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An ERP study on visual spatial priming with peripheral onsets

MARTIN EIMER
Universität München, Institut für Psychologie, München, Germany

Abstract

Visual event-related potentials were measured for peripheral target stimuli that were preceded by a peripheral square. Targets appeared either at the same location as the square or in the opposite visual hemifield. In Experiment 1, 75% of the trials were same-location trials, and in Experiment 2, same- and opposite-location trials were equiprobable. The subject's overt response was dependent either on the identity or on the location of the target. In both experiments, opposite-location targets elicited an enhanced P1 at posterior electrodes ipsilateral to the position of the letter. This enhancement may be due to a sensory inhibition of same-location targets. Same-location targets elicited an enhanced negativity between 130 and 300 ms, with a first peak located parietally and a second peak broadly distributed over midline electrodes. This effect was larger in Experiment 1 than in Experiment 2 and is interpreted as enhanced processing of same-location targets due to an attentional orienting process elicited by the peripheral square.

Descriptors: Visual-spatial attention, Event-related brain potentials, P1, Processing negativity, Trial-by-trial cueing

When visual attention is directed to specific objects and locations within the visual field, stimuli at to-be-attended locations are detected with higher speed and accuracy than are stimuli presented outside the attentional focus. Such attentional orienting processes may occur covertly, that is, independent of overt behavior such as eye movements (Jonides, 1981; Müller & Findlay, 1987; Müller & Rabbitt, 1989; Posner, Cohen, & Rafal, 1982; Posner, Nissen, & Ogden, 1978; Posner, Snyder, & Davidson, 1980). The covert orienting of visual-spatial attention has been investigated by both behavioral and electrophysiological studies. In most reaction time studies, trial-by-trial cueing paradigms were used, where a precue informs subjects about the likely position of an upcoming imperative stimulus. Reactions to targets at validly indicated locations were faster than reactions to targets occurring at unprimed positions (Posner et al., 1978, 1980, 1982). In most event-related potential (ERP) studies on visual spatial orienting, sustained attention paradigms were used. Subjects were required to focus attention on one visual hemifield and to ignore events in the opposite hemifield. Attended stimuli elicit enhanced P1 and N1 components as compared with stimuli that occurred in the unattended hemifield (Eason, 1981; Harter, Aine, & Schroeder, 1982; Hillyard & Mangun, 1987; Hillyard & Münte, 1984; Mangun & Hillyard, 1988). These findings have been interpreted as evidence for intraperceptual sensory gating mechanisms that favor processing of valid stimuli (see Mangun & Hillyard, 1990, for an overview).

In contrast to most behavioral experiments, few ERP studies have investigated effects of visual spatial orienting induced by trial-by-trial cueing. Mangun and Hillyard (1991; see also Mangun, Hansen, & Hillyard, 1987) found enhancements of P1 and N1 amplitudes at occipital scalp sites for attended targets. Because these effects were similar to ERP modulations found with the sustained-attention paradigm, they suggested that functionally similar sensory gating mechanisms are active both in trial-by-trial cueing situations and during sustained attention. In addition to these early modulations of sensory-evoked components, validly cued stimuli also elicit an enhanced negativity as compared with invalid stimuli. Mangun and Hillyard (1991) reported a negative modulation for valid stimuli at lateral central, parietal, and occipital electrodes between 230 and 400 ms, which they attributed to an enlarged P3 elicited by low-probability invalid stimuli. Eimer (1993) found a negative enhancement for valid trials at midline electrodes with an onset of about 180 ms. This effect was interpreted tentatively as an analogue of the Nd effects reported for the auditory modality (Näätänen, 1982), which indicate the selection and/or the enhanced processing of stimuli within the attentional focus.

In the former trial-by-trial cueing ERP studies, centrally presented symbolic precues (arrows pointing either to the left or to the right) were used to induce attentional orienting. In the experiments reported here, I investigated whether similar ERP effects

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Address reprint requests to: Martin Eimer, Universität München, Institut für Psychologie, Leopoldstr. 13, 80802 München 40, Germany.

of attentional orienting can be induced when peripheral onsets are used as precues. In Experiment 1, the target was presented with 75% probability at the position where a peripheral cue stimulus had occurred. In Experiment 2, the peripheral cue was noninformative; the target appeared with equal probability at the cued location and in the opposite visual hemifield. Behavioral studies have shown that peripheral cues elicit fast, reflexive, automatic attentional shifts, whereas central symbolic cues lead to slower, controlled, and voluntary orienting processes (Jonides, 1981; Müller & Rabbitt, 1989). However, when the interval between a peripheral cue and a subsequent target is sufficiently long and the cue is informative with respect to target position (i.e., when the cue usually occurs at locations at which a subsequent target will appear), it may be assumed that peripheral cues elicit voluntary attentional shifts comparable to orienting processes induced by central symbolic cues. If this assumption is true, Experiment 1 should yield ERP modulations for valid (as compared with invalid) trials that are similar to those found with central cues.

However, there may be other functional differences between central and peripheral cues. Most importantly, valid and invalid trials are not physically identical in peripheral cueing studies. In valid trials, the target stimulus is preceded by a cue at the same location, whereas in invalid trials, cue and target occur in opposite hemifields. Thus in valid trials, cue and target activate the same or similar pathways within the visual system. Upon arrival of the target stimulus, these pathways may still be in a refractory state because of the prior processing of the cue. This is not the case for invalid trials. If such sensory interactions between cues and targets occur in valid trials, effects on ERP waveforms may be confounded with ERP modulations related to attentional orienting. In addition, a number of behavioral studies that employed noninformative peripheral cues (Maylor, 1985; Maylor & Hockey, 1987; Posner & Cohen, 1980, 1984) have shown that when the cue–target interval is longer than 300 ms, reaction time for targets that occur at the same position as the cue is slower than for opposite-location targets. According to Posner and Cohen (1984), this inhibition of return can be regarded as an automatic and purely sensory phenomenon that is due to reduced processing efficiency for stimuli presented at previously cued locations.

These presumed differences between symbolic and peripheral cueing suggest that the effects of peripheral cues on early sensory-evoked ERP components may be quite distinct from the effects reported in experiments using central cues. Whereas informative central symbolic cues are assumed to induce enhanced sensory processing for validly cued targets, peripheral cues may cause an initial inhibition of sensory processing for targets at cued locations. Moreover, this sensory inhibition should be largely independent of the informativeness of the peripheral cue.

Methods

Experiment 1

Subjects. Ten paid volunteers (five female, five male), ages 20–39 years ($M = 25.6$ years), participated in the experiment. Subjects were right handed and had normal or corrected-to-normal vision.

Stimuli and apparatus. Subjects were seated in a dimly lit, electrically shielded sound-attenuated cabin, with response buttons under their left and right hands. A computer screen was placed 100 cm in front of the subject's eyes and carefully positioned so that the stimuli (presented white-on-gray) occurred on the subject's horizontal straight-ahead line of sight. A fixation cross was presented continuously at the center of the screen. Each trial began with a 200-ms presentation of a peripheral square that subtended a visual angle of 1.2° × 1.2°. The square appeared either in the left or right visual hemifield at a horizontal distance of 6° from the fixation cross. Seven hundred milliseconds after the offset of the square, an uppercase letter (an M or W), subtending an angle of 1° × 1°, appeared for 100 ms in the left or right hemifield (6° horizontal distance from the fixation cross). The intertrial interval between letter offset and onset of the next square was 2 s.

Procedure. Two experimental halves (Experiments 1a and 1b), each consisting of 12 blocks, were run successively, resulting in a total of 24 experimental blocks. Each block consisted of 60 trials and lasted approximately 2.5 min. Both letter stimuli appeared randomly and with equal probability on the left and right side and were preceded either by a peripheral square at the same location (valid trials) or by a square in the opposite hemifield (invalid trials). Forty-four of 60 trials (73.3%) per block were valid. In Experiment 1a, subjects had to respond with the left hand to the occurrence of the letter M and with the right hand when a W appeared on the screen (response cue: letter identity). In Experiment 1b, response was contingent upon the location of the letter: left-side letters required a left-hand reaction, right-side letters required a right-hand reaction (response cue: letter location). The order of experimental halves was balanced across subjects. Subjects were instructed to respond as quickly and accurately as possible and to fixate the central cross during the trials. To make subjects familiar with these specific task requirements, several training blocks were run at the beginning of the experiment.

Recording. The electroencephalogram (EEG) was recorded with Ag-AgCl electrodes from Fz, Cz, and Pz (according to the 10-20 system), from PL and PR (located halfway between Pz and the ear channel), and from OL and OR (located halfway between O_1 and T_5, and O_2 and T_6, respectively). Additionally, the EEG was recorded from C_3' and C_4' (1 cm in front of C_3 and C_4).[1] All electrodes were referenced to the right earlobe. The horizontal electrooculogram (EOG) was recorded bipolarly from electrodes at the outer canthi of both eyes, and the vertical EOG was recorded from electrodes above and beside the right eye. Electrode impedance was kept below 5 kΩ. The amplifier bandpass was 0.016–70 Hz. The EEG and EOG were sampled on-line every 7 ms and stored on disk. Reaction times were recorded for each trial.

Data analysis. The EEG and EOG were epoched off-line into periods of 1,800 ms, starting 100 ms prior to the onset of the peripheral square and ending 800 ms after letter onset. Trials with eyeblinks, horizontal eye movements, or overt response errors were excluded from analysis. The EEG was averaged sep-

[1] These additional recordings were made to obtain the lateralized readiness potential (LRP) as an index of selective motor preparation processes (see Coles, Gratton, & Donchin, 1988). Because this aspect of the research is beyond the scope of the present article, these data will not be reported.

arately for all combinations of conditions (response cue: letter identity/letter position; trial validity: valid/invalid; visual field of presentation: left/right; stimulus identity: M/W), resulting in 16 average waveforms for each subject and electrode site. All measures were taken relative to the mean voltage of the 100-ms interval preceding letter onset.

Effects of experimental variables on the ERP evoked by the imperative stimulus were determined separately for lateral posterior and central recording sites. Separate repeated measures analyses of variance (ANOVAs) were performed on amplitude and latency measures for the following variables: electrode location, recording side (for lateral electrodes), trial validity (valid vs. invalid), stimulus–response compatibility (in Experiment 1a), and letter location (left vs. right). When appropriate, a Greenhouse–Geisser adjustment was made to the degrees of freedom. Additional repeated measures ANOVAs were performed for single recording sites. At lateral posterior sites, P1 and N1 peak amplitudes were determined as maximum positive and negative voltages within the intervals 92–148 ms (P1) and 140–200 ms (N1) after letter onset. To test specific effects of trial validity, two-tailed paired t tests were used. Mean amplitude values were computed for the time interval 130–180 ms (Nd_1) at lateral posterior and midline sites and for the time interval 220–280 ms (Nd_2) at midline electrodes. To test the significance of the Nd effects related to trial validity within these time windows, one-tailed paired t tests were used. P3 amplitudes at midline sites were determined as maximum positive voltages between 300 and 500 ms poststimulus. For the reaction time data, repeated measures ANOVAs were performed separately for Experiments 1a and 1b for the factors trial validity, response side, and stimulus–response compatibility (for Experiment 1a).

Experiment 2

Subjects. Eleven paid volunteers participated in the experiment. One subject had to be excluded because of delayed response times accompanied by a large number of response errors; therefore, 10 subjects (four female, six male), ages 23–31 years ($M = 26.0$ years), remained in the sample. Subjects were right handed and had normal or corrected-to-normal vision.

Stimuli and apparatus. Stimuli and apparatus were identical to those in Experiment 1.

Procedure. Two experimental halves (Experiments 2a and 2b) were run. The overall procedure and response instructions were identical to those in Experiment 1, with letter identity serving as response cue in Experiment 2a and letter position being response relevant during Experiment 2b. In contrast to Experiment 1, the letter appeared with equal probability in the visual hemifield where the square had been presented and in the opposite hemifield. As before, these trials are called valid and invalid, respectively.

Recording and data analysis. These procedures were identical to those in Experiment 1.

Results

Experiment 1

Behavioral performance. Reaction times for valid letters were significantly shorter than reaction times for invalid letters. When letter identity served as the response cue (Experiment 1a), mean reaction time for valid trials was 456 ms, as compared with 469 ms for invalid trials ($F[1,9] = 25.00$, $p < .001$). When letter position was the response cue (Experiment 1b), mean reaction times were 262 and 296 ms, respectively ($F[1,9] = 31.15$, $p < .001$). Stimulus–response compatibility had no effect on reaction time. Right-hand reactions were faster than left-hand reactions in Experiment 1a (453 vs. 471 ms; $F[1,9] = 8.19$, $p < .019$).

Effects of trial validity at lateral parietooccipital sites. Figure 1 shows the ERPs for imperative letter stimuli for valid and invalid trials at lateral parietal and occipital electrodes ipsilateral and contralateral to the position of the letter. A main effect of trial validity on P1 amplitude was found for Experiment 1b ($F[1,9] = 11.52$, $p < .008$) but was missing in Experiment 1a. In both experimental halves, three-way interactions (Recording Side × Trial Validity × Letter Location: $F[1,9] = 8.12$, $p < .019$ and $F[1,9] = 7.15$, $p < .025$ for Experiments 1a and 1b, respectively) indicated that trial validity influenced P1 amplitude differently at recording sides ipsilateral and contralateral to the letter position. At ipsilateral recording sites, P1 amplitude was enhanced for invalid trials (see Figure 1). In Experiment 1a, this effect was present at occipital electrodes ($t[1,9] = 2.40$, $p < .040$) and approached significance at parietal electrodes ($t[1,9] = 2.23$, $p < .053$). In Experiment 1b, the effect was found both at occipital ($t[1,9] = 3.70$, $p < .005$) and at parietal ($t[1,9] = 3.72$, $p < .005$) recording sites. A significant P1 enhancement for invalid trials at contralateral sites could only be observed at occipital sites in Experiment 1b ($t[1,9] = 3.51$, $p < .007$).[2]

N1 amplitude was influenced by trial validity in Experiment 1a ($F[1,9] = 9.94$, $p < .012$). The N1 for valid trials was greater than for invalid trials. This effect was significant at parietal sites ($F[1,9] = 13.16$, $p < .006$) and approached significance at occipital electrodes ($F[1,9] = 4.77$, $p < .057$). In Experiment 1b, no effect of trial validity on N1 amplitude was found. For the mean amplitude in the Nd_1 time range (130–180 ms), there were no main effects of trial validity. However, in Experiment 1a, three-way interactions (Recording Side × Trial Validity × Letter Location: $F[1,9] = 19.06$, $p < .002$ and $F[1,9] = 10.94$, $p < .009$ for parietal and occipital electrodes, respectively) indicated an enhanced negativity for valid trials at ipsilateral electrodes (see also the difference waveforms in Figure 3). Subsequent t tests revealed that this effect was present both at parietal ($t[1,9] = 2.45$, $p < .037$) and at occipital ($t[1,9] = 2.88$, $p < .018$) electrodes. In Experiment 1b, similar tendencies were present but did not reach significance.

Effects of trial validity at central electrodes. Figure 2 shows the ERP waveforms for valid and invalid trials at central electrodes. These waveforms and the difference waveforms in Figure 3 indicate that valid trials elicited an enhanced negativity as compared with invalid trials with an onset of about 210 ms (Nd_2). This negative shift was preceded by another negative enhancement for valid trials at 130–180 ms that is visible mainly

[2] An unexpected interaction between cue validity and letter location was found in Experiment 1a ($F[1,9] = 16.41$, $p < .003$). Subsequent t tests revealed that the P1 enhancement elicited by invalid trials was more pronounced for stimuli presented in the left visual hemifield. However, this finding could not be replicated in Experiment 1b or Experiment 2.

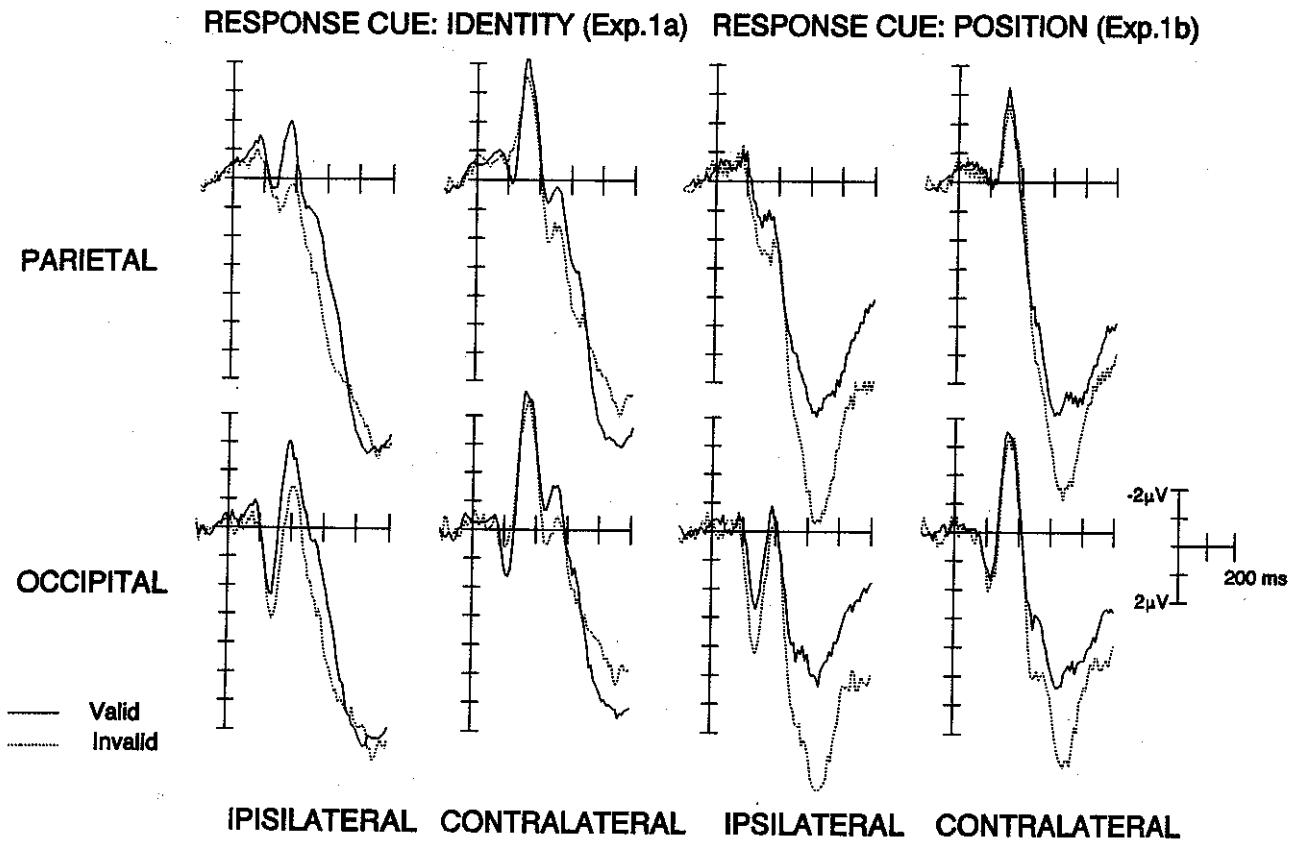

Figure 1. Grand-averaged ERPs at parietal and occipital recording sites for valid and invalid trials, Experiment 1. ERPs recorded from hemispheres ipsilateral and contralateral to the visual field of stimulation are presented separately.

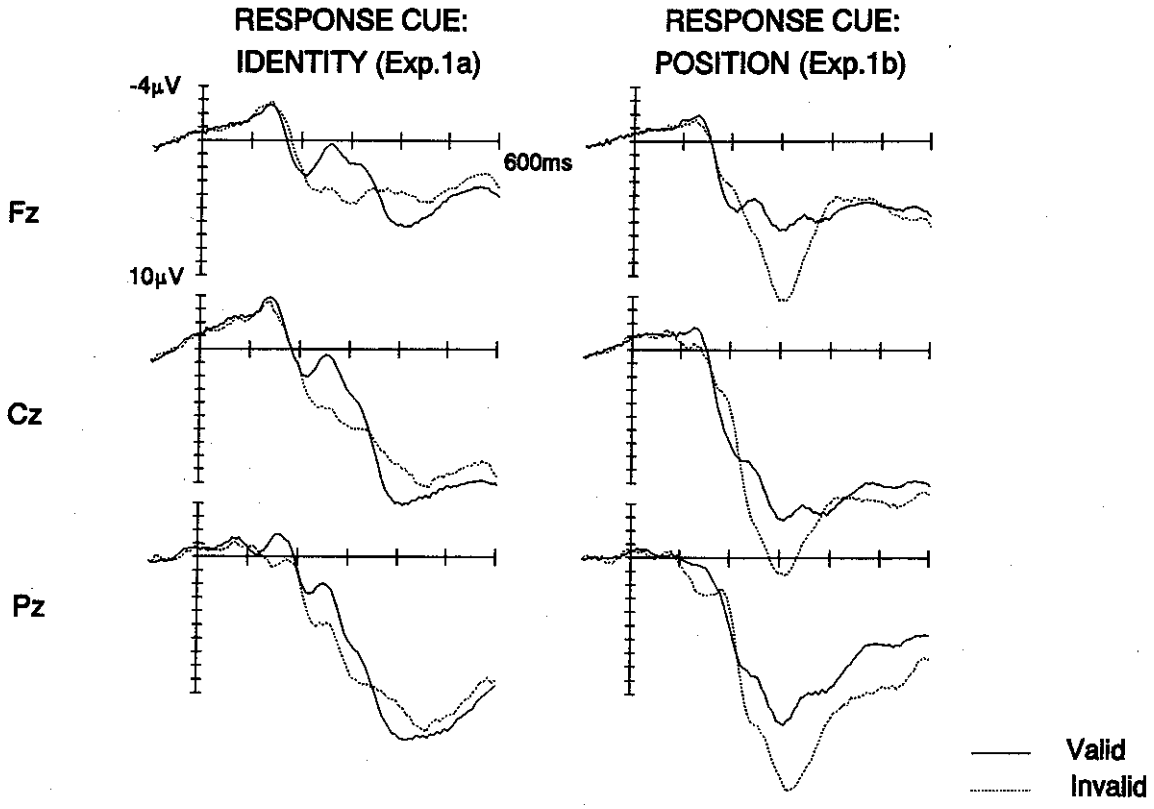

Figure 2. Grand averaged ERPs at Fz, Cz, and Pz for valid and invalid trials, Experiment 1.

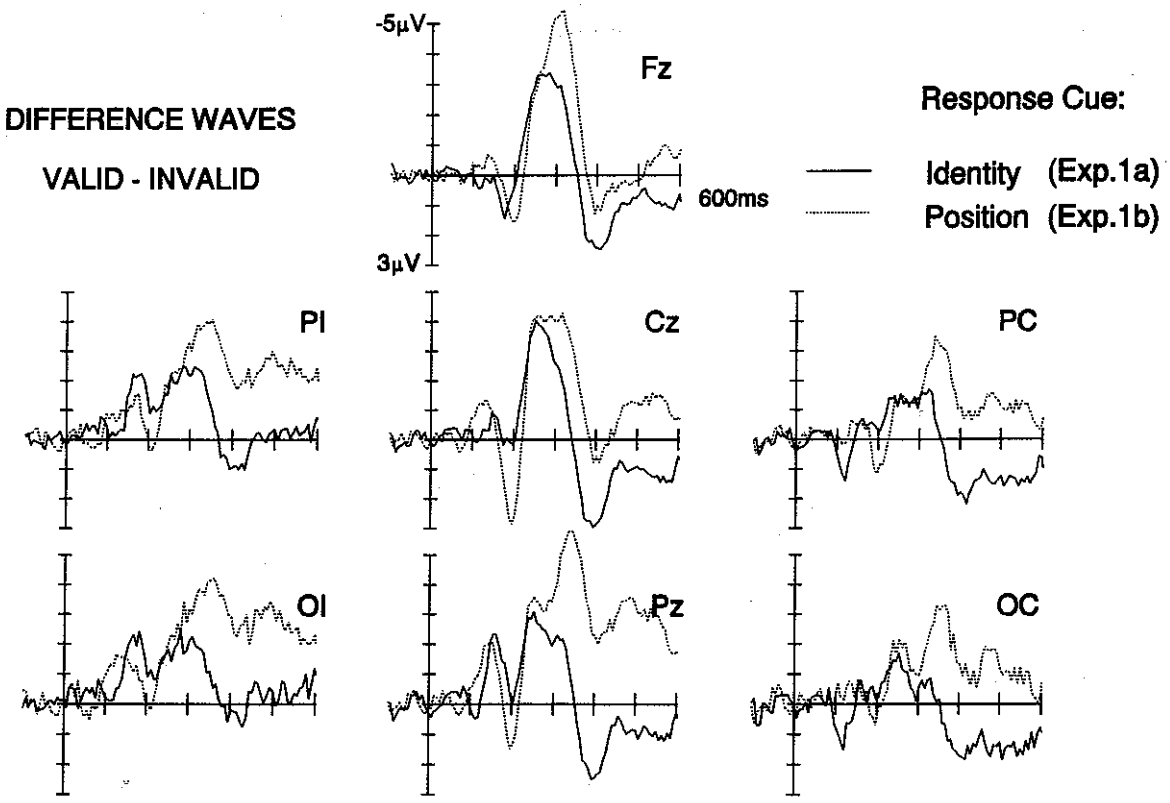

Figure 3. Difference waveforms obtained by subtracting ERPs for invalid trials from ERPs for valid trials for Experiment 1a (solid line) and Experiment 1b (dotted line) for midline electrodes and for lateral parietal and occipital electrodes ipsilateral (PI and OI) and contralateral (PC and OC) to the visual field of the target.

at Pz (Nd_1). In the Nd_1 range, no main effect of trial validity on mean amplitude was found. However, there was an interaction between electrode location and trial validity that was significant for Experiment 1a ($F[2,18] = 9.93$, $p < .01$, $\epsilon = 0.534$) and almost approached significance for Experiment 1b ($F[2,18] = 3.44$, $p < .09$, $\epsilon = 0.567$). Subsequent t tests revealed that the Nd_1 effect was significant at Pz in Experiment 1a ($t[1,9] = 2.14$, $p < .031$) and approached significance at Pz in Experiment 1b ($t[1,9] = 1.78$, $p < .059$). In the Nd_2 interval, there was an effect of trial validity both for Experiment 1a ($F[1,9] = 17.52$, $p < .009$) and for Experiment 1b ($F[1,9] = 7.98$, $p < .020$). Subsequent t tests revealed that this effect was significant at all midline electrodes.

In Experiment 1a, there was no effect of trial validity on P3 amplitude. In Experiment 1b, invalid trials elicited enhanced P3 amplitudes as compared with valid trials ($F[1,9] = 11.35$, $p < .008$).

Experiment 2

Behavioral performance. No effect of trial validity on response latency was found. In Experiment 2a, mean reaction times for valid and invalid trials were 486 and 484 ms, respectively. In Experiment 2b, mean reaction time was 302 ms both for valid and invalid trials. As in Experiment 1, stimulus-response compatibility had no significant effect on reaction time. Right-hand reactions were faster than left-hand reactions in Experiment 2a (476 vs. 493 ms; $F[1,9] = 7.13$, $p < .026$).

Effects of trial validity at lateral parietooccipital sites. Figure 4 shows the ERPs for valid and invalid trials at lateral parietal and occipital electrodes ipsilateral and contralateral to the visual field of presentation. There was an effect of trial validity on P1 in Experiment 2a ($F[1,9] = 5.31$, $p < .047$). This effect almost reached significance for Experiment 2b ($F[1,9] = 4.33$, $p < .067$). As in Experiment 1, three-way interactions were obtained (Recording Side × Trial Validity × Letter Location: $F[1,9] = 11.25$, $p < .008$ and $F[1,9] = 15.00$, $p < .004$ for Experiments 2a and 2b, respectively), indicating a differential influence of trial validity on P1 for ipsilateral and contralateral recording sites. Again, P1 amplitude was enhanced for invalid trials at electrodes ipsilateral to the stimulus (see Figure 3). In Experiment 2a, this effect was present both at occipital ($t[1,9] = 3.39$, $p < .008$) and parietal ($t[1,9] = 2.71$, $p < .024$) electrodes. A similar result was obtained in Experiment 2b for occipital ($t[1,9] = 2.49$, $p < .034$) and parietal ($t[1,9] = 3.33$, $p < .009$) ipsilateral electrodes. At sites contralateral to the visual field of presentation, no systematic P1 modulation was observed.

N1 amplitude was influenced by trial validity in Experiment 2a ($F[1,9] = 7.27$, $p < .025$), with an enhanced N1 for valid trials. However, this effect was significant only at parietal sites ($F[1,9] = 8.97$, $p < .015$). As in Experiment 1, no main effect of trial validity on N1 amplitude was found when letter position was the response cue (Experiment 2b). Valid trials elicited an enhanced N1 at parietal sites ($F[1,9] = 7.78$, $p < .021$). Three-way interactions were obtained in Experiment 2a (Recording

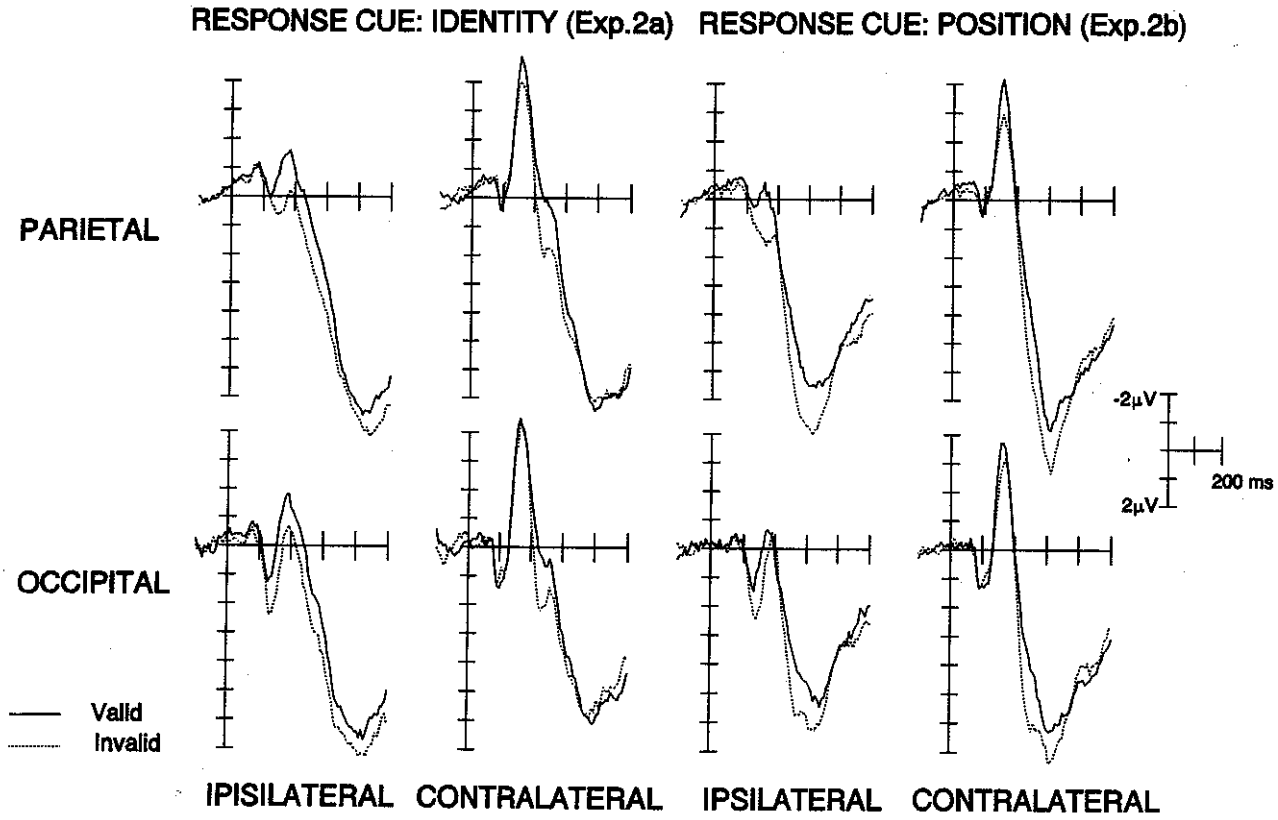

Figure 4. Grand-averaged ERPs at parietal and occipital recording sites for valid and invalid trials, Experiment 2. ERPs recorded from hemispheres ipsilateral and contralateral to the visual field of stimulation are presented separately.

Side × Trial Validity × Letter Location: $F[1,9] = 9.95$, $p < .012$ and $F[1,9] = 10.25$, $p < .011$ for parietal and occipital electrodes, respectively) for the mean amplitude in the Nd_1 time range (130–180 ms), indicating an enhanced negativity for valid trials at ipsilateral electrodes (see also the difference waveforms in Figure 6). The t tests revealed that this effect was present both at parietal ($t[1,9] = 3.61$, $p < .006$) and at occipital ($t[1,9] = 2.41$, $p < .039$) electrodes. In Experiment 2b, a main effect of cue validity on mean amplitude in the Nd_1 time range was found ($F[1,9] = 15.97$, $p < .003$ and $F[1,9] = 7.23$, $p < .025$ for parietal and occipital electrodes, respectively). Additionally, interactions (Recording Side × Trial Validity × Letter Location: $F[1,9] = 9.12$, $p < .014$ and $F[1,9] = 8.41$, $p < .018$ for parietal and occipital electrodes, respectively) indicated that this effect again tended to be located at ipsilateral electrodes.

Effects of trial validity at central electrodes. Figure 5 shows the ERPs for valid and invalid trials recorded at central electrodes. Again, valid trials elicited an enhanced negativity as compared with invalid trials. A bimodal pattern similar to that of Experiment 1 is visible in the difference waveforms (Figure 6). In the Nd_1 range (130–180 ms), trial validity influenced mean amplitude significantly both for Experiment 2a ($F[1,9] = 14.20$, $p < .004$) and for Experiment 2b ($F[1,9] = 7.52$, $p < .023$). Interactions were obtained between electrode location and trial validity for Experiment 2a ($F[2,18] = 4.65$, $p < .054$, $\epsilon = 0.546$) and for Experiment 2b ($F[2,18] = 5.32$, $p < .032$, $\epsilon = 0.664$). Subsequent t tests revealed that the Nd_1 effect was missing at Fz. In Experiment 2a, it was present both at Cz ($t[1,9] = 2.57$, $p < .015$) and at Pz ($t[1,9] = 3.76$, $p < .002$). In Experiment 2b, the effect was significant at Pz ($t[1,9] = 4.68$, $p < .001$) and approached significance at Cz ($t[1,9] = 1.57$, $p < .076$).

In the Nd_2 interval (220–280 ms), there was a highly significant influence of trial validity on mean amplitude for Experiment 2a ($F[1,9] = 15.69$, $p < .009$) and for Experiment 2b ($F[1,9] = 43.75$, $p < .001$). Subsequent t tests showed that this effect was significant at all midline electrodes.

In Experiment 2a, there was no effect of trial validity on P3 amplitude. As in Experiment 1, however, invalid trials elicited enhanced P3 amplitudes as compared with valid trials ($F[1,9] = 7.47$, $p < .023$) when letter location served as the response cue (Experiment 2b).

Discussion

The present experiments were designed to investigate the effects of peripheral trial-by-trial cueing on visual evoked potentials. Two main results were obtained. First, the posterior P1 was enhanced for targets that were preceded by a peripheral cue in the opposite hemifield (invalid trials) as compared with targets following a cue at the same side (valid trials). Second, valid trials elicited an enhanced negativity that emerged as a bimodal Nd effect in the valid–invalid difference waveforms. These effects were quite similar in Experiment 1 and Experiment 2, although cue informativeness was high in Experiment 1 but at chance level in the Experiment 2. Behavioral performance was massively influenced by the level of cue informativeness, with reaction time benefits for valid trials in Experiment 1 and no

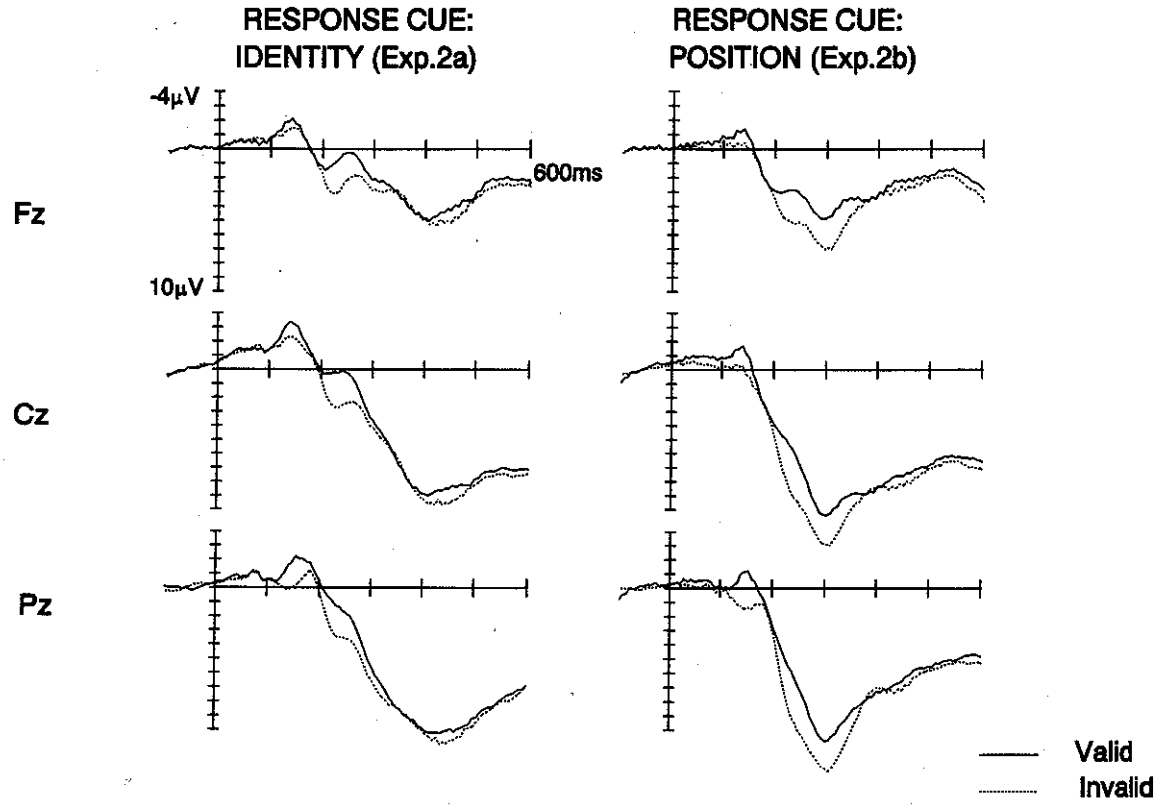

Figure 5. Grand averaged ERPs at Fz, Cz, and Pz for valid and invalid trials, Experiment 2.

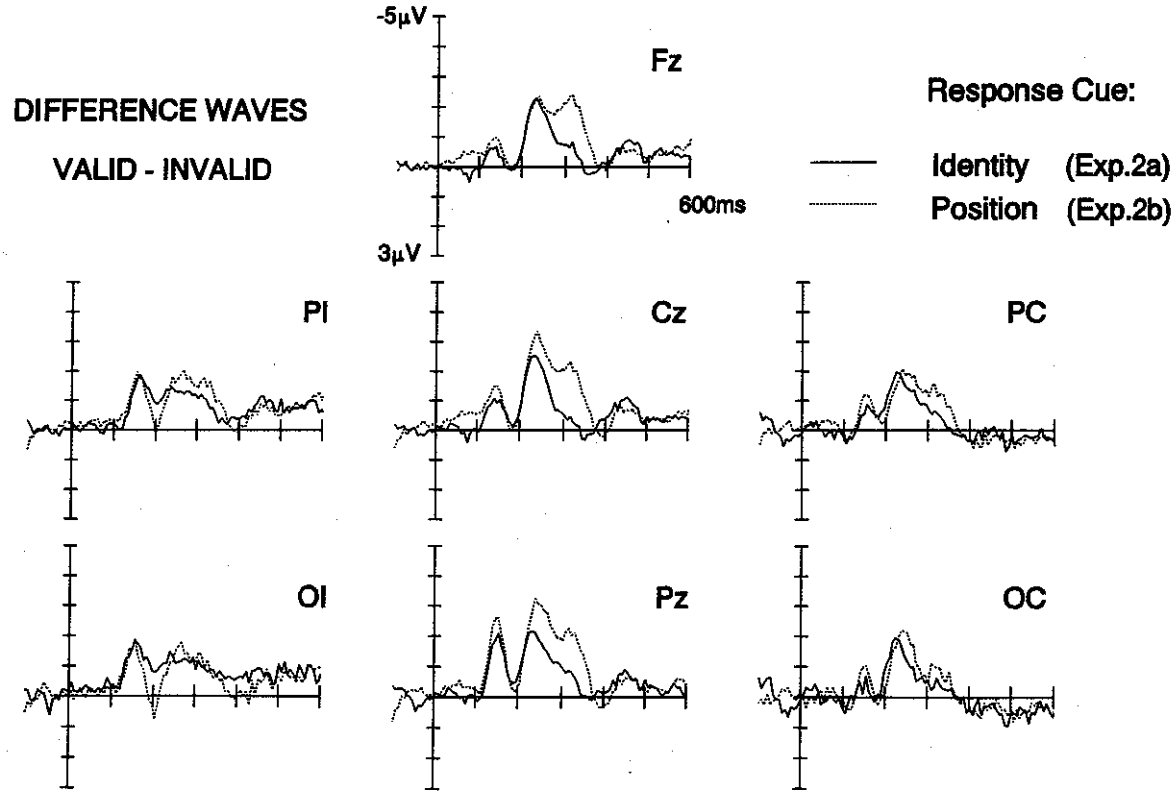

Figure 6. Difference waveforms obtained by subtracting ERPs for invalid trials from ERPs for valid trials for Experiment 2a (solid line) and Experiment 2b (dotted line) for midline electrodes and for lateral parietal and occipital electrodes ipsilateral (PI and OI) and contralateral (PC and OC) to the visual field of the target.

effect of cue validity on reaction time in Experiment 2. On the basis of these behavioral data, one may assume that an attentional orienting process is elicited by informative peripheral cues in Experiment 1 that is comparable to orienting processes caused by central symbolic cues. For Experiment 2, the absence of any validity effect on reaction time data suggests that no attentional orienting process has occurred but also that no inhibition of the type reported by Maylor (1985) has been elicited by the peripheral cue.

The P1 component was enhanced for invalid as compared with valid trials at lateral parietal and occipital electrodes ipsilateral to the visual field of presentation. This result stands in marked contrast to the P1 enhancements for valid trials that have been found in trial-by-trial cueing experiments using symbolic precues (Mangun & Hillyard, 1991) and may have been caused by a sensory interaction between cues and targets on valid trials, when both occurred within the same region of visual space. A P1 reduction in valid trials may be due to either the refractoriness of sensory neurons responsible for the processing of the target or a sensory inhibition of the type described by Maylor (1985). Because the reaction time data of Experiment 2 showed no indication of an inhibition of return, the latter possibility seems rather unlikely. To further distinguish between these alternative explanations, cue–target intervals should be varied systematically in future experiments. Sensory refractoriness should be maximal for short intervals, whereas the inhibitory process postulated by Maylor (1985) is supposed to occur with longer cue–target intervals.

Another possible explanation is that the reduced P1 for valid trials reflects the same phenomenon as the parietal Nd_1, which peaked at about 150 ms poststimulus. The difference waveforms in Figures 3 and 6 show an enhanced negativity for valid trials (reflected in a significant Nd_1 effect for Experiments 1a, 2a, and 2b) at ipsilateral posterior electrodes in the Nd_1 time range, which may have overlapped with the later phase of the ipsilateral P1. Because the peak latency of the ipsilateral P1 was at about 130 ms, whereas the contralateral P1 peaked about 20 ms earlier, this interpretation may account for the fact that a smaller P1 for valid trials was found at ipsilateral but not at contralateral electrodes. However, this interpretation is in conflict with the fact that nearly all ERP studies on visual spatial attention (using both sustained and trial-by-trial cueing paradigms) have reported an enhanced P1 for valid trials, and enhanced negativities to attended stimuli occur later. Further ERP experiments using peripheral cueing are needed to determine whether these differential modulations of the P1 component reflect a general functional difference between the attentional processes elicited by central and peripheral cues.

As in previous ERP studies investigating trial-by-trial cueing (Mangun & Hillyard, 1991; Eimer, 1993), valid trials elicited an enhanced negativity as compared with invalid trials at midline electrodes. Similar to prior findings by Eimer (1993), this effect consisted of two phases. An early phase (Nd_1) between 130 and 180 ms was centered at Pz, and a second phase (Nd_2), with an onset beyond 200 ms, was more broadly distributed over midline recording sites. Additionally, an N1 enhancement was found for valid trials at lateral parietal electrodes and may reflect the same underlying process as the parietal Nd_1 effect, because both effects occur within the same time interval and both almost disappear together in Experiment 1b. Contrary to Mangun and Hillyard (1991), who found an enhancement of the posterior N1 for valid trials only when stimuli had to be discriminated on the basis of their features, N1 validity effects were found in the present study also when the response was dependent on letter position. These effects may reflect another functional difference between peripheral and central cueing. In peripheral cueing, the process reflected by the N1 validity effect may be activated regardless of specific task demands, whereas with central cues, this process is engaged only when focused attention is needed to discriminate between task-relevant stimulus features.[3]

The Nd_2 effect between 220 and 280 ms was highly significant at all midline electrode sites in both experiments and for both response cues. As revealed by the difference waveforms (Figures 3 and 6), the negative enhancement for valid trials extended well beyond 300 ms, most notably when letter position served as the response cue. However, this later phase should not necessarily be identified with the preceding Nd_2 effect because it may be due to the enlarged P3 for invalid trials that was found for Experiment 1b and Experiment 2b. However, the larger P3 amplitude for invalid trials may have been caused by an overlap of the valid P3 with an ongoing enhanced negativity reflecting further processing of the attended stimulus.

Before interpreting the Nd effects in attentional terms, alternative explanations must be ruled out. Because the experiments employed an S1–S2 paradigm, a large contingent negative variation (CNV) was built up in the cue–target interval. Although the CNV is not a topic of the present article, the question arises of whether the Nd effects are a result of differential CNV resolution times for valid and invalid trials. In Experiment 1, reaction times for valid trials were faster than for invalid trials, thus making an explanation in terms of differential CNV resolution seem plausible. If CNV resolution occurred earlier for valid than for invalid trials, this may result in a positive shift in the valid–invalid difference waveforms. Some indications for this are found in the difference waveforms for Experiment 1 (Figure 3), most notably at Cz. In Experiment 2, however, there is no reaction time difference between valid and invalid trials and therefore presumably no systematic difference in CNV resolution. Nevertheless, the Nd effects remained. Moreover, reaction time was almost 200 ms faster when letter location served as the response cue when letter identity was relevant. Therefore, systematic effects of differential CNV resolution should be visible at different time points for both response assignment conditions. However, onset and peak latencies of the Nd_1 and Nd_2 effects are almost identical in the difference waveforms for both response cues (see Figures 3 and 6). Therefore, CNV resolution is not the main cause for the Nd effects.

In addition, these effects cannot be traced back to the fact that for valid trials, imperative stimuli are always preceded by cues in the same hemifield, whereas for invalid trials, cues and targets occur in opposite hemifields. If this physical difference was the sole cause of the enhanced negativity for valid trials, no such effect would be expected for a trial-by-trial cueing experiment using central cues. However, strikingly similar bimodal Nd effects have been found in a visual experiment using central cues (Eimer, 1993) and for peripheral auditory stimuli that were cued by a visually presented central arrow (Schröger & Eimer, 1993).

As an alternative, the Nd effects may be interpreted in terms of the selection or enhanced sensory processing of attended let-

[3] I thank Ron Mangun for making this point.

ters. This assumption seems plausible for Experiment 1, where the cue was informative and thus may have elicited a voluntary shift of attention to the cued side where the imperative stimulus was expected. However, similar Nd effects were recorded in Experiment 2, where the peripheral cue was not informative with regard to letter position and there was no reaction time benefit for valid trials. According to the present interpretation, this finding indicates a processing bias in favor of cued locations even when the cue is not informative. This bias may counteract an initial sensory inhibition of valid trials. However, this presumed processing advantage for valid trials is not reflected in a corresponding reaction time advantage for validly cued targets in Experiment 2. Thus, the processing differences reflected in the ERPs for valid and invalid trials may be largely uncoupled from overt behavior.

However, an enhanced processing of targets at validly cued locations should be elicited more strongly by informative cues. Evidence for this comes from a comparison of the mean amplitudes within the Nd_2 interval of the valid–invalid difference waveforms for Experiment 1 and Experiment 2 (Figure 7). The absolute value of the Nd_2 effect was greater for Experiment 1 than for Experiment 2 for each midline electrode and both response assignment conditions.[4]

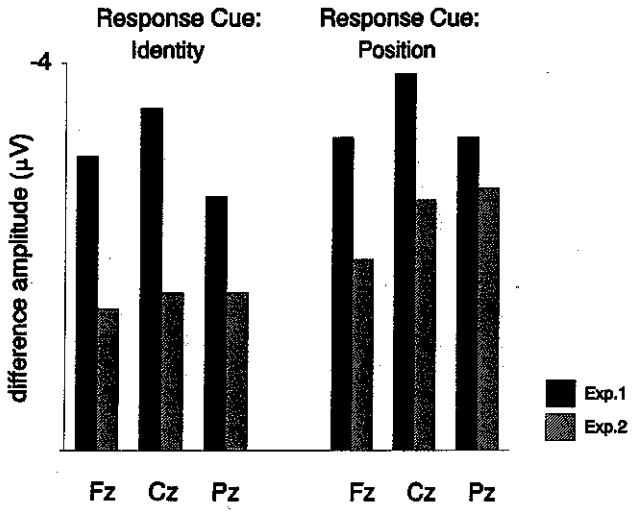

Figure 7. Mean voltage values for the Nd_2 time window (220–280 ms poststimulus) at midline electrodes, Experiment 1 and Experiment 2.

These interpretations are highly speculative. Whether P1 enhancements for invalid trials actually reflect an inhibition of sensory processing for stimuli at locations that have recently been stimulated by a peripheral cue and the assumption that the Nd effects reported here reflect an enhanced processing for stimuli at attended locations merit further experimental investigation. One reason to remain skeptical with regard to the latter assumption is the fact that Nd effects were present in a situation where the precue was uninformative. Moreover, because the Nd_1 and Nd_2 effects occur within different time windows and have different scalp distributions, they may reflect different underlying processes. To answer these questions, additional research is necessary that employs ERP measurements in different experimental situations where visual-spatial attention is directed by peripheral cues.

[4]To test whether these differences between experiments were statistically significant, one-tailed two-sample *t* tests were used. When letter identity served as the response cue (Experiments 1a and 2a), the Nd_2 effect was indeed significantly larger at midline electrodes when cues were informative ($t[18] = 1.79$, $p < .046$). For letter position as the response cue, this difference failed to reach significance.

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