

PRIMARY AND SECONDARY SACCADDES TO GOALS DEFINED BY INSTRUCTIONS

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(Received 6 July 1977; in revised form 7 December 1977)

Abstract—A luminous point steps horizontally in the dark, and the subject tracks it (normal task), or is instructed to respond by some other horizontal eye movement (e.g. an equal and opposite movement—the “anti-saccade” task). Eye movements in the “anti-task” are characterized by long latency, inaccurate primary saccades which sometimes show minor anomalies in velocity profile. The secondary saccades are large, corrective, of shorter than primary latency, and are not based on retinal feedback. Thus, the human saccadic system is optimized for, but not restricted to, foveation. The highly idiosyncratic “anti” latency data can be normalized by reference to the Wheelless 2-step paradigm. A mechanism is proposed.

INTRODUCTION

There is a common divergence of opinion as to whether saccadic eye movements are best regarded as being the versatile result of high level, voluntary, cognitive and perceptual processes, or as being more limited, lowly reflex, servomechanistic responses, which form an early part of general bodily orientation to a lateralized stimulus. Recent examples of these views are shown in Monty and Senders (1976). Of course, both viewpoints may be correct, the former being appropriate, possibly, to visual search in a complex environment, the latter to tracking a single visual stimulus in the dark.

With respect to orientation, a recent issue is whether saccades are simply responses to retinal error alone (for example, see review of Robinson, 1973), or whether saccades are directed at the real positions of objects—which requires in most cases additional accurate information about current or anticipated eye position. Thus a flashed target, presented at the time of a saccade, may be perceptually mislocated (e.g. Matin and Pearce, 1965; Matin, 1976; Pola, 1976), but in similar oculomotor experiments the eye executes a normal latency, normal accuracy saccade to the now extinguished target (Hallett and Lightstone, 1973a,b, 1974, 1976 a,b; Hallett, 1976). Accurate ocular orientation has also been claimed by Levy-Schoen and Blanc-Garin (1974), Becker and Jurgens (1975), Gresty and Leech (1976), and Ono and Nakamizo (1977), in different tasks and situations.

Thus, a reasonable hypothesis is that the saccadic mechanism is specialized for reliably foveating real objects, but maintains some limited capacity to point the fovea in any direction at will. This hypothesis is supported for the present experimental conditions, and is in sharp contrast to my original working hypothesis: I hoped to show that the fovea is only one of many possible retinal goals, and that a subject could drive the retinal image anywhere at will over

the retina, without special training, *by making use of retinal feedback* as to the error between the retinal image and the “retinal goal”. This ought to be so. I reasoned, because the foveal region is night-blind, and the optimal location for a dim image is in the retinal periphery (e.g. Stiles and Crawford, 1937; Hallett, Marriott and Rodger, 1962; but see Steinman and Cunitz, 1968, for a cautionary clue), and I was also reacting against the possibility that the human saccadic system might be limited or inflexible.

I use the term “primary” saccade for the first saccade in a response, and “secondary” saccade for the second response if there is one within 350 msec of the end of the primary. I make use of Becker’s concept (Becker, 1972; Prablanc and Jeannerod, 1975; Becker, 1976) that secondary saccades are pre-programmed, and that there are varieties of secondary saccades which differ substantially in their use of retinal feedback. Results for three novel tasks will be presented. The subject can be instructed to make an eye movement only half as large as the stimulus step (the “hypo” task), or a fixed amount larger (the “hyper” task), or of equal and opposite magnitude (the “anti” task), i.e. to change the gain, offset or sign of the saccadic response to a random horizontal stimulus step. From the Results section it seems clear that saccades are optimized for foveation. In the case of the most fully studied task (the “anti” task) the subjects can drive the retinal image to a specified position (i.e. deeper into the periphery), but they do so after a longer than normal latency, with a primary saccade which sometimes shows an anomalous velocity profile, and which usually leaves a large end-saccadic error. Secondary saccades are certainly corrective, on average, but are not based on retinal feedback. Goals are achieved, but only with low accuracy and long total latency. Practice does lead to a striking reduction in accidental or reflex foveations of the retinal image, but practice of 1000 or so trials (with rich feedback of performance) does not reduce the

latency or angular errors of peripheralizing ("anti") saccades. "Anti" latencies are predictably related to normal latencies (see item 17 below), so the saccadic system does seem to follow normal timing rules, once the reflex response is aborted.

METHODS

These have been fully described in the previous papers (Hallett and Lightstone, 1976a,b). Eye movements are measured by means of a non-contacting near-infrared technique which monitors the left and right edges of the dark-adapted pupil. The original electronic equipment has been refined by F. G. Oakham, who has also further stabilized the light output of the oscilloscopes and built special computer interfaces. Figure 1 shows the detailed protocols. Measurements of time, target and eye position are made on stimulus steps and when eye velocity passes through low trigger values, specified in later figures.

The target was a nominally 8' subtense oscilloscope spot at 100-times foveal threshold (fast decay P15 blue-green phosphor), viewed monocularly at an effective distance of 72.5 cm. Peak-to-peak electrical noise in the eye movement monitor is 6'; digitization is in steps of 2.3'; linearity is within 5%; frequency response (20 Hz corner frequency) follows a linear phase characteristic so as to optimize the

step response: latencies are measured to 1 msec. Only the horizontal component of eye movement is measured—the device is insensitive to vertical movements or large sudden changes in pupil size, or to lid droop if the eye is kept reasonably open.

Subject H was 37, PEH 39 and EC 23 years old at the time of the experiments. The subjects had 20/15 vision in their left eyes, which they used to view the target. All were right-handed and PEH differed in being left eye dominant. Subject H had suffered an inferior temporal branch vessel block in her right eye 1 year previously, which is probably irrelevant to the present study. Subject PEH is 2.5 D myopic in his right eye.

Feedback of performance and other arrangements

For the experiments on the N (normal) and A (anti) tasks, with subjects PEH and EC, automatic feedback was introduced with the aim of optimizing performance. Figure 2 shows that a trial is divided into 3 phases: fixation, task and reinforcement. (Ignore for now the full and broken ellipses at the lower left—they were only displayed in the instruction-on-the-step experiment). The subject triggers the equipment, and after a delay the stimulus lights to initiate the fixation phase, which establishes the initial conditions. At the instant of the random target step a low pleasant tone commences—it is terminated by the primary

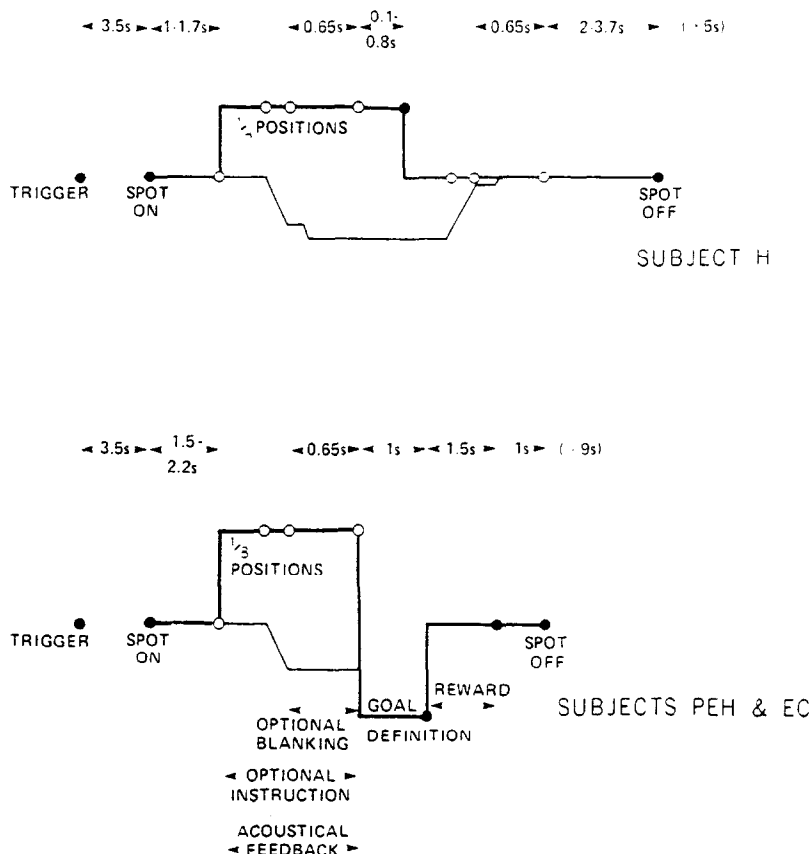


Fig. 1. Format for a trial (not to scale). The subject triggers the trial, and after a delay the target lights, steps, and eventually returns to centre, and is extinguished. Certain phases of the trial have fixed durations, as shown, but the trial length varies because the reaction time varies, and because other phases are of random duration (for these a range of times is indicated, and the range is covered by eight possible values in steps of 0.1 sec). The horizontal target step is one of eight values ($\pm 3.8^\circ$, $\pm 7.6^\circ$, $\pm 11.5^\circ$, $\pm 15.3^\circ$). Eye position (thin line) is measured at the times shown by open circles: at the initial stimulus step, at the beginning and end of the primary saccade, and 0.65 sec after the end of the primary saccade. Top: Arrangements for subject H, who received no feedback whatsoever as to performance. Bottom: Arrangements for the other two subjects, who received rich feedback (see Fig. 2).

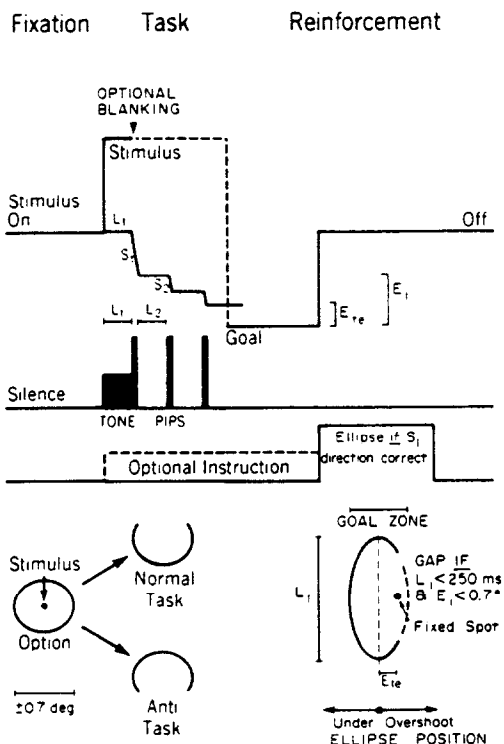


Fig. 2. Feedback arrangements (Subjects PEH and EC only). Schematic, not to scale, showing the 3 phases of the trial (fixation, task and reinforcement), the stimulus motion and lighting, the acoustic feedback, the goal definition at the end of the task, one example of the reward pattern, and the optional visual instruction (Figs 9 and 11 only), and optional blanking arrangements (Figs 8 and 10 only). S_1 and S_2 are primary and secondary saccades, L_1 and L_2 are their latencies, E_1 is end-primary error and E_{te} is terminal error at the end of the task period. Other details in Methods and Fig. 1. The breaks in the ellipses are actually larger than are shown, almost yielding semi-ellipses.

saccade, and this signals primary latency and provides a small sense of urgency. Each saccade generates a tone pip. At 650 msec after the end of the primary the stimulus steps to define the goal position (A-task), or remains at the same position (in order to establish the N-goal), for 1 sec prior to returning to centre. The acoustic and goal-defining feedbacks establish latency, number of saccades and terminal angular error in a very natural manner, which is reinforced more intellectually as well. When the stimulus returns to centre, an ellipse (Fig. 2, bottom right) is lit as a partial "reward", if the primary is toward the stimulus in the N-task, or away from the stimulus in the A-task. The width of the ellipse ($\pm 0.7^\circ$) defines the size of the "goal zone"; the horizontal position of the ellipse relative to the stimulus spot is the terminal error which remains at the end of the task period, 650 msec after the end of the primary saccade; the height of the ellipse is one of eight values which are graded to span the range of primary latency. Finally, this partial reward pattern is supplemented by a right-hand gap as a full reward, if primary latency is less than 250 msec and if the end-primary error is less than $\pm 0.7^\circ$. The reward patterns are not shown if the primary goes towards the stimulus in the A-task or away from the stimulus in the N-task (a direction error), and the subject is obliged to press extra buttons and reject the trial from analysis.

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One can paraphrase the feedback arrangements by saying that the subject is playing a solo game of skill with the intention of securing a short C centred on the central dot. His ideal performance is a quick hit of the goal zone with his primary saccade, followed by either no secondary, or else by a secondary which corrects his aim. The probability of the "full" reward (C shape) is not given, but equals the probability pQ of a short latency ("quick") primary times the probability pH that end-primary error is less than $\pm 0.7^\circ$ (a "hit"), as shown in the later figures. This suggests independence of primary latency and end-primary error (Becker, 1972; Prablanc and Jeannerod, 1975) which was confirmed by scatter plots of these quantities. Thus the acoustic feedback of latency does not give immediately useful indication of error during the task period. Also, it is obvious from pQ and end-primary hit rate pH in the figures that full rewards are quite common in the N-task, but rare in the A-task.

In the sessions with blanking (Figs 8 and 10) the stimulus is blanked with 50% probability when the specified velocity trigger level is exceeded at the very beginning of the primary saccade, so as to deny retinal feedback of intrasaccadic retinal image motion and end-primary error. In the instruction-on-the-step experiment (Figs 9 and 11) the arrangements at the bottom left of Fig. 2 apply—the central stimulus is surrounded by an ellipse with $0.5 \times 0.7^\circ$ minor and major semi-axes. This is broken at top and bottom randomly, with 50% probability, to signal N- or A-task at the instant of the stimulus step, thus delaying the subject's response. The visual instruction remains lit at centre until it is blanked at the end of the task period, and also serves as a landmark of the straight-ahead.

OVERVIEW

Subject H

The subject was given the instructions for the task at the beginning of that session's dark-adaptation period. When ready, she assumed a good posture, biting into a deep, hard dental impression, and triggered a trial. The small, blue-green stimulus lit, and then stepped randomly to one of eight positions in the range $\pm 15.3^\circ$. These positions will be called "positions 1,2,3,4, left or right" for convenience. Her eye movements were aimed at the goal position defined by the instruction and the stimulus step. Apart from the stimulus nothing else was visible—the very dim near infrared source for the eye movement monitor was usually invisible, because of the Troxler fading effect, and was too remote to serve as a landmark. At a random time after the end of the first (i.e. primary) saccade the stimulus returned to centre, and was extinguished some time later, after the end of her return (primary) saccade. Although subject H was given encouragement she was given no feedback whatsoever as to the nature of her responses or performance. Figure 3 illustrates the results for the different instructions, retinal image position being the electrical difference between stimulus and left eye position. The base-line is the fovea in these records. Figures 4 and 5 show the data that Fig. 3 illustrates.

Normal (N) sessions

In this case the task was simply to track a stimulus which would step to 4 leftward or 4 rightward positions. The symbols at the extreme left of Fig. 3 (top) show the eight possible retinal image positions and the single foveal goal. The left trace shows that the

MODIFICATION BY INSTRUCTIONS OF SACCADES TO HORIZONTAL STEPS

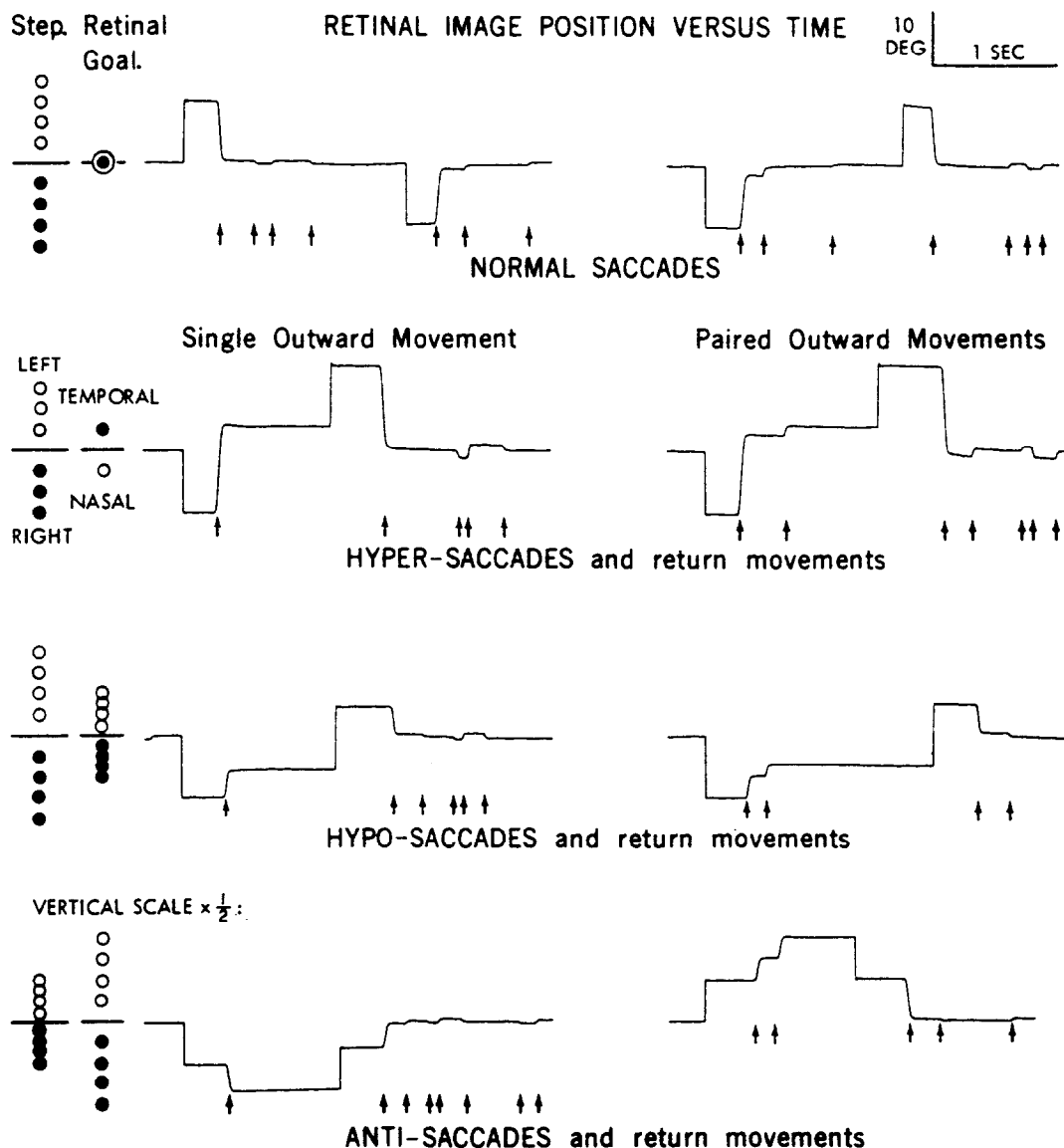


Fig. 3. (Subject H, no feedback.) Representative trials. The baseline is the fovea, with left (temporal) hemi-retina above and right (nasal) hemi-retina below. The symbols at the left show the retinal positions of the stimuli, and the retinal goals implicit in the instructions. The left-hand traces are more common outcomes than the right-hand traces. The steps in the retinal image traces are due to the stimulus stepping away from, and back to, the straight-ahead. The arrows show saccades. Primary latencies, and end-primary and terminal angular errors are worse in the three instructed tasks. Secondary saccades are less frequent, large and corrective.

retinal image steps into the periphery and is brought nearly back to the fovea by the first, or primary, saccade. Shortly afterwards smaller secondary saccades bring the target to the fovea. The right-hand response in Fig. 3 (top) is less usual because the corrective, short latency, secondary saccade is so large. The traces also show the retinal image stepping again into the retinal periphery, when the stimulus returns to the straight-ahead, and the consequent return saccades.

Hyper-saccade (Hr) sessions

For technical reasons the stimulus at $\pm 15.3^\circ$ ("4L

or 4R") was deleted. The instruction was "The stimulus will step to the 3 inner right and 3 inner left positions as usual. You should aim to make an eye movement which is 1 unit bigger; so if the stimulus goes to position 2 left (say) you should aim to go past it 1 unit to position 3 left, and so on". The symbols at the extreme left of the appropriate row in Fig. 3 show that the two "retinal goals" implicit in the instructions each lie a fixed distance into the opposite hemi-retina to the retinal stimulus. The left trial shows the usual result—after a slightly long latency the retinal image is swept through the fovea to a position which misses the exact "retinal goal", but there

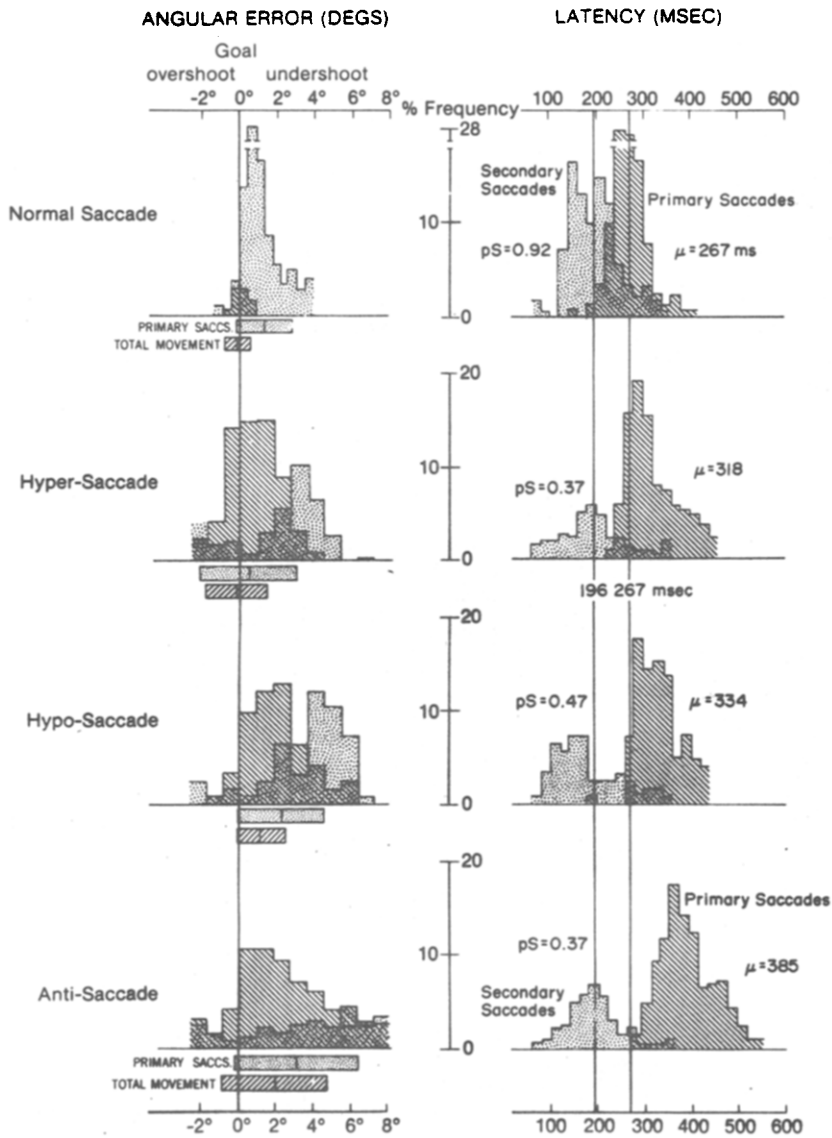


Fig. 4. (Subject H, no feedback.) Each row is for a separate instruction and shows about 300 trials accepted over three sessions, after rejecting an initial practice of about 40 trials. Right: Secondary saccade latency is relative to the end of the primary saccade, and is truncated at 350 msec. The proportion of primaries followed by secondaries, pS, is reduced, and primary latency is increased, in the 3 instructed tasks. Left: The horizontal rectangles below the histograms (Primary Saccs. and Total Movement) show $\mu \pm \sigma$ for end-primary angular error and terminal error 650 msec later, errors being relative to the goals defined by the instructions. Total movement is better, so secondaries are corrective on average. The superimposed histograms show end-primary errors sorted according to whether there is (stippled) or is not (hatched) a secondary saccade within 350 msec—the larger errors tend to elicit secondaries. The Hr- and Ho-tasks are intermediate in latency and angular error between the N- and A-tasks. Trials with accidental fixations towards the stimulus were not accepted for analysis and occurred with about 8% frequency. Trigger levels 12.5°/sec for N and 50°/sec for the other tasks.

are no corrective secondary saccades. The right-hand trial shows the less usual result—the end-primary saccade error is particularly large, and there is a conspicuous secondary saccade, which is corrective. The returns at the ends of the trials are quite normal.

Hypo-saccade (Ho) sessions

In this case the instruction was: "If the stimulus steps to position 4 left (say), then you should aim to go only half as far, i.e. to position 2 left, and so

on". The symbols, at the extreme left of the appropriate row in Fig. 3, show that the instructions imply eight "retinal goals", each in the ipsilateral retina, half way between the retinal stimulus and the fovea. The more common (left) and less common responses (right) show that the task is executed with a longer-than-normal latency, inaccurate, primary saccade, which is only followed by a short latency, secondary corrective saccade when the end-primary error is large. The secondary saccade is either absent (left) or is conspicuously large (right).

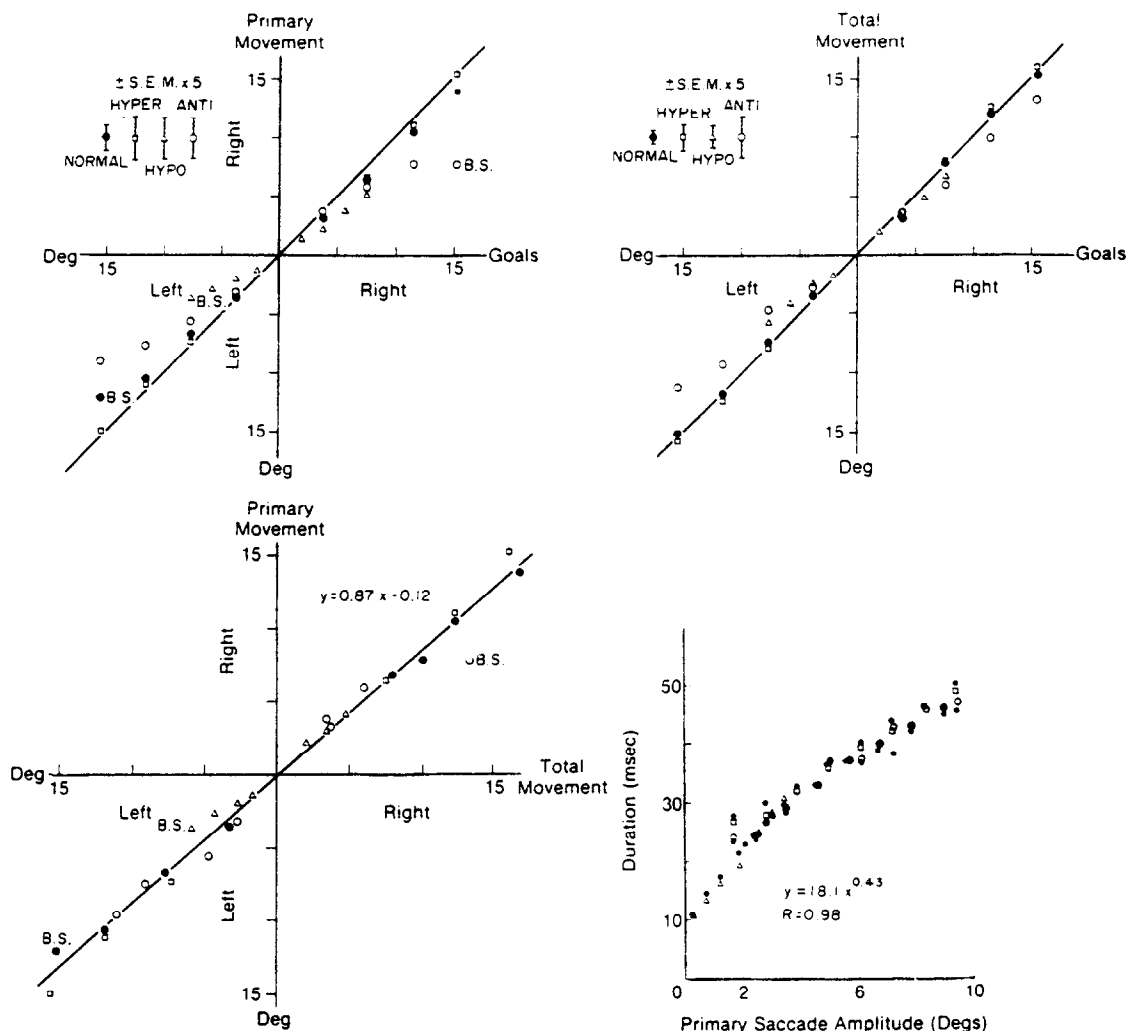


Fig. 5. (Subject H, no feedback). Data of Fig. 4 replotted. Top left: Primary movement is here graded to goal (reference line is slope 1). Top right: Mean total movement is better (line is slope 1). Bottom left: Primary movement is 0.87 of total movement. Bottom right: The primary saccades for the various tasks, and their return saccades to centre (shown as dots), approximate a single amplitude-duration relation. BS—the stimulus at Position 4L (-15.3°) falls on the physiological blind-spot. S.E.M. is standard error of the mean, enlarged 5 times.

Anti-saccade (A) sessions

The instruction was: "If the stimulus steps to position 4 left (say), then you should aim to go an equal and opposite distance, i.e. to position 4 right, and so on". The symbols at the left in the bottom row of Fig. 3 show that the instructions imply eight "retinal goals" each in the ipsilateral retina, twice as far from the fovea as the corresponding retinal stimulus. The results are much the same as for the Ho and Hr conditions, but more striking. The usual response (left) is a delayed, very inaccurate, primary saccade, which is more usually not followed by secondary saccades. If the end-primary error is unusually large, however (Fig. 3, right), then shorter latency, large, corrective secondary saccades tend to occur.

The instructions compared

The subject was able to execute any of the tasks on the first or second attempt, but error trials (a pri-

mary saccade to fixate the stimulus) were more frequent in the first 50 or so trials. A complete search through all the data (3000 or so trials for all three subjects) has failed to show any small saccades towards the stimulus, during the latent period between the stimulus step and the instructed Hr, Ho or A saccade (though, as expected, there were a very few tiny spontaneous shifts of fixation in either direction within 100 msec of the step)—accidental or reflex saccades to the stimulus are either large conspicuous errors (e.g. Fig. 13, bottom, record 3) or else are completely absent. Each type of instruction was given to subject H in sessions on 3–4 days, but the only noticeable effect of training was a steady reduction in the frequency of error trials. Primary saccade latency showed the usual day-to-day fluctuation, but no trend. There were, however, suggestive trends in the amplitude of the primary saccade towards the instructed goal. Thus the Ho-saccades progressively diminished, and in fact the smallest stimulus step of

$\pm 3.8^\circ$ has to be eliminated because of the subject's distress as to whether or not she had responded (she always did, but, of course, feedback as to performance was denied). The Hr-saccades tended to increase in mean amplitude, and the A-saccades were roughly constant, from session to session.

Subjects PEH and EC

Following these results with subject H, automatic feedback of performance (primary latency, angular errors and number of saccades) was introduced in the hope of stabilizing and optimizing the performance of the next two subjects, PEH and EC, and they were allowed to see their records between sessions. These arrangements are illustrated in Fig. 2 and are described in Methods. Of the three novel tasks only the A-task was investigated, for the practical reasons that performance is relatively stable and errors (reflex fixation towards the stimulus) are easily and unambiguously recognized by the experimenter "on-line". There is also the theoretical advantage that the A-task

is comparable (see later) with the extensively investigated "pulse over" pattern of the Wheelless two-step experiment (Wheelless, Boynton and Cohen, 1966), because in both tasks the subject can substitute an oppositely directed saccade for the initial saccade that he might be expected to make. (The Hr and Ho tasks may also be comparable with the "pulse over" and "stair-case" patterns of the Wheelless paradigm, but those patterns have not been much investigated, except by Becker and Jurgens, 1975.)

The first experiments on PEH and EC are just like those described for subject H, and are called "session experiments" because whole sessions are devoted to a single instruction, either N-task or A-task. The next experiments are "sessions with blanking", where the stimulus is blanked with 50% probability at the very beginning of the primary saccade. These experiments show that the A-secondary saccades are corrective on average, but differ from normal in being quite insensitive to blanking. The concept of "retinal goal" is therefore quite inappropriate in the A-task, at least,

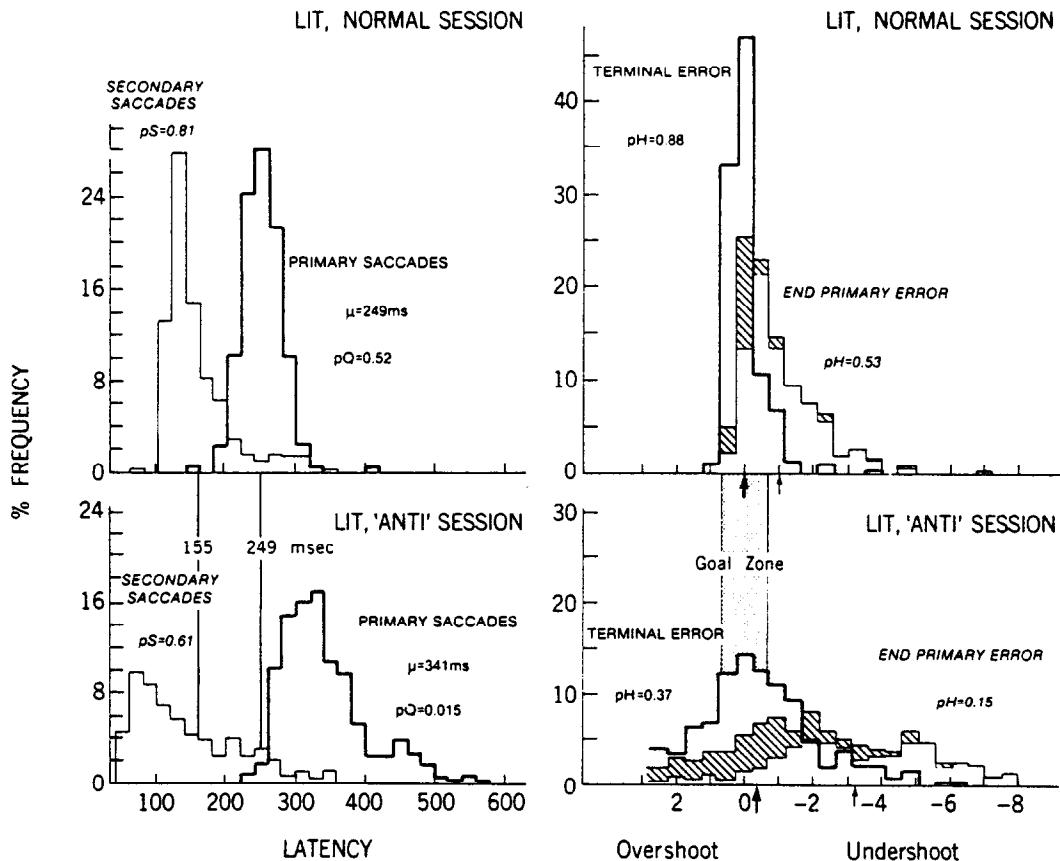


Fig. 6. (Subject PEH, feedback.) N- and A-session experiments of about 400 trials each, begun after about 300 trials practice at the A-task. Right: Goal zone is goal position $\pm 0.7^\circ$. The conspicuous histogram in heavy line (bold data) is terminal error, 650 msec after the end of the primary, and the heavy arrow points to its mean. The less conspicuous doubly-shaded histogram (italic data) is end-primary angular error and the light arrow points to its mean—the empty zones are the trials where the primary saccade is followed within 350 msec by a secondary. pH is the proportion of end-primary or terminal eye positions that hit the goal zone. A-angular errors are large. Larger errors are more likely to evoke secondaries. Secondaries reduce error on average. Left: Latencies as in earlier figures. In the A-task primary latency is long— pQ is the proportion of quick primaries of less than 250 msec latency. The proportion of primaries pS followed by a secondary in less than 350 msec is reduced, but the absolute frequency of very short latency secondaries is much increased. Trigger levels 12.5 and $20^\circ/\text{sec}$ for the N and A sessions, respectively.

because there is no useful intra-saccadic or post-primary retinal feedback—it is better to say that instructions and stimulus step define an “eye position” goal. The last experiment on subjects PEH and EC is called “instruction on the step”, in which the instructions N and A are randomly mixed, being given by a visual pattern at the instant of the stimulus step. The object of this experiment is to force the subject to delay his response, to provide him with a visual land-mark of the straight-ahead, and to permit him to compare the N and A tasks in the same session with the aid of automatically fed-back performance. Nevertheless, angular errors and secondary latencies are much as usual, and the very small improvement in EC’s end-primary aim could equally be attributed to practice. The various results are shown in the figures, which are grouped by subject, and are in the order of the experiments. The general reader may wish, on a first reading, to pass swiftly over the details of the Results to the Discussion.

RESULTS

The Normal (N) and Anti- (A) Tasks Compared

1. *A-primaries usually have longer latencies than N-primaries*

This is clearly true in the mean—for session experiments

(Figs 4 and 6) and sessions with blanking (Figs 8 and 10), the increase being 40–120 msec, depending on the subject—but is not true for individual trials because the distributions partly overlap. Giving the instruction on the step, with 50% probability of N or A being required, increases both N and A-primary latencies as might be expected, but the latency advantage of N-primaries over A-primaries is preserved for EC (Fig. 11) and lost in the data shown for PEH (Fig. 9).

2. *Primary latencies may be multimodal*

Primary latencies are never realistically Gaussian, but are typically skewed to longer latencies (Figs 4, 6, 8 and 10), and may suggest minor late peaks (Figs 4, 6, and 8) in some subjects. If confirmed, these suggestive peaks imply that latency increases quantally if internal deadlines are not met.

3. *A-angular errors are worse than N-angular errors*

This is very obvious, for both end-primary and terminal errors, whether one uses the criterion of accuracy in spread, probability of hitting the “goal zone”, pH , or the shape of the histogram (Figs 4, 6, 8, 9, 10 and 11). Accuracy in mean terminal error, however, may be very similar in the N and A tasks, showing that the subject knows what is required, but cannot achieve it reliably.

4. *Primaries usually undershoot the goal*

This is certainly true in the mean for both N- and A-primary saccades, as can be seen when the end-primary

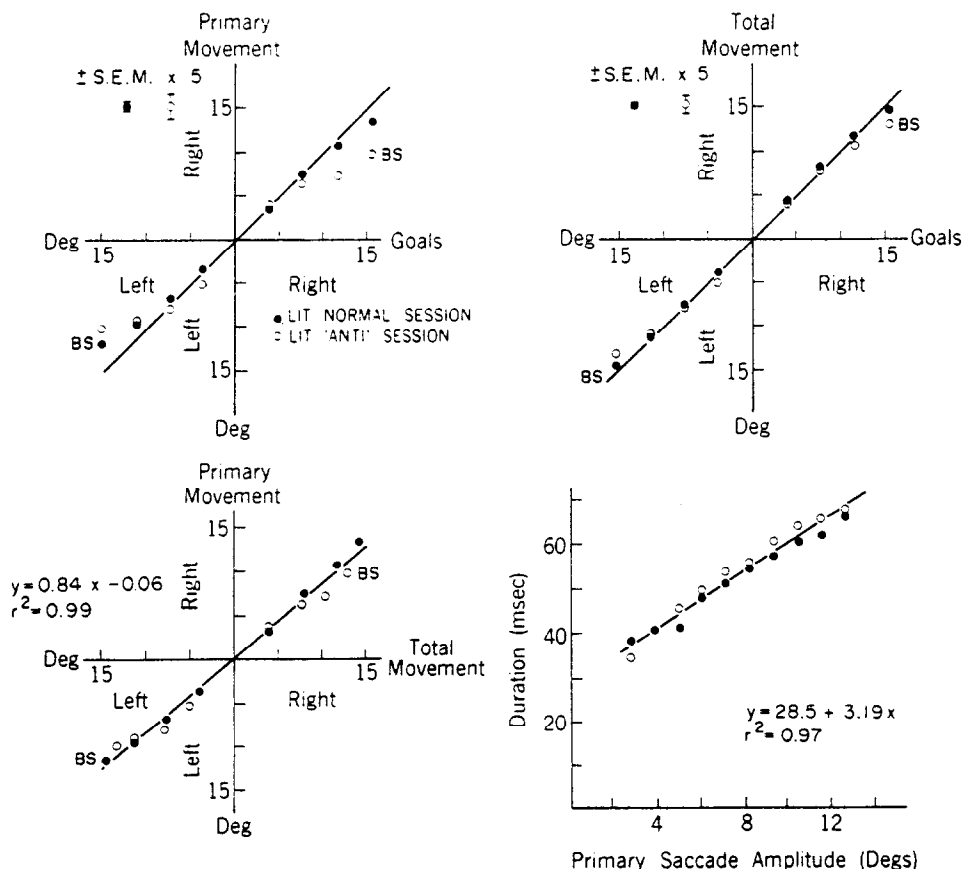


Fig. 7. (Subject PEH, feedback.) Same data as Fig. 6 replotted. Top left: Primary amplitude is graded and idiosyncratic, (line is slope 1). Top right: Mean total movement better approximates the goals (line is slope 1). Bottom left: Primary amplitude is 0.84 of total movement. Bottom right: The amplitude-duration relation for primary saccades is linear. For this subject A-primaries tend to be longer than normal by about 2 msec, and velocity profiles of A-primaries show minor anomalies in most of his trials.

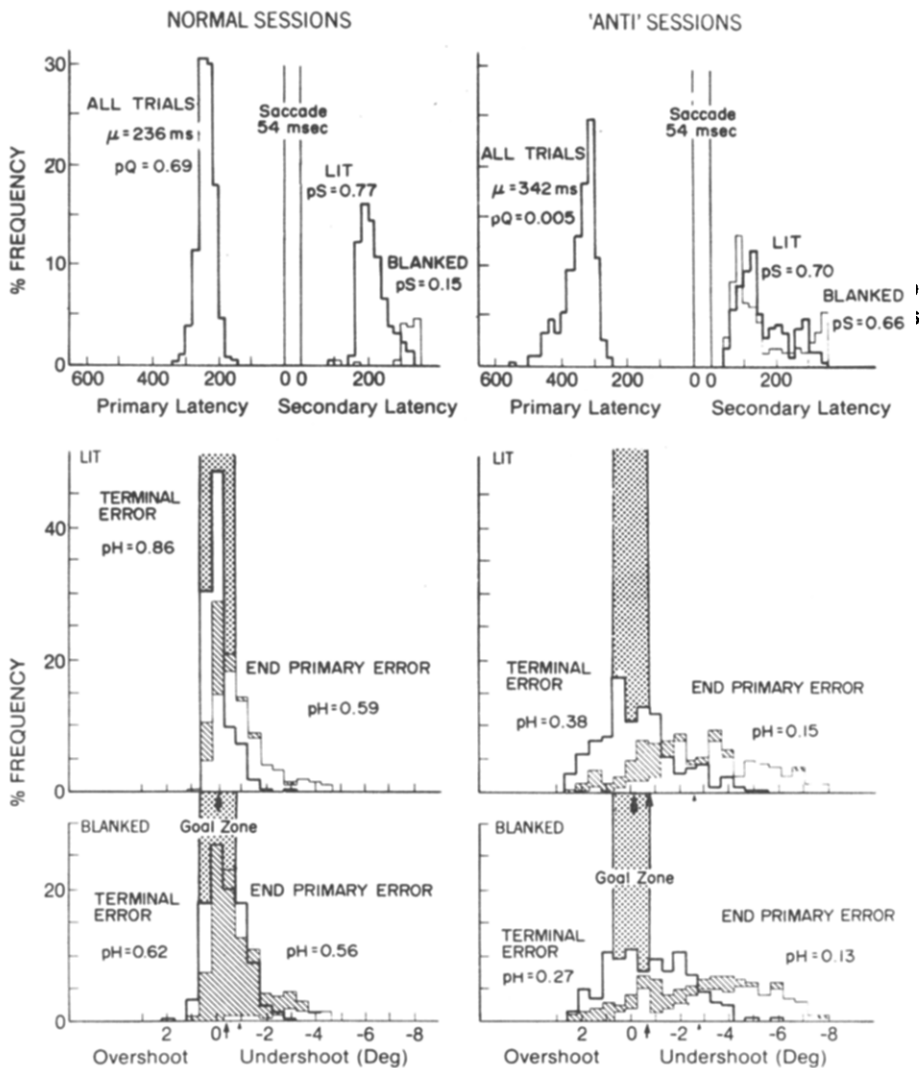


Fig. 8. (Subject PEH, feedback.) Sessions with blanking at the start of the primary saccade with 50% probability. About 430 trials for each instruction. Top: Primary latencies are plotted backwards and so indicate time of stimulus relative to the start of the primary saccade. Secondary latency histogram in heavy line is for lit trials, light line is for blanked trials. Blanking eliminates most N-secondaries, sparing the shortest latencies and increasing the frequency of the longest (which are truncated at 350 msec). Blanking does not affect A-secondaries. Middle and bottom: Plots of angular errors as in Fig. 6. Terminal errors in heavy line with means indicated by heavy arrows. End-primary errors doubly shaded (the clear regions being those errors which elicit secondaries), with the mean indicated by a light arrow. End-primary errors identical for lighting and blanking as they should be. N-terminal errors worse for blanking, but A-terminal errors not significantly affected. Data collected after about 723 trials practice at the A-task. Trigger levels $12.5^\circ/\text{sec}$ for the N-task and $20^\circ/\text{sec}$ for the A-task.

errors for all goals are pooled (Figs 4, 6, 8, 9, 10 and 11), but all of these distributions show a proportion of over-shoots.

5. A-primary amplitude is idiosyncratic

When the A-primary amplitude data are broken down by goals interesting idiosyncracies are revealed. Thus, subject H shows a graded undershooting response to A-goals, which is curvilinear rather than proportional (Fig. 5, top left), while PEH consistently overshoots the A-goal at "position 1L", approximately hits the A-goals at "2L" and "1R", and undershoots the others (Fig. 7, top left). These 2 subjects were able to execute the A-task on the first or second trial, and their initial tendency to fixate the stimulus, though high (e.g. 30%), dropped very rapidly in

the course of the first 30–100 trials. Subject EC's accidental or reflex fixations were very frequent in his first 100 trials (80% or so), and although this quickly fell with practice to the same low rate as other subjects (5–7% of trials) he was not able to grade his A-primaries to the A-goals in most experiments—his A-primaries were in the correct direction but were of crudely fixed amplitude (Fig. 12, bottom left, $\circ \bullet \Delta \square$). This fixity of amplitude accounts for the flat distribution of end-primary A-errors in Fig. 10, which is in contrast to the weak undershooting peaks shown by the other subjects (Figs 4, 6, 8 and 9) for the A-task. In his last three sessions, after 1200 trials practice, and when he was forced to delay by giving the "instruction on the step", EC's A-primaries became slightly more graded to the goals (Fig. 12, bottom left, \blacktriangle), though only

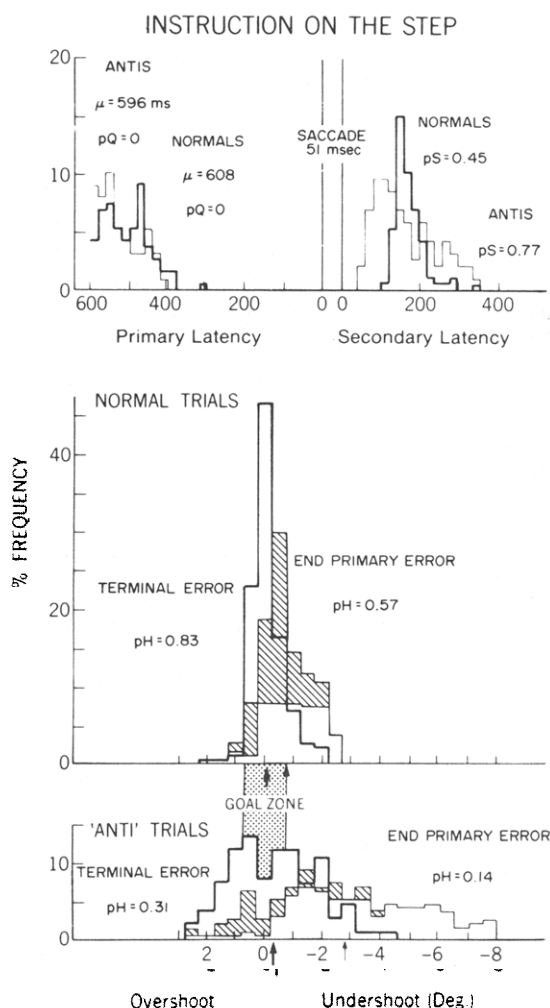


Fig. 9. (Subject PEH, feedback.) Instruction-on-the-step experiment. About 180 trials for each instruction. Top: Latencies plotted as in Fig. 8, but many primary latencies off scale. N-task latencies in heavy line, A-task in light line. N- and A-primaries equally delayed in these data. A-secondaries increased in absolute frequency at short and long latencies relative to N-task. Middle and bottom: Angular errors plotted as in Fig. 6. Terminal errors in heavy line, with the mean indicated by an heavy arrow. End-primary errors shown doubly shaded (the clear regions being errors which elicit secondaries), the mean indicated by a light arrow. The extra delay and persistent landmark have no effect on the angular errors—A-errors still much worse than N-errors. Data collected after 1126 trials practice at the A-task. Trigger levels $20^\circ/\text{sec}$ for both N- and A-tasks.

crudely so, and the pooled end-primary errors (Fig. 11) now show a hint of a weak undershooting peak.

6. A-primary velocity profiles are sometimes anomalous

Figure 13 (top, N) shows position and velocity profiles for the N-task. The velocity profiles for this amplitude range and instrumentation are smooth, narrow, and fairly symmetrical, with tight peaks and without conspicuous tails. Similar profiles are common in the instructed tasks, and even occur sometimes in darkness (Fig. 13, top, Hr, Ho, A, dark). However, in some trials for some tasks and some subjects there are qualitative anomalies that are not seen for the same subject in the N-task: most striking are

abrupt inflections or discontinuities indicated by short lines in Fig. 13 (bottom) for subject H (some Hr-trials) and PEH (some A-trials). A fourth subject, EGM, shows anomalous profiles (mainly discontinuities) in about half of his A-trials. Lesser criteria are broadening of the peak and asymmetry. If the occasional minor anomalies are equated with clumsiness, then they tend to support the present view that the saccadic system is optimized or specialized for foveation. Anomalies are more likely to be seen in the instructed saccade which follows an accidental fixation towards the stimulus (Fig. 13, bottom, trials 3, 5 and 7), perhaps because of the much larger position change. The occasional minor anomalies differ from the well-known, frequent and much more severe disturbances of velocity profile seen when the same subjects attempt eye movements towards imagined goals in darkness (Fig. 13, bottom, trials 8, 9 and 10) or in other circumstances where there is no retinal image (e.g. Becker and Fuchs, 1969; Sharpe *et al.*, 1975). As the A-task almost certainly involves a switch of function from one half-brain to the other at some level (Discussion), the occasional anomalies may reflect a more indirect use of retinal information than is normal.

7. A-primary amplitude vs duration is essentially normal

Perturbations and asymmetries in velocity profile will not alter amplitude-duration relationships unless the area under the velocity profile or the duration are altered. The present amplitude-duration data (Figs 5, 7 and 12) are well fitted by a single Yarbus or Robinson relationship, depending on the amplitude range, irrespective of task. For PEH, however, A-durations are found to be significantly longer than N, but only by about 2–3 msec, on testing comparable N and A-task data obtained in the same session at the same velocity trigger levels. Thus the anomalous velocity profiles of PEH's A-primary saccades are only minor anomalies.

8. Secondary latency is usually less than primary latency

This is true in the mean for both N- and A-tasks for subjects H and PEH, but even in those subjects the primary and secondary latency distributions partly overlap (Figs 4, 6 and 8). For EC (Fig. 10, left, lit) matters are more complicated: the primary and secondary distributions for the lit trials still overlap, but the latter is larger in the mean by 36 msec, and would be even larger if the secondary data were not truncated at 350 msec (which is done throughout this paper in order to restrict attention to those secondary saccades which are closely related to the primary). However, close examination of EC's data shows a small peak at short latencies from which the atypical main peak may be derived by the imposition of additional delay. A similar subject to EC is Shebilske's (1976) subject KS—accuracy arguments and the effects of blanking suggest that KS delays for additional post-primary retinal feedback.

9. Secondary latencies may be multimodal

Skewness is seen in Figs 4 (Ho), 6, 8 (N lit, A), 9 and 10. Suggestions of extra peaks occur in Figs 4 (N), 8 (N blanked, A), 10 (N) and 11. Heterogeneity of population is an expectation of Becker's hypothesis (Becker, 1972; Prablanc and Jeannerod, 1975; Becker, 1976) that secondary saccades are of 3 "types": (1) short latency large corrections for large errors, which do not wait for retinal feedback; (2) medium sized corrections for medium errors, which are expedited by retinal feedback, if available, after a medium latency, but are otherwise delayed; (3) small responses to small errors, which are completely dependent on retinal feedback. Heterogeneity is also to be expected for general reasons, e.g. if there is stochastic non-stationarity (drift).

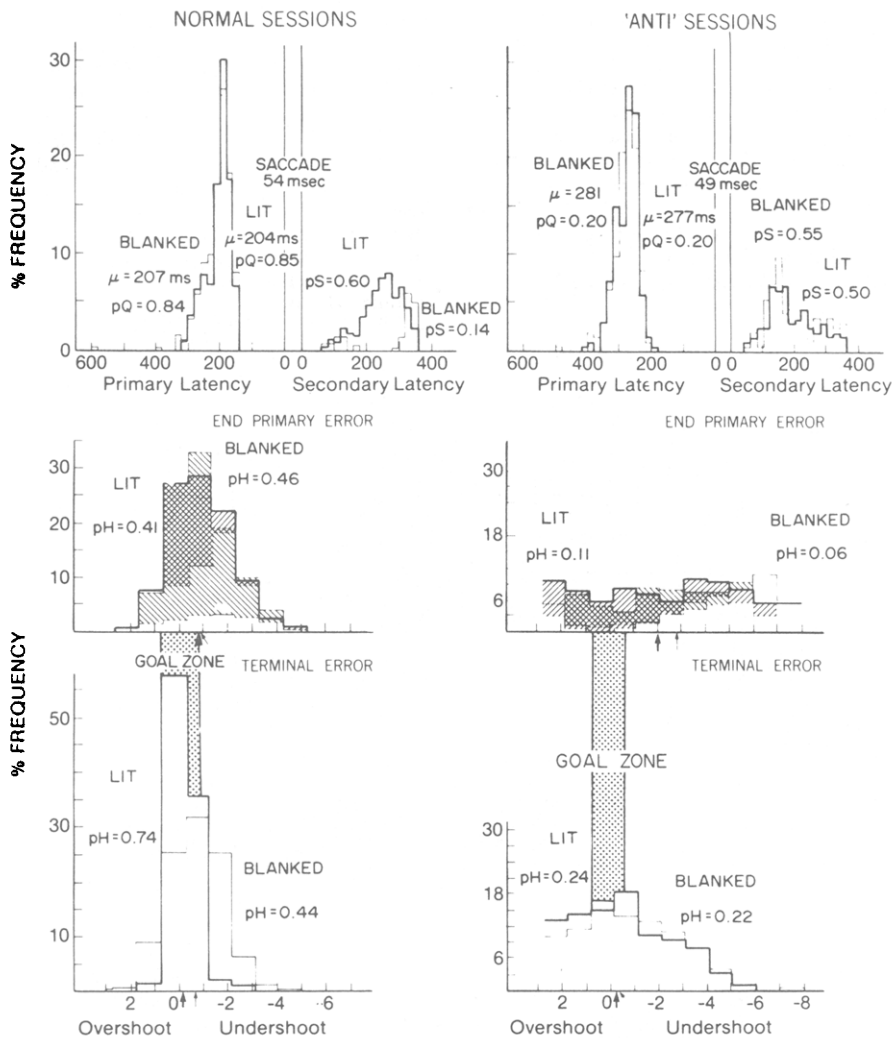


Fig. 10. (Subject EC, feedback.) N- and A-sessions with blanking, begun after about 400 trials at the A-task. About 260 trials for each of the four combinations of instruction and lighting. Top: Latencies as in Fig. 8. Lit histograms in heavy line, blanked histograms in light outline. A-primaries have long latencies. Lit N-secondaries show a minor short latency peak and a major, unusually long, latency peak. Blanking eliminates most N-secondaries, sparing the briefest and increasing the number of long latencies, but does not affect A-secondaries. Middle and bottom: A-angular errors are bad. Angular errors for lit trials in heavy outlines, their means shown by heavy arrows. Histograms for blanked trials in a light line, with means shown by light arrows. End-primary errors are the same, of course, whether lit or blanked, and secondaries reduce these errors to the terminal values, so terminal errors are affected by blanking in the N-task, and are independent of blanking in the A-task. End-primary errors that do not evoke secondaries within 350 msec are shown as 8–2 o'clock hatching for lit trials and 10–4 o'clock hatching for blanked trials. The hatching is greatest in the region of the goal zone, showing that the smaller errors do not tend to evoke secondaries. Correction is lighting sensitive in the N-task. Trigger levels are 12.5°/sec for the N and 20°/sec for the A-task.

10. Blanking profoundly disturbs most N-secondaries

Figures 8 and 10 (top left) show that blanking at the very beginning of primary saccades eliminates many N-secondaries, but spares the very rare N-secondaries of about 100 msec latency, and leads to an increase in the number of long latency secondaries which occur just prior to the 350 msec cut-off for the data. This is consistent with Becker (1976) that "type 3" secondaries, as just defined above, are lost, while "type 2" are much delayed, and "type 1" (which are probably scarce for the present angular range of N-primaries) are unaffected. The reader can be confident that many corrective secondaries have been lost, and not just excluded by the arbitrary data cut, because the ter-

minal errors, which are measured 650 msec after the end of the primary, are worse in the blanked than in the lit trials, and post-primary correction is almost entirely by saccades, not by drift. Of course, retinal feedback for N-secondaries is not simply post-primary saccade. By blanking, or shifting and blanking, the target during the primary saccade, Hallett and Lightstone (1976a, p. 103) showed that intrasaccadic lighting may release or suppress secondaries.

11. Blanking does not affect A-secondaries

See Figs 8 and 10 (right). This is a reasonable expectation on Becker's hypothesis because end-primary errors are

large, and the secondaries are large and so more likely to be "type 1". It is also an expectation of the present hypothesis that saccades are specialized to foveate real objects, because there is no usable error signal relative to the fovea in the lit A-task, e.g. if an A-primary undershoots the goal the retinal error relative to fovea must be increased if the secondary is to correct the aim at the goal. The results are quite contrary to the original working hypothesis that there might be useful feedback of error between the retinal image and the hypothetical peripheral "retinal goal" implicit in the A-instruction.

One may add that the latencies of secondaries to im-

agined targets in complete darkness (136 msec, $\sigma = 75$, for PEH; 187 msec, $\sigma = 71$, for EC) are almost identical in μ and σ to their A-secondary data, and the amplitudes also look similar.

12. The distributions of N- and A-secondary latencies differ

There is a substantial increase in the number of secondaries of about 100 msec or less in the A-task relative to the N-task for subjects PEH and EC (Figs 6, 8, 9, 10 and 11), which is compatible with an increase of "type 1" secondaries due to an increase in the number of large end-primary errors. However, this is not seen for subject H.

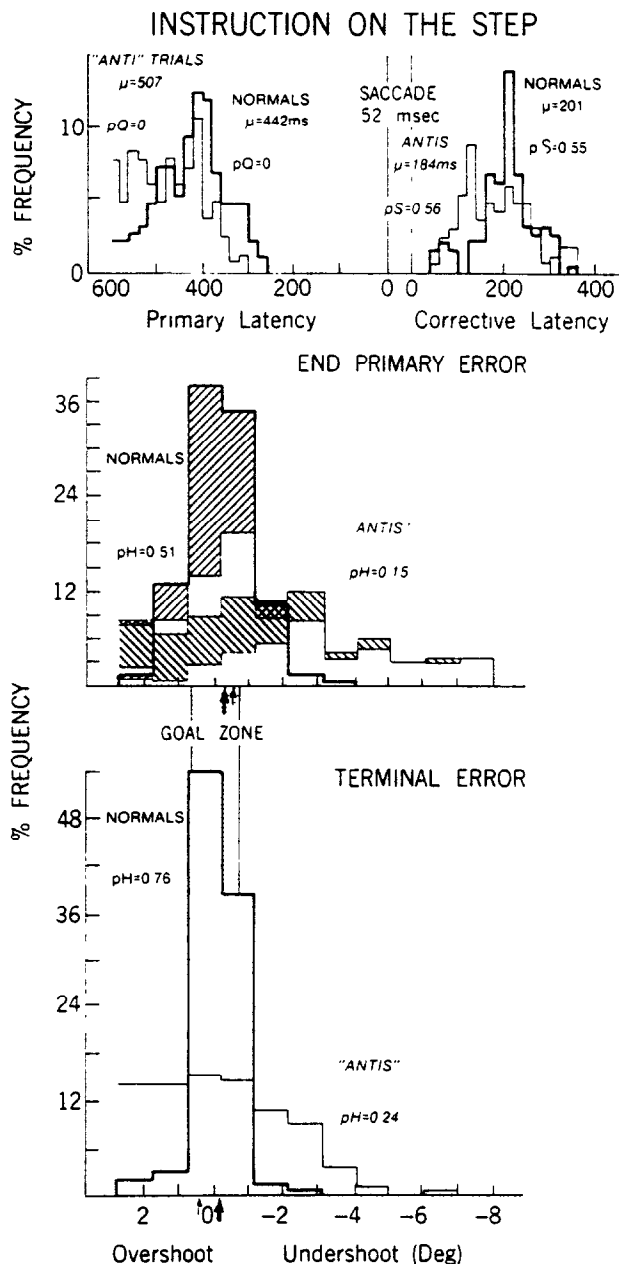


Fig. 11. (Subject EC, feedback.) Instruction-on-the-step experiment begun after about 1300–1400 trials practice at the A-task. About 180 trials for each instruction. Top: Latency plotted as in Fig. 8. A-primaries delayed more than N. A-secondaries show more medium latencies. Middle and bottom: Angular errors plotted as in Fig. 10. A-errors still much worse than N, but practice, or the visual instruction, exerts a small effect, as the end-primary errors now suggest a gentle peak instead of being flat. Secondaries reduce the end-primary errors to the terminal values. The hatched zones of the end-primary error histograms show that those errors which do not elicit secondaries are usually the smaller errors.

Trigger levels 20°/sec for both N- and A-tasks.

although about one-third of her corrected end-primary errors were $>8^\circ$ (off-scale in Fig. 4). PEH also shows an increase in his longer latency A-secondaries, which is not seen for H and EC, but is possibly in keeping with individual differences in the frequencies of "medium" end-primary errors and delayed "type 2" secondaries. In the final analysis, however, the currently available joint distributions of end-primary error and secondary latency are difficult to reconcile with the details of Becker's hypothesis, as given in section 9 above. Part of the difficulty may be that the types may vary quantitatively from subject to subject, or may drift during prolonged data collections, or may not be sharply demarcated from each other. Also, end-primary errors are defined objectively, relative to goals set by the experimenter, and it is by no means clear what the subject's own goals are, and thus very uncertain what end-primary errors are from the viewpoint of his oculomotor system. Possibly the amplitudes of the secondaries reflect this internal error more simply, but this has not been examined. The present results do, however, confirm Becker's main idea, which is that secondary saccades differ substantially in their use of retinal feedback.

13. Large end-primary errors are more likely to cause secondaries

The end-primary error histograms (Figs 4, 6, 8, 9, 10 and 11) are subdivided to show those errors which are followed by secondaries within 350 msec. All sizes of error may evoke secondaries within 350 msec but larger errors are more likely to do so.

14. Post-primary movement is corrective

Figures 4, 6, 8, 9, 10 and 11 show that the distribution of terminal errors is better than the distribution of end-primary errors, for all tasks, no matter what criterion is applied (mean, S.D., pH or distribution). Similarly, Figs 5, 7 and 12 show that the total movement conforms better to the objective goal than the primary movement. Post-primary movement is almost entirely secondary saccades, and drift is trivial. As corrective secondary saccades occur without post-primary retinal feedback in the A-task, the oculomotor system must possess better information about the goal than the end-primary errors would suggest. Subject EC's data are striking in this respect, as his primary in the A-task is not graded to the goal, and so the observed terminal accuracy is entirely due to the secondary saccade. There are no current theories as to the source of primary saccade inaccuracy, but a testable suggestion is that it may reflect the characteristics of fast peripheral retinal acuity channels (see Discussion).

15. Practice does not reduce overall latency and error

The duration of the task period is mainly determined by latency, since the task period ends 650 msec after the end of the primary saccade, and the duration of the primary saccade is fixed by primary amplitude. With respect to terminal error, the overall mean in the A-task is often not much different to the normal value of a few min of arc; more strikingly abnormal is the terminal S.D. or the terminal hit rate pH . Examination of the A-task data for subjects H (4 sessions of about 80 trials each), PEH (6×150) and EC (6×125) shows no trends in primary latency, terminal error or hit rate, or full reward rate. Performance is thus relatively constant, but not fixed in all respects: the striking reduction in direction errors, and the very late tendency of EC to aim his primary saccades in a manner slightly more graded to the goal, have already been mentioned in section 5 above.

16. Laterality effects are small

The histograms of latency and angular errors conceal relatively small left-right or abduction-adduction asymmetries, which vary with task and subject, and do not seem

to support Heywood's (1973) proposal that there may be differences related to hand and eye dominance. The latency asymmetry for the present subjects and experiments is a 10% difference between the fast and slow directions, but this may not apply to other subjects or tasks (subject H is fast to the right, PEH to the left, while EC is fast to the right for N-task and fast to the left for A-task. For dominance see Methods).

17. The A-task is related to the 2-step experiment

Subject-to-subject variability in A-primary latency is considerable. From the figures one obtains values of 385 msec for subject H, 341 and 342 for PEH, and 281 and 277 for EC, to which one may add a value of 237 msec for another subject, EGM (experiments made in collaboration with B. Adams). Is there a normalizing procedure which reduces this variability? The A-task is like a 2-step experiment in which the subject eliminates his "reflex" to the first step and then creates an equal and opposite mental step to which he reflexly responds.

Let us suppose that the A-primary saccade occurs one normal latency after a fictitious equivalent stimulus step; then when does this equivalent step occur? If one calculates $\bar{A} - \bar{N}$ from the figures, where \bar{A} is mean A-primary latency and \bar{N} is mean N-primary latency, one obtains widely ranging values (118 msec for subject H; 92 and 106 for PEH; 73 and 74 for EC; 40 msec for EGM) for the time of equivalent step relative to the initial step. A better normalization is $\bar{A} - 2\bar{N}$, which is much less widely ranging (-149 ; -130 ; -157 ; -133 ; -131 ; -157) and represents the time of the equivalent stimulus step relative to the missing N-primary saccade. The assumption that the A-primary follows the equivalent step by about 1 normal reaction time is not unreasonable, as the response to the second step in 2-step experiments of the pulse-over pattern does so by about 1.1 reaction times (1 ± 0.1 from Lisberger, Fuchs, King and Evinger, 1975, their Fig. 2; 1.15 from Wheelless *et al.*, 1966; 1.05 from Komoda, Festinger, Phillips, Duckman and Young, 1973, their Fig. 4, top; approximately 1, Becker and Jurgens, 1975; 1.1–1.3 from Carlow, Dell'Osso, Troost, Daroff and Birkett, 1975, their Fig. 6; and 1.2 from Hallett and Lightstone, 1976b, p. 122, their P10 and P14 patterns). Now, in such 2-step experiments the probability of a saccade towards the first step is a sigmoid function of \bar{N} minus the inter-step interval (Lisberger *et al.*, 1975, their Fig. 4), which resembles the integral of the N-primary latency histogram, but which has a mean (or 50%) position which is 124–136 msec earlier than \bar{N} . One obvious interpretation of the sigmoid function is that the missing reflex saccade is deleted by retinal information 124–136 msec prior to the point of final motor neuron discharge. There seems, then, to be a strong parallel between the A-task and 2-step experiments of the pulse-over type: in both cases the subject substitutes an oppositely directed saccade for the expected response, and the internal substitution seems to occur approximately 130–150 msec before the missing saccade, roughly 1 normal latency before the observed saccade.

DISCUSSION

Different tasks

It would not be very surprising if the present tasks were to elicit somewhat wildly directed primary saccades but it is surprising that the aim of the primary saccades can be roughly graded to the goal, and that it is definitely refined, on average, by the secondary saccade. This ability to change the sign, gain or offset of the saccadic response to a stimulus step shows a surprising degree of voluntary control, and a high metric sense of what is required (best revealed in mean terminal error), so that one might wish to argue

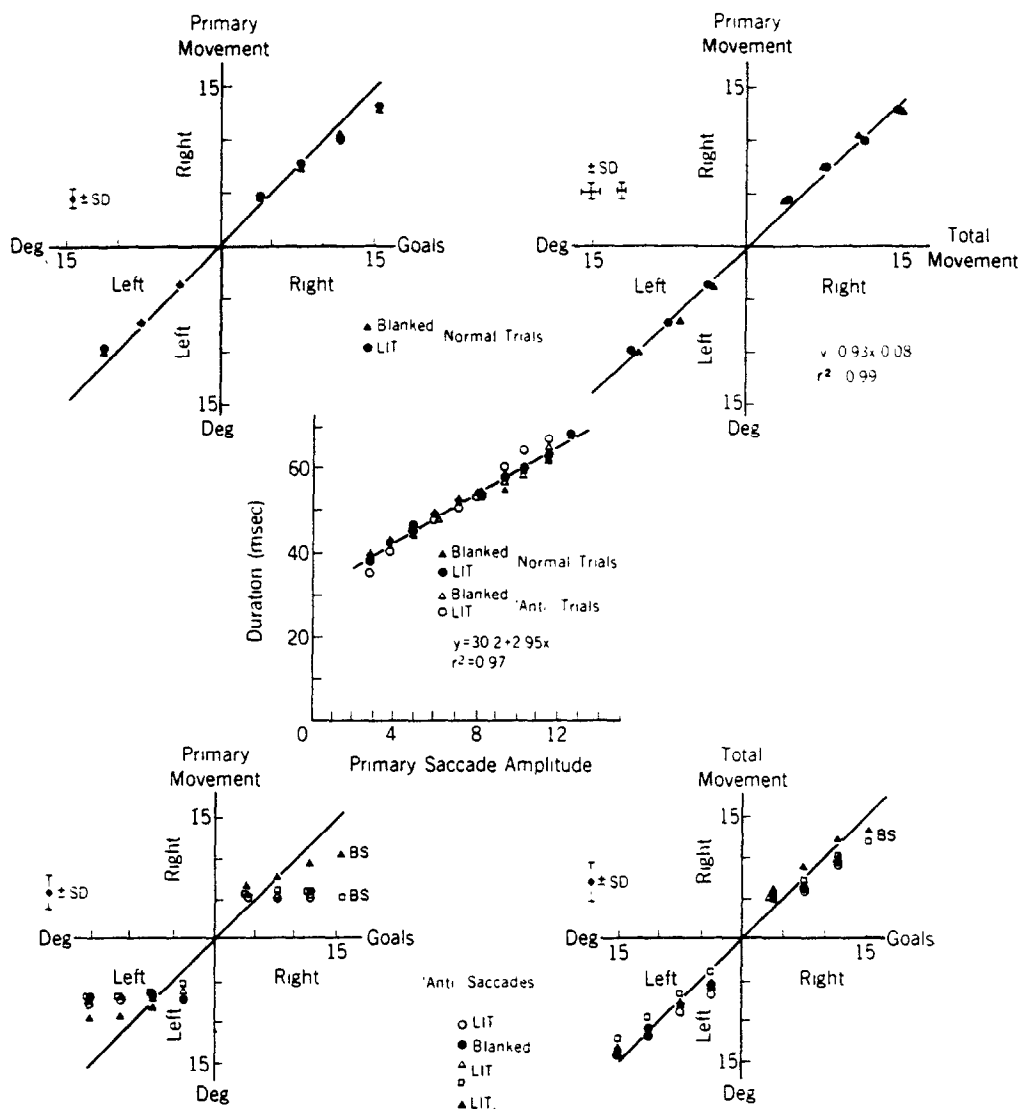
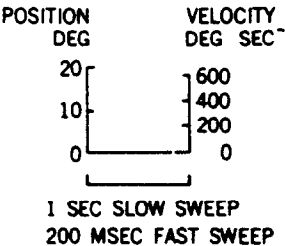
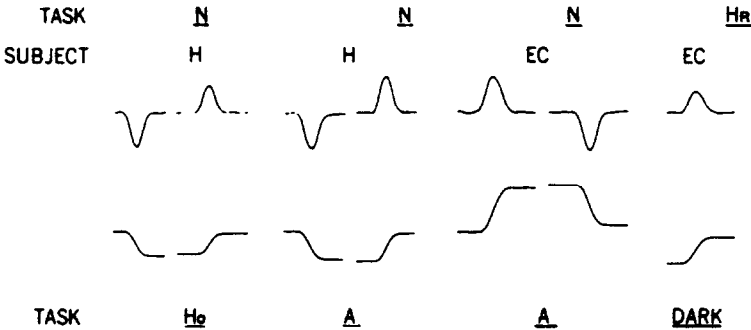
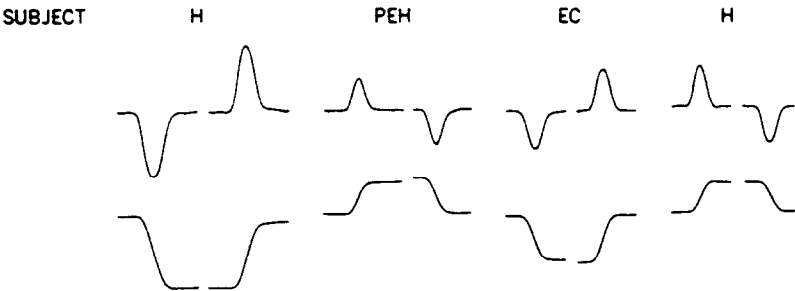


Fig. 12. (Subject EC, feedback.) Data of Figs 10 and 11 replotted. Top left: N-primary amplitude is closely related to goal (reference line is slope 1). Top right: N-primary amplitude is 0.93 of total movement irrespective of lighting. Centre: N- and A-primaries have essentially similar amplitude-duration characteristics, and velocity profiles as well. Bottom left: The various experiments were performed in the order of the symbol list. A-primary amplitude was roughly constant for the first 1300 or so trials, but in the last experiment (\blacktriangle instruction-on-the-step) the primary aim is slightly graded to the goal. Bottom right: As a result of secondaries, mean A-total movement is a fair match to goals in all experiments.

Fig. 13. Velocity and position profiles for primary saccades. All of the records (except 3 and 5) were made at the fast sweep. Inflections or discontinuities marked by short lines. Top: The first seven sets of records show outward and return pairs of saccades for the various tasks. The equivalence of out and return in the N-task justifies the use of the return saccade as a normal control with which instructed saccades can be compared. Normal velocity profiles do not show inflections or discontinuities, and are fairly symmetrical, and usually show sharp peaks. Normal profiles are seen for all subjects and all instructions, and are occasionally seen as responses to imagined goals in darkness. (NB Anomalous or abnormal profiles are never seen in the N-task). Bottom: Subject H shows anomalous profiles in over half of her Hr-trials: in record 1 the outward saccade is prolonged and asymmetric; in record 2 the velocity profile shows inflections marked with short lines. Her A-primaries were always normal, except in her accidental direction errors: the second, or instructed, saccade in record 3 can be compared with the first, or accidental fixation. Record 4 is an A-saccade from early training for PEH, showing velocity and position overshoot. The outward saccade in record 6 is a more typical anomalous A-primary saccade for this subject. Records 5 and 7 are direction errors—the second, or instructed, saccade being prolonged in 5, and with an inflection as well in 7. A fourth subject, EGM, resembles PEH in showing anomalous profiles (mainly discontinuities) in about half of his A-trials. Subject EC does not show any anomalies in the A-task, but most of his saccades to imagined goals in darkness are highly abnormal (records 9 and 10), as are those of PEH (record 8).

NORMAL PROFILES



ABNORMAL PROFILES

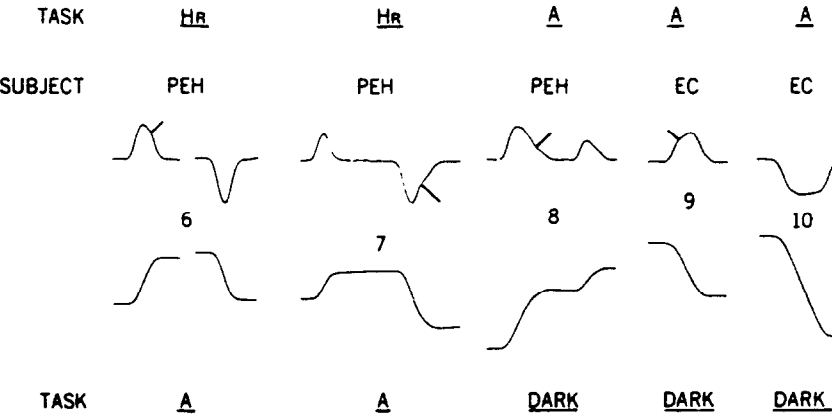
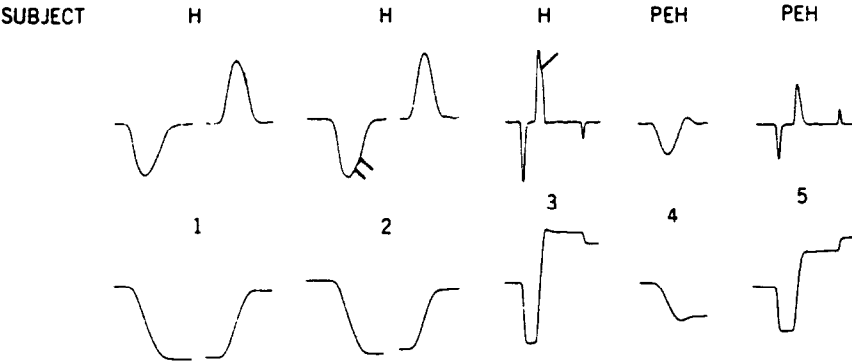


Fig. 13.

that there is only a degree of difference between the N- and A-tasks that should largely disappear with suitable training. The present contention, however, is that the saccadic system is clearly optimized for foveation in the present experimental conditions: instructed goals are achieved with only very poor accuracy (in terminal S.D. or hit rate pH) after long latency, despite rich feedback of performance for hundreds of trials, and most importantly, there is no use of intrasaccadic or post-primary retinal feedback for releasing or aiming the secondary saccades.

Neural paths—a proposal

How are the neural paths actually exercised by the present tasks? If $\Delta E(\text{deg})$ is the total change in eye position caused by saccades in response to a horizontal target step $\Delta T(\text{deg})$, and if m is gain and c is offset, then we can write:

$$\Delta E = m \cdot \Delta T + c$$

where

$m = 1, c = 0$	(N-task)
$m \approx 1, c \approx 3.8^\circ$	(Hr-task)
$m \approx 0.5, c \approx 0$	(Ho-task)
$m \approx -1, c \approx 0$	(A-task).

For all instructions a leftward step of the monocularly viewed fixation point shifts the retinal image into the right hemi-retina of the left eye, and the sensory processing into the right "half-brain". In the N, Hr and Ho tasks, respectively, the motor commands (eye position goals) are presumed to issue from the same (right) "half-brain" and drive the retinal image to the fovea, and so into both half-brains (e.g. Stone, Leicester and Sherman, 1973), into the opposite (left) hemi-retina and half-brain, or leave it in the same (right) hemi-retina and half-brain, as the case may be. In the A-task a motor command from the opposite (left) half-brain drives the retinal image deeper into the same (right) hemi-retina—and a long conduction time through fine unmyelinated visual fibres of the corpus callosum (Swadlow and Waxman, 1976) is one of several possible explanations for the 70–120 msec delay observed.

It is clear from the present results that retinal error is not the sole determinant of eye movements (e.g. review by Robinson, 1973), and there is, in any case, already a considerable body of evidence for the importance of eye position signals and corollary discharge in oculomotor processing (e.g. Becker and Fuchs, 1969; Becker, 1972, 1976; Skavenski and Steinman, 1970; Skavenski, 1971, 1972; Becker and Klein, 1973; Hallett and Lightstone, 1976a,b; Hallett, 1976). Nevertheless, the multiplicity of retinotopic (i.e. binocular retinal hemifield) mapping within the brainstem and cerebrum suggests a possible means whereby "selective attention" may change m and c at hemifield levels. Consider the N-task first. Let us suppose that there is a hemifield representation that projects, not the whole scene, but only the goal detail, into the visual-motor paths, where it combines with eye position information to define a desired future eye pos-

ition. This mechanism is fast, being based on large receptive fields and brief processing delays, and yields the first coarse approximation to the goal, which is realized by the primary saccade. A second, co-activated, but slower system, with small retinal receptive fields, gives rise to a later and better estimate of retinal image position, which combines with current eye position, and with the primary saccade corollary discharge, in order to give a high accuracy secondary estimate of the goal, and of anticipated end-primary error. A secondary saccade is implemented with very short latency, without the use of intrasaccadic or post-primary retinal feedback, if the anticipated error is large (type 1 secondary); or is delayed only so long for expediting by retinal feedback if the anticipated error is small (type 2 secondary); or is discarded entirely in favour of post-primary retinal feedback if the anticipated error is insignificant.¹

Selective attention on this scheme is the ability to create a neural image, or goal detail, in the hypothetical visuo-motor hemifield, which is based on other modalities, or on memories or abstractions. When such a neural image arises *de novo* it triggers saccades in darkness or visually empty fields. Such a neural image does not originate in the retina, and so it is not likely to be sharp and normal on the horizontal retinotopic meridian. Consequently, saccadic horizontal velocity profiles would be expected to be abnormal, as is the case. When the neural image is related to a retinal image by some rule, it triggers saccades to goals defined by instructions. The scaled neural image needs to be ipsilateral to the retinal image, roughly c° more peripheral in the Hr-task, roughly half as peripheral in the Ho-task, and roughly in the mirror position (in the contralateral hemifield representation) in the A-task. These neural images are related to the retinal image, though indirectly, and should therefore be better defined and closer to the horizontal retinotopic meridian than a neural image due to imagination alone. Consequently, the horizontal velocity profiles in the present instructed tasks should show only the observed occasional minor anomalies. The primary aim at goal is degraded in the A-task, because it reflects the errors of the large receptive field mechanism plus the errors of translation into the contralateral retinotopic hemifield. The resulting retinotopic goal detail is realized roughly 130–150 msec before the deleted reflex saccade to the stimulus, about 1 normal latency before the eventual A-primary, and as a consequence A-primary latency is much less than twice normal.² The later, more accurate, retinal information from the presumptive small receptive field mechanism is similarly degraded, by translation into the contralateral hemifield in the A-task, and similarly combines first with current eye position and then with primary saccade corollary discharge, in order to give a more accurate appraisal of the eye position goal, and of anticipated end-primary error. If the anticipated error is large it elicits a secondary saccade independently of retinal feedback, but many medium and small anticipated errors do not elicit secondaries because there is no useful retinal feedback. The most plausible reason for retinal feedback being useless is that the fovea is the sole possible retinal goal, or retinotopic zero point, on which feedback can be based (and it is directed at

¹ See sections 9–12 above for a discussion of the varieties of secondary saccade.

² See section 17 for a discussion of timing.

empty space). Other possibilities are that post-primary retinal feedback is delayed too long by transformation or scaling operations, or that it conveys no useful position information, as in the A-task where the retinal image is peripheralized by the primary saccade into even lower acuity regions.

Certain aspects of this proposal must be considered conjectural. For example, there is evidence for low and high acuity mechanisms co-existing in the visual periphery (subject PEH in Hallett, 1961), and there is a current interest in low and high acuity channels which differ by 70–100 msec in their latency (e.g. review of Breitmeyer and Ganz, 1976). But it is obviously speculative or even unnecessary to associate primary and secondary saccades with such perceptual mechanisms; in any case, a perceptual unit size of 0.5° at 7° eccentricity from the fovea would require an additional source of position noise, or convergence from the perceptual to the oculomotor level, in order to account for end-primary errors with $\sigma = 1^\circ$ in the N-task. Also, Becker's (1976) varieties of secondary saccade may not be sharp cut in latency and amplitude characteristics, and the process of scaling by m and c could, perhaps, occur at motor rather than retinotopic levels. (However, common experience suggests that human versatility is primarily perceptual rather than motor.) Nevertheless, the present proposal is worth considering because it is largely testable, and because it is useful to show how saccades according to instructions may require only minimal high level, or cognitive, intervention in the operation of a more or less specialized, low level, oculomotor mechanism.

Acknowledgements—This work was supported by Grant MT 4092 of the Medical Research Council of Canada. I am indebted to H. M. E. Hallett and especially to E. Conway (medical student) for considerable assistance. The experiments were made possible by electronic equipment built by F. G. Oakham to supplement that due to A. D. Lightstone. I am grateful for comments by W. Becker (Ulm) and B. Adams.

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