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Ipsilesional Biases in Saccades but not Perception after Lesions of the Human Inferior Parietal Lobule

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Abstract

■ We examined the effects of chronic unilateral lesions to either the inferior parietal lobe, or to the dorsolateral prefrontal cortex including the frontal eye fields (FEFs), upon human visual perception and saccades in temporal-order-judgment (TOJ) tasks. Two visual events were presented on each trial, one in each hemifield at various stimulus onset asynchronies (SOAs). In the saccade task, patients moved their eyes to whichever stimulus attracted gaze first. In the perceptual-manual task, they pressed a button to indicate which stimulus was perceived first. Frontal patients showed appropriate TOJs for visual targets in both tasks. Parietal patients showed appropriate TOJs in the perceptual-manual but not the saccade task; their saccades tended to be ipsilesional unless the contralesional target led substantially. This reveals a bias in saccade choice after parietal damage that cannot be attributed to deficient visual perception. These results challenge previous claims that only anterior lesions produce motoric spatial biases in humans. However, they are in accord with recent neurophysiological evidence for parietal involvement in saccade generation, and also with suggestions that visuomotor transformations in the parietal lobe serving direct spatial motor responses can dissociate from conscious perception as indicated by indirect arbitrary responses. ■

INTRODUCTION

Unilateral lesions of the parietal or frontal lobes can produce a variety of spatial disorders in humans. For instance, contralesional neglect commonly occurs after large lesions in the right hemisphere (see Heilman, Valenstein, & Watson, 1985; Mesulam, 1981). Such neglect is a complex phenomenon, involving several components (see Driver & Mattingley, 1998; Rafal, 1994). The characteristic failure to respond appropriately to contralesional stimuli may involve not only perceptual/attentional deficits, but also motor impairments. Several reports (Tegner & Levander, 1991; Bisiach, Geminiani, Berti, & Rusconi, 1990; Mesulam, 1981) suggested that neglect patients with anterior lesions may have a deficit in directing movements towards contralesional space (with the ipsilesional hand); while more posterior lesions to the inferior parietal lobe may produce purely perceptual biases. However, recent studies have challenged this dichotomy (Bisiach, Ricci, Lualdi, & Colombo, 1998; Mattingley, Husain, Rorden, Kennard, & Driver, 1998). Moreover, evidence from monkey single-unit recordings suggests that parietal regions may be involved in initial stages of spatially selective motor planning, for both hand and eye movements (Li, Mazzoni, & Andersen, 1999; Snyder, Batista, & Andersen, 1997, 1998; Andersen, Snyder, Bradley, & Xing, 1997; Colby, Duhamel, & Goldberg, 1996; Duhamel, Colby, & Goldberg, 1992).

Here we provide a new test of whether specific motor deficits for contralesional visual targets can be distinguished from a more general perceptual deficit in patients with chronic unilateral lesions restricted to either the parietal or frontal lobe. We focus on saccades, unlike previous patient studies that considered perceptual versus motor biases only for the control of hand movements (e.g., Mattingley et al., 1998; Tegner & Levander, 1991; Bisiach et al., 1990). Although some saccadic abnormalities have been shown in parietal patients (Heide, Blankenburg, Zimmermann, & Kompf, 1995; Duhamel, Goldberg, Fitzgibbon, Sirigu, & Grafman, 1992; Pierrot-Deseilligny, Rivaud, Gaymard, & Agid, 1991; Pierrot-Deseilligny, Rivaud, Gaymard, Muri, & Vermersch, 1995), further perceptual tasks were never implemented for comparison, so the saccadic deficits could have been a consequence of abnormal visual perception. We compared two versions of a temporal-order-judgment (TOJ) task (Posner & Cohen, 1980), recently shown to provide sensitive measures for lateral biases in saccades (Schiller & Chou, 1998) or perception (Rorden, Mattingley, Karnath, & Driver, 1997). Both tasks were implemented in two patient groups: one with unilateral lesions involving the inferior parietal lobule (IPL; see Figure 1 and Table 1), and one with more anterior lesions to dorsolateral prefrontal cortex, including the frontal eye field (FEF; see Figure 2 and Table 1).

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Figure 1. Lesion reconstructions for the parietal patients, mapped into standardized space (Montreal Neurological Institute representative brain, pitched 12° to match the scanning angle) using MRIcro software (see text). Group average is shown at top, with color coding the percentage of patients with structural damage to particular regions. The rows below show the lesion in individual patients. Note that the lateral cortical view of the lesions also includes subcortical damage.

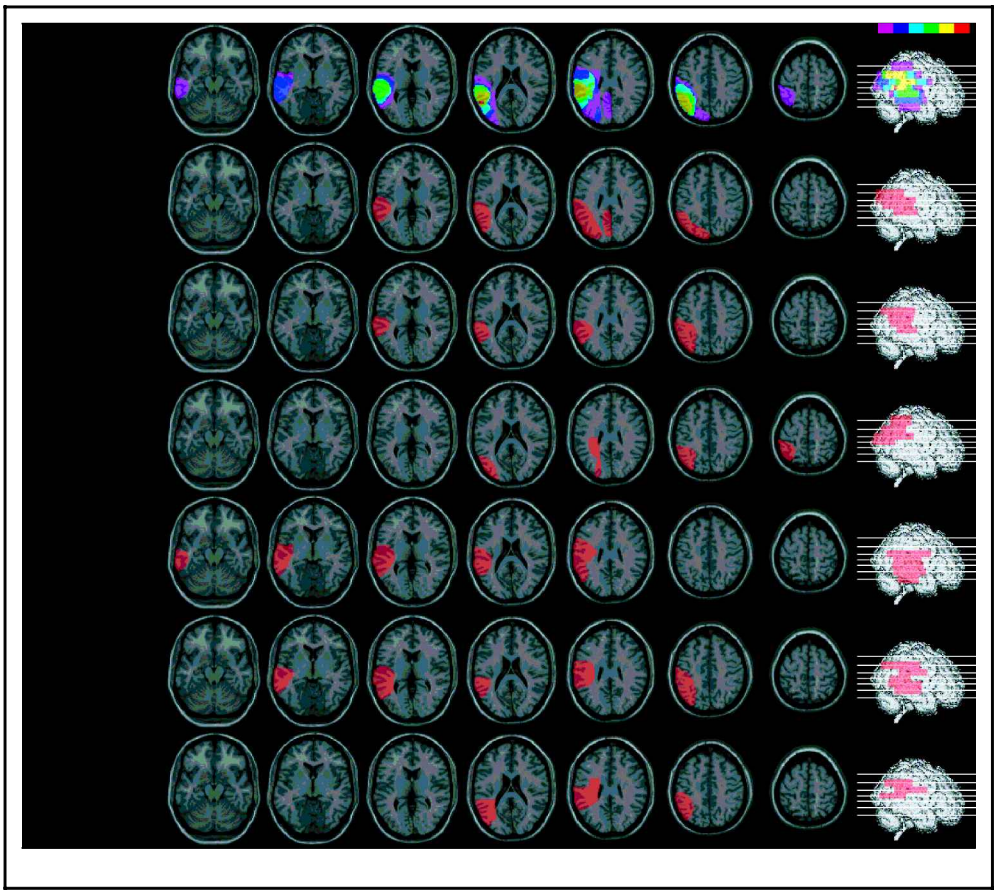

Figure 2. Lesion reconstructions for the frontal patients, in the same format as for the parietal patients in Figure 1.

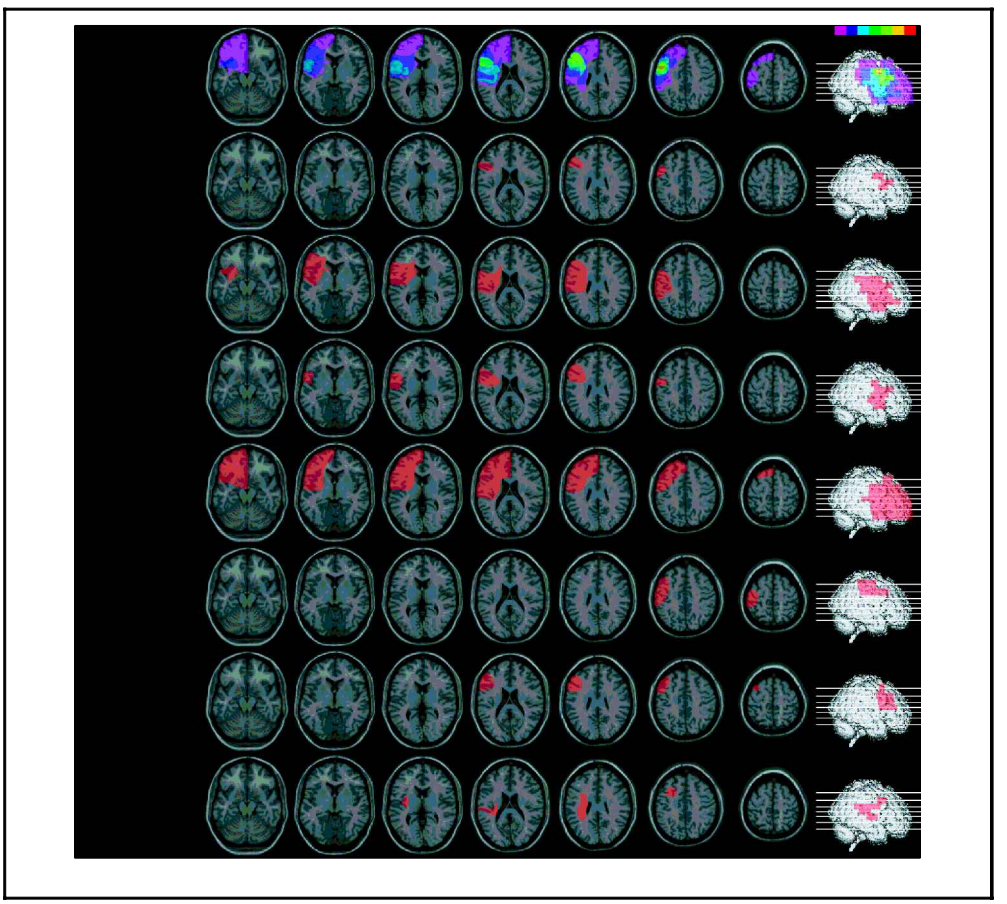

Table 1. Patient Information

				Lesion Information			
Patient	*Age*	*Sex*	*Clinical*	*Hemisphere*	*Vol.*	*Vint.*	*Etiology*
RA	66	M	A, H	Left	71	8	Stroke
LL	73	M	A	Left	40	2	Stroke
RR	69	M		Right	34	11	Stroke
JS	39	M		Right	73	11	Shrapnel
RS	51	M		Right	80	6	Stroke
KT	48	F	H	Right	46	22	Resection
Parietal mean	58				57	10	
OA	65	M		Left	18	13	Stroke
WA	75	F	A	Left	26	11	Stroke
JC	73	M	A, G	Left	106	11	Stroke
EE	68	M	A, G	Left	41	4	Stroke
JM	71	M		Left	15	3	Shrapnel
MK	65	M	H	Right	200	18	Aneurysm
SR	77	F		Right	13	3	Stroke
Frontal mean	71				60	9	

Vol. = Volume (in cm^3); Vint. = Vintage (rounded to the nearest year); A = Aphasia, H = Hemiparesis; G = Hemiplegia.

Two visual events were presented on each trial, one in each hemifield at various stimulus onset asynchronies (SOAs) (see Figure 3). In the saccade task, patients moved their eyes to whichever stimulus attracted gaze first, thus making a direct motor response towards the actual location of the leading stimulus. In the perceptual-manual task, patients indicated which stimulus was perceived first by pressing one of two central buttons (*not* located directly at the target positions) with their ipsilesional hand (this hand was used since many of the patients were contralesionally hemiparetic or hemiplegic; see Table 1). Normals show no bias towards one side (e.g., see Rorden et al., 1997). Any contralesional deficit in the patients should lead to an advantage for ipsilesional targets when appearing simultaneously with contralesional targets (cf. Schiller & Chou, 1998; Rorden et al., 1997), with contralesional targets receiving responses only when leading substantially in time. If the patient's lesion disrupted or delayed afferent inputs from the contralesional side, such a bias should be found for both the saccadic and the perceptual-manual task. On the other hand, if the lesion affected the planning or execution of contralateral saccades, one would predict a bias in the saccade task only.

If motoric spatial deficits in humans depend on more anterior lesions, and perceptual deficits on posterior lesions (Mesulam, 1981), the frontal group might be expected to show a purely saccadic TOJ deficit, while the parietal group might show a TOJ deficit in both tasks (or only when indicating their conscious perception via an arbitrary response, as in the perceptual-manual task). However, given recent single-unit findings on saccade generation in parietal regions (Li et al., 1999; Andersen et al., 1997; Snyder et al., 1997, 1998), a specific saccade deficit might be observed within the parietal group instead.

RESULTS

The TOJ data for the saccade and perceptual tasks were scored separately. For analysis, the percentage of responses made to the contralesional target in each task was arcsine transformed for each patient, and then subjected to a 2×11 mixed ANOVA with lesion location (frontal vs. parietal) as the between-subject factor and SOA (-250 to $+250$ msec in 50-msec intervals) as the within-subject factor.

Saccadic TOJs

The mean percentage (untransformed) of contralesional responses in the saccade task, as a function of the SOA between targets, is shown separately for the two patient groups in Figure 4, where it can be seen that the parietal and frontal group show a different pattern. The ANOVA found a main effect of SOA [$F(10,110) = 31.93$, $p < .001$] simply reflecting the

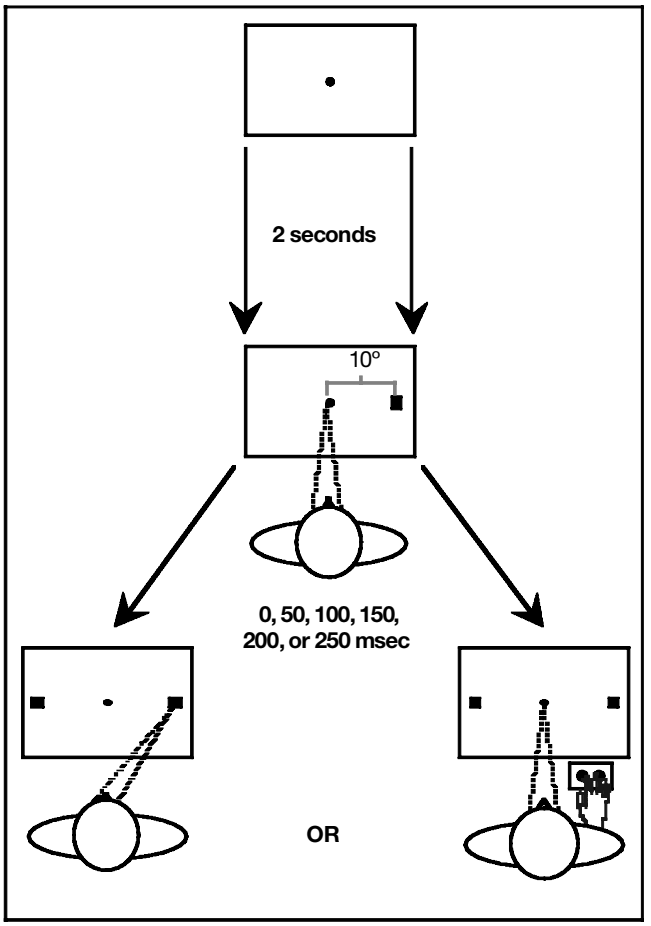

Figure 3. Sequence of events for one trial (time runs top to bottom). A visual target appeared in each hemifield, with variable temporal SOA. As indicated by the fork at the bottom, patients made either a saccadic response or a button-press perceptual judgment, depending on the task.

increased responses towards the contralesional field when the contralesional stimulus led by an increasing amount of time. More importantly, there was also a Lesion × SOA interaction [$F(10,110) = 2.24$, $p < .02$]. This interaction reflected a saccadic bias towards the ipsilesional visual hemifield in the parietal group only (they made significantly more ipsilesional than contralesional saccades when targets were simultaneous, at the 0-msec SOA, [$t(5) = 2.15$, $p < .05$]). Because these experiments were designed as a group study, we had insufficient power to analyze the psychometric functions for each patient individually. However, inspection of the data from each patient in the parietal group revealed that five of the six parietal patients made more ipsilesional than contralesional saccades (the only exception was case KT). By contrast, the frontal group showed a symmetrical pattern of ipsilesional and contralesional saccades against SOA (ipsilesional saccades were no more likely than contralesional saccades for them at the 0-msec SOA, [$t < 1$, *ns*]). This difference between groups was further confirmed by an ANOVA, which tested the symmetry of the response pattern against SOA, using the percentage of contralesional saccades at negative SOAs, but rescoring the positive SOAs in terms of the percentage of *ipsilesional* saccades (which yields the inverse of the pattern shown in the right half of Figure 4). The data from all SOAs except zero were then submitted to a two-way within-subject ANOVA with absolute SOA (regardless of sign) as one factor (five levels), and direction of saccade (contralesional vs. ipsilesional) as the other factor (two levels). Asymmetry should be apparent as an interaction. This was reliable for the parietal group [$F(4,20) = 3.89$, $p < .02$], but did not approach significance for the frontal group [$F < 1.0$, *ns*].

Perceptual-Manual TOJs

The mean percentage (untransformed) of contralesional choices in the perceptual-manual task, as a function of the SOA between targets, is shown separately for the two patient groups in Figure 5. It can be seen that the difference between groups that was so apparent in the saccade task is no longer evident (compare with Figure 4), as both groups now show a fairly symmetrical pattern around zero against SOA. Indeed, at the zero SOA, ipsilesional choices were no more likely than contralesional choices in either group [for both patient groups, $t < 1$, *ns*]. In the mixed ANOVA on contralesional choices against SOA, with lesion group as the between-subject factor, there was

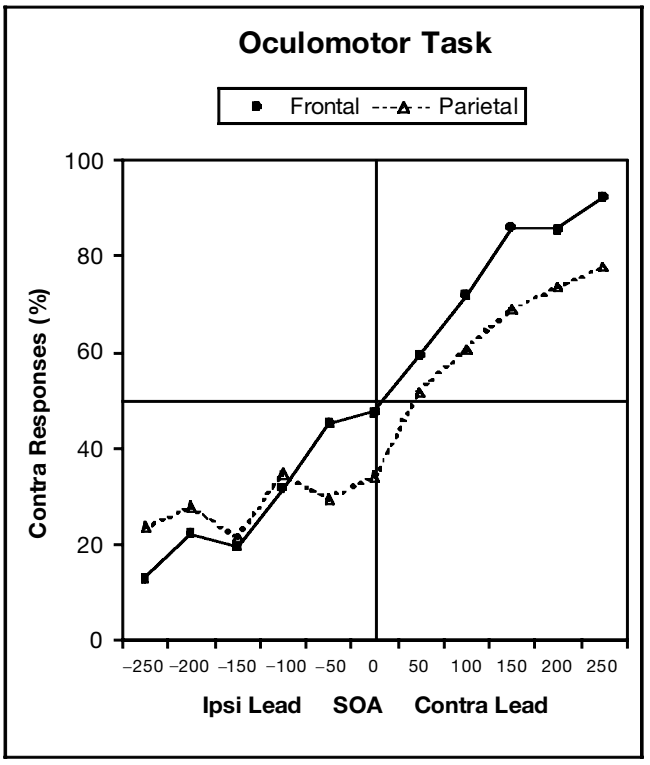

Figure 4. The proportion of contralesional saccades made by the frontal group (solid line) and by the parietal group (dotted line) as a function of contralesional to ipsilesional SOA.

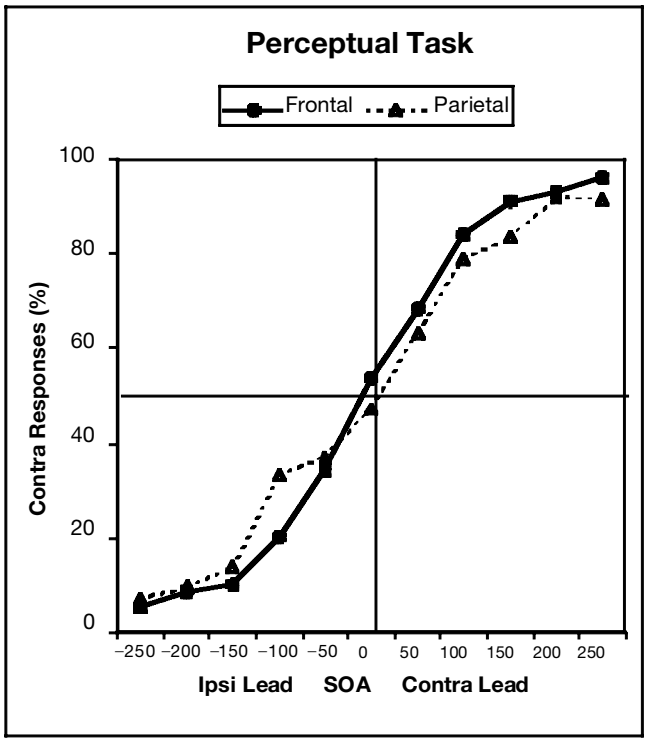

Figure 5. The proportion of contralesional perceptual choices made by the frontal group (solid line) and by the parietal (dotted line) group as a function of contralesional to ipsilesional SOA.

again a main effect of SOA due simply to the increased proportion of contralesional choices when the contralesional stimulus led by increasing amounts of time [$F(10,110) = 88.36$, $p < .001$]. However, in contrast to the saccade task, the critical Lesion × SOA interaction was no longer significant [$F(10, 110) = 1.09$, *ns*]. ANOVAs testing for symmetry around the zero SOA now found no significant deviation within either group, unlike the parietal result for the saccade task. The apparent trend for a group difference at the SOA of – 100 (see Figure 5) did not approach significance ($t(11) = 1.59$, *ns*).

Reaction Times (RTs)

Although the above TOJs provide the critical data, we were able to record RTs for the responses also (though note that our instructions placed no emphasis on speed). The average RTs are shown for each group and task in Table 2, for just the middle three SOAs (– 50, 0, 50) as the others contained too few data points to allow a meaningful comparison of contralesional and ipsilesional targets (i.e., there were virtually no responses in the "wrong" direction at the longer SOAs for some patients). For each task, we conducted a mixed ANOVA on RTs, with the factors of group, hemifield, and SOA. The saccade RT analysis found only a slight trend for a three-way interaction [$F(1,11) = 3.22$, $p < .10$], due mainly to frontal patients being faster to make contralesional saccades (421 msec) than ipsilesional ones (509 msec) when the contralesional stimulus led by 50 msec [$t(6) = 3.14$, $p < .02$], a pattern which is reminiscent of Henik, Rafal, and Rhodes' (1994) saccade findings in a similar frontal group. The three-way interaction was significant for RTs in the perceptual-manual task [$F(2,22) = 5.11$, $p < .02$], now due to frontal patients tending to make slower contralesional (617 msec) than ipsilesional (565 msec) responses overall, regardless of SOA [$F(1,6) = 4.84$, $p < .07$], while there was a trend for parietal patients to respond slower whenever reporting (erroneously) that a nonleading stimulus appeared first [$F(2,10) = 3.82$, $p < .06$]. Finally, the parietal patients were slower overall (978 msec) than the frontal patients (591 msec) in the manual response task [$F(1,11) = 11.74$, $p < .01$). Thus, while there were some group differences in RT (as discussed below), these do not seem to explain the TOJ results presented earlier (see Figures 4 and 5). In particular, the groups did not differ in overall RT for the saccadic task, even though they did show a group difference in TOJs (with a specific parietal abnormality; see Figure 4). Moreover, the parietal patients showed no reliable latency differences between ipsilesional and contralesional saccades, despite their TOJ bias towards the ipsilesional side in the saccadic task. Indeed, parietal saccade latency was virtually identical for ipsilesional and contralesional targets in the simultaneous (0 msec SOA) condition (see Table 2), even though for this condition the TOJ data revealed that the parietal patients saccaded ipsilesionally much more often.

Saccade Metrics

Mean saccadic amplitudes were scored for the middle three SOAs in the saccade task (see Table 2; the other SOAs had too few saccades towards the target that appeared second for any meaningful comparison of saccades in opposite directions). Saccade amplitudes for these middle SOAs were analyzed with a three-way mixed ANOVA (Group × Direction × SOA). The only term to approach significance was a main effect of saccade direction [$F(1,11) = 4.26$, $p = .06$]. Mean amplitude was 6.95° for contralesional saccades versus 8.02° for ipsilesional saccades. This suggests a more pronounced saccadic hypometria in the contralesional direction, for both patient groups.[1]

DISCUSSION

These results suggest that the human IPL is involved with saccade generation. Moreover, this oculomotor role of the IPL may be independent of conscious visual perception (as indicated by more arbitrary responses such as the button presses in the perceptual-manual

Table 2. Proportion of Responses, Latencies, and Amplitudes (Saccade Task Only)

			SOA		
Patient Group	*Task*	*Field*	*– 50*	*0*	*50*
Frontal	Oculomotor	Contra	45.1% 413 msec	47.6% 439 msec	59.5% 421 msec
			7.0°	7.3°	7.0°
		Ipsi	54.9%	52.4%	40.5%
			414 msec	442 msec	509 msec
			8.6°	7.5°	7.2°
	Perceptual-Manual	Contra	34.3%	53.9%	68.4%
			593 msec	633 msec	625 msec
		Ipsi	65.7%	46.1%	31.6%
			572 msec	540 msec	584 msec
Parietal	Oculomotor	Contra	29.4%	34.3%	51.8%
			630 msec	560 msec	590 msec
			7.5°	6.4°	6.5°
		Ipsi	70.6%	65.7%	48.2%
			556 msec	565 msec	572 msec
			8.2°	8.6°	8.1°
	Perceptual-Manual	Contra	37.1%	47.2%	63.3%
			1028 msec	952 msec	944 msec
		Ipsi	62.9%	52.8%	36.7%
			938 msec	976 msec	1032 msec

task). The parietal group showed an ipsilesional bias in the saccadic TOJ task (Figure 4, dotted line), yet exhibited unbiased TOJs in the perceptual-manual version of this task for the very same stimuli (Figure 5, dotted line). This extends previous reports of saccade biases following posterior parietal damage in humans (Heide et al., 1995; Pierrot-Deseilligny et al., 1991, 1995; Duhamel, Goldberg, et al., 1992) since the ipsilesional oculomotor biases observed here cannot be reduced to any primary deficit in visual perception (e.g., delayed afferent input for the contralesional side).

The frontal group did not show any ipsilesional bias in their directional choices in the TOJ task, neither in the saccadic task nor in the perceptual-manual task (see solid lines in Figures 4 and 5). The saccadic result for this chronically lesioned frontal group is in accord with the good performance recently shown on a similar saccade task in monkeys with chronic FEF lesions (Schiller & Chou, 1998). However, this monkey study did find an "acute" saccade bias immediately following an FEF lesion that resolved with time, perhaps due to an initial disruption of interconnected areas. No perceptual task was run for comparison in the monkey study. Adding this may be important in future animal work, given the different outcomes for the saccadic and the perceptual-manual tasks in our parietal patients.

Two previous patient studies (Robertson, Mattingley, Rorden, & Driver, 1998; Rorden et al., 1997) used only a perceptual version of the temporal-order task, yet did find ipsilesional biases. These studies selected acute patients on the basis of showing florid clinical neglect, rather than selecting chronic patients on anatomical grounds as in this study. Their patients typically had very extensive lesions, involving not only the parietal cortex, but also the temporal and frontal lobes. Purely perceptual deficits (as found in their studies) clearly contribute to clinical neglect (e.g., see Driver & Mattingley, 1998; Rafal, 1994; Milner, Harvey, Roberts, & Forster, 1993). However, the saccadic bias identified in the parietal group here may constitute a further, exacerbating component to clinical neglect, particularly since the inferior parietal lobe is commonly included in the large lesions of most neglect patients (Vallar, 1993). If so, then neglect patients should exhibit a more severe ipsilesional bias in the saccadic task than the perceptual-manual version of the temporal-order task

(as for our chronic inferior parietal group), even though they may also show some perceptual bias in the latter task (unlike our patients). We find exactly this when applying the present two tasks to patients selected for showing clinical neglect or extinction (Rorden, Ro, Harvey, Kramer, & Driver, unpublished observations). That is, such patients show larger biases with the saccade task than with the perceptual-manual task, but they do exhibit some pathological ipsilesional bias for both. Thus, they have a saccade deficit over and above any perceptual deficit.

The finding of just a saccadic bias in the present parietal group, in the absence of any deficit on the perceptual-manual version of the task, is in accord with recent proposals that circuits computing visuomotor transformations for direct spatial responses may dissociate from those for conscious visual perception (e.g., Ladavas, Zeloni, Zaccara, & Gangemi, 1997; Milner & Goodale, 1995). Although the present "perceptual" task did involve manual responses, these were arbitrary button presses rather than direct reaches to the target location, and so would be considered to reflect conscious perception rather than direct control of action under Milner and Goodale's (1995) influential formulation. From this perspective, our parietal group might be considered to have damage to that part of the "dorsal" stream involved in the initial generation of saccades. Consistent with this, recent single-cell studies in monkey have suggested that the posterior parietal cortex is involved in the initial, spatially selective stages of motor planning (e.g., Snyder et al., 1997; Bracewell, Mazzoni, Barash, & Andersen, 1996; Mazzoni, Bracewell, Barash, & Andersen, 1996), with the lateral intraparietal area particularly implicated for saccades (Andersen et al., 1997; Duhamel, Colbe, et al., 1992; but see Gottlieb & Goldberg, 1999; Snyder et al., 1997). Moreover, unilateral lesions to this area in the monkey (with reversible muscimol deactivation) have recently been shown to produce an ipsilesional saccadic bias, in a task similar to the saccadic temporal-order task used here with the patients (Li & Andersen, 1997). The *inferior* parietal lobule was involved in all our parietal patients, whereas the *superior* parietal lobule was also additionally involved in only one (RR). Damage to the IPL may thus be critical for the bias in saccadic choice, which we report. The critical lesion may involve the lateral intraparietal region, as implicated in initial saccade choice for monkeys (Andersen et al., 1997; Li & Andersen, 1997; Snyder et al., 1997; Duhamel, Colby, et al., 1992). Some part of this region was involved in all our parietal patients, typically in its posterior extent (see Figure 1 and cf. Pierrot-Deseilligny et al., 1995).

Although the TOJ saccade task did not reveal any bias in saccade choice for our frontal group, previous work in this laboratory on such patients (i.e., with chronic lesions involving the FEF)—including those who participated in the current study—has revealed slowed voluntary saccades to the contralesional field (Henik et al., 1994). Moreover, contralesional saccades tended to be hypometric in the present frontal group, as for the parietal group, confirming that their saccades were not completely symmetrical. Given that the FEF is thought to be involved in a decision stage for voluntary saccades (see Hanes, Patterson, & Schall, 1998), we should consider why the saccadic TOJs themselves showed no ipsilesional bias in the frontal group. One speculative possibility may be the following. Note that the saccadic TOJ task requires a specific type of "decision," between competing demands for prosaccades towards one or the other stimulus in bilateral displays. Using displays without such bilateral competition, Henik et al. (1994) found that while voluntary saccades are slower contralesionally than ipsilesionally in unilateral FEF patients, the reverse was observed for reflexive saccades (which they attributed to "disinhibition" of contralesional reflexes, see also Rafal, Machado, Ro, & Ingle, 2000; Guitton, Buchtel, & Douglas, 1985). One aspect of the present frontal results is reminiscent of Henik et al.'s findings. When the contralesional stimulus led by 50 msec, contralesional prosaccades had shorter latencies than ipsilesional ones, for the frontal group only. Therefore, it is possible that the bias to choose an ipsilesional saccade may have been counteracted by a disinhibition of reflexive orienting toward the contralesional target.

The key new finding of our study is the directional saccadic bias in the parietal group. Finding such a bias in these parietal patients does not fit conventional wisdom that only lesions involving the frontal lobe produce motoric spatial biases in humans (e.g., Tegner & Levander, 1991; Bisiach et al., 1990; Mesulam, 1981). However, our parietal patient result agrees with single-cell findings showing parietal involvement in initial stages of motor planning (e.g., Snyder et al., 1997), and specific involvement of the lateral intraparietal area in saccade choice (e.g., Andersen et al., 1997; Li & Andersen, 1997; Li et al., 1999).

METHODS

Patients

Six patients with unilateral lesions restricted to the posterior association cortex including the IPL, and seven with more anterior lesions restricted to the dorsolateral prefrontal cortex including the FEF, participated after informed consent. The FEF was located as approximately 2 cm anterior to the motor hand area, at the junction of the superior frontal sulcus and the precentral sulcus (see Ro, Cheifet, Ingle, Shoup, & Rafal, 1999; Paus, 1996). Note that each patient group served as a control group for comparison with the other. All were recruited from the patient population of the Veterans

Administration in Martinez, CA and were in the chronic stages of their neurological disorder, being tested no earlier than 1 year from lesion onset, to minimize any influence from diaschisis. All the patients had intact visual fields and normal oculomotor function on standard clinical testing of saccades, pursuit eye movements, and optokinetic nystagmus. None had any clinical signs of hemispatial neglect, or a coexisting neurological disorder (e.g., Parkinson's disease) at the time of testing. The magnetic resonance images or computerized tomographic images of the patient's brain were used to transform each lesion onto standardized transaxial templates for subsequent computerized coregistration and reconstruction (see Frey, Woods, Knight, & Scabini, 1987). These reconstructions were then translated onto the corresponding matching MRI templates via MRIcro software (www.psychology.nottingham.ac.uk/staff/cr1/mricro.html).

Figure 1 shows the lesion reconstructions for the parietal group, with the group-averaged reconstruction in the top row, and the individual patients in the rows below. Figure 2 shows the lesion reconstructions for the frontal group, with the same format. Clinical details of each patient are given in Table 1. Although the patients in the parietal group were somewhat younger (mean = 58, *SD* = 14) than the patients in the frontal group (mean = 71, *SD* = 5), the two groups did not differ in mean lesion volume (Parietal = 57 cc, *SD* = 20; Frontal = 60, *SD* = 70). Note that while the lesions in the frontal group include various dorsolateral prefrontal regions in addition to the area of maximal overlap centered on the FEF, previous comparisons with other frontal groups (who had dorsolateral prefrontal lesions that spared the FEF) in experiments from the same laboratory (Rafal et al., in press; Henik et al., 1994) suggest that this lesion group has some specific deficits in saccade execution consistent with damage to the human homologue of FEF (see Discussion).

Apparatus

All patients were tested on a PC connected to a NEC Multisync VGA monitor set to 640 × 480 pixels. The timing of the visual displays was synchronized with the vertical synchronization of the monitor at 60 Hz. For the perceptual-manual task, judgments were made by pressing one of two buttons on a joystick, with the index or second finger of the ipsilesional hand that rested on the buttons, indicating in this arbitrary manner which stimulus was consciously perceived first. In the saccade task, eye position was monitored using an Applied Science Laboratories (Bedford, MA) Eye-Trac 210 connected to the parallel port of the computer. The digital output from the eye tracker was sampled at 1000 Hz, and subsequently treated with a 200-Hz low pass filter. Saccadic eye movements were identified via an automated analysis program, with their initiation defined as the point at which velocity exceeded 50°/sec. Saccadic amplitude was calculated by measuring the distance traversed by the eye from the point at which eye velocity exceeded 50°/sec to the point where it dropped below this.

Stimuli and Procedures

All the stimuli were filled, dark gray shapes on a light gray background. The circular fixation point measured 0.1° at the center of the computer monitor. The target stimuli were squares measuring 1° appearing 10° to the left or right of the center (see Figure 3).

At the start of each trial, the central fixation point appeared for 2 sec before the onset of a left or a right square target. The second target appeared on the other side, either simultaneous with (0 msec condition), or 50, 100, 150, 200, or 250 msec after onset of the first stimulus. For ease of exposition (given that both left- and right-hemisphere patients were tested), negative SOA values between the two target stimuli will refer to conditions where the *ipsilesional* target led in time, whereas positive SOA values will refer to the *contralesional* target leading. For example, an SOA of – 150 refers to the condition where the ipsilesional target led the contralesional target by 150 msec.

The patients sat approximately 57 cm from the computer monitor. In the perceptual-manual task, they were instructed to press the left button on the joystick if they perceived the left target as appearing first, and the right joystick button if they saw the right target first. In the saccade task, the patients were asked simply to move their eyes to whichever peripheral square drew their gaze first. The order of tasks was counterbalanced across patients. For the first eight patients run, the first stimulus event was presented for 1000 msec and the second stimulus for 1000 msec minus the asynchrony, giving the patients a 1000-msec time window from the first stimulus event to make their response while the targets were still visible. This was extended for the last five patients tested (one frontal patient and four of the six parietal patients) to a 2000-msec window, since some responses were slower than anticipated, especially in the parietal group. Note that this extended presentation applied equivalently for both tasks.

Each patient first completed one or two practice blocks, consisting of 11 trials each, before experimental data were collected for either task. Following this practice, a minimum of 220 trials were completed at each task (330 trials for the last five patients), comprising 20 (or 30) trials at each of the 11 different SOA conditions within each patient, all intermingled in a random order. The critical dependent measure was the side responded to, which provides a TOJ. The instructions emphasized accuracy in these judgments rather than speed.

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Note

1. Although saccades generated towards the ipsilesional targets were also hypometric, it is the difference between contralesional and ipsilesional saccades that is of interest. Inspection of the individual eye movement traces from the patients in this study showed the stereotypical metric properties of saccadic eye movements. Specifically, saccades to all targets were somewhat hypometric, and subsequent corrective saccades and postsaccadic drifts were generated to adjust for this hypometria. The abnormal metric pattern was that contralesional saccades were significantly more hypometric than ipsilesional saccades.

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