Spatial attention triggered by eye gaze increases and speeds up early visual activity

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What are the neuronal correlates of reflexive shifts of attention triggered by eye gaze direction? Event related potentials (ERPs) were measured on 14 subjects performing a spatial attention task where eye gaze direction of a face cued the location of a forthcoming target. Subjects were faster in detecting a validly cued target, i.e. one appearing at the location the eye was gazing at, compared to invalidly cued targets, despite the non-predictive value of the eye cues. ERP results showed an enhanced and earlier occipito-parietal P1 and N1 for valid trials, demonstrating the early modulation of visual input by attentional allocation. These findings provide the first evidence that social attention can rapidly modify the processing of visual information in extrastriate cortex. *NeuroReport* 12:2381–2386 © 2001 Lippincott Williams & Wilkins.

Key words: ERPs; Eye gaze; N1; P1; Social attention; Spatial attention

INTRODUCTION

When visual attention is directed toward a specific spatial location, stimuli presented to that location are detected and discriminated with greater speed and accuracy then when they are presented outside the attended focus [1]. These effects are independent of eye movements (covert orienting of attention) and have been interpreted as the results of enhanced sensory–perceptual processing for stimuli at attended location [1]. This hypothesis of a perceptual locus of attentional selection has been largely confirmed in humans by event-related potential (ERP) studies [2,3] (for a review see [4].

The classic ERP paradigm to study spatial attention consists of comparing ERP waveforms elicited by stimuli flashed in an attended visual hemifield to electrophysiological responses evoked by the same physical stimuli presented in an unattended visual hemifield [5]. These studies have demonstrated amplitude increases of the occipital P1 wave, a positive deflection originating from extrastriate areas 70–100 ms post-stimulus, and of the parieto-occipital N1 component (150–200 ms) but the peak latency of these components does not appear to be reliably modulated by spatial attention. Similar results have been observed with spatial cueing tasks when attention is modulated trial-by-trial using a symbolic cue (e.g. an arrow) priming the most likely location of a forthcoming target [2]. In these conditions, subjects' responses are faster and more accurate when the target appears at the cued location (valid trials) than when the target appears at an uncued location (invalid trials) [1]. Similarly, larger sensory-evoked responses are observed for the targets on valid than invalid trials, indicating that the enhanced behavioural speed and accuracy is caused, at least partly, by enhanced sensory processes [5]. Cueing with a central arrow has been defined as endogenous cueing because a decoding of cue information must occur first, this can then be followed by a voluntary attentional allocation [4]. In the same way, sustained attention by instructions can be defined as a kind of endogenous spatial attention cueing. On the other hand, exogenous or reflexive effects of attention have also been described for peripheral cues (at the same locations than the forthcoming targets), with overall similarities in the ERP modulations for symbolic and peripheral cueing, but also notable differences, such as an absence of P1 effect for reflexive attention by peripheral targets [4].

Despite these differences, the general interpretation of these ERP observations during spatial attention tasks is that sensory gain control mechanisms enhance neural activity in visual extrastriate cortex [6], facilitating the processing of attended stimuli.

Recently, several studies have replaced symbolic predictive cueing in spatial attention tasks by socially relevant cues such as eye gaze direction and head orientation [7–11]. A lateralized target is preceded by a face gazing either in the same direction (valid trials) or in the opposite direction (invalid trials) and subjects have to detect the occurrence of the target, or make a discrimination judgement on it, as fast as possible. In this situation, subjects' responses are faster in valid trials, even when subjects are aware that the cue (the eye gaze direction) is not predictive of the localization of the forthcoming target. This spatial attention modulation by eye gaze direction is robust and has been described in paradigms with [7] or without eye

movement of the cue [8,9,11,12], with schematic [8,12] or real face photographs [10,11], various stimulus onset asynchronies (SOAs) between the eye gaze cue and the target, and for different types of target responses (e.g. detection, location discrimination or identification tasks [8]). Because the preceding central cue is not predictive of the location of the forthcoming target, it has been suggested that the facilitation effect produced by the gaze cue reflects the involvement of exogenous or reflexive covert attention [8].

In the present experiment, we used ERPs to investigate the onset of spatial attention triggered by eye gaze direction on visual processing in the human brain. More precisely, we aimed to test whether attention triggered by eye gaze, or reflexive attention by a central non-predictive cue, modulates early extrastriate activity, represented by visual P1 and N1 components. Such findings would provide the first evidence that social attention can rapidly modify the processing of visual information in extrastriate cortex, and help to clarify the locus of reflexive attentional selection. To do so, we used a spatial attention task, where eye gaze direction cued the forthcoming location of a target in an ecological way. Considering the behavioural results on eye gaze cues and the ERP results on spatial attention, we predicted that the cueing effects should be at least as powerful as those observed in symbolic cueing paradigms. If similar neuronal mechanisms are underlying the attentional shift mechanisms as for symbolic cueing, enhanced P1 and N1 should be observed for targets following congruent eye gaze directions.

MATERIALS AND METHODS

Subjects: Fourteen paid volunteers (seven males, one male left handed), aged 21–27 years participated in the study. All had normal or corrected to normal vision.

Stimuli and procedure: Subjects were seated in a comfortable chair in a dimly lit, electrically shielded room, at a distance of 80 cm from a monitor screen, their head restrained by a chin rest. Stimuli were one picture of a full front female face with the eyes fixating the viewer, and two pictures of the same face with eye gaze averted, towards the left and right visual fields (Fig. 1). All face photographs subtended a visual angle of 7.15° horizontally and 3.9° vertically, and were presented on a white background. A simple black cross (0.5/0.5°) was used as a target.

A trial was always made of the following events: a face gazing at the viewer (500 ms), followed by the same face with eyes averted (left or right, 500 ms), and then the lateralized target cross appearing randomly in half of the trials in the right visual field (RVF), in the other half in the left visual field (LVF) at a distance of 6.8° from the centre of the screen (Fig. 1). The face with eyes averted and the target remained on the screen until the subject's response. The next trial was presented after a 500 ms intertrial delay. Given the continuous stimulus presentation, the whole sequence, as illustrated in Fig. 1, was perceived as a face moving the eyes towards the left or right side of the screen followed by a target cross at a congruent or incongruent position. In total, four combinations were used: target location was congruent with the eye direction (valid-LVF/valid-RVF), or opposite to the gaze direction (invalid-LVF/invalid-RVF).

Subjects ran four blocks of 45 trials with a rest between each block. Ninety of the trials were valid trials, where eye direction and target location were congruent, and 90 were invalid trials, in which eye direction and target location were in opposition.

Throughout the experiment, subjects were instructed to maintain fixation to the central face. Twenty practice trials were run before starting the experiment and subjects knew that the direction of eye gaze was not predictive of the location of the following target. They were required to press the left button of the response box when the target

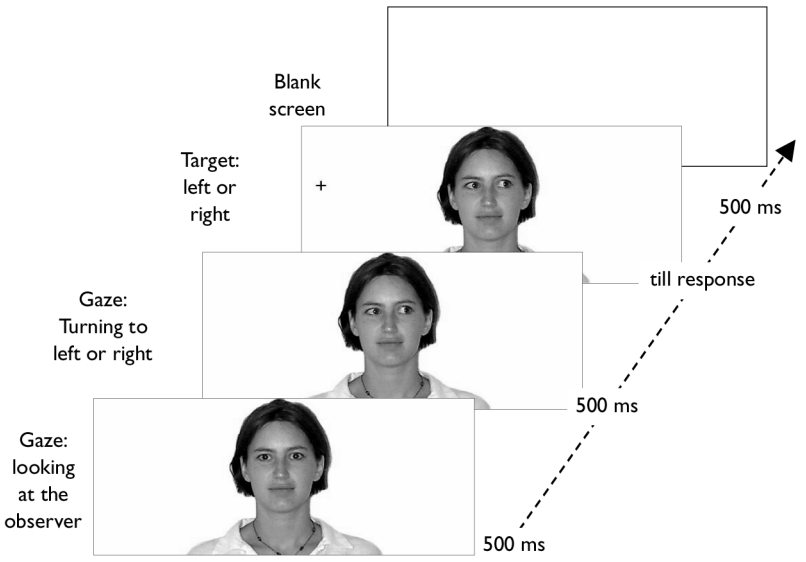

Fig. 1. Stimuli and procedure used in this study (here an invalid trial, with the target appearing in the opposite direction of the eye gaze cue).

was shown in the LVF and the right button when the target was presented in the RVF, and to be as accurate and as fast as possible, using their dominant hand to respond.

ERP recordings and data analysis: Recordings were made using tin electrodes in a 64 channel modified quick-cap (Neuromedical Supplies, Inc.), adapted from the 10-20 montage. Electrodes FC3/FC4, C3/C4, CP3/CP4 were removed and replaced by six additional low occipito-temporal electrodes (TP9/TP10, P9/P10, PO9/PO10; Fig. 3). Horizontal EOG recording electrodes were positioned at the outer canthi of both eyes and vertical EOG recording electrodes were placed above and below the left eye. The reference electrode was positioned on the tip of the nose. EEG was amplified with a gain of 30 K and bandpass filtered at 0.01–100 Hz. Electrode impedance was kept $< 5\,k\Omega$. EEG and EOG were sampled at a digitization rate of 500 Hz.

After removal of EEG and EOG artefacts, averages were generated for each subject and each of the four conditions in epochs of −200 to 824 ms (valid-LVF/valid-RVF/invalid-LVF/invalid-RVF). The ERPs were then filtered with a lowpass filter of 20 Hz and a highpass of 2 Hz, to facilitate automatic latency peak detection. Based on the topographical maps (Fig. 3), P1 amplitude was automatically computed as the mean amplitude values in a window of 90–150 ms, relative to a 200 ms pre-stimulus baseline on parietal–occipital electrodes (PO3/PO4, P3/P4, P5/P6). The highest amplitude value in this window was taken as the P1 peak latency. N1 peak latency and amplitude were similarly measured in a 140–220 ms window, on the same parietal–occipital electrodes (PO3/PO4, P3/P4, P5/P6). In addition, the late positivity (P3) was measured on electrodes Pz, POz and Cz in a 280–340 ms window.

A four-ways repeated measures ANOVA was conducted on the amplitude and latency data measured on the parietal-occipital P1 and N1 with cue validity (valid/invalid), visual field stimulation (LFV *vs* RVF), recording site (left *vs* right, for lateral electrodes) and electrode location as factors. Additional *post-hoc* comparisons were performed as required. For the P3 analysis, left and right target averages were grouped and a two-ways ANOVA with cue validity and electrode location as factors was computed.

RESULTS

Behavioural performance: Mean correct response times were 306 ms for the valid trials and 325 ms for the invalid trials ($F(1,13) = 17.919$; $p = 0.001$). There was no any main effect of visual field stimulation nor interaction. Errors occurred on $< 3\%$ of the trials and were excluded from ERP analyses.

ERPs: Grand-average ERPs over the 14 subjects elicited by the left- and right-field stimulation are illustrated in Fig. 2. Three clear components were elicited following the target onset (Fig. 2, Fig. 3): a positive deflection at posterior sites peaking at around 130 ms and starting in the contralateral hemisphere, termed the P1; a negative occipital/parietal component also starting at contralateral sites and peaking around 170 ms (N1), and a late positivity over central sites peaking around 300 ms, which was identified as a P300.

There was a significant enhancement of the P1 amplitude for validly cued targets as compared to invalidly cued targets ($F(1,13) = 5.332$; $p = 0.038$; Table 1). This effect was qualified by a significant interaction between the factors cue validity and visual field stimulation ($F(1,13) = 10.648$; $p = 0.006$): there was a significant P1 amplitude increase for valid trials for RVF (*post-hoc t*-tests: $p = 0.0007$) but not for LVF stimulation ($p = 0.928$). Furthermore, there was an interaction between visual field stimulation and electrodes ($F(2,26) = 1.197$; $p = 0.0038$) due to a larger P1 for LVF than RVF stimulation for electrode pairs PO3/PO4 and P3/P4 (*post-hoc*: $p < 0.0007$) but not for the more lateral pair P5/P6 (*post-hoc*: $p = 0.0007$).

The ANOVA on P1 latency also revealed a main effect of cue validity ($F(1,13) = 6.70$; $p = 0.02$), reflecting an earlier P1 to validly cued targets compared to invalid trials. Moreover, P1 latency was modulated by visual field stimulation, peaking earlier in the contralateral hemisphere ($F(1,13) = 12.670$; $p = 0.0028$; Fig. 3). This contralateral effect was modulated by electrode laterality ($F(2,26) = 8.008$; $p = 0.002$): RVF stimulation gave rise to a significant earlier P1 in the left hemisphere (*post-hoc*: $p = 0.002$) but this effect was only significant for electrode P4 in the right hemisphere ($p = 0.0229$ for P4; other all p values > 0.11).

ERPs to the validly cued targets also showed a significant shorter N1 latency than in invalid trials ($F(1,13) = 16.81$; $p = 0.0012$). N1 latency was also modulated by visual field stimulation, thus peaking earlier in the contralateral hemisphere ($F(1,13) = 12.16$; $p = 0.004$; Fig. 3). This contralateral effect was modulated by electrode laterality ($F(2,26) = 6.663$; $p = 0.0046$): RVF stimulation gave rise to a significant earlier N1 in the left hemisphere (*post-hoc*: $p < 0.01$) and targets presented in the LVF evoke an earlier N1 on all the right-sided electrodes ($p < 0.01$) but PO4 ($p = 0.067$).

Because of the large latency difference between valid and invalid trials (Fig. 2), comparison of the amplitude values were made over two different time windows, in the rising edge of the N1 (140–190 ms) and the descending slope of the component (180–220 ms) [2]. The amplitude of the N1 peak measured out on the first window (140–190 ms) was significantly larger for validly cued targets ($F(1,13) = 13.858$; $p = 0.0025$) but there was no such effect of cue validity on the second part of the N1 component ($F(1,13) = 0.307$; $p = 0.588$; Fig. 2). During the first window, there was a significant interaction between visual field stimulation and electrode laterality, reflecting an enhanced N1 for controlateral hemifield stimulation ($F(1,13) = 34.996$; $p = 0.00005$). *Post-hoc* tests showed that this contralaterality effect was significant for every electrode but PO4 ($p = 0.37$ for PO4; other p values < 0.0001). During the second time window (180–200 ms), there was a significant interaction between visual field stimulation, electrode laterality and electrodes ($F(2,26) = 4.099$; $p = 0.028$): for each left sided electrode, no significant differences between LVF and RVF stimulation were found (all $p > 0.41$) but for electrode PO4, P4 and P6, RVF targets elicited a larger N1 than LVF stimulation (all $p < 0.01$).

The latency of the P3 was not significantly modulated by cue validity or by any other factor. However, the ampli-

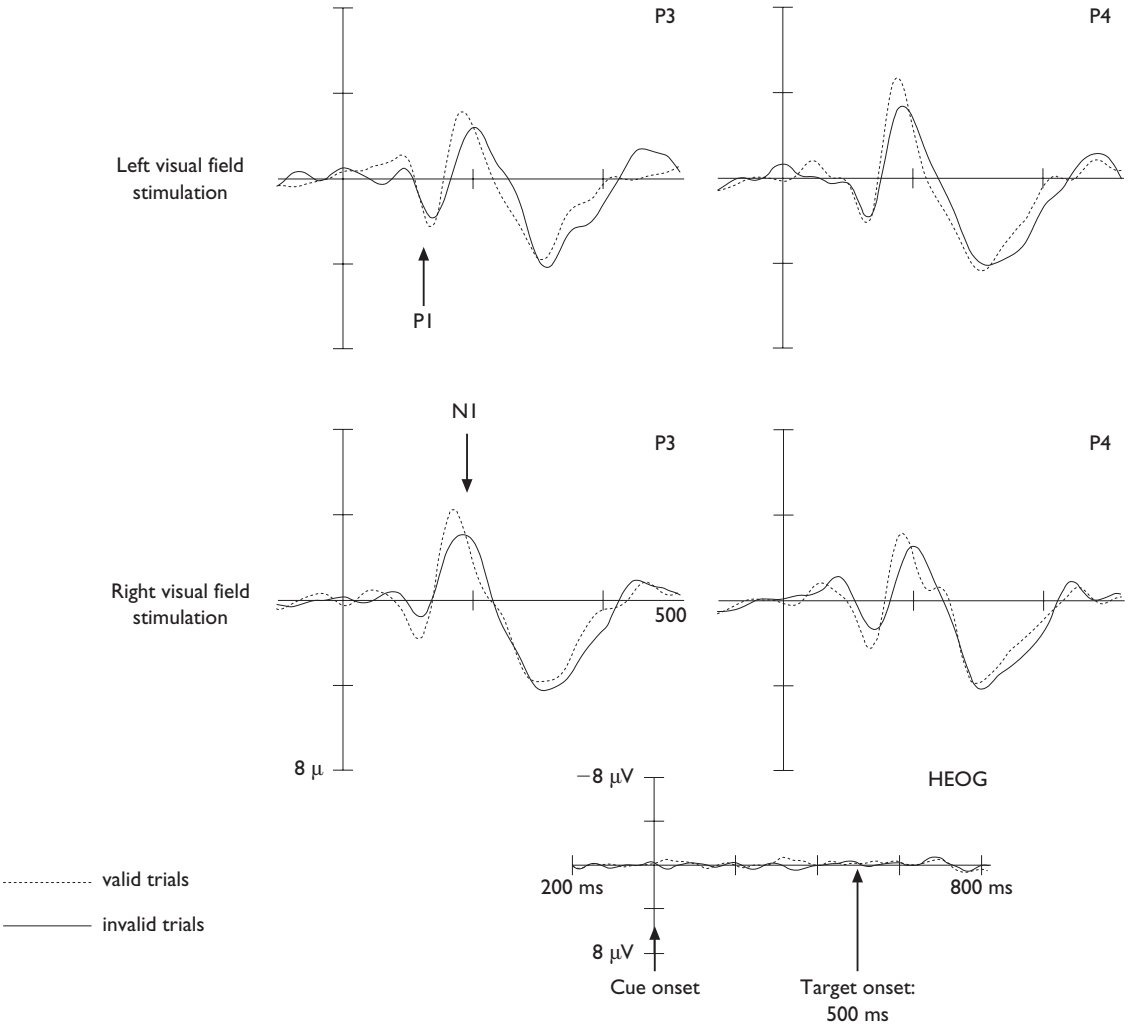

Fig. 2. Grand-averaged ERPs over 14 subjects to validly (dotted lines) and invalidly (solid lines) cued targets at posterior electrodes P3 (left hemisphere) and P4 (right hemisphere) when the targets are presented in the LVF and in the RVF. Below, the grand-averaged horizontal EOG from the onset of the cue (dotted line = eye gaze towrd the right; solid line = eye gaze toward the left).

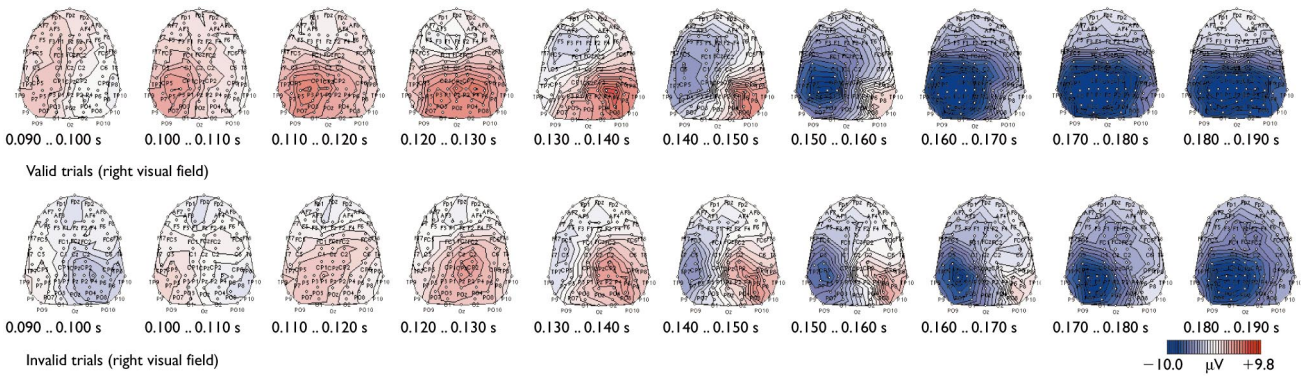

Fig. 3. Grand-averaged scalp voltage distribution at 10 ms latency ranges for valid and invalid trials when targets are presented in the RVF. Note the parieto-occipital distribution of the P1 and N1. The P1 (positivity in red, starting around 90 ms) and N1 (negativity in blue, starting around 150 ms) are larger for valid trials. Note also that these components start in the contralateral (here the left) hemisphere and then spread to the ipsilateral side. The contralateral hemispheric dominance of visual field stimulation in P1 latency and N1 latency and amplitude (expressed by statistically significant interactions between visual field stimulation and electrode side) reflects the anatomical projections of the geniculostriate pathway to the visual cortex.

Table 1. Mean latencies and amplitudes (grand averaged data) of P1 and N1 components measured on electrodes P3 and P4 for valid and invalid trials during LVF and RVF stimulation.

		Left hemisphere (P3)		Right hemisphere (P4)	
		Valid trials	Invalid trials	Valid trials	Invalid trials
P1					
RVF stimulation	Latencies (ms)	116	120	132	138
	Amplitudes (μV)	1.78	0.80	2.23	1.36
LVF stimulation	Latencies (ms)	136	138	128	130
	Amplitudes (μV)	2.27	1.86	2.12	1.83
N1					
RVF stimulation	Latencies (ms)	170	184	182	198
	Amplitudes (μV)	−4.26	−3.06	−3.15	−2.58
LVF stimulation	Latencies (ms)	184	202	176	184
	Amplitudes (μV)	−3.16	−2.42	−4.69	−3.37

tude of the component was significantly enhanced for invalidly cued targets ($F(1,13) = 9.489$, $p = 0.0088$). There was also a significant main effect of electrode ($F(1,13) = 36.603$, $p = 0.0001$), due to a reduced P3 amplitude at POz compared to Pz and CPz ($p = 0.0001$). These differences were also reflected by the significant interaction between cue validity and electrodes ($F(2,26) = 17.422$ $p = 0.0001$), although *post-hoc* tests revealed that invalid trials led to an enhanced P3 amplitude for every single central electrode (every electrode: $p < 0.02$).

DISCUSSION

Confirming previous behavioural observations [8,9,11], targets appearing at a gazed-at location were detected faster than invalidly cued stimuli, despite the fact that subjects were fully aware of the non-predictive gaze direction-cue and kept central fixation (Fig. 2). These behavioural results confirm the reflexive, automatic nature of eye gaze as a central attentional cue [8,13]. As a novel finding, our ERP results clearly demonstrate that these facilitations of visual processing by spatial attention are reflected by enhanced early visual evoked potentials (P1 and N1), much in the same way as previously demonstrated by non-reflexive spatial attention studies with ERPs [2,14]. These results thus confirm the early sensory nature of attentional mechanisms in spatial attention tasks [4] and extend it to reflexive spatial attention triggered by a socially relevant cue, namely eye gaze direction. In addition to an increase of amplitude, both the P1 and N1 components peaked earlier in the congruent condition (Fig. 2, Fig. 3), showing that reflexive attention not only increases visual activity but speeds up the processing of visual information at least as soon as it reaches the extrastriate visual cortex, although the latency modulation was rather small on the P1 component (Fig. 2). A last observation was the enhanced P3 for invalid trials, an effect reported earlier [2] and interpreted as an increase with decreasing target probability, providing additional evidence that the eye gaze cue was effectively manipulating the subject's expectancy for target location.

To our knowledge, the increase of P1 amplitude had not been described previously for reflexive attention, which was always tested with peripheral cues [4]. Our results thus reinforce the idea that the lack of early attentional modulation (P1) with the peripheral cues [15] may be related to some form of sensory–sensory interaction (when the cue and the target are presented at the same location) rather than to the reflexive nature of the spatial attention task *per se* [4].

Based on scalp topographies, peak latency and source analyses, as well as co-registration with PET and fMRI recordings, it has been suggested that the lateral extrastriate P1 component do not originate from the primary visual cortex but rather in the posterior fusiform gyrus and ventral-lateral extrastriate cortex of the middle occipital gyrus [3,16–18]. The results described here thus suggest that reflexive attention triggered by eye gaze direction does not modulate the primary visual cortex, although our experiment did not manipulate the upper/lower placement of the evoking target in the visual field [3], which allows us to identify more clearly the C1 component evoked by the primary visual cortex (onset at 50 ± 60 ms, peak at 80 ± 90 ms). The amplitude increases of the P1 and N1 components have traditionally been related to a mechanism of gain control or selective amplification of sensory information flow in the visual pathways, giving inputs from attended locations an improved signal-to-noise ratio so that more information can be extracted from relevant portions of the visual field [5]. This gain multiplication view is supported by several studies of single-cell recordings in the monkey brain [19].

In sum, the present study shows that when there is a shift of eye gaze toward a given location, there is an automatic expectation which leads to an amplification of early visual activity to facilitate perceptual processing of forthcoming stimuli at the location indicated by the gaze direction.

In addition to an amplitude increase of P1 and N1, reflexive attention triggered by eye gaze also caused earlier peak latency of the P1 and N1 responses (Fig. 2, Fig. 3). To our knowledge, such latency effects have never been described in the spatial attention literature [5]. How could these effects be explained in the present study? First, they could reflect an artefact of subjects moving their eyes in the direction indicated by the eye gaze cue. If subjects were to make either fast saccades or slower drifts toward the cued visual field in these tasks, stimuli presented in the valid

visual field would fall closer to the fovea, whereas stimuli presented to the opposite (invalid) visual field would fall farther from the fovea. These differences might well result in ERP latency changes thus erroneously attributed to the effect of selective attention. However, careful inspection of the individual and grand-averaged horizontal EOG channel does not support this possibility (Fig. 2). Moreover, the latency effect has been found to be larger on the N1 component than the P1 component, so that eye movements would have to take place especially in between these two components, very late after the cue onset (500 ms before the target onset), which is highly implausible. A possible explanation would be a higher rate of accumulation of activity in neuronal populations processing the targets at cued location, leading to an earlier response at the level of the population potential. Such a mechanism has been proposed to account for the variation in speed recognition of objects presented in different views [20]. An important factor that may be related to these differences of type of attentional effect is that the sources of spatial attention triggered by eye gaze are likely to differ, at least partly, from those involved in sustained attention from instructions or symbolic cueing. In the last case, a network of prefrontal, parietal and temporal areas [21] is most probably involved. However, the attentional effects to eye gaze such as those reported in the present study are more likely to originate from regions of the STS, where cells sensitive to eye gaze have been recorded in the monkey brain [22] or the intraparietal sulcus, which is highly connected to the STS and also involved in eye gaze detection [23] and selective attention [24]. Lesion studies in monkey and human brains [25], as well as neuroimaging evidence [26] also points to the STS as a locus of gaze direction analysis. The clear lateral parieto-occipital topography of the P1 and N1 attentional effects observed in the present study (Fig. 3) also reinforces the idea that the STS and intraparietal sulcus may play a role in these effects. Direct comparisons of reflexive attention triggered by eye gaze direction and non-reflexive symbolic attention in future studies will help clarifying the similarities and differences between these two types of attentional modulations of extrastriate activity. More generally, the effects observed here are also probably due to the specific nature of the cues we used. Eye gaze detection is a very powerful cue and its role in human and animal behaviour is known to be fundamental for social interaction [27]. The ability to follow eye gaze is present for non-human primates [28] and appears very early in human development; children aged between 14 and 18 months can follow eye gaze without any head cues [29]. These observations and other evidence [27] have led several authors to propose that gaze processing is hard-wired in the brain in circuits including the STS. The present study indicates that these regions may strongly influence spatial attention networks and early visual processing of information.

In conclusion, this study has shown that the automatic processing of eye gaze direction, and consequently the shift of the viewer's attention, enhances and speeds up sensory processing at the location indicated by the viewer. This result indicates that visual information from gaze direction is processed in areas interacting with spatial attention networks modulating early visual processing. The increase and acceleration of visual information processing at a location indicated by another person's gaze reflects the social and biological value of eye gaze direction in humans.

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