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EFFECTS OF PARIETAL INJURY ON COVERT ORIENTING OF ATTENTION[1]

MICHAEL I. POSNER,[2] JOHN A. WALKER, FRANCES J. FRIEDRICH, AND ROBERT D. RAFAL

University of Oregon and Cognitive Neuropsychology Laboratory, Good Samaritan Hospital, Portland, Oregon 97210

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Abstract

The cognitive act of shifting attention from one place in the visual field to another can be accomplished covertly without muscular changes. The act can be viewed in terms of three internal mental operations: disengagement of attention from its current focus, moving attention to the target, and engagement of the target. Our results show that damage to the parietal lobe produces a deficit in the disengage operation when the target is contralateral to the lesion. Effects may also be found on engagement with the target. The effects of brain injury on disengagement of attention seem to be unique to the parietal lobe and do not appear to occur with our frontal, midbrain, and temporal control series. These results confirm the close connection between parietal lobes and selective attention suggested by single cell recording. They indicate more specifically the role that parietal function has on attention and suggest one mechanism of the effects of parietal lesions reported in clinical neurology.

It has been shown in animal studies and in studies of humans with brain injury that the parietal cortex plays a significant role in the ability to select information for the highest levels of conscious integration. When monkeys are trained to attend to stimuli eccentric to their current line of vision, single cells in the parietal cortex whose receptive field is the focus of attention show an enhancement in the rate of firing to target events. This selective enhancement of single cells independent of eye movements does not occur at other anatomical sites studied (see Wurtz et al., 1980).

Similarly, results with patient populations suffering from posterior brain injury (Weinstein and Friedland, 1977; De Renzi, 1982) have shown that these patients tend to neglect information coming from the side of space opposite the lesion. This neglect includes what in clinical neurology is called extinction. When simultaneous conflicting stimuli to the left and right of fixation occur, the patient is not aware of the one that occurs contralateral to the lesion. On the other hand, when the patient is fixated but receives no conflicting stimulation, the patient may be able to detect events contralateral to the lesion with little problem.

Both the monkey and patient studies suggest that parietal cells are closely involved in attention in some way. However, they have not provided us a detailed account of exactly how these areas of the brain control visual attention. Indeed, Wurtz et al. (1980) have argued that, although the activity of these cells seems related to visual attention in some way, it is not possible to determine whether they are actually part of the neural systems for visual attention or simply correlated with it.

In the area of cognitive psychology, studies of attention emphasize the sequence of internal operations involved in the act of attending (Broadbent, 1982; Posner et al., 1982). While we usually use head and eye movements to orient toward a visual stimulus, it is quite possible to show that attention can be oriented toward a location in the visual field entirely covertly. While foveation improves the efficiency of processing visual events in terms of increased acuity, directing attention covertly to a visual location improves the speed and reduces the threshold for processing events that occur there. One method to study these changes in efficiency is to present a cue which informs the person about where a later target will occur. The cue may be an arrow or word introduced at fixation that informs the person where the target is most likely to occur (Posner, 1980), or it may be a sensory change that occurs near the place where the target will

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[2] To whom correspondence should be addressed, at Department of Psychology, University of Oregon, Eugene, OR 97403.

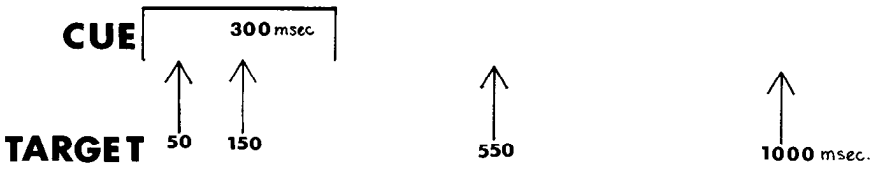

Figure 1. Timing of cues and targets for the main experiment.

be introduced which draws attention to that location (Posner and Cohen, 1984). Both cuing methods make it possible to compare the speed and accuracy of dealing with targets at both cued and uncued locations. These studies indicate facilitation of processing efficiency at the cued location in comparison to other locations of similar eccentricity. Careful monitoring of eye movements and other peripheral adjustments ensures that this facilitation occurs covertly and not by any overt change. The time course of facilitation can be studied by introducing the target at varying intervals following the cue. Speed of processing at the cued location improves more rapidly than at other locations during the first 50 to 150 msec after the cue.

It is also possible to distinguish more finely the mental operations involved in target detection. One can consider the act of orienting attention toward the target in terms of three more elementary mental operations: disengaging from the current focus of attention, moving attention to the location of the target, and engaging the target. Consider a person facing a blank visual display. If a cue is given to expect a target at some location and sufficient time is also given to orient attention there, then the only remaining operation required when the target appears is to engage it. If the person facing the blank screen is given no cue, when the target arrives attention must be moved to the target location, and then the target can be engaged. Finally, if a cue is first given and a target subsequently appears at a location other than the cued one, the person must first disengage attention from the cued location, move it to the target location, and engage the target. Each of these operations has been studied in separate cognitive experiments (LaBerge, 1974; Shulman et al., 1979). While such studies have documented the existence of time-dependent component stages in shifting attention, they have not explored them within a single paradigm and have not provided any real hint as to the underlying neural machinery involved.

In order to attack this question, we (Posner et al., 1982) have reported on studies of patients suffering from progressive supranuclear palsy (PSP), a disease that involves degeneration of the midbrain structures responsible for control of the saccadic eye movements. We found that subjects unable to move their eyes in certain directions (usually vertical) are able to move attention in these directions, although they are slow in so doing. The delay for both cued and uncued targets indicated a slowing of the attention movement operation. In the same paper we reported preliminary results from two parietal patients. We found that a peripheral cue that summoned attention to the side of the lesion (the nonneglected field) produced a massive interference in the time to report targets contralateral to the lesion. The advantage of the cued over the uncued side for contralateral targets appeared to be earlier and larger than for ipsilateral targets or for normals. This result was quite different than in the PSP patients, who showed a delay in taking advantage of the cue for the most damaged directions, and suggested that the parietal injury might have affected specifically the ability of the patient to disengage from an ipsilateral cue. In the current paper we report the results of studies of 13 parietal patients which are designed to confirm the effect of parietal injury on covert orienting and to locate the particular components of the task that are most impaired by the injury and, thus, the aspects of covert orienting that depend upon intact parietal functioning.

Materials and Methods

The basic experimental method is the same in all of the experiments. Basically, it is a reaction time task in which subjects are asked to detect a bright asterisk (target) at one of two positions in the visual field. Prior to the target, cues are introduced that direct the subject's attention either to the location of the target or to some other location. We measure the reaction time to the target as a function of the location of the cue and of the time between cue and target. In some experiments the target usually occurs on the side of the cue, whereas in other experiments the target occurs equally often on the cued side and on the noncued side.

In all experiments the subject faces a cathode ray tube in which visual cues and targets are presented. The task is to press a single key as quickly as possible with the index finger of the hand ipsilateral to the lesion whenever a bright target asterisk is presented. A video camera is used to watch the patients' eyes during the experiment. The patients are instructed to keep their eyes fixed on the center of the screen at which is present an outline box of about 0.5° size.

Trials. Each trial begins with a cue event which is the brightening of one of the two peripheral boxes located about 8°[3] to the left or right of the fixation box.[4] The brightening lasts 300 msec. At varying intervals of 0, 150, 550, or 1000 msec after onset of the cue, the target is presented in one of the two peripheral boxes. The

[3] The patient's head was not held during this experiment so the visual angles subtended by the display may vary by up to a degree.

[4] For patients J. C. and J. L. the standard eccentricity of the peripheral boxes was about 2° since the presence of a right hemianopsia for stimuli further out in the periphery prevented running them at the usual eccentricities.

target remained present until a response was made or for a maximum of 5 sec. The time lines for events in the basic experiment are shown in Figure 1.

In the basic experiments the target occurred on the side of the cue on four-fifths of the trials (valid) and on the side opposite the cue on one-fifth of the trials (invalid).

Two subjects (R. S. and R. E.) were run in a slightly different version of the experiment in which the onset of the peripheral box rather than merely brightening it was used as the cue. For these two patients the target was present in the field for 1000 msec and then went off. Five seconds were allowed for a motor response, and there were 1000 msec between trials.[5]

For six patients the standard paradigm was augmented by neutral trials which occurred on one-fifth of the total trials. In these trials the central box brightened as the cue. For neutral trials, targets were equally likely to occur in either of the two peripheral boxes.

Three patients were run using a central arrow cue rather than a peripheral cue. The central arrow was introduced at the start of each trial in the central box. The arrow was approximately 0.25° in size and pointed left or right. The target was presented on the side indicated by the arrow on four-fifths of the trials and on the side opposite the arrow on one-fifth of the trials.

Procedure. Each patient was run in one or more sessions of approximately 200 trials each. The total number of trials collected from each person and the number of sessions were dictated by their general condition, energy level, and considerations of other forms of therapy to which they were committed. All of the patients reported formally in this paper had at least 10 reaction times in each condition reported.

Patients. Thirteen patients with acquired lesions of the parietal lobes were recruited from the inpatient and outpatient neurology services of Good Samaritan Hospital and Medical Center. Referrals were solicited on the basis of the anatomical location of the lesion rather than the presence of clinical neglect; however, it is likely that our sample was biased toward patients with signs of neglect. In six patients the parietal lesion was in the right hemisphere, and in seven patients it was in the left hemisphere. One of the right parietal patients, R. E., had a smaller left parietal lesion from radiation necrosis; while one of the left hemisphere patients (L. M.) had a small, older infarction of the right parietal lobe from which she had recovered.

Table I describes the clinical features of the 13 parietal patients who constitute the main subject of this investigation. The table indicates whether clinical signs of neglect were present and rates the severity on a five-point scale. These were as follows: (*1*) Minimal—findings of neglect (e.g., extinction on double simultaneous visual stimulation) found inconsistently; neglect does not contribute to disability in everyday activities. (*2*) Mild-extinction and/or other signs were found consistently on examination. (*3*) Minor—problems with neglect occurred infrequently in everyday life. (*4*) Moderate—inattention to everything occurring on the neglected side; neglect disables patients in most activities of daily living. (*5*) Severe-dense neglect with tonic head and/or eye deviation. There were no patients included in the study who had neglect classified as severe. All patients gave their consent to participate in the experiments.

TABLE I
Clinical data on parietal patients

Patient	Age/Hand/Sex	Lesion				
		Type[a]	Side	General Location[b]	Vintage[c]	Clinical Neglect[d]
R. S.	65/R/M	T[e]	R	Deep parietal	2 mo	None
R. E.	62/L/M	T[f]	R[g]	Parieto-occipital	3 yr	Mild
W. K.	65/R/M	S	R	Fronto-parietal	7 yr	Mild
C. W.	83/R/F	S	R	Fronto-parietal	4 yr	Minimal
J. C.	61/L/M	S	R	Tempero-parietal	10 yr	Minimal[h]
J. L.	75/R/M	S	R	No scan	2 mo	Moderate[h]
L. M.	69/R/F	S	L[i]	Front-temp parietal	1 mo	Mild
E. A.	55/R/F	S	L	Tempero-parietal	7 yr	None
R. J.	65/R/F	T	L[j]	Fronto-parietal	2 wk	None
A. R.	65/R/F	S	L	Posterior parietal	2 wk	Mild
N. D.	43/R/M	T	L[k]	Deep fronto-parietal	1 mo	None
J. G.	63/R/M	S	L	Lateral parietal	1 mo	Mild
W. W.	74/R/M	S	L	Dorsal-occipital	2 wk	None

[a] S, stroke; T, tumor.
[b] Rough clinical description of location. For quantitative data see Table II.
[c] Time since onset of symptoms.
[d] A five-category scale was used to rate severity (see the text).
[e] Metastatic tumor.
[f] Resected glioma.
[g] A small secondary lesion from radiation necrosis was present in the left parietal region.
[h] Homonymous hemianopsia.
[i] A small older right parietal lesion was seen from a stroke which had occurred 4 years earlier and from which she had recovered prior to the curren stroke.
[j] Vertex meningioma.
[k] Glioblastoma.

Neurological controls. Neurological controls included three patients with lesions of the frontal lobes (two right hemisphere and one left), and four patients who had undergone temporal lobectomies for epileptic seizures.

[5] Actually, eight patients were run in this procedure. However, only three of them had a sufficient number of invalid trials for analysis. These are patients C. W., R. S., and R. E. Patient C. W. was also run in the later version of the experiment and provided comparable data. The early version with 1-sec exposure duration was marked by many more trials on which there was no response to the target when it was invalid and on the contralateral side. This is discussed in detail later in the paper.

Scans. Of the 13 patients constituting this study, CT scans were available for 12. These scans were used to confirm the presence of the reported lesion and to provide evidence on the anatomical location of the phenomena we report. Since there was no evidence of neglect in the control patients, no effort is made to present detailed anatomical data on them.

Quantification of scans. After examination of the scans, we decided to present the results mainly in quantitative form. In analyzing CT scans, each section was projected onto corresponding plates constructed from both the photographic atlas of DeArmond et al. (1976) and the diagrams of Gado, et al. (1979). These two sources, in combination, provide for sufficiently detailed correspondence between CT scans and gross brain structures, so that boundaries between areas such as frontal, parietal, occipital, and temporal lobes can be made with certainty. Each section of these plates represents a view about 8 mm from the adjacent views. Although the actual scans from a patient may not correspond precisely to these plates, each CT section can be matched to the most appropriate plate. For each section, the area of lesion was drawn onto the corresponding plate, with particular attention to the boundaries of the lesion. In two cases, W. K. and W. W., adjustments were necessary because the head was tilted from the usual angle of CT scan. After each plate was drawn, three scores were computed for the affected hemisphere: (*1*) the total two-dimensional area involved in the lesion, as a percentage of the total area at that level of section; (*2*) the total amount of parietal lobe tissue involved in the lesion, as a percentage of total parietal lobe area at that level; and (*3*) the distance, in millimeters, from the posterior edge of cortex to the posterior edge of the lesion. To compute the total lesion size, the values for each section were added together and calculated as a percentage of total area covered by the plates. From these values we derived a score for total lesion size, total amount of parietal lesion size, subdivided into superior and inferior parietal areas, and posteriority, derived from the most posterior part of the lesion (see Table II). If a plate could not be reconstructed due to the absence of an appropriate CT scan section, the values for that plate were estimated by averaging the two adjacent sections. These calculations, based on two-dimensional reconstructions, have the drawback of failing to represent adequately the three-dimensional characteristics of the lesion, but they offer the best estimate of the lesion as judged from the available CT scan data.

Results

Peripheral cues. The basic results of the major experiment are summarized in Table III. The median reaction time in each condition was calculated for each subject. To obtain these data, all reaction times less than 100 msec and more than 2500 msec were eliminated from consideration. Except for the two subjects who were run with target durations of 1000 msec, this resulted in the elimination of only a few trials. For the patients run with the 1000 msec exposure, misses (e.g., reaction times of 5000 msec) were quite frequent at the 50- and 150-msec durations. It was this fact that produced the change in method of leaving the target present until a response. Thus, quite a number of trials were eliminated from patients R. E. and R. S., who were run under the limited exposure conditions, but their remaining data were similar to the other 11 subjects of Table III.

The data of Table III are shown graphically for the six patients with right-sided lesions in Figure 2 and for the seven patients with left-sided lesions in Figure 3. The data of Table III were subjected to a repeated measures analysis of variance with lesion site (left versus right), validity (cued side versus uncued side), target side (contra- versus ipsi-), and interval as the independent variables. Lesion site was a between-subjects factor, whereas the other factors were manipulated within subjects. All of the main effects of the variables proved to be significant beyond the 0.01 level. The only significant interaction was between target side and validity conditions ($F(1,11) = 7.4, p < 0.02$). The interaction of target side with interval also approached but did not reach significance ($F(3,33) = 2.59, p < 0.07$).

The statistical analysis confirms the most obvious findings available from inspection of figures 2 and 3. The left-sided lesion group produced faster reaction times than did the right-sided group. Much of this effect appears to be due to the very long times that occur when the target is contralateral to the lesion while the cue is ipsilateral (invalid). The effect of ipsilateral cues in slowing responses to contralateral targets is similar to what is called extinction in the neurological literature (Weinstein and Friedland, 1977). It is the cause of the significant interaction between target side and validity, and, while it occurs for both right and left patients, it is considerably larger on the average for right-sided patients. Moreover, this extinction-like reaction time pattern is strongest at the shortest intervals between cue and target. However, the three-way interaction between target side, validity, and interval does not reach statistical significance ($F(3,33) = 1.98, p < 0.13$).

The main effect of target side is also important. For valid trials, there is a modest advantage of targets on the side ipsilateral to the lesion. This advantage is present in 11 of the 13 subjects whose data are shown in Table

TABLE II

Quantitative estimates of lesions from CT scans

Patient	Total Lesion Size	Percentage of Parietal Area			Posterior Score
		Total	Superior	Inferior	
	%				*mm*
W. W.	1	1	0	2	0
A. R.	6	21	25	17	1
R. J. Tumor	8	3	30	15	5
J. G.	8	20	0	40	6
L. M.	10	33	50	25	5
E. A.	14	15	5	27	0
N. D. Tumor	18	33	30	35	4
R. S. Tumor	8	16	15	17	2
J. C.	16	34	30	37	1
B. E. Tumor	19	21	20	22	0
W. K.	27	71	65	77	2
C. W. (J. L.)	33	49	40	57	4

TABLE III

Reaction times (milliseconds) as a function of side of target and validity interval for left- and right-sided lesions

Patient	Ipsilateral								Contralateral							
	Valid				Invalid				Valid				Invalid			
	50	150	500	1000	50	100	500	1000	50	150	500	1000	50	100	500	1000
L. M.	497	425	372	427	537	569	510	350	603	624	504	392	1034	883	821	725
E. A.	557	576	531	535	600	633	692	511	676	625	515	474	745	739	705	523
R. J.	375	407	425	320	490	460	425	405	380	395	405	375	510	470	440	420
A. R.	412	375	341	309	445	357	538	396	473	460	410	261	741	1028	840	487
N. O.	426	391	383	392	469	409	388	517	496	408	404	439	588	515	428	575
J. G.	441	349	351	371	550	436	380	362	500	395	358	379	589	546	447	428
W. W.	515	538	408	424	613	579	352	311	591	511	417	355	694	606	562	302
R. S.	500	490	410	350	540	500	530	470	615	600	425	430	940	730	940	1030
B. E.	770	650	650	750	780	910	750	810	800	730	800	870	1090	950	1050	1000
W. K.	571	446	432	359	543	540	716	943	633	503	488	450	855	699	821	682
C. W.	543	544	462	425	534	548	601	616	593	535	464	495	982	593	817	558
J. C.	637	546	605	502	706	579	616	561	650	555	587	660	1032	946	942	434
J. L.	406	389	379	379	399	384	405	373	420	480	459	477	1536	1632	1558	1729

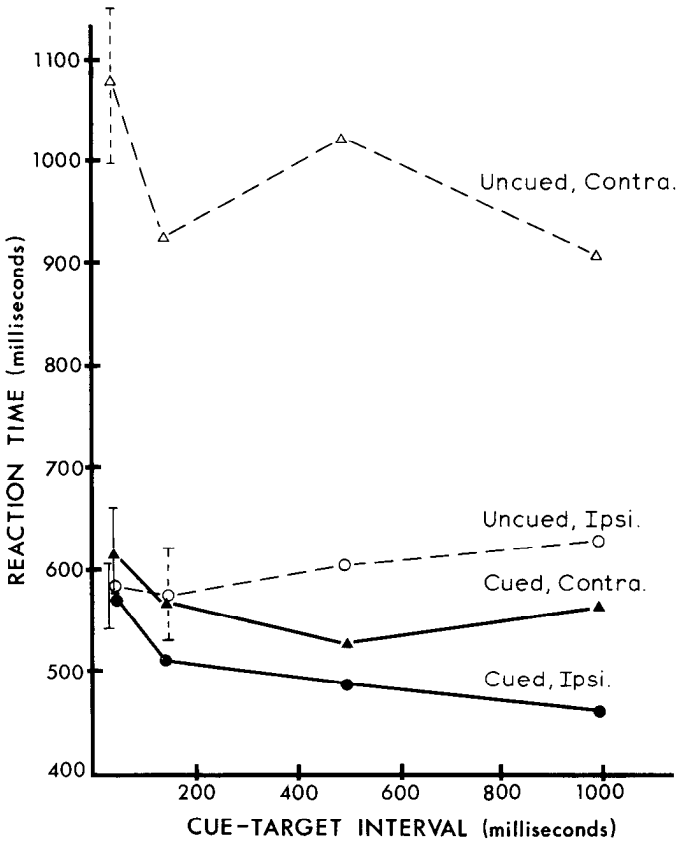

Figure 2. Reaction time for six right parietal patients in the main experiment. *Solid lines* are for targets on the cued side, and *dashed lines* are for targets on the uncued side. *Triangles* are contralateral targets, and *circles* are ipsilateral targets. *Bars* indicate ±1 SE for representative points.

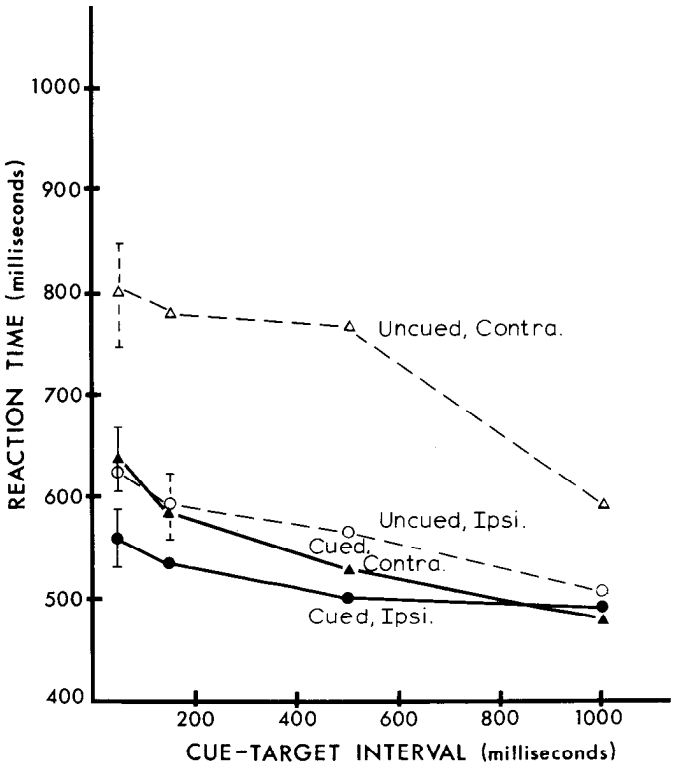

Figure 3. Reaction times for seven left parietal patients in the main experiment. *Solid lines* are targets on the cued side, and *dashed lines* are for targets on the uncued side. *Triangles* are contralateral targets, and *circles* are ipsilateral targets. *Bars* indicate ±1 SE for representative points.

III. Two subjects show no difference between contralateral and ipsilateral targets when they have been cued as to correct position. Even for those subjects who show an advantage to the ipsilateral side on valid trials, this tends to be quite small in comparison to effects found on invalid trials. Another important effect is the fact that the improvement in reaction time with interval for valid trials is equivalent for targets on the two sides. Since the improvement in reaction time with interval for valid trials is partly due to attracting attention toward the cue, this implies that the cue is equally effective in attracting attention to either the ipsilateral or contralateral field.

There is a significant advantage for valid trials overall. For right hemisphere patients, the cued side advantage

TABLE IV

Reaction times (milliseconds) for central and peripheral cues

Patient	Neutral								Invalid							
	Ipsilateral				Contralateral				Ipsilateral				Contralateral			
	50	150	500	1000	50	150	500	1000	50	150	500	1000	50	150	500	1000
Central																
C. W.	571	521	421	527	625	564	375	355	560	585	406	389	720	521	439	605
R. S.	534	504	471	426	908	887	608	658	512	529	464	451	760	797	1500	1200
L. M.	558	527	489	453	642	610	453	544	589	522	599	522	950	898	818	512
Peripheral																
C. W.	543	544	462	425	593	535	464	495	534	548	601	616	982	593	817	558
R. S.	500	490	410	350	615	600	425	430	540	500	530	470	940	730	940	1030
L. M.	497	425	372	427	603	624	504	392	537	569	510	350	1034	883	821	725

for ipsilateral targets seems to occur only after 150 msec, while for left patients it is present at the earliest interval. Normals usually show a validity effect by 50 msec that increases over the first 150 msec following the cue (Posner and Cohen, 1984). This effect for ipsilateral targets contrasts markedly with the huge validity effects for contralateral targets found particularly for the right-sided lesions which we are calling the extinction-like reaction time pattern.

Arrow cues. According to our view, the difference between valid and invalid trials is due to covert orienting of attention in the direction of the cue. Three alternative interpretations are also possible. The first is that the peripheral cue may have its influence by a purely sensory mechanism. The second is that some overt shift such as eye movements may mediate the effect. The third is that facilitation might be due solely to the fact that the target is more likely to be on the cued than the uncued side. All of these issues have been investigated in normal subjects. The first is countered by showing that a central arrow pointing to left or right, like a peripheral cue, has a facilitatory effect (Posner, 1980), the second by careful monitoring of eye position (Posner et al., 1977), and the third by showing that peripheral cues are effective even when they provide no information about the likelihood that the target will occur at the cued position (Posner and Cohen, 1984). In this and the next section ("Neutral Cues"), we seek to show that the effects in patients are also due to covert attention shifts.

Three patients were run in blocks with central arrow cues which were valid (i.e., target on cued side) 80% of the time. The method was described under "Materials and Methods." Table IV summarizes the median reaction times in each condition for the blocks with central cues for these three patients and the results for peripheral cues with the same three patients. The data of Table IV are shown graphically in Figure 4.

An analysis of variance over the data of Table IV indicates significant main effects of validity condition ($F(1,2) = 23.9$, $p < 0.04$) with valid trials faster than invalid and of validity condition × target side ($F(1,2) = 17.7$, $p < 0.05$) indicating that invalid trials are especially long when they are contralateral to the lesion (the extinction-like reaction time pattern). Finally, there is also a significant interaction of cue (central versus peripheral) and validity ($F(1,2) = 23.7$, $p < 0.04$). This interaction is apparently due to the long reaction times found on valid trials with a central arrow cue when the target is on the side contralateral to the lesion (*solid squares* in Fig. 4). The lengthening of reaction times is particularly marked at the short cue to target interval and appears to indicate that subjects have trouble shifting attention from the central arrow when it points in the contralateral direction. Even though the arrow is telling subjects where to attend, when this is to the side opposite the lesion they are so slow in executing the instruction that at 50 msec their reaction times are almost as long as when the arrow is in the wrong direction (Fig. 4, *dotted line, open squares*).

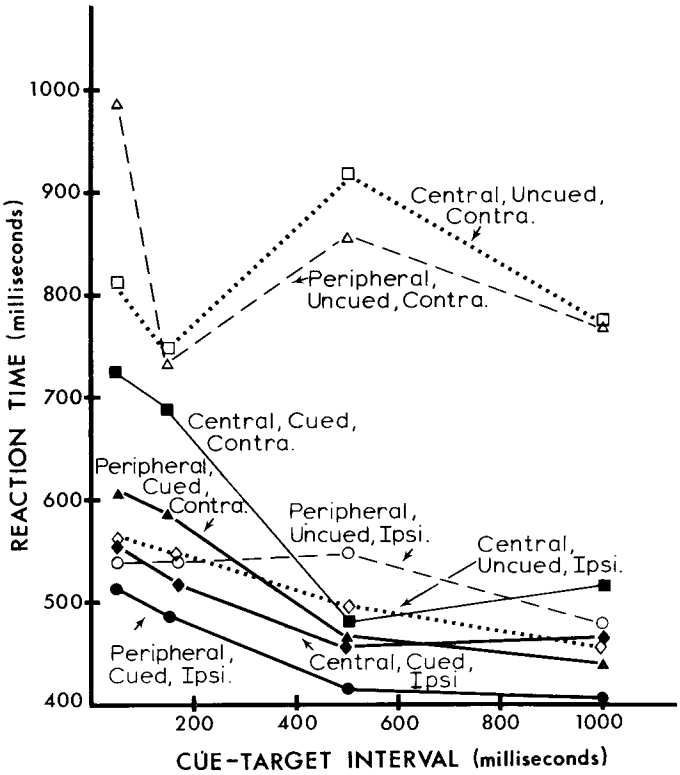

Figure 4. Reaction times for three patients' central cues (*diamonds* and *squares*) and peripheral cues (*circles* and *triangles*). *Solid lines* are cued targets, and *dashed lines* are uncued targets. *Circles* and *diamonds* involve targets ipsilateral to the lesion, and *triangles* and *squares* involve contralateral targets.

These results show that the basic facilitation pattern and the extinction-like reaction time pattern found with the peripheral cue also extend to a central arrow cue. Thus, the facilitation effect cannot be entirely sensory in origin. Moreover, it indicates that parietal damage affects shifts of attention even when the cue is not at the target location. This last finding is interesting since eye movements made as part of a search plan or to nonvisual events are sometimes thought to be controlled by more anterior systems (frontal eyefields), while reflexive eye movements to visual input are controlled from more posterior or midbrain systems. Irrespective of the case for eye movements, it appears clear that parietal lesions affect both of these types of covert attention shifts.

Neutral cues. Both central arrow and peripheral cues provide the patient with information about where the target is most likely to occur. This information may be used either to shift the eyes (overt attention) or might affect reaction time performance directly by some motor adjustment that does not involve an attention system. In normal subjects we carefully monitored eye movements to ensure that only trials with no eye movements were included (Posner et al., 1977). This is easy to do in normals because with luminance detection tasks subjects spontaneously supress eye movements after a few trials even if they are allowed to make them, since eye movements actually slow down overall reaction time (Posner et al., 1977). However, it was much harder to achieve this level of control in patients. There is much spontaneous eye movement in these patients, and often they do not appear to know whether their eyes are stationary or exactly where they are fixated.

We did monitor eye position over the television screen, and it was clear that at least several of the patients did not move their eyes when cues were presented. These patients did not appear to give grossly different data than those who were noted as making many eye movements. However, it was difficult to determine with our methods how the eye movements that did occur were related to the cues. Therefore, we employed a condition in which the cue did not provide any information about where the target is likely to occur. In this case, patients could not use the cue to direct eye movements, nor could they make any other differential preparation based on knowledge of where a target was likely to be.

Six patients were run; one-fifth of the trials were neutral, while the remaining trials were the same as in the main experiment ("Materials and Methods"). In neutral trials, the central fixation box was brightened, and targets could occur with equal frequency on either side.

The data for these six patients are presented in Table IV, which compares the neutral trials with invalid trials (from Table III) for the same patients. These data are shown graphically in Figure 5. The figure shows quite clearly that the neutral condition produces the same extinction-like reaction time pattern that was previously found for invalid trials. An analysis of variance of the data of Table IV showed significant differences of interval ($F(3, 15) = 8.5$, $p < 0.01$) and of target location by interval ($F(3, 15) = 5.9$, $p < 0.01$). There was clearly no effect of condition (invalid versus neutral) where F was less than 1.

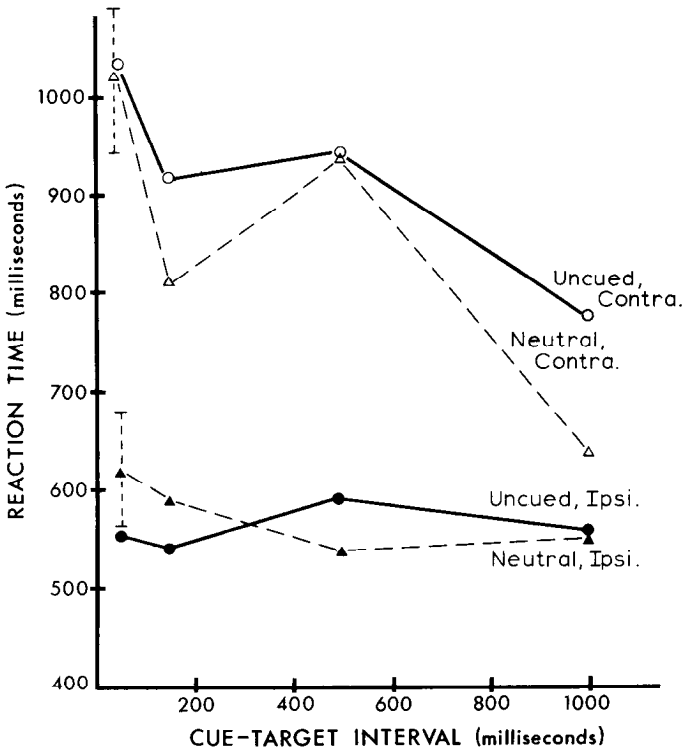

Figure 5. Reaction times for six patients for targets following neutral cues (*triangles*) and for the uncued side with a peripheral cue (*circles*). *Solid symbols* are for ipsilateral targets, and *open symbols* are for contralateral targets. *Bars* indicate ±1 SE for representative points.

These data are quite clear in showing the full extinction-like reaction time pattern in the neutral trials that was found previously for the invalid trials. The neutral cue appears to serve two functions. First, it alerts the subject as to the impending target (warning signal function). However, both valid and invalid peripheral and arrow cues also provide a warning signal, but only invalid cues produce the extinction-like reaction time pattern. Second, the neutral cue serves to engage the patient's attention at fixation. It appears to be this function that is crucial to producing the extinction effect. This result is important. It confirms the arrow study by showing that the extinction-like reaction time effect does not depend on peripheral energy in the ipsilateral field that occurs at the same time or shortly before the contralateral target. It shows that the effects do not depend on the fact that the cued side is likely to get a target since in the neutral condition the cue provides no information about the location of the target. For the same reason this condition eliminates the possibility that the cue has its effect by directing the patient's eye movements toward the wrong field, since the cue is at fixation in this condition.

Anatomical correlates. The parietal patients show a large increase in reaction time to invalid trials contralateral to the lesion which is particularly strong at the short intervals between cue and target. This is the extinction-like reaction time pattern. Table V shows for each patient the validity effect (invalid reaction time − valid reaction time) at 50 msec for ipsilateral and contralateral targets.

TABLE V

Reaction times (milliseconds) for neutral and invalid cues

Patient	Neutral								Invalid							
	Ipsilateral				Contralateral				Ipsilateral				Contralateral			
	50	150	500	1000	50	150	500	1000	50	150	500	1000	50	150	500	1000
W. K.	816	502	551	462	946	794	766	578	543	540	716	943	855	699	821	682
J. C.	676	691	579	716	1039	993	898	547	706	579	616	561	1032	946	942	434
C. W.	635	708	484	543	1041	749	1044	535	534	548	601	616	982	593	817	558
J. L.	417	419	407	408	1371	892	1685	1718	399	384	405	373	1536	1642	1558	1729
E. A.	580	646	636	662	834	689	631	629	600	633	692	511	745	739	705	523
L. M.	586	558	550	548	912	725	618	416	537	569	510	350	1034	883	821	725

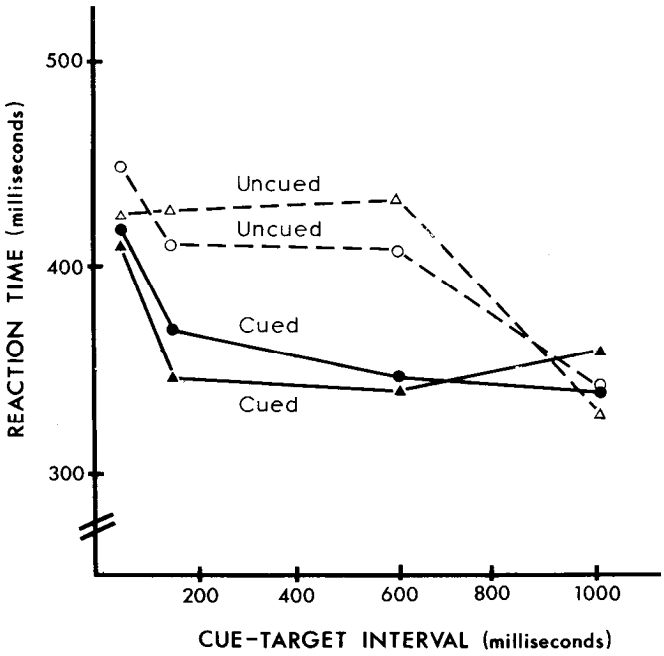

Figure 6. Reaction times for temporal lobectomy control patients in the peripheral cue experiment. *Solid lines* are cued targets, and *dashed lines* are uncued targets. *Circles* represent ipsilateral targets, and *triangles* represent contralateral targets.

TABLE VI

Size of validity effect at 50-msec interval for left and right parietal patients, temporal and frontal controls

Patient	Contralateral	Ipsilateral	Contralateral-Ipsilateral
Right parietal (n = 6)			
R. S.	325	40	285
B. E.	290	10	280
W. K.	222	−18	240
C. W.	391	−9	398
J. C.	382	69	313
J. L.	1116	−7	1123
		Mean	441
Left parietal (n = 7)			
L. M.	431	40	391
E. A.	69	43	26
R. J.	130	115	15
A. R.	298	33	265
N. D.	92	43	49
J. G.	89	109	−20
W. W.	103	98	5
		Mean	106
Temporal frontal (n = 7)			
C. B.	30	40	−10
P. M.	92	211	−119
J. S.	23	43	−20
J. B.	94	34	60
H. M.	0	40	−40
S. Z.	110	110	0
H. Z.	100	100	0
		Mean	−19

In addition to the 13 parietal patients, these data are compared to seven control patients (three frontal and four temporal). These data show that the advantage in the validity effect for contralateral over ipsilateral targets is uniformly large for all right parietal patients, is positive for six of seven left parietal patients, and is positive for only one of seven control subjects.

The data of the four temporal patients are displayed graphically in Figure 6. This figure indicates that there are no important differences between ipsilateral and contralateral targets for either valid or invalid trials. However, they do confirm the usual effects of validity found in the parietal patients and normals for ipsilateral targets.

These data leave little doubt that the extinction-like reaction time pattern is characteristic of parietal patients and is not a strong property of all unilateral cortical lesions.

Right versus left. A second anatomical question concerns the differences between the effects of right- and left-sided lesions. The analysis of variance of Table II supports the data shown graphically in Figures 2 and 3 indicating that patients with left lesions are faster, particularly on invalid trials contralateral to the lesion. This is strongly supported by the mean difference in the size of the validity effect at 50 msec (441 msec for right patients and 106 for lefts, see Table VI). In general these data support the clinical observation (De Renzi, 1982) that right-sided lesions are more likely to produce neglect. However, with such a small number of patients and with problems of selection, it is difficult to be sure that the subjects are comparable. For example, Table II suggests that the right-sided lesions are somewhat larger, particularly in the parietal area. Moreover, the two left-sided patients showing the largest difference between ipsilateral and contralateral validity scores (see Table VI) are both rather special. Patient L. M. had a new left-sided stroke which followed an earlier right-sided lesion

and, thus, had bilateral involvement. Patient A. R. was tested rather soon after the stroke. While the patients with right-sided lesions seem to show uniformly large effects even when tested years after the lesion, we do not have a single patient with left-sided lesions who was tested a long time after a purely left lesion who had a powerful extinction-like reaction time pattern. The most convincing patient in this category was E. A. Fortunately, a right-sided patient, J. C., has a somewhat similar lesion to E. A. Thus, a comparison of the data of these two cases might be instructive (see Fig. 7). It seems clear from the data that both patients show the same overall pattern in which contralateral invalid trials are long; however, the effects in the right-sided patient are much stronger.

Neural system. The data we have presented argue that right parietal lesions produce the strongest extinction-like reaction time patterns. These patients appear to show the same pattern whether the injury was induced by tumor or stroke, whether it is a new or old injury, and whether or not they were diagnosed as showing clear neglect. However, it is also clear that the lesions of our patients are not discrete lesions but large ones often extending far in the posterior (occipital lobe) and anterior (frontal lobe) directions. We divided our measures of the 12 scans into five categories (see Table II). These were converted to rank orders. In addition, the same 12 patients were given ranks on the degree of their extinction-like reaction time pattern by ranking the differences between contralateral and ipsilateral validity effects at 50 msec. The five rank orders from the CT data and the behavioral ranks were then intercorrelated by rank order correlation coefficient, and the resulting correlation matrix is shown in Table VII.

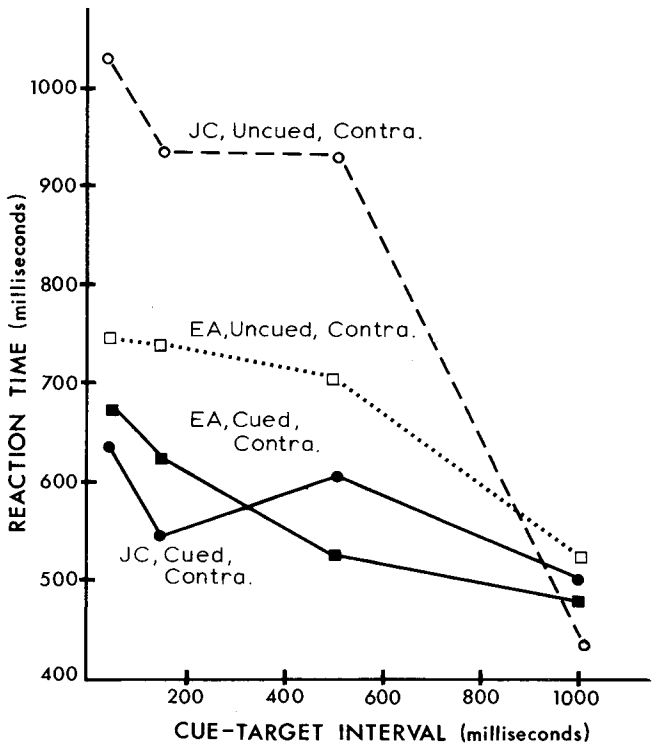

Figure 7. Reaction time for a left parietal patient (E. A., *squares*) and a right parietal patient (J. C., *circles*) with similar size and extent of lesion. *Solid lines* are for cued targets, and *dashed lines* are for uncued targets.

TABLE VII

Rank order correlation matrix between size of validity effects and measures of lesion characteristics from CT scans

	Size	Posterior	Parietal	Inferior Parietal	Superior Parietal
Behavioral score	0.450	−0.003	0.526	0.210	0.603
Size		0.021	0.717	0.747	0.607
Posterior			−0.382	0.294	−0.354
Parietal				0.655	0.923
Inferior parietal					0.441

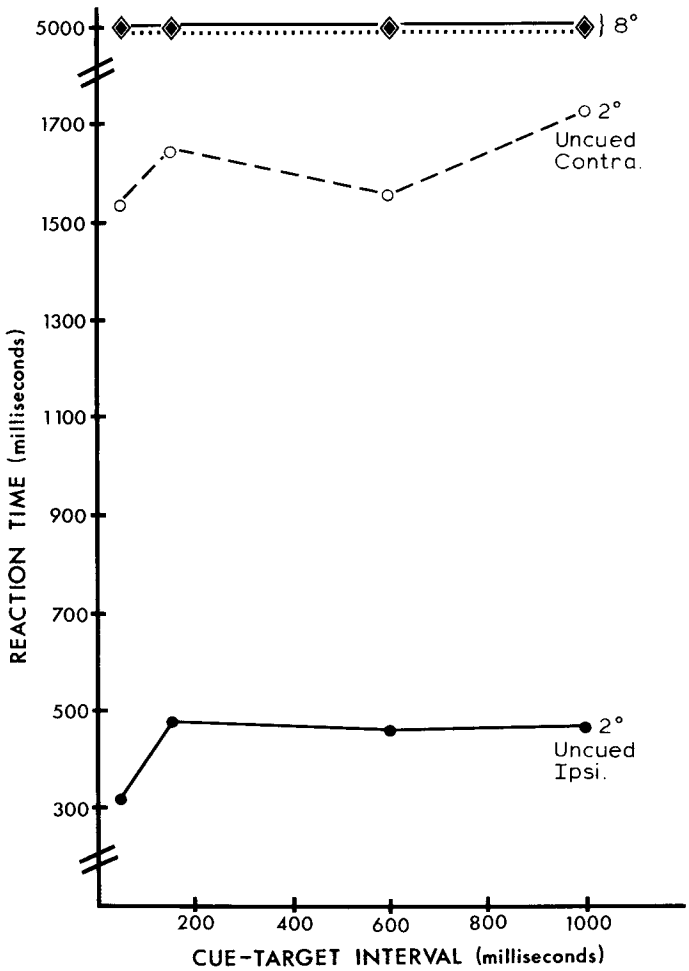

Figure 8. Reaction times for patient J. L., who had a field cut as well as neglect. Targets at the standard 8° distance are simply not seen at all on cued or uncued trials (*diamonds*). At 2° the extinction-like reaction time pattern is found. The *solid line* and *solid circles* are for uncued ipsilateral target, and the *dashed line* and *open circles* are for uncued contralateral targets.

The behavioral measure correlated best with overall lesion size, percentage of parietal lobe, and percentage of superior parietal lobe, although only the percentage of the superior parietal lobe reached statistical significance $p < 0.05$. Since we have only 12 cases, these results are only suggestive, but our data indicate that the extinction-like reaction time pattern is most highly related to the

extent of right superior parietal lobe damage. Left-sided lesions may also appear to produce the effect, although it may be smaller and shorter lasting.

Hemianopsia. Two of the patients in these experiments had clear evidence of field cuts (J. C. and J. L.). Both of these patients had lesions of the occipital lobe causing blindness in parts of the left visual field. In these cases we had to determine if there was a portion of the field at which sufficient vision could be obtained so that we could test the patient. Both patients proved to have sparing of vision within 2 to 3° of fixation. They could be tested by moving the cues and targets sufficiently close to the fovea on both sides that they responded well on valid trials. A comparison of trials for patient J. L. (see Fig. 8) illustrates the contrast in the pattern found within the hemianopic area (8° from fixation) and within the neglected area. The two conditions are quite discriminable in this type of testing. The results for J. C. were even more dramatic. Within the central 2°, he showed equivalent reaction times on valid trials (see Table III), but he was unable to respond to either valid or invalid trials for targets beyond 5°.

In testing normal subjects, it has been shown that the facilitation effects from cueing can be obtained within the fovea as well as at eccentric peripheral positions. Apparently, posterior lesions may produce a combination of blindness in some portions of the visual field with a pattern of poor orienting of attention within parts of the field where vision is spared. The type of testing used here may improve our ability to understand the relationship between purely sensory effects on vision and those relating to central attentional systems.

Conclusions

Elementary mental operations. In the introduction, we suggested that in the act of covert orienting three mental operations can be identified. These are disengaging from the current focus of attention, moving to the target, and engaging the target.

The first of these, disengaging from the current attentional focus, is a function of the depth of involvement in the current task (LaBerge, 1974). It is not possible to observe the disengagement operation directly. However, we can compare the efficiency with which targets on either side summon attention in conditions in which the patient's attention has first been actively engaged elsewhere and in conditions where it has not. At the time a valid cue is presented, the patient is alert and waiting, but attention has not been engaged by a cue. We can ask how efficiently the cue summons attention to the ipsilateral versus the contralateral field. There is no significant interaction between the side of the cue and interval between cue and target. This means that cues on either side summon attention equally well. It might be argued that the relatively few short cue to target intervals (e.g., 50 and 150 msec) and the lack of a no-cue condition prevented us from finding such an interaction. However, in a subsequent control study we measured reaction time to targets when no cue was presented. We found only slight differences between the two fields in this condition. As far as we can determine, the amount of improvement in reaction time from a cue over the first 150 msec following the cue is the same for the ipsilateral and contralateral fields.

These symmetric benefits of valid cues can be contrasted with the marked differences in reaction time to ipsilateral and contralateral targets following an invalid peripheral cue, a central arrow cue, or a neutral cue. All three of these conditions produce a markedly greater reaction time on the contralateral side, particularly at short intervals. What is the difference between a valid peripheral cue, on the one hand, and the invalid and neutral conditions, on the other? The main difference appears to be that target detection in the invalid and neutral trials first requires that attention be disengaged from a location other than the target.

Perhaps some alternatives are possible. For example, one might believe that the patient shifts attention toward the ipsilateral field in all conditions other than when a cue comes from the contralateral field. This explanation does not predict the results in the arrow cue experiment. The central arrow pointing toward either field does produce a shift of attention toward that field. However, any central arrow valid or invalid appears to delay the attention shift toward the contralateral field as though it caused a brief engagement of attention at the center, thus increasing the difficulty of a contralateral shift. It is clear that the patients can use central and peripheral cues to shift attention to the correct location. Thus, we favor the disruption of the disengage operation rather than an effect on the movement operation (see below). However, it should be noted that the effect on disengagement is spatial, not general. That is, it occurs selectively for targets in the contralateral visual field.

The movement of attention from a current focus to a new target position is a time-locked analogue mental operation. In studies of normal subjects using central cues, it was possible to show that low probability events introduced between fixation and the target location were facilitated in reaction time at intervals between presenting the cue and maximal facilitation at the target location (Shulman et al., 1979). Tsal (1983) has provided evidence that distance between cue and target can be used to predict the time required to obtain maximal facilitation as though attention movements occurred at a rate of about 8 msec/degree. Thus, there appears to be a separable mental operation related to the movement of attention from its current focus to the target, although we do not know how this is accomplished physiologically. If parietal lesions affected this component, we would expect attention to move slowly to contralateral cues irrespective of the degree to which attention was engaged at its current focus. Since no such effects are found on valid trials, it appears that the parietal lesion is not slowing the attention movement. Moreover, there is no correlation between the efficiency of moving to a contralateral peripheral cue and the size of the extinction-like reaction time pattern. This gives more evidence that the parietal lesion affects are not on the movement operation.

These findings contrast markedly with those we previously reported for lesions involving the colliculus and surrounding midbrain areas (Posner et al., 1982). The midbrain lesions slow orienting on both valid and invalid trials when they are in the direction in which the saccadic

eye movement system has been most impaired. The finding of similar slowing on valid and invalid trials is quite different than what we find for parietal lesions and implicates the movement component as a major effect of the midbrain lesion. Allocation of the disengage operation to cortical control and the move operation to midbrain control would fit rather well with views of how cortical and midbrain systems cooperate to control eye movements and other bodily movements (Wurtz et al., 1980). Our results suggest that purely cognitive functions may involve similar allocation of control to levels of the nervous system.

The final component of attention is engagement of the target after the attention movement is completed. This component can be measured by differences in the reaction time to contralateral and ipsilateral targets at sufficiently long intervals between the valid cue and target for attention to have moved to the target. On the average, reaction times to contralateral targets are longer, indicating overall interference with this component. However, two right parietal patients who show strong extinction-like reaction time patterns (C. W. and J. C.) show no difference in reaction time to left targets following valid cues. Thus, parietal damage sufficient to cause the extinction-like reaction time pattern need not affect the engagement component.

This finding is important because it relates to whether the parietal lobe should be regarded as the neural system most responsible for the efficiency of processing that we associate with attention. Since some parietal patients with large deficits in performance show complete ability to compensate for their deficit when attention is drawn to the contralateral side, we conclude that the improvement in performance associated with attention does not itself require an intact parietal lobe.

Of course, it is reasonable to expect that improvement in performance due to attending involves a complex neural network with many distributed locations (Mesulam, 1981). Many views would predict an important role for frontal sites in this function. Our study indicates that the parietal lobe is particularly important for the operation of disengaging attention and of less importance once attention is correctly oriented toward a contralateral target.

Relation to cognitive studies. In our studies we used the efficiency of responding to a stimulus as a measure of the patient's attention being directed to the stimulus. How well does this measure capture our intuitive notion of attention? When people move their eyes to a stimulus, they improve the efficiency with which they process it by bringing the high acuity portion of the visual system to bear upon the stimulus. Similarly, a covert attention shift provides an efficient routing of the stimulus to centers responsible for awareness (as measured by the ability to make an arbitrary response, such as a key press). The results of our preliminary experiments seem to us to provide strong evidence for this view. When we left the target present for only 1 sec, we found many invalid trials where patients failed to report contralateral targets. They were simply unaware of the presence of the target. It seems to us that this is exactly what one wants to mean by a failure of attention. We found that these misses were clearly related to the long delays that occur even when the interval between cue and target was increased. By leaving the stimulus present until a response, by using patients in better health, and by training, we found that most targets were reported, but the delays remained present. It seems to us that the delayed reaction times we find in the case of contralateral targets when attention is engaged elsewhere are a good measure of the effectiveness of the target in reaching the systems leading to awareness.

Support for this view also comes from studies in normals that have used either reaction time measures of attentional orienting (Posner, 1980; Jonides, 1981) or threshold measures (Bashinksi and Bachrach, 1980; Remington, 1980). Both kinds of studies have shown that under threshold conditions unattended stimuli are simply not seen or reported, while in above-threshold viewing conditions reaction times are delayed. These findings argue that the delay in reaction time in these experiments indicates a delay in reaching systems that produce awareness of the stimulus by the usual criteria of reports based on arbitrary responses that we use for studies of humans.

Relation to neuroscience studies. How do our results fit with the findings of a parietal locus for attention-related cellular responses in alert monkeys (Mountcastle, 1978; Wurtz et al., 1980)? In general, our anatomical measurements, as crude as they are, fit very well with a crucial role of the parietal lobe in orienting of attention. The main thrust of our results is to qualify the exact role they play. However, we have two related anatomical findings that need to be considered in relation to the monkey work. First, it appears that the attention-orienting system in the human may be asymmetric, in that right-sided lesions have greater effects. While this result is consonant with much clinical observation, such hemispheric differences do not appear to be characteristic of the monkey. Moreover, we had thought it likely that the clinical observations arose because the right hemisphere was responsible for the spatial representation of the outside world (Bisiach et al., 1979) rather than because the attention-orienting system was itself asymmetric. Thus, if our results had proven symmetric, we would have been inclinded to the view that the act of orienting was controlled equivalently by the two hemispheres but that the spatial organization of the outside world was primarily a right-sided function. However, at this point the data appear to support an asymmetry in the control of attentional orienting that favors the right side.

Our second result concerns the brain area most related to the extinction-like reaction time pattern we have observed. We find that the best anatomical correlate of this pattern is the extent of involvement of the superior parietal lobe (see Table VII). While our results must be taken with caution because of the size and variability of the lesions involved, they may, at first, seem to conflict with the data from studies using single cell recording in the alert monkey which we cited earlier. However, the organizations of the parietal lobe in human and monkey are quite different. In his book on human cortex, Baraak (1980) labels as a mistake the idea that the inferior parietal lobule in humans corresponds to area 7 in the monkey. Rather, in the human areas 39 and 40 have

greatly enlarged (Ariens et al., 1967) and occupy much of the region that corresponds to the inferior parietal area in the monkey. The human has two distinct portions of the parietal lobe that have different gross structures. The superior parietal lobe according to Economo's classification (Critcheley, 1953, p. 39) has a structure distinct from that found in the inferior parietal and temporal lobe. According to this view, Brodmann's area 7 can be identified with the superior parietal lobe in humans (see also Critchley, 1953, Table 7, p. 41). In studies of the visual system, it has been common to stress the similarity in neuroanatomy between human and infrahuman species. It may be important that as we move toward complex cognitive function, differences in the anatomical location and, perhaps, function of cortical structures will be more commonly found.

We believe that the simplicity of our behavioral assay and the relative quantitative precision by which we can time lock effects to cues and separate patterns of neglect and hemianopsia suggest important clinical implications of this research. It should be possible to assay the course of spontaneous recovery or direct therapeutic interventions with greater precision. Beyond the methodology, it seems to us that the ability to relate anatomical areas to elementary operations has both fundamental and applied importance. It should be possible to determine whether the neural tissue involved in disengaging attention in these spatial tasks is also used for other nonspatial operations. Such findings would advance our understanding of the brain's control of cognition. The ability of clinicians to associate an anatomical insult detectable in the CT scan with an elementary operation may permit a more rational clinical analysis of the resulting symptom complex.

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