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Saccadic latency effects of progressively deleting stimulus offsets and onsets

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Abstract

We designed two extensions of Saslow's well-known gap and overlap conditions that require increased voluntary effort because of the progressive elimination of target onsets and fixation point offsets, and obtained repeatable data obeying simple numerical relations. For each of the five stimulus lighting conditions, saccadic latency was measured as a function of the retinal eccentricity or displacement of the target. Latencies were fitted by a serial processing model in which the visually guided minimum tracking latency $\boldsymbol{VGL}_{\min}$ is supplemented by two types of delay, dubbed 'unlock' and 'target', that can be either short or long ('direct' or 'indirect'), depending on the conditions. There are two findings: (1) The model has utility. The rank order of saccadic latencies for the five stimulus lighting conditions was constant across all subjects, sessions and eccentricities in the range 7.5′–6° left or right. For pooled data, and the saccadic latency plateau (1–6°), the model was also within ±3 ms of the mean latencies. (2) Latencies of tiny saccades to intrafoveolar stimulation (7.5–45′) were invariably long in all five stimulus conditions. One factor here must be the experimentally measured local prolongation of $\boldsymbol{VGL}_{\min}$.

Keywords: Eye movements; Successive planning; Serial processing; Express; Gap; Overlap; Foveating saccades; Anti-saccades

1. Introduction

1.1. Saccadic latency and stimulus lighting

Saccadic eye movements are often triggered in the laboratory by an instantaneous step displacement of the fixation target (here called the normal condition) involving a simultaneous offset of the fixation point and onset of the target. Saslow (1967) demonstrated that onset–offset asynchrony strongly affects the latency of a foveating saccadic response. Saccadic latency was decreased in the gap condition, where the fixation point is turned off before the target is lit, and increased in overlap where the fixation point remains on after the appearance of the target. His study triggered a great deal of work in both humans and monkeys (e.g., Cohen & Ross, 1977; Ross & Ross, 1980, 1981; Reulen, 1984a,b; Mayfrank, Mobashery, Kimmig, & Fischer, 1986; Kalesnykas & Hallett, 1987; Reuter-Lorenz, Hughes, & Fendrich, 1991; Fischer & Weber, 1992, 1993; Wenban-Smith & Findlay, 1991; Kingstone & Klein, 1993; Tam & Ono, 1994; Kopecz, 1995; Dorris & Munoz, 1995; Munoz & Corneil, 1995; Walker, Kentridge, & Findlay, 1995; Dorris, Paré, & Munoz, 1997; Everling, Paré, Dorris, & Munoz, 1998; Everling, Dorris, Klein, & Munoz, 1999; Spantekow, Krappmann, Everling, & Flohr, 1999; Walker, Walker, Husain, & Kennard, 2000; and many others).

The new study extends these stimulus lighting conditions to a range of central retinal eccentricities (±6°), with foveation as the most extensively studied response, to investigate the effects of systematically removing target onsets and fixation point offsets on saccadic latencies. We have supplemented Saslow's gap (G), normal (N) and overlap (O) with the persistent target (PT) and the persistent fixation point and target (PFPT) conditions. It will often be convenient to use the ordinary initial letters for the conditions and boldface for the corresponding mean latencies **G**, **N**, **O**, **PT** or **PFPT**. The gap and normal conditions display both a fixation point offset and a target onset, the offset in gap

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occurring well before the latency measuring period. The other three conditions progressively remove one or other or both dynamic elements. Overlap has no fixation point offset but does have a target onset, the PT condition has a fixation point offset but no target onset, and PFPT has neither a fixation point offset nor a target onset. [1] As will become apparent later, the two new conditions depend on an extra stimulus or nontarget at the mirror position to the lit target, which hides the identity of the target until the nontarget is extinguished, at which time only the target remains and a saccadic foveating response follows. To compare the lighting conditions reliably we used standardized retinal stimulus intensities, many different retinal eccentricities, and several subjects, taking care to always compare two conditions in the same session to allow for session to session variation. [2]

1.2. The 'unlock and target' model

In the process of measuring saccadic eye movement latency, one looks for distinguishing characteristics related to stimulus conditions to simplify the understanding of events without actually being able to directly see the underlying physiological processes. We found two such characteristics, one of which (process A) was related to properties of the foveal fixation stimulus, and the other (process B) to the eccentric target. Processes A and B in their short and long forms provide a serial mathematical description of our data. With diffidence, because single words are usually more attractive than symbols, and open to many interpretations, we will refer to processes A and B as 'unlock' and 'target', respectively. The unlock process may or may not be related to fixation mechanisms, the ability to suppress saccades, or the disengagement of attention, but it certainly should not be considered equivalent to a saccade trigger signal. The target process may or may not be related to a spatial shift of limited attentional resources; we consider it in general terms to be a targeting mechanism, the completion of which results in a spatial saccadic command and a saccade trigger signal. These processes are serial, and the completion of the A or unlock process allows initiation of the B or targeting process.

Latency predictions for the five stimulus lighting conditions follow from the notions (a) that the extinction of a fixation point provides a direct sensory release or *direct unlocking* of the saccadic oculomotor system (perhaps by releasing the frontal eye field and superior colliculus (SC) from inhibition, e.g. Hikosaka & Wurtz, 1983; Munoz & Guitton, 1989, 1991; Munoz, Guitton, & Pelisson, 1991; Munoz & Wurtz, 1993a,b; Dias & Bruce, 1994; Forbes & Klein, 1996; Dorris et al., 1997; Munoz & Istvan, 1998; Abrams, Oonk, & Pratt, 1998; Taylor, Kingstone, & Klein, 1998; Taylor, Klein, & Munoz, 1999) and (b) that the recent lighting of the target triggers a direct sensory request for targeting a saccadic eye movement (*direct targeting*, e.g., by means of the onset transient, Todd & van Gelder, 1979). If these sensory triggers are missing, oculomotor processing must be voluntarily (*indirectly*) disengaged from a fixation point which persists without offset, and voluntarily shifted to the target if this lacks an onset during the latency measurement period (cf. exogenous vs. endogenous targeting, Forbes & Klein, 1996). Unlocking is associated with a pathway from the retinal region that receives the fixation point, the foveola. The unlock process may be either direct (= short latency or sensory driven) or indirect (= longer latency or endogenously driven) with delays *du* or *iu*, respectively. Targeting is due to projections from the entire retina and the process is similarly direct or indirect with delays *dt* or *it*. We make no claims where in the brain these processes occur. Our unlock and target model is distinct to the when/where model of Findlay and Walker (1999) which is based on *parallel* competing processes, and the SC-based competitive integration model of Trappenberg, Dorris, Munoz, and Klein (2001). By contrast, our model is serial and noncompetitive and the two variable processes each have two forms, as stated above, depending on stimulus conditions.

Exact predictions for the unlock and target model and the extended range of Saslow conditions would be very straightforward if the unlock and target delays were exactly replicated across different conditions and subjects, as can be seen from the relations. [3]

$$\mathbf{E} = 0 + 0 + \boldsymbol{VGL}_{\min} + \varepsilon, \tag{1a}$$

$$\mathbf{G} = 0 + dt + \boldsymbol{VGL}_{\min} + \varepsilon, \tag{1b}$$

$$\mathbf{N} = du + dt + \boldsymbol{VGL}_{\min} + \varepsilon, \tag{1c}$$

$$\mathbf{O} = iu + dt + \boldsymbol{VGL}_{\min} + \varepsilon, \tag{1d}$$

$$\mathbf{PT} = du + it + \boldsymbol{VGL}_{\min} + \varepsilon, \tag{1e}$$

and

$$\mathbf{PFPT} = iu + it + \boldsymbol{VGL}_{\min} + \varepsilon. \tag{1f}$$

[1] We are not the first to use a PT-like condition or to stress the difference between transient and sustained illumination but the stimulus arrangements of Todd and van Gelder (1979) were more complicated and their comparisons less detailed.

[2] A report of exploratory findings was given by Kalesnykas and Hallett (1989).

[3] We use ordinary case letters to denote lighting conditions (e.g., PT), bold to denote observed latency means (**PT**), italic to denote hypothetical delays (e.g., *dt*) and bold italic to denote a latency boundary ($\boldsymbol{VGL}_{\min}$).

Here $\boldsymbol{VGL}_{\min}$ is the experimentally measured visually guided *minimum* latency, the latency boundary below which direction errors and anticipatory responses occur with equal frequency (see Kalesnykas & Hallett, 1987; Kalesnykas, 1994). ε is a correction for the difference between the $\boldsymbol{VGL}_{\min}$ *boundary* and the next higher latency *mean* **E** (Eq. (1a)) which is often referred to as the "express saccade mode". [4] Eq. (1b) indicates that there is directly initiated targeting during the latency measurement period of the gap condition, unlocking having occurred earlier (Mayfrank et al., 1986). Eq. (1c) shows both direct unlocking and targeting in the normal condition. Finally, Eqs. (1d)–(1f) indicate the increasing participation of the indirect processes already mentioned. Interestingly, if this formulation were exactly true, there would be a redundancy in the equations that would allow two checks on the validity of the model (see Section 4). However, given the variability in most behavioral experiments, it seems wiser as a first step to relax the assumptions of the model and allow that the unlock and target delays (*du*, *iu*, *dt*, *it*) might possibly vary with the conditions and subjects, and assume only that delays are positive quantities with direct delays shorter than the corresponding indirect delays, i.e., only that $du < iu$ and $dt < it$. The predicted rank order of the measurements by increasing latency is then

$$\boldsymbol{VGL}_{\min} < \mathbf{E} < \mathbf{G} < \mathbf{N} < (\mathbf{O}?\mathbf{PT}) < \mathbf{PFPT}. \quad (2)$$

The ? symbol notes that the relaxed model is ambiguous as to the a priori order of **O** and **PT**. This is because the **O** and **PT** latencies each involve one direct and one indirect operation in Eqs. (1d) and (1e), but of different kinds and unknown values; the order of **O** and **PT** might even vary with subject or retinal eccentricity depending on the relative sizes of the delays *du*, *iu*, *dt* and *it*.

[4] We follow current usage in calling the shortest latency visually guided saccades express saccades but note some difficulties. In experiments with well-randomized targets, like the present ones, $\boldsymbol{VGL}_{\min}$ is easily measured, while the modal mean latency **E** is hard to measure because express saccades are few and not well segregated from longer latency responses (see Section 2; also Wenban-Smith & Findlay, 1991). In human experiments in which randomization of target position is weak, or in primate experiments, "express saccades" are a well-defined mode (e.g., Fischer & Weber, 1993)—however Kalesnykas and Hallett (1987) regarded such responses in humans as really being amplitude-modified, correctly-directed anticipatory saccades. On their view (i) $\boldsymbol{VGL}_{\min}$ should be identical, after correction for stimulus intensity, to the classic latency for visual cancellation of a latent saccade, (ii) $\mathbf{E} = \boldsymbol{VGL}_{\min} + \varepsilon$ in Eqs. (1a)–(1f) is the shortest latency for amplitude modification of a latent saccade, and (iii) express saccades can never consistently and significantly exceed 50% of the total responses because misdirected anticipatory responses are cancelled and replaced by longer latency, correctly aimed, saccades.

1.3. Intrafoveolar target displacements

One condition to which the relaxed model (Eq. (2)) might be sensitive is very small target displacements. It is known that the foveolar pathways have special properties: (a) both the initiation (Steinman, Haddad, Skavenski, & Wyman, 1973; Wyman & Steinman, 1973a,b; Winterson & Collewijn, 1976) and suppression (Becker, 1972; Prablanc & Jeannerod, 1975; Adams, 1978; Hallett, 1986) of very small amplitude saccades can require effort; (b) the functions of the foveolar pathways (= hold fixation and scrutinize) must be very different from those of the remaining retina (= shift fixation); (c) indeed foveolar stimuli can inhibit or delay saccades of all sizes (Kalesnykas & Hallett, 1996)—behavior that may be the result of neural interactions within the SC (Munoz & Wurtz, 1992, 1993a,b); (d) target displacements within the foveola are associated with long saccadic latencies (Wyman & Steinman, 1973a,b), a function referred to as the central or foveolar saccadic latency peak; (e) this peak is a robust phenomenon that has resisted attempts to eliminate it (Kalesnykas & Hallett, 1994, 1996). One possibility, then, is that the rank-order of latencies (Eq. (2)) will prove to be different for intrafoveolar target displacements than for larger displacements within the region of the latency plateau (e.g., because of special foveolar values for the delays *du*, *iu*, *dt* and *it*). Even if the rank orders hold constant across eccentricity it is still not possible to predict whether the latency peak will persist unchanged across all target conditions. The peak might persist, growing in absolute latency on passing through the lighting conditions from G via N, O and PT to PFPT, if latencies are intrinsically long in the foveola, or absolute latency might become constant if foveolar or small saccade [5] processing time cannot be protracted by more than a certain amount, in which case the peak might relatively dwindle and shrink in width (see Section 4.1). In order to gain some interpretive power in this situation we directly determined the minimum visually guided latency $\boldsymbol{VGL}_{\min}$ for each subject for small and large target displacements, using the method of Kalesnykas and Hallett (1987).

1.4. Summary

The major results are as follows. (i) A single rank order for latencies $\boldsymbol{VGL}_{\min} < \mathbf{E} < \mathbf{G} < \mathbf{N} < \mathbf{O} < \mathbf{PT} <$

[5] We generally prefer the term central latency peak to foveolar peak as this would seem to meet both sensory and motor explanations. This study does not attempt to dissociate retinal and motor factors. Possibly the Hypo and Hyper tasks of Hallett (1978) would help here. Kalesnykas and Hallett (1996) attributed the peak to the properties of central sensory motor maps.

PFPT applies across all subjects, sessions and target eccentricities, so the relaxed model has generality. From Eqs. (1a)–(1f) this rank order is suggestive of indirect targeting *it* always inflating latencies more than indirect unlocking *iu* (see Section 4). (ii) The central latency peak persists, though possibly narrowed in the long latency conditions, and $\boldsymbol{VGL}_{\min}$ is prolonged in the same retinal region. We therefore see $\boldsymbol{VGL}_{\min}$, and intrinsic limitations on foveola or small saccade processing, as being the major temporal basis for the latency peak. (iii) Finally, the exact form of the model (Eqs. (1a)–(1f)) passes its two checks when tested with sufficiently tightly defined mean latencies (a test that could only be performed for the extensive data at larger displacements)—suggesting that the model's motivating arguments are not without value.

2. Methods

The experimental design aims at direct comparisons of latencies, so we always compared two stimulus lighting conditions in each session to allow for small intersession variations. The points in the plots are for at least 50 trials. Because there are many points in the region of the latency plateau (Kalesnykas & Hallett, 1994; Fig. 3), the typical latency of the plateau can be well estimated by pooling across eccentricities of 1–6°.

2.1. Apparatus

We used our previous techniques (e.g., Kalesnykas & Hallett, 1996). The two-dimensional corneal-reflex tracker was that of Frecker, Eizenman, and Hallett (1984). Photometrically balanced, flatfaced light-emitting diodes provided very small stimuli (4′ as in Kalesnykas & Hallett, 1994) that were set monocularly each session to be 1000 times the dark-adapted foveal threshold, so as to eliminate latency variations due to nonstandard stimulus intensity (Hallett & Lightstone, 1976). A mydriatic (1% Mydriacyl), to stabilize pupil size and thus retinal illuminance and reaction time, was used in the PT and PFPT conditions where the number of lit stimuli varied from moment to moment (this did not change mean latency).

2.2. Gap, normal and overlap conditions

These were fairly conventional. Fig. 1 and its legend describe the timing. Fig. 2 and its legend describe the spatial positions of stimuli—it is a frame-by-frame representation which shows two successive trials with target (solid circle), fixation point (open circle) and eye positions (cross). An initial training of three to four 800-trial sessions of the N condition was used to stabilize foveating latency. We tried to get the shortest possible gap and the longest overlap latencies. The optimal gap interval V was 150 or 200 ms depending on the subject. In overlap, the fixation point remained lit until the start of the primary saccade. There were no special instructions, except to track the stimulus as quickly and accurately as possible, and no admonishments when responses were anticipatory. Up to 22 target positions were possible in a trial (±0.25°, 0.5°, 0.75°, 1°, 1.5°, 2.25°, 2.75°, 3°, 3.25°, 3.75°, and 6°), and the target position for one trial was the fixation point for the next. Thus fixation was always direct and foveal (never eccentric) with the average target or fixation point 2.3° to left or right of the straight ahead midline. This procedure was checked. (i) The shifting fixation point method gave the same results as a fixation point on the midline. (ii) Separately accumulating centripetal and centrifugal saccades revealed no latency differences, and this practice was discontinued. From Accardo, Inchingolo, and Pensiero (1987), who pooled subjects and target directions for larger saccadic amplitudes, one might perhaps expect a small (6 ms) effect. We, however, will plot subjects and left and right target data separately, as there can be appreciable idiosyncratic differences, as the data of Accardo et al. also show. The lighting conditions were run as separate blocks of 800 trials, two conditions per session. The minimum visually guided latency $\boldsymbol{VGL}_{\min}$ was measured as in Kalesnykas and Hallett (1987), for small (0.25° displacements) and large (pooled 0.75–6°) amplitude saccades.

2.3. 'Persistent target' and 'persistent fixation point and target' conditions

The lower parts of Figs. 1 and 2 illustrate one trial, followed by a control trial, for each condition. Both a target and a nontarget (NT in the figures) are lit at the beginning of a trial, and extinguishing the NT defines the saccadic target and the beginning of the latency measurement. Twenty to thirty practice trials were given for familiarity whenever experimental conditions changed. Five blocks of 200 trials each were collected in a 1 h session for each of the two conditions to give at least 50 trials per latency point in the later figures. There were two minor simplifications from the earlier G, N and O experimental block forced by the potential complexity of the new displays: (i) the fixation point remained at centre (for checks see Section 2.2 above), and (ii) there were only two PT or PFPT target positions in a block of trials (e.g., x° left or right). To eliminate any possibility of either guessing or latency inflation in the new conditions, special control trials (N for PT and O for PFPT) were intermixed with 0.5 probability. These trials began exactly like PT or PFPT trials with the typical fixation point changing to a target and nontarget display (i.e., frame 1 is the same as frame 6 in Fig. 2 bottom) but all or part of this display disappeared, as

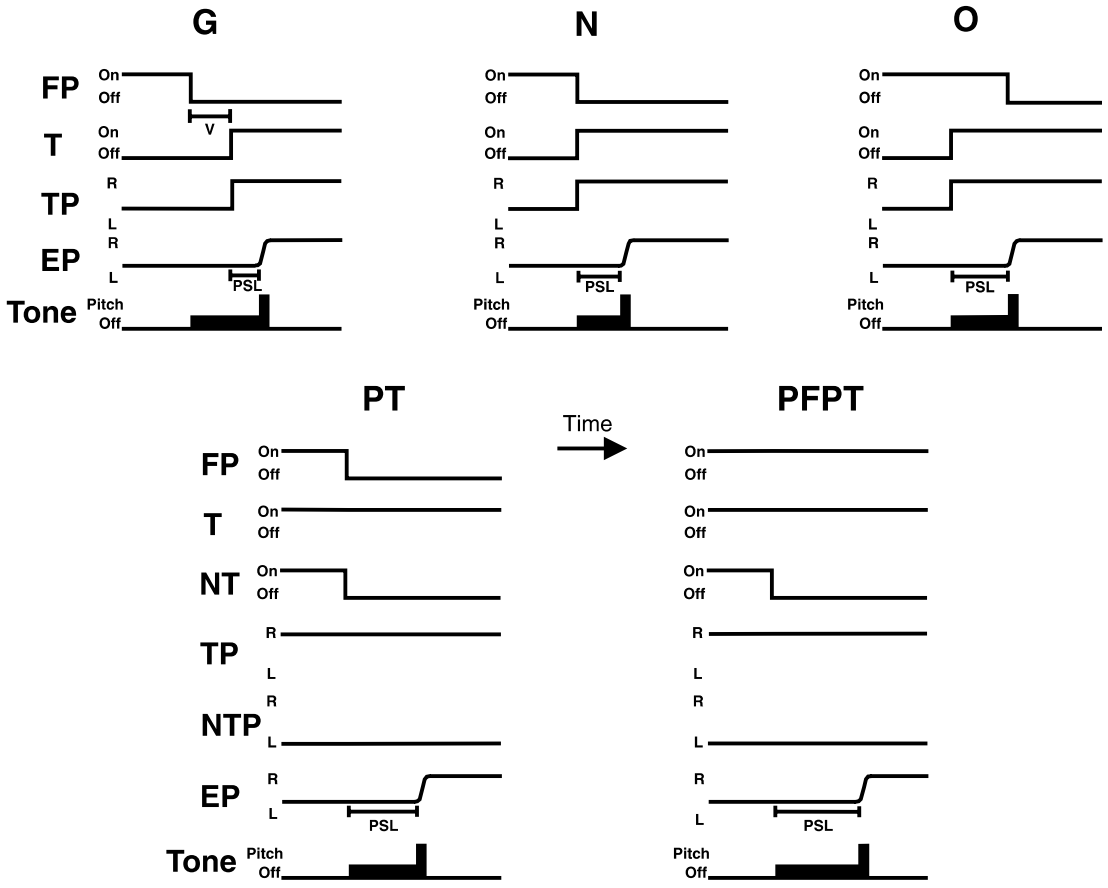

Fig. 1. Schematic diagram showing the timing sequence of stimulus events for the gap (G), normal (N), overlap (O), persistent target (PT), and persistent fixation point and target (PFPT) conditions. The interval of darkness is shown as period V in the gap condition. Lighting states (Off/On) of the fixation point (FP), target (T), and nontarget (NT) stimuli are shown. Eye (EP), target (TP), and nontarget (NTP) positions of the stimuli are displayed graphically within right (R) and left (L) limits. Time elapses from left to right. The trial begins with a random foreperiod of 1.5–2.4 s in 0.1 s steps. Primary saccade latency (PSL) is from the onset of the target stimulus to the onset of the primary saccade and is marked by a low-pitched tone that changes to a higher-pitched "pip" for the duration of the saccade. Each session involved two blocked lighting conditions to allow for session to session latency variation.

appropriate, and simultaneously the control target appeared—at 0.25° left or right for PT and PFPT targets and nontargets at ±3–4° (as in frame 7 in Fig. 2 bottom) or at 4° left or right for the other target/nontarget positions. No cross-effects of the new lighting conditions upon the control latencies were found (e.g., panels 3 and 4 in Fig. 3 and its legend). Pilot studies did show unusually long latencies for the most peripheral nontargets (±3–4°). We were able to correct these to the typical plateau value with a supplementary nontarget placed 1° or 2° vertically above the fixation point, and extinguished at the same time as the main nontarget, but we decided to omit this technical refinement in favor of simpler reporting.

2.4. Subjects

There were five volunteers. Subjects HDK and RPK were highly experienced, the others naïve as to the purpose of the experiments. Subjects HDK, MLZ, and RPK participated fully in all experiments. Subject AJB used fewer target positions in the PT and PFPT conditions. Subject HMH contributed ancillary results, as noted.

2.5. Supplementary analyses

Not all of the findings are reported. Unremarkable analyses reported in Kalesnykas (1994) included the frequencies of the anticipatory and few express saccades in the gap condition (much as in Kalesnykas & Hallett, 1987), and typical main sequence relations for foveating saccades (saccadic amplitude versus peak velocity and duration of the primary saccades) for all conditions (much as in Hallett, 1986).

3. Results

The saccade latencies for some pairs of stimulus conditions are summarized in Fig. 3 for the typical

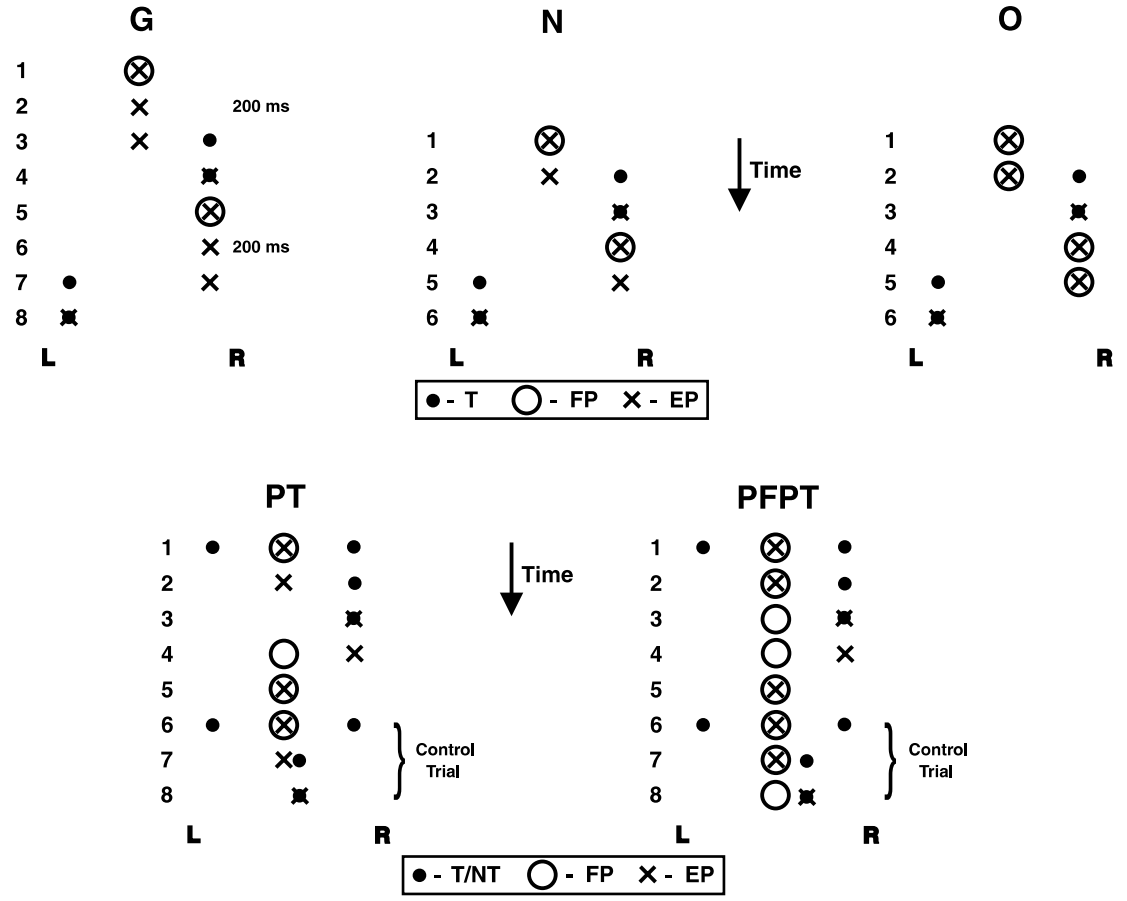

Fig. 2. A frame-by-frame cartoon of the sequences of spatial relations between eye position (×), target/nontarget (●) and fixation point (○) along the horizontal meridian in the five stimulus lighting conditions. Time elapses from top to bottom of each panel. The sequences for the G, N and O conditions show two consecutive trials (frames 1–4 and 5–8, respectively, for G; frames 1–3 and 4–6 for N and O); the fixation point shifts in these three conditions from trial to trial. In the gap condition frames 2 and 6 are 200 ms dark intervals. The sequences for the PT and PFPT conditions show a single trial (frames 1–5) followed by a randomly interleaved Control trial (frames 6–8), which is essentially a normal and overlap condition, respectively. The fixation point remains central in these two conditions.

subject. Fig. 4 shows the latency ordering for the five stimulus lighting conditions in the main subjects. Fig. 5 shows supplementary results with 7.5′ as the smallest target displacement.

3.1. Central or foveolar saccadic latency peak

Saccades $< 0.5°$ were rarely anticipatory or of "express" latency (also Weber, Aiple, Fischer, & Latanov, 1992). Mean latencies generally increased for both intrafoveolar and larger target displacements on proceeding through the sequence of lighting conditions from G, via N, O, PT, to PFPT. There were two exceptions for small target displacements where a change in lighting condition produced little change in latency (N and O in Fig. 3, panel 2 at $-0.25°$, and in Fig. 4, top, at $+0.25°$). The foveolar peak appeared narrowed for all subjects in the PFPT condition, and apparently missing occasionally in overlap (subjects HDK and RPK in Fig. 4), so this was investigated with 7.5′ displacements of the target (Fig. 5). However, every check showed that foveolar latency increased sharply for a small enough target displacement.

The visually guided minimum latency $\boldsymbol{VGL}_{\min}$ was measured in each subject by the latency in the gap condition at which there is a transition from an equal mixture of correctly directed and direction error saccades at shorter latencies (anticipations) to all correctly directed (tracking responses) at longer latencies (Kalesnykas & Hallett, 1987). For displacements equal to 15′ and four subjects, $\boldsymbol{VGL}_{\min}$ was 151 ± 32 ms. For larger displacements it was considerably shorter at 97 ± 5 ms. This last is smaller than our earlier value of 120 ± 10 ms, because the targets are now 10 times more intense than in Kalesnykas and Hallett (1987). Note that the new value is limiting, as latency cannot not be further reduced by brightening of the target (Kalesnykas & Hallett, 1994). The central peak was virtually free of presumptive "express" saccades. Only two (for a 0.25° step) were detected among the gap condition saccades.

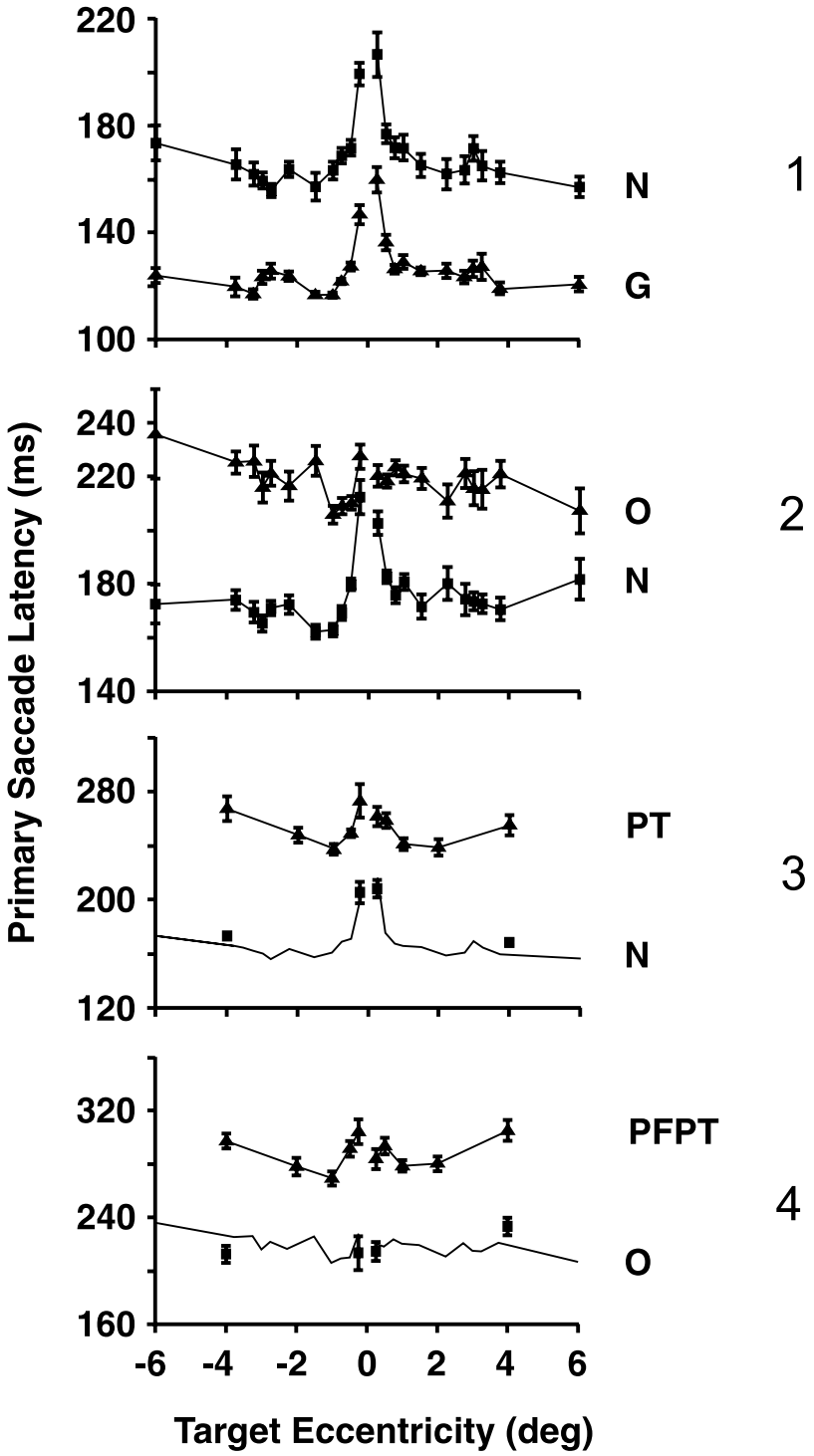

Fig. 3. Primary saccade latency as a function of target eccentricity for the five stimulus conditions and a representative subject, HDK. The control conditions (shown as single mean latency points) in the bottom two panels are randomly interleaved in each session to permit direct latency comparisons. The lower curves in those panels are from panels 1 and 2, rescaled. Target eccentricities in the nasal and temporal hemifields are negative and positive, respectively. Mean latencies ±1 SEM.

All others were at larger (>0.75°) displacements and were poorly separable from the anticipatory or tracking populations (Fig. 6, top; also Wenban-Smith & Findlay, 1991).

3.2. Plateau latencies

The plots in Fig. 5 show that latency generally increases by a constant across all eccentricities of the

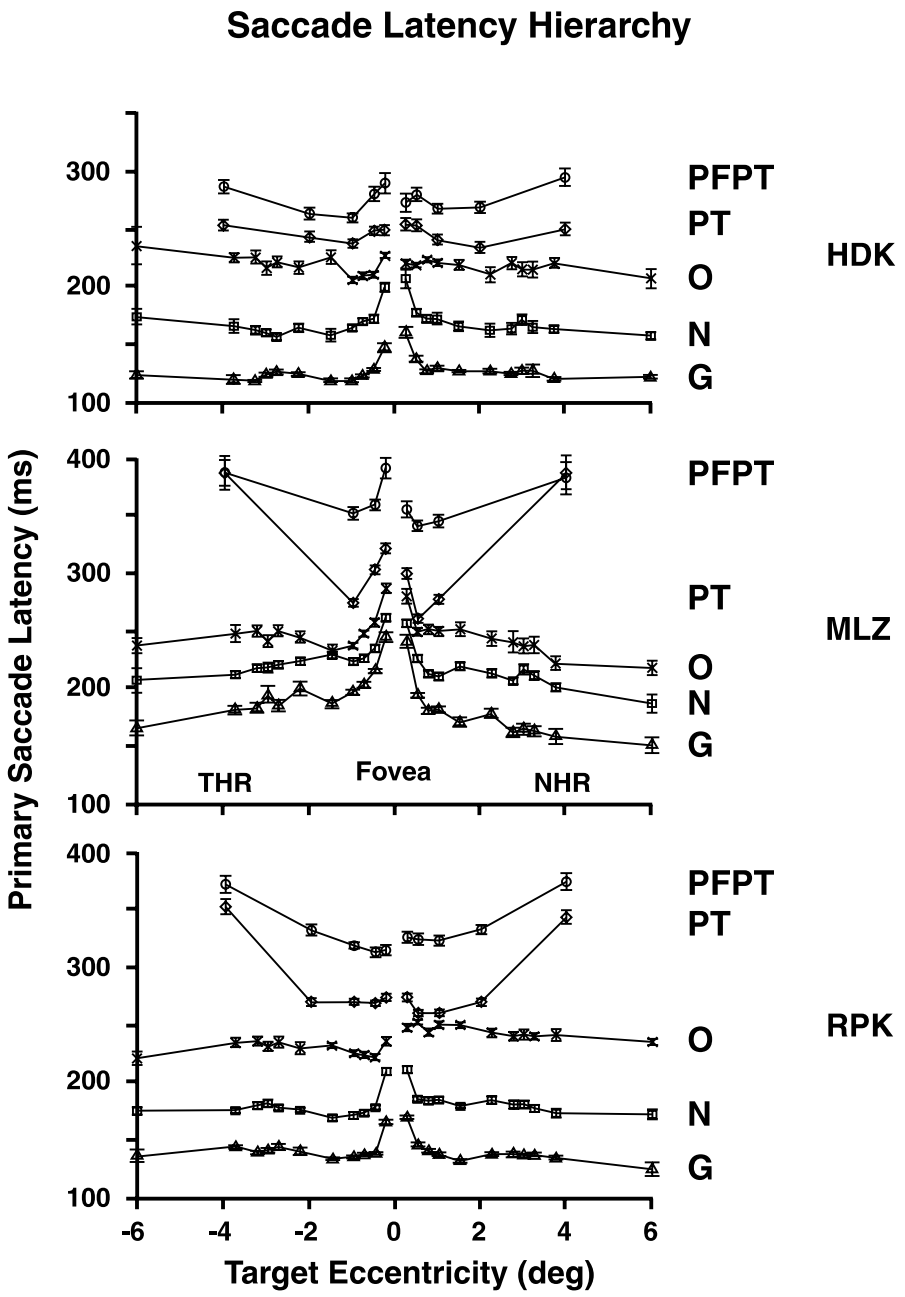

Fig. 4. Plot of primary saccade latency versus target eccentricity for the five stimulus lighting conditions for three subjects. THR and NHR represent temporal and nasal hemiretinas. The data form a latency hierarchy based upon the presence or absence of the transient offset and onset components of fixation point and target lighting. The reason for the anomalous PT and PFPT latencies at ±4° is given in Sections 2.3 and 3.2. Mean latencies ±1 SEM.

latency plateau from one lighting condition to the next (the only exception was the similarity of normal and overlap latencies at −1.5° for subject MLZ in Fig. 4). There are two qualifications to the general rule. (A) In any given subject the overall increase in latency from any one lighting condition to the next is not a constant across all lighting conditions. (B) Latency was exceptionally long at the ±3–4° positions in the PT and PFPT conditions (Figs. 3–5). Additional experiments on subject RPK suggested an artefact due to insufficient salience of the nontarget offset. The increase at ±3–4° could (a) be *avoided* by increasing salience, by providing an additional and simultaneous nontarget offset on the vertical midline reasonably [6] close to the foveola (see Section 2.3), or (b) further *increased* by 20–40 ms by

[6] Not too close, because foveolar nontargets, depending on their position, lead to global or hemifield latency increases (Kalesnykas & Hallett, 1996).

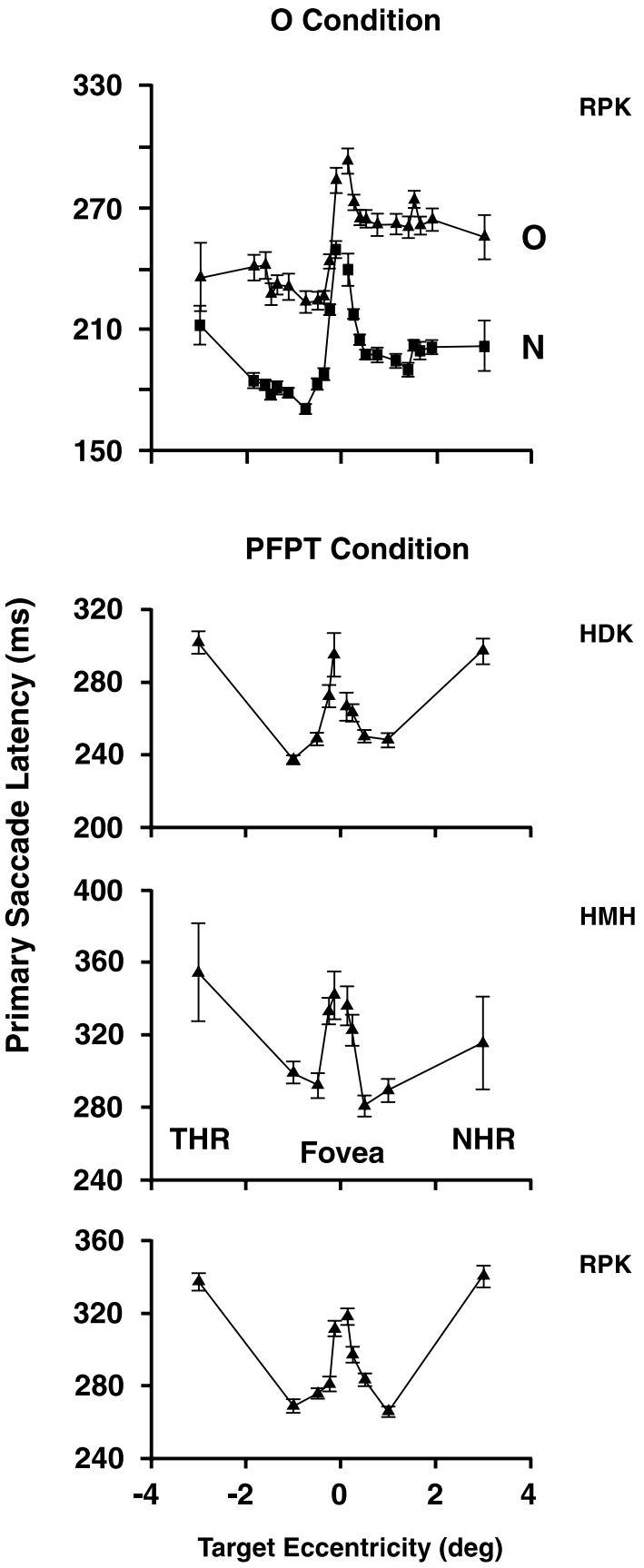

Fig. 5. Primary saccade latency/target eccentricity functions for target displacements as small as 7.5′ in the overlap and PFPT conditions, to show the central latency peak. Other details as in Fig. 4. Mean latencies ±1 SEM.

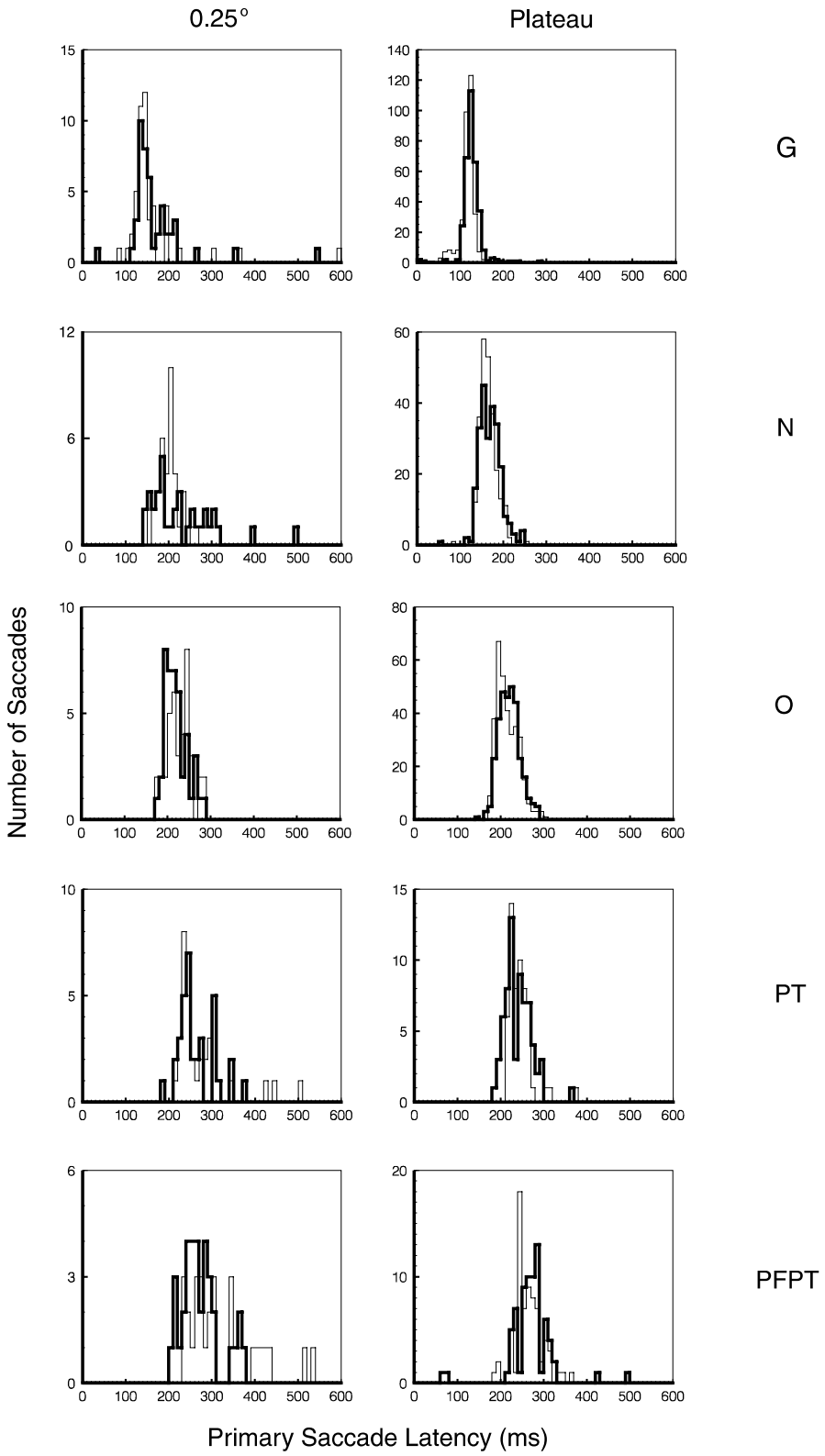

Fig. 6. Histograms of primary saccade latency in the five stimulus lighting conditions for small (0.25°) and larger (plateau) target displacements. Left-going (abducting) and right-going (adducting) saccades graphed with heavy and light outlines, respectively. Distributions appear unimodal, and variability increases with mean latency. Subject HDK.

weakening the usual cueing by omitting the auditory tone that signals the onset of target displacement (Fig. 1).

3.3. Rank order of latencies

The rank order of the measured latencies was the same across subjects, sessions and eccentricities, i.e.,

$$\boldsymbol{VGL}_{\min} < \mathbf{E} < \mathbf{G} < \mathbf{N} < \mathbf{O} < \mathbf{PT} < \mathbf{PFPT}. \quad (3)$$

3.4. Latency distributions

These are shown in Fig. 6 for 15′ and for larger target displacements. The distribution standard deviations increase with mean latency, and the distributions appear unimodal when examined with two different sizes of latency bin.

4. Discussion

The latency rank order predictions of the relaxed version of the unlock and target model (see Section 1.2) were surpassed, in the sense that there was only one rank order for the mean latencies for the five lighting conditions across subjects, sessions and target displacements.

4.1. The central latency peak

The general persistence of the peak, with possible indications of narrowing in the O, PFPT and possibly PT lighting conditions, throws some light on the peak's nature.

4.1.1. Persistence

This is the third time that we have been unable to eliminate the central peak and make the central retina a "level playing field" in which target displacements of any size (±6° range) elicit saccadic responses with the same latency. Kalesnykas and Hallett (1994) failed to eliminate the peak by manipulating sensory factors such as target intensity, wavelength and rod-cone interactions. Kalesnykas and Hallett (1996) failed for special displays in which fixation point or target were virtual. They found that the fovea has special properties, because a stimulus there (depending on its precise position) exerted either hemifield or global saccadic inhibition, as judged by latency increases, rather than a local effect—and this inhibition had sensitive, fast dynamics, in the sense that it could be partially extinguished by a 1 ms extinction of the fixation point. The present study has failed to eliminate the central peak by eliminating stimulus transients. When we progressively removed the direct sensory triggers to saccadic eye movements the peak persisted, latencies for very small target displacements increasing by roughly the same amount as for larger displacements. We have also found that a central latency peak persists if we change the required motor response from foveation (pro-saccades) to aversion (anti-saccades) for the gap, normal and overlap lighting conditions (AG, A and AO in Fig. 7). We therefore attach importance to the finding that the visually guided minimum latency $\boldsymbol{VGL}_{\min}$ is prolonged for very small displacements (see Section 3.1)—it seems likely that the central latency peak is at least partly due to an intrinsically long latency for foveola or small saccade processing (also Section 1.3) whatever the conditions. This could arise at various neural levels. (a) *Afferent*: Foveal processing might be heavily dependent on a retinocortical pathway with small receptive fields and fine, slow conducting axons, e.g., the parvocellular pathway from the midget ganglion cells (Hubel & Wiesel, 1972; Dreher, Fukuda, & Rodieck, 1976; Schiller & Malpeli, 1978; Creutzfeldt, Lee, & Elepfandt, 1979; Shapley, Kaplan, & Soodak, 1981; Kaplan & Shapley, 1982). Or central processing might be long because targeting requires prolonged measurement when the target displacement is very small and intrafoveolar. (b) *Motor*: Small amplitude saccade latency might be long because the putative foveolar fixation zone is continuous with the targeting zone in some neural map (Kalesnykas & Hallett, 1996; Krauzlis, Basso, & Wurtz, 1997; Walker, Deubel, Schneider, & Findlay, 1997), which raises the possibility of fixation and saccade being interfering operations at sufficiently small target displacements. In brief, the persistence of the central latency peak in the present experiments seems at least partly attributable to the empirical prolongation of $\boldsymbol{VGL}_{\min}$ in Eqs. (1a)–(1f), and this is consistent with intrinsic limitations on foveolar processing in other types of experiments.

4.1.2. Narrowing

Although we regret not including a larger variety of intrafoveolar target displacements there are clear indications that the central latency peak is narrowed in the O, PFPT and possibly PT conditions. There are two nonexclusive interpretations here. (a) To greatly exaggerate the first possibility, suppose that intrafoveolar processings or small saccades were always highly voluntary, i.e., only indirect delays *iu* and *it* ever occur, then the absolute latencies of the central peak would be fixed, and moving systematically through the lighting conditions from G to PFPT would reduce the peak's height relative to the increasing height of the latency plateau. The peak would also be narrowed because only the longest, most central latencies would still be apparent. This explanation is wrong because the peak does systematically increase in absolute latency, but the observed narrowing leaves a suspicion that delays other than $\boldsymbol{VGL}_{\min}$ are altered in the region of the peak. (b) Considerable widening of the behavioral latency peak, in experiments with virtual stimuli, was held to be

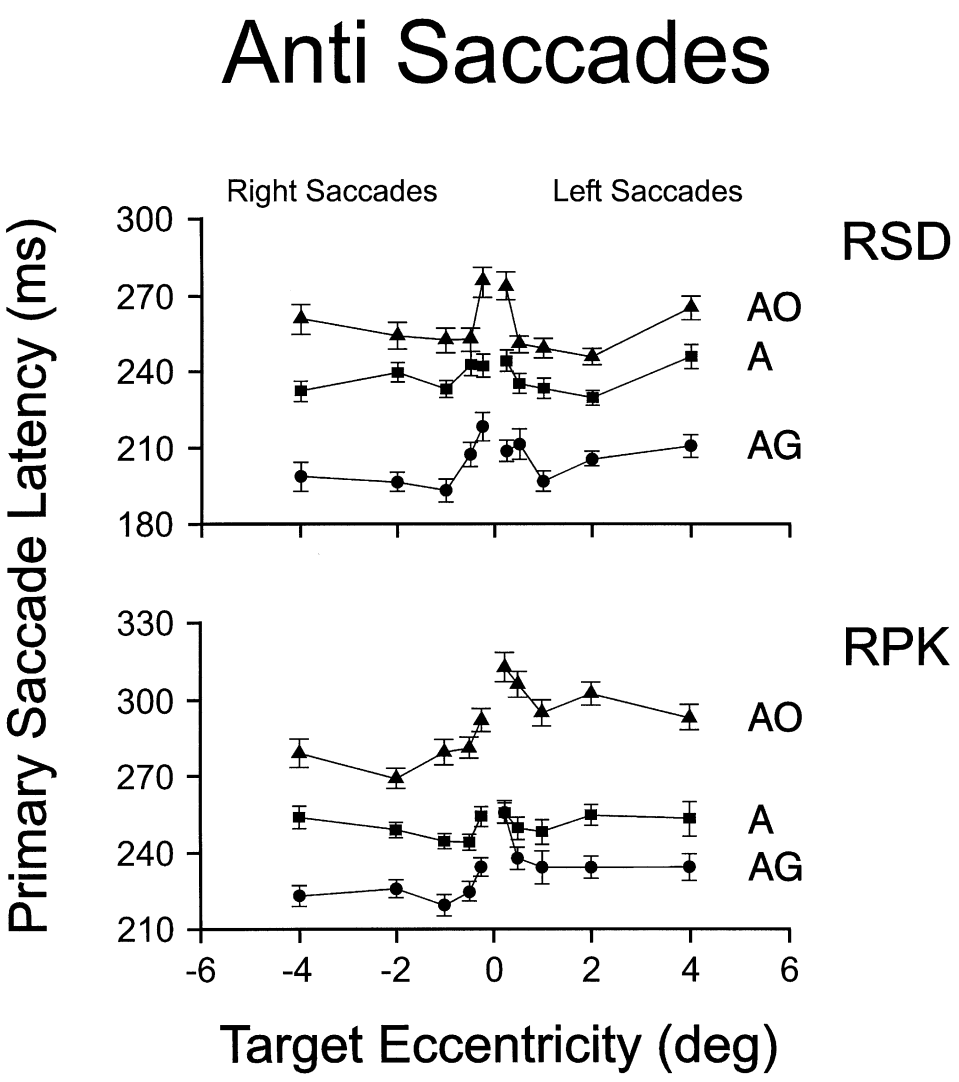

Fig. 7. Latency experiments for lighting conditions similar to the gap, normal and overlap data of Figs. 3 and 4, but with an anti-saccade as the required response. The corresponding mean latencies are designated **AG**, **A** and **AO**. Although 7.5′ target displacements were not used the data show that the foveolar latency peak persists with this change of task if plotted against retinal eccentricity. The peak is broader if plotted against saccade amplitude. The fixation point was always at the midline. Mean latencies ±1 SEM.

consistent with latency depending on broad representations of the virtual fixation point and target in some oculomotor map (Kalesnykas & Hallett, 1996). Conceivably sensory adaptation to the persistent fixation point contributes to narrowing of the neural image and thus of the central latency peak in the O and PFPT lighting conditions.

4.2. The rank order of latencies for the plateau

It was impressive that there was only one rank order of mean latencies for the five lighting conditions across subjects, sessions and target displacements. Given that mean latency $\mathbf{O} < \mathbf{PT}$, it follows from Eqs. (1d) and (1e), after rearrangement, that

$$iu_{\mathrm{O}} - du_{\mathrm{PT}} < it_{\mathrm{PT}} - dt_{\mathrm{O}},$$

where the subscripts allow that the delays might be lighting condition dependent. This inequality becomes a simple result when the dependencies are weak, as it reduces to the latency increase for indirect unlocking being smaller than for indirect targeting.

4.3. The actual latency values for the plateau

It is uncommon in behavioral studies to attempt to attach any meaning to absolute latency values because latencies typically vary from subject to subject and session to session, data are often sparse, and most theories are qualitative rather than quantitative. Two recent exceptions include a numerical formula for mean anti-saccade latency (Hallett & Adams, 1980), never to our knowledge replicated, and claims for a fixed latency peak for express saccades, irrespective of target salience,

Table 1
Solutions for direct and indirect delays of the unlock and target serial processing model (four subjects, latency plateau)

Theoretical delay	*du*	*dt*	*iu–du*	*iu–du*	*it–dt*	*it–dt*
Method of calculation	**N–G**	**G–E**	**PFPT–PT**	**O–N**	**PT–N**	**PFPT–O**
Grand mean, ms (SE)	43.8 (1.0)	32.0 (2.3)	45.3 (4.1)	42.7 (2.2)	80.4 (1.8)	83.1 (2.5)

which have been denied (e.g., Fischer & Weber, 1993, versus McPeek & Schiller, 1994). The present case seems more promising, as the exact form of the unlock and target model is overdetermined in Eqs. (1a)–(1f) of Section 1, so a rigorous test is possible. The model does seem useful so far as pooled data are concerned. Table 1 sets out the latency differences and standard errors (SEs) for pairs of lighting conditions, pooled across the eccentricities of the latency plateau and across the four subjects; from Eqs. (1a)–(1f) these differences could be used to estimate the four unlock and target delays. There is good agreement between the two estimates of the latency inflation, *iu–du*, due to indirect unlocking, and between the two estimates of inflation, *it–dt*, for indirect targeting. The SEs are also small, so the exact form of the model passes its validity checks with pooled data. Averaging across the four subjects appears to have been successful in minimizing *lighting condition* × *subject* interactions. Also, as already anticipated from the single observed pattern of rank orderings (see Section 4.2), delay *iu* appears appreciably smaller than *it*, by about 25 ms on average. Some authors might look further into data of this sort for evidence of 'temporal quanta' (Geissler, 2000; Kalesnykas, 1994; Kristofferson, 1990; Hallett, 1986).

4.4. Is the anti-saccade task relevant here?

We are not the first to study the gap and overlap lighting conditions with making an anti-saccade as the task, though the latency peak is a new finding. The latency plateau results in Fig. 7 match some studies (human: Fischer & Weber, 1992; primate: Bell, Everling, & Munoz, 2000; Everling & Munoz, 2000) in finding appreciable latency differences between the three conditions of Fig. 7. Others have found rather modest latency increases for overlap (Reuter-Lorenz et al., 1991; Reuter-Lorenz, Oonk, Barnes, & Hughes, 1995). Naturally we regret [7] that our anti-task data are not sufficiently extensive to develop a full hierarchy of latency plots, comparable to the foveating results in Fig. 4, and so lead to an anti-task model *analogous* to Eqs. (1a)–(1f). From our viewpoint, indirect targeting in the anti-task is a special delay (*at*) which likely involves cognitive switching of the nontarget across the midline, whereas foveating delay *it* of Eqs. (1a)–(1f) is a result of there being no recent target onset transient. Providing the viewing conditions are simple, it would be unlikely that *it* would equal *at*. In fact, the indications are that $at > it$ (given $\mathbf{A} - \mathbf{PT} = 16$ ms, *sig.*, $p < 0.05$, 3 subjects). What is perhaps of more immediate interest, though, is some form of counter proposal to the present unlock and target hypothesis—that the PT and PFPT conditions might be the anti-saccade task (Hallett, 1978) in disguise. There are two possibilities.

4.4.1. Anti-saccades to the nontargets?

Perhaps saccades in the PT and PFPT conditions are really 'anti-saccades' to the offset of the nontarget (Fig. 2, bottom, frame 2). This suggestion fails a number of tests. (A) *Main sequences*: Extensive analyses of the PT and PFPT saccades were normal, which is quite strongly against the presence of anti-saccades of generally longer duration, slower velocity and anomalous velocity profile (Hallett & Adams, 1980; Smit, van Gisbergen, & Cools, 1987; Kalesnykas, 1994). (B) *Accuracies*: An invariable characteristic of PT and PFPT, and the G, N and O foveating conditions, is that a small displacement of the target evokes an appropriately small saccade. This is not true when a simple aversion response is required; this laboratory has never encountered a subject who could make small anti-saccades matching the amplitude of target displacement (data not shown). (C) *Direction errors*: Some DEs occur in the PT and PFPT lighting conditions. However, DEs in the anti-task are generally at quite a different latency (≈**N**) to the DEs in the new lighting condition. The general rarity of errors frustrates strong claims but there seem to be two latency groups in the new conditions: anticipatory (earlier than $\boldsymbol{VGL}_{\min}$) and late (at about the same latency as the correctly directed saccades). Both groups were common in the PT and PFPT conditions only in our shortest latency subject (the naïve subject AJB with 4% and 21–26% of anticipatory and late DEs, respectively), infrequent for HDK (1% and 5–9%) despite his earlier experience of the anti-task, and rare for the other two main subjects (<1% and 1–2% for anticipatory and late DEs, respectively), one of whom was new to oculomotor studies. Very small numbers of DEs occur in the G, N, and O conditions as well, so the occurrence of DEs can scarcely

[7] We also regret that it is not easy to apply our model to the wider reaction time literature. The number of compared conditions is often too few (e.g., Saslow, 1967) and the stimuli, and possibly task, complicated. For example, in some studies fixation 'point' cues and targets change in outline or fill (Walker et al., 2000) and there are persistent fixation guidelines intruding on the foveola (Ross & Ross, 1980), that are expected to cause inhibition (Weber & Fischer, 1994; Kalesnykas & Hallett, 1996; Walker et al., 1997).

be a completely reliable diagnostic of surreptitious anti-saccades. (D) *Latencies*: One would expect latencies **PT** = **A** and **PFPT** = **AO**. We do not have extensive data on this issue but have apparently just refuted the first equality. *In brief*, there are currently no grounds for believing that the PT and PFPT saccades are anti-saccades.

4.4.2. A mixed process?

An alternative proposal is that a PT or PFPT saccade is a foveating response to the persistent target which is, in some way, expedited by concurrent latent anti-saccade processing in response to the offset of the nontarget. This might be compatible with the observations of normal accuracy and a normal velocity profile. We do not see this proposal as being a counter proposal because the latency equations (Eqs. (1a)–(1f)) do not change. The only change is that the indirect targeting delay *it* is now reinterpreted as being a mixed process, a possibility that is susceptible to further investigation. [8] In short, a simple unlock and target serial processing model remains for us the parsimonious, practical explanation of all the experiments.

4.5. Multiple modes of successive planning

Even if one had some reason for rejecting the unlock and target hypothesis, one would still be left with distinct latency modes for the five stimulus lighting conditions. There are two possible objections to the idea that lighting manipulations have isolated natural modes of successive planning. (i) *The modes might be elastic artefacts*: Distinct modes might merely arise from very large, discrete changes in the lighting conditions. If so, it should be possible to produce mean latencies intermediate between, e.g., **N** and **O** or between **PT** and **PFPT**, by dimming the fixation point rather than switching it off in the normal or PT conditions (cf., Newman, 1971). If this were to prove true, we would still expect (if the terms direct or reflexive, and indirect or voluntary, are in fact useful dichotomies) that intermediate mean latencies in dimming experiments will prove to be mixtures of **N** and **O** or **PT** and **PFPT** population means. A related idea, that some latency means may represent variable mixtures of separate but overlapping latency populations, has recently passed two computer-intensive statistical tests for two very different sets of viewing conditions (Gezeck, Fischer, & Timmer, 1997; Lau, 1998, this laboratory). (ii) *The modes do not coexist*: Another objection is that we never demonstrate the presence of two latency modes for the same lighting condition. We counter that isolating modes is an important part, though admittedly just one part, of demonstrating their existence. For old evidence of mixed latency modes in two-step tracking experiments see Hallett (1986). In conclusion, the unlock and target serial processing model seems a promising summary of our data.

[8] A minor experimental change that would test for any contribution from latent anti-saccade processing would be to repeat our PT and PFPT lighting conditions but with multiple simultaneous nontargets (after the fashion of Todd & van Gelder, 1979). Such responses should be pure foveations.

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