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SACCADIC DYSMETRIA IN A PATIENT WITH A RIGHT FRONTOPARIETAL LESION

THE IMPORTANCE OF COROLLARY DISCHARGE FOR ACCURATE SPATIAL BEHAVIOUR

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SUMMARY

Double-step experiments have demonstrated that retinotopic coding is inadequate to explain the spatial performance of the saccadic system. In such experiments a subject is asked to make two successive saccades to fixate two sequentially flashed targets each of which disappears before the first saccade. Despite the dissonance thus created between the retinal location of the second target and the saccade necessary to acquire it, normal humans and monkeys perform the task perfectly well. Single unit recording in monkeys indicates that neurons in the superior colliculus, frontal eye fields and in parietal cortex generate a spatially accurate signal during the performance of double-step saccades, which is thought to be obtained by combining a retinotopic signal with a signal corollary to the previous saccadic eye movement.

We studied saccadic eye movements in a patient with a right fronto-parietal lesion using single- and double-step tasks. Single saccades into the left (contralesional) hemifield had longer latency and were hypometric relative to those into the right (ipsilesional) hemifield. Varying the initial orbital position had no effect on the latency and accuracy of saccades to left and right retinal stimuli. When the patient was asked to do a double-step task with targets flashed first into the right field and then into the left field, she performed well. When she was asked to do the same task with a target flashed first into the left field and then into the right field she made the first saccade correctly but never acquired the second target, even though this required her to make a saccade in the normal direction to a stimulus that appeared in the normal field. Such a deficit therefore cannot be one of retinotopic or spatial coding, nor can it be one of generating a certain direction of saccade. We suggest that the deficit is a failure of corollary discharge, the inability to register the amplitude and direction of a saccade into the contralesional field, and use that information to update the representation of the location of the next saccade target.

INTRODUCTION

One of the basic tasks that the brain performs is to coordinate information about the environment and information about the internal state of the organism. In the visual-oculomotor system, saccadic eye movements have long been recognized as a useful tool to study the interactions between retinal and extra-retinal signals in different brain regions (Goldberg and Wurtz, 1972; Bushnell *et al.*, 1981; Fischer and Boch, 1981). The question

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of the role of extra-retinal signals in eye movements addresses directly the broader issues of reference frames and spatial representation.

From a behavioural standpoint, it has been established that the processing taking place during saccadic eye movements requires both visual and non-visual signals. In a now classic experiment, Hallett and Lightstone (1976) devised an oculomotor task, the double-step paradigm, in which two consecutive saccades were made in response to two sequentially flashed visual targets (Fig. 1). Because the duration of the stimuli was very brief, the saccades were initiated and executed in darkness. Normal human subjects can perform this task quite accurately. This finding refutes the notion that only retinal information is available to the oculomotor system in planning an eye movement: since the second saccade does not start at the spatial location from which the second visual target was seen, the double-step task introduces a spatial dissonance between the retinal coordinates of the target and the motor coordinates of the required eye movement. In order to compensate for this and to compute an accurate saccade, non-retinal information about the displacement of the eye since the target was seen must therefore be used.

How this computation is accomplished remains a subject of debate. One position states that retinal and eye position data are added to produce a supra-retinal craniotopic representation of visual space (Mays and Sparks, 1980; Zipser and Andersen, 1988;

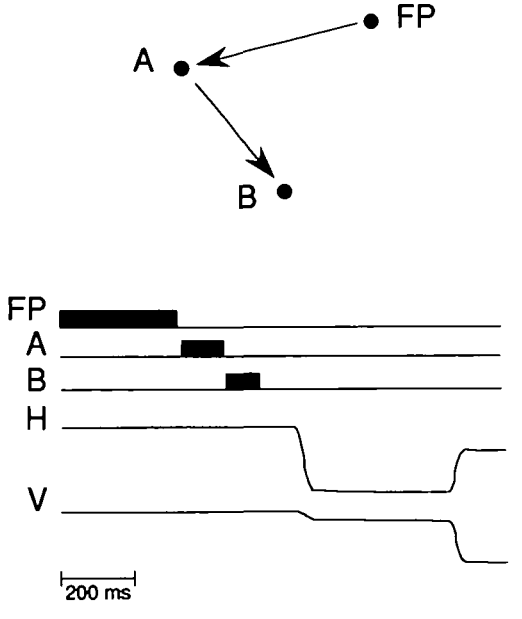

FIG. 1. Double-step saccade experiment. FP = fixation point; A and B, first and second saccade targets; H and V, horizontal and vertical eye position. The top part of the figure shows the location of the targets and the sequence of saccades. The bottom part shows the timing of visual and oculomotor events (time runs from left to right). Note that the targets were extinguished before any eye movement took place. This created a dissonance between the retinal coordinates of the stimulus at B and the motor coordinates of the saccade to it: B was flashed in the left visual field but had to be acquired with a rightward eye movement.

Schlag-Rey *et al.*, 1989). In principle, such a representation could be used in the double-step case to compare the current position of the eye and that of the stimulus with respect to a common cranial reference. An alternative view suggests that locations in space are encoded relative to the centre of gaze (Goldberg and Bruce, 1990; Goldberg *et al.*, 1990; Duhamel *et al.*, 1992). To account for accurate double-step performance it proposes that the coordinates of the second saccade are obtained by subtracting the direction and amplitude of the first eye movement from the original retinal coordinates of the second target. Rather than an eye position signal, this form of coordinate transformation uses an efferent copy of the saccade as extra-retinal input.

Studies of eye movements in monkeys and humans with lesions in frontal or in parietal cortex have revealed anomalies in preparing and executing saccades toward the visual field contralateral to the lesion (Guitton *et al.*, 1982; Pierrot-Deseilligny *et al.*, 1987; Lynch and McLaren, 1989). It is not clear, however, whether these visuo-motor impairments are purely retinotopic, affecting eye movements in response to the stimulation of a specific part of the retina, or whether they also affect saccades made into a certain part of space or in a certain direction. This can only be established by experimentally dissociating the retinal coordinates of a stimulus from its spatial coordinates and from the movement coordinates of the saccade necessary to acquire it.

We recently had the opportunity to examine the eye movements of a patient with a long-standing fronto-parietal lesion in the right cerebral hemisphere, using the magnetic search coil technique. Single saccades made to targets presented in the contralesional visual hemifield were found to have increased latency and reduced amplitude relative to saccades made towards the opposite visual hemifield. In order to address the issues described above, we further studied the patient's eye movements in specially designed tasks. Three major questions were asked:

(i) Is the oculomotor impairment retinotopic or spatial? This question was addressed using a single-step paradigm in which the patient made visually guided saccades from different initial orbital position. This allowed us to study eye movements to targets whose eccentricity in head-centred space varied while their retinal eccentricity remained constant.

(ii) Is the impairment retinotopic or directional? This question was addressed using a double-step task in which the spatial arrangement of the stimuli was such that both appeared in the normal, ipsilesional visual hemifield but required that the second saccade be made in the contralesional direction. This task was designed to distinguish between an impairment in using visual information from a particular portion of the retina and one in making eye movements in a particular direction.

(iii) Is there an impairment in the ability to use extra-retinal information for the purpose of calculating the dimensions of a saccadic eye movement? This question was addressed using a double-step paradigm in which one stimulus appeared briefly in each visual hemifield. The patient's task was to saccade to the stimuli in the order that they appeared, left to right or right to left. This is a situation in which the dimensions of the second eye movement are determined jointly by the location of the stimuli (identical in both conditions) and by the direction and amplitude of the first eye movement (which varies with target order). This task therefore allowed us to compare, under identical retinal conditions, the use of extra-retinal information associated with eye movement made in the ipsi- and contralesional directions.

METHODS

Case report

The patient, E.C., is a 66-yr-old right-handed female who, in 1977, suffered a rupture of aneurysm in the territory of the right internal carotid artery which was subsequently clipped. When seen as an outpatient in 1990 at the National Institutes of Health for participation in a comprehensive neuropsychological study, E.C. presented residual contralateral hemiparesis. Visual fields measured with both static and dynamic perimetry showed the presence of a left hemianopia which spared vision in about 10° on the horizontal meridian and in the lower quadrant and to 20° or more in the upper quadrant (Fig. 2). Vision was normal

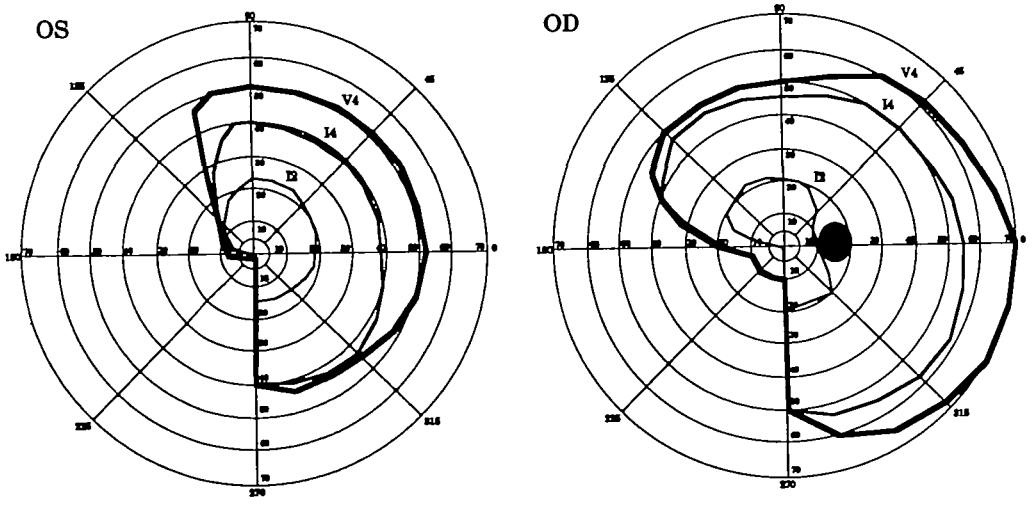

FIG. 2. Monocular visual fields of patient E.C. showing left homonymous hemianopia predominating in the lower quadrant and sparing the central 10° on the horizontal meridian. OS = left eye; OD = right eye.

in the right homonymous hemifields. Residual visuo-spatial impairments were still present, including moderate left visual neglect as established in spontaneous drawing and copying tasks. In a line bisection test she had a mean rightward bias of 18% (eight trials, lines were 15 cm long). A computerized tomography (CT) scan performed in October 1990 (Fig. 3) shows the presence of a large lesion in the right cerebral hemisphere which extends from the cortex to the wall of the right lateral ventricle. The cortical lesion involves the frontal and parietal lobes and encompasses the right frontal eye field and the region of the right intra-parietal sulcus.

Visual stimulation and eye movement recording procedures

The patient sat in a chair in a dimly illuminated room, her head resting against a chin and forehead support. For all tasks the stimulus was a small white spot of light (0.1°; >2 log units above threshold luminance) reflected from a pair of mirror galvanometers and projected on the back of a translucent tangent screen. The mirrors were used to control the vertical and horizontal position of the stimulus. The stimulus was turned on and off by means of a shutter mounted on a rotary solenoid. Eye position was recorded with the scleral search coil technique (Robinson, 1963), using eye coils made by Skalar and 6 ft field coils (CNC Engineering, USA). The system had a resolution, after analogue to digital conversion, of about 1/20°. At the beginning of each recording session the coil was placed in the left eye following a drop of topical anaesthetic (proparacaine hydrochloride) and remained in place for about 25 min. Before collecting

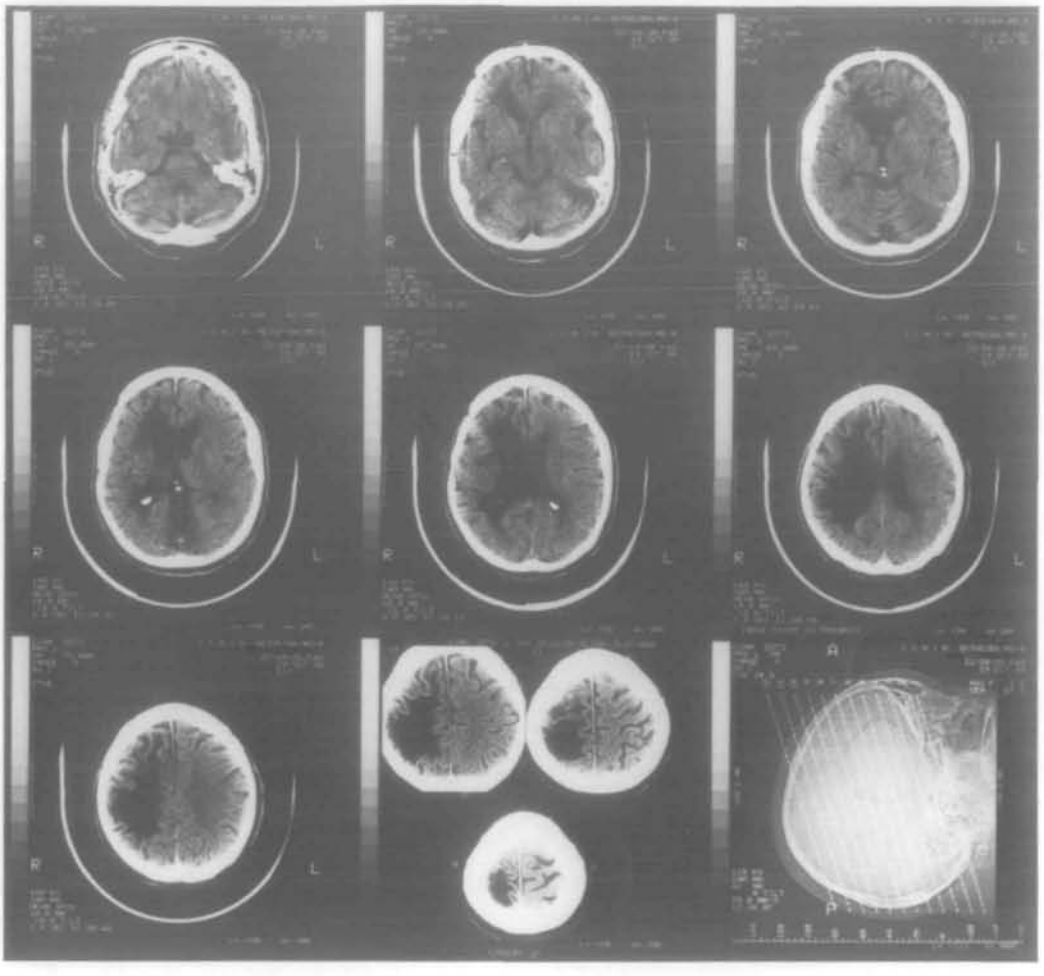

FIG. 3. Patient's CT scan showing large right hemispheric lesions (details in text).

data, the signals from the coil were calibrated by having the subject fixate spots of light located at the centre of the screen and 10° right, left, above and below this point. Experiments were carried out under computer control (PDP 11/73). Horizontal and vertical eye position and target position were sampled at either 250 Hz or 1000 Hz. The computer monitored fixation, determined the order in which trials were presented, controlled the position of the target, digitized analogue signals and stored the data.

Oculomotor tasks

Visually guided saccades. The subject fixated the stimulus located in the centre of the screen. After an unpredictable delay, the spot of light disappeared and reappeared pseudo-randomly at one of 12 locations on the horizontal meridian (2°, 4°, 6°, 8° in the left visual field; 2°, 4°, 6°, 8°, 10°, 12°, 14° in the right visual field). The subject's task was simply to make a saccade to the stimulus and maintain fixation at the new location until the end of the trial. Each target location was tested 8–10 times. The main purpose of this task was to measure the latency, amplitude and velocity of single saccades to targets in the left and right visual fields.

Visually guided saccades from multiple initial positions. The block of trials began with the subject fixating a target at the centre of the screen. At an unpredictable time after the onset of the fixation target, one of two targets located 5° to the left or to the right would appear and the subject would saccade to it. This target would provide the fixation point for the next trial, and the next target would appear pseudo-randomly 5° to the left or to the right. Therefore, although all saccades were of the same required amplitude and only differed in direction, their goal in head-centred space varied systematically as a function of initial eye position. Initial position ranged, in 5° steps, from 20° to the left to 20° to the right of primary position (which coincided with the centre of the screen). Eleven trials were collected for each direction of saccades from each initial eye position.

Double-step saccades. After an unpredictable delay from the onset of the fixation period, the central fixation stimulus disappeared and the first saccade target was flashed for 100 ms. This flash was immediately followed by a second saccade target which was flashed for 80 ms. The patient's task was to shift rapidly her gaze from the fixation to the first target location and from there to the second target location. Since the latency of the first saccade was longer than the total duration of the two stimuli, both saccades were made while the screen was blank. These two saccades can therefore be said to be visually triggered, but they are not visually guided. In this paradigm visual information about the second target is sampled prior to the beginning of the first eye movement. Unequal stimulus durations were used because previous experience with this paradigm has shown that this reduces the probability that a subject will make the saccades in the reverse order (from the fixation point to second target to first target) or make a single direct saccade to the second target.

Four different pairs of double-step target combinations were used. Two of the pairs consisted of stimuli appearing in the same visual hemifield and the two other pairs consisted of one stimulus in each visual hemifield. Further details about the location of the stimuli are given in Results. Each stimulus combination was presented 12 times in a series of randomly intermingled trials. A slowed-down version was also used as a control task for some of the target combinations, with each stimulus remaining on the screen for 500 ms. Since the latency of the first saccade was invariably less than 500 ms, this amounted to a sequence of two visually guided saccades. The spatial aspects of the slow and fast trials are identical, but the visual information available to the oculomotor system is different since in the slower case the second stimulus appears at the end of the first saccade.

Data analysis

Data analysis was performed off-line. Trials were examined for blinks and other disruptive artefacts, and for first-saccade latencies shorter than 180 ms in the double-step experiments; such trials were excluded. The computer differentiated each eye position record and used the resulting velocity record to search for saccades. The computer identified the time of onset and end of each saccade, and measured the eye and target positions at these times. Most saccades were horizontal but in the double-step experiments some saccades had a vertical component. The analysis program detected the beginning and end of the vertical component of the (oblique) saccade, and then measured the horizontal component during this period. We assured ourselves, by visual inspection, that the horizontal component of the saccade occurred within the timing of the vertical component, so that our measure of the horizontal component was indeed accurate. Data reported in the tables are only for the horizontal components of those saccades.

RESULTS

Visually guided saccades: effect of retinal location

Sample eye movement traces for all stimulus eccentricities are shown in Fig. 4 and averaged data for each set of trials are presented in Table 1. For both leftward and rightward saccades, large eye movements were associated with longer latencies than small eye movements. However, saccades to stimuli in the contralesional visual field had markedly increased initiation latencies relative to symmetrical locations in the ipsilesional visual field. The mean latency difference between leftward and rightward eye movements is about 78 ms. The left-right differences are significant for all symmetrical target pairs ($P < 0.01$ or less). Data reported elsewhere on the same patient

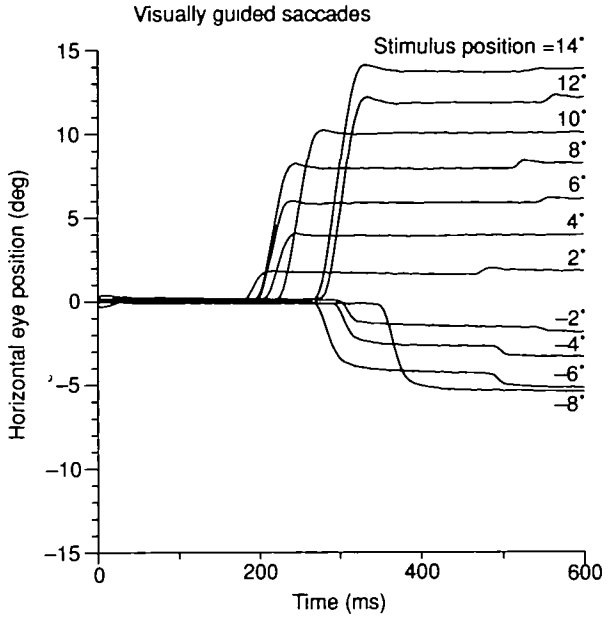

FIG. 4. Sample horizontal eye traces for saccades to left (−) and right (+) visual field targets. The saccade target remained visible throughout the trial.

TABLE 1 AMPLITUDE AND LATENCY OF VISUALLY GUIDED SACCADES TO RIGHT (+) AND LEFT (−) VISUAL FIELD STIMULI

Stimulus location (degrees)	*Latency (ms) mean (SD)*	*Amplitude (degrees) mean (SD)*	*Percentage undershoot*
14	362 (117)	13.5 (0.6)	3
12	300 (82)	11.5 (0.4)	4
10	242 (50)	9.7 (0.5)	3
8	242 (72)	7.6 (0.6)	5
6	216 (21)	5.7 (0.3)	5
4	219 (16)	4.0 (0.3)	0
2	213 (28)	1.8 (0.1)	10
−2	305 (13)	−1.6 (0.5)	20
−4	303 (16)	−2.3 (0.3)	42
−6	289 (8)	−4.0 (0.4)	32
−8	363	−4.8	40

indicate that the increased saccade latencies may in part be due to attentional factors (Duhamel *et al.*, 1990) though other variables such as increased delays in visuo-spatial analysis and/or response programming could play a role as well. There was no convincing evidence for blindsight. The patient made saccades into the seeing field, but almost never beyond the border of the scotoma. She did make one extremely hypometric saccade to a target 8° into the contralesional field, which is reported in Table 1 without a standard deviation.

Saccades to contralesional stimuli undershot their target by an average of 34% of the required movement amplitude. Thus undershoot ranged from 20% to 42% depending on target eccentricity but the amount of undershoot was not related in any systematic way to saccade amplitude. Saccades to ipsilesional targets had near perfect accuracy, with a mean undershoot of only 4% with a range between 0% and 10%, again without any clear relationship between amount of undershoot and saccade amplitude. The left-right differences were significant for all symmetrical target pairs ($P < 0.001$) except at 2°, by Student's t test. The amplitude/velocity profile of eye movements was similar for eye movements in the two visual hemifields (Fig. 5), indicating that the dysmetria of leftward saccades was not associated with abnormal saccade dynamics (Becker, 1989).

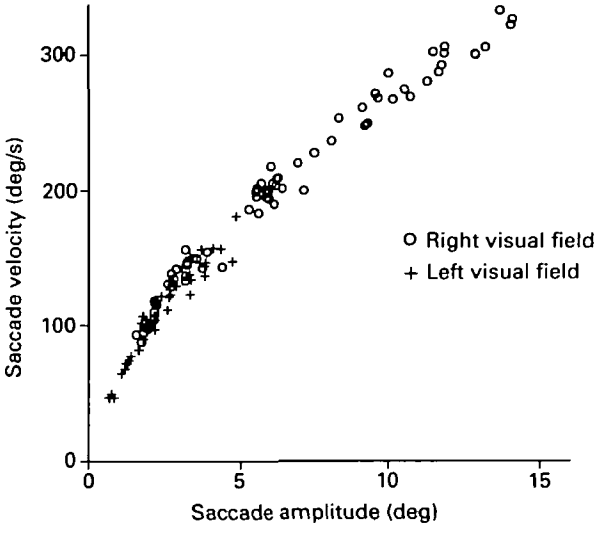

FIG. 5. Velocity of eye movements as a function of their amplitude for visually guided saccades.

Visually guided saccades: effects of orbital position

In order to determine the influence of the craniotopic spatial location of a visual stimulus on eye movements, the initial orbital position of the fixation stimulus was varied from 20° left to 20° right of primary position. The saccade targets were always presented at one of two possible retinal locations: 5° to the left or to the right of the fixation point. Analysis of variance revealed a strong effect of the stimulus' retinal location on both saccade latency and amplitude but there was no significant effect of angle of gaze (Fig. 6). Saccades had longer latencies and shorter amplitudes for left than right visual field targets (both $P < 0.0001$) at all spatial locations, consistent with the results reported above. An unexpected influence of orbital position on saccade velocity was found, however, in an interaction between angle of gaze and saccade direction ($P < 0.005$). The velocity of leftward saccades was consistently slower than that of rightward saccades as shown in Fig. 6, in keeping with their smaller amplitude. In addition, for leftward saccades

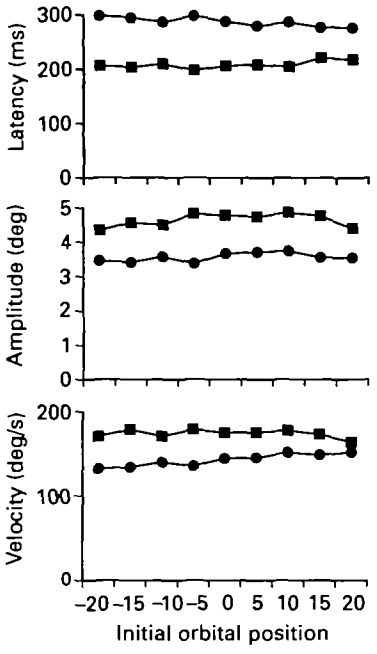

FIG. 6. Latency, amplitude and velocity of saccades to left (●) and right (■) 5° horizontal targets made from initial orbital positions ranging from 20° to the left to 20° to the right of primary position.

only, *a posteriori* contrasts revealed a linear trend toward a decrease in velocity as the fixation target moved from the right to the left of the orbit. However, this effect was modest, as a regression analysis showed that orbital position accounted for only 19% of the total variance in the velocity of these saccades.

In conclusion, it appears that saccades to left visual field targets were harder to initiate and less accurate than saccades to right visual field targets and mostly independent of orbital position.

Double-step saccades

Two stimuli in the same visual field: effect of stimulus location. These pairs of double-step stimuli were used to determine whether the inaccuracy of E.C.'s saccades was related to the retinal location of the targets (left versus right visual hemifield) or to the direction of the eye movements (leftward versus rightward saccades). Two stimuli were presented in rapid succession in the same hemifield, and the patient was required to saccade first to the most eccentric stimulus, and then back to the most central stimulus. For example, as illustrated in the top part of Fig. 7, sequence B, a stimulus was flashed for 100 ms at 6° right and 1.5° down and then for 80 ms at 2° right and 1.5° down. The patient had to execute a rightward saccade to the first stimulus location, and then a leftward saccade to the second stimulus location. The critical aspect of this experiment is the timing of the visual events: both stimuli went on and off before the beginning of the first saccade. This manipulation allowed us to measure an eye movement in the

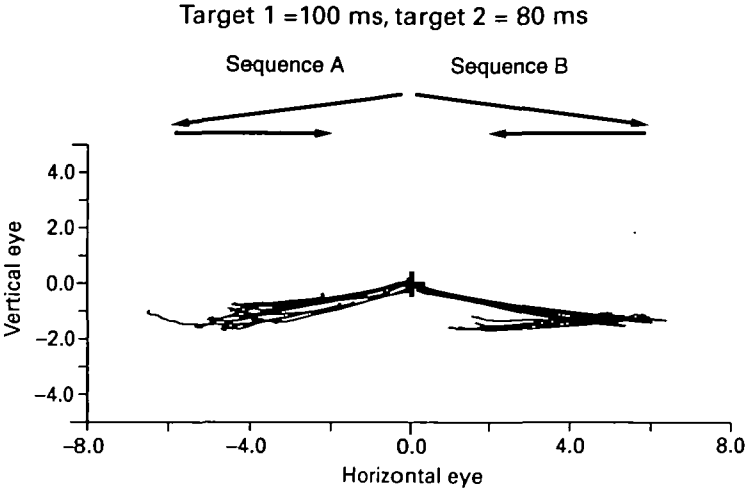

FIG. 7. Superimposed *x-y* plots of double-step saccades to a pair of left visual field stimuli and to a pair of right visual field stimuli. Stimuli were extinguished prior to the beginning of the first saccade. Arrows above the eye movement traces illustrate the two combinations of target steps.

contraversive direction with respect to the lesional hemisphere, but in response to a visual stimulus presented in the normal hemifield. As a control condition, the reverse pattern (sequence A), a rightward saccade to a left visual field target, was also tested by flashing the stimuli at the symmetrical locations to the left of the fovea. If the unilateral impairment observed in visually guided single-step saccades is determined by visual factors alone, saccades to left visual field targets (A) in the double-step task should be inaccurate, and saccades to right visual field targets (B) should be accurate, regardless of their direction. If the saccadic impairment is directional in nature, all leftward saccades should be inaccurate, including those made in the right visual field.

The results show that in the right visual field (ipsilesional, condition B), the first saccades had normal latency and accuracy, while in the left visual field (contralesional, condition A), the first saccades had increased latency and reduced amplitude, respectively $P < 0.01$ and $P < 0.02$ (Fig. 7, Table 2). This was expected from our earlier observations, since the first saccade in this task is analogous to a direct single step saccade, where the required movement amplitude matches exactly the retinal distance of the target

TABLE 2. SACCADE AMPLITUDE AND LATENCY OF E.C. IN THE DOUBLE-STEP EXPERIMENTS WITH TWO STIMULI IN THE SAME VISUAL HEMIFIELD

Step order	*Right* ⟶ *Left*		*Left* ⟶ *Right*	
Step duration	100	80	100	80
Stimulus eccentricity	6	2	−6	−2
Required saccade amplitude	6	−4	−6	4
Actual saccade amplitude (degrees)	5.3 (0.4)	−3.5 (0.9)	−3.8 (1.1)	0.9 (3.0)
Saccade latency (ms)	199 (20)	144 (30)	299 (36)	426 (153)

Standard deviations are given in brackets.

from the fovea. More critical is the second saccade of the double-step pair. Interestingly, leftward saccades to the 2° right visual field target (B) were initiated with a very short latency and were quite accurate. In contrast, rightward saccades to the left visual target (A) had long latencies, or were not executed at all, and never reached the target. The accuracy of leftward saccades made in response to a right visual field target thus rules out the hypothesis of a directional saccadic impairment and appears to be consistent with a retinotopic impairment. The marked inaccuracy of rightward movements in the left visual field condition (A), however, requires further considerations that are addressed below.

One stimulus in each visual hemifield: effect of saccade direction. The above results confirm the contribution of stimulus location to the performance of double-step saccades. As described in the Introduction, in this paradigm, while the retinal location of the first target uniquely determines the dimensions of the first saccade, the retinal vector of the second stimulus and the dimensions of the second saccade are not equivalent. Accurate coding of the latter requires a compensation for the displacement of the eyes associated with the first saccade. As suggested previously, this is only possible if there exists a stabilized representation of the stimulus with respect to the head, or if the visual system can update the gaze-centred representation of the location where the stimulus was flashed in conjunction with the first eye movement. Either solution necessitates a computation that combines the retinal coordinates of the stimulus and extra-retinal information about the eye (static eye position or corollary discharge signal).

To study this computation further, we had the patient make two opposite pairs of saccades to the same physical locations. By keeping the retinal information constant, the only variable is the required order of saccade directions; right-left or left-right. We used symmetrical locations at 3° out and 1.5° down in each visual hemifield, and the same stimulus exposure times as above for the first and second stimuli. The direction of the first and second saccade was determined by the order of appearance of the two stimuli. For example, when the stimulus sequence was right-left, the patient attempted to make a rightward saccade to the right visual field target followed by a leftward saccade to the left visual field target. Latency and accuracy of these eye movements were compared with that of the same saccades made in the context of a slow version of the double-step task. In the slow version, the two stimuli were presented for 500 ms, leaving ample time for the patient to execute the first saccade before the onset of the second stimulus. This slow version involved the same eye movements as the fast version, but the motor coordinates of both the first and second saccades were congruent with the visual coordinates of their respective targets. Since one of the two stimuli was presented in the left (contralesional) visual field, none of the double-step pairs were expected to be perfectly accurate. We expected the left saccades in both the left-right and right-left pairs to be hypometric. However, if the damaged cortical regions of the right hemisphere play a role in generating or channelling extra-retinal information associated with leftward eye movements, we might expect increased latencies and inaccuracy of the rightward movement which follows it as well.

The results for the slow version of the double-step task indicate latencies and amplitudes that are consistent with results obtained with visually guided single step saccades: leftward, contraversive saccades were hypometric ($P < 0.01$) and their latencies were longer than those of rightward, ipsiversive saccades ($P < 0.02$), regardless of saccade order

(Fig. 8 top two panels, Table 3A). In the fast version, the critical question is the accuracy of the second saccade and its capacity to reach the spatial location of a target whose retinal coordinates do not match the required saccade vector (Fig. 8 bottom two panels, Table 3B) In the right-left condition, the eye movement required to reach a target which appeared at 3° left and 1.5° down was a straight 6° leftward saccades. The actual saccades made by the patient had a mean latency of 196 ms and a mean amplitude of −4.8°. Since it followed a nearly perfectly accurate first saccade, the second eye movement undershot the target by about 20%. In the left-right condition, the results were strikingly different. In two out of 10 trials, the patient performed the saccades in the wrong sequence, going from the second target to the first target, and on other trials she made two consecutive leftward eye movements. These trials were excluded from quantitative analysis. For the remaining trials, the mean latency of the second saccade was 337 ms (SD = ±177), much longer than the corresponding rightward saccades in the slow version of the double-step task ($P < 0.01$). These saccades were also inaccurate, with a mean amplitude of 2.2°. In most csaes it was impossible to determine whether the second saccades were actually directed at the second target or were simply return saccades to the fixation location.

In conclusion, in response to identical retinal stimuli, the patient made very different eye movements depending on the sequence that she had to follow. While she performed right-left combinations with adequate latency and accuracy, rightward movements in the left-right combination were markedly impaired. This occurred only for brief target presentations which created a spatial mismatch between the retinal input and the required motor output.

DISCUSSION

The patient described in this report had two sorts of deficits: (i) an increase in latency and reduced amplitude for visually guided saccades into the contralesional hemifield, and (ii) a marked deficit in compensating for prior contralesionally directed saccades when she was asked to make sequences of saccades to briefly flashed targets. She had no consistent deficit for saccades to a particular location in cranio- or egocentric space. We will discuss these deficits in terms of current concepts of the saccadic system and the analysis of space by the cerebral cortex.

Retinal factors

Saccades made into the contralesional field were hypometric and had a slight increase in latency. This deficit was relatively independent of the orbital position from which the saccade was initiated. Saccadic impairments can be explained by either sensory or motor factors. Three findings rule out a motor intepretation of this deficit: (i) all saccades, both normal ipsilesional and hypometric contralesional ones, lay on the main sequence velocity-amplitude curve that defines normal saccadic performance (Bahill *et al.*, 1975; Becker, 1989); (ii) the hypometria increased with increasing target eccentricity in a non-linear fashion, so the deficit was not a simple decrease in saccadic gain; (iii) the double-step experiments showed that the patient could make perfectly accurate saccades in the contralesional direction when the targets that evoked those saccades were in the ipsilesional retinal field.

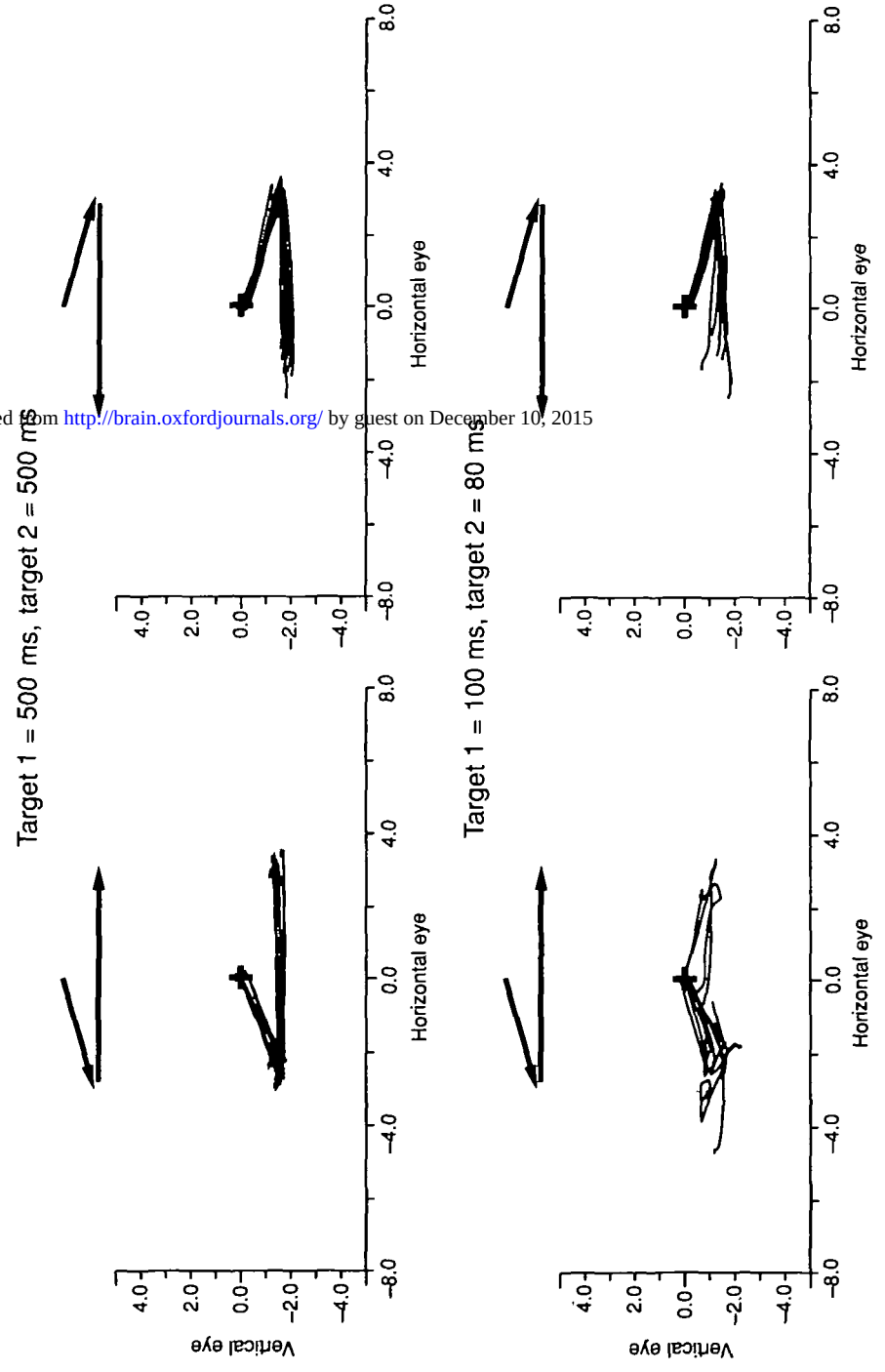

FIG. 8. Superimposed x-y plots of double-step saccades to pairs of left and right stimuli. Two different sequences of saccades were determined by the order of appearance of the stimuli, as indicated by the arrows above each plot. In the top two panels there is no dissonance between retinal and saccade vectors because the saccades were executed while their respective targets were visible. In the bottom two panels, retinal and saccade vectors for the second step are unequal because *the second target was extinguished before the first saccade was initiated.*

TABLE 3. SACCADE AMPLITUDE AND LATENCY OF E C IN THE DOUBLE-STEP EXPERIMENTS WITH ONE STIMULUS IN EACH VISUAL HEMIFIELD

Step order	*Right* → *Left*		*Left* → *Right*	
(A) Two visually guided saccades				
Step duration (ms)	500	500	500	500
Stimulus eccentricity (degrees)	3	−6	−3	6
Required saccade amplitude (degrees)	3	−6	−3	6
Actual saccade amplitude (degrees)	3.1 (0.3)	−4.5 (0.7)	−2.2 (0.6)	5.8 (0.5)
Saccade latency (ms)	190 (36)	218 (33)	288 (30)	162 (177)
(B) Two visually-triggered saccades				
Step duration (ms)	100	80	100	80
Stimulus eccentricity (degrees)	3	−3	−3	3
Required saccade amplitude (degrees)	3	−6	−3	6
Actual saccade amplitude (degrees)	3.0 (0.3)	−4.8 (1.2)	−2.4 (0.6)	2.2 (2.6)
Saccade latency (ms)	186 (9)	196 (36)	363 (104)	337 (177)

Standard deviations are given in brackets.

It is not clear why a target in the contralesional retinal field should evoke a well-made hypometric saccade. One possible explanation is in terms of a distortion of the cortical input to the superior colliculus. Lee *et al.* (1988) demonstrated the importance of the active ensemble in the colliculus for the control of saccadic amplitude. They made small lesions in the retinotopic map in the intermediate layers of the superior colliculus. Saccades made to the centre of the collicular scotoma were accurate but slow. Saccades made to the near side of the scotoma were hypometric, and those made to the far side of the scotoma hypermetric. Because of our patient's hemianopia, it is reasonable to suggest that visual activity in the lesioned hemisphere is skewed toward the centre of the visual field. Since both parietal and frontal cortex project to the superior colliculus (Lynch *et al.*, 1985; Stanton *et al.*, 1988), the corticotectal projection in this case must excite a collicular ensemble also weighted toward the centre of the visual field, resulting in hypometria. The relative mildness of the deficit may be due to its chronic nature, which had allowed a significant adaptation to the effects of the lesion over the years.

Spatial factors

Cortical deficits have been classified as having a combination of hemifield and hemispace aspects (Heilman, 1979; Ladavas, 1987; Farah *et al.*, 1990). In a hemispatial deficit the patient has difficulty with targets lying in a particular region of space relative to an egocentric coordinate frame, regardless of the target's location on the retina. In a hemifield deficit the patient has difficulty with a certain area of the retina regardless of the spatial location of the stimulus. The saccadic deficits in this case were predominantly hemifield deficits, as was shown by the task in which 5° saccades in ipsilesional and contralesional directions were made from different orbital positions.

Varying the initial orbital position dissociates the retinal and spatial coordinates of the target, but preserves the equivalence between the target's retinal vector and the required eye movement vector. In the double-step task, the goal of the second saccade cannot be defined in retinotopic coordinates, because the first saccade modifies the relationship between the centre of gaze and the location of the target. In order to maintain

the spatial accuracy of the second saccade, the extent of eye displacement associated with the first saccade has to be compensated for using information of extra-retinal origin.

The patient showed a unilateral impairment in this task but not in the direction that would be expected from a hemispatial deficit. When two stimuli were presented in sequence, one on each side of the central fixation point, the patient was able to generate a properly oriented leftward saccade following a prior rightward saccade. The leftward saccade undershot the target, as was expected from the mild retinotopic deficit. However, when the same locations were stimulated in the reverse sequence, the patient failed to make a rightward saccade following a prior leftward saccade. This impairment was neither spatial nor retinotopic. It was an impairment in compensating for an eye movement made in the contralesional direction: it seemed that the computation of a saccade in the 'normal' direction in response to a stimulus in the 'normal' visual field was aborted because of an inability to register the amplitude and direction of the immediately preceding saccade.

Can this pattern of results be accounted for without invoking extra-retinal factors? A perceptual rivalry effect is unlikely. It could explain that on two occasions the left-right target sequence triggered a right-left eye movement sequence, the ipsilesional target being responded to before the contralesional one. But on most trials the patient failed to respond to the ipsilesional target. An extinction effect, which was observed with simultaneous bilateral stimuli in patients with unilateral lesions (Bender and Teuber, 1947), would predict a pattern opposite to the one observed: the ipsilesional stimulus obliterating the contralesional one. Another possibility is that the trace of the right visual field target in 'working memory' was lost during the increased processing time of the first saccade to the left visual target. This is also unlikely since one would predict a more stable representation of ipsilesional than contralesional stimuli.

The results support the hypothesis that cortical sites in the frontal and parietal lobes are critical for performing spatial computations involving both retinal information and information corollary to eye movements. Area 8 neurons and neurons in the lateral intraparietal area, studied while monkeys performed a double-jump task, were found to encode a specific direction and distance relative to the centre of gaze, irrespective of the retinal location of the stimulus (Goldberg and Bruce, 1990; Goldberg *et al.*, 1990; Barash *et al.*, 1991). We recently demonstrated that, in the case of parietal cortex, this occurs because neural activity triggered by a previous visual input is remapped in conjunction with every eye movement into the coordinates of the current gaze location (Duhamel *et al.*, 1992). The present results indicate that such a mechanism is critical for double-step saccade performance, and perhaps more generally whenever spatial coding is required to transcend the inherent limitations of the retinal image.

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