 p=.008 a)			F(2,16)=6.8 p=.026 a)		F(2,16)=7.8 p=.022 b)				F(2,16)=6.4 p=.033 a)	
	'early' vs. 'late'														

Notes. No significant effects were found at frontal electrodes. 'Ipsi' and 'contra' stand for ipsilateral and contralateral targets with respect to the response side. It denotes, for which levels of factor 'direction' the differences between 'sequenced' and 'random' trials were significant. a) In the 'random' condition, the direction effect was reduced or absent. b) In the 'random' condition, the direction effect was inverted. Only significant effects with $p < .05$ are shown.

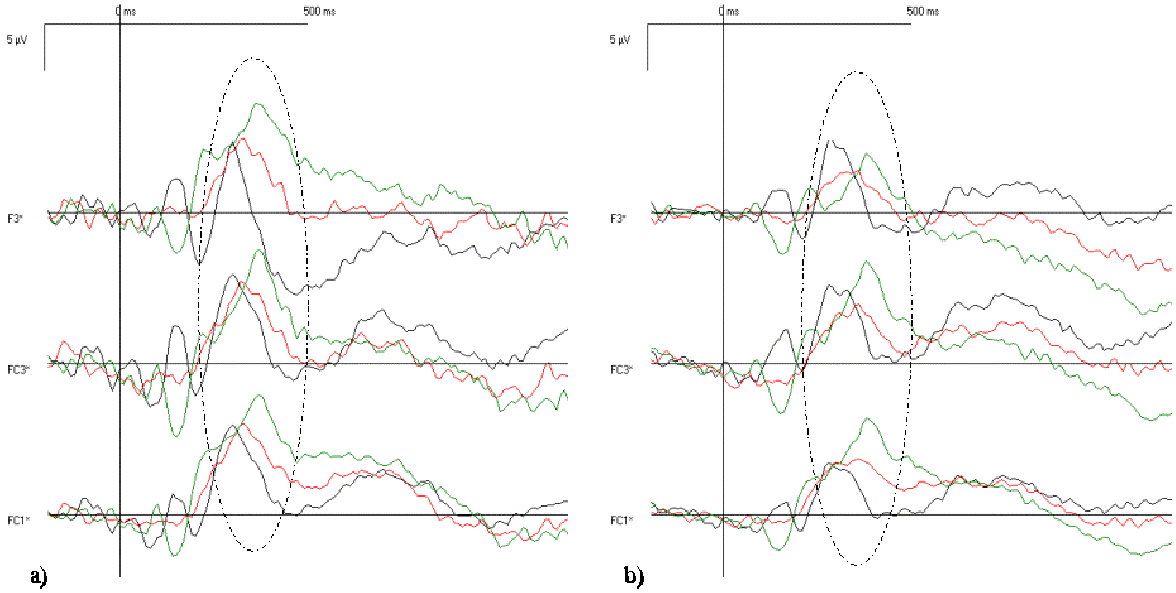

Fig. 4: Stimulus-locked difference potentials at exemplary anterior sites. Hemisphere contralateral to response plotted upwards. a) 'random', b) 'sequenced'. Black lines: targets ipsilateral to response side, green lines: contralateral targets, red lines: central targets. 0 ms is target onset. Note latency differences for intermediate ERL peaks (broken ellipses): ipsilateral < central < contralateral targets.

b) A rather long-lasting intermediate ERL component was different at anterior and posterior sites. Figure 4 shows ERLs at anterior electrode pairs.

The posterior component had it's maximum at parieto-temporal and parieto-occipital sites (P7/8, PO7/8) and spread up to central (motor) sites. It was measured as the mean amplitude between 280 and 430 ms from target onset in stimulus-locked averages and lasted further than movement onset (average response time was 423 ms in the 'random' condition and 417 ms in the 'sequenced' condition). When stimuli were presented laterally, ERLs deviated from those with central targets towards the hemisphere ipsilateral to the target. When stimuli were central, activity tended to be greater in the hemisphere ipsilateral to the pointing arm at parietal and parieto-occipital sites and in the contralateral hemisphere at central sites, thereby shifting the 'baseline'. Target predictability ('sequenced' vs. 'random') did not have a main effect on this component, but interacted with target position such that in the 'sequenced' condition, the direction effect was smaller or absent. Comparably, 'early' trials in the sequenced condition showed a greater intermediate ERL than 'late' trials. Difference potentials for 'early' and 'late' trials are plotted in Figure 5.

c) At frontal (F3/4) and fronto-central sites (FC3/4 and FC1/2), the intermediate ERL component differed from that at central, parietal and parieto-occipital sites. For all target positions, ERLs were greater in the hemisphere contralateral to the responding arm, but peak latency, measured in the same time interval as the posterior part (between 280 ms and 430 ms after target onset), varied with target position (F3/4, FC3/4 and FC1/2) and with predictability (F3/4 and FC1/2). ERLs peaked earlier when stimuli were presented ipsilateral to the active arm and latest with contralateral targets and earlier in 'random' than in 'sequenced' trials, respectively. At site FC1/2 peak amplitude varied with predictability and was higher in the 'random' condition. Levels of significance for the anterior intermediate ERL are shown in Table 2.

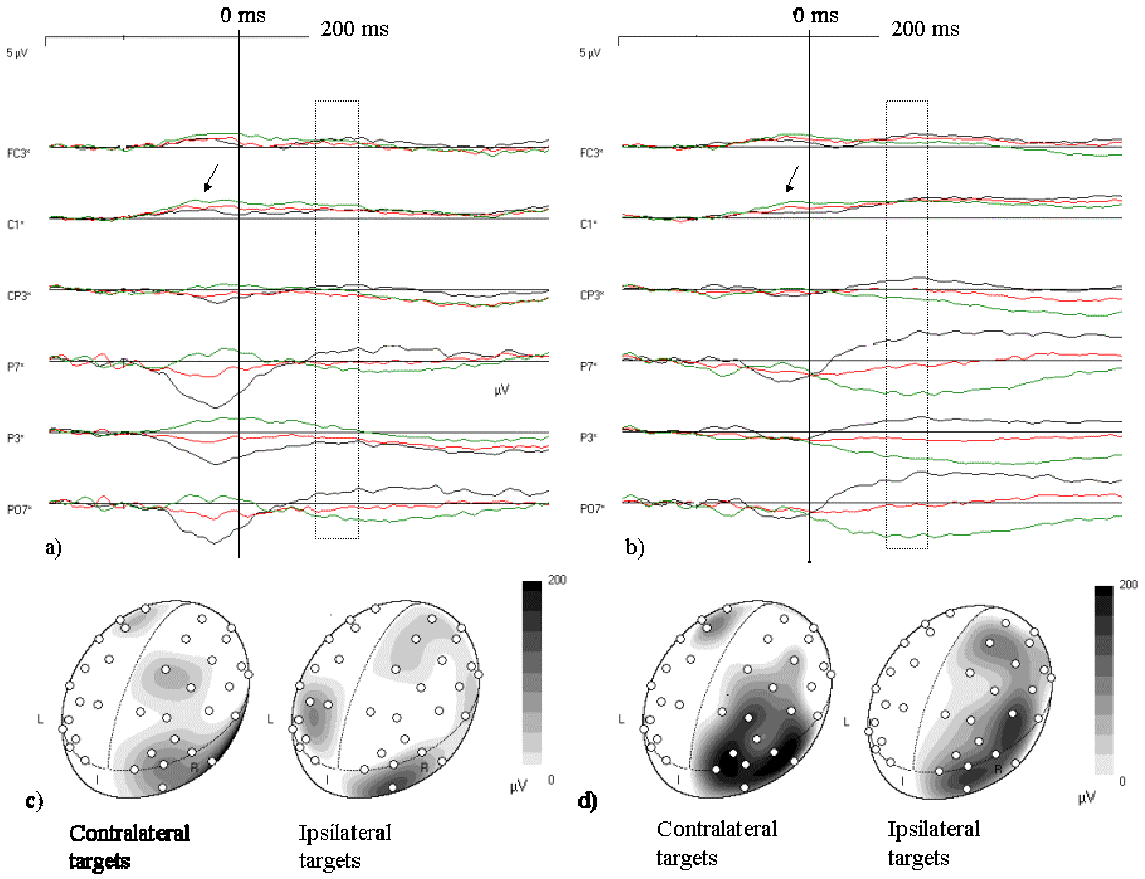

Fig. 5: Stimulus-locked difference potentials. 'Early' vs. 'late' trials in the 'sequenced' condition. a) Exemplary frontal to parieto-occipital sites. Hemisphere contralateral to target plotted upwards. Black lines: 'early' trials, red lines: 'late' trials. 0 ms is target onset. Note increased early ERL at lateral central sites, higher 2nd peak in early ERL and decreased intermediate ERL in 'late' trials. (Decrease in early ERL at parieto-temporal sites is not significant.) b) Topography maps at 350 ms. The right hemisphere shows activity that is higher ipsilateral to the target. The decreased intermediate ERL for 'late' vs. 'early' trials is visible.

Table 2
F- and p-values of effects of factors direction and predictability on peaks of anterior intermediate ERL in experiment 1.

	electrode sites				
	F7/8	F3/4	FC5/6	FC3/4	FC1/2
effect of direction on peak amplitude					
effect of predictability on peak amplitude					F(1,8)=11.2 p=.010
interaction					
effect of direction on latency		F(2,16)=5.8 p=.013	F(2,16)=4.9 p=.038	F(2,16)=11.9 p=.001	F(2,16)=6.4 p=.017
effect of predictability on latency				F(2,16)=8.4 p=.020	F(2,16)=15.2 p=.005
interaction	F(2,16)=6.0 p=.018 a)				

Note. a) In the 'random' condition, the direction effect was absent. Only significant effects with $p < .05$ are shown.

d) During ongoing pointing movement, ERLs also varied with horizontal target position. Response-locked difference potentials are shown in Figure 6. ERLs with lateral targets deviated from those with central targets to the hemisphere contralateral to the target. This late component was measured as the average amplitude between 200 and 300 ms after movement onset in response-locked averages. The main effect of target position was significant at PO9/10, PO7/8 and P7/8. There was no main effect of direction predictability, but there was an interaction with target position at various sites (central, parietal and parieto-occipital): The target position effect was smaller or absent in the 'random' condition and at site PO3/4, had the opposite sign. Figure 7 illustrates differences in ERLs between the 'random' and 'sequenced' condition at site P7/8. No marked differences between 'early' and 'late' trials in a sequence were evident.

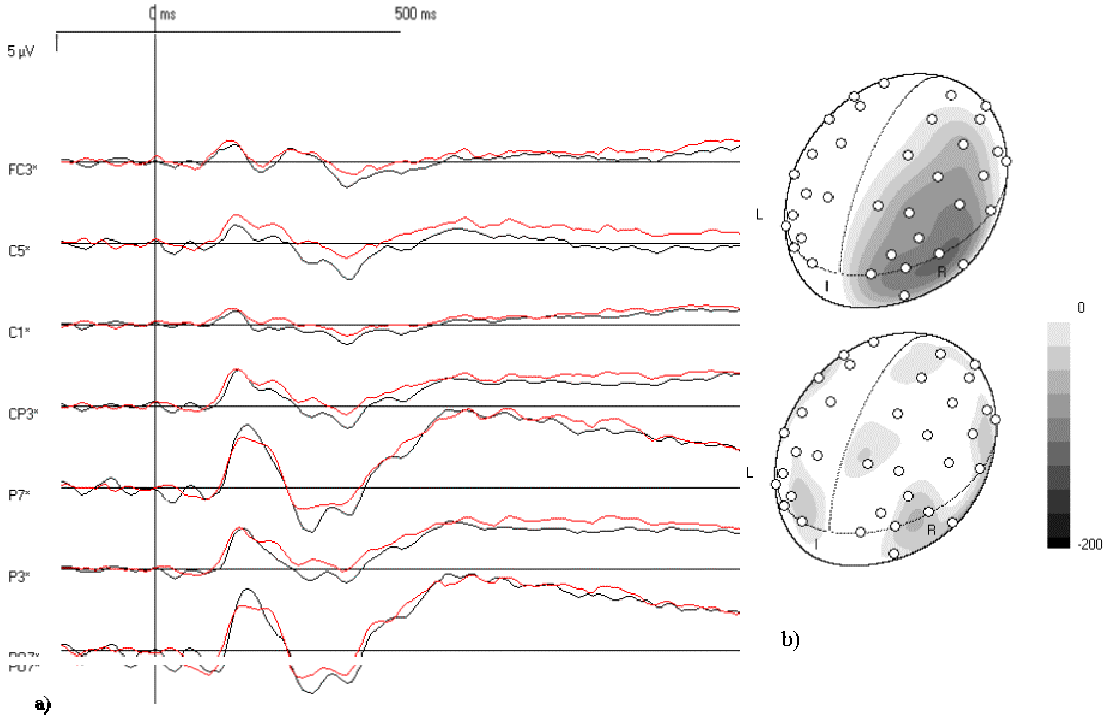

Fig. 6: Response-locked difference potentials. a) and c) 'random', b) and d) 'sequenced'. a) and b): Exemplary frontal to parieto-occipital sites. Hemisphere contralateral to response plotted up. Black lines: target ipsilateral to response side, green lines: contralateral targets, red lines: central targets. 0 ms is movement onset. The dotted rectangle depicts the time interval in which the late ERL was measured. Note coincidence of motor-LRP (arrows) and intermediate ERL component. The effect of horizontal target position on the late ERL is reduced in 'random' compared to 'sequenced' trials. (Difference between target positions in motor-LRP is not significant.) c) and d): Topography maps at 250 ms after movement onset. The right hemisphere shows activity that is higher contralateral to the target. Note that in the 'sequenced' condition, parietal activity was higher in the hemisphere contralateral to the target whereas in the 'random' condition, the component was decreased in amplitude and expansion. Activity over motor sites was higher in the hemisphere contralateral to the responding arm.

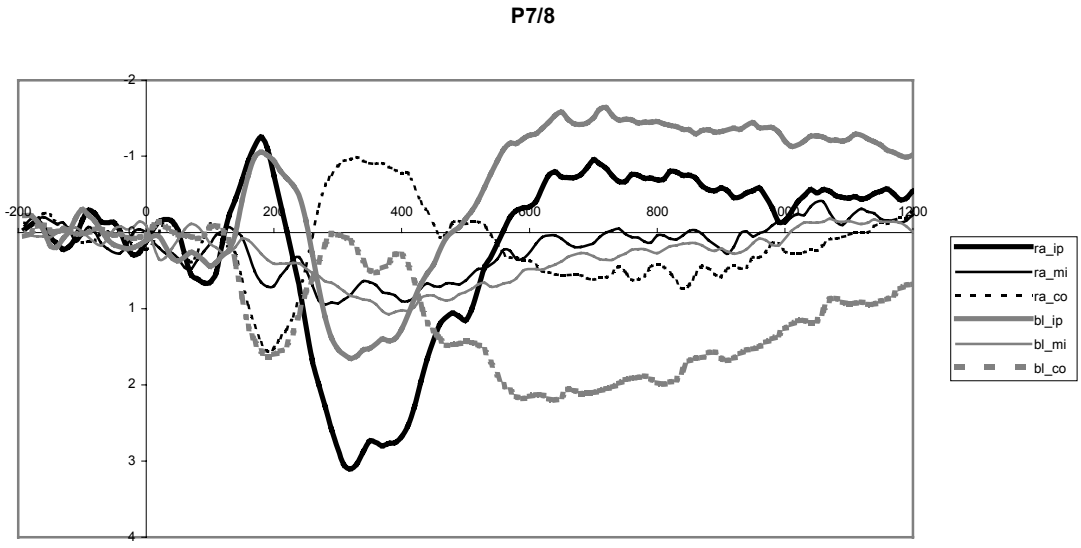

Fig. 7: Stimulus-locked difference potentials at electrode pair P7/8. 'Random' and 'sequenced' trials compared. Hemisphere contralateral to response plotted upwards. Black lines: 'random' trials, grey lines: 'sequenced' trials. Bold lines: target ipsilateral to response side, solid lines: central targets, broken lines: target contralateral to response side. Note the second peak in the early ERL in ipsilateral 'sequenced' trials, the reduced intermediate ERL in 'sequenced' trials and the reduced late ERL in 'random' trials.

Discussion

We identified distinct ERL components in the visuomotor transformation process from target onset to the execution of the pointing movement. These ERLs varied with horizontal target position and therefore with pointing direction.

About 150 to 190 ms after the onset of the target, cortical activity was higher in the hemisphere contralateral to the target. This ERL component reached highest amplitudes at electrode sites over cortical visual areas[1] (parieto-temporal and parieto-occipital) and was evident up to motor and premotor areas (central and fronto-central). This component may well be related to the visual N2 of the event-related potential (ERP). Several studies have found that the visual N2 is increased contralateral to stimulus position (for example Luck&Hilliard, 1994a; Luck&Hilliard, 1994b). This component was called the N2pc (posterior contralateral) and was proposed to indicate attentional selection of task relevant stimuli (Eimer, 1996) and to accompany the voluntary focusing of a visual target (Luck & Hilliard, 1994a). This may be the function of the early ERL component in the present study. However, the ERL component peaked earlier than the N2 of the ERP. This contrasts findings by Wascher et al. (submitted) who found the temporal relation between ERP and ERL in the N2 time span to be the other way around. A possible explanation for the earlier maximal amplitude of the component in the present study may be the fact, that when the target came on, it was the only visual stimulus. In the other studies, additional (task irrelevant) stimuli were visible as well, which may result in a prolonged time to identify the task relevant stimulus and account for the later peak of the N2pc.

The presence of this early component over motor cortical areas might reflect the resourcing of visual information (i.e., target location) for immediate goal directed responses and might trigger automated response activation (Eimer, 1995; Wascher & Waschkuhn, 1996). It is known that there are connections between visual cortical areas and motor areas of the brain via cortico-cortical routes and via subcortical connections (for review see Glickstein, 2000). It is remarkable though, that the latency of this component was shorter over motor and pre-motor areas, compared to that over visual areas of the cortex, suggesting that target location coding information reaches these areas before it is reflected in primary visual cortical areas. Evidence for a separate generation of target evoked visual attention in separate brain areas comes from a recent study by Praamstra & Plat (2001). In addition to the occipito-temporal source of the N2pc, they found a second

[1] Effects at posterior sites only marginally reached significance

CSD source over central scalp sites.

The early ERL did not differ in amplitude between the 'random' and 'sequenced' conditions (except for site O1/2), but amplitudes increased in the ‘late’ trials compared to the ‘early’ trials in a sequence, in which the target appeared in the same hemifield in five subsequent trials. This effect was significant over temporal sites (C5/6, C3/4). This might be due to a shift of visual attention towards the hemifield, in which the target would predictably appear, therefore enhancing or facilitating target selection and location specific processing.

With higher predictability of target position ('sequenced' vs. 'random' and 'late' vs. 'early’ trials) a second peak in the early component became more prominent. This difference was mainly evident in trials with ipsilateral targets: In trials with contralateral targets, a second peak was vaguely visible in the ‘random’ condition as well. The second peak may reflect a separate overlying component, which becomes more prominent with predictable direction. It may reflect enhanced representation of the task relevant stimulus. Contrastingly, Wascher et al. (submitted) found evidence for a preceding ERL component in trials when the position of an imperative stimulus corresponded (i.e. was ipsilateral) to the side of the limb that was selected for response (according to a preceding cue). They found the main peak of the N2pc to emerge at around 250 ms after the stimulus and concluded a preceding component at around 180 ms in trials with ipsilateral imperative stimuli, not with contralateral stimuli. However, in the present study, a second peak was visible with contralateral stimuli as well.

The intermediate ERL component was divided into an anterior part over premotor sites and a posterior part over visual cortex. The posterior ERL consisted of a deviance of activity towards the hemisphere ipsilateral to the target, when compared to central targets. It had maximal amplitudes at parieto-temporal and parieto-occipital electrode sites and extended in time to the point of movement execution and beyond. This effect of target position decreased with target, and therefore movement, direction predictability. Over premotor sites, the component consisted of an increase of activity in the hemisphere contralateral to the responding arm with peak latency varying with target position and predictability. The question arises as to whether the intermediate ERL component reflects target localization or movement preparation, as the direction of both target and movement were identical in this task. Evidence for a movement related function of this component comes from the fact that it can be seen at a time before movement, when there is already increased activity over contralateral motor cortex. This increase of activity prior to the start of the movement was called the motor-LRP to distinguish it from the LRP that can be seen before the imperative stimulus and which reflects the selection of the response side (Verleger et al., 2000). The motor-LRP most likely does not reflect the selection of an effector, as such a selection is not required in this task, but rather reflects the preparation of the movement that is to be executed. The coincidence of the intermediate ERL component and the motor-LRP could therefore be interpreted as the intermediate component being movement related. However, we suggest that the intermediate ERL component is movement related as well as stimulus related, in that it may reflect encoding of target position for motor response. Evidence comes from Verleger et al. (2000), who investigated contra-ipsilateral differences in the EEG after the presentation of arrows as cuing stimuli. Arrows pointing in identical directions were always presented on both sides of a central fixation point. Either saccades or left or right key presses were required after a second stimulus indicated whether or not participants had to respond with the hand indicated by arrow direction. That is, response side matched arrow direction unless the cue was defined as invalid by the second stimulus. At about 400 ms after the arrows, they found increased potentials in the hemisphere contralateral to arrow direction (indicating response side) maximal at fronto-central sites. Verleger et al. called the component L-400 and suggested that it reflects encoding of response relevant spatial properties of the stimulus for action in premotor cortex. The component may have a similar function as the fronto-central part of the intermediate ERL in the present study. Our finding of increased activity over premotor cortex, contralateral to the responding arm (indicating the motor relevance of the component), which differed in latency with target position (indicating the stimulus dependent aspect) is congruent with this interpretation.

Likewise, the parietal part of the intermediate ERL component may be interpreted in terms of encoding spatial stimulus properties for motor response, an interpretation that is suggested by it's dependence on target position and it's maximal amplitude preceding movement onset. Wascher et al. (submitted) also found a direction specific asymmetric ERL component at around 400 ms after stimulus onset, which indicated the start of directed arm movements, but did not represent the target as in the present study. Movement direction was independent of stimulus position. They found the component to be an

increase of activity in the hemisphere contralateral to the arm, which had been selected for movement. The component varied with movement direction, which could either be straight, inward (crossing the body center) or outward (away from the body). The scalp topography of this component in stimulus-locked averages was similar to the parietal part of the intermediate ERL component in the present study, with a focus in parieto-temporal regions. They explained the direction dependent property of the component with an additional ERL component that coded movement direction independently of the responding arm. The fact, that in the study by Wascher et al., activity was higher in the hemisphere contralateral to the response side whereas the posterior intermediate ERL in the present study was lateralized in the hemisphere ipsilateral to the target may be explainable by the following considerations: In the Wascher et al. study, a selection of the responding arm was required, which was not the case in the present study. As is known from the LRP, selection of an effector is reflected in a contralateral increase of activity. We suggest, that if no effector selection were required in the Wascher et al. study, the pattern of lateralisations would be similar to the present study with lateralisations being dependent on movement direction.

The fact that target position predictability interacted with factor ‘direction’ in that it reduced the parietal ERL component may reflect the reduced processing effort when movement direction can be predefined. The reduced component in 'late' trials in a sequence compared to 'early' trials could also be interpreted in that way. Evidence for this interpretation also comes from a study by Deiber et al. (1997). They found decreasing rCBF in posterior parietal cortex during learning of stimulus-response mapping tasks, in which participants had to move a joystick in the direction indicated by a stimulus. They concluded that in the tasks used, a conversion of visual information into the spatial/motor domain was required and that this mapping may be reflected by the changes of activity in the posterior parietal cortex. Furthermore, as the coordinate transformation process becomes routine (i.e., more automatic), the importance of posterior parietal cortex decreases. A common basis for the premotor and parietal part of the intermediate ERL component may be assumed, due to anatomical fronto-parietal connections mediating exchange of information.

An exeptional feature of the posterior intermediate ERL is that activity is higher in the hemisphere ipsilateral to the target. Because of the common principle of contralaterality in cortical processing, one might expect an increase of activity in the hemisphere contralateral to either the target or response side. In the EEG, it is assumed that negativity reflects activation and positivity reflects inhibition in the cortical areas beneath the recording electrodes. Therefore, the relative increase of activity ipsilateral to the target might as well be due to an increased inhibition contralateral to the target. One possible interpretation may be that this inhibition reflects the suppression of processing of visual stimuli in the hemisphere contralateral to the target, to which attention has been directed. Inhibition of visual processing preceding execution of a motor response has been reported in several studies (for example Müsseler et al., 1997a, 1997b). This effect has been named “blindness to response compatible stimuli”, and was initially used to describe the finding that the identification of a right-pointing arrow was impaired when presented during the execution of a right response compared with a left response and vice versa. A major problem to this inhibition of visual processing explanation here is that it cannot explain the decrease of this component if the direction of the pointing movement is predictable. On the contrary, an increase of suppression could be expected if the target predictably appeared on one side of the screen, because visual attention may be more strongly focused on the corresponding hemifield. Thus, even though the posterior intermediate ERL may reflect an increase of positivity in the hemisphere contralateral to the target rather than an ipsilateral increase of negativity, it still seems likely that it reflects visuomotor transformation processes.

In the ‘sequenced’ condition, the late ERL component during movement execution varied with horizontal target position. In the ‘random’ condition, this effect was reduced and evident at fewer sites. One possible origin of the component could be visual feedback from the hand, as it becomes visible during the pointing movement. This is unlikely for two reasons: First, once the visual focus had been directed to the target, it remained on the target during the whole trial (Neggers & Bekkering, 2000). Therefore, when the hand approached the target, it was always visible in the same hemifield, irrespective of target position. Second, the target positions were the same in the 'random' and 'sequenced' condition and visual feedback from the hand during movement was the same during a 'random' condition trial and a 'sequenced' trial. The effect of horizontal target position on the late ERL component though, was evident mainly in the 'sequenced' condition. We

suggest that the component reflects efficient directing of the movement to the target, thereby keeping the target in the focus of attention. If the movement is performed repeatedly, as in the 'sequenced' condition, attention may be more efficiently focused on the target and the directional aspect of the movement, whereas the visuomotor transformation processes in the preparatory phase get less important.

The question came up, whether the effects of horizontal target position that we found in this experiment were dependent on the degree of target laterality. To address this question, we designed a further experiment in which we reduced target laterality.

Experiment 2

In Experiment 2, we investigated whether directional effects were stable if target laterality was reduced. The lateral target positions were moved closer to the initial fixation point in the center of the screen. By reducing their laterality, visual detection of the targets should be facilitated, but directional differences in the task would be reduced. Additionally, to prevent vertical eye movements, which interfered with ERP analysis in Experiment 1, there was no variation in vertical target position. We further reduced pointing direction differences by moving the starting positions closer to the centerline. Moving the starting positions closer to the screen as well, allowed us to further reduce disadvantages in the EEG recordings caused by movement dimensions. Due to these changes, the difference between the contralateral and ipsilateral pointing movements was reduced.

Methods

Unless stated explicitly, setup and methods were the same as in Experiment 1.

Participants

Twelve healthy right-handed participants (8 female) aged 17 to 27 (average 22.42 years) took part in this experiment. Vision was normal or corrected to normal.

Apparatus

The starting positions were moved closer to each other and to the screen (distance between starting positions 7 cm and to screen 7.2 cm). Similar to Experiment 1, the pointing hand became visible once the movement was in progress and the hand approached the screen. In the second part of Experiment 2, which will not be reported here, the visual field was reduced both horizontally and vertically by a prism device, which mirrored participants' vision. To allow direct comparison between the two parts of Experiment 2, in the first part of the experiment, the visual field was also restricted to a similar degree. It yielded full vision of the screen.

Stimuli

In Experiment 1, vertical offset of the target positions caused the participants to produce vertical eye movements to the target, which interfered with ERP analysis. To prevent this, we excluded vertical variation of target position in Experiment 2. We reduced horizontal offset of the target locations (1.7 deg. to the left or right from the center). Consequently, the variations in pointing direction were also reduced. The three possible target locations were presented in random order.

Procedure

Participants performed this part of the experiment with their left and right hands in separate blocks. One block consisted of 450 trials (3 positions * 150 repetitions).

Data Processing and Analysis

The accuracy criterion was loosened in this experiment due to the increased difficulty in the part with mirrored vision. A trial was considered if the participant first touched the screen either at the target location or within 20 mm below the target, independent of horizontal deviance. ANOVAs were performed with factor target position (levels 'ipsilateral', 'central', and 'contralateral').

Results

Figure 8 shows the ERLs in Experiment 2. Since target positions were always presented in random order in Experiment 2, comparisons will only be made to the 'random' condition of Experiment 1. Though Experiment 2 differed from Experiment 1 in at least three factors (no variance of vertical target position, reduced movement distance and reduced distance of lateral stimuli to the center), overall asymmetry of cortical activity showed a similar pattern of lateralisations. The differences will be discussed in more detail here.

Early components: With the smaller lateral distance of the stimuli, the early ERL component turned out to be more pronounced with sharper and higher

amplitudes than in Experiment 1. An additional earlier component, which had maximal amplitudes around 100 ms after target onset, became prominent. It was measured as the mean amplitude between 80 ms and 110 ms after target onset. In this component, which was not well pronounced in Experiment 1, hemispheric lateralisations were reversed (i.e. higher activation in the hemisphere ipsilateral to the target), compared to the proximate. The component was evident up to premotor areas. The following early ERL, measured between 150 and 190 ms after target onset, also peaked earlier at anterior than at posterior sites ($t(11)=2.6$, $p=.024$). The posterior intermediate ERL component, measured as the mean amplitude between 280 ms and 430 ms after target onset, tended to be reduced in Experiment 2 compared to Experiment 1. It did not spread up to central sites, as was the case in Experiment 1. Significant direction effects were found at parieto-occipital and parieto-temporal sites (PO7/8, PO9/10, P7/8). Tables 3 and 4 show levels of significance in Experiment 2. Whereas in Experiment 1, activity in trials with central targets tended to be asymmetric, there was no such lateralisation of activity for central targets over non-motor areas in Experiment 2.

At frontal sites, the intermediate component showed effects of target position on both amplitude and latency of negative peaks, measured in the same time interval as the posterior component (see Table 4). In trials with ipsilateral targets, the component peaked earlier, than with central and contralateral targets. It peaked highest with ipsilateral and lowest with contralateral targets. This contrasts Experiment 1, where target position had significant effects mainly on peak latency. It has to be noted though, that the peaks were not well defined in this ERL in Experiment 2. Therefore, measurement of the peaks here may not be as reliable as in Experiment 1.

In Experiment 2, there was no effect of target position on the ERL after movement onset.

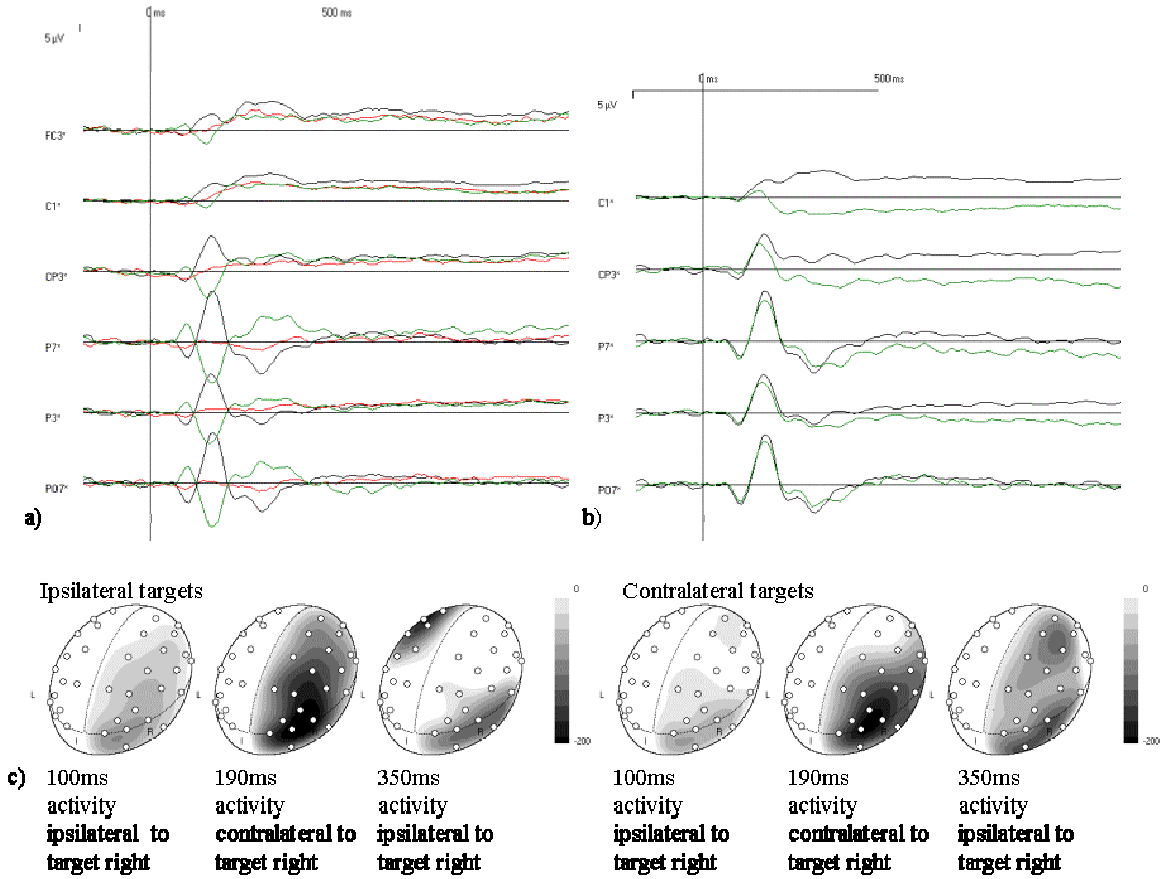

Fig. 8: Experiment 2. Stimulus-locked difference potentials. a) and b) Exemplary frontal to parieto-occipital sites. a) Hemisphere contralateral to response plotted upward, b) hemisphere contralateral to target up. Black lines: target ipsilateral to response side, green lines: contralateral targets, red lines: central targets. 0 ms is target onset. a): Note effect of target position on amplitudes of frontal intermediate ERL. In Experiment 2, an ERL at about 100 ms after target onset is distinct, with higher amplitudes in the hemisphere ipsilateral to the target. c): Topography maps at 100 ms, 190 ms and 350 ms. At 100 ms and 350 ms the right hemisphere shows activity that is higher ipsilateral to the target and at 190 ms activity that is higher contralateral to the target.

Table 3
F- and p-values of effects of factor direction on mean amplitudes in experiment 2.

	electrode sites																
component	FC5/6	FC3/4	FC1/2	C5/6	C3/4	C1/2	CP5/6	CP3/4	CP1/2	P7/8	P5/6	P3/4	P1/2	PO9/10	PO7/8	PO3/4	O1/2
early ERL at 100 ms	F(2,22)=3.9 p=.046	F(2,22)=7.9 p=.003	F(2,22)=7.6 p=.004	F(2,22)=5.0 p=.020	F(2,22)=12.1 p<.001	F(2,22)=9.5 p=.001	F(2,22)=10.4 p=.001	F(2,22)=9.9 p=.001	F(2,22)=7.4 p=.004	F(2,22)=13.1 p=.002	F(2,22)=12.6 p=.001	F(2,22)=19.8 p<.001	F(2,22)=19.7 p<.001	F(2,22)=17.8 p<.001	F(2,22)=13.3 p=.002	F(2,22)=16.7 p<.001	F(2,22)=19.0 p<.001
early ERL at 170 ms	F(2,22)=26.2 p<.001	F(2,22)=14.8 p<.001	F(2,22)=22.4 p<.001	F(2,22)=32.4 p<.001	F(2,22)=23.7 p<.001	F(2,22)=20.7 p<.001	F(2,22)=39.4 p<.001	F(2,22)=51.6 p<.001	F(2,22)=41.9 p<.001	F(2,22)=41.6 p<.001	F(2,22)=67.7 p<.001	F(2,22)=67.8 p<.001	F(2,22)=26.9 p<.001	F(2,22)=16.8 p=.001	F(2,22)=52.2 p<.001	F(2,22)=44.6 p<.001	F(2,22)=46.1 p<.001
intermediate ERL										F(2,22)=5.7 p=.032				F(2,22)=8.0 p=.008	F(2,22)=4.8 p=.041		
late ERL																	

Note. No significant effects were found at sites F3/4 and F7/8. Only significant effects with $p < .05$ are shown.

Table 4
F- and p-values of effects of factor direction on peaks of anterior intermediate ERL in experiment 2.

	electrode sites				
	F7/8	F3/4	FC5/6	FC3/4	FC1/2
effect of direction on latency			F(2,22)=5.0 p=.021	F(2,22)=3.9 p=.040	
effect of direction on peak amplitude		F(2,22)=4.6 p=.041		F(2,22)=6.0 p=.021	F(2,22)=5.9 p=.023

Note. Only significant effects with $p < .05$ are shown.

Discussion

Even with smaller lateral distance of the targets, similar effects of direction on hemispheric EEG differences were evident. In Experiment 2, lateral target positions were closer to the initial visual focus. This resulted in a facilitation of target localization, which was reflected in the finding that early ERL components after target onset were more sharply pronounced and had higher amplitudes. The posterior intermediate ERL component was more sharply focused at parieto-occipital and parieto-temporal sites, where it also had highest amplitudes in Experiment 1. In Experiment 2, it did not spread to the motor cortex though. In Experiment 1, we argued that the component reflects the transformation of visual information about target position into the motor domain. Given its relation to movement direction, the decrease in this component in Experiment 2 seems plausible due to the smaller differences between the potential movement directions.

During movement, there were no directional effects on ERLs. The lack of target position effects on the late component in Experiment 2 may be explainable by the reduced differences in the movements directed to the different target positions. It may also serve as additional evidence that the function of the late ERL component is not primarily visual.

General Discussion and Conclusions

Asymmetries in the activation of the hemispheres provide valuable information about the cortical processes underlying visually triggered pointing movements from target onset to the ongoing movement with especially high resolution in the time domain. These processes involve target localization, response preparation and execution.

In Experiment 1, we investigated the effect of horizontal target position and target position predictability on ERLs. Both factors influenced the pattern of hemispheric activation asymmetries. In Experiment 2, we found these effects across variations in movement distance and pointing and target laterality.

Three components of visuomotor processing were modified by pointing direction: An early stage, probably reflecting attentional selection of the target; an intermediate stage preceding the start of the pointing movement that seems to reflect encoding of target position for motor response; and finally movement execution.

ERLs that preceded the start of the movement differed at posterior and at anterior sites. Whereas lateralisation of activity in parietal and parieto-occipital cortex changed with pointing direction, activity in premotor cortex was consistently higher in the hemisphere contralateral to the response side with peak latency and amplitudes of the ERL depending on pointing direction. Parietal cortex is known to be involved in visuomotor coordination (Kertzman et al., 1997; Laquantini & Caminti, 1998) and the selection of directed movements (Grafton et al., 1992). Many parietal neurons in monkey have multisensory receptive fields allowing the integration of proprioceptive and visual information for goal directed reaching movements (Graziano et al., 2000). Sensory locations of stimuli are thereby converted into appropriate motor coordinates required for directed movements (for review see Andersen et al., 1997). Evidence for the coding of spatial stimulus and response properties in premotor cortex comes from monkey studies. Graziano et al. (1997) found neurons in the ventral premotor cortex of primates that coded the location of a target in space after it had disappeared. They concluded that those neurons underlie the ability to reach for a target that is no longer visible. In an instructed delay task, in which monkeys had to reach to a visual target, Crammond et al. (1994) found activity of dorsal premotor cortex neurons to reflect spatial features of both the cueing stimulus and the motor response. Early instructed delay period (IDP) activity covaried with cue location, whereas late IDP activity covaried with the direction of movement signaled by the cue, independent of cue location. They therefore concluded, that IDP activity in dorsal premotor cortex ultimately encodes direction of intended reaching movements. These findings provide evidence for the interpretation that the movement preceding components, posterior as well as anterior, reflect the transformation of spatial target properties into motor coordinates.

Predictability of horizontal target position added a second peak to the early ERL that may reflect an additional overlying component in the process of target localization. Predictability of pointing direction was also reflected in a reduction of the ERL component preceding movement onset that had its focus in parieto-temporal cortex. Taking into account the function of parietal cortex in accurate goal directed movements, we suggest that this reduced ERL reflects the lesser processing effort in visuomotor transformation if the direction of movement is predictable. During movement execution, repeated pointing direction may allow more efficient attending to target position and the directional aspect of the movement, reflected in an increased ERL in that phase.

References

Andersen R.A., Snyder L.H., Bradley D.C., Xing J. (1997). *Multimodal representation of space in the posterior parietal cortex and its use in planning movements.* Annual Reviews Neuroscience, 20, 303-330.

Clower M. D., Hoffman J.M., Votaw J.R., Faber T.L., Woods R.P., Alexander G.E. (1996). *Role of posterior parietal cortex in the recalibration of visually guided reaching*. Nature, 383, 618-621.

Crammond D.J., Kalaska J.F. (1994). *Modulation of preparatory neuronal activity in dorsal premotor cored due to stimulus-response compatibility.* Journal of Neurophysiology, 71, 1281-1284.

De Jong R., Wierda M., Mulder G., Mulder L.J.M. (1988). *Use of partial stimulus information in response processing.* Journal of Experimental Psychology: Human Perception and Performance, 14, 682-692.

Deiber M.P., Passingham R.E., Colebatch F.G., Friston K.J., Nixon P.D., Frackowiak F.S. (1991). *Cortical areas and the selection of movement: A study with positron emission tomography.* Experimental Brain Research, 84, 393-402.

Deiber M.P., Wise S.P. Honda M., Catalan M.J., Grafman J., Hallett M. (1997). *Frontal and parietal networks for conditional motor learning: a positron emission tomography study.* Journal of Neurophysiology, 78(2), 977-91.

Eimer M. (1995). *Stimulus-response compatibility and automatic response activation: Evidence from psychophysiological studies.* Journal of Experimental Psychology: Human Perception and Performance, 21(4), 837-854.

Eimer M. (1996). *The N2pc component as an indicator of attentional selectivity*. Electroencephalography and Clinical Neurophysiology, 99, 225-234.

Fisk J.D., Goodale M.A. (1885). *The organization of eye and limb movements during unrestricted reaching to targets in contralateral and ipsilateral visual space*. Experimental Brain Research, 60, 159-178.

Georgopoulos A. P., Lurito J.T., Petrides M., Schwartz A.B., Massey J.T. (1989). *Mental rotation of the neuronal population vector.* Science, 243, 234-236

Glickstein M. (2000). *How are visual areas of the brain connected to motor areas for the sensory guidance of movements.* Trends in Neuroscience, 23, 613-617.

Grafton S.T., Mazziotta J.C., Woods R.P., Phelps M.E. (1992). *Human functional anatomy of visually guided finger movements.* Brain, 115(2), 565-587.

Gratton G., Coles M.G.H., Sirevaag E.J., Eriksen C.W., Donchin E. (1988). *Pre- and poststimulus activation of response channels: A psychophysiological analysis.* Journal of Experimental Psychology: Human Perception and Performance, 14, 331-344.

Graziano M.S., Hu X.T., Gross C.G. (1997). *Coding the locations of objects in the dark.* Science, 277, 239-241.

Graziano M.S.A., Cooke D.F., Taylor S.R. (2000). *Coding the location of arm by sight.* Science, 290, 1782-1786.

Inoue K., Kawashima R., Satoh K., Kinomura S., Goto R., Koyama M., Sugiura M., Ito M., Fukuda H. (1998). *PET study of pointing with visual feedback of moving hands*. Journal of Neurophysiology ,79(1), 117-125.

Kertzman C., Schwarz U., Zeffiro T.A., Hallett M. (1997). *The role of posterior parietal cortex in visually guided reaching movements in humans.* Experimental Brain Research, 114(1), 170-83.

Laquantini F., Caminti R. (1998). *Visuo-motor transformations for arm reaching.* European Journal of Neuroscience, 10, 195-203.

Luck S.J., Hillyard S.A. (1994a). *Electrophysiological correlates of feature analysis during visual search*. Psychophysiology, 31, 291-308.

Luck S.J., Hillyard S.A. (1994b). *Spatial filtering during visual search: Evidence from human electrophysiology.* Journal of Experimental Psychology: Human Perception and Performance, 20, 1000-1014.

Müsseler J., & Hommel B. (1997). *Blindness to response-compatible stimuli.* Journal of Experimental

Psychology: Human Perception & Performance, 23(3), 861-872.

Müsseler J., & Hommel B. (1997). *Detecting and identifying response-compatible stimuli.* Psychonomic Bulletin & Review, 4, 125-129.

Neggers S.F., Bekkering H. (2000). *Ocular gaze is anchored to the target of an ongoing pointing movement*. Journal of Neurophysiology, 83(2), 639-51.

Praamstra P., Plat F.M. (2001). *Failed suppression of direct visuomotor activation in Parkinson's Desease*. Journal of Cognitive Neuroscience, 13, 31-43.

Verleger R., Vollmer C., Waschkuhn B., van der Lubbe R.H.J., Wascher E. (2000). *Dimensional overlap between arrows as cueing stimuli and responses? Evidence from contra-ipsilateral differences in EEG potentials.* Cognitive Brain Research, 10, 99-109.

Wascher E., Waschkuhn B. (1996). *The interaction of stimulus- and response-related processes measured by event-related lateralisations of the EEG.* Electroencephalography and Clinical Neurophysiology, 99, 149-162.

Wascher E., Wolber M., Schoenstein S. (submitted). *Tracking the visuomotor system by measuring event-related asymmetries of the EEG in a task requiring directed arm movements.*

Wojciulik E., Kanwisher N. (1999). *The generality of parietal involvement in visual attention*. Neuron, 23, 747-764.