The disengagement of covert attention and the neglect syndrome

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Parietal lesions can cause impairment of the ability to disengage attention from an ipsilateral stimulus prior to shifting the focus of attention contralaterally. The present study addresses two main questions concerning the parietal-based disengagement process. First, what is the relationship between the disengagement process, as operationalized by a cued simple reaction-time task, and the neglect syndrome? Second, is there an asymmetry in the roles of the left and right hemisphere in the disengagement of attention? We found a very strong relationship between clinical neglect and impairment of the disengagement operation across patients, and longitudinally, within patients. Furthermore, we found that right-hemisphere lesions cause greater impairment in the disengage process than do left-hemisphere lesions.

Many patients confronted with a stimulus on the side of the brain contralateral to a lesion behave as if they have not perceived that stimulus. This syndrome of unilateral neglect, or hemi-inattention, is most common after damage to the right parietal area, but it has been found in patients with lesions in other areas, including the left hemisphere (Ogden, 1985), the right frontal lobe (Butter, Rapcsak, Watson, & Heilman, 1988; Damasio, Damasio, & Chang Chui, 1980; Heilman & Valenstein, 1972; Van Der Linden, Seron, Gillet, & Bredart, 1980), and subcortical structures (Watson & Heilman, 1979).

A number of theories have been proposed to account for the neglect syndrome. Many early studies hypothesized that neglect was simply the result of a loss of sensation or perception (Battersby, Bender, Pollack, & Kahn, 1956; Denny-Brown, Meyer, & Horenstein, 1952; McFie, Piercy, & Zangwill, 1950). Subsequent theorists have proposed that neglect is due either to an imbalance of activation between the two hemispheres (Kinsbourne, 1970, 1977) or to a deficit in arousal that is concomitant with a unilateral disruption of a cortico-limbic-reticular loop (Heilman, 1979; Heilman & Valenstein, 1972; Heilman & Van Den Abel, 1979; Heilman & Watson, 1977). More recently, Posner, Walker, Friedrich, and Rafal

(1984) have studied attentional processes in parietal-damaged patients by using a theoretical framework and an experimental paradigm developed in the cognitive psychology laboratory. They have found evidence that lesions of the parietal lobe disrupt one specific operation required for spatial attention.

More specifically, the model proposed by Posner et al. (1984) assumes that the orienting of spatial attention includes both overt and covert operations, directed by specific neural systems. For example, when a signal in the environment alerts an organism, the head and eyes usually move towards the stimulus—an overt alignment of attention. However, attention may also be directed towards the stimulus without any head or eye movements—a covert alignment of attention. Results from visual reaction-time studies with normal subjects (Posner, 1980) suggest that three specific mental operations—"disengage," "move," and "engage"—are involved in the covert orienting of attention. When a spatial cue signifies an upcoming target in the same position, one needs only to engage attention when the target appears. If no cue is given as to the location of an upcoming target, the appearance of a target will result first in attention being moved to the target location, and then in engagement of the target. Finally, if a cue is presented in one place and the target appears elsewhere, attention must first be disengaged from the invalidly cued location and moved to the new location, where the target is then engaged.

To test their model of covert orienting, Posner and colleagues (Posner, Cohen, & Rafal, 1982; Posner et al., 1984) administered the visual reaction-time paradigm to patients with cerebral lesions. They found an interesting dissociation: patients with parietal lesions were impaired on the disengage operation, while patients with damage to the midbrain were impaired on the move operation.

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Impairment on the disengage operation has not, however, been related to the neglect syndrome (Posner et al., 1982; Posner, Inhoff, Friedrich, & Rafal, 1987; Posner et al., 1984)—although the clinical phenomenon is quite consistent with a disengage explanation (Mark, Kooistra, & Heilman, 1987). The majority of patients in the Posner et al. (1984) study showed no evidence of clinical neglect. Furthermore, whereas neglect is more common after right, as opposed to left, parietal damage, they did not find significant differences between the groups in severity of impairment on the disengage operation. It might then be argued that the disengage deficit and the neglect are distinct and separate phenomena. This apparent support is limited, however, by the small number of patients tested—6 patients with right parietal lesions and 7 patients with left parietal lesions. Moreover, many of the patients were 3 to 10 years postlesion onset.

In the present study, we ask two questions: (1) What is the relationship between clinical neglect and the ability to disengage attention to the contralateral hemisphere? (2) In a larger sample of patients, with more recent lesion onset, will an asymmetry be found in the disengage operation?

METHOD

Subjects

Twelve patients with right-hemisphere lesions (RHLs) and 10 patients with left-hemisphere lesions (LHLs) were recruited from the inpatient and outpatient clinics at Harnarville Rehabilitation Center. Patients either had a lesion that included the parietal lobe or showed evidence of neglect, as indicated in their medical charts. All patients were right-handed and none had any history of psychiatric disorder.

The sides of the lesions were established by reference to history, a neurological report, and CT scan data. Three scans were done within 24 h of admission and were reported to be negative. Each patient had a contralateral hemiparesis. Visual fields were full to confrontation within 45° of fixation. The majority of patients had sustained vascular lesions and were tested within 2 months of lesion onset. The median lesion-to-test interval was 55 days for the RHL group and 40 days for the LHL group. Demographic data for the patients are presented in Table 1.

Apparatus and Design

Tests for neglect. Three tests specifically designed to assess clinical neglect were administered: a letter-cancellation test, a line-bisection test, and a visuoconstruction test. On the letter-cancellation task, patients were presented with an 8 × 11 in. page containing 17 lines of random letters. There were 22 letters on each line, and patients were asked to cross out all occurrences of the letter E. On the line-bisection test, patients were given five pages, each of which contained a single dark horizontal line, and they were asked to make a mark at the center point of the line. Lines of various lengths (3, 10, 15, 20, and 25 cm) were presented randomly to each patient. The visuoconstruction test required patients to copy the Rey-Osterrieth complex figure (Rey, 1941).

The patients were assigned a score for each of these tests. The scores for the letter-cancellation test consisted of the number of specified letters omitted on the neglected side (no patient omitted a letter on the nonneglected side). Scores on the line-bisection test were obtained by measuring the number of millimeters each patient's mark deviated from the center (again, no patient deviated to the nonneglected side). The Rey figure was scored in the standard fashion (Taylor, 1959); in addition, scores for those units, or elements, that were to the left or right of the midline were added separately and a difference score between the two was computed. The neglect score for the Rey figure was based on this difference. To obtain an overall score for neglect, scores on the three tests were converted to *z* scores and averaged.

Table 1
Summary of Demographic and Clinical Data

Patients	Age*	Sex	DX	CT Report	Medical Report	Lesion-to-Test†
RH1	77	M	CVA	negative	mild neglect	54
RH2	65	M	CVA	negative	mild neglect	32
RH3	70	M	CVA	parietal/frontal	moderate neglect	420
RH4	65	M	Tumor	parietal craniotomy	no neglect	80
RH5	60	F	CVA	temporal/parietal	severe neglect	42
RH6	54	M	CVA	parietal	severe neglect	56
RH7	61	M	CVA	parietal	mild neglect	45
RH8	41	M	CVA	frontal/parietal	moderate neglect	31
RH9	66	M	CVA	parietal	moderate neglect	46
RH10	56	F	CVA	negative	moderate neglect	364
RH11	57	M	CVA	parietal	moderate neglect	59
RH12	59	M	CVA	parietal	moderate neglect	161
LH1	73	F	CVA	parietal	mild neglect	60
LH2	60	F	CVA	temporal/parietal	no neglect	36
LH3	41	M	CVA	parietal	no neglect	28
LH4	47	M	CVA	negative	mild neglect	32
LH5	44	M	CVA	parietal	mild neglect	56
LH6	58	F	CVA	parietal	no neglect	65
LH7	62	M	CVA	parietal/occipital	no neglect	49
LH8	69	F	CVA	negative	mild neglect	38
LH9	39	F	AVM	parietal craniotomy	mild neglect	44
LH10	53	F	CVA	temporal/parietal	no neglect	38

*years. †days.

RESULTS

Reaction time to visual targets. For the visual reaction-time task, the subjects were seated in front of a video monitor at a distance of .8 m. The stimuli were generated by an Apple IIE computer, and the reaction times were stored on a diskette. For each trial, three squares of equal dimension (4 × 4 cm) appeared at the same horizontal level on the monitor, with the left and right squares placed 7° from the center square. A trial was initiated by increasing the brightness of one of the three squares (cue). Following this cue, a cross (target) appeared in the center of one of the two side squares. This target was presented at intervals of 50, 150, 550, or 1,000 msec after completion of the cue onset. The subject's task was to respond as quickly as possible to the target by depressing the space bar. Trials were either *valid* (i.e., the cue and target occurred in the same square) or *invalid* (i.e., the target occurred in the square opposite the one that displayed the cue). Seventy-five percent of the trials were valid; 25% were invalid.

Procedure

The display was viewed binocularly at eye level, in a darkened room. A patient was instructed to rest his/her eyes fixated on the center of the middle square during each trial. Sixteen practice trials and 352 test trials were administered. The test trials were divided into four blocks of 88 trials. Each block contained, in pseudo-random order, both valid and invalid targets at the various cue-target intervals. The order of the blocks was randomized across patients, and one of the clinical neglect tests was administered between each block of trials.

To avoid including anticipatory responses or trials that clearly were misses, reaction times under 150 msec or over 5,000 msec were excluded from the data analysis. Less than 3% of the trials were excluded. For the remaining reaction times, medians were calculated for each patient in each of the conditions. Table 2 presents the individual data for each of the conditions, as well as the means and standard deviations for the two groups. These data are shown graphically in Figure 1.

Between-Group Comparisons

The median reaction-time data were analyzed by a repeated measures analysis of variance with group (RHL, LHL) as a between-subjects factor and validity (valid, invalid), target side (left, right), and interval (50, 150, 550, 1,000 msec) as within-subjects factors. Significant main effects were found for validity [$F(1,20) = 15.4, p < .001$] and interval [$F(3,60) = 11.3, p < .001$]. In addition, there were a number of two-way and three-way interactions: group × validity [$F(1,20) = 7.6, p < .01$], group × target side [$F(1,20) = 6.4, p < .01$], validity × target [$F(1,20) = 5.5, p < .02$], interval × group

Table 2
Median Reaction Times for Ipsilateral and Contralateral Targets
at the Valid and Invalid Conditions Across Intervals*

Patient	Valid				Invalid				Valid				Invalid			
	50	150	550	1000	50	150	550	1000	50	150	550	1000	50	150	550	1000
Right Side (Ipsilateral)									Left Side (Contralateral)							
RH1	390	375	359	328	476	375	359	398	453	421	382	351	531	437	445	392
RH2	484	453	453	453	515	484	445	437	484	484	484	453	484	500	484	597
RH3	453	468	390	484	609	562	484	515	390	453	453	468	718	578	593	515
RH4	343	296	281	242	304	304	250	234	335	320	304	265	304	281	250	242
RH5	687	718	595	640	765	851	726	593	757	750	585	632	1125	1250	1273	1078
RH6	546	578	640	593	671	828	710	546	1000	820	750	664	1546	1406	991	828
RH7	406	375	367	406	406	398	414	437	390	453	453	421	546	476	676	496
RH8	500	546	445	453	578	617	515	500	548	585	515	515	671	656	718	618
RH9	640	710	671	453	640	484	726	578	1031	703	976	820	1640	1270	750	710
RH10	531	593	515	515	601	703	632	671	609	609	585	468	940	734	664	609
RH11	710	640	562	609	695	750	679	656	1609	1093	2562	1742	2859	3687	2281	1062
RH12	515	453	515	515	578	625	562	585	515	468	515	500	1390	1546	1773	648
Mean	517	517	482	474	569	581	541	512	676	596	713	608	1062	1068	908	649
SD	115	135	121	114	128	178	157	122	371	215	607	385	716	928	593	246
Left Side (Ipsilateral)									Right Side (Contralateral)							
LH1	546	562	515	515	593	588	578	546	679	867	671	578	820	773	765	710
LH2	484	500	515	484	515	484	484	484	578	546	546	515	584	601	515	546
LH3	265	218	218	203	257	218	210	218	281	265	218	234	289	250	226	234
LH4	289	289	257	265	296	265	281	296	359	421	343	335	515	425	359	328
LH5	540	550	562	405	540	535	411	411	525	460	459	482	620	510	488	555
LH6	367	398	406	406	468	510	445	439	476	476	468	445	468	500	460	429
LH7	312	281	281	265	321	320	289	250	359	328	312	296	421	382	351	335
LH8	421	398	328	359	390	343	343	296	421	406	375	367	453	406	421	328
LH9	687	617	625	648	671	703	773	671	687	671	671	718	781	765	804	734
LH10	445	453	421	390	562	500	414	453	484	578	531	484	703	593	531	515
Mean	435	426	412	394	461	446	422	406	484	501	459	445	565	520	492	471
SD	133	133	139	132	139	154	163	142	135	174	150	143	168	166	178	169

Note.—*Intervals and reaction times in milliseconds.

Figure 1. Mean reaction time for right-hemisphere (Panel A) and left-hemisphere (Panel B) lesioned groups. ● Valid ipsi, ○ invalid ipsi, ■ valid contra, □ invalid contra.

[$F(3,60) = 2.6, p < .05$], validity $\times$ target $\times$ group [$F(1,20) = 8.3, p < .01$], and target $\times$ interval $\times$ group [$F(3,60) = 2.7, p < .05$].

The main effects of validity and interval revealed that valid trials elicit faster responses than invalid trials, and that the longer the time between the cue and the target, the faster the reaction time. Pairwise comparisons of the cue-target interval means revealed significant decreases in reaction time between 150- and 550-msec ($p < .01$) and 550- and 1,000-msec intervals ($p < .01$).

Tests of simple main effects and Dunn's *a priori* comparisons (Kirk, 1982) were used to identify the nature of the interactions. The group $\times$ validity interaction demonstrates that patients with RHL are slower to respond to invalid trials than patients with LHL ($p < .05$). The group $\times$ target-side interaction is the result of the increase in reaction times for the RHL group whenever a target occurs on the left side, contralateral to the lesion ($p < .001$), and of a corresponding increase in response latencies for the LHL group when the target appears on the right side, contralateral to the lesion ($p < .01$). Because patients with RHL show such a large increase in reaction times to targets on the neglected side, the validity $\times$ target-side interaction reached significance. Moreover, since this increase is most pronounced at the early cue-target intervals, the interaction between validity and interval was significant.

The group $\times$ validity $\times$ target-side interaction is the result of the large increase in reaction times to invalid left-side targets in the RHL group. That is, for patients with lesions in the right hemisphere, when a cue appears on the side ipsilateral to the lesion and is followed by a target on the contralateral side, there is a large increase in the time taken to respond to that target. This behavior is typically referred to as extinction, and Posner et al. (1984, p. 1866) labeled this the "extinction-like reaction time pattern" (see Figure 1A, top line). Patients with LHL, however, do *not* take significantly more time to respond to a target on the side contralateral to the lesion when the target is preceded by a cue on the ipsilateral side.

Tests of simple main effects for the above three-way interaction demonstrate that, within the RHL group, the difference between valid and invalid trials is significant ($p < .001$). Furthermore, reaction times to invalid contralateral targets are significantly longer than reaction times to valid contralateral targets ($p < .01$). There are no differences between valid and invalid conditions on the ipsilateral side. Although patients with LHL take longer to react to invalid trials, and take more time overall to respond to targets on the contralateral side, there is no interaction between side and validity ($p > .05$).

The group $\times$ target side $\times$ interval interaction demonstrates the facilitation effect of increasing cue-target intervals for patients with RHL when the target occurs on the contralateral (left) side. That is, when the cue and target are separated by relatively long intervals (1,000 msec), the time to respond to targets on the left side is much quicker than when the cue and target are separated by short intervals (50 msec). We find this to be true only for patients with RHL.

An inspection of Figure 1 will show that this large decrease in reaction time across intervals for patients with RHL is most dramatic for invalid contralateral targets (the extinction-like reaction-time pattern). Although the four-way interaction is not significant, these data seem to warrant attention. For patients with RHL, when a cue summons attention to the ipsilateral side and a target occurs 50 or 150 msec later on the contralateral side, reaction times to the target are quite long. However, reaction times decrease steadily as the cue-target interval increases: by the 1,000-msec interval, there is only a 40-msec difference between invalid and valid contralateral targets. That is, when a cue that occurs on the ipsilateral side is followed by a target on the contralateral side, patients with RHL, when given enough time, will respond to the target almost as quickly as if they had been cued to the contralateral side. Because the four-way interaction did not reach significance, we cannot test these comparisons. This decrease does not seem to be due to a small number of outliers, but appears to be a consistent pattern for the majority of patients with RHL (see Table 2).

Relationship Between Neglect and the Disengagement of Covert Attention

One of the objectives of this study was to test the relationship between the severity of clinical neglect and the

time taken to respond to invalidly cued targets in the contralateral field. To test this, we computed the validity effect, at the 50-msec interval, for each patient: we determined the differences in reaction-time latencies between the valid and invalid trials at the 50-msec interval for targets on the ipsilateral side and targets on the contralateral side, and then the differences between the two sides. For example, at the 50-msec cue-target interval, Patient RH12 had a difference of 875 msec for contralateral targets (1390–515) and a difference of 63 msec for ipsilateral targets (578–515). His validity effect, then, is 812 (875–63). These difference scores were then correlated with each patient's neglect *z* score. The clinical-neglect *z* scores and the validity effects for the individual patients are presented in Table 3.

Rank-order correlations revealed a highly significant correlation between clinical neglect and the reaction-time difference scores for patients with RHL ($\rho = .91$, $p < .001$), but a nonsignificant correlation for patients with LHL ($\rho = .05$).

Although the correlations in the RHL group are quite large, it is conceivable that several other factors might affect performance. To test whether the number of days postonset or the patient's age had an effect on the reaction-time difference score, a regression analysis was run separately for each group, with age, days postonset, and neglect scores as the independent variables. In the LHL group, no variables entered the equation; in the RHL group only the neglect scores entered it ($R^2 = .75$).

Longitudinal Assessment of Neglect and the Disengagement of Covert Attention

The effects of recovery on covert orienting were tested by reevaluating 4 patients shortly after their initial assessment. All of the patients (RH5, RH7, RH8, and RH12) were administered the neglect measures and the reaction-time task on both occasions. Patient RH5 was initially tested 42 days postonset and, at that time, showed clear evidence of left-side neglect ($z = 0.81$), as well as a distinct pattern of increased reaction times to invalid contralateral targets. When reevaluated at 134 days postonset, Patient RH5 showed virtually no improvement on the neglect tests ($z = 0.89$) and almost no change in reaction times. Patient RH12, tested on Days 161 and 246 poststroke, demonstrated a similar pattern of performance: clinical neglect at both occasions (z s = 2.14 and 2.33, respectively), and the extinction-like reaction-time pattern at both assessments. Graphs of these patients' reaction times and their copies of the Rey figure are shown in Figures 2 and 3.

A somewhat different pattern was found for Patients RH7 and RH8. Patient RH7 was tested initially at 45 days poststroke and again at 139 days poststroke. At the first test session, RH7 obtained a clinical-neglect *z* score of 0.20. At the second test session, his clinical-neglect score improved to 0.02. Similarly, Patient RH8, who was tested at 31 and 65 days poststroke, showed evidence of recovery on the clinical-neglect measures (z s = 0.62 and –0.40, respectively). In addition, both of these patients

Table 3
Clinical Neglect Scores and Validity Effect*

Patient	Z score	Validity Effect
RH1	0.066	–8
RH2	0.099	–31
RH3	0.231	172
RH4	–0.511	70
RH5	0.811	290
RH6	1.037	421
RH7	0.209	156
RH8	0.628	45
RH9	1.586	609
RH10	0.761	261
RH11	2.231	1265
RH12	2.146	812
Mean	0.774	338
LH1	0.210	94
LH2	–0.341	–25
LH3	–0.551	16
LH4	–0.661	149
LH5	–0.275	95
LH6	–0.451	–109
LH7	0.491	53
LH8	–0.065	63
LH9	–0.154	110
LH10	–0.253	102
Mean	–0.205	54

*Higher scores represent poorer performance. The validity effect is for invalid–valid RT at 50 msec, contralateral–ipsilateral.

demonstrated quicker reaction times to invalid contralateral targets at their second assessment—that is, a decrease in their extinction-like reaction-time pattern. Figures 4 and 5 present graphs of reaction times and copies of the Rey figure at both assessments for these 2 patients.

Precautions Against Eye Movements

Although the patients in this study were instructed to maintain fixation at center, it was possible that they might have moved their eyes in the direction of the cue—especially since they were told that the cue and target would usually occur in the same location. To try to control for this, we administered a small set of “neutral” trials. On these trials, the cue occurred at center and the target was presented in either the left or the right box with equal frequency. Thus the cue was in the same location as eye fixation, and was not informative regarding the placement of the upcoming target. The pattern of performance on the neutral trials was identical to that seen on the invalid trials. That is, for patients with RHL, when the cue was at fixation, the reaction times to targets in the neglected hemisphere were significantly longer than the reaction times to ipsilateral targets. This would suggest that the cue was not being utilized by the patients to direct their eye movements.

DISCUSSION

In the present study, the operations of covert attention were examined in patients who showed evidence of clinical neglect or had sustained left or right parietal lobe le-

Figure 2. Mean reaction time and Rey figure copy for Patient RH5 at initial testing (Panel A) and follow-up testing (Panel B).

Figure 3. Mean reaction time and Rey figure copy for Patient RH12 at initial testing (Panel A) and follow-up testing (Panel B).

Figure 4. Mean reaction time and Rey figure copy for Patient RH7 at initial testing (Panel A) and follow-up testing (Panel B).

Figure 5. Mean reaction time and Rey figure copy for Patient RH8 at initial testing (Panel A) and follow-up testing (Panel B).

sions. The results demonstrate that patients with RHL show a distinct pattern of reaction times on a visual orienting task that is quantitatively different from that of patients with LHL.

When a warning stimulus in the environment signals an upcoming target in the same spatial location, patients with RHL react quickly to the target if it is in the right

hemisphere, ipsilateral to the lesion, but they take more time to respond to a spatially primed target in the contralateral hemisphere. Likewise, when a cue occurs on one side and is followed by a target on the other side, these patients make a rapid response if the target is located on the ipsilateral side. However, if the target is located in the contralateral hemisphere and preceded by an ipsilateral

cue, there is a significant increase in reaction time. There is a tendency for patients with RHL to respond more quickly to invalid contralateral targets if the cue and target are separated by longer intervals (1,000 msec), although this tendency is not significant.

Surprisingly, patients with LHL did not show the extinction-like reaction-time pattern, even though they did show slightly longer latencies for invalidly cued targets on the contralateral side at the 50 msec cue-target interval. Posner et al. (1984) did find a significant increase in reaction times to invalid contralateral targets in their sample of left parietal lesioned patients, but it was not as pronounced as that found in patients with right parietal lesions. Although utilizing a somewhat different paradigm, Baynes, Holtzman, and Volpe (1986) also found that patients with RHL, when compared with controls and patients with LHL, were especially slow to shift attention in the neglected hemifield.

Our data support the hypothesis that there is a selective, directional impairment of the disengage operation in patients with right-hemisphere lesions. However, while Posner et al. (1984) found that responses to valid contralateral targets (cue and target on the neglected side) were faster than those to invalid ipsilateral targets (cue on the neglected side, target on the nonneglected side), we found the reverse to be true. Posner suggested that the facilitation effect of spatially primed contralateral targets indicated that the move operation was not responsible for increasing reaction times to invalidly cued contralateral targets. Since, in the present study, we found significant reductions in reaction times to valid contralateral targets, relative to either invalid or valid ipsilateral targets, there may be some slowing of the move operation. Alternatively, even though the patients reported here did not have visual field deficits on confrontation, the increased latency of response time to stimuli presented to the contralateral side may have been due to subtle sensory loss.

For most of the patients with RHL, the responses to invalid contralateral targets were faster on the trials with longer cue-target intervals. Although the four-way interaction did not reach significance, the steep reduction in reaction times across intervals for invalid contralateral targets in the RHL group is intriguing. This might suggest that the disengage impairment may be refractory. That is, if attention has been engaged to the ipsilateral side, a certain time interval (for our sample, approximately 550 msec) must elapse before the disengage operation can be initiated. It is interesting that one of the patients reported by Posner et al. (1984), Patient J.C., also showed a steep decline in reaction times to invalidly cued contralateral targets at the longest cue-target interval, dropping in reaction time from 1,032 to 946, to 942, to 434 msec across the four intervals.

One of the differences between the present study and that of Posner et al. (1984) is the lesion-to-test interval. In Posner's sample, 4 of the 6 patients with RHL had sustained their lesions 3 to 10 years previously. Furthermore,

very few were noted to have signs of clinical neglect—as determined by an estimate based on activities of daily living. The authors concluded that the extinction-like reaction-time pattern is most likely related to the amount of damage done to the superior parietal lobe. Interestingly, however, their one patient who did show a “moderate” amount of neglect was only 2 months poststroke and also demonstrated the largest difference between invalidly cued targets on the two sides.

In our sample, the majority of the patients with RHL demonstrated at least some clinical neglect. Indeed, the patients who had the most severe neglect also had the largest increase in reaction time for invalid targets presented to the neglected side. For these patients with RHL, it seems that the magnitude of the extinction-like reaction-time effect depended on the severity of clinical neglect. For the patients with LHL, we cannot draw the same conclusions, mainly because these patients exhibited minimal neglect and very little evidence of the extinction-like reaction-time pattern.

The longitudinal results of 4 patients in this study lend further support to the hypothesis that clinical neglect may have significant bearing on the extinction-like reaction-time pattern. The 2 patients who improved on clinical neglect measures also decreased their reaction times to invalid contralateral targets. The 2 patients whose clinical neglect remained unchanged showed little change in reaction times. In fact, Patient RH5's reaction times to invalid contralateral targets were almost identical at the two testing sessions.

In addition, it seems that the patients reported by Posner et al. (1984) had lesions that were quite extensive, “large ones often extending far in the posterior (occipital lobe) and anterior (frontal lobe) directions” (p. 1871). It may be that these patients' lesions were so large, and encompassed so much cortical area, that the extinction-like effects were more pronounced even 10 years following lesion onset. In the present patient sample, the documented lesions were probably less extensive (although we do not have exact measurements); otherwise the patients would not have been well enough to participate in an extensive rehabilitation program so soon after onset, nor would they have been able to carry out the task. The majority of the CT scan reports for the present sample noted damage mainly to the parietal lobe, without extensive damage to a large surrounding area. Although it is not reported, the patients in Posner's sample may have had severe neglect initially after their lesions and subsequently, over the years, may have learned to compensate enough for the neglect to have become less evident on clinical measures. The extent of injury along with the amount of behavioral neglect may therefore contribute to this extinction-like reaction-time pattern.

In this connection, we note that our only patient with a right-hemisphere lesion who did not demonstrate a dissociation between reaction times to invalid ipsilateral and contralateral targets had a parietal craniotomy and no evidence of neglect (Patient RH4). Furthermore, we found

the extinction-like pattern in a neglecting patient whose lesion was predominantly frontal, although there was some extension into the parietal convexity (Patient RH8). These data would be consistent with several theories.

Heilman (1979), for example, has suggested that lesions outside the parietal area that disrupt the corticolimbic reticular loop can result in a failure to attend to the contralateral hemispace. Rizzolatti, Gentilucci, and Matelli (1985) have also speculated, on the basis of animal data, that there are a number of cortical areas that control attention, and that each area has the ability to produce covert attentional shifts. Since cuing attention to the neglected hemispace facilitates reaction times, this suggests that if either the frontal or parietal lobe is intact, there remains the ability to shift attention to the neglected hemispace when only a single sensory stimulus is present. However, if attention is drawn to somewhere outside of the neglected hemispace, neither the frontal nor the parietal area can adjust as quickly to a subsequent contralateral target.

Recently, Posner et al. (1987) have outlined a hierarchical model of attention in relation to subcortical and cortical networks. The authors hypothesize two distinct neural systems: a parietal system that is specific to visuospatial attention, and a more general system that monitors both language and visuospatial attention. They have speculated that the common anatomical system may involve the frontal area.

CONCLUSION

In the overall results of the present study and of Posner et al. (1984), a consistent pattern emerges regarding patients with right-hemisphere lesions. Once the attention has been drawn to areas outside the contralateral hemifield, there is a significant delay in responding to stimuli presented to the neglected side. Responses to targets in the contralateral hemifield are much faster if attention has been engaged there first. This may be taken as evidence of a failure in the disengage operation. Moreover, for patients tested shortly after lesion onset, there seems to be a relationship between the magnitude of the extinction-like reaction-time pattern and the amount of clinical neglect. Patients tested many years afterwards may show no signs of neglect yet retain an impairment of the disengage operation (Posner et al., 1984). Chronic disruption of the disengage operation is most severe when a lesion involves the right parietal lobe and the patient displays clinical neglect.

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