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Clinical Neurophysiology 116 (2005) 2565–2576

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Spatial attention triggered by eye gaze enhances and speeds up visual processing in upper and lower visual fields beyond early striate visual processing

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Accepted 29 July 2005

Abstract

Objective: The detection of a lateralized visual target is faster when preceded by a face gazing to the location of this stimulus. Here we aimed to clarify the time-course of the visual processing modulated by these reflexive shifts of attention.

Methods: ERPs were measured on 16 subjects performing a speeded location task on a circular checkerboard. The checkerboard target appeared either on the left or right of the upper or lower visual field, and was preceded by a central face orienting its gaze obliquely to one of the four possible corner locations for the target to appear.

Results: Congruently cued targets were located faster than incongruently cued targets and were associated with larger and earlier occipital P1 (~110 ms) and occipito-parieto-temporal N1 (~150 ms) components. However, no such attentional modulations were found on the earlier C1 visual component, best observed with a negative polarity for upper visual field stimulations, and thought to originate largely from primary visual cortex.

Conclusions and significance: These results show that reflexive shifts of attention following oblique eye gaze to upper and lower visual fields increase and speed up the processing of visual information beyond the feedforward flow of information in primary visual cortex.

Keywords: Eye gaze; spatial attention; primary visual cortex; C1

1. Introduction

The direction of others' gaze is a crucial signal in processing social information and the eye region is often used to predict the intentions and goals of others (Baron-Cohen, 1995; Kleinke, 1986). The importance of eye gaze in both human and non-human primates is well documented by behavioral, neurophysiological and neuroimaging studies (for reviews see Emery, 2000; Langton et al., 2000). The power of the eyes in signaling the direction of another person's attention is illustrated by a series of recent studies demonstrating that gaze cues are able to trigger an automatic shift of the focus of the viewers' visual attention (Driver et al., 1999; Farroni et al., 2003; Friesen and Kingstone, 1998; Friesen et al., 2004; Kingstone et al., 2000; Hietanen, 1999; Hietanen and Leppanen, 2003; Hood et al., 1998; Langton and Bruce, 1999). In these studies, a gazing face stimulus (photograph or schematic) is presented to the viewer in the center of the visual field, followed by a target presented randomly at the gazed-at location or another location. Subjects are faster to detect, localize and identify the gazed-at target, providing evidence that their attention is automatically displaced following the direction of the eye cue. This orienting is considered to be reflexive because it is fast, emerging at about 100 ms after the eyes are presented (e.g. Friesen and Kingstone, 1998), and it occurs even though subjects know that the direction of the eye gaze is not predictive of the location of the forthcoming target (in all studies cited above). Furthermore, this orienting effect is so strong that it even persists when

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1388-2457/$30.00
doi:10.1016/j.clinph.2005.07.021

the cue prediction probability is reduced to 25%, i.e. is counterpredictive (Driver et al., 1999; Friesen et al., 2004; Langton and Bruce, 1999; Vuilleumier, 2002). Eye gaze direction seems therefore to be processed obligatory and shifts the viewer's attention automatically towards the cued region.

Recent electrophysiological studies have investigated the time course of the spatial attention triggered by gaze perception (Schuller and Rossion, 2001; 2004). In line with behavioral work, these studies used a spatial attention paradigm in which subjects had to detect a lateralized target following the presentation of central face gazing to the left or right of the display. It was found that the parieto-occipital components P1 (~110 ms) and N1 (~150 ms) were speeded up and amplified when the target appeared at a gazed-at location compared to an incongruent (or neutral) condition, whether the eye cues were dynamic (eye-gaze motion, Schuller and Rossion, 2001) or static (Schuller and Rossion, 2004). The amplitude enhancement of the P1 and N1 was similar to the typical ERP findings on spatial attention (see Luck et al., 2000), which is thought to exert a selective gain control or amplification on the sensory visual processing flow (Hillyard et al., 1998). However, ERP latency differences between congruent and incongruent targets are usually not disclosed in studies of sustained spatial attention, or trial-by-trial cueing using predictive cues (e.g. Mangun and Hillyard, 1991; Luck et al., 2000) and may therefore be specific to spatial attention triggered by central reflexive cues such as eye-gaze direction

To support this view, several sources point to specialized cerebral mechanisms being involved in the perception of eye gaze. First, single-cell recording studies in monkeys have shown that in the superior temporal sulcus (STS) there are neurons specifically sensitive to the orientation of the eyes (Perrett et al., 1985), which may be involved in the recognition of the location where another individual is looking (Perrett et al., 1992). In humans, neuroimaging studies show that the posterior STS region is implicated in gaze processing (Hoffman and Haxby, 2000; Hooker et al., 2003; Pelphreys et al., 2003; Puce et al., 1998; for a review see Allison et al., 2000). The intraparietal sulcus (IPS), which participates in covert shifts of attention (e.g. Corbetta, 1998; Nobre et al., 1997), is also significantly activated during averted eye gaze perception (George et al., 2001; Hoffman and Haxby, 2000; Hooker et al., 2003; Pelphreys et al., 2003; Puce et al., 1998; Wicker et al., 1998). Electrophysiological studies in humans corroborate the 'special' nature of the eyes in several ways. First, it has been shown that isolated human eyes evoke particularly large and early visual responses compared to whole face stimuli or other isolated facial features (e.g. Bentin et al., 1996; Taylor et al., 2001). Second, the occipito-temporal N170 (see e.g. Bentin et al., 1996; Rossion et al., 2000) evoked by isolated eyes is present earlier in development than the same component elicited by whole face stimuli, suggesting a faster maturation of the eye processing system compared to general face processes (Taylor et al., 2001; for evidence of early processing of gaze in infancy see also Farroni et al., 2003; 2004). Finally, recent evidence using response classification methods in adults suggest that the eyes of a face evoke the earliest and largest face-sensitive ERP responses (Schyns et al., 2003; but see Eimer, 1998).

In the present study, considering this evidence supporting the 'special' nature of the eyes, as well as the power of eye gaze as a socially relevant cue to orient spatial attention, we investigated whether the peculiar effects of spatial attention triggered by eye gaze—amplitude increase and speeding up of information processing—may take place as early as the afferent volley in the primary visual cortex. The earliest visual ERP response is reflected on the scalp surface by a relatively small component referred to as the C1 or N70[1], starting in humans around 50 ms following stimulus onset, and best observed on the midline at occipito-parietal sites (Aine et al., 1996; Bodis-Wollner et al., 1981; Clark et al., 1995; Foxe and Simpson, 2002; Jeffreys and Axford, 1972a; Martinez et al., 1999; Moradi et al., 2003; Tzelepi et al., 2001). A particular feature of the C1 component is its opposite polarity according to the location of the visual stimulation in the upper (negative C1 polarity) or the lower (positive C1 polarity) visual field (Jeffreys and Axford, 1972a; Clark et al., 1995). This polarity reversal appears to be due to the activation of the upper versus the lower banks of the calcarine sulcus for lower and upper visual field stimulation respectively (e.g. Aine et al., 1996; Jeffreys and Axford, 1972a), and is thus a critical marker of V1 activation (Di Russo et al., 2003). Consequently, the sources of the C1 component are usually found in striate cortex (e.g. Clark et al., 1995; Di Russo et al., 2002; Martinez et al., 1999; Moradi et al., 2003) although extrastriate areas such as V2 and V5 may also contribute to the late part of the C1 component (Moradi et al., 2003; Foxe and Simpson, 2002; Tzelepi et al., 2001).

Together with the amplitude enhancement of the visual P1 and N1 (see Hillyard and Anllo-Vento, 1998; Luck et al., 2000), a consistent finding of visual attention studies has been that the C1 component is not modulated by spatial attention (e.g. Clark and Hillyard, 1996; Martinez et al., 1999) although primary visual cortex appears to be modulated later in time probably through feedback inputs from higher cortical areas (e.g. Martinez et al., 1999; Noesselt et al., 2002). However, the previous studies testing the influence of spatial attention on visual information processing at the level of the primary visual cortex (C1) all used sustained attention paradigms, or symbolic cues (e.g. an arrow) which were predictive of the forthcoming location of the target (see Hillyard and Anllo-Vento, 1998; Di Russo et al., 2002). To our knowledge, the question of whether the early volley to the primary visual cortex, as reflected largely

[1] Or the M70 in studies using magnetoencephalography (MEG; e.g. Tzelepi et al., 2001; Moradi et al., 2003).

by the C1 component, can be modulated following reflexive shifts of attention to central biological cues such as eye gaze has not been addressed previously. This question is of theoretical interest given that spatial attention shifts following eye gaze cueing appears to be qualitatively different from other forms of spatial attention (e.g. Friesen and Kingstone, 1998; Friesen and Kingstone, 2003a; Friesen et al., 2004). As noted above, the facilitatory effect of gaze direction is both rapid and occurs in response to nonpredictive cues, suggesting a reflexive attentional orienting rather than an endogenous or volitional orienting. However, orientation to gaze direction does not exhibit all of the characteristics associated with reflexive shifts of attention using nonbiological cues: (1) the cue is presented centrally, not in a peripheral location; (2) the orienting to gaze direction persists well beyond a cue-target SOA of 500 ms whereas the reflexive orienting effect produced by nonpredictive peripheral cues disappears at SOAs above 300 ms (Klein et al., 1992); and (3) there is no inhibition of return effect (Klein, 2000) at long SOAs following eye gaze cues. Considered together, these differences suggest that attention to gaze direction might represent a different type of reflexive orienting (Friesen and Kingstone, 1998).

Furthermore, there is evidence from several studies (Kingstone et al., 2000; Friesen and Kingstone, 2003a; 2003b; Ristic et al., 2002; Hood et al., 1998; Vuilleumier, 2002) that the neural circuits subtending the attentional effects following eye gaze cues are different than the superior colliculus (SC)-parietal pathway involved in the reflexive orienting to a peripheral location (Rafal et al., 1991), and also different than the endogenous orienting to an expected target location (following a predictive cue), thought to involve a network of prefrontal and parietal areas (Corbetta et al., 1993; Posner, 1995). Rather, the gaze-triggered reflexive attention pathway, which seems to be lateralized to the dominant hemisphere for face and gaze processing (Kingstone et al., 2000) may be subserved by a temporo-parietal pathway, with cells in inferotemporal cortex processing face and eye information, cells in the superior temporal sulcus processing the direction of gaze, and cells in the parietal cortex shifting attention to the gazed-at-location (Kingstone et al., 2000; Friesen et al., 2004).

Here we aimed to test the hypothesis that the C1 would be modulated by spatial attention triggered by eye gaze. In previous ERP studies using eye-gaze cueing, the C1 component in response to targets of attention could not be measured adequately because these stimuli were presented on the horizontal meridian, canceling out or at least strongly reducing the C1 peak and making difficult to distinguish the C1 from the subsequent P1 (Clark et al., 1995). Accordingly, in the present study, in order to be able to measure the C1 accurately, we presented the target stimuli—a circular checkerboard—in the four quadrants of the visual field (Fig. 1). This target stimulus appearing in one out of the four corners of the visual display was preceded by a face gazing to either its location (congruent trial), or the diametrically opposite location (incongruent trial; see Fig. 1). Thus, another additional feature as compared to previous behavioral and neurophysiological work was the use of oblique eye gaze cues, directed to the upper or lower visual fields.

2. Methods

2.1. Participants

Twenty-one volunteers were paid for taking part in the experiment. All of them had normal or corrected to normal vision. Five subjects had to be excluded because of too many ocular movements and/or movement artefacts contaminating EEG. Thus, data from 16 subjects (6 females, 1 left-handed, aged 18–34, mean age 24 years) are reported.

2.2. Stimuli and procedure

Subjects were seated in a comfortable chair in a dimly lit, electrically shielded room, at a distance of 82.5 cm from a monitor screen, their head restrained by a chin rest. Stimuli included one picture of a full front female face with the eyes fixating the viewer, and four pictures of the same face with

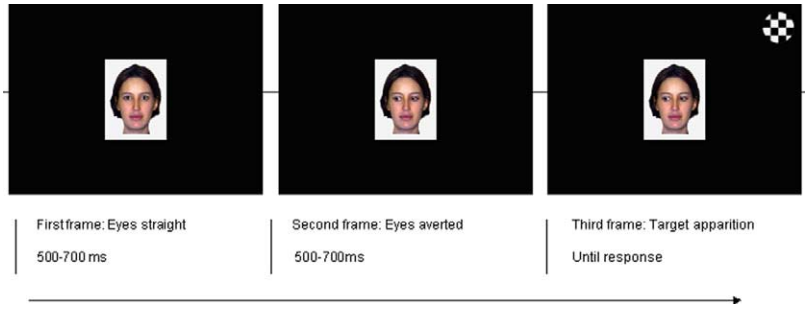

Fig. 1. Stimulation sequence, beginning with a straight gazing face, then glancing obliquely to one of four possible locations (to the left/right upper or lower corner), and followed by an annulus checkerboard target appearing either in the corner where the face is looking, or in the opposite corner. After the subject's response ('left or right'), a blank screen was presented for 800 ms. Here an incongruent trial, with the target presented in the upper RVF.

eye gaze averted 45° to the upper left visual field (LVF) or right visual field (RVF), or downwards to the LVF or the RVF, hence glancing to one of the 4 corners of the screen (see Fig. 1). All face photographs were surrounded by a white box measuring 7.5 cm vertically and 5.5 cm horizontally. The face picture sized 3.8° on 2° of visual angle. The face inside the white box was presented on a black background in the centre of the screen (Fig. 1). Targets consisted of small circular checkerboards of 2° diameter of visual angle and a check size of 35 min. They were presented at four locations in the visual field, centered along an arc that was equidistant (8.2° visual angle) from the centre of the face stimulus, at a position located at 45, 135, 225 or 315° arcs of polar angle (Fig. 1). Thus the four target locations were: in the upper or lower LVF, or in the upper or lower RVF. The target eccentricity of 8.2° was chosen in order to activate regions of striate cortex close to the average midpoint of its anterior–posterior length (Tollhurst and Ling, 1988). Target size and eccentricity corresponded to the parameters used by Clark et al. (1995).

A trial began with the presentation of a straight gazing face, lasting between 500 and 700 ms and followed by one of the 4 averted eye gaze pictures appearing also for a random time period (500–700 ms). After this delay, a lateralized target appeared, either at the position where the face was looking at (congruent trials) or in the diametrically opposite location (incongruent trials) (Fig. 1). The central face and the target remained on the screen until the subject's response. The next trial was presented after an 800 ms delay. Given the continuous stimulus presentation, the whole sequence, as illustrated in Fig. 1, was perceived as a face moving the eyes obliquely downwards or upwards to the left or right side of the screen. In total, eight combinations were used: the target location was either congruent with the eye gaze direction (Congruent-LVF-up/Congruent-RVF-up/Congruent-LVF-down/Congruent-RVF-down), or opposite to the gaze direction (Incongruent-LVF-up/Incongruent-RVF-up/Incongruent-LVF-down/Incongruent-RVF-down). Thus, for each possible location of the target, there was only one valid and one invalid (diametrically opposite) location used regarding the direction indicated by the eye-gaze cue. In other words, the eye-gaze cue was valid in half of the trials.

Throughout the experiment, subjects were instructed to maintain fixation to the central face. They were also reminded that the eye gaze was not predictive of the forthcoming location of the target stimuli. Twenty trials were run before starting the experiment to familiarize the subjects with the task. They were required to press the left button of the response box when the target was shown in the LVF and the right button when the target was presented in the RVF, independent of the upper or lower position of the target in the respective visual field. Responses were given with the index finger of the left and right hand in respect to the target location, and subjects were asked to be as accurate and as fast as possible. Five blocks of 144 trials (order randomized) were run, giving 85 trials/condition and 40 catch trials. The catch trials, with no target following the cue, were added to sustain the attention of the subjects and to prevent anticipations.

2.3. 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 electrode system. During the recording of EEG, the electrodes were referenced to linked-earlobe electrodes. An additional electrode was placed on the tip of the nose and used for offline-re-referencing. 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. EEG was amplified with a gain of 30 K and bandpass filtered between 0.01–70 Hz. A notch filter of 50 Hz was used during acquisition. Electrode impedance was kept below 5 kOhms. EEG and EOG were digitized at a sampling rate of 500 Hz.

Off-line, the EEG was filtered between 2 and 20 Hz. The high-pass cut-off (2 Hz) was used to get rid of drifts and slow waves (e.g. CNV) related to subject's anticipation (see e.g. Vogel and Luck, 2000) and the low-pass cut-off (20 Hz) to smooth the waves and facilitate automatic peak detection on ERP waveforms. Then EEG and EOG artefacts were removed using a $[-40;+40\ \mu V]$ deviation over 200 ms intervals on all electrodes. In case of too many blink artefacts (in 5 subjects out of the16) they were corrected by a subtraction of VEOG propagation factors, based on PCA-transformed EOG components (Nowagk and Pfeifer, 1996). Then, averages were generated for each subject and each of the 8 conditions in epochs of -200 to 800 ms, time-locked to the onset of the target. Averages were then re-referenced offline to the nose electrode in order to maximize the amplitude of posterior (visual) components.

2.3.1. Data analysis

Following the identification of the C1 component by comparing upper and lower VF stimulations (Fig. 3), mean amplitude relative to a 200 ms pre-stimulus baseline was extracted automatically on each subject and condition data on two central electrodes PZ and POZ where the C1 was most prominent during the time period of 60–90 ms. The peak latency of the visual components P1 and N1 was extracted automatically on a single pair of electrode (P3/P4) where these components were largest on grand-average data and could be extracted accurately for all averages. Given the significant differences of peak latencies between the congruent and incongruent trials, and between the four locations of the targets and their subsequent differential processing of each hemisphere, amplitude values were extracted during a 20 ms window centered on the grand-average peak latencies for each attentional condition, for

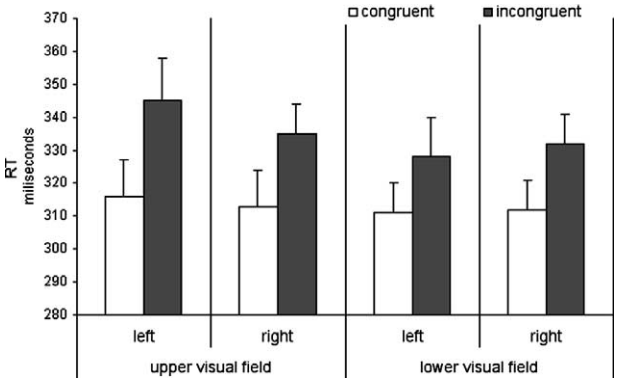

Fig. 2. Mean reaction times (RTs, in milliseconds) and standard errors (SE) for congruent (white bars) and incongruent (grey bars) trials according to the target location in the 4 quadrants of the visual field. RTs were significantly faster for congruent than for incongruent trials.

each visual quadrant and each hemisphere separately. After visual inspection of the components distribution on the scalp (Fig. 6), mean amplitude values were measured on 9 pairs of electrodes for the P1 (CP1/2; CP3/4; P1/2, P3/4; P5/6, P7/8; PO3/PO4, PO5/PO6; PO7/PO8). Mean amplitude for the N1 component was measured on 6 pairs of electrodes CP1/2, CP3/4; P1/2, P3/4; PO3/PO4, PO5/PO6. To take into account P1 differences in assessing the N1 effects, analyses were also computed on peak-to-peak differences between N1 and P1 amplitude and latency values. The P300 was measured in a time period of 260–360 ms on 3 central leads: CZ, CPZ, and PZ.

Repeated-measures analyses of variance (ANOVA) were performed on the reaction time (RT) and ERP values. The factors were: *Congruency* (congruent/ incongruent) and *Visual Quadrant* of stimulation (LVF up/ RVF up/ LVF down/ RVF down). The additional factors for ERP values were: *Hemisphere* of recording (LH, left hemisphere /RH, right hemisphere) and *Electrode site*. Planned comparisons were performed to evaluate the specific attentional modulations. Greenhouse–Geisser correction was used to correct p values when appropriate.

3. Results

3.1. Behavioral results

Reaction times were significantly faster for congruent than for incongruent trials ($F(1, 15) = 40.434$, $P < 0.0001$; Fig. 2) expressing the attentional orienting produced by eye gaze. The main effect of visual quadrant of stimulation and the interaction between congruency and visual quadrant of stimulation were not significant.

3.2. Event-related potentials

ERP waveforms for congruent and incongruent trials evoked by lateral targets are illustrated on Figs. 4 and 5. The topography of the components C1, P1, N1 and P300 obtained in this experiment are shown on Fig. 3 and will be described in detail below.

The C1 was clearly negative for upper visual field stimulation and had an average onset latency of about 50–65 ms and a peak latency of about 70 ms for upper visual field stimulations (Figs. 3 and 4). As illustrated on Fig. 5 for each of the four stimulated corners of the display, the C1 was most prominent over ipsilateral parietal sites for upper visual field stimulations, whereas for lower visual field stimulations, it was positive at contralateral centro-parietal sites.

The P1 component had an average onset latency of 90 ms and its early part was elicited at contralateral parietal sites, sprading to ipsilateral sites (Fig. 3, 2nd and 3rd column). For lower visual field stimulation, the early P1 was overlapping in time with the positive

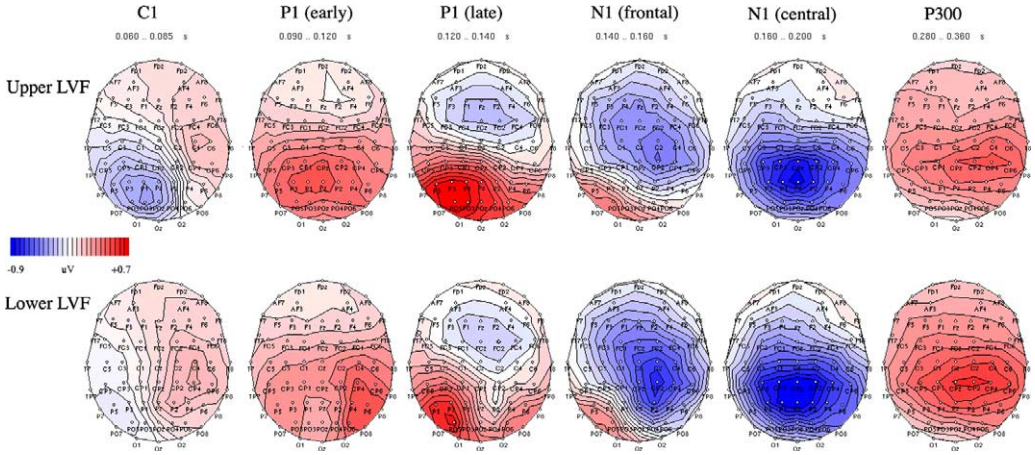

Fig. 3. Topographical maps of the sequence of ERP components evoked by the LVF target, for upper (top part of the figure) and lower (bottom part) visual field stimulations. Congruent and incongruent trials are averaged together here.

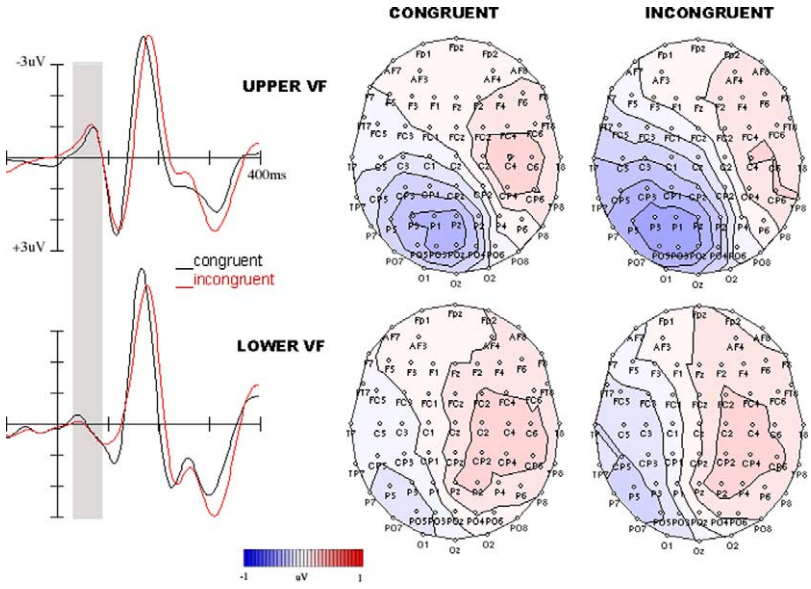

Fig. 4. Grand-averaged ERPs on PZ (left) and topographical maps for the C1 component, shown for congruent and incongruent conditions separately.

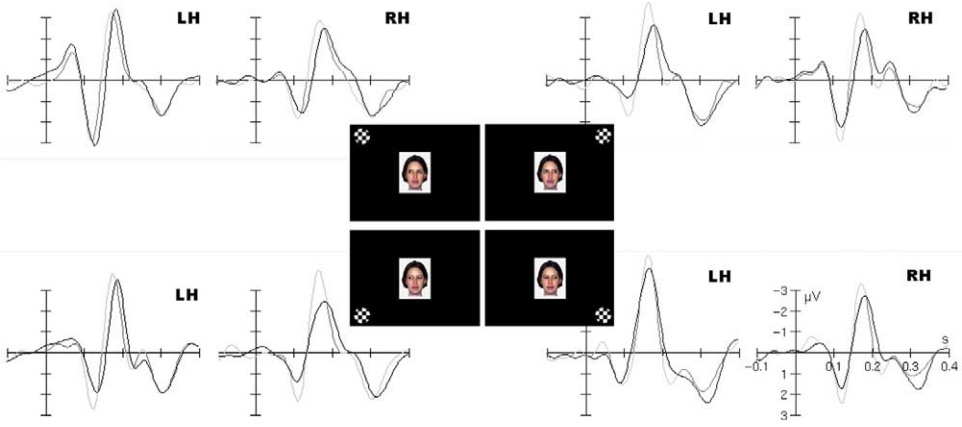

Fig. 5. Grand-averaged waveforms for the four visual field locations of the target stimuli. ERPs are represented for the left (P3) and the right (P4) hemisphere and show the congruent (grey) and incongruent (black) trials. Note the larger ipsilateral response for the C1 component (paradoxical lateralization). The P1 also appear larger on ipsilateral sites, but topographical maps indicate a contralateral onset (Fig. 6).

contralateral C1 (peak at −90 ms) (Fig. 5). The early phase of the P1 component was peaking around 114 ms for upper VF (visual field) stimuli. The most prominent phase of the P1 was located over the ipsilateral occipito-temporal sites and peaked slightly later, around 122 ms. The P1 did not change polarity and varied slightly in amplitude for upper versus lower VF stimulations (Fig. 5).

Following the P1, a broadly distributed negativity appears in the time range of 130–200 ms (Figs. 5 and 6).

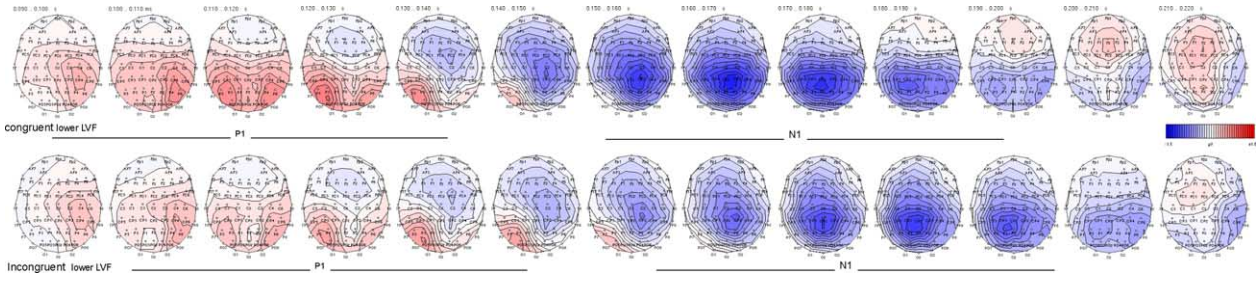

Fig. 6. Topographical maps representing the P1 and N1 attentional effects for lower LVF trials (congruent and incongruent).

The early phase of this N1 (130–160 ms) was frontally distributed, peaking at contralateral sites for both upper and lower VF stimuli around 150–158 ms (Fig. 3, 4th column). The later phase of the N1 (160–200 ms) was elicited at bilateral centro-parietal sites and peaked on average around 168–174 ms (Fig. 3, 5th column). The N1 components showed no differences to upper versus lower visual field stimulations (Figs. 3 and 5).

Finally, in the time range between 260–360 ms a largely distributed positivity, which can be identified as a P300 component, was most prominent at central sites and peaked around 310 ms (Fig. 3, 6th column).

4. Statistical analysis

4.1. C1

No significant *congruency* effects on the mean amplitude of the C1 were found ($F(1, 15)=0.66$, $P=0.4272$). There was a significant effect of the factor *visual quadrant* ($F(2.40, 36.10)=12.041$, $P<0.0001$), expressing the reversed polarity of the C1 according to the upper/lower VF stimulation, stimulations in the upper VF stimuli evoking a more negative C1 than lower VF targets ($P<0.0001$; see Fig. 4).

4.2. P1

The analysis of the P1 *latency* measured on electrode P3 and P4 showed a significant main effect of *congruency* ($F(1, 15)=10.543$, $P<0.005$), and an interaction of *congruency* with *visual quadrant* ($F(2.17, 32.63)=6.632$, $P<0.003$) (see Fig. 5). These effects reflect a P1 peaking earlier for congruent compared to incongruent trials for both upper and lower LVF stimulations (both P's <0.002), but only a trend for this latency difference for upper RVF stimulations ($P=0.078$) and no effect for lower RVF targets ($P=0.11$) (see Fig. 5). Besides the attentional effects, the analysis showed a significant main effect of *visual quadrant* ($F(2.68, 40.25)=3.703$, $P<0.05$) and a significant two-ways interaction between *visual quadrant* and *hemisphere* ($F(2.37, 35.64)=14.083$, $P<0.0001$). This last interaction reflects a P1 peaking earlier for contralateral targets compared to ipsilateral stimulations (early contralateral P1 phase), particularly in the left hemisphere (effect of visual quadrant: LH: $P<0.0001$, RH $P<0.02$).

P1 mean *amplitude* was significantly increased for congruent trials ($F(1, 15)=7.695$, $P<0.01$). The ANOVA yielded a marginally significant two-way interaction between *congruency* and *visual quadrant* ($F(2.07, 30.01)=3.174$, $P=0.054$), indicating a significant amplitude difference between congruent and incongruent conditions in all (all P's <0.05) but the upper LVF ($P=0.39$). The analysis of P1 amplitude further showed a significant effect of *electrode site*, because of larger ERPs recorded on lateral posterior parietal electrodes ($F(2.6, 38.94)=11,34$, $P<0.0001$). Furthermore, there was a significant interaction between *electrode site* and *visual quadrant* ($F(4.46, 66.87)=4.863$, $P<0.001$), *hemisphere* and *visual quadrant* ($F(2.24, 33.6)=3.83$, $P<0.03$) as well as a three-way interaction between the factors *electrode site*, *hemisphere* and *visual quadrant* ($F(4.36, 65.41)=2.49$, $P<0.05$), reflecting minor differences between the amplitude values for each VF stimulation recorded on the different pairs of electrodes.

4.3. N1

The N1 *latency* yielded a significant main effect of *congruency* ($F(1, 15)=21.923$, $P<0.0003$), due to the N1 peaking earlier for congruent trials than for incongruent trials (see Figs. 5 and 6). No other effects were found on the N1 latency. The same *congruency* effect ($F(1, 15)=5.995$, $P<0.027$) was found on the N1 when P1 effects were taken into account (peak-to-peak analysis, see methods).

There was no significant effect of *congruency* on the N1 mean *amplitude* ($F(1,15)=1.421$, $P=0.25$). However, a significant interaction of *congruency* with *electrode site* ($F(2.04,30.53)=3.85$, $P<0.03$) indicates a larger N1 response for congruent than for incongruent trials for all electrode sites, however slightly stronger for lateral electrodes (P's <0.005) than for the more central electrode pairs CP1/CP2 and P1/P2 (P's <0.03). The ANOVA also yielded a three-way interaction between *congruency, hemisphere,* and *visual quadrant* ($F(2.09, 31.32)=5.508$, $P<0.008$). The planned comparisons of this three-way interaction showed enhanced N1 amplitude for congruent trials compared to incongruent trials over both hemispheres in all visual quadrants (P's <0.05), except for upper LVF stimulations (LH: $P=0.15$, RH: $P=0.42$) and for lower LVF stimulations over the LH ($P=0.25$). The analysis of N1 amplitude showed a significant effect of *electrode site,* due to larger ERPs recorded on lateral posterior parietal electrodes ($F(1.64,24.53)=8.065$, $P<0.003$). The factor *hemisphere* yielded a significant main effect, because of a larger N1 amplitude in the left hemisphere ($F(1,15)=9.575$, $P<0.007$). There was a significant interaction between the two factors *hemisphere and visual quadrant* ($F(2.27, 34.11)=4.407$, $P<0.02$). *Electrode site* was also significantly modulated by *visual quadrant* ($F(4.94,74.03)=2.392$, $P<0.05$), reflecting minor differences between the amplitude values for each VF stimulation recorded on the 6 pairs of electrodes.

The ANOVA on the peak-to-peak amplitude of N1–P1 showed a significant main effect of *congruency* ($F(1, 15)=4.912$, $P<0.05$), reflecting the larger N1 amplitude for congruent trials. The interaction of *congruency* with *visual quadrant* was significant ($F(2.86, 42.84)=2.865$, $P=0.05$). Planned comparisons showed that the N1 amplitude difference between congruent and incongruent trials was statistically significant for upper RVF ($P<0.01$) and lower

LVF stimulations ($P<0.03$), a non-significant trend for lower RVF stimulations ($P=0.06$) and no difference for upper LVF stimulations ($P=0.55$) (see Fig. 3). A further significant effect of this analysis was the interaction between *congruency * visual field * hemisphere* ($F(2.84, 42.57)=3.005$, $P<0.04$), showing that the N1 amplitude was larger for congruent trials over both hemispheres and for all visual field stimulations (P's <0.03) except for upper LVF stimuli over the RH ($P=0.58$). Furthermore, *congruency* interacted significantly with *electrode site* ($F(2.45, 36.68)=3.306$, $P<0.04$). Planned comparison show larger N1 for congruent trials on all electrode pairs (P's <0.0001). The ANOVA also yielded a significant main effect on factor *electrode site* ($F(1.71, 25.65)=4.459$, $P<0.03$). A two-ways interaction between *electrode site* and *visual quadrant* was also significant ($F(4.19, 62.78)=2.564$, $P<0.04$).

4.4. P3

The P3 peaked earlier for congruent compared to incongruent trials ($F(1, 15)=8.602$, $P<0.01$). P3 *amplitude* analysis revealed a main effect of *congruency* ($F(1, 15)=11.959$, $P<0.005$), reflecting an increased P3 amplitude for incongruent trials. Further significant main factors of the amplitude analysis were *electrode site* ($F(1.254, 18.81)=12.176$, $P<0.001$), and *visual quadrant* ($F(2.631, 39.465)=6{,}866$, $P<0.001$). These effects reflect a larger P3 over CPz compared to Cz and Pz ($P<0.001$), and an enhanced P3 for lower VF compared to upper VF stimulations ($P<0.001$).

In summary, there was no attentional modulation of the C1 component, whereas P1, N1 and P3 amplitude and latency were significantly modulated by spatial attention driven by eye gaze cues. The P1 and N1 peaked earlier for congruent compared to incongruent trials and spatial attention enhanced the P1 and the N1 amplitude in most visual quadrants.

5. Discussion

The goal of this study was to test the hypothesis that eye gaze direction, a particularly powerful spatial attentional cue presenting specific properties (Friesen and Kingstone, 1998; Friesen et al., 2004) would modulate the processing of visual information as early as the primary visual cortex. To test the hypothesis that this early volley of inputs in the primary visual cortex—as represented by the C1 here—was modulated by spatial attention, we modified our previous paradigm (Schuller and Rossion, 2001; 2004) in two ways. First, the target stimuli were presented in the upper and lower visual fields instead of being presented on the horizontal meridian. Previous studies suggest that spatial attention enhances visual processing in a retinotopical way, i.e. in dorsal visual areas for lower VF stimuli and ventral areas for upper VF stimuli (Woldorff et al., 1997). Here attention was explicitly attracted towards either upper or lower visual corners, and the target location was either fully congruent or completely incongruent (opposite corner). During social interactions, attention to upper and lower visual field may carry different meanings than eye gaze cues directed laterally, pointing to objects to grab (lower visual field) rather than to people for social interaction for instance. Yet, strong effects of attention are found with these stimuli also, supporting the robustness of such cues for directing attention. Second, we used *oblique* eye gaze cues directed towards the upper or lower corners, instead of horizontal eye gaze cues. This decision was made to maximize the effects of attention at the cued location, rather than using a diffuse attention spread to the left or right visual fields.

In these conditions of oblique eye gaze cues towards upper/lower corners of the visual field, subjects were faster at detecting targets presented in locations of the visual field cued by a face gazing to this location, even though the direction of gaze was not predictive (50% validity) of the location of the forthcoming target. To our knowledge, only one previous study showed such effects, using oblique gaze cues in a schematic face (experiment 4, Vuilleumier, 2002). Similarly to this work, subjects' RTs were shorter for congruent/attended stimuli in all of the four quadrants of the visual field in the present study (Fig. 2). The behavioral effects with oblique gaze cues found here are roughly of the same magnitude as was observed with horizontal averted gaze cues with the same stimuli (Schuller and Rossion, 2001, 2004). Thus. oblique eye-gaze cues causes powerful spatial attention shifts to upper and lower part of the visual field, in line with several sources of evidence with lateral cues (Driver et al., 1999; Friesen and Kingstone, 1998; Friesen et al., 2004; Kingstone et al., 2000; Hietanen, 1999; Hietanen and Leppanen, 2003; Hood et al., 1998; Langton and Bruce, 1999).

Our previous work has shown that horizontal eye gaze cues from face photographs causes amplitude increases and speeding up of information processing at the level of the P1 and N1 components evoked by lateralized targets (Schuller and Rossion, 2001; 2004). These effects are also found here in a new paradigm, both at the level of the amplitude and latency of these components, reinforcing these previous findings. However, there was no modulation of the preceding C1 component, neither in latency nor in amplitude. Although this absence of modulation at the level of the C1 may be considered as a null effect, the large significant differences found at the level of the P1, N1 and P3 suggest that the absence of C1 modulation was not due to a lack of power: Our data support the view that the modulations of visual processes following reflexive attention triggered by social cues such as eye gaze start *after* the initial volley to the primary visual cortex.

The C1 observed in the present study had an early latency onset (50–65 ms) and a focal central distribution (Fig. 4). The short onset latency, scalp topography and polarity reversal fit with the characteristics defining the C1 in

previous studies (e.g. Clark et al., 1995; Jeffreys and Axford, 1972a; Di Russo et al., 2002). Its topographical distribution was slightly contralateral for the lower VF stimulations and slightly ipsilateral for the upper VF stimuli (Fig. 3). The positive C1, induced by lower VF stimulation, was clearly visible only in the contralateral hemisphere (Fig. 5). These responses on the scalp have been described previously and are thought to reflect the retinotopic properties of the primary visual cortex and its distribution around the calcarine fissure ('the modified crucifix model', e.g. Aine et al., 1996; Clark et al., 1995; Jeffreys and Axford, 1972a). The localization of the C1 in the primary visual cortex has been confirmed by several studies using low posterior electrodes, and locating the dipole sources around the calcarine sulcus (Clark et al., 1995; Clark and Hillyard, 1996; Di Russo et al., 2002; 2003; Martinez et al., 1999; 2001; Noesselt et al., 2002). These studies usually found a single cortical source in the primary visual cortex, accounting for most of the C1 variance, varying systematically in orientation around the calcarine fissure as a function of stimulus elevation (e.g. Clark et al., 1995). Although it has recently been shown that the late parts of the C1 component may also reflect the activation of extrastriate visual areas (Moradi et al., 2003; Foxe and Simpson, 2002; Tzelepi et al., 2001), a large part of the C1 clearly originates from the earliest activation of the primary visual cortex, (Di Russo et al., 2002; 2003).

The absence of modulation of the C1 by spatial attention triggered by eye gaze is in agreement with previous spatial attention studies with non-gaze cues or sustained attention (Clark and Hillyard, 1996; Di Russo et al., 2003; Martinez et al., 1999; 2001; Noesselt et al., 2002). This indicates that despite the high saliency and power of the eyes as attentional cues the increases and speeding up of visual processing by reflexive attention to such central cues start beyond the first feedfoward processing flow in V1.

In contrast with the absence of C1 modulation, large effects of attention were observed at the occipito-parietal components P1 (peaking at ~110 ms) and N1 (~150 ms), which were enhanced, and peaked earlier, when elicited by a target checkerboard preceded by a congruent eye gaze cue. Peak-to-peak analyses for both amplitude and latency measurements further indicate that the N1 effects were not due to effects observed at the preceding P1 with an overlapping topography over posterior sites, but on the contrary that spatial attention goes on further modulating perceptual processes taking place after the extrastriate processing reflected by the P1.

P1 and N1 amplitude enhancements have been found in numerous spatial attention studies, using endogenous or exogenous (peripheral) cues, such as instructions to attend a particular location (or central cues predicting the forthcoming stimulus location), or peripheral cues respectively (for reviews see Mangun and Hillyard, 1995; Luck et al., 2000). The sources of the attentional enhancements (P1 and N1 effects) and of the original components in the extrastriate visual cortex are generally similar (Di Russo et al., 2003; see below), suggesting an increase of activation due to attention rather than the recruitment of additional regions (see, e.g. Martinez et al., 2001 for fMRI evidence). These findings have been taken as evidence that sensory gain control mechanisms in higher-order areas amplify neural activity in extrastriate cortex, facilitating the processing of stimuli (Hillyard et al., 1998).

The sources of the P1 component have been located in ventral-lateral cortex/posterior fusiform gyrus for stimuli presented in upper visual field (Gomez Gonzales et al., 1994; Heinze et al., 1994; Mangun et al., 2001), and in dorsal extrastriate cortex when the P1 was evoked by lower visual field stimulation (Woldorff et al., 1997). Recent evidence has supported this dorsal/ventral dissociation for the P1 attentional effects, suggesting that the early enhancement of the P1 (80–100 ms) due to spatial attention can be accounted for by dorsal sources, in the lateral mid-occipital cortex for both upper and lower field stimuli, whereas the latter phase of the P1 (100–130 ms) originates from the ventral occipital cortex (Martinez et al., 1999; 2001; Di Russo et al., 2002; 2003). The enhanced N1 component was estimated to arise from multiple generators in the occipito-parietal and occipito-temporal cortex, partly overlapping with the P1 generators (Clark et al., 1995; Clark and Hillyard, 1996; Gomez Gonzales et al., 1994; Di Russo et al., 2003). In a recent combined ERP and fMRI study, the early part of the N1 (130–160 ms) has been modeled by a dipolar source in the superior parietal cortex (Di Russo et al., 2003).

Although the cues that were used to trigger spatial attention mechanisms in the present study were of a different nature, the sources of these P1 and N1 components, evoked by quite simple visual stimuli, are probably highly similar to the aforementioned studies. On the other hand, the attentional effects may have been triggered by different regions than the fronto-parietal attentional network thought to be involved in modulating the early processing of visual targets in previous paradigms (e.g. Corbetta, 1998; Kastner and Ungerleider, 2000). A likely candidate is the STS-region, where cells sensitive to eye gaze have been reported in monkeys (Perrett et al., 1985; 1992). The STS shares reciprocal connections to the intraparietal sulcus, an area that could mediate the transfer of information about socially relevant stimuli to parietal neural systems for directing spatial attention (Harries and Perrett, 1991). In humans the perception of gaze direction also recruit the intraparietal sulcus (Puce et al., 1998; Wicker et al., 1998; Hoffman and Haxby, 2000; Calder et al., 2002; Pelphreys et al., 2003; Hooker et al., 2003), a region participating in spatial perception and covert shifts of attention (Corbetta, 1998; Nobre et al., 1997), and the STS during reflexive shifts of attention (Kingstone et al., 2004). The reflexive shifts of attention triggered by averted eye gaze perception may thus to be mediated by an interaction between face-responses in

the STS and the spatial attention system in the IPS (Kingstone et al., 2000; Friesen et al., 2004).

In the present study, the P1 and N1 components to upper/lower visual field stimuli were not only enhanced, but their latency was also decreased following congruent eye gaze cues. These effects have been reported previously with or without motion of the lateral eye gaze cue (Schuller and Rossion, 2001; 2004, respectively). It is the first time that they are observed with oblique eye gaze cues directed towards lower and upper portions of the visual field. Such latency effects appear to be characterized by speeding up or facilitation mechanisms rather than a delayed processing for targets following incongruent eye gaze cues (Schuller and Rossion, 2004). ERP latency modulations have been only reported in few spatial attention studies (Di Russo and Spinelli, 1999a; 1999b; 2002), but not during trial-by-trial cueing. In their first study, Di Russo and Spinelli (1999a) reported an amplitude increase and latency decrease of both the P1 and N1 components during sustained attention. The latency effects ($\sim$10 ms) for the two components were in the same range as what is found with reflexive shifts of attention following eye gaze cues (Schuller and Rossion, 2001; 2004; the present study). It is currently unclear why latency effects are observed in some studies of spatial attention and not others (e.g. Di Russo et al., 2003). Small deviations from eye fixation on the centre of the screen are unlikely to be the cause of the latency differences, for several reasons. First, as in previous experiments (see Schuller and Rossion, 2001; 2004), eye movements were controlled by EOG inspection, allowing the rejection of trials associated with detectable ocular deflections. Second, if anything, small gaze shifts in the direction of the target lead to increases of amplitude of the stimulus evoked potentials, but have little or no effect on latency variations (Di Russo and Spinelli, 1999a). Third and most importantly, in the present study, contrary to previous paradigms of sustained attention where latency modulations of visual components were found (Di Russo and Spinelli, 1999a; 1999b; 2002), the target following the eye gaze cues could appear either at the congruent or the incongruent location (diametrically opposite corner) with equal probability, giving no advantage in detection to the subject who would move the eyes towards one of the corners. Furthermore, the catch trials used in the present study also prevented subjects to anticipate. All these reasons explain why subjects normally maintain accurate fixation and avoid moving their eyes during covert orienting involving detection tasks (Posner and Cohen, 1994). Fourth, the fact that the latency (and amplitude) effects do not start at the level of the C1, as could be shown in the present study, but only at the level of the P1 and N1, also suggest that anticipatory eye movements (i.e. before the target appears) are not responsible for the latency differences reported here. Note that eye gaze shifts could still have been made *after* the target appears, but then, given the time required to make a saccadic movement to the target, they are unlikely to be at the basis of latency modulation of components occurring before 200 ms after the target onset. Finally, there was no evidence whatsoever of vertical or horizontal eye movements on the ocular channels (VEOG and HEOG) folowing the presentation of the eye-gaze cues until the appearance of the target[2].

The decreases of latency for visual components evoked by eye-gaze cued targets is in line with the 'prior entry' hypothesis, which proposes that paying attention to a stimulus accelerates the sensory processing of this stimulus (see Schneider and Bavelier, 2003). However, the neural mechanisms underlying these effects are currently unclear. The onset times of the response of single cells in areas MT (e.g. Treue and Maunsell, 1996; 1999), V2 and V4 (e.g. Luck et al., 1997; McAdams and Maunsell, 1999) of the monkey do not appear to be modulated by attention. Yet, in some cases, attentional changes were evident early in the stimulus response, with the congruent/attended response beginning at the same time as the incongruent/unattended response but increasing somewhat more steeply. Such effects may be related to a larger number of cells recruited for attended than unattended conditions, even though the onset discharge time of individual neurons is identical in the two conditions. Neural activity among the population of cells sensitive to the visual features may accumulate more slowly when these features are unattended, leading to response delays observed at the level of field potentials, decisional and motor responses (see Perrett et al., 1998).

Finally, the P3 component was enhanced for incongruent trials, as observed previously (Hugdahl and Nordby, 1994; Mangun and Hillyard, 1991; Schuller and Rossion, 2001). This last effect could be related to the subjects' expectancy of a target appearing in the congruent location. Because incongruent trials are 'unexpected' (although their probability of occurrence is the same as congruent trials) given the direction of the eye gaze cue, they give rise to an amplified P3, in line with the observation that this component's amplitude is usually inversely proportional to target probability. The P3 also peaked earlier for congruent trials, consistent with the reaction time advantages observed for congruently cued target locations (Wright et al., 1995) and the idea that attention accelerates perceptual decision mechanisms (Carrasco and McElree, 2001; Schneider and Bavelier, 2003).

6. Conclusions

Eye-gaze cueing to portions of the upper and lower visual field trigger powerful reflexive attention shifts to these locations, speeding up and enhancing visual processing of

[2] On the 4 waveforms corresponding to the 4 types of cues (eye gaze to the right superior corner, left superior corner, right inferior, left inferior), the maximal variation of amplitude during a 800 ms epoch time-locked to the cue was below 1 μV for the HEOG electrode, and below 0.3 μV for the VEOG electrode.

targets appearing in these locations. Despite the special nature of eye gaze as attentional cues, which is thought to be subtended by specific neural mechanisms, these reflexive effects of attention to central cues appear to take place only after the first activation of the primary visual cortex, similarly to modulations of visual processing following sustained attention or trial-by-trial cueing with predictive cues.

Acknowledgements

The authors thank Laurent Dehaye for his precious help during stimulus preparation and data acquisition, and Christine Schiltz for helpful suggestions on a previous version of this manuscript. This work was supported by a grant ARC 01/06-267 (Communauté Française de Belgique—Actions de Recherche Concertées), the Belgian National Fund of Scientific Research (FNRS) for BR. AMS was supported by the section Research and Development of the Luxembourg Research Ministry and by a grant from the Faculty of Medecine, UCL. This publication was gratefully supported by the Fonds National de la Rechenche, Luxemboung.

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