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Regional cerebral correlates of global motion perception

Evidence from unilateral cerebral brain damage

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Summary

We used a psychophysical task to measure sensitivity to motion direction in 50 stroke patients with unilateral brain lesions and 85 control subjects. Subjects were asked to discriminate the overall direction of motion in dynamic stochastic random dot displays in which only a variable proportion of the spots moved in a single direction while the remainder moved randomly. Behavioural and neurophysiological evidence shows that the middle temporal (MT/V5) and middle superior temporal (MST) areas in the macaque monkey are indispensably involved in the perception of this type of motion. In human subjects too, lesions in the same region disrupt performance on this task. Here we assessed more extensively the correlation between direction sensitivity for global motion and the anatomical locus of the lesion. Thresholds for perceiving the direction of global motion were impaired in the visual field contralateral to the lesion in patients with lesions in the occipitoparietal and parietotemporal areas involving the human analogue of areas MT/V5 and MST, but not by lesions in the occipito-temporal or anterior frontal areas. Patients with lesions involving the anterior temporal or parietal lobes displayed poor performance for stimuli presented in either visual field, which is consistent with the large and bilateral receptive fields in these areas in monkeys. The perception of global motion was also more impaired in the centripetal than the centrifugal direction in the hemifield contralateral to the MT/V5 lesion. Surprisingly, thresholds were normal in all patients when the displays contained static but not dynamic visual noise, suggesting that their deficit reflects an inability to filter out dynamic noise. Although frequent repeated testing of some patients whose lesion involved the human homologue of MT was accompanied by an improvement in performance, this was no greater than in other patients who received training on different motion tasks.

Keywords: brain damage; motion processing; visual areas

Abbreviations: fMRI = functional MRI; MCT = motion coherence task; MST = middle superior temporal; MT = middle temporal

Introduction

Two major processing streams have been identified in the visually responsive cortex of the macaque monkey, each involving several areas defined on anatomical and physiological grounds (Ungerleider and Mishkin, 1982; for a review, see Ungerleider, 1996). Cortically, both routes originate in the striate cortex (VI). One route courses ventrally into the posterior temporal lobe and primarily mediates colour perception and the analysis of features indispensable for object recognition, while the other distributes dorsally into the posterior parietal lobe and is chiefly involved in analysing visual–spatial characteristics of the scene. Motion analysis is more prominent in the dorsal pathway (Maunsell and Newsome, 1987; Albright, 1992; Andersen, 1997; Vaina, 1998), and electrophysiological recordings, selective

chemical lesions and microstimulation (for a review, see Wurtz *et al*., 1990) have established that the middle temporal area (MT) is particularly important for motion perception, perhaps because it is the first level in the hierarchy where global motion is analysed.

A common example of global motion is the perception of a coherent group of dots moving cohesively in a particular direction among masking dots moving randomly. For example, MT lesions in macaque monkeys temporarily elevate motion coherence thresholds (the proportion of dots that have to move coherently for their direction to be discriminated within the field defect contralateral to the lesion) and by concomitantly measuring neuronal responses and psychophysical performance, Newsome *et al*. and Britten *et al*. showed that the MT neurones signal the direction of global motion with a fidelity that matches the monkey's psychophysical performance (Newsome *et al*., 1986; Britten *et al*., 1992). Subsequently, Celebrini and Newsome demonstrated that neurones in the adjacent region MST also mediate this motion discrimination task (Celebrini and Newsome, 1995). The strongly direction-selective neurones of the MT area also integrate direction over global motion fields that arise during eye movements or when navigating through the environment. The motion analysis continues and is elaborated beyond MT, in visual areas of the posterior parietal lobe and the anterior portions of the temporal lobe, which are also involved in aspects of motion perception (Boussaoud *et al*., 1990).

Area MT/V5 of the macaque is histologically identifiable by its distinctive heavy myelination, and this was one of the first indications that a similar region in the human brain, first described on the basis of its myeloarchitecture by Flechsig (Flechsig, 1920), might correspond to macaque MT/V5. Recent functional imaging studies by positron emission tomography (Miezin *et al*., 1987; Corbetta *et al*., 1990; Zeki *et al*., 1991; Watson *et al*., 1993) and functional MRI (fMRI) (McCarthy *et al*., 1995; Tootell *et al*., 1995; McKeefry *et al*., 1997; Sunaert *et al*., 1999) indicate that human MT/V5 is situated ventrolaterally, at the confluence of the occipital and temporal lobes and the junction of Brodmann areas 19 and 37 inferiorly, and does coincide with Flechsig's area 16 (see Fig. 1) (Thurston *et al.*, 1988; Vaina *et al.*, 1998). The source of the dipoles evoked by moving stimuli was also localized to this region (Probst, 1993). Transcranial magnetic stimulation of this region transiently disturbs motion perception (Beckers and Homberg, 1992; Hotson *et al.*, 1994; Beckers and Zeki, 1995; Walsh *et al*., 1999), as well as eliciting moving visual phosphenes (Stewart *et al*., 1999). Furthermore, the large lesions that cause the rare, severe and permanent motion perception deficits in several patients include this region (Zihl *et al*., 1983, 1991; Vaina, 1989; Vaina *et al*., 1990; Baker *et al*., 1991; Morrow and Sharpe, 1993; Plant and Nayakama, 1993; Plant *et al*., 1993; Barton *et al*., 1995; Barton and Sharpe, 1997). Finally, there is neuroanatomical evidence, including fibre degeneration (Clarke and Miklossy, 1990) and cytochrome oxidase histochemistry (Tootell and Taylor, 1995), that MT/V5 lies in this region.

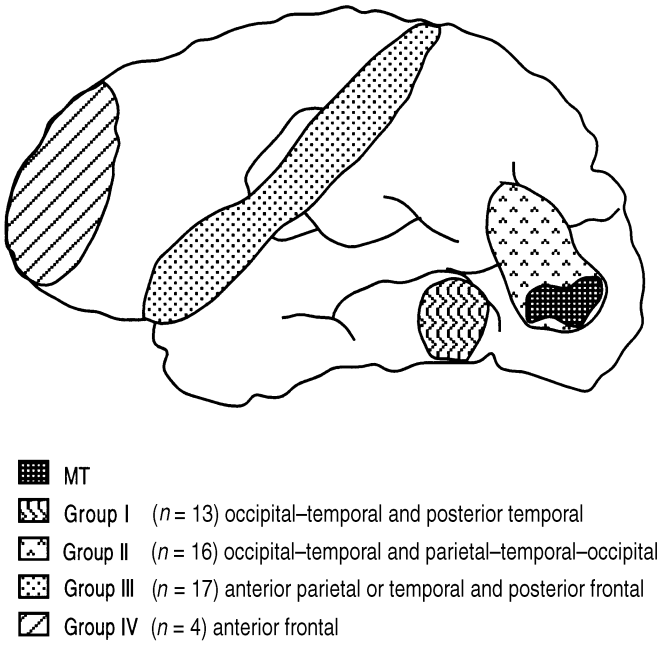

Fig. 1 Outline diagram of the left hemisphere showing the region of cortex and underlying white matter that was involved in every patient in each of the four groups. Human MT/V5 lies within the common area for Group II. The common area for each group was estimated by drawing the borders of the lesion in each patient, as visible in CAT or MR scans, on to the standard outline shown here and as described by Vaina *et al*. (Vaina *et al.*, 1990).

One of the tasks used to probe psychophysically the human homologue of the macaque area MT/V5 is adapted from studies by Newsome and Paré, who introduced the task to investigate the receptive field properties of neurones in MT and the effects of neurotoxic lesions in MT on motion sensitivity thresholds in the macaque monkey (Newsome and Paré, 1988). The monkeys performed a two-alternative, forced-choice discrimination of motion direction in a set of dynamic random dot patterns that contained a unidirectional motion signal embedded in a field of masking motion noise. The strength of the motion signal (termed coherence) was varied from trial to trial by changing the proportion of dots that moved coherently. They found that the threshold coherence for discriminating direction of global motion was elevated from ~5% to nearly 100% shortly after the lesion, although it rapidly returned to normal levels. In the present study we examined any association between the anatomical locus of cortical damage and performance on this task and whether deficits on this task are specific to aspects of the display, such as the type of masking noise and the direction of motion signal.

Although isolated single case studies are important in showing that a particular lesion can produce a selective deficit (e.g. Zihl *et al*., 1983; Vaina and Cowey, 1996; Vaina *et al*., 1998*a*, *b*), they do not reveal whether other lesions, whether within the same territory or outside it, have similar

consequences. Given the scarcity of studies in which large groups of neurological patients are screened with respect to psychophysical tasks used to investigate motion processing in area MT/V5, we report here the results of measuring the thresholds for the perception of motion coherence (motion coherence task, MCT) in 50 patients with unilateral cortical damage caused by a single stroke, sometimes involving area MT/V5. Schenk and Zihl investigated perception of motion coherence in 32 patients with unilateral lesions, as determined by CT or MRI studies, and found that three of these patients needed an up to four times more coherent motion signal to accurately discriminate the direction of motion for stimuli presented in the the contralesional visual hemifield, while their performance in the ipsilateral field was no different from that of the normal controls (Schenk and Zihl, 1997). Although the lesions in these three patients were large, they involved the junction of the Brodmann areas 21, 22 and 37, and the authors suggest that this region is functionally equivalent to area MT/V5 in the macaque. However, unlike monkeys with lesions confined to area MT, the deficits were permanent in two of the three patients.

In a study primarily concerned with defects in smooth pursuit eye movements in 26 patients with unilateral cortical lesions (Barton *et al*., 1996), the authors found six patients with elevated thresholds for motion leftward or rightward and two patients with a bi-directional increase. In seven of these patients the region of overlap in the lesions included human MT/V5. However, as the patients were instructed to look directly at the displays, motion coherence could not be measured for each hemifield separately, which is the main concern of the present paper. In an earlier study, Barton and colleagues were the first to report that discrimination of the direction of global motion in centrally fixated displays was worse for motion towards the side of the lesion, i.e. towards the fovea in the contralateral hemifield (Barton *et al*., 1995). However, all of the patients studied by Barton *et al*. also had complete or incomplete hemianopic or quadrantanopic field defects, making it difficult to compare performance in the two hemifields. We therefore confined our measurements to the separate hemifields of patients whose field defects had resolved or who had never had a clinically detected field defect. We were interested in the severity of any deficit, given reports that motion perception can be virtually abolished in patients with large lesions that include MT/V5 (Zihl *et al*., 1983, 1991; Vaina, 1989, 1994) but not in monkeys with smaller lesions that nevertheless involve total destruction of MT/V5 (Cowey and Marcar, 1992; Marcar and Cowey, 1992; Rudolf and Pasternak, 1999). We also looked for evidence of hemispheric asymmetry in the perception of coherent motion in a large group of control subjects, given the evidence that only patients with left sided lesions had bidirectional impairments in motion coherence (Barton *et al*., 1996). Finally, we studied any improvement in the extent of the impairment of motion perception with practice in selected patients, given the evidence that in monkeys the immediate and severe effects of lesions to MT/V5 diminish even within days or weeks, sometimes with apparently complete recovery (Newsome and Paré, 1988) or at least with marked improvement (Rudolf and Pasternak, 1999).

Methods and patients/subjects

From a group of 120 stroke patients who participated over 4 years in psychophysical studies of visual motion perception, we selected a subset of 50 right-handed patients (16 female, 34 male) with unilateral lesions (right or left hemisphere) caused by embolic strokes in 42 cases, haemorrhagic stroke in seven cases and aneurism in one case. All the patients were first evaluated with the motion coherence test between 7 and 12 days from the cerebrovascular accident, after their discharge from the acute care hospital, and except for the sub-set given repeated testing, the testing described here was completed between 3 and 4 weeks after the lesion. At the time of the study all patients had normal visual acuity for their age, after optical correction where necessary, and most had full visual fields in both eyes by tangent Goldmann perimetry and/or tangent screen perimetry. Patients with a field defect approaching the central retina had already been excluded. The few patients who had a field defect who were included had the defect beyond the central 30 degrees. All patients had normal vision as judged perimetrically in the central 30 degrees. (The patients who participated in Experiment 2, in which stimuli were shown at larger eccentricities, all had full visual fields.) Patients' age ranged between 27 and 65 years. Neuropsychological examinations did not reveal any signs of visual neglect. Contrast sensitivity for detection and discrimination of moving sinusoidal gratings was normal in both visual fields in 47 of the patients, at spatial frequencies of 0.2, 0.5 and 1.0 cycles/degree and temporal frequency of 6 Hz. Three patients were slightly impaired. All patients and subjects gave informed consent according to the declaration of Helsinki and in accordance with the Boston University Human Subjects Committee and, where appropriate, the Brigham and Women's Hospital Human Subjects Committee, who gave approval for the study.

The patients were divided into four groups according to the site of the lesion, as shown on CT or MRI scans. The groups are defined and the lesions localized schematically in Fig. 1. Lesions in Group I were confined to the caudal part of the occipitotemporal ventral pathway, which is predominantly involved in form perception (Ungerleider and Mishkin, 1982; Ungerleider, 1996). The patients in Group II had lesions in the dorsal visual pathway, with direct or indirect (white matter) involvement of the human homologue of MT/V5. Group III lesions involved the rostrolateral temporal lobe and therefore the higher-level motion system. Group IV lesions involved the rostral frontal lobe, always in the right hemisphere. Since the imaging data were obtained for clinical purposes in different scanners and over a period of years, and the methods of imaging (i.e. angulation, slice thickness T_1- or T_2-weighting) and imaging modality (CT or MRI) varied, a precise localization of the lesion expressed in

Talairach coordinates was not possible. Moreover, the confluence of the parietal–temporal–occipital cortex, the area we focus on in this study, is extremely gyrified (Zilles *et al*., 1998) and therefore much of its surface area is buried within sulci. The borders of the lesion are rarely sharp and on each anatomical slice they were drawn by eye along the most obvious border between normal and abnormal tissue. Many lesions clearly involved white matter. The borders were then transferred onto a lateral view of a standard hemisphere as described by Vaina and colleagues (Vaina *et al*., 1990; Fig. 1). For each group the region involved in every lesion was then determined by superimposing all the outlines. Although the heterogenous functional imaging methods available provide a consistent localization of the human MT/V5, none of them establishes a consistent relationship between this motion responsive area and the sulcal and gyral pattern in this region. A recent fMRI study provides a stable anatomical landmark that will in future permit a more detailed correlation of structural MRI and CT images (Dumoulin *et al.*, 2000).

The patients' results on the motion task (see below) were compared with the results from 85 normal control subjects ranging in age from 25 to 66 years. Like the patients, males outnumbered females by about two to one. In all the psychophysical tasks subjects responded verbally and the responses were promptly entered by the examiner on the computer keyboard. The responses were given after the stimulus disappeared, and during the period of the stimulus presentation the examiner monitored the patient's fixation. Any trials with noticeable eye movements were promptly discarded and the stimulus repeated.

Experiment 1: motion coherence

As in behavioural studies of monkeys, the aim of this task was to determine the threshold level of motion coherence at which a subject could reliably discriminate the prominent direction of motion. The stimuli were random dot kinematograms in which a specifiable proportion of spatially dispersed dots provided a correlated motion signal among uncorrelated masking noise (Fig. 2A). Two types of masking noise were used. In the first, a proportion of the noise dots were plotted at random locations within the display, giving the impression of flickering dots. In the second, the noise dots were immortal and therefore static throughout each presentation.

The signal dots moved globally and cohesively in one of the four cardinal directions and subjects were asked to indicate the direction of global motion (up, down, left, right). Threshold, the proportion of signal dots necessary for 63.3% correct direction discrimination, was determined using an adaptive staircase procedure (Saiviroporoon, 1992) in which the size of the change varied according to how close the subject was to reaching 63% correct. The latter is the percentage reached in four-alternative forced-choice when the titration rule is to increase task difficulty after three consecutive correct judgements and to decrease it after any

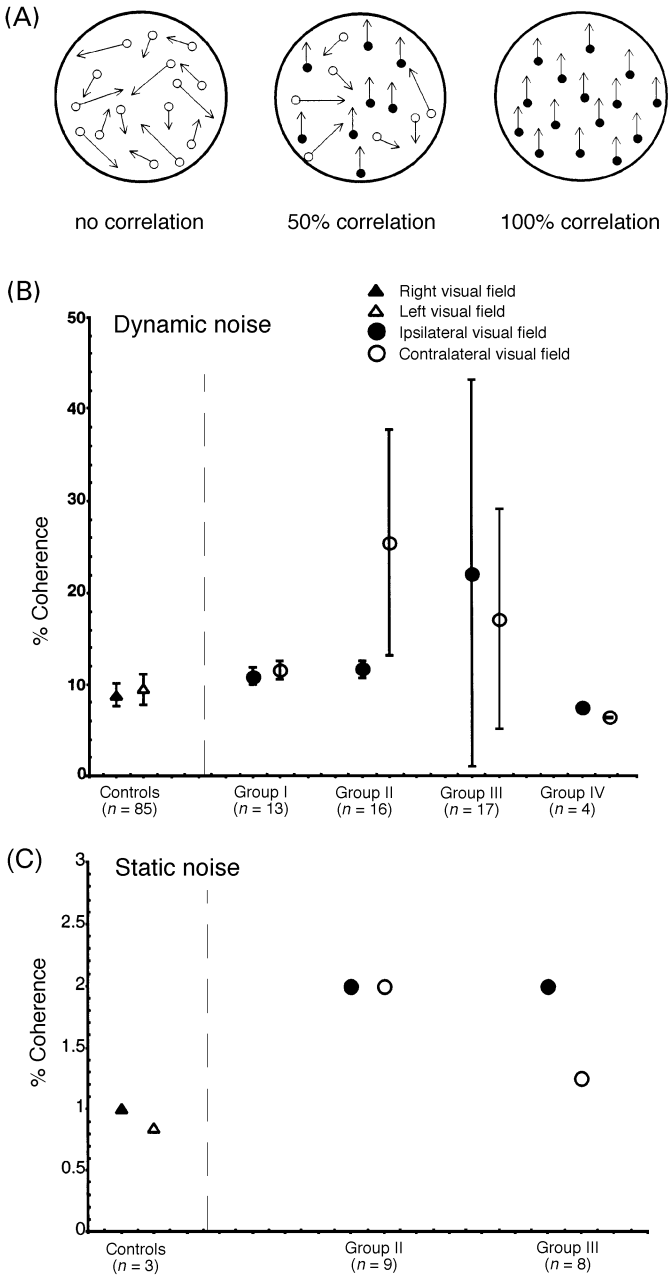

Fig. 2 (**A**) Schematic representation of the motion display; the actual display had many more dots. At 0% there was no coherent motion signal and at 100% all the dots moved in the same direction. On most of the trials the subjects viewed a stimulus that was somewhere between 0 and 100% correlation. The middle sketch shows a stimulus with 50% correlation. (**B**) Proportion of coherent motion for 63.3% correct discrimination threshold in left and right visual hemifields of 85 normal control subjects and in four groups of patients. Threshold was significantly higher in the visual hemifield contralateral to the lesion in Group II. (**C**) Threshold proportion of coherence in the condition where the noise dots were static. There was no significant difference between the thresholds in hemifields ipsilateral and contralateral to the cerebral lesion. There are no standard error bars in **C** because the variance was so small. The actual standard deviations were: normal controls, right visual field 0.28, left visual field 0.56; Group II, ipsilateral field 0.34, contralateral field 0.14; Group III, ipsilateral field 0.37, contralateral field 0.52.

error. For two-alternative forced-choice, as in Experiment 2, the same rule yields 79% correct. The stimuli were presented in each visual hemifield separately to assess processing by the normal and the damaged hemispheres.

Random dot kinematograms (white dots on a black background) were generated by a Macintosh Centris computer and displayed on the Macintosh RGB monitor. The stimuli, displayed in a circular aperture 10° in diameter, were random dot kinematograms with a correlated motion signal of variable strength embedded in motion noise. Each frame was 45 ms in duration with no interframe interval and the stimulus was presented for 22 frames, i.e. 1 s. The percentage of the dots moving in the same direction varied from 100% (total correlation) to 0% (noise). Signal dots were displaced by 8 arcmin in one direction, giving an effective velocity of 2.96°/s. The remaining dots were randomly repositioned within the stimulus aperture. On every trial, for successive displacements the identity of the signal dots was randomly assigned (Newsome and Paré, 1988; Vaina *et al.*, 1990, 1994). Therefore, the impression of coherent movement in some dominant direction had to be derived from a global computation which spatially integrated the local motion measurements, making it almost impossible for the observer to perceive direction by tracking a single dot or a local cluster of dots over several frames, especially at low coherence levels.

Although eye movements were not quantitatively assessed, the subjects' eyes were watched while they fixated a small square located 2° to one or other side of the edge of the display and performed the tasks. When the experimenter started the trial the eyes were watched during the brief period that the display was present. Any discernible eye movement led to the cancellation of the trial. Both saccadic eye movements greater than 2° and slow pursuit movements are easy to detect even though their extent and gain, respectively, would be impossible to know. Fortunately, neither the control subjects nor the patients made frequent discernible eye movements, i.e. they usually followed instructions to maintain fixation.

Results

To determine whether the site of the lesion was consistently related to any impairments we first compared the performance of the various groups on the task of discriminating global motion embedded in dynamic masking noise. For the 85 control subjects, the threshold coherence for 63.3% correct was 8.81 (SD = 1.24) in the right visual field and 9.5 (SD = 1.69) in the left visual field. These differences were not significant, given that the slope of the regression line predicting the results for stimuli presented in the right visual field from the thresholds in the left visual field was 1 (β_1 = 1.0; $P < 0.05$), and the intercept was close to 0 (β_0 = 0.9). This indicates that the computation of motion coherence is not mediated preferentially by one hemisphere or the other, which is important in evaluating the effects of unilateral lesions. The results for the patients are shown in Fig. 2B, where performance is given with respect to the hemifield ipsilateral to the lesion or contralateral to the lesion.

Contingency table analyses were computed to assess any relationship between the side of the lesion (left or right hemisphere) and the sign of visual hemifield difference (left visual field thresholds – right visual field thresholds). In this analysis all the patients were first considered only as left or right damaged. A significant relationship was found between the side of the lesion (left or right) and threshold coherence values (χ^2 = 4.46, $P < 0.03$; Fisher's exact value = 0.07). Thus, on the significance level of 90%, the null hypothesis of independence of lesion side versus performance is rejected. The analysis was then repeated for the 16 patients in Group II, the only group where there was a large difference between performance in the ipsilateral and contralateral hemifields. Not surprisingly the patients' thresholds were higher for stimuli presented in the visual field contralateral to the lesion, as shown in Fig. 2B.

How does the performance of patients in different groups compare?

Post hoc analysis with Duncan's multiple comparison test for pairwise difference showed that all patient groups, except Group I, differed significantly from the normal controls ($P < 0.05$). A between-groups comparison showed that the normal controls and Group I differed significantly from Group II and III for stimuli presented in either visual field ($P < 0.05$). Group IV contained only four subjects, but their data showed that performance on motion coherence was not affected by the lesion. A significant difference between normal control subjects and patients by lesion type (except for Group I and IV) was found even when we included in the analysis only the patients without any visual field loss at any stage [$F(5,115) = 19.47$, $P < 0.0001$]. The performance of the normal controls, patients with occipito-temporal lesions (Group I) and patients with anterior frontal lesions (Group IV) was not statistically different (Group IV performed better than the majority of controls).

Does the result on the MCT depend on field loss?

Since several patients had some visual field loss we assessed whether their performance on the MCT depended on the intactness of the visual field, even though all patients were able to perceive bright moving stimuli throughout the hemifield contralateral to the lesion, especially at the eccentricity of the motion coherence display, whose medial edge was 2 degrees from the visual midline. Their performance on the contrast sensitivity task—both detection and discrimination—was also normal, although this was presented with central fixation.

A lesion group × field loss ANOVA (analysis of variance) revealed that there was no effect of field loss [$F(1,28) < 1$;

$P = 0.785$] on the difference threshold (threshold from left visual field – threshold from right visual field) or on the overall threshold (threshold left visual field + threshold right visual field) [$F(1,28) = 1.103$; $P = 0.303$]. The patients involved in this analysis were from Groups III and IV.

Static noise condition

To assess whether the deficits on the MCT were specific to displays containing dynamic noise, 17 patients from Groups II and III, all impaired on motion coherence in the dynamic noise condition, were also tested on the static noise condition, as shown in Fig. 2C. For the three control subjects, the percentage of dots that had to move coherently for the subject to achieve 63.3% correct in either left or right visual field was 1. In nine patients from Group II it was 2 in each hemifield and in eight patients from Group III it was 1.25 in the contralateral visual field and 2 in the ipsilateral visual field.

Experiment 2: anisotropy of motion sensitivity?

Experiment 2 specifically examined discrimination of centrifugal and centripetal motion in view of evidence for anisotropy (see Discussion). The stimulus display was a modification of the motion coherence stimulus described in Experiment 1. To minimize any possible effect of smooth pursuit the display was on for only 120 ms (four frames, with frame duration 30 ms;) and the display appeared at either 5° or 8° eccentricity from the fixation spot to the nearest edge of the display. Signal dots were displaced laterally by 8 arcmin giving an overall velocity of roughly 4.4°/s. As in Experiment 1, for successive displacements the signal dots were reselected randomly from the entire number of dots without regard to the motion of each dot in the previous frames. In a two-alternative, forced-choice discrimination paradigm subjects had to decide whether global motion was left or right. Using an adaptive staircase, as in Experiment 1, thresholds for motion coherence were measured, but now for leftward and for rightward motion, i.e. either centrifugal or centripetal depending on the hemifield in which the display was presented. Threshold was computed as the mean of the last six reversals in the staircase, tracking 79% correct performance.

A subgroup of 12 patients from Group II (whose damage impinged on area MT/V5) without clinically detected visual field loss, were tested. Ten of these patients were impaired on the previous motion coherence test (Experiment 1) for stimuli presented in the visual hemifield contralateral to their lesion. Two patients, although impaired on other motion tasks, had normal performance on motion coherence.

Results

The patients were first tested in the normal visual hemifield ipsilateral to the lesion, and then in the contralateral field (in which most of them were impaired on Experiment 1). This arrangement should minimize any deficit given that subjects usually improve with practice on a new task. The results are shown in Table 1, which gives the percentage of total errors that were made when the motion signal was moving in the centrifugal direction, i.e. away from the fixation point. In the normal, ipsilateral hemifield, direction anisotropy in favour of centripetal motion (fewer errors) was present in seven patients. Five patients made fewer errors in the centrifugal direction. However, in the hemifield contralateral to the lesion the pattern of errors was different in that all 12 patients made more errors in the centripetal direction, indicating a bias in favour of the centrifugal direction in the impaired hemifield (Table 1). Overall, in the first seven patients the directional bias switched from centripetal to centrifugal in the field contralateral to the lesion and in which their performance on the MCT was impaired in Experiment 1. In the five patients with better discrimination of centrifugal motion in the normal ipsilateral hemifield field the direction of bias was maintained in the impaired contralateral visual field but was reduced in three of them. The results therefore show that lesions to the occipitotemporoparietal area, involving the MT/V5 complex, produce a deficit on the discrimination of centripetal motion in the visual field contralateral to the lesion, no matter what directional bias, if any, the patients showed for stimuli presented in the normal ipsilateral hemifield. This was significant even with the conservative sign test ($n = 12$, $r = 2$, $P = 0.004$, two-tailed where $r =$ the number of successes in the binomial).

Transient or permanent deficits of motion sensitivity?

Several studies report that monkeys in which motion coherence in the corresponding region of the retina is impaired by neurotoxic lesions within area MT, improve or even completely recover their previous perceptual abilities within a few days after the lesion (Newsome *et al*., 1985; Newsome and Paré, 1988; Yamasaki, and Wurtz, 1991). On the other hand, Marcar and Cowey reported that complete surgical removal of MT apparently permanently impaired the animals' ability to perform a direction discrimination task; threshold of coherence for direction discrimination was impaired even 1 year after the surgery (Marcar and Cowey, 1992). Similarly, Pasternak and Merigan reported that large bilateral MT and MST lesions result in permanent deficits in direction discrimination in 'noisy' global motion stimuli, but no lasting impairments in contrast sensitivity (Pasternak and Merigan, 1994). More recently, Rudolf and Pasternak found that, although complete unilateral destruction of areas MT and MST in the macaque produce enduring deficits in direction discrimination in noisy global motion stimuli, repeated training was accompanied by an improvement in coherence thresholds that was attributed to the training rather than to other factors (Rudolf and Pasternak, 1999).

Table 1 *Percentage of total errors made to global motion in the centifugal direction in the normal (ipsilateral) and impaired (contralateral) visual hemifields of patients in Group II, with unilateral cortical damage that included area MT/V5*

Subject	Age (years)	Sex	Lesion	% centrifugal errors	
				Ipsi	Contra
1	53	F	L. posterior parietal	67	44
2	41	M	L. posterior parietotemporal	62	32
3	62	M	R. posterior parietotemporal	72	42
4	47	F	R. parietotemporal	65	32
5	62	M	R. parietotemporal	62	40
6	47	F	R. parietotemporal	59	40
7	63	M	L. occipital posterior medial parietal and temporal	52	35
8	55	M	R. parietoanterior–temporal	47	40
9	29	F	R. occipitoparietal	35	40
10	57	M	R. occipitoparietal	42	43
11	57	M	R. posterior temporal parietal and occipital	40	35
12	53	M	R. temporal–parietal	40	30

F = female; M = male; L. = left; R. = right; Ipsi = ipsilateral; Contra = contralteral.

We were interested to learn whether training on the MCT leads to specific improvement of performance, and whether the improvement was causally linked to training or to its specificity. Therefore, we studied a subset of 14 patients from Group II and measured their threshold on the MCT at ~1 week after the lesion and then again 5 weeks later. The patients were selected because they were initially impaired on the MCT and because they were able and willing to participate in further extended testing. Six of these patients (Group A) participated in 45 min weekly training sessions with the MCT only. Five patients (Group B) did not participate in any training in the intervening weeks between the two threshold measurements. Three patients (Group C) participated in weekly sessions of motion assessments with local motion tasks of speed and direction discrimination. They were given the MCT only on weeks 1 and 5.

Table 2 shows the changes in initial and final threshold and the Z scores for each patient in the three subgroups. Patients in Groups A and C significantly improved their performance on the MCT whether they were trained with MCT or other motion tasks. The improvement occurred in both hemifields. Of the nine patients involved in motion training (Groups A and C), six were initially impaired (threshold of motion coherence necessary for reliable performance was > 2 SD away from the mean threshold of a large number of normal control subjects). Subsequent to training, either specifically with the MCT or generally with other motion tasks, four of these six patients recovered normal performance for stimuli presented in the visual field ipsilateral to the lesion. Performance for stimuli presented in the contralateral visual field, was initially impaired (compared with the normal observers) in all nine patients in Groups A and C. However, after training, six patients were not significantly different from the normal observers on the MCT ($Z < 2$). On the other hand, the five patients in Group B who did not undergo any training in the intervening period between the two performance assessments on MCT improved less. Two of these patients (Subjects 7 and 10), however, initially could do the task only when the coherence level was very high in both visual fields. At first they were significantly more impaired on this task than any of the patients in Groups A and C. Two patients were normal in the ipsilateral field, and only one patient had an initial performance comparable to that of the impaired subjects from the other two groups. This patient (Subject 8) significantly improved for stimuli shown in his ipsilateral visual field, but not in the contralateral field.

Discussion

We studied motion sensitivity of a group of 50 patients with unilateral lesions resulting from a single stroke using a psychophysical task of direction discrimination in stochastic dynamic random dot displays, where a variable proportion of the dots created a single directional motion signal and the remainder provided masking motion noise. No deficits on this task were found in patients with lesions along the ventral route involving the caudal temporal area (Group I). In striking contrast, global motion discrimination in the condition of dynamic noise was severely affected by lesions (in either hemisphere) along the dorsal route (Group II), which presumably involved the human homologue of areas MT/V5 and MST. However, these patients' performance was normal when the masking noise was static (Fig. 2C), indicating that the deficit with dynamic noise reflects an inability to filter out dynamic noise, as in our previously reported case A.F. (Vaina *et al*., 1990). The present patients may therefore be different from the motion-blind patient L.M. Her much larger lesion left her severely impaired when viewing random dot kinematograms with static noise (Baker *et al.*, 1991) or

Table 2 *Thresholds and* Z *scores illustrating performance on the MCT at 1 month apart in three subgroups of patients selected from Group II*

Subject	Age (years)	Sex	Lesion	Ipsilateral threshold				Contralateral threshold			
				t_1	*Z*	t_2	*Z*	t_1	*Z*	t_2	*Z*
Group A											
1	41	M	L. posterior parieto-temporal (small)	12.07	0.39	8.12	–0.64	21.75	2.92	9.64	–0.24
2	57	F	R. parietooccipital	24.41	3.62	9.10	–0.38	28.56	4.70	17.03	1.69
3	59	M	R. posterior parieto-temporal, anterior temporal	26.68	4.21	14.60	1.05	34.24	6.18	17.97	1.93
4	39	F	R. posterior parieto-temporal	44.04	8.74	22.16	3.03	75.98	17.08	25.07	3.79
5	62	M	L. occipitoparietal	42.35	8.30	36.78	6.85	47.40	9.62	32.86	5.82
6	63	M	R. occipital posterior parietal	19.82	2.42	15.44	1.27	20.08	2.49	20.57	2.61
Group B											
7	53	M	R. parietotemporal	89.13	20.51	85.79	19.64	83.29	18.99	73.57	16.45
8	59	M	R. posterior parietal	33.62	6.02	21.29	2.80	32.20	5.65	31.81	5.55
9	51	M	R. posterior parieto-temporal, anterior temporal	9.63	–0.24	8.06	–0.65	24.70	3.69	19.07	2.22
10	51	F	R. parietotemporal	52.88	11.05	50.36	10.39	64.36	14.05	58.17	12.43
11	51	F	R. occipitoposterior parietal	7.50	–0.80	7.60	–0.77	15.46	1.28	9.39	–0.31
Group C											
12	63	F	L. occipital	15.36	1.25	7.47	–0.81	26.66	4.20	14.07	0.92
13	53	M	R. posterior parieto-temporal	11.70	0.30	10.00	–2.76	36.41	6.75	13.08	0.66
14	49	F	R. parietal	34.39	6.22	12.49	0.50	67.34	14.83	12.56	0.52

The data are shown for stimuli presented in the visual field ipsilateral to the lesion and the visual field contralateral to the lesion. Group A: six patients who underwent weekly training sessions with the MCT. Group B: five patients who were not tested in the intervening time interval (1 month) between the first threshold and the second. Group C: three patients who underwent weekly training sessions with motion tasks other than MCT, especially local direction, speed and discrimination, and discrimination of 2D form defined by motion cues. F = female; M = male; t_1 = time 1; t_2 = time 2; L = left; R = right.

when viewing the biological figures of Johansson against a background of static noise (McCleod *et al.*, 1996).

Although the deficits were long lasting—and perhaps permanent—in the sense that they were present months after the cortical damage in those patients tested repeatedly, it is possible that they were even more severe at an earlier stage. Sub-total neurotoxic lesions in area MT of macaque monkeys characteristically produce prominent immediate effects on a similar task of discriminating the direction of global motion in random dot kinetograms, followed by total recovery within 2 weeks or so (Newsome *et al*., 1985; Yamasaki *et al*., 1991). But when area MT is totally destroyed the effects last at least for months, and in some cases are permanent (Cowey and Marcar, 1992; Marcar and Cowey, 1992), but in no investigation of which we are aware has direction discrimination been completely abolished. In this sense the results of studies on macaque monkeys and those reported here are similar. It is not clear why the deficits are partial but the simplest explanation is that the multiple cortical areas in which motion is analysed (Dupont *et al*., 1994; Gulyas *et al.*, 1994; Sunaert *et al*., 1999; Vaina *et al*., 2000) show overlap and functional redundancy and, in addition, that the initial cortical damage is almost always more extensive than the final damage. Presumably the rare patients who lose almost all ability to discriminate motion, such as patient L.M. described by Zihl *et al.* (Zihl *et al*., 1983, 1991), do so because the large lesion encompasses so many of the extrastriate visual areas in the dorsal stream, or disables them by damaging white matter.

Bilateral deficits

A large subset of patients in Group III, with the most dorsolateral and rostral temporal lesions, were impaired when displays were presented in either visual field, independently of whether the visual hemifields were perimetrically intact. A possible explanation for this bilateral deficit is that beyond MT, which is still retinotopically mapped, receptive field sizes become larger and extensively straddle the vertical meridian. Even further in the anterior temporal or parietal lobes, they can encompass almost the entire visual field (Gross *et al.*, 1985). Anterior frontal lesions, some of which almost certainly involved the frontal eye fields in Brodmann area 8, had no effect on performance in this task.

Is there a directional bias in sensitivity for detecting direction in global motion?

Raymond reported the first clear evidence in normal observers of directional anisotropy (centripetal superiority) of direction discrimination in global motion stimuli (similar to the stimulus described in Experiment 1) presented at eccentricities of 5 degrees or more (Raymond, 1994). Edwards and Badcock also reported a centripetal bias for sensitivity of motion in depth (Edwards and Badcock, 1993). Initiation of smooth pursuit was also found to be faster for centripetal motion than for centrifugal motion, varying systematically with the eccentricity of the stimulus (being smallest in the fovea). These reports are interesting since the major cortical areas involved in both smooth pursuit and perception of motion coherence are believed to be MT/V5 and MST (Newsome *et al*., 1985, 1986; Dürsteler and Wurtz, 1988; Dürsteler *et al*., 1987). However, when Ball and Sekuler examined the response latencies to motion onset in a dynamic random dot field they found that latencies were shorter for motion away from the fovea than towards the fovea, suggesting a centrifugal bias for motion sensitivity (Ball and Sekuler, 1980). Support for greater sensitivity to centrifugal flow fields also comes from electrophysiological studies of neurones in MT in the macaque monkey (Albright, 1989), where centrifugal bias increased with eccentricity (maximum between 12 and 30 degrees). Unfortunately, the present study of anisotropy of sensitivity for global motion stimuli presented at an eccentricity of 5 degrees was not totally conclusive, although it indicates a greater impairment of the perception of centripetal motion, as did the study by Barton *et al*., in which five patients had directional defects even with central fixation of the display (Barton *et al*., 1995). It is conceivable that the anisotropy could be accounted for by a deficit in smooth pursuit (which we minimized by requiring and monitoring fixation but might not have eliminated) since all lesions involved, directly or indirectly, the underlying white matter, and the parietal–occipital–temporal junction is an important structure in the cortical control of smooth pursuit (Newsome *et al*., 1985; Dürsteler *et al*., 1987; Dürsteler and Wurtz, 1988; Barton *et al.*, 1996). Clinically, there is a system of ipsilateral control: in the right hemisphere the parietal–occipital–temporal junction controls smooth pursuit to the right, and in the left hemisphere controls smooth pursuit to the left. We are now conducting a study in which stimuli are presented extrafoveally at even larger eccentricities and in larger angular displays.

Effects of training

In a sub-group of patients from Group II we explored whether training significantly influences functional recovery. Several studies over the last 10 years report that after circumscribed cortical lesions the adult brain has the capacity to recover language and motor functions with training. Most of the studies focus on the early segments of the motor systems, especially the primary motor area M1. More recently, restorative plasticity was reported from functional neuroimaging studies even in higher brain areas, and it was demonstrated that recovery from aphasia (Weiller *et al.*, 1995; Thulborn *et al*., 1999), especially with training (Musso *et al.*, 1999), or auditory agnosia after left hemisphere stroke (Engelien *et al.*, 1995) involved the right hemisphere in language processing. Similarly, in recovery of motor function after unilateral stroke, functional recovery is linked to activations of homologous areas of the contralateral hemisphere. Thus it has been suggested that the possible bilateral representation of functions may provide an insight into how recovery occurs (Chollet *et al.*, 1991; Frackowiak, 1997). In this study we found significant improvement of performance with training with motion stimuli in both the intact and the damaged hemisphere. Because it is likely that the MCT is mediated by neurones in the MT complex in either hemisphere, it is possible to interpret this dual improvement in different ways. Improvement which occurred for stimuli presented to the intact contralateral hemisphere could be mediated by perceptual learning, as shown in fMRI studies of normal observers (Vaina *et al*., 1998*c*). The question arises as to what underlies improvement when the MCT is mediated by neuronal circuitry in the lesioned hemisphere? Is it recruitment of neurones in the perilesional region, or are other motion responsive areas taking over the function? This question can only be answered by investigating recovery of motion deficits after stroke.

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