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Attentional control parameters following parietal-lobe damage: evidence from normal subjects

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Abstract

Attentional control involves the factors, or cognitive parameters, that determine which environmental inputs receive attention and which do not. Cognitive studies of attentional control have highlighted two general classes of control parameters, bottom-up (data driven or exogenous) parameters and top-down (goal driven or endogenous) parameters. Which of these control parameters is affected following parietal-lobe damage? In parietal-damaged patients, it is possible that a disorder in one control parameter (e.g. goal driven) would appear as a disorder in another parameter (e.g. data driven). To investigate the control parameters that might be affected in parietal patients, we simulated neglect in normal participants by disrupting data-driven information processing. When half of a computer monitor was degraded by translucent tracing paper while normal participants performed a cued spatial attention task (Experiment 1), the normal participants showed a pattern of results similar to patients with unilateral parietal-lobe damage—the so-called "disengage deficit." This pattern of results replicated when neutral attentional cues were included in the experiment (Experiment 2). However, the disengage deficit was not simulated in normal participants with predictive central symbolic cues (Experiment 3) or predictive peripheral cues (Experiment 4). Because perceptual degradation influences data-driven attentional control parameters, we suggest that these control parameters may be disrupted following parietal-lobe damage.

Keywords: Parietal-lobe damage; Attentional control; Symbolic cue

Of the multiple cortical and subcortical areas that participate in the control of spatial attention, perhaps none has been studied as extensively as the posterior parietal region. Damage to the parietal region (especially the right parietal region) in humans results in a profound attentional impairment referred to as neglect. The performance of neglect patients has provided important insights about the operation and mechanisms of attention. Patients with neglect present with a variety of symptoms (e.g. see Bisiach & Vallar, 1988; Heilman, Watson, & Valenstein, 1993; Heilman, Watson, & Valenstein, 2000; Rafal & Robertson, 1995; Rafal, 2000), including the failure to pay attention to stimuli falling on the side of space opposite to the lesion (the contralesional side); a patient with damage to the right parietal lobe may fail to eat food on the left half of the plate or may not read words on the left half of a page. Neglect patients often do not make head or eye movements in the contralesional direction. These failures to respond to contralesional stimuli are not due to sensory deficits (e.g. a visual scotoma or hemianopia). As a patient recovers and the neglect becomes less severe, patients can process a single stimulus presented in the contralesional visual field. However, these recovering patients continue to show a subtle attentional impairment known as extinction: when two stimuli are presented simultaneously in opposite visual fields, patients will extinguish, or fail to notice, the stimulus in the contralesional field. In other words, extinction patients exhibit neglect of contralesional stimuli only in the presence of ipsilateral stimuli. Although many of these characteristics can follow damage to other brain regions (e.g. frontal lobe areas and subcortical areas such as the pulvinar), our focus is on neglect and extinction phenomena that follow damage to parietal lobe areas. Hence, we use the terms 'neglect' and 'ex-

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tinction' as shorthand for 'neglect (or extinction) following parietal-lobe damage.'

Different interpretations of the attentional deficits in neglect and the mechanisms of those deficits have been offered. For example, neglect has been interpreted as a difficulty with visuospatial attention (see Driver, 1998; Posner, Walker, Friedrich, & Rafal, 1984; Rafal, 2000; Rafal & Robertson, 1995), with object-based attention (see Behrmann & Moscovitch, 1994; Behrmann & Tipper, 1994; Egly, Driver, & Rafal, 1994), and with orienting attention to temporal events (Husain, Shapiro, Martin, & Kennard, 1997), suggesting that the parietal lobe plays a role in many attentional phenomena. In addition to these varieties of attentional impairments, many theorists have proposed different mechanisms to explain the performance of neglect patients. Kinsbourne (1993) proposed that competition between the two cerebral hemispheres could explain neglect; because the damaged hemisphere cannot compete with the intact hemisphere, attention is biased toward the ipsilesional visual field, which is controlled by the intact hemisphere (also see Cohen, Romero, Servan-Schreiber, & Farah, 1994; Làdavas, 1990; Làdavas, Petronio, & Umiltà, 1990; Làdavas, Umiltà, Ziani, Brogi, & Minarini, 1993). Posner et al. (1984) proposed that the parietal lobes are involved in the disengagement of attention; patients with unilateral parietal damage have difficulty disengaging spatial attention from the ipsilesional visual field, making it difficult to direct attention to contralesional stimuli.

Attempts to understand the mechanisms that are damaged in parietal-damaged patients have not always been informed by information processing theories of normal attentional processes (see Posner et al., 1984, for an exception). Information processing, or cognitive, studies of attention in neurologically normal participants focus on two important issues, the control of attention and the effects of attention (see Luck & Vecera, 2002; Vecera & Luck, 2002). Studies of attentional control involve isolating the factors that determine which inputs receive attention and which do not. That is, how does a viewer attend to a red circle when it appears in a scene filled with red squares and green circles? Studies of the effects of attention involve specifying the processing differences between attended and unattended items. We focus on the control of visuospatial attention in neglect following recent theoretical developments on this topic. We ask: What attentional control parameters (i.e. factors) are impaired in parietal-damaged patients?

To address this question, we must consider the possible sources of attentional control because there are different parameters or processes that can influence where attention is directed. Two general sources of attentional control are goal-driven (also called conceptually-driven, top down, or endogenous) sources that arise from the current behavioral goals and data-driven (also called bottom up or exogenous) sources that arise from sensory stimuli present in a scene (Klein, Kingstone, & Pontefract, 1992; Yantis, 1998). We refer to these two sources broadly as "attentional control parameters" because the factors that control attention are parameterized by involving (1) the type of factor affecting attention (top-down or bottom-up, for example) and (2) the value of that factor. The different terms (e.g. endogenous versus top-down) typically are used in different paradigms. For example, in peripheral cuing paradigms, attentional control is discussed in terms of exogenous and endogenous processes, whereas in visual search, attentional control may be discussed in terms of bottom-up and top-down processes. Because we intend to discuss attentional control broadly, we will tend to use the most inclusive terms to discuss control parameters; that is, instead of using a term that typically is tied to a paradigm (e.g. exogenous), we will use a more general term (e.g. bottom-up or data-driven). The terms we have chosen to use will not affect the interpretation of our results.

Recently, Desimone and Duncan (1995) have proposed a "biased competition" account of attention that integrates both goal-driven and data-driven attentional control. The biased competition account was developed to explain visual search in which a subject looks for a target among distractors. The target searched for is held in working memory and guides attention in a goal-directed manner. The image being searched provides sensory information that guides attention in a data-driven manner. The multiple objects present in the scene compete for limited attentional resources. The goal-driven attentional signal serves to bias this competition to favor objects that are similar to the target item. The biased competition account provides a useful conceptual framework for studies of overt orienting (Trappenberg, Dorris, Munoz, & Klein, 2001), visual search, visuospatial attention (Desimone & Duncan, 1995), and object-based attention (Vecera, 2000).

Applying the biased competition account to neglect patients suggests at least two possible processes that might be disrupted in these patients. Attentional impairments following parietal damage could arise from an inability to use goal-directed, or endogenous, attention to orient to the contralesional visual field (the neglected or extinguished field). Patients may have damage to the representation of the contralesional space, and they may be unable to use this representation to control the allocation of attention in a goal-directed manner. Or, the attentional impairments following parietal damage could arise from an inability of perceptual inputs to control attention in a data-driven, or exogenous, manner. Events in the contralesional field may fail to engage attention as quickly or effectively as events in the ipsilesional field (e.g. Làdavas, 1990; Làdavas et al., 1990, 1993).

Although either goal-driven or data-driven control sources might be disrupted in parietal-damaged patients, it is difficult to disentangle the effects of these control parameters in patients. Damage to one control parameter may appear as an impairment in the other parameter. For example, parietal-damaged patients make fewer exploratory eye movements into the contralesional field than in to the ipsilesional field. This suggests damage to goal-driven control parameters which are responsible for visual search via eye movements. If data-driven parameters were disrupted, however, patients might fail to make exploratory eye movements because the

eye movement system does not receive sufficient inputs from the contralesional field to program and execute eye movements. Thus, the patients' behavior could be the result of damage to either control parameter.

We have attempted to overcome the difficulty of separating goal-driven and data-driven control by studying neurologically normal research participants and selectively manipulating attentional control parameters. Simulation studies, in which normal participants are made to perform as a brain-damaged patient performs, have been used to investigate various neuropsychological syndromes. Such studies have been useful in resolving theoretical accounts of various syndromes, including visual form agnosia (Vecera & Gilds, 1998), neglect (Farah, Monheit, & Wallace, 1991; Graves & Jones, 1992; Michel, Pisella, Halligan, Luauté, Rode, Boisson & Rossetti, 2003), and aphasia (Miyake, Carpenter, & Just, 1994). These examples demonstrate that simulating neuropsychological disorders in normal participants can be important for addressing theoretical issues in neuropsychology.

In the present studies, we report four studies in which we simulate the results of parietal-damaged patients in normal participants to investigate the attentional control parameters that might be affected in these patients. Participants performed a simple visuospatial attention task, Posner's cued detection task (Posner, 1980; Posner & Cohen, 1984; Posner, Snyder, & Davidson, 1980). In this task, depicted in Fig. 1 with peripheral cues, attention is summoned to a location by a peripheral cue (a flicker in the visual periphery). A small target then appears, and participants press a key as soon as they detect the onset of the target. The target is either validly cued (the target appears at the cued location) or invalidly cued (the target appears at the uncued location). Neurologically normal participants are faster to detect validly-cued targets than invalidly-cued targets at short cue-to-target intervals. If validly cued- and invalidly-cued targets appear equally (i.e. 50% valid and 50% invalid), this task appears to involve exogenous, or data-driven, spatial attention (see Jonides, 1981; Klein et al., 1992; Müller & Rabbit, 1989), and no goal-directed control parameters need to be involved. In contrast, when central, symbolic cues (e.g. arrows) are used to direct attention to a peripheral location, endogenous, or goal-driven, parameters are required: such symbolic cues typically must predict the upcoming target's location to encourage participants to direct attention from the cue (Jonides, 1981 p. 803, but see Tipples, 2002), and such symbolic cues can be interfered with by a concurrent memory load (Jonides, 1981). Mixtures of the two control parameters are possible, as when peripheral cues predict the upcoming target's location on a majority of trials; such situations would tap both data-driven and goal-driven control processes.

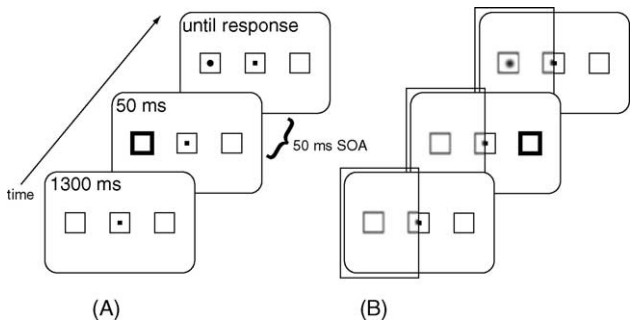

Fig. 1. Order of events in the spatial precuing task. (A) No screen degradation (validly-cued target). (B) An example of degrading the left side of a display (invalidly-cued target, with target appearing on degraded side).

When neglect/extinction patients perform Posner's cued detection task, they show an attentional asymmetry referred to as a "disengage deficit": they are slower to detect invalidly-cued targets appearing in the contralesional field than targets appearing in the ipsilesional field (Posner et al., 1984). A common interpretation of these results is that the parietal lobe contains the neural circuitry that controls the disengagement of attention from a currently-attended location. Following parietal damage, attention has difficulty disengaging from the ipsilesional field (e.g. Posner et al., 1984; Rafal, 2000). Therefore, when the precue appears in the ipsilesional field and the target appears in the contralesional field, it takes longer for attention to be disengaged from the precued location; when the precue appears in the contralesional field and the target appears in the ipsilesional field, attention can be disengaged from the contralesional field relatively quickly and easily, allowing the target to be detected rapidly.

Cohen and colleagues (1994) offered an alternative explanation of the "disengage deficit pattern" in neglect patients. By creating a simple neural network model that could perform the cued detection task, Cohen et al. (1994) demonstrated that poor detection of invalidly-cued targets appearing in the contralesional field could be produced by asymmetric damage to a bilateral attentional system. If one "hemisphere" in the model was damaged, this hemisphere could not compete strongly with the intact hemisphere, which made it difficult for the model to detect targets appearing in its contralesional field. The model did not contain an attentional disengager, indicating that the disengage deficit pattern could be an emergent property of a damaged attentional network that did not contain an explicit disengage process. Because of the potentially confusing terminology, we use "disengage deficit pattern" to refer to the pattern of reaction time (RT) results exhibited by patients with parietal-lobe damage; this pattern of results should be distinguished from a disengage process because, as we discuss next, the disengage pattern could arise from damage to a process that is not a disengage process per se.

Based on Cohen et al.'s (1994) interpretation of neglect and extinction, we hypothesized that data-driven control parameters might result in the disengage deficit pattern of results. This hypothesis allows us to integrate theoretical models of extinction (Cohen et al., 1994) with current theoretical perspectives on attentional control (Luck & Vecera, 2002; Vecera & Luck, 2002; Yantis, 1998). In the following experiments, we manipulated data-driven (exogenous) attentional control parameters by varying the perceptual quality of the

displays while participants performed Posner's cued detection task. In Experiment 1, one-half of the computer's monitor was occluded with translucent tracing paper which blurred the image from that side of the monitor (see Farah et al. (1991) for this procedure). If the disengage deficit pattern is caused by an inability of contralesional events to adequately capture attention in a data-driven manner, then we should be able to simulate the disengage deficit pattern of results in normal participants. Specifically, when the clear (intact) field is cued, then targets in the degraded ("neglected") field should be detected relatively slowly; in contrast, when the degraded field is precued and the target appears in the clear field, the target should be detected relatively quickly. We should note that effect sizes from our normal participants might be smaller overall than those from parietal patients because of baseline RT differences between these groups; normal participants may show smaller effects because their RTs are faster than those of parietal-damaged patients.

1. Experiment 1: unilateral "lesion"

1.1. Method

1.1.1. Participants

The participants were 12 University of Iowa undergraduates who received partial course credit for their time. All had normal or corrected vision.

1.1.2. Stimuli

All stimuli were viewed from a distance of 80 cm. The stimuli were three small boxes, each measuring 1.2° of visual angle on each side and arranged side-by-side across the computer monitor. All of the displays consisted of black lines on a white background. The two peripheral boxes were located 4.3° to the left and right of the middle (fixation) box. The peripheral boxes were cued by thickening the border of the box by 13 pixels, or approximately 0.5° of visual angle (see Fig. 1). The target consisted of a small black circle which was 0.35° in diameter and appeared in one of the two peripheral boxes.

One side of the display was degraded by covering it with standard translucent tracing paper purchased from an art supply store. Half of the participants received left-side degradation and half received right-side degradation. We used tracing paper for degradation instead of an electronic manipulation (e.g. reducing contrast) because tracing paper has been shown to successfully simulate aspects of performance following parietal-lobe damage (Farah et al., 1991).

1.1.3. Procedure

An individual trial began with the three boxes present on the screen for 1300 ms, and participants were instructed to maintain fixation on the center box. The cue was then presented for 50 ms in one of the two peripheral boxes and was replaced immediately by a target, producing a 50 ms stimulus-onset asynchrony (SOA). We used a short SOA in the present experiments because parietal-damaged patients show the largest impairments at short SOAs (Losier & Klein, 2001). The target remained visible until the participant responded. Participants responded by pressing the spacebar on a standard keyboard. All displays were presented on a Macintosh computer with a 15 in. monitor and viewed from a distance of 80 cm. Each participant received eight blocks of experimental trials, with each block containing 80 trials; 20% of these trials were catch trials in which no target appeared. The participants were asked to withhold responses to the catch trials. In trials in which a target appeared, the cue was valid in 50% of trials and invalid in the other 50% of trials. Targets appeared equally to the left and right of fixation.

1.2. Results and discussion

Because the median can produce biased estimates of central tendency under some experimental conditions, such as those we use in later experiments (see Miller, 1988), mean reaction times were computed for each condition. RTs greater than two standard deviations above the mean were excluded from the analyses. This trimming excluded less than 5% of the data.[1]

The mean RTs were analyzed with a two-factor ANOVA with target field (clear versus degraded) and trial type (validly cued versus invalidly cued) as factors. The mean RTs appear in Fig. 2, which shows a larger validity effect in the degraded ("neglected") field than in the clear field. This pattern of results replicates the "disengage deficit" pattern observed in patients with parietal damage (Posner et al., 1984).

A detailed analysis supported the disengage pattern that is evident in Fig. 2. There was a main effect of target field, with faster RTs to targets appearing in the clear field (314.8 ms) than to those appearing in the degraded field (324.0 ms), $F(1, 11) = 7.3$, $p < 0.05$. There was also a main effect for trial type; participants detected validly-cued targets faster than invalidly-cued targets (307.2 ms versus 331.6 ms, respectively), $F(1, 11) = 37.1$, $p < 0.0001$. Most important, there was a statistically significant interaction between target field and trial type, $F(1, 11) = 23.5$, $p < 0.001$, indicating that the difference between validly- and invalidly-cued targets was larger in the degraded field than in the clear field. Planned pairwise comparisons verified the presence of a spatial precuing effect in both the clear and degraded fields. Validly-cued targets were detected faster than invalidly-cued targets in the clear field, $t(11) = 3.3$, $p < 0.01$, and in the degraded field, $t(11) = 9.2$, $p < 0.0001$.

We performed an additional analysis to examine the disengage pattern across the visual fields to determine if this pattern was asymmetric. Neglect typically follows damage

[1] We should note that the analyses of all of our experiments were qualitatively similar when median RTs were computed for each condition and when means were used with a 2.5 standard deviation exclusion; see Ratcliff (1993) for the suggestion that analysis of RT data be confirmed using different measures of central tendency.

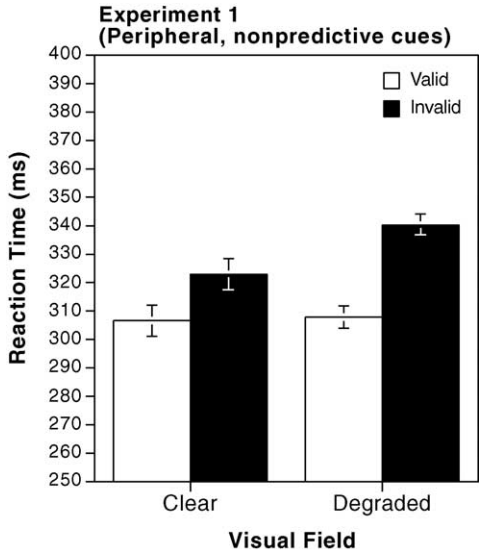

Fig. 2. Results from Experiment 1. Neurologically normal participants are disproportionately slower to detect invalidly-cued targets appearing in the degraded field than those appearing in the clear field. Neglect and extinction patients show a qualitatively similar pattern of results, the so-called "disengage pattern" (error bars are 95% within-subject confidence intervals on the valid vs. invalid comparisons).

to the right parietal lobe, but extinction can follow damage to both the left and right parietal lobes (see Bisiach & Vallar, 1988). If our simulation procedure mimics neglect, then we might expect to find a larger disengage pattern following degradation of the left visual field than of the right visual field. If our procedure mimics extinction, then we might expect a similar disengage pattern across the visual fields.

We analyzed the results separately for participants whose left visual field was degraded and those whose right visual field was degraded. The means for these conditions appear in Table 1. As is evident from the results, there was a disengage pattern for both groups, and the magnitude of this pattern was similar for left and right visual field degradation. These observations were corroborated by a three-way ANOVA, with target field (clear versus degraded), trial type (valid versus invalid), and side of degradation (left versus right visual field) as factors. RTs were similar for detecting targets in a degraded left visual field (319.3 ms) and a degraded right visual field (319.5 ms), F (1, 10) < 1. The main effect of cue type remained significant, F (1, 10) = 41.0, $p < 0.0001$, with faster RTs to detect validly-cued targets than invalidly-cued targets. There was also a main effect for target field, with faster responses to detect targets in the clear field than in the degraded field, F (1, 10) = 6.8, $p < 0.03$. The only two-way interaction that was significant was the two-way interaction between cue type and target field, F (1, 10) = 22.3, $p < 0.0005$, which replicated the disengage pattern observed when the degraded field was not included as a factor. There was no three-way interaction, F (1, 10) < 1, indicating that the disengage deficit pattern was similar following both left and right visual field degradation.

The neurologically normal participants in Experiment 1 show the same qualitative pattern of results as patients with unilateral parietal lobe lesions: invalidly-cued targets that appear in the degraded field are detected more slowly than invalidly-cued targets that appear in the intact (clear) field. In parietal-damaged patients, this pattern has been attributed to a damaged attentional "disengager." However, the normal participants in Experiment 1 presumably did not have damage to their attentional processes, making damage to an attentional disengage process unlikely. A more straightforward interpretation of the present results, and, similarly, of the results from parietal-damaged patients, is that the data-driven capture of attention has been weakened in the degraded field. In the normal participants, targets appearing in the degraded field are harder to detect; therefore, when attention is summoned to the clear field and the target appears in the degraded field, the target has a difficult time summoning attention away from its current location in the clear field. When attention is summoned to the degraded field and the target appears in the clear field, the target can be detected quickly for two reasons. First, attention may be summoned to the degraded field only weakly because the cue appears degraded; second, the target, which appears in the good field, may have a relatively easy time summoning attention away from the degraded field. The same analysis can explain the pattern of results from parietal patients (see Cohen et al., 1994). Of course, we acknowledge that the present pattern of results is consistent with a disengage deficit and that it will be difficult to disentangle a disengager account from a data-driven capture account within a single experiment. Nevertheless, our results show that there are alternative accounts for the spatial cuing data from parietal lobe patients.

There are several issues raised by the results of Experiment 1 that deserve discussion. As one reviewer noted, our degradation manipulation not only degrades the target's representation, but also degrades the cue's representation. The consequence of this fact is that the disengage pattern might be the result of a degraded cue representation, which makes it difficult to detect nondegraded targets that are preceded by degraded cues, an interpretation consistent with a disengage mechanism. We admit that unilateral degradation affects both

Table 1
Mean RTs from Experiment 1 analyzed by visual field (standard errors appear in parentheses)

	Left field degraded		Right field degraded	
	Clear field	Degraded field	Clear field	Degraded field
Validly-cued target	310.5 (13.9)	312.0 (17.0)	303.9 (13.5)	304.9 (10.5)
Invalidly-cued target	317.4 (17.0)	337.3 (17.0)	326.9 (12.0)	342.4 (9.8)

the cue and the target, but we should point out that the same is true for peripherally-presented cues that are presented to parietal patients. Indeed, the fact that both cue and target are degraded when presented contralesionally in parietal patients might partially explain why the disengage deficit is small or absent when central cues (e.g. arrows presented at fixation) are used to direct spatial attention (see Losier & Klein, 2001). We return to this issue in Experiment 3, in which we attempt to simulate the performance of parietal patients using central arrow cues.

Another relevant point is that we found a very small difference between validly-cued targets appearing in the clear field (306.6 ms) and those appearing in the degraded field (307.9 ms), $t(11) < 1$. This appears at odds with an explanation that appeals to damaged data-driven attention in parietal patients: if parietal patients, and our simulated patients, had problems with data-driven attentional parameters, should not responses be slower to validly-cued targets in the degraded field than those in the clear field? Not necessarily. Data-driven information accumulation might occur extremely rapidly at a validly-cued location because (1) attention has been directed to this location and (2) attention can enhance the degraded stimulus (or reduce the perceptual noise or uncertainty associated with this stimulus). This would be particularly true in younger, neurologically normal participants who could rapidly deploy attention to the cued region, preventing us from observing a difference between valid trials in the degraded and clear fields because of a reaction time floor effect. Indeed, in our following experiments, the experiments with slower overall RTs are more likely to show a difference between valid trials on the degraded versus clear fields. Parietal patients, who tend to take longer to respond than control subjects, tend to show a moderate difference between validly-cued targets in the contra- and ipsilesional fields (see Losier & Klein, 2001; Morrow & Ratcliff, 1988; Posner, Cohen, & Rafal, 1982; Posner, Inhoff, Friedrich, & Cohen, 1987). Further, the extent of the damage (real or simulated) could affect the RT differences to the validly-cued targets, such that greater damage impairs not only data-driven attentional control but also perceptual-level processes. Our current results could reflect the fact that our simulated parietal damage was 'mild' damage. Greater degradation could produce a difference in valid trials in the degraded and clear fields and maintain the 'disengage deficit' pattern of results. Of course, greater degradation could encourage our normal participants to engage in compensatory behaviors, such as willfully monitoring the degraded field for targets to prevent missing a target. The same compensatory behaviors do not appear to operate in neglect and extinction patients, suggesting that such compensatory processes might be bootstrapped by data-driven attentional processes damaged in patients.

Finally, our simulated parietal-lobe damage was not affected by the visual field that was degraded; a similar disengage pattern was observed for left and right visual field degradation. The lack of a visual field difference mimics observations from parietal patients who exhibit extinction, as extinction can occur after either left or right parietal damage (Bisiach & Vallar, 1988; but, because neglect more often follows right parietal damage, there might also exist an asymmetry in extinction). There is, however, a puzzle regarding parietal-damaged patients: neglect more often occurs after damage to the right parietal lobe than to the left parietal lobe. How would such an asymmetry fit with a data-driven account of parietal-lobe attention processes? It might be the case that the right parietal lobe is more sensitive to data-driven control parameters than the left parietal lobe, resulting in greater attentional impairments following damage to the right parietal lobe. Also, the asymmetry between left and right parietal damage would be compounded based on the right parietal lobe's representation of both contra- and ipsilateral space (Corbetta, Miezin, Shulman, & Petersen, 1993). In sum, the asymmetries that arise following parietal-lobe damage are not inconsistent with the view that the parietal lobes participate in the data-driven control of attention.

Having demonstrated that a unilateral stimulus degradation can produce data qualitatively similar to those from parietal-damaged patients, we now explore how well this simulated parietal damage follows the performance of patients. Because our stimulus degradation affects the data-driven control of attention, if degradation allows us to simulate other results from parietal patients, the better support we have for the parietal lobes being involved in data-driven attentional control. In Experiment 2, we ask if we can replicate the results of Experiment 1. We also ask if the disengage pattern we have observed holds when neutrally-cued targets are compared to invalidly-cued targets, as has been reported in some studies (Posner et al., 1984). Posner and colleagues (1984) demonstrated that extinction patients showed similar RTs for invalidly-cued targets and neutrally-cued targets, with slower responses in the contralesional field than in the ipsilesional field. This finding has been interpreted as suggesting that disengaging attention—either from an invalidly-cued location or a neutrally-cued location—determines the responses of extinction patients. Experiment 2 was identical to Experiment 1, with the exception of added neutral trials in which a centrally-located cue preceded the target. If a data-driven manipulation (i.e. unilateral degradation) can produce results that appear to support an attentional disengager, then we should see a larger discrepancy between neutrally-cued targets appearing in the degraded and clear fields than for validly-cued targets appearing in these fields. That is, there should be a cue type (valid versus neutral) by field interaction, consistent with data reported by Posner et al. (1984) to support a disengage deficit.

2. Experiment 2: neutral cues

2.1. Method

2.1.1. Participants

The participants were 12 University of Iowa undergraduates who received partial course credit for their time. All

had normal or corrected vision, and none had participated in Experiment 1.

2.1.2. Stimuli and procedure

The stimuli and procedure were identical to those used in Experiment 1, except for the addition of a neutral cue condition, in which the central box was cued in the same manner as the peripheral boxes. Each cue type (valid, neutral, and invalid) appeared equally often. Peripheral cues were again unpredictive of the targets location; half of the peripheral cues were valid and half were invalid. There were 96 trials per block in which a target appeared, and participants viewed six blocks of trials with a short rest between each block. In each block, there were 12 catch trials (four for each of the three possible locations in which the cue could appear).

2.2. Results and discussion

As in Experiment 1, after excluding RTs greater than two standard deviations above the condition mean, mean RTs were computed for each participant in each of the six conditions. The trimming procedure eliminated less than 5% of the data. These mean RTs were then analyzed with a two-factor ANOVA with target field (clear versus degraded) and trial type (valid, neutral, or invalid cue) as factors. The mean RTs appear in Fig. 3. Inspection of the valid and invalid trials reveals a replication of the disengage pattern observed in Experiment 1. There was a larger validity effect in the degraded visual field than in the clear visual field.

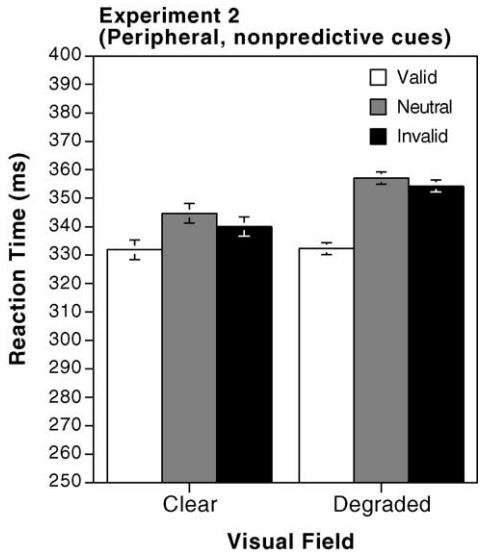

Fig. 3. Results from Experiment 2. Neurologically normal participants produce the disengage pattern of RT results when neutrally-cued targets are added to the experiment. Importantly, both neutrally- and invalidly-cued targets show the "disengage" pattern, in which RTs are slower in the degraded visual field than in the clear visual field, further replicating results from parietal-damaged patients (see text for additional discussion) (error bars are 95% within-subject confidence intervals for the cue type effects within each visual field condition).

A detailed analysis supported the disengage pattern that is evident in Fig. 3. There was a main effect of target field, with faster RTs to targets appearing in the clear field (338.9 ms) than to those appearing in the degraded field (347.9 ms), $F(1, 11) = 10.9$, $p < 0.01$. There was also a main effect for trial type; participants detected targets differently based on the preceding cue (332.1 ms for valid trials, 351.0 ms for neutral trials, and 347.1 ms for invalid trials), $F(2, 22) = 29.9$, $p < 0.0001$. Most important, there was a statistically significant interaction between target field and trial type, $F(2, 22) = 7.1$, $p < 0.005$, indicating that the RTs to targets in the clear and degraded fields differed for the three cue types. We explored this interaction further with two additional analyses, one comparing valid and invalid trials to verify the pattern observed in Experiment 1 and another comparing valid trials to neutral trials.

To verify the results from Experiment 1, we compared valid and invalid trials using a two-factor ANOVA, with cue type (valid versus invalid) and target field (clear versus degraded) as factors. There was a main effect of target field, with shorter responses to targets in the clear field (336.0 ms) than to those in the degraded field (343.3 ms), $F(1, 11) = 5.7$, $p < 0.04$. There was also a main effect of cue type, with shorter RTs on valid trials (332.1 ms) than on invalid trials (347.1 ms), $F(1, 11) = 31.4$, $p < 0.0005$. Finally, these two factors interacted, $F(1, 11) = 15.5$, $p < 0.005$, reflecting the larger validity effect (valid versus invalid RTs) for targets appearing in the degraded field than those appearing in the clear field. These findings replicate those from Experiment 1.

Next, to determine if a disengage pattern can be produced in a neutral cue condition, we also compared valid trials to neutral trials using a two-factor ANOVA with cue type (valid versus neutral) and target field (clear versus degraded as factors). Recall that Posner et al. (1984) reported that both neutral trials and invalid trials produced a disengage pattern (i.e. slower RTs to contralesional targets than to ipsilesional targets following both neutral and invalid cues). Our analyses indicate that we simulated the disengage deficit with neutral cues. There was a main effect of target field, with significantly faster responses to targets in the clear field (338.3 ms) than to targets in the degraded field (344.7 ms), $F(1, 11) = 5.6$, $p < 0.04$. There was a main effect of trial type, with faster RTs on valid trials than on neutral trials (332.1 ms versus 350.1 ms, respectively), $F(1, 11) = 54.7$, $p < 0.0001$. Finally these two factors interacted, $F(1, 11) = 7.4$, $p < 0.02$, indicating that degradation had a larger effect for neutrally-cued targets than for validly-cued targets. This interaction is qualitatively similar to that observed when valid trials are compared with invalid trials, reflecting a disengage pattern. Further, both neutrally- and invalidly-cued targets were detected slower in the degraded field than the intact field, and this visual field effect did not differ significantly between neutrally- and invalidly-cued targets.

The present findings demonstrate, importantly, that we can replicate the results of Experiment 1, even when the experimental parameters are changed to include neutral trials. Also,

we have demonstrated that centrally-presented neutral cues, which offer no information regarding the upcoming location of the target, can produce a disengage pattern of results. This latter result is important for several reasons. First, it shows that impairing data-driven attentional control can produce a disengage deficit pattern of results. Thus, one does not need to hypothesize damage to an attentional disengage process to explain these results; an impairment in data-driven control parameters can produce this disengage deficit pattern of response times, suggesting that patients with parietal damage could have impairments with their data-driven control parameters. Second, slower responses to neutrally-cued targets in the degraded field than to those in the clear field indicate that our degradation manipulation indeed affects bottom-up or exogenous attentional capture. As we discussed with Experiment 1, the absence of a difference between validly-cued targets appearing in the degraded and clear fields is difficult to interpret because the spatial cue may have allowed information to accumulate extremely rapidly these validly-cued locations. The neutral cue condition provides a purer measure of the effect of unilateral degradation because (1) attention is maintained at fixation and (2) the target appeared in each field equally following a neutral cue. Both of these factors should have reduced or eliminated the effect that the cue would have on information accumulation (or attentional capture) in the periphery, thereby providing us with a better measure of the effectiveness of our degradation manipulation.

We should acknowledge that neutral cues are problematic in attentional cuing studies: there is no perfect neutral cue. Some studies with neurologically normal participants us a no-cue condition for a neutral condition; this neutral condition is not ideal because it lacks the alerting provided by the cue. Other studies use a centrally-presented cue, as we have done; this neutral condition is not ideal because it cues attention to a non-target location, much as an invalid cue does, making this type of neutral cue more similar to an invalid cue than to a true neutral cue. Irrespective of these issues, the important result in the present study is that our neurologically normal participants show a pattern similar to parietal-damaged patients.

Having simulated a disengage deficit in Experiments 1 and 2 under a variety of conditions, we now investigate the role of predictive, centrally-presented symbolic cues on the disengage deficit. Such endogenous attentional cues appear to involve different control parameters than peripheral exogenous cues (see Jonides, 1981; Klein et al., 1992; Luck & Vecera, 2002; Müller & Rabbit, 1989; Vecera & Luck, 2002; Yantis, 1998), as we discussed in the introduction. Further, patients with parietal damage appear to show a smaller disengage deficit when tested with highly predictive symbolic cues (see Losier & Klein, 2001 for a review). One interpretation of this smaller disengage deficit is that parietal patients might be able to orient attention strategically based on the predictive nature of symbolic cues, thereby reducing the disengage pattern of results. Similarly, pure endogenous (central) cues might allow our neurologically intact participants to strategically orient attention based on the cue and thereby minimize the effects of unilateral degradation.

3. Experiment 3: predictive symbolic cues

Experiment 3 involved a unilateral screen degradation identical to that used in Experiments 1 and 2. Participants in the current study, however, viewed centrally presented arrow cues that pointed to one of the peripheral locations. These arrow cues were predictive of the upcoming target's location.

3.1. Method

3.1.1. Participants

The participants were 12 University of Iowa undergraduates who received partial course credit for their time. All had normal or corrected vision, and none had participated in the previous experiments.

3.1.2. Stimuli and procedure

The stimuli and procedure were similar to those used in Experiment 1, except the cues were centrally-presented symbolic arrows that were, overall, 1.1° of visual angle long and 0.5° tall. The arrow head was a filled triangle that was 0.8° wide and 0.5° tall. The arrow's tail was a 2 pixel-wide line that was approximately 0.3° long. The arrow cue was presented for 100 ms, because such centrally-presented cues require more time to orient attention from than do peripheral cues (see Müller & Rabbit, 1989). Because central, symbolic cues direct attention in a more goal-driven, endogenous manner (Jonides, 1981; Klein et al., 1992), the cues were now predictive regarding the target's location. On 75% of trials, the target was validly cued, and on 25% of trials the target was invalidly cued.

3.2. Results and discussion

As in the previous experiments, mean reaction times were again computed after excluding RTs that fell more than two standard deviations above the condition mean. Trimming eliminated less than 5% of the data. These mean RTs were then analyzed with a two-factor ANOVA with target field (clear versus degraded) and trial type (valid or invalid cue) as factors. The mean RTs appear in Fig. 4. Interestingly, inspection of Fig. 4 reveals that the disengage pattern observed in Experiments 1 and 2 was not replicated with predictive symbolic cues: the difference between responses to validly- and invalidly-cued targets was approximately equal in both the clear and degraded visual fields.

A detailed analysis supported the lack of a disengage pattern. There was no main effect of target field, with similar RTs to targets appearing in the clear field (301.7 ms) and the degraded field (300.3 ms), $F(1, 11) < 1$. There was a highly-significant main effect for trial type; participants detected validly-cued targets faster than invalidly-cued targets

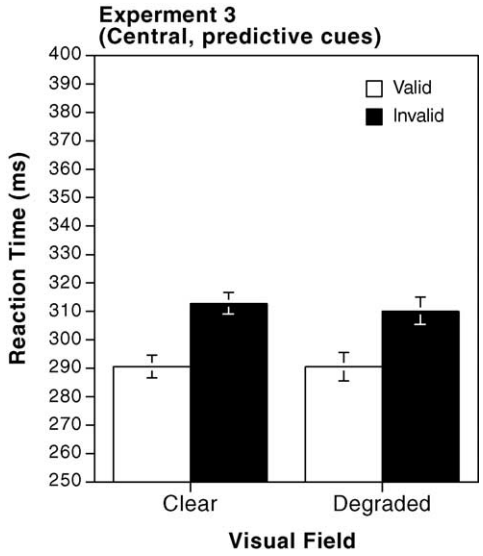

Fig. 4. Results from Experiment 3. Similar to parietal-damaged patients, neurologically normal participants do not show the "disengage" pattern of RTs when attention is directed with highly predictive symbolic cues that are presented at fixation (error bars are 95% within-subject confidence intervals on the valid vs. invalid comparisons).

(290.6 ms versus 311.3 ms, respectively), F (1, 11) = 88.2, $p < 0.001$. These two factors did not interact, F (1, 11) < 1, indicating that the difference between validly- and invalidly-cued targets was not statistically different between the degraded and clear fields. The cuing effects (invalid minus valid RT) were 22.0 ms for the clear field and 19.4 ms for the degraded field. As in Experiments 1 and 2, planned pairwise comparisons verified the presence of a spatial precuing effect in both the clear and degraded fields. Validly-cued targets were detected faster than invalidly-cued targets in the clear field, $t(11) = 6.1$, $p < 0.0001$, and in the degraded field, $t(11) = 4.3$, $p < 0.005$.

The present results are straightforward: unlike the previous two experiments, there was no disengage pattern when attention was oriented from predictive peripheral (endogenous) cues. These results are consistent with the results from parietal patients, who tend to show a small disengage deficit when endogenous cues are used. Presumably, when cues are highly predictive of the target's location, goal-driven control parameters can compensate for the degraded data-driven inputs. A related explanation is that data- and goal-driven attentional control might depend on different anatomical and functional systems, allowing these two forms of control to be dissociated in parietal patients and in our intact participants.

Our findings in Experiment 3 can speak to several issues that are currently unresolved in studies of orienting following parietal-lobe damage. The general issue is why parietal patients appear to show a smaller disengage pattern when endogenous (central arrow) cues are used than when exogenous (peripheral flash) cues are used. A recent review of the 'disengage deficit' suggests two possibilities (Losier & Klein, 2001): first, cuing effects (valid versus invalid RTs) are often smaller for endogenous cues than for exogenous cues, and a smaller disengage deficit might appear reduced for endogenous cues because this baseline effect is reduced. Second, as discussed by Losier & Klein (2001), most of the data from parietal patients that show a diminished disengage deficit come from one publication (Nagel-Leiby, Buchtel, & Welch, 1990), and this study did not use peripheral boxes to mark the locations in which the targets would appear. It is conceivable that failing to mark the targets' locations with a placeholder object prevents attention from becoming strongly engaged at the cued location; consequently, on invalid trials, it is much easier to disengage attention from the cued location, and a smaller disengage deficit is produced.

Our results speak to the smaller disengage pattern exhibited by parietal-damaged patients. The overall cuing effects (valid versus invalid) in Experiment 3 are of similar magnitude to the cuing effects in Experiments 1 and 2 (24.7 ms for Experiment 3 and 14.3 ms and 22.7 ms for Experiments 1 and 2, respectively). Consequently, the smaller disengage pattern following central predictive cues may not be due to a smaller cuing effect, at least in our data. Also, we observed a reduced disengage pattern in Experiment 3, even when placeholders were used to mark the locations of the peripheral targets. Thus, the absence of peripheral placeholders does not appear to explain the smaller disengage pattern that arises when central predictive cues are used.

We interpret the results of Experiment 3 as being consistent with other theoretical views (Jonides, 1981; Klein et al., 1992; Klein & Shore, 2000; Luck & Vecera, 2002; Müller & Rabbit, 1989) in suggesting that endogenous attentional orienting (tapped by central predictive cues) is at least partially dissociable from exogenous attentional orienting (tapped by unpredictive peripheral cues). We hypothesize that the predictive nature of endogenous cues (goal-driven attentional parameters) allows our participants, and perhaps parietal patients, to compensate partially for the degraded exogenous cues (data-driven attentional parameters). Again, as with the previous experiments, we can interpret the pattern of results of our participants without appealing to a damaged attentional 'disengager.' We would argue that the same logic applies to parietal-damaged patients: the results from parietal patients can be explained without appealing to an attentional disengager.

There is one final pattern of results from parietal-damaged patients that is relevant to our present data: parietal patients appear to show a large disengage deficit when predictive peripheral cues are used to orient attention (e.g. Friedrich, Egly, Rafal, & Beck, 1998; Morrow & Ratcliff, 1988; Petersen, Robinson, & Currie, 1989; Posner et al., 1984). These cues contain both data-driven (exogenous) and goal-driven (endogenous) components. Because predictive peripheral cues (with both data- and goal-driven components) show a larger disengage deficit pattern than predictive central cues (with only goal-driven components), the attentional impairment in parietal patients appears to be restricted to data-driven (exogenous) control parameters.

In our final experiment, we investigate the role of predictive, peripherally-presented cues on the disengage deficit pattern. Parietal patients might be unable to use the predictive nature of the cue to orient attention because the goal-driven (endogenous) system receives inputs from the data-driven (exogenous) system; the damaged data-driven system might impair the ability of the goal-driven system to take cue predictability into account. Parietal patients may be able to orient attention from central predictive cues because these cues do not fully engage the data-driven system, thereby minimizing the orienting impairments that typically follow parietal damage. By extension, our neurologically normal participants might be unable to strategically orient attention from predictive peripheral cues because of the degraded data-driven inputs. However, it is also possible that our neurologically normal participants may not have as degraded data-driven inputs as parietal patients, which would allow our participants to partially engage the goal-driven system and minimize the effects of the unilateral degradation.

4. Experiment 4: predictive peripheral cues

4.1. Method

4.1.1. Participants

The participants were 12 University of Iowa undergraduates who received partial course credit for their time. Again, all had normal or corrected vision, and none had participated in the previous experiments.

4.1.2. Stimuli and procedure

The stimuli and procedure were identical to those used in Experiment 1, except the cues were now predictive peripheral cues: the cues predicted the target's location on 75% of trials (validly-cued targets) and did not predict the target's location on 25% of trials (invalidly-cued targets). All timing parameters were identical to those in Experiment 1.

4.2. Results and discussion

As in the previous experiments, mean reaction times were computed for each participant in each of the four conditions, and means that were more than two standard deviations above the mean were excluded from the analyses. Trimming excluded less than 5% of the data. These mean RTs were then analyzed with a two-factor ANOVA with target field (clear versus degraded) and trial type (valid or invalid cue) as factors. The mean RTs appear in Fig. 5. The results replicate those of Experiment 3: the disengage pattern observed in Experiments 1 and 2 was not replicated with predictive cues, even when these cues were peripheral. As is clear from Fig. 5, the difference between responses to validly and invalidly-cued targets was approximately equal in both the clear and degraded visual fields.

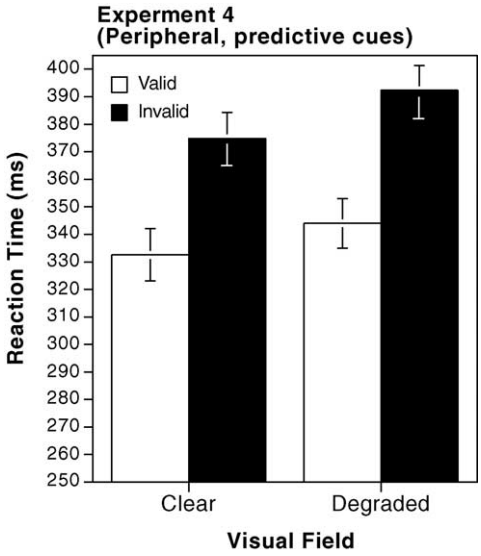

Fig. 5. Results from Experiment 4. Unlike parietal-damaged patients, neurologically normal participants do not show the "disengage" pattern of RTs when attention is directed with highly predictive peripheral cues. Normal participants appear able to engage goal-driven attentional control processes to overcome the effects of unilateral stimulus degradation. Parietal-damaged patients may be unable to engage these goal-driven processes because of the degraded input (error bars are 95% within-subject confidence intervals on the valid vs. invalid comparisons).

As in Experiment 3, the lack of a disengage pattern was supported by statistical analyses. There was a marginal main effect of target field, with faster RTs to targets appearing in the clear field (353.7 ms) than to those appearing in the degraded field (368.2 ms), $F\ (1,\ 11) = 3.3$, $p < 0.10$. There was also a highly-significant main effect for trial type, with faster RTs to detect validly-cued targets (338.3 ms) than to detect invalidly-cued targets (383.6 ms), $F\ (1,\ 11) = 40.4$, $p < 0.0001$. Target field and trial type did not interact, however, $F\ (1,\ 11) < 1$, indicating that the difference between validly-cued and invalidly-cued targets was similar in the degraded and clear fields. The cuing effects (invalid minus valid RT) were 42.3 ms for the clear field and 48.4 ms for the degraded field. As in the previous experiments, planned pairwise comparisons verified the presence of a spatial precuing effect in both the clear and degraded fields. Validly-cued targets were detected faster than invalidly-cued targets in the clear field, $t(11) = 4.9$, $p < 0.0005$, and in the degraded field, $t(11) = 5.9$, $p < 0.0001$.

The present results appear to run counter to those from parietal patients, who show a strong disengage pattern when cued with predictive peripheral cues. The difference between the results of Experiment 4 and the results from parietal patients suggests that in patients, the impairment in data-driven attentional control prevents goal-driven parameters from overcoming the data-driven impairments. Our neurologically normal participants appear to have sufficiently intact data-driven parameters to permit goal-driven parameters to compensate for the unilateral degradation. This interpretation of Experiment 4 leads to two testable predictions which

extend beyond the scope of the present paper. First, when tested with predictive peripheral cues, parietal-damaged patients with relatively minor data-driven attentional impairments should show a smaller disengage pattern than parietal patients with more severe data-driven impairments. Second, in normal participants, a greater unilateral degradation might produce a larger disengage pattern than that observed in the current experiment. Of course, as we noted earlier, greater unilateral degradation could induce normal participants to orient attention strategically using goal-driven control parameters to minimize the effects of the unilateral degradation.

5. General discussion

The results from two experiments suggest that data-driven attentional control parameters may be damaged in patients with neglect and extinction. We replicated a pattern of results obtained with parietal-damaged patients, the disengage deficit pattern, in neurologically normal participants. When a single side of a display was degraded, our participants were slower to detect invalidly-cued targets appearing in the degraded field than to detect an invalidly-cued target appearing in the clear field. Because our participants are unlikely to have an attentional impairment, the simplest explanation of our results is that stimuli in the degraded (i.e. extinguished) field do not capture visuospatial attention as effectively as stimuli in the clear field. Because this difficulty with data-driven capture occurs for only one field, the degraded field is unable to compete adequately with the clear field for attentional processing. As a result, invalidly-cued targets in the degraded field are detected slower than invalidly-cued targets in the clear field. This same "disengage" pattern is simulated in neurologically normal participants when neutral cues are added, indicating that our results are replicable under other experimental conditions. However, the disengage pattern is absent in our participants when predictive central (symbolic) cues or predictive peripheral cues are presented. In these last two experiments, goal-driven parameters, tapped by the predictive cues, may minimize or abolish the effects of unilateral stimulus degradation.

Before discussing the theoretical implications of our results, there are several issues that should be discussed. The first issue for discussion, which we discussed earlier, is that main effect of degradation in some of our experiments revealed no significant difference between targets appearing in the clear field and those appearing in the degraded field (Experiments 3 and 4, although Experiment 4 exhibited a marginal effect of degradation). To examine the effect of target field across the four experiments, we combined these experiments into a three-way analysis, with cue type (valid versus invalid) and target field (clear versus degraded) as within-subjects factors and experiment as a between-subjects factor. Although there were differences across the individual experiments, the main effect of target field remains statistically significant across the four experiments, with faster RTs to detect targets in the clear field (326.5 ms) than in the degraded field (333.9 ms), $F\ (1,\ 44) = 7.2,\ p < 0.01$. Further, target field only interacted with cue type, $F\ (1,\ 44) = 7.4,\ p < 0.01$, reflecting the large disengage pattern evident in Experiments 1 and 2. Target field did not interact with experiment, $F\ (3,\ 44) = 1.4,\ p > 0.20$, and there was no three-way interaction, $F\ (3,\ 44) = 1.9,\ p > 0.14$.

On a related point, we should note that when the experiments are combined, there remains no difference between validly-cued targets appearing in the clear field (315.4 ms) and those appearing in the degraded field (318.9 ms), $t(47) = 1.2,\ p > 0.20$. Most of the main effects of target field were due to difference in the invalidly-cued trials. Some might argue that this result seems to suggest that our degradation manipulation was insufficient to affect data-driven attentional control. However, we should again point out that the valid-cue condition is not the ideal situation to examine target field differences because summoning attention to the degraded field may enhance processing at this location and decrease RTs (and directing attention to the clear field may affect RTs less because they are already rapid and thus suffer from a floor effect). Of course, the invalid-cue is not ideal for examining target field differences for similar reasons, except that the absence of attention on the degraded side now slows target detection and produces a disengage pattern of results. Perhaps the best condition that we have for examining target field effects in the current set of experiments is the neutral-cue condition in Experiment 2. In this condition, attention is likely to remain at fixation (the cued location), allowing a reasonable baseline measure of sensory effects in the clear and degraded fields. In Experiment 2, we find a large difference between neutrally-cued targets appearing in the clear field (344.7 ms) and neutrally-cued targets appearing in the degraded field (357.1 ms), $t(11) = 3.4,\ p < 0.006$, suggesting that our degradation was sufficient to slow data-driven factors when attention was (roughly) equated between the clear and degraded fields.

A second issue for discussion is the role of fixation position. Although we were cautious to use cue and target timing parameters that would minimize eye movements to the cued location, the presence of a degraded field throughout the experiment could have allowed participants to fixate a location within the degraded field. Shifting fixation to the degraded field would be the natural strategy for improving performance because targets in this field are harder to detect. This fixation-shift strategy could explain why there was little, if any, difference between detecting validly-cued targets in the clear and degraded fields. However, this fixation-shift strategy would not have produced the disengage pattern we have reported. If anything, shifting fixation to the degraded field would dilute a disengage pattern because invalidly-cued targets appearing in the degraded field should be detected relatively quickly because they appear nearer fixation. Future studies that attempt to simulate neglect should bear these issues in mind and strongly consider monitoring eye fixation location to re-

duce the opportunity of missing a disengage pattern because of a secondary strategy.

Another issue concerns the role of goal-driven control parameters in modulating the disengage pattern. Our results demonstrate that the disengage pattern is eliminated in normal participants by adding structure to the cuing task with a predictive cue. In these experiments, 'goal driven' refers to the predictive information carried by the cue; but, goal-driven is also discussed in terms of other mechanisms, such as following task instructions to search for a specific target in a visual search task. For parsimony, we have assumed that different goal-driven control parameters are handled by a system (or systems) outside of parietal-lobe attention areas and that parietal areas are sensitive to data-driven parameters. Failures to see the disengage pattern with predictive central cues in parietal-damaged patients (and in Experiment 3) arise because an intact data-driven system is required to provide inputs to the goal-driven system. Further, our failure to find a disengage pattern in with predictive peripheral cues (Experiment 4) might be due to the fact that our normal participants have intact parietal lobes, which can allow goal-driven parameters to be bootstrapped by the degraded, but not absent, data-driven signals.

An alternative possibility suggested by a reviewer is that parietal-lobe areas might be sensitive to cue predictability, which also would explain why we failed to see a disengage pattern in Experiment 4: our participants had intact parietal lobes and could use cue predictability to direct attention independent of the screen degradation. Consistent with this possibility, parietal-damaged patients do not appear to benefit from the use of predictable cues: these patients show as large of a disengage pattern with predictive peripheral cues as they do with non-informative peripheral cues (see Losier & Klein, 2001). Our current results cannot distinguish between our view of parietal-control processes being primarily data driven and an alternative view in which parietal-control processes are sensitive to cue predictability. This could prove to be an interesting issue for exploration with patient groups who might have damage to goal-driven parameters (e.g. frontally-damaged patients). If such patients did not benefit from the use of predictable cues, as well as an inability to use other goal-driven control parameters, then the neural region(s) damaged in these patients might provide the circuitry for goal-driven attentional control.

The present results have at least two implications for neuropsychological studies of neglect and extinction patients. First, as other theorists have noted (Cohen et al., 1994), an attentional disengage mechanism does not need to be hypothesized to explain the performance of parietal-damaged patients on Posner's spatial precuing task (and perhaps other tasks as well, such as visual search tasks). Second, and more important, our results offer a potential explanation for at least some of the attentional impairments in neglect and extinction: patients may fail to attend items in contralesional space because these items do not fully engage the attentional system in a data-driven, or bottom-up, manner.

Our reliance on Cohen et al.'s model to think about the disengage pattern of results raises a natural question: Would this model exhibit the disengage pattern if the inputs to the model were degraded, as in the current studies? Although a full simulation of all of the experiments reported here is beyond the scope of the present paper, we have started to simulate the basic result presented in Experiment 1 and have found that an interactive variant of Cohen et al.'s model produces a disengage pattern following a unilateral reduction of the bottom-up input to the model. These preliminary results are depicted in Fig. 6, which also shows results from 16 normal human participants who had not taken part in any of the present studies. The model's input was degraded on one side by reducing the input value from 1.0 to 0.75, and the human participants' input was reduced as in Experiment 1. The model was allowed to cycle until it settled into a stable pattern of activity in which the units' activation values were no longer changing. The number of cycles to settle was measured; to compare human RT with the models' cycles, we computed an RT for the model by first performing a linear regression of the model's cycles for each condition on the humans' mean RTs for each condition. We used the resulting regression equation to convert the model's cycles to settle to an RT. As Fig. 6 shows, the model does an excellent job simulating the human results ($R^2 = 0.79$), suggesting that our variant of the Cohen et al. model produces a disengage pattern when provided degraded inputs. Interestingly, the simulated results show a larger effect of degradation on valid trials than do the behavioral results, suggesting that our human participants may be using a compensatory strategy—such as endogenous orienting—to reduce the effects of unilateral degradation on valid trials. Future simulations could examine this latter suggestion and focus on the results of our other experiments.

Our focus on data-driven parameters in parietal-damaged patients may be a theoretically useful way to stimulate further research. For example, Yantis and colleagues have demonstrated that abrupt visual onsets capture attention automatically, perhaps in a data-driven fashion (see Yantis, 1998). Neglect and extinction patients might be slowed to detect abruptly appearing targets in the contralesional field compared to those appearing in the ipsilesional field. Other data-driven control parameters, such as the detection of local inhomogeneities in a cluttered image (e.g. Sagi & Julesz, 1984), could be explored in parietal-damaged patients to determine if data-driven control is indeed disrupted.

Although our simple degradation manipulation allows us to simulate the effects of parietal-lobe damage in normal participants, we acknowledge that parietal-damaged patients exhibit several effects that might not be simulated with a simple unilateral screen degradation. For example, there have been many reports of object-centered neglect, in which patients not only neglect the contralesional side of space but also the contralesional side of objects (Behrmann & Moscovitch, 1994; also see Behrmann & Tipper, 1994; see Rafal, 2000, for a recent review), even when the entire object appears in the good (ipsilesional) field. We have not explored the possi-

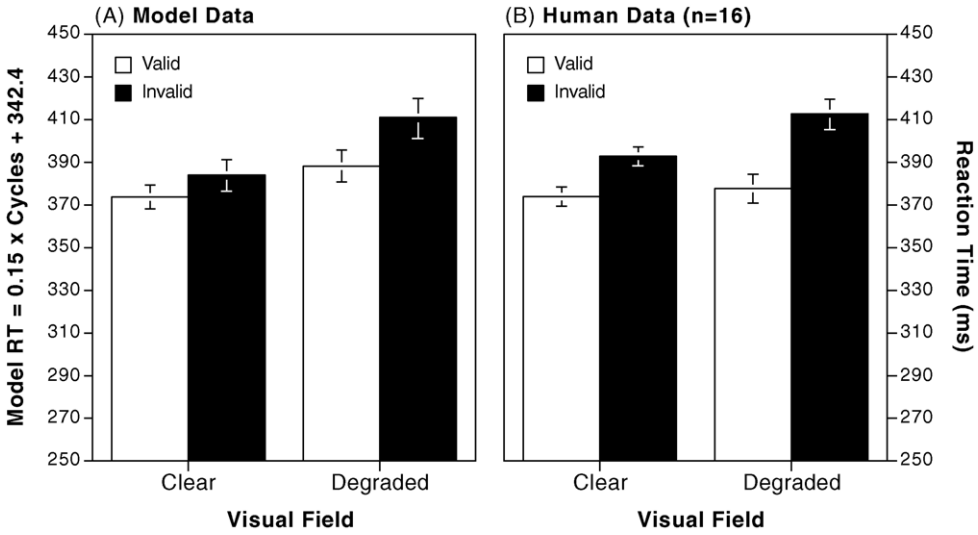

Fig. 6. Preliminary results from an interactive model designed to simulate performance on a spatial cuing task. (A) Results from the model when inputs were degraded. (B) Results from 16 normal human participants whose results replicated Experiment 1. The model exhibits the disengage pattern of results following unilateral input degradation. (Error bars on the human data are 95% within-subject confidence intervals on the valid vs. invalid comparisons, and error bars on the model's data are 95% confidence intervals of each condition mean).

bility that our simple screen degradation could mimic these results. However, if our degradation procedure did not produce object-centered neglect, this failure would not rule out data-driven attentional control as a problem in neglect patients. Our screen degradation may only tap data-driven spatial attention, not object-based attention. Recent theoretical perspectives on object-centered neglect suggest that object-centered neglect might arise from egocentric neglect (Driver & Pouget, 2000; Mozer, 1999, 2002; Mozer & Vecera, in press; Pouget & Driver, 2000; Pouget & Sejnowski, 1997), in which the left side of an object is less likely to control attention, even when the object appears in the intact field. A damaged 'gradient' of attention, in which data-driven attentional control gets progressively poorer from the patients right to left, can explain most of the object-centered neglect literature (Mozer, 1999, 2002). Our degradation procedure would not reproduce this damaged gradient in normal participants because the degradation is too discrete and transitions abruptly from 'clear' to 'degraded' at fixation. However, object-centered neglect might be simulated if some form of degradation could be found to replicate the damaged gradient predicted by Mozer's model, in which degradation gradually increases from the ipsilesional field to the contralesional field.

Another important issue that warrants discussion is a potential confusion over the term 'data-driven control parameters'. We do not mean to imply that perceptual impairments per se are the cause of phenomena observed in neglect and extinction patients. Although our screen degradation was a direct manipulation of perceptual parameters (i.e. the degradation impaired perception), the effects we observed were attentional effects, not merely perceptual effects (e.g. a scotoma). Specifically, unilateral screen degradation had the effect of causing an imbalance in the attentional competition between representations of the two fields; the degraded field could not compete for attention as well as the intact field. Other studies have ruled out simple perceptual impairments as the cause of neglect and extinction behavior (e.g. Baylis, Driver, & Rafal, 1993). Further, our use of a detection task might rely minimally on perceptual processes (unlike a discrimination task), making our screen degradation manipulation affect attentional processes more than perceptual processes.

Although we would agree that perception is intact in neglect/extinction patients, we should acknowledge that it may be difficult to distinguish between attentional effects and perceptual effects, both in our data and in data from neglect and extinction patients. If attention acts to enhance perceptual representations, then a failure to enhance a perceptual representation could be caused by damage to either attention or perception (or damage to both). In general, if one assumes that attention and perception mutually influence one another, as many accounts of attention indicate (Cohen et al., 1994; Mozer, 1991; Mozer & Sitton, 1998), then it may be impossible to cleanly separate attentional processes from perceptual processes (see Vecera & O'Reilly, 2000, for discussion of the difficulty of disentangling interacting processes).

Our studies have investigated only the issue of attentional control. Another major focus in attention research are the effects of attention. We do not want to argue that all of the effects of attention are produced by a common mechanism (e.g. data-driven attentional control). Indeed, some attentional effects, such as simple target detection, as we have studied, may be linked with data-driven control parameters, but other effects, such as the integration or binding of features into whole objects, may be linked with goal-driven control parameters. Although our present results do not directly speak to this issue, our screen-degradation procedure could be used

to determine those attentional effects that, perhaps like neglect and extinction, are produced by data-driven attentional control or impairments in data-driven attentional control.

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