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Impaired Orienting of Attention in Left Unilateral Neglect: A Componential Analysis

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Twenty-six patients suffering from damage to the right side of the brain, 19 of whom exhibited signs of left neglect, as well as 32 matched controls, ran 3 spatial cuing tasks. Patients were also tested with 2 cancellation tests, a line-bisection test, the copy of a complex drawing, and a visual extinction procedure. Results first showed correlations between extinction and cancellation tests performance on one hand, and between line bisection and copy on the other hand. Second, results demonstrated that an engagement deficit toward contralesional targets appeared to be the most striking feature of neglect, and the engagement score was correlated with the cancellation score and extinction. Most patients with neglect also presented a deficit in disengagement, a deficit of inhibition of return, and probably a deficit of alertness. Deficits in engagement and in disengagement, as well as poor scores in cancellation tests, seemed to be related with posterior cortical and subcortical lesions. Most important, even if an endogenous deficit (frequently related with a thalamic lesion) could aggravate the neglect behavior, neglect syndrome was mainly explained by a deficit of exogenous attention.

Keywords: spatial neglect, spatial cuing, right hemisphere brain damage, orienting of attention

Unilateral neglect is a relatively frequent and disabling disorder occurring after unilateral brain lesions, mainly in the right hemisphere. Patients with neglect syndrome may omit contralesional targets in cancellation tasks or left-sided details in copying tasks and may shift rightward the subjective center of horizontal lines. They typically show difficulties in orienting spatial selective attention toward the contralesional hemispace. A question remains as to the exact nature of the attentional deficit in neglect. Attention is not a unitary construct, and several components or elementary operations of spatial attention have been identified on the basis of the cuing paradigm developed by Posner and colleagues (Posner & Cohen, 1984; Posner, Walker, Friedrich, & Rafal, 1984). The main goal of the present study is to evaluate the relationship between these different attentional operations or components and unilateral neglect signs measured through paper-and-pencil tests.

Posner et al. (1984) tested patients with posterior parietal lesions and showed that, although detection of contralesional and ipsilesional targets evoked broadly similar response times (RTs), target detection in the contralesional hemifield was abnormally slowed when attention had previously been engaged on an invalid cue in the ipsilesional hemifield. The authors interpreted this deficit as a difficulty in disengaging attention from its current focus in order to move toward the contralesional direction. A disengage deficit has been demonstrated in patients with parietal lesions even when clinical signs of neglect or extinction were absent (Egly, Driver, & Rafal, 1994; Friedrich, Egly, Rafal, & Beck, 1998; Friedrich & Margolin, 1993; Posner et al., 1984; Posner, Walker, Friedrich, & Rafal, 1987). However, the severity of the disengage deficit has been correlated with the severity of clinical neglect (Morrow & Ratcliff, 1988).

Such a disengage deficit could explain why contralesional omissions on cancellation tasks are reduced when targets are erased instead of marked (Eglin, Robertson, & Knight, 1989; Mark, Kooistra, & Heilman, 1988). If the presence of the rightmost marked targets renders difficult the disengagement operation, the hypothesis is that disengaging would be facilitated with deletion of these targets. A disengage deficit could also explain why some patients are able to correctly describe the drawing of a flower and

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notice the petals in the contralesional part, while forgetting these petals when asked to copy the flower (Ishiai, Seki, Koyama, & Yokota, 1996). The copying task supposedly enhances focusing of attention toward the rightmost details of the drawing, from which patients are slow to disengage (see also the differences in dealing with hierarchical stimuli as a function of the task; Marshall & Halligan, 1995a; Worthington & Young, 1996). Moreover, it has been found that suppressing the visual feedback (by drawing with eyes closed) frequently reduces neglect signs compared with drawing under visual control (Chokron, Colliot, & Bartolomeo, 2004; Mesulam, 2000). However, a deficit in the operation of disengaging attention cannot account for several aspects of neglect behavior, and it has been argued that other attentional deficits might be present in patients with neglect syndrome. Indeed, in cancellation tasks, most patients still show a nonnegligible amount of neglect even if rightmost targets have been erased (Mark et al., 1988). Also, if the disengage deficit can explain why patients have difficulties in processing contralesional targets once they have engaged on ipsilesional targets, the question remains of why patients with neglect syndrome usually start to explore the external world by rightmost stimuli.

Losier and Klein (2001), in a meta-analysis of several previously published spatial cuing studies in neglect, showed that responses to validly cued targets in the contralesional hemispace were significantly slower than responses to validly cued targets in the ipsilesional hemispace, although the fact is hardly mentioned in most of the individual studies. They concluded that patients with neglect syndrome present an engage deficit of attention toward contralesional targets, because patients did not fully benefit from contralesional cues. An abnormal orienting bias of attention toward the ipsilesional hemispace (lateral preference) could be an important component of neglect (Gainotti, D'Erme, & Bartolomeo, 1991; Kinsbourne, 1993; Làdavas, 1993; Mattingley, Bradshaw, Nettleton, & Bradshaw, 1994). Patients with neglect syndrome frequently start scanning from the right side of a spatial display (for example, in cancellation tasks; see Jalas, Lindell, Brunila, Tenovuo, & Hämäläinen, 2002), a fact easier to explain by an abnormal ipsilesional capture of attention or a bias in engaging attention than by a disengage deficit, even if motor-directional akinesia could also, at least partially, explain this phenomenon. Finally, the mere appearance on the computer screen of the peripheral boxes in which a target can appear elicits a shift of patients' attention toward the rightmost box (D'Erme, Robertson, Bartolomeo, Daniele, & Gainotti, 1992), an experimental fact which is clearly in favor of an ipsilesional bias in engaging attention.

Another component of attention, inhibition of return (IOR), has also been implicated in neglect. In spatial cuing tasks using peripheral cues, RTs in valid trials are normally faster than RTs in invalid trials for short (less than 300 ms) stimulus onset asynchronies (SOAs), but the opposite pattern of results occurs for longer SOAs, with RTs being the slowest in valid trials, as if attention was inhibited from returning to previously explored objects (Maylor & Hockey, 1985; Posner & Cohen, 1984). An absence of IOR in the ipsilesional hemifield has been described in patients with neglect syndrome (Bartolomeo, Chokron, & Siéroff, 1999; Bartolomeo, Siéroff, Chokron, & Decaix, 2001) as well as in parietal patients without signs of neglect (Vivas, Humphreys, & Fuentes, 2003).

In summary, the orienting deficit in neglect could have at least three components: (a) a bias in engaging attention in favor of the ipsilesional hemispace, which would explain why patients process and respond to ipsilesional stimuli first even when stimuli are simultaneously presented in each hemispace; (b) a deficit in disengaging of attention, explaining why attention to contralesional stimuli is reduced when patients have engaged their attention on ipsilesional stimuli; (c) and a deficit of ipsilesional IOR, contributing to the favoring of ipsilesional "anchoring" of attention. A possibility is that the operations involved in neglect behavior are depending on the anatomical locus of the lesion. A distinction has been proposed with the disengage operation in the posterior parietal areas (Posner et al., 1984, 1987) or in the temporo-parietal junction (Friedrich et al., 1998), the engagement operation in the pulvinar (Rafal & Posner, 1987), and the IOR in the superior colliculus (Posner, Choate, Rafal, & Vaughn, 1985).

Whatever the exact nature of the orienting component involved in neglect, most authors seem to agree with a deficit of exogenous attention rather than endogenous attention (Bartolomeo, Siéroff, Decaix, & Chokron, 2001; Gainotti, 1996; Làdavas, 1993; Làdavas, Carletti, & Gori, 1994; Luo, Anderson, & Caramazza, 1998; Natale, Posteraro, Prior, & Marzi, 2005; Smania et al., 1998). In other words, patients present difficulties in orienting when exogenous events capture their attention, but their voluntary orienting is more or less intact and can overcome the exogenous deficit under some conditions. The spatial cuing method is well suited to dissociate endogenous and exogenous orienting of attention, specifically by varying the proportion of valid and invalid trials (see e.g., Berger, Henik, & Rafal, 2005). The disengage deficit in patients with neglect syndrome has been described with different cuing procedures, but is usually stronger in experiments using peripheral cues and exogenous attention conditions (no predictive information of the cue, i.e., in experiments using as many invalid trials as valid trials) than in experiments using central or symbolic (arrows) cues and endogenous conditions, namely when the cue is informative because most of the trials are valid (Losier & Klein, 2001). The disengage cost can even be reversed to an advantage for left invalidly cued targets, if most cues predict that the target will occur on the opposite side (Bartolomeo, Siéroff, Decaix, & Chokron, 2001). In this case, patients may use preserved endogenous processes to orient leftward after a right-sided cue and obtain relatively fast RTs to left invalidly cued targets. A predominant exogenous deficit in the neglect syndrome is also in agreement with brain imaging studies showing that exogenous orienting of spatial attention predominantly activates the right hemisphere in the ventral part of parietal and frontal regions overlapping those whose lesion provokes neglect (Corbetta & Shulman, 2002).

What is the relationship between performance in the spatial cuing task and performance in clinical neglect tests? Morrow and Ratcliff (1988), using an overall score of neglect with different tests, found a positive correlation between the disengage deficit and the severity of neglect. However, the exact relationship between each of the main neglect tests (cancellation, line bisection, copy) and the different component scores (disengagement, engagement, IOR) in the cuing task has not been established. Even if the different clinical tests of neglect are frequently disturbed in the same patients, dissociations have clearly been found, for example between cancellation and line bisection (Binder, Marshall, Lazar, Benjamin, & Mohr, 1992; Halligan & Marshall, 1992; Seki, 1996),

with most authors recognizing the composite nature of neglect. According to McGlinchey-Berroth et al. (1996), whether an individual will be impaired solely on one of these tests could not be predicted on the basis of lesion location. However, other authors have maintained that separable components of neglect may be associated with damage to discrete areas of the right hemisphere. Binder et al. (1992) found a relation between cancellation scores and frontal or deep lesions, on one hand, and between line bisection performance and posterior lesion on the other (see also Rorden, Fruhmann, Berger, & Karnath, 2006). Still, Marshall and Halligan (1995b) found the opposite pattern. We hypothesized that, if performance in one clinical test of neglect depends on the integrity of posterior areas in the brain, it should be correlated with elementary operations involved in orienting spatial attention, such as engagement or disengagement, the deficits of which follow posterior lesions.

Furthermore, authors frequently argue for some common underlying deficit in visual extinction and clinical neglect (see Driver & Vuilleumier, 2001). However, visual extinction has been found using different materials in left hemisphere lesions as well as in right hemisphere lesions, although neglect is prevalent following right hemisphere lesions (Friedland & Weinstein, 1977; Siéroff & Michel, 1987). Also, the lesion site may differ between extinction and neglect (Karnath, Himmelbach, & Küker, 2002; Vallar, Rusconi, Bignamini, Geminiani, & Perani, 1994). The relationship between the extinction phenomenon and the deficits in the different components of attentional orienting or the deficits in paper-and-pencil neglect tests remains to be established.

In this study, we present the results of three cuing experiments using peripheral cues and the detection of simple targets in patients suffering from a right hemispheric lesion. These patients were distinguished by the presence or absence of symptoms associated with left neglect syndrome. The experiments differed only in the proportion of information given by the cue in each condition. In the first experiment, in which 50% were valid trials and 50% were invalid trials, the cue provided no information on the location of the target. Thus, RT differences between valid and invalid trials were supposedly caused by exogenous orienting of attention. The second experiment used informational cues (80% valid trials), and effects should have been at least partially caused by endogenous orienting of attention. Although central cues are typical in this type of experiment, we preferred to use the same peripheral cues as in the previous experiment, to make results comparable. In the third experiment, most trials were invalid: For example, a right cue was followed by a left target in 80% of the cases and by a right target in 20% of the cases only. Here, the endogenous component of orienting of attention consists of the ability to inhibit the attentional capture by the cue and to reorient attention from the location of the cue to the opposite location.

This study aims to evaluate the different components of attention (disengagement, engagement, and IOR) in three groups: those suffering from right brain damage *with* signs of neglect, those suffering from right brain damage *without* signs of neglect, and age-matched healthy participants. By comparing results between the three experiments, we determine the endogenous or exogenous nature of the deficits. Finally, we calculate correlations between scores evaluating the different components of attention and patients' performance on clinical tests of neglect and of extinction.

Method

Participants

Twenty-six patients (13 men and 13 women) and 32 controls (13 men and 19 women) participated in the study. All participants were right-handed. Mean age was 63.96 years (SD = 13.09, range = 29–80) for patients and 60.19 (SD = 13.00, range = 39–81) for controls. Mean sociocultural level was 4.82 (SD = 1.85) for patients and 4.81 (SD = 1.47) for controls. Table 1 shows the demographic and clinical characteristics of patients. Figure 1 and Table 2 show the anatomical sites of the lesion for the 14 patients who had available MRI or CT scan.

Patients were selected on the basis of the lesion location in the right hemisphere and the absence of hemianopia (all patients had full visual field to confrontation within 30° of fixation). The study was carried out by following the guidelines of the Ethics Committee of the Cochin Hospital in Paris.

Clinical Tests

Unilateral neglect was assessed by means of a battery of paper-and-pencil neglect tests (Bartolomeo & Chokron, 1999a), including tasks of target cancellation, line bisection, and drawing copy. Only the 26 patients performed these tests.

Line bisection. Patients were asked to mark the middle of 8 lines of 1-mm width and of different lengths (6, 18, 10, 10, 18, 6, 6, and 10 cm), arranged on the left, the middle, or the right part of a vertical A4 sheet of paper. Deviation from the true middle was measured in mm. Then, a line-bisection score of rightward deviation was calculated: The deviation from the middle was expressed as a percentage of half the length of the line. A positive score indicates a rightward deviation, whereas a negative one indicates a leftward deviation. Pathological left neglect scores correspond to rightward deviations superior to 11.1% (Bartolomeo & Chokron, 1999a).

Bells cancellation test (Gauthier, Dehaut, & Joanette, 1989). Patients were asked to circle 35 targets (black-ink drawings of bells) presented on an horizontal A4 sheet, among 280 distracters. Targets were presented in a pseudorandomized way and were equally distributed in seven columns. Only the targets of the three lateral columns were taken into consideration (15 targets each). The bells laterality score is the difference between the number of cancelled bells on the left and on the right. Positive scores indicate more omissions in the left half than in the right half. Scores superior to 2 are considered pathological (Rousseaux et al., 2001).

Albert cancellation test (Albert, 1973). Patients were asked to mark all 60 of the short lines randomly presented on a horizontal A4 sheet. The Albert laterality score is the difference between the number of cancelled lines on the left and on the right (30 lines each). Positive scores indicate more omissions in the left half than in the right half. Scores are considered as pathological when superior to 2.

Copy. Patients had to copy a scene on a horizontal A4 sheet (Gainotti, D'Erme, Monteleone, & Silveri, 1986). The total score is 6 points: 1 for the trees on the right (omission of the left half of

Table 1
Demographic and Clinical Characteristics of Patients

Patients	Sex	Age	Sociocultural level	Onset of illness (days)	Hemiplegia	Anosognosia	Visual extinction	Neglect tests: Bisection deviation	Neglect tests: Bells score	Neglect tests: Albert score	Neglect tests: Copy	*Z* score	Pathological neglect tests
AD	M	60	2	45	**Y**	N	**13**	**16.9**	0	**9**	2	0.87	**3**
BL	F	67	3	92	**Y**	N	**11**	−2.6	1	**4**	0	−0.49	**1**
CB	F	47	7	0	N	N	0	**17.9**	0	0	0	−0.26	**1**
DB	M	48	7	30	Y	N	0	10.4	**5**	**4**	1	0.13	**3**
DM	F	72	7	98	**Y**	Y	**13**	1.9	**4**	**3**	0	−0.18	**2**
HC	M	74	3	184	**Y**	N	0	**11.8**	0	0	0	−0.45	**1**
JB	F	70	5	19	**Y**	N	**18**	**16.9**	**5**	**9**	2.5	1.39	**4**
JD1	M	61	5	296	N	N	**15**	10.7	**6**	**7**	0	0.34	**2**
JD2	M	73	6	278	**Y**	N	**16**	8.8	**6**	**5**	0	0.26	**2**
JL1	M	75	3	12	**Y**	N	0	−0.5	**5**	−2	0	−0.66	**1**
JL2	F	52	7	110	**Y**	N	0	6.3	**4**	1	0	−0.42	**1**
MB1	F	80	4	40	Y	N	**18**	7.9	**8**	**23**	0	0.77	**2**
MB2	F	80	4	35	**Y**	N	3	**16.9**	**13**	**23**	1	1.21	**4**
MM	M	60	7	74	**Y**	N	0	**17.6**	1	0	**0.5**	−0.07	**2**
MS	F	75	3	50	**Y**	Y	**18**	8.4	**15**	**26**	**0.5**	1.33	**3**
MV1	M	66	2	36	**Y**	N	0	1.4	2	**6**	0	−0.57	**1**
MV2	M	73	4	66	**Y**	N	**18**	10.9	0	**24**	0	0.52	**1**
PB	F	78	6	28	N	N	**5**	**15.5**	**11**	**24**	**0.5**	0.99	**4**
VG	F	29	7	280	N	N	0	1.6	**6**	0	0	−0.50	**1**
BN	M	44	3	85	N	N	0	3.2	0	0	0	−0.73	**0**
EN	F	52	6	343	**Y**	N	0	−0.7	1	0	0	−0.81	**0**
HL	F	79	3	71	**Y**	N	0	10.0	0	0	0	−0.51	**0**
HT	M	58	7	23	N	N	0	8.8	−0	2	0	−0.51	**0**
JC	M	52	7	290	N	N	0	4.6	−1	0	0	−0.73	**0**
MH	M	68	4	179	**Y**	N	0	4.6	2	0	0	−0.59	**0**
SM	F	70	3	32	**Y**	N	**11**	4.2	1	2	0	−0.32	**0**

Note. Pathological scores are in bold. M = male; F = female; Y = yes; N = no.

a tree = 0.5), 2 for the house (omission of a window, the door or a part of the roof = 0.5; omission of the left half of the house = 1), and 1 for the trees on the left. Scores are considered as pathological when superior or equal to 0.5.

Extinction. The presence of visual extinction was clinically tested by briefly wiggling fingers for 2 s in one or both visual fields. The examiner controlled central gaze fixation, and 36 trials were given in a fixed pseudorandom sequence including 18 unilateral trials (9 on each side) and 18 simultaneous bilateral trials. Extinction was considered as present when a patient failed at least once to report a contralesional stimulus during bilateral simultaneous presentation, while accurately detecting unilateral stimuli (Azouvi et al., 2002). An extinction score was calculated by the difference between correct detection on the right and on the left (maximum = 18) in the bilateral condition.

Response Time to Visual Targets

In all experiments, participants sat facing a computer monitor at a distance of approximately 50–60 cm. Stimulus presentation and response collection were controlled by the Psychlab software (Bub & Gum, 1995). The method was directly inspired from the method used by Posner et al. (1984).

Each trial began with the appearance of a horizontal display of three black unfilled square boxes on a white background. Each square box was 10 mm wide (approximately 1° of visual angle) and each side of the square was 0.35 mm thick. The distance between the boxes was 30 mm (approximately 3° of visual angle). Patients were instructed to fixate a black dot located in the central box (remaining present in the whole trial). Eye movements were observed by one of the experimenters, so that such trials could be discarded; however, discarded trials were not replaced by new trials. After 500 ms, a cue (thickening of the boxes by 0.7 mm) followed during 300 ms. The target (a black asterisk, 0.5° of visual angle in diameter) appeared in one of the peripheral boxes after a variable delay (SOA = 100, 500 or 1,000 ms) from the cue and remained visible until a response was made. The task was to press the space bar of the keyboard as quickly as possible with the index finger of the right hand as soon as the target appeared. Patients were instructed to respond exclusively to the target and not to the cue. The intertrial interval was 1,500 ms.

There were three cuing conditions. On valid trials, the target appeared at the same location as the cue, whereas on invalid trials the target appeared on the side opposite the cue. Before each experiment, participants were informed about the level of predictability of the cue. In Experiment 1, 50% of valid trials and 50% of invalid trials were presented (for a total of 252 trials, with three blocks of 84 trials each). In Experiment 2, 80% of valid trials and 20% of invalid trials were presented (for a total of 270 trials, with three blocks of 90 trials). In Experiment 3, 20% of valid trials and 80% of invalid trials were presented (for a total of 270 trials, with three blocks of 90 trials). Also, neutral trials were presented in separate blocks, with the neutral cue consisting of the thickening of the central

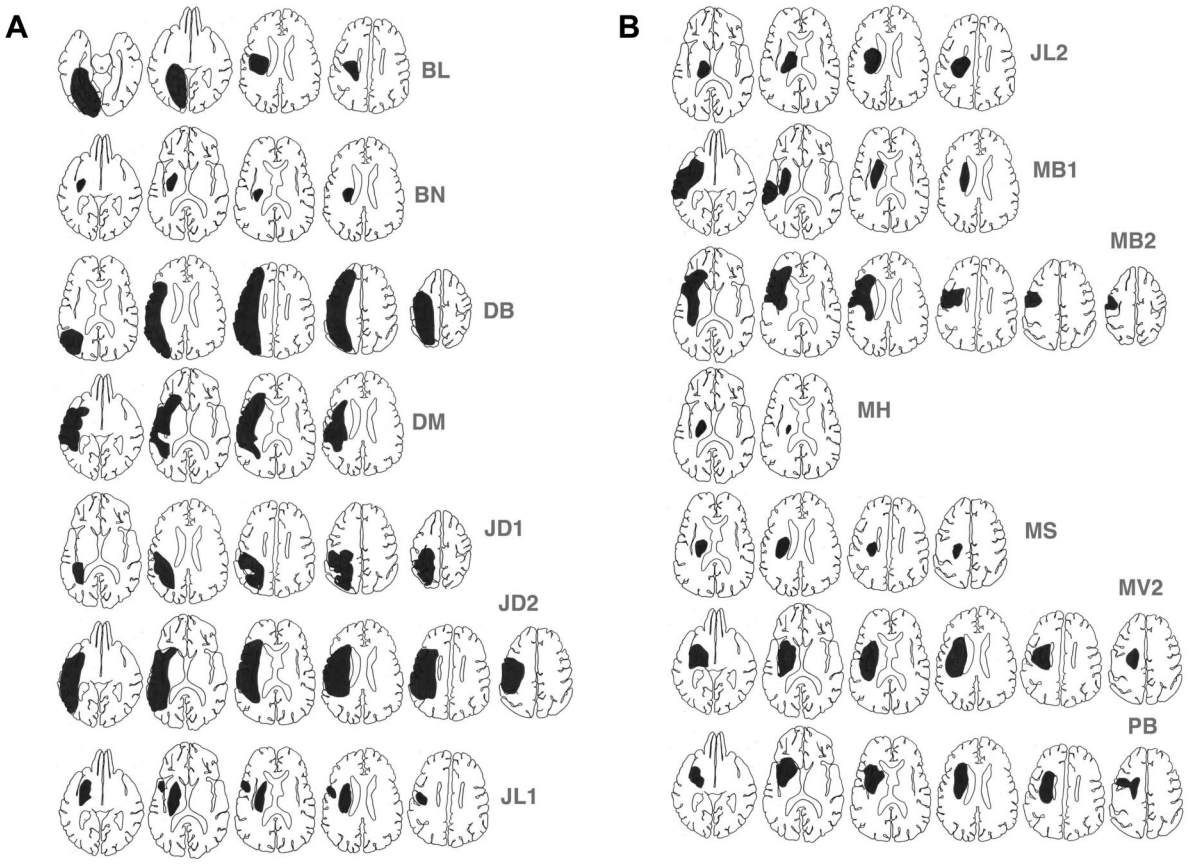

Figure 1. A and B: MRI/CT scans of patients' lesions plotted on Damasio and Damasio's (1989) templates (some of the slices are missing for patient BL). Adapted with permission from *Lesion Analysis in Neuropsychology*, by H. Damasio and A. R. Damasio, 1989, New York: Oxford University Press. Copyright 1989 by Oxford University Press.

square box (for a total of 252 trials, with three blocks of 84 trials). Note, however, that this condition may not represent a true neutral condition in patients with neglect syndrome, as suggested by Posner et al. (1984; see results of Experiment 1 for further discussion). Blocks of trials, corresponding to the three experiments varying the percentage of valid and invalid trials and to the neutral condition, were presented in a counterbalanced way over three sessions separated by 1 or 2 days in patients and 1 hr in healthy controls. Rest periods were provided between each block of a session. Participants practiced the task (30 trials) before the data was collected while the experimenter observed to ascertain that the directions were understood and that the participant was not making eye movements.

Results

Analyses of Results

Clinical tests were analyzed by use of raw scores for copy and line bisection and the right–left difference for extinction and both cancellation tests (Albert, bells). Correlations were calculated between clinical tests themselves and between tests and elementary operations revealed by the cuing task. However, because more than half of the patients were at ceiling in the extinction and the copy tasks, an analysis considering only the failure or the success of these tests was conducted, and *t* tests were calculated when correlations were not significant. Because of the finding of a positive correlation between the disengage deficit and the severity of neglect using an overall score (Morrow & Ratcliff, 1988), an overall neglect score was calculated by converting and averaging scores of the four neglect tests and of extinction test to *z* scores.

In the cuing experiments, RTs exceeding the range of 150–5,000 ms were discarded from the analysis. For each experiment, median RTs were entered in a repeated-measures ANOVA, with group (patients with neglect, patients without neglect, healthy controls) as a between-factors variable, and field (left, right), cue type (valid, invalid, neutral) and SOA (100 ms, 500 ms, 1,000 ms) as within-factor variables.

We also tried to relate the neuroanatomical findings to the behavioral data. However, MRI or CT-scan was available for 14 patients only. Because of the diversity of the lesions presented by these patients (see Figure 1 and Table 2), multiple component analyses were not conclusive. Consequently, we chose to argue mainly on the basis of the dissociations between patients.

Table 2
Anatomical Sites of the Lesions in Patients for Whom We Have Brain Imagery, and Pathological Scores for the Experiments and Clinical Tests

	Patients													
Lesion sites, experiments, and clinical tests	BL	DB	DM	JD1	JD2	JL1	JL2	MB1	MB2	MS	MV2	PB	MH	BN
Lesion sites														
Frontal lobe														
Lateral: rolandic region		++	+		++	++			++			++		
Operculum		++			++				++					
Parietal lobe														
Inferior		++	+	++										
Lateral		++		++										
Para-/supraventricular	++			++										
Temporal lobe: lateral														
Inferior gyrus (posterior)					++									
Middle gyrus (posterior)			++	++	++			++						
Posterior to auditory region			++		++									
Auditory region			++		++				++					
Anterior to auditory region			++		++	++			++			++		
Middle gyrus (anterior)			++		++			++						
Temporal lobe: mesial														
Anterior (amygdala)												++		
Posterior (hippocampus)											++			
Occipital lobe														
Mesial	++													
Lateral: inferior					++									
Paraventricular area	++			++	++									
Insula			++		++	++			++			++		++
Subcortical														
Head of caudate nucleus			+		+	++		+	+			++		
Body of caudate nucleus						++								
Lenticular nucleus/internal capsule	+		+		+	++	++	++	+	+	+	++		
Thalamus: anterior part												+		
Thalamus: posterior part	++						++			++	++			
Thalamus: lateral part	+					++	+			+	+		++	
Experiments														
Experiment 1														
Disengagement SOA 100	+			+	+	+	+	+			+	+		
Disengagement SOA 500	+	+		+	+	+	+		+	+	+	+		
Engagement SOA 100	+	+	+	+	+					+	+	+		
Engagement SOA 500	+		+	+	+					+		+		
Engagement SOA 1,000	+	+	+	+	+	+			+	+	+	+		
Right IOR	+		+								+	+		
Experiment 2														
Disengagement SOA 100				+				+		+	+			
Engagement SOA 100	+	+	+	+	+					+	+	+		
Engagement SOA 500	+	+	+	+	+					+	+	+		
Engagement SOA 1,000	+		+	+	+					+	+	+		
Experiment 3														
Inhibition of RVF cue	POS		+	POS		POS			POS	+	POS	POS		
Reorienting of attention			+	+		POS			POS	+	+	+		
Clinical tests														
Bisection deviation								+				+		
Bells score		+	+	+	+	+	+	+	+	+		+		
Albert score	+	+	+	+	+			+	+	+	+	+		
Copy		+							+	+		+		
Exinction	+		+	+	+			+		+	+	+		

Note. The signs + or ++ indicate a lesion or a pathological score. In Experiment 3, "POS" indicates that patients were able to inhibit the right hemifield capture of attention or to efficiently reorient attention toward the left hemifield. SOA = stimulus onset asynchrony; IOR = inhibition of return; RVF = right visual field.

Results in Clinical Tests

Table 1 shows the performance of the patients on the neglect battery and on the test of visual extinction. Nineteen patients had at least one pathological score in neglect tests (cancellation, bisection, copy) and, consequently, were considered as suffering from neglect. They form the right brain damage group with neglect syndrome, or RBDN+ group. Seven patients with right brain damage had no pathological score on neglect tests. They form the RBDN− group.

We analyzed and compared the performance in the visual extinction task, two cancellation tests (Albert test, bells test), and the line-bisection and copy tasks in the 26 patients. As could be expected, performance on both cancellation tests (Albert and bells tests) was positively correlated ($r = .69$, $p < .01$). Also, the Albert test performance was correlated with extinction ($r = .60$, $p < .01$). However, neither the cancellation tests nor the extinction test correlated with the line-bisection task ($r = .37$ for the Albert test, $r = .06$ for the bells test, $r = .15$ of the extinction score). Finally, only the line-bisection test correlated with the copy task ($r = .58$, $p < .01$).

As can be seen in Tables 1 and 2, clear dissociations emerged between the different tests. Only 4 patients showed a deficit in all five clinical tests. The anatomical data were available for only two of them (MB2 and PB). Both of them revealed a lesion involving the inferior frontal and the superior part of anterior-middle temporal cortex, the anterior part of the capsulo-lenticular region. This result is consistent with recent data (Karnath, Fruhmann Berger, Küker, & Rorden, 2004; Karnath, Himmelbach, & Rorden, 2002), even if the parietal lobe has also been incriminated in neglect syndrome and in extinction (see Doricchi & Tomaiuolo, 2003; Mort et al., 2003). Cases of neglect are frequently caused by lesions in different parts of the right hemisphere, the neglect syndrome being now considered as a deficit of a complex attentional network (Mesulam, 2000).

Seven patients presented a left extinction and a pathological score in one or two cancellation tests without a pathological rightward deviation in the line-bisection test (BL, DM, JD1, JD2, MB1, MS, and MV2). Five lesions involved the temporal lobe, three involved the parietal lobe, four involved the capsulo-lenticular region, and three involved the thalamus. Five other patients presented a deficit in at least one cancellation test but no extinction and no deficit in the bisection test. These patients had a lesser deficit on the cancellation tests than the previous patients. Scans were available for only three of them (DB, JL1, and JL2). One showed a parietal lesion and one showed a lesion of the temporal lobe; all patients also showed a thalamic lesion. Overall, only 3 of the patients presenting a deficit in cancellation tests (with or without extinction) and not in the bisection test had a lesion involving the frontal lobe (DB, JD2, and JL1).

Finally, three patients presented a clear rightward deviation in the line-bisection test without any deficit in any of the cancellation tests and without extinction. Unfortunately, no scan was available for these patients. Note, however, that the 2 patients showing a deficit in the line-bisection test (as well as in other tests), for whom we have precise anatomical data on the lesion site (MB2 and PB), seemed to present lesions including more anterior regions (lateral and posterior frontal areas, anterior part of the superior temporal gyrus, and anterior part of the capsulo-lenticular area).

In conclusion, these results are compatible with Marshall and Halligan's (1995b) hypothesis of a relationship between cancellation deficit and posterior lesions (temporal and parietal lobes, posterior subcortical structures), and between line bisection deficit and more anterior lesions, although exceptions can be found.

Experiment 1

In Experiment 1, the ratio of valid and invalid conditions was 50:50. The peripheral cue occurred at the target location in only 50% of the trials, thus providing no useful information. With this type of experiment using noninformative cues, exogenous orienting of attention can be evaluated without much influence from endogenous components, at least when the differential effects of the cue are considered. Results for the three groups are presented in Figure 2.

Global RT analysis. All main effects were significant. The main effect of Group, $F(2, 55) = 29.46$, $MSE = 639E{+}03$, $p < .01$, reflected slower RTs for RBDN+ patients ($M = 833$ ms, $SE = 65$ ms) and for RBDN− patients ($M = 700$ ms, $SE = 86$ ms) than for the control group ($M = 424$ ms, $SE = 15$ ms). However, RTs were not significantly different between both groups of patients, $F(1, 55) = 2.57$, $MSE = 639E{+}03$, *ns*. This result is congruent with the usual finding of slowness following a right hemisphere lesion, a deficit, which could be related, at least partially, to a difficulty in being alerted by external events (Posner & Petersen, 1990).

There was a main effect of SOA, $F(2, 110) = 32.59$, $MSE = 20.8E{+}03$, $p < .01$, and the interaction of Group × SOA, $F(4, 110) = 3.84$, $MSE = 20.8E{+}03$, $p < .01$, reflected a stronger effect of the delay between the cue and the target (for difference between the shortest and the longest SOAs, $M = 135$ ms, $SE = 29$ ms) in RBDN+ patients than in controls ($M = 63$ ms, $SE = 10$ ms), with the difference obtained in RBDN− patients ($M = 103$ ms, $SE = 47$ ms) being intermediate. The stronger effect of SOA found in RBDN+ patients compared with other groups could simply be explained by the fact that RTs were slower in RBDN+ patients, leaving room for a strong improvement with SOA. In this case the effect should be stronger in slower left hemifield targets. However, the interaction of Group × Field × SOA was not significant, $F(4, 110) = 1.94$, $MSE = 13.7E{+}03$, *ns*. Another explanation is that patients with neglect syndrome present an additional deficit of alertness (Posner & Petersen, 1990; Robertson, 1993). In our procedure, the boxes in which the target was presented disappeared at the end of each trial and reappeared at the beginning of each trial, 500 ms before the appearance of the cue, which is different from the procedure used by Posner et al. (1984), in which the boxes were always present on the screen. A possibility is that in control participants, the first event (sudden appearance of the boxes) could summon a maximum of alert effect at a fast rate, and the effect of SOA after the cue would consequently be less visible. In RBDN+ patients, the alerting effect caused by the sudden boxes appearance was smaller (or slower), and alert could be additionally recruited by another following event, the cue (the visual cue can also act as an alerting device; Fernandez-Duque & Posner, 1997; Posner, 1978). If the difference in the SOA effect is actually due to a deficit in alertness in patients with neglect syndrome, it could explain why the effect did not interact with the hemifield, alertness being a change in the internal state indepen-

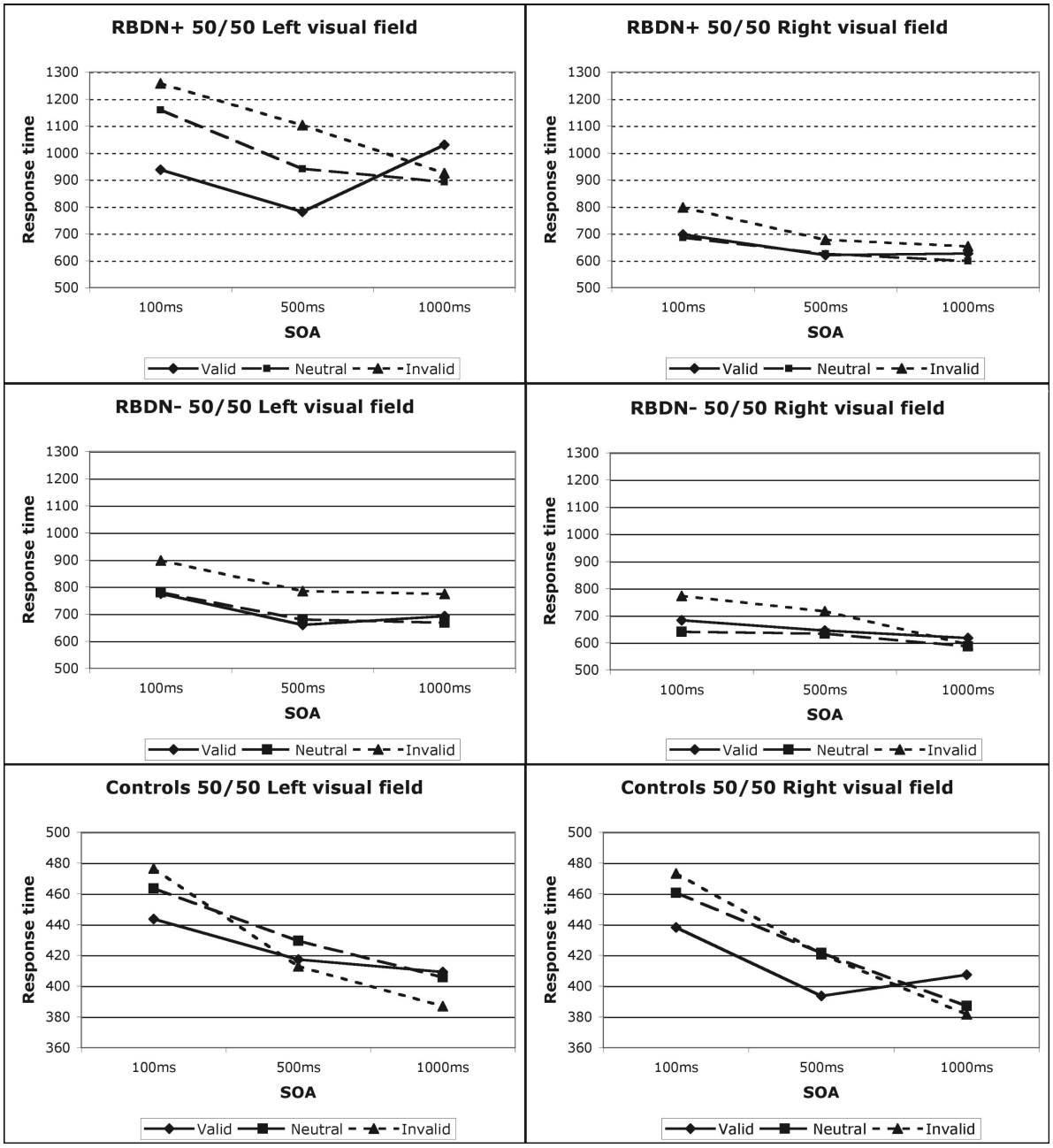

Figure 2. Response times for the three groups in Experiment 1: RBDN+ patients (right brain damage with left neglect syndrome), RBDN− patients (without neglect syndrome), and controls.

dent from orienting (Fernandez-Duque & Posner, 1997). A deficit in alertness should be found with right hemifield targets as well as with left hemifield targets.

The field effect, $F(1, 55) = 20.74$, $MSE = 182E{+}03$, $p < .01$, showed faster RTs for the right hemifield, but the interaction of Group × Field was also significant, $F(2, 55) = 16.27$, $MSE = 182E{+}03$, $p < .01$. Only the RBDN+ patients showed a field effect (in favor of right hemifield targets, $M = +338$ ms, $SE = 78$ ms), $F(1, 55) = 53.61$, $MSE = 182E{+}03$, $p < .01$, the difference between both hemifield being not significant in RBDN− patients ($M = +92$ ms, $SE = 62$ ms) and in controls ($M = +7$ ms, $SE = 3$ ms). Because of the small number of patients in the RBDN− group and their heterogeneity, results considering this group have to be taken cautiously. Still, it remains interesting that only the RBDN+ patients showed faster RTs for right hemifield targets than for left hemifield targets, even when global measures independent from validity effects were used.

Finally, the cue type was also significant, $F(2, 110) = 11.04$, $MSE = 29.2E{+}03$, $p < .01$. Of great interest is the significant interaction of Group × Field × Cue Type × SOA, $F(8,$

220) = 6.04, MSE = 8.7E+03, $p < .01$. Further analysis explored the deficits in disengagement of attention, engagement of attention and IOR in neglect and found differences, which can clarify this interaction.

Disengagement of attention. An initial question is whether patients with neglect syndrome show a specific deficit in disengaging attention from right-sided ipsilesional stimuli before moving it toward left-sided contralesional stimuli. To measure a differential deficit of disengagement between hemifields, we calculated the difference between valid and invalid conditions in the left and in the right hemifield, then a disengage score (Losier & Klein, 2001; Morrow & Ratcliff, 1988), as follows: (left invalid – left valid) – (right invalid – right valid).

RBDN+ patients showed a larger disengagement effect for left hemifield targets (M = +319 ms, SE = 74 ms; M = +321 ms, SE = 106 ms, respectively) than for right hemifield targets (M = +101 ms, SE = 47 ms; M = +57 ms, SE = 35 ms, respectively) at the shortest (100 ms) SOA, $F(1, 55) = 34.95$, MSE = 6.5E+03, $p < .01$, and at the 500-ms SOA, $F(1, 55) = 19.17$, MSE = 17.3E+03, $p < .01$. The disengage score was +218 ms (SE = 57 ms) at the 100-ms SOA and +264 ms (SE = 103 ms) at the 500-ms SOA. RBDN− patients did not show a significant difference of disengagement between hemifields (for disengage score at the 100 ms SOA, M = +32 ms, SE = 71 ms; at the 500-ms SOA, M = +53 ms, SE = 38 ms), and neither did controls (M = −2 ms, SE = 10 ms; M = −32 ms, SE = 10 ms). The asymmetry of disengagement was significantly larger in patients with neglect syndrome than in controls at the shortest 100-ms SOA, $F(1, 55) = 22.38$, MSE = 6.5E+03, $p < .01$, as well as at the 500-ms SOA, $F(1, 55) = 15.07$, MSE = 17.3E+03, $p < .01$, showing a clear disengage deficit. RBDN− patients did not differ from the control group at both these SOAs. The asymmetry of disengagement was also significantly larger in patients with neglect syndrome than in RBDN− patients at the 100-ms SOA, $F(1, 55) = 6.83$, MSE = 6.5E+03, $p < .05$. Our results confirm the strong deficit in disengaging attention from an ipsilesional cue in patients with neglect syndrome in order to orient toward contralesional targets (Morrow & Ratcliff, 1988), specifically for short and medium SOAs (100 ms and 500 ms).

Note that an asymmetry for patients with neglect syndrome was also found when we considered the difference between valid and neutral conditions at both 100- and 500-ms SOAs (M = +233 ms, SE = 72 ms; M = +156 ms, SE = 59 ms, respectively), showing that neutral conditions using a cue in the central box can elicit a deficit in disengagement (Posner et al., 1984). In other words, the disengage difficulty of patients with neglect syndrome occurs whenever the cue is located to the right of the target, regardless of whether it occurs in the opposite ipsilesional hemifield (Posner et al., 1987).

Also, if patients with neglect syndrome showed a larger valid–invalid difference for left targets than for right targets as compared with controls, note that there was a tendency for a disengage deficit at the 100-ms SOA, even for right hemifield targets in RBDN+ patients (M = 101 ms, SE = 47 ms) compared with the control group (M = 35 ms, SE = 9 ms), $F(1, 55) = 3.23$, MSE = 8.05E+03, $p = .078$.

To document the relationship between the contralesional disengage deficit and the severity of neglect, we compared scores obtained in neglect tests with the disengage score. Results from all 26 of the patients were entered in the analysis. The disengage score at the 100-ms SOA did not correlate with any of the clinical tests: Albert test (r = .09), bells test (r = .02), line-bisection task (r = −.03), and copy task (r = .03). Finally, no correlation was found between the disengage deficit and the overall neglect score (r = .12). Similar results were obtained when only those 19 patients actually showing neglect were entered in the analysis. Morrow and Ratcliff (1988) found no correlation between the severity of neglect and the contralesional disengage deficit in left hemispheric patients (n = 10), but a strong correlation in right hemispheric patients (n = 12). Our results are in partial agreement with theirs. A stronger disengage deficit was found in RBDN+ patients than in RBDN− patients, showing that the disengage deficit is a rather specific feature of neglect, but its magnitude seems to have no specific relationship with the severity of neglect. This may be due to differences between our analysis and that of Morrow and Ratcliff. First, the number of patients in their study was only 12 (for the right hemispheric group). Second, they calculated an overall score of neglect, using performance on a letter-cancellation test, line-bisection test, and the copy of the Rey figure. Our measures are more precise in evaluating correlations with each of the different clinical tests, but they did not include the copy of the Rey figure. Third, their cuing procedure was slightly different from ours, because they used informative cues (75% valid trials with peripheral cues) and calculated correlations for the 50-ms SOA. We examine, in Experiment 2, whether the disengage deficit is correlated with clinical tests when cues are informative.

The disengage deficit has been called the *extinction-like* deficit (Posner et al., 1984), because the invalid condition (the cue on one side and the target on the other) resembles the typical extinction in which one stimulus is presented on each side of the fixation. In our study, no significant correlation occurred between the extinction score and the disengage score (r = .26), perhaps because many patients were at ceiling on the extinction test. However, as expected, patients with extinction had a disengage deficit (M = 281 ms, SE = 76 ms), which was significantly larger than that showed by patients without extinction (M = 83 ms, SE = 59 ms) $t(23) = 2.09$, $p < .05$.

Fourteen of the 19 RBDN+ patients (3 of the 7 RBDN− patients) showed a valid–invalid difference for left hemifield targets at more than two standard deviations from the controls' mean. Eleven of these patients showing a disengage deficit had brain imagery (see Table 2). Lesions involved various parts of the brain: parieto-occipital (BL, DB, and JD1), postero-temporal (JD1, JD2, and MB1), occipital (BL, JD1, and JD2), and subcortical (JD2, JL1, JL2, MB1, MB2, MS, MV2, and PB). Three patients with imagery did not present a disengage deficit (BN, DM, and MH); none of these patients had a large lesion in the parietal or the occipital lobe, and DM had a large temporal lesion. We found the same result when considering other patients for whom we had only the reports of their scan (AD, HL, and VG). So, apparently, a disengage deficit is most probable when the lesion involves the posterior part of the right hemisphere, including the parietal lobe, but a right parietal lesion is not necessary, and other lesions sparing the right posterior cortex can give rise to a disengage deficit.

Interestingly, 6 RBDN+ patients and 2 RBDN− patients showed a valid–invalid difference for right hemifield targets at more than two standard deviations from the controls' mean. All 6

of the RBDN+ patients also had a deficit for left hemifield targets, and this left hemifield deficit was even stronger than the deficit for right hemifield targets in 4 patients. Among these 6 patients, 5 had a lesion including the lenticular capsule (JL1, MB2, MS, MV2, and PB) and 5 had a cortical lesion, including the frontal rolandic areas (JL1, MB2, and PB), the anterior part of the temporal lobe (JL1, MB2, and PB), the mesial part of the temporal lobe (MV2), and the parieto-occipital areas (BL). Thus, lesions seem slightly more anterior in this group.

Engagement of attention. The engagement component of orienting was evaluated with the score (left valid) – (right valid), following Losier and Klein (2001). RBDN+ patients showed significantly larger differences between left and right valid conditions than the control group. At the 100-ms SOA, the engage score was +241 ms in favor of right hemifield targets (SE = 96 ms) for RBDN+ patients and +5 ms (SE = 5 ms) for controls, $F(1, 55) = 11.07$, $MSE = 29.9\text{E}{+}03$, $p < .01$; +161 ms (SE = 70 ms) and +24 ms (SE = 7 ms) at the 500-ms SOA, $F(1, 55) = 6.98$, $MSE = 16.0\text{E}{+}03$, $p < .05$; and +403 ms (SE = 108 ms) and +2 ms (SE = 8 ms) at the 1,000 ms SOA, $F(1, 55) = 24.54$, $MSE = 39.1\text{E}{+}03$, $p < .01$. The RBDN− patients were at an intermediate level (M = +93 ms, SE = 51 ms; M = +15 ms, SE = 34 ms; M = +75 ms, SE = 73 ms). This result is in agreement with Losier and Klein (2001) and shows the importance of the deficit in engaging spatial attention toward contralesional targets in the neglect syndrome.

Considering the 26 patients, the 100-ms SOA engage score was correlated with performance in the Albert test ($r = .53$, $p < .01$) and in the bells test ($r = .52$, $p < .01$), but not with the performance in the line-bisection test ($r = .13$) or the copy test ($r = .20$). The correlation with extinction was weaker but still significant ($r = .39$, $p < .05$). Similar results were obtained when we considered only the 19 patients with neglect. Patients with visual extinction had a 100-ms SOA engage score (M = 432 ms, SE = 140 ms) significantly stronger than patients without extinction (M = 29 ms, SE = 33 ms), $t(23) = 3.1$, $p < .01$. A similar result was obtained in patients who showed a copying deficit (M = 455 ms, SE = 141 ms) and in those who did not (M = 108 ms, SE = 62 ms), $t(23) = 2.3$, $p < .05$. The positive correlation between the engage deficit and the overall neglect score was also significant ($r = .50$, $p < .01$). Also, the engage score at the 1,000-ms SOA correlated with the Albert test score ($r = .52$, $p < .01$).

Ten of the 19 RBDN+ patients and 4 of the 7 RBDN− patients showed a 100-ms SOA engage score at more than two standard deviations from the controls' mean. Interestingly, 6 patients (CB, HC, JL1, JL2, MB1, and MV1) presented a pathological disengage score without a pathological engage score, and 3 patients showed the opposite pattern (DM, JC, and VG). Parieto-occipital, posterior temporal, caudate, capsulo-lenticular, and thalamic lesions are all concerned by an engage deficit (see Table 2). However, none of these regions (not even the thalamus) seems crucial for this attentional operation because some patients whose lesions involved these regions (for posterior temporal, MB1; for subcortical, BN, JL2, MB1, and MH) did not present any engage deficit.

IOR. IOR was evaluated by the invalid–valid difference in each visual field in the longest SOA condition (1,000 ms). In the right hemifield, IOR was −26 ms (SE = 7 ms) in the control group and −24 ms (SE = 18 ms) in RBDN− patients; however, RBDN+ patients had faster RTs for valid trials than for invalid trials (M = +26 ms, SE = 22 ms). The difference between RBDN+ patients and controls was significant, $F(1, 55) = 7.72$, $MSE = 2.07\text{E}{+}03$, $p < .01$, and there was a tendency for a difference between RBDN+ patients and RBDN− patients, $F(1, 55) = 3.11$, $MSE = 2.07\text{E}{+}03$, $p = .083$. Thus, our results clearly indicate an IOR deficit for ipsilesional right hemifield targets in patients with neglect syndrome, even replaced by facilitation—a result, that is consistent with several previous results (Bartolomeo et al., 1999; Bartolomeo, Siéroff, Decaix, & Chokron, 2001). Also, there was a correlation between the amount of disengage deficit for left targets at the 100-ms SOA and the amount of facilitation of return ($r = .46$, $p < .05$). As suggested elsewhere (Bartolomeo et al., 1999), facilitation of return in patients with neglect syndrome could be caused, at least partially, by the fact that once a cue occurred in the right hemifield, patients had difficulties in disengaging from the cue location, thus facilitating this location for an abnormally long period of time.

The difference between valid and invalid trials in the 1,000-ms SOA condition (IOR) in the right hemifield did not correlate either with the overall neglect score ($r = .09$) or with any clinical test of neglect in the totality of patients: extinction ($r = .18$), Albert test ($r = .12$), bells test ($r = .10$), line-bisection task ($r = -.09$), and copy task ($r = .01$). Similar results were obtained when we considered only the 19 RBDN+ patients.

Eight of the 19 RBDN+ patients, but no patient without neglect, showed a right facilitation of return at more than two standard deviations compared with the controls' mean. Lesions involved the temporal lobe in 3 patients (completely in DM, of the mesial part in MV2, and anterior in PB), and the mesial part of the parietal and occipital lobes in 1 patient (BL). Subcortical structures were also involved in 3 patients (DM, MV2, and PB).

In the left hemifield, the invalid minus valid difference at the 1,000-ms SOA was −105 ms (SE = 96 ms) for the RBDN+ patients, −22 ms (SE = 8 ms) for controls and +82 ms (SE = 40 ms) for RBDN− patients. However, there was no significant difference between groups, probably because of the large variance characterizing patients' performance with left-sided targets (see Anderson, Mennemeier, & Chatterjee, 2000; Bartolomeo, 1997; Bartolomeo, Siéroff, Chokron, & Decaix, 2001).

Experiment 2

Experiment 1 has shown that left unilateral neglect can be related to a deficit in exogenous attention. The aim of Experiment 2 was, first, to evaluate the frequency of a deficit in endogenous attention in patients with neglect syndrome and, second, to evaluate whether a deficit in endogenous attention can aggravate neglect. In Experiment 2, the ratio between valid and invalid conditions was 80:20. The peripheral cue occurred at the target location in 80% of the trials, thus providing useful information to anticipate and move or shift attention in advance toward the location of the target. Both exogenous and endogenous orienting of attention should contribute the detection processes. Exogenous orienting should affect RTs for short SOAs, like in Experiment 1. Endogenous orienting should influence later processes, anticipation being usually present with SOAs superior to 300 ms (Müller & Findlay, 1988). Results of the three groups are presented in Figure 3.

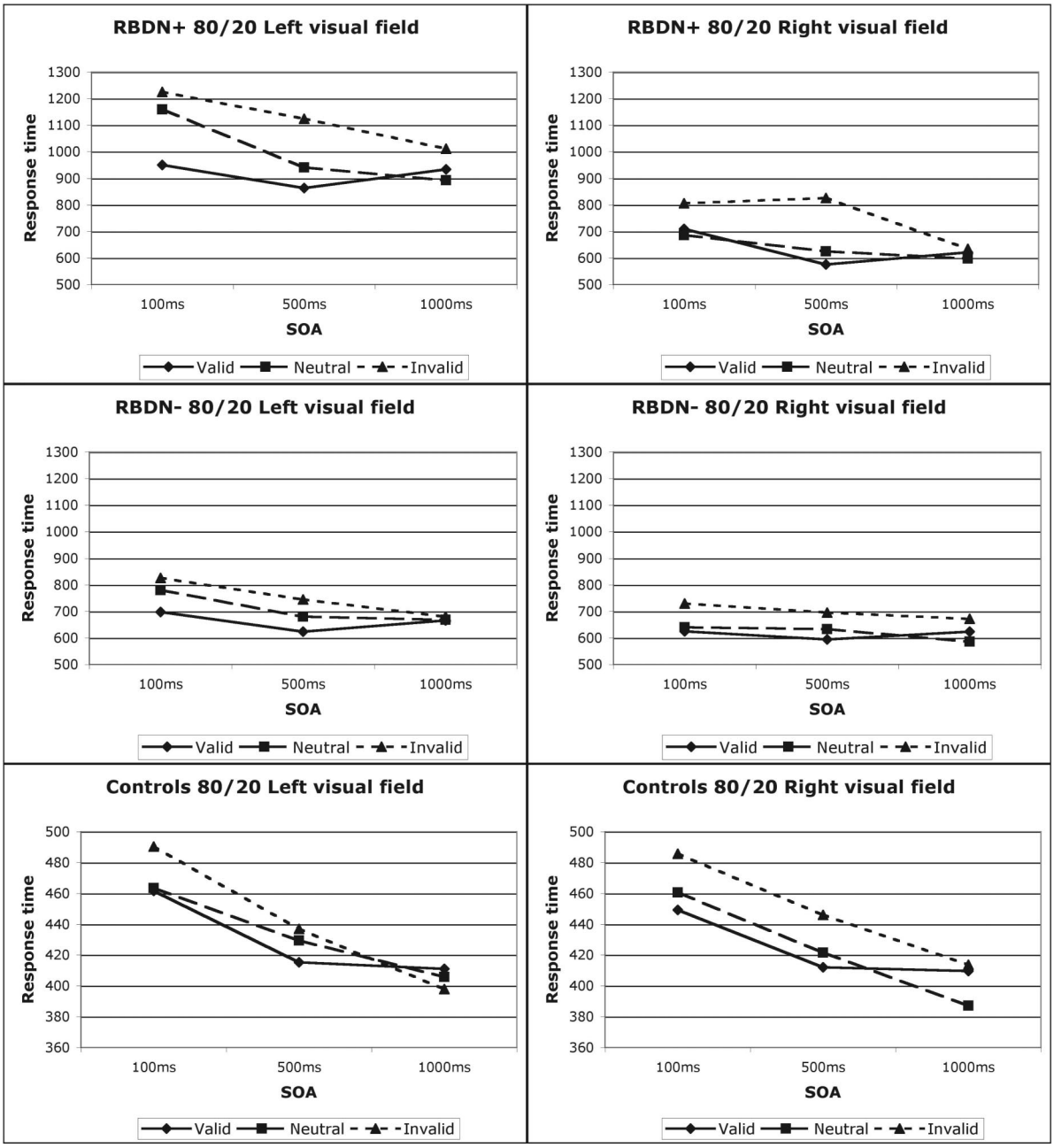

Figure 3. Response times for the three groups in Experiment 2: RBDN+ patients (right brain damage with left neglect syndrome), RBDN− patients (without neglect syndrome), and controls.

Global RT analysis. All main effects were significant. The main effect of group, $F(2, 55) = 27.19$, $MSE = 680E{+}03$, $p < .01$, reflected, as in Experiment 1, slower overall RTs for RBDN+ patients ($M = 843$ ms, $SE = 69$ ms) and for RBDN− patients ($M = 676$ ms, $SE = 72$ ms) than for the control group ($M = 433$ ms, $SE = 15$ ms).

There was also a main effect of SOA, $F(2, 110) = 23.09$, $MSE = 22.4E{+}03$, $p < .01$, and the interaction Group × SOA, $F(4, 110) = 2.63$, $MSE = 22.4E{+}03$, $p < .05$, reflected a stronger effect of SOA (141 ms of difference between the short and the long SOAs, $SE = 33$ ms) in RBDN+ patients than in controls ($M = 64$ ms, $SE = 11$ ms) and in RBDN− patients ($M = 67$ ms, $SE = 25$ ms). However, as in Experiment 1, the interaction Group × Field × SOA was not significant, $F(4, 110) < 1$, $MSE = 19.5E{+}03$, *ns*.

The field effect, $F(1, 55) = 14.26$, $MSE = 224E{+}03$, $p < .01$, showed faster RTs for the right hemifield, but the interaction of Group × Field was also significant, $F(2, 55) = 13.48$, $MSE = 224E{+}03$, $p < .01$. Only RBDN+ patients showed a field effect (+336 ms in favor of the right hemifield, $SE = 89$ ms), $F(1,$

55) = 42.92, *MSE* = 224E+03, $p < .01$, the difference between both hemifields being not significant in RBDN− patients (*M* = +63 ms, *SE* = 35 ms) and in controls (*M* = +3 ms, *SE* = 3 ms).

The effect of cue type was significant, $F(2, 110) = 21.57$, *MSE* = 23.6E+03, $p < .01$, and there was an interaction of Group × Cue Type, $F(4, 110) = 8.38$, *MSE* = 23.6E+03, $p < .01$. Valid trials showed the fastest RTs (M = 566 ms, *SE* = 32 ms), followed by neutral trials (*M* = 584 ms, *SE* = 36 ms) and by invalid trials (*M* = 640 ms, *SE* = 40 ms). RBDN+ patients showed a larger difference between valid and invalid trials (*M* = 162 ms, *SE* = 39 ms) than controls (*M* = 19 ms, *SE* = 3 ms), $F(1, 55) = 25.18$, *MSE* = 29.3E+03, $p < .01$, with the results of RBDN− patients being at an intermediate level (M = 86 ms, *SE* = 17 ms). Also, the difference between invalid and neutral trials was larger in RBDN+ patients (*M* = 121 ms, *SE* = 33 ms) than in controls (*M* = 17 ms, *SE* = 3 ms), $F(1, 55) = 15.71$, *MSE* = 24.5E+03, $p < .01$, RBDN− patients being at an intermediate level (*M* = 60 ms, *SE* = 23 ms). However, contrary to Experiment 1, neither the Group × Field × Cue Type × SOA nor the Group × Field × Cue Type interactions were significant, $F(8, 220) < 1$, *MSE* = 21.3E+03, *ns*; $F(4, 110) = 1.06$, *MSE* = 23.0E+03, *ns*, respectively. Theoretically relevant results were followed up by paired associations.

Disengagement of attention. At the shortest 100-ms SOA, the disengage score was significantly larger for patients with neglect syndrome (*M* = +178 ms in favor of right hemifield targets, *SE* = 122 ms) than for controls (*M* = −8 ms, *SE* = 13 ms), $F(1, 55) = 4.23$, *MSE* = 24.3E+03, $p < .05$; the score of RBDN− patient was at an intermediary level (*M* = +22 ms, *SE* = 40 ms). None of the other SOAs showed differences in the disengage score between groups. Thus, high predictability of the target location did not increase the disengage deficit, and even tended to reduce this deficit. The disengage deficit in RBDN+ patients was numerically smaller in Experiment 2 (*M* = 178 ms, *SE* = 122 ms) than in Experiment 1 (*M* = 218 ms, *SE* = 57 ms), and occurred only for the 100-ms SOA. Therefore, the disengage deficit obtained in Experiment 2 could be explained by the fact that our cues are peripheral and still call for exogenous attention at short SOAs. Losier and Klein (2001) have found, in their review, that the disengage deficit in patients with neglect syndrome was frequently stronger in conditions of exogenous orienting of attention (peripheral cues) than in conditions of endogenous orienting of attention (symbolic central cues). Our results are in agreement with this meta-analysis and present the advantage of comparing exogenous and endogenous attention by using the same type (peripheral) of cue.

As expected, there was a positive correlation between the disengage score and the extinction score ($r = .51$, $p < .01$). However, no correlation with the clinical tests of neglect was obtained: Albert test ($r = .07$), bells test ($r = 34$), line-bisection task ($r = 25$), and copy task ($r = .12$).

Engagement of attention. At the 100-ms SOA, the engage score (valid difference between hemifields) was significantly larger in RBDN+ patients (*M* = +241 ms, *SE* = 80 ms) than in controls (*M* = +12 ms, *SE* = 6 ms), $F(1, 55) = 14.99$, *MSE* = 20.8E+03, $p < .01$, and only marginally different between RBDN+ patients and RBDN− patients (*M* = +74 ms, *SE* = 41 ms), $F(1, 55) = 3.43$, *MSE* = 20.8E+03, $p = .069$, RBDN− patients being not significantly different from controls. This engage score is numerically equivalent to the one obtained in Experiment 1. The engage score at the 500-ms SOA was significantly stronger in RBDN+ patients (*M* = +289 ms, *SE* = 90 ms) than in controls (*M* = +3 ms, *SE* = 5 ms), $F(1, 55) = 18.74$, *MSE* = 25.9E+03, $p < .01$, and RBDN− patients (*M* = +29 ms, *SE* = 28 ms), $F(1, 55) = 6.67$, *MSE* = 25.9E+03, $p < .05$. The engage score at the 1,000-ms SOA was also significantly stronger in RBDN+ patients (*M* = +312 ms, *SE* = 105 ms) than in controls (*M* = +1 ms, *SE* = 8 ms), $F(1, 55) = 16.30$, *MSE* = 35.4E+03, $p < .01$, and RBDN− patients (*M* = +42 ms, *SE* = 41 ms), $F(1, 55) = 5.27$, *MSE* = 35.4E+03, $p < .05$. Interestingly, the engage score of patients with neglect syndrome is numerically smaller at the longest SOA in Experiment 2 with informative cues than in Experiment 1 (*M* = +403 ms, *SE* = 108 ms) with noninformative cues.

As in Experiment 1, at the 100-ms SOA, the engage score correlated with extinction ($r = .41$, $p < .05$), but with none of the neglect scores, which is at variance with the results of Experiment 1: Albert test ($r = .26$), bells test ($r = .18$), line-bisection task ($r = .14$), copy task ($r = .28$), and overall neglect score ($r = .36$). This difference between Experiment 1 (noninformative cues) and Experiment 2 (informative cues) again suggests the predominance of exogenous impairments in left neglect. Still, at the 1,000-ms SOA, the engage score correlated with the Albert test ($r = .51$, $p < .01$), the Bells test ($r = .50$, $p < .01$), and the extinction score ($r = .44$, $p < .05$).

Because of the predictability of the cue, endogenous strategies can be produced to anticipate the side of the target. Despite this, with endogenous orienting of attention being slower than exogenous orienting, deficits of endogenous orienting should appear for long SOAs. A score of endogenous engagement and maintenance of attention toward the left hemifield was therefore calculated by use of the difference in the valid left condition between the shortest (100 ms) and the longest (1,000 ms) SOAs. A positive score indicates that subjects were faster at the long SOA.

The left endogenous score (+16 ms in RBDN+ patients, *SE* = 77 ms; +32 ms in RBDN− patients, *SE* = 49 ms; and +50 ms in controls, *SE* = 12 ms) was not significantly different between the three groups, $F(1, 55) < 1$ for each comparison, which militates against a deficit in endogenous attention in neglect syndrome. However, RBDN+ patients' variability of performance was substantial, and 4 RBDN+ patients and 1 RBDN− patient showed a strong deficit in endogenous engaging and/or maintaining of attention in the left hemifield after a left hemifield cue. Among these patients, four had an available CT scan (BL, −221 ms; MB2, −248 ms; MS, −1,185 ms; and MV2, −155 ms). For all these patients, the lesion involved the thalamus and/or the capsulo-lenticular regions. These results are compatible with the role of the thalamus in engagement (Petersen, Robinson, & Morris, 1987; Rafal & Posner, 1987) and maintenance of attention (LaBerge, 1995; LaBerge & Buchsbaum, 1990). However, some patients showing a thalamic lesion, although partial, did not present a deficit in maintaining attention toward the left hemifield (JL1, +10 ms; JL2, +140 ms; and MH, +2 ms). Note also that 2 patients (BL, −221 ms, and JD1, +200 ms) clearly showed a lesion in the superior part of the parietal lobe (which has been included in an endogenous network of attention by Corbetta and Shulman, 2002, dorsal fronto-parietal network), although this did not include superior frontal areas. Only BL, whose lesion was also

involving the thalamus, actually showed a deficit in endogenous orienting and/or attention maintenance.

The left endogenous score was not correlated with extinction ($r = -.18$), the line bisection task ($r = .16$), the copy task ($r = .21$), or the global neglect score ($r = -.21$), but was negatively correlated with both cancellation tests: Albert test ($r = -.42$, $p < .05$), bells test ($r = -.53$, $p < .01$). Patients with a deficit in endogenous attention toward the left hemifield showed the strongest deficit in these tests. This pattern of results suggests that, if neglect behavior is mainly related to an exogenous deficit, it can be aggravated by an additional deficit in endogenous attention. Indeed, only 2 of the 19 patients with neglect syndrome (AD and MM) presented an engage and/or a disengage deficit in Experiment 2 without any deficit in Experiment 1, thus showing a rather pure endogenous deficit.

Experiment 3

The aim of Experiment 3 was to evaluate the possibility of patients with or without neglect syndrome to endogenously reorient attention to the contralesional hemifield when the cue is located in the "good" ipsilesional hemifield. In Experiment 3, the ratio between valid and invalid conditions was 20:80. The target occurred at the location opposite the peripheral cue in 80% of the trials. Thus, participants could anticipate and shift attention in advance toward the location of the target. In Experiment 3, both exogenous and endogenous orienting of attention should contribute to the orienting process. At short SOAs, peripheral cues should capture attention, but this exogenous process might be endogenously inhibited because participants were expecting the target to occur at the opposite location. The endogenous component could also occur at longer SOAs and consists of reorienting attention from the invalid cue location toward the contralateral target location. Thus, in Experiment 3, we explored the endogenous ability of patients with neglect syndrome to inhibit ipsilesional capture of attention and to reorient attention toward contralesional targets. Results of the three groups are presented in Figure 4.

Global RT analysis. The main effect of group, $F(2, 55) = 28.32$, $MSE = 703E+03$, $p < .01$, reflected slower overall RTs for RBDN+ patients ($M = 858$ ms, $SE = 70$ ms) than for RBDN− patients ($M = 674$ ms, $SE = 74$ ms) and for RBDN− patients compared with the control group ($M = 431$ ms, $SE = 16$ ms).

There was a main effect of SOA, $F(2, 110) = 22.52$, $MSE = 24.0E+03$, $p < .01$, and the interaction of Group × SOA was only marginally significant, $F(4, 110) = 2.42$, $MSE = 24.0E+03$, $p = .053$. RBDN+ patients showed a 121-ms ($SE = 38$ ms) difference between the short and the long SOAs, 62 ms ($SE = 37$ ms) for RBDN− patients and 73 ms ($SE = 13$ ms) for controls. Once again, the interaction of Group × Field × SOA was not significant, $F(4, 110) = 1.70$, $MSE = 16.0E+03$, *ns*.

The field effect, $F(1, 55) = 19.98$, $MSE = 227E+03$, $p < .01$, showed faster RTs for the right hemifield, but the interaction of Group × Field was also significant, $F(2, 55) = 19.83$, $MSE = 227E+03$, $p < .01$. As in previous experiments, only RBDN+ patients showed a field effect ($M = +406$ ms in favor of the right hemifield, $SE = 88$ ms), $F(1, 55) = 62.12$, $MSE = 227E+03$, $p < .01$, the difference between both hemifield being not significant in RBDN− patients ($M = +71$ ms, $SE = 57$ ms) and in controls ($M = +1$ ms, $SE = 3$ ms).

Cue type was not significant, $F(2, 110) = 2.17$, $MSE = 28.7E+03$, *ns*, and neither were the interaction of Group × Cue Type, $F(4, 110) = 1.70$, $MSE = 28.7E+03$, *ns*, and the interaction of Group × Field × Cue Type × SOA, $F(8, 220) < 1$, $MSE = 15.1E+03$, *ns*. However, there was an interaction of Cue Type × SOA, $F(4, 220) = 2.48$, $MSE = 17.3E+03$, $p < .05$, because of the stronger improvement between the 100-ms and the 1,000-ms SOAs for invalid ($M = 116$ ms, $SE = 15$ ms) and neutral trials ($M = 104$ ms, $SE = 16$ ms) than for valid trials ($M = 43$ ms, $SE = 29$ ms). Finally, there was an interaction of Group × Field × Cue Type, $F(4, 110) = 3.75$, $MSE = 14.6E+03$, $p < .01$. The only significant differences between cue type conditions, independent from SOA, were found in RBDN+ patients. For left hemifield targets, there was a difference between valid and invalid trials in favor of invalid trials ($M = +90$ ms, $SE = 65$ ms; for comparison, $M = -36$ ms, $SE = 14$ ms, in RBDN− patients; $M = +21$ ms, $SE = 8$ ms, in controls), $F(1, 55) = 5.26$, $MSE = 232E+03$, $p < .05$. There was a difference between invalid and neutral trials in favor of neutral trials ($M = +50$ ms, $SE = 32$ ms; for comparison, $M = +19$ ms, $SE = 14$ ms, in RBDN− patients; $M = -13$ ms, $SE = 10$ ms, in controls), $F(1, 55) = 5.81$, $MSE = 70.1E+03$, $p < .05$. There was also a difference between valid and neutral trials ($M = +140$ ms, $SE = 58$ ms; for comparison, $M = -17$ ms, $SE = 59$ ms, in RBDN− patients; $M = +8$ ms, $SE = 10$ ms, in controls), $F(1, 55) = 14.86$, $MSE = 557E+03$, $p < .01$. For right hemifield targets, there was a difference between invalid and neutral trials only in RBDN+ patients (in favor of neutral trials, $M = +43$ ms, $SE = 27$ ms), $F(1, 55) = 5.80$, $MSE = 51.6E+03$, $p < .05$.

The triple interaction is mainly explained by a stronger left hemifield difference between valid and invalid conditions in patients with neglect syndrome than in other groups. Invalid trials gave faster RT than valid trials. However, this result cannot be entirely explained by an efficient endogenous reorienting of attention to the left hemifield, because the difference between Experiment 3 and Experiment 1 was a small gain in invalid trials (1,047 ms vs. 1,094 ms) in favor of an endogenous reorienting of attention, as well as a large cost in valid trials (1,137 ms vs. 916 ms). Patients with neglect syndrome were specifically slow in valid left trials in this experiment, certainly because a left cue indicating the frequent occurrence of a right target (80% invalid conditions) led to an endogenous orienting toward the right hemifield. When the cue was valid, patients had to shift the direction of attention and return to left locations. Most probably, this shifting in the direction of attention was difficult in patients with neglect syndrome.

Disengagement and engagement. In Experiment 3, there was no disengagement deficit for left hemifield targets in any group of patients at the 100-ms SOA, RTs being even faster in invalid trials (1,167 ms) than in valid trials (1,192 ms) in RBDN+ patients. A deficit in engaging attention at short SOA was found in RBDN+ patients ($M = +524$ ms, $SE = 133$ ms) but not in RBDN− patients ($M = +9$ ms, $SE = 49$ ms).

Inhibition of right hemifield cue. We were interested in the possibility for the patients with neglect syndrome to inhibit the location of the right hemifield cue when right targets rarely followed right cues, that is, to resist to right attentional capture.

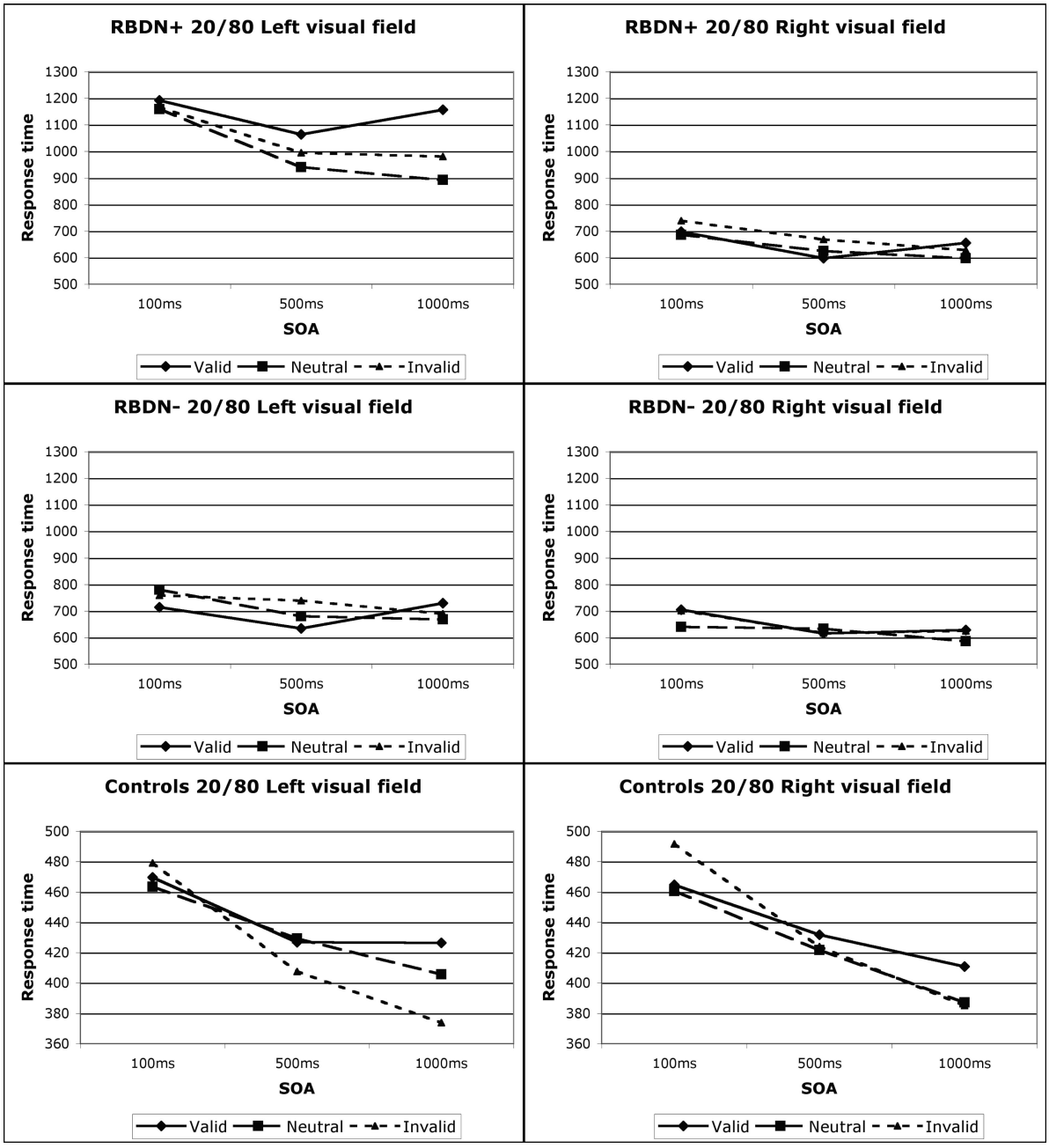

Figure 4. Response times for the three groups in Experiment 3: RBDN+ patients (right brain damage with left neglect syndrome), RBDN− patients (without neglect syndrome), and controls.

We calculated the gain in RTs to invalid left targets (right cue), independently from SOA, between Experiment 1 and Experiment 3. This gain was 47 ms in RBDN+ patients (*SE* = 44 ms), 90 ms in RBDN− patients (*SE* = 37 ms), and 5 ms in controls (*SE* = 5 ms). Eight of the 19 RBDN+ patients and 5 of the 7 RBDN− patients showed a large gain (more than two standard deviations from controls' mean), demonstrating an effective inhibition of right attentional capture. However, analyses of lesions in RBDN+ patients showing or not showing inhibition of right cues did not point to specific regions in the brain (see Table 2).

Reorienting of attention. To further examine the endogenous attention abilities in patients with neglect syndrome, we looked closely at their reorienting of spatial attention from a right cue toward a contralateral left target. Because endogenous control of attention is a slower process than exogenous orienting (Posner & Snyder, 1975), a comparison was made in left invalid targets at the longest SOA, between Experiment 1 and Experiment 3. A score of reorienting of attention was calculated as follows: (left invalid E1) – (left invalid E3) at the 1,000-ms SOA. RBDN+ patients showed a negative score (*M* = −56 ms, *SE* = 41 ms), demonstrating poor abilities in reorienting of

attention from ipsilesional right cue toward left contralesional targets. RBDN− patients showed a positive score ($M = +86$ ms, $SE = 48$ ms) as well as controls ($M = +13$ ms, $SE = 8$ ms). Three RBDN+ patients and 4 RBDN− patients had a positive reorienting score more than two standard deviations away from controls' mean, showing effective reorienting attention toward contralesional targets. Table 2 indicates patients presenting a deficit in reorienting of attention. Unfortunately, no anatomical conclusion could be drawn.

Discussion

The present study supports claims to the heterogeneous nature of left neglect, because we showed that neglect could be composed of dissociable deficits in several paper-and-pencil tests and in elementary operations, like engagement or disengagement of attention. However, in keeping with previous results, our findings also stress that an asymmetry of attentional engagement, with faster engagement for right-sided than for left-sided objects, is a major component deficit in left neglect. Also, our results clearly demonstrate that neglect is mainly explained by an exogenous deficit, even if a possible endogenous deficit could exacerbate neglect behavior.

Dissociations Among Paper-and-Pencil Tests

Results in the paper-and-pencil tests of neglect indicate that not every test is correlated with others. Even if spatial neglect is sometimes considered as a globally unitary syndrome, several authors have described dissociations, for example between performance on cancellation tests and the line-bisection test (Binder et al., 1992; Halligan & Marshall, 1992; McGlinchey-Berroth et al., 1996; Seki, 1996). Our results clearly confirm such dissociations. Although performances on both cancellation tests (Albert and bells tests) were unsurprisingly correlated, only 4 patients presented a deficit in both cancellation and line-bisection tests. Twelve patients showed a deficit in the cancellation tests only, and 3 patients showed a deficit in the line-bisection test only, an asymmetry, that has already been noted (Ferber & Karnath, 2001). Also, performance on the cancellation tests were correlated with extinction, in agreement with previous findings (McGlinchey-Berroth et al., 1996), and line bisection was correlated with copy, a correlation that has not been described in the literature.

Available CT scans tend to indicate that lesions are mainly posterior (including the parietal lobe, the temporal lobe, and the posterior part of subcortical structures) when a cancellation deficit exists, and more anterior (the anterior part of the temporal lobe, the frontal lobe, and the anterior part of the capsulo-lenticular region) in patients presenting, although not isolated to, a significant bias in line bisection, a result that is similar to the dichotomy proposed by Marshall and Halligan (1995b). Finally, all lesions of patients showing extinction involved the temporo-parietal region, in agreement with the findings of Karnath, Himmelbach, and Küker (2002).

The presence of double dissociations between cancellation tests and the line-bisection test rule out explanations due to the differential complexity of the task, even if those patients showing a deficit in the cancellation tests without deficit in the line-bisection test did not present the strongest cancellation deficit in our group of patients. The cognitive explanations for the cancellation–line-bisection dissociations are currently in debate. Marshall and Halligan (1995b) have argued that line bisection requires the computation of a midpoint that is not physically present in the stimulus array. Still, according to the results reported here, we also have to explain, first, why cancellation tests and extinction are correlated and, second, why line bisection and copy are correlated.

A motor involvement exists in cancellation tests and in line bisection (as well as in copy), but not in extinction, and cannot easily represent an explanation. A possibility is that the type of motor involvement differs between tasks. Even if cancellation tasks require a motor response, and the performance can be affected by a motor-directional akinesia, the evaluation of the performance is not based on a fine analysis of the quality of the response (for example, a circle around the bell is counted as a correct response even if the circle is not perfect and complete). On the contrary, the motor response in copy or even in line-bisection tasks has to be much more precise.

Another explanation is that several objects are presented to the patient in the cancellation and extinction tests, whereas only one is presented in the bisection test, so that results in the different tests could differ according to the presence or absence of object- versus viewer-centered neglect (Chatterjee, 1994). The distribution of attention on several separated objects could play a role in cancellation and extinction tests. Conversely, in both copy and line-bisection tests, attention on individual objects could be involved. Although several objects were presented in our copy test, our score depended mostly on the left neglect of individual objects (Behrmann, 1994; Driver & Halligan, 1991; Gainotti, Messerli, & Tissot, 1972). Similarly, several lines were presented on the same sheet of paper in our line-bisection test; however, in this task, patients had to focus attention on each individual line. Riestra, Crucian, Burks, Womack, and Heilman (2001) have shown that patients had more difficulties in judging the similarity between the left and right segments when presented with a pre-bisected line than when presented with sequential, individual segments. If this result pleads in favor of an extinction-like phenomenon playing a role in line bisection bias, it can also be argued that left and right segments in a pre-bisected line can be considered as two parts of the same object and not two different objects, although the sequential individual segments could involve the processing of two different objects. It would be interesting to see whether patients exist with a stronger deficit in the sequential segments judgment task than in the pre-bisected judgment task, and whether such a deficit is correlated with other tests like the cancellation test.

Finally, only cancellation and extinction scores were correlated with scores obtained in the cuing task, specifically the engagement and the disengagement scores. As in these clinical tests, targets in the cuing task could appear in several (two) locations, these locations being marked by empty boxes, which were present during the whole trial. Possibly, good performance in these clinical tests and in our cuing task shares the need for the distribution of attention over several objects and for the orienting of attention between object locations.

Left Neglect and the Elementary Operations of Attention

Both groups of patients, distinguished by the presence or absence of neglect syndrome, were slower than controls in the cuing

experiments. However, only for patients with neglect syndrome were RTs to left hemifield targets systematically slower, in all three experiments, than RTs to right hemifield targets.

We first found some argument in favor of a deficit in phasic alertness in patients with neglect syndrome. They improved their RTs with SOA more than other groups, which can be caused by some slowness in the alerting effect of the sudden appearance of the boxes in the beginning of each trial. However, even if this result is in agreement with other results using physiological or reaction time data, we are careful about the conclusion of a deficit in phasic alertness in our patients, because our measure of alertness was quite indirect.

Most important, patients with left neglect syndrome clearly differ from controls and from patients suffering from right brain damage without neglect syndrome in terms of the effects of a spatial peripheral precue on the detection of a single target. Concerning the question of the locus of the cuing effect, there is evidence that location cuing does affect perceptual processing (Bonnel, Possamaï, & Schmitt, 1987; Müller & Humphreys, 1991). Cue could also have an effect on motor preparedness. However, most our patients did not produce any false alarm (responding to the cue instead of the target, specifically with long SOAs), and the total percentage of false alarms was less than 3%. Consequently, the results presented here, using a peripheral spatial cuing procedure, are indicative of which elementary operations of attention are specifically associated with left neglect syndrome.

Disengagement. In agreement with Morrow and Ratcliff (1988), there was a clear deficit in disengaging from ipsilesional stimuli in patients with neglect syndrome, and patients without neglect did not show a strong deficit in disengaging attention. Furthermore, in both Experiments 1 and 2, we found a close relationship between a disengage deficit and the extinction phenomenon (Posner et al., 1984). However, the disengage deficit in Experiment 1 (noninformative cues) as well as in Experiment 2 (informative cues) did not correlate with neglect severity, either measured by scores on individual paper-and-pencil tests or by a global neglect score, contrary to the study of Morrow and Ratcliff (1988). A possible explanation of this difference between our results and theirs is that these authors included the copy of the Rey figure. Impairment on this task frequently results from right hemispheric lesion, and left neglect could make it worse (Pillon, 1981; Rapport, Farchione, Dutra, Webster, & Charter, 1996). Despite this, our results suggest that other components of orienting of attention might be more closely related to left neglect clinical signs.

Concerning the lesion location, our results are compatible with the proposed relationship between disengaging of attention and posterior cortex (Egly et al., 1994; Friedrich et al., 1998; Friedrich & Margolin, 1993; Posner et al., 1984, 1987). Still, it is difficult to delimit a specific area for a deficit in disengaging of attention, for example concerning the superior parietal lobule, as proposed by Posner et al. (1984), or the temporo-parietal junction, as proposed by Friedrich et al. (1998). The analysis of morphological data may not be sufficient to the comprehension of the pathophysiology of neglect. All our patients were tested less than 1 year after the ictus and, for 17 of them, less than 3 months. Lesions could therefore be accompanied by diaschisis effects within an attentional neural network, as suggested by several authors (e.g., Demeurisse, Hublet, Paternot, Colson, & Serniclaes, 1997). Also, subcortical lesions could provoke a disconnection of some cortical areas. Accordingly, some lesions not involving the temporo-parietal junction or the superior parietal lobule could still be accompanied by a disengage deficit.

Finally, a nonnegligible number of patients with neglect syndrome suffering from a right hemisphere lesion showed a disengage deficit in both hemifields, a result in agreement with the clinical fact that some patients with neglect syndrome have difficulties even in the right hemispace (see Bartolomeo & Chokron, 1999b). This result cannot be totally explained, at least in our patients, by a tendency to compensate for the contralateral neglect, because all patients showing a disengage deficit in the right hemifield showed an even stronger disengage deficit in the left hemifield. Such a bilateral deficit in orienting of attention is compatible with evidence from other methods, showing that the right hemisphere processors play a role in orienting of attention in both hemifields or hemispaces (Corbetta, Kincade, Ollinger, McAvoy, & Shulman, 2000; Heilman & Van der Abell, 1980; Mesulam, 1981; Perry & Zeki, 2000). However, another possibility is that some diaschisis effect occurred in the left parietal lobe, a phenomenon that has been described with acute right parietal lesion (Perani, Vallar, Paulesu, Alberoni, & Fazio, 1993). The fact that lesions provoking a bilateral disengage deficit seem more anterior than lesions provoking a unilateral disengage deficit does not allow us to conclude in favor of one or the other explanation.

Engagement. In their review, Losier and Klein (2001) wrote that

> patients with posterior, right-hemisphere damage responded significantly more slowly to validly cued targets in the contralesional than ipsilesional hemispace. Although this pattern is present in each of the five studies contributing to this analysis, in no case did the authors indicate whether the trend was significant. (p. 9)

In the present study, the patients with neglect syndrome showed a strong difference between left and right engagement of attention compared with controls, which is in agreement with Losier and Klein's meta-analysis. Is it really a deficit in engaging of attention? In principle, this could be ascertained by a comparison between valid and neutral conditions (absence of benefit in valid condition). However, the definition of a neutral condition in cuing procedures is always problematic (Jonides & Mack, 1984), and all the more so in patients with neglect syndrome, for whom neutral cues occurring in the central box can act as invalid cues for left targets (Posner et al., 1984). A possibility is that the difference between left and right valid conditions could be caused by a low-level sensory deficit. Patients suffering from an acuity deficit in the left hemifield should be slower for left targets compared with right targets even if this target is validly cued. Two issues militate against this hypothesis. First, according to our inclusion criteria, none of our patients suffered from left hemianopia. Second, the left–right difference for valid targets was obtained even with relatively long SOAs, which should have permitted compensation for a visual sensory deficit without engage deficit.

We not only were able, like Losier and Klein (2001), to conclude that a deficit in engaging attention toward contralesional targets is frequent in neglect, but we also found that the engage deficit was correlated with the deficit in the cancellation tests and the overall neglect score (at least in Experiment 1). Our findings

confirm the pervasive nature of a left-sided engage deficit in left neglect (D'Erme et al., 1992; Gainotti et al., 1991).

Results also indicate that patients who present a visual extinction not only have difficulty disengaging attention from ipsilesional stimuli but also have a strong deficit in engaging attention toward left contralesional stimuli. This result is important because relating the extinction phenomenon solely to a deficit in disengaging from ipsilesional stimuli does not explain why patients prefer to report ipsilesional targets first (unless if we consider that their attention was strongly engaged on the fixation item).

Anatomically, the engage deficit seems related to posterior cortical and subcortical lesions, like the deficit in cancellation tests. However, here again it remains difficult to point to a specific locus. Only studies of patients with a longer delay from the ictus could give more precise information on the anatomical locus of the elementary operations.

Even if we were unable to delimit different anatomical loci for disengage and engage deficits, double dissociations clearly exist. According to several theories, orienting of attention could be explained by an imbalance between hemispheres (Farah, 1994; Kinsbourne, 1993). A unilateral lesion should provoke a bias in orienting of attention toward ipsilesional targets. Such theories explain why patients with neglect syndrome have a contralesional deficit even in valid conditions, but they predict that the contralesional deficit should be stronger in the invalid condition, because the invalid cues would reinforce the bias in favor of ipsilesional targets. Such theories do not predict dissociations between deficits in engaging of attention and deficits in disengaging of attention. An engage deficit should always been accompanied by a disengage deficit. Cohen, Romero, Farah, and Servan-Schreiber (1994) have argued that the difficulty in disengaging attention can be interpreted as an emergent property of interactions among the remaining parts of the system, without the need to invoke a specific disengager. Contrary to this prediction, we found instances of engage deficit without disengage deficits, as well as the opposite dissociation, showing that these elementary operations are produced by different anatomical processors, in agreement with Posner's theory (Posner et al., 1984).

IOR. In addition to attentional engagement and disengagement, other components of attention were also impaired in our patients with neglect syndrome. We found a clear deficit of IOR in the ipsilesional hemifield of patients with neglect syndrome, even replaced by a facilitation, but not in patients suffering from a right brain damage without neglect, confirming previous results (Bartolomeo et al., 1999; Bartolomeo, Siéroff, Decaix, & Chokron, 2001). The absence of IOR, or even the facilitation of return in the ipsilesional hemifield could explain why patients with neglect syndrome repeatedly process ipsilesional targets (Bartolomeo et al., 1999). In our patients, right facilitation of return was correlated with the left target disengage score. Thus, a strong possibility is that facilitation of return is the consequence of a difficulty in disengaging attention from right hemifield locations.

Exogenous Versus Endogenous Deficit

Our results clearly support the hypothesis of a deficit in exogenous orienting of attention in the neglect syndrome (Bartolomeo, Siéroff, Decaix, & Chokron, 2001; Gainotti, 1996; Làdavas et al., 1994; Luo et al., 1998; Natale et al., 2005). A substantial deficit in endogenous orienting in patients with neglect syndrome would have led to a stronger left–right RT asymmetry in Experiment 2 (80% valid cues) than in Experiment 1 (noninformative cues), which was not observed. On the contrary, the disengage deficit was stronger with noninformative cues (Experiment 1) than with informative cues (Experiment 2) and was found only with short SOAs between the cue and the target (100 ms and 500 ms in Experiment 1; 100 ms in Experiment 2), thus confirming the results of the meta-analysis of Losier and Klein (2001). Moreover, the engage deficit was correlated with different clinical tests of neglect in Experiment 1, but less systematically so in Experiment 2. This result is important because most (but not all) studies exploring orienting in patients with neglect syndrome have used informative cues.

Corbetta and Shulman (2002) have developed a model of attention in which a ventral network in the parietal (temporo-parietal junction) and frontal lobes sustains exogenous orienting, and a dorsal parieto-frontal network sustains endogenous orienting. Most of our patients showed lesions in the ventral network and showed signs of deficits in exogenous orienting.

As seen in Experiment 2, few patients presented a clear deficit in endogenous engagement and/or maintaining of attention in the left hemifield (and only 2 out of the 19 patients with neglect syndrome showed an apparently pure endogenous deficit). These patients also presented more severe signs of left neglect. Thus, even if neglect is mainly explained by a deficit in exogenous orienting of attention, it is not surprising that an additional deficit in endogenous orienting could aggravate the neglect behavior. We found that those patients with an endogenous deficit suffered mainly from thalamic lesions, in agreement with the role of the thalamus in endogenous orienting of attention (LaBerge & Buchsbaum, 1990; Petersen et al., 1987; Rafal & Posner, 1987). Only 2 of our patients suffered from a lesion in the superior part of the parietal lobe (thus in the posterior part of the dorsal network of Corbetta & Shulman, 2002), but only 1, also showing a thalamic lesion, presented a deficit in endogenous orienting.

Finally, contrary to Experiments 1 and 2, patients with neglect syndrome did not present a disengage deficit in Experiment 3, in which 80% of the trials were invalid. Two issues may account for this dissociation. First, RTs to left valid trials were slower than in the other experiments, possibly because left cues incited patients to orient towards the right, and the few cases in which a left target actually occurred after a left cue engendered a disproportionate cost in RT, because patients had to shift the direction of attention. Second, patients with neglect syndrome showed, as did other groups, the possibility of inhibiting the capture of attention from right ipsilesional cues. The possibility of endogenously inhibiting the attentional capture exerted by right-sided objects is also consistent with a substantial sparing of endogenous processes in left neglect. Still, most of our patients with neglect syndrome did not present the possibility of developing an efficient endogenous reorienting of attention toward contralesional targets, even when ipsilesional cues were most probably followed by contralesional targets (Experiment 3). This result is different from that obtained by Bartolomeo, Siéroff, Decaix, and Chokron (2001) in a study using a similar methodology. A possible explanation underlying this difference is that patients in the present study had fewer trials per block than in the study by Bartolomeo, Siéroff, Decaix, and Chokron

(2001), and the time to build and develop endogenous strategies of leftward reorienting might have been insufficient. Regardless, it is difficult to ascertain that a problem in reorienting of attention from an ipsilesional right cue to a contralesional left target is caused by a deficit in endogenous orienting. Because of the fact that patients may have an exogenous bias in attention toward the right hemifield and a difficulty in disengaging of attention, reorienting should be more difficult to operate even when the endogenous mechanisms are not deteriorated. More studies are necessary, varying the number of trials in a block and using longer delays (greater than 1,000 ms) in order to better evaluate endogenous reorienting in patients with neglect syndrome, but our results suggest that the endogenous inhibition of right hemifield capture of attention is easier than the efficient reorienting of attention toward left hemifield in patients with neglect syndrome.

In conclusion, our findings point out the importance of adapting the rehabilitation of neglect to these various attentional impairments. Rehabilitation should thus include not only the classic reorientation of attention towards the contralesional hemifield but also a reduction of the rightward attraction of attention in the ipsilesonal hemifield. In addition, it appears that the attentional training should take into account the clear dissociation between exogenous versus endogenous orientation of attention observed in most patients with left neglect syndrome.

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