SPATIAL ANISOTROPY OF SACCADIC LATENCY IN NORMAL SUBJECTS AND BRAIN-DAMAGED PATIENTS

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ABSTRACT

In the present study, reaction time of oblique and orthogonal saccades was investigated in normal subjects and in two groups of patients with right (RBD) and left (LBD) vascular cerebral lesions and no signs of spatial neglect. Clear altitudinal effects were present in each group of subjects: saccadic latencies were longer in the lower than in the upper part of the visual field for both orthogonal and oblique saccades. Asymmetry along the horizontal meridian was present only in case of right hemisphere damage. This supports the view that a lesion in the right hemisphere causes a greater deficit of visual-spatial processing than a left hemisphere lesion. A cerebral lesion in the right and/or left hemisphere produces a general slowing in the saccadic latency and a general reduction in the accuracy of saccades with respect to normal subjects performance. Further, it seems that making saccades in oblique direction reduces the general saccade efficiency.

INTRODUCTION

Most of the studies on saccadic latency in patients with right and left vascular cerebral lesions are concerned with the presence of an asymmetry in the horizontal dimension. Several studies, reviewed by Cochin et al. (1996), have shown contradictory results. Some authors found differences in the performance along the horizontal meridian; the most common result is that contralesional saccadic latency is longer than ipsilesional one (Heide et al., 1995; Cochin et al., 1996). This was found both in RBD (Cochin et al., 1996; Heide and Kömpf, 1994, 1998; Heide et al., 1995) and in LBD patients (Pierrot-Deseilligny et al., 1991; Cochin et al., 1996). However, there is also evidence in the opposite direction (Zihl, 1980), or even a failure to find asymmetries both in RBD (Pierrot-Deseilligny et al., 1991; Braun and Breitmeyer, 1992; Girotti et al., 1983) and in LBD patients (Girotti et al., 1983; Karnath et al., 1991; Braun and Breitmeyer, 1992; Heide and Kömpf, 1994, 1998; Troost et al., 1972; Sharpe et al., 1979). Therefore, the horizontal asymmetry underlying saccadic latency in brain-damaged patients still needs to be clarified.

None of these studies on brain-damaged patients have considered the vertical dimension, although several studies on normal subjects suggest a major division of the visual and oculomotor functions between the upper and lower visual field. Indeed, vertical asymmetries occur in a variety of perceptual-motor tasks such as visual search, motor reaction times to visual stimuli, motion perception, eye movement and visual evoked potential latencies (see reviews in Skrandies, 1987,

Furthermore, most of the studies of saccades have mainly dealt with orthogonal saccadic movements, while most saccades are neither horizontal nor vertical, but oblique. An oblique saccade is coded both along the horizontal (left-right) and vertical (upper-lower) dimension. To date, there are no data in brain-damaged patients for the reaction time in oblique saccades. Considering that the present paper focuses on studying saccadic latency between different spatial positions, vertical (upper/lower) and oblique saccades are essential to testing the spatial anisotropy of the visual field in a more fine-grained fashion. We wanted also to find out if the vertical and horizontal asymmetries observed in the orthogonal saccades, if any, were preserved in the oblique saccades.

Overall, the aim of the present research was to investigate the latency of orthogonal (vertical and horizontal) and oblique saccades in patients with unilateral right and left vascular cerebral lesions.

While previous studies were based on a small number of patients, a large group was investigated here. Both patients and normal subjects were tested with the same experimental paradigm, to allow a direct comparison between groups. The prior offset of the fixation point (gap paradigm) reduces the latency of saccades directed to a peripheral target compared to a condition in which the fixation point remains visible (overlap paradigm; see a review in Fischer and Weber, 1993). Moreover, an accurate screening of unilateral visuospatial neglect was performed to exclude patients with this syndrome, which dramatically affects the exploration of the contralesional side. Neglect is much more frequent after right brain damage than following left brain damage (Bisiach and Vallar, 1988). Thus, the exclusion of cases of neglect allows a less-biased comparison of left and right brain damaged patients.

Another variable that was controlled in the present research was eye stability during fixation. This is a critical factor for ensuring that all the stimuli were presented at the same eccentricity, and inclusion of patients with fixation deficits could add unrelated variance to the data on saccadic latency. Fixation instability was observed in various functional pathologies (e.g., degenerative cerebellar disorders, Hotson, 1982; functional macular impairments, Rohrschneider et al., 1995), and following cerebral lesions involving the prefrontal cortex (e.g., Pierrot-Deseilligny et al., 1995), the ventromedial frontal lobe (e.g., Paus et al., 1991) or the vestibular system (e.g., Foster et al., 1997). Therefore, fixation stability was measured first and patients with poor ability to maintain stable fixation were excluded from the study.

To obtain normative data, all the experimental conditions (saccadic tasks and fixation test) were preliminarily studied in a group of normal subjects. Experiments on normal subjects have shown that vertical asymmetries in eye movements depend on the type of movement executed. In the lower visual field, faster pursuit movements (Tychsen and Lisberger, 1986; Schlykowa et al., 1996) and greater slow-phase gain of horizontal optokinetic nystagmus (Murasugi and Howard, 1989) were found. Conversely, saccades to static targets generally showed an advantage for the upper visual field (e.g., van Leeuwen et al., 1998), although some work reported an opposite trend (Miller, 1969; Cohen and Ross, 1977). Moreover, there are no data in normal subjects on the reaction time in
oblique saccades. In fact, all studies on oblique saccades in humans dealt with parameters, such as amplitude, peak velocity, duration and trajectory (Bahill and Stark, 1977; Evinger et al., 1981; Oohira et al., 1983; van Gisbergen et al., 1985). Thus, the present investigation also adds new original data for normal subjects, at least for the latency of oblique saccades.

**MATERIALS AND METHODS**

**Subjects**

One hundred and twenty-six (126) patients admitted to the hospital in the last five years were examined in the present study. An initial general screening was aimed at excluding patients with (a) bilateral lesions (assessed by CT or MRI scan); (b) head trauma or tumoral pathology; (c) visual field defects (assessed by a standard kinetic Goldmann perimetry); (d) oculomotor deficits, such as diplopia and pupillar reflex deficits (assessed by a neurological examination); (e) visuo-spatial neglect (assessed on the basis of a standard neuropsychological battery, including two cancellation tasks, that is Barrage and Letter Cancellation tests, the Wundt-Jastrow area illusion test and a Sentence Reading test; Pizzamiglio et al., 1989; see last four columns of Tables I and II) and (f) fixation instability. After selection, only 58 brain-damaged patients (46%) were submitted to the measurement of saccadic latency.

The RBD group was composed of 25 patients, 21 males and 4 females, mean age 48.4 (S.D. ± 11.2). All patients had vascular pathology. The mean time interval between the onset of the disease and the examination was 15 months (S.D. ± 11; range 10-66 months) (see Table I). The LBD group was composed of 33 patients, 28 males and 5 females, mean age 48.7 (S.D. ± 10.5). All patients had vascular pathology. The interval between the onset of the disease and the examination was 15 months (S.D. ± 9.6; range 12-60 months), (see Table II). Twenty-two normal subjects (with normal or corrected to normal vision) also participated to the study. The subjects were 11 males and 11 females; the mean age was 40.8 years, S.D. ± 16.5. A t-test statistic showed that the three groups were age-matched (p > 0.05). Informed consent was obtained from all participants.

**Stimuli**

A Pentium PC generated stimuli on a NEC multisync 17" SVGA monitor. The screen subtended 37° × 28° of visual angle. Stimuli were presented on a black background.

In the **Saccades tasks** the target was a red spot subtending 0.5° of visual angle displayed at an eccentricity of 6° from the central fixation point (a white spot subtending 0.3°). The target could be displayed at 0, 90, 180 and 270 degrees of polar angle (orthogonal condition), or at 45, 135, 225 and 315 degrees of polar angle (oblique condition). In all, eight different locations separated by 45° of polar angle were tested. All targets appeared on the invisible outline of a circle. The central fixation point was presented 5° below the height of the eyes (a comfortable position for the observers) for 2200 ms. At fixation point offset the target was displayed for 800 ms. There was no delay between fixation point offset and the target onset (gap = 0).

In the **Fixation task** the target was a white spot subtending 0.3° displayed in the center of the screen for 10 sec. Height of the target was 5° below the eyes height.

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1 Previous studies on cases with functional impairment of eye motion described unstable fixation with an averaged SD greater than 3 deg (Rohrschneider et al., 1995). Considering the small stimuli eccentricity used in the saccadic task, we chose a more conservative cut-off. Patients with SD three times higher than the SD observed in normal subjects (i.e., higher than 1.2° on the horizontal dimension and 0.9 on the vertical dimension) were not included in the study.

2 After selection, the two groups of patients turned out to be sex un-matched. By the way, many previous studies have specifically reported that the saccadic reaction time to a visual stimulus does not show a sex difference (Nagel-Leiby et al., 1990; Fujiwara et al., 2000).
### TABLE I

Demographic and Clinical Data for 25 RBD Patients

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<thead>
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<th>Subject</th>
<th>Sex</th>
<th>Age</th>
<th>Years of schooling</th>
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<th>Aetiology</th>
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<th>Visual field</th>
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**Note**

Aetiology: H, haemorrhagic; I, ischaemic; SAH, subarachnoid hemorrhage. Lesion site: P, parietal; F, frontal; T, temporal; O, occipital; BG, basal ganglia; IC, internal capsule; EC, external capsule; Th, Thalamus. Neuropsychological tests: The presence (+) or absence (-) of a pathological score in the four neuropsychological tests used to screen neglect is shown in the last four columns. Patients failing in at least two out of four tests are usually classified as neglect patients (Pizzamiglio et al., 1989).
### TABLE II

**Demographic and Clinical Data for 33 LBD**

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*Note – See Table I.*
Procedure

Subjects were seated in a chair in a dimly illuminated room with the head fixed with a chin-rest and a forehead-rest at a viewing distance of 63 cm. Before collecting the data, the signals were calibrated by the presentation of nine white 0.8° boxes, located according to a 3×3 matrix subtending 7°×7°. The first box appeared at the top-left location then it jumped to the next position and so on (from left to right and top to bottom). The subject was instructed to track it. Calibration procedure was repeated three times, and then the experiment started. To correct small head movements during the course of an experimental session, the origin of the system's co-ordinate frame was reset before, during, and after each experimental trial by asking subject to fixate a visual spot at the center of the screen (where the fixation point was placed during the experimental session).

Saccade Tasks

Oblique and orthogonal conditions were tested in separate sessions. Subjects were instructed to maintain fixation on the central point until the target appeared unpredictably at one of the four locations; then they had to perform, as soon as possible, a saccade to the target. When the target went off the subject’s gaze was supposed to return to the central fixation point. It was emphasized that only one target at time was displayed and that the subject should not try to anticipate the target appearance. In both experimental sessions, at least five trials for each location were presented in a random sequence. The 20 stimuli run were repeatedly delivered until at least five repetitions with no artifacts for each spatial position were recorded (range 5-30 repetitions). As central tendency indicator, we used the median value. The order of the two conditions (oblique and orthogonal) was randomized between subjects.

Fixation Task

The subject was asked to maintain fixation as still as possible on the central point. Measurements were made based on a 10-sec fixation period, to stress the fixation system.

Eye Movement Recording and Data Analysis

Horizontal and vertical eye movements were recorded from the right eye, by means of an infrared pupil reflection system (AMTech ET3 eye tracking system). Filtered (DC-125Hz) signals were sent to a computer and recorded on a disk. The temporal resolution of the system was 5 ms (sampling rate 200 Hz); spatial resolution was at best 0.025° but somewhat lower in practice (0.03°), similar to other studies (Foster et al., 2000). Signals were analyzed off-line after each run. Blinks were automatically detected. The portions of traces contaminated by blinks and artifacts due to occasional head movements were rejected. In the saccadic tasks, data were excluded from the analysis for each one of the following reasons: (a) recording was contaminated by blinking; (b) the saccades were performed in the wrong direction; (c) eyes moved before the target onset; (d) saccade latency was shorter than 80 ms or longer than 500 ms; (e) the saccadic amplitude was less than five degrees; (f) the subject performed two saccades instead of one. The number of all responses rejected provided a rejection rate, taken as an index of the signal reliability. In normal subjects, the

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3 In agreement with earlier investigations (Wenban-Smith and Findlay, 1991; Fischer and Weber, 1993) we used 80 ms as the upper time limit for the presence of anticipatory saccades, and as the lower limit for visually guided saccades.
rejection rate was 13.5% in the oblique condition and 11.7% in the orthogonal condition. In the Oblique condition, rejection rates were 28.2% and 29.5% for RBD and LBD, respectively. In the Orthogonal condition, rejection rates for RBD and LBD patients were 26.2% and 25.7%, respectively.

The number of responses rejected for reasons (b), (e) and (f) provided an index of the performance accuracy (expressed as percentage of error). A preliminary analysis of variance was carried out on the accuracy of saccades.

Saccadic latency to the target stimulus was measured automatically. For technical reasons, in the oblique condition data were recorded only in two subgroups (23 LBD and 23 RBD patients). Moreover, to directly compare the orthogonal and oblique saccades, data obtained in the four different spatial positions were averaged. Then, two separate ANOVAs were conducted on the saccadic reaction times (TRs) and accuracy (percentage of error). In both cases, Group (controls, LBD and RBD) was the between factor and Task (oblique and orthogonal) was the repeated factor. Considering the unequal size of our three groups of subjects, the post-hoc comparisons were based on Tukey Honest Difference test for unequivalent sample.

In the fixation task, the position of the right eye in the focal plane (fixation stability) was evaluated by calculating the standard deviation of the eye position averaged along both the horizontal and vertical dimensions (in deg of visual angle).

**RESULTS**

**Accuracy in the Saccade Tasks**

Mean accuracy (expressed as percentage of error) obtained in oblique and orthogonal saccades is reported in Figure 1 for the three groups of subject. Inspection of the figure clearly indicates that in both kinds of saccades the two groups of patients were less accurate than the normal subjects. It is also evident that each group of subjects was much less accurate in the oblique saccades than in the orthogonal ones.

In the *oblique condition*, the ANOVA showed that only the main effect Group was significant ($F_{2, 65} = 4.20; p < 0.05$). Post-hoc analyses showed that control subjects made a smaller number of errors ($p < 0.05$) than both groups of patients. Accuracy in RBD and LBD patients was not different ($p = 0.26$, n.s.). The mean percentages of errors were 10.0% (control subjects), 21.4% (LBD) and 23.3% (RBD).

In the *orthogonal condition*, the ANOVA showed that only the main effect Group was significant ($F_{2, 76} = 4.46; p < 0.05$). Post-hoc analyses showed that control subjects made a smaller number of errors ($p < 0.05$) than both groups of patients. Accuracy in RBD and LBD patients was not different ($p = 0.88$, n.s.). The mean percentages of errors were 7.2% (control subjects), 18.9% (LBD) and 16.5% (RBD).
Comparisons between Orthogonal and Oblique Saccades

ANOVA showed that both the main effects, Group (F_{2, 65} = 3.48; p < 0.05) and Task (F_{1, 65} = 6.3; p < 0.05) were significant. Particularly relevant in this ANOVA is the main factor Task. All subjects made a bigger number of errors when performing oblique saccades (18.2%) than orthogonal ones (14.2%). The interaction was not significant (F_{2, 65} = 1.07).

Saccade Latencies in the Oblique Condition

Individual saccadic latencies are reported in the correlogram of Figure 2 for both left (LVF) and right (RVF) visual field targets. Inspection of the figure clearly indicates that in each group of subjects saccadic latencies to upper targets were shorter than those obtained for lower targets.

The ANOVA showed that both the main effects, Group (F_{2, 65} = 3.24; p < 0.05) and Position (F_{3, 195} = 60.03; p < 0.001) and the interaction (F_{6, 195} = 2.15; p < 0.05) were significant. Post-hoc analyses showed that in control subjects both saccadic latencies to lower targets (247 ms left and 243 ms right) were significantly longer than that to upper left (213 ms, p < 0.001) and right (211 ms, p < 0.001) targets. The other differences were not significant. In LBD patients, both saccadic latencies to lower targets (264 ms left and 261 ms right) were significantly longer than that to upper left (230 ms, p < 0.001) and right (234 ms, p < 0.001) targets. The other differences were not significant. In RBD patients, both saccadic latencies to lower targets (273 ms left and 264 ms right)
Spatial anisotropy of saccadic latency

Fig. 2 – Correlograms show the individual saccadic latencies obtained in upper visual field as a function of lower visual field (oblique saccades) in the 22 normal subjects (left), in the 23 LBD (middle) and in the 23 RBD patients (right). Filled circles refer to left visual field and open circles refer to right visual field. In this and in the following pictures it’s also shown a schematic representation of the stimuli position.

were significantly longer than that to upper left (231 ms, p < 0.001) and right (231 ms, p < 0.001) targets. Moreover, saccadic latency to lower left targets was significantly longer than that to lower right (p < 0.05). Saccadic latencies to upper left and right targets were not different. For each tested spatial positions, saccadic latencies observed in RBD and LBD patients were significantly longer than those observed in control subjects (p < 0.05). Furthermore, for lower left targets RBD patients had a saccadic latency longer than that observed in LBD patients (p < 0.05).

Saccade Latencies in the Orthogonal Condition

Individual saccadic latencies obtained in the upper visual field as a function of lower visual field for each group of subjects are reported in Figure 3. Individual saccadic latencies obtained in the right visual field as a function of left visual field for each group of subjects is reported in Figure 4.

The ANOVA showed that main effect Group was not significant (F_{2, 77} = 1.44; n.s.). The main effect Position (F_{3, 195} = 60.03; p < 0.001) was significant (F_{3, 231} = 26.79; p < 0.001). Interaction was also significant (F_{6, 231} = 3.65; p < 0.005). Post-hoc analyses showed that in normal subjects the mean saccadic latency to lower target (265 ms) was significantly longer than that to upper target (230 ms; p < 0.001), left target (232 ms, p < 0.001) and right target (234 ms, p < 0.005). The other differences were not significant. In LBD patients, the mean saccadic latency to lower target (268 ms) was significantly longer than that to upper target (241 ms; p < 0.005), left target (250 ms, p < 0.05) and marginally to right target (252 ms, p < 0.08). The other differences were not significant. In RBD patients, the mean saccadic latency to lower target (267 ms) was significantly longer than that to upper target (234 ms; p < 0.001), and right target (229 ms, p < 0.001). The mean saccadic latency to left target (263 ms) was significantly longer than that to upper target (p < 0.005) and right target...
Fig. 3 – Correlogram shows the individual saccadic latencies obtained in 22 normal subjects (filled squares), in the 33 LBD (open circles) and in the 25 RBD (filled triangle) patients in the orthogonal saccades: upper visual field as a function of lower visual field.

Fig. 4 – Correlograms show the individual saccadic latencies obtained in right visual field as a function of left visual field (orthogonal saccades) in the 22 normal subjects (left), in the 33 LBD (middle) and in the 25 RBD patients (right).
(p < 0.005). The other differences were not significant. For left targets, RBD patients had a saccadic latency significantly longer than that observed in control subjects (p < 0.005). For each tested spatial positions, saccadic latencies observed in RBD and LBD patients were significantly longer than those observed in control subjects (p < 0.05).

**Comparisons between Orthogonal and Oblique Saccades**

ANOVA showed that neither the main effects, Group (F_{2, 65} = 2.98) and Task (F_{1, 65} = 2.2) or the interaction were not significant (F_{2, 65} = 1.48).

**Fixation Task**

In normal subjects, the mean standard deviation of eye movement during fixation was 0.40° on the horizontal axis and 0.32° for the vertical axis. In the RBD group, the mean SD was 0.71° on the horizontal dimension and 0.90° on the vertical dimension. In the LBD group, it was 0.69° on the horizontal dimension and 0.62° on the vertical dimension. The values for the two groups of patients were not different neither along the horizontal (t < 1; n.s.) or along the vertical (t_{61} = 1.42; n.s.) dimension. This suggests that results obtained in the saccadic task were not due to differences in stability of fixation between the two groups. The fixation test showed a high degree of stability and a shift around the fixation point of the same order of magnitude as that reported in previous studies on normal subjects (Rohrschneider et al., 1995; Ukwade and Bedell, 1993; Eizenman et al., 1992).

**DISCUSSION**

The main purpose of this research was to study saccadic latency toward targets located in various space positions in patients with unilateral brain lesions. The summary of results obtained in both saccade tasks for the two groups of patients with right and left cerebral damage is reported in the polar plot of Figure 5. In addition, normal subject data are reported for a comparison in the same figure. Clear altitudinal effects are present for the saccadic reaction time in each group of subjects. The figure shows that all patients with unilateral brain lesion showed saccadic latencies longer towards targets placed in the lower part of the visual field. This trend is similar to what is observed in normal subjects and is preserved after the occurrence of brain lesions (see the polar plot in Figure 5). It’s worth noting that in all subjects the altitudinal asymmetry was similarly present in both orthogonal and oblique saccades. This implies that the vertical effect is present not only when the stimuli are separated by a polar angle of 180 deg (upper vs. lower target in the orthogonal condition) but also for a separation of just 90 deg (i.e., upper-left vs. lower-left or upper-right vs. lower-right in the oblique condition).

The direction of the asymmetry for the vertical dimension (an advantage for the upper visual field) that was observed in both controls and brain damaged
patients, is the opposite to that observed in studies on other types of eye movements. For example, Tychsen and Lisberger (1986) reported an asymmetry in eye movement accelerations to pursuit targets in the two hemifields, with greater accelerations in the lower visual field for both upward and downward target motion. The lower visual field superiority in pursuit initiation is also paralleled by the greater slow-phase gain of horizontal optokinetic nystagmus in the lower visual field (Murasugi and Howard, 1989). The longer latency for saccades towards the lower targets is also in contrast with findings in other kinds of tasks. Indeed, lower-visual field advantage has been also observed for temporal and spatial contrast sensitivity (e.g., Skrandies, 1985a, 1985b), visual acuity (Millodot and Lamont, 1974), motor reaction time (e.g., Payne, 1967), visual evoked potentials latencies (Lehmann and Skrandies, 1979; Skrandies, 1987; Rudell et al., 1993; Pitzalis et al., 1997) and functional activation (Portin et al., 1999). Differences in the processing of the upper and lower retinal visual stimuli could be explained by a general superiority of the upper over the lower hemiretinal system. This would be due to an over-sampling at the retinal level and an over-representation at the cortical level for the lower portion of the visual field with respect to the upper field. In humans, there is a higher density of both classes of photoreceptors in the upper and nasal field when compared to the lower and temporal parts of the retina at eccentricities exceeding 6° degrees (e.g., Østerberg, 1935; Curcio and Allen, 1990), a result which fits, at least in part, with the over-sampling hypothesis. Considering the wealth of data supporting a superiority of the lower visual field, the opposite asymmetry found for saccadic latency remains odd. An interpretation of the results has been proposed within the framework of an ecological theory concerning the relationships of the upper and lower visual fields to extrapersonal and peripersonal space, respectively (Previc, 1990). Previc (1990) argued that vertical perceptual-motor asymmetries might result from the biases of the lower and upper visual field toward proximal and distal space. Different neural systems specialized for operations in near and far space would be biased towards the lower and the upper visual fields, respectively. The lower field advantage
observed in many studies is probably related to the fact that stimuli occurring below the visual horizon carry different meaning and have a different degree of importance than stimuli above the visual horizon. Indeed, for humans many objects of interest in the visual world are at reaching distance and below the horizon. On the other hand, it might be proposed that the shorter latency of saccades for the upper portion of space is related to the specific role of this type of eye movement. The function of saccades is to scan and explore space, but particularly extrapersonal space; this may explain the bias toward the upper visual field. Support for the ecological hypothesis comes from the notion that the centers involved in the control of eye movements seem to be preferentially activated by stimuli located at far distances. For instance, recordings from frontal eye field neurons showed that far space has no relation to somatosensory inputs but appears instead to be strictly linked with oculomotion and, more generally, with contralateral exploratory movements (Goldberg and Bushnell, 1981; Bruce and Goldberg, 1985). Based on the physiological properties of area 8 neurons and on ablation studies showing that lesions of monkeys’ frontal eye fields causes a decrease of eye movements contralateral to the lesion and a visual neglect in the far space (Rizzolatti et al., 1983; Rizzolatti et al., 1985), Rizzolatti and Gallese (1988) proposed that area 8 is involved in far space representation. Furthermore, area LIP neurons also have independent sensory and motor responses as well as saccade-related bursts. Colby et al. (1996) showed that area LIP (which is richly connected with, and physiologically similar to, area 8) might be another neural substrate for the representation of far space in monkeys. The hypothesis of saccades having a preferential relationship with upper space is supported also by data on visual search tasks, where there was a slight preference to start the scanning in the upper quadrants (Chedru et al., 1973; Previc and Blume, 1993; Previc, 1996; Behrmann et al., 1997), even in complete darkness (Karnath and Fetter, 1995). Also, upward shifts of gaze are more frequent than downward shifts during visuospatial imagery tasks (Kinsbourne, 1972; Previc and Murphy, 1997). The dominance of the upper over the lower retina frequently observed in humans is not found in all species. For instance, the visual system of rabbits (e.g., Oyster et al., 1980) and cats (Tusa and Palmer, 1980), which are small-sized animals and thus vulnerable to predators from above, shows signs of the opposite organization. It is possible that the oculomotor system of a particular species has evolved to best meet the demands of its environment. Therefore, the shorter saccadic latency toward objects above the eye level might fit with the importance of saccades in the exploration of human extrapersonal space.

Along the horizontal dimension, different results have been observed depending on the group tested. In normal subjects, saccadic latencies towards the left and the right side were comparable in both oblique and orthogonal saccades, confirming with most of the previous studies (Saslow, 1967; Miller, 1969; 4 Recent neuropsychological investigations seem to suggest that in humans, space is coded in a way similar to monkeys. For instance, studies of line bisection in neglect patients have shown that bisection performance can be severely impaired in either near or far space while remaining within normal limits in the other spatial domain (Halligan and Marshall, 1991; Cowey et al., 1994, 1999; Vuilleumier et al., 1998; Pitzalis et al., 2000; Berti and Frassinetti, 2000).
Michard et al., 1974; Cohen and Ross, 1977; Heywood and Churcher, 1980; Karnath et al., 1991; Cochin et al., 1996). As with normal subjects, LBD patients did not show any horizontal asymmetry either in oblique or orthogonal saccades, thus confirming only some of the previous studies (Pierrot-Deseilligny et al., 1991; Cochin et al., 1996). On the other hand, damage to the right hemisphere disrupts the normal temporal symmetry of saccades across left and right space. Saccadic latency towards contralesional targets was slower than that for ipsilesional targets. This horizontal asymmetry was present in the left vs. right orthogonal saccades and only in the lower-left vs. lower-right oblique saccades. Thus, the horizontal asymmetry was absent in the upper hemifield. Interestingly, this result is similar to what has been observed in patients with damage to the right hemisphere and visual neglect. Indeed, in these patients a prevalence of a greater lateral deficit for stimuli positioned in the lower than in the upper space has been frequently reported (e.g., Pitzalis et al., 2000).

The presence of the horizontal asymmetry only in the RBD patients suggests that a right cerebral lesion has a more marked effect on the latency of contralesional saccades than that observed for left hemisphere damage. We excluded neglect patients from our group of RBD and, therefore, the results cannot be ascribed to spurious differences present between the two groups, as indicated by the similar accuracy in performance. The presence of a horizontal asymmetry in patients with right but not left brain damage supports the view that a lesion in the right hemisphere causes a greater deficit of the visual-spatial computation than a left hemisphere lesion (Mesulam, 1981; Heilman et al., 1987), even when patients with unilateral heminattention are excluded. This suggests that there is some degree of cerebral lateralization for saccades, just as for a number of other visuospatial functions. Indeed, previous studies also found that the right hemisphere is dominant both in programming eye movements (e.g., Pierrot-Deseilligny et al., 1995) and in executing simple reactions to lateralized unstructured visual stimuli (Sava et al., 1988). Moreover, the right hemisphere dominance in spatial attention is well known (e.g., Heilman and Van Den Abell, 1980; Heilman et al., 1985). In humans, for instance, unilateral neglect syndromes are more frequent and severe after lesions in the right hemisphere (e.g., Bisiach and Vallar, 1988). In addition, during motor exploration of space (Gitelman et al., 1996) and lateral orientation of visuo-spatial attention (Nobre et al., 1997), parietal areas have been reported to be active, with a prevailing or exclusive lateralization to the right cerebral hemisphere. In a recent fMRI study, Vallar et al. (1999) reported evidences of a fronto-parietal network, with a major right hemisphere-based component, for computing the egocentric frames of reference in humans. All this evidence strongly supports a model of right-hemispheric dominance for the distribution of attention within the extrapersonal space and supports the view that spatial attention and saccade programming share the same neural substrates (Sheliga et al., 1995a; Sheliga et al., 1995b; Sheliga et al., 1997). As summarized in the introduction, conflicting results are present in the literature on brain-damaged patients regarding the horizontal asymmetry in the saccadic latency. It seems that the present investigation, extended to a large group of patients and using strict selection criteria on fixation stability and absence of unilateral neglect, might offer normative data, at
least for the gap paradigm condition.5

Finally, a cerebral lesion in the right and/or left hemisphere produces a
general slowing in the saccadic latency (about 23 ms) and a general reduction in
the accuracy of saccades with respect to normal subjects’ performance (more
than a factor of two). Moreover, direct comparison between LBD and RBD
patients showed that the saccades’ latency is similarly affected in both groups
along the vertical dimension but not along the horizontal one, where only RBD
patients had longer contralesional saccadic latency than ipsilesional. By contrast,
performance accuracy was similarly affected in both groups of patients,
independent of the side of the lesion. Interestingly, the direct comparison
between the accuracy in the oblique and orthogonal saccades showed that, while
there were no differences in the saccadic reaction times, all subjects made a
larger number of errors in the oblique condition than in the orthogonal one (see
also Figure 1). Thus, it seems that making saccades in an oblique direction does
not affect the saccadic latency but reduces general saccade efficiency and
represents a more difficult condition than making saccades purely along the
orthogonal axes.

In summary, functional differences between different regions of the visual
field cannot be discussed only in terms of a left/right distinction, because the
upper/lower distinction plays a role in the visual perception. The normal
advantage of saccadic latency for targets located in the upper portion of space
survives with unilateral brain damage. This holds for patients with stable
fixation and when only accurate saccades are selected. On the other hand, the
symmetry of temporal properties of saccades along the horizontal meridian is
lost only for damage to the right hemisphere.

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5 The gap paradigm changes the attentional demands of the task (with respect to overlap condition) because the offset
of fixation point eliminates the cost of disengagement, reducing the saccadic latency. PET studies indicate that the
different paradigms used (gap and overlap) can produce distinct and robust changes in regional activation in the brain
(e.g., Corbetta et al., 1993; Doricchi et al., 1997). Consequently, it is clear that results obtained with a temporal gap
cannot be compared with those obtained with an overlap paradigm because they probably arise from different visual
mechanisms.


KARNATH, H.O., SCHENKEL, P., and FISCHER, B. Trunk orientation as the determining factor of the “contralateral” deficit in the neglect syndrome and as the physical anchor of the internal representation of body orientation in space. Brain, 114: 1997-2014, 1991.


