Kybernetik 14, 71—83 (1973)

Models of the Saccadic Eye Movement Control System

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Received: May 14, 1973

Abstract

1. A sequence of four models is proposed for the saccadic eye movement control system. The models become increasingly complex as they are made to respond to increasingly more complicated target movements in accordance with experimental results. Compatibility with neurological structure and function is stressed in the formation of the models. In each case, the elements of the models are constructed to conform as closely as possible to neuroanatomical structures and behave in a way that has been established or suggested by neurophysiology.

2. The dynamic behavior of the mechanics of the extraocular muscles and eyeball suspensory tissues has been established by recording from oculomotoneurons in alert monkeys. The transfer function of this mechanical system is used in these models.

3. Recent experiments on the neural circuits in the brain stem that are responsible for saccadic eye movements suggest an arrangement of the premotor circuitry that contains two principal neural networks; an integrator and a pulse generator. This circuitry is used in the models.

4. When the above modifications are made to existing models of the saccadic system, they remove the necessity of supposing that the visual information is sampled by the nervous system. The models do not include a sampler although the saccadic pulse generator still makes the overall system behavior similar to that of a sampled-data system.

5. The basic model is modified to make its behavior agree with experimental eye movement responses to target ramps and step-ramps. This is done by using error and its rate of change to estimate the error that will exist one reaction time in the future.

6. Parallel processing of data is a well recognized property of the nervous system. By utilizing it in combination with a random decision threshold, the model is extended to produce results in agreement with experiments for double-step target movements in which the second step occurs less than 0.2 sec after the first.

7. Finally, a model is presented which incorporates a continuum of parallel processing to represent the retinotopic spatial organization of the visual system and the tecto-bulbar motor commands. The model is conceptual; it was not constructed or tested but is used to discuss more complex eye movement phenomena such as those that appear to occur when the decision process must shift between hemispheres and how the system might produce quick correcting saccades with latencies as short as 85 msec.

Zusammenfassung

1. Es wird eine Folge von vier Modellen für das System der sakkadischen Augenbewegungen vorgeschlagen. Die Modelle werden von Stufe zu Stufe komplexer und bilden die experimentell gefundenen Antworten auf zunehmend kompliziertere Zielbewegungen nach. Bei der Konzeption der Modelle wird der Akzent auf Vereinbarkeit mit den strukturellen und funktionellen Gegebenheiten der Neurologie gelegt. In jeder Stufe werden die Elemente dieser Modelle so gewählt, daß sie möglichst genau neuro-anatomischen Strukturen antsprechen und daß ihr Verhalten sich mit dem neurophysiologisch nachgewiesenen oder wahrscheinlich gemachten deckt.

2. Durch Ableitung von oculomotorischen Neuronen beim wachen Affen wurde die Dynamik des mechanischen Systems, bestehend aus den äußeren Augenmuskeln und dem Bindegewebe, in dem der Augapfel gelagert ist, erfaßt. Die Übergangsfunktion dieses Systems ist in die Modelle eingearbeitet.

3. Neuere Untersuchungen an den Strukturen des Hirnstamms, die für sakkadische Augenbewegungen verantwortlich sind, lassen im prämotorischen Apparat eine Anordnung vermuten, die im wesentlichen zwei neuronale Netzwerke enthält: einen Integrator und einen Pulsgenerator. Diese Schaltungen werden in den Modellen verwandt.

4. Nach Einarbeitung der obengenannten Änderungen in bestehende Modelle des sakkadischen Systems wird die Annahme, daß die visuelle Information durch das Nervensystem diskontinuierlich abgetastet wird, überflüssig. Die Modelle enthalten keine Abtastung, obwohl in Folge des sakkadischen Pulsgenerators ihr Verhalten als Ganzes noch immer das einer getasteten Regelung ist.

5. Das Grundmodell wird so modifiziert, daß sein Verhalten mit den Augenbewegungen übereinstimmt, die experimentell als Antwort auf kombinierte Sprung- und Rampenbewegungen des Ziels gefunden werden. Dies geschieht, indem der nach einer Reaktionszeit zu erwartende Fehler aufgrund des Momentanfehlers und seines Differentialquotienten geschätzt wird.

6. Parallele Datenverarbeitung ist eine allgemein bekannte Eigenschaft des Nervensystems. Durch Kombination dieser Eigenschaft mit einer zufälligen Entscheidungsschwelle wird das Modell so erweitert, daß es sich mit den experimentellen Befunden auch bei solchen Doppelsprüngen des Ziels deckt, bei denen der Sprungabstand kleiner als 0.2 sec ist.

7. Abschließend wird ein Modell vorgestellt, das ein Kontinuum von parallelen Datenverarbeitungskanälen einschließt und damit die retinotope räumliche Organisation des visuellen Systems sowie die tecto-bulbären motorischen Signale nachbildet. Es handelt sich hierbei um ein Denkmodell, das weder realisiert noch geprüft wurde. Es wird vielmehr dazu verwendet, komplexere Formen von Augenbewegungen zu diskutieren, wie sie z. B. aufzutreten scheinen, wenn der Entscheidungsprozeß zwischen den Hemisphären wechseln muß. Ebenso wird erörtert, wie das System schnelle Korrektursakkaden auslösen kann, deren Latenzen bis zu 85 msec kurz sind.

1. Introduction

In 1963, Young and Stark developed a sampled-data model of the nonpredictive saccadic eye movement system. The elements of that model were not intended to resemble actual brain structures because so little was known at that time about the brain stem organization of eye movements. Much remains unknown but recent techniques which allow us to record from single neurons in the brain stems of behaving monkeys has given us a few firm facts and strongly suggested others. Consequently, it seems worth-while to modify the model of the saccadic system to incorporate these findings. When this is done, surprisingly enough, it appears to be unnecessary to hypothesize that the brain samples visual data before processing it.

The model of Young and Stark (1963) also did not predict the correct experimental responses when the target moved in a ramp, a step-ramp and in two steps in rapid succession. The revised model proposed here is modified to obtain correct responses to these classes of inputs. These modifications are also compatible with neurophysiology and the general belief that parallel data processing is an important and powerful property of brain function.

2. Model I

The model of Young and Stark (1963) is shown in Fig. 1A. Their description of the sampler (M) indicated that so long as the error exceeded a certain threshold, the sampler would operate every 0.2 sec, but, when the error was below threshold, the sampler would stop until the threshold was once more exceeded. This timing regimen is made explicit in Fig. 1A by adding a timing circuit which now contains the dead zone DZ. When the error e exceeds the threshold e_t, the pulse generator PG is triggered which causes a sample to be taken. At the same time the $INHBT$ element blocks DZ for 0.2 sec. Consequently, so long as $|e| \geqq e_t$, samples will occur every 0.2 sec. When $|e| < e_t$, the sampler will stop. The model is thus completely equivalent to that of Young and Stark.

The Plant

The model of the extraocular muscle and eyeball mechanics in Fig. 1A, proposed by Westheimer (1954), is incorrect. It is underdamped and produces a saccade-like output for a step input. The actual plant is overdamped and is driven by a pulse-step to produce a saccade. While several models have been proposed for the plant based on mechanical experiments

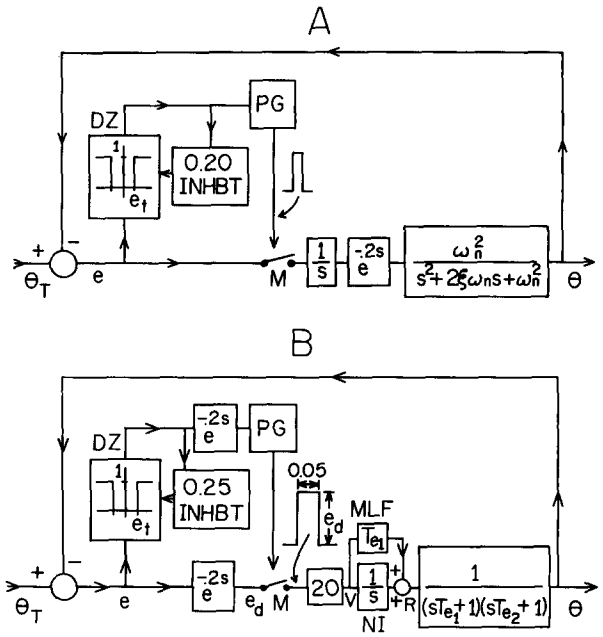

Fig. 1. A The model of Young and Stark (1963). The only modification is to make the timing circuit explicit according to their description. DZ threshold element with dead zone. PG pulse generator to actuate sampler, M. $INHBT$ a device to inhibit the timing circuit for 0.2 sec. θ_T is target position, θ is eye position, ω_n is 240 rad/sec, ζ is 0.7. B A modification of A which incorporates the correct plant model and premotor circuits suggested by physiology. NI, neural integrator; MLF, medial longitudinal fasciculus. PG and M create a pulse to make saccades

(Robinson, 1964, 1965; Childress and Jones, 1967; Thomas, 1969) and theoretical considerations (Cook and Stark, 1967, 1968), the only certain way to find the relationship between eye movement and motor nerve activity is to either control or monitor the latter. Zuber (1968) stimulated the oculomotor nerves of cats and obtained the transfer function,

$$\frac{\theta}{S} = \frac{1}{sT_e + 1} \tag{1}$$

where θ is eye position, S is the stimulus rate and s is the Laplace transform variable. This result was confirmed by recording the firing rate R of individual oculomotoneurons in the behaving monkey (Robinson, 1970; Robinson and Keller, 1972). The result was,

$$R = k(\theta - \theta_T) + r\frac{d\theta}{dt}. \tag{2}$$

The threshold θ_T is that eye angle at which the motoneuron is recruited into the active pool (it is understood that R is non-negative). The coefficient k is the slope of the linear regression fit to many different (R, θ) points during fixation when $d\theta/dt$ is zero.

Correlation coefficients are about 0.95 (Skavenski and Robinson, 1973). The coefficient r is the slope of the linear regression fit to many $(R, d\theta/dt)$ points during smooth pursuit movements for a fixed value of θ. Correlation coefficients are about 0.90 (Skavenski and Robinson, 1973). Equation (1) is, of course, the transfer function one would obtain from (2) in relating changes in R to changes in θ. The time constant, T_e, is r/k. In view of known non-linearities in muscle mechanics, it is surprising that the experimental data is so compatible with a linear model.

The order of the plant is actually greater than two. Close examination of records (Keller and Robinson, 1972) show a dependence of R on eye acceleration,

$$R = k(\theta - \theta_T) + r\frac{d\theta}{dt} + m\frac{d^2\theta}{dt^2}. \quad (3)$$

The coefficient m is very small and the system is still heavily overdamped. Mechanical studies in man (Robinson, 1964; Childress and Jones, 1967; Thomas, 1969) agree that there is a complex pole pair in the transfer function, probably associated with globe moment of inertia and muscle series elastic elements, with a resonant frequency variously reported to lie in the range 50–87 Hz and a damping coefficient in the range 0.51–0.84. However, the effects of this are small. The noisy fluctuations in $R(t)$ (about 6% of mean rate, Keller and Robinson, 1972) prevent the detection of such a phenomenon in neurophysiological experiments and it is certainly unimportant in modelling the plant for practical purposes. Putting Eq. (3) in the form of a transfer function, one gets

$$\frac{\theta}{R} = \frac{1}{(sT_{e_1}+1)(sT_{e_2}+1)}. \quad (4)$$

Since $m \ll r \ll k$, T_{e_1} is approximately r/k, the larger time constant, and T_{e_2}, the smaller time constant, is approximately m/r.

Numerically, T_{e_2} is about 7 msec. It could be neglected without seriously affecting the main results. Its retention, in a model, does prevent eye velocity from changing instantaneously and therefore makes its saccades look more natural. There is a question about which value for T_{e_1} is appropriate for man. In Eq. (1), Zuber (1968) found T_e to be 64 msec. The mean of r/k in Eq. (2) over a population of motoneurons for the rhesus monkey is about 200 msec (Robinson, 1970; Keller and Robinson, 1972; Skavenski and Robinson, 1973). However, quick release mechanical experiments in the monkey (Keller and Robinson, 1971) gave 90 msec and in man (Robinson, 1964), approximately 150 msec. It is not known whether these differences are species differences or, as is suggested in the monkey, due to differences in experimental procedures. More work is clearly needed although it is very likely that T_{e_1}, for man, is bounded from below by about 100 msec and from above by 200 msec. For the present simulation, T_{e_1} was chosen as 150 msec.

Finally, it is useful to know that there is no stretch reflex in the extraocular muscles of the rhesus monkey (Keller and Robinson, 1971). There are conflicting reports about such a reflex in man (Breinen, 1957; Sears *et al.*, 1959; Maruo, 1964) but the complete failure to find this reflex in lower animals and now in a primate makes it unlikely that man alone possesses it. Its absence simplifies our model.

The most important consequence of the correct plant transfer function is the fact that, in order to overcome the viscous drag due to the $r\frac{d\theta}{dt}$ terms in Eq. (2) or (3), the input $R(t)$ required to produce a saccade is a pulse-step. This is seen dramatically in the behavior of single motoneurons (Fuchs and Luschei, 1970; Schiller, 1970; Robinson, 1970) almost all of which burst into high firing rates (400–600 spikes/sec) during saccades. The question now is, how does the premotor circuitry produce the pulse and step needed to make a saccade?

Premotor Circuitry

We shall suggest, as shown in Fig. 1B, that the premotor circuitry consists of a neural integrator, NI, in parallel with a proportional path, MLF, whose gain is T_{e_1}. This network is fed by a pulse generator PG. The area under the pulse equals the desired saccade amplitude. The direct path, MLF, creates the pulse seen in the motoneurons, R, while the integrator, in response to the pulse, produces the needed step. Analytically, the transmission from V to θ in Fig. 1B is

$$\frac{\theta}{V} = \frac{(T_{e_1}+\frac{1}{s})}{(sT_{e_1}+1)(sT_{e_2}+1)} = \frac{1}{s}\cdot\frac{1}{(sT_{e_2}+1)} \quad (5)$$

which makes it clear that, if one ignores the slight filtering added by the T_{e_2} term, eye position is the integral of the signal put in at V. Thus V may be thought of as a velocity command. If V is an approximation to a pulse, the output is an approximation to a step, that is, a saccade.

The evidence for this circuit comes from the vestibulo-ocular reflex. Below about 1 Hz, Eq. (4) indicates that, for sinusoidal stimuli, motoneuron firing rate is in phase with eye position. In this reflex, eye position is equal and opposite to head position (Skavenski and Robinson, 1973). Thus, except for a sign change, R

is in phase with head *position*. But the firing rates of first and second order vestibular neurons from the semicircular canals are in phase with head *velocity* (Fernandez and Goldberg, 1971; Melvill Jones and Milsum, 1965, 1971; Young, 1969). Consequently, there exists a neural integrator between the vestibular and oculomotor nuclei. This idea is thoroughly confirmed by neurophysiology (Fernandez and Goldberg, 1971; Skavenski and Robinson, 1973) and, at this writing, work is underway to locate this integrator anatomically. Carpenter (1972) already has evidence that it is, in part, in the cerebellum.

The integrator bypass, *MLF*, is a well known anatomical fiber tract called the medial longitudinal fasciculus. That it forms a lead network to compensate the lag of the plant is a role forced upon it by its known location in the circuit. Experimentally, no phase lag is seen in the vestibulo-ocular reflex in man (Benson, 1970) or animals (Skavenski and Robinson, 1973) at frequencies above 1 Hz so something is compensating for the plant lag. There is little reason to doubt that it is the *MLF*. Thus, the existence of the arrangement in Fig. 1B of *NI* and *MLF* rests on good neurophysiological and anatomical data.

The hypothesis that saccades are the result of a pulse input to this network is more circumstantial. It rests partly on the fact that it is the simplest arrangement. However, several investigations (Luschei and Fuchs, 1972; Sparks and Travis, 1971) have found brain stem neurons or fibers in the behaving monkey in premotor areas such as the reticular formation, vestibular nuclei and dentate nuclei that burst at high discharge rates in exact synchrony with the saccadic bursts seen in oculomotoneurons. It is difficult to dismiss the idea that these units are part of a neural pulse generator mechanism. Stimulation studies (Robinson, 1972; Robinson and Fuchs, 1969) also suggest a saccadic neural pulse generator.

Indirect evidence for a pulse generator comes from the fact that the integrator *NI must* be reset during quick phases of vestibular nystagmus. There is good evidence to suppose that saccades and quick phases are made by the same neural circuit (Ron *et al.*, 1972) so that quick phases and saccades are made by the same neural pulse generator. Passing the quick phase pulse through the integrator automatically resets it to a new level. Any alternate model must propose some necessarily more complex way of resetting the integrator. Finally, it's conceivable that resetting could be avoided by assuming two integrators, one of which accumulated slow phases only, the other accumulating quick phases only. This theory is most unlikely. Rotation of a subject for long periods can create unidirectional nystagmus of long duration. The accumulation of all slow phases in one integrator and all quick phases in another would soon lead to a condition in which each integrator had stored within it signals proportional to many complete rotations of the eye in the head. This arrangement is theoretically untenable. It is more likely, on the basis of simplicity, that there is a single integrator, shared by the vestibular and saccadic systems in the arrangement shown in Fig. 1B.

Overall Organization

If one incorporates the correct plant and the suggested premotor circuitry into the saccadic system model, one is struck by the fact that the pulse generator and neural integrator of Fig. 1B perform exactly the same sample-and-hold function as the sampler and integrator in Fig. 1A. It seems unlikely that one needs two time discrete elements, a sampler and a saccadic pulse generator. One can avoid this by interchanging the order of the delay and the sample-and-hold elements in Fig. 1A and then renaming the sample-and-hold element as the neural pulse generator and integrator. The result is to do away with the concept of sampling, or perhaps, to recognize that the motor circuitry samples the decisions of the brain's data processing rather than that the brain samples its visual input.

This rearrangement, shown in Fig. 1B, also puts the 0.2 sec delay first which seems reasonable anyway since it represents the time taken by the brain to process visual (and non-visual) information and reach a decision. The pulse is created by *PG* in the timing circuit which momentarily closes the switch *M* and creates a pulse whose amplitude depends on the delayed error e_d. The 0.2 sec delay must be placed in both the timing and amplitude branches of the circuit. A fixed representative saccade duration of 50 msec was chosen. Saccade duration is well known to be a function of saccade amplitude (Hyde, 1959; Robinson, 1964) but it was felt that to include this feature added a complexity that brought little but prettiness to the model. To make the pulse area, which is initially 0.05 e_d, equal to e_d, the desired saccade amplitude, the raw pulse is multiplied by 20. Finally, *DZ* is inhibited for 0.25 sec so that the timing circuit will not be reactivated until 0.2 sec after the end of a saccade.

Dead Zone

Actually there is no dead zone. Wyman and Steinman (personal communication) showed that

subjects can make saccadic responses to 3.5 min arc target steps. Haddad and Steinman (1973) showed that subjects can make equally small voluntary saccades. The results from Steinman's laboratory (Steinman *et al.*, 1973) indicate that the small saccades of fixation serve exactly the same purpose as large saccades; to bring fresh information to the fovea. They are voluntary and can be made or not made at will. The dead zones that have been observed are psychological and depend on the criterion unconsciously set by the subject as to what will be accepted as "looking at the target". The subject *can* respond to small target steps but will only if specifically instructed to do so. If e_t is zero, the model will make very small, frequent saccades in response to system noise but this, of course, is just what the human eye does. Consequently the choice of e_t is somewhat arbitrary and may be determined by the type of behavior to be simulated. In what follows, e_t was set at 0.24 deg.

Performance

The circuit of Fig. 1B is identical in its performance to the model of Young and Stark in Fig. 1A. Its capabilities are neither more nor less. From the standpoint of "black box" modelling, the rearrangement is irrelevant. Only from the standpoint of neurophysiological simulation is it important. The model correctly predicts the results of simple steps and of visual feedback which causes a saccadic run-away in the open loop condition and oscillations for external negative feedback (Young and Stark, 1963; Robinson, 1965). It also does not correctly predict the responses to ramps, step-ramps and double target jumps. There is no need to reproduce these responses again here.

Only one type of response is of interest at this stage. Just at the end of a saccade, the pulse has brought the eye to a new position. The job of the integrator is to hold it there. If the ratio of neural

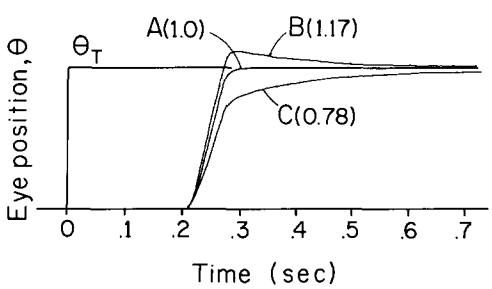

Fig. 2. Normal and abnormal saccades. Three responses are shown to a step in target position, θ_T. The gain of the *MLF* path in Fig. 1B is normal in A, is 17% too large in B and is 22% too small in C

transmissions through *NI* and *MLF* is not T_{e_1}, because of spontaneous variability, this will not happen and the eye will undershoot or overshoot and then exponentially approach a new steady-state position with a time constant of T_{e_1} (0.15 sec). The results of changing the *MLF* gain are shown in Fig. 2. These sorts of saccades are occasionally seen in eye movement records. Vossius (1960) has called attention to similar shape differences and Weber and Daroff (1972) have given the name glissade to the rising tail of curve *C*, the most common variation seen.

3. Model II

The element in Figs. 1A and B which insures a minimum intersaccadic interval of 0.2 sec is the *INHBT* element. This seems to be an artificial device without much neurophysiological significance. It is, in fact, ironic that the most sophisticated of the brain's processes is represented merely by a 0.2 sec delay. It is proposed instead that when an error occurs, it starts a process that will eventually culminate in a saccade. In Fig. 3, the variable *n*, the output of an analog low-pass filter, may be thought of as the number of completed calculations (sub-routines) needed to reach a decision. When (and if) *n* rises to the threshold level *m* of *THR*, which is set to occur normally in about 0.23 sec, the *PG* is triggered and a saccade is made. When this happens, all visual images change place on the retina and the process of calculation must start all over again from the beginning. In a digital analogy, this indicates erasing or dumping from memory all results from the previous calculation. In the analog model of Fig. 3, it requires that all capacitors in the active *RC* filter that was used be momentarily shorted out. This is symbolized by the dump command *DMP* in Fig. 3.

This arrangement accomplishes the desired goal, no saccade can occur sooner than 0.23 sec after a previous one. It also does it in a way that may be more realistic than in Figs. 1A and B. It already behaves differently from those models (assume momentarily that the part marked VELOCITY CIRCUIT is not included): if the target jumps to one side for, say, 50 msec or less, and then returns, the system in Fig. 3 will not make a saccade because $n(t)$ will never reach threshold. This is what the real system also does.

The *DEL* filters are three pole approximations to a delay line. Their transfer function is,

$$DEL(s) = \frac{1}{(1+0.6sT_d)(1+0.4sT_d+0.16(sT_d)^2)}. \quad (6)$$

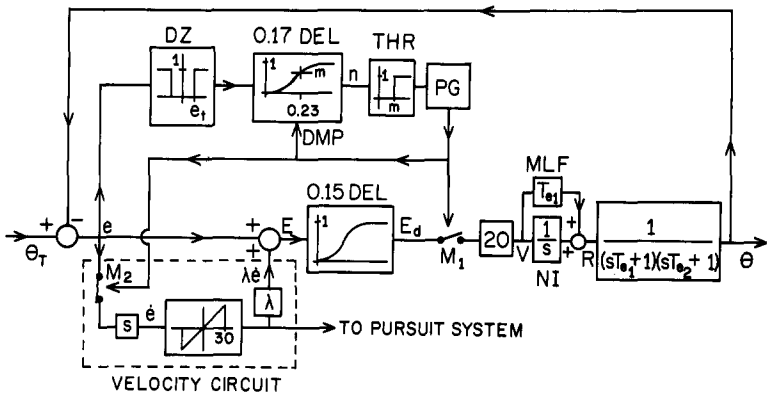

Fig. 3. A modification of Fig. 1B which achieves a 0.23 sec minimum intersaccadic interval by requiring that a decision process, $n(t)$, reach a threshold m in element *THR* to make a saccade. The decision memory is erased or dumped, *DMP*, after each saccade. *DEL* are analog approximations to delay lines. The approximate step response of these delays is indicated by the curve within each block. A velocity correction circuit for saccade amplitude, E, is included. M_2 creates saccadic suppression in the velocity circuit

T_d is 0.17 sec in the timing circuit. The threshold value m was 0.54. A similar delay line approximation with T_d equal to 0.15 sec was also used to delay the error e. This was done only for the practical reason that simulation was done on an analog computer and no pure delay lines were available.

Ramps and Step Ramps

The models of Figs. 1A and B make saccades whose amplitudes are equal to the error that existed 0.2 sec ago. But when the eye makes a saccade 0.2 sec after the start of a ramp, the error 0.2 sec ago was zero. Yet, the actual saccade is usually large enough to put the eye on target. Conversely, if the target jumps in one direction and moves back in a ramp whose velocity causes the target to recross its original position in 0.2 sec, the eye gets on target with only a smooth pursuit movement (Rashbass, 1961; Robinson, 1965). No saccade is made although the error 0.2 sec ago was large.

Clearly the brain is clever enough to lead the target as a hunter leads a moving target with his gun. The saccade is made not to where the target was but to where it will be in 0.2 sec. This amount is estimated by multiplying target velocity by 0.2 sec. Target velocity is a signal readily available for this calculation as the output of directionally selective neurons in the visual system (Oyster *et al.*, 1972; Pettigrew *et al.*, 1968) which, as suggested in Fig. 3, is also undoubtedly the signal which drives the smooth pursuit system. The predicted target error E is then simply,

$$E = e + \lambda \dot{e} \tag{7}$$

where λ should be about 0.2.

As usual, in deriving error velocity, it is necessary to simulate the fact that directionally selective visual units saturate at large values of $\dot{e}$ and cease responding altogether for very large values. This is approximated by the high velocity cut-out non-linearity shown in the velocity circuit of Fig. 3. Little is known about the actual shape of this non-linearity in foveate animals [unlike the rabbit (Collewijn, 1972)] and a 30 deg/sec cutout velocity was chosen rather arbitrarily. This non-linearity eliminates impulses associated with target steps. Interestingly, the velocity circuit (and the entire pursuit system) needed saccadic suppression to keep it from reacting to saccades themselves. A 1.5 deg saccade lasting 50 msec has a mean velocity of only 30 deg/sec; a signal which could pass into the velocity system to influence future saccades. A second modulator, M_2, which opens during saccades, removes this problem.

To produce pursuit movements, a model similar to Collewijn's (1972) was used. The signal marked "to pursuit system" in Fig. 3 was passed through the transfer function $7.5/(2s+1)$ and then through a low pass approximation of a delay line as in Eq. (6) with T_d equal to 0.15 sec to simulate the latency of the pursuit system. The output of this was added to the input of the premotor circuitry at point V. Since the open loop pursuit gain was only 7.5 (for higher gains the system approached instability), the eye tracked the target at only 88% of target velocity in the steady state.

Performance

Figure 4 shows the system's response to ramps and step-ramps. The correct value for λ was experimentally determined to be 0.17. In each case a saccade occurs in about 0.23 sec which is just large enough to get the eye on target. Trace 3 in Fig. 4B is the well known

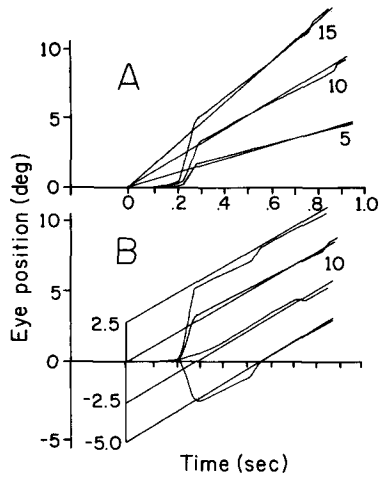

Fig. 4. A Ramp responses of the model in Fig. 3 for 5, 10 and 15 deg/sec ramps. The inclusion of velocity compensation insures that the first saccade gets the eye on target. B Step-ramp responses for steps of 2.5, 0, −2.5, −5.0 deg and a 10 deg/sec ramp velocity. The −2.5 deg step-ramp is the Rashbass (1961) input which ellicits no saccade. All other responses evoke saccades that get the eye on target

Rashbass (1961) step-ramp which evokes no saccade in its response. Whether the saccade is positive or negative, it is always appropriate to the anticipated error given by Eq. (7). This is the behavior seen experimentally (Robinson, 1965).

Young *et al.* (1968), in a revised model, suggested that if sampling were non-synchronized, that is, the sampler ran all the time whether or not there was any target movement, then, for various accidental time lapses between target movement and sampling, a variety of step-ramp responses appeared, among them, ones similar to the Rashbass response. Fuchs (1971) correctly pointed out that the frequency of occurrance of the various responses, especially the Rashbass response, predicted by the revised model of Young *et al.* (1968) does not correspond to experimental results. All of the responses in Fig. 4, given a certain randomness in the relative timing of pursuit and saccadic latencies, occur with great regularity in actual oculomotor testing, especially the Rashbass response which, far from being rare, is the usual response to that stimulus. Of course, if the idea that there is no sampler in the visual system is borne out, the question of whether sampling is synchronized or not is not relevant.

An interesting phenomenon is seen in Fig. 4. The pursuit velocity just before and after a saccade are usually noticeably different. This is because, for a 10 deg/sec ramp, the pursuit system is accelerating the eye at about 66.7 deg/sec^2. During the 50 msec duration of a saccade, the velocity changes by 3.3 deg/sec or, in general, by about one third of the total ramp velocity. There is thus an "apparent" jump in eye velocity associated with a saccade. This has been used to suggest sampling in the pursuit system. Although other evidence has been presented that that system is not sampled (Robinson, 1965), the responses in Fig. 4 further suggest that the jump in velocity is only apparent and does not indicate a true, rapid change in the pursuit velocity component of the eye movement.

4. Model III

The model of Fig. 3, as well as Fig. 1, will not correctly predict the eye's response to double target jumps; that is, when the target jumps to point *A* for *T* msec and then to point *B*. Wheeless *et al.* (1966), Newman (1970) and others have shown that if *T* is, say, 100 msec, there is a certain probability that the eye will first go to *A* and then *B*, (response type *a*) and the rest of the time will go directly to *B*, ignoring *A* (response type *b*). Often, the eye will first go to a position somewhere between *A* and *B*. The probabilities of what will happen depends on *T*.

To simulate such responses (the previous models *always* make response *a*) it is necessary to clearly recognize what an enormously artificial constraint the forms of Figs. 1 and 3 place upon the model by neglecting the fact that neural processing takes place in *space* (within the brain), not just in *time*. The retina is a two dimensional sheet of neural tissue. So are the superior colliculi and visual cortices. In fact, there is an orderly, continuous projection of the retina on these structures so that a target in space evokes electrical activity of neurons in a certain location in them according to that retinotopic map. When a target jumps from one part of the retina to another, one part of the visual system must recognize that the target is no longer there, while another part must recognize that the target is now here. This implies parallel data processing by the brain. It is generally recognized in neural modelling that the only way the brain could possibly handle the information rates that it does is by parallel processing. This is a fundamental concept in modelling CNS function.

In Fig. 3, the 0.17 *DEL* and *THR* elements represent only one tiny part of this vast parallel processing system. A very modest suggestion is that there are two such parts, one to decide what to do about the target at *A*, another to worry about the target at *B*. Consequently, Fig. 5 shows a system similar to that in Fig. 3 except that there are two parallel decision paths, 0.17 *DEL* 1 and 2 and *THR* 1 and 2 to

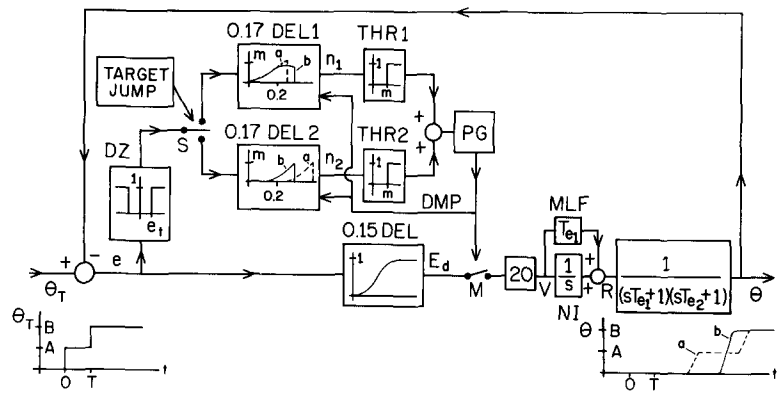

Fig. 5. An extension of Fig. 3 to include double target jumps such as that shown at the lower left. All elements are similar to those in Fig. 3. Switch *S* changes state when the target jumps from *A* to *B* to represent the fact that a new part of the visual system is now processing the data. The variable *m* is made random so that two types of responses can occur as shown at lower right. Within the *DEL* 1 and 2 blocks is indicated their responses for the two response types, *a* (dashed lines), *b* (continuous lines)

represent the two parts of the visual system excited by the target locations *A* and *B*. The switch *S* decides which circuit is excited by visual input. Consequently, whenever the target jumps from *A* to *B*, switch *S* is made to change its state to represent the fact that a new part of the visual system is excited and processing data. For simplicity, the error velocity circuit has been left out since only target jumps are to be considered, although its inclusion should not lead to any problems. This model is, of course, limited because it can only deal with targets that jump no more than twice between two successive saccades.

When the target jumps to point *A*, *DEL* 1 begins to respond. Variable $n_1(t)$ starts to rise toward the threshold *m*. When the target jumps to *B* and *S* changes state, n_2 starts to rise. The signal n_1 does not immediately return to zero but continues to rise because of the delay-line nature of these filters. It may or may not reach threshold depending on whether *T* is large or small. If *T* is large enough, n_1 reaches *m*, and a saccade is made to point *A*. Both *DEL* circuits are reset by the dump command (*DMP*) and n_2 now starts to rise again. It will cross *m* about 0.23 sec later and a second saccade will be made to *B*. If *T* is too small and n_1 does not reach threshold, no saccade to *A* is made and when n_2 reaches threshold, a single saccade to *B* occurs.

Thus the model can make two possible responses; type *a* or *b*. If *m* were fixed, then, depending on *T*, all responses would be either of one type or the other. However, in view of the number of non-target-related decisions the brain must also make (involving other sense modalities) it clearly will not reach the same decision in the same time for each target presentation. It seems reasonable that this variability can be represented by making *m* a random variable. In Fig. 5, *m* (for both *THR*) was chosen to have a mean of 0.54 (so that mean saccade latency was 0.23 sec), a range of $\pm 0.216 (\pm 40\%)$ from the mean with a probability distribution that was constant over this range (for simplicity).

Performance

For plain steps, the mean latency was 0.23 sec with a range of 0.18–0.28 sec. The distribution was approximately constant over this range but could be tailored to whatever one liked by chosing a different probability distribution for $m(t)$. Fig. 6 shows the results for double target jumps; first 4 deg to one side, then 8 deg to the same side as in Newman's (1970) experiments. Three values of *T* were chosen: 60, 100 and 150 msec. The probability of type *a* and type *b* responses depended on *T*.

A noticeable fault of the model is that saccades are often not either 4 or 8 deg but to points in between. This is because of the poor approximation of 0.15 *DEL* to a pure delay line. If the latter had been used, saccades would always be one or the other. However that, too, would be incorrect since Newman (1970) has shown that intermediate saccades do occur not infrequently. A better, but not perfect, approximation to a delay line would improve the model.

When *T* is 0.15 sec (Fig. 6C) there is a sharp division in response types when *m* is 0.66 so that 22% of the responses are of type *b* (the first saccade is about 8 deg in size with a latency of about 0.4 sec) while 78% are of type *a* (about 4 deg in amplitude with a mean latency of 0.205 sec). When *T* is 0.10 sec (Fig. 6B), type *a* responses occur only 34% of the time, type *b* responses,

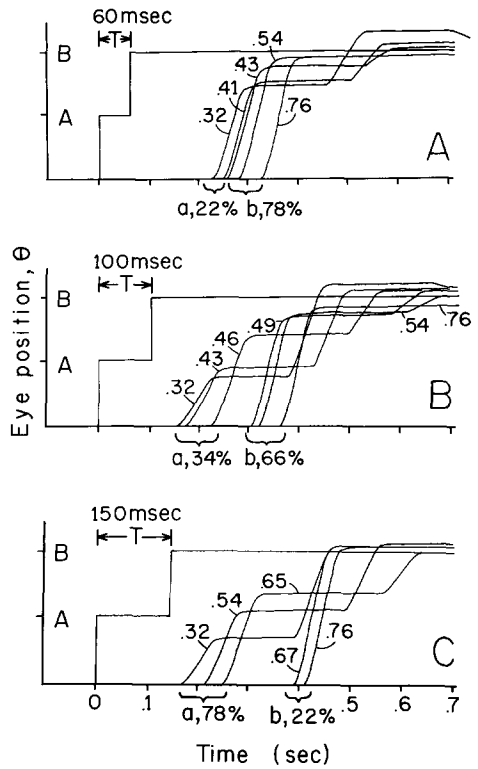

Fig. 6. The responses of the model of Fig. 5 to double target jumps with intervals T of 60, 100 and 150 msec. The value of m is given next to each response. The responses are divided into two types, a and b and the frequency with which each occurs is given in each case

66% of the time. When T is 60 msec (Fig. 6A) the distinction between the two types is no longer so sharp on the basis of latency. However, when $m \leqq 0.41$, the saccade amplitude is much less than 8 deg. Using this criterion, 22% of the responses are type a, 78% are type b with a mean latency of 0.30 sec.

Newman (1970) has shown that saccades to A are influenced in latency and amplitude by the subsequent target jump to B and *vice versa*. Consequently the division of movements into types a and b is actually an oversimplification for the real system just as in this model. Not enough experimental statistics exist to be able to try to adjust the transfer function of the delay line between e and E_d and the probability distributions of $m(t)$ to better fit actual behavior. The purpose in presenting this model is not to offer exact, final solutions but to suggest that the inclusion of spatial (parallel) processing is the appropriate way to develop models that can deal more realistically with target behaviors more complicated than simple steps and ramps and to do so in a way that is compatible with brain stem motor organization and higher cortical data processing.

5. Model IV

Spatial processing in the visual system is probably so basic, that advanced models of the saccadic system should abandon the artificial constraints inherent in all the models we have just considered (such as the separation of timing and amplitude into different circuits) and go directly to a spatial-temporal arrangement. One reason for doing this is simply that the additional degree of freedom allows one to model more complicated phenomena in saccadic control. But the basic reason is that neurophysiological experiments clearly show that vision and saccades are spatially organized in the same structures. It's well known that the retinal visual field projects onto the optic tectum or superior colliculi so as to form a sensory map of visual space. A motor map also exists in the colliculus (Robinson, 1972). If a local region of the colliculus is electrically stimulated in the alert monkey, it makes a saccade of a fixed amplitude and direction which depends only on the location of the stimulus site and not on stimulus parameters or initial eye position. Thus, to each site can be assigned a fixed amplitude and direction forming a continuous motor map. Of course, the sensory and motor maps overlay each other. If a light is flashed, say, 20 deg to the right and 10 deg up (with respect to the fovea), cells are excited (after a 50 msec delay) in only one local region of the superior colliculus to which that point projects. If that same locus is stimulated, the monkey makes a saccade (after a delay of about 35 msec) which is 20 deg to the right and 10 deg up.

Schiller (1972) has demonstrated this spatial sensory-motor mapping even more clearly. He has found neurons in the intermediate gray layers of the colliculus which are not only excited by visual stimuli in a certain part of the visual field but also fire in a burst during saccades and do so most vigorously only for those saccades which carry the fovea to that particular visual stimulus. These neural elements seem to be concerned with detecting the presence of visual stimuli in a local region of the retina and also in the decision to make a saccade to look at it. Many of these cells begin firing (they are not spontaneously active) about 100 msec before the saccade and reach a crescendo when the saccade starts. There is a suggestion here that the location of visual stimuli are being held in spatial storage awaiting decisions to arrive from other brain areas which release this activity to the motor circuits.

The released message is clearly spatially coded. That is, the pulse generator obtains the information specifying saccade size not from the temporal nature

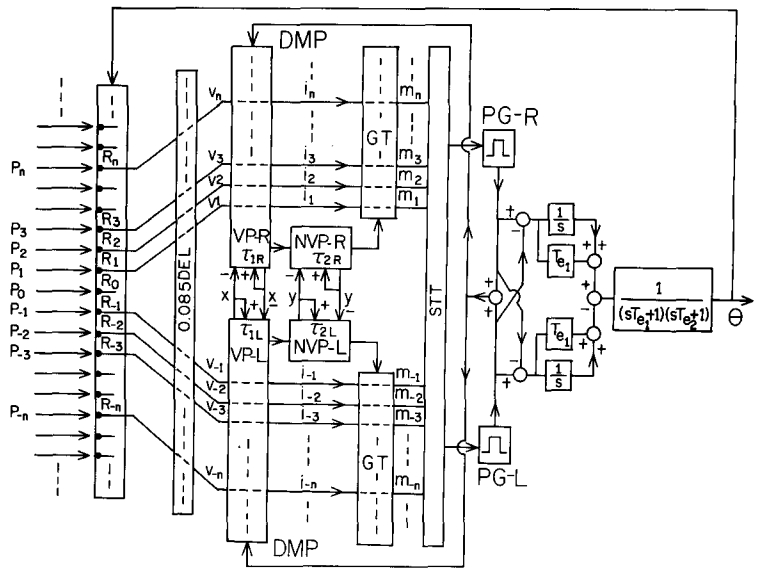

Fig. 7. A complete spatial-temporal model of the saccadic system. $\{P_n\}$, the set of possible target positions in space; $\{R_n\}$, the set of retinal locations (the fovea is at R_0); $\{v_n\}$, the set of neural receiving sites in primary visual cortex and upper layers of the superior colliculi; $\{i_n\}$, the set of neural processing sites similar to those in the intermediate gray layers of the superior colliculi; $\{m_n\}$, motor commands descending from deep tectal layers. *VP-R*, *VP-L*, visual processing elements like those in Fig. 5 in the right and left visual cortices; *NVP-R*, *NVP-L*, non-visual data processing by association cortex; *STT*, a spatial-temporal translator that calculates the correct saccade size from spatially coded input information. The premotor circuitry is shown more completely as push-pull, brain stem elements

of the activity but from the location in the visual system from which it came. How this is accomplished is not known. We can only assume the existence of some neural network caudal to the mesencephalon which converts spatially coded signals into pulses of the correct width and height to make the appropriate saccade. Such a network might be called a spatial-temporal translator.

Figure 7 shows a rough model which embodies these ideas. The retina is divided into many locations $\{\ldots R_{-2}, R_{-1}, R_0, R_1, R_2 \ldots\}$ onto which, in the primary position, project targets of spatial locations $\{\ldots P_{-2}, P_{-1}, P_0, P_1, P_2 \ldots\}$. Whenever the eye moves, the set of retinal loci $\{R_n\}$ slide with respect to the external visual loci $\{P_n\}$ by the amount θ. The 50 msec delay before visually evoked activity appears in central visual structures plus the 35 msec delay between collicular stimulation and a saccade, constitute an 85 msec minimum reaction time for an eye movement. This total delay is lumped into a single element, 0.085 *DEL*. The signals $\{v_n\}$ represent neuron activity in striate cortex and the upper layers of the superior colliculi. *VP-L* and *-R* represent visual processing of this spatial-temporal information in the left and right hemispheres. Assume this process takes τ_1 milliseconds. When this system has identified and located the target it holds the information spatially $\{i_n\}$ and informs higher centers, *NVP-R* and *-L*, which process non-visual data and reaches a decision in τ_2 msec. These decisions may be concerned with whether or not some non-visual stimulus indicates that some other action should take priority over the impending saccade. This decision gates (*GT*) the spatially held target position information (one of the i_n lines), and the activity passes to one of the spatially coded motor command lines $\{m_n\}$. Neuroanatomically, the signals $\{i_n\}$ may be similar to unit activity in the intermediate gray layers of the superior colliculus and $\{m_n\}$ represents the tecto-bulbar output of the colliculus to lower brain stem motor circuits. These latter commands are decoded by the spatial-temporal translator (*STT*) whose properties are fairly well defined by electrical stimulation experiments (Robinson, 1972). If a target appears at, say, R_3 and this finally excites line m_3, the *STT* causes the pulse generator *PG-R* to produce a pulse whose area is just large enough to create a saccade that brings the fovea, R_0, to occupy the point formerly occupied by R_3. In this model there is no need to propose separate timing and amplitude circuits. The occurrence of activity on line m_n carries with it both pieces of information.

The motor circuitry has been amplified to show that it actually consists of pairs of elements in push-pull, symmetrically arranged around the midline.

This is generally understood and ignored for simplicity in most models. It is included here only to remind us of the actual circuitry because this becomes very important in considering models of oculomotor disorders such as those seen in neuro-ophthalmology clinics. The modelling of such disorders is still very primitive at present but badly needs whatever help it can get from systems analysis.

Spatial Response Properties

One advantage of spatial models such as that in Fig. 7 is their ability to cope with behavior that seems spatial in origin such as interhemispheric transfer. Newman (1970) showed that in double target jumps (as in Fig. 6) the system could more quickly change its mind, cancel plans to saccade to point A and go to B directly (type b response), if target B was on the same side of the fovea as A than if it crossed to the opposite side. The most obvious physiological correlate of this is that in the first case, the same hemisphere did all the processing while in the second case, the processing had to be shifted between hemispheres. This suggests that when one hemisphere has started the analysis process, it is facilitated in reidentifying and relocating the target if it moves but stays in the same hemisphere. In fact, the opposite hemisphere seems to be actually disfacilitated in the process. Specifically, activity in VP-R causes τ_{1R} to decrease and τ_{1L} to increase for the analysis of a second target jump. This is suggested symbolically by the signals x.

A similar phenomenon is seen in normal saccades. Most large saccades (e.g. greater than about 15 deg) only go about 90% of the distance to the target. A second saccade closes the 10% gap. It occurs about 130 msec after the first (Becker and Fuchs, 1969). This appears to be a deliberate strategy used by the brain. The second saccade is thought to be preprogrammed because of its short latency. The preprogram may occur as follows in the model of Fig. 7. There is no need to remake all the decisions involved in NVP for every saccade especially when there is a high expectency that the first saccade will fall short. There may be a mechanism by which NVP could be held on temporarily so that the delay τ_2 is removed for the second saccade. Since the eye almost always falls short of the target after the first saccade, the residual error is processed by the same hemisphere so that some of the time saved (100 msec) may also come from a decrease in τ_1, as already suggested.

Young *et al.* (1968) found that if the target jumped immediately after a saccade, the latency to the next saccade was 200 msec if it jumped in the same direction (stayed in the same hemisphere) but was 400 msec if it jumped in the opposite direction (changed hemispheres). These results neither confirmed or denied their theory about non-synchronized sampling but do seem consistent with some phenomenon associated with a time penalty incurred when saccadic control is switched between hemispheres. It suggests, in Fig. 7, that the NVP may also interact like the VP, facilitating themselves and disfacilitating their counterparts in the other hemisphere when they have identified and are processing the appearance of the target. This is suggested by the connections y. However, Becker and Fuchs (1969), in a similar experiment, found quite different results. If the second target jump took the target outside the expected 10% residual error range, the latency to the next saccade was 390 msec regardless of which way the target jumped. Clearly these differences must be resolved before one can get very specific in Fig. 7.

It may even be possible to model secondary saccades that occur with latencies of only 90 msec. If, in Fig. 7, τ_2 were removed by temporarily holding NVP on, and VP were stripped to the bare essentials of a pattern recognition program, reducing τ_1 to a small value, a secondary saccade latency may well approach the absolute minimum of 85 msec. Johnson (1963) proposed a model which used model feedback, that is, an internal model of the oculomotor system was used to obtain a quick estimate of whether or not any given command was adequate to reduce the error to zero. If it were not, quick remedial action could be taken. This theory may well be correct but it should also be recognized that alternate theories are possible.

6. Discussion

Clearly a great deal has been left unspecified and untested in Fig. 7. Its main purpose is to suggest that models that fail to take spatial processing into account have gone about as far as they can go. In Fig. 7, all the elements between $\{m_n\}$ and θ are compatible with everything we know so far about the brain stem organization of eye movements in general and saccades in particular. We may not know where these elements are, anatomically, or how they are arranged as nerve nets but their input-output relationships are experimentally known and the evidence for this arrangement is mounting so rapidly in neurophysiology that it will probably soon move from theory to fact. Granting this, the greatest implication of the models presented here is to remove

the concept of sampling from considerations of how the brain processes visual data. The elements that challenge the theoretician in Fig. 7 lie between the inputs $\{v_n\}$ and the outputs $\{m_n\}$, that is, the higher processing functions of the cerebral cortex. There, no sampling features are required or suggested by this model. The model as a whole behaves, of course, like a sampling system in the general sense that it processes data, reaches decisions and acts upon them in a time discrete fashion. But this does not imply that any sampling occurs before the mesencephalic output to the brain stem. No doubt future experiments designed to discover whether or not the decision processes can be compartmentalized and what kind of algorithms they might use will further test this general hypothesis.

Acknowledgment. This investigation was supported by U.S.P.H.S. Research Grant EY-00598 of the Eye Institute, the National Institutes of Health. I also thank W. Becker of the Sektion Neurophysiologie, Universität Ulm for assistance with the manuscript.

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