Exp Brain Res (2001) 137:45–57
DOI 10.1007/s002210000546

RESEARCH ARTICLE

Chiang-Shan Ray Li · Richard A. Andersen

Inactivation of macaque lateral intraparietal area delays initiation of the second saccade predominantly from contralesional eye positions in a double-saccade task

Received: 8 December 1999 / Accepted: 29 June 2000 / Published online: 8 February 2001
© Springer-Verlag 2001

Abstract Previous studies have shown that, although lateral intraparietal (LIP) area neurons have retinotopic receptive fields, the response strength of these cells is modulated by eye position. This combining of retinal and eye position information can form a distributed coding of target locations in a head-centered coordinate frame. Such an implicit head-centered coding offers one mechanism for maintaining spatial stability across eye movements and can be used to compute new oculomotor error vectors after each eye movement. An alternative mechanism is to use eye displacement signals rather than eye position signals to maintain spatial stability. The aim of this study was to distinguish which of these two extraretinal signals (or perhaps both signals) are employed in a double saccade task, which required the monkey to use extraretinal information associated with the first saccade to localize a remembered target for a second saccade. By varying the direction and the end point of the first saccade and selectively inactivating area LIP in one hemisphere with muscimol injection, we were able to distinguish between the two mechanisms by observing how the second saccade was impaired in this task. The displacement mechanism predicts that, if the first saccade is in the contralesional direction, the second saccade will be impaired, and the end point of the first saccade would not be important. The eye position mechanism predicts that if the first saccade ended in the contralesional head-centered space, the second saccade will be impaired, no matter in which direction the first saccade is made. Results showed that, after area LIP lesion, when the first saccade stepped into the contralesional field, the error rate of the second saccade became higher and the latency longer. However, when the end point of the first saccade was constant, the direction of the first saccade had much less effect on the second saccade. These results suggest that eye position, and not eye displacement, is the more predominant factor in this task. In a different behavioral paradigm, the monkeys performed single visual and memory saccades from different initial eye positions. It was found that the impairment of either the metrics or dynamics of visual and memory saccades did not significantly vary with the different eye positions. It thus appears that the performance of single visual and memory saccades is best described in an oculocentric coordinate frame that does not rely on extraretinal signals. Altogether these results lend further support to the hypothesis that, by combining retinal and eye position signals, area LIP contains concurrent eye-centered and head-centered representations of the visual space. Depending on the task, either representation can be used.

Keywords Posterior parietal cortex · Eye position · Saccade · Extraretinal signal · Muscimol · Monkey

C.-S.R. Li · R.A. Andersen (✉)
Division of Biology, 216-76,
California Institute of Technology, Pasadena,
CA 91125, USA
e-mail: andersen@vis.caltech.edu
Tel.: +1-626-3958336, Fax: +1-626-7952397

Present address:
C.-S.R. Li, Departments of Psychiatry and Physiology,
Chang Gung Memorial Hospital and Chang Gung University,
Tao-Yuan 333, Taiwan

Introduction

An important problem in studies of spatial vision is to understand how we can maintain stable percepts and behaviors despite constant eye and head movements. A candidate cortical area where such an important function could be carried out is the posterior parietal cortex (PPC). One among the many neural algorithms to solve the problem of spatial stability is to have neurons with explicit head- or body-centered receptive fields. Physiological studies, however, have failed to find large numbers of such cells in the PPC.

Although the construction of head- and body-centered visual receptive fields requires extraretinal signals related to eye and head position, there are alternative ways of achieving spatial stability using extraretinal signals that do not require explicit representations of space in these

coordinate frames. One possible way this might be achieved neurally has been suggested by experiments which showed that visual information and eye and head signals converged onto individual parietal neurons (Andersen and Mountcastle 1983; Andersen et al. 1985, 1990; Brotchie et al. 1995). Parietal neurons, recorded from area 7a and the lateral intraparietal area (area LIP), maintain retinotopic visual receptive fields, but their overall response magnitudes are modulated by eye and head positions. It has been proposed that object locations in a head- or body-centered space could be encoded through a distributed population of such parietal neurons. How such activity patterns could form the basis of a spatial coding mechanism has been suggested by modeling studies, which show that visual and eye position activities could indeed be combined in a multiplicative manner to encode target locations in a distributed framework (Goodman and Andersen 1990; Zipser and Andersen 1988). Further exploration of the network model has revealed neuronal properties similar to those observed in physiological experiments (Andersen et al. 1985; Brotchie et al. 1995; Goodman and Andersen 1989; Thier and Andersen 1996). Such a distributed head-centered scheme implicitly encodes target locations through a population of cells.

In the abovementioned distributed encoding scheme, when the eyes move in the dark, the retinal vector of a remembered target location must be recomputed (updated). This updating is required because the eye position signals and eye position-dependent gains change with each new eye position. Thus the coding of the retinal location of the remembered target must also be adjusted to indicate, in the population, the correct location of the target in head- or body-centered coordinates. This retinal updating has been found to occur in LIP (Duhamel et al. 1992a; Gnadt and Andersen 1988; Mazzoni et al. 1996). Interestingly, recent modeling experiments have shown that updating can also be accomplished by eye position gain fields (Xing and Andersen 1995). Thus both the updating of retinal position information across saccades, as well as the read-out of the population in head-centered coordinates, can be accomplished by eye position-dependent gain fields.

Another way in which visual stability might be maintained across eye movements is by making use of eye displacement signals (Duhamel et al. 1992a; Goldberg and Bruce 1990). A corrected retinotopic representation is maintained by subtracting the displacement vector of the saccades from the retinal vector of the stimuli. In this scheme an eye position signal is not required to update retinal locations after saccades. However, this method would not provide a way to read out the population code of LIP in coordinate frames that are nonretinotopic (i.e., head- or body-centered), and such additional coordinate frames would need to be computed elsewhere.

The present study aimed to distinguish between these two different hypotheses by using a double saccade task, modified from the one used by Hallet and Lightstone and others in psychophysical and physiological experiments (Gnadt and Andersen 1988; Goldberg and Bruce 1990; Hallet and Lightstone 1976; Mays and Sparks 1980; Mazzoni et al. 1996). This task required the monkey to use the information associated with the first saccade to be able to make the second saccade correctly. Since both saccade-related and eye position activity generally shows a contralateral bias (Barash et al. 1991; Lynch et al. 1977; C.-S. R. Li and R. A. Andersen, unpublished data; but see Platt and Glimcher 1997 for some different results regarding saccade activity) by selectively lesioning this area in one hemisphere, we disrupted contralateral eye position and saccade-related activities. We then systematically varied the direction and end point of the first saccade and tried to determine how the impairment of the saccade and eye position activities might affect the second saccade in this task. A predominantly directional effect would favor the displacement model, whereas an eye position effect would support the eye position model.

A preliminary report of part of this study has been presented in abstract form (Li et al. 1995).

Materials and methods

Surgery, animal care, unit recording, eye position monitoring, and muscimol injections

Two macaque monkeys, LBZ and NWT, were used in this experiment. Surgical procedures and animal care are described in detail in a previous paper (Li et al. 1999). Eye position was monitored by the scleral search coil technique (Fuchs and Robinson 1966; Judge et al. 1980), and calibration was performed daily before experiments started. After behavioral training was completed, recordings and lesions were performed in area LIP in three hemispheres of the two monkeys. Pressure injection of muscimol was made with a Hamilton syringe, which was held by an adapted Narishigi microdrive. Two to three microliters of muscimol (8 mg/ml; Sigma, St. Louis, Mo.) were used in each lesion in this experiment. In each experiment, injections were usually made at two different locations where saccade-related and eye position activities were recorded. The injections were slowly administered, typically over a period of 2–3 min. On two separate occasions, saline injections were used for controls. The behavioral tasks usually started within 10 min after the injections. As the amounts of muscimol injected in these experiments were relatively large, no specific effort was made to estimate how much of the cortical tissue in area LIP was inactivated in each injection. However, as was described in the previous paper, the effect of muscimol injections of this size appeared to be confined within this cortical area, since no deficits other than saccades were found (Li et al. 1999). No specific effort was made to examine whether the deficits were topographical in this study, as again the results obtained in the previous experiment suggested that such effects were generally not present. A total of six lesions were performed in two different hemispheres (four in the right hemisphere and two in the left) of monkey LBZ and four lesions in the left hemisphere of monkey NWT. NIH guidelines were strictly followed for the care and use of the animals.

One monkey was killed after both hemispheres were explored in the recording and lesion experiments. The monkey was given an overdose of pentobarbital sodium and then perfused transcardially with heparinized saline, followed by buffered formalin. Examination of the penetration marks on the surface of the brain showed that they were mostly concentrated on the lateral bank of the intraparietal sulcus. Fifty-micrometer-thick sections of the brain were cut and stained with neutral red for cytoarchitectural investigation. The lesion marks were clearly visible and located in the lateral bank of the intraparietal sulcus.

Behavioral tasks and training

Double saccade task

The monkeys were trained in a task requiring two sequential saccades. After the monkey acquired a fixation point (total duration of fixation, 1200 ms), a small target (T2) was briefly flashed (100 ms). The fixation light was turned off 800 ms after the presentation of T2 and, at the same time, another light target (T1) was presented. The monkeys were trained to make a visually guided saccade to T1 within a time window of 400 ms and to stay there for a criterion duration (600 ms for one monkey and 400 ms for the second), after which T1 was turned off. The monkey then had to make another saccade to the remembered location of T2 within a time window of 500 ms. Therefore two saccades were involved in this task; the first was a visual saccade and the second a memory saccade. Since the experiments were carried out in otherwise total darkness, the animals had to compute the second eye movement taking into account the first saccade. The spatial windows were typically 4° and 12° in diameter, respectively, for the visual and memory saccades.

The locations of the fixation light and saccade targets were arranged so that the fixation light could appear at one of four different locations on the horizontal axis: (–15,0), (–5,0), (5,0), and (15,0), respectively, relative to straight ahead: (0,0). Target T1 was also always positioned on the horizontal axis, so that the first saccade either went right or left, and was always 10° in amplitude. Target T2 was located on one of the two diagonals centered at the end point of the first saccade, so that the second saccade was also 10° in amplitude, and directed either up left, up right, down left, or down right. The direction and amplitude of the two saccades were chosen such that the oculomotor behaviors were within a reasonable range of motion. A distinct combination of each first and second saccade was called a "class." This behavioral paradigm is schematically illustrated for one class in Fig. 1.

This double saccade paradigm is different from the one introduced by Hallet and Lightstone (Hallet and Lightstone 1976) and later refined by Mays and Sparks for physiological experiments (Mays and Sparks 1980). This new paradigm was used in this study for two reasons. First, we have shown in a previous study that lesioning of area LIP disrupted the metrics of memory saccades (Li et al. 1999). Thus employment of the traditional double saccade paradigm, in which two memory saccades are executed, would make it difficult to evaluate the impairment of the eye position signal in this behavioral task. Any deterioration of performance would have to take into account the impairment of the first memory saccade per se. Second, by making the first saccade visually guided and imposing a criterion stay at the end of this first eye movement, we could make a precise measurement of the latency of the second saccade, which would be difficult to obtain in the Hallet and Lightstone paradigm.

As a control experiment, data were also collected from monkey LBZ in a task where two successive, visually guided saccades were performed. In this task, instead of presenting target T2 for the monkey to memorize while fixating, T2 was presented immediately after the criterion stay at T1, and he was required to make a second visual saccade within the same time window to acquire the target. The spatial window for acquiring each target was a circle 6° in diameter.

Different classes were presented in a pseudorandom manner during the experiment. To ensure an even sampling of all classes, those classes in which the monkey had successfully completed fewer trials had a higher priority of being presented until the same number of successful (HIT) trials was achieved for all classes. However, to avoid repeatedly sampling a particular class, if the monkey failed a class for a successive five trials, the class was temporarily switched off. It was switched on again after the monkey successfully performed trials in at least two other classes. This procedure prevented the monkeys from becoming frustrated after repeated failures. Other than in one initial experiment with monkey LBZ, the sampling of different classes was even (the difference in HIT numbers between different classes in this initial experiment was 1 at most). The task was run until an equal number of HIT trials was collected for each class in each control and lesion experiment. Typically a total of 10 or 12 HIT trials was collected for each class in each block of the experiment. Each lesion and its corresponding control experiment had the same number of HIT trials. MISS trials were those in which the monkey failed to acquire the fixation point or to fulfill the initial stay at the fixation light for the criteria duration (300 ms). An ERROR trial was one in which the monkey failed to complete the task after the trial started. In a HIT trial the animal completed the entire trial successfully and received a reward.

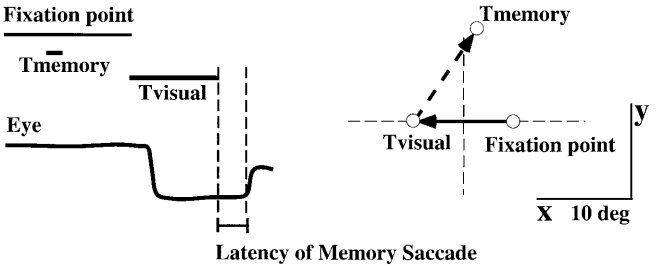

Fig. 1 The sequential saccade paradigm. While the monkey fixated, a target, *Tmemory*, appeared briefly. At the end of the fixation, another light target, *Tvisual*, appeared. The monkey was required to make a saccade to Tvisual and to stay there for a criterion duration, after which Tvisual was turned off, signaling him to make another saccade to the location where Tmemory had appeared before. The targets were arranged so that all the first saccades were on the horizontal axis and the second saccades were on the two diagonals centered on the end point of the first saccades. Both saccades were 10° in amplitude. The latency of the second saccade was measured as the time it took the monkey to initiate the saccade after the offset of Tvisual. Tvisual and Tmemory are represented as T1 and T2, respectively, in the text

Training and experiments were conducted in otherwise total darkness, so the monkeys had no access to any other visual information in any phases of these experiments. The room light was turned on briefly every 5–10 min to prevent the monkey from becoming dark-adapted or falling asleep. Training for both monkeys in this experiment started with the double visual saccade task, which showed them that there would be two saccades involved in the task. Training for the sequential visual and memory saccades started with a short criterion stay at the end of the first saccade, which was gradually increased through training. After approximately 1 month of training, one monkey was able to stay at target T1 for a duration of 600 ms and the other monkey, 400 ms, successfully completing the task over 80% of the time.

Single saccades from different eye positions

In separate blocks of experiments, the monkeys performed single visual and memory saccades from different initial eye positions. Three different eye positions on the horizontal axis were tested: (–10, 0), (0,0), (10,0), and targets were presented at eight different locations equally spaced on a circle 15° in radius in this task. In visual saccades, the monkey fixated the initial fixation point for 1200 ms and then was required to make a saccadic eye movement within 350 ms after the offset of the fixation light and the simultaneous onset of the saccade target randomly chosen from one of the eight possible locations. The animal was required to maintain fixation on the saccade target for another 800 ms within an 8° diameter window to obtain the reward of a drop of juice. In the memory saccade trials, the saccade target was briefly flashed at one of the eight locations for 100 ms. After a delay of 950 ms from the offset of the flash, the fixation light went off and the animal was required to saccade to the remembered location of the flash within 450 ms. The spatial window for the memory saccades was larger

(15° diameter), since memory saccades are generally less accurate than visual saccades.

Data analysis

The amplitude, latency, and velocity of the first saccade was first computed to document the effect of lesion on the visual saccades, as was described in the previous paper (Li et al. 1999). A preliminary analysis of variance (ANOVA) showed that, for both ipsilesional or contralesional saccades, these parameters did not vary significantly with different second saccades. The data of the first saccades were pooled for analyses. There were three groups of rightward saccades, starting at (–15,0), (–5,0), and (5,0), and three other groups of leftward saccades, starting at (–5,0), (5,0), and (15,0). Analyses of variance for amplitude, velocity, and latency were performed on the rightward and leftward saccades for the lesion and control data with respect to different starting positions. The computation of saccade latency, velocity, and metrics has been described in detail in the previous paper (Li et al. 1999). The control data used for comparison were usually collected the day before and after the lesion experiments.

The major goal of the study was to see whether, after muscimol injection into area LIP, the execution of the second (memory) saccade was impaired after the first (visual) saccade was made. It was then determined how the impairment might differ according to the directions and/or end points of the first saccade. To characterize the performance of the second saccade, we examined the error rate, latency, and metrics in each condition. Since there were four different directions of the second saccades, we first examined whether or not the saccades of the four different directions could be combined in the analyses. Analyses of variance showed that in most experiments the error rate, latency, and scatter of the end points did not vary significantly among the four different directions of the second saccades which could follow a common first saccade; therefore, the following analyses of error rate, latency, and scatter of the end points were applied to the pooled data of all four second saccade directions for each direction and end point of the first saccade. For instance, the data of all four different directions of the second saccades following the same first saccade beginning at (–15,0) and ending at (–5,0) were combined for analysis. On the other hand, since the amplitudes of the four directions of the second saccades did vary systematically, analysis of the saccade accuracy in terms of amplitude change was performed separately for each of the four second saccades. Likewise, the analysis for the change of saccade direction was performed separately for each of the four second saccades. Finally, there were six different groups of such second saccades for both control and lesion data; three of them with the first saccade in a rightward direction, ending at (–5,0), (5,0), and (15,0), and the other three with the first saccade in a leftward direction, ending at (–15,0), (–5,0), and (5,0), respectively.

There were two variables in this experiment that might contribute to the impairment of the second saccade in the lesion condition. The first one was the direction of the first saccade – whether it was in the contralesional or ipsilesional direction. The second variable was the end position of the first saccade – whether it was on the contralesional or ipsilesional side, relative to the midline. A schematic is shown in Fig. 2 to demonstrate the predictions about how the first saccades would affect the performance of the second saccades according to the two different hypotheses. In the first analysis, we looked at how the impairment of the second saccade varied with the end point of the first saccade. Analysis of variance was applied to the data using general linear models (McNeil et al. 1996). In essence, a linear model composed of terms of independent variables and of interactions of these variables was used to fit the data. We tested whether or not the fit would be significantly different when one of these independent variables or interaction terms was left out of the model. This was carried out by performing a general *F*-test using the goodness-of-fit of the full model, where all variables were included, and that of the restricted model in which a variable or the interaction term was not included. In the first analysis, there were two independent variables: experimental condition (i.e., lesion vs control) and saccade eye position (i.e., the three different end points of the first saccade). This analysis was done separately for the two different directions, contralesional and ipsilesional, of the first saccade to see how the impairments of the second saccade might vary depending on the end points of the first saccade. Using the same set of data, we then looked at the instances in which the end points of the first saccade were the same. There were two such cases, one with two different groups of contralesional and ipsilesional saccades ending at (–5,0) and the other one with those ending at (5,0). Therefore, in the second analysis, the two independent variables were lesion versus control and the two different directions of the first saccade. In other words, we controlled the end point of the first saccade and examined whether the impairment of the second saccade depended on the direction of the first saccade.

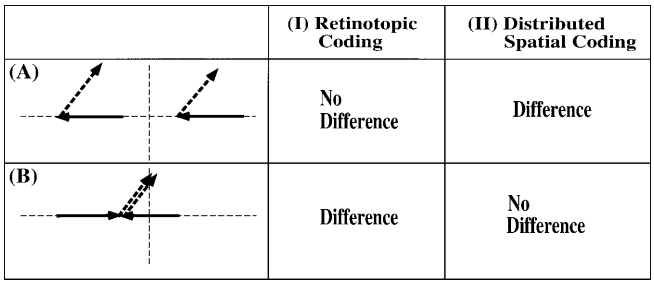

Fig. 2A, B Predictions of the hypotheses. A schematic showing the predictions of the two models as to the performance of the second saccades (*dashed arrows*) as a function of the end point and direction of the first saccades (*solid arrows*). Assuming that the lesion is in the right hemisphere, the efference copy of the leftward (contralesional) saccade as well as the eye position signal in the left hemifield would then be impaired. According to the displacement hypothesis, as long as the first saccade is in the contralesional direction, the second saccade would be impaired; in other words, it predicts no difference in the performance of the second saccades for the two conditions in **A**, in which the first saccades both go left. On the other hand, it predicts a difference for the two conditions in **B**, since the first saccades are not of the same direction. The eye position hypothesis predicts the opposite result. In **B**, where the end points of the first saccades are the same for the two conditions, the impairment of the second saccades should not be different. And in **A**, where the first saccades are of different directions but ending up in different locations, this model predicts that the performance of the second saccades would be worse for the condition where the end point of the first saccade is in the contralesional field. The *thin dashed lines* in both **A** and **B** are the horizontal and vertical axes of the screen

These analyses were applied to the error rate, latency, and metrics of the second saccade. Only trials that the monkeys successfully completed were included for the analysis of latency and metrics. The latency of the second saccade was defined as the time it took for the second saccade to be initiated (to reach a velocity criterion of 20°/s) after the offset of target T1. For the metrics of the second saccade, we computed both the amplitude, the direction, and the scatter of the end points of the second saccades. The direction of the second saccade was calculated as the *arctangent* (in degrees) of the ratio of the *y*- component over the *x*-component of the saccade. Those saccades directed toward the upper left and lower right, therefore, had a negative value, and those of the other two directions a positive one. The magnitude of the scatter was computed as the mean distance between the end points of individual saccades and the corresponding target location. Finally, we computed the number of the error trials in each block of experiment, in which 10 or 12 HIT trials were collected for each class. Since only one error measurement was available for each of the six different groups of data in a given lesion experiment, an ANOVA with repeated measures was used for this analysis. Error

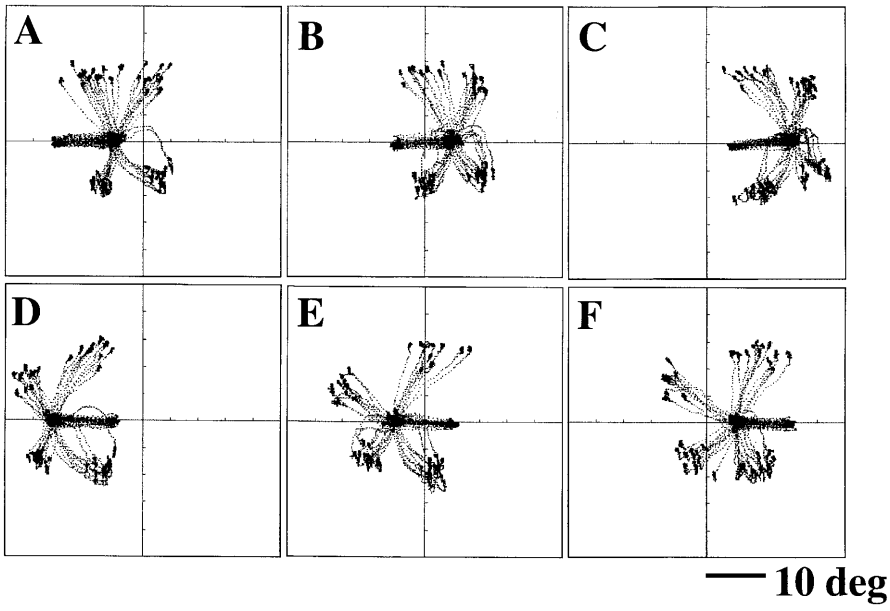

Fig. 3A–F Typical eye traces from the double saccade experiment. **A**–**C** showed the trials where the first saccades were all directed to the right and ended at (–5,0), (5,0), (15,0), respectively; and **D**–**F** those to the left and ending at (–15,0), (–5,0), (5,0), respectively. Trials with different second saccades were grouped and plotted in the same panel according to the same first saccades. It can be seen that the scatters of the end points of second (memory) saccades are much greater, and some of the second saccades show the characteristic up-shift of the end points. The saccades made further into the orbital periphery are hypometric

rate in this case is the mean number of errors for a particular condition, across experiments. Similarly, to obtain the mean change of saccade latency across experiments, the difference between control and lesion data was first obtained for each injection experiment and the results were further analyzed with an ANOVA with repeated measures. Given that a disruption of the saccade accuracy could be reflected in an increased error rate and altered metrics, the combination of error rate and metrics jointly determined the accuracy of the second saccades.

Results

General performance

Example eye traces from a control experiment with monkey LBZ are shown in Fig. 3. Different classes composed of ten trials each are shown, and the second saccades are grouped and plotted in the same panel according to the first saccade. In general, the first (visual) saccade was fairly precise, and the second (memory) saccade was not as precise. Some of these saccades showed a characteristic "upshift" of end points, such that the upward saccades were hypermetric and the downward saccades hypometric. Saccades made toward the orbital periphery from an already peripheral position also tended to be hypometric.

We first examined the effect of lesioning on the visual (first) saccade. The results showed that the latency of the contralesional saccades increased after muscimol injection. The latency increased from 206 to 240 ms for contralesional saccades ($P<0.001$, data from two hemispheres combined) and from 215 to 220 ms for ipsilesional saccades for monkey LBZ. It increased from 204 to 248 ms for contralesional saccades ($P<0.001$) and from 203 to 208 ms for ipsilesional saccades for monkey NWT. An ANOVA showed that the latency increase did not vary with starting position of the saccade: $P>0.5$ for both monkeys. The amplitude and velocity of the first saccade were also not significantly different from those of the control data for different starting positions. Similar results were obtained for both monkeys.

A previous study showed that single memory saccades were impaired after muscimol lesion of area LIP (Li et al. 1999). To document the impairment of the second (memory) saccade, therefore, data from all of the conditions (different directions and end points of the first saccades) were combined according to the direction of the second saccade. Table 1 shows the data from the two monkeys for saccade amplitude, latency, and velocity. Except for the saccades in the upper contralesional direction ($P<0.01$) in monkey NWT, the mean amplitude of the second saccade did not seem to be different from the controls, but the latency of the second saccade increased ($P<0.001$) and the velocity became slower ($P<0.001$) for both monkeys after area LIP lesion. Given that the memory saccades in this task were of one fixed amplitude, we were not able to deduce the relationship between the peak velocity and amplitude. However, since the saccade amplitudes were not significantly reduced in most cases, the decrease in velocity was probably a result of altered processing of saccade dynamics due to the lesion. These results reproduced the effects on memory saccades described in a previous study (Li et al. 1999).

Impairment of eye position signal in the double saccade task

Latency

Figure 4 shows the results of an ANOVA for the mean increase in latency (with standard errors) across lesions. To make the comparison easier, note that in Fig. 4 the data were presented as though the contralesional fields were all on the left (i.e., as though the lesions were all in

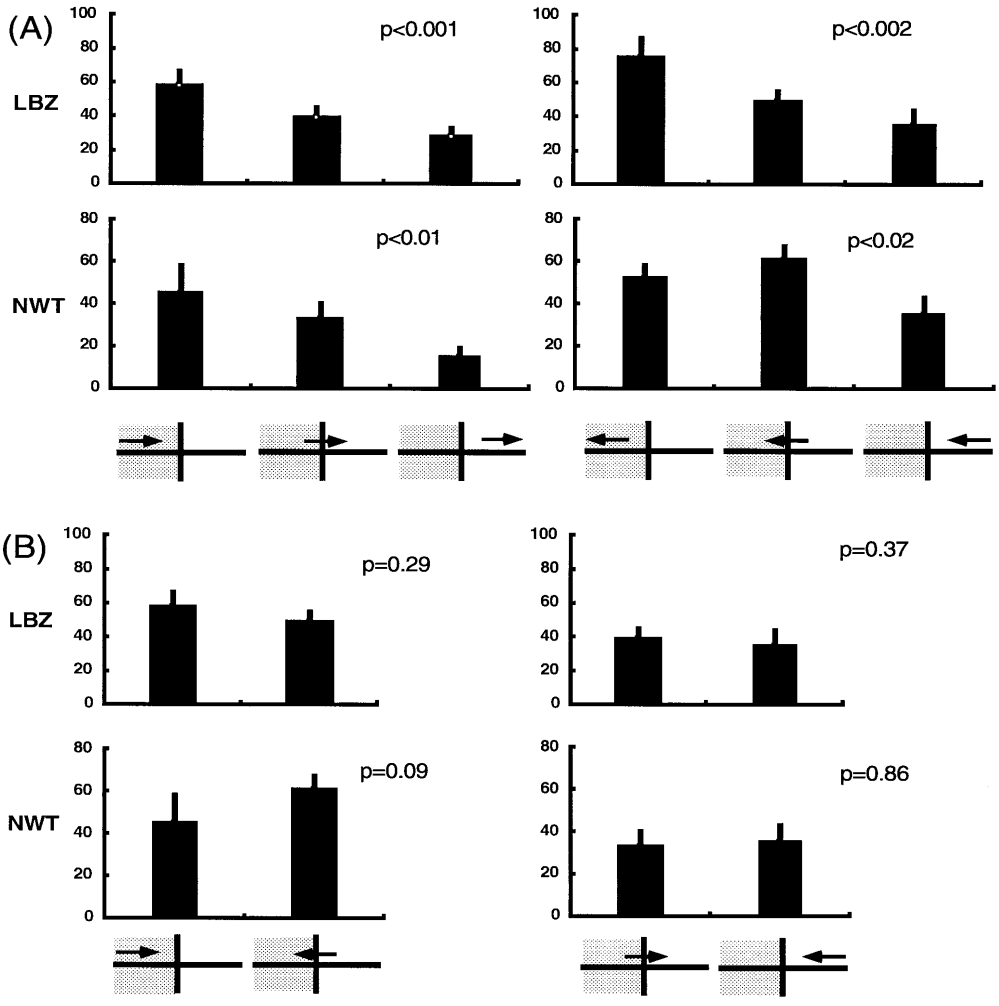

Fig. 4A, B The increase in the latency of the second saccades for both monkeys in the sequential visual and memory saccade task. Each *vertical bar* represents the increase in the latency of the second saccade in each condition averaged from all experiments for each monkey. To compare the data from the two monkeys, note that the data (monkey LBZ in the *upper row* and NWT in the *lower row*) are plotted in the same format with the contralesional field on the left. The schematics at the *bottom* show the direction and end point of the first saccade in screen coordinates. The contralesional field is *shaded*. **A** tested the effect of the end point and **B** that of the direction of the first saccade on the increase in the latency of the second saccade after muscimol injection. The results showed that the latency increase varied significantly with the end point, but not with the direction, of the first saccade. Similar results were obtained for both monkeys. See text for further explanation

Table 1 The mean latency, amplitude, and velocity of the second saccade in the double saccade task. See text for statistics

Saccade direction		Up contra		Up ipsi		Down contra		Down ipsi	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD
Monkey LBZ (R)									
Latency	Control	182	34	174	29	192	33	187	34
	Lesion	228	27	211	30	237	49	213	42
Amplitude	Control	12.3	2.9	11.6	1.8	11.3	3.0	11.2	3.1
	Lesion	12.1	2.2	11.6	2.6	10.9	2.8	10.8	3.3
Velocity	Control	295	30	290	40	204	29	213	35
	Lesion	210	39	315	36	130	67	246	22
Monkey LBZ (L)									
Latency	Control	168	21	163	26	174	25	186	25
	Lesion	234	29	209	28	258	24	250	39
Amplitude	Control	11.8	2.0	11.7	1.6	10.8	2.0	10.0	1.1
	Lesion	12.1	1.6	11.6	1.8	10.6	1.9	10.0	0.9
Velocity	Control	288	32	261	31	209	40	192	30
	Lesion	261	40	238	53	134	19	154	28
Monkey NWT (L)									
Latency	Control	179	30	190	24	216	39	201	34
	Lesion	231	48	224	33	260	51	234	28
Amplitude	Control	12.4	1.7	11.7	2.4	11.0	2.6	10.5	2.7
	Lesion	11.4	1.8	11.5	2.0	10.7	2.8	10.4	2.5
Velocity	Control	287	42	301	39	239	39	224	49
	Lesion	247	34	298	47	189	27	228	50

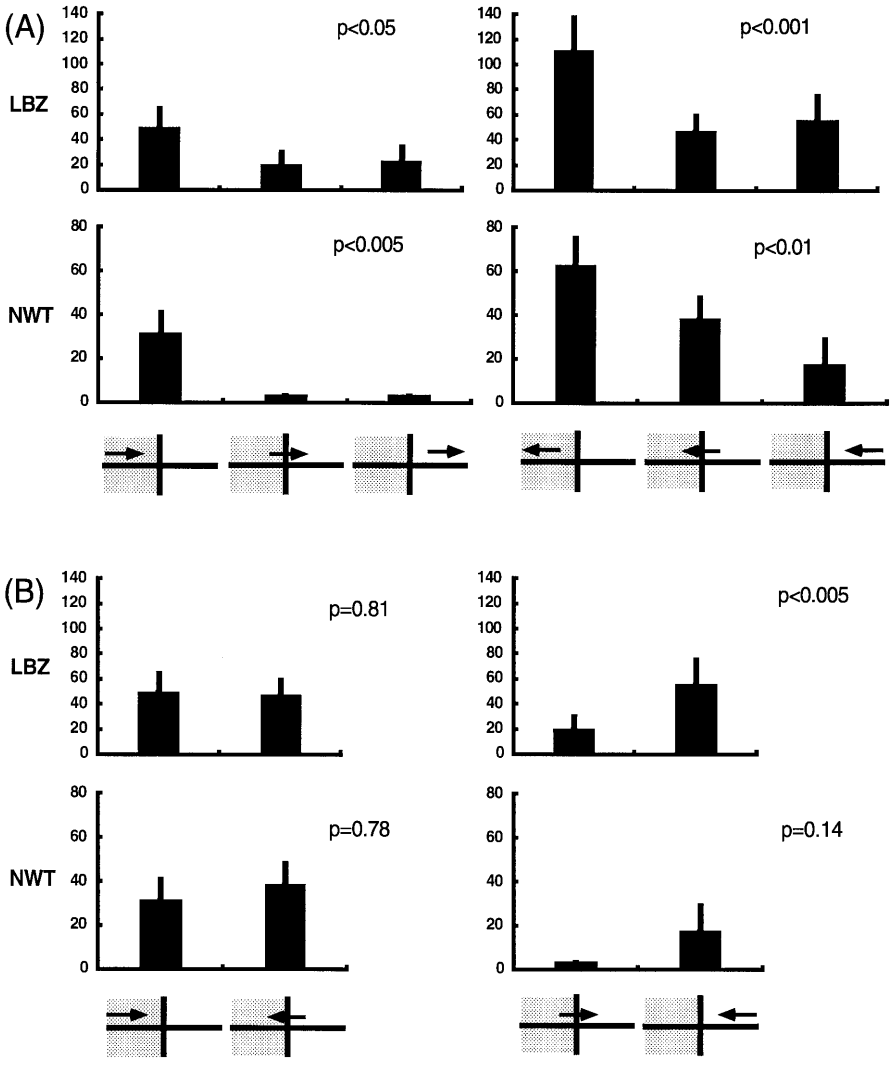

Fig. 5A, B The increase in the error rate after muscimol lesion for both monkeys in the sequential visual and memory saccade task. The arrangement of the data is the same as in Fig. 4. Again, note that the data from the two monkeys are plotted as though the contralesional fields were both on the left. **A** and **B** compared the effect of the end points and directions, respectively, of the first saccade on the increase in the error rate of the second saccade. For both monkeys the increase in the error rate of the second saccade varied significantly with the end point of the first saccade. Furthermore, except for one case in monkey LBZ, the direction of the second saccade did not significantly contribute to the increase in the error rate after muscimol injection

the right hemisphere). Thus, for example, the data of which the first saccades are in the contralesional direction and ending at (15,0) for monkey NWT are now those ending at (–15,0). Figure 4A shows the effect of varying the end points of the first saccade on the increase in the latency of the second saccade, whereas Fig. 4B shows the effect of varying the directions of the first saccade. For both monkeys the increase in latency differed significantly with respect to the end point of the first saccade (Fig. 4A). When the end point of the first saccade was controlled (Fig. 4B), the latency increase was not significantly different between the two different directions of the first saccade.

Error rate

Overall the number of errors in the second saccade increased (from 10.2 to 52.7 errors for monkey LBZ and from 8.2 to 33.8 errors for monkey NWT) after muscimol lesion [$P<0.001$ for both monkeys, results from LBZ(R) and LBZ(L) combined]. Figure 5 shows the results of the ANOVA for the increase in error numbers (with standard errors) averaged across lesions, for monkey LBZ and NWT, respectively. The data were again presented as though the contralesional fields were all in the left and organized in the same format as in Fig. 4. Figure 5A shows the dependence of the increase in the error number on the end point of the first saccade. The left half of Fig. 4A shows the data in which the first saccades are all in the ipsilesional direction but end in different positions, while in the right half the first saccades are all in the contralesional direction. It was found that, in both monkeys, the dependence on end points was significant for both saccade directions. Figure 5B shows the results of an analysis taking the same set of data and examining how the direction of the first saccade might affect the impairment of the second saccade. The end point of saccades were fixed in both cases, at (–5,0) on the left and (5,0) on the right. It could be seen that, except for the one case in monkey LBZ, the direction of the first saccade did not contribute significantly to an increase in the number of errors of the second saccade. Since there was only one direction (either contralesional or ipsilesio-

nal) of the first saccade tested at (–15,0) and (15,0), analyses for the directional effect were not possible at these two locations.

Scatter of the end points

The same analyses were applied to the scatter of the end points of the second saccades. The results showed that the scatter was not significantly different between the lesion and control cases (4.0° for control vs 4.7° for lesion, P=0.36 for monkey LBZ; 4.3° for control vs 4.5° for lesion, P=0.17 for monkey NWT). Furthermore, neither the direction nor the end position of the first saccade contributed to a difference in the scatter between lesion and control. The effects of end position led to P-values of 0.11 and 0.36, for monkey NWT, and 0.21 and 0.19 for monkey LBZ, for ipsilesional and contralesional first saccades, respectively. The effects of saccade direction led to P-values of 0.88 and 0.61, for monkey NWT, and 0.42 and 0.25 for monkey LBZ, for end points of (–5,0) and (5,0), respectively.

Amplitude and direction

Finally, ANOVAs were performed on the amplitude and direction of the second saccades. Since the four second saccades were of different directions and amplitudes (upward saccades tended to be hypermetric and downward saccades hypometric), this analysis was done for each of the four different classes of the second saccade. The results showed that neither the end position nor the direction of the first saccade contributed to an amplitude difference in the second saccade between the lesion and control experiments, and this was true for all four directions of the second saccades (P>0.17 and P>0.37 for all cases regarding upper contralesional saccades; P>0.11 and P>0.36, upper ipsilesional; P>0.29 and P>0.17, lower contralesional; P>0.38 and P>0.18, lower ipsilesional; each for monkey LBZ and NWT, respectively). The lesion also did not affect the direction of the second saccade (P>0.27 and P>0.28 for all cases regarding upper contralesional saccades; P>0.30 and P>0.09, upper ipsilesional; P>0.28 and P>0.07, lower contralesional; P>0.07 and P>0.06, lower ipsilesional; each for monkey LBZ and NWT, respectively).

Double visual saccades

The results from monkey LBZ in the control task of double visual saccades showed that there was an increase in latency of the second saccade after muscimol lesion (P<0.05). However, this latency increase did not depend on either the end point (P=0.33 and P=0.54, for ipsilesional and contralesional first saccades, respectively) or the direction [P=0.16 and P=0.14 for (–5,0) and (5,0), respectively] of the first saccade. Post hoc analysis

Table 2 The error rate, latency, scatter of the end points, and amplitude of the second saccade in the double visual saccade task. See text for statistics

		First saccade contralesional						First saccade ipsilesional					
		(–15,0)		(–5,0)		(5,0)		(–15,0)		(–5,0)		(5,0)	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Error rate (%)	Control	4.4	2.8	5.2	3.3	4.9	2.7			2.2	1.2	4.1	2.3
	Lesion	3.6	1.5	6.1	2.9	5.3	2.1			3.4	1.5	4.5	2.5
Latency (ms)	Control	226	34	217	25	221	31			213	35	209	35
	Lesion	239	40	228	36	243	37			240	37	235	38
Scatter (deg)	Control	0.77	0.19	0.76	0.25	0.65	0.14			0.82	0.28	0.77	0.13
	Lesion	0.71	0.11	0.69	0.11	0.56	0.23			0.74	0.12	0.83	0.19
Amplitude(deg)	Control	9.8	0.4	9.5	0.5	9.6	0.4			9.6	0.4	9.7	0.3
	Lesion	9.5	0.3	9.7	0.5	9.7	0.4			9.6	0.4	9.5	0.5

		(15,0)	
		Mean	SD
Error rate (%)	Control	4.8	2.8
	Lesion	4.5	1.9
Latency (ms)	Control	217	34
	Lesion	232	44
Scatter (deg)	Control	0.76	0.22
	Lesion	0.79	0.20
Amplitude(deg)	Control	9.8	0.6
	Lesion	9.5	0.5

Table 3 The amplitude and latency of single visual and memory saccades from different eye positions (mean ± SD). (*iep* Initial eye position)

	IEP	Monkey LBZ Memory saccade				Monkey NWT Memory saccade			
		Contralesional Lesion/control		Ipsilesional Lesion/control		Contralesional Lesion/control		Ipsilesional Lesion/control	
Amplitude (gain in deg)	(–10,0)	0.80 (0.09)		0.97 (0.07)		0.86 (0.10)		1.01 (0.05)	
	(0,0)	0.82 (0.07)		0.98 (0.07)		0.84 (0.05)		1.03 (0.09)	
	(10,0)	0.82 (0.10)		1.01 (0.08)		0.87 (0.08)		1.06 (0.10)	
	ANOVA	*P*=0.86		*P*=0.73		*P*=0.66		*P*=0.69	
		Control	Lesion	Control	Lesion	Control	Lesion	Control	Lesion
Latency (ms)	(–10,0)	202 (33)	237 (35)	234 (37)	244 (25)	249 (30)	284 (45)	208 (29)	202 (32)
	(0,0)	210 (27)	244 (30)	206 (30)	218 (38)	227 (26)	259 (36)	215 (34)	212 (26)
	(10,0)	230 (36)	277 (49)	198 (32)	208 (24)	216 (22)	245 (38)	235 (40)	247 (37)
	ANOVA	*P*=0.89		*P*=0.87		*P*=0.72		*P*=0.25	
		Visual saccade				Visual saccade			
		Contralesional Lesion/control		Ipsilesional Lesion/control		Contralesional Lesion/control		Ipsilesional Lesion/control	
Amplitude (gain in deg)	(–10,0)	0.97 (0.04)		0.97 (0.05)		0.97 (0.03)		0.99 (0.05)	
	(0,0)	1.01 (0.07)		0.98 (0.05)		0.98 (0.04)		0.98 (0.04)	
	(10,0)	0.99 (0.04)		1.01 (0.06)		0.99 (0.04)		0.99 (0.05)	
	ANOVA	*P*=0.54		*P*=0.69		*P*=0.82		*P*=0.91	
		Control	Lesion	Control	Lesion	Control	Lesion	Control	Lesion
Latency (ms)	(–10,0)	191 (22)	220 (29)	207 (21)	222 (31)	221 (27)	246 (41)	198 (19)	205 (32)
	(0,0)	197 (27)	235 (21)	200 (25)	206 (30)	210 (26)	239 (36)	206 (30)	216 (28)
	(10,0)	216 (30)	248 (44)	189 (29)	191 (29)	201 (35)	229 (29)	214 (39)	219 (42)
	ANOVA	*P*=0.47		*P*=0.33		*P*=0.85		*P*=0.86	

showed that the latency increase in the second saccade occurred primarily in the saccades in the contralesional direction. The metrics of the second saccade in this task, both in terms of the amplitude and scatter of end points, were not significantly different from the control, and no variation was found among different end points (P=0.82 and P=0.86 for amplitude; and P=0.85 and P=0.41 for scatter, for ipsilesional, and contralesional first saccades, respectively) and directions [P=0.95 and P=0.90 for amplitude and P=0.89 and P=0.24 for scatter, for (–5,0) and (5,0), respectively] of the first saccade. Finally, the overall mean error rates were both very low, 4.2% (control) and 4.6% (lesion), and did not vary either with the end position (P=0.67 and P=0.59, for ipsilesional and contralesional first saccades, respectively) or with the direction [P=0.87 and P=0.91 for (–5,0) and (5,0), respectively] of the first saccade. These results are listed in Table 2. Overall, other than an increase in the latency of contralesional saccades, the performance of the second saccades in the double visual saccade task was the same after muscimol lesion.

The effect of initial eye position on single visual and memory saccades

The results of varying the initial eye position for both the visual and memory saccade tasks are shown in Table 3 for both monkeys. Data were taken from four different lesions in monkey LBZ and three lesions in monkey NWT. Since the results were obtained using only one saccade amplitude, we were not able to determine the main sequences of peak velocity versus amplitude of the saccades. The ratio (lesion to control) of the saccade amplitude was calculated for each individual saccade direction in each lesion experiment and the results were averaged for contralesional and ipsilesional saccades in a given lesion experiment. The results shown were means of amplitude ratios across all lesions for different eye positions. The saccade latencies were means for contralesional and ipsilesional saccades from all lesion experiments. Note that the lesion was in the left hemisphere for monkey LBZ and in the right for monkey NWT. A one-factor ANOVA was performed for the amplitude data and a two-factor ANVOA (initial eye position × lesion vs control) for the latency data. The results showed that the initial eye position did not have an effect on the impairment either of saccade amplitude or latency.

Discussion

The most important result obtained in this study is that an impairment in eye position signal delays the processing and increases the errors of the second eye movement in the double saccade task. Moreover, whether extraretinal signals are used for processing eye movements appears to be task dependent. We will first address how the neurons in area LIP form a distributed head-centered representation of target locations in space. We then discuss two neural algorithms for combining retinal information and extraretinal signals to maintain visual stability and how the present study provides evidence favoring an implicit head-centered scheme.

Neurons in area LIP form a distributed representation of the space in multiple coordinate frames

Even though there is considerable evidence that extraretinal signals are important for spatial localization (Bridgeman et al. 1994; Dassonville et al. 1992; Grossberg and Kuperstein 1986; Grüsser 1986; Guthrie et al. 1983; Hansen and Skavenski 1985; Honda 1989, 1990; Howard 1982; Matin 1972; Mays and Sparks 1980, 1983; Skavenski 1990; Sparks 1989; Stark and Bridgeman 1983; Viviani and Velay 1987), evidence about how the combination of retinal and extraretinal signals is implemented in the nervous system has remained elusive until only recently. In the recording studies of parietal areas 7a and LIP, we found that the visual and saccade-related activities of these parietal neurons were oculocentric but their overall magnitudes of response were modulated by eye position (Andersen and Mountcastle 1983; Andersen et al. 1985, 1990). These results suggested that signals in area LIP could provide oculocentric position information. Furthermore, it was proposed that a population of cells with such eye position modulations might also serve to encode target locations in a head-centered coordinate frame (Zipser and Andersen 1988). In contrast to the explicit coding scheme (in which individual neurons have head-centered receptive fields), such a distributed representation can be simultaneously read out in different coordinate frames depending on the needs of particular visuomotor behaviors.

How does such a head-centered mechanism allow the monkey to perform the double saccade task in this experiment or similar paradigms in other experiments? The mechanism could be as follows: when the target is presented while the monkey fixates, information about the retinal location of the target is combined with the current eye position signal to form a distributed head-centered representation of the target location in space. Then with the first saccade planned and executed, new eye position information comes in and is subtracted from the implicit head-centered representation to compute the correct second saccade. Such a neural algorithm requires the use of eye position information to form an intermediate coding of head-centered space. We demonstrated in a modeling study that a push-pull mechanism together with the use of eye position signal was indeed sufficient to allow the network to perform the double saccade task successfully (Xing et al. 1995). After the network was trained to do the double saccade task, excitatory connections were found among units with similar directional tuning and inhibitory ones among those with dissimilar tuning. Such a pattern of neuronal connections locked in the ongoing activity in the network for the first saccade until the move-

ment was made, at which point, with the new eye position integrated, a new pattern of activity developed for the second eye movement. The cells in this network remained largely retinotopic, but with eye position gains, and the second target was updated in eye-centered coordinates after the first eye movement. Thus the network showed similar properties to those recorded from LIP neurons. Together with the results obtained in previous modeling studies (Xing et al. 1994; Zipser and Andersen 1988), this study demonstrated that, in addition to forming a distributed representation of head-centered space, eye position signals could also be used dynamically to maintain spatial stability for second saccades in eye coordinates.

The nature of the extraretinal signal: eye position or eye displacement?

Drawing on recording results from primate frontal eye field (FEF), Goldberg and Bruce suggested a different mechanism in which the computation for the second saccade in the double saccade task could be accomplished (Goldberg and Bruce 1990). In this coding scheme, the efference copy from the first saccadic eye movement was used as a displacement vector, which was reflected in the postsaccadic activity of some FEF neurons. They proposed that the displacement vector was subtracted from the retinotopic memory of the target for the second saccade to derive the correct amplitude and direction of the second saccade. In other words, the computation was achieved through a vector subtraction mechanism in a retinotopic framework and thus neither eye position nor an implicit head-centered representation was necessary.

Additional evidence in favor of the vector subtraction mechanism has been obtained from some clinical studies, in which parietal patients were asked to perform the same double saccade task (Duhamel et al. 1992b; Heide et al. 1995). It was found that, if the first saccade stepped into the contralesional field, the patients made more errors in the second saccade. The interpretation is that parietal patients fail to use the displacement information associated with the first saccadic eye movement to perform the second saccade. Because, in these studies, whenever the patient made a saccade in the contralesional direction, the eye position also stepped into the contralesional field, any information concerning eye displacement and eye position was essentially confounded. Therefore, although these studies are instrumental in documenting the failure of parietal patients to use extraretinal signals, they do not address the nature of this extraretinal information.

The experiments in the present study were specifically designed to test which one of these two hypotheses was correct. In other words, it was clear that efference copy information was required for accurate performance in the double saccade task. However, the nature of the efference copy signal is not clear; i.e., it could be a displacement vector or an eye position signal. Previous studies have indicated that neurons in area LIP contain saccade-related and eye position activities, both of which show a contralateral bias (Andersen et al. 1990; Barash et al. 1991; Lynch et al. 1977; C.-S. R. Li and R. A. Andersen, unpublished data). In other words, the saccade-related neurons generally become active when the monkey makes or prepares to make a saccade into the contralateral field. Similarly, cells with eye position activity generally raise their firing rates when the monkey moves his eyes to the contralateral space and decrease firing when the animal looks to the ipsilateral side. Therefore, when area LIP was lesioned by muscimol and contralesional saccade-related and eye position activities were impaired, the two hypotheses predicted different results for the experiments: If the vector subtraction hypotheses was correct, and since muscimol injection would inactivate neurons subserving saccades in the contralesional direction, whenever the first saccade went in the contralesional direction, the second saccade would be impaired. On the other hand, if the distributed head-centered scheme was correct, as long as the first saccade ended in the contralesional space, the utilization of eye position information would be impaired and the monkey would not be able to perform the second saccade correctly. In this latter case, it would not matter whether the first saccade was in the ipsilesional or contralesional direction. Given that area LIP contains the two extraretinal signals, it is not inconceivable that both an eye-centered and a head-centered representation could be used and be affected by muscimol lesioning. Finally, it is quite possible that the eye position signal is derived by integrating eye displacement signals, in which case the two extraretinal signals may not be entirely independent of one another.

Results in this study showed that it was mainly the end point of the first saccade that determined the performance of the second saccade. Whenever the first saccade ended in the contralesional space, the latency and the number of errors increased in making these second saccades. This was true even when the second saccade was in the ipsilesional direction. Therefore, the results overall favored the distributed head-centered scheme and provide evidence that the efference copy information used in the double saccade task in this study depends on eye position. The large window size used in this experiment may account for the lack of significant difference in terms of the amplitude, direction, and the scatter of the end points between the second saccades in the control experiments and those successfully performed in the lesion experiments. This large window was required because there was a large scatter of second saccade endpoints even in the control condition. Given the limited number of locations where the target of the second saccade was presented and the repetitive nature of the task, it is also possible that the performance of the monkey became somewhat automated and ceased to demand the posterior parietal cortex to recompute the metrics of the second saccade in each trial.

Besides the position-dependent impairment in the second saccade, there was some evidence in the error rates

that a displacement signal might be used for the computation as well. For both monkeys, when the end position of the first saccade was in the "healthy" field, the direction of the first saccades seemed to affect the number of errors the monkeys made in doing the second saccade (even though the results did not reach statistical significance in one monkey). That is, when the first saccade was in the contralesional direction, the monkeys' performance was worse. It thus appeared that a displacement signal might also be used to compute the second saccade, although it played a less important role than the eye position signals. Such a directional effect was not observed when the end points of the first saccades were in the lesioned field, probably because it was overshadowed by the predominant eye position effect. This result is not inconsistent with the hypothesis that both eye position and eye displacement signals are used as extraretinal cues for spatial computations in the posterior parietal cortex.

In sum, the results obtained in this study do not disprove the vector subtraction mechanism. However, they do show that, in the double saccade task employed in this experiment, eye position information plays a more dominant role in achieving spatial stability.

Using extraretinal signals is task-dependent

The results obtained in the first experiment overall support the idea that the extraretinal signal used for computing the second saccade in the double saccade task is eye-position dependent. In a separate set of experiments, we showed that single visual and memory saccades were impaired after lesioning of area LIP. However, varying the initial eye position did not have an effect on the severity of these impairments (similar results have been obtained in the human study by Duhamel et al. 1992b). Therefore, it seems that when the monkeys were only required to make a single visual or memory saccade, a task in which the retinal information of the target location alone was sufficient to compute the eye movement, the eye position information does not enter into the computation.

Altogether the results from these two experiments support the existence of multiple representations of space in the posterior parietal cortex. The computation may be carried out in an oculocentric framework for a task that requires only a simple eye displacement, as in visual and memory saccade tasks. However, in the double saccade task, the data in this study support the notion that the programming of the second saccade requires the use of an eye position signal, and the computation is performed using an implicit, head-centered coordinate frame.

Acknowledgements We thank David Bradley, Maneesh Sahani, and Larry Snyder for discussions, and Ken Grieve, Grace Chang, and Pietro Mazzoni for comments on an earlier version of manuscript. We also thank Jason Liao, Debbie Ward, and Betty Gillikin for technical assistance, and Ken Grieve and Larry Snyder for assistance in histology and many other aspects of the experiments. This work was supported by the National Eye Institute.

References

Andersen RA, Mountcastle VB (1983)The influence of the angle of gaze upon the excitability of the light-sensitive neurons of the posterior parietal cortex. J Neurosci 3:532–548

Andersen RA, Essick GK, Siegel RM (1985) Encoding of spatial location by posterior parietal neurons. Science 230:456–458

Andersen RA, Bracewell RM, Barash S, Gnadt JW, Fogassi L (1990) Eye position effects on visual, memory, and saccade-related activity in areas LIP and 7a of macaque. J Neurosci 10: 1176–1196

Barash S, Bracewell RM, Fogassi L, Gnadt JW, Andersen RA (1991) Saccade-related activity in the lateral intraparietal area II Spatial properties. J Neurophysiol 66:1109–1124

Bridgeman B, Van der Heijen AHC, Velichkovsky BM (1994) A theory of visual stability across saccadic eye movements. Behav Brain Sci 17:247–292

Brotchie PB, Andersen RA, Snyder LH, Goodman SJ (1995) Head position signals used by parietal neurons to encode locations of visual stimuli. Nature 375:232–235

Colby CL, Duhamel J-R, Goldberg ME (1995) Oculocentric spatial representation in parietal cortex. Cereb Cortex 5:470–481

Dassonville P, Schalg J, Schlag-Rey M (1992) Oculomotor localization relies on a damped representation of saccadic eye displacement in human and nonhuman primates. Vis Neurosci 9: 261–269

Duhamel JR, Colby C, Goldberg M (1992a) The updating of the representation of the visual space in parietal cortex by intended eye movement Science 255:90–92

Duhamel J-R, Goldberg M, Fitzgiboon EJ, Sirigu A, Grafman J (1992b) Saccadic dysmetria in a patient with a right frontoparietal lesion. Brain 115:1387–1402

Fuchs AF, Robinson RA (1966) A method for measuring horizontal and vertical eye movement chronically in the monkey. J Appl Physiol 21:1068–70

Gnadt JW, Andersen RA (1988) Memory related motor planning activity in posterior parietal cortex of macaque. Exp Brain Res 70:216–220

Goldberg M, Bruce CJ (1990) Primate frontal eye fields. III. Maintenance of a spatially accurate saccade signal. J Neurophysiol 64:489–508

Goodman SJ, Andersen RA (1989) Microstimulation of a neural-network model for visually guided saccades. J Cogn Neurosci 1:317–326

Goodman SJ, Andersen RA (1990) Algorithm programmed by a neural network model for coordinate transformation. Proc Int Joint Conf Neural Networks, San Diego. Erlbaum, Hillsdale, NJ, pp 381–386

Grossberg S, Kuperstein M (1986) Neural dynamics of adaptive sensory-motor control. Elsevier, North Holland

Grüsser O-J (1986) Interaction of efferent and afferent signals in visual perception: a history of ideas and experimental paradigms. Acta Psychol 63:3–21

Guthrie BL, Porter JD, Sparks DL (1983) Corollary discharge provides accurate eye position information to the oculomotor system. Science 221:1193–1195

Hallett PE, Lightstone AD (1976) Saccadic eye movements towards stimuli triggered by prior saccades. Vision Res 16: 99–106

Hansen RM, Skavenski AA (1985) Accuracy of spatial localization near the time of saccadic eye movements. Vision Res 25: 1077–1082

Heide W, Blankenburg M, Zimmermann E, Koempf D (1995) Cortical control of double-step saccades: implications for spatial orientation. Ann Neurol 38:739–748

Honda H (1989) Perceptual localization of visual stimuli flashed during saccades. Percept Psychophys 45:162–174

Honda H (1990) The extraretinal signal from the pursuit-eye-movement system: its role in the perceptual and the egocentric localization systems. Percept Psychophys 48:509–515

Howard IP (1982) Human visual orientation. Wiley, Chichester, pp 275–340

Judge SJ, Richmond BJ, Chu FC (1980) Implantation of magnetic search coils for measurement of eye position: an improved method. Vision Res 20:535–538

Li C-SR, Mazzoni P, Andersen RA (1995) Reversible inactivation of area LIP disrupts saccadic eye movements. Soc Neurosci Abstr 21:281

Li C-SR, Mazzoni P, Andersen RA (1999) The effect of reversible inactivation of macaque lateral intraparietal area on visual and memory saccades. J Neurophysiol 81:1827–1838

Lynch JC, Mountcastle VB, Talbot WH, Yin TCT (1977) Parietal lobe mechanisms for directed visual attention. J Neurophysiol 40:362–389

Matin L (1972) Eye movements and perceived visual direction In: Jameson D, Hurvitch L (eds) Handbook of sensory physiology, vol 7, part 4. Springer, Berlin Heidelberg New York

Mays LE, Sparks DL (1980) Dissociation of visual and saccade-related responses in superior colliculus neurons. J Neurophysiol 43:207–231

Mays LE, Sparks DL (1983) Saccades are spatially, not retinocentrically, coded. Science 208:1163–1165

Mazzoni P, Bracewell R M, Barash S, Andersen RA (1996) Motor intention activity in the macaque's lateral intraparietal area. I. Dissociation of motor plan from sensory memory. J Neurophysiol 76:1439–1456

McNeil K, Newman I, Kelly F (1996) Testing research hypotheses with the general linear model. Southern Illinois University Press, Carbondale, IL

Platt ML, Glimcher PW (1997) Responses of intraparietal neurons to saccadic targets and visual distractors. J Neurophys 78: 1574–1589

Skavenski AA (1990) Eye movement and visual localization of objects in space. In: Kowler E (ed) Eye movements and their roles in visual and cognitive processes. Elsevier, Amsterdam, pp 263–287

Sparks DL (1989) The neural encoding of the location of targets for saccadic eye movements. J Exp Biol 146:195–207

Stark L, Bridgeman B (1983) Role of corollary discharge in space constancy. Percept Psychophys 34:371–380

Thier P, Andersen RA (1996) Electrical microstimulation suggests two different forms of representation of head-centered space in the intraparietal sulcus of rhesus monkeys. Proc Natl Acad Sci USA 93:4962–4967

Viviani P, Velay J-L (1987) Spatial coding of voluntary saccades in man In: O'Regan JK, Levy-Schoen A (eds) Eye movements: from physiology to cognition. Elsevier/North-Holland, Amsterdam, pp 69–78

Xing J, Stricanne B, Andersen RA (1994) A neural network model for sensorimotor transformation in macaque area LIP. Soc Neurosci Abstr 20:143

Xing J, Li C-S, Andersen RA (1995) The temporal and spatial properties of LIP neurons in sequential eye movements simulated with neural networks. Soc Neurosci Abstr 21:281

Zipser D, Andersen RA (1988) A back-propagation programmed network that simulates response properties of a subset of posterior parietal neurons. Nature 331:679–684