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Increased Effect of Target Eccentricity on Covert Shifts of Visual Attention in Patients With Neglect

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Abstract

Debate continues regarding the mechanisms underlying covert shifts of visual attention. We examined the relationship between target eccentricity and the speed of covert shifts of attention in normal subjects and patients with brain lesions using a cued-response task in which cues and targets were presented at 2° or 8° lateral to the fixation point. Normal subjects were slower on invalid trials in the 8° as compared to 2° condition. Patients with right hemisphere stroke with neglect were slower in their responses to left-sided invalid targets compared to valid targets, and demonstrated a significant increase in the effect of target validity as a function of target eccentricity. Additional data from one neglect patient (JM) demonstrated an exaggerated validity × eccentricity × side interaction for contralesional targets on a cued reaction time task with a central (arrow) cue. We frame these results in the context of a continuous ‘moving spotlight’ model of attention, and also consider the potential role of spatial saliency maps. By either account, we argue that neglect is characterized by an eccentricity-dependent deficit in the allocation of attention.

Keywords

neglect; disengage; attention; eccentricity; validity

1. Introduction

An enduring controversy in the study of visual cognition is the question of how attention is allocated across the visual field. Several accounts of the mechanisms by which attention focuses on certain regions of the visual field and how it moves between targets have been proposed. One model compares visual attention to a moveable spotlight (Eriksen et al., 1978; Crick, 1984; Posner et al., 1980). According to this metaphorical model, visual attention is restricted to a single region of visual space and/or object. A second key characteristic of this model is that when attention moves from one target location to another, it moves continuously through the intervening space in an analog fashion (Schulman et al., 1979). One prediction of a moving spotlight model of attention is that the time required to shift between visual targets will increase as a function of the distance between them. This distance effect has been observed in normal adults (Tsal, 1983; Henderson and Macquistan, 1993).

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A second influential model of the movement of spatial attention proposes that attention is a distributed process that can be shifted in a more ‘quantal’ fashion. A number of investigators have suggested that attention may be able to skip over points in space, suggesting a noncontinuous trajectory (Sagi and Julesz, 1985; Kwak et al., 1991; Sperling & Weichselgartner, 1995; LaBerge et al., 1997). One alternative to a moving locus model of attention was proposed by LaBerge and colleagues, who proposed the activity-distribution model, in which attention shifts from one location to another by means of the opening and closing of channels (LaBerge et al., 1983; LaBerge et al., 1986; LaBerge et al., 1997). The channels are defined as areas of relatively confined activity within more spatially diffuse regions that exist in prolonged preparatory states (LaBerge et al., 1997). According to this model, the time taken for attention to shift from one location to another does not depend on the distance between the two locations, but rather is a function of how much attentional activity exists at the location prior to the appearance of a target (LaBerge et al., 1997; Pratt and Quilty, 2002).

A related account proposes that bottom-up information is used to generate saliency maps that dictate the movement of attention to relevant targets in the visual field. By this account, visual features of a stimulus such as color, orientation, and intensity are extracted from an image and used to generate separate conspicuity maps related to feature domains. Information from these maps is combined into a saliency map, and the most active location on the map becomes the destination for the next shift of visual attention (Koch & Ullman, 1985). According to this model, the time required to shift attention from one location to another depends on the encoded salience of the target toward which attention is being directed. This original model has been refined and reinforced by computational models (Nieber & Koch, 1996; Itti & Koch, 2001).

Many observations regarding the movement of attention have been made in neurologically intact subjects; to date, shifts of attention through space have not been thoroughly explored in patients suffering from deficits in this domain. We therefore investigated the effect of brain lesions on the ability to shift attention through space.

We employed a modified version of the well-known cued-response task (Posner et al., 1978; Posner, 1980; Posner et al., 1984) in which subjects attend to peripheral targets while maintaining central fixation, giving rise to covert shifts of attention. The target is preceded by a peripheral cue, which may correctly indicate the site of the subsequent target (a “valid” cue), incorrectly indicate the site of the target (an “invalid” cue) or appear at fixation (a “neutral” cue). In normal subjects, responses for valid trials are more rapid than those for invalid trials. The “validity effect” refers to the difference in reaction time (RT) on invalid as opposed to valid trials at the same target location and is a measure of the cost of invalid cueing. This paradigm has been used to test both exogenous orienting of attention, when the cues are located peripherally in the visual field and endogenous orienting of visual attention, when the cue is located centrally in relation to the targets (Posner, 1980). We modified the cued response task by manipulating the eccentricity of the targets and cues such that the validity effect could be measured as a function of target distance and examined the performance of normal subjects and patients with stroke.

The cued-response paradigm has been employed in a number of studies of patients with brain lesions. Posner and colleagues (1984) originally reported that patients with right and left parietal lesions exhibited a substantial slowing of response time to invalidly cued targets presented in the contralesional hemispace. Because they found little difference in performance for validly cued targets in the ipsilesional as compared to contralesional hemispace, they concluded that parietal lobe lesions produced an impairment in the ability to “disengage” attention from the cued location. Since subjects with parietal lesions did not exhibit exaggerated costs with invalidly cued targets presented in the ipsilesional hemispace, the putative disengage

deficit was assumed to be present only in the ipsilesional hemisphere in which visual processing was assumed to be otherwise relatively preserved. Patients with right parietal lesions exhibited a significantly greater validity effect for targets in the contralesional hemisphere, prompting some investigators to suggest that the failure to disengage attention in the ipsilesional hemisphere was a major contributor to the neglect syndrome (Farah et al., 1989; D'Erme et al., 1992; Egly et al., 1994; Friedrich et al., 1998; Bartolomeo et al., 2001). More recently, the notion of spatial saliency maps has also been used to conceptualize this disengagement. By this account, the saliency of targets in neglected hemisphere is poorly encoded, making it difficult for individuals with neglect to direct attention away from ipsilesional targets toward contralesional ones. If one further assumes that targets placed more eccentrically in neglected hemisphere are less salient than those near the midline, this model would predict that the validity effect would increase in proportion to the eccentricity of the targets (Parkhurst et al., 2002).

An alternative (but not mutually exclusive) explanation for the significantly greater validity effect for contralesional targets in neglect is that the syndrome is characterized by impairment in the movement of selective attention into or toward the neglected hemisphere. Such an account is consistent with previous demonstrations that neglect is associated with a slowing of arm/hand (Heilman et al., 1985; Mattingly et al., 1994) and eye (Behrmann et al., 2001–2002) movements into or toward the neglected hemisphere; this phenomenon has been designated “directional hypokinesia.” If subjects with neglect are also slow in moving visual attention into the neglected hemisphere one would predict that the validity effect would increase in proportion to the eccentricity of the targets because more time would be required to move attention to targets further into the impaired field. This finding would be consistent with a model in which shifts of attention are mediated by the analog movement of a locus of attention.

The experiments described were performed in accordance with the Declaration of Helsinki.

Experiment 1: Cued-Response Task

In this experiment we examined the effect of target eccentricity on the performance of normal subjects as well as subjects with stroke.

2. Methods

Subjects—Subjects included 11 normal controls (mean age = 58.8 years; range = 43 to 72 years), 3 Right Hemisphere subjects with Neglect (RH+N; mean age = 60.7 years; range = 52 to 71 years), 2 Right Hemisphere subjects without Neglect (RH; mean age = 43 years; range = 30 to 56 years); and 7 Left Hemisphere subjects (LH; mean age = 52.3 years; range = 31 to 65 years). All subjects had normal or corrected-to-normal vision, and full visual fields confirmed by confrontational testing. The presence of neglect was determined using four measures developed in our laboratory involving line cancellation, line bisection, tactile extinction, and visual extinction. Neglect was judged to be absent in subjects who performed within the normal range on all measures and present in subjects who performed outside this range on one or more of these measures.

Visual stimuli were presented using a Macintosh desktop computer and color monitor, and reaction times (RTs) were recorded by depressions of a telegraph key with the right hand for normal subjects or the ipsilesional hand for brain lesion subjects.

Stimuli consisted of three outline boxes, one presented at the midline, and one presented to either side of the midline at 2° or 8° of visual arc, depending on the condition being tested. Stimulus presentation was blocked so that only one cue/target eccentricity was tested at a time. The outline boxes remained on the screen throughout the experiment. Each trial consisted of the brightening of one of the three boxes (CUE) followed by the presentation of a solid diamond

shape (TARGET) within one of the peripheral boxes (See Figure 1). Cue duration was 300 msec, with target presentation following cue onset at a stimulus onset asynchrony (SOA) of 50, 150, 500, or 1000 msec. Targets persisted until subjects responded. The CUE correctly indicated the location of the target on 72% of the trials (VALID trials), and incorrectly indicated target location (INVALID trials) on 20% of the trials (e.g., the cue appears in the rightmost box and the target in the leftmost box). On the remaining 8% of the trials, the CUE appeared at the middle location, giving no location information since targets never appeared in the middle box (NEUTRAL trials). Subjects were instructed to maintain fixation on the central box and to depress the telegraph key as quickly as possible when a target was presented. Trials on which eye movements were detected by an examiner sitting behind the Cathode Ray Tube (CRT) and monitoring eye position or on which the subject failed to respond were repeated at the end of the session. Each subject received 2 sessions of 200 trials each, and each session was preceded by 40 practice trials.

3. Results

The data from the 11 normal subjects was subjected to an analysis of variance (ANOVA) in which eccentricity (2°, 8°), SOA (50, 150, 500, 1000 msec.), cue validity (valid, invalid, neutral) and side of target (right, left) were treated as within subject factors (See Figure 2a). Significant main effects were observed for eccentricity ($F [1,10] = 20.0, p = .001$), validity ($F [1,10] = 16.47, p = .002$) and SOA ($F [1,10] = 91.86, p < .00001$). No effect of side was obtained ($F [1,10] = .003, p = .955$). Normal subjects responded significantly slower to targets at 8° of eccentricity (459.3 msec.) as compared to 2° of eccentricity (416.9 msec.) (post-hoc t-test; $p < .01$). RTs were shortest for valid (406.5 msec) trials whereas RTs for invalid (454.1 msec) and neutral trials (453.8 msec) were similar; post-hoc analysis using t-tests revealed that RTs for valid trials were significantly shorter than the RTs for neutral and invalid trials, which did not differ (both $p < .01$). Finally, there was an inverse relationship between SOA and RT; post-hoc t-tests indicated that RTs at SOAs of 50 (485.3 msec) and 150 msec (468.7 msec) did not differ from each other ($p = .188$) and were significantly slower than RTs at SOAs of 500 msec (413.7 msec) and 1000 msec (384.7 msec) (all $p < .001$). A significant SOA \times validity interaction was obtained ($F [1,10] = 5.13, p = .047$); RTs for invalid and neutral trials were significantly longer than those for valid trials at every SOA (t-tests; all $p < .01$) except 1000 msec ($p = .248$). Second, a marginally significant eccentricity \times validity interaction ($F [1,10] = 4.62, p = .057$) was noted; a post-hoc comparison of RTs for invalidly cued targets at 2° (424.4 msec) and 8° (483.7 msec) was significant ($p < .001$). There was no significant interaction between side \times eccentricity \times validity.

Because the numbers of subjects with right and left-sided brain lesions were relatively small and unequal to controls, contrasts between groups were analyzed using nonparametric measures. In order to characterize the relationship between target eccentricity and validity across patient groups we determined the difference in validity effect (Δ VE) between targets at 2° and targets at 8° for each subject. The Δ VE for left-sided targets for right-hemisphere patients with neglect (mean = 309.22 ms; $SD = \pm 140.61$ ms) was significantly higher than for controls (mean = 42.12 ms; $SD = \pm 44.71$ ms), right hemisphere patients without neglect (mean = 41.8 ms, $SD = \pm 43.6$ ms), or left hemisphere patients (mean = 9.53 ms, $SD = 26.2$ ms) (Mann Whitney; $p = .001$), indicating that the increase in response time attributable to target eccentricity was greater for left-sided targets in right hemisphere patients with neglect than in other patient groups. By contrast, the Δ VE for right-sided targets for right-hemisphere patients with neglect (mean = 69.01 ms, $SD = 7.8$ ms) did not differ significantly from controls or other patient groups (Mann Whitney; $p = .115$). Excluding control subjects and looking only at patient groups (RH+N, RH, & LH), the Δ VE for left-sided targets for right-hemisphere patients with neglect was again significantly higher than for all other patient groups for left-sided targets (Mann Whitney; $p = .009$), but not for right-sided targets (Mann Whitney; $p = .145$), further

indicating that subjects with neglect have prolonged RTs for invalid left-sided targets as a function of eccentricity compared to other brain-lesioned subjects.

There were very few misses by any groups of subjects (<3% of trials), yielding insufficient data for error analysis.

4. Discussion

A number of informative results emerged from this experiment. First, there was a significant overall effect of target validity for all subject groups, reproducing the basic pattern of results reported by other investigators (Posner et al., 1978; Farah et al., 1989; D'Erme et al., 1992; Egly et al., 1994; Friedrich et al., 1998; Bartolomeo et al., 2001). Second, analysis of normal subject performance revealed a strong trend for an interaction between target validity and eccentricity, supporting prior studies that demonstrated that target distance impacts the speed with which normal adults can make rapid covert shifts of attention (Tsal, 1983; Henderson and Macquistan, 1993).

This latter finding of an interaction between validity and eccentricity in normal subjects is consistent with a spotlight model of attention. Subjects are assumed to covertly shift attention initially to the site of the cue; on invalid trials, the appearance of the target on the opposite side is assumed to require that the subject disengage attention from the cued location and shift attention to the site of the target before generating a response. In the 2° condition, subjects move the “spotlight” only 4° of visual angle whereas on the 8° condition the movement is 16°. By the spotlight account, the interaction between validity and eccentricity is thus attributable to the increased distance in the path of attention. However, the saliency map model also adequately explains this finding. Prior data suggest that, in normal subjects, visual targets in the periphery may be less salient than parafoveal targets (Parkhurst et al., 2002). Such a gradient of saliency could account for differences in validity effect as a function of target eccentricity.

Compared to all other groups, subjects with neglect showed a greater effect of eccentricity on the validity effect for left-sided targets, but not for right-sided ones. The validity effect for left-sided targets was an order of magnitude higher (See Appendix 1) for this group than for normal subjects or stroke subjects without neglect, underscoring the difficulty that patients with neglect experienced when attempting to attend to eccentric targets in neglected hemisphere. This finding is consistent with either a spotlight model of analog attention shifts or a model based on saliency maps. By the spotlight account, the movement of attention is slowed as it navigates through contralesional space, resulting in an increased effect of eccentricity on validity in neglected space compared to non-neglected space. In the framework of the saliency map model, targets in contralesional space are less potent attractors of attention than targets in ipsilesional space (Pouget & Driver, 2000; Driver & Vuilleumier 2001). If one assumes a gradient of attention in the neglected hemisphere characterized by decreasing attentional allocation with increasing eccentricity, more laterally situated targets in neglected hemisphere may be even further diminished in their salience, giving rise to an increased validity effect.

EXPERIMENT 2: Presentation of a Central Cue

1. Introduction

Experiment 1 raises an issue that merits further investigation. Posner and colleagues (1984) attributed increased validity effects to invalidly cued target in the neglected hemisphere to a failure to disengage attention from invalidly cued target in the good hemisphere. An unelaborated version of this account does not, however, predict the striking hemisphere × validity × distance interaction described above, because it does not predict that the eccentricity of targets in neglected hemisphere would affect the ability to disengage from invalidly cued targets in intact hemisphere. To determine whether the exaggerated validity × eccentricity

interaction seen in subjects with neglect for neglected hemispace was due to differences in the ability to disengage attention from invalidly cued locations at different eccentricities in intact hemispace, we conducted Experiment 2, in which one patient with neglect and control subjects responded to valid or invalid presentation of a central cue.

2. Method

Subjects included a patient with neglect (JM) who had participated in Experiment 1 as well as seven age-matched normal controls (mean age = 53.6 years; range = 42–72 years). JM was a 59-year old right-handed male who had suffered a right parietal and posterior temporal infarction. He exhibited neglect of the left body and left extrapersonal space on a variety of tasks including line cancellation, line bisection, and visual search (See Figure 3). He had minimal left-sided clumsiness but no weakness, and visual fields were full to confrontation. Testing was performed over a 4-month interval approximately 8–12 months following infarction.

Experimental details were the same as Experiment 1, except that a central fixation point replaced the midline box and became an arrow indicating either the correct location of the subsequent target (VALID trial), an incorrect location (INVALID trial), or no location information by becoming a double-headed arrow (NEUTRAL trial).

3. Results

A comparison of the results for normal subjects and for patient JM on both the centrally cued task and to his prior performance on the peripheral cued-response task is shown (See Figure 4). The data from the 7 normal controls was subjected to an analysis of variance (ANOVA) in which eccentricity (2° and 8°), SOA (50, 150, 500 and 1000 msec.), cue validity (valid, invalid and neutral) and side of target (right, left) were treated as within subject factors. Significant main effects were noted for eccentricity ($F[1,6] = 10.442$; $p = .018$) validity ($F[1,6] = 8.536$; $p = .027$), and a marginally significant effect of SOA was noted ($F[1,6] = 5.772$; $p = .053$). No significant interactions were observed between factors.

For JM a separate univariate ANOVA was performed on individual trial data in which eccentricity, SOA, validity, and side, were treated as fixed factors. All main effects were significant ($p < .001$ in all cases), and a number of significant interactions emerged. Pertinent to our hypotheses, significant interactions were noted for eccentricity \times side ($F[1, 11] = 14.801$; $p < .001$), reflecting a greater effect of target eccentricity for left-sided targets than for targets on the right. A significant side \times validity interaction ($F[2, 11] = 11.176$; $p < .001$), was consistent with the observation that responses to left-sided invalidly cued targets (mean RT = 643.48 msec) were slower than responses to right-sided invalidly cued targets (mean RT = 531.38). Finally, there was a significant three-way interaction between eccentricity \times side \times validity ($F[2, 745] = 3.152$; $p = .043$), consistent with the finding that JM demonstrated prolonged RTs for invalid left-sided targets as a function of target eccentricity. There were insufficient errors (<3% trials) in either control subjects or for JM to pursue error analysis.

4. Discussion

The principal finding from Experiment 2 is that, compared to control subjects, JM demonstrates an exaggerated interaction of validity and eccentricity for contralesional stimuli for both centrally and peripherally cued targets. This result weighs against the notion that eccentricity \times validity interactions are mediated by differential deficits in disengagement from cues in the ipsilesional hemispace. Were this the case, one would have predicted that a centrally cued task would not be affected by variations in target eccentricity. Our results instead suggest that covert shifts of attention, regardless of whether they are oriented by exogenous or endogenous mechanisms, require the movement of a locus of attention through space, and that this process

would be slowed in the affected hemifield of patients with neglect regardless of the nature of the cue.

These findings are consistent with prior data indicating that the validity effect seen in response to endogenously cued stimuli is larger in patients with neglect than normal subjects for contralesional space (Bartolomeo et al., 2001). While a number of prior studies have demonstrated differences in normal subjects for exogenous as compared to endogenous cues (Briand, 1998; Lu and Doshier 2000, Klein and Shore, 2000), other data demonstrate that validity effects can be seen in either condition (Müller and Rabbit, 1989). The results of Experiment 2 are consistent with prior data that indicate that the validity effect seen in response to endogenously cued stimuli is larger in patients with neglect for contralesional space than in normal subjects (Bartolomeo et al., 2001).

Our results indicate that the slowing of attentional shifts that occurs in patients with neglect occurs on the basis of damage to mechanisms that impact both peripherally (Experiment 1) and centrally (Experiment 2) cued targets. While this implies that both exogenous and endogenous mechanisms of attention are affected, one important caveat is that Experiments 1 and 2 may not perfectly delineate these two mechanisms. In Experiment 1 peripheral cues were somewhat (72%) predictive of target location. It is possible that shifts of attention may contain an endogenous component, since subjects could develop expectations regarding the location of targets based on the appearance of cues. Prior investigations that have employed informative peripheral cues demonstrate validity effects that are presumed to be attributable to endogenously generated expectations about target location (Müller and Findlay, 1988). Thus, it may be the case that endogenous mechanisms may be contributing to the exaggerated validity and eccentricity effects observed for neglect patients in Experiment 1. This possibility is itself interesting, because it contrasts with prior studies demonstrating relative preservation of endogenous mechanisms for orienting attention into contralesional space in patients with neglect (Decaix et al., 2002).

5. General Discussion

Normal subjects exhibit an effect of target eccentricity on invalid trials. Additionally, subjects with neglect demonstrate an exaggerated interaction between validity and target eccentricity, but only for targets in their impaired hemispacial field. Further testing with a single illustrative patient demonstrated that the interaction between target eccentricity and validity is present for both exogenously and endogenously cued attention. The performance of neglect patients is quite variable (Hamilton et al., 2008); as a consequence, the results obtained from Experiment 2 must be interpreted with caution. However, taken together, these experiments suggest that neglect is characterized by a deficit in the ability to relocate attention to targets in contralesional hemispaces and that, consistent with the view that attention moves through space in an analog fashion (Schulman, et al, 1979; Tsal, 1983; Henderson and Macquistan, 1993), this deficit varies as a function of target eccentricity. More generally, the hypothesis that neglect is characterized by a slowing of the movement of attention into or toward the contralesional hemispaces is consistent with previous demonstrations that movements of the hands (Heilman et al., 1985; Mattingly et al., 1994) and eyes (Behrmann et al., 2001 & 2002) into the neglected hemispaces are pathologically slowed.

Recent work by Grubb and Reed (2002) is relevant to this claim. In our investigations subjects sat with their heads, bodies and eyes aligned; thus, the hemispaces defined by these coordinate systems were aligned. Grubb and Reed (2002) found that, in normal subjects, rotating the trunk results in increased RTs to invalidly cued targets on the side opposite to the direction of rotation and decreased RTs toward the direction of rotation. Moreover, they noted an interaction between target distance relative to the trunk, and the validity effect. Thus, creating a lateral

bias of attention in which representation of the body is shifted away from visual targets appears to impact the movement of visual attention through space. These findings are consistent with the claim that the locus of attention moves in an analog fashion and that this movement can be affected by manipulation of nonvisual frames of reference.

Because of the relatively small number of subjects and variability in lesion location, our data do not permit one to draw strong inferences regarding the anatomic basis of the effects reported here. A growing body of functional imaging data in humans and single-unit recordings in primates, however, demonstrates that shifts of attention are associated with retinotopic changes in cortical activity in striate and extrastriate areas corresponding to locations to which attention has been oriented (Tootell et al., 1998; Muller et al., 2003; McAdams and Reid, 2005). These visual cortical regions receive extensive projections from multimodal cortical areas, including the parietal lobe, that may modulate visual cortex excitation (Hillyard et al., 2004). Consistent with this notion, prior functional brain imaging data in humans and neurophysiologic data in primates demonstrate that covert shifts of attention are associated with enhancement of neural activity in the parietal lobe (Wurtz and Mohler, 1976; Corbetta et al., 1993).

Computational models based on saliency maps capture many elements of selective visual attention, and can account for the results of the current study. Decreased salience of neglected hemispace may lead to slowing of the reallocation of attention from salient ipsilesional targets to contralesional ones (Nieber & Koch, 1996; Itti & Koch). Computational models based on saliency maps have been used to model validity effects and neglect symptoms. For example, the Selective Attention for Identification Model proposed by Heinke and Humphreys (2003) uses both space-based and object-based constraints to determine visual selection and generate translation invariant recognition. This model reproduces a number of attentional phenomena, including the validity effect, and algorithms that replicate many aspects of neglect. However, to our knowledge, no computational model to date has been used to predict or explain the interaction between target eccentricity and validity that we observed in patients with neglect.

Our finding that the validity effect is exaggerated in neglect patients sheds further light on the phenomenology of this syndrome. Posner and colleagues proposed that patients with neglect have a diminished ability to disengage attention from a location to which it had been invalidly cued in order to initiate a shift to a new location (Posner et al, 1984). An unelaborated version of this disengage deficit does not adequately explain the interaction between eccentricity and validity for contralesional targets seen in patients with neglect. It is possible that an ipsilesional disengage deficit could interact with other mechanisms of visual attention to contribute the current results. For example, eccentricity-dependent deficits in contralesional saliency coding may lead to increasingly inefficient driving of attention toward contralesional targets. This could, in turn, have differential effects on the ability to overcome an ipsilesional disengagement deficit. However, while such a model would incorporate the notion of a disengage deficit into the current results, the differential effect of eccentricity would primarily be a function of contralesional saliency coding rather than ipsilesional disengagement. While the current data do not permit one to draw conclusions about the presence or absence of a disengage deficit in neglect, they do suggest that such a deficit alone does not offer a complete account.

A comparison of JM's performance on centrally and peripherally cued tasks further informs our understanding of the mechanisms that underlie covert shifts of attention. Several studies have demonstrated that a cued-response task with central cues leads to smaller validity effects in normal subjects (Briand, 1998; Lu and Doshier, 2000). On this basis, some have suggested that there may be a fundamental difference in the attentional resources allocated in response to exogenous and endogenous cues (Klein and Shore, 2000). While patient JM did show less difference in RT between validly and invalidly cued targets in contralesional space in a centrally cued task compared to a peripherally cued task, he demonstrated a robust effect of target

eccentricity in contralesional space regardless of whether the cue preceding target stimuli was central or peripheral. This finding suggests that at least some of the mechanisms that subserve shifts of attention through the visual field are common to both endogenously and exogenously cued attention, even though dissociable neural substrates may underlie other aspects of processing of these two types of attentional cues.

Finally, one potential objection to our findings is that subjects may have directed eye movements toward valid and invalid cues and to targets. A recent study investigating the activity-distribution model of visual attention shifts demonstrated that the presence of previously undetected saccadic eye movements can have dramatic effects on reaction times (Turk-Browne and Pratt, 2005). We attempted to control for eye movements by visually monitoring subjects' fixation performance of the task and discarding trials in which any eye movement was observed but may have been unable to detect small eye movements (e.g., less than 1°). The finding that the interaction of validity and target eccentricity is present for neglect subjects even at Inter stimulus intervals (ISI) of 50 ms, an interval too brief to permit visually guided saccadic eye movements (Schall and Thompson, 1999), suggests that inadequate fixation does not explain our findings. Also relevant in this context is the fact that Horowitz and colleagues (2006) recently used a cued-response paradigm to demonstrate that microsaccadic eye movements that occur during fixation may not accurately reflect the direction of shifts of attention.

References

- Bartolomeo P, Siéhoff E, Decaix C. Modulating the attentional bias in unilateral neglect: the effects of the strategic set. *Experimental Brain Research* 2001;137:432–444.
- Behrmann M, Ghiselli-Crippa T, Dimatteo I. Impaired initiation but not execution of contralesional saccades in hemispatial neglect. *Behavioral Neurology* 2002;13:39–60. [PubMed: 12118150]
- Brand KA. Feature integration and spatial attention: more evidence of a dissociation between endogenous and exogenous orienting. *Journal of Experimental Psychology: Human Perception and Performance* 1998;24:1243–1256.
- Carrasco M, Giordano AM, McElree B. Attention speeds processing across eccentricity: feature and conjunction searches. *Vision Research* 2006;46:2028–2040. [PubMed: 16481020]
- Cheal ML, Lyon D. Attention effects on form discrimination at different eccentricities. *Quarterly Journal of Experimental Psychology* 1989;41:719–46. [PubMed: 2587796]
- Corbetta M, Miezin FM, Shulman GL, Petersen SE. A PET study of visuospatial attention. *Journal of Neuroscience* 1993;13:1202–1226. [PubMed: 8441008]
- Crick F. The function of the thalamic reticular complex: The searchlight hypothesis. *Proceedings of the National Academy of Science* 1984;81:4586–4590.
- Decaix C, Sieroff E, Bartolomeo P. How voluntary is 'voluntary' orienting of attention? *Cortex* 2002;38:841–845.
- D'érme P, Robertson I, Bartolomeo P, Daniele A, Gainotti G. Early rightward orienting of attention on simple reaction time performance in patients with left-sided neglect. *Neuropsychologia* 1992;30:989–1000. [PubMed: 1470341]
- Driver J, Vuilleumier P. Perceptual awareness and its loss in unilateral neglect and extinction. *Cognition* 2001;79:39–88. [PubMed: 11164023]
- Egley R, Driver J, Rafal RD. Shifting visual attention between objects and locations: evidence from normal and parietal lesion subjects. *Journal of Experimental Psychology: General* 1994;123:161–177. [PubMed: 8014611]
- Eriksen, CW.; Schultz, DW. Temporal factors in visual information processing: A tutorial review. In: Requin, J., editor. *Attention and Performance VII*. New York: Academic Press; 1978.
- Farah MJ, Wong AB, Monheit MA, Morrow LA. Parietal lobe mechanisms of spatial attention: modality-specific or supramodal? *Neuropsychologia* 1989;27:461–470. [PubMed: 2733819]

- Friedrich FJ, Egley R, Rafal RD, Beck D. Spatial attention deficits in humans: a comparison of superior parietal and temporo-parietal junction lesions. *Neuropsychologia* 1998;12:193–207.
- Grubb JD, Reed CL. Trunk orientation induces neglect-like lateral biases in covert attention. *Psychological Science* 2002;13:553–556. [PubMed: 12430841]
- Hamilton RH, Coslett HB, Buxbaum LJ, Whyte J, Farne A, Frassinetti F, Ferraro MK. Inconsistency of performance on neglect subtype tests following acute right hemisphere stroke. *Journal of the International Neuropsychological Society* 2008;14:23–32. [PubMed: 18078528]
- Henderson JM, Macquistan AD. The spatial distribution of attention following an exogenous cue. *Perception and Psychophysics* 1993;53:221–230. [PubMed: 8433920]
- Heilman KD, Bowers D, Coslett HB, Whelan H, Watson RT. Directional hypokinesia: prolonged reaction times for leftward movements in patients with right hemisphere lesions and neglect. *Neurology* 1985;35:855–859. [PubMed: 4000486]
- Heilman KM, Valenstein E, Watson RT. Neglect and related disorders. *Seminars in Neurology* 2000;20:463–470. [PubMed: 11149702]
- Heinke D, Humphreys GW. Attention, spatial representation, and visual neglect: simulating emergent attention and spatial memory in the Selective Attention for Identification Model (SAIM). *Psychological Review* 2003;110:29–87. [PubMed: 12529057]
- Hillyard, SA.; Di Russo, F.; Martinez, A. The imaging of visual attention. In: Kanwisher, N.; Duncan, J., editors. *Attention and Performance XX: Functional Neuroimaging of Visual cognition*. Oxford: Oxford University; 2004. p. 381–388.
- Horowitz TS, Fine EM, Fencsik DE, Yurgenson S, Wolfe JM. Fixational eye movements are not an index of covert attention. *Psychological Science* 2006;18:356–363. [PubMed: 17470262]
- Itti L, Koch C. Computational modeling of visual attention. *Nature Reviews Neuroscience* 2001;2:194–203.
- Klein, RM.; Shore, DI. Relations among modes of visual orienting. In: Monsell, S.; Driver, J., editors. *Attention and Performance XVII: Control of Cognitive Processes*. Cambridge: MIT Press; 2000. p. 195–208.
- Koch C, Ullman S. Shifts in selective visual attention: towards the underlying neural circuitry. *Human Neurobiology* 1985;4:219–227. [PubMed: 3836989]
- Kwak HW, Dagenbach D, Egeth H. Further evidence for a time-independent shift of the focus of attention. *Perception and Psychophysics* 1991;51:455–464. [PubMed: 1594435]
- LaBerge D, Carlson RL, Williams JK, Bunney BG. Shifting attention in visual space: Tests of moving spotlight models versus an activity-distribution model. *Journal of Experimental Psychology: Human Perception and Performance* 1997;23:1380–1392. [PubMed: 9336958]
- Lu Z, Doshier BA. Spatial attention: Different mechanisms for central and peripheral temporal precues? *Journal of Experimental Psychology: Human Perception and Performance* 2000;26:1534–1548. [PubMed: 11039483]
- Mattingly JB, Phillips JG, Bradshaw JL. Impairments of movement execution in unilateral neglect: a kinematic analysis of directional bradykinesia. *Neuropsychologia* 1994;32:1111–1134. [PubMed: 7991078]
- Muller NG, Bartelt OA, Donner TH, Villringer A, Brandt SA. A physiological correlate of the “Zoom Lens” of visual attention. *Journal of Neuroscience* 2003;23:3561–5. [PubMed: 12736325]
- Müller HJ, Findlay JM. The effect of visual attention on peripheral discrimination thresholds in single and multiple element displays. *Acta Psychologica* 1988;69:129–155. [PubMed: 3245479]
- Müller HJ, Rabbit PMA. Reflexive and voluntary orienting of visual attention: Time course of activation and resistance to interruption. *Journal of Experimental Psychology: Human Perception and Performance* 1989;15:315–330. [PubMed: 2525601]
- Niebur E, Koch C. Control of selective visual attention: modeling the ‘where’ pathway. *Neural Information Processing Systems* 1996;8:802–808.
- Parkhurst D, Law K, Niebur E. Modeling the role of salience in the allocation of overt visual attention. *Vision Research* 2002;42:107–123. [PubMed: 11804636]
- Posner, MI. *Chronometric explorations of mind*. Lawrence Erlbaum; Hillsdale NJ: 1978.

- Posner MI. Orienting of attention. The 7th Sir F.C. Bartlett Lecture. Quarterly Journal of Experimental Psychology 1980;32:3–25. [PubMed: 7367577]
- Posner MI, Snyder CRR, Davidson BJ. Attention and the detection of signals. Journal of Experimental Psychology: General 1980;109:160–174.
- Posner, MI.; Cohen, Y. Components of visual orienting. In: Bouma; Bouwhuis, DG., editors. Attention and performance X. Hove, UK: Lawrence Erlbaum; 1984.
- Pouget A, Driver J. Relating unilateral neglect to the neural coding of space. Current Opinion in Neurobiology 2000;10:242–249. [PubMed: 10753799]
- Pratt J, Quilty L. Examining the activity-distribution model of visual attention with exogenous cues and targets. The Quarterly Journal of Experimental Psychology 2002;55:627–641. [PubMed: 12047063]
- Remington R, Pierce L. Moving attention: Evidence for time-invariant shifts of visual selective attention. Perception and Psychophysics 1984;35:393–399. [PubMed: 6739275]
- Sagi D, Julesz B. Fast noninertial shifts of attention. Spatial Vision 1985;2:141–149. [PubMed: 3940055]
- Schall JD, Thompson KG. Neural selection and control of visually guided eye movements. Annual Review of Neuroscience 1999;22:241–59.
- Schulman GL, Remington RW, McLean JP. Moving attention through visual space. Journal of Experimental Psychology: Human Perception and Performance 1979;5:522–526. [PubMed: 528957]
- Sperling G, Weichselgartner E. Episodic theory of the dynamics of spatial attention. Psychology Review 1995;102:503–532.
- Tootell RB, Hadjikhani M, Hall EK, Marrett S, Vanduffel W, Vaughan JT, Dale AM. The retinotopy of visual spatial attention. Neuron 1998;21:1409–1422. [PubMed: 9883733]
- Turk-Browne NB, Pratt J. Attending to eye movements and retinal eccentricity: evidence for the activity distribution model of attention reconsidered. Journal of Experimental Psychology Human Perception and Performance 2005;31:1061–1066. [PubMed: 16262498]
- Tsal Y. Movements of attention across the visual field. Journal of Experimental Psychology: Human Perception and Performance 1983;9:523–530. [PubMed: 6224890]
- Wurtz RH, Mohler CW. Enhancement of visual responses in monkey striate cortex and frontal eye fields. Journal of Neurophysiology 1976;39:766–772. [PubMed: 823304]

Appendix 1

Control (n=11)	2 Degrees				8 Degrees			
ISI (ms):	50	150	500	1000	50	150	500	1000
Left Targets								
Valid	439.5	419.0	350.5	360.8	476.6	442.4	368.5	372.7
Invalid	466.9	446.2	398.5	382.4	510.2	496.3	494.3	452.2
Neutral	484.9	490.2	402.5	354.2	537.3	534.3	445.5	393.2
Right Targets								
Valid	422.7	423.6	375.1	370.1	478.9	451.7	370.3	381.2
Invalid	476.1	415.0	438.9	371.2	530.1	515.9	471.5	399.2
Neutral	461.0	486.2	400.7	368.2	539.0	503.7	447.6	410.9

Right Hemisphere with Neglect (n=3)								
ISI (ms):	50	150	500	1000	50	150	500	1000
Left Targets								
Valid	631.0	582.2	467.6	437.6	771.2	768.7	553.2	615.9
Invalid	900.5	896.0	723.0	549.2	1537.0	1504.6	1070.0	784.4

Right Hemisphere with Neglect (n=3)								
ISI (ms):	50	150	500	1000	50	150	500	1000
Neutral	1011.3	813.0	615.4	524.4	1579.4	1261.2	932.5	799.3
Right Targets								
Valid	601.8	556.2	453.6	412.2	716.4	635.5	505.9	475.5
Invalid	608.9	539.5	496.1	421.8	747.1	692.2	617.1	595.4
Neutral	553.9	568.6	493.1	471.9	695.7	730.8	633.1	493.2

Right Hemisphere without Neglect (n=2)								
ISI (ms):	50	150	500	1000	50	150	500	1000
Left Targets								
Valid	495.0	427.1	370.0	359.1	491.0	456.1	324.5	349.6
Invalid	527.5	466.9	429.1	395.5	535.1	575.0	435.1	411.4
Neutral	520.4	513.7	414.6	380.2	671.1	526.6	469.1	362.3
Right Targets								
Valid	485.4	448.1	352.4	343.0	545.1	468.4	354.7	368.6
Invalid	542.4	415.0	390.2	400.5	528.4	447.2	438.8	431.3
Neutral	546.9	425.1	348.0	346.3	509.0	495.8	304.9	332.0

Left Hemisphere (n=7)								
ISI (ms):	50	150	500	1000	50	150	500	1000
Left Targets								
Valid	545.3	501.5	394.4	382.0	611.1	568.8	456.9	463.1
Invalid	581.5	541.8	444.1	411.8	641.8	639.2	541.7	471.2
Neutral	589.1	584.3	458.3	406.2	694.1	632.9	515.9	483.7
Right Targets								
Valid	542.4	514.1	416.5	407.1	682.8	598.5	494.4	484.5
Invalid	582.4	535.1	455.2	405.3	747.1	683.8	587.2	481.5
Neutral	585.8	606.5	512.8	410.8	740.8	663.2	543.6	501.3

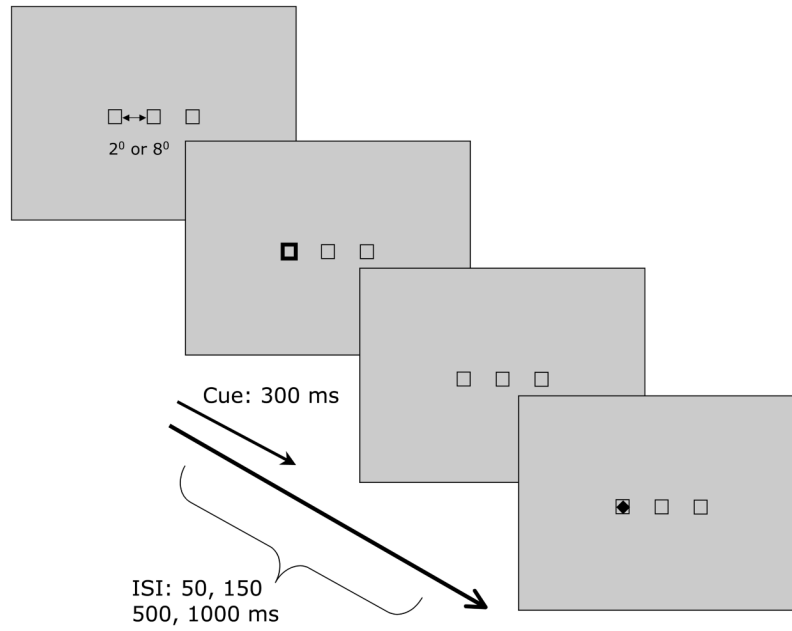


Figure 1.

Summary of the cued-response task used in Experiment 1. Subjects fixated at the central location. Each trial consisted of the brightening of one of three boxes (cue) followed by the presentation of a diamond shape (target) inside a peripheral box. The cue duration was 300 msec; target presentation followed a stimulus onset asynchrony (SOA) of 50, 150, 500, or 1000 msec. Cues either correctly indicated the location of the target (valid trials), incorrectly indicated target location (invalid trials), or appeared at the middle location (neutral trials). In each block, lateral targets were either 2° or 8° of visual arc away from the central fixation point.

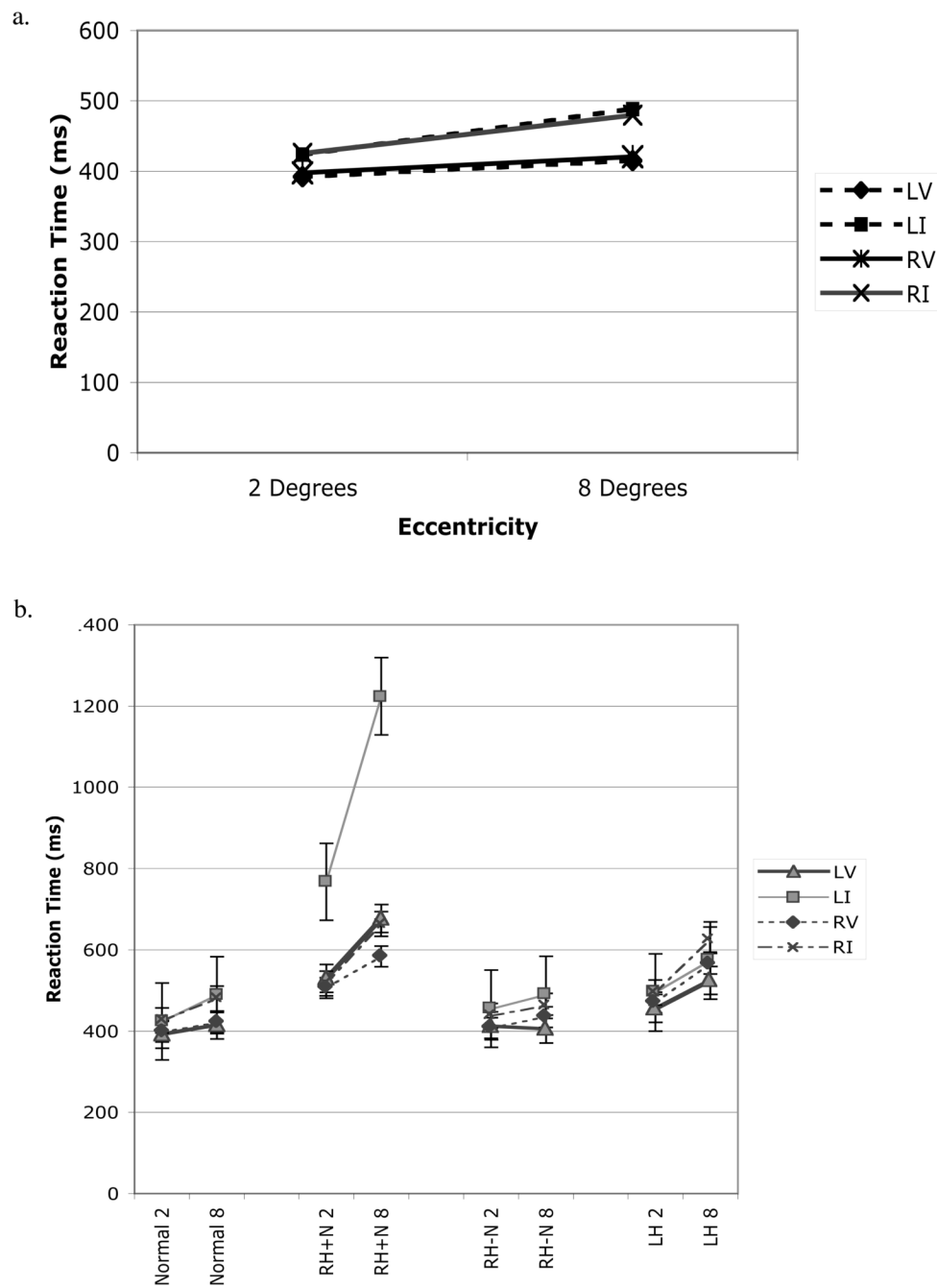


Figure 2.

2a. Results for normal controls (Normal, n=11) on a peripheral cued-response task in which the eccentricity of targets was either 2° or 8°.

2b. Comparison of normal controls (Normal, n=11), right hemisphere patients with neglect (RH+N, n=3), right hemisphere patients without neglect (RH-N, n=2), and left hemisphere patients (LH, n=7) on the same task.

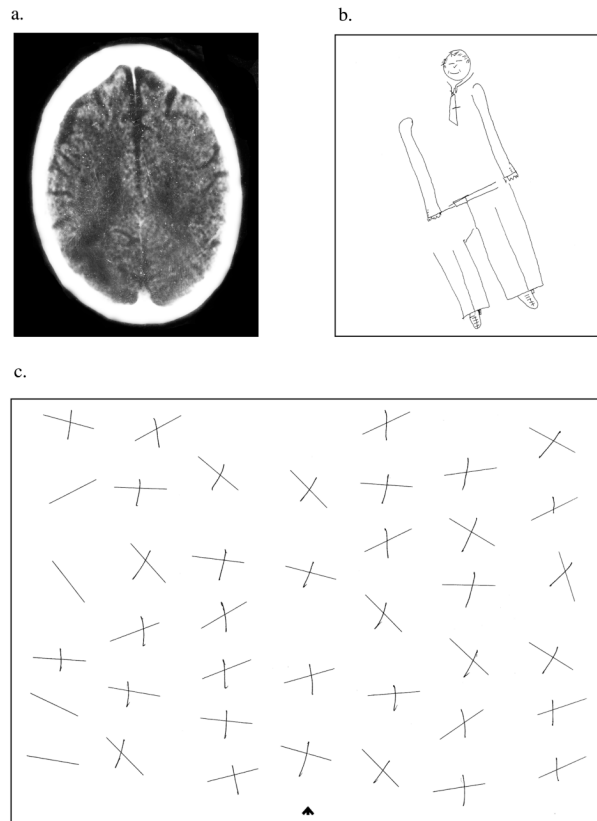


Figure 3.

Clinical data for JM, a patient with a right parietotemporal stroke and neglect.

3a. Axial slice from JM's head CT, demonstrating a right parietal infarct (seen in radiologic view).

3b. JM's performance on a drawing task in which he has been asked to generate the figure of a man.

3c. JM's performance on a line cancellation task, demonstrating neglect for left-sided targets.

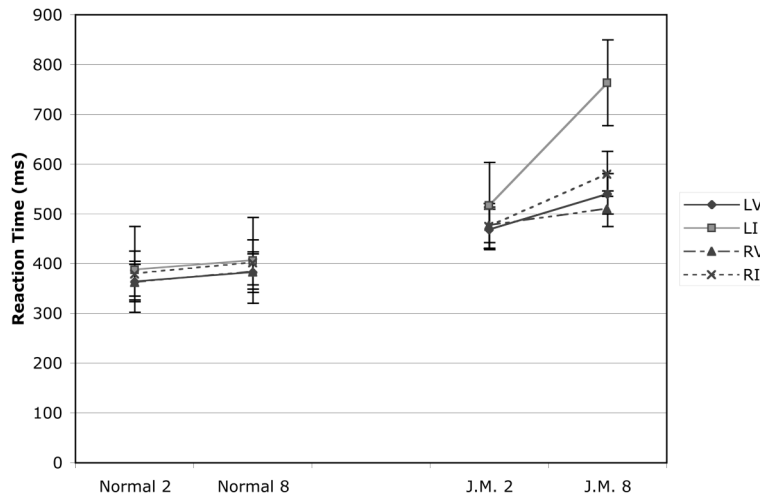


Figure 4. Results for JM and seven age-matched controls on a centrally cued response task with peripheral targets at 2° or 8° of eccentricity.