



Perception and action in hemispatial neglect

PAOLO BARTOLOMEO,*‡ PATRIZIA D'ERME,† ROBERTA PERRI† and GUIDO GAINOTTI†

*INSERM Unit 324, Centre Paul Broca, Paris, France; †Institute of Neurology of the Catholic University, Rome, Italy

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Abstract—Hemispatial neglect is a neurological disorder which entails a spatial bias that penalizes events occurring in the hemispace contralateral to a brain lesion. Mechanisms operating upon various stages ranging from perception to action have been invoked to explain neglect. The present study explores the contribution of a defective programming of arm movements towards the neglected hemispace to neglect behaviour. Two reaction time tasks — a “perceptual” task and a “motor” task — were performed by right brain-damaged (RBD) patients with left hemispatial neglect, RBD patients without signs of neglect and control subjects. The perceptual task consisted of lateralized visual stimuli and central motor responses, whereas the motor task consisted of visual stimuli presented on the vertical midline and hand responses to be produced in either hemispace. Neglect patients showed a rightward bias on the perceptual task, but only two RBD patients (showing no signs of severe neglect) were consistently slowed in producing leftward motor responses. Different reference frames may thus be used in perceptual tasks and tasks involving arm movements. We conclude that hemispatial neglect commonly results from attentional impairments operating upon a visual perceptual frame of reference; additional deficits appear to be necessary to produce a directional motor disorder. © 1998 Elsevier Science Ltd. All rights reserved

Key Words: directional hypokinesia; spatial reference frames.

Introduction

Hemispatial neglect is characterized by the failure to respond or orient towards events that occur in the hemispace contralateral to a brain lesion. Lesions responsible for neglect usually involve the right parietal lobe [50], and patients are consequently impaired in performance on the left side of space. They typically copy only the right side of drawings [25], deviate towards the right when bisecting a horizontal line [47], and cancel small lines scattered on a paper sheet only on the right side of the sheet [1]. Each of these behavioural patterns involve visuomotor transformations. Perceptual representations and motor plans mutually update each other as action changes the perceived environment (action–perception cycles [2]). Neglect can involve an impairment at different levels of the processing of information from perception to action.

The first possible level of impairment might be an elementary sensory deficit. Patients would neglect the

left side of their world because they do not see it [5]. This hypothesis has been falsified by the reports of double dissociations between hemianopia and neglect [23, 39].

A second level of impairment was hypothesized by Bisiach and associates (see [6] for review). These authors reported neglect patients who omitted left details in describing well-known places from memory. Bisiach *et al.* argued that neglect is caused by an inability to build a symmetrical mental representation of space. However, using a multiple single-case approach we found that only a minority of neglect patients show such an imaginal impairment, and concluded in favour of an important role of ipsilesional visual stimuli in triggering neglect behaviour [4].

A third putative level of impairment consists in an attentional imbalance which somehow enhances the processing of right-sided stimuli [21, 34], or which disrupts the processing of left-sided items [28, 40, 44]. We have demonstrated that neglect patients indeed show an early orientation of attention towards the stimuli ipsilateral to their brain lesion [18, 24].

The last possible level of impairment in the action–perception cycle is the programming of arm movements in or towards the neglected hemispace. This “motor” deficit would express itself as a reluctance or a slowing in

‡ Address for correspondence: INSERM Unit 324, Centre Paul Broca, 2ter rue d'Alésia, F-75014 Paris, France; tel.: +33 1 40789210; fax: +33 1 45896848 or 45807293; e-mail: paolo@broca.inserm.fr.

performing movements towards left-sided targets. The aim of the present study was to analyse the contribution of this directional motor disorder to neglect manifestations. Mesulam [40] proposed that the motor aspect of neglect reflects involvement of the frontal component of an attentional network including also the posterior parietal and cingulate cortices and the brainstem reticular formation. Heilman *et al.* [27] found that a group of six right brain-damaged (RBD) patients with left neglect were slower to initiate hand movements towards the left side of space than rightward-directed movements. Once the movement was initiated its speed did not vary, regardless of the direction. Heilman *et al.* termed the described impairment "directional hypokinesia". Their findings suggest that a motor disorder participates in neglect symptoms, but do not demonstrate whether it is a necessary component of neglect or whether it is present only in some patients. The possibly related concept of directional hypometria, i.e. insufficient amplitude of contralesionally directed movements, was originally introduced to define hypometric leftward saccades in a patient with right frontal lesion [13], and was subsequently used to describe the performance of a patient showing rightward line bisection errors in the absence of other signs of left neglect [37]. Mattingley *et al.* [38] requested brain-damaged patients to press buttons which were horizontally arranged and illuminated in sequence from left to right or in the opposite direction. RBD neglect patients were slower when executing leftward movements than when moving rightward. In particular, patients with retro-rolandic lesions were slowed when initiating movements towards a button illuminated on the left side, whereas patients with anterior or subcortical lesions showed a decreased speed of leftward movements. Nevertheless, in the paradigm of Mattingley *et al.* the slowing of the initiation time showed by neglect patients with posterior lesions is not unambiguously interpretable in terms of directional hypokinesia, because patients had to detect the occurrence (lighting) of a left-sided stimulus before moving to reach it. The confounding effect of this perceptual-attentional component might thus have added to the motor component in slowing down patients' performance.

Other studies employed paradigms other than reaction time detection. Bisiach *et al.* [7] asked 16 left neglect patients to press left- or right-sided buttons in response to lateralized visual stimuli. Crossed and uncrossed conditions were performed, in which the side of stimulation and the side of motor response were, respectively, the opposite or the same. Most errors concerned left-sided responses, irrespective of the side of stimulation. Bisiach *et al.* concluded that an "output neglect" was present in their patients. However, in the right stimulus/left response condition, crucial for demonstrating the output component, the ipsilesional stimulation could have captured patients' attention (see e.g. [24] and [18]), thus decreasing accuracy on contralesional responses.

Other attempts to disentangle perceptual and motor

aspects of neglect include a line bisection test, in which a pointer could be moved by a pulley in the direction opposite to the hand movement [8], and a line cancellation test where left and right sides could be reversed using a mirror [9, 49] or an epidiascope [43]. These studies demonstrated instances of "motor" and "perceptual" forms of neglect. Whereas perceptual factors prevailed in most neglect patients, motor factors seemed more pronounced in patients with lesions involving the frontal lobes, which appeared consistent with evidence from case reports [10, 17, 19, 36] (see also [35] and [9]). However, the "motor" paradigms used in these studies were again characterized by the presence of lateralized visual feedback, thus complicating the interpretation of results. For example, in order to align the non-congruent visual and proprioceptive sensory inputs produced by the motor condition in these tasks, some patients might have mentally rotated by 180° their visual representations, thus transforming their perceptual neglect in apparent directional hypokinesia. This is not an unlikely possibility, as neglect patients may show an apparent "object-based" neglect when mentally restoring the upright position of 90° rotated stimuli [14].

Attempts to explore directional motor disorders using more "ecological" paradigms include the studies of Ishiai *et al.* [30, 31], who asked neglect patients to extend a line leftward to double its original length. However, both patients with parietal lesions and patients with frontal lesions performed accurately on this task, thus not showing any signs of directional hypokinesia. Harvey *et al.* [26] required eight neglect patients to point to either of the ends of a mid-transected line which they judged closer to the transection. Seven patients pointed consistently leftward, thus showing perceptual forms of neglect. Only one patient pointed predominantly rightward, a pattern suggestive of directional motor deficit. Mijovic [41] failed to find any evidence of directional hypokinesia in a series of 40 right brain-damaged patients who searched for a target by moving the stimulus display board under a panel until the target appeared in a window (e.g., to bring a right-sided target into view, the board was to be moved towards the left).

The conflicting results obtained in the above reviewed studies may suggest that a directional motor disorder is a comparatively rare phenomenon in neglect, or that the employed tasks did not always suitably disclose this deficit. We have developed a task of motor reaction times (RTs) to visual stimuli on the grounds that RT detection provides a sensitive behavioural measure of visuospatial disorders. To disentangle the relative contribution of perceptual and motor factors in neglect, we devised two test conditions: a perceptual task, characterized by lateralized visual stimuli and central motor responses, and a motor task, consisting of central visual stimuli and lateralized motor responses. This experimental paradigm allowed a direct comparison between the results of the two tasks and was simple enough to be performed by the vast majority of patients.

Methods

Subjects

Thirty-four right brain-damaged patients and 15 age-matched control subjects free of neurological damage consented to participate in this study. Table 1 reports subjects' demographic and clinical data.

Hemispatial neglect was assessed using a battery of visuospatial tests [4], which included tasks of line cancellation, identification of overlapping figures and line bisection. A laterality score was assigned to each patient, indicating the magnitude of rightward bias. A cut-off score was determined on the basis of the performance of a group of normal control subjects [4]. Fourteen patients, whose laterality scores fell above the cut-off, were considered to be showing left hemispatial neglect (patients 21–34 in Fig. 3).

Table 1. Demographic and clinical data for RBD patients (P1–34) and control subjects (C1–15)

Subject	Sex, age, years of schooling	Onset of illness (days)	Aetiology	Locus of lesion	Visual field	Left visual extinctions
P1	M, 46, 17	82	Neoplastic	TP	SQ	No
P2	F, 77, 6	8	Ischaemic	FP	Normal	No
P3	M, 58, 20	171	Ischaemic	FPO	Normal	Yes
P4	M, 53, 18	39	Ischaemic	IC, BG	Normal	Yes
P5	M, 53, 5	370	Haemorrhagic	IC, BG	Normal	Yes
P6	M, 26, 13	22	Haemorrhagic	P	Normal	No
P7	F, 52, 8	29	Ischaemic	FP	Normal	Yes
P8	M, 59, 8	8	Haemorrhagic	IC, Th	Normal	No
P9	M, 63, 11	10	Ischaemic	FP	Normal	No
P10	F, 76, 19	10	Ischaemic	FP	Normal	Yes
P11	M, 55, 22	77	Haemorrhagic	PI	Normal	Yes
P12	M, 80, 17	173	Ischaemic	TO	H	–
P13	M, 74, 10	21	Neoplastic	TP	IQ	Yes
P14	M, 75, 18	111	Ischaemic	FP	Normal	No
P15	M, 57, 5	129	Ischaemic	FT, BG	H	–
P16	M, 40, 6	33	Ischaemic	FTP, BG	Normal	No
P17	M, 68, 8	78	Ischaemic	FP	Normal	Yes
P18	M, 76, 13	60	Ischaemic	P	Normal	Yes
P19	F, 74, 8	485	Neoplastic	FP	Normal	Yes
P20	M, 46, 5	57	Ischaemic	FP	Normal	Yes
P21	M, 61, 8	135	Traumatic	TP	Normal	No
P22	F, 66, 11	20	Neoplastic	TP	Normal	Yes
P23	M, 67, 18	37	Ischaemic	Th	SQ	Yes
P24	M, 77, 12	30	Ischaemic	FP	Normal	Yes
P25	M, 76, 5	15	Ischaemic	P	IQ	Yes
P26	M, 43, 8	119	Haemorrhagic	IC, Th	Normal	No
P27	M, 50, 7	306	Ischaemic	FTP	IQ	Yes
P28	M, 79, 5	761	Ischaemic	TO	H	–
P29	F, 53, 7	76	Ischaemic	FP	Normal	Yes
P30	M, 69, 13	251	Ischaemic	IC, BG	H	–
P31	M, 65, 12	52	Haemorrhagic	FP	Normal	Yes
P32	M, 62, 12	449	Haemorrhagic	TO	H	–
P33	F, 62, 15	90	Ischaemic	O, Th	H	–
P34	M, 66, 13	12	Ischaemic	TP	Normal	Yes
C1	M, 74, 10					
C2	F, 72, 12					
C3	F, 21, 15					
C4	F, 31, 15					
C5	F, 75, 17					
C6	M, 70, 8					
C7	F, 63, 20					
C8	F, 69, 15					
C9	F, 34, 18					
C10	F, 72, 17					
C11	F, 73, 19					
C12	F, 57, 11					
C13	M, 68, 12					
C14	F, 30, 24					
C15	F, 66, 15					

IQ, inferior left quadrantanopia; SQ, superior left quadrantanopia; H, left hemianopia (with macular sparing); F, frontal; T, temporal; P, parietal; O, occipital; Th, thalamic; IC, internal capsule, BG, basal ganglia.

Apparatus and procedure

Perceptual task. Subjects sat in front of a computer monitor at a distance of approximately 50 cm. Three horizontally arranged black circles were displayed, the central circle being located at the centre of the screen. The distance between circles was 23 mm. During the test, the circles were always present on the screen. After an interval of 2000 msec, one of the circles became grey (target: Fig. 1).

When a right- or a left-side target appeared, subjects had to respond by pressing the computer spacebar with the index finger of the right hand as quickly as possible. Subjects had to refrain from responding when the middle circle became grey (catch trials, a procedure devised to avoid the possibility of subjects responding to the fixed time interval). Response time was measured from target onset to key press. The target disappeared when a response was made or after 5000 ms. One block of six practice trials and 10 blocks of four right- and four left-sided trials each were presented. The order of trials within a block was randomized. In order to minimize the influence of possible oculomotor components, subjects were instructed to keep fixation on the central circle. Eye position was monitored during the practice trials, and subjects were given appropriate feedback.

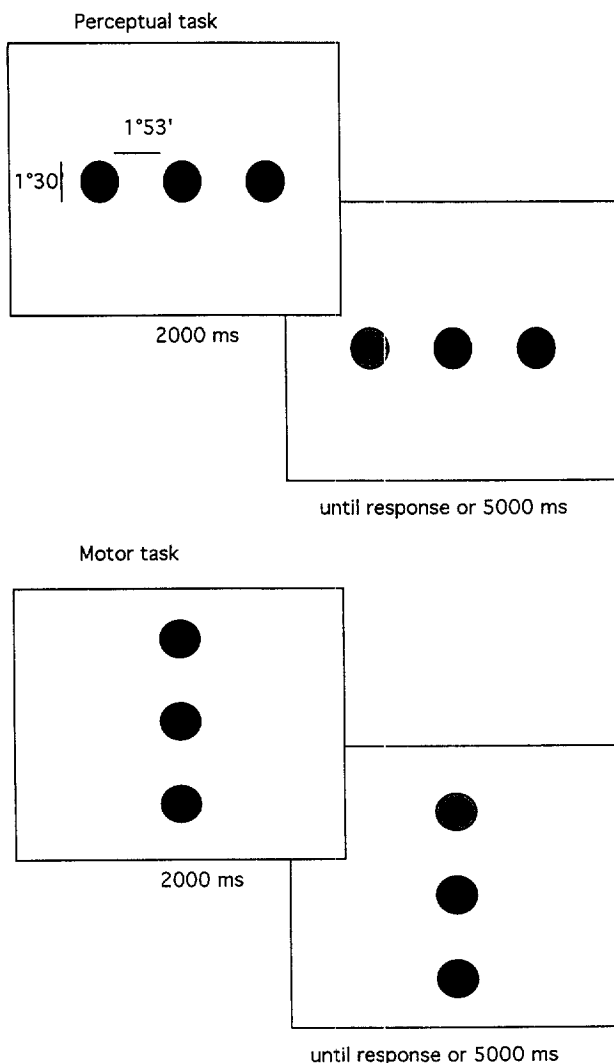


Fig. 1. Schematic drawing of trial events in perceptual and motor tasks. Distances are in degrees of visual angle.

Motor task. A paper board was placed on the computer keyboard, leaving three windows open on three different positions: a right-side area (keys 7, 8, 9, 4, 5, 6 of the numeric keypad), a middle area (keys i, o, p, k, l; of the American keyboard), and a left-side area (keys q, w, e, a, s, d). Right- and left-side areas were about 13 cm distant from the middle area. As in the perceptual task, three circles were presented on the computer screen, but they were arranged in a vertical array at the midsection of the screen. The distance between circles was 23 mm. After an interval of 2000 msec, one of the circles became grey (target: Fig. 1). Upon the appearance of an upper target, subjects had to move their right hand from the home position at the centre of the keyboard to whatever key was situated in the right-side area, at a distance of about 20 cm from the home position. When a middle target appeared, response keys were in the middle area, about 10 cm from the home position; when a lower target occurred, subjects had to press a key on the left-side area, about 20 cm to the left of the home position. The target disappeared when a response was made or after 5000 msec. After every trial, subjects had again to place their hand at the home position. Response time was measured from target onset to key press (our paradigm did not allow us to differentiate between the time of onset of the movement and its duration). Each area of response included six keys (instead of only one) in order to make the task easier for the subjects, by not demanding a precision reaching movement. One block of 12 practice trials and 10 blocks of four upper-, four middle-, and four lower-target trials each were presented. The order of trials within a block was randomized. A typical experimental session began with the motor task. The perceptual task and the visuospatial battery followed. At the end of the session, an inverted version of the motor task was performed (upper target → left-sided response, lower target → right-sided response). The response times for the two versions of the motor task were pooled, in order to minimize possible effects due to vertical neglect or stimulus-response compatibility.

Data analysis

Only correct responses were taken into account in the RT analysis. All responses with RTs either less than 100 msec or more than 4500 msec were discarded from analysis. For the motor task, only lateralized responses, i.e., responses directed to the right or the left side area, were taken into account.

To measure subjects' spatial bias independent of their overall performance level, laterality scores derived from Bryden and Sprott [12] were used. The rightward bias was estimated by

$$\lambda = \ln(x_1/x_2).$$

The "normal" range of spatial bias in the perceptual and motor task was defined by controls mean λ score ± 2 S.D.

A set of λ scores was obtained for each patient in order to perform the principal component factor analysis. The following measures were used to compute λ scores:

1. Visuospatial battery (see procedure in [4]).
2. RTs on the perceptual task (x_1 = mean RT to left-sided targets; x_2 = mean RT to right-sided targets).
3. Accuracy on the perceptual task (x_1 = number of responses to right-sided targets; x_2 = number of responses to left-sided targets).
4. RTs on the motor task (x_1 = mean RT for responses on the left side; x_2 = mean RT for responses on the right side).
5. Accuracy on the motor task (x_1 = number of correct right-sided responses; x_2 = number of correct left-sided responses).

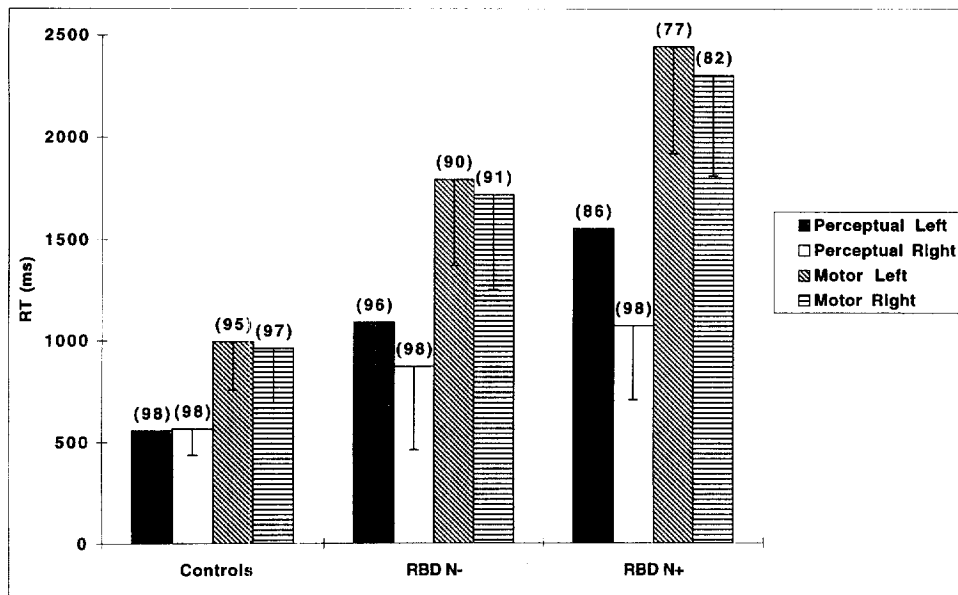


Fig. 2. Mean RTs and percentage of accuracy (in parentheses) for control subjects, RBD patients without (N-) and with neglect (N+) in the perceptual and the motor tasks. The error bars represent the standard deviations.

Results

Figure 2 presents the performance of control subjects, RBD patients without signs of neglect and RBD neglect patients on the perceptual and motor RT tasks. Accuracy and response times will be analysed separately.

Accuracy

The three groups of subjects exhibited different degrees of accuracy in the two tasks and the two sides of space [$\chi^2(11)=631.48$, $P<0.0001$]. Multiple paired comparisons were performed within each group and each task by subdividing the contingency table in 2×2 tables and using the Yates and Bonferroni corrections. The only significant differences came from performance of RBD neglect patients, who produced more correct responses to right-sided than to left-sided targets in the perceptual task [$\chi^2(1)=46.12$, $P<0.0001$], and more correct responses on the right side than on the left side in the motor task [$\chi^2(1)=8.97$, $P<0.005$].

Response times

A repeated measures analysis of variance was performed on the mean RTs with group (neglect, non-neglect, control) as between factor and target side (right, left) and test (perceptual, motor) as within factors. The analysis revealed a significant difference between groups [$F(2,46)=36.49$, $P<0.0001$]. *Post hoc* pairwise comparisons were carried out using Fisher's protected least significant difference. The two groups of RBD patients

were slower than the control group on both tests and both sides of space (all effects, $P<0.05$). Neglect patients were slower on the left than on the right side for the perceptual task (mean difference (MD)=479 msec, $P<0.005$), but not for the motor task (MD=141 msec, $P>0.3$). Neglect patients as a group did thus not present any evidence of directional hypokinesia. RBD patients without neglect showed a tendency to respond faster to right-sided than to left-sided targets in the perceptual task (MD=217 msec, $P=0.09$), but no left/right difference in the motor task (MD=76 msec, $P>0.5$). Control subjects did not present any evidence of spatial bias in either task (perceptual: MD=10 msec, $P>0.9$; motor: MD=33 msec, $P>0.8$).

The finding that our group of patients with neglect did not show any evidence for a leftward motor deficit in RT performance might be due to the fact that these two disorders did not always co-occur. To investigate this possibility, we performed a principal component factor analysis on the whole sample of RBD patients. Data for this analysis were laterality scores measuring rightward bias, obtained by each patient's performance on the visuospatial test battery and on the computerized tasks, for which both RTs and accuracy were taken into account. Table 2 shows the correlation matrix for the obtained laterality scores. The rotated factor matrix (Table 3) indicated two factors that accounted for 83% of the total variance.

Interestingly, all laterality measures loaded on factor 1 (which can be termed the "perceptual" factor), except scores derived from the RTs on the motor task, which loaded on factor 2 (which can be defined as the "motor" factor). Accuracy scores for the motor task also loaded on the perceptual factor. This result suggests that in the

Table 2. Correlation matrix of RBD patients' laterality scores

	Visuospatial battery	RTs perceptual	Accuracy perceptual	RTs motor
Visuospatial battery				
RTs perceptual	0.53			
Accuracy perceptual	0.71	0.51		
RTs motor	0.02	0.06	-0.11	
Accuracy motor	0.40	0.19	0.01	-0.04

Table 3. Varimax rotated factor matrix for the principal components analysis of laterality scores

	Loading on factor 1	Loading on factor 2
Visuospatial battery	0.92	-0.07
RTs perceptual	0.77	0.04
Accuracy perceptual	0.82	-0.30
RTs motor	-0.001	0.97
Accuracy motor	0.41	0.09

motor task neglect patients were impaired in the spatial coding of the left-side response site, but not in producing the response itself.

In order to analyse further the relationships between directional motor disorder and perceptual neglect, and to investigate the possible anatomical correlates of these deficits, we examined the individual patterns of performance of our patients using a multiple single-case approach. Figure 3 shows the laterality scores obtained by each patient on the visuospatial battery and on the perceptual and motor RT tasks.

Most RBD patients showed a rightward bias on the perceptual task (patients 1, 3, 4, 8, 11, 15-17, 19, 20, 23-25, 28-34), but on the motor task a definite rightward bias was present only in two patients (6 and 17). Patient 6 had a haematoma involving the superior parietal region and no signs of neglect on visuospatial testing. Some degree of rightward bias was present on the perceptual RT task, but it remained within the control range. Patient 17 had an ischaemic lesion centred on the superior parietal region and the adjacent dorsolateral part of the frontal lobe (Fig. 4). He showed signs of mild neglect on the visuospatial battery and slowed responses to left-sided targets on the perceptual task.

Discussion

We used reaction time tasks to visual stimuli to explore perceptual and motor aspects of spatial hemineglect in an unselected sample of right brain-damaged patients. In particular, our study aimed to investigate the role of a

possible deficit in programming arm movements towards the neglected space. The main advantage of our motor task compared with most previous paradigms used to study directional motor disorders was that no lateralized stimuli were present in subjects' visual fields. This was in order to minimize the influence of perceptual factors on the motor task. However, to detect a directional motor disorder the sites of response had to be lateralized; consequently, a slowing of left-directed responses would have been difficult to interpret as arising from purely motor problems and not, for example, from a deficit in the perceptual encoding of the left-side response area. Fortunately, most RBD patients did not show this directional slowing; they showed instead a decreased accuracy in left-directed responses. This finding strongly suggests that our paradigm was indeed apt to dissociate perceptual from praxic factors in neglect, as the encoding of response location apparently did not influence RTs once the movement was started.

Results of visuospatial testing suggest a continuum of impairment rather than a sharp dichotomy between neglect patients and patients without neglect (Fig. 3). This finding replicates previous results obtained with the same laterality score on different series of RBD and LBD patients (see [4], Fig. 2), and is consistent with the view that neglect is not a unitary, all-or-none phenomenon, but a complex behaviour resulting from the interaction of multiple deficits and compensatory mechanisms (see [3] for a discussion of this issue). The relative contribution of these multiple components might vary across different patients, thus causing different degrees of spatial bias.

Our controls did not show statistically reliable evidence for laterality effects, either in the perceptual or in the motor task. The notion of pseudoneglect [11], i.e., a leftward deviation on line bisection in normals, would have predicted an advantage of left-sided over right-sided stimuli in the perceptual task, and perhaps an advantage of left-directed responses over right-directed responses in the motor task. A 10 msec RT difference in the predicted direction was indeed observed in the perceptual task, but it did not reach statistical significance. In the motor task, a non-significant 33 msec difference was present, but with an advantage for right-directed responses. There was thus no evidence of pseudoneglect in our control sample. Indeed, pseudoneglect might be related to scan strategies

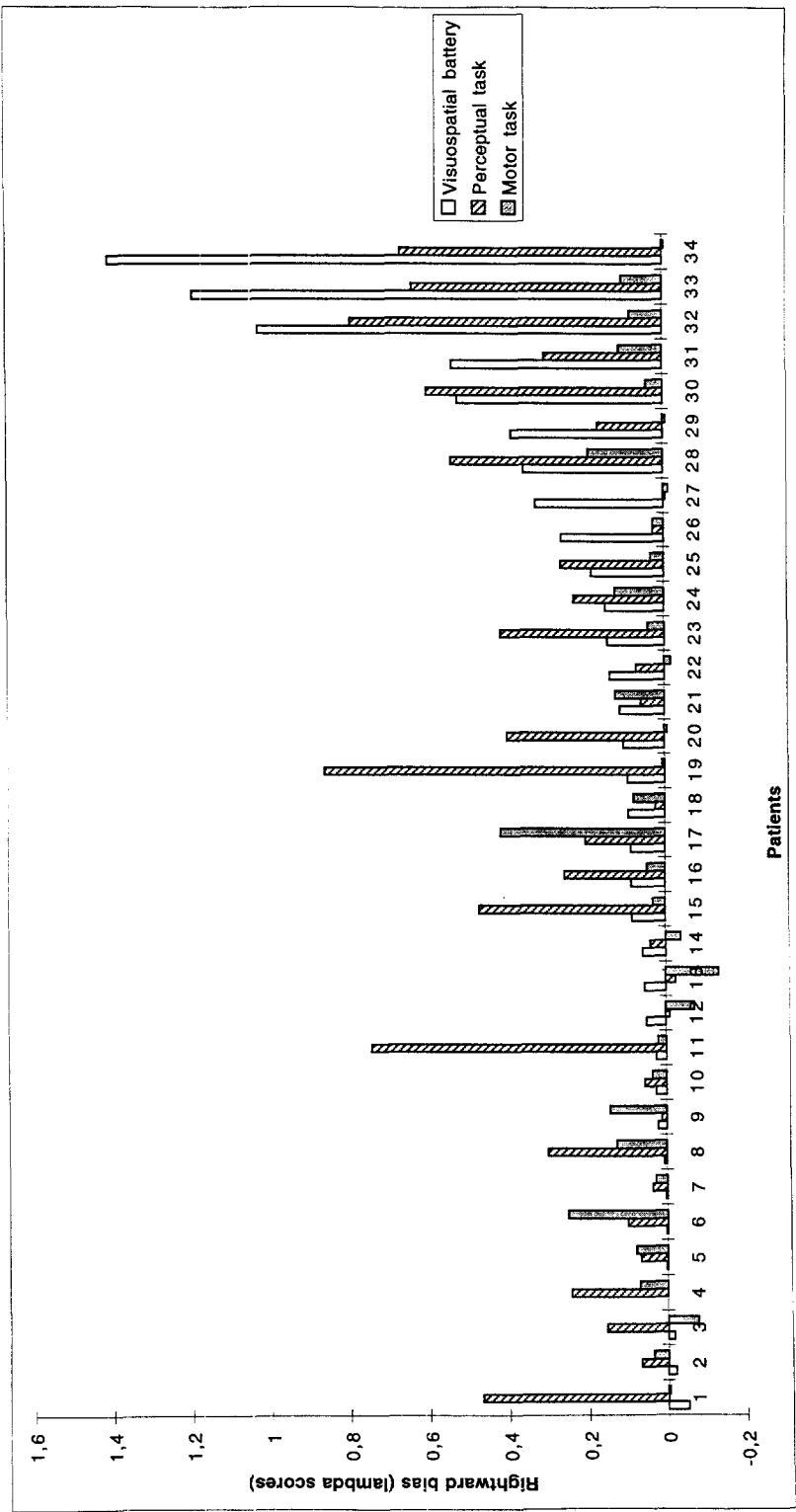


Fig. 3. Performance of individual patients on the visuospatial battery and the RT tasks. Patients are ordered according to the magnitude of rightward bias in the visuospatial battery. Lesion sites are reported. F, frontal; P, parietal; T, temporal; O, occipital; I, insula; IC, internal capsule; Th, thalamus; BG, basal ganglia.

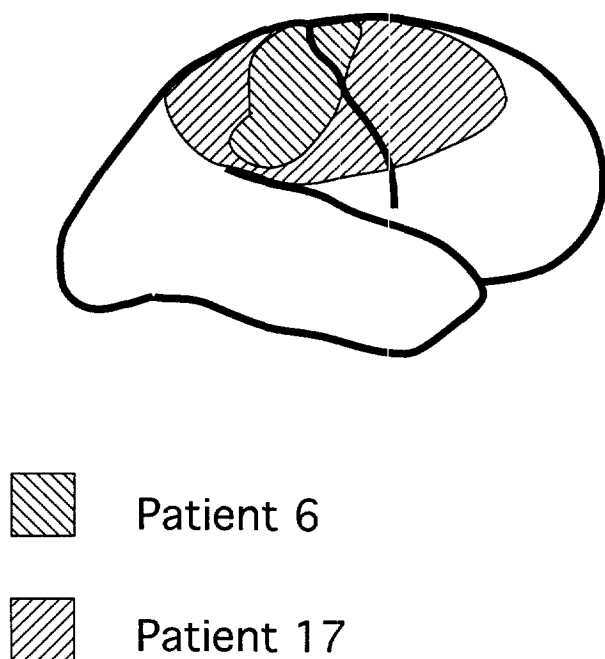


Fig. 4. Schematic reconstruction of lesions of patients 6 and 17, following the method of Damasio and Damasio [20].

more than to hemispheric activation, as suggested by the finding that right-to-left Israeli readers show a rightward deviation in line bisection [15, 16]; pseudoneglect might therefore be preferentially elicited by tasks (as line bisection) that emphasize scanning behaviour.

The response times of RBD patients were slower than controls in all test conditions, irrespective of the side of stimulation or of the side of response. Previous studies have shown that RBD patients are slower than left brain-damaged patients when responding to non-lateralized or ipsilesional stimuli [18, 29], thus suggesting the presence of a defective arousal in RBD patients.

The evaluation of RTs did not provide any evidence of directional motor disorder of the ipsilesional arm in our patients taken as a group. Principal components analysis revealed that the spatial bias on the perceptual RT task generally paralleled performance on the visuospatial neglect battery. A perceptual rightward bias was also disclosed by RT measure in patients without signs of neglect on the visuospatial battery (Fig. 3), thus confirming the greater sensitivity for visuospatial disorders of RT tests as opposed to paper-and-pencil tests [22, 44]. In particular, RT paradigms may foil compensatory strategies and reveal a residual spatial bias in patients who had recovered from neglect [3].

The possibility remains that the spatial bias shown by neglect patients in the perceptual task was influenced by a directional oculomotor deficit. If neglect patients did not follow examiner's instructions to maintain central fixation and made saccades to targets, they might have been slower to move their eyes towards left targets than towards right targets, thus producing longer RTs to left targets. Whereas this possibility is to be borne in mind as

a general caveat when the term "perceptual" is encountered in this study, it must be stressed that the focus of the present research, as well as of most previous studies on directional hypokinesia, is on arm movements. Indeed, oculomotor deficits may well have an independent status from arm motor deficits in neglect [9].

Our result, that impaired accuracy and decreased speed of response for leftward arm movements in the same (motor) task dissociate in different RBD patients, suggests that different cognitive processes are involved in localizing a target and reaching for it. The pattern of dissociations between these two deficits indicates that when performing the motor task, neglect patients are impaired in the initial selection of left-sided target location, as assessed by their defective accuracy of response. However, once this goal selection is accomplished, neglect patients are not, as a rule, slowed in performing the relative reaching movement. Looking at individual data, it appears that this directional slowing occurs only in a small subset of patients, thus perhaps explaining the lack of evidence for directional motor disorders in some of the studies reviewed in the Introduction. In our series, the only two patients who showed an unmistakable slowing of left-directed arm movements had lesions involving the superior part of the right parietal lobe. Some degree of rightward spatial bias was present in these two patients on the perceptual RT task, but they did not show signs of severe hemineglect on the visuospatial battery.

Consistent with the view that different systems are involved in the perceptual analysis of a stimulus and in making movements towards it [32, 42], evidence from normal subjects suggests that arm movements involved in aiming at a target require a coordinate transformation from a retinotopic frame of reference to other coordinate systems (e.g., shoulder-centred [48]). In this framework, if a visual perceptual frame of reference were the principal locus of impairment in neglect, one would expect exactly the present pattern of results, that is, a decreased accuracy in reaching left targets but a normal speed of leftward responses in those trials in which perceptual information was correctly translated into the appropriate coordinate frames. Additional levels of impairment seem to be required for a directional motor disorder to occur. In our sample, this deficit appears to be more related to parietal than to frontal lesions, contrary to the predictions of Mesulam [40]. However, it must be noted that we did not have the possibility of testing patients with isolated frontal lesions. Further investigations with our paradigm might disclose "frontal" forms of directional hypokinesia. A relationship between parietal lesions and directional motor disorders, although requiring confirmation, would not be surprising. The superior parietal cortex is the end-point of the visuomotor stream, and is connected in a modular fashion with the premotor areas of the frontal lobe, thus forming a parietofrontal visuomotor system [42].

Results of the principal components analysis suggest

that the defective accuracy to left side events in the perceptual and motor tasks may be accounted for by one single factor. However, neglect patients were more accurate in responding to left side events in the perceptual (86%) than in the motor (77%) task. To explain this difference, one may consider that the perceptual task was a simple RT task, whereas the motor task was a choice RT task rather demanding in terms of memory and sustained attention. This type of task demands are known to increase spatial bias in neglect (see e.g. [45, 46]). Therefore, we expected that neglect patients showed more rightward bias on the motor than on the perceptual task; the finding that this effect was evident only for accuracy, and not for RTs, further strengthen our conclusion that directional motor disorders do not play an important role in neglect.

Neglect patients showed similar accuracy when responding to left-sided stimuli in the perceptual task (86%) and when pressing the right-side keys in the motor task (82%; see Fig. 2). This finding may be merely coincidental, and reflect once again the more demanding nature of the motor task, which could also have decreased neglect patients' accuracy of performance on the ipsilesional side. This worsening of the overall performance in the motor task compared with the perceptual task might have masked in some instances a directional motor disorder. However, it seems unlikely that this mechanism could explain the failure to observe this disorder in the great majority of patients. Moreover, this mechanism leaves unexplained the observed dissociation between accuracy and time of performance, with a definite rightward bias observed only with accuracy.

Our results bear implications for theories of hemispatial neglect. Karnath [33] claims that an ipsilesional deviation of an egocentric reference frame is the crucial causal mechanism of neglect. Our finding of neglect patients' rightward bias in the perceptual condition is broadly consistent with this hypothesis (but see [3]). However, Karnath's hypothesis of a single frame of reference subserving the orienting of attention as well as eye and limb movements is challenged by the dissociation between performance on perceptual and motor tasks in our patients. If a systematic ipsilesional deviation of this reference frame determines neglect, then it should also manifest itself in tasks requiring a lateralized arm response.

To conclude, spatial hemineglect is a complex disorder resulting from the interaction of multiple attentional deficits [24] and compensatory mechanisms [3]. These processes may operate on various reference frames. A visual perceptual frame of reference appears to be the most common locus of impairment in neglect. In some patients, other spatial representations can be affected, including internally generated images and visuomotor programmes.

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References

1. Albert, M. L., A simple test of visual neglect. *Neurology*, 1973, **23**, 658–664.
2. Arbib, M. A., Perceptual structures and distributed motor control. In *Handbook of Physiology: The Nervous System II. Motor Control*, ed. V. B. Brooks. American Physiological Society, Bethesda, MD, 1981, pp. 1449–1480.
3. Bartolomeo, P., The novelty effect in recovered hemineglect. *Cortex*, 1997, **33**, 323–332.
4. Bartolomeo, P., D'Erme, P. and Gainotti, G., The relationship between visuospatial and representational neglect. *Neurology*, 1994, **44**, 1710–1714.
5. Battersby, W. S., Bender, M. B., Pollack, M. and Kahn, R. L., Unilateral "spatial agnosia" ("inattention") in patients with cerebral lesions. *Brain*, 1956, **79**, 68–93.
6. Bisiach, E., Mental representation in unilateral neglect and related disorders: the Twentieth Bartlett Memorial Lecture. *The Quarterly Journal of Experimental Psychology*, 1993, **46A**, 435–461.
7. Bisiach, E., Berti, A. and Vallar, G., Analogical and logical disorders underlying unilateral neglect of space. In *Attention and Performance XI*, eds M. I. Posner and O. S. Marin. Lawrence Erlbaum Associates, Hillsdale, NJ, 1985, pp. 239–249.
8. Bisiach, E., Geminiani, G., Berti, A. and Rusconi, M. L., Perceptual and premotor factors of unilateral neglect. *Neurology*, 1990, **40**, 1278–1281.
9. Bisiach, E., Tegnér, R., Ládavas, E., Rusconi, M. L., Mijovic', D. and Hjalton, H., Dissociation of ophthalmokinetic and melokinetic attention in unilateral neglect. *Cerebral Cortex*, 1995, **5**, 439–447.
10. Bottini, G., Sterzi, R. and Vallar, G., Directional hypokinesia in spatial hemineglect: a case study. *Journal of Neurology, Neurosurgery and Psychiatry*, 1992, **55**, 562–565.
11. Bowers, D. and Heilman, K. M., Pseudoneglect: effects of hemispace on a tactile line bisection task. *Neuropsychologia*, 1980, **18**, 491–498.
12. Bryden, M. P. and Sprott, D. A., Statistical determination of degree of laterality. *Neuropsychologia*, 1981, **19**, 571–581.
13. Butter, C. M., Rapsak, S., Watson, R. T. and Heilman, K. M., Changes in sensory inattention, directional motor neglect and "release" of the fixation reflex following a unilateral frontal lesion: a case report. *Neuropsychologia*, 1988, **26**, 533–545.
14. Buxbaum, L. J., Coslett, H. B., Montgomery, M. W. and Farah, M. J., Mental rotation may underlie apparent object-based neglect. *Neuropsychologia*, 1996, **34**, 113–126.

15. Chokron, S. and De Agostini, M., Reading habits and line bisection: a developmental approach. *Cognitive Brain Research*, 1995, **3**, 51–58.
16. Chokron, S. and Imbert, M., Influence of reading habits on line bisection. *Cognitive Brain Research*, 1993, **1**, 219–222.
17. Coslett, H. B., Bowers, D., Fitzpatrick, E., Haws, B. and Heilman, K. M., Directional hypokinesia and hemispatial inattention in neglect. *Brain*, 1990, **113**, 475–486.
18. D'Erme, P., Robertson, I., Bartolomeo, P., Daniele, A. and Gainotti, G., Early rightwards orienting of attention on simple reaction time performance in patients with left-sided neglect. *Neuropsychologia*, 1992, **30**, 989–1000.
19. Daffner, K. R., Ahern, G. L., Weintraub, S. and Mesulam, M.-M., Dissociated neglect behaviour following sequential strokes in the right hemisphere. *Annals of Neurology*, 1990, **28**, 97–101.
20. Damasio, H. and Damasio, A. R. (1989) *Lesion Analysis in Neuropsychology*. Oxford University Press, New York.
21. De Renzi, E., Gentilini, M., Faglioni, P. and Barbieri, C., Attentional shifts toward the rightmost stimuli in patients with left visual neglect. *Cortex*, 1989, **25**, 231–237.
22. Friedrich, F. J. and Margolin, D. I., Responses time measures of hemi-inattention: a longitudinal case report. *Neuropsychiatry, Neuropsychology and Behavioral Neurology*, 1993, **6**, 54–59.
23. Gainotti, G., Les manifestations de négligence et d'inattention pour l'hémi-espace. *Cortex*, 1968, **4**, 64–91.
24. Gainotti, G., D'Erme, P. and Bartolomeo, P., Early orientation of attention toward the half space ipsilateral to the lesion in patients with unilateral brain damage. *Journal of Neurology, Neurosurgery and Psychiatry*, 1991, **54**, 1082–1089.
25. Gainotti, G., Messlerli, P. and Tissot, R., Qualitative analysis of unilateral spatial neglect in relation to the laterality of cerebral lesions. *Journal of Neurology, Neurosurgery and Psychiatry*, 1972, **35**, 545–550.
26. Harvey, M., Milner, A. D. and Roberts, R. C., An investigation of hemispatial neglect using the landmark task. *Brain and Cognition*, 1995, **27**, 59–78.
27. Heilman, K. M., Bowers, D., Coslett, H. B., Whelan, H. and Watson, R. T., Directional hypokinesia: prolonged reaction times for leftward movements in patients with right hemisphere lesions and neglect. *Neurology*, 1985, **35**, 855–859.
28. Heilman, K. M., Watson, R. T. and Valenstein, E., Neglect and related disorders. In *Clinical Neuropsychology*, eds K. M. Heilman and E. Valenstein. Oxford University Press, New York, 1993, pp. 279–336.
29. Howes, D. and Boller, F., Simple reaction time: evidence for focal impairment from lesions of the right hemisphere. *Brain*, 1975, **98**, 317–332.
30. Ishiai, S., Sugushita, M., Watabiki, S., Nakayama, T., Kotera, M. and Gono, S., Improvement of left unilateral spatial neglect in a line extension task. *Neurology*, 1994, **44**, 294–298.
31. Ishiai, S., Watabiki, S., Lee, E., Kanouchi, T. and Odajima, N., Preserved leftward movement in left unilateral spatial neglect due to frontal lesions. *Journal of Neurology, Neurosurgery and Psychiatry*, 1994, **57**, 1085–1090.
32. Jeannerod, M., The representing brain: neural correlates of motor intention and imagery. *Behavioral and Brain Sciences*, 1994, **17**, 187–245.
33. Karnath, H.-O., Disturbed coordinate transformation in the neural representation of space as the crucial mechanism leading to neglect. *Neuropsychological Rehabilitation*, 1994, **4**, 147–150.
34. Kinsbourne, M., Orientational bias model of unilateral neglect: evidence from attentional gradients within hemispace. In *Unilateral Neglect: Clinical and Experimental Studies*, eds I. H. Robertson and J. C. Marshall. Lawrence Erlbaum Associates, Hove, U.K., 1993, pp. 63–86.
35. Ládavas, E., Umiltà, C., Ziani, P., Brogi, A. and Minarini, M., The role of right-sided objects in left side neglect: a dissociation between perceptual and directional motor neglect. *Neuropsychologia*, 1993, **31**, 761–773.
36. Liu, G. T., Bolton, A. R., Price, B. H. and Weintraub, S., Dissociated perceptual-sensory and exploratory-motor neglect. *Journal of Neurology, Neurosurgery and Psychiatry*, 1992, **55**, 701–706.
37. Marshall, J. C. and Halligan, P., Within- and between-task dissociations in visuo-spatial neglect: a case study. *Cortex*, 1995, **31**, 367–376.
38. Mattingley, J. B., Bradshaw, J. L. and Phillips, J. G., Impairments of movement initiation and execution in unilateral neglect. *Brain*, 1992, **115**, 1849–1874.
39. McFie, J., Piercy, M. F. and Zangwill, O. L., Visual spatial agnosia associated with lesions of the right hemisphere. *Brain*, 1950, **73**, 167–190.
40. Mesulam, M.-M., A cortical network for directed attention and unilateral neglect. *Annals of Neurology*, 1981, **10**, 309–325.
41. Mijovic, D., Mechanisms of visual spatial neglect: absence of directional hypokinesia in spatial exploration. *Brain*, 1991, **114**, 1575–1593.
42. Milner, A. D. and Goodale, M. A., *The visual brain in action*. Oxford University Press, Oxford, 1995.
43. Nico, D., Detecting directional hypokinesia: the epidiascope technique. *Neuropsychologia*, 1996, **34**, 471–474.
44. Posner, M. I., Walker, J. A., Friedrich, F. J. and Rafal, R. D., Effects of parietal injury on covert orienting of attention. *Journal of Neuroscience*, 1984, **4**, 1863–1864.
45. Rapcsak, S. Z., Verfaellie, M., Fleet, W. S. and Heilman, K. M., Selective attention in hemispatial neglect. *Archives of Neurology*, 1989, **46**, 178–182.
46. Robertson, I. H., The relationship between lateralised and non-lateralised attentional deficits in unilateral neglect. In *Unilateral Neglect: Clinical and Experimental Studies*, eds I. H. Robertson and J. C. Marshall. Lawrence Erlbaum Associates, Hove U.K., 1993, pp. 257–275.
47. Schenkenberg, T., Bradford, D. C. and Ajax, E. T., Line bisection and unilateral visual neglect in patients with neurologic impairment. *Neurology*, 1980, **30**, 509–517.

48. Soechting, J. F., Tillery, S. I. H. and Flanders, M., Transformation from head- to shoulder-centered representation of target direction in arm movements. *Journal of Cognitive Neuroscience*, 1990, **2**, 32–43.
49. Tegnér, R. and Levander, M., Through a looking glass. A new technique to demonstrate directional hypokinesia in unilateral neglect. *Brain*, 1991, **114**, 1943–1951.
50. Vallar, G., The anatomical basis of spatial hemi-neglect in humans. In *Unilateral Neglect: Clinical and Experimental Studies*, eds I. H. Robertson and J. C. Marshall. Lawrence Erlbaum Associates, Hove, U.K., 1993, pp. 27–59.