Journal of Experimental Psychology:
Human Perception and Performance
1981, Vol. 7, No. 2, 356–366

0096-1523/81/0702-0356$00.75

Saccadic Plasticity: Parametric Adaptive Control by Retinal Feedback

Joel M. Miller, Tania Anstis, and W. B. Templeton
York University, Downsview, Ontario, Canada

Saccadic gain (the ratio of saccadic amplitude to target eccentricity) was experimentally altered as normal human observers made refixation saccades to the right, which caused step changes in the horizontal position of the target. Eye movements were monitored by diffuse infrared limbus reflection. We found that decreases in saccadic gain reached 60% of completeness, whereas increases were only 25% complete. This asymmetry in adaptive capacity may reflect the saccadic system's need to avoid overshooting a target. With a single target, adaptation is rapid (time constant = 6 saccades); if training is distributed over six different targets, adaptation is considerably slower (time constant = 57 saccades). Gain changes that result from training with a given target do not transfer strongly to other targets in the same horizontal direction and may not transfer at all to targets in the opposite direction. The gain of saccades to one target may be decreased, and simultaneously the gain of saccades to another target at a different distance in the same direction is increased. These results suggest that each element of a sensory–motor structure underlying saccadic plasticity is associated with a particular retinal or spatial sensory locus and can alter its motor response without much affecting the response of neighboring elements. This is consistent with the finding that distributed training slows adaptation.

The saccadic branch of the oculomotor system is responsible for quick and accurate changes of fixation between targets at a given distance from an observer and must deal with threats to refixation accuracy by maturational and pathological changes in the system's parts. Two sorts of problems may result. One is primarily a motor problem: A saccade fails to reach its target, and additional, corrective saccades are needed. Depending on the task, acuity or information acquisition may suffer as a consequence.

The other problem arises in the perception of egocentric direction around the time a saccade occurs. Little quantitative information about eye or target position could be available from the image smeared across the retina during a saccade (Festinger & Holtzman, 1978). Then, the egocentric direction of a target viewed after, relative to before, a saccade would be judged on the basis of the change in position of its retinal image from before to after the saccade and on extraretinal information about the saccade's amplitude. There is good evidence that this extraretinal information is derived from the efferent command rather than from orbital or muscular receptors (Helmholtz, 1866; Skavenski, Haddad, & Steinman, 1972) although the matter cannot be declared settled (e.g., Brindley, Goodwin, Kulikowski, & Leighton, 1976; Steinbach & Smith, 1976). To the extent that extraretinal information is efferent, if the eye fails to move as far as efference specifies, there will be an error in localizing a target after a saccade (assuming it was accurately localized before). If this error is large enough, we might expect a stationary target to appear to have jumped, as when the target itself is displaced (Bridgeman, Hendry, & Stark, 1975; MacKay, 1973).

Disturbances in the saccadic system are thought to be compensated for with a certain

---

This research was supported by Natural Sciences Engineering Research Council (NSERC) (Canada) Grant 296 to H. Ono, NSERC Grant 299 to W. B. Templeton, and a grant from the President's National Research Council Fund to J. M. Miller.

W. B. Templeton is now deceased.

Requests for reprints should be sent to J. M. Miller, who is now at the Department of Ophthalmology, The Wilmer Institute, Room 355, Johns Hopkins University School of Medicine, Baltimore, Maryland 21205.

kind of feedback called *parametric adaptive control*. Should saccades become regularly and unacceptably in error, it is supposed that the operating parameters of the saccadic system are gradually modified to improve future accuracy. Two distinct feedback loops are thought to provide error information to this plastic adaptive mechanism. The first, proposed by Ludvigh (1952), involves a comparison of "intended" eye movement with actual eye movement. Ludvigh took the monitored efferent command or efference copy to indicate the intended eye movement, and feedback from extraretinal proprioceptors to indicate the actual eye movement. Since neither extraretinal feedback itself nor the result of its comparison with efference copy is assumed to be consciously available, this theory does not conflict with the theory that judgments of egocentric direction do not use extraretinal proprioception. There seem to have been no attempts, however, to show that Ludvigh's mechanism actually exists; in referring to it we must remain aware of its uncertain status.

The second parametric feedback loop was demonstrated by McLaughlin (1967) to mediate adaptive plastic motor change if there is a mismatch between intended and actual change in retinal image position. As with Ludvigh's (1952) theory, the intended movement may be presumed to be signaled by efference copy.[1] The two theories are distinguished by the nature of the feedback. In contrast to Ludvigh's extraretinal feedback, McLaughlin considered feedback of the retinal fixation error that exists after a saccade. It is this retinal feedback loop with which the present experiments are concerned.

The motor system of the eye is somewhat unusual in that its function is not to produce a particular movement but to achieve a particular sensory state. The function of a saccade is to bring the image of a target onto the fovea; the ocular rotation must be whatever this requires. Thus, the retinal feedback loop provides a more appropriate referent for system calibration than does the extraretinal loop. Nevertheless, a peripheral efferent disturbance (in the motor endplate, for instance) might be detected by both retinal and extraretinal feedback loops, so that the two systems could work in concert. However, if retinal and extraretinal feedback signals were inconsistent, the system would do well to ignore the extraretinal signal; the disturbance could be in the extraretinal loop itself. If the disturbance were in the retinal signal, it would, in many cases, be caused by damage to the retina, in which case eye movement accuracy becomes unimportant.

Two sorts of studies of parametric feedback are found in the literature. One deals with paretic eyes, either in humans (Abel, Schmidt, Dell'Osso, & Daroff, 1978) or in animals (Optican & Robinson, 1977). However, because subjects in these studies made dysmetric saccades in a more or less normal visual environment, the effects of retinal and extraretinal feedback were confounded. For example, Abel et al. studied a man who suddenly developed a right third nerve palsy. If, with left eye patched, this subject attempted fixation of a spot 10° to his left, he made a saccade of only 4° or 5°. Suppose that the system has information regarding its intention to make a 10° leftward saccade and, for comparison with this, information regarding the outcome of the movement. First, there is extraretinal feedback: Length and tension receptors in the muscles and such orbital pressure receptors as may exist signal that a saccade considerably smaller than 10° has actually occurred. Second, there is retinal feedback: The visual target is still 5° or 6° off fovea. If visual feedback following saccades had been eliminated, the extraretinal feedback loop would have been isolated. However, this was not done.

The other type of parametric feedback study involves normal eyes that faithfully execute the intended saccade and presumably would not give rise to error signals by way of the extraretinal loop. The relationship between eye movement and image movement, however, is made abnormal by optical or electronic means so that an error signal is transmitted by the retinal feedback loop. These studies have used normal human

---

[1] For both Ludvigh's (1952) and McLaughlin's (1967) models, there are other potential sources of intended eye movement signals: The retinal eccentricity of the target prior to the saccade may directly specify the intended movement, or the intended outcome of the saccade—reasonable foveation of the target—might be what is specified.

subjects. McLaughlin (1967) had subjects monocularly view a pair of lights separated horizontally by 10°. On each pretraining trial the subject fixated the right light and on signal shifted fixation to the left light. On the average, a saccade of about 9.75° was made. On training trials, during saccades from right to left, the left light was replaced by a light 1° to its right. Initially this would produce retinal feedback that indicated that the eye had overshot its target by almost 1°. After five to nine training trials, the amplitude of saccades to the 10° target had decreased to 9.07°. In another study (McLaughlin, Kelly, Anderson, & Wenz, 1968), a 10° target jumped 5° back toward the initial fixation point during the saccade. The amplitude of the initial saccade made to the 10° target decreased from 9.4° to 5.8° with comparable rapidity.

There are a few studies in the literature that pursue McLaughlin's (1967) finding. Weisfeld (1972) found that a saccade to a target in one direction could be reduced in size without much affecting the return saccade (which of course is in the other direction). Miller and Festinger (1977) found that plastic changes in the vertical component of an oblique saccade could be easily induced even if the required adaptation was contingent on the direction of the saccade and/or the direction of gaze. Henson (1978) considered that the retinal parametric feedback loop might maintain the normal behavior in which a saccade undershoots its target by about 10% to 15%. He used an optical system (similar in effect to McLaughlin's electronic system) to eliminate normal undershooting and found that after 100–200 saccades, the normal pattern of undershooting had been restored.

There are several reasons why a tendency to undershoot is desirable in a refixation system subject to disturbance. An argument by Becker (1972) suggests the advantage of any simple bias in the initial saccadic response: The subsequent correction may be made more economically, since its direction at least is predictable. Following an unbiased response, the direction of the required correction is not predictable. In humans, an undershooting rather than overshooting bias is chosen, and it is interesting to ask why. Robinson (1973) points out that undershooting insures that the target will be in the same hemifield after the initial saccade as it was before and suggests

> that when a [cerebral] hemisphere has started the analysis process, it is facilitated in reidentifying and relocating the target if it moves but stays in the same hemisphere (p. 81).

Since the saccadic oculomotor system is also lateralized—stimulation of the frontal eye fields (Robinson & Fuchs, 1969) and the superior colliculus (Robinson, 1972), for instance, produce contralateral saccades—such "hemispheric facilitation" might also exist in the motor system. A final virtue of the undershooting strategy is that it requires a minimum of muscular energy (Frost & Pöppel, 1976).

Let us assume, then, that the saccadic system's effectiveness would suffer if for any reason the eye consistently overshot its target. Exaggeration of normal undershooting would not create these difficulties. This implies that a mechanism for plastic change in saccades evolved under pressure to repair overshooting (by shortening the saccade to a given target) but without the need to repair undershooting (by lengthening saccades). We expect to find, then, that plasticity is asymmetric: Saccades to a given target can be shortened by McLaughlin's (1967) technique more quickly or to a greater extent than they can be lengthened. Our first aim in the present study was to examine this prediction.

We have already mentioned that on theoretical grounds, the paretic eye experiments are not directly comparable with McLaughlin's (1967) retinal feedback type of experiment. The two sorts of experiments have also tended to differ methodologically. The subject's visual surroundings during adaptation were fairly normal in the paretic eye experiments but were restricted in the studies with McLaughlin's paradigm. Thus, in the former, saccades were made to many different targets at different eccentricities, directions, and distances, whereas in the latter only a small subset of possible saccades was retrained. Adaptation with McLaughlin's paradigm is fast, having a time constant of perhaps 5–50 saccades. In contrast, time constants of about 1 day, time enough to make perhaps 100,000 saccades, are reported in paretic eye studies. Thus, our second aim was to study the effect of the number of training targets on the speed of adaptive plastic change.

Finally, by means of transfer tests and differential training, we studied the generality of plastic change in the saccade to a given target. This is of interest in connection with the issue of the speed of adaptive change and as a basis for certain inferences about the organization of the saccadic system.

Method

In four experiments eight subjects were tested: Three subjects were informed and experienced (EH, JM, and TA), and five were naive (DW, ME, SM, GG, and MT). All had normal acuity, stereopsis, phoria, and fusional range as measured by the Keystone Professional Performance Test. No more than a single test was made with a subject on a given day.

Each subject viewed binocularly a single 2-mm diameter spot on a Cathode-ray tube (CRT) (Wavetek model 1901C, P4 phosphor) at a distance of 60 cm in an otherwise dark room. The head was held by a dental impression bite. Spot intensity was adjusted to provide a clearly visible spot which, even to the completely dark-adapted eye, left no visible traces on the CRT when it moved and did not perceptibly illuminate the CRT face or its borders. In the various conditions and phases of the experiments, the spot appeared at different horizontal positions, always at eye level. The set of spot positions for a given experiment was centered with respect to straight ahead.

Binocular eye positions were measured by diffuse photoelectric limbus reflection (Biometrics SGH/V-2). Left and right eye position signals were summed and input to circuits that both detected the beginning and end of saccades by a velocity criterion and sequenced the trial events as described below. The summed eye position signal was also recorded on frequency modulated magnetic tape, later written out on a (Beckman Type R) polygraph along with a trace indicating spot position and digitized by hand. These data were then analyzed by computer.

Experiment 1 had three parts: baseline, training, and transfer testing. Experiments 2, 3, and 4 had two parts: baseline and training. Each experiment was preceded and followed by collection of calibration data, which were used to linearize the baseline, training, and transfer data.

On each baseline trial (see Figure 1) the subject tracked the spot as it jumped between its Initial Position F and each Destination Position D1 or D2 to be used in the subsequent training phase (Experiments 1, 2, 3, and 4) or Test Position T to be used in the later transfer phase (Experiment 1). Subjects were instructed to refixate the jumping spot as quickly and accurately as possible. Eye positions during steady fixation of F, D1, D2, and T are denoted f, d1, d2, and t, respectively. The ends of the initial saccades made to D1, D2, and T on baseline trials are called d1′, d2′, and t′, respectively.

Whereas the nonlinearities in the eye position signal remain stable for a given subject and position of the infrared light sources and sensors, the scale factor of the monitor (mV/degree) may drift by 10% or more during a test. Therefore, assuming that before the saccade to D1, D2, or T, fixation of F was accurate (f = F), and that after any corrective saccades had been made, fixation of D1, D2, or T was accurate (d1 = D1; d2 = D2; t = T), we computed a "local" scale factor for each trial that was applied, along with linearity corrections, to arrive at d1′, d2′, or t′.

We calculated saccadic gain as the amplitude of the initial saccade made to a target divided by the eccentricity of that target prior to the saccade. Baseline trials provided measures of the normal, unadapted saccadic gains to the various targets.

After subjects had a short rest with room lights on, the room was again darkened and training begun (see Figure 1). On each training trial the spot jumped to the right, from F to the first position of the destination target, D1. When the end (Experiment 1) or the beginning (Experiments 2, 3, and 4) of the saccade to D1 was detected, the spot jumped to its second position, D2, and remained there for 2 sec before sliding smoothly back to F. Instructions to the subject were simply to look directly at the spot at all times, refixating it as quickly as possible when it jumped and following it smoothly when it moved.

To summarize the results of an entire training run, gain (G) was assumed to vary as

$$G(n) = G_{\infty} - (G_{\infty} - G_0)e^{-n/\tau}, \tag{1}$$

where e is the base of natural logarithms, n is the trial number ($n \geq 0$), G_{∞} is the asymptotic gain, G_0 is the gain before training, and τ is the time constant. The three parameters (G_{∞}, G_0, and τ) were estimated from the data by a least squares method.

The two dependent variables used to characterize a training run were the time constant τ and a percentage of training index based on G_{∞}. Percentage of training is calculated so that if the size of the initial saccade to D1 at the estimated training asymptote is the same as on baseline trials ($d^{*}_{\infty} - f = d1' - f$), percentage of training is 0. If, at the other extreme, it is the same as a normal, baseline saccade to D2 ($d^{*}_{\infty} - f = d2' - f$), training is 100%.

The various training conditions differed in several respects. Two factors had interesting effects on the dependent measures:

1. The direction of the D1 to D2 jump was either (a) back toward F (to train for decreased gain) or (b) further from F (to increase gain).
2. There was either (a) just a single training stimulus (by which we mean the entire F − D1 − D2 sequence) with the F to D1 distance of 8° being the same on every trial, or (b) there were multiple training stimuli with F to D1 distances of 2°, 4°, 6°, 8°, 10°, and 12° presented in a standardized series of random permutations.

Three factors had no systematic effects on the dependent variables; we will consider the means across these factors. These factors were:

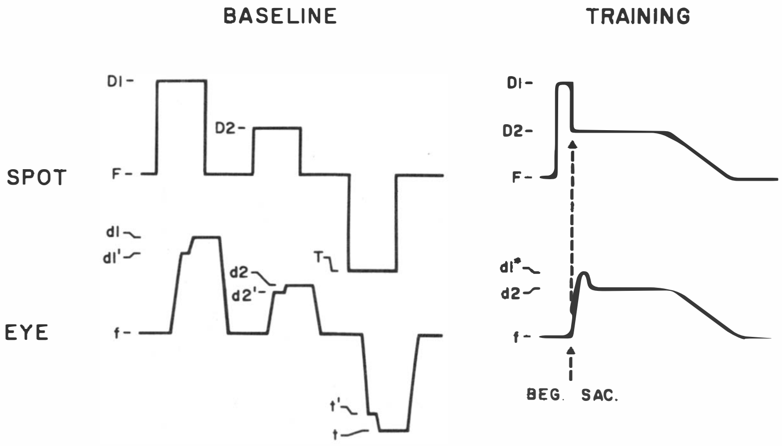


Figure 1. Schematics of target spot positions and corresponding eye positions in baseline and training phases of an experiment intended to decrease gain by 50%. (Baseline spot position T was only presented in Experiment 1. The timing of the D1 to D2 training step is shown for Experiments 2, 3, and 4; in Experiment 1 the step occurred at the end of the saccade.)

3. The spot was switched from D1 to D2 either (a) at the beginning or (b) at the end of the initial saccade made to D1.

4. The size of the D1 to D2 jump was either 25% or 50% of the F to D1 distance.

5. The gain-decreasing and gain-increasing conditions compared were matched with either (a) F to D1 or (b) F to D2 distances the same.

The conditions in Experiments 1–3 are summarized in Table 1. Experiment 4 is described in the Discussion section. The training phase of Experiments 1 and 2 consisted of 100 trials. In Experiment 3 we ran either 100 (single training stimulus) or 200 (multiple training stimuli) training trials. Experiment 4 had 200 trials.

Immediately following training in Experiment 1, we tested whether the experimentally adapted response to D1 transferred to other destination targets, specifically, (a) to a target T the same distance from F as D1 but on the opposite side from F (direction transfer), (b) to a target T 50% closer to F and on the same side as D1 (small transfer), and (c) to a target T 50% farther from F on the same side as D1 (large transfer). Only one type of test was made in a given run. Transfer trials (each having the same form as a baseline trial) were presented as 10 pairs, each pair separated from the next by a block of 10 training trials (to restore the state of adaptation that is altered by the transfer trial itself). We computed percentage of transfer so that if the gain of the initial saccade to T after training was unchanged from that in baseline, percentage of transfer was 0. If there was the same fractional change from before to after training in the gain of the initial saccade to T as there was in that to D1, transfer was 100%.

Table 1
Conditions of Experiments 1-3

Experiment	Subjects	Factors
1	EH, JM, TA	1a, 1b, 2a, 3b, 4b, 5a, 5b
2	DW, ME, SM	1a, 1b, 2b, 3a, 4a, 5a, 5b
3	EH, JM	1a, 1b, 2a, 2b, 3a, 4a, 4b, 5a
3	GG, MT	1a, 1b, 2a, 3a, 4b, 5a, 5b

Results

Adaptation

Figure 2 shows how saccadic gain (size of initial saccade divided by target distance) changed with training in four representative conditions (Experiment 3, Subject EH, 50% D1 to D2 jump). The abscissa is the trial number (by pairs for graphic convenience), and the ordinates are gain and percentage of training.

Figure 2 (A) shows the effects of multiple training stimuli intended to decrease gain. It can be seen that gain did decrease, reaching at asymptote 69% of training with a time constant of 42 trials. Figure 2 (B) shows data from the corresponding condition in which training was intended to increase gain. Here, there was little change at asymptote (18%), and that was achieved slowly (time constant = 146 trials). Figure 2 (C and D) shows gain-decreasing and gain-increasing conditions in which only a single training stimulus was used. Asymptotes were similar to those in the corresponding conditions with a single training stimulus; see Figure 2 (A

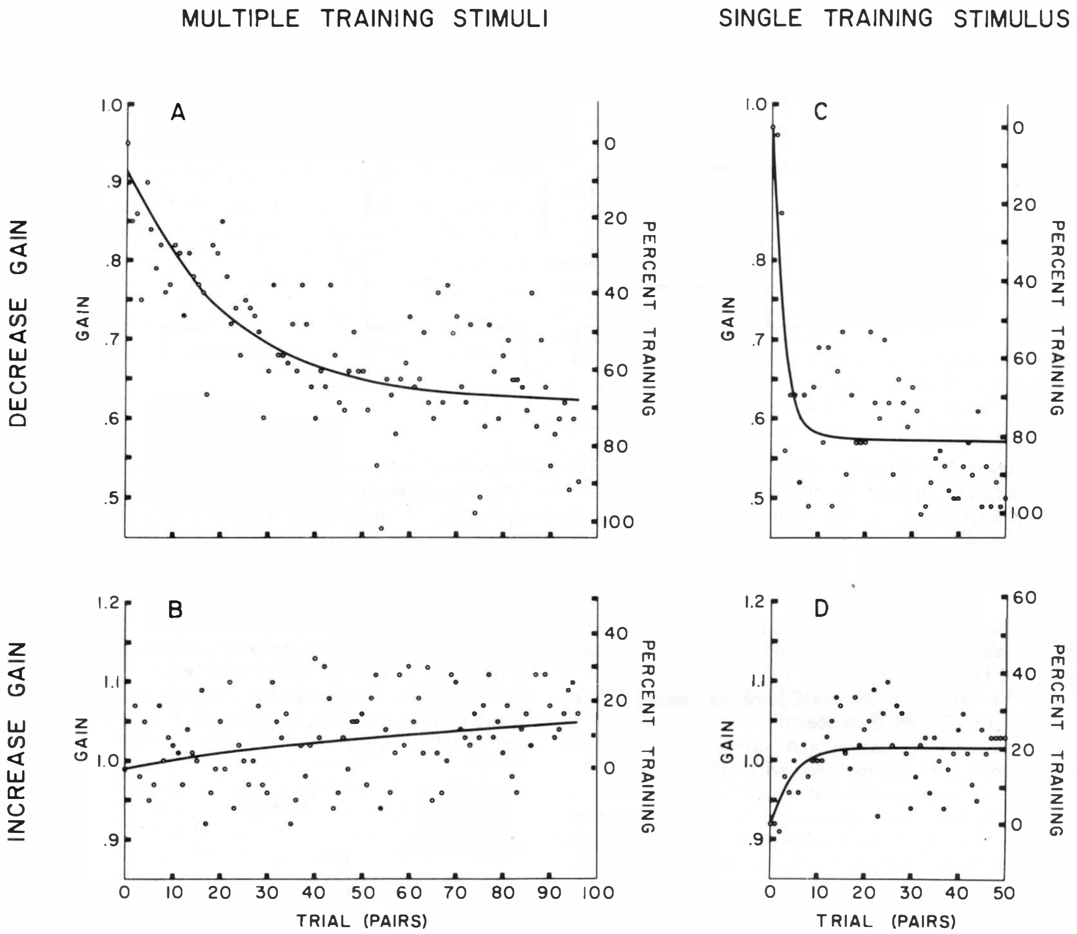


Figure 2. Gain and percentage of training as a function of trial number (by pairs) in Experiment 3 for the 50% D1 to D2 jump and Subject EH. (The circles are experimental data, and the line is the best fitting exponential function. Zero and 100% training points are determined from baseline measures. The baseline value corresponding to 0% training is shown as a data point on the left ordinate.) (A) Multiple training stimuli intended to decrease gain. (The curve is given by $G = .62 + .29\,e^{-n/42}$.) (B) Multiple training stimuli to increase gain: $G = 1.07 - .08\,e^{-n/146}$. (C) Single training stimulus to decrease gain: $G = .57 + .41e^{-n/4.5}$. (D) Single training stimulus to increase gain: $G = 1.02 - .10e^{-n/8.7}$. (In these equations n is the number of trials [not pairs of trials].)

and B). Here gain could be decreased by 81% but increased by only 20% of what would be complete adaptation. These changes, however, occurred much more quickly than in the multiple training stimulus conditions; time constants were 4.5 and 8.7 trials, again with increases in gain being slower.

Consider next the percentage of training asymptotes for the two subjects (EH and JM) who completed all eight conditions of Experiment 3 (Table 2). It is clear that the asymptotes were greater in gain-decreasing conditions: On the average, gain could be decreased to 68% completeness but only increased to 26% completeness, a factor of 2.6. We will see that most other subjects showed the same pattern.

The remaining two factors of Experiment 3 do not systematically affect the asymptotes. Whether the D1 to D2 jump is small (25% of the F to D1 distance) or large (50%) has no systematic effect on the percentage of training asymptote. That is, if the "re-

Table 2
Percentage of Training Asymptotes in Experiment 3

Training stimulus/subject	Gain: Decrease[a] 25% jump	Gain: Decrease[a] 50% jump	Gain: Increase[b] 25% jump	Gain: Increase[b] 50% jump	Decrease/Increase[c] 25% jump	Decrease/Increase[c] 50% jump
Single						
EH	46	81	38	20	1.2	4.1
JM	79	77	32	15	2.5	5.1
Multiple						
EH	68	69	14	18	4.9	3.8
JM	63	59	34	36	1.9	1.6

[a] $M = 68$. [b] $M = 26$. [c] Ratio of $M = 2.6$.

quired" gain change increases, the actual asymptotic gain change increases in the same proportion. The multiplicity of training stimuli is also seen to have no effect on the asymptotes.

It might be objected that gain increases are less complete than gain decreases simply because the system tends not to make large saccades (Bahill, Adler, & Stark, 1975). Consider, for instance, a 50% target jump condition in which D1 is 8° from F for both gain-increasing and gain-decreasing conditions. Complete adaptation to the former requires initial saccades of about 12°, whereas complete adaptation to the latter requires saccades of only about 4°. The size of the required saccade per se and not whether it is larger or smaller than a normal saccade to D1 might conceivably be the important factor. To check this, in Experiments 1 and 2, we used gain-decreasing conditions that were compared with gain-increasing conditions sharing the same D2 (so involving relatively small saccades), as well as the usual gain-increasing conditions matched in D1. There was no significant effect of saccade size—only a slight tendency to lower percentage of training asymptotes in the "small saccade" condition, which is the opposite of what is required by the objection we have been entertaining.

Comparing percentage of training asymptotes in Experiments 1 and 3, it was apparent to us that they were not affected by the time of D1 to D2 switching (Factor 3 in the Method section). We have already discussed the lack of effect of the size of the D1 to D2 jump (Factor 4), saccade size (Factor 5), and multiplicity of training stimuli (Factor 2) on the asymptote. Thus, by taking means across these factors, we are able to bring all the relevant data from Experiments 1–3 to bear on the matter of training asymmetry (Table 3). One difficulty must be pointed out. For four runs (flagged by Footnotes e and f in Table 3) the data were too variable to fit Equation 1. In these cases we report instead a value based on the mean of the last 10 training trials.

Table 3 shows that the asymmetry of percentage of training asymptotes is found in most subjects, there being only one exception (ME) in our sample of eight ($t(7) = 3.26$, $p < .02$, two-tailed). Overall, gain decreases reach 60% completeness, whereas increases reach only 25% completeness, a factor of 2.4.

Time constants are shown in Table 4 for EH and JM. Again, values for the 25% and 50% target jumps are similar. There is a tendency for gain increases to be slower than decreases, but it is not reliable. The variable that affects the time constant most strongly is that of single versus multiple training stimuli, with the former producing much more rapid adaptation. Out of the eight comparisons, however, there is one reversal (decrease gain, 25% jump, JM), and it must be noted that these data are from only two subjects. Unfortunately, we have no additional data that bear on this issue.

Transfer

Following 100 training trials in Experiment 1, we tested whether the experimentally adapted response to D1 transferred to other destination targets, specifically, to a target the same distance from F as D1 but on the opposite side (direction transfer), to a target 50% closer to F and on the same side as D1 (short transfer), and to a target 50% farther from F on the same side (long transfer). The three transfer tests were performed following both gain-decreasing and gain-increasing training. The training and transfer testing stimuli are shown schematically in Figure 3. At the bottom of the figure are the values of percentage of transfer for the three subjects (EH, JM, & TA). Two conclusions may be drawn: There is no significant transfer of adaptation to a target in the opposite direction, and there is an average of 42% transfer to a target in the same direction at a larger or smaller distance than that used in training. The statistical significance of the results concerning transfer to targets in the same direction, however, is marginal. In part, this is probably because of the small number of subjects, the small amount of adaptation "available for transfer" in the gain-increasing conditions, and subject fatigue (although the subject was rested when the baseline measures were taken, he or she had just been through 100–200 training trials when the transfer tests were made). Thus, although the mean values for transfer in the same direction as training are suggestive, we cannot say whether such transfer was weak or merely obscured by large experimental errors.

Table 3
Percentage of Training Asymptotes in Experiments 1, 2, and 3

Subject	Gain Decrease	 Increase	Decrease/ increase
TA[a]	46	26	1.8
DW[b]	83	−12	—
ME[b]	40	44	.9
SM[b,e]	86	38	2.3
GG[c,f]	69	36	1.9
MT[c]	23	18	1.3
EH[d]	65	22	3.0
JM[d]	71	29	2.4
M	60	25	
Ratio of *M*s			2.4

Note. The factors referred to in the following notes are described in the Method section.
[a] Experiment 1: Decrease is the mean of three runs; Increase is the mean of three runs (across Factor 5).
[b] Experiment 2: Decrease is based on a single value; Increase is the mean of two runs (across Factor 5).
[c] Experiment 3: Decrease is based on a single value; Increase is the mean of two runs (across Factor 5).
[d] Experiments 1 and 3: Decrease is the mean of seven runs (across Factors 2, 3, and 4); Increase is the mean of eight runs (across Factors 2, 3, 4, and 5).
[e] Curve fit was not possible; values are the means of the last 10 training trials (see text).
[f] Curve fit was not possible for one run in increase; the mean of the last 10 training trials was used (see text).

Table 4
Training Time Constants and Number of Trials in Experiment 3

Training stimulus/ subject	Gain Decrease[a] 25% jump	 50% jump	 Increase[b] 25% jump	 50% jump
Single[c]				
EH	2	5	9	9
JM	8	6	4	3
Multiple[d]				
EH	29	42	43	146
JM	4	62	80	50

[a] $M = 20$. [b] $M = 43$. [c] $M = 6$. [d] $M = 57$.

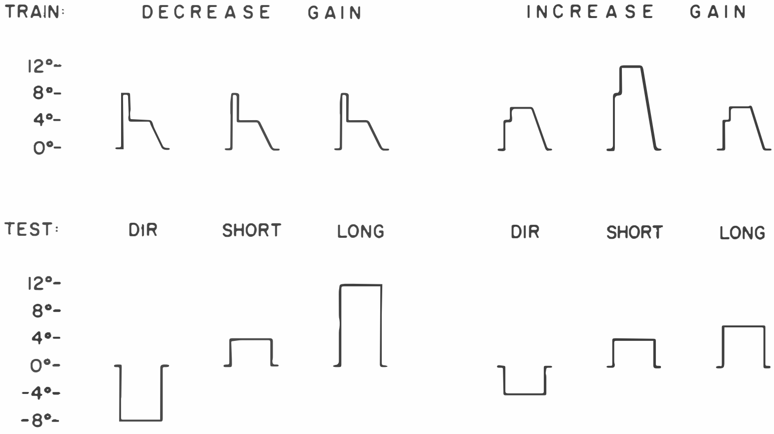
Discussion

Three main conclusions may be drawn from Experiments 1–3:

1. Saccadic gain decreases produced by retinal error feedback are more complete by a factor of 2 or 3 than are gain increases. Gain decreases reach about 60% of complete adaptation, whereas increases reach only about 25% of what is required.

2. The speed of adaptation is substantially reduced if several targets are used rather than just one. In our data, increasing the number of training stimuli from one to six slowed adaptation by a factor of 10.

3. Adaptation to a target in one direction does not transfer to a target in the opposite (horizontal) direction. There probably is



	DIR	SHORT	LONG	DIR	SHORT	LONG
EH:	-18	54	-21	-53	-9	36
JM:	8	62	47	87	25	56
TA:	11	76	31	11	73	75
MEAN:	0	42		15	43	

Figure 3. Schematic diagram of the training and transfer testing stimuli used in Experiment 1. (Direction, short, and long transfer were tested after both gain-decreasing and gain-increasing training. Values of percentage of transfer are shown at bottom for Subjects EH, JM, and TA).

some transfer, however, to a target in the same direction but at a different eccentricity.

It is of course true that of these conclusions, Conclusions 2 and 3 are especially in need of confirmation. The effect of multiple targets on speed of adaptation should be replicated with additional subjects and the transfer tests repeated under conditions that minimize fatigue. (A full-field stimulus would probably be an improvement over the single bright spot we used.) Still, some light is shed, and a number of interesting issues are raised by these results.

Spatial Organization

We expected that decreases in saccadic gain might be faster or larger than increases. There is little evidence that gain decreases are faster, but it is clear that they are larger. Such an adaptive asymmetry is what one would expect in a system oriented to avoid saccadic hypermetria but willing to tolerate hypometria. As we discussed, lateralization of sensory functions, motor functions, or both, could underlie this bias.

Lateralization could also account for the absence of direction transfer in our data. If structures subserving saccades to the right are segregated from those subserving saccades to the left, there would be little mutual influence.

Unfortunately, the transfer data were no more than suggestive concerning the independence of adaptations of different saccades in the same direction. To pursue the matter, we took a different approach in Experiment 4, which may be described briefly:

Subjects DW, GG, and JM were given alternating gain-increasing and gain-decreasing trials. Gain-increasing training was applied to a small saccade (F to D1 distance of 3°) and gain-decreasing training to a large saccade (F to D1 distance of 12°). Switching was at the beginning of the initial saccade made to D1, and the D1 to D2 jump was 33% of the F to D1 distance. A total of 200 trials were run with each subject. All other conditions were the same as in Experiments 1–3.

Percentage of training was calculated separately for gain-increasing and gain-decreasing trials. Adaptation was slow, and asymptote was not reached for either the small or large saccade. Equation 1 could not, therefore, reasonably fit the data, so instead of calculating the training asymptote, we used the mean of the last 10 trials as G_x in calculating percentage of training. The results are given in Table 5.

Subjects GG and JM show a simultaneous decrease in the gain of a large saccade and increase in the gain of a small saccade. DW shows a gain decrease for both large and small saccades, however, the differential gain change of the two is still considerable.

Thus, retraining one or several saccades does not necessarily alter the production of all saccades.

Incomplete Adaptation

We found that retinal error feedback was only able to adjust gain by a fraction of what was required for accurate refixation (60% of a required decrease and 25% of a required increase). We can think of three ways to understand this rather poor performance: (a) It is a poor system, at least with such stimuli as we provided. (b) Parametric adaptation under retinal feedback is a two-stage process with a small and a large time constant, of which we measured only the first. (c) The retinal error feedback loop only provides fine adjustment of gain that is grossly determined elsewhere. Perhaps Ludvigh's (1952) (putative) extraretinal loop could determine such a coarse gain setting.

With respect to Point a, it appears that the more "natural" multiple target situation does not influence the final extent of adaptation, only its speed. Point b could be decided by use of a less fatiguing procedure, which would allow the length of a run to be increased.

Table 5
Percentage of Training at End of 100 Trials to Increase Gain of a Small Saccade, Interlaced With 100 Trials to Decrease Gain of a Large Saccade in Experiment 4

Subject	Increase gain of small saccade	Decrease gain of large saccade
DW	−10[a]	63
GG	7	44
JM	30	64

[a] Gain decreased.

Point c is an interesting one. Since retinal feedback defines the success of refixation, it is well suited to mediate its fine adjustment. Extraretinal feedback is not appropriate for fine adjustment and so (if it is involved in parametric adaptation at all) could only determine gross adjustments.

Another related argument can be made. It is a shame we did not measure the decay of adaptation with repeated saccades to normal, unswitched targets. If we had, we would probably have found that gain quickly returns to its normal value. (Otherwise, EH and JM would have soon become unusable as subjects.) If this is so, then only gain changes departing from some "normal" set point are asymmetrical in completeness; return is always complete, whether it involves an increase or decrease in gain. This set point might be determined by the extraretinal feedback loop.

Speed of Adaptation

As expected, the adaptive time constant increases substantially if saccades are made to several targets instead of just one. But, can this effect account for the factor of 10^4 which exists between time constants reported for "free" training in paretic eye experiments and those reported for the more constrained training with normal eyes using McLaughlin's (1967) paradigm? Perhaps it can, since the former involves saccades to many more targets than the latter in two or three spatial dimensions. We would also expect that adapting saccades with both leftward and rightward horizontal components as in free training would double the time constant compared with training constrained to one side. But there are other differences between the two paradigms, such as the presence in free training of distracting stimuli, which may reduce the salience of refixation errors. Another difference, of course, is the possible involvement in paretic eye experiments of extraretinal feedback.

Conscious Correction

One reason for using naive subjects and for presenting 25% target jumps was to learn if adaptation was affected if the subject did not know that the targets were displaced contingent on his or her saccade, or if the displacements were imperceptible (Bridgeman, Hendry, & Stark, 1975), or both. Direct comparison of adaptation to small and large target jumps in Experiment 3 showed that for informed subjects, there were no differences. Experiment 2, with naive subjects and the small target jump, gave comparable results (except in the case of Subject ME—but even in this case adaptation did occur, though without the usual asymmetry). Naive subjects did not, in fact, perceive the small target jump and were surprised when told about it after the experiment. The fast training with a single training stimulus cannot be explained as conscious correction.

References

Abel, L. A., Schmidt, D., Dell'Osso, L. F., & Daroff, R. B. Saccadic system plasticity in humans. *Annals of Neurology*, 1978, *4*, 313–318.

Bahill, A. T., Adler, D., & Stark, L. Most naturally occurring human saccades have magnitudes of 15 degrees or less. *Investigative Ophthalmology*, 1975, *14*, 468–469.

Becker, W. The control of eye movements in the saccadic system. *Bibliotheca Ophthalmologica*, 1972, *82*, 233–243.

Bridgeman, B., Hendry, D., & Stark, L. Failure to detect displacement of the visual world during saccadic eye movements. *Vision Research*, 1975, *15*, 719–722.

Brindley, G. S., Goodwin, G. M., Kulikowski, J. J., & Leighton, D. Stability of vision with a paralysed eye. *Journal of Physiology*, 1976, *258*, 65P–66P.

Festinger, L., & Holtzman, J. D. Retinal image smear as a source of information about magnitude of eye movement. *Journal of Experimental Psychology: Human Perception and Performance*, 1978, *4*, 573–585.

Frost, D., & Pöppel, E. Different programming modes of human saccadic eye movements as a function of stimulus eccentricity: Indications of a functional subdivision of the visual field. *Biological Cybernetics*, 1976, *23*, 39–48.

Helmholtz, H. von. *Handbuch der Physiologischen Optik* (Vol. 3). Leipzig, W. Germany: Voss, 1866.

Henson, D. B. Corrective saccades: Effects of altering visual feedback. *Vision Research*, 1978, *18*, 63–67.

Ludvigh, E. Control of ocular movements and visual interpretation of environment. *A.M.A. Archives of Ophthalmology*, 1952, *48*, 442–448.

MacKay, D. M. Visual stability and voluntary eye movements. In R. Jung (Ed.), *Handbook of sensory physiology*, (Vol. 7/3). Berlin, W. Germany: Springer, 1973.

McLaughlin, S. C. Parametric adjustment in saccadic eye movements. *Perception & Psychophysics*, 1967, *2*, 359–362.

McLaughlin, S. C., Kelly, M. J., Anderson, R. E., & Wenz, T. G. Localization of a peripheral target during parametric adjustment of saccadic eye movements. *Perception & Psychophysics*, 1968, *4*, 45–48.

Miller, J. M., & Festinger, L. Impact of oculomotor retraining on the visual perception of curvature. *Journal of Experimental Psychology: Human Perception and Performance*, 1977, *3*, 187–200.

Optican, L. M., & Robinson, D. A. Plastic adaptations of saccadic dysmetria. *Abstracts, Society for Neuroscience*, 1977, *3*, 157.

Robinson, D. A. Eye movements evoked by collicular stimulation in the alert monkey. *Vision Research*, 1972, *12*, 1795–1808.

Robinson, D. A. Models of the saccadic eye movement control system. *Kybernetic*, 1973, *14*, 71–83.

Robinson, D. A., & Fuchs, A. F. Eye movements evoked by stimulation of frontal eye fields. *Journal of Neurophysiology*, 1969, *32*, 637–649.

Skavenski, A. A., Haddad, G., & Steinman, R. M. The extraretinal signal for the visual perception of direction. *Perception & Psychophysics*, 1972, *11*, 287–290.

Steinbach, M. J., & Smith, D. R. Effects of strabismus surgery on spatial localization. *Neuroscience abstracts* (Vol. 2, Pt. 1). Bethesda, Md.: Society for Neuroscience, 1976.

Weisfeld, G. E. Parametric adjustment to a shifting target alternating with saccades to a stationary reference point. *Psychonomic Science*, 1972, *28*, 72–74.

Received December 10, 1979 ■