

Chapter 4

Adaptive properties of the saccadic system

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1. Introduction

Visual perceptions are formed after acquiring and stabilizing images on the retinal area with the highest visual acuity, the fovea. Preceding chapters have tended to concentrate on those eye movements used to stabilize a given retinal image during head and/or image movements. In foveate animals the voluntary shifts of gaze, made with rapid eye movements called saccades, are of equal functional significance. Normally these saccades are characterized by both a high degree of accuracy (corresponding to the small foveal diameter, e.g., 1–2° in the monkey) and a high angular velocity (100–900°/s in the same species). Another important characteristic, which is particularly relevant to the present chapter, is that saccades end abruptly, i.e., without post-saccadic ocular drift, thereby permitting good visual perception immediately after completion of each saccadic movement. Over the past decade much has been learned about the way the brain generates saccadic eye movements. The present chapter focuses on one aspect of the neural control of saccades: the adaptive ability to maintain saccadic performance (in particular the ability to get the eyes accurately on target and to hold them there without ensuing ocular drift).

The command for a saccade is produced by neurons within the brain stem that are part of the oculomotor control system (Robinson, 1975a; Keller, 1981). When a target is selected as the item

of interest, it is the task of this subsystem to generate the neural signal required to take the fovea to the newly selected image and hold it there. As we shall see below, functionally significant aspects of the motor performance produced by this signal are monitored by other parts of the brain. Those other parts can advantageously alter critical parameters within the saccadic motor subsystem, thereby acting as an adaptive control system (Optican & Robinson, 1980).

In natural life these adaptive mechanisms serve to offset degradation of oculomotor performance resulting from interference along the chain of neural conduction, neuromuscular transmission and muscle function, due for example to such factors as aging, injury and disease. Clinical aspects of this matter are reviewed by Zee and Optican in Chapter 11, and by others elsewhere (e.g., Optican, 1982; Stark, 1982). Experimental laboratory studies have shown that at least two mechanisms, one fast and one slow, can alter saccadic movements in response to inappropriate oculomotor performance. The present chapter reviews some of the basic experimental studies which demonstrate the presence of these adaptive mechanisms, and attempts a systematic interpretation of their functional structure and operation.

2. Generation of saccades

As shown by Robinson (1964), the description of the mechanical properties of the eyeball, suspen-

sory tissues and extraocular muscles can be simplified by lumping them into viscous and elastic elements, the inertia of the globe being insignificant. To move the eyes rapidly, the muscles must develop a large transient force to overcome the viscous drag of the orbital tissues. To hold the eyes at one point in the orbit, the muscles must develop just enough maintained tension to balance the elastic restoring force of the orbital tissues. Hence a saccade requires a large, brief pulse of torque to move the eye rapidly and a smaller, sustained torque to hold the eye in its new position.

These two components of torque are caused by two corresponding elements in the efferent neural discharge to the extraocular muscles. Thus, the large transient motor output needed to turn the eyes rapidly against viscous drag is generated by a high-frequency burst, or pulse, of phasic neural activity; while the sustained muscle force opposing the elastic tension is generated by a step change in tonic activity (Robinson, 1973). This combined pulse-step pattern of neural activity is now known to be formulated within the brain stem by the motor control subsystem mentioned above, and constitutes the basis of all normal saccade generation (Keller & Robinson, 1972; Luschei & Fuchs, 1972; Keller, 1974).

As implied above, to achieve an ideal saccade the pulse and step of innervation must be adjusted so that the pulse drives the eye to the desired position and the step holds it there. Inappropriate innervations give rise to inaccurate, or dysmetric, eye movements. If both the pulse and step are too small, the eye will not reach the target (hypometria), while if they are too large, the eye will overshoot the target (hypermetria). If the pulse and step are not matched, the eye will move rapidly until the pulse of innervation ends, but then it will drift exponentially to the orbital position determined by the step (glissade). If the step is too large for the pulse, the eye drifts onwards; if too small, it drifts back (see Fig. 2).

3. Maintaining saccadic accuracy

The accuracy of saccades appears to be governed by three different mechanisms. One mechanism is under voluntary control and operates over a time scale of seconds, while the other two are involun-

tary and operate over slower time scales of minutes and days. For reasons given below, only the latter two are considered to function as adaptive mechanisms.

3.1. Voluntary goal adjustment

The voluntary control of saccadic accuracy is achieved by changing the selection of the saccade goal. Vossius (1972) studied the ability of subjects to acquire targets that moved when they tried to look at them. By monitoring eye position and adding a fraction of the eye position to the target position signal, the target was made to jump with every saccade in a predictable fashion. After just a few attempts subjects learned to make a single saccade to a location where there was no target, but to which the feedback signal would bring the target. Hallett (1978) has shown that subjects can voluntarily make saccades that are smaller than, larger than, or oppositely directed to target jumps. Such studies indicate a high degree of volitional control over the selection of saccadic goals.

Such a goal changing strategy may be of limited usefulness outside the laboratory. For example, patients with a sixth nerve palsy take several days to improve the accuracy of their saccades, apparently making no use of saccadic goal selection strategies (see below). In the laboratory, goal selection tasks are done with a few points of light that move in a stereotyped fashion. When patients have to cope with saccadic dysmetrias, however, the visual scene is rich with items of interest, and the required goal modification may depend on the position of the eyes in the orbit. Thus, there may not be enough time to make the volitional effort needed to reprogram the goal for every saccade under these conditions. Because of the volitional nature of goal selections, these essentially instantaneous alterations of saccadic gain are excluded from the rest of the discussion of the adaptive properties of saccades.

3.2. Normal hypometria

The definition of an adaptive mechanism must be broad enough to deal with more than just simple accuracy, for despite the ability of subjects to rapidly adjust saccadic goals, normal saccades are

not always accurate. When the required refixation exceeds 10° of arc in man or monkey, it is usually made with more than one saccade. The first, or primary, saccade almost always falls short of the target, and is quickly followed by a secondary, or corrective, saccade (Becker & Jürgens, 1975; Hallett & Lightstone, 1976). If the target light is turned off just before the primary saccade, an accurate secondary saccade can still be made. Hence, this falling short, or hypometria, does not reflect an inaccuracy of the saccadic system. Instead, saccadic undershoot seems to be preprogrammed (Becker & Fuchs, 1969).

3.3. Rapid saccadic adaptation

This normal hypometria has been studied by altering visual feedback conditions. Henson (1978) used an optical system to magnify the effective size of a subject's saccades. This magnification resulted in the usually hypometric saccade falling exactly on the target (orthometria) or even overshooting it (hypermetria). With 10 to 15 min of this altered visual experience the subject's saccades again became usually hypometric. This mechanism seems qualitatively different from the voluntary selection of saccadic goals: it involves a smooth change in saccadic accuracy over a period of time. These experiments indicate that the gain of the saccadic system (size of the primary saccade divided by the size of the initial target eccentricity) is under rapid adaptive control. Furthermore, they demonstrate that the goal of the first saccade is not simply to acquire the target: the primary saccade is intended to fall short.

Most explanations for this undershooting are based on the observation that corrective saccades in the same direction as the primary saccade follow it with a shorter latency than corrective saccades in the opposite direction (Hallett & Lightstone, 1976; Henson, 1978). If the first saccade falls short, the target will still be on the same side of the fovea, and hence its image will project to the side of the brain that generated the primary saccade. If the first saccade overshoots the target, it will then be on the opposite side of the fovea, and its image will project to the other side of the brain. The increase in time required to correct for a hypermetric movement is presumed to have two compo-

nents: one for consolidating information between the hemispheres and one for reprogramming the direction of the corrective saccade.

Two mechanisms have been proposed to account for this form of hypometria. Hallett (1978) proposed a dual saccadic system in which one component is fast, but has a coarse retinal resolution, while the other is slower but more accurate. The primary saccade is made by the faster system. The second, co-activated system gives a better estimate of the target's position. This estimate is combined with eye position information to obtain an anticipated error, which may give rise to a secondary saccade. This dual control would explain the need for corrective saccades, but it would not explain why the primary saccade is usually hypometric. Since the saccadic gain is adaptable, the average of a large number of coarsely programmed saccades should still be orthometric, i.e., exactly on target.

In an attempt to overcome this difficulty, Optican (1982) hypothesized that the undershoot of large saccades is purposeful. Since most saccades are small (e.g., under 15° in amplitude according to Bahill et al., 1975a), an undershoot only on large saccades would provide an occasional perturbation of known size, allowing a 'corrective system' to monitor the accuracy of the saccadic motor system. In order to derive an error signal for saccadic gain control, the target must not move before the result of this perturbation is measured. When the retinal image projects to the opposite hemisphere, the information on target position must be sent back to the original hemisphere to allow for a comparison with the expected error. This must take longer than if the image stayed in the same hemisphere. Since the measurement time will be shorter if the retinal error stays in the same hemisphere, the primary saccade is made hypometric to reduce the probability that the target will move before the actual retinal error can be compared with the expected retinal error. Furthermore, programming a corrective saccade in the same direction as the primary saccade takes less time than programming one in the opposite direction (Hallett & Lightstone, 1976). Hence, the total time to actually reach the target will be less if the perturbed primary saccade falls short, rather than long.

The purpose of the rapid adaptive system that maintains hypometria may be to guarantee that the first saccade falls short. This could be achieved with a relatively simple system, allowing a more complex, but slower, controller to adjust saccadic accuracy by monitoring the effects of amplitude perturbations. The existence of such a gradual controller has been established in both human patients and monkeys.

3.4. *Gradual saccadic adaptation*

The ability of subjects to rapidly restore hypometria in response to altered visual feedback represents a fast form of plasticity, i.e., a gain change that is maintained without conscious effort. The voluntary changes of saccadic goals described earlier represent another fast form of saccadic control. Despite these abilities patients take several days to increase their saccadic system gains to compensate for muscle paralysis (Kommerell et al., 1976; Abel et al., 1978). Presumably patients with peripheral weakness can still alter their saccadic goals voluntarily, but they do not use this ability to compensate for their weakness. Perhaps this is because such compensation requires a large volitional effort for each saccade, especially when oculomotor deficits depend on orbital position. A qualitative difference also exists between the types of saccadic adaptation seen in patients and in the subjects of altered feedback experiments: in altered visual feedback experiments no post-saccadic ocular drift develops. Yet Kommerell et al. (1976) found that patients with a unilateral abducens nerve palsy, when viewing with the normal eye, made saccades with the paretic eye that not only fell short of the target, but also had post-saccadic drift back. Patching the normal eye led to an increase in the size of its saccades, and produced post-saccadic drift in it. These changes were not evident after a short time (15 min), but took several days to develop. Hence patterns of innervation change slowly in patients with abducens nerve palsies.

A gradual form of adaptation for recalibrating the motor system would obviate the need for volitional control over every saccade. The longer time course of a gradual adaptive mechanism would allow it to take into account effects, such as

orbital position dependence or post-saccadic drift, which were not evident from the accuracy of a single saccade. These interesting clinical observations have led to controlled experimental studies on laboratory animals. A form of gradual adaptation to peripheral weakness has been studied in monkeys. After weakening the muscles of one eye, patching one eye at a time allows the visual experience of the animal to be controlled. Using this patching paradigm Optican and Robinson (1980) found that adaptive control of saccadic accuracy occurred gradually; compensation for peripheral weakness had a (roughly) exponential time constant of 1.5 days, while return to the original condition occurred more rapidly.

Weakening the muscles of these animals caused a decrease in the ratio of orbital viscosity to elasticity, presumably due to the increased elasticity of scar tissue. Hence, saccades in the weakened eye not only fell short of the target, but were also followed by post-saccadic drift back. When the monkey's normal eye was patched, the saccades of the paretic eye gradually became larger, and its drift was suppressed. The corresponding saccades in the normal eye became too large and had post-saccadic drift onward. This is consistent with Hering's Law, that the same innervation is sent to both eyes. The time course of the amplitude correction and the drift suppression were the same.

These changes in saccadic waveforms can be interpreted in terms of the efferent pulse and step of innervation. When the normal eye is first patched, the brain views the world with an eye that falls short of the target and drifts back. In an effort to improve vision, the pulse is increased until the paretic, viewing eye can reach the target in one saccade. The normal, patched, eye overshoots the target, and thus appears to also receive this larger innervation. The step of innervation is also increased to offset the increase in the orbital elasticity. This suppresses the post-saccadic drift back in the paretic eye. The normal, patched, eye develops a post-saccadic drift onward when it receives the same step.

Optican and Robinson (1980) used this patching paradigm to investigate the dependence of the adaptive control system on the cerebellum. The direct effects of ablating portions of the cerebel-

lum on the saccadic system can be assessed by observing the saccades made by the normal eye. The indirect effect of such ablations on the adaptive mechanism can be assessed by observing the changes in saccades following patching of the normal eye. After the midline portions of the cerebellum (the vermis, paravermis and fastigial nuclei) were ablated in monkeys, the saccades in the normal eye became hypermetric. Patching either eye for several weeks did not cause any change in the saccadic amplitude. However, post-saccadic drift was still suppressed, after several days, in whichever eye was viewing. After total cerebellar ablations, the saccades in the normal eye became hypermetric, and were followed by post-saccadic drift. The direction and amount of the drift depended on orbital position. Neither the hypermetria nor the drift were ever altered by the patching of either eye. These two findings suggest a separate mechanism for drift suppression. Following this lead, it was found that when the floccular lobes of the cerebellum were ablated in monkeys, their saccades did not become hypermetric, but had an enduring post-saccadic drift that could not be modified by optical means (Optican et al., 1980).

Vilis and Hore (1981) found that cooling the midline cerebellar nuclei of monkeys caused dysmetric saccades with post-saccadic drift. When cooling ceased, saccades returned to normal. Vilis et al. (1982) have also found that unilateral cooling of the medial cerebellar nuclei resulted in different degrees of dysmetria in the two eyes. In both the ablation and the cooling experiments the amount of dysmetria and drift depended on orbital position and saccade amplitude, indicating that the cerebellum is involved in matching the pulse to the step as a function of orbital position. Neither study found any change in the dynamic characteristics of saccades (their amplitude-duration and amplitude-peak velocity relationships). An earlier study by Ritchie (1976) did report an altered amplitude-duration relationship in primates with small midline cerebellar ablations, but no explanation exists for this discrepancy.

These experiments indicate that the cerebellum is involved in the adaptive control of both the pulse and step gains of the saccadic system (for

recent reviews see Optican, 1982 and Stark, 1982). The pulse gain control seems to depend on midline structures (vermis and fastigial nuclei) while the step gain control seems to depend on lateral structures (flocculus). The cerebellum may also be involved in maintaining conjugacy of the two eyes, and in compensating for position-dependent orbital mechanics. How the cerebellum interacts with the brain stem circuits that generate saccades is not yet known. It will be a great help in studying such interactions if the functional roles of the two regions can be understood in detail. One approach to such a functional description is through modeling.

4. Error signals for adaptive control

Quantitative models of physiological processes not only summarize what is known about a system, but allow hypotheses to be tested and new experiments to be designed. Modeling an adaptive control system requires knowledge of two parts of the system. First, the motor subsystem must be understood. This subsystem actually moves the eye, and it will contain parameters, such as gains and time constants, that determine its performance. Second, the adaptive mechanism must be described. This subsystem monitors the performance of the saccadic system. Some measure of performance is combined with other information to derive an error signal. It is the goal of the adaptive mechanism to reduce this error signal by adjusting the parameters of the motor control subsystem, a form of interaction termed parametric feedback.

While quantitative models of the motor control part of the saccadic system have progressed rapidly (Robinson, 1973, 1975a; Zee et al., 1976c), very little is known about the error signals used to achieve adaptive control of saccades. The adaptive control of the pulse gain, which determines saccadic accuracy, is greatly complicated by the need to recognize the target. Since a saccade is made to only one item within a visually rich scene, knowledge that a saccade was inaccurate requires that the same item be identified in different retinal locations before and after the saccade. These different locations must then be compared in some way to determine the error

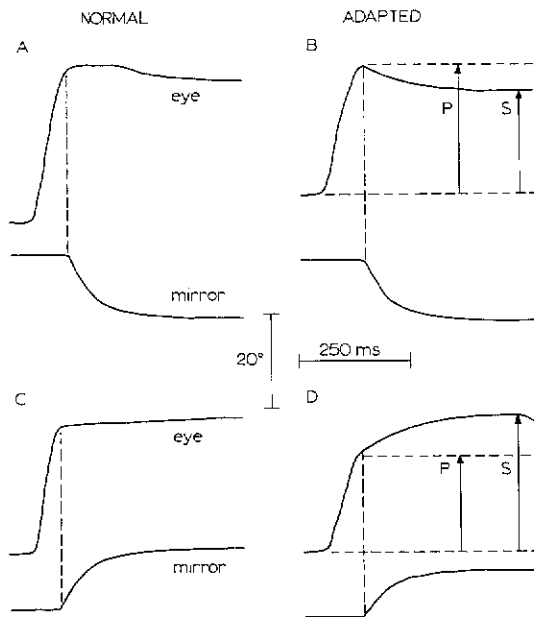


Fig. 1. Suppression of optically induced retinal slip. The left panels show the movement of the visual field (mirror) triggered by a saccade before the animal has been adapted. The visual field movement in part A causes retinal slip backward, while that in part C causes retinal slip onward. Note the pursuit system response after about 110 ms. The right panels show the same types of movements after several days of experiencing the optically induced slip. There is now a zero-latency ocular drift in the compensatory direction. Part B shows that the gain of the step driven part of the movement (S) has been reduced relative to the gain of the pulse driven part (P). In part D, the gain of the step has been increased.

signal. A preliminary attempt at modeling the adaptive control of saccadic accuracy has been described elsewhere (Optican, 1982). That model depends on a corrective system that generates secondary saccades and derives a post-saccadic amplitude error signal for a cerebellar-dependent adaptive gain controller. The corrective system derives an error signal by comparing the actual retinal error after a saccade with the retinal error expected because of the intentional hypometria of the primary saccade.

Adaptive control of the step gain (to suppress post-saccadic ocular drift) is much easier to model, since drift can be monitored either by extraocular muscle proprioception or by full-field retinal slip. Optican and Miles (1979) reported

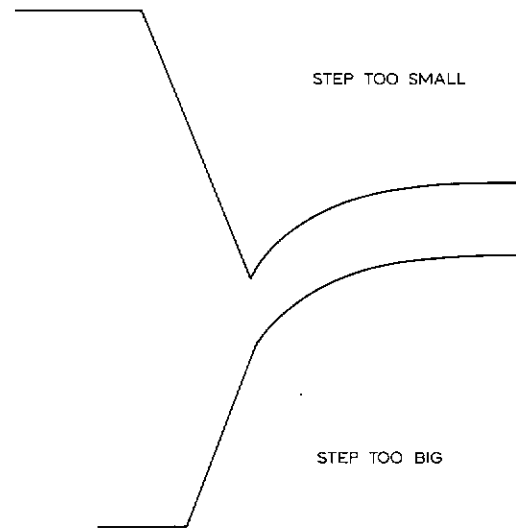
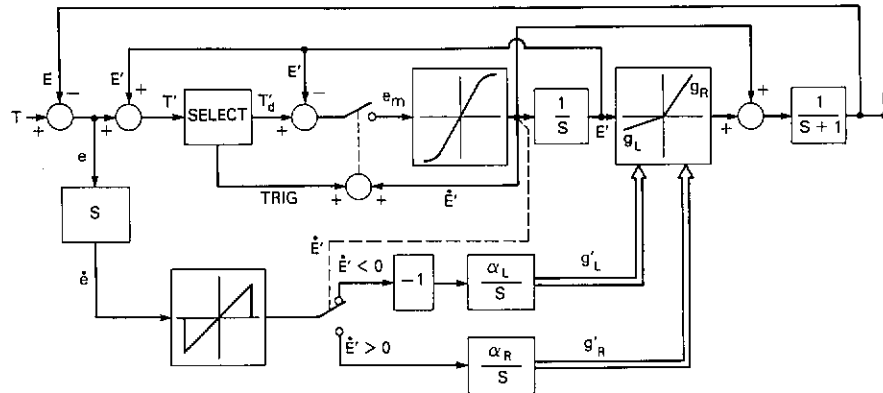


Fig. 2. Schematic representation of a glissade. In the top trace, the step of innervation is too small for the pulse of innervation, causing the eye to drift back. In the bottom trace, the step is too large for the pulse, causing the eye to drift onward. Note that the drift portion of the eye movement is identical in either case, making post-saccadic retinal slip alone insufficient to determine whether the step gain is too large or too small.

studies in primates subjected to saccade-triggered visual slip. In these experiments the visual world was caused to drift after each saccade by a computer controlled optical system involving a mirror galvanometer and a projected visual scene. In one series of experiments the slip was in the same direction as the antecedent saccade (Fig. 1C), while in another series the slip was oppositely directed (Fig. 1A). After several days of such optically-induced slip, the monkey's saccades showed adaptive changes (Fig. 1B,D). These experiments showed that retinal slip is sufficient to elicit adaptive changes in the saccadic system. However, slip alone does not indicate the sign of the necessary gain change. Fig. 2, for example, shows that a saccade to the left when the step is too small for the pulse of innervation, and a saccade to the right when the step is too large, both have identical ocular drifts. Based on the experimental findings given above an attempt has been made to propose a mechanism for adaptive drift suppression in which the relevant error signal is derived by correlating retinal slip im-



ADAPTIVE CONTROL OF POST-SACCADIC DRIFT

Fig. 3. Block diagram of a model for the suppression of post-saccadic drift. The upper pathways represent the saccadic motor control system. The step gain is represented by the box on the right with two line segments whose slopes are variable. The slopes, g_L and g_R , represent the gain for leftward and rightward saccades. The bottom pathways represent the adaptive control mechanism for adjusting the step gains. The signal indicating the step gain error is derived by correlating retinal slip with the direction of the antecedent saccade. Single lines represent signal processing paths. Dashed lines indicate switching signals. Double lines represent variable gain controls. See text for explanation of signals.

mediately following a saccade with the direction of that saccade. Fig. 3 shows a model based on this hypothesis.

5. An adaptive model of post-saccadic drift suppression

To model drift suppression we start with a model of the normal saccadic system (Robinson, 1975a; Zee et al., 1976c). Fig. 3 shows the neural integrator ($1/S$) and ocular motor plant ($1/S+1$) in Laplace transform notation (Robinson, 1973). There is also a branch for velocity feed-forward compensation of the lag in the plant dynamics.

To make a saccade, a velocity command, or pulse, must be generated and sent both directly and indirectly, through the neural integrator, to the ocular motor neurons; the two pathways contributing the pulse and step signals, respectively (Robinson, 1973). The pulse therefore completely determines the dynamic properties of the saccade. In this model, a pulse generator that is not pre-programmed, and that has as its input the desired target position in spatial, rather than retinal, coordinates is used (Robinson, 1975a). This spatial estimate (T') is obtained by adding the brain's best estimate of where the eye is

(efference copy, E'), to the retinal error (e). Retinal error (e) is determined by the position of the target in space (T) relative to the eye position in space (E). A selection process determines the desired eye position in spatial coordinates (T'_d). A high-gain, nonlinear element generates a large output, or burst, based on an estimate of the motor error (e_m) between where the eye is (E') and where it is going (T'_d).

Since e_m depends on E' , the burst is generated continuously, automatically ending when the motor error (e_m) becomes zero. The high gain, non-linear element is based on the 'short lead' burst cells identified by single-unit recordings within the brain stem (Luschei and Fuchs, 1972; Keller, 1974). The burster's high gain makes the saccadic system unstable. Hence to prevent the eyes from oscillating a switch, modeled after the pause cells found in the pons, is included (Keller, 1974). When a trigger is received from some other part of the brain, the switch closes and the burst begins. When e_m reaches zero, the switch opens and the eye is held on target by the neural integrator. Once again, it is important to realize that each part of the model reflects some aspect of the available data. The structure is a hypothesis, but no physiologically unrealistic parts have been

added (Zee et al., 1976c).

When assembling these various components, we must include a variable gain element that will allow post-saccadic drift to be adaptively suppressed. Experiments on monkeys (see above) have shown that lesions of the posterior vermis can abolish pulse gain adaptation while sparing step gain adaptation. That being the case, there is one, and only one, place where the variable gain element may be located. Since the step is obtained by integrating the pulse, everything upstream from the neural integrator affects both the pulse and the step. Since the ocular motor neurons carry the combined pulse and step of innervation, everything downstream from this point also affects them both. Hence the variable gain element must be located just after the neural integrator. Since final eye position is based on efference copy (E'), the gain element must be outside the efference copy feedback loop. (If it were inside the feedback loop, the steady state eye position would not be affected by the gain, since the burst continues until the desired eye position is reached.) The gain of the step can be altered independently for leftward and rightward saccades, so it is represented in Fig. 3 by two line segments with different slopes: g_L and g_R .

The parts of the model described so far represent the structure needed to make the saccade, but not the structure needed to adjust the variable gain element. In order to extend this model, two further components must be added. The first is a parametric gain adjuster. The second is a mechanism for deriving a step gain error signal. The step gain does not jump instantly to new values, but changes gradually over several days. This suggests that the step gain could be set by the output of a neural integrator with a low gain. The step gain would then change gradually, with a time course determined by the gain of the neural integrator. Such slow integration acts as an averager. The retinal slip seen by normal subjects can have any direction, but on the average it should occur equally often in all directions, i.e., the average slip should be zero. A subject with post-saccadic ocular drift, however, would have retinal slip in a direction correlated with the direction of the antecedent eye movement. Hence a retinal slip signal could be used as a step-gain

error signal if it is integrated in a way correlated with saccade direction.

The adaptive controller on the bottom of Fig. 3 monitors retinal slip velocity (\dot{e}), with high saccadic speeds clipped out, by feeding them into one of two integrators. Which integrator the slip goes into is determined by a switch set by the direction of the antecedent saccade (\dot{E}' dotted line). The output of the two integrators (α_L/S and α_R/S) sets the desired step gains (g'_L and g'_R) for the elimination of post-saccadic ocular drift. The gains of the integrators, α_L and α_R , determine the time course of the adaptation.

This model is useful in two ways. First, it summarizes what is known about the structure and function of the saccadic system, and second, it allows predictions about the behavior of the system to be made. By extending this model to include other ocular motor subsystems, such as smooth pursuit or the vestibulo-ocular reflex, predictions can be made about the interaction between saccadic and other types of eye movements. Then, by studying the effects of changing the saccadic step gain on other types of eye movements more can be learned about the structure of the ocular motor control system as a whole.

6. Conclusion

The saccadic system exhibits both a rapid and a gradual adaptive capability, with the gradual mechanism being known to depend on the cerebellum. Fast adaptation keeps the eyes from overshooting the target, while gradual adaptation appears to compensate for deficits in the motor system that affect saccadic accuracy (even in an orbital-position-dependent way), or that cause post-saccadic ocular drift. These two systems may interact, with the gradual system depending on the faster one for the derivation of its error signal.

The interaction between the visual sensory system and the saccadic motor system is not limited to the selection of visual targets, but also includes complex interactions that are required to maintain saccadic performance. Saccadic amplitude control may depend on the ability to compare the actual retinal error after a saccade (a visual system signal) with the expected retinal

error (a motor system signal). Post-saccadic drift suppression may require the correlation of retinal slip (a visual system signal) with the antecedent saccade's direction (a motor system signal). By extending the mathematical models of saccadic

motor performance to include adaptation, new roles for visual inputs to the saccadic system are opened to quantitative investigation (an example is given in Ch. 12).