Biol. Cybern. 55, 43–57 (1986)

Biological Cybernetics

A Model of the Smooth Pursuit Eye Movement System

D. A. Robinson, J. L. Gordon*, and S. E. Gordon

Departments of Ophthalmology and Neurology, The Johns Hopkins University, School of Medicine, Baltimore, MD 21205, USA

Abstract. Human, horizontal, smooth-pursuit eye movements were recorded by the search coil method in response to Rashbass step-ramp stimuli of 5 to 30 deg/s. Eye velocity records were analyzed by measuring features such as the time, velocity and acceleration of the point of peak acceleration, the time and velocity of the peaks and troughs of ringing and steady-state velocity. These values were averaged and mean responses reconstructed. Three normal subjects were studied and their responses averaged. All showed a peak acceleration-velocity saturation. All had ringing frequencies near 3.8 Hz and the mean steady-state gain was 0.95.

It is argued that a single, linear forward path with any transfer function $G(s)$ and a 100 ms delay (latency) cannot simultaneously simulate the initial rise of acceleration and ring at 3.8 Hz based on a Bode analysis. Also such a simple negative feedback model cannot have a steady-state gain greater than 1.0; a situation that occurs frequently experimentally. L.R. Young's model, which employs internal positive feedback to eliminate the built-in unity negative feedback, was felt necessary to resolve this problem and a modification of that model is proposed which simulates the data base. Acceleration saturation is achieved by borrowing the idea of the local feedback model for saccades so that one nonlinearity can account for the acceleration-velocity saturation: the main sequence for pursuit. Motor plasticity or motor learning, recently demonstrated for pursuit, is also incorporated and simulated.

It was noticed that the offset of pursuit did not show the ringing seen in the onset so this was quantified in one subject. Offset velocity could be characterized by a single exponential with a time constant of about 90 ms. This observation suggests that fixation is not pursuit at zero velocity and that the pursuit system is turned on when needed and off during fixation.

1 Introduction

Smooth pursuit eye movements are normally made when we track an object moving smoothly in the visual environment. Their purpose is to keep the image of the object near the fovea. Large errors are eliminated by saccadic eye movements and the role of the pursuit system is subsequently to match eye velocity to target velocity. Young and his colleagues proposed a model for the pursuit system in 1968 (Young et al. 1968; Young 1971) but since then there have been no further efforts to extend it or replace it. Meanwhile, several studies have provided additional data. Lisberger et al. (1981) demonstrated a saturation effect on eye acceleration and Optican et al. (1985) showed plasticity or motor learning in the pursuit system – a form of parametric, adaptive control. Moreover, no one has looked at the dynamics of pursuit movements in sufficient detail to form a basis for the sort of model presented here and this was one of our first tasks. It then seemed appropriate to see if Young's model could be extended to include all these phenomena.

It is important to point out what the proposed model does not address: 1) It assumes the head is stationary and consequently is not involved with interactions between the pursuit system and the vestibulo-ocular reflex; 2) When a target moves back and forth in a periodic, predictable manner, the pursuit system quickly learns to anticipate the target and tracks it with little or no delay compared to the usual latency of 130 ms seen when targets move unpredictably (e.g. Michael and Melvill Jones 1966). This interesting phenomenon has not been adequately

* J. L. Gordon is currently in the Dept. of Otolaryngology, University of Pittsburgh, Pittsburgh, PA, USA

modelled and no such attempt is offered here – only unpredictable movements are considered; 3) The relationship between pursuit and optokinetic effects from a moving or stationary background are not included in this model which only considers the situation of a small, (foveal), clearly-visible target moving across a tecture-free background; 4) Wyatt and Pola (1981) have confirmed the observation that the pursuit system will respond to an error in retinal image position in the absence of retinal image motion – the latter being the stimulus usually thought to drive the pursuit system. Nevertheless, when a subject tracks a moving target, it is generally believed that image motion is a much stronger stimulus than image position (e.g. Rashbass 1961) and consequently, the proposed model will concentrate on the response to the former stimulus.

The most common, unpredictable, test stimulus is a ramp of target motion; the target is initially stationary and, at an unexpected time, begins to move at a constant velocity and this study concentrates on this situation. Looked at as a velocity servo, we examined and modelled the response to a step in target velocity. Because no previous studies have examined the fine structure of this oculomotor response (time to peak acceleration, amplitude and frequency of ringing, etc.) we examined it in three normal subjects for horizontal target motion to provide a data base for the model. Because it was noticed that the dynamics, when the eye stopped pursuing, differed from those when it started, stopping was further examined in one of these subjects.

2 Methods

For the experimental data base, three subjects participated; all were experienced in eye-movement recording sessions and all knew considerable oculomotor physiology. The target was a small, bright spot of white light projected onto the back of a translucent tangent screen 1 m in front of the subject. The head was stabilized with a chin rest. Eye movements were recorded by the magnetic-field/eye-coil method set at a full-scale range where the accuracy was 0.25 deg. The bandwidth was 0–80 Hz (limited by a pen recorder). Horizontal eye velocity was obtained by an analogue differentiator with a band-width of 0–50 Hz. The target followed a step-ramp time course first suggested by Rashbass (1961): it stepped in one direction and immediately moved at a constant velocity in the other. The step amplitude and ramp velocity were adjusted so that the target recrossed its starting position in 0.2 s, a value intended to allow a pursuit movement to occur without the interruption of a catch-up saccade.

For target velocities of 5, 10, and 20 deg/s, the target started at primary position (straight ahead) and moved left or right at unexpected times and velocities for about 1.5 s in a random sequence. In order to prolong the duration of tracking at 30 deg/s, the target started from 10 or 30 deg left for rightward ramps and vice versa so that, in this case, the subject knew the direction but not the timing of the target motion. Data were recorded on a chart recorder and analyzed by hand as described in the text. When it was suspected that the dynamics of the offset of pursuit differed from its onset, the paradigm was modified so that the target stopped, in a Rashbass step-stop fashion (see Fig. 1) at a random time between 1 and 2 s. One of the three subjects was rerun under these circumstances and the dynamics with which the eye came to rest were measured, again as described in the text.

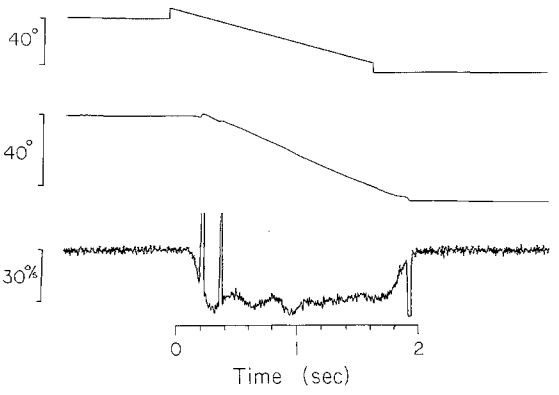

Fig. 1. A recording of target position *(top trace)*, eye position *(middle trace)*, and eye velocity *(bottom trace)*, during the start and stop of a Rashbass step-ramp stimulus for pursuit at 30 deg/s for subject DR

Computer simulation was accomplished by a simulation program called ASP written by Drs. L.M. Optican and H.P. Goldstein in the language C and run on a PDP 11/45 under the UNIX operating system.

3 Results

The Human, Pursuit Ramp Response

The sample record in Fig. 1 shows most of the features to be described: an increasing rise in velocity to a peak acceleration; velocity overshoot and ringing settling to a steady state; spontaneous oscillations during pursuit; a simple exponential decay of velocity to zero when the target stopped. Note that the step of the target at start and stop was too large for this subject so that saccades (back-up saccades in this case) were reduced in size but not eliminated as hoped. This was the usual case for all subjects and trials.

Onset. Because of trial-to-trial variations in the time of events in the ramp response, averaging over trials

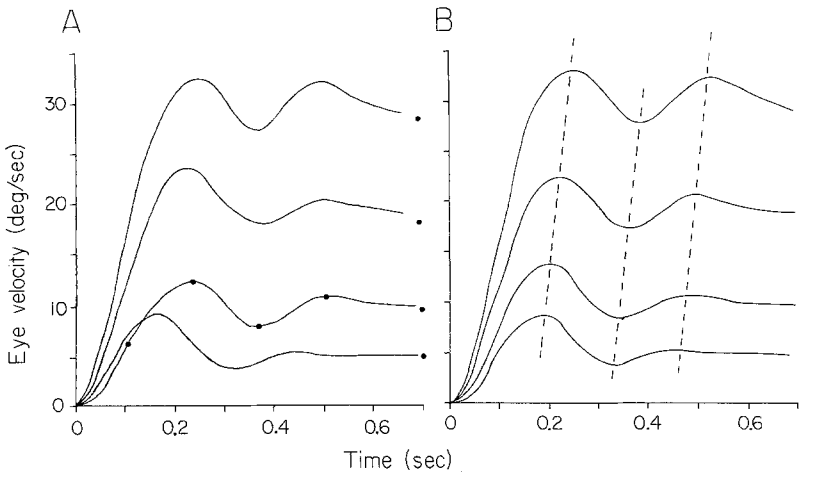

Fig. 2A and B. The ramp response for subject DR. **A** Filled circles on the 10 deg/s response show those features that were averaged over individual trials to obtain the averaged results shown. Circles at the end of all traces indicate the estimate of steady-state eye velocity. **B** The curves in **A** were further regularized by finding the linear regressions between the feature values and target velocity and using the values given by the regression line. Those involved in timing are shown by dashed lines

would smooth out and blurr its features. Consequently, the data were analyzed by picking out the amplitude and timing of its important features, averaging them, and then reconstructing a response that reflected these averaged features. The features were: latency (after this all times were measured from the beginning of the eye movement); peak acceleration, the time to reach it and the velocity at that time; peak velocity of the first overshoot and the time to reach it; velocities and time of occurrence of subsequent valleys and peaks during the ringing; steady-state eye velocity. These values were averaged for 15–20 trials at each velocity and a curve was then drawn by hand between these points to resemble a typical response. Results for subject DR are shown in Fig. 2A.

Moderate, idiosynchratic differences in the dynamics (mainly in acceleration) occurred between pursuit to the left and right. Since the purpose of the model was to simulate more global features, left and right movements were averaged together. A phenomenon appeared at 20 and 30 deg/s that we have commonly observed but has seldom been mentioned in the literature. When a subject knows in advance that the target is going to stop moving at a certain position or time, pursuit begins to slow down many hundreds of msec before the target stops. Subjects who are aware of this phenomenon still cannot seem to stop themselves from doing it by concentrated effort. At target velocities of 20 and 30 deg/s, this decrease in eye velocity often encroached on the ringing period and it became difficult to estimate what would have been steady-state eye velocity. The values shown are the result, in part, of subjective judgements from individual records. This phenomenon is not seen in Fig. 1 because the time of stopping there was unexpected, a method that often prevented it, but this was not discovered until pursuit onset had already been well documented in the three subjects with a fixed pursuit duration where the phenomenon always appeared.

Figure 2A shows considerable fluctuations in feature points as target velocity increased. Each subject behaved differently in this regard. A general trend due to an acceleration saturation was evident; as target velocity increased it usually took longer for the various feature points to occur. It would be possible, but not useful, to produce a model with parameters that varied with eye velocity (nonlinearities) in order to simulate each of these individual curves for each subject. Consequently, the fluctuations were smoothed out to leave only general trends. Each feature was plotted against target velocity and fit by a linear regression. The value of the feature was when altered to lie on the regression line at each target velocity. The result is shown in Fig. 2B. The feature points now vary in a regular way with target velocity as shown by the dashed lines but note that they are only the regression lines of the times of features and do not reflect the regression values of the amplitudes (accelerations and velocities) of the features. For these curves, there is some hope of simulating them all with just one nonlinearity.

Data from the other two subjects were treated similarly and Fig. 3A shows results for all three subjects for a target velocity of 20 deg/s. Note the remarkable differences in just these three normal subjects. Subject DZ showed a steady-state gain greater than 1.0 which has important theoretical implications for the model (see below). His steady-state gains for target velocities of 5, 10, 20, and 30 deg/s were 0.94, 0.96, 1.13, and 1.12 respectively (1.04 mean) so that his gain being near or greater than 1.0 was not a peculiarity at only one velocity but a general feature of his pursuit. RT had low gains at the higher velocities. His steady-state gains for the four velocities were 1.02, 0.94, 0.75, and 0.77 respectively. RT also had low accelerations. The data for DR lay in between. All had pronounced ringing. For purposes of modelling, the mean values of their features were averaged for the

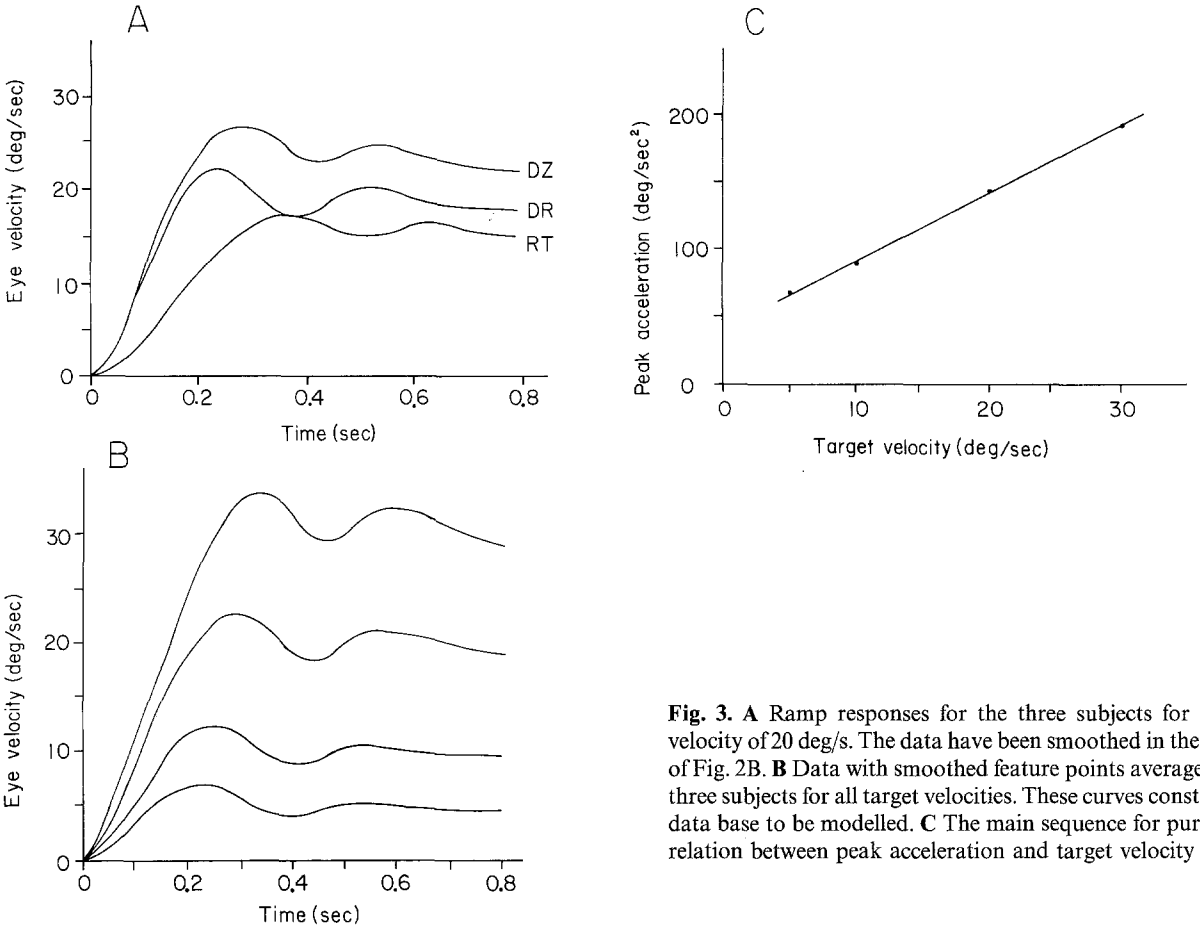

Fig. 3. A Ramp responses for the three subjects for a target velocity of 20 deg/s. The data have been smoothed in the manner of Fig. 2B. **B** Data with smoothed feature points averaged for all three subjects for all target velocities. These curves constitute the data base to be modelled. **C** The main sequence for pursuit; the relation between peak acceleration and target velocity from **B**

Table 1. The features of the data base for the four target velocities. t_1, time of peak acceleration; v_1, velocity at time t_1; a_1, peak acceleration; t_2, time of peak velocity; v_2, peak velocity; t_3, time of first valley in ringing; v_3, velocity of first valley in ringing; t_4, time of second peak in ringing; v_4, velocity of second peak; v_{ss}, steady-state velocity; f, frequency of ringing; $v_2 - v_{ss}$, amount of velocity overshoot above steady-state velocity; G_0, steady-state gain

Target velocity	t_1	v_1	a_1	t_2	v_2	t_3	v_3	t_4	v_4	v_{ss}	f	$v_2 - v_{ss}$	G_0
5	113	4.0	61.7	227	7.1	381	4.0	510	5.2	4.8	3.4	2.3	0.96
10	118	6.6	89.2	249	12.2	395	8.9	524	10.4	9.5	4.0	2.7	0.95
20	128	11.8	141.2	289	22.3	424	18.3	553	20.7	18.6	3.9	3.7	0.93
30	138	17.3	190.1	334	33.1	455	28.5	584	31.7	28.4	3.9	4.7	0.95

three subjects. It was felt that if a model could simulate mean subject behavior it could easily be altered to simulate individual variations.

In Fig. 3B, the features, not the curves, were averaged across subjects and representative curves drawn through these average features. They constitute the data to be simulated by the model. The features themselves are given in Table 1. Three aspects of these data are noteworthy. Like saccades, the peak rate of change of these curves is not proportional to their final displacement. For saccades this represents a velocity saturation and results in the well-known velocity-amplitude relationship (e.g. Boghen et al. 1974), frequently referred to as the main sequence (Bahill et al. 1975). For pursuit, it represents an acceleration saturation (Lisberger et al. 1981) and results in an acceleration-velocity relationship shown in Fig. 3C which may be thought of as a main sequence for pursuit. Note, however, that this applies only to unpredictable pursuit stimuli and not to periodic, predictable stimuli that produce much larger accelerations (Lisberger et al. 1981).

A second aspect is the ringing shown by all three subjects. For each subject the frequency of ringing

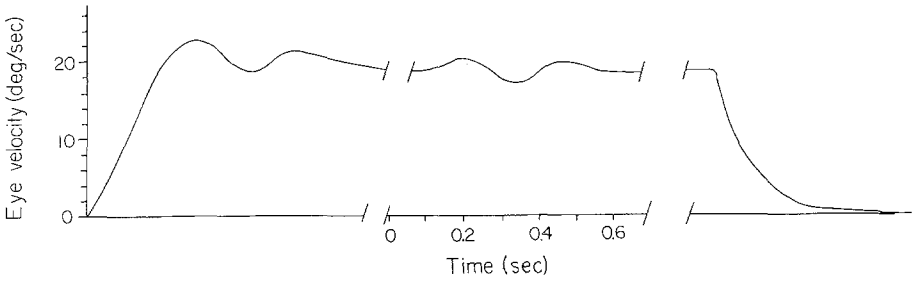

Fig. 4. Comparison of onset (left) and offset (right) of pursuit of a target a 20 deg/s for subject DR. The onset is copied from Fig. 2B. The offset is approximated by an exponential with a time constant of 90 ms. The center section calls attention to spontaneous oscillations that occur occasionally at about 4 Hz during pursuit. These curves are a reconstruction based on averaging

depended very little on target velocity and for the three subjects, averaged over target velocities, was 3.7, 4.0, and 3.6 Hz. These frequencies are similar and the overall mean frequency was 3.8 Hz averaged over all three subjects and all target velocities. A third aspect is that the amount of velocity overshoot did not rise linearly with target velocity. In Table 1, this overshoot is $(v_2 - v_{ss})$. Its value rose from 2.3 to 4.7 deg/s (a factor of 2) while target velocity rose from 5 to 30 deg/s (a factor of 6). Thus the ringing amplitude decreased as did acceleration, relative to increases in target velocity, suggesting that ringing is created in some part of the system that shares the acceleration saturation.

Offset. Any quasi-linear model of pursuit will not distinguish between a transition from 0 to 20 deg/s and one from 20 to 0 deg/s. Tracking a target moving at 0 deg/s, however, is often called fixation and it has been suspected that this activity is different from pursuit. Consequently, the cessation of pursuit was examined in detail for subject DR.

As already mentioned, causing the target to stop at unexpected times proved an excellent remedy for the problem that when the subject knows when (or where) the target will stop, pursuit slows almost to a stop hundreds of msec before that time. As shown in Fig. 1, pursuit usually continued, under this paradigm, without a decrease in velocity, right up to the time the target stopped. This strategy also allowed the observation of spontaneous bouts of oscillations during what would otherwise be the steady-state portion of tracking (Fig. 4). Samples of such oscillations showed a mean frequency of 3.96 Hz and this did not depend on eye velocity.

When the target jumped and stopped, eye velocity began to decrease after 137 ms on average, about 30 ms less than the latency for the on response. Peak deceleration occurred almost at once and closely followed the behavior for acceleration shown in Fig. 3C. Specifically, for target velocities of 5, 10, 20, and 30 deg/s, peak deceleration was 69, 95, 122, and 179 deg/s^2, values that varied by less than 14% from the appropriate values of acceleration (Table 1).

The subsequent time course was, however, very different; there was no overshoot or ringing. Instead, eye velocity decayed to zero following a time course that could be described roughly by an exponential with a single time constant. To approximate it as such, the time was measured for eye velocity to fall to 37% of its initial value. For target velocities of 5, 10, 20, and 30 deg/s, the mean time constants were 82, 67, 115, and 98 ms respectively. No trend was obvious; the overall mean value was 90 ms. As a check, the time was measured to the point where eye velocity had returned to zero. Again, no trend was seen with target velocity and the overall mean was 282 ms ($N = 28$), close to the expected value of three time constants.

Nonetheless, a discrepancy arises when one recalls that the time constant of a single exponential should equal initial eye velocity divided by initial acceleration. Since, because of the acceleration saturation, these two variables do not increase in proportion as target velocity increases, one should expect an increase in the time constant, predicted in this way, at larger velocities. These predicted values were found to be 77, 80, 160, and 142 ms respectively. The first two agree with the mean value of 90 ms, the last two are about 50% larger. This means that at the two higher target velocities, eye velocity dropped more rapidly than would be expected by a single exponential, and a double exponential would have been a better fit.

In summary, a fair approximation to the time course of offset is a single exponential with a time constant of 90 ms. Since the model, proposed here, does not deal with the offset (see Discussion), further characterization was not done. The main point is that offset is quite different from onset as shown for emphasis in Fig. 4. In particular, there was no ringing

at offset and spontaneous oscillations, frequently seen during pursuit, were absent during subsequent fixation.

The Model

Figure 5A summarizes what little we know of the neurophysiology of pursuit. The velocity of the target's image across the retina, $\dot{e}$, is taken as the major stimulus to pursuit and the error that the system tries to minimize. It is extracted by direction-selective, motion-sensitive cells, probably in primary visual cortex. The signal is currently believed to leave the cortex, at least in part, through the medial temporal area (e.g. Newsome et al. 1985), descend to the dorsolateral pontine nuclei (e.g. Mustari et al. 1984) to be relayed to the cerebellar vermis (Suzuki et al. 1981) and flocculi (Miles and Fuller 1975; Lisberger and Fuchs 1978) where the signal has been transformed into an eye velocity command, $\dot{E}'$, observed on Purkinje cells.

From there the signal $\dot{E}'$ passes directly (in a signal sense) to the motoneurons, with gain T_{e1}, and indirectly through a neural integrator, NI, located in the vestibular-prepositus hypoglossi nuclear complex (Cannon and Robinson 1986) to allow the motoneurons to receive the correct combination of eye-position and eye-velocity commands. The plant (eye muscles and orbital tissues) can be approximated by two lags with time constants T_{e1} and T_{e2} of about 224 and 13 ms respectively (Keller 1973). The combination of the two paths, T_{e1} and 1/s, cancels the pole $1/(sT_{e1}+1)$ in the plant leaving a pure integration and the second plant lag $1/(sT_{e2}+1)$. The initial differentiator and final integrator may be dispensed with by choosing target and eye velocities as the variables of interest. Thus the scheme in Fig. 5A simplifies to that in Fig. 5B.

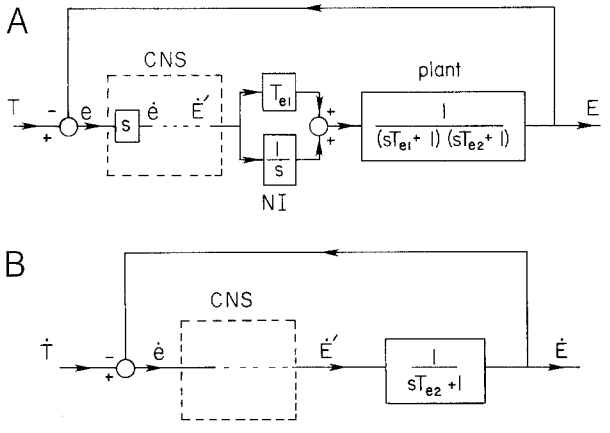

Fig. 5A and B. A summary of known signal processing in pursuit. **A** T is target position, E is eye position, e is retinal position error and $\dot{e}$ its velocity. See text for a review of the neurophysiology. **B** The premotor circuits and plant in A have been combined and the system redrawn with $\dot{T}$, target velocity, and $\dot{E}$, eye velocity, as the input and output variables of interest

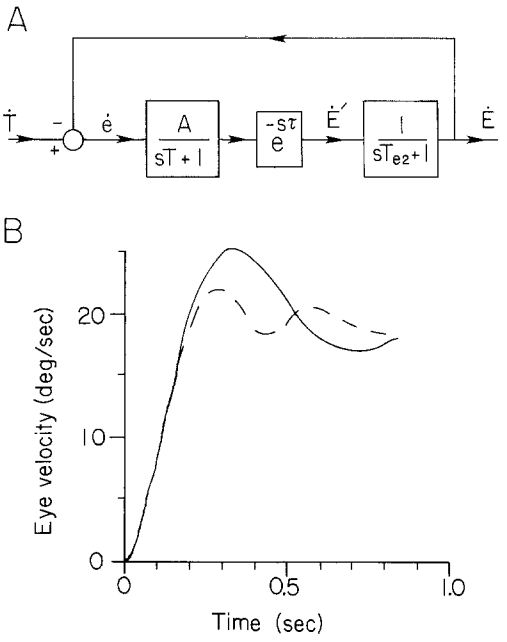

Fig. 6. A A simple model of pursuit. T_{e2} is 15 ms, the delay τ is 130 ms, A is 20 and T must be 3.5 s to match the initial rate of rise of $\dot{E}$. **B** Computer simulation of model in A for a target velocity of 20 deg/s, compared with the experimental data *(dashed line)* from Fig. 3B

To appreciate the need for internal positive feedback, one may commence with the most simple sort of velocity tracking model: a forward path consisting of a gain A and a lag of time constant T as shown in Fig. 6A. The gain A determines the closed-loop, steady-state pursuit gain $\dot{E}/\dot{T}$ or G_0 which is $A/(1+A)$. This value is about 0.9–0.95 in most young, healthy subjects in this velocity range (e.g. Schalén 1980; Meyer et al. 1985; this study). We have seen gains as low as 0.7 in healthy, naive subjects, unknowledgeable about eye movements, but gains below this, we would regard as abnormal. To fit our data base where G_0 is 0.95, A should equal 20.

To estimate T one can match the early accelerations seen in Fig. 3B. For a target speed of 20 deg/s this should be close to 141 deg/s^2 (Table 1). In the model of Fig. 6A, this early acceleration will be $20(A/T)$ deg/s^2 since $A/(sT+1)$ will look, initially $(sT \gg 1)$, like a pure integrator with a gain A/T and this will determine behavior in the first 130 ms before any feedback signals can have an effect. Thus A/T should be 141/20 or 7.1 and T should then be 20/7.1 or 2.8 s. Finally a pure delay of 130 ms was added. Figure 6B shows the response to a step in target velocity of 20 deg/s together with the experimental result (dashed

line) from Fig. 3B. T was readjusted slightly to 3.5 s in order to improve the match over the whole rising phase of $\dot{E}$. In obtaining this trace, T_{e2} was 15 ms but its effect on the general behavior is small.

The response shows overshoot and ringing that is qualitatively similar to the experimental results but the frequency of ringing is much too small. This frequency, f, is close to that for which the loop phase lag is 180 deg (because the system is nearing instability). Since the element $A/(sT+1)$ has a phase lag of almost 90 deg in this range, ringing occurs when the phase lag of the delay, τ, which is (360τ) deg, equals another 90 deg. If τ is 0.13 s, f is 1.9 Hz as shown in Fig. 6B. It is only half the experimental value of 3.8 Hz, a value that was fairly consistent across subjects and target velocities. To make such a model ring at 3.8 Hz due to the delay τ, it would have to be reduced to 65 ms, in conflict with the observed latency.

Before abandoning all models of the form shown in Fig. 6A for something more complicated, it is important to demonstrate that adding more lead and lag elements cannot salvage this topology. Specifically, if all the elements in Fig. 4A other than the delay τ are gathered into a linear operator $G(s)$, it should be shown that no choice of $G(s)$ can produce the dashed curve (experimental data) in Fig. 6B. The argument, briefly, is that the frequency of ringing tells us something about the properties of $G(s)$ in the region around 3.8 Hz while the shape of the initial rise of $\dot{E}(t)$ also requires certain high-frequency properties of $G(s)$ and the two requirements are incompatible. Specifically, the phase lag of a 100 ms delay, τ, at 3.8 Hz is 137 deg. This small a delay has been reported by Gellman and Carl (1985) and is chosen here to be as kind as possible to the model in Fig. 6A. Since the phase lag around the loop must be close to 180 deg at 3.8 Hz, the lag of $G(s)$ at this frequency can then only be about 45 deg. It will always be necessary to include at least one term such as $A/(sT+1)$ to reduce the low-frequency gain A to a value just below 1.0 at 3.8 Hz (for stability) and this term will have a phase lag of 90 deg at this frequency. Consequently, one must introduce a lead element to create a lead of about 45 deg at 3.8 Hz and reduce the overall phase lag back to 45 deg. Any $G(s)$ that does this will have the appearance of $(0.042s+1)/s$ in the region around 3.8 Hz. Here the break point of the lead was taken at 3.8 Hz itself giving a time constant $(1/\omega)$ of 0.042 s. The pole, 1/s, represents the phase lag of 90 deg from $A/(sT+1)$, the zero represents the lead needed for the 45 deg phase advance.

The initial response to the step is a lagged ramp seen clearly in Fig. 3B. The 20 deg/s target velocity step appears to be converted into a ramp with a slope that represents an acceleration of 141 deg/s^2 followed by another lag that causes eye velocity to initially rise more slowly to that value. An estimate of the time constant of the latter is roughly 40 ms. Consequently, from this standpoint, the high-frequency behavior of $G(s)$ should have the appearance of $1/(s(0.04s+1))$. The break point of the lag $(1/(2\pi \times 0.04))$ is 4 Hz, nearly the same frequency as that of the ringing. At this frequency the phase lag of $G(s)$ must now be around 135 deg.

These requirements are contradictory. To ring at 3.8 Hz, $G(s)$ must behave like a lag-lead with a phase lag not more than about 45 deg in this region. To simulate the sluggish rising phase, $G(s)$ must behave like a lag-lag with a phase lag of about 135 deg. Put another way, if one matched the initial rise only, the system could still be made to ring but at too low a frequency as shown in Fig. 6B. If one matched the frequency of ringing, the system would have too high a gain at higher frequencies and the initial response would be too prompt. Because of the required lead element, one could even get a small, step increase in eye velocity for a step of target velocity, a behavior that has never been observed.

We feel that, given the data base, the above analysis is conclusive: no $G(s)$ exists that can simulate the curves of Fig. 3B with the form of the model in Fig. 6A. Adding an acceleration saturation would require a zero-memory nonlinearity operating directly on $\dot{e}$ in Fig. 6A. This would not help since it would only effect gain changes and the above argument is based entirely on phase shifts. In the model, we shall propose (Fig. 8) that the feedback is removed so that the system can be split into two parts where the initial rise and subsequent ringing can be controlled independently. Finally, there is another argument against the model in Fig. 6A. Subject DZ in this study had steady-state gains greater than 1.0 at target velocities of 20 and 30 deg/s (1.13 and 1.12 respectively). Moreover, an average gain of 0.95 with a standard deviation of $\pm 6\%$ meant that in many individual trials, other subjects frequently began tracking the target with a steady-state eye velocity larger than target velocity. Such behavior is impossible for the system in Fig. 6A.

Internal Positive Feedback. Young et al. (1968) were attempting to model the pursuit system with a major, closed loop time constant on the order of 40 ms. This meant they were using a value of T in Fig. 6A about ten times smaller than the value of 3.5 s required to reproduce the correct initial acceleration. This made the high-frequency gain in their model so large that their system was very unstable and oscillated perniciously. These authors took a bold approach to this problem: if negative feedback causes the system to oscillate, get rid of it. Since the negative feedback comes about because the retina is attached to the eye,

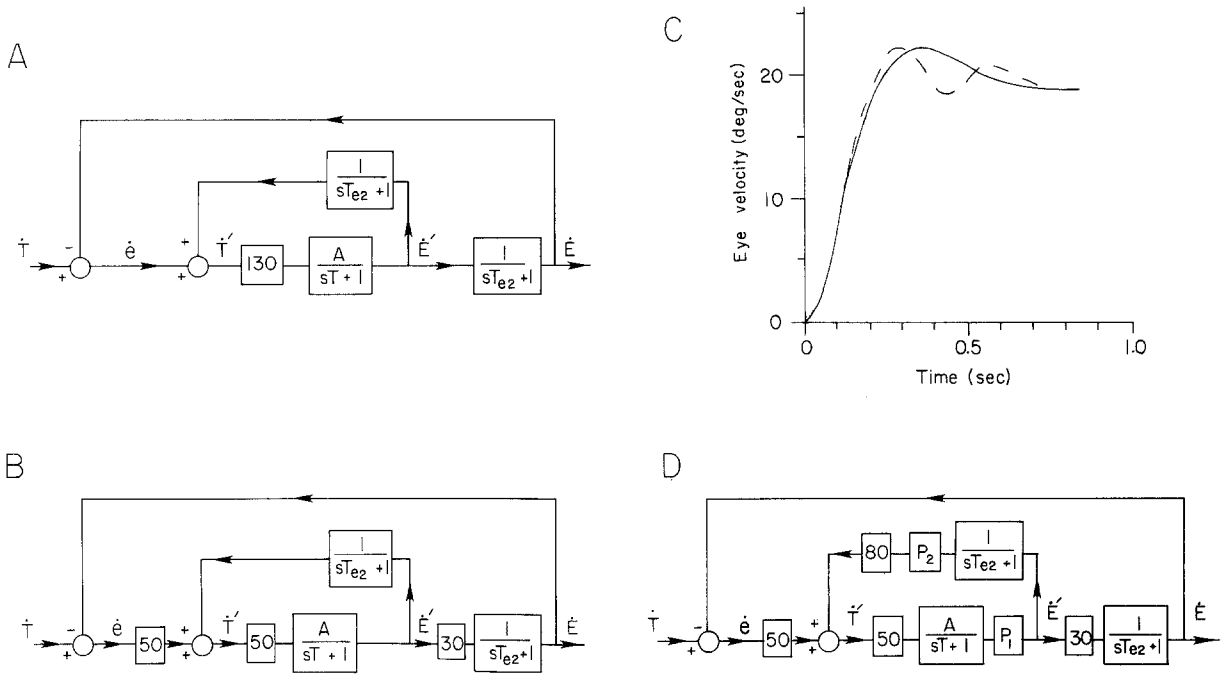

Fig. 7A–D. Evolution of the positive feedback model. Numbers in boxes refer to the number of ms of a pure delay. **A** A slightly modified version of the model of Young et al. (1968). $\dot{T}'$ is a centrally reconstructed copy of target velocity. Other symbols are as in Fig. 6. **B** A redistribution of delays to conform to physiological findings. **C** The response of the model in **B** to a step of target velocity to show serious differences from experimental data *(dashed curve)* from Fig. 3B (20 deg/s). **D** Adding 80 ms in the positive feedback brings efference copy $\dot{E}'$ and reafference, $\dot{e}$, back into registration and eliminates all ringing. P_1 and P_2 are associated with plasticity (see text)

one cannot, of course, literally get rid of it, but one can cancel it out by internal positive feedback as shown in Fig. 7A. This figure is modified from that of Young et al. (1968) by ignoring, for the present, nonlinearities and including a more realistic plant model. If the inner positive feedback path is to cancel the outer, it too must contain a model of the uncompensated, second plant lag $1/(sT_{e2}+1)$. Although T_{e2} is only 15 ms, its absence in this path will create peculiar transients. Now that there is effectively no feedback, A should be 0.95 and T 134 ms (so that A/T times target velocity (20 deg/s) equals desired acceleration, 141 deg/s^2). This model is, of course, completely stable since it has no feedback.

Using positive feedback is not a totally novel approach. Such feedback is a copy of the motor command and has been called efference copy (von Holst and Mittelstaedt 1950) or corollary discharge (Sperry 1950) and has been proposed to allow the nervous system to tell whether a retinal image motion was due to target motion or eye motion. This allows perceptual stability of stationary objects when the eye moves. Specifically, in Fig. 7A, the addition of $\dot{E}'$ (the efference copy) and $\dot{e}$ (here called reafference because it is a direct sensory consequence of eye motion) recreates a central copy of target velocity, $\dot{T}'$, that is independent of eye movements. Whether this signal leads to a perception of target velocity is not the subject of this investigation but, conversely, in a number of situations in which pursuit occurs together with a perception of a target velocity while $\dot{e}$ is missing, it may be that a perceptual signal can be injected into the model at the site $\dot{T}'$ to produce the pursuit. Pursuing one's hand in total darkness (Steinbach 1969) is one example but there are many others (Young 1977; Steinbach 1976; Yasui and Young 1975). This idea will not be considered further; the point being that the basic concept is not new and has its historical basis in the perceptual and psychophysical literature.

There is, however, a problem with Fig. 7A. The internal positive feedback path cannot possibly enclose the entire 130 ms delay. It is known that there is a 50 ms delay in the retina before light, striking the retina, creates electrical activity in visual cortex. This delay depends on stimulus intensity and 50 ms is chosen only to be representative. It is also the case that, when one stimulates pre-oculomotor areas such as the superior colliculi (Robinson 1972) or cerebellum (Ron and Robinson 1973) eye movement does not begin until about 30 ms later. Again, this delay depends on stimulus intensity and 30 ms is taken as representative. About 10 ms is lost in the muscles alone (Fuchs and Luschei 1970) and may be regarded as a minimum value for this delay but where the motor copy signal

originates is unknown. If 30 ms is accepted, it leaves only 50 ms (130 less 50 less 30) for central processing to be included under the positive feedback loop. This distribution of delays is shown in Fig. 7B where delays, as in all subsequent figures, will be represented simply as the number of msec enclosed in a box.

Having removed 80 ms or 62% of the total delay from the protection of the positive feedback loop, we have the situation that the delay from $\dot{E}'$ to $\dot{T}'$ around the outer loop has a delay of 80 ms while that around the inner loop has none. How this mismatch affects behavior is shown in Fig. 7C. Overshoot and ringing persist and the frequency of ringing is less than 2 Hz. The reason is much the same as that in Fig. 6; ringing occurs near that frequency at which a delay of 130 ms has a phase lag of 90 deg. It would be desirable to eliminate this behavior. In addition there is a conceptual problem. If efference copy, $\dot{E}'$, via the positive feedback path is to be compared to reafference, $\dot{e}$, via the outer negative loop, it makes no sense to compare the former with no delay to the latter delayed by 80 ms. This can be easily corrected by delaying the former by 80 ms so that $\dot{T}'$ is estimated from two signals in the same time frame. This arrangement is shown in Fig. 7D. It, like the model in Fig. 7A, is absolutely stable since it effectively has no feedback; the inner and outer feedback loops exactly cancelling each other. The step response of this model resembles a single exponential largely governed by the time constant T; there is no ringing. The addition of the two gains P_1 and P_2, with normal values near 1.0 is discussed next.

Strong supporting evidence for positive feedback comes from studies of plasticity or motor learning or, more properly, parametric adaptive feedback. The classic example is the vestibulo-ocular reflex that transduces head velocity via the semicircular canals and causes eye velocity to be equal and opposite. Thus the line of sight remains stationary in space and images do not slip on the retina when the head moves. This system operates without the benefit of visual feedback which would be at least 80 ms too late, yet it retains its calibration over a life time. Clearly its integrity must be monitored by some system and its gain (eye velocity/head velocity) maintained by parametric control. This has been amply demonstrated by studies that artificially alter the relation between head velocity and retinal image motion and cause large changes in the gain of this reflex (Gonshor and Melvill Jones 1976; Robinson 1976; Ito 1982). The message of these studies is clear: any system without the benefit of on-line feedback must be protected against dysmetria by a system that can detect it and subsequently alter parameters in the system to restore normal function. It operates on a trial-by-trial basis and experiments show that readjustments require hours to days.

The scheme in Fig. 7D fits this situation perfectly. Feedback has been eliminated and with it has gone a major purpose of feedback – to protect a system against changes in internal parameters. Consequently, one can predict that the pursuit system should be capable of plastic changes and Optican et al. (1985) have shown just that. Details of their experiments are given later when the model will be asked to simulate their results. Conversely, any system that exhibits motor plasticity is behaving like a system that does not depend on negative feedback to make its performance insensitive to parameter changes. While the demonstration of plasticity in the pursuit system does not prove that it has gotten rid of its negative feedback by internal positive feedback, it nevertheless offers strong support.

P_1 in Fig. 7D is an adjustable gain in the forward loop. If, due to a lesion anywhere in the system, pursuit becomes abnormal, some system that undoubtedly uses visual clues, detects the dysmetria and increases or decreases P_1 until G_0 returns to its normal value of about 0.95. P_1 is placed within the positive feedback loop only because it is thought to be fairly central; other locations are possible. If A should change, a compensatory change in P_1 is enough but if, as will be considered later, a change occurs in the plant due to muscle weakness, P_1 must be increased but $\dot{E}'$ will then no longer be a copy of $\dot{E}$. This requires a decrease in P_2 so that the signal sent back to calculate $\dot{T}'$ remains veridical. Note that in Fig. 7D it is very simple to simulate the behavior of subjects with steady-state gains G_0 greater than 1.0 – another argument in favor of the basic topology of this model.

The use of internal positive feedback to get rid of negative feedback is the crux of this model. It has three main results: it provides a solution to the inadequacy of models of the form of Fig. 6A; it is compatible with the discovery of plasticity in the pursuit system; and it permits gains G_0 that are equal to or larger than 1.0. There may be other ways to solve these problems but this is, at least, a simple one.

Ringing and Acceleration Saturation. The model in Fig. 7D does not ring and being linear would not exhibit an acceleration saturation. It is tempting to introduce ringing by deliberately mismatching the dynamics of the inner and outer feedback paths. There are two ways to do this: make their gains differ or make their delays differ. Fig. 7C is an extreme example of the latter. The gains may be made to differ by increasing P_1 and decreasing P_2 by the same ratio in order to keep G_0 at 0.95. The model then rings but at a frequency around 2.0 Hz. Such a scheme can be reduced to an equivalent, single, negative feedback path with a gain of $(1-P_2)$ and a loop delay of 0.13 s. The latter largely determines

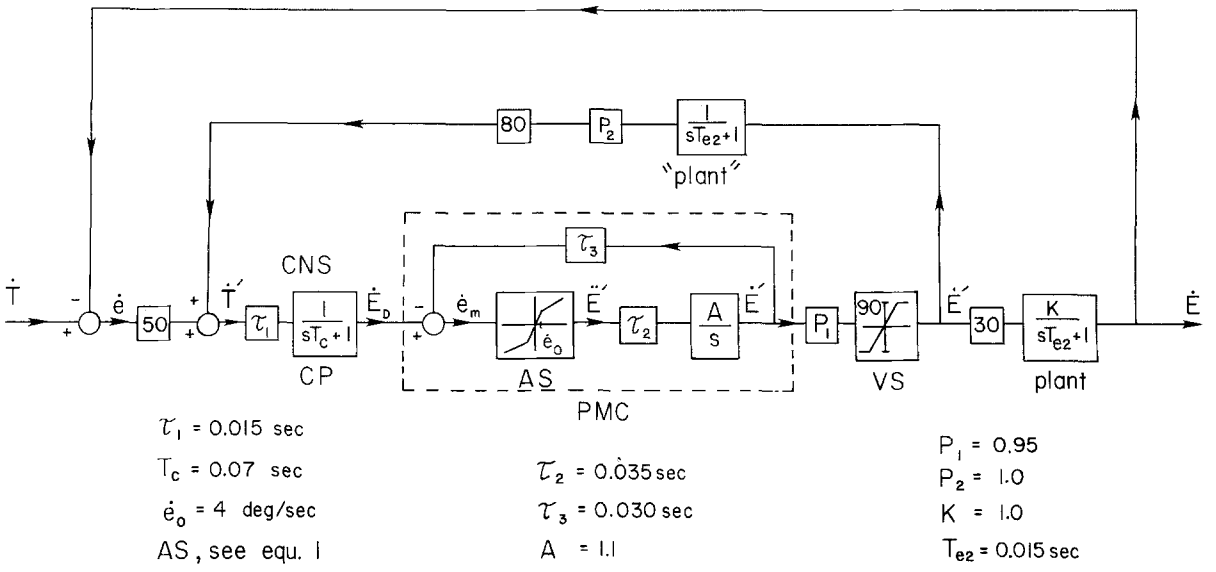

Fig. 8. The final version of the model and the parameters used. CP, central processing felt to reflect activity of the central nervous system, CNS; PMC, premotor circuitry; AS, acceleration saturation; VS, velocity saturation; K, gain of plant; τ_1, τ_2, τ_3, internal delays; T_c, time constant of CP; $\dot{E}_D$, desired eye velocity; $\dot{e}_m$, motor error; $\dot{e}_0$, break in nonlinearity AS; $\dot{E}'$, desired eye acceleration. Other symbols as in Fig. 7

the frequency of ringing as described above. This frequency is too small.

If the delay of 80 ms in the positive feedback path is changed to 60 or 40 ms, ringing occurs but at two frequencies, a basic one at about 2.0 Hz probably due to the average delay around the two loops that is a little less than 130 ms with an oscillation at a much higher frequency riding atop it that probably is connected with the difference in delays around the two loops. The latter was so small that it would be unobservable in experimental records. The main reason for rejecting this approach is that the main ringing frequency of 2 Hz is too low.

There is another problem with these two ways of producing ringing: they still leave the system linear. The amplitude of the ringing does not rise linearly with steady-state eye velocity. From Table 1 the amplitude of the first velocity overshoot above steady-state velocity was 2.3 deg/s for a 5 deg/s target and rose to only 4.7 deg/s for a 30 deg/s target. This is an increase of only 2.0 for a 6 fold increase in target speed. This means that the ringing must be created in some part of the circuit where there is a saturating nonlinearity. Only two nonlinearities are known for pursuit. One is a velocity saturation at 90 deg/s (Meyer et al. 1985). This is clearly not involved with the observed ringing which shows nonlinearities at much lower velocities. The other is the acceleration saturation.

The only signal in Fig. 7D proportional to eye acceleration is retinal slip, $\dot{e}$. It is a velocity error and is consequently a command to change eye velocity; that is, accelerate the eye. If, however, a saturating nonlinearity were added in the $\dot{e}$ pathway, the model would fail to track correctly because it could not reconstruct the signal $\dot{T}'$. If, for example, $\dot{T}$ were 20 deg/s but, in the first 130 ms, before the eye began to move, $\dot{e}$ were only 10 deg/s because of a saturating nonlinearity, the command $\dot{E}'$ would initially be only 10 deg/s and the eye would aim for this velocity over the next 130 ms. This simply does not happen. With the topology of this model, there must be no nonlinearities in the signals leading to the estimate of $\dot{T}'$ or the system could not make a correct, initial estimate of target speed which it clearly does. One must not confuse this situation with the optokinetic system where nonlinearities in the $\dot{e}$ pathway are clearly present (e.g. Demer 1981; Zee et al. 1982).

To add an acceleration nonlinearity to Fig. 7D it must be modified, subsequent to $\dot{T}'$, to provide a signal proportional to eye acceleration. Figure 8 shows one way to create such a signal. Reconstructed target velocity, $\dot{T}'$, is declared by central processing (CP) to be desired eye velocity $\dot{E}_D$. A premotor circuit, PMC, compares current eye velocity $\dot{E}'$ to $\dot{E}_D$ to create a motor error $\dot{e}_m$ that is amplified to drive $\dot{E}'$ closer to $\dot{E}_D$. The principal point is that $\dot{e}_m$ is proportional to a desired change in eye velocity so it is proportional to acceleration. The nonlinearity AS will therefore provide an acceleration saturation.

This is a major complication of the model and was included reluctantly. Given the topology of Fig. 7D, any modification that permitted an acceleration saturation would have been major and the question was not whether a major revision was necessary but which one.

The one adopted was suggested by an analogy with the saccadic system. Robinson (1975) proposed that current eye position (from the output of the neural integrator in Fig. 5A) be compared to desired eye position, derived from higher centers, to create a motor error. The latter appeared as the output of burst neurons which drove the eye quickly to the desired position thereby creating a saccade. A major virtue of this scheme is that a single velocity nonlinearity in the burst neurons could account for a velocity saturation seen in saccades. Peak eye velocity does not increase linearly with saccade size – a feature called the velocity-amplitude relation (e.g. Boghen et al. 1974) or main sequence (Bahill et al. 1975).

This hypothesis, expounded most fully in van Gisbergen et al. (1981), has been used to account for clinical abnormalities (Zee and Robinson 1979) and has received support from physiological studies (Mays and Sparks 1980; Jürgens et al. 1981). It is, of course, still an hypothesis but, while various improvements have been suggested (Jürgens et al. 1981; Fuchs et al. 1985) the basic idea of local negative feedback has consistently been supported. The hypothesis is a variation of a general hypothesis in motor physiology in which final, desired limb position is decided and the appropriate signals sent out to bring the limb to this final end point. The proposal for PMC in Fig. 8 is another example of this general hypothesis. It is exactly analogous to the local-feedback model for saccades with position variables changed to velocities and velocity variables changed to accelerations. In the same way, a single nonlinearity (AS), can produce the major nonlinearity of the pursuit system, the acceleration-velocity relationship; the main sequence equivalent for pursuit. Thus, the local feedback hypothesis proposed in Fig. 8 is not novel but, whether right or wrong, has considerable precedence and historical background.

Three other items have been included in Fig. 8. The conversion from $\dot{T}'$ to $\dot{E}_D$ by the central nervous system is unlikely to be instantaneous so a lag of time constant T_c has been included. The central delay of 50 ms may lie partly in PMC so it has been assigned delay τ_2 with τ_1 left in central processing and the understanding that τ_1 plus τ_2 equal 50 ms. Saccadic oscillations, normal and pathological, have been simulated by delays in the local, negative feedback loop on the basis that any neural pathway has delays. This is represented by τ_3. A velocity saturation, VS, has been included for completeness but since it is only important above 90 deg/s, it does not affect the present simulations and can be ignored.

Given the topology of Fig. 8, picking appropriate values for the constants is fairly straightforward. As already indicated, T_{e2} was taken at 15 ms from Keller (1973). P_1 was set at 0.95 to establish mean, steady-state gain. Consequently, P_2 equals 1.0. The steady-state gain of the innermost feedback loop is 1.0 because it has an integrator in its forward path so that overall gain is established by P_1. Other possibilities would work but this seemed simplest. The frequency of ringing, 3.8 Hz, demands that τ_2 plus τ_3 be 65 ms. To share synaptic delays between the forward and feedback paths, τ_2 was taken as 35 ms, τ_3 as 30 ms. Since τ_1 plus τ_2 equalled 50 ms, τ_1 was 15 ms. The acceleration nonlinearity, AS, is given by Fig. 3C. The four data points lie on a straight line but it was assumed that below some retinal slip velocity, $\dot{e}_0$, the function fell along a straight line to the origin. Thus, the input-output relation of AS is

$$\begin{aligned} \ddot{E}' &= 40 + 5\dot{e}_m \qquad \dot{e}_m > \dot{e}_0 , \\ \ddot{E}' &= \left(5 + \frac{40}{\dot{e}_0}\right)\dot{e}_m \qquad \dot{e}_m \leqq \dot{e}_0 . \end{aligned} \tag{1}$$

Theoretically, A should be 1.0 so as not to interfere with the values for $\dot{e}_m$ larger than $\dot{e}_0$ given in Fig. 3C. Around the origin of AS, where all ringing occurs, the loop gain $\left(5 + \frac{40}{\dot{e}_0}\right)$ can become quite large as $\dot{e}_0$ decreases so that the intensity of ringing (not the frequency) can be independently controlled. Finally, the time constant T_c was manipulated to control the initial rate of rise of acceleration. The only trade-offs left in this model were A, $\dot{e}_0$ and T_c which all influenced, in different ways, the initial rise of $\dot{E}$, the amplitude of ringing and its rate of damping. Values that gave satisfactory results are shown in Fig. 8.

Figure 9 shows the results of simulation using these values in the topology of Fig. 8. A best fit was attempted for 20 deg/s and full reliance on the acceleration data in Fig. 3C was used, via AS, for other velocities. The match was considered good enough. Obviously one could include mild nonlinearities in a

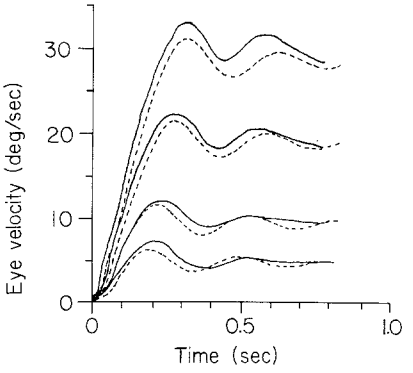

Fig. 9. Computer simulation *(dashed lines)* of the experimental curves *(solid lines)* copied from Fig. 3B for the target velocities 5, 10, 20, and 30 deg/s using the model of Fig. 8

number of parameters in Fig. 8 (e.g. A, τ_2, T_c) causing them to depend on $\dot{T}'$ but this would address trivial issues and obscure the major phenomena which are well simulated. There is one general trend evident in the mismatches of Fig. 9 already mentioned: when the subject knows when or where the target will stop, eye velocity begins to slow down well before that point. At 20 and 30 deg/s this caused a steady drop in the velocity about which the system was ringing throughout the period examined (0.8 s). This could have been simulated by creating an overshoot and decline in $\dot{E}_D$ coming from CP in Fig. 8. It was felt, however, that such an embellishment would not, at this stage, add anything to the usefulness of the simulation.

Motor Plasticity. An important correlate of effectively removing negative feedback in the model was that parametric adaptive control be present as demonstrated by Optican et al. (1985) to maintain orthometria. These authors took advantage of the opportunity offered by patients with one horizontal rectus muscle partially paralyzed. For clarity, consider a right abducens nerve paralysis. When the patient patches this eye, the normal eye is reinforced by normal pursuit which in terms of Fig. 8 means establishing P_1 at 0.95 (ideally) and P_2 at 1.0. If the patient is forced by a patch to look out of the right eye for one week, all movements to the right, especially in the right hemifield will initially be too slow. This may be simulated in Fig. 8 by decreasing the plant gain, K, by, say, 25%. Here we neglect, for simplicity, the continuously changing dependence of K on the position of the palsied eye and assume that over some oculomotor range of interest to the right, K is around 0.75. If, ideally, the CNS completely compensated for this dysmetria it would increase P_1 to 1.27 (0.95/0.75) in this range and decrease P_2 to 0.75 to simulate the weak plant. The result would be that with this eye viewing, the net gain G_0, would become normal (0.95) in this range, although this idealistic result was usually not completely achieved.

The increased innervation to the right eye for rightward pursuit could be measured by suddenly switching the patch back to the right eye and asking the left eye to pursue. In the model, this means returning K to 1.0 while leaving P_1 at 1.27 and P_2 at 0.75. The gains (from $\dot{E}'$ to $\dot{T}'$) around the inner and outer loops are now 0.95 (1.27×0.75) and 1.27 (1.27×1.0) respectively and this mismatch will cause pronounced ringing in the model and even sustained oscillations if the mismatch is great enough. These phenomena were just what were seen experimentally (Optican et al. 1985).

There was, however, a problem with this part of the simulation. According to Optican et al. (1985), the frequency of these oscillations was around 3 Hz. In the model, however, the frequency of ringing dropped from 3.8 Hz as the mismatch in P_1 and P_2 increased. When P_1 reached 1.4, the frequency was 2.5 Hz and tended toward a limit of 2 Hz as P_1 approached or exceeded 2.0 (a 50% drop in K). The reason has already been given: the system is equivalent to one with a single negative feedback path with a gain of $1 - P_2$ and a total loop delay of 130 ms and will ring at a frequency for which this delay produces a phase lag of about 90 deg since in this frequency range the other elements (CP, PMC, Fig. 8) will have produced the other 90 deg to create the requisite total loop phase lag of 180 deg.

Qualitatively, then, the model simulates motor learning or motor plasticity as observed experimentally in the pursuit system. How serious the quantitative mismatch is remains to be seen. It would be important to know the frequency of ringing in the patients in the normal eye when it was the experienced eye to see if it differed significantly from the frequency of the exaggerated ringing when the palsied eye was experienced. It would also be of interest to measure pursuit latency in these patients.

4 Discussion

We feel that to a reasonable extent the model was driven by the data. We are convinced that models such as that shown in Fig. 6 cannot do the job but cannot, of course, say that using internal positive feedback (Fig. 7D) is the only solution to that problem or even that it is the simplest. Once committed to positive feedback, the use of local feedback (PMC, Fig. 8) seems a reasonable, but not the only, way to add an acceleration saturation so the major hypothesis remains the use of positive feedback. We are a long way from any neurophysiological support for this hypothesis. Suzuki et al. (1981) have discovered neurons in the cerebellar vermis that appear to carry the signal $\dot{T}'$; they modulated their discharge rates in phase with a sinusoidally-moving target whether or not the monkey tracked it but other studies (e.g. Suzuki and Keller 1983; Mustari et al. 1984) find such bewildering combinations of $\dot{e}$ and $\dot{E}'$ signals in the pons and cerebellum that it would be premature to say that the scheme in Fig. 8 was supported by them. This scheme is mainly presented as a target for further research.

Pursuit Offset. Our data show that the eye comes to rest in a manner very different from when it responds to pursuit and is compatible with the idea that when target motion is detected, the pursuit system (whether or not represented by Fig. 8) is turned on and when the target is seen to stop, it is turned off and replaced by fixation. There are two other observations of which we are aware that support this idea. Some cells in the

parietal cortex of trained monkeys have discharge rates that increase during the state of fixation but not in the state of pursuit while others do just the opposite (Lynch et al. 1977). Also, D.S. Zee and one of us (D.A.R.) have found three patients with normal fixation yet their eyes break into oscillations similar to those seen in congenital nystagmus whenever they try to pursue (unpublished observations). These fragmentary clues are less compelling than the observations of this study (Fig. 4) which we believe are the best to date to support the idea that fixation is not pursuit at zero velocity.

It is possible, of course, that a serious, overlooked nonlinearity might exist in the pursuit system that causes it to react one way to acceleration and another to deceleration. Preliminary studies in collaboration with A.E. Luebke (unpublished observations) indicate that this is not the case. If borne out, it means that there is something very special about the velocity 0 deg/s and the system that tracks it is not the pursuit system. In the model of Fig. 8, one would wish to turn the pursuit off somewhere within the innermost negative feedback loop since it is this that creates the ringing seen at onset but not offset. In the saccadic system, burst neurons are inhibited by pause neurons when saccades are not wanted and, in analogy, it may be that pursuit is turned off by inhibition of neurons that mediate the forward path AS in Fig. 8. No attempt was made to model pursuit offset since that would appear to involve a fixation system that lay beyond the range of this study.

Another test of the model is what happens if, during pursuit, the target disappears and the subject is left in darkness (branch $\dot{e}$ in Fig. 8 opens suddenly). We did not attempt to simulate this because we are convinced that the result is greatly influenced by instructions to the subject. Becker and Fuchs (1985) showed that if a subject knew the pattern of target motion and was told to imagine it and continue to track it after it disappeared, the eyes could be kept going for several seconds at about 60% of prior velocity. We suspect that if a subject were told to imagine a stationary spot and fixate it when the target disappeared, eye velocity would go to zero very quickly. We would guess that in the former case a substitute value for $\dot{T}'$ was handed down to the model in Fig. 8 from perceptual centers and in the latter case, the pursuit system would probably just be shut off. It is possible to give no instructions to a subject but it is probably impossible to keep the subject from establishing some expectation, consciously or otherwise, of what one is supposed to do when the target disappears. We are unaware of experiments that control for this and feel that no data base exists for such a simulation.

If one does open the $\dot{e}$ pathway in the model, $\dot{E}$ decays very slowly to zero because of the perseverating action of the positive feedback loop but we doubt that the parameters of the system will remain unchanged for very long in the absence of any visible target. A better paradigm to consider is opening the loop by electronic feedback that allows eye motion to drive target motion. When this is done in the model the result is the same; eye velocity continues because of the positive feedback. If, for example, P_1 and P_2 were both 1.0, creating perfect tracking, $\dot{E}$ would continue unchanged indefinitely because unity positive feedback essentially converts the system into a perfect integrator. Such behavior has been seen in trained monkeys (Morris and Lisberger 1983). If, as in humans, tracking is less than perfect, $\dot{E}$ will decrease when the loop is opened, more rapidly as the product P_1P_2 decreases but, again, we are unaware of any systematic studies of this phenomenon in humans.

Pursuit Latency. The latency found in this study, 167 ms, is long compared to older reports of 130 ms (Robinson 1965) and a recent report of 100 ms (Gellman and Carl 1985; Carl and Gellman 1985). The latter authors have found that the Rashbass step-ramp used in this study is a mixed blessing; it may eliminate the initial catch-up saccade but the step itself apparently causes a pursuit response in the direction of the step that delays the response in the other direction to the ramp. These authors report that the response to the step begins after 100 ms and an additional 60 ms passes before acceleration reverses to the ramp direction thus causing an apparent latency of 160 ms. The response to the step is very small creating velocities on the order of 1.0 deg/s or less that were not visible in our recordings. Thus, the step appears to simply increase the latency to pursuit.

A latency of 130 ms was used in the simulations simply because this value has been commonly accepted for so long. It may well be an overestimate since it was often determined from a position rather than a velocity trace and, of course, the latter would be more accurate. It is important that the model tolerate a latency of 100 ms. To accommodate it, one should recognize that 50 ms is a generous estimate for the retinal delay and, for the luminance levels of targets usually used, could be dropped to 40 ms. Similarly a motor delay of 30 ms is generous; stronger stimulation of, say, the cerebellum can create eye movements in less than 20 ms (Ron and Robinson 1973). Using these values leaves 40 ms for central processing and could be accommodated in Fig. 8 by decreasing τ_1 to 5 ms. It should be noted that decreasing the latency to 100 ms will not make it easier to achieve ringing at 3.8 Hz. Using the rough criteria that ringing occurs when the phase of the total delay is 90 deg, decreasing the delay from 130 to 100 ms, only raises the frequency from this source from 2.0 to 2.6 Hz.

Thus, one still needs to suppress oscillations from this source (by effectively removing the feedback) and create another component, such as PMC, capable of ringing at 3.8 Hz.

The observation that the latency for pursuit offset was 30 ms less than for onset is, we think, new. Offset was also done in a Rashbass manner (Fig. 1). Carl and Gellman (1985), however, report that acceleration due to forward steps during pursuit is much lower than to backward steps so it is quite possible that such considerations, associated with the use of a step, could account for the decrease in latency without attributing it to an intrinsic property of the pursuit system.

Retinal Position Error. Wyatt and Pola (1981) showed conclusively what had long been suspected, that the pursuit system responded to the position of images on the retina, relative to the fovea, as well as their velocity. Gellman and Carl and Carl and Gellman (1985) report accelerations to target position steps on the order of 10 deg/s^2 whereas, in this study, we are concerned with much larger accelerations (Table 1) and so felt justified in ignoring the position response, at least initially. It should be noted from the start, however, that any model that responds literally to position error, must have a steady-state velocity gain of 1.0. In any of the velocity models considered here, the inclusion of a position error, e, as an input requires an integrator in the forward path (to change $\dot{e}$ to e) and this requires that $\dot{e}$ be zero in the steady state. Since this does not happen, we cannot take this proposal literally.

It is well known that a jumping target can appear to move smoothly if the jumps are small enough and frequent enough, as in motion pictures. Whatever filters this sampled motion into a smooth interpretation would be stimulated, although feebly, by a target that jumps only once. The finding of Gellman and Carl (1985) that the response of the step decreases as step size increases is compatible with this notion since the larger the step the less reasonable it is to interpret it as the start of a smooth motion. Consequently, we would propose that the response to a step is not due to a position error but to a misinterpretation of the step as a velocity impulse – a very different situation.

Nevertheless, the pursuit system does respond to error position as shown clearly by Wyatt and Pola (1981) and others. Pursuit of a slightly eccentric after-image is one simple demonstration. The latencies and accelerations of such movements, separated from the step-stimulus response, have not been well studied. Introspection with after-images suggest it is slow. Wyatt and Pola found that predictability of target behavior and experience improved the response to position errors. These considerations make it uncertain whether there is much of a role for such responses in the experiments reported here where retinal velocity errors were large and probably dominated behavior.

Acknowledgements. We thank A.L. McCracken for preparing the manuscript and C.V. Bridges for the illustrations. A.G. Lasker provided essential technical assistance. This research was supported by Grants EY00598 (D.A.R.), EY07047 (S.E. Gordon), computer facilities were supported in part by Grant EY01765 from the National Eye Institute, National Institutes of Health, U.S. Public Health Service.

References

Bahill AT, Clark RM, Stark L (1975) The main sequence, a tool for studying human eye movements. Math Biosci 24:191–204

Becker W, Fuchs AF (1985) Prediction in the oculomotor system: smooth pursuit during transient disappearance of a visual target. Exp Brain Res 57:562–575

Boghen D, Troost BT, Daroff RB, Dell'Osso LF, Birkett JE (1974) Velocity characteristics of normal human saccades. Invest Ophthalmol 13:619–623

Cannon SC, Robinson DA (1986) The final common integrator is in the prepositus and vestibular nuclei. In: Keller EL, Zee DS (eds) Adaptive processes in eye movements and vision. Pergamon Press, Oxford (in press)

Carl JR, Gellman RS (1985) Human smooth pursuit: the response to conflicting velocity and position stimuli. Soc Neurosci Abstr, vol 11, part 1, p 78

Demer JL (1981) The variable gain element of the vestibulo-ocular reflex is common to the optokinetic system of the cat. Brain Res 229:1–13

Fuchs AF, Luschei ES (1970) Firing patterns of abducens neurons of alert monkeys in relationship to horizontal eye movement. J Neurophysiol 33:382–392

Fuchs AF, Kaneko CRS, Scudder CA (1985) Brain stem control of saccadic eye movements. Annu Rev Neurosci 8:307–337

Gisbergen JAM van, Robinson DA, Gielen S (1981) A quantitative analysis of the generation of saccadic eye movements by burst neurons. J Neurophysiol 45:417–442

Gellman RS, Carl JR (1985) Human smooth pursuit: early responses to sudden changes in target velocity. Soc Neurosci Abstr, vol 11, part 1, p 79

Gonshor A, Melvill Jones G (1976) Extreme vestibulo-ocular adaptation induced by prolonged optical reversal of vision. J Physiol (London) 256:381–414

Holst E von, Mittelstaedt H (1950) Das reafferenzprincip. Naturwissenschaften 37:464–476

Ito M (1982) Cerebellar control of the vestibulo-ocular reflex – around the flocculus hypothesis. Annu Rev Neurosci 5:275–296

Jürgens R, Becker W, Kornhuber HH (1981) Natural and drug-induced variations of velocity and duration of human saccadic eye movements: evidence for a control of the neural pulse generator by local feedback. Biol Cybern 39:87–96

Keller EL (1973) Accommodative vergence in the alert monkey. Vision Res 13:1565–1575

Lisberger SG, Fuchs AF (1978) Role of primate flocculus during rapid behavioral modification of vestibuloocular reflex. I. Purkinje cell activity during visually guided horizontal smooth-pursuit eye movements and passive head rotation. J Neurophysiol 41:733–763

Lisberger SG, Evinger C, Johanson GW, Fuchs AF (1981) Relationship between eye acceleration and retinal image velocity during foveal smooth pursuit in man and monkey. J Neurophysiol 46:229–249

Lynch JC, Mountcastle VB, Talbot WH, Yin TCT (1977) Parietal lobe mechanisms for directed visual attention. J Neurophysiol 40:362–389

Mays LE, Sparks DL (1980) Saccades are spatially, not retinocentrically coded. Science 208:1163–1165

Meyer CH, Lasker AG, Robinson DA (1985) The upper limit of human smooth pursuit velocity. Vision Res 25:561–563

Michael JA, Melvill Jones G (1966) Dependence of visual tracking capability upon stimulus predictability. Vision Res 6:707–716

Miles FA, Fuller JH (1975) Visual tracking and the primate flocculus. Science 189:1000–1002

Morris EJ, Lisberger SG (1983) Signals used to maintain smooth pursuit eye movements in monkeys: effects of small retinal position and velocity errors. Soc Neurosci Abstr, vol 9, part 2, p 866

Mustari MJ, Fuchs AF, Wallman J (1984) Smooth-pursuit-related units in the dorsolateral pons of the rhesus macaque. Soc Neurosci Abstr, vol 10, part 2, p 987

Newsome WT, Wurtz RH, Dürsteler MR, Mikami A (1985) Deficits in visual motion processing following ibotenic acid lesions of the middle temporal visual area of the macaque monkey. J Neurosci 5:825–840

Optican LM, Zee DS, Chu FC (1985) Adaptive responses to ocular muscle weakness in human pursuit and saccadic eye movements. J Neurophysiol 54:110–122

Rashbass C (1961) The relationship between saccadic and smooth tracking eye movements. J Physiol (London) 159:326–338

Robinson DA (1965) The mechanics of human smooth pursuit eye movement. J Physiol (London) 180:569–591

Robinson DA (1972) Eye movements evoked by collicular stimulation in the alert monkey. Vision Res 12:1795–1808

Robinson DA (1975) Oculomotor control signals. In: Lennerstrand G, Bach-y-Rita P (eds) Basic mechanisms of ocular motility and their clinical implications. Pergamon Press, Oxford, pp 337–374

Robinson DA (1976) Adaptive gain control of vestibuloocular reflex by the cerebellum. J Neurophysiol 39:954–969

Ron S, Robinson DA (1973) Eye movements evoked by cerebellar stimulation in the alert monkey. J Neurophysiol 36:1004–1022

Schalén L (1980) Quantification of tracking eye movements in normal subjects. Acta Otolaryngol 90:404–413

Sperry RW (1950) Neural basis of spontaneous optokinetic response produced by visual inversion. J Comp Physiol Psychol 43:482–489

Steinbach MJ (1969) Eye tracking of self-moved targets: the role of efference. J Exp Psychol 82:366–376

Steinbach MJ (1976) Pursuing the perceptual rather than the retinal stimulus. Vision Res 16:1371–1376

Suzuki DA, Keller EL (1983) Sensory-oculomotor interactions in primate cerebellar vermis: a role in smooth pursuit control. Soc Neurosci Abstr, vol 9, part 1, p 606

Suzuki DA, Noda H, Kase M (1981) Visual and pursuit eye movement-related activity in posterior vermis of monkey cerebellum. J Neurophysiol 46:1120–1139

Wyatt HJ, Pola J (1981) Slow eye movements to eccentric targets. Invest Ophthalmol Vis Sci 21:477–483

Yasui S, Young LR (1975) Eye movements during after-image tracking under sinusoidal and random vestibular stimulation. In: Lennerstrand G, Bach-y-Rita P (eds) Basic mechanisms of ocular motility and their clinical implications. Pergamon Press, Oxford, pp 509–513

Young LR (1971) Pursuit eye tracking movements. In: Bach-y-Rita P, Collins CC, Hyde JE (eds) Control of eye movements. Academic Press, New York, pp 429–443

Young LR (1977) Pursuit eye movements – what is being pursued? In: Baker R, Berthoz A (eds) Control of gaze by brain stem neurons. Elsevier, Amsterdam, pp 29–36

Young LR, Forster JD, van Houtte N (1968) A revised stochastic sampled data model for eye tracking movements. Fourth Ann NASA – University Conference on Manual Control, University of Michigan, Ann Arbor, Michigan

Zee DS, Robinson DA (1979) An hypothetical explanation of saccadic oscillations. Ann Neurol 5:405–414

Zee DS, Butler PH, Optican LM, Tusa RJ, Gücer G (1982) Effects of bilateral occipital lobectomies on eye movements in monkeys: preliminary observations. In: Roucoux A, Crommelinck M (eds) Physiological and pathological aspects of eye movements. Junk, The Hague, pp 225–232

Received: April 16, 1986

Dr. David A. Robinson
Room 355 Woods Research Bldg.
The Wilmer Institute
The Johns Hopkins Hospital
601 N. Broadway
Baltimore, MD 21205
USA