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CORRECTIVE SACCADES: DEPENDENCE ON RETINAL REAFFERENT SIGNALS[1]

C. PRABLANC and M. JEANNEROD
Laboratoire de Neuropsychologie Expérimentale, Unité U 94 INSERM, 69500 Bron, France

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Abstract—The saccadic response to a peripheral step stimulus is composed of a main saccade, and a corrective saccade with a shorter latency. When a single peripheral pulse stimulus is presented with a duration shorter than the latency of the response, the main saccade is not followed by a corrective one, though it is inaccurate. However when a second pulse synchronized to the first saccade is presented within some degrees around the new visual axis, it elicits a saccadic correction with a short latency. If the second pulse is presented at a larger retinal eccentricity, the saccadic correction is performed with a normal latency.

The corrective saccade mechanism can be interpreted as a by-pass of decision time at the end of the main saccade if the residual retinal error does not exceed some degrees.

INTRODUCTION

Any model of saccade initiation must consider the existence of corrective saccades, which occur in most cases when the target step amplitude exceeds 10° of arc. Their amplitude represents typically 10 per cent of the step amplitude (Becker, 1972), and their latency is smaller (by about 50 per cent) than that of the main saccade. This difference in latency is not predicted by the sampled-data models, if it is thought that the corrective saccade is due to a new sampling of the image position error at the end of the main saccade. Since sampling can only begin at the end of the main saccade the corrective saccade should be delayed at least by 200 msec. For this reason, Becker and Fuchs (1969) have proposed that the corrective saccade is "preprogrammed" together with the main saccade, thus allowing a reduction of computation and decision time. In the present study, however, we could not observe corrective saccades when visual input was no longer present at the end of the main saccade, although they were systematically elicited even by a very brief restoration of visual input. Furthermore we investigated the necessary conditions for the occurrence of corrective saccades.

METHODS

Normal subjects participated on these experiments. They were seated at a distance of 30 cm from a circular screen. Head movements were restricted by a chin-holder and a forehead rest. Experiments were performed with monocular viewing of the left eye, in complete darkness. Subjects were allowed to dark adapt for about 10 min.

The circular screen was equipped with gallium arsenide phosphide diodes, emitting in the red spectrum (6500 Å) with a rise time of 50 nsec. Red lights were chosen because perceptual threshold for this wavelength is practically constant over the retinal surface, up to 40° from the fovea (see Pirenne, 1972). A preliminary curve of saccade latency to stimulus intensity was constructed. Latency decreased down to a minimum with increasing intensity. In the present experiments, intensity was set at a value corresponding to a latency slightly above this minimum (about 10 per cent). The luminous surface of the diodes subtended a 0·5° solid angle at the eye. One of the diodes represented the central fixation point (LC), the peripheral diodes (LP) being at 5, 10 and 20° on each side in the horizontal plane. Diodes were energised via a logic circuit which allowed randomized presentation of the peripheral targets.

Eye position in the horizontal plane was recorded with an opto-electronic device. The left eye was illuminated by an infra-red source on both the nasal and temporal side, and the image of the eye was projected through a lens on two photo-transistor arrays mounted differentially; this set-up allowed measurement of the position of the border between iris and sclera to be made without drift (Masse, 1971). Accuracy of measurement of eye position was ± 20′ over the full range. During calibration, a graph of eye position against voltage was constructed for each subject, before and after testing. If the two calibrations differed, head displacement had occurred during the experiment, and the results were rejected.

Records were taken with a d.c. ink-jet writer with a 700 Hz cut off frequency, and with a paper speed of 100 mm/sec.

RESULTS

Experiment 1

Five subjects were tested. Steps, and pulses from 20 msec to 200 msec in duration were applied to the peripheral targets. The central fixation spot was always turned off when the peripheral target was turned on, and was kept off for 1 sec. Main saccade latency (T_1) was measured from each pulse presentation (single

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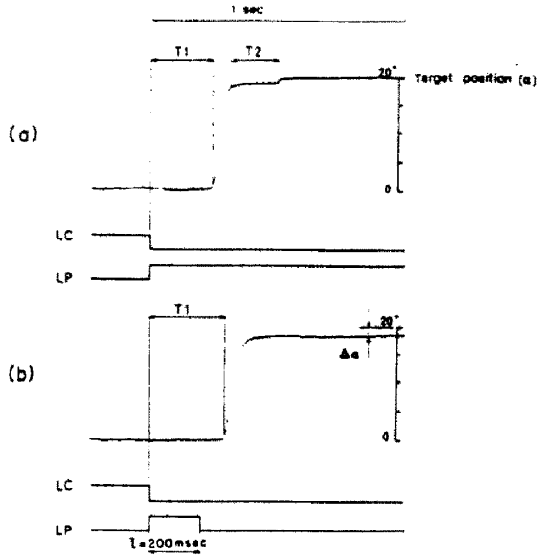

Fig. 1. Saccadic responses in a step situation (a), and a single pulse situation [(τ = 200 msec) (b)]; LC: central fixation point; LP: 20° nasal target. T_1 is the latency of the main saccade with respect to the onset of LP. T_2 is the delay between the end of the main saccade, and the beginning of the corrective saccade. Note absence of corrective saccade in the single pulse situation, and the persistent residual retinal error ($\Delta\alpha$), at the end of the main saccade.

pulse situation) with respect to the onset of the corresponding saccade. The angular error ($\Delta\alpha$) between eye position at the end of the main saccade and target position, was also measured [Fig. 1(b)]. For presentation of steps (step situation) the same measures were taken [Fig. 1(a)]. Each peripheral stimulus was given three times; all targets were presented. Stimulus presentation was randomized.

Pulses at any target location and of any duration (as short as 20 msec) elicited a saccadic response in more than 90 per cent of cases. After the main saccade, eye position was maintained for about 400 msec. In some subjects, however, a slow drift towards resting position was observed.

Angular error ($\Delta\alpha$) varied with pulse duration. For a given target location, it increased when pulse duration was decreased. For instance, for a 20° nasal target, the error was about 1° when pulse duration was 200 msec and increased up to 3° for a 20-msec pulse duration.

As regards relations of angular error with target location, the error increased systematically with the eccentricity (see also Becker, 1972). This effect occurred in both nasal and temporal fields and was more marked with short pulses than with steps (see Prablanc and Jeannerod, 1974).

For any given target location, and any pulse duration, no correlation between amplitude of the error and latency of the saccade was found. For instance, the linear correlation coefficient was $r = 0{\cdot}33$ (n.s.) for a 20° nasal target displacement (100 msec pulse), and $r = 0{\cdot}18$ (n.s.) for the same target with a step.

In the experiments reported here, which involved 300 stimulations with pulses of different duration (20–200 msec) and at different locations, only three corrective saccades were found. Conversely, with step stimuli, corrective saccades appeared in all cases, when the main saccade was inaccurate, i.e. for target locations beyond 5°. The latency (T_1) of the main saccade was 217 msec (S.E. = 6·9), while delay (T_2) between main and corrective saccades was 190 msec (S.E. = 8·2), significantly shorter than T_1 ($P < 0{\cdot}1$; Fig. 3). Delays reported in the literature for corrective saccades are usually shorter (125–150 msec according to Bartz, 1967; Becker and Fuchs, 1969). This difference might be explained by individual factors and by computational procedures: for instance, in our experiment, occasional delays as long as 300 msec were taken into account for the computation of T_2.

Experiment 2

This experiment was aimed at the prerequisites for the occurrence of corrective saccades. Subjects were

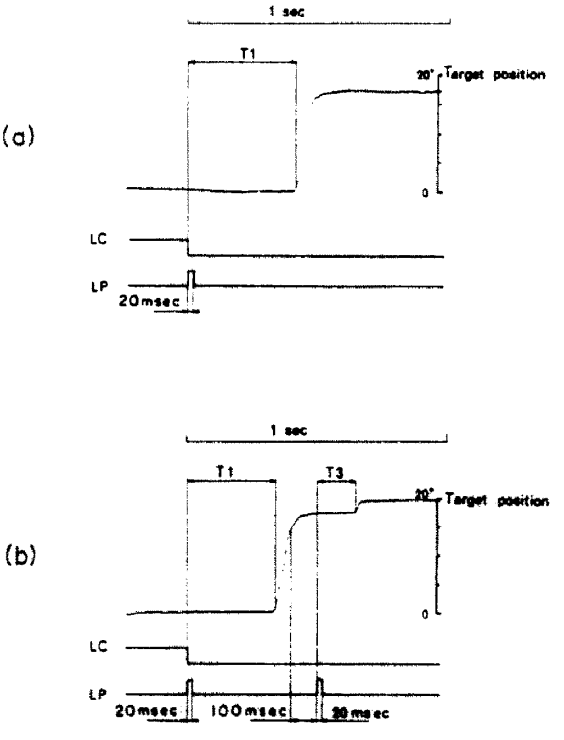

Fig. 2. Comparison of saccadic responses in a single pulse situation (a), and a double pulse situation (b). Pulse duration: 20 msec. Target location: 20° nasal. (a) To be compared with Fig. 1(b). Note more important angular error related to a shorter pulse duration. (b) The second pulse is delayed by 100 msec from a triggering point corresponding to about the 2/3 of the main saccade amplitude. Note occurrence of a corrective saccade with a latency T_3 shorter than T_1.

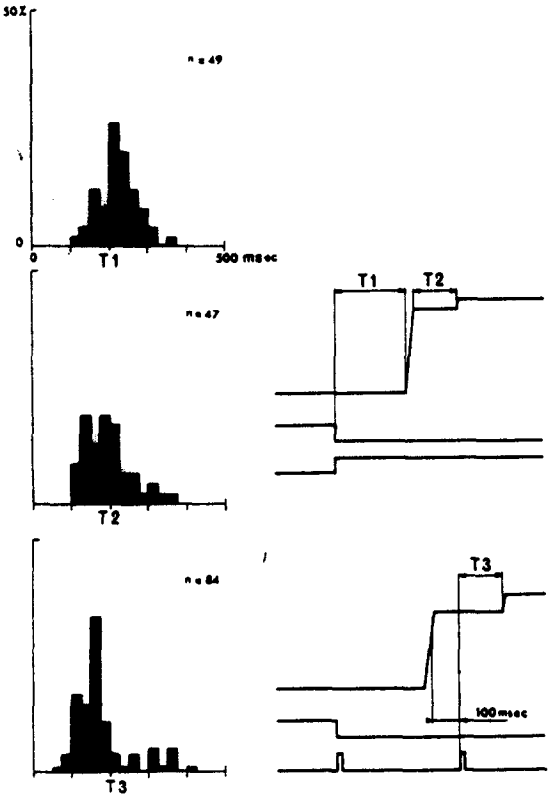

Fig. 3. Histograms of latencies T_1 and T_2 in the step situation, and latency T_3 in the double pulse situation. Values from 10° nasal and temporal, and 20° nasal targets have been pooled.

presented with pulse stimuli at any LP location, in random order. In this case the main saccade could trigger off a second pulse of the same duration, at the same or at a different LP location (double pulse situation). This second pulse could be delayed by up to 100 msec from the triggering point. Latency (T_1) of the main first saccade and latency between the second pulse and the following saccade were measured.

(1) *Double pulse at the same target location.* Five subjects were tested. Stimulus duration was fixed at 20 msec. As in Experiment 1, corrective saccades were almost never observed when a single pulse was presented [Fig. 1(b), 2(a)]. However, when the second pulse occurred within a delay of more than 50 msec after the main saccade, a secondary saccade of small amplitude was observed. The secondary saccade was "corrective", i.e. completed exactly the amplitude of the main saccade, so the eye position became concordant with the target location. This effect was less constant when the second pulse was within 50 msec from the end of the main saccade.

Latency (T_3) between the second pulse and the onset of the corrective saccade was averaged over 84 trials for the 10 and 20° peripheral targets. It was found to be 176 msec (S.E. = 8·4), i.e. significantly shorter than latency T_1 ($P < 0{\cdot}01$), [Figs. 2(b) and 3].

(2) *Double pulse at two different locations.* Ten other subjects were tested. Pulse duration was fixed at 50 msec or at 200 msec for both pulses, and delay between the first saccade and the onset of the second pulse was fixed at 50 msec. Pulses were presented at 5, 10, 12, 20 and 22°, with seven combinations of double pulses intermingled in a random order (10L–12L, 20R–10R, 22R–20R, 10L–10R, 12L–10L, 20R–22R).

As regards latency we could separate secondary saccades into two classes over the 240 trials:

(a) Saccades corresponding to stimuli of small eccentricity, (i.e. in the parafoveal retinal region). For a 22° LP following a 20°LP, for instance [Fig. 4(a)], secondary saccades were corrective and had brief latencies (T_4 = 152 msec, S.E. = 3·6), significantly shorter than corresponding latencies T_1 ($P < 0{\cdot}001$), (Fig. 5). When the second pulse was nearer the central fixation point than the first (for instance 20° following 22° or 10° following 12°) secondary saccades did not occur systematically, since the main saccadic response undershot and thus the visual axis could match the location of the second target. However, when secondary saccades

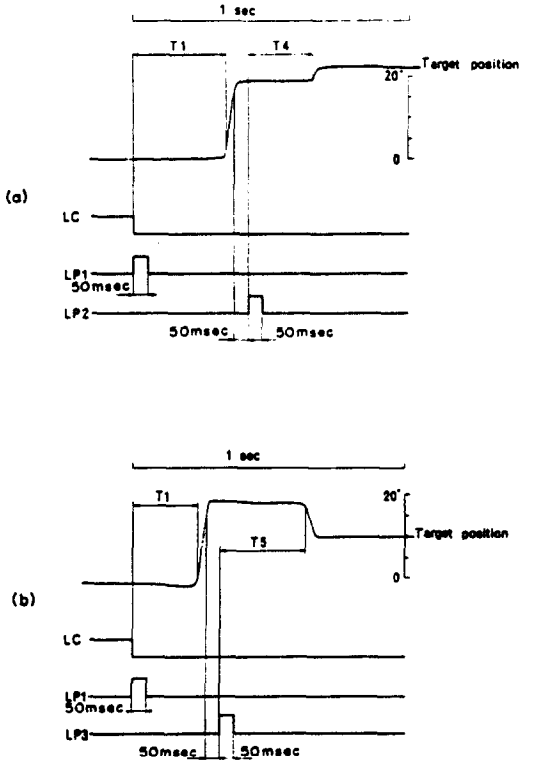

Fig. 4. Comparison of saccadic responses in two double pulse situations. Pulse duration: 50 msec. The second pulse is delayed by 50 msec from the main saccade. Target locations: (a) LP1: 20° nasal; (b) LP1: 20° nasal; LP2: 22° nasal; LP3: 10° nasal. Note that the latency T_4 of the secondary saccade is shorter than T_1 for a small retinal error; while T_5 is longer for a large retinal error.

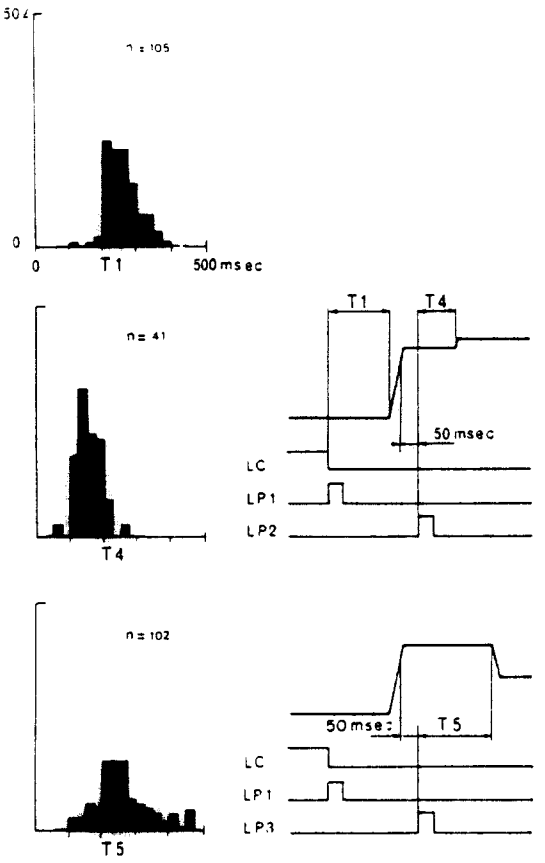

Fig. 5. Histograms of latencies T_1, T_4, for a 20–22° nasal target locations, and of latencies T_5 for combinations of more distant targets. Note that latency T_4 is much shorter than T_1 and T_5.

occurred they were corrective and had latencies T_4' very similar to T_4 (T_4' = 158 msec, S.E. = 7).

(b) Saccades corresponding to large eccentricities, i.e. to stimuli falling on a peripheral area of the retina at the end of the first saccade [Fig. 4(b)]. These saccades had latencies (T_5 = 275 msec, S.E. = 10) which were slightly longer than T_1. Comparison of latencies T_5 (Fig. 5) with latencies T_4 or T_4' obtained with stimuli of small eccentricity shows a significant difference ($P < 0.001$).

DISCUSSION

In a previous study with saccades of large amplitude, Becker (1972) observed frequent corrective saccades in single pulse situations. Only when the same target was presented repeatedly without being intermingled with other targets were we occasionally able to observe corrective saccades in the single pulse situation. However this fact cannot explain the discrepancy between Becker's study and ours, since target presentation was randomized in both experiments. One explanation could be that in Becker's experiment, after a pulse was presented, the same peripheral lamp was turned on: the subject thus could know that his saccade was systematically "hypometric". This condition could have introduced a cognitive factor, leading to a corrective mechanism. In our experiment, corrective saccades could be systematically elicited by a second pulse at the same location, and synchronized with the end of the main saccade. Absence of corrective saccades in the single pulse situation, and occurrence of corrective saccades when a second pulse is given after the main saccade, suggests that the visual input resulting from a saccade (reafferent input) is a necessary condition to bring the fovea at the target location, provided the subjects do not know the target position. Each corrective saccade is thus programmed individually, as a discrete correction of retinal error. Becker and Fuchs (1969) found "corrective saccades" with short delays when asking a subject to reproduce in the dark saccades learnt with luminous targets. In this situation, the previously stored position of the target could act as an internal signal, thus explaining the shorter latencies.

Latency T_5 of the corrective saccade with respect to the second pulse was shorter than latency T_1 of the main saccade and was in the same range (or shorter) than delay T_2, observed for corrective saccades in the step situation. This difference between T_1, on one hand, and T_2, T_3, on the other, might be explained by the shortening, or the absence of "decision time" in the sequence of events which lead to a corrective saccade. Becker and Fuchs (1969) suggested that the main and corrective saccade could be programmed together, the role of the reafferent signal at the end of the main saccade being to allow, or to cancel the execution of the preprogrammed corrective saccade if the error is too large or in a direction opposite to that expected. In their step–step experiment (40–45°) very similar to our pulse–pulse experiment, these authors found a latency of 228 msec for saccades to the second (45°) target. This value is that of main saccades, and not of corrective saccades. Considering that the main saccade brings the eye at a position which corresponds to about 90% of the step amplitude (in this case, approx 36°), the second step at the 45° target actually represented a retinal error of about 9°. In our double pulse situation, for a 20–22° double pulse, the main saccade had an amplitude of about 18°, so that the retinal error of the second pulse was only 4°, and the saccade which followed had a latency of a corrective saccade. This difference in magnitude of the error signal at the end of the main saccade may explain the difference in latency between the two situations. In addition a short latency was also found when the second pulse was presented in an opposite direction to the initial saccade, and with a small retinal eccentricity (approximately 2°). Small retinal error, however, is not sufficient *per se* to explain shorter latencies. Saccades in response to small initial target step (0·5–2°) may have latencies as long as 250 msec or more (Komoda, Festinger, Phillips, Duckman and Young, 1973; Wyman and Steinman, 1973).

In our experiment, latency of saccades to a 5° initial stimulus was 202 msec.

These results indicate that the modification of the response characteristics of the saccadic system occurs only when two conditions are fulfilled: that a previous saccade has been made and that the residual retinal error is of small amplitude, i.e. within approximately 4°, without respect to its direction. If the error is too large a new decision has to be made, resulting in an increase in latency of the secondary saccade, in the same range as the latency of an initial saccade. Faster decision would be more readily explained by the intervention of efferent signals, resulting from the command of the main saccade. These signals could operate as an "internal feedback" at the level of the centres where the saccadic decision is taken. This operation could allow a by-pass of the normal pure decision delay, and thus an immediate computation of the residual retinal error, if it does not exceed a certain value. A similar hypothesis has been advanced by Robinson (1973) in a theoretical model. Another possible explanation, by analogy with computers, could be that in all cases, at the end of a saccade, a standard subroutine is started. If the error is too large it cannot be treated by this subroutine and the general routine of saccadic initiation has to be called in. In this case a time consuming process could not be located in specific decision "centres". It is hardly conceivable presently to choose between these two "models" by psychophysiological studies only, and further neurophysiological investigations are required.

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