# SACCADIC EYE MOVEMENTS TO FLASHED TARGETS

P. E. HALLETT and A. D. LIGHTSTONE

Department of Physiology and Institute of Biomedical Engineering, University of Toronto, Toronto M5S 1A8, Ontario, Canada

# (Received 4 March 1975)

Abstract—A target presented as a flash in darkness, before, during or after a saccade, elicits a subsequent goal-directed saccade of normal amplitude and appropriate latency. In a flashed target variation of the Wheeless paradigm, "cancellation time" is not observed in circumstances where the first target is believed to be ineffective. Latency is approximately the same whether target steps are synchronized to saccades or not. Little or no processing for a primary saccade occurs before the prior primary saccade.

# INTRODUCTION

In the previous paper (Hallett and Lightstone, 1976) the *beginning* of a saccade triggers the exposure of the fixation target at a new position and its extinction 1-300 msec later. Surprisingly, the duration of the exposure has no effect on the accuracy of the eventual saccadic response, although exposure duration substantially affects the length and amount of light in the retinal image. This paper presents more evidence that saccades are towards the *physical* positions of targets—which is only possible if retinal image position and eye position information are correlated.

Sections in small type deal with saccade latency and may be read separately.

#### METHODS

The present experimental arrangements and subjects are described in Hallett and Lightstone (1976) and Lightstone (1973). An oscilloscope spot (very fast decay P15 phosphor), viewed through a low power microscope of large exit pupil, is stepped and lit by a PDP8 computer according to times and positions randomly selected from tables. A near i.r. image of the eye (sharp band pass 800-1100 nm) falls upon a bisected horizontal slit, each half of the slit being imaged by microscope objectives on special high impedance diodes. As the eye rotates, the image of the large black pupil shifts on the slit and causes reciprocal changes in the lengths of the images on the diodes which, after electronic amplification and filtering, give a voltage proportional to the sine of eye rotation in the range  $\pm 15^\circ$ . The S.D. of the system noise is 3', and tests show that the deep dental impression is very effective in maintaining head position. The eye tracker is insensitive to change in pupil size or to vertical eye movement. Saccade latency and amplitude are read from Brush 260 chart paper (125 mm sec<sup>-1</sup>) to an accuracy of  $\pm 4$  msec and  $\pm 12$ . The beginnings and ends of saccades are determined from expanded velocity traces.

#### Trials with two cues

In the double true and partial cue trials (Figs. 2 and 3) there is usually a single saccade in the dark period. In 06 of trials the saccade can be confidently associated with either cue 1 or in cue 2 because it follows *only* one cue with normal (150-350 msec) latency. In the remaining trials a saccade is simply allocated to the cue which precedes it by most nearly 256 msec. In the false cue trials

(Figs. 4 and 5) saccades can be allocated to cues by direction and, because of the falseness of the cue, saccades to cue 2 can be recognized long after the final re-lighting of the target, e.g. Fig. 4, P10  $\bullet$  max. Such saccades cannot be recognized for P1 to P8, unless they occur immediately after re-lighting (e.g. Fig. 3, P5  $\circ \blacktriangle$  min.).

# RESULTS

#### Cues during saccadic velocity peaks

The otherwise fully dark-adapted subject fixates the 2 log supra-foveal threshold, nominally 8' subtense, blue-green target, and follows its subsequent instantaneous steps as best he can. After a random delay the target steps 7.65°, left (-) or right (+), to elicit saccade  $S_0$ , which triggers the subsequent randomly selected pattern of target motion and lighting. When saccade  $S_0$  reaches a velocity of about 37° sec<sup>-1</sup> (after about 8-12 msec and 6-10' into the movement) it initiates several actions. The target is (1) blanked out for 10 msec, (2) re-illuminated for  $\Delta = 20$  msec at a position randomly selected from  $\pm 3.83$  and  $\pm 11.5$ deg, (3) blanked out for 340 msec, and (4) re-illuminated for 750 msec before returning to the instrument axis. The net result is that the  $\Delta$  cue is exposed only from the time that the eye has accelerated to about 330° sec<sup>-1</sup> until it decelerates to about 295° sec<sup>-1</sup>.

Figure 1 illustrates the variety of trials, ignoring mirror image types. The common response to the intrasaccadic cue is a saccade  $S_1$  of normal latency, usually occurring in the dark towards the position of the invisible target. The second record in Fig. 1 is of interest. Although the 20 msec cue illuminates a retinal track of roughly 3°-4° length, which includes the fovea in this case, there is a subsequent saccade  $S_1$  towards the position of the invisible target. This would not be expected if retinal image position were the sole information used, but is consistent with saccade  $S_1$  making allowance for eye movement during saccade  $S_0$ .

Latencies. As a control the latency from a continually-lit target step of  $2^{\circ}-12^{\circ}$  to the resulting saccade has mean 263 msec (S.D. = 39), and is never shorter than 150 msec. In the trials where the cue is crossed with respect to the saccade  $S_0$  (Fig. 1, bottom 3 patterns)  $17/84 S_1$  saccades



Fig. 1. Presentation of a cue during peak velocity section of a saccade. The position of the cue is indicated by the centre of the triangle  $\blacktriangle$ . Blanking out of the target is indicated by absence of the dotted target trace. The time mark of 150, 250 and 350 msec corresponds to the lower limit, mean, and upper limit of primary saccade latency. The interval between the eye and target position times is retinal position relative to the foveal line-of-sight. Subject PEH.

occur more than 150 msec after the final re-lighting of the target—the earlier analysis (Hallett and Lightstone, 1976) suggests that these 17 saccades are not responses to re-lighting but are very late responses, due to the reduced luminous energy of the crossed intrasaccadic cue. The remaining 67.84 saccades have a normal latency of mean 248 msec (S.D. = 48 msec) relative to the beginning of the intrasaccadic cue. However, in all 26/26 trials with the *uncrossed* cue (Fig. 1, top) the  $S_1$  saccade occurs after the end of blanking. In 21/26 trials the  $S_1$  are evenly distributed from 6 to 134 msec after blanking, and are thus too early to be responses to re-lighting, but must be long latency responses (mean 439, S.D. = 47 msec) to the intrasaccadic cue. The remaining 5/26 delayed  $S_1$  saccades occur more than 150 msec after the end of blanking, and may be due to the re-lighting, the uncrossed  $\Delta$  cue being relatively ineffective on occasions (Hallett and Lightstone, 1975).

Amplitudes. In Fig. 6 the amplitudes of the  $S_1$  saccades that occur during blanking (Fig. 1, bottom 3 patterns), or just after ( $\leq 150$  msec) the re-lighting of the target (Fig. 1, top), are plotted as (I) against the error between eye position and target position that remains to be corrected at the start of saccade  $S_1$ . A regression line through the four points passes through the origin. The four points also fall close to the regression line shown, which is fitted to the data of the next experiment in which the eye is stationary at the time of the cue. Primary saccades are proportional to what needs to be corrected. Retinal image position is not the sufficient stimulus for saccades to flashed targets-appropriate allowance is also made for saccadic movement in the latent period between cue and response.



Fig. 2. Presentation of two cues: cue 1 is at the beginning of triggering saccade  $S_0$ ; cue 2 follows after a random dark interval of 20-200 msec. P1-P4 are the double true cue varieties of target position pattern. In the left margin O denotes saccade  $S_1$  to cue 1,  $\bullet$  is saccade to cue 2, cue 1 having been missed, and  $\blacktriangle$  is saccade  $S_2$  to cue 2 following an  $S_1$  saccade to cue 1. Max means common response pattern, maj less common, min rare.



Fig. 3. The P5-P8 partial cue varieties of target position pattern.

#### Trials with two cues

Arrangements. In this experiment of 787 trials on the two subjects the beginning of the saccade  $S_0$  triggers first a brief "true" cue  $\Delta_1$ , which indicates the final position of the target, and then, after delay, a cue  $\Delta_2$  at some other position.

The 16 position patterns, and the various responses to the cues, are illustrated in Figs. 2-5 for subject PEH. After a random delay the target initially steps randomly, left (-) or right (+), by  $\pm 3.83^{\circ}$  to elicit the triggering saccade  $S_0$ , the beginning of which triggers a brief cue flash of  $\Delta_1 = 5$  or 10 msec, at a random position chosen from  $\pm 7.65^{\circ}$  and  $\pm 11.5^{\circ}$ . The target is extinguished for a randomly chosen period ( $\omega_1 = 20, 100, 150$  or 200 msec) and is then exposed briefly, as a second cue flash of  $\Delta_2 = 10$ or 16 msec, at a position randomly chosen from  $\pm$ 7.65° and  $\pm$ 11.5°. The target is blanked after cue 2 for  $\omega_2 = 334$  or 150 msec. The target finally reappears at the same position as the first  $(\Delta_1)$  cue for 750 msec before stepping back to the instrument axis. The symbols in the left hand margins of the figures link the examples to the subsequent analysis of saccade amplitude (Figs. 6 and 7). As a cue is only effective with probability 0.4-0.7 there are four possible responses: (1) a single saccade  $S_1$  during blanking towards the position of cue 1, cue 2 being missed (O); (2) cue 1 is missed, and a single saccade  $S_2$  occurs towards cue 2 ( $\bullet$ ); (3) there are a pair of saccades: saccade  $S_1$  is towards the cue 1, and saccade  $S_2$  towards cue 2 (OA): (4) there are no saccades until after the final re-lighting of the target (not illustrated)—this was rare for PEH but more common for AMR. The abbreviations max, maj and min, give an indication of the relative frequencies of the outcomes—thus max is common, maj less common, and min rare. Note that cue 1 is at the same position for pattern numbers that differ by 4.

Double true cues P1-P4. In these experiments cues 1 and 2 are at the same physical position, but, because saccade  $S_0$  intervenes, the cues are at different retinal positions. The usual response is a single saccade during blanking in the direction of the cues, but in rare cases ( $\bigcirc \blacktriangle$  min) there is a second saccade, during blanking, that brings the fovea still closer to the cue. Using the latency criteria (Methods), most single saccades are due to cue 2 in P1, about equally often to cue 1 or cue 2 in P2, and almost always to cue 1 in P3 and P4. The 11/154 rare responses attributable to both cues ( $\bigcirc \bigstar$  min) are of special interest, since the second saccade makes allowance for the size of the preceding saccade, and is not based on retinal



Fig. 4. The P9-P12 false cue varieties of target position pattern.

position alone. In the figures, retinal image position relative to the fovea is the interval between the eye and target traces. Thus, in the example P4  $\bigcirc \blacktriangle$  min (Fig. 2), the retinal distance of cue 2 from the fovea is about 1·1 that of cue 1, but saccade  $S_2$  to cue 2 is only 0·5 of the amplitude of saccade  $S_1$  to cue 1. In the example P3  $\bigcirc \blacktriangle$  min, the fortuitous occurrence of cue 2, during saccade  $S_1$ , provides a spontaneous example of the effectiveness of a stimulus delivered at the peak velocity of a saccade.

Partial cue trials (P5-P8). In these patterns cue 2 is within  $\pm 3.83^{\circ}$  of the physical position of cue 1; but, because of the size and direction of the intervening saccade  $S_0$ , cue 2 is nearly at the same retinal position as cue 1, in the case of P5 and P8. Latency criteria show that the common response-a single saccade during blanking-is usually to cue 2 for P5, about equally often to cue 1 or cue 2 for P6, and nearly always to cue 1 for P7 and P8. As before, for P1-P4, there are also (12/175) rare responses attributable to both cues when cue 2 occurs just before, or during, saccade  $S_1$  to cue 1. P7  $\bigcirc \blacktriangle$  min is interesting: the retinal distance of cue 2 from the fovea is almost twice that of cue 1, but the saccades to cue 1 and cue 2 are of nearly equal size. In P8 O A min the two cues are at roughly the same retinal position, but saccade  $S_2$  to cue 2 is only 0.5 of the amplitude of saccade  $S_1$  to cue 1. Clearly allowance is being made for the intervention of saccade  $S_1$  between cue 2 and its response  $S_2$ .

False cue trials (P9-P16). As before, single saccades are usually to cue 2 for patterns P9 and 13, about equally to either cue 1 or cue 2 for P10 and 14, and nearly always to cue 1 for P11 and 15, and P12 and 16. Responses to both cues  $(O, \blacktriangle)$  are easy to recognize, and the illustrations (e.g. P11  $\bigcirc \blacktriangle$  min, P12  $\bigcirc$  $\bigstar$  min, P15  $\bigcirc \bigstar$  max) show large  $S_2$  saccades based not just on the retinal position of cue 2, but also on the size and direction of the intervening saccade  $S_1$ .

## Amplitude of saccades

In this, and the previous paper, primary saccades undershoot their targets but are always directed at the physical position of the target.

As a first illustration, saccades have been allocated to cues, and plotted in Fig.6 against the error that remains between eye and target position just prior to the saccade. Most of the grouped points coincide, but have been separated out for illustrative purposes. The deviant points at lower left reflect very few trials  $(n \ge 5)$ . As a better illustration regression lines through the data of the *individual* trials pass through



Fig. 5. The P12-P16 false cue varieties of target position pattern. (The return to the target by the fourth, fifth and sixth saccades in the bottom trial is unusual. This saccade group is possibly a primary-corrective saccade pattern in which the primary saccade is small and the first corrective saccade very large. The sixth saccade is a corrective saccade of more usual size.)



Fig. 6. Plot of mean data for the P1-P16 target positions: O saccade  $S_1$ ;  $\oplus$  saccade  $S_2$ . cue 1 being missed;  $\blacktriangle$  saccade  $S_2$  cue 2 following saccade  $S_1$  to cue 1. Also shown (**II**) are the data from the experiment of Fig. 1. Data of observer AMR displaced 11.5° to right. The scatter of the data is exaggerated for illustrative purposes—many of the clumped points superimpose.



Fig. 7. The mean saccade amplitude data of Fig. 6 plotted against the retinal position of the cue flash. The scatter of the points is real.

the origin, to within a few min of arc, and have correlation coefficients of 0.94–0.99. For both observers the slopes of regression lines are essentially the same (actually 10% shallower) as in the earlier experiments of Hallett and Lightstone (1976). in which saccade  $S_0$  triggered a single cue  $\triangle$  of variable duration (1– 300 msec). The coefficients of variation of saccade size (S.D./mean) are also the same (21% for PEH and 12% for AMR). As the  $\Delta = 300$  msec condition is equivalent to a normal, continually lit, target step, we conclude that saccade accuracy is unaffected by the experimental manoeuvres of this and the earlier paper.

Saccade amplitudes are plotted against the angle of the cue from the fovea in Fig. 7. The greater scatter is real, and not due to illustration. Although the  $(\bullet)$ points are invariant in the two plots, the  $(\bigcirc)$  and  $(\blacktriangle)$ points shift, since their abscissae are affected by failure to allow for the saccadic movement that intervenes between the  $\bigcirc$  or  $\blacktriangle$  saccade and its cue. The regression line may pass close to the origin, as a result of fortuitious pooling of opposing effects, but regression lines through sub-sets of the data do not pass through the origin, as is required if retinal position is the sole determinant of saccade amplitude.

#### (i) Frequency of the different response patterns

(a) Relative effectiveness of cue 1. In the simpler experiments of Hallett and Lightstone (1976) the beginning of the  $S_0$  saccade triggered a single cue  $\Delta$ , which was followed by a single blanking period  $\omega$ . When the  $\triangle$  cue was in the same direction as the  $S_0$  saccade ("uncrossed cue") there was a tendency for the  $\triangle$  cue to be missed. This was occasionally seen for  $\triangle$  as long as 200 msec, but was frequent for the shorter, intrasaccadic cues. In the present experiment cue 1 is brief, and intrasaccadic, and the same effect is seen. Recall that pattern numbers that differ by 4, in Figs. 2-5, are for the same position of cue 1. There are two sets of patterns in which cue 1 is uncrossed with respect to saccade S<sub>0</sub> (P1, 5, 9, 13 and P2, 6, 10, 14) and two sets in which cue 1 is crossed (P3, 7, 11, 15, and P4, 8, 12, 16). Cue 1 is rarely effective in the first uncrossed set, cues 1 and 2 are more nearly equally effective in the second uncrossed set, and cue 1 is very effective in both crossed sets.

(b) Cue 2 effect. Pairs of saccades to both cues are apparently 3 times as common for the false cue trials (P9-16), in which cue 2 is remote from cue 1, as for the other trials (P1-8) in which cue 2 is spatially coincident or adjacent to cue 1. However, this may simply reflect the greater ease of identifying saccades to false cues.

(c)  $\omega_1$  effect. For the false cue patterns (P9-16) responses to both cues are common when the inter-cue interval  $\omega_1 = 200$  msec, and are virtually absent when cue 2 occurs in the terminal part of saccade  $S_0$  ( $\omega_1 = 20$  msec). For other patterns (P1-8) these responses are rare (or identified with difficulty) and are mainly for  $\omega_1 = 200$  msec. This is in keeping with what has often been found for continually-lit targets (Bartlett, Eason and White, 1961; Wheeless, Boynton and Cohen, 1967; Feinstein and Williams, 1972; Komoda, Festinger, Phillips, Duckman and Young, 1973)—the neural processes in the first half of the latent period are not entirely ballistic, in the sense that they can be cancelled by a second stimulus occurring shortly after the first.

(d)  $\omega_2$  effect. For the false cue patterns P9-16, with  $\omega_2 = 150$  msec, the effectiveness of cue 1 is unaltered, and the effectiveness of cue 2 is reduced, relative to the frequency of the responses for  $\omega_1 = 350$  msec. Presumably some responses to false cues are cancelled by early relighting of the target.

(e) Observer effect. Although observer PEH rarely fails to respond to one of the cues, AMR shows a relatively greater tendency to respond only to the re-lighting of the target, and also (for P1-P8) a slightly greater tendency to respond to cue 2. AMR's mild tendency to delay has already been noted (Hallett and Lightstone, 1976).

# (ii) Saccade latencies (P1-P8)

In the case of the double true cue (P1-4) and partial cue (P5-8) patterns there are rare pairs of saccades during blanking, occurring with a frequency of 7%. These saccades are only seen when the latency of saccade  $S_1$  to cue 1 is such that cue 2 occurs during (-), or just before (-), saccade  $S_1$  (+18 to -165 msec, mean -32 msec). Possibly there are other saccade pairs that cannot be recognized because  $S_2$  occurs after re-lighting. The second saccade of the pair is not a corrective. or secondary, saccade, because corrective saccades always require visual inflow during the prior saccade, and are cancelled if the target shifts (Hallett and Lightstone. 1976). The second saccade has a normal primary saccade latency, relative to cue 2 (mean 265, S.D. 48 msec, n = 24; cf. 256 and 40 msec for saccades to con-tinually lit targets) but a short latency (mean 233 S.D. 35 msec) relative to the beginning of saccade S1. One interpretation is that 0-25 msec of processing for  $S_2$  is possible before the start of saccade  $S_1$ .

#### (iii) Saccade latencies (P9-16)

(a) Latency to cue 1 (O saccades). As cue 1 is weak, in patterns P9 and P13, only the other patterns contribute responses to cue 1. In each of these patterns, the latency of the  $S_1$  saccade to cue 1 (pattern means 236–253, n = 10–25) does not differ significantly from the latency of the  $S_0$  triggering saccade (pattern means 237–270 msec) to the continually-lit initial step, nor does this differ (grand mean 259 msec, n = 108) from the control data for continually-lit steps of  $2^3-12^3$  (mean 256 msec, S.D. 40 msec). As cue 1 occurs at the beginning of saccade  $S_0$  one can conclude that the occurrence of  $S_0$  and cue 2 do not delay the processing of cue 1. Single cue experiments suggest a similar conclusion with respect to saccade  $S_0$  (Figs. 2 and 4 of Hallett and Lightstone. 1976).

(b) Latency to cue 2 ( $\bullet$  saccades). The bulk of the  $S_2$ saccades to cue 2 only (cue 1 missed) occur for the uncrossed patterns in which cue 1 is relatively weak (P9 and 13, P10 and 14). Now, Wheeless et al. (1967) found that the latency of the response to their second stimulus was longer by about 40 msec ("cancellation time") when the expected response to the first stimulus was cancelled. Is a cancellation time apparent in the present data? In P9 and P13 the uncrossed cue 1 is frequently ineffective-perhaps in these cases there are no latent neural processes for cue 2 to cancel? On the other hand, in P10 and P14, cues 1 and 2 are of more nearly equal strength. In fact, for P9 and P13 ( $\bullet$ ) the mean latency of saccade S<sub>2</sub> to cue 2 is normal (269 msec, n = 68), and for P10 and 14 (•) it is prolonged (308 msec. n = 49), relative to control data for continually-lit steps (mean 256, S.D. 40 msec). Thus the present data agree with a "cancellation time" of 39-52 msec, in the Wheeless et al. (1967) sense, provided that there is a neural process to cancel. In false cue trials, cue 2 activates the motor nucleus antagonistic to that activated by cue 1, and can be expected to primarily activate the opposite hemisphere and superior colliculus. It is of interest, then, that Komoda et al. (1973, their Fig.4) find a small "cancellation time" in similar (continually lit) trials. but find a latency saving (dependent on step timing and pattern) when cue 2 activates the same motor nucleus and half brain as cue 1. Perhaps a cancellation time is only found if there is a latent process to cancel and if, as Robinson (1973) suggests, cue 2 causes latent responses in quite different neural channels to cue 1.

(c) Latencies of saccade  $S_2$  to cue 2 ( $\blacktriangle$  saccades). In this case both cue 1 and cue 2 cause responses, and so patterns P9 and P13 are not involved because of the weakness of cue 1. In the remaining false cue patterns cue 2 typically occurs some 0-100 msec before  $S_1$  to cue 1, but a very few cues occur very early at the end of triggering saccade  $S_0$ , and a few occur late during saccade  $S_1$ . The latency distribution of saccade  $S_2$ , relative to the beginning of cue 2, is delayed (mean 334 msec. S.D. = 47, n = 45 for PEH), but is normal if the beginning of saccade  $S_1$  is taken as origin (mean 257, S.D. = 61 msec). AMR's latencies are similar, though somewhat longer. It seems that cue 2 is remembered, or stored, but is not successfully acted upon until saccade  $S_1$  to cue 1 is being executed. This is, broadly speaking, in keeping with the concept from more general serial reaction time studies (Feinstein and Williams, 1972; Smith, 1967; Welford, 1959) that there seems to be a single decision channel which cannot be occupied by more than one latent response.

#### (iv) Intersaccadic interval

This interval is *not* necessarily of the order of 150-350 msec. The present observers have yielded, in a variety of situations, saccade pairs of roughly equal size  $(1^{\circ}-4^{\circ})$ , in the same or opposite direction, in which the second saccade starts 0-80 msec after the eye comes to rest (Light-stone. 1973). This is also seen in the monkey (Barmack, 1970) and is more common in the presence of the drug Diazepan (Frecker. 1973). A near zero dead-time requires parallel processing, or serial processing (*vide infra*) in which the minimum delay from retina to movement is not much longer than a saccade duration (40-70 msec)—a possibility suggested by the very different approach of St.-Cyr and Fender (1969).

#### DISCUSSION

#### Saccade size

Perceptual suppression and mislocation effects, and some relevant oculomotor and neurophysiological findings, are discussed in Hallett and Lightstone (1976) and in Dichgans and Bizzi (1972). The oculomotor paths probably discard a great deal of information under continually-lit viewing conditions-eye position information is then unnecessary, and retinal position alone suffices to define the goal. Better use must be made of all available information under conditions of intermittent lighting. At any rate, the mean size, variability, and goal-directedness of saccades is maintained by our subjects, for targets which are briefly exposed before, during and after saccades, or for targets which are exposed throughout the saccade (Hallett and Lightstone, 1976). Although perceptual observations show that briefly exposed targets are mislocalized, when presented in the interval of  $\pm 200$ msec about a saccade, there is no evidence from our experiments that saccade size is affected by illusory processes. Cue 1 is relatively ineffective when it steps to a position just ahead of a saccade: this is shown by delayed responses (p1). infrequent responses (ia), and the absence of a "cancellation time" (iiib). Ineffectiveness may be due to poor localization, or there may be some other explanation. We suggest that the final oculomotor output is spared most of the illusion because low oculomotor levels have direct access to the retina and to precise up-to-date information about eye position in the orbit. In addition, higher oculomotor levels may be able to correctly categorize the true position of a target, if the illusion is small relative

to the known spacing of target positions; or, in the case of true cues, higher oculomotor levels may be able to adapt to distortion of the visual world, if this is simple and *not noisy*, by correlating the appearances of the cue flash with its veridical position as revealed by the final re-lighting of the target. Whatever the case, no special training or practice is required, latency is usually normal, and the saccadic system has the option of not responding if localization is ever ambiguous.

## Saccade frequency and latency

The present experience is that saccades are ballistic but can be cancelled (e.g. Westheimer, 1954; Feinstein and Williams, 1972; Wheeless *et al.*, 1967; Komoda *et al.*, 1973). To this we add that useful visual stimulation can occur at any time, before, during or after a saccade; and that certain stimulus configurations (i.e. uncrossed cues at the time of saccades) sometimes fail to set up latent processes which lead to a saccade, or which require cancellation.

If new information arrives too late to cancel ongoing events, it is apparently stored until the beginning of the next saccade, and little or no oculomotor processing occurs. The data for rare pairs of saccades to patterns P1-8 (see ii), where the second saccade is small and in the same direction as the first, suggest that possibly 0-25 msec of latency-reducing processing for the second saccade can occur before the beginning of the first saccade. When the second saccade is large, and in the opposite direction, (P9-16  $\blacktriangle$ ), then its processing seems to wait until the beginning of the first saccade (see iiic). Komoda et al. (1973, their Fig. 2) claim more substantial latency savings, e.g. up to 60 msec, but their computation assumes that there is no latent processing during a saccade (to which the present work is opposed). If a correction is made their latencysaving may be 35 msec at best. Savings of 0-35 msec are not especially convincing in view of the large number of psychological and physiological factors involved.

Are there other arguments that the latent processes of two saccades can overlap substantially? The existence of short interval saccade pairs (see iv) would seem to indicate as much as 210 msec overlap on occasions, but this figure nearly vanishes if one accepts the possibility that the minimal oculomotor delay from retina to movement (65 msec, according to the extrapolation of St.-Cyr and Fender, 1969) is comparable to a saccade duration of 40-70 msec. Nor does the apparent short latency of corrective saccades provide evidence for substantial overlap. Visual processing for a corrective saccade can occur during the prior primary saccade (Hallett and Lightstone, 1976)-a possibility suspected by Becker and Fuchs (1969). Consequently, even if the neural process leading to a corrective saccade is of the same duration as that for a primary saccade, the corrective saccade process need not begin much more than 45 msec prior to the primary saccade—and, of course, it is quite possible that the corrective saccade process is briefer, because the goal is already defined at low oculomotor levels.

# Other work

Levy-Schoen and Blanc-Garin (1974) find in the Wheeless paradigm that a saccade can be shortened, or re-oriented, following cancellation, but not lengthened. This does not conflict with Figs. 2-5; responses to cue 2 only are common ( $\odot$  max) for patterns P5, 9, 10, 13, 14, and would involve lengthening if the saccade to cue 1 were really cancelled, but according to present arguments cue 1 is relatively ineffective when it steps to a position ahead of saccade  $S_0$ , and there is nothing to cancel. Levy-Schoen and Blanc-Garin also report that the serial orders of saccades and targets may differ—which we have not seen.

Acknowledgements—This work was supported by grants MRC MA 4092 and DRB 9310-122 from the Medical Research Council and Defence Research Board of Canada. We are indebted to Ann Rose for acting as an observer, to L. Phillips, H. Hallett and G. Oakham for technical help, and to R. Winterink and Y. Torgov for translations.

#### REFERENCES

- Barmack N. H. (1970) Modification of eye movements by instantaneous changes in the velocity of visual targets. Vision Res. 10, 1431-1441.
- Bartlett N. R., Eason R. G. and White C. T. (1961) Latency of ocular fixation upon the second of two successive stimuli. *Percept. Mot. Skills* 13, 259–268.
- Becker W. and Fuchs A. F. (1969) Further properties of the human saccadic system: eye movements and correction saccades with and without visual fixation points. *Vision Res.* 9, 1247–1257.
- Dichgans J. and Bizzi E. (1972) (Eds.) Cerebral control of eye movements and motion perception. *Biblthca* ophth. 82.
- Feinstein R. and Williams W. J. (1972) Interactions of horizontal and vertical human oculomotor systems: the saccadic systems. Vision Res. 12, 33–44.

- Frecker R. C. (1973) Effects on human saccadic eye movements of diazepan, pentobarbitol and dextroamphetamine. Ph.D. Thesis, University of Toronto.
- Hallett P. E. and Lightstone A. D. (1976) Saccadic eye movements due to stimuli triggered during prior saccades. Vision Res. 16, 99-106.
- Komoda M. K., Festinger L., Phillips L. J., Duckman R. H. and Young R. A. (1973) Some observations concerning saccadic eye movements. Vision Res. 13, 1009–1020.
- Levy-Schoen A. and Blanc-Garin J. (1974) On oculomotor programming and perception. Brain Res. 71, 443-450.
- Lightstone A. D. (1973) Visual stimuli for saccadic and smooth pursuit eye movements. Ph.D. Thesis, University of Toronto.
- Robinson D. A. (1973) Models of the saccadic eye control system. Kybernetik 14, 71-83.
- Smith M. C. (1967) Theories of the physchological refractory period. Psychol. Bull. 67, 202-213.
- St.-Cyr G. J. and Fender D. H. (1969) Non-linearities of the human oculomotor system: time delays. *Vision Res.* 9, 1491-1503.
- Welford A. T. (1959) Evidence of a single channel decision mechanism limiting performance in a serial reaction time task. Q. Jl exp. Psychol. 11, 193–210.
- Westheimer G. (1954) Mechanism of saccadic movements. A.M.A. Archs Ophthal. 52, 710-724.
- Wheeless L. L., Boynton R. M. and Cohen G. H. (1967) Eye movement responses to step and pulse-step stimuli. J. opt. Soc. Am. 56, 956–960.

**Résumé**—Quand on presente une cible de durée brève, avant, pendant, ou après une saccade, on déclenche une autre saccade de precision normale, et délai propre. Quand on étincele la cible dans une variation de l'expérience de Wheeless on n'exige pas un temps d'annulation si la cible est peut-etre inefficace. La latence approximative est la même soit que la cible est déclenche par une saccade ou non. Il n'y a pas raisons fortes de croire qu'une reduction de latence pour une saccade première est possible avant la saccade première et anterieuse.

Zusammenfassung—Ein Ziel, präsentiert als ein Blitz im Dunkel, vor, während oder nach einer Sakkade, entlockt eine spätere zielorientierte Sakkade von normaler Grösze und geeigneter Latenz. In einer angeblitzten Zielvariation des "Wheeless-Versuches", "Erlöschungszeit" ist nicht bemerkbar unter Umstände worunder das erste Ziel ist geglaubt nicht effektiv zu sein. Die Latenz ist ungefähr dieselbe, ob Zielschritte synchronisiert sind mit Sakkaden oder nicht. Es gibt keine starke Gründe zum Glauben dasz Latenzreduktion für eine spätere primäre Sakkade stattfinden kann bevor die vorgehende primäre Sakkade.