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A saccade is a rapid shift of the position of the eyes (< 100 ms). Saccades are generally considered too quick to be influenced by retinal signals. To address this idea, we displaced the visual target of a rightward horizontal saccade at eye movement onset (when there is suppression of conscious perception). To prevent adaptive and learning effects to occur, jump saccades were always followed by a random series of 10 no-jump saccades. Results indicated that the target jump influenced significantly the amplitude and the peak velocity of the ongoing saccade (opposite effects were found for rightward and leftward jumps). Changes in saccade kinematics occurred as early as 50 ms after the target jump. These results show that retinal information is processed quickly during eye movements, presumably through sub-cortical pathways. *NeuroReport* 14:000–000 © 2003 Lippincott Williams & Wilkins.

Key words: Feedback; Human; Motor control; Retinal feedback; Saccade; Superior colliculus

INTRODUCTION

Saccades are rapid eye movements that shift the point of gaze from one position to another. These movements are known to be very accurate and remarkably quick (20° saccades can occur in < 70 ms [1]). Based on these characteristic features, most researchers agree that saccadic eye movements are ballistic, i.e. independent of retinal feedback. For example, Becker [1] noted that a retinal comparison between the current and desired eye position, even if taken right at the beginning of a saccade, would be too late to influence the course of the saccade. In apparent contradiction with this claim, on-line corrections in saccades were however reported in some studies. For instance, it was shown that the direction or amplitude of visually directed saccades could change in-flight when vertical target steps (22.5°) were delivered during the reaction time of large horizontal saccadic eye movements (50°) [2], or when detectable target jumps were triggered during abnormally slow saccades in spino-cerebellar patients (4–30 times slower than normal [3]). Although widely acknowledged, these data were considered too specific to seriously challenge the canonical idea that normal saccades unfolded uninfluenced by retinal signals.

A puzzling observation questioning the idea that normal saccades could not be corrected on-line was reported by Prablanc and Martin [4]. These authors studied automatic limb corrections to a perturbed stimulus by asking human subjects to look and point to visual targets whose location could change undetectably during the saccade (due to saccadic suppression, subjects were not aware of this jump). The authors noted that the saccadic response was modulated by the nature of the intra-saccadic target jump: the amplitude of the primary saccade was slightly increased or decreased when the target jumped forward or backward, respectively. However, this observation was questioned because of the methodological limitations related to the recording technique (EOG) and because of the characteristics of the task which involved a simultaneous eye-hand movement. A similar modulation of the saccadic response was observed, by our group, in two subsequent experiments using identical experimental designs (unpublished results).

To specifically investigate the possibility that saccadic eye movements can be modified in response to a change of intra-saccadic visual information, we designed an experiment in which: (1) eye movements were recorded with a high resolution technique; (2) intra-saccadic perturbation was not consciously detected by the subjects; (3) intra-saccadic visual perturbation of target position occurred only once every 10 saccades to remove any possible learning effect and to prevent the potential influence of a large post-saccadic error on the characteristics of next saccade (this has been observed in adaptation paradigms; see [5] for a review, [6]); (4) no hand movement was associated with the saccadic response task.

MATERIALS AND METHODS

Behavioral task: Fourteen subjects participated in this study. They were required to look at visual targets presented in their peripheral visual field in an otherwise dark room. Horizontal eye movements were recorded with an infrared optometric system (EyeLink, Ontario; nominal accuracy 0.1°) at a frequency of 250 Hz. Eye velocity was detected on-line using a two-point central difference algorithm [7]. Red light-emitting diodes (LEDs, diameter 5 mm) were located on a semicircle centered on the subjects' cyclopean eye (radius 110 cm). LEDs were placed in a range from −17.5° (left) to 32.5° (right) in 2.5° intervals. Four LEDs were designated as fixation LEDs (−17.5, −10, −5 and −2.5°), whereas the others were considered target LEDs. Each subject performed 200 trials in a single session. Each trial unfolded as follows: (1) the subjects looked at one of the fixation LEDs (illumination period, 2 s ± 300 ms); (2) the fixation LED was turned off and one of the target LEDs was simultaneously turned on (illumination period, 2 s ± 300 ms) signaling the subject to perform a rightward saccade; (3) for the leftward return saccade, the target LED was turned off while one of the fixation LEDs was turned on.

For convenience, trials were segmented into 20 blocks of 10 trials. For each block three types of trials were presented: jump (J), reference (R), or standard (S). Trial 10 was always a J trial. It started with a −10° fixation point followed by an initial target at 20°. During the saccadic displacement, the target location was modified once eye velocity exceeded 30 deg/s. In one group of subjects (G−; $n = 7$) the jump was backwards (20 → 12.5°) while in a second group (G+; $n = 7$) it was forward (20 → 27.5°). Either trial 7 or trial 8 or trial 9 was an R trial (random selection). This R trial was identical to a J trial except that the target was not displaced during the saccadic response. The 8 remaining trials were S trials (considered as distractors from the experimenter's point of view) with single target step varying randomly within a range of 12.5° to 35° (2.5° intervals).

Analysis: Following the experiment, eye position signals were numerically filtered at 30 Hz with a finite impulse response dual pass filter, using 33 coefficients. Eye velocity was computed from the filtered position signal using a two-point central difference derivative algorithm [7]. The beginning and the end of the primary and corrective saccades were automatically detected using a velocity threshold procedure (30 deg/s). The results of this procedure were verified off-line and corrected, if necessary. The main saccade-related parameters analyzed in this experiment were the reaction time, the movement duration (MD), the magnitude and the instant of occurrence of the eye peak velocity (PV and IPV) and the amplitude of the primary saccade (AMP). For each experimental group, a one-way ANOVA with repeated measures was used to test the effect of the intra-saccadic target step (R *vs* J trials) on these parameters. For the sake of simplicity, R−, J− and R+, J+ denote R and J trials recorded in the G− and G+ paradigms, respectively (signs − meaning backward and + meaning forward).

RESULTS

Statistical results and mean values for the main experimental parameters are reported in Table 1.

When questioned at the end of the experiment, only two of the subjects (S1 G+ and S5 G−) were able to report the occurrence of the intra-saccadic target jump. As shown below, the behavior of the two subjects who detected the jump was qualitatively and quantitatively coherent with the behavior of the subjects who had not detected the jump.

At a quantitative level, no significant difference was observed for RT, MD and IPV between R+ and J+ and between R− and J−. In contrast, significant differences were observed for AMP and PV (Table 1). In group G+ (forward jump), an increase in saccadic amplitude (1.2° on average) and in saccadic maximal velocity (14.2 deg/s on average) was observed. In group G−, a decrease in saccadic amplitude (0.5° on average) and in saccadic maximal velocity (8.6 deg/s on average) was detected. As can be seen in Fig. 1, the response to the target jump was very consistent across participants: in all seven subjects, AMP and PV showed an increase in group G+ and a decrease in group G−. Interestingly, the modification of PV in both G− and G+ indicates that the target jump already affected the course of the saccade at IPV. To estimate the latency of the saccadic reaction to perturbation, the delay between target jump and IPV was computed. This delay was equal to 47 ms in G+ and to 52 ms in G−.

Table I. Means and standard deviations of main saccade parameters.

	R+	J+	Statistical results	R−	J−	Statistical results
Reaction time (ms)	234 ± 41	232 ± 41	$F(1,6) = 0.23$; $p = 0.6469$	176 ± 43	185 ± 53	$F(1,6) = 3.97$; $p = 0.0934$
MD (ms)	144 ± 24	146 ± 24	$F(1,6) = 3.19$; $p = 0.1242$	127 ± 14	127 ± 15	$F(1,6) = 0.71$; $p = 0.4331$
IPV (ms)	47 ± 6	47 ± 7	$F(1,6) = 0.00$; $p = 0.9631$	51 ± 6	52 ± 8	$F(1,6) = 0.49$; $p = 0.5093$
PV (deg/s)	412.9 ± 60.7	427.1 ± 60.7	$F(1,6) = 35.83$; $p = 0.0010$*	410.11 ± 24.5	401.5 ± 28.9	$F(1,6) = 10.04$; $p = 0.0194$*
AMP (deg)	28.3 ± 1.7	29.6 ± 1.5	$F(1,6) = 61.60$; $p = 0.0002$*	27.0 ± 1.2	26.5 ± 1.5	$F(1,6) = 10.89$; $p = 0.0164$*

R refers to a reference rightward saccade and J refers to a jump saccade. R+, J+ (forward jump saccade) and R−, J− (backward jump saccade) denote R and J trials recorded in the G+ and G− paradigms. Several saccade parameters were calculated: reaction time, MD (movement duration), PV and IPV (magnitude and instant of occurrence of the eye peak velocity) and AMP (amplitude of the primary saccade).

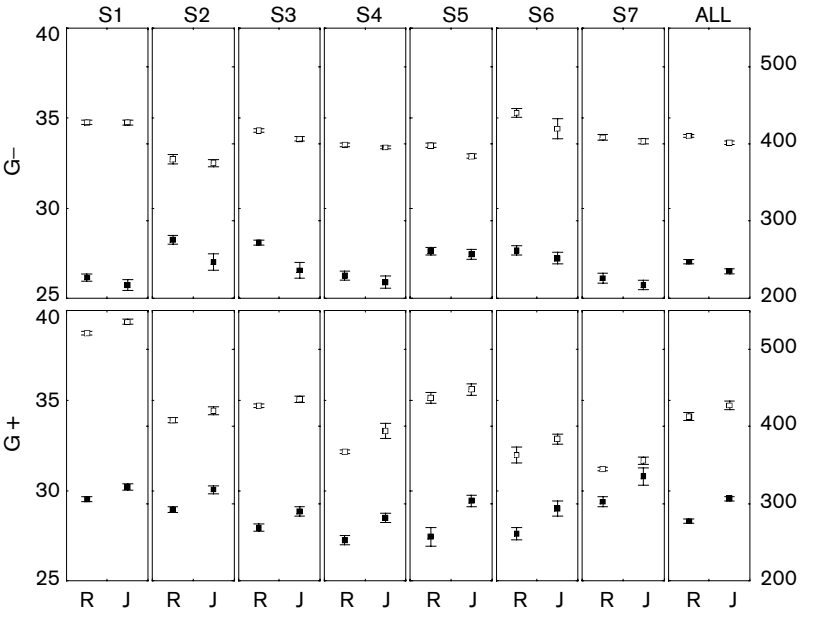

Fig. I. Mean ($\pm$ s.e.) amplitude of primary saccade (black squares) and peak velocity (white squares) for each subject (SI–S7) and group (ALL) for the two paradigms (G− and G+).

Specific longitudinal analyses were carried out to determine whether a learning or adaptive effect could account for our data. The results of these analyses failed to reveal any significant modification of the characteristics of the J saccades over time (i.e. the effect of the target jump was identical for the first and last saccade of the session). Specifically, none of the subjects showed a significant correlation between AMP and the saccade rank (mean r for G+ $= -0.05$, $p = 0.293$; mean r for G− $= 0.008$, $p = 0.866$).

DISCUSSION

The major finding of this study is that both the amplitude and the peak velocity of the ongoing saccadic response can be modified on-line when the target is displaced at the time of saccade onset. This result indicates that the oculo-motor system is able to detect the target jump in the J trials and to use this information to rapidly modify the oculomotor response.

Previous studies have shown that visual processing occurs during the planning phase preceding saccadic displacement [2,8] or during the saccade itself when it is abnormally slowed in spino-cerebellar patients [3]. Typically, these modifications were not thought to reflect the modulation of the amplitude of the ongoing saccade. They were rather considered as the expression of the ability of the motor system to generate secondary corrective saccades added to the ongoing one, without refractory period.

Our data extend and generalize the previous observations by showing that small but significant in-flight changes in saccadic trajectory: (1) can be elicited by unconsciously detected visual stimuli, (2) may occur during the acceleration period (time from target jump to IPV), i.e. within a mean latency of 50 ms, (3) affect the saccadic velocity without modifying the saccadic duration, and (4) are specific of the direction of the intra-saccadic jump. The idea that the constant rank, in the sequence of trials, of the J+ or J− saccades was unconsciously detected by the subjects cannot be formally rejected. However, this factor cannot account for our results. Indeed, longitudinal analysis failed to reveal any trend for the J+ or J− saccades to change over time (i.e., the effect of the jump was not different for the first and last J saccades of the sequence), as would have been expected if the subjects had learned progressively the rank of the J saccades.

Although the anatomical substrates of these rapid eye trajectory modifications remain to be identified, the rapidity of the response might suggest a subcortical route. A first candidate pathway involves the superior colliculus (SC), a structure that has been demonstrated to be involved in the control of saccadic eye movements [9,10]. The SC receives visual information directly from the retina and indirectly from the cortex. It projects to the ponto-medullary reticular formation where the saccadic pulse generator for horizontal saccades is located. Some neurons located in the deep layers of the SC both respond to the presentation of a visual target and exhibit a burst of activity prior to the execution of saccades. Data collected in the cat suggest that the minimum visuo-motor delay is close to 60 ms. This value is computed from adding the delay of the visual response (mean of the earliest response latency 47 ms [11]) to the minimum latency of saccadic modification following abrupt changes in deep SC activity (10 ms [12]). In the monkey, the delay of the visual response is either of similar magnitude (51 ms [13]; 40–50 ms [14]) or slightly longer (70 ms [15,16]). Although these values slightly exceed the saccade modification delay estimated in our study, it is important to note that the collicular visual response latencies have always been tested with a spot of light presented to stationary eyes. In contrast, in our study the eyes were moving at 30 deg/s and quickly accelerating at the time the second target was presented.

The SC visual response to such a stimulus sweeping quickly on the retina might be shorter. If such is the case, one might speculate that the sudden jump of the target location at saccade onset triggers a secondary burst of activity in the SC, resulting in a specific change in the metrics of the ongoing saccade. The existence of alternative routes to this direct retino-collicular-pontine pathway can of course not be rejected at this point. Given the very short reaction time to the target jump and the fact that the visual stimulus swept on the retina during the saccade acceleration phase, one may suggest, for instance, a contribution of the nucleus of the optic tract [17] to the rapid saccade modification that was demonstrated by our study.

The observation that the amplitude of the ongoing saccade can be modified on-line by the retinal input contrasts with the classical view of physiological oculomotor control, which denies any retinal feedback action during a normal saccade particularly during its acceleration phase (see Introduction). The small extent of the saccadic modifications observed in the present study (0.5–1.2° compared with the 7.5° jump) might explain this discrepancy. Indeed, our data show that both a specific protocol and a very accurate eye movement recording system are necessary to unravel the phenomenon of saccadic flexibility. Beyond this remark, the functional purpose of the fast on-line saccadic adjustments observed in the present study remains puzzling. With respect to this issue it may be emphasized that saccadic flexibility seems to represent a robust phenomenon: we have now observed it repeatedly in different subjects groups, with various paradigms and with different eye movement recording techniques (present study, [4], Desmurget *et al.* unpublished data). These different studies, and in particular the present one, have indicated that the in-flight saccadic changes, although small when compared to the initially intended saccade, are specific to the direction of the target step and do not simply represent a default response to the target perturbation. Even if the amount of saccade flexibility (about 1°) may appear limited in magnitude, it is far from negligible in the context of normal saccades performed toward stationary objects. Thus, we propose that in-flight modifications of the saccadic amplitude represent a as yet unidentified corrective process modulating saccade accuracy. To test this original hypothesis, other experiments with varied intra-saccadic target step amplitudes and directions as well as varied saccadic amplitudes could be used. There should be a saccadic amplitude (and duration) under which the afferent and efferent delays become too long to produce any effect on the ongoing saccade. In the same vein, one may expect large saccadic shift to trigger larger kinematic modifications, as those observed by Prablanc and Martin [4]. Finally, removing the retinal sweep during the saccade could produce a slighter amplitude correction as compared to a normal saccade towards a continuously lit target.

In conclusion, our data show that retinal information can influence the ongoing saccadic response. Further experiments remain to be carried out to determine the exact functional role of this fast feedback loop and to unravel its anatomical substrate.

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