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"Sensory gating" as a mechanism for visuospatial orienting: Electrophysiological evidence from trial-by-trial cuing experiments

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Stimuli at attended-to locations in visual space are usually detected with higher speed and accuracy than stimuli at unattended positions. It has been argued that this effect is due to "sensory gating" mechanisms that modulate the flow of perceptual information from attended and unattended positions. In the present experiments, event-related potentials (ERPs) were recorded to stimuli that were preceded either by a valid or by an invalid positional cue (trial-by-trial cuing). When overt responses were required only to infrequent target stimuli on valid trials (Experiment 1) or to all validly cued stimuli (Experiment 2B), but not to invalid trials, systematic enhancements of early sensory-evoked potentials were found. These effects were smaller when both validly and invalidly cued stimuli required a response (Experiment 2A). These findings are interpreted as evidence that sensory gating processes are activated during the trial-by-trial cuing of spatial attention. Furthermore, valid stimuli elicited a greater negativity than invalid stimuli at midline electrodes following the early enhancements of sensory-evoked potentials. This possibly reflects an additional enhanced processing of attended-to locations.

When subjects are instructed to selectively attend to a specific spatial location, visual stimuli at these locations are detected with higher speed and accuracy than are stimuli presented outside the attentional focus (Bashinski & Bacharach, 1980; Downing, 1988; Hawkins et al., 1990; Müller & Findlay, 1987; Müller & Rabbitt, 1989; Posner, Nissen, & Ogden, 1978). These spatial priming effects can be observed even when eye movements to the attended location are excluded ("covert orienting of attention"). Different mechanisms have been advocated to explain these effects of visuospatial orienting. On the one hand, spatial attention may directly influence perceptual processing, so that stimuli at attended locations are analyzed more rapidly and/or intensively. This intraperceptual model of spatial orienting ("sensory gating") has been advocated, among others, by Posner (1980). On the other hand, the orienting of attention in the visual field might influence primarily postperceptual processes, such as response selection (Sperling, 1984).

When investigating visuospatial attention, the recording of event-related brain potentials (ERPs) may be a useful tool. If it could be shown that spatial attention systematically affects early, sensory-evoked brain potentials, this may be taken as positive evidence for sensory gating mechanisms. In a number of recent ERP studies on visuospatial attention, such effects have indeed been reported (Eason, 1981; Harter, Aine, & Schroeder, 1982; Hillyard & Mangun, 1987; Hillyard & Münte, 1984; Mangun & Hillyard, 1988; Neville & Lawson, 1987; Rugg, Milner, Lines, & Phalp, 1987). In these experiments, subjects were instructed to keep their attention focused on one visual hemifield during a block of stimuli lasting one to several minutes. A response was required only to infrequent target stimuli at the attended side, whereas stimuli at the unattended side were to be ignored. Stimuli at the attended side elicited larger P1 and N1 components at lateral posterior electrodes than did stimuli in the unattended hemifield. These effects start quite early (usually between 80 and 110 msec poststimulus), and neither scalp distributions nor onset latencies of the sensory-evoked P1 and N1 components seem to be altered (Hillyard & Mangun, 1987; Mangun & Hillyard, 1988). These findings have been interpreted as evidence that the behavioral effects of visuospatial orienting are at least partially due to "sensory gain control" mechanisms that modulate the flow of perceptual information from attended and unattended locations (see Mangun & Hillyard, 1990b, for an overview).

A sustained attention paradigm was used in these ERP studies, but evidence for reaction time facilitation has primarily been obtained in situations in which attentional orienting is induced on a trial-by-trial basis by a symbolic precue (e.g., an arrow) that informs subjects on each trial about the likely position of the next imperative stimulus (Posner paradigm). A response is required for both correctly (valid) and incorrectly (invalid) indicated stimuli. There may be important differences between attentional processes elicited within these two paradigms, so it is

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problematic to explain the behavioral effects obtained in a trial-by-trial cuing situation with reference to ERP data collected within the sustained attention paradigm. However, only a few ERP studies on spatial attention have employed the Posner paradigm. In a trial-by-trial cuing study by Mangun and Hillyard (1991; see also Mangun, Hansen, & Hillyard, 1987), enhancements of sensory-evoked potentials to attended stimuli that were comparable to the effects in the sustained attention paradigm were observed. Similar results have been reported by Harter, Miller, Price, LaLonde, and Keyes (1989) for an experiment that differed from the standard Posner paradigm in that overt responses were to be given only to validly cued, imperative stimuli.

On the basis of these findings, Mangun and Hillyard (1991) suggested that functionally similar sensory gating mechanisms may be active in trial-by-trial cuing experiments as well as during sustained attention. However, other experimental results pose some difficulty for this interpretation. In an experiment by Hillyard, Munte, and Neville (1985), subjects were required to attend to one hemifield that was indicated by a precue while short series of flashes were presented randomly in the left or right visual field. P1 enhancement was not visible in response to the first four stimuli presented at the attended location. This lack of a P1 validity effect may have been due to the rather long cue–target interval (1,800 msec) used in that study. In a series of trial-by-trial cuing experiments conducted in our laboratories (Eimer, 1993a, 1993b), enhancements of sensory-evoked potentials to attended stimuli were found to be dependent on specific task characteristics; these effects were present only when the selection of the correct response required a discrimination between different letter stimuli on the basis of single features. They were absent when the response-relevant discrimination was between left- and right-presented stimuli. A second, rather stable effect of spatial orienting was a greater negativity elicited by attended than by unattended stimuli with an onset latency of about 150–200 msec. It is still unclear how this negative enhancement can be interpreted in functional terms.

Thus, it seems that early effects of visuospatial attention on event-related brain potentials are more easily and reliably obtained with sustained attention paradigms than with trial-by-trial cuing. This may be due to the different response assignments used in these two paradigms. In the sustained attention situation, stimuli at the unattended side never require a response and can therefore be completely ignored, but in the Posner paradigm, a response is assigned to all stimuli (including those at the uncued side). In this situation, subjects may not fully focus attention at the side indicated by the precue. Compared with the sustained paradigm, the trial-by-trial cuing situation may thus be understood as a divided-attention condition (see Rugg et al., 1987, and Wijers, 1989, for similar considerations). This would explain why sensory gating effects, as mirrored by P1 and N1 enhancements, are easier to obtain in the sustained paradigm.

The present experiments were designed to investigate this issue by using variations of the Posner paradigm. In the first experiment, responses were required only to infrequent target stimuli at the cued side. For nontargets at the cued side as well as for all stimuli at the uncued side, no response was to be given. These response assignments are equivalent to the response instructions in the sustained attention paradigm usually employed by ERP studies on visuospatial attention. In contrast to the standard Posner paradigm, this experimental situation should presumably constitute a condition in which attention is fully focused at the cued side. If the modulation of P1 and N1 components actually reflects the activity of sensory gating mechanisms, these effects should be present in these experiments. To test whether these attentional modulations are influenced by specific task assignments, as suggested by the results of Eimer (1993b), responses were made dependent on the identity of the target letters in one half of the experiment (Experiment 1A) and on the location of the target letters in the other half (Experiment 1B).

In a second experiment, ERPs recorded in a trial-by-trial cuing situation in which invalidly cued targets had to be ignored was directly compared with ERPs recorded in a situation in which both validly and invalidly cued targets required a response. In the latter case, attention was expected to be partially divided between cued and uncued locations, so the effect of spatial cuing on sensory-evoked potentials was predicted to be smaller or possibly even absent. In addition to investigating these early effects of spatial orienting, another aim of these experiments was to confirm prior findings that visuospatial attention also systematically affects later parts of the ERP waveforms.

EXPERIMENT 1

Method

Subjects. Eight paid volunteers (2 female, 6 male), aged 20–35 years (mean age, 25.4 years) participated in the experiment. All the subjects were right-handed and had normal or corrected-to-normal vision.

Stimuli and Apparatus. The subjects were seated in a dimly lit, electrically shielded and sound-attenuated chamber, with response buttons under their left and right hands. A computer screen was placed 100 cm in front of the subjects' eyes and carefully positioned so that the stimuli (presented as white on gray) occurred on their horizontal straight-ahead line of sight. Each trial began with a 200-msec presentation of a centrally located arrow (subtending a visual angle of 1.5°×0.6°), pointing to either the left or the right side. Seven hundred milliseconds after cue offset, an uppercase letter (M, N, or W) appeared for 100 msec on the left or right side (6° horizontal distance from the screen center), subtending an angle of 1°×1°. The intertrial interval between letter offset and onset of the next arrow was 2 sec.

Procedure. The experiment was divided in half (described as Experiments 1A and 1B), with each part consisting of 12 blocks, resulting in a total of 24 experimental blocks. Each block consisted of 60 trials and had a duration of 2.5 min. Letter stimuli appeared with equal probability on both the left and right sides and were preceded either by an arrow pointing to the side where the letter appeared (valid trials) or by an arrow pointing to the opposite side (invalid trials). The subjects were instructed to react to Ws and Ns

("go" stimuli) when they were preceded by a valid cue and to withhold reaction if the letter M was presented ("no-go" stimulus). The no-go stimulus was presented on 44 trials. It was preceded by a valid cue on 32 trials and by an invalid cue on 12 trials. On the remaining 16 trials, a go stimulus was presented. It was preceded by a valid cue (and thus required a response) on 12 trials and by an invalid cue on 4 trials. Thus, 44 out of 60 trials (73.3%) were valid. The two halves of the experiment differed with respect to the response instructions. In Experiment 1A, the response to valid go stimuli depended on letter identity—the letter W required a left-hand response, and the letter N required a right-hand response. In Experiment 1B, response was conditional on letter position—validly indicated go stimuli on the left and right sides required left and right responses, respectively. The order of the two halves of the experiment was balanced across subjects. ERP results are reported only for those trials in which the no-go letter was presented and no overt response was recorded. The subjects were instructed to respond as quickly and accurately as possible and to maintain central eye fixation during the trials. To familiarize them with these specific task requirements, several training blocks were run at the beginning of the experiment.

Recording. EEG was recorded with Ag-AgCl electrodes from Fz, C3′ (1 cm in front of C3), Cz, C4′ (1 cm in front of C4), Pz (according to the 10–20 system), from PL and PR (located halfway between Pz and the ear channels), and from OL and OR (located halfway between O1 and T5, and O2 and T6, respectively). All the electrodes were referenced to the right earlobe. Horizontal EOG was recorded bipolarly from electrodes at the outer canthi of both eyes, and vertical EOG was recorded from electrodes above and beside the right eye. Electrode impedance was kept below 5 kΩ. The amplifier bandpass was 0.10–70 Hz. EEG and EOG were sampled on line every 7 msec and stored on disk. Reaction times were recorded for each trial.

Data Analysis. EEG and EOG were averaged off line for epochs of 1,800 msec, starting 100 msec prior to arrow (stimulus) onset and ending 800 msec after letter onset. Trials with eyeblinks, horizontal eye movements, overt response errors, or responses in no-go trials were excluded from analysis. After artifact removal, the computer-averaged horizontal EOG for each subject was scored for systematic deviations of eye position in the cue–target interval. If the maximal residual EOG deviation exceeded ± 1 μV (usually indicating a tendency to move the eyes in the arrows' direction), the subject was disqualified. EEG was averaged separately for all combinations of task conditions (response cue, letter identity/letter position; validity, valid/invalid; visual field of presentation, left/right; stimulus identity, M/W/N), resulting in 32 average waveforms for each subject and electrode site. Statistical analyses of ERP components were conducted only for the ERP waveforms elicited by no-go stimuli. Components were measured relative to the mean voltage of the 100-msec interval preceding letter onset. Mean amplitudes were computed over the following latency windows (times given in milliseconds after letter onset): P1 at lateral posterior sites (80–110 msec for electrodes contralateral to the visual field of stimulus presentation; 100–130 msec for electrodes ipsilateral to the stimulus side), N1 at lateral posterior sites (140–200 msec), N1 at midline sites (130–190 msec), and N2 at midline sites (220–280 msec). P3 peak amplitudes at midline electrodes were determined as maximum amplitude values between 300 and 550 msec poststimulus. Separate repeated measures analyses of variance (ANOVAs) were performed on these values for the following factors: electrode location, recording side (left vs. right, for lateral electrodes), cue validity (valid vs. invalid), and letter location (left vs right). When appropriate, a Greenhouse-Geisser adjustment to the degrees of freedom was performed (indicated in the results section by "GG"). For the RT data, repeated measures ANOVAs were performed for the factors of stimulus–response compatibility and response side

Results and Discussion

Behavioral performance. Mean reaction time (RT) to correctly indicated stimuli was 558 msec (Experiment 1A) and 473 msec (Experiment 1B). Overt response errors were recorded for 3% (Experiment 1A) and 0.7% (Experiment 1B) of the go trials. On no-go trials, the rate of false alarms was 0.6% (Experiment 1A) and 0.5% (Experiment 1B). Neither response side nor stimulus–response compatibility influenced reaction time significantly.

ERPs at lateral posterior recording sites. As can be seen from Figure 1, the P1 tended to be enhanced for validly indicated letters at recording sites ipsilateral to the visual field of presentation in Experiments 1A and 1B. This effect reached significance at parietal sites for Experiment 1A [$F(1,7) = 5.70, p < .048$] and at occipital sites for Experiment 1B [$F(1,7) = 5.86, p < .046$], and it approached significance at occipital sites when data of Experiments 1A and 1B were collapsed [$F(1,7) = 4.55$, $p < .070$]. At electrodes contralateral to the stimulus side, no effect of cue validity on P1 amplitude could be found. Valid trials elicited enhanced N1 components over both parts of the experiment both at parietal and occipital electrodes [$F(1,7) = 10.88$, $p < .013$ for parietal sites; $F(1,7) = 11.49, p < .012$ for occipital sites]. A three-way interaction (cue validity × electrode location × visual field of presentation) at parietal [$F(1,7) = 5.88$, $p < .046$] and occipital [$F(1,7) = 7.89, p < .026$] electrodes indicated that this effect was more pronounced over ipsilateral recording sites. These results may be interpreted as evidence for sensory gating. Contrary to the results reported by Eimer (1993b), validity effects on the P1 and N1 were not missing when the response was conditional upon the position of the imperative stimulus (Experiment 1B). This difference is presumably due to the fact that in the experiments reported by Eimer (1993b), identifying the location of a stimulus was sufficient for response decision, whereas in the present Experiment 1B, the subjects had the additional task of discriminating between go and no-go letters.

ERPs at midline recording sites. As can be seen from Figure 2, valid trials elicited a greater negativity at midline electrodes than invalid trials. This negativity showed a first, posterior peak in the N1 range, followed by a second peak that was more anteriorly distributed. This pattern is very similar to prior results that were obtained with the standard Posner paradigm (see Eimer, 1993b). In both parts of the experiment, valid trials elicited a larger negativity in the N1 range than invalid trials [$F(1,7) = 9.82$, $p < .017$]. As revealed by an interaction between validity and electrode location [$F(2,14) = 6.96, p < .018$, GG], this effect was not equally distributed over all midline electrodes (see Figure 2). Subsequent t tests revealed that it was not significant at Fz, but reached significance both at Cz [$t(1,7) = -3.29, p < .013$] and at Pz [$t(1,7) = -3.42, p < .011$]. In the N2 range, a greater negativity for valid than for invalid trials was elicited at all midline electrodes. No overt response was produced in either valid

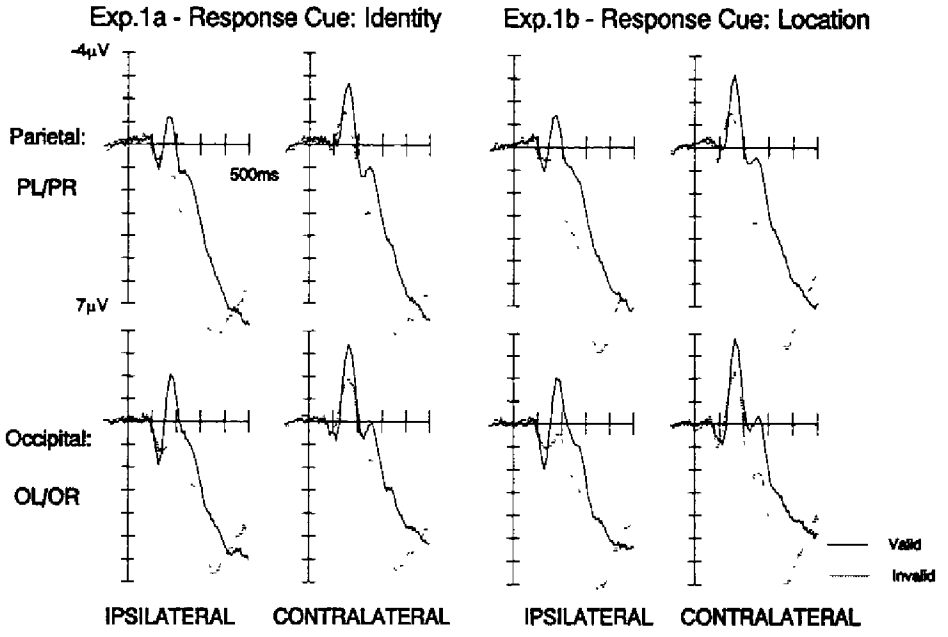

Figure 1. Grand-averaged ERPs to validly (solid lines) and invalidly (dotted lines) cued no-go letters at lateral parietal and occipital electrodes in Experiment 1. ERPs recorded at electrodes ipsilateral and contralateral to the visual field of stimulus presentation are displayed separately: left, Experiment 1A; right, Experiment 1B.

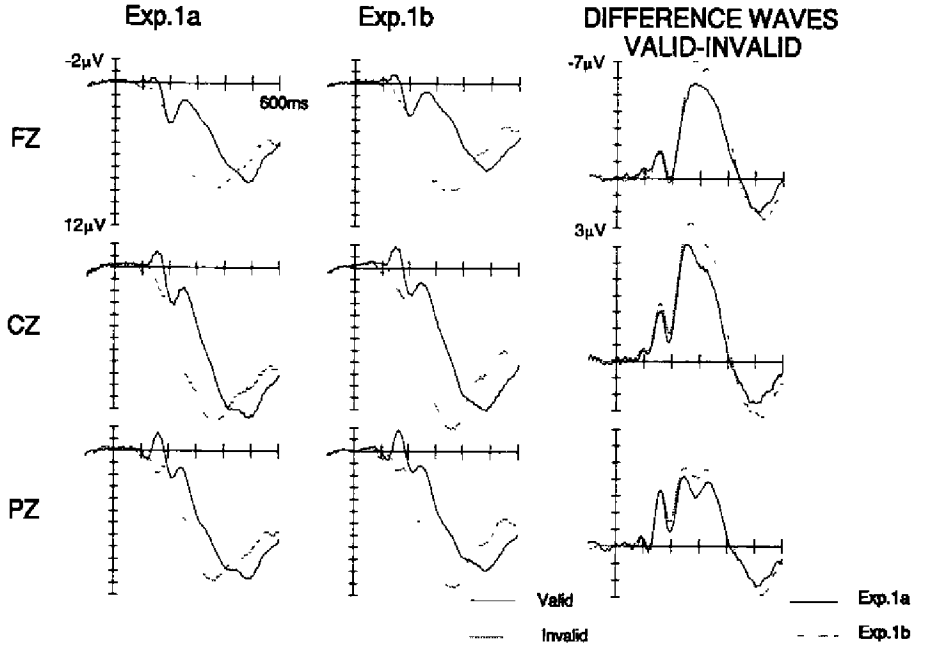

Figure 2. Left and middle columns: Grand-averaged ERPs to validly (solid lines) and invalidly (dotted lines) cued no-go letters at midline electrodes in Experiments 1A and 1B. Right column: Difference curves obtained by subtracting ERPs to invalid trials from those for valid trials. Experiment 1A (solid lines); Experiment 1B (dotted lines).

or invalid trials, so these findings cannot be due to overlap with motor potentials. However, differential CNV (contingent negative variation) resolution times for valid- and invalid-trial effects cannot be completely discounted as one possible source for this effect. Letters occurring at uncued locations never required a response, whereas letters at cued locations sometimes did. In the former case, response preparation may end earlier because no stimulus identification process is neccessary, resulting in an earlier CNV resolution for invalid trials.

Alternatively, it may be considered that the later part of the greater negativity to valid trials is a reflection of enlarged or shorter-latency P3s elicited by the lower-probability invalid trials. An indication for this was found at Fz, where the P3 for invalid trials tended to be larger than that for valid trials over both halves of the experiment. However, this effect failed to reach significance [$F(1,7) = 5.47, p < .052$], and there was no main effect of cue validity on P3 amplitude over all midline electrodes. P3 latency was influenced by cue validity [$F(1,7) = 78.56, p < .001$], with shorter-latency P3s to invalid trials at all midline electrodes.

Experiment 1 provided evidence for the existence of sensory gating mechanisms underlying the orienting of visuospatial attention in a trial-by-trial cuing situation when responses are assigned only to (a subset of) validly cued stimuli. Enhancements of early sensory-evoked potentials to attended stimuli were visible even when they did not require an overt response. In addition to these early effects, Experiment 1 also confirmed the presence of a distinctive cue-validity effect at midline electrodes, consisting of a posterior negativity peaking around 160 msec and followed by a broad frontocentral negativity with an onset of about 200 msec. Experiment 2 was conducted in order to directly compare the modulations of ERP waveforms to valid and invalid trials in a situation in which only validly indicated stimuli require a response with cue-validity effects on the ERP that are obtained with the standard Posner paradigm. To do this, Experiment 2 was also divided in half, with identical stimuli but different response assignments for invalid stimuli.

EXPERIMENT 2

Method

Subjects. Sixteen paid volunteers participated in the experiment. Two of them had to be excluded because of poor eye fixation control in the cue-target interval. Thus, 14 subjects (6 female, 8 male), aged 21–34 years (mean age, 26.1 years) remained in the sample. All the subjects were right-handed and had normal or corrected-to-normal vision.

Stimuli, Apparatus, Procedure, and Data Analysis. These were similar to Experiment 1, except that only two imperative letter stimuli (M and W) were used, and the response instructions were different. Again, 24 experimental blocks were run, each consisting of 60 trials. Both letter stimuli appeared randomly and equally often on both the left and right sides and were preceded either by an arrow pointing to the side where the letter appeared (valid trials) or by an arrow pointing to the opposite side (invalid trials). As in Experiment 1, 44 out of 60 trials (73.3%) per block were valid. In the first half of the experiment (Experiment 2A), the subjects were instructed to respond to validly cued stimuli as well as to invalidly cued stimuli (response to all). In the second half (Experiment 2B), only valid trials required a response (response to valid). As before, the order of the two halves of the experiment was balanced across subjects. In both halves, letter identity determined the response side: the letter M required a left response, and the letter W required a right-hand buttonpress.

Results and Discussion

Behavioral performance. In Experiment 2A, a rather small, but highly significant RT benefit for valid (vs. invalid) trials was found [448 vs. 457 msec; $F(1,13) = 17.15, p < .001$]. The rate of response errors was 4.1% for valid and 3.6% for invalid trials. An additional t test showed that this difference was not significant. Right-hand responses were faster than left-hand responses [445 vs. 461 msec, $F(1,13) = 8.11, p < .014$], and compatible reactions were faster than incompatible reactions [443 vs. 461 msec; $F(1,13) = 10.25, p < .007$]. In Experiment 2B, mean RT to correctly indicated stimuli was 477 msec. Response errors were recorded on 2.3% of the valid trials. The rate of false alarms in invalid trials was also 2.3%. Again, right-hand reactions were faster than left-hand reactions [466 vs. 487 msec; $F(1,13) = 5.79, p < .032$], and compatible reactions were faster than incompatible reactions [465 vs. 489 msec; $F(1,13) = 23.10, p < .001$].

ERPs at lateral posterior recording sites. When only valid trials required a response (in Experiment 2B), occipital P1 amplitude was enhanced to valid trials at ipsilateral sites [$F(1,13) = 8.84, p < .011$]. At contralateral electrodes, this effect approached significance [$F(1,13) = 3.75, p < .075$].[1] When both valid and invalid trials were response relevant (in Experiment 2A), no significant enhancement of occipital P1 amplitude was found at ipsilateral and contralateral electrodes (see Figure 3). The fact that the different response assigments in Experiments 2A and 2B influenced the occipital P1 validity effect was also reflected in an interaction between cue validity and experimental half [$F(1,13) = 5.15, p < .041$, for ipsilateral occipital electrodes]. These results suggest that attentional allocation may indeed have been different in Experiment 2A (in which attention was expected to be partially divided between cued and uncued locations) and in Experiment 2B (which was thought to represent a situation in which attention was fully focused). Similarly, the N1 validity effect was significantly influenced by the different response instructions [as indicated by an interaction of experimental half × cue validity; $F(1,13) = 26.14$, $p < .001$ for parietal electrodes; $F(1,13) = 7.15, p < .019$ for occipital electrodes]. When all stimuli required a response (in Experiment 2A), there was a moderate effect of cue validity on parietal N1 amplitude [$F(1,13) = 5.13, p < .041$], but no validity effect on occipital N1. Three-way interactions [cue validity × recording side × visual field of presentation; $F(1,13) = 9.64, p < .008$ for parietal electrodes; $F(1,13) = 11.45, p < .005$ for occipital electrodes] indicated that valid trials tended to elicit an enhanced N1 at electrodes ipsilateral to the im-

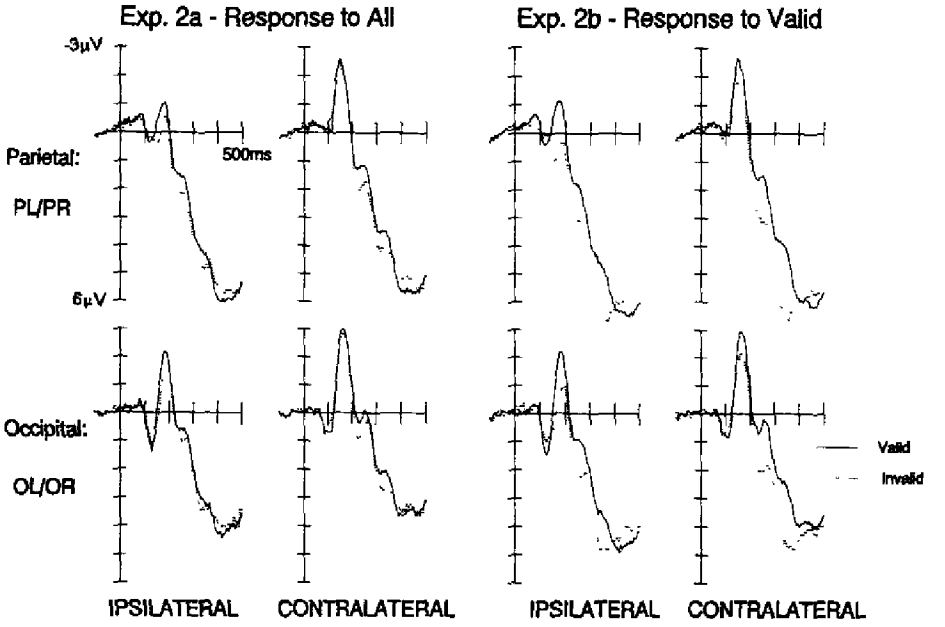

Figure 3. Grand-averaged ERPs to validly (solid lines) and invalidly (dotted lines) cued letter stimuli at lateral parietal and occipital electrodes in Experiment 2. ERPs recorded at electrodes ipsilateral and contralateral to the visual field of stimulus presentation are displayed separately: left, Experiment 2A; right, Experiment 2B.

perative stimulus (see Figure 3). In Experiment 2B, a main effect of cue validity on N1 amplitude was found both at parietal and at occipital electrodes [$F(1,13) = 27.95, p < .001$ for parietal sites; $F(1,13) = 7.04, p < .020$ for occipital sites]. Again, three-way interactions [cue validity × recording side × visual field of presentation; $F(1,13) = 7.69, p < .016$ for parietal electrodes; $F(1,13) = 6.77, p < .022$ for occipital electrodes] showed that this effect was at a location that was primarily ipsilateral to the stimulus presentation side.

When a response was required to both valid and invalid trials, P1 and N1 validity effects were smaller, as compared with the situation in which a response was assigned solely to valid trials. This may be interpreted as an indication that sensory gating mechanisms were activated more strongly in the latter condition. However, the differential N1 validity effects obtained for both response instructions may at least in part result from an earlier onset of CNV resolution for invalid (no-go) trials in Experiment 2B.

ERPs at midline recording sites. When a response was required in all trials (in Experiment 2A), a parietal negativity to valid trials in the N1 range that almost reached significance was found [$F(1,13) = 4.58, p < .052$]. In Experiment 2B, there was a validity effect in the N1 range [$F(1,13) = 20.94, p < .001$] as well as an interaction between cue validity and electrode location [$F(2,26) = 13.51, p < .001$, GG]. Further t tests revealed that the validity effect was present at Pz and Cz, but was not significant at Fz (see Figure 4).

In the N2 range, a greater negativity to valid trials was found in both halves of the experiment [$F(1,13) = 27.61, p < .001$ for Experiment 2A; $F(1,13) = 23.21, p < .001$ for Experiment 2B]. This effect was significant at all midline electrodes and was larger in Experiment 2B than in Experiment 2A [as indicated by an interaction between cue validity and experimental half: $F(1,13) = 7.61, p < .016$]. In Experiment 2A, there was no effect of cue validity on P3 amplitude or latency. When only valid trials were response relevant (in Experiment 2B), these trials elicited a longer-latency P3 than did invalid trials [$F(1,13) = 28.54, p < .001$]. Cue validity also influenced P3 amplitude in Experiment 2B, with larger P3s to invalid trials [$F(1,13) = 8.41, p < .012$]. An interaction between cue validity and electrode location [$F(2,26) = 18.13, p < .001$, GG] indicated that enlarged P3 amplitudes to invalid (as compared with valid) trials were present at Fz and Cz, but not at Pz (see Figure 4). Again, it is unclear whether these effects were primarily caused by attentional processes or whether they were partially due to an earlier CNV resolution in the no-go trials of Experiment 2B.

GENERAL DISCUSSION

The aim of the present experiments was to look for electrophysiological evidence for the "sensory gating" mechanisms underlying processes of visuospatial orienting in a trial-by-trial cuing situation. It was found that early sensory-evoked potentials are affected by the direction of

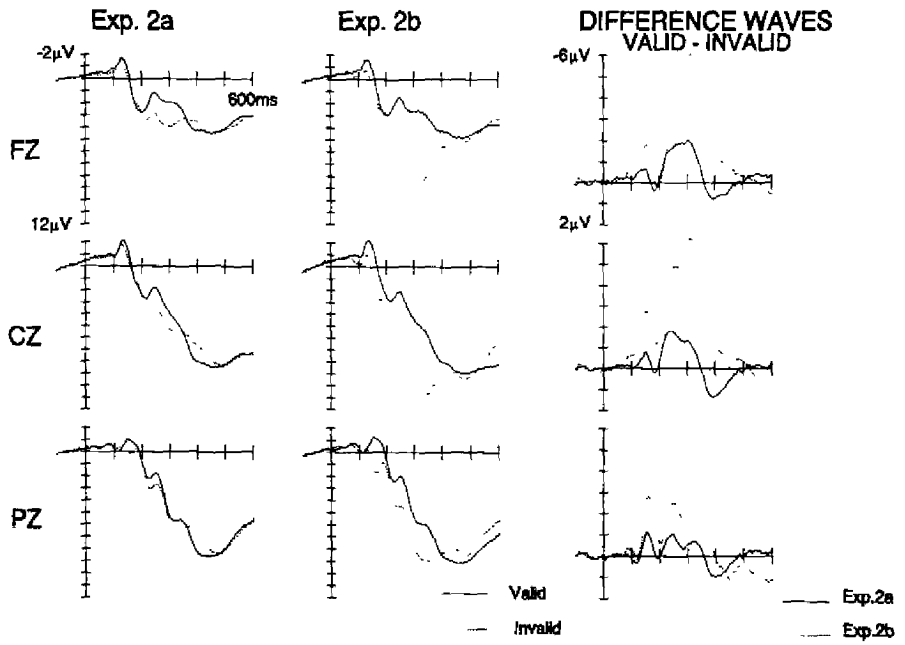

Figure 4. Left and middle columns: Grand-averaged ERPs to validly (solid lines) and invalidly (dotted lines) cued letter stimuli at midline electrodes in Experiments 2A and 2B. Right column: Difference curves obtained by subtracting ERPs to invalid trials from those for valid trials. Experiment 2A (solid lines); Experiment 2B (dotted lines).

spatial attention. Stimuli at attended locations usually elicit larger P1 and N1 components at lateral parietal and occipital sites than stimuli at unattended locations. Figure 5 shows a summarized plot of these effects. These components are considered to be primarily exogeneous, so this finding can be interpreted as positive evidence that spatial attention has a modulatory influence on the flow of perceptual information from specific locations in visual space.

Enhanced P1 and N1 components to validly indicated letters were found in Experiment 1, in which invalidly cued stimuli were to be ignored by the subjects. This is in line with the hypothesis that sensory gating mechanisms are activated when unattended stimuli are irrelevant. In Experiment 2, validity effects on lateral posterior P1 and N1 components were found in the condition in which only validly cued letters required a response. When both valid and invalid trials were response relevant, the N1 validity effect was considerably smaller, and no significant enhancement of the occipital P1 component was found for valid trials (see Figure 5). These differences were reflected in the significant interactions between cue validity and response assignments for parietal and occipital N1 amplitudes and for the ipsilateral occipital P1. However, it should be noted that in the case of the N1 component, these interactions may have been due in part to differential CNV resolution times for valid and invalid trials in both halves of the experiment. Nevertheless, the differential effects of spatial attention on sensory-evoked potentials obtained in Experiments 2A and 2B suggest that different attentional allocation strategies may be involved in the standard Posner paradigm and in trial-by-trial cuing situations in which the response requirements are equivalent to the sustained attention paradigm. In the former case, attention may be divided between cued and uncued locations, whereas in the latter case, attention may be strictly focused at the cued location. The fact that different attentional allocation strategies (divided vs. focused attention) are reflected in differential modulations of P1 and N1 components has recently been demonstrated by Mangun and Hillyard (1990a). Sensory gating mechanisms may thus play a less prominent role in the standard Posner paradigm than they do in experimental circumstances in which invalid trials are always no-go trials.

It has been argued by Mangun and Hillyard (1991) that similar mechanisms of visuospatial attention operate during sustained attention and trial-by-trial cuing. In most ERP studies on visuospatial attention using sustained attention paradigms, attentional modulations of P1 amplitude have been found to be either bilaterally symmetric or maximal at contralateral recording sites. The N1 attention effect has usually been found to be larger at recording sites that are contralateral to the visual field of presentation. This pattern could not be replicated in the present experiments; both the P1 and the N1 validity effect were larger over scalp sites that were ipsilateral to the imperative stimulus (see Figure 5).[2] Although an ipsilateral N1 validity effect has already been reported by Mangun and Hillyard (1991), the ipsilaterality of the P1

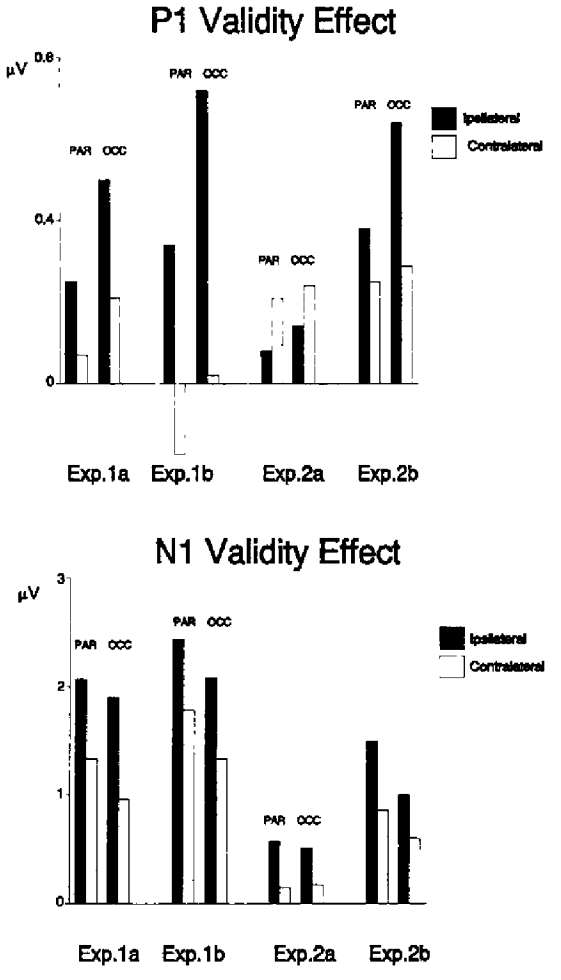

Figure 5. Bar graphs showing the effects of cue validity on the amplitudes of the P1 and N1 components for Experiments 1 and 2. Top: Mean amplitudes of the difference between ERPs to valid and invalid trials (valid minus invalid) for the P1 component at lateral parietal (PAR) and occipital (OCC) electrodes ipsilateral (100–130 msec) and contralateral (80–110 msec) to the visual field of stimulus presentation. Upward bars indicate that that the P1 amplitude was more positive for valid than for invalid trials. Bottom: Mean amplitudes of the difference between ERPs to valid and invalid trials (valid minus invalid) for the N1 component (140–200 msec) at lateral parietal (PAR) and occipital (OCC) electrodes ipsilateral and contralateral to the visual field of stimulus presentation. Upward bars indicate that that the N1 amplitude was more negative for valid than for invalid trials.

attention effect is a novel finding. As can be seen in Figures 1 and 3, a P1 enhancement to valid trials could be found only during the later phase of this component—that is, usually beyond 100 msec poststimulus.[3] At this time, the P1 maximum had already switched from the contralateral to the ipsilateral scalp side. It is therefore quite likely that the ipsilaterality of the P1 validity effect is due to the fact that it develops only during the later, ipsilaterally distributed portions of the P1 component. Although it is not clear whether these different scalp distributions of attentional effects on sensory-evoked brain potentials reflect important functional differences in the mechanisms underlying attentional orienting in both experimental situations, they will have to be taken into account by theories assuming that similar processes are elicited both during sustained attention and during trial-by-trial cuing.

Another dispute concerns the question of whether sensory gating is the most prominent or possibly the only mechanism underlying visuospatial orienting. In the present experiments, a systematic pattern of negative enhancements to validly cued trials was found at midline electrodes. The first peak of this negativity was found at Pz at about 150 msec; a second negative peak was located more anteriorly and had an onset beyond 200 msec poststimulus. The latter effect may be partially explained by reference to differential CNV resolution times for valid and invalid trials or to an enlarged P3 to lower-probability invalid trials. The fact that no-go trials usually elicit an enlarged and more anteriorly distributed P3 than go trials (see Eimer, 1993a) may also partially be responsible for the greater negativity elicited by valid trials. However, these explanations cannot fully account for the fact that quite time-invariant bimodal patterns with distinct scalp distributions for both negative peaks were found in all four parts of the experiments as well as in previous trial-by-trial cuing experiments (Eimer, 1993a, 1993b), in which response requirements were rather different. It may thus be considered whether these negativities are a reflection of an enhanced postperceptual processing of attended-to stimuli that follows earlier intraperceptual sensory gating mechanisms. This hypothetical process may also be responsible for the RT benefits for valid trials that are usually observed for Posner-like trial-by-trial cuing situations, even when there is no ERP evidence for early sensory gating.

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NOTES

1. As can be seen in Figure 3, the enhancement of the contralateral P1 for valid trials seems to be restricted to the later portion of this component. Further analyses revealed that for the leading edge of the contralateral P1 (75-95 msec), no validity effect was present, but for its second phase (95-115 msec), valid trials elicited an enhanced positivity both at parietal and occipital contralateral electrodes. At occipital electrodes, this effect was significant for Experiment 2B [$t(1,13) = 3.31$, $p < .006$], but failed to reach significance in Experiment 2A [$t(1,13) = 1.86$, $p < .086$].

2. The only exception is Experiment 2A, in which the P1 enhancement to valid trials was found to be larger at contralateral posterior electrodes (see Figure 5). However, this contralateral enhancement failed to reach statistical significance.

3. This finding differs from the results reported by Mangun and Hillyard (1991), who found a P1 validity effect also for the first phase of this component. During this phase it was maximal over contralateral scalp sites, but it was ipsilaterally located during the later portion of the P1.

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