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Abnormal inhibition of return: A review and new data on patients with parietal lobe damage

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The study of the performance of patients with neurological disorders has been fruitful in revealing the nature and neural basis of inhibition of return (IOR). Thus, in recent years, studies have reported abnormal IOR in patients with Alzheimer's disease, patients diagnosed with schizophrenia, and brain-damaged patients. In the present study, we investigated the hypothesis that a spatial "disengagement deficit" (DD; Posner, Walker, Friedrich, & Rafal, 1984) contributed to the pattern of impaired IOR in the ipsilesional field of parietal patients, found in a previous work (Vivas, Humphreys, & Fuentes, 2003). In a first experiment, we replicated the attenuation of IOR for ipsilesional targets on those trials with a lateralized IOR procedure. With stimuli vertically aligned about fixation, we found intact IOR for both up and down targets. Most important, when we ameliorated the potential impact of a spatial DD by presenting both cues and target in the same hemifield, still we found impaired IOR in the ipsilesional field. We interpret these findings in terms of unilateral parietal damage leading to an imbalance of the relative salience of signals represented in a spatial map for directing attention.

INTRODUCTION

Twenty years have passed since Posner and collaborators (Posner, Rafal, Choate, & Vaughan, 1985) referred with the term *inhibition of return* (IOR) to the empirical observation, which they reported a year earlier (Posner & Cohen, 1984), of slowed response times to peripheral targets that follow the earlier presentation (about 600 ms) of an irrelevant spatial cue. During these years, considerable research has been undertaken to determine both the nature and boundary conditions of IOR, and the progressively increasing number of articles and reviews published on this phenomenon speaks eloquently for its relevance (Klein, 2000; Klein & Taylor, 1994; Taylor & Klein, 1998). Currently, work on IOR generates around 40 publications per year in journals listed in the ISI

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Journal Reports Index (Lupiáñez, Klein, & Bartolomeo, 2006). The present paper is written in recognition of the seminal work of Posner and collaborators, which has had such an influence on our own research.

In general, research has reported abnormal IOR in several populations with neurological and psychiatric pathologies, as well as in normal ageing.

Abnormal IOR in Alzheimer's disease and ageing

Many studies converge to the idea that healthy ageing and Alzheimer's disease (AD) are related with changes in inhibitory processes of attention that might be correlated with specific alterations in the brain. In relation to normal ageing, studies have found IOR effects in healthy old adults as large as those found in young adults, when different tasks were employed such as target onset detection (Faust & Balota, 1997; Hartley & Kieley, 1995), letter (Hartley & Kieley, 1995) and colour discrimination (Langley, Vivas, Fuentes, & Bagne, 2005), and categorization (Langley, Fuentes, Hochhalter, Brandt, & Overmier, 2001), and regardless of whether a central cue was used or not to return attention to the fixated location before the target was presented. However, IOR seems to be more resistant to resolution with SOA changes in healthy older adults than in young adults (Hartley & Kieley, 1995; Langley et al., 2001). Although, these studies suggest that location-based IOR appears to be relatively unaffected with normal ageing, evidence shows that object-based IOR is impaired in old adults (McCrae & Abrams, 2001). That is, Tipper, Driver, and Weaver (1991) showed that if the original visuospatial task employed by Posner and Cohen (1984) changed, and participants had to response to targets that appeared inside boxes that moved across the field, then the inhibition moved with the object. In this case, response times (RTs) were slowed to stimuli that appeared inside the previously cued object, relative to stimuli appearing in an uncued object that had moved into a new location (e.g., after the peripheral spatial cue the boxes rotated 90° in polar coordinates). Tipper, Weaver, Jerreat, and Burak (1994) proposed that these two different forms of IOR, location based and object based, would implement attentional biases towards novelty, in visual search tasks, under different conditions (e.g., looking for a stationary object and looking for a friend in the airport, respectively), and that they might be mediated by different cortical systems (Tipper et al., 1994, 1997). In agreement with previous findings, McCrae and Abrams (2001) reported intact location-based IOR in a group of older adults; however, unlike the group of young adults, the old adults showed object-based facilitation instead of inhibition with the dynamic display. This finding suggests a differential pattern of breakdown of these two inhibitory effects, which agrees with the hypothesis of task-specific inhibitory deficits in normal ageing (Kramer, Humphrey, Larish, Logan, & Strayer, 1994).

The evidence regarding IOR in AD is not as consistent. Faust and Balota (1997) first reported impaired IOR in AD patients using a single-cue paradigm (Experiment 1), whereas they showed intact IOR when a second central cue was employed (Experiment 2). The authors concluded that the failure to observe IOR with a single spatial cue was due to a problem in spontaneous disengagement from the peripheral cue, in order to endogenously reorient attention back to the centre. However, in a later study, Danckert and colleagues (Danckert, Maruff, Crowe, & Currie, 1998) found normal IOR effects in a group of AD patients using both single-cue and double-cue paradigms, and, similar to the findings with old adults, this effect seemed more resistant to resolution with longer stimulus onset asynchronies (SOAs). Danckert et al. (1998) noted that Faust and Balota (1997, Exp. 1) used spatial cues with predictive value and concluded that under these circumstances both modes of attentional control—endogenous and exogenous—could have been confounded, leading to impaired performance in AD patients. Other authors have suggested that task demands might be crucial to observed impaired IOR in AD patients even when a central cue is employed in order to

automatically shift attention back to the centre. Thus, Langley et al. (2001) showed intact IOR with a double-cue procedure and onset detection responses in a group of AD patients, but failed to find significant IOR effects with a more complex categorization task. These later findings suggest a cortical involvement in the generation of location-based IOR especially when more sophisticated tasks than onset detection are used. Thus, a broader neural network than it was originally thought might be involved in the late inhibitory effects observed in visuospatial tasks like the one employed by Posner and Cohen (1984). In this sense posterior areas might be concerned with inhibition of locations in simple spatial detection tasks, whereas more anterior areas might play a role in generating high-level properties of IOR related with feature or response selection processes in more sophisticated tasks (Langley et al., 2001; Vivas, Humphreys, & Fuentes, 2003).

Abnormal IOR in schizophrenia

IOR has also been tested in patients diagnosed with schizophrenia, although the literature reflects rather contradictory results (Fuentes, 2001a, 2001b). Inconsistency in findings might be due to differences across studies in relevant aspects associated with the disease (heterogeneity of the disorder; different medications; onset and severity of the illness), but also to differences in the procedure used to assess IOR effects. For instance, Fuentes and Santiago (1999) found preserved IOR in a group of medicated schizophrenic in-patients with predominance of positive symptoms. However, these results contrast with those of Huey and Wexler (1994) and Gouzoulis-Mayfrank et al. (2004) where blunted IOR has been reported in medicated patients. The different procedures used in the studies to measure IOR may account for the discrepant results. Huey and Wexler (1994) and Gouzoulis-Mayfrank et al. (2004; see also Sapir, Henik, Dobrusin, & Hochman, 2001, Exp. 1) did not use a second central cue to withdraw attention from the periphery. The second central cue in the Fuentes and Santiago study could have speeded up the development of IOR and together with the use of very long SOAs might have overcome the initial deficit in the inhibitory process (although see Larrison-Faucher, Briand, & Sereno, 2002, for delayed onset but normal magnitude of IOR with the longest SOAs). In addition, similar to the explanation proposed for abnormal IOR in AD patients, the observed impairment in IOR with a single peripheral cue could have been due to a deficit in the processes that control intrinsic reorientation of attention rather than a deficit in filtering information that appears at inhibited locations.

Two studies compared IOR effects under single peripheral and second central cue procedures with 1,200-ms SOA, with contradictory results. In the Fuentes, Boucart, Alvarez, Vivas, and Zimmerman (1999) study the schizophrenic patients showed IOR effects in both cueing conditions similar to those exhibited by healthy adults. In contrast, Sapir et al. (2001) showed preserved IOR effects with the second central cue condition, but lack of IOR with the single peripheral cue condition. The different results might be due to the severity of the illness in the schizophrenic participants of the two studies. Fuentes et al.'s participants were out-patients, whereas Sapir et al.'s were in-patients. In line with this, Fuentes and Santiago (2002) observed reduced IOR with a group of medicated psychotic patients with predominant negative symptoms in the single-cue procedure compared with the second central cue procedure.

Medication seems to be relevant for IOR as well. AL, a patient diagnosed with schizophrenia who usually refuses to take medication, carried out the Fuentes et al.'s (1999) experiment (reported in Fuentes, 2001a). The results showed preserved IOR with the second central cue condition, but no IOR at all with the single-cue procedure. Taken together these results suggest that both severity of the disease and medication may play a fundamental role in the process that leads to voluntary reorient attention to the centre when noninformative cues are presented, locating the deficit in the processes that lead to IOR rather than in this inhibitory mechanism per se.

However, studies of patients on medication have also revealed some contradictory findings. Fuentes and Santiago (1999) examined medicated patients and reported preserved IOR; Huey and Wexler (1994), Gouzoulis-Mayfrank et al. (2004), and Sapir et al. (2001) examined medicated patients and reported blunted IOR. Carter, Robertson, Chaderjian, Celaya, and Nordahl (1992) reported normal IOR in nonmedicated patients, but the same authors reported IOR deficits in a group of nonmedicated patients diagnosed with schizophrenia of the paranoid type (Carter, Robertson, Chaderjian, O'Shora-Celaya, & Nordahl, 1994).

Briefly, despite some inconsistencies, all these results suggest that processes leading to IOR might be affected in schizophrenia. However, certain procedural manipulations (e.g., the use of a second central cue and a very long SOA), the degree of severity of the disease, and medication might reduce the deficit in IOR, at least in some types of schizophrenia. Furthermore, given that, similar to AD patients, several cortical alterations have been hypothesized to be at the core of the cognitive symptoms in schizophrenia, the finding of abnormal IOR in these patients suggests that an intact cortex may be necessary for this effect to be observed. However, the relatively diffuse nature of the brain abnormalities associated with AD and schizophrenia does not allow linking IOR to any particular site within the cortex.

Abnormal IOR in brain-damaged patients

The first study that aimed at identifying particular brain areas associated with IOR examined the performance of 6 patients with progressive supranuclear palsy (PSP; affecting the superior colliculus), 4 patients with Parkinson's disease, and 5 and 7 patients with focal cortical lesions in the frontal and parietal lobes, respectively (Posner et al., 1985). As the authors predicted, the group of patients with PSP failed to show IOR when the spatial locations were arranged vertically (4 ms), but showed normal IOR with the typical horizontal display (46 ms; see Table 2 in Posner et al., 1985). The authors concluded that the failure to observe IOR for the vertical display in the PSP group was not due to a general motor deficit, since the group of Parkinson patients showed intact IOR, but it was linked to the impaired ability of these patients to make voluntary saccadic eye movement in the vertical direction. Furthermore, they reported typical inhibitory effects with the horizontal display in parietal and frontal lobe patients. From this the authors argued that IOR is mediated by lower level neural structures such as the superior colliculus (Sapir, Soroker, Berger, & Henik, 1999) and consequently suggested that high-level cortical processes are not involved in the generation of IOR. However, a closer look to the pattern of data exhibited by the patients with parietal damage shows that the IOR effect seemed to be larger for the contralateral visual field (31 ms) tha for the ipsilesional field (19 ms; see Table 2 in Posner et al., 1985). Although this tendency was not examined further in the seminal paper of Posner and colleagues, later neuropsychological studies of IOR have confirmed this pattern of results in parietal patients and patients with the neglect syndrome.

Thus, Bartolomeo and colleagues (Bartolomeo, Chokron, & Siéroff, 1999; Bartolomeo, Siéroff, Decaix, & Chokron, 2001) reported abnormal IOR in left unilateral neglect patients, using both a response–response paradigm (where two successive targets could appear at the same or a different location) and a cue–target paradigm (with a single peripheral cue). In both studies, the neglect patients showed a positive advantage for repeated ipsilesional targets as compared to unrepeated ipsilesaional targets (Bartolomeo et al., 1999) and for ipsilesional target that appeared in the previously cued location (Bartolomeo et al., 2001, Exp. 1), under general conditions that would normally generate IOR. The finding of facilitation instead of inhibition in the right (ipsilesional) field was attributed by the authors to a strong attentional bias towards right-side objects in left unilateral neglect patients (Bartolomeo et al., 1999). In addition, a difficulty in disengaging their attention from ipsilesional cues in order to respond to contralesional targets (Posner, Walker, Friedrich, & Rafal, 1984) could

have contributed to this pattern of results, although as P. Bartolomeo pointed out (personal communication, February 7, 2005), the decrease of the disengagement deficit (DD)[1] with longer SOAs, when the IOR impairment was still present (Bartolomeo et al., 2001, Exp. 1), suggests that these two deficits might be independent in neglect patients.

In agreement with these findings, we found that a group of 4 patients with left and right brain lesions in the inferior parietal lobe, who had extinction but not clinical neglect at the time of testing, showed attenuated IOR for ipsilesional targets in both detection (Vivas et al., 2003, Exp. 1) and colour discrimination tasks (Vivas et al., 2003, Exp. 2). However, unlike in Bartolomeo and colleagues' studies (Bartolomeo et al., 1999, 2001), only one patient showed a tendency for facilitation instead of inhibition in the ipsilesional field. This difference could be explained in terms of a stronger bias towards the ipsilesional field in parietal patients with neglect, leading to a stronger facilitation for targets presented in this side (Vivas et al., 2003). Most importantly, we concluded that the parietal lobe plays a crucial role in the generation of IOR, and that the pattern of impaired IOR in neglect and parietal patients could have been due to either of two deficits: a strong orienting response to ipsilesional targets (e.g., Ladavas, Petronio, & Umiltà, 1990; Shalev & Humphreys, 2000) that overruled any IOR applied there, and/or slowed disengagement of attention from ipsilesional cues in order to respond to contralateral targets (the disengagement deficit, DD; Posner et al., 1984). The DD was first proposed by Posner et al. (1984) in order to account for a pattern of abnormally increased response times to invalid cued targets in the contralesional hemifield, as compared to the ipsilesional hemifield (extinction-like RTs pattern), in unilateral parietal patients.

Although the DD was originally reported in studies that used informative cues (eliciting an endogenous control of attention), later studies have shown that: (a) the DD is particularly associated with purely exogenous orienting of attention (noninformative exogenous cues); (b) the DD decreases with longer SOAs in right brain-damaged patients, whereas in left brain-damaged patients the overall size of the DD is smaller but remains constant with increasing cue–target intervals; and (c) although the DD is significantly reduced with SOAs longer than ~200 ms, it remains significant with cue–target SOA of 1,000 ms (see Losier & Klein, 2001, for a review). Note that both impaired IOR and a spatial DD can generate a strong attentional orienting response towards the ipsilesional field, and any unique problem in IOR may have combined with a DD in prior studies showing impaired IOR in parietal patients (Bartolomeo et al., 1999, 2001; Vivas et al., 2003). From prior data, any independent deficits are difficult to disentangle. The purpose of the present study was to investigate the contribution of a possible DD to the finding of attenuated IOR, for ipsilesional targets, in parietal patients. We report two studies, the first demonstrating basic IOR effects across the horizontal and vertical meridians in parietal patients, the second showing IOR effects within each field (to test the DD account).

EXPERIMENT 1: IOR IN THE HORIZONTAL AND VERTICAL MIDDLE AXES

The present experiment had two purposes: (a) to replicate the pattern of attenuated IOR for the ipsilesional field (Vivas et al., 2003) in a group of parietal patients without clinical signs of neglect; and (b) to test whether IOR can be elicited with vertically arranged boxes in this group of patients. In order to do so, we employed the same IOR procedure, with an array of three boxes arranged

[1] Note that this DD, which is related to automatic and exogenous control of attention, differs from the deficit in intrinsic spontaneous shift of attention that has been attributed to AD and schizophrenia.

horizontally, as in Vivas et al. (2003) on half of the trials, but with shorter SOA (660 ms). In the remaining trials the three boxes were arranged vertically, centred on the middle axes of the computer's screen.

Method

Participants

A total of 5 healthy adults participated in this experiment. Their age ranged from 41 to 78 years, with a mean of 59 years. They all had normal or corrected-to-normal vision. We examined 4 patients, all with unilateral lesions affecting the inferior parietal lobe (M.H., P.F., R.H., J.B.), in the left hemisphere in 3 (M.H., P.F., R.H.), and in the right hemisphere in 1 (J.B.). Clinical details of the patients are presented in Table 1, and transcriptions of their lesions are shown in Figure 1. All the patients showed visual extinction but none manifested unilateral neglect on standard tests requiring spatial scanning of attention (e.g., star or line cancellation).

Stimuli

The experimental task was created using MEL (Micro Experimental Laboratory; Schneider, 1988). The target consisted of a small red square that could appear inside of one of the four lateral boxes (left, right, up, and down). Participants had to press the space bar as soon as they saw the target stimulus.

Procedure

A trial began with a fixation cross. The fixation cross remained on the screen until the experimenter judged that the patient was looking at the cross and was ready to perform the task. The fixation cross was then replaced by three white boxes that could be arranged horizontally or vertically for 1,000 ms (see Figure 2). Then, one of the outside boxes thickened (the peripheral cue) for 100 ms. After an interval of 100 ms, the central box thickened (the central cue) for 130 ms. The interstimulus interval between the central cue and the target was 330 ms. The target appeared on 83% of the trials and was absent on the remaining 17% of the trials. The target stimulus appeared either in the location previously cued or in the opposite peripheral uncued location, on either the vertical or the horizontal axis. The target remained on the screen until the participant responded by pressing the space bar. On catch trials without a target, participants were instructed not to respond.

Table 1. *Age, sex, aetiology, location of the lesion, and neurological signs of the patients who served as participants in Experiments 1 and 2*

Patient	*Age*[a]*/Sex*	*Aetiology*	*Location*	*Neurological signs*
M.H.	48/M	Anoxia	Left inferior parietal, angular gyrus, extending to superior parietal lobule	Extinction, mislocalization
D.B.	62/M	Stroke	Left middle and superior temporal, sylvian fissure, angular gyrus, inferior parietal	Anomia, extinction
P.F.	53/F	Stroke	Left parietal (angular gyrus, supramarginal gyrus), superior temporal gyrus	Extinction
R.H.	70/M	Stroke	Left parietal (angular and supramarginal gyri), superior temporal gyrus	Anomia, extinction
J.B.	67/F	Stroke	Right parietal (angular gyrus, supramarginal gyrus), superior temporal gyrus, extending to inferior frontal	Extinction

Note: M.H., P.F., R.H., and J.B. took part in Experiment 1; M.H., P.F., R.H., J.B., and D.B. took part in Experiment 2. Extinction was demonstrated by a selective deficit in reporting a contralesional event under conditions of double simultaneous stimulation. None of the patients demonstrated unilateral neglect on standard tests requiring spatial scanning, such as line or star cancellation.
[a]In years.

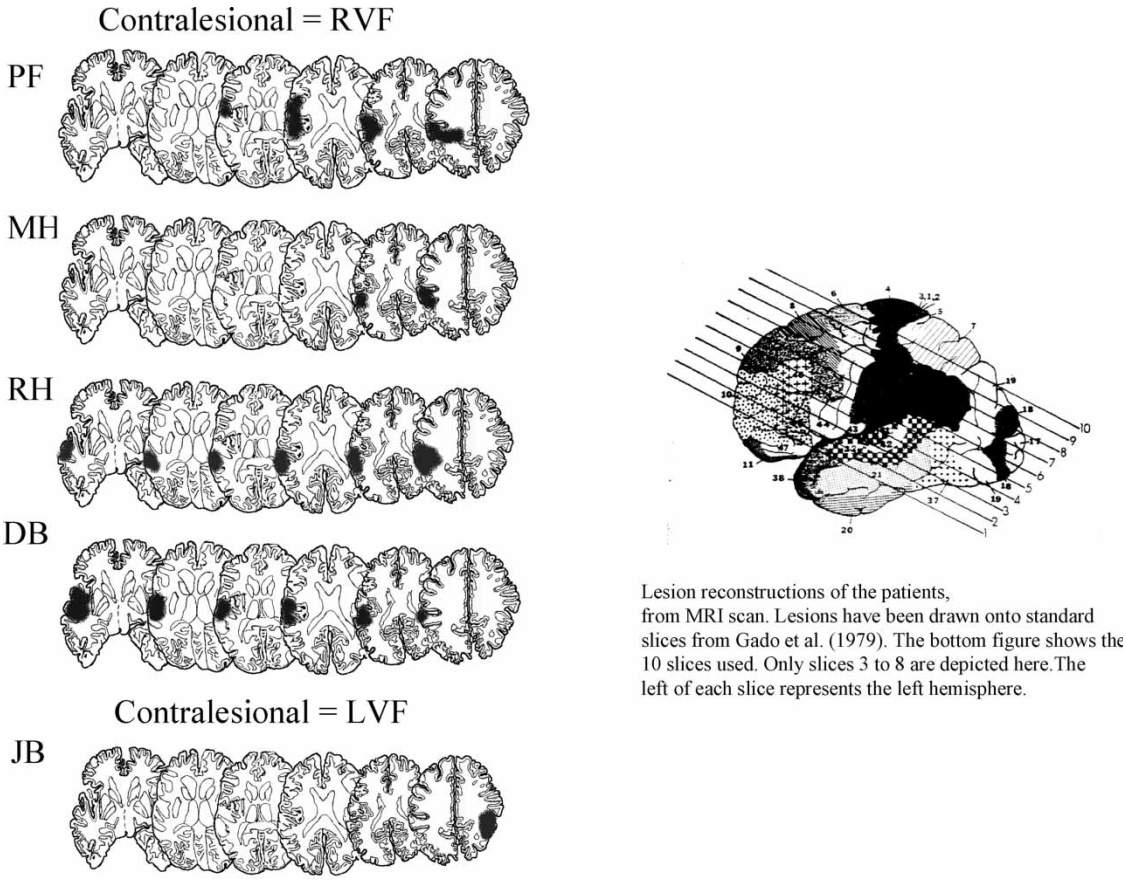

Figure 1. *MRI scans plotted onto standardized slices of the 5 patients (M.H., D.B., P.F., R.H., J.B.) who served as participants. The standardized plates are taken from Gado, Hanaway, and Frank (1979). Only Slices 3 to 8 are depicted here.*

The patients and controls ran one practice block of 24 trials followed by two experimental blocks of 96 trials. In the experimental block the target was presented on 80 trials (83%), and it was absent on 16 trials (17%). On half of the present trials (40), the three boxes were arranged horizontally, and in the remaining trials they were arranged vertically. Also, for each spatial arrangement the targets appeared in the left hemifield (or ipsilesional hemifield for the group of patients) or in the upper hemifield, respectively, on half of the trials (20), and on the remaining trials they fell in the right hemifield (or contralesional hemifield for the group of patients) or in the lower hemifield, respectively. Also, for each hemifield the target appeared at the previously cued location on half of the trials (10) and at the uncued location on the other trials.

Results and discussion

The mean correct response times for the horizontal and the vertical trials were submitted separately to a mixed analysis of variance (ANOVA) with group (healthy adults and patients) as the between-subject factor and field (left and right for horizontal trials, and up and down for vertical trials) and cueing (cued and uncued) as within-subject factors (see Table 2). For the horizontal trials, the results yielded a significant main effect of cueing $F(1, 7) = 13.14$, $p < .05$. Response

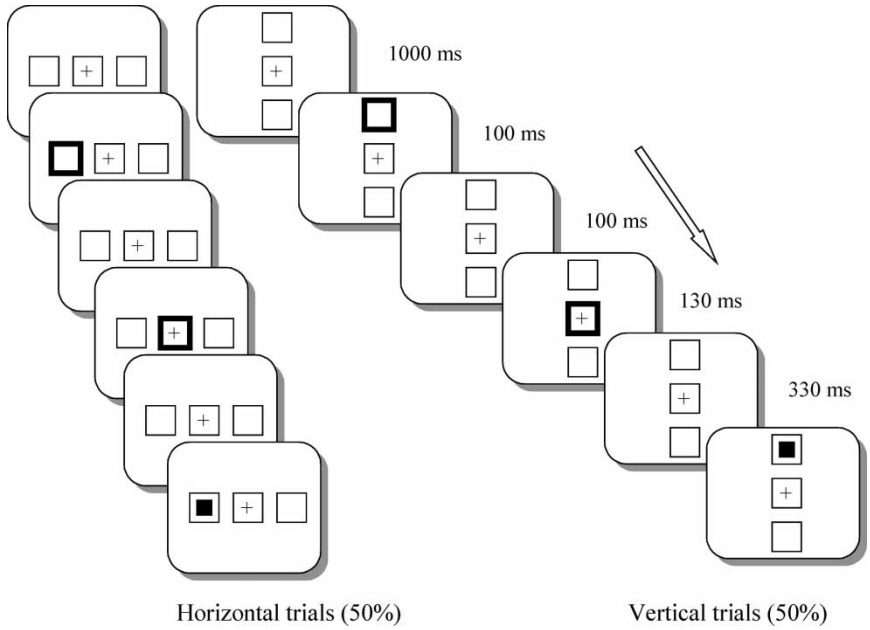

Figure 2. *Sequence of events and exposure duration of stimuli for a cued trial in horizontal and vertical trials in Experiment 1.*

times were higher for targets presented at the cued location (509 ms) than for those at the uncued location (490 ms). There were also significant field by cueing, and group by field by cueing interactions, $F(1, 7) = 24.52$, $p < .05$, and $F(1, 7) = 8.35$, $p < .05$, respectively. The interaction field by cueing was due to a IOR effect (38 ms) for left/ipsilesional field, whereas this effect did not emerge (1 ms) for targets presented in the right/contralesional field. However, this interaction was modulated by the three-way interaction between group, field, and cueing. The interaction analyses showed significant differences between the cued and the uncued location for both fields left (17 ms of effect), $t(4) = 2.94$, $p < .05$, and right (33 ms of effect), $t(4) = 2.91$, $p < .05$, in the group of healthy adults. However, in the group of patients, there was a significant difference between the cued and the uncued location only for the contralesional field (43 ms of effect), $t(3) = 2.91$, $p < .05$, but this effect did not emerge for targets presented in the ipsilesional field (−15 ms of effect), $t(3) = -1.44$, $p > .05$. No other effects reached statistical significance, ps $> .05$.

The analysis of the vertical trials yielded a main significant effect of cueing, $F(1, 7) = 17.34$, $p < .01$. No other effect, nor their interaction, reached statistical significance, ps $> .05$.

There were no anticipated responses or errors by the group of patients. In the group of healthy adults, only one of the participants made two anticipated responses.

The group of healthy adults showed a typical IOR effect for both horizontal (25 ms of effect) and vertical (35 ms of effect) trials, and this effect did not interact with the field (left, right, up, and down). The finding of IOR with vertically arranged locations agrees with previous results found in a similar procedure with healthy university students (Fuentes, Vivas, de Labra, Valle-Inclan, & Alonso, 2002).

For the group of parietal patients, the results from the horizontal trials replicated the data from our previous study (Vivas et al., 2003) using a shorter SOA value (660-ms SOA compared to 1,000-ms SOA in Vivas et al., 2003) and were similar to those reported by Bartolomeo and collaborators (Bartolomeo et al.,

Table 2. *Mean correct response times as a function of field and location for the group of controls and parietal patients in Experiment 1*

		Field			
	Target location	*Left*	*Right*	*Up*	*Down*
Controls	Cued	458 (94)	454 (106)	494 (74)	444 (102)
	Uncued	441 (102)	421 (120)	454 (101)	414 (109)
	IOR	17*	33*	40*	30*
		Ipsilesional	*Contralesional*	*Up*	*Down*
Patients	Cued	541 (55)	584 (89)	553 (95)	551 (71)
	Uncued	556 (69)	541 (84)	537 (89)	525 (84)
	IOR	−15	43*	17*	26*

Note: IOR (inhibition of return) = cued RT − uncued RT (mean difference score). RT = response time. Standard deviations are in parentheses.
*$p < .05$.

1999, 2001). That is, we found an IOR effect for contralesional targets (43 ms) and a nonsignificant tendency for a facilitation effect in the ipsilesional field (−15 ms). Furthermore, this pattern of results cannot be explained in terms of a tendency for smaller IOR for left visual field targets, relative to right visual field targets, operating in both healthy controls and patients, given that the right brain damage patient (J.B.) showed the same tendency of attenuated IOR for the ipsilesional field as the left brain damage patients (see Table 3). This result adds to a growing body of evidence suggesting that the parietal lobe is involved in the generation of IOR (Bartolomeo et al., 1999, 2001; Dorris, Klein, Everling, & Muñoz, 2002; Lepsien & Pollmann, 2002; Rosen et al., 1999). We suggest that two different deficits may be contributing to the lack of IOR for ipsilesional targets: (a) a strong ipsilesional orienting bias/an imbalance in a map determining competition for orienting, and/or (b) selectively slowed disengagement of attention from ipsilesional cues (to the more central cue, in the double-cueing procedure).

The results from the vertical trials showed an intact IOR effect for targets falling in both the up (17 ms) and the down (26 ms) locations. Although this vertical spatial procedure has been used in a previous studies to elicit IOR in healthy participants (e.g., Fuentes, Vivas, de Labra, Valle-Inclan, & Alonso, 2002), and in PSP and Parkinson patients (Posner et al., 1985), we are not aware of any other study that has employed this procedure with parietal patients. The finding of intact IOR in the middle vertical plane agrees with the hypothesis of a lateral (left-to-right) gradient of attention determined by the body's midsagittal plane (Kinsbourne, 1977), with poor performance in the contralesional field and hyperattention in the ipsilesional field, increasing with eccentricity. More importantly, for our present study, this finding enables us to study IOR for ipsilesional and contralesional targets using a vertical spatial procedure.

EXPERIMENT 2: IOR WITH A SPATIALLY LATERALIZED VERTICAL PROCEDURE

The purpose of the present study was to test the hypothesis that a deficit in disengaging attention from ipsilesional cues to more contralesional events contributed to the pattern of impaired IOR in the ipsilesional field, found in Experiment 1 and in our previous study (Vivas et al., 2003). In order to test this, we modified the original IOR procedure so that the cue and target always appeared in the same visual hemifield. That is, we used the same vertical IOR procedure employed in Experiment 1, but now with the stimuli spatially lateralized to the ipsilesional

Table 3. *Patients' individual means for correct response times as a function of field and location for horizontal trials, in Experiment 1*

	Ipsilesional			*Contralesional*		
	Cued	*Uncued*	*IOR effect*	*Cued*	*Uncued*	*IOR effect*
P.F.	554	561	−7	542	518	24
J.B.	538	534	4	668	607	61
R.H.	601	647	−46	647	606	41
M.H.	469	482	−13	479	432	47

or contralesional field. If patients in Experiment 1 (and in our previous work) had difficulty only when attention had to be disengaged from an ipsilesional cue to a more contralesional (central) cue, then we should expect to find IOR effects in both fields here, since a lateralized attentional shift is never required. The disengagement hypothesis, as originally proposed by Posner and collaborators (Posner et al., 1984), did not explicitly refer to vertical shifts of attention among different locations within the same hemifield; thus we assumed that parietal patients should be able to shift attention along the vertical axis within their ipsi- as well as their contralesional field, and so IOR effects should emerge in both cases. In contrast, if deficits in spatial attention after parietal damage reflect a strong ipsilesional orienting bias and/or an imbalance in signal competition[2] in a spatial map governing orienting, then IOR may still be impaired to targets in the ipsilesional field. This would follow if the originally cued ipsilesional boxes, and the targets that subsequently fall there, remain as powerful attractors of attention even when attention is shifted to another cue in the intervening interval.

Method

Participants

A total of 6 healthy adults participated in this experiment. Their age ranged from 41 to 68 years, with a mean of 55.8 years. They all had normal or corrected-to-normal vision. We examined the same 4 patients who participated in Experiment 1 and a new patient (D.B.) with a left unilateral lesion including the left inferior parietal lobe (see Table 1 and Figure 1).

Stimuli

The stimuli were the same as those used in Experiment 1, except that nine boxes, instead of three boxes, were presented on each trial.

Procedure

A trial began with a fixation cross. The fixation cross remained on the screen until the experimenter judged that the patient was looking at the cross and was ready to perform the task. The fixation cross was then replaced by nine white boxes arranged in three vertical arrays of three boxes each (one in the left side, a second one in the centre, and a third one in the right side of the screen) for 1,000 ms (see Figure 3). Then, one of the boxes in one of the upper or lower corners thickened for 300 ms (the peripheral cue). After an interval of 200 ms, the middle box of this side thickened for 300 ms (the central cue). Finally, after a further interval of 200 ms, the target could appear inside one of the lateral boxes in the upper or lower quadrant. The target remained on the screen until the participant responded by pressing the space bar. On catch trials without a target, participants were instructed not to respond.

The patients ran one practice block of 24 trials followed by two experimental blocks of 100 trials. In the experimental block the target was presented on 80 trials (80%), and it was absent on 20 trials (20%). On half of the present trials (40), the target appeared in the left hemifield (or ipsilesional for the group of patients), and it appeared in the right hemifield (or contralesional for the group of patients) in the remaining trials. Also, for each hemifield, the target appeared in the upper quadrant on half of the trials (20), and on the remaining trials it fell on the lower quadrant.

[2] Note that although all the events (cues and targets) are now presented only in one visual hemifield, there is still competition from the placeholder boxes in the opposite field.

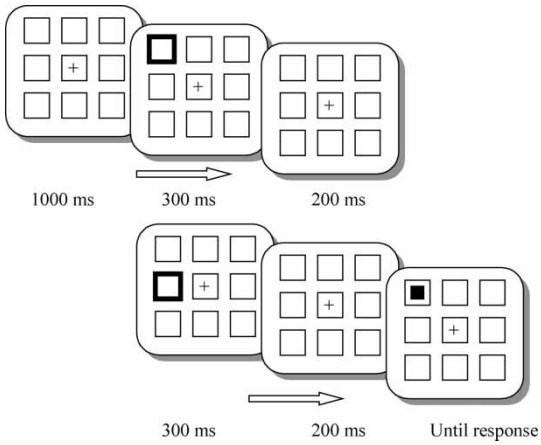

Figure 3. *Sequence of event and exposure duration of stimuli for a cued trial in Experiment 2.*

Finally, for each quadrant, the target appeared at the previously cued location on half of the trials (10) and at the uncued location on the other trials.

Results and discussion

The mean correct response times were submitted to a mixed ANOVA with group (healthy adults and patients) as the between-subjects factor and hemifield (left and right), quadrant (up and down), and cueing (cued and uncued) as within-subject factors (see Table 4). The results showed significant main effects of group, field, and cueing, $F(1, 9) = 5.85$, $p < .05$, $F(1, 9) = 6.35$, $p < .05$, and $F(1, 9) = 28.33$, $p < .05$, respectively. The following interactions were also significant: group by field, and group by field by cueing, $F(1, 9) = 28.33$, $p < .05$, and $F(1, 9) = 8.08$, $p < .05$. The analysis of the two-way interaction showed significant differences between the ipsilesional (495 ms) and the contralesional hemifields (558 ms) for the group of patients, $t(4) = -2.81$, $p < .05$, whereas there were no differences between the left (404 ms) and the right (404 ms) hemifields for the group of healthy adults, $t(5) < 1$. The analysis of the three-way interaction group by field and by cueing showed significant differences between the cued and the uncued location for both visual fields left (28 ms of IOR effect), $t(5) = 4.37$, $p < .05$, and right (18 ms of IOR effect), $t(5) = 5.26$, $p < .05$, for the group of healthy adults. However, for the group of patients, there were significant differences between the cued and uncued location (28 ms of effect) only for the contralesional field, $t(4) = 3.52$, $p < .05$; however, this effect did not emerge for ipsilesional targets (2 ms of effect), $t(4) < 1$. No other effects reached statistical significance, ps $> .05$.

There were no anticipated responses or errors by the patients and the healthy adults.

The group of healthy adults showed a typical effect of IOR (24 ms). More important, this effect did not interact with either the field (left and right) or the quadrant (up and down). This finding confirms previous research, which showed that a vertical spatial procedure is effective in generating IOR (Experiment 1, present study; Fuentes et al., 2002).

The group of parietal patients showed similar results to those found in Experiment 1—that is, IOR did not emerge for targets falling in the ipsilesional field (2 ms), whereas there was

Table 4. *Mean response times as a function of field, place, and location for the group of controls and parietal patients in Experiment 2*

		Field/place			
		Left		*Right*	
	Target location	*Up*	*Down*	*Up*	*Down*
Controls	Cued	426 (26)	410 (16)	424 (40)	402 (30)
	Uncued	397 (33)	383 (17)	400 (29)	389 (39)
	IOR	29*	27*	24*	13
		Ipsilesional		*Contralesional*	
		Up	*Down*	*Up*	*Down*
Patients	Cued	493 (98)	490 (84)	542 (127)	528 (114)
	Uncued	491 (113)	487 (85)	502 (109)	513 (108)
	IOR	2	3	40*	15 †

Note: IOR (inhibition of return) = cued RT − uncued RT (mean difference score). RT = response time. Standard deviations are in parentheses.
*$p < .05$; †$p = .08$.

intact IOR when the targets appeared in the contralesional field (28 ms). These findings suggest that a DD did not contribute to the attenuation of IOR for ipsilesional targets found in Experiment 1 and in previous studies (Vivas et al., 2003). In contrast, the failure to observe IOR in the ipsilesional field, even when attention was shifted to a cue in the same field, fits quite well with the hypothesis of a strong ipsilesional orienting bias and/or an attentional imbalance in visual selection. This bias/imbalance can be so strong that any IOR on the ipsilesional side is overruled either by sustained orienting to the original cue or by the orienting response to a new ipsilesional event.

GENERAL DISCUSSION

One of the most intense debates on IOR has been concerned with whether it reflects the inhibition of attentional orienting processes (Posner & Cohen, 1984), the inhibition of oculomotor programmes (Sapir, Soroker, Berger, & Henik, 1999), or the inhibition of response selection processes (Klein & Taylor, 1994). Although, initially, IOR was strongly linked to the subcortical structures subserving oculomotor programmes (Posner et al., 1985; Sapir et al., 1999), more recent work has suggested that cortical areas, related to visuospatial attention, may also play an important role (Lepsien & Pollmann, 2002; Rosen et al., 1999). Indeed, functional magnetic resonance imaging (fMRI) studies have not found activation in the superior colliculus, but they have reported significant activation associated with IOR in several cortical areas including the frontal eye fields, the superior parietal cortex, and anterior motor areas (Lepsien & Pollman, 2002; Rosen et al., 1999). The hypothesis that IOR might be generated in the cerebral cortex is also strengthened by one study with single unit recording in monkeys, where there was no inhibition in the superior colliculus although there was a behavioural IOR effect (Dorris et al., 2002). The present finding (Experiment 1), of nonsignificant facilitation instead of inhibition in the ipsilesional field of parietal patients, replicates our previous data (Vivas et al., 2003) and provides additional support to this hypothesis.

Our new finding is the lack of an IOR effect in the ipsilesional field even when a vertical procedure is employed, so that attention does not shift laterally (Experiment 2). This result indicates that a selective spatial DD (Posner et al., 1984) is insufficient to account for the pattern of results found in the present (Experiment 1) and previous work (Vivas et al., 2003).

Posner and collaborators (see Posner & Raichle, 1994, for a review) have proposed a theoretical framework for attention in terms of a distributed network of brain systems. According to this framework, the orienting of attention involves three separate elementary operations: *disengaging* from the current object/location, *moving* towards the target, and *engaging* the target. In later studies with brain-damaged patients, this disengaging operation was localized in the parietal lobe, whereas the moving and engaging operations were located in the midbrain and thalamus, respectively (Friedrich, Egly, Rafal, & Beck, 1998; Posner et al., 1984). The argument concerning the effects of parietal damage was based on Friedrich et al.'s (1998) finding that parietal patients showed an extinction-like pattern (very slow detection times) for targets presented in the contralesional field but only for invalid conditions—when the spatial cue was presented in the ipsilesional field. They suggested that there was a specific problem in disengaging attention from an ipsilesional stimulus in order to orient to a more contralesional event (a spatial disengagement problem).[3] Later on Posner, Walker, Friedrich, and Rafal (1987) slightly modified this model and proposed that unilateral damage to the parietal lobe would result in a DD when attention must be moved in a contralateral direction

[3] However, see Cohen, Romero, Farah, and Servan-Schreiber (1994) for a simulation of the extinction-like RT pattern using a model of attention based on competitive interactions that do not require a "disengagement" operation.

regardless of the field in which attention was engaged previously. This model of spatial attention has been put forward to explain the pattern of performance of patients with neglect and extinction. Thus, poor performance in the contralesional hemifield would reflect a deficit in disengaging attention from ipsilesional stimulation in order to orient/respond to contralesional targets. However, other alternative explanations have been offered to explain orienting deficits in parietal patients. Thus, it has been proposed that parietal damage may result in an ipsilesional attention bias or ipsilesional "hyperattention" (Ladavas et al., 1990). Another account, proposed by Duncan, Humphreys, and Ward (1997), suggests that unilateral parietal damage produces an imbalance in visual selection and action, with attentional competition being biased towards ipsilesional signals. Although these three accounts can be seen as complementary, it is important to explore their contribution to the different pattern of performance exhibited by parietal patients. The validity of these models to explain cognitive deficits in parietal patients is also of great relevance because of its implications for understanding normal functioning attention. Our data suggest that a lateral DD is not contributing to the pattern of impaired IOR in the ipsilesional field found in the present study (Experiments 1 and 2) or, by extension, in our previous work with a similar group of parietal patients (Vivas et al., 2003). In contrast, our results are clearly compatible with the role of the posterior parietal cortex in implementing competitive biases in spatial attention. As we have proposed previously, "the posterior parietal cortex may contain a spatial map that signals the relative salience of locations for attention" (Vivas et al., 2003, p. 1539). In this map, previously cued locations, in an IOR procedure, may normally be marked by a reduced relative salience. Unilateral parietal damage may produce an imbalance in the relative salience of locations in this map, so that ipsilesional locations may be more salient and receive less competition from contralesional locations. The increased relative salience of the ipsilesional signals in the spatial map may be sufficient to overrule any IOR applied to ipsilateral locations, in parietal patients.

Further, a review of abnormal IOR in patients with Alzheimer's disease and schizophrenia, and of IOR in healthy old adults, also suggests that a wide network (probably involving several cortical areas) may be involved when properties of the stimulus other than its location are strongly weighted in an IOR procedure (i.e., object-based properties such as colour or identity). For instance, as mentioned, Langley et al. (2001) found that any impairments in the performance of AD patients in an IOR procedure were modulated by task demands. Also, patients diagnosed with schizophrenia do not exhibit the response-related inhibitory effects associated with IOR that have been previously reported in healthy populations (Fuentes, Boucart, Vivas, Alvarez, & Zimmerman, 2000). Further research is needed in order to directly compare the performance of different groups of patients in equivalent IOR tasks, to enhance our understanding of how the parietal lobe collaborates with other cortical areas such as the frontal lobe (Vivas et al., 2003), in order to implement inhibitory biases in visuospatial attention.

In their seminal paper, Posner et al. (1985) first demonstrated different patterns of IOR as function of the site of brain damage. Most importantly, this study stimulated a line of neuropsychological research that has investigated the brain mechanisms of IOR. Although, Posner et al. (1985) suggested that the parietal lobe was not involved in the generation of this inhibitory effect, later studies (Bartolomeo et al., 1999, 2001) and our own results suggest that this phenomenon does reflect the function of attentional processes associated with the parietal lobe. In addition, our results do not support the hypothesis of a lateral disengagement deficit as an explanation for impaired IOR in parietal patients. However, this does not imply that a DD may not contribute to the pattern of performance of parietal patients in other tasks, or in patients with a complex clinical syndrome. For example, Bartolomeo and collaborators (Bartolomeo et al., 1999, 2001) have reported facilitation, instead of IOR, in the

ipsilesional field of a group of patients with unilateral neglect. This might reflect poor spatial disengagement to the ipsilateral event and/or that there is sustained attentional orienting to the ipsilesional side in neglect. In contrast, in the present group of patients who showed extinction without neglect, there may be an imbalance in spatial attention when new events are presented (so that an ipsilesional target overwhelms any IOR), without sustained chronic orienting to that side. Further studies in which patients with neglect and those with extinction are examined together may address this question.

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