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THE MECHANISM OF PREDICTION IN HUMAN SMOOTH PURSUIT EYE MOVEMENTS

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SUMMARY

1. Experiments have been conducted on human subjects to determine the role of prediction in smooth eye movement control. Subjects were required to actively pursue a small target or stare passively at a larger display as it moved in the horizontal plane.

2. Target motion was basically periodic, but, after a random number of cycles an unexpected change was made in the amplitude, direction or frequency of target motion. Initially, the periodic stimulus took the form of a square waveform. In subsequent experiments, a triangular or sawtooth waveform was used, but in order to examine the timing of the response in relation to stimulus appearance, the target was tachistoscopically illuminated for 40–320 ms at the time that it passed through the mid-line position.

3. When subjects either actively pursued the target or stared passively at the larger display a characteristic pattern of steady-state eye movement was evoked composed of two phases, an initial build-up of eye velocity that reached a peak after 200 ms, followed by a decay phase with a time constant of 0·5–2 s. The build-up phase was initiated prior to target displacement for square-wave motion and before onset of target illumination for other waveforms.

4. The peak eye velocity evoked gradually increased over the first two to four cycles of repeated stimulation. Simultaneously, the response became more phase advanced, the reaction time between stimulus onset and the time at which peak velocity occurred decreasing from an average of 300 to 200 ms for triangular waveform stimuli.

5. When there was a sudden and unexpected change in amplitude and direction of the stimulus waveform, the eye movement induced had a peak velocity and direction that was inappropriate for the current visual stimulus, but which was highly correlated with the features of the preceding sequence in the stimulus.

6. When there was a sudden change in the frequency of the stimulus waveform the predictive eye movement was induced with a timing appropriate to the periodicity of the previous sequence but inappropriate to the new sequence.

7. The results indicate that prediction is carried out through the storage of information about both the magnitude and timing of eye velocity. The trajectory of the averaged eye velocity response was similar in form irrespective of the duration

of target exposure or basic stimulus frequency, suggesting that the predictive estimate is released as a stereotyped volley of constant duration but varying magnitude under the control of a periodicity estimator.

8. It is probable that the stored eye velocity estimate is derived from the sampling of an efference copy of eye velocity and is dependent on the fidelity of retinal velocity error input.

9. The normal mode of operation of the pursuit reflex appears to be one in which continuous visual feedback is enhanced by predictive estimates of eye velocity initiated under the control of the periodicity estimator and only corrected if retinal error conflict indicates the predictive estimate to be inappropriate.

INTRODUCTION

It has long been realized that the pursuit reflex in humans exhibits properties of prediction. Dallos & Jones (1963), for example, recognized that the performance of the pursuit response was much better than might be expected on the basis of a simple retinal velocity error feedback mechanism, given the limitations in the system such as the large processing delays. Their conclusions were largely based on the observation that the response to a regular sinusoidal oscillation of the target generally exhibited a very high proportion of smooth eye movement whereas in that evoked by a random target motion, eye velocity no longer matched target velocity so well and the presence of catch-up saccades led to the appearance of broken pursuit. On the basis of these findings Dallos and Jones suggested that the improvement in gain and phase might be carried out by a predictive operator consisting of a short-term store under the control of a periodicity estimator. More recently this concept has been further elaborated by Bahill & McDonald (1983), who have suggested that the predictive mechanism is used to reconstruct the required trajectory of a regular waveform such as a sinusoid from a menu of learned waveforms.

In early experiments the pursuit response was considered as a whole, not split into its smooth and saccadic components. From the work of Rashbass (1961) and numerous later studies it has become apparent that the fast and slow mechanisms are essentially independent. The break-down in the pursuit response to random target motion is caused by an inability to match eye velocity to target velocity, even though overall eye displacement tends to match target displacement very closely because of the foveating characteristics of the saccadic system.

In our own more recent experiments, in which the pursuit response to mixed-frequency pseudo-random stimuli has been investigated (Barnes, Donnelly & Eason, 1987; Barnes & Ruddock, 1989) it has been possible to show that the break-down in pursuit is not actually associated with the randomness of the stimulus, but with the frequency of its highest frequency component. When this frequency exceeds approximately 0·4 Hz, pursuit becomes broken, even when the stimulus is composed of as few as two sinusoids. One of the notable features of the results of these experiments was that although the lower frequency components of the response exhibited a reduction in eye velocity gain, the highest frequency component itself tended to have an enhanced gain comparable to that of a sinusoidal response. This led to the suggestion (Barnes & Ruddock, 1989) that the predictive mechanism might operate selectively on the highest frequency component by a predictive velocity estimator under the control of a periodicity estimator in a manner similar to that suggested by Bahill & McDonald (1983). In an attempt to isolate the predictive mechanism of pursuit we have now examined two particular features of smooth pursuit frequently associated with prediction, the ability to produce anticipatory eye movements and the continued pursuit of a target when it has disappeared from view.

Although the presence of prediction may be inferred from conventional pursuit responses, it is well established that most individuals are unable to initiate anticipatory smooth eye movements in the absence of a visual target (von Noorden & Mackensen 1962; Heywood, 1972). One way of demonstrating the effects of prediction is to examine the response to square-wave target motion (Kowler & Steinman, 1979; Barnes *et al.* 1987). Such stimuli are found to elicit not only the expected saccadic eye movements, but also smooth eye movements that precede actual target displacement. However, the eye velocities generated in this type of response are rarely greater than 4–5 deg/s and it is difficult to see how they might be used for prediction of targets moving at higher velocities. In order to demonstrate whether a similar predictive mechanism might operate in response to a continuous target motion we have developed a new technique for examining the oculomotor response in which a target moving at velocities up to 45 deg/s is presented for brief periods at regular intervals. This has allowed the timing of the response with respect to target appearance to be examined in detail, thus clearly revealing evidence of prediction similar to that evoked by square-wave target motion, but with much higher velocity.

The continuation of eye movements that occurs when the pursued target suddenly disappears from view has been examined by a number of investigators (von Noorden & Mackensen, 1962; Eckmiller & Mackeben, 1978; Mitrani & Dimitrov, 1978; Whittaker & Eaholtz, 1982; Becker & Fuchs, 1985; Barnes *et al.* 1987). Although the results demonstrate the persistence of the smooth eye movement response for as much as 3–4 s, there has been little quantitative assessment of the velocity and frequency characteristics of these movements which would unequivocally demonstrate that they are the result of prediction and do not simply represent the transient decay of the oculomotor system. In order to assess these aspects in detail we have examined the oculomotor response that is present when, following a number of cycles of similar stimuli, there is a sudden change in the frequency, direction or amplitude of target motion. Such a change has been found to evoke a predictive eye movement with a peak velocity as high as 40 deg/s in a manner inappropriate to the current visual stimulus velocity with a timing that is determined by the periodicity of the previous part of the stimulus. A preliminary report of this work has been published previously (Asselman & Barnes, 1989*a*).

METHODS

Subjects were seated in a totally darkened room with head fixed at the centre of a screen of radius 2 m on to which a moving visual display was projected by a mirror galvanometer. The illumination of the moving display was controlled by an electromechanical shutter that could produce pulse durations down to 8 ms. Two types of display were used; a target comprising a circle of radius 25 min of arc with fine cross-hairs superimposed on it and a larger background display subtending 12 deg in width by 8 deg in height composed of horizontal stripes (spatial frequency 2 cycles/deg). The motion of the target was controlled by a computer-generated waveform that took various forms as described later. Eye movements were transduced by an infra-red limbus tracking technique with a resolution of 5–10 min of arc (Skalar Iris). The eye movement recorders were rigidly coupled to the head by a dental bite and helmet assembly.

In order to examine the predictive component of the smooth eye movement response a novel target waveform was devised in which sudden and unexpected changes were introduced into an otherwise regular periodic motion. The stimulus was split into five consecutive sequences of randomized and unequal duration that contained between six and twelve cycles of a periodic waveform. At the transition between sequences the waveform was suddenly changed in amplitude, direction or frequency. In some trials the stimulus was blanked out at the transition for two complete cycles of the new sequence and eye movements were recorded in the absence of any visual stimulus. In other trials, there was no blanking period at the transition so that the conflict between intended eye movement and target movement was immediately apparent to the subject. In experiment I the periodic stimulus was a square-waveform target motion, the target being either continuously illuminated or tachistoscopically illuminated for 20 ms immediately after peak step displacement. In the remaining experiments the periodic stimulus was a triangular or sawtooth waveform, the target or display being illuminated for varying pulse durations at the time when the target passed through the mid-line position. In all of these experiments, the subject was required either actively to pursue the small fixation target or to view passively the larger background display. In the passive condition, subjects were required either to attempt to hold fixation in a central position and ignore the motion of the display or to make small saccadic eye movements in the opposite direction to display motion. In practice it was not actually possible to actively pursue any particular point in the display because the stripes were orientated parallel to the direction of motion. Details of the individual conditions and the waveforms used are explained in more detail in the Results section.

The results reported here were all obtained on the same group of four normal subjects, one of whom required refractive correction in order to see the target clearly. However, most test procedures were also carried out on a larger group of subjects in order to verify the generality of the effects described. All subjects participated with informed consent and the experimental procedure was approved by the local ethics committee.

Eye movements were analysed using an interactive computer graphics technique similar to that described previously (Barnes, 1982). After removal of the fast-phase components of the eye movements the slow-phase eye velocity was compared with target velocity. Cycle-by-cycle averages of slow-phase eye velocity were obtained by overlaying and averaging successive cycles of the response (excluding the first two cycles) during each sequence of constant frequency periodic stimulation. Time constants of transient decay were obtained by the fitting of an exponential function using an iterative, least squares error curve-fitting procedure.

RESULTS

Responses to changes of amplitude in square wave stimuli (experiment I)

In the initial experiments, either the small target or the display of horizontal stripes made square-wave displacements in the horizontal plane about the mid-line position at frequencies of 0·52–1·30 Hz. Maximum amplitude of the step motion was varied in equal increments from ±1·6 to 8·0 deg, changing at random in successive sequences of target motion. There was no blanking period between sequences.

A varying pattern of eye movement was evoked dependent on the instructions to the subject and on the frequency of the waveform (Fig. 1). If the subject was asked to actively follow the motion of the target, regular saccadic eye movements were made with a small proportion of smooth eye movement tending to precede each saccadic component as noted previously (Kowler & Steinman, 1979; Barnes *et al.* 1987). If the subject simply stared at the centre of the larger background display a pattern of nystagmic eye movements was elicited in which the slow and fast movements were frequently of opposite polarity, similar to a conventional optokinetic response. If the subject was instructed to make random saccades around the inner part of the display a hybrid pattern could be seen. In all these responses, however, the velocity of the smooth (slow-phase) component of eye movement was very similar and did not obviously change with the actual position of the display on the retina as indicated in the example shown in Fig. 1, in which eye position changed considerably over the duration of presentation. It would thus appear that this response was elicited not by positional error as referred to the fovea, but by the *change* of position information presented. The smooth eye movement responses induced during passive stimulation were considerably easier to analyse because of the absence of very large amplitude saccadic components and in the discussion that follows, only these responses will be described.

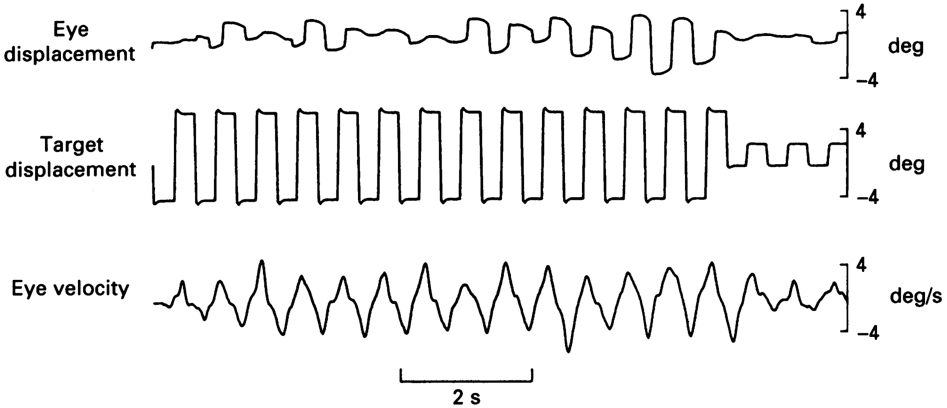

Fig. 1. Examples of eye movements evoked by square-wave target displacement at a frequency of 1·3 Hz. The subject was instructed to make small saccadic eye movements at random around the inner part of a large moving display (12 × 8 deg) and not to actively follow display motion. Fast-phase components have been removed from the eye velocity trace.

One of the most notable features of both the active and passive responses was that eye velocity generally took some two to four cycles of stimulation to build up to an asymptotic level as indicated by the changes in root mean square (r.m.s.) eye velocity during each half-cycle of the stimulus (Fig. 2*B*). There was normally no measurable smooth eye movement during the first half-cycle of stimulation.

After the initial build-up of the response there was a very characteristic pattern of slow-phase eye velocity which was revealed by carrying out a cycle-by-cycle average excluding the first two cycles (Fig. 2*A*). There were essentially two phases of the response. First, there was a build-up of eye velocity that was initiated prior to the onset of the target stimulus and rose to a peak that terminated 0–150 ms (mean 87 ms) after the actual target step displacement. Second, there was a decay of eye velocity towards zero which frequently reversed in direction after a period of 1–2 s. When a low-frequency stimulus was used these two components could be seen to be clearly separated and the reversal of eye velocity became more evident. Mean eye velocity was normally no more than 4–6 deg/s in response to square-wave stimulation, although one subject exhibited mean velocities as high as 10 deg/s. Peak velocity increased with the amplitude of target displacement and also varied with frequency, the average levels for the largest target displacement (8 deg) being 5·8, 9·0, 10·1 and 5·9 deg/s at frequencies of 0·52 , 0·65 , 0·86 and 1·30 Hz respectively.

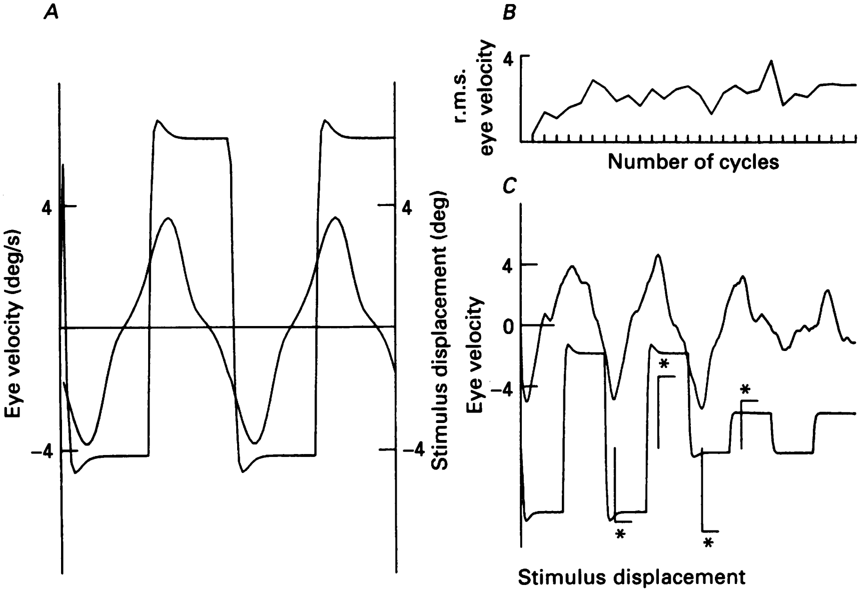

Fig. 2. *A*, cycle-by-cycle average of the smooth eye velocity (repeated for clarity) shown in Fig. 1, indicating the manner in which eye velocity builds up in the direction of target motion prior to actual displacement. Stimulus frequency, 1·3 Hz. *B*, changes in root mean square (r.m.s.) eye velocity as a function of the number of cycles of stimulation. C, smooth eye velocity at the transition when there was an unexpected change in target displacement. Bar lines marked by asterisks in lower trace indicate the timing and magnitude of peak eye velocity in relation to target displacement.

At the transition from one sequence to the next, when there was an unexpected change in target amplitude (Fig. 2*C*), subjects continued to produce a peak eye velocity which was more appropriate to that of the preceding sequence than that for the current target displacement. The effect is demonstrated by the high level of correlation and appropriate, positive slope in the relationship between peak eye velocity at the transition and the mean peak velocity for the prior sequence (Fig. 3). In contrast, the relationship to the mean peak velocity of the ensuing sequence exhibited a poorer correlation and inappropriate slope in all subjects. This clear demonstration of the predictive nature of the eye velocity pulse was evident in both active and passive responses.

Changes of amplitude/direction in triangular waveform stimuli (experiment II)

In the second experiment the target or background display was made to move with a triangular waveform in the horizontal plane, but it was exposed for only a brief duration each time that it passed through the mid-line position. Pulse duration (PD) was varied from 40–320 ms, whilst the frequency of the basic triangular waveform

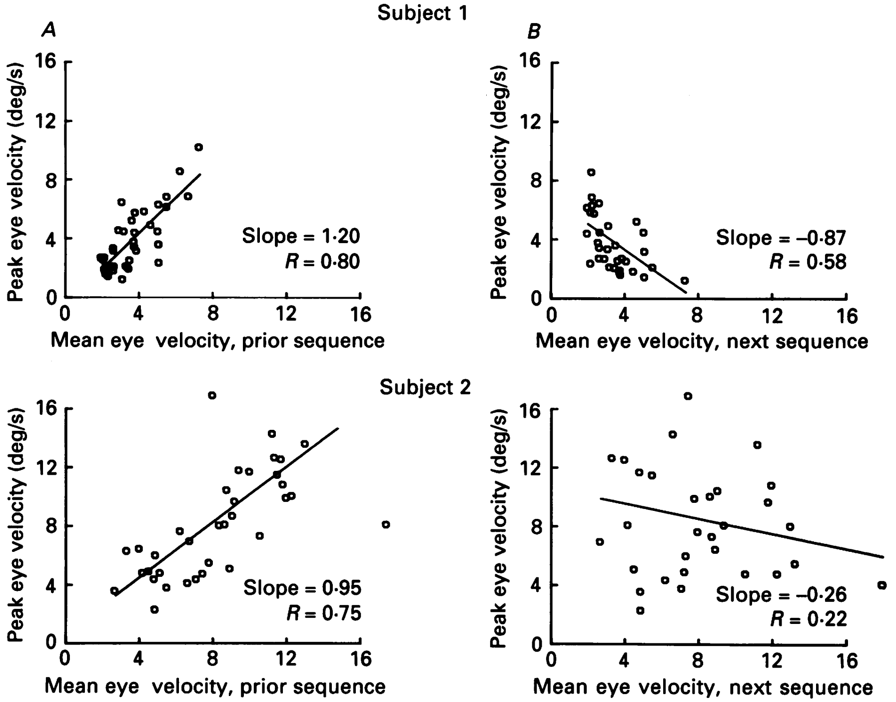

Fig. 3. The correlation in two subjects between peak eye velocity at the transition and mean peak velocity for the previous sequence of stimulation (*A*) or mean peak velocity for the ensuing sequence (*B*).

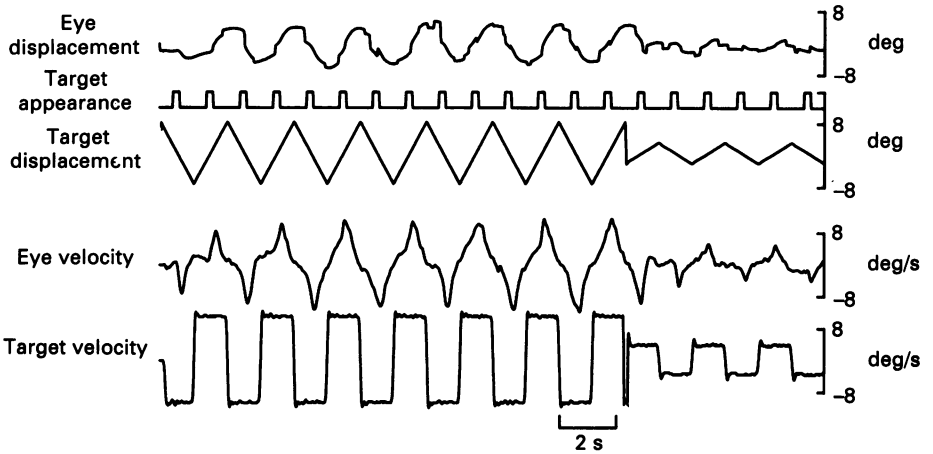

Fig. 4. Examples of eye movements induced by the tachistoscopic presentation of a target moving at constant velocity as it passed through the centre of the visual field. Pulses (trace 2) indicate the timing of target appearance which was for a duration of 240 ms. Stimulus triangular waveform at a frequency of 0·69 Hz, peak velocity ± 10 deg/s. Fast-phase components have been removed from the eye velocity trace.

was held constant at 0·69 Hz. At the transition between sequences, which were of differing, randomly assigned velocity between 9 and 45 deg/s, both the velocity and direction of target motion were unexpectedly changed. Passive responses were examined with a blanking period between sequences; active responses were examined

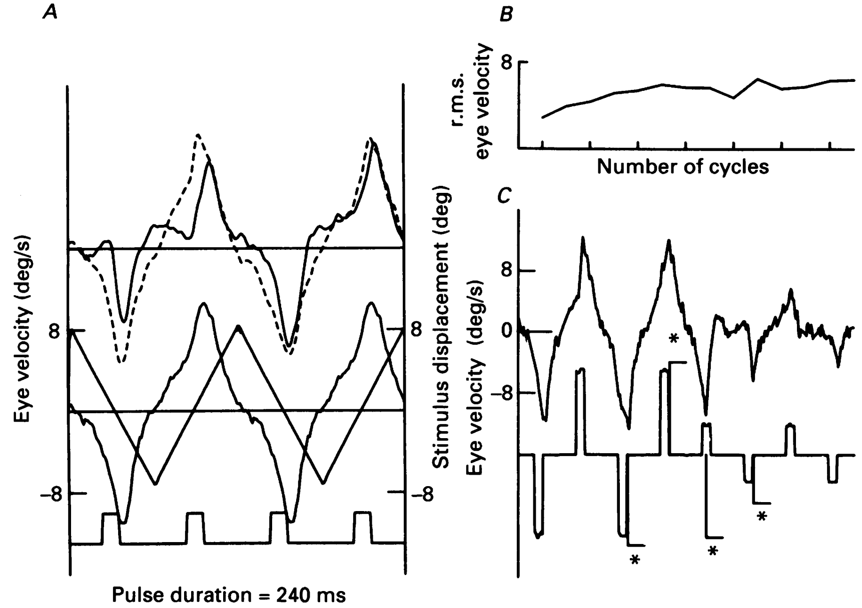

Fig. 5. Centre traces in *A*, cycle-by-cycle averages of eye velocity (repeated) and target displacement derived from the responses in Fig. 4, indicating the build-up of eye velocity prior to target appearance. Stimulus frequency 0·69 Hz. Upper traces in *A*, the first two cycles (continuous line) and second two cycles (dashed line) of eye velocity plotted to the same time scale as the averaged responses so as to indicate the manner in which the magnitude and timing of the eye velocity response changes with repeated stimulation. Lowest trace in *A*, pulses indicating timing of target exposure. *B*, changes in r.m.s. eye velocity with number of cycles of stimulation. *C*, changes in eye velocity at the transition when there was an unexpected change in both velocity and direction of the target motion. Bar lines in lower trace marked by asterisks indicate the magnitude and timing of peak velocity points in relation to target velocity pulses.

both with and without blanking. Typical eye movements induced during active pursuit of the target when there was no blanking are shown in Fig. 4, from which four important features may be noted.

Firstly, in all subjects, regardless of the duration of exposure, eye velocity gradually built up to reach an asymptotic level after about two cycles of stimulation in the same manner as for the square-wave responses. The effect could be demonstrated by changes in both the peak velocity and the r.m.s. eye velocity for each half-cycle (Fig. 5*B*). The detail of the eye velocity trajectory during the first two pairs of stimulus cycles is shown in Fig. 5*A* (upper traces). As might be expected, peak eye velocity to the first presentation was never negligible and much higher velocities were achieved than for square-wave stimuli. The increase in peak velocity during the first four presentations was dependent on both target velocity and PD.

When pulse duration was 40 ms there was no significant effect of target velocity, but average peak eye velocity increased from 7·5 to 10·3 deg/s between the first and fourth presentations. When pulse duration was 160 ms mean peak velocity increased in a progressive and significant manner ($P < 0{\cdot}01$ by ANOVA) from 11·1 to 13·3 deg/s at a target velocity of 9 deg/s and from 12·8 to 35·7 deg/s for a target velocity of 45 deg/s (Fig. 6*B*). There was a strong tendency to overestimate the required velocity at the third presentation. Associated with the changes in eye velocity there were also modifications in the timing of the responses with repeated exposure which were best observed when the target was blanked between sequences. At the beginning of each sequence of the stimulus the eye movements were initiated, on average, 110 ms after stimulus onset, and reached a peak after a mean reaction time of 310 ms (Fig. 6*A*). With repeated exposure, eye velocity became gradually more anticipatory so that it passed through zero velocity before target exposure and mean reaction time from onset of target illumination to peak velocity was reduced to 210 ms after four exposures (Fig. 6*A*).

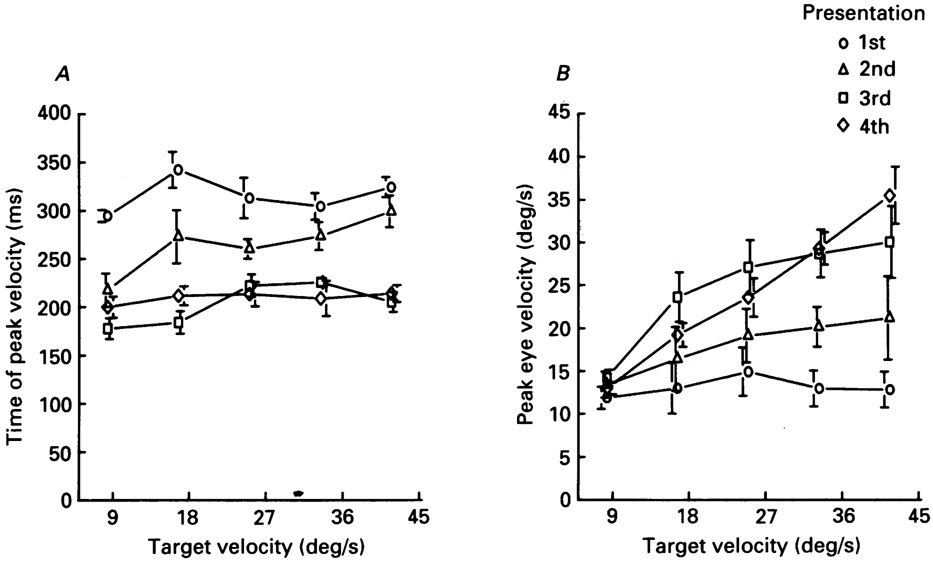

Fig. 6. Changes in the timing (*A*) and the magnitude (*B*) of peak eye velocity during the first four presentations of the moving target as a function of the velocity of target motion. The time of peak velocity is a reaction time referred to onset of target exposure. Results shown are for a pulse duration (PD) of 120 ms during *active* pursuit and are representative of all values of PD tested. Mean of four subjects ± 1 S.E.M. (shown as a vertical bar).

Secondly, the cycle-by-cycle average of eye velocity (Fig. 5*A*) exhibited a trajectory which was similar to that for the square-wave responses (Fig. 2*A*). Peak velocity increased significantly with the duration of target exposure from 40–320 ms. It also increased with target velocity, although a saturation effect was evident for exposures less than 160 ms (Fig. 7). When the pulse duration was only 40 ms peak velocity remained essentially constant at a mean of 9·7 deg/s. The initial build-up of eye velocity started well before the onset of target exposure, with a mean reaction time of −150 ms, and reached approximately 32% of its peak value at the time of target exposure. Eye velocity measured 100 ms after the onset of target exposure (i.e. at the time when visual feedback might be expected to become effective) reached, on average, 66% of peak velocity. The velocity achieved at these times was considerably greater than obtained previously for the square-wave responses. For example, when the pulse duration was 240 ms and target velocity was 45 deg/s mean peak eye velocity was 41 deg/s, the velocity at target onset was 12 deg/s and the velocity 100 ms after exposure was 26 deg/s. The reaction time between target onset and peak velocity varied very little with exposure duration, with a mean of 215 ms. As a consequence, peak velocity was frequently achieved before the target disappeared when exposure duration was more than 240 ms, though not in the example shown in Fig. 5*A*. After the attainment of peak response, eye velocity decayed towards zero with a measured time constant that varied from 0·5–2 s. Differences in time constant showed no consistent change with exposure duration, velocity or any other variables investigated.

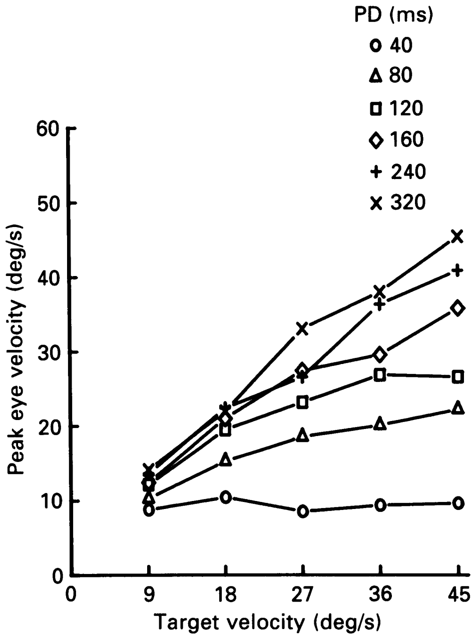

Fig. 7. The relationship between average peak eye velocity and target velocity as a function of the PD of target exposure from 40–320 ms. Mean of four subjects, S.E.M. (omitted for clarity) ranged from 1·4–3·8 deg/s.

Thirdly, when the target was made to change direction unexpectedly at the transition from one sequence to the next and the target was not blanked out, eye velocity was always inappropriate in both amplitude and direction as indicated in Fig. 5*C*. Peak eye velocity at the transition was positively correlated with the mean peak velocity of the previous sequence of the response but negatively correlated with the mean peak velocity for the ensuing sequence (Fig. 8). As soon as the conflict between target motion and this predictive eye movement became apparent eye movement was rapidly terminated with an average latency of 116 ms. Consequently when target exposure was increased beyond 120 ms there was no further significant increase in peak eye velocity, because visual feedback was clearly being used to terminate the incorrect predictive estimate. When the visual conflict was avoided by blanking the display at the transition, peak eye velocity rose to higher levels for longer pulse durations.

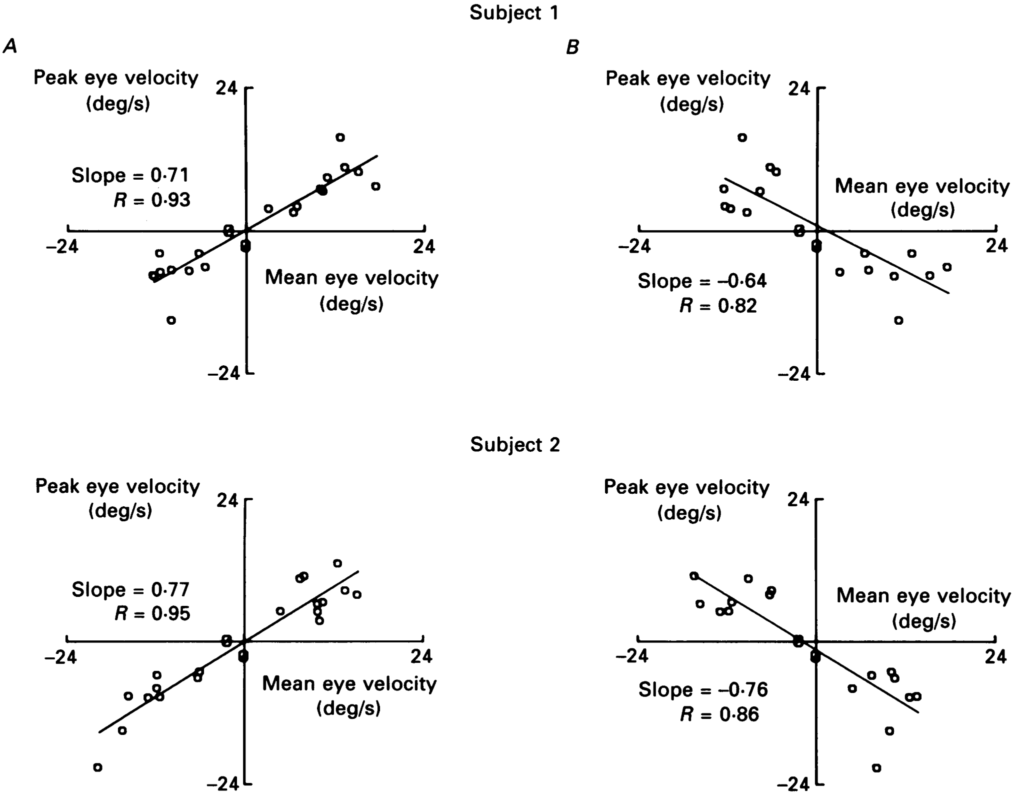

Fig. 8. Correlation in two subjects of peak eye velocity at the transition (Fig. 5*C*) with mean peak eye velocity for the previous (*A*) and ensuing (*B*) sequences. PD = 120 ms.

Fourthly, the eye movements induced during passive stimulation, which were all examined with a blanking period between sequences, had very similar velocity characteristics to those for active pursuit, even though the pattern of eye displacement was similar to that of optokinetic nystagmus, with the fast phases acting predominantly in the opposite direction to slow-phase components. At the start of each new sequence there was a steady rise in peak eye velocity over the first four presentations as shown in Fig. 9*A* and the changes in timing of the response were very similar to those for active pursuit (Fig. 6*A*). The averaged eye velocity profiles were indistinguishable from those induced by active pursuit. In fact, the only measurable difference between active and passive responses, which is evident by comparison of Figs 6*B* and 9*A*, was that the peak velocities generated during active pursuit were, on average, 30% higher than those for passive stimulation, a difference which was somewhat less than that recorded in previous comparisons of active and passive pursuit (Barnes & Hill, 1984; Barnes & Crombie, 1985; Pola & Wyatt, 1985). At the transition between sequences, predictive but inappropriate velocity estimates were generated in the same way as for active pursuit. Figure 9*B* shows a comparison, for a pulse duration of 240 ms, between peak eye velocity before and after the transition and three measures derived from the averaged eye velocity profile. It is evident that there is a very close correspondence between peak eye velocity at the transition and the averaged eye velocity 100 ms after the onset of target exposure. Exactly similar results were also obtained during active pursuit when a blanking period was left between sequences.

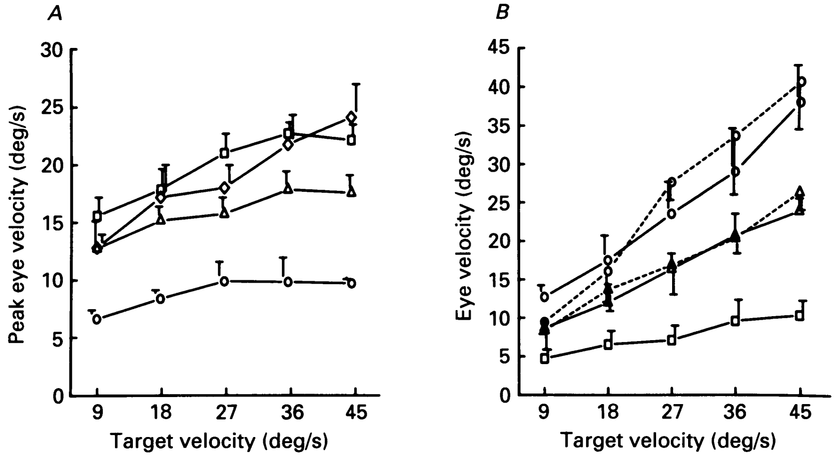

Fig. 9. *A*, changes in peak velocity during the first four presentations of the moving target as a function of the velocity of target motion. Symbols as Fig. 6. Results shown are for a PD of 120 ms during *passive* stimulation and are representative of all values of PD tested. Mean of four subjects ± 1 S.E.M.. *B*, continuous lines represent three measures of eye velocity derived from the cycle-by-cycle average (Fig. 5*A*) and plotted as a function of target velocity: velocity at target onset (□), velocity 100 ms after target onset (△) and peak velocity (○). The dashed lines represent the values of peak eye velocity prior to (○) and at the time of (△) the transition in amplitude and direction of the target motion as a function of target velocity. Target exposure (PD) = 240 ms. Mean of four subjects ± 1 S.E.M. Note the similarity between velocity at the transition and 100 ms after target exposure.

Changes of frequency in triangular waveform stimuli (*experiment III*)

Similar evidence of prediction during active and passive stimulation was also obtained when the frequency of the target motion was changed unexpectedly as indicated in Fig. 10. In this experiment, the pulse duration was held constant at 240 ms and the frequency of the triangular waveform was varied from 0·32 to 0·96 Hz. Four velocity levels, 10, 20, 30 and 40 deg/s were examined in each of four subjects. The effects of prediction were particularly evident at the transition between sequences when the period between target presentations was increased, the eye reaching a peak velocity prior to the time at which the target actually appeared (Fig. 10*A*). This effect was quantified by measurement of the latency of peak eye velocity with respect to the appearance of the target. A negative latency indicated attainment of peak velocity prior to target appearance and as indicated in Fig. 10*B*, this negative latency was observed to increase when there was an increase in the difference between the half-periods of consecutive sequences, the slope determined by linear regression having a value of $-1{\cdot}01$. In effect this demonstrates that the peak response had occurred at a time appropriate for the periodicity of the previous half-cycle showing that both the timing and the amplitude of the response were generated on a predictive basis. In contrast, when the frequency of the target motion was increased the appearance of the target prior to the expected time resulted in a rapid correction of the predictive response. Consequently, peak velocity for this category of response occurred with a positive latency that was essentially constant (mean 315 ms) regardless of the difference in half-period between sequences.

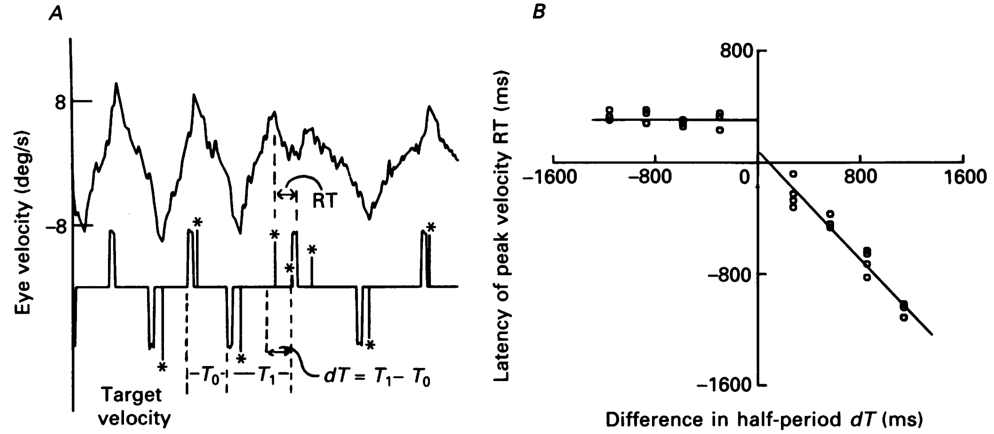

Fig. 10. *A*, eye velocity at the transition when there was a sudden decrease in stimulus frequency, indicating the manner in which the predictive eye velocity trajectory continues to be made at a time prior to actual target appearance. Asterisks in lower trace indicate the magnitude and timing of peak eye velocity in relation to target velocity pulses. *B*, the reaction time (RT) between attainment of peak eye velocity and the appearance of the target plotted as a function of the difference in half-period (dT) between successive sequences. The derivation of RT and dT is shown in *A*.

Examination of the cycle-by-cycle average of eye velocity (Fig. 10*A*) revealed a similar profile to that shown in Fig. 5*A*, except that there were significant changes with the frequency of stimulation. At the lowest frequency the separation of the two components of the response, the predictive build-up and the transient decay, could be clearly distinguished. Overlaying the responses from different stimulus frequencies (Fig. 11) revealed that the predictive component and the initial part of the transient decay exhibited a very similar time course irrespective of stimulus frequency. However, at low frequencies, decay of the previous transient component was largely complete before initiation of the next predictive, reversed response, whereas at higher frequencies there was interaction between the responses to successive stimuli. An estimate of the trajectory of a single predictive pulse was derived by assuming there to be negligible interaction between successive predictive pulses in the response to the lowest frequency stimulus, a process which has been fully justified by investigation of the response to even lower frequency (0·125 Hz) stimuli (Asselman & Barnes, 1989*b*). The fully developed trajectory is illustrated in Fig. 11*A*, in which the response to each of the four stimulus velocity levels has been normalized by dividing through by peak eye velocity. Overlaying these normalized responses reveals the remarkable similarity in the time course irrespective of target velocity. Note, in particular, the timing of the initial response onset in relation to the pulse representing target exposure and the timing of the response decay, which normally passed through zero velocity after approximately 0·5–0·7 s and was followed by a period of low-amplitude, reversed velocity. When this derived trajectory was overlaid with opposing polarity at decreasing intervals in order to simulate the

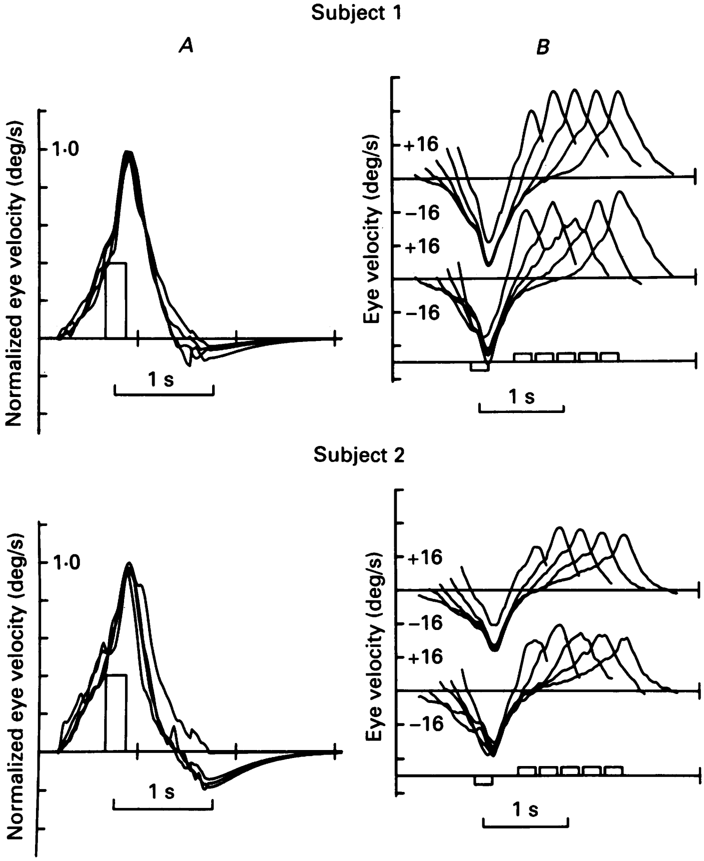

Fig. 11. *A*, the estimated velocity trajectory of a single predictive pulse derived from one half-cycle of the response to the lowest frequency stimulus. Responses for the two subjects have been normalized by dividing through by peak eye velocity in order to compare the overlaid response to four stimulus velocities (8, 16, 24 and 32 deg/s). Centre trace in *B*, cycle-by-cycle averages of eye velocity obtained for different frequencies of stimulation from 0·32 to 0·96 Hz as indicated. Traces are time-locked to the onset of the target in the first half-cycle at each frequency. Top traces in *B*, simulated responses to different stimulus frequencies derived from the linear summation of the estimated predictive pulse shown in *A* with periodicity and polarity appropriate to the stimulus that is shown in the bottom trace in *B*. Results of two subjects shown separately.

effects of higher frequency stimulation (Fig. 11*B*, upper traces) there was a notable similarity to the observed responses (Fig. 11*B*, lower traces) in the timing of the peaks and zero velocity cross-overs. At frequencies up to 0·65 Hz the low magnitude of the reversal phase had little summative effect on the ensuing peak response and, as a consequence, average peak eye velocity remained constant (Fig. 12*A*). However, at the highest frequency (0·96 Hz), the initial phase of the transient decay was not complete before the initiation of the next predictive response, leading to a reduction of peak velocity (Figs 11*B* and 12*A*). At the highest frequency the overall velocity profile had a quasi-sinusoidal form, in which the predictive and transient decay components could no longer be clearly distinguished. In contrast to peak velocity, r.m.s. velocity decreased with decreasing frequency (Fig. 12*B*) because the same temporal profile was averaged over an increasing period of time, the time constant of transient decay, as noted above, remaining essentially unchanged by stimulus frequency.

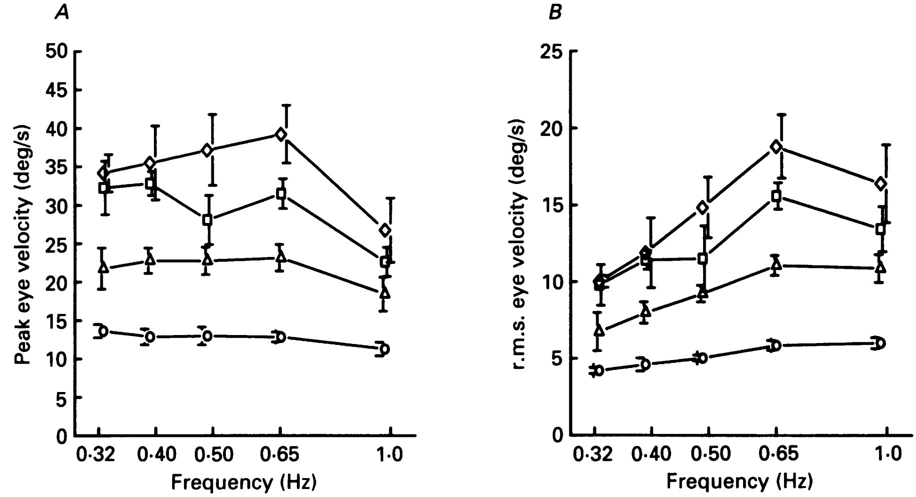

Fig. 12. Peak (*A*), and r.m.s. (*B*), eye velocity as a function of the frequency and velocity (*v*) of target motion. PD = 240 ms. Values for *v* were: 10, ○; 20, △; 30, □ and 40, ◇, deg/s. Mean of four subjects ± 1 S.E.M.

Responses to sawtooth waveform stimuli (*experiment IV*)

In the final experiment we attempted to determine whether the predictive effects demonstrated for direction-changing stimuli could also be observed when target motion was unidirectional. The underlying stimulus was a sawtooth waveform that varied in velocity from 10 to 40 deg/s and in frequency from 0·32 to 0·96 Hz. In order to facilitate comparison with the previous experiment one cycle was unconventionally defined as including two ramp components so that target exposure actually occurred at the same intervals as in the previous experiment. The target was exposed for 240 ms as it passed through the mid-line position.

The cycle-by-cycle average of eye velocity (Fig. 13*A*) associated with each target presentation exhibited a similar pattern to that seen previously for direction-changing stimuli, although there was considerably more variability in the response. The eye velocity trajectory was initiated prior to target onset, reached a peak, on average, 240 ms after target onset and subsequently decayed towards zero with a mean time constant of 0·7 s. When there was a sudden and unexpected change in the frequency of target motion, a predictive eye velocity was normally evoked (Fig. 13*C*) with a timing that was clearly based on the periodicity of the previous half-cycle as demonstrated previously (Fig. 10*B*). However, peak velocity at the transition attained with this stimulus was frequently lower than that obtained when the target reversed direction at each exposure and the predictive component was sometimes suppressed altogether.

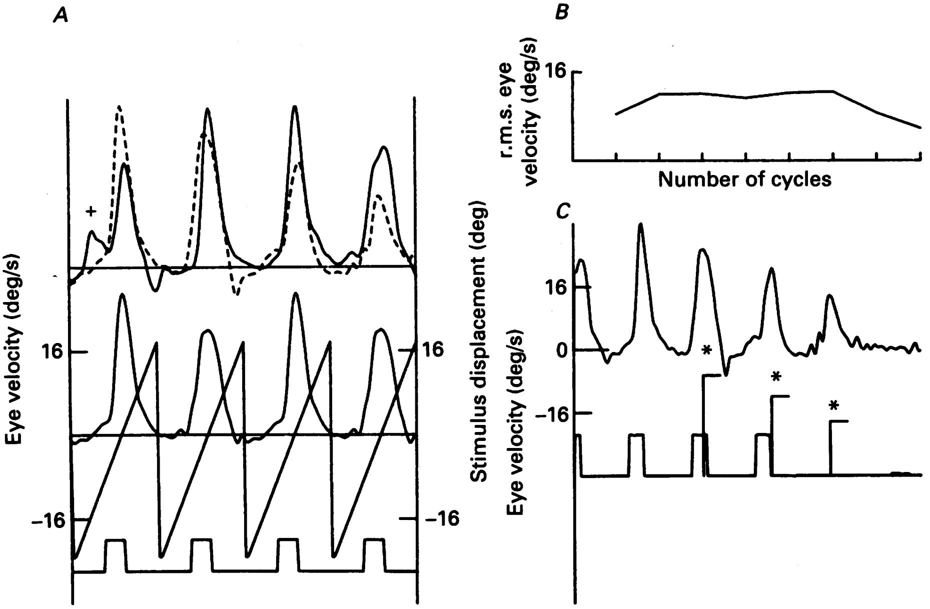

Fig. 13. Centre trace in *A*, cycle-by-cycle average of eye velocity (repeated) in response to brief (240 ms) exposure of a target moving with a constant velocity sawtooth waveform. One cycle unconventionally defined as two ramp components for comparison with Fig. 5. Upper traces in *A*, detail of first two cycles (continuous line) and second two cycles (dashed line) of response. Peak marked by + is a predictive response to previous stimulus of higher frequency. *B*, r.m.s. eye velocity as a function of the number of stimulus cycles. *C*, the continuation of the eye velocity response when the target was blanked out at the termination of the sequence. Pulses indicate time of target exposure. Note the occurrence of the final eye velocity pulse in the absence of a visual stimulus at a time appropriate to the periodicity of the preceding stimulus.

DISCUSSION

The evidence for prediction in the pursuit response

The results of the experiments presented here provide a positive demonstration of the activity of the mechanism of prediction in smooth eye movement control and allow the following five conclusions to be made about the manner in which this effect is achieved.

Firstly, it is evident that repeated exposure to a moving target leads to a modification of both the magnitude and timing of the eye velocity trajectory in relation to the visual stimulus. Initially, the response occurs with a latency of approximately 100 ms after the onset of target exposure in the manner demonstrated previously (Carl & Gellman, 1987). However, after three or four exposures the smooth eye movement has increased in peak velocity by a factor of 1·5–2 whilst simultaneously becoming more phase advanced with respect to the stimulus. This type of response almost certainly underlies the changes in gain and phase that are seen within the first half-cycle of the response to a sinusoidal stimulus (Dallos & Jones, 1963; Bahill & McDonald, 1983). The tachistoscopic presentation technique allows the effect of prediction to be directly observed rather than being inferred from changes in phase in the response to a continuous periodic stimulus. The changes in the timing of the response with repeated stimulation are quite incompatible with the behaviour of a linear feedback system. It is evident, especially from the lowest frequency responses shown in Fig. 11 for example, that the build-up of eye velocity prior to target illumination could not be attributed to the persistence of the transient response to the previous stimulus. Although the initial transient response often exhibited some reversal of eye velocity (e.g. in the response to the first presentation in Fig. 4*A*), which might be expected to make some contribution towards the build-up of velocity in the opposite direction, the magnitude of reversed velocity was rarely more than 20 % of the initial peak velocity. In contrast, during repetitive stimulation, 50–70 % of peak velocity was achieved within 100 ms of the onset of target exposure, that is, at the time when visual feedback might be expected to become effective. This indicates that the enhancement of the response with repeated stimulation must be carried out, not through direct summation, but through the charging of an underlying internal storage mechanism. The model presented in Fig. 14, which has been developed from that proposed previously (Barnes & Ruddock, 1989), illustrates one way in which such a system might be realized through the temporary storage of an efference copy of eye velocity. This model is similar in concept to those proposed by a number of authors (e.g. Robinson, 1982; Bahill & McDonald, 1983), but with important differences which will be discussed later.

The second important manifestation of the action of the predictive mechanism lies in the response observed when there is a sudden and unexpected change in the nature of the stimulus. A number of authors have demonstrated that there is a continuation of the response following target disappearance (von Noorden & Mackensen, 1962; Eckmiller & Mackeben, 1978; Mitrani & Dimitrov, 1978; Whittaker & Eaholtz, 1982; Becker & Fuchs, 1985; Barnes *et al.* 1987), but there has been no clear demonstration that such a response represents the result of true prediction rather than a simple transient decay process. The results of the experiments presented here demonstrate clearly that when there is an unexpected change in the target waveform, the eye movement continues to be made even when it is completely inappropriate to the current visual input and is only corrected when the conflict has been registered. Most importantly, both the amplitude and timing of peak eye velocity at the transition from one sequence to the next were found to be more highly correlated with the velocity and periodicity of the previous sequence than with the characteristics of the current visual stimulus. However, it is important to note that the transient decay pattern itself did not appear to be modified, only the timing of its occurrence, so that the persistence of the response cannot be considered to be part of the predictive mechanism.

Thirdly, the behaviour at the transition is exactly what would be expected from the averaged responses shown in Fig. 5. Eye velocity at the transition reaches a peak that is similar in magnitude to that achieved in the averaged response 100 ms after the onset of target exposure (Fig. 9*B*), i.e. before visual feedback has had time to become effective. These results demonstrate that the eye velocity information stored in memory must be released prior to the expected time of target onset under the control of a periodicity estimator. The periodicity is presumably assessed on the basis of landmarks in the stimulus waveform, such as changes in direction. Previous

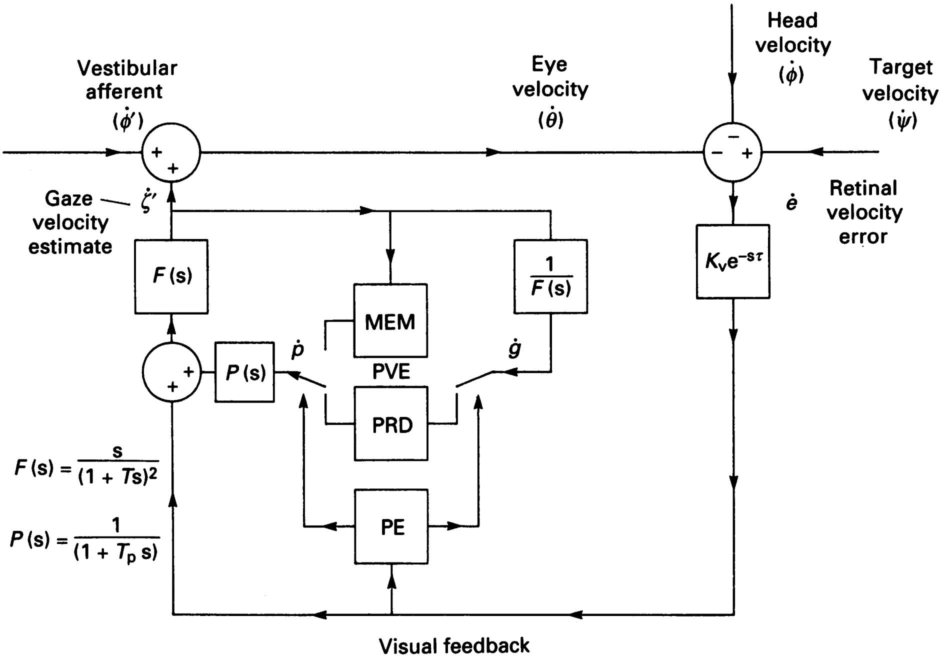

Fig. 14. A model of the pursuit eye movement control mechanisms which are necessary to explain the reported experimental findings. The visual feedback is composed of two basic components, a direct feedback of retinal velocity error and a secondary pathway incorporating a predictive velocity estimator (PVE). The PVE is a sample and hold mechanism that takes its input from an efference copy of gaze velocity ($\dot{\zeta}'$ = eye velocity if head velocity = 0). Output from the PVE is controlled by a periodicity estimator (PE) that derives its control from the retinal velocity error signal and thereby estimates the periodicity of the most frequently changing component of the stimulus. The PVE may obtain estimates of required eye velocity either from direct sampling of efference copy, through the predictor (PRD), or from information stored in memory (MEM) based on the previous half-cycle of the response, depending on stimulus frequency. The output from PVE is partially integrated by the filter $P(s)$, which has a time constant T_p of approximately 0·15 s, and summated with the retinal velocity error signal before passing through a band-pass filter $F(s)$ with a time constant T of approximately 0·5–1 s. $F(s)$ and $P(s)$ have dynamic characteristics as defined in the diagram, where s is the Laplace operator. K_v = visual feedback gain. τ = delay in visual feedback = 0·1 s.

work (Barnes *et al.* 1987) has indicated that periodicity is probably based simply on the duration of the previous half-cycle of stimulation. This effect is represented in the model (Fig. 14) by the periodicity estimator, which senses the appropriate frequency on the basis of retinal velocity error and releases the output of the store.

Fourthly, one of the objectives of the experiments presented here was to demonstrate unequivocally that predictive eye movements of reasonable velocity could be achieved. The results show that peak eye velocities as high as 40 deg/s may be attained when pulse duration is as long as 320 ms whereas previous experiments had not demonstrated eye movements of more than 4–5 deg/s initiated in the absence of a target (Becker & Fuchs, 1985; Barnes *et al.* 1987). However, in order for the velocity to reach this level the internal store must be suitably charged by previous visual input. The results shown in Fig. 7 indicate that the higher the velocity level of the stimulus, the longer the exposure that is required to fully charge the store.

Finally, it is evident from these experiments that one important role for visual feedback is to check the validity of the velocity estimate in the predictive process. If there is a conflict between the estimate and the current visual input the estimation system is shut down. The pursuit system then falls back on the use of conventional visual feedback in order to build up a new estimate of velocity. In doing so the reaction time to peak response is increased to 300 ms for the initial response but becomes reduced to 200 ms after two or three presentations. The required information for the assessment of conflict is, of course, contained within the retinal velocity error signal, so that visual feedback is vital to the process. As a consequence, if the visual feedback is degraded, for example by location of targets only in the peripheral retina, the velocity estimate built up in the store will also be degraded proportionately, so that both predictive and non-predictive pursuit will be impaired (Barnes & Hill, 1984). If the visual input is absent then presumably the storage mechanism cannot be charged and eye velocity will never rise above the baseline level of 4–5 deg/s that can be achieved in the dark. Conversely, if the visual image is stabilized on the retina (Heywood & Churcher, 1971; Kommerell & Taumer, 1972), the velocity estimate is never incorrect and the subject can produce any desired velocity drive to the eye without producing conflict. A similar argument may explain the ability to track a stroboscopically illuminated stationary target (Heywood, 1973; Behrens & Grusser, 1979). Once the eye has started in motion, that motion can be continued if the positional error is not too great because the brief exposure of the target would not allow velocity error to be perceived and the estimate of eye velocity would thus be confirmed.

Mode of operation of pursuit reflex

The model of the pursuit reflex shown in Fig. 14 has been simulated by computer and found to give reliable predictions of the behaviour of the pursuit reflex. The mode of operation may be summarized as follows. Motion of a target initially creates a retinal velocity error that drives the oculomotor system in a conventional feedback manner. However, this initial retinal velocity error signal is also stored in the form of an efference copy of eye velocity within the memory of the predictive velocity estimator and may be added to the visual feedback signal to enhance system performance. This concept is essentially identical to that originally proposed by Yasui & Young (1975) and subsequently developed by others (Young, 1977; Lisberger & Fuchs, 1978*a*, *b*; Robinson, 1982; Bahill & McDonald, 1983), except that there seems little possibility that this is a continuous positive feedback as originally suggested. The results of our experiments show clearly that the information stored in the memory can be held for some time and released at the appropriate moment under the influence of the periodicity estimator. This implies that the information held in memory must have been sampled at a previous time, since there was no continuous visual input present in the experiments described here. The predictive velocity estimator may thus be regarded as a sample and hold module, a concept similar to that proposed previously by Young & Stark (1963). The similarity of the eye velocity trajectories obtained with differing exposure durations and repetition frequencies (Fig. 11) indicates that the volley of eye velocity drive that is released by the predictive velocity estimator is probably a stereotyped pulse of activity of fixed duration but variable magnitude. The dynamics of the visual feedback pathways, which appear to be those of a bandpass filter (F(s) in Fig. 14) as suggested previously (Barnes & Ruddock, 1989) then shape this pulse to give the observed eye velocity trajectory.

How could such a system operate to boost performance during pursuit of a pseudo-random target motion, giving rise to the changes in gain observed when the frequency content of the stimulus is varied (Barnes *et al.* 1987)? The pursuit of low-frequency (i.e. $< 0{\cdot}4$ Hz) target motion could be achieved very effectively by the continuous storage and release of eye velocity estimates with a regular sampling period (Δt) that is somewhere in the region of 250 ms. This function would be carried out by the predictor (PRD in Fig. 14), which, in its simplest form, could derive its predictive velocity estimate ($\dot{p}$) on the basis of extrapolation from the current and previous samples of the efference copy signal ($\dot{g}$): that is $\dot{p}(t) = \beta[2\,\dot{g}(t)+\dot{g}(t-\Delta t)] \approx \beta\,\dot{g}(t+\Delta t)$, where β is the gain of the predictor pathway. Simulation has shown that if β is in the region of 0·9 pursuit of either a single sinusoid or a mixed sinusoid (Fig. 15*A*) can take place with eye velocity gain greater than 0·9 and negligible phase error at frequencies up to 0·3 Hz. The sampling of efference copy results in a very low-amplitude oscillation at 3–4 Hz, a feature that is frequently evident in recorded pursuit eye movements (Robinson, Gordon & Gordon, 1986).

This mode of operation could clearly no longer function when any frequency component of the stimulus exceeded a certain level (approximately 0·4 Hz) because it would not be possible to generate a reliable estimate of eye velocity within the sampling period as shown in the initial half-cycle in Fig. 15*B* and *C*. When there is a higher frequency component present in the stimulus the best attempt that such a system can make in achieving an optimum response is to determine the periodicity of the stimulus and provide estimates from memory (MEM in Fig. 14) of the required velocity at the appropriate times based on previous response cycles. For those frequencies in the important range between 0·4 and 2 Hz the result of such activity produces a response which appears quasi-sinusoidal in form (Fig. 11) and without detailed examination could easily be mistaken for a sinusoidal response. Examination of averaged eye velocity trajectories evoked by conventional sinusoidal target motion at frequencies of 1–2 Hz do indeed reveal a non-linear form similar to that evoked by the stimuli used here. Bahill & McDonald (1983), who derived a very similar model, have claimed that the system is capable of storing a fairly detailed representation of a complete periodic waveform. However, the simulations shown in Fig. 15*B* and *C* indicate that the storage of as few as four samples derived from the previous half-cycle of the efference copy of eye velocity are quite sufficient to generate a very realistic smooth eye movement response at frequencies down to 0·5 Hz. The timing of the release of this output from the memory (MEM) is critical to the control and varies as a function of the frequency determined by the periodicity estimator.

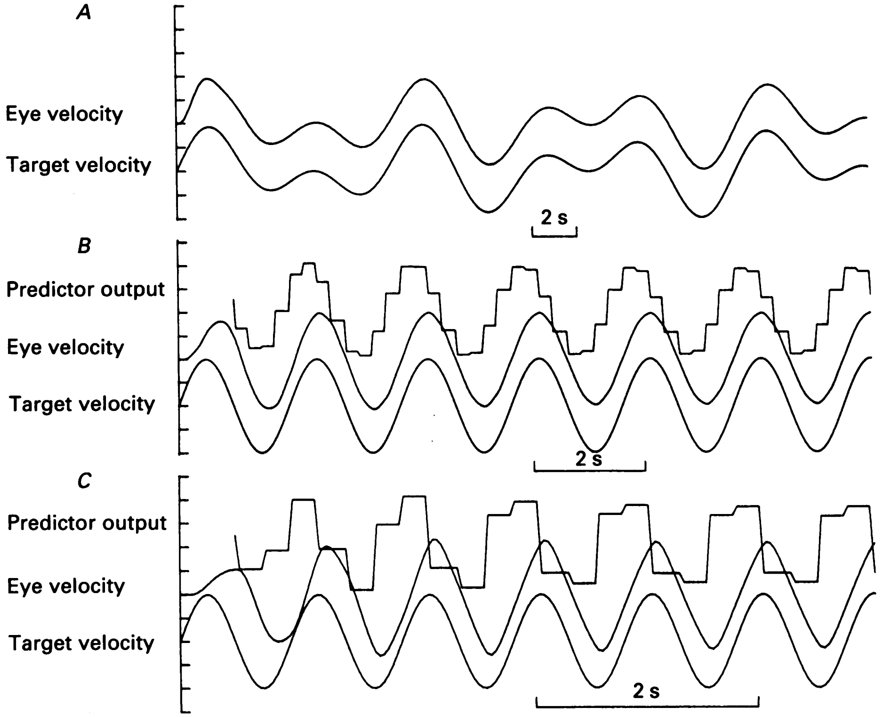

Fig. 15. Simulated slow-phase eye velocity traces derived from the model of Fig. 14. *A* shows the response to a mixed sinusoid (frequency 0·11+0·19 Hz) using the continuous predictor (PRD) which samples the efference copy signal ($\dot{g}$) every 250 ms and outputs a predictive drive signal ($\dot{p}$) derived from the current and previous samples. *B* and *C* show the responses to single sinusoids of frequency 0·5 and 1 Hz, respectively. Continuous prediction has been invoked for the first half-cycle; thereafter the predictive estimate is taken from the memory system (MEM) which derives its output from samples of the previous half-cycle of the efference copy signal. The output of MEM is shown in the uppermost traces. Values of the parameters defined in Fig. 14 are: $T = 0{\cdot}5$ s; $\tau = 0{\cdot}1$ s; $T_p = 0{\cdot}15$ s; $K_v = 1$; β (gain of PRD) $= 0{\cdot}9$.

The neurophysiological basis of the predictive mechanism

The site of the predictive mechanism in pursuit is essentially unknown at present. However, a number of recordings from the flocculus of the cerebellum (Miles & Fuller, 1975; Lisberger & Fuchs, 1977, 1978*a*; Noda, 1986; Noda & Warabi, 1986,

1987) give some indication that it is likely to be closely associated with this mechanism. Signals representing retinal slip velocity, eye velocity (or rather, gaze velocity if there is head movement) and target velocity, variables that are important to the function of the model, have all been observed in primates. Of particular interest is a study by Noda (1986) of the mossy fibre inputs to the flocculus. The so-called visuomotor units, which appear to give an estimate of target velocity, exhibited three properties that would be expected from the output of the predictive velocity estimator: (1) the response exhibited considerable phase advance (equivalent to a lead time of approximately 125 ms) with respect to target velocity; (2) at low-stimulus frequencies the neural excitation was not maintained so that two or three peaks appeared during each cycle at 0·2 Hz, which might be indicative of a sampling process in operation; (3) when there was a sudden decrease in stimulus frequency the effect of the previous frequency sometimes persisted in a manner akin to the responses shown in Fig. 10. The existence of a second area within the vermis of lobules VI and VII with properties similar to those of the flocculus has also been described (Kase, Noda, Suzuki & Miller, 1979). This area is also important in pursuit since some function remains if it is spared and only total cerebellectomy completely abolishes the pursuit response (Westheimer & Blair, 1973). Whether the flocculus and vermis serve different functions in pursuit has yet to be determined (Keller, 1988).

REFERENCES

Asselman, P. T. & Barnes, G. R. (1989*a*). The contribution of predictive mechanisms to smooth eye movement control in humans. *Journal of Physiology* **420**, 17*P*.

Asselman, P. T. & Barnes, G. R. (1989*b*). The effects of intermittent exposure of a moving target on smooth pursuit in humans. *Journal of Physiology* **423**, 69*P*.

Bahill, A. T. & McDonald, J. D. (1983). Model emulates human smooth pursuit system producing zero-latency target tracking. *Biological Cybernetics* **48**, 213–222.

Barnes, G. R. (1982). A procedure for the analysis of nystagmus and other eye movements. *Aviation Space and Environmental Medicine* **53**, 676–682.

Barnes, G. R. & Crombie, J. W. (1985). The interaction of conflicting retinal motion stimuli in oculomotor control. *Experimental Brain Research* **59**, 548–558.

Barnes, G. R., Donnelly, S. F. & Eason, R. D. (1987). Predictive velocity estimation in the pursuit reflex response to pseudo-random and step displacement stimuli in man. *Journal of Physiology* **389**, 111–136.

Barnes, G. R. & Hill, T. (1984). The influence of display characteristics on active pursuit and passively induced eye movements. *Experimental Brain Research* **56**, 438–447.

Barnes, G. R. & Ruddock, C. J. S. (1989). Factors affecting the predictability of pseudo-random motion stimuli in the pursuit reflex of man. *Journal of Physiology* **408**, 137–165.

Becker, W. & Fuchs, A. F. (1985). Prediction in the oculomotor system: smooth pursuit during transient disappearance of a visual target. *Experimental Brain Research* **57**, 562–575.

Behrens, F. & Grusser, O. J. (1979). Smooth pursuit eye movements and optokinetic nystagmus elicited by intermittently illuminated stationary patterns. *Experimental Brain Research* **37**, 317–336.

Carl, J. R. & Gellman, R. S. (1987). Human smooth pursuit: stimulus-dependent responses. *Journal of Neurophysiology* **57**, 1446–1463.

Dallos, P. J. & Jones, R. W. (1963). Learning behaviour of the eye fixation control system. *IEEE Transactions* AC **8**, 218–227.

Eckmiller, R. & Mackeben, M. (1978). Pursuit eye movements and their neural control in the monkey. *Pflügers Archiv* **377**, 15–23.

Heywood, S. (1972). Voluntary control of smooth eye movements and their velocity. *Nature* **238**, 408–410.

HEYWOOD, S. (1973). Pursuing stationary dots: smooth eye movements and apparent movements. *Perception* **2**, 181–195.

HEYWOOD, S. & CHURCHER, J. (1971). Eye movements and the afterimage. 1. Tracking the afterimage. *Vision Research* **11**, 1163–1168.

KASE, M., NODA, H., SUZUKI, D. A. & MILLER, D. C. (1979). Target velocity signals of visual tracking in vermal Purkinje cells of the monkey. *Science* **205**, 717–720.

KELLER, E. (1988). Cerebellar involvement in smooth pursuit eye movement generation: flocculus and vermis. In *Physiological Aspects of Clinical Neuro-Ophthalmology*, ed. KENNARD, C. & ROSE, F. C., pp. 341–355. Chapman & Hall, London.

KOMMERELL, G. & TAUMER, R. (1972). Investigations of the eye tracking system through stabilized retinal images. In *Cerebral Control of Eye Movements and Motion Perception*, ed. DICHGANS, J. & BIZZI, E., pp. 288–297. Karger, Basel.

KOWLER, E. & STEINMAN, R. M. (1979). The effect of expectations on slow oculomotor control. I. Periodic target steps. *Vision Research* **19**, 619–632.

LISBERGER, S. G. & FUCHS, A. F. (1977). Role of the primate flocculus in smooth pursuit eye movements and rapid behavioural modification of the vestibulo-ocular reflex. In *Control of Gaze by Brainstem Neurons. Developments in Neuroscience*, ed. BAKER, R. & BERTHOZ, A., pp. 381–389. Elsevier, Amsterdam.

LISBERGER, S. G. & FUCHS, A. F. (1978*a*). Role of primate flocculus during rapid behavioural modification of vestibulo-ocular reflex. I. Purkinje cell activity during visually guided horizontal smooth-pursuit eye movements and passive head rotation. *Journal of Neurophysiology* **41**, 733–763.

LISBERGER, S. G. & FUCHS, A. F. (1978*b*). Role of primate flocculus during rapid behavioral modification of vestibulo-ocular reflex. II. Mossy fiber firing patterns during horizontal head rotation and eye movement. *Journal of Neurophysiology* **41**, 764–777.

MILES, F. A. & FULLER, J. H. (1975). Visual tracking and the primate flocculus. *Science* **189**, 1000–1002.

MITRANI, L. & DIMITROV, G. (1978). Pursuit eye movements of a disappearing moving target. *Vision Research* **18**, 537–539.

NODA, H. (1986). Mossy fibres sending retinal-slip, eye and head velocity signals to the flocculus of the monkey. *Journal of Physiology* **379**, 39–60.

NODA, H. & WARABI, T. (1986). Discharges of Purkinje cells in monkey's flucculus during smooth eye movements and visual stimulus movements. *Experimental Neurology* **93**, 390–403.

NODA, H. & WARABI, T. (1987). Responses of Purkinje cells and mossy fibres in the flocculus of the monkey during sinusoidal movements of a visual pattern. *Journal of Physiology* **387**, 611–628.

POLA, J. & WYATT, H. J. (1985). Active and passive smooth eye movements: effects of stimulus size and location. *Vision Research* **25**, 1063–1076.

RASHBASS, C. (1961). The relationship between saccadic and smooth tracking eye movements. *Journal of Physiology* **159**, 326–338.

ROBINSON, D. A. (1982). A model of cancellation of the vestibulo-ocular reflex. In *Functional Basis of Ocular Motility Disorders*, ed. LENNERSTRAND, G., ZEE, D. S. & KELLER, E. L., pp. 5–13. Pergamon Press, Oxford.

ROBINSON, D. A., GORDON, J. L. & GORDON, S. E. (1986). A model of the smooth pursuit eye movement system. *Biological Cybernetics* **55**, 43–57.

VON NOORDEN, G. K. & MACKENSEN, G. (1962). Pursuit movements of normal and amblyopic eyes. *American Journal of Ophthalmology* **53**, 325–336.

WESTHEIMER, G. & BLAIR, S. M. (1973). Oculomotor defect in cerebellectomized monkeys. *Investigate Ophthalmology and Visual Science* **12**, 618–620.

WHITTAKER, S. G. & EAHOLTZ, G. (1982). Learning patterns of eye motion for foveal pursuit. *Investigative Ophthalmology and Visual Science* **23**, 393–397.

YASUI, S. & YOUNG, L. R. (1975). Perceived visual motion as effective stimulus to pursuit eye movement system. *Science* **190**, 906–908.

YOUNG, L. (1977). Pursuit eye movement – what is being pursued? In *Control of Gaze by Brainstem Neurones. Developments in Neuroscience*, ed. BAKER, R. & BERTHOZ, A., pp. 29–36. Elsevier, Amsterdam.

YOUNG, L. R. & STARK, L. (1963). Variable feedback experiments testing a sampled data model for eye tracking movements. *IEEE Transactions* HFE **4**, 38–51.