Saccade preparation is required for exogenous attention but not endogenous attention or IOR

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Abstract

Covert attention is tightly coupled with the control of eye-movements but there is controversy about how tight this coupling is. The Premotor theory of attention proposes that activation of the eye-movement system is necessary to produce shifts of attention. In this study we experimentally prevented healthy participants from planning or executing eye-movements and observed the effect on exogenous attention, Inhibition of Return (IOR) and endogenous attention. The participants experienced a deficit of exogenous attentional facilitation that was specific to locations that were inaccessible by saccade. In contrast their ability to endogenously orient attention was preserved as was IOR. These results show that (a) exogenous attention depends on motor preparation (b) IOR is independent of motor preparation and exogenous attention and (c) endogenous attention is independent of motor preparation. Although these data are consistent with a weak version of the Premotor theory, we believe they can be better explained by a biased competition account of visual attention.

Introduction

Attention can be oriented endogenously in response to the goals and desires of the observer (e.g. looking both ways before crossing the street) or reflexively in response to salient events in the environment (e.g. responding to an unexpected car horn). Endogenous attention is relatively slow to deploy, but creates a sustained enhancement at the attended location (e.g. Muller & Rabbitt, 1989). Reflexive attention shifts are short-lived and superseded by a sustained inhibitory effect at the location of the salient event, known as Inhibition of Return (Posner, Rafal, Choate, & Vaughan, 1985). There is considerable controversy surrounding the neural mechanisms that control these different modes of attention, but one enduring idea is that covert visuospatial attention is somehow linked to the eye-movement system.

The Oculomotor Readiness hypothesis (OMRH) hypothesised that covert spatial attention was the product of planned but unexecuted eye-movements. This idea was initially proposed and rejected by Klein (1980), on the basis that preparing but withholding a saccadic eye-movement did not enhance perceptual performance at the saccade goal. However, methodological issues with this study made interpretation of the results problematic (e.g. Rizzolatti, Riggio, & Sheliga, 1994; Shepherd, Findlay, & Hockey, 1986) and OMRH was subsequently reformulated and expanded by Rizzolatti and colleagues (Rizzolatti, Riggio, Dascola, & Umilta, 1987; Rizzolatti et al., 1994), who made four explicit predictions about the relationship between activity in the motor system and covert attention. Specifically, they proposed that (1) visual spatial attention

arises from activation in the parts of the brain used to prepare actions, (2) that covert attention is dependent on this activation, (3) that action preparation in any effector system is sufficient to produce spatial attention, but the (4) the oculomotor system plays a privileged role in visual spatial attention. This "Premotor" theory of attention has been extremely influential, and there is compelling evidence that preparing to move the eyes to a location engages the same frontoparietal network (including Frontal Eye Field, FEF and Lateral Intraparietal Cortex, LIP) as covertly attending to that location (Awh, Armstrong, & Moore, 2006; Beauchamp, Petit, Ellmore, Ingeholm, & Haxby, 2001; Corbetta et al., 1998; de Haan, Moryan, & Rorden, 2008; Grosbras & Paus, 2002; Kustov & Robinson, 1996; Muggleton, Juan, Cowey, & Walsh, 2003; Nobre, Gitelman, Dias, & Mesulam, 2000; O'Shea, Muggleton, Cowey, & Walsh, 2004; Perry & Zeki, 2000; Smith, Jackson, & Rorden, 2005; Smith, Jackson, & Rorden, 2009). However, a finer grained analysis of the oculomotor and attentional functions of Frontal Eye Field using single cell recording in awake monkeys shows that covert attention and saccade preparation are controlled by overlapping *but independent* neural populations (Sato & Schall, 2003; Thompson, Bichot, & Sato, 2005; Thompson, Bichot, & Schall, 1997), indicating that covert attention and oculomotor control have separate neural substrates. Furthermore, an elegant study by Juan, Shorter-Jacobi and Schall (2004) has successfully dissociated activity related to saccade preparation from covert attention in the macaque FEF, suggesting that covert attention is not contingent on oculomotor planning in FEF.

The claim that activity in the motor system is necessary and sufficient for spatial attention is also controversial. Consistent with the Premotor theory, there is strong behavioural evidence that attention is locked onto the goal of eye-movements in the 120 or so milliseconds prior to the onset of a voluntary eye or arm movement (Baldauf & Deubel, 2008a, 2008b; Deubel, 2008; Deubel, Schneider, & Paprotta, 1998; Dore-Mazars, Pouget, & Beauvillain, 2004; Hoffman & Subramaniam, 1995; Shepherd et al., 1986). However, when eye-movements are planned but *not* executed, no attentional enhancement is observed at the goal of the intended movement (Klein, 1980). Importantly, this dissociation between motor preparation and covert attention is still observed when eye-movements are monitored and attention is assessed using an unspeeded discrimination task (Hunt & Kingstone, 2003a; Klein & Pontefract, 1994). These behavioural studies suggest that that motor preparation alone is not a sufficient condition for covert endogenous attention, and are incompatible with the Premotor theory of attention.

Studies of neuropsychological patients are also inconclusive with respect to the role of motor preparation in covert attention. For example, patients with progressive supranuclear palsy (a disease that affects brain structures involved in saccade control) are significantly impaired on tasks that engage exogenous attention, but relatively good at tasks which require endogenous attention shifts (Rafal, Posner, Friedman, Inhoff, & Bernstein, 1988). Sereno, Briand, Amador & Szapiel (2006) described a single case with a collicular lesion who showed abnormal reflexive eye-movements and unreliable

exogenous attention in a peripheral cueing task (peripheral cueing effects were only observed in 2/4 conditions where they would be predicted to occur), again suggesting that exogenous attention is dependent on oculomotor control. Interestingly, at long cue-target SOAs the patient showed a reliable facilitation at the cued location, suggesting that the patents ability to endogenously orient attention was intact. Similarly, patients with chronic lesions of the cortical eye-movement system, specifically the FEF, have a saccadic deficit but no deficit of endogenous attention (Henik, Rafal, & Rhodes, 1994), suggesting that endogenous attention is not reliant on the ability to plan eye-movements.

Data from two studies of patients with chronic ophthalmoplegia (paralysis of the eyes) argues for a partial dissociation between the oculomotor system and covert attention. Smith, Jackson & Rorden (2004) reported the case of AI, a participant with congenital ophthalmoplegia who had never made an eye-movement (see Gilchrist, Brown, & Findlay (1997) for a full description). AI performed normally on tasks requiring endogenous attentional orienting, but had a deficit of reflexive attention (although interestingly, IOR was also preserved). Gabay and colleagues (Gabay, Henik, & Gradstein, 2010) observed the same pattern of impaired exogenous attention with preserved endogenous attention and IOR in 3 patients with Duanes Syndrome, a developmental disorder characterised by the inability to make lateral eye-movements, typically affecting the left eye.

One appealing interpretation of this neuropsychological data is that saccade preparation is required for exogenous attention but not for endogenous attention, which would explain why endogenous saccade preparation does not necessarily produce a shift of attention. However, it is necessary to be cautious when interpreting this neuropsychological data, given the low sample sizes (the largest study included only 7 participants) and the potential for development of compensatory strategies in chronic conditions, particularly as there is some evidence that patients with *acute* ophthalmoplegia exhibit a deficit of endogenous attention (they fail to orient in response to predictive central cues (Craighero, Carta, & Fadiga, 2001)).

The suggestion that the functional equivalence of saccade preparation and covert attention proposed by the Premotor theory of attention is only true for exogenous attention leads to a clear prediction. If saccade preparation is prevented, participants should experience a deficit of exogenous attention but endogenous attention should be preserved. One elegant method to prevent saccade preparation in healthy participants was introduced by Craighero, Nascimben and Fadiga (2004). Participants were asked to abduct the eye by 40 degrees, such that they were fixating a point near the limit of their oculomotor range (see Figure 1a). When the eye is in this position it is impossible to plan or execute eye-movements further into the temporal hemifield. At this stage, stimuli are presented to the left and right of the gaze direction. Note that peripheral stimuli are equidistant from fixation, so fall on equally sensitive portions of the retina.

Craighero et al., (2004) found that this manipulation disrupted participant's ability to orient attention in response to spatially predictive cues presented close to fixation. They interpret this deficit as a failure of endogenous attention. However, this conclusion may be premature for the following reason. An endogenous attention shift requires the observer process semantic information about a cue in order to consciously orient attention to the cued location. The cue employed by Craighero et al., was a sudden onset pointer that was lateralised to the left or right hemispace. This type of cue directly signals probe location, so does not require the observer to consciously process the symbolic content of the cue. If the cue does not require conscious processing, it seems unlikely to engage endogenous attention. In other words, it is possible that the cues used by Craighero elicited very limited engagement of endogenous attention, and that the attentional effects they observed were primarily due to activation of the exogenous attentional system.

More recently, we have shown that the eye-abduction manipulation slows visual search for a feature singleton in the temporal hemispace when the search is easy and increases omission errors when the search is made more difficult (Smith, Ball, Ellison, & Schenk, 2010). Although this result is consistent with the view that preventing saccade preparation disrupts exogenous attentional capture by salient items, it did not offer a strong test of the role of saccadic preparation in endogenous attention.

In summary, the Premotor theory of attention holds that covert spatial attention is dependent on activation of the eye-movement system but the existing empirical

evidence is more consistent with the more conservative interpretation that exogenous attention is tightly coupled to the oculomotor system whereas endogenous attention is independent of oculomotor control. Here we test the role of saccade preparation in both exogenous and endogenous attention using the eye-abduction technique to create a brief and reversible inability to generate eye-movements in one direction. Experiment 1 examines the influence of transient eye-abduction on exogenous attention and inhibition of return for the first time. Experiment 2 examines whether eye-abduction affects endogenous attention in a symbolic number-cueing task, extending the previous work of Craighero et al. (2004) and testing the oculomotor readiness hypothesis of Klein (1980). If covert spatial attention is dependent on the ability to plan eye-movements, abduction of the eye should lead to deficits of spatial attention at locations which are inaccessible to the eye-movement system.

Experiment 1: Effect of eye-abduction on exogenous attention and Inhibition of Return.

Methods

Participants

Nineteen participants drawn from the undergraduate and postgraduate population of Durham University volunteered for the experiment (13 female). Nine participants were right eyed. Ages ranged from 18-32 (mean 21.8). Undergraduate

participants enrolled on the Psychology or Applied Psychology degree programs

received credit in the Department of Psychology participant pool for taking part. Other

undergraduates were paid £4 for participating. All participants gave informed consent

before participating. The study was approved by the Department of Psychology Ethics

Committee.

Apparatus

Stimuli were generated using a Cambridge Research Systems ViSaGe graphics

card and displayed on a 17-inch Sony Trinitron CRT monitor with a refresh rate of 100Hz.

Responses were collected using a button-box with two response buttons. A laser pointer

was attached to the centre of the forehead of the participant using a Velcro headband.

A point corresponding to a 40 degree angle of abduction was marked on the wall. In the

abducted condition the participant fixated the centre of the monitor and turned the

body and head such that the laser beam intersected with this point. If the participant

moved their head to reduce the angle of abduction, the experimenter asked them to

return to the correct position and marked the trial number so it could be excluded from

the analysis. In the central condition the laser beam intersected with the horizontal

midline of the monitor.

Stimuli

The array consisted of three black 3° x 3° boxes arranged on the horizontal

meridian on a grey background. The central box contained a black fixation point. The

centres of the two peripheral boxes were 7.5° from fixation. Boxes were cued by changing the colour of the box from black to white for 150ms. The target was a 0.5° x 0.5° white filled white square which could appear at the centre of either peripheral box. Figure 1 illustrates the experimental setup.

Procedure

Participants used the dominant eye for the experiment. The task was performed monocularly, with the non-preferred eye patched. Eye dominance was assessed for each participant by having them sit ~2 meters away from the experimenter and fixate on the nose of the experimenter. Participants were then told to extend their arms and bring their hands together in front of the eyes leaving only a gap through which the experimenter could see the dominant eye.

Participants took part in two conditions. In the Central condition the eye was in the canonical position (i.e. the centre of the orbit). In the Abducted condition the participant fixated the centre of the display and the head and body were rotated to the right if the left eye was in use and to the left if the right eye was in use. This manipulation produced an abduction of the eye of 40° into the temporal hemifield. The response box was placed in line with the horizontal midline of the body. Participants responded with the preferred hand (see Figure 1). Participants were told to press the upper button of the response box as quickly as possible when they saw the target and the other button if no target appeared.

Figure 1 around here

Participants were positioned 57cm away from the display with their head resting in a chinrest. They were instructed to fixate the central fixation point and not move their eyes. Trials began with the presentation of the stimulus array for 1500ms. A non-predictive cue was then presented for 150ms at one of the 3 locations in the array. On half the trials there was a 66% probability of a target appearing in the centre of one of the 2 peripheral boxes immediately following the offset of the cue (150ms SOA). On the other half of the trials the central fixation point was re-cued for 50ms immediately following the offset of the cue. On these trials there was 66% probability of a target appearing in the centre of one of the 2 peripheral boxes 550ms following the offset of the cue (750ms SOA). Peripheral cues correctly predicted target location with an accuracy of 50%.

Each participant completed one block of fifty four practice trials (with eye in the central position), four blocks of fifty four trials in the Central condition and four blocks of fifty four trials in the Abducted condition. There were 12 trials in each Hemifield at each level of Validity, Eye-Position and SOA, plus 144 no-target trials where one of the boxes was cued but no target presented. Figure 1b shows the time-course of events.

Results

Two participants requested to withdraw from the experiment before testing had been completed and were excluded from the analysis. One participant was unable to maintain a stable fixation in the eye-abduction condition and was also excluded.

Data were filtered to anticipations (responses faster than 100ms) and outliers that were more than 2.5 standard deviations greater than an individual's mean reaction time. This procedure resulted in the exclusion of 201/6912 (2.9%) of trials. We also removed 5 trials during which participants had been requested to adjust their head position. All these trials came from the Eye Abducted condition. Target-absent trials were not analysed.

False alarms were rare and evenly distributed across the eye-abduction conditions (18/1152 in the Central condition, 24/1152 in the Abducted condition). Misses were also rare (36/2304 in the central condition, 20/2304 in the abducted condition).

Mean reaction times were submitted to a 2x2x2x3 repeated measures ANOVA with factors of SOA (150ms and 750ms), Eye Position (Central and Abducted), Hemifield (Nasal and Temporal) and Validity (Valid, Invalid and Neutral). The results revealed a significant main effect of SOA ($F_{(1,15)}$ = 11.32, $p < 0.05$, $\acute{\eta}_p^2$ = 0.43) and a significant 4-way interaction between SOA, Eye Position, Hemifield and Validity ($F_{(1,15)}$ = 4.01, $p < 0.05$, $\acute{\eta}_p^2$ = 0.21).

To investigate this interaction we analysed the data from the 150 and 750ms

SOAs in separated 2x2x3 ANOVAs. The analysis of the 150ms SOA revealed a significant

main effect of Validity ($F_{(2,15)}$ = 5.52, p < 0.05, η_p^2 = 0.27) and a significant 3-way

interaction between Eye Position, Hemifield and Validity ($F_{(2,30)}$ = 5.67, p < 0.05, η_p^2 =

0.275). We therefore ran two 2 (Eye Position) x 3 (Validity) ANOVAs at each level of Eye

Position. For the Central eye position the analysis revealed a main effect of Validity

($F_{(1,15)}$ = 6.4, p < 0.05, η_p^2 = 0.3), such that reaction times were significantly faster to

validly cued targets than invalidly cued targets. Planned comparisons (1-tailed t-tests)

confirmed that this validity effect was present in both nasal ($t_{(1,15)}$ =2.81, p < 0.025, d =

0.25) and temporal hemispheres ($t_{(1,15)}$ =3.15, p < 0.025, d=0.43). In the Abducted eye

position the analysis revealed no main effects and a trend towards a significant

Hemifield x Validity interaction ($F_{(1,15)}$ = 2.81, p = 0.076, η_p^2 = 0.16). Here, planned

comparisons (1-tailed t-tests) revealed a significant validity effect in the nasal hemifield

($t_{(1,15)}$ =4.64, p < 0.025, d=0.32), but no validity effect in the temporal hemifield ($t_{(1,15)}$

=0.56, p = 0.956, d=0). Figure 2 illustrates the interactions in the 150m SOA.

Figure 2 around here

Analysis of the 750ms SOA revealed a significant main effect of Validity ($F_{(2,15)}$ =

5.63, p < 0.05, η_p^2 = 0.27), such that reaction times to validly cued trials were significantly

slower than both Invalid trials ($t_{(1,15)}$ =2.95, p < 0.025, d =0.28) and Neutral trials ($t_{(1,15)}$

=3.22, p < 0.025, d =0.22), and a significant 2-way interaction between Eye Position and

Hemifield ($F_{(1,15)}$ = 4.75, p < 0.05, η_p^2 = 0.275). Inspection of Figure 3 suggests that this

interaction was driven by a slowing of reaction times to all targets that appeared in the temporal hemifield in the Abducted condition. However, there was no 3-way interaction between Eye-Position, Hemifield and Validity ($F_{(2,30)}$ = 0.434, p = 0.65, $\acute{\eta}_p^2$= 0.028), indicating that abduction of the eye did not affect Inhibition of Return.

Figure 3 around here

No cueing effect was observed in the temporal hemifield of in the eye-abducted position at 150ms SOA (i.e. participants did not attend the peripheral cue). If IOR is caused by an exogenous attention shift, the inability to exogenously attend peripheral cues in the abducted position at short SOAs should also be associated with a reduced or absent IOR effect at long SOAs. To test this hypothesis we investigated the IOR effect in the Nasal and Temporal hemifields when the eye was in the Abducted position. There were significant IOR effects in the Nasal hemifield (Valid = 432ms, Invalid =402ms; $t_{(1,15)}$ =3.29, p < 0.025, d =0.34) and Temporal hemifield (Valid = 445ms, Invalid =416ms; $t_{(1,15)}$ =2.85, p < 0.025, d =0.33).

Discussion

Our aim was to see if extreme abduction of the eye influences exogenous attentional orienting in the temporal hemispace (i.e. beyond the range of eye movements). The results show that non-predictive peripheral cues in the temporal hemispace failed to generate an initial cuing effect (SOA = 150ms) when the eye was

abducted. This data offers clear evidence that exogenous shifts of attention are dependent on the ability to plan and execute an eye-movement and confirms the predictions of the Premotor theory of attention. A second noteworthy finding was that peripheral cues presented in the temporal hemifield in the eye abducted position were able to elicit Inhibition of Return at the 750ms SOA even though they were unattended at the 150ms SOA. This result is consistent with the view that IOR is caused by the onset of a peripheral cue, not a shift of attention to that cue. This conclusion is consistent with theoretical accounts of IOR which propose that exogenous attention and IOR are distinct processes (Posner & Cohen 1984), and previous empirical evidence that IOR can be dissociated from exogenous attention (Danziger & Kingstone, 1999; Smith, Jackson, & Rorden, 2009; Smith, Rorden, & Jackson, 2004; Smith & Schenk, 2010; Tassinari, Aglioti, Chelazzi, Peru, & Berlucchi, 1994).

Superficially, these findings are inconsistent with a previous study by Hunt & Kingstone (2003b), who argued that exogenous attention was *independent* of saccade preparation. However, their conclusion was based on the observation that saccade planning did not elicit Inhibition of Return in a manual response task. Critically, Hunt & Kingstone (2003b) did not directly measure exogenous attention but rather used IOR as a marker for an unobserved exogenous attention shift, based on the assumption that IOR is preceded and caused by reflexive attention. Given the evidence that IOR and exogenous attention are generated by different cognitive mechanisms we believe that this assumption is not valid, and so their result does not provide convincing evidence of

a dissociation between exogenous attention and oculomotor preparation. A more parsimonious interpretation of Hunt & Kingstone (2003b) is that IOR in manual response tasks is independent of saccade preparation, a result that we have replicated in this study and that is consistent with other data showing dissociations between activity in the oculomotor system and IOR (Chica, Klein, Rafal, & Hopfinger, 2010; Sumner, Nachev, Vora, Husain, & Kennard, 2004).

Previous studies have shown a temporal hemifield advantage for both exogenous cueing effects and IOR when the eye is in the canonical position (Rafal, Henik, & Smith, 1991). We did not observe a statistically significant temporal hemifield advantage for cueing or IOR. However, consistent with previous results, examination of the effect size of the cueing effect at 150ms SOA (Cohen's d) indicated that the effects of the cue were greater in the temporal hemifield (d =0.43) than in the nasal hemifield (d =0.25). In contrast, a post-hoc analysis of the size of the IOR effect in the nasal and temporal hemifields (collapsed across eye-position) suggested that the effect size was similar for both hemifields (Temporal d= 0.32, Nasal d= 0.27). The reason for this discrepancy is not clear but there were a number of differences between Experiment 1 of this study and the earlier study by Rafal et al., (1991). Specifically, Rafal et al., (1991) presented targets further into the periphery (10° vs 7.5° in our study), the fixation point was removed prior to the onset of cue and target whereas we left the fixation point on, cue and target remained present until response whereas our cue was only presented for

150ms, and Rafal et al., (1991) required participants to make a choice reaction time, whereas we used a simple detection reaction time.

One alternative explanation for the results is that the onset of the peripheral cue triggered small breaks of fixation in the direction of the cue. These small changes in fixation may have brought the cued location closer to the fovea, thus enhancing the sensory representation of the cue. We did not monitor eye-movements, so cannot definitely rule out this possibility. However, if the RT benefit occurs because the participant fixated the cued location, one would predict that Inhibition of Return effects would be smaller or absent because the probe will appear at the center of vision. In contrast to this prediction, we observed intact IOR in all conditions. Furthermore, consistent with the assumption that participants were maintaining fixation, the magnitude of IOR was similar in the nasal and temporal hemifields. It is also worth noting that in a previous study utilizing the eye-abduction technique we monitored eye-movements during a pop-out visual search task (Smith et al., 2010). Fewer than 1% of trials had to be discarded due to breaks of fixation, which lead us to conclude that participants were very good at maintaining fixation, even under conditions where the properties of the visual array afford the execution of eye-movements.

The results of Experiment 1 demonstrate that exogenous attention is dependent on saccade preparation and are consistent with previous neuropsychological evidence that patients with chronic deficits of eye-movements have deficits of exogenous attention. These data suggest that saccade preparation is a necessary precondition for a covert, exogenous attention shift, as predicted by the Premotor theory.

The Premotor theory also predicts that endogenous attention is dependent on saccade preparation, a prediction that is partially supported by data from Craighero and colleagues, who reported deficits of endogenous attention in patients with acute ophthalmoplegia (Craighero et al., 2001) and healthy participants whose eye-position was constrained using the eye-abduction technique (Craighero et al., 2004). However, as described in the introduction, these experiments used a lateralised, directional cue which was likely to have activated the exogenous attention mechanisms, and so provide a poor test of the causal role of saccade preparation in endogenous attention. In order to provide a more rigorous test of the Premotor theory with respect to endogenous attention we conducted a second experiment in which participants were cued using a centrally presented number cue. Number cues have no spatial component and are a more reliable method of activating only endogenous attentional mechanisms.

Experiment 2: Effect of eye-abduction on endogenous attention.

Methods

Participants

Twelve participants drawn from the undergraduate and postgraduate population of Durham University volunteered for the experiment (3 male). Nine participants were right eyed. Ages ranged from 19-36 (mean 24). Undergraduate participants received credit in the Dept of Psychology participant pool.

Apparatus

Apparatus were the same as in Experiment 1.

Stimuli

The stimulus array consisted of three black 3° x 3° boxes arranged on the horizontal meridian on a grey background. The centres of the two peripheral boxes were 7.5° from fixation. The cue to orient attention was the numeral 1 (attend left), 2 (attend right) or 3 (get ready). These numerals had a height of 12 pixels. The target was a 0.5° x 0.5° filled white square.

P*rocedure*

Participants used their dominant eye. The manipulation of eye-position was the same as in Experiment 1. Participants were instructed to attend left if they saw a 1, attend right if they saw a 2 and to get ready (but not attend left or right) if they saw a 3. They were told to press the upper button of the response box as quickly as possible when they saw the target. If no target appeared they should press the lower button.

Participants were positioned 57cm away from the display with their head resting in a chinrest. They were instructed to fixate the central fixation point and not move their eyes during a trial. On each trial a fixation point appeared for 1000ms, followed by the array of 3 rectangles. One of the three numerals then appeared at fixation for 150ms. Five hundred milliseconds after cue onset a target could appear in one of the peripheral boxes. On 12.5% of trials no target appeared. Left and right cues predicted the location

of the target with an accuracy of 75%. Left, right and get ready cues appeared an equal number of times. Each participant completed one block of fifty-four practice trials, three blocks of fifty-four trials in the Central condition and three blocks of fifty four trials in the Abducted condition (i.e. 36 valid, 12 invalid, 24 neutral and 18 no-target trials in each hemifield in each eye-position).

Results

The data were filtered to remove error trials, anticipations (response times faster than 100ms) and outliers (reaction times that were more than 2.5 standard deviations greater than an individual's mean reaction time). This procedure resulted in the exclusion of 2.5% of trials. We also removed 2 trials in which a participant had been requested to change their head position. Target-absent trials were not analysed. One participant withdrew from the experiment and their data has been excluded from the analysis.

False alarms were evenly distributed across the eye-abduction conditions (10/216 in the Central condition, 7/216 in the Abducted condition). Misses were very rare and also evenly distributed across the eye-abduction conditions (14/1728 in the Central condition, 19/1728 in the Abducted condition).

Participants mean reaction times were subjected to a 2 (Eye Position: Central/Abducted) x 2 (Hemifield: Nasal/Temporal) x 3 (Validity: Valid/Invalid/Neutral) repeated measures ANOVA. The ANOVA revealed a significant main effect of Validity

($F_{(1,10)}$ = 6.59, p < 0.01, $ή_p^2$= 0.38). Analysis of simple main effects revealed that reaction

times to Validly cued targets were significantly faster than RTs to Invalidly cued targets

(Valid = 291ms, Invalid= 386ms; $t_{(10)}$ = 2.08, p< 0.05, d =0.99) and neutrally cued targets

(Valid = 291ms, Neutral= 319ms; $t_{(10)}$ = 4.27, p< 0.05, d=0.38). Critically, there was no

evidence of a significant two-way interaction between Eye Position and Validity ($F_{(1,10)}$ =

0.61, p = 0.55, $ή_p^2$= 0.06) or a significant three-way interaction between Eye Position,

Hemifield and Validity ($F_{(1,10)}$ = 0.161, p = 0.85, $ή_p^2$= 0.02). Figure 4 shows the cueing

effects in the Eye Central and Eye Abducted conditions. Given the a-priori hypothesis

that cueing effects would be absent in the Temporal hemifield in the Eye Abducted

condition we ran two planned comparisons (1 tailed t-tests) to test for the cueing

effects in the eye-abducted condition. These t-tests confirmed that endogenous cueing

effects survived abduction of the eye in both the temporal hemifield (Valid 298ms,

Invalid 408ms; $t_{(10)}$ = 2.94, p< 0.05, d =1.04) and the nasal hemifield (Valid 294ms, Invalid

388ms; $t_{(10)}$ = 2.40, p< 0.05, d =0.94).

Figure 4 around here

Discussion

This experiment examined the role of eye position on endogenous cueing. The

results show no evidence of an interaction between validity and eye-position and clear

evidence of an attentional cueing effect in the temporal hemisphere in the Eye

Abducted condition. This result demonstrates that the ability to endogenously orient

attention is not dependent on the ability to generate an eye-movement to the cued

location and is contrary to the predictions of the Premotor theory. These data complement previous work demonstrating that saccade preparation is insufficient to trigger an endogenous shift of attention (Hunt & Kingstone, 2003a; Klein, 1980; Klein & Pontefract, 1994) by demonstrating that the ability to generate saccades is also not required for endogenous attention.

Superficially, the results of Experiment 2 appear contrary to the previous work of (Craighero et al., 2004), who reported disrupted endogenous attention following eye abduction. However, there are compelling reasons to believe that the cueing task employed by Craighero only weakly activated the endogenous attention system (see Introduction), and that the attentional effects they observed were primarily due to activation of the exogenous attention system. If the results of Craighero et al., (2004) are interpreted as a deficit of exogenous attentional orienting they can be reconciled with both the data from the experiments described here, and other studies showing that disruption to the oculomotor system disrupts exogenous attention but spares endogenous attention (Smith et al., 2004; Smith et al., 2010; Gabay et al., 2010; Rafal et al., 1988). This interpretation is also consistent with the findings of Posner, Cohen & Rafal (1982) who reported that rapid exogenous attention shifts generated by a predictive peripheral cue (80% valid) were disrupted in Progressive Supranuclear Palsy patients with opthalmoplegia, whereas endogenous attentional orienting was relatively intact.

General Discussion

The result of experiment 1 demonstrated that saccade preparation is required for exogenous attention but is not required to generate Inhibition of Return. This result is consistent with the predictions of the Premotor theory. However, the result of Experiment 2 showed that saccade preparation is not necessary for endogenous attentional orienting, a finding that is directly contrary to the predictions of the Premotor theory. These data suggest that the strong version of the Premotor theory, in which oculomotor preparation is a necessary precondition for *all* shifts of spatial attention should be rejected. However, our data are consistent with a weak version of the Premotor theory, in which only exogenous attention is dependent on motor preparation. This version of the Premotor theory fits well with existing empirical data showing that disruption to the oculomotor system generally leaves endogenous attention intact, but severely disrupts exogenous attention (Gabay et al., 2010; Rafal et al., 1988; Smith et al., 2010; Smith et al., 2004)

A weak version of the Premotor theory can be accommodated within a broader theoretical framework developed to account for both spatial and non-spatial visual attention. More specifically, the biased competition account of visual attention (e.g. Desimone & Duncan, 1995) proposes that attention is the consequence of competition within and across different sensory-motor systems. In this view, sensory inputs compete for neural representation within sensory and motor systems. The more physically salient an input is, the greater its representation. The competition between representations is integrated across sensory and motor systems so that different

systems converge on a single representation. This representation is the 'winner' of the competition and is attended, in the sense that it becomes available to higher cognitive processes such as awareness and response systems. Critically, competition can be biased towards less physically salient stimuli by top down factors such as our current goals and the content of working memory.

During the exogenous cueing task the sudden appearance of the cue briefly increases the physical salience of the cued location and triggers the preparation of a saccade, creating a powerful bias in the visual and oculomotor system towards the cued location. This bias propagates through the perceptual-motor system and facilitates processing of subsequent visual events at the cued location (i.e. the cued location becomes attended). However, when the eye is in the abducted position locations in the temporal hemifield cannot become the goal of a saccadic eye-movement, so the onset of the cue does not create a bias in the oculomotor system. As a consequence, visual events from the cued location are not prioritized in the visuomotor system so no cueing effect is observed. In contrast, during the endogenous cueing task the participant knows that the cue accurately predicts target location and can use top-down cognitive processes that are independent of the eye-movement system to bias the visual system towards the cued location.

These data are also relevant to other theoretical perspectives. For example, the Visual Attention Model (VAM: Schneider, 1995) proposes that the role of endogenous attention is to create an 'object token' that allows the attended object to be

differentiated from other objects in the scene throughout the visual system. The spatial

location of the object is encoded as part of this object token. According to this model,

attention and motor control are coupled prior to action execution, but if no action is

executed (e.g. during covert attention), then attention and motor control are

functionally dissociated. This prediction is entirely consistent with the results of

Experiment 2, which demonstrated that covert endogenous attention is dissociated

from motor preparation.

However, it is not clear how VAM can account for the very tight coupling

between motor preparation and covert exogenous attention observed in Experiment 1

and in our previous study of pop-out visual search (Smith et al., 2010). According to

VAM, exogenous cueing effects occur because the peripheral cue triggers the

segmentation of the cued location into an object token. When the target appears at the

cued location it is processed as a change to an existing object. It does not require a

separate segmentation process and is therefore detected quickly. In contrast, targets

appearing at uncued locations must be segmented from the rest of the scene which

takes time, thus slowing detection (see also Lupianez, Milliken, Solano, Weaver, &

Tipper, 2001). In this model, a failure of exogenous attention is attributable to an

inability to encode the cue and target in the same object token. However, it is not clear

from this model why disrupting oculomotor preparation should prevent the visual

system from encoding the peripheral cue and target in the same token. Indeed, VAM

specifically predicts that spatial attention is independent of motor control (as long as no

goal directed actions are being prepared), so has no mechanism that can account for attentional failures that occur as a consequence of deficits in the motor system. We therefore propose that although VAM offers a plausible theoretical account of endogenous attention, the models account of exogenous attention is less compelling.

It has been argued that the primary mechanism underlying the failure of exogenous attention in Experiment 1 was the limitation on oculomotor preparation imposed by abducting the eye. However, our manipulation may potentially have modulated attention via other mechanisms. Specifically, there is evidence that spatial processing can be profoundly affected by the perceived position of the body midline. For example, brain-injured patients with hemispatial neglect misperceive their body midline into in the ipsilesional hemispace but rotation of the trunk into the contralesional hemispace can attenuate the severity of neglect (Karnath, Christ, & Hartje, 1993; Karnath, Schenkel, & Fischer, 1991). Similarly, Grubb and Reed (2002) found that a 15 degree rotation of the trunk created an attentional bias towards locations near the trunk midline in healthy participants. In a recent study, Balslev, Gowen and Miall (2011) demonstrated that passive abduction of one eye created a spatial bias such that the perceived midline was shifted in the direction of eye-abduction. They observed that participants were faster to respond to targets that were closer to the perceived midline than those that were farther from the perceived midline, even though they were equidistant from the actual midline.

In our eye-abduction paradigm, the trunk and head were rotated by 40 degrees, such that locations in the nasal hemifield were closer to the midline and locations in temporal hemifield were farther from the midline. Given the effects of manipulating the body midline on spatial attention described above, it might be argued that the failure to observe exogenous cueing effects was due to the eccentricity of temporal hemifield locations relative to the midline and not due to the inability to plan saccades per se. Indeed, this interpretation would be consistent with our previous finding of impaired pop-out search in the temporal hemifield (i.e. the hemifield farthest from the trunk midline) (Smith et al., 2010).

However, there is a critical difference between the results of Experiment 1 of this study and the previous studies of Balslev et al., (2011) and Grubb and Reed (2002). Specifically, all three studies used salient peripheral cues to engage exogenous attention. However, only our manipulation created a specific deficit of exogenous attention. In contrast, the manipulations of Balslev et al., (2011) and Grubb and Reed (2002) changed the overall distribution of spatial attention, but did not disrupt exogenous cueing effects (i.e. peripheral cues still summoned attention, regardless of their position relative to the perceived midline). Furthermore, Rorden, Karnath and Driver (2001) failed to find any effect of manipulating the perceived midline on spatial attention using temporal order judgement and simple detection tasks. Taken together, this pattern of data is suggestive of a dissociation between the effect of eye-abduction and trunk rotation on spatial attention, such that eye-abduction disrupts exogenous

attentional capture by inhibiting oculomotor preparation whereas trunk rotation

modulates spatial attention by shifting the perceived body midline.

Participants had intact IOR effects in the abducted position despite being unable

to exogenously attend to the cued location. This result is a clear demonstration that

exogenous attention and IOR are produced by dissociated cognitive mechanisms, and is

consistent with the view that IOR is triggered by the onset of a salient peripheral event,

rather than a shift of attention (Posner & Cohen, 1984). Furthermore, our finding that

IOR was unaffected by abduction of the eye demonstrates that inhibition of return in

manual response tasks is not dependent on activation of the oculomotor system. This

result is consistent with the view that IOR is composed of at least two different

processes, a motor inhibition which slows the execution of movements (typically

saccades) towards the cued location, and a perceptual inhibition that disrupts the

processing of visual information (Taylor & Klein, 2000) and that these different

processes are mediated by different neural mechanisms (Sumner et al., 2004).

These findings are consistent with a growing body of evidence that activation of

the oculomotor system can exert a powerful influence on cognitive processing in other

domains. However, the nature of the interactions between different motor and

cognitive systems is not well understood and a number of issues remain unresolved. For

example, there has been considerable recent interest in 'social attention' (rapid attention shifts

in response to the gaze of another person). There is considerable controversy surrounding the

mechanisms that mediate social attention but recent studies suggest that social attention may

engage the oculomotor system (e.g. Kuhn & Kingstone, 2009). One unresolved issue in this domain is the extent to which social attention is *dependent* on the activation of the oculomotor system. Similarly, overt oculomotor activation is known to disrupt spatial working memory (e.g. Pearson & Sahraie, 2003) and visual imagery but the precise role of the oculomotor system in these cognitive processes is unknown. The eye-abduction technique permits transient and specific disruption of the oculomotor system, and therefore offers the potential to explore the role of the oculomotor system in a wide range of cognitive functions including attention, visual imagery, working memory and social cognition

In summary, these experiments have shown that disrupting saccade preparation by abducting the eye creates a deficit of reflexive attention, but has no effect on endogenous attention or inhibition of return in healthy participants. Previous neuropsychological studies have reported similar dissociations, but given the chronic nature of the deficits and the small sample sizes, it was impossible to rule out the possibility that the results were due to compensatory changes to the cognitive system, rather than being indicative of the organisation of the attention system in the healthy brain. The fact that the same dissociation can be observed in healthy participants with an acute and reversible disruption of the oculomotor system is consistent with the view that exogenous attention is dependent on activation of the eye-movement system whereas endogenous attention and IOR are not.
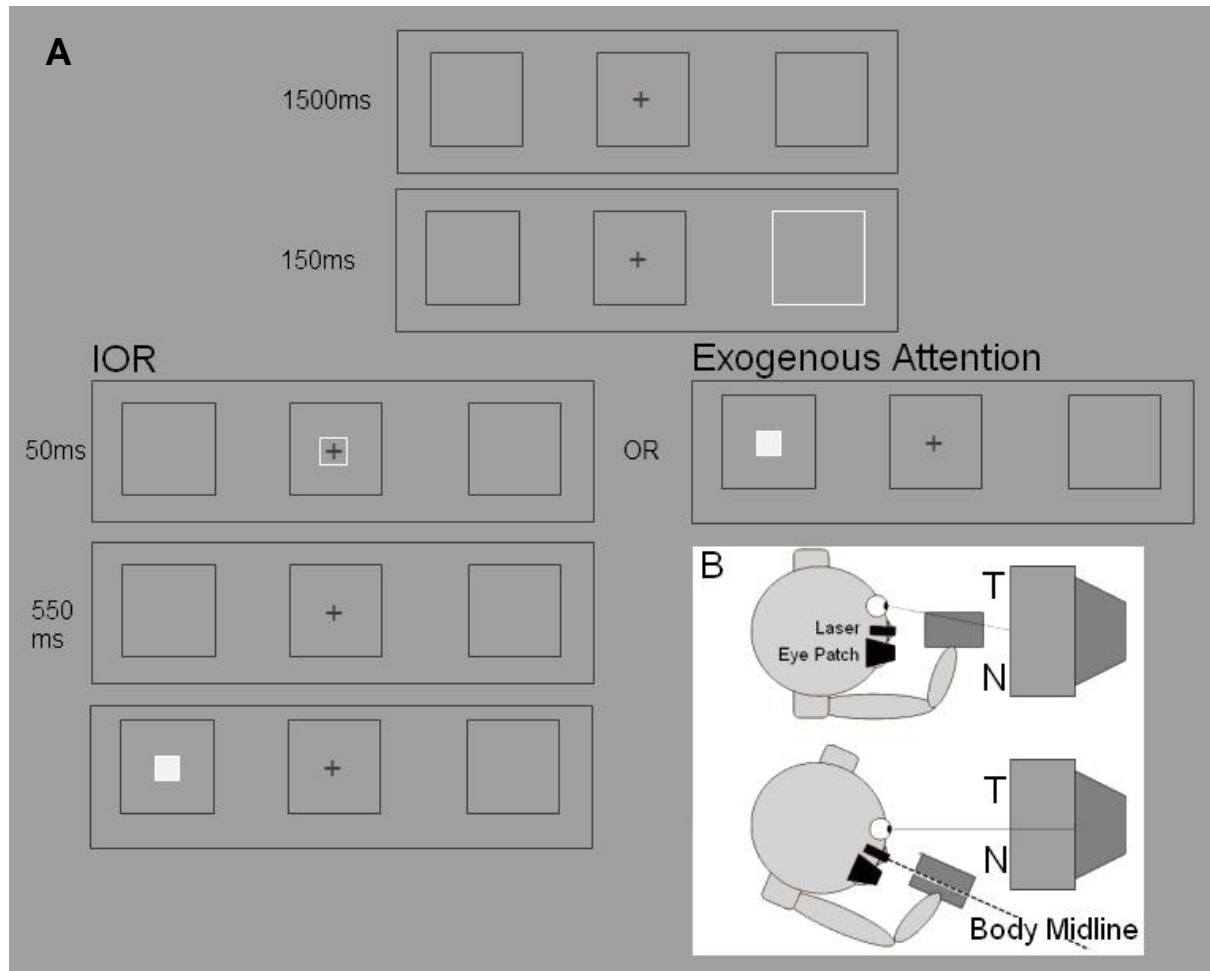
Figures and Tables

Figure 1: Cartoon showing experimental setup. Panel A shows the time-course of events during the cueing task. The figure shows an invalid trial. Panel B shows the 2 eye-positions. The upper position is the canonical central position, the lower the abducted eye position. The angle of abduction was 40 degrees. Participants performed the task monocularly with the preferred eye. The non-preferred eye was patched (in this figure the right eye). T= temporal hemifield, N= nasal hemifield.
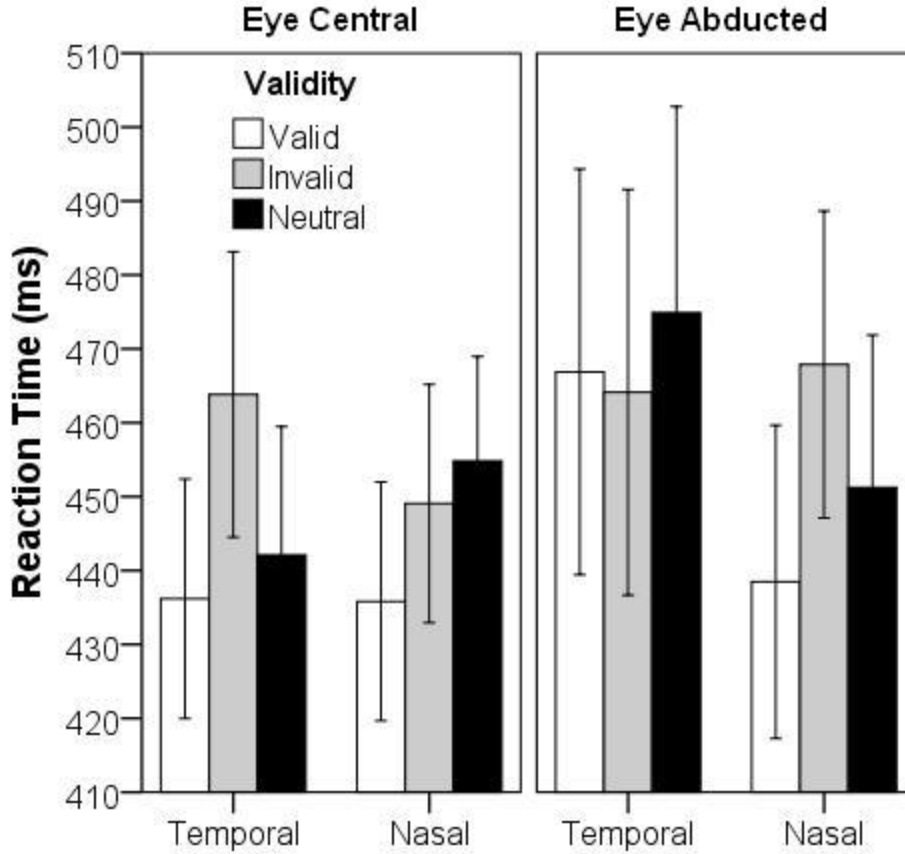
Figure 2: The 3-way Eye-Position x Hemifield x Validity interaction at 150ms SOA. Bars represent reaction times on valid (white bars), invalid (grey bars) and neutral (black bars) trials. In the central condition the cueing effect is significant in both nasal and temporal hemifields. In the abducted condition the cueing effect is significant in the nasal hemifield but there is no cueing effect in the temporal hemifield. Error bars show +/-1 SEM.
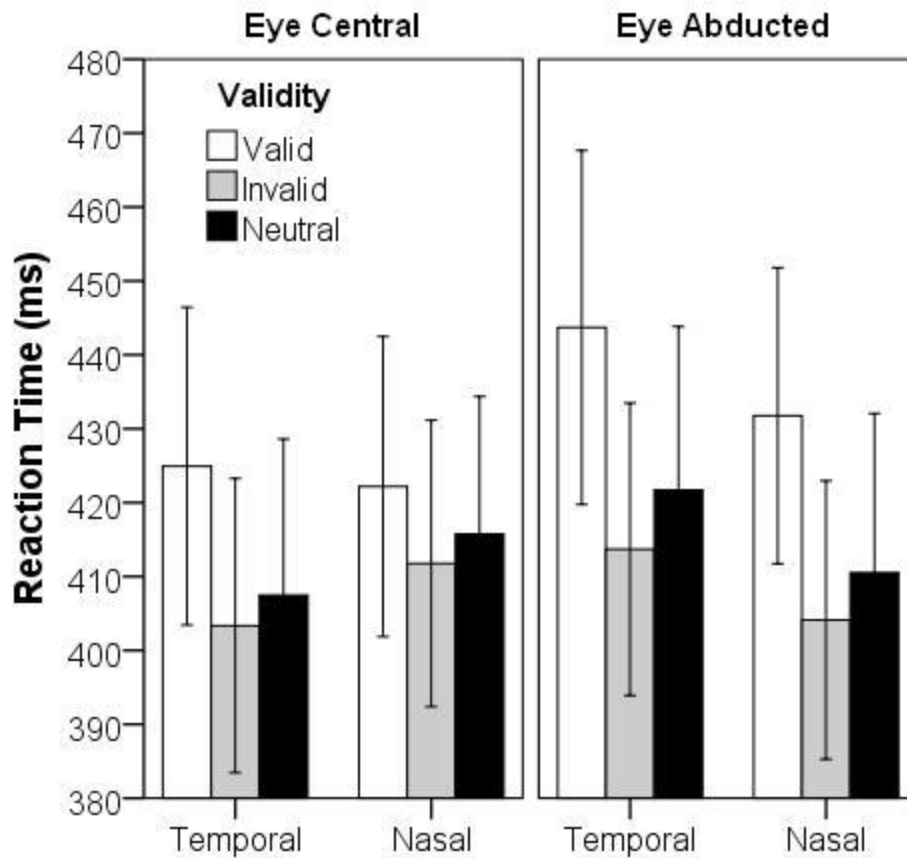
Figure 3: Reaction times at 750ms SOA during the exogenous cueing task. Bars represent reaction times on valid (white bars), invalid (grey bars) and neutral (black bars) trials. Inhibition of Return is present in both nasal and temporal hemifields in the central and abducted conditions. Error bars show +/-1 SEM.
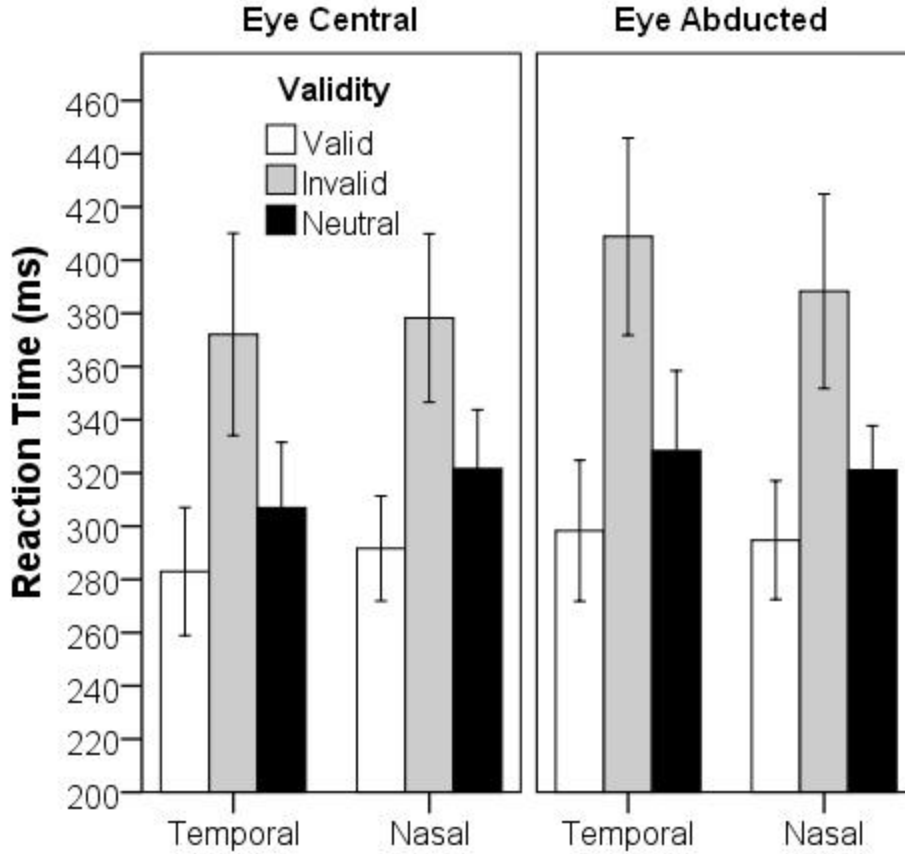
Figure 4: Results of the endogenous cuing task. Bars represent reaction times on valid (white bars), invalid (grey bars) and neutral (black bars) trials. Significant endogenous cueing effects are present in the nasal and temporal hemifields in both eye central and eye abducted conditions. Error bars show +/-1SEM

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