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Adaptive Modification of Saccadic Eye Movements

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Experiments are reported in which the target for a saccadic eye movement was displaced during the saccade. Subjects adapted to the displacement by altering the amplitudes of subsequent saccades to compensate for it. Analysis of kinematic details of the saccade trajectories revealed that the adaptation did not arise from a simple remapping of perceived target locations. Instead, the adaptation appeared to be accomplished by a change in the gain of the saccadic system. The gain change arose primarily from a change in the magnitude of the force pulse for the saccade, not a change in the duration of the pulse. These results have implications for the mechanisms that underlie saccades in normal situations. In particular, people can separately adjust the magnitudes and durations of the force pulses used to produce saccades.

Saccadic eye movements play an important role in people's ability to scan complex visual scenes such as the ones they are confronted with every day. Saccades also represent a nearly ideal movement for researchers interested in motor-control mechanisms because the eye is, for the most part, unaffected by the physical and mechanical restrictions that can influence movements of other body parts such as the head and limbs. Because of their importance, saccades have been studied extensively, in part to learn more about their role in the perception and representation of extrapersonal space and in part to learn about details of the underlying motor-control mechanisms.

Some important insights into the mechanisms underlying the production of movements in general and saccades in particular have been gleaned by studying behavior after altering the normal relation between a movement and the perceptual consequences of that movement. Such studies have examined movements of the limbs and eyes during and after exposure to (a) displaced vision through wedge prisms (Harris, 1965; Held & Gottlieb, 1958), (b) modification of the vestibulo-ocular reflex (Gonshor & Melvill Jones, 1976), (c) proprioceptive illusions induced in the limb (Lackner & Levine, 1981), and (d) illusions caused by paralysis of the eye muscles (Matin et al., 1982). When feedback is available about the perceptual–motor rearrangement, people typically adapt to it quite readily. Substantial effort has been expended to learn about the details of the adaptive processes and the nature of the changes that take place. We continue in that spirit, focusing on the changes that occur in response to an alteration in the relation between an eye movement and the retinal consequences of that movement.

Saccade Adaptation

Many investigators have altered the relation between an eye movement and its perceptual (i.e., retinal) consequences by displacing the target for a saccade during the eye movement (Henson, 1978; Mack, Fendrich, & Pleune, 1978; McLaughlin, 1967; McLaughlin, Kelly, Anderson, & Wenz, 1968; Miller, Anstis, & Templeton, 1981; Semmlow, Gauthier, & Vercher, 1989; Wolf, Deubel, & Hauske, 1984). Typically, subjects will initially miss the displaced target and must make secondary, corrective movements to fixate it. Nevertheless, they soon learn to fixate the target nearly accurately in a single saccade. Thus, the attempt to fixate a particular visual stimulus after adaptation elicits a different-size saccade than does the same stimulus before adaptation.

To make inferences about the mechanisms underlying the adaptive change, researchers have focused on determining the extent to which the adaptive modification influences saccades directed at targets other than those that were initially adapted to. This is of interest because the nature of any transfer of the adaptation may provide insight into details of the oculomotor-control mechanisms. The adaptation does indeed transfer to other targets in the same direction as the adapting stimulus (Deubel, 1987; Miller et al., 1981; Semmlow et al., 1989; Wolf et al., 1984). But several researchers have failed to find transfer of the adaptation either to saccades in a different direction or to saccades directed to the opposite side of fixation (Deubel, 1987; Miller et al., 1981; Semmlow et al., 1989; Wolf et al., 1984).

Parametric Gain Change Versus Spatial Remapping

The finding that there is at least some transfer of the adaptation to other targets suggests that the adaptation is accomplished with a general change in some "parameter" of the motor programs used to produce saccades. Hence this type of adaptation has been referred to as *parametric* (McLaughlin, 1967). Presumably the adaptation involves a change in a gain parameter such that the amplitude of an adapted saccade to a given target would be some constant proportion of the amplitude of saccades to the same target before adaptation. An alternative to such a parametric gain-change mech-

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anism, however, does exist. According to this alternative, adaptation involves a remapping of perceived target locations such that affected saccades would end some constant distance away from their preadaptation end locations.[1]

Distinguishing between these two possibilities has important implications for the nature of the mechanisms underlying saccades. In particular, a change in gain is consistent with the view that saccades are prepared by specifying values for individual parameters of the movement such as direction and amplitude (Abrams & Jonides, 1988) or force and time (Abrams, Meyer, & Kornblum, 1989). Presumably, the gain change could be accomplished by proportionally adjusting the value of an amplitude parameter (e.g., the magnitude or duration of the force pulse applied to the eye; see Abrams et al., 1989). Alternatively, a remapping of target locations is more consistent with the view that saccades are programmed by specifying the final desired location of the eyes (Mays & Sparks, 1980). According to this view, the motor commands needed to fixate a given target location after adaptation would be the same commands that were associated with some other target location prior to adaptation. Of course, these two views of saccade programming are not mutually exclusive. It is entirely possible that saccades are programmed in terms of the desired final location at one level of the oculomotor system and in terms of direction and amplitude parameters at some other level. The question of interest presently is, How are saccades coded at the level at which adaptation occurs?

Attempts to distinguish between these two possibilities have revealed that the adaptation appears to arise from a change in the gain of the saccadic system (Miller et al., 1981; Semmlow et al., 1989; Wolf et al., 1984).[2] Thus, saccades after adaptation to a variety of different target locations may all have amplitudes that are some constant proportion of amplitudes to the same targets before adaptation. Nevertheless, there are some reasons to question this conclusion. First, a gain change is not easy to distinguish from a remapping of target locations if the remapping is not applied uniformly for each affected spatial location. For example, subjects may simply recalibrate the mapping between a retinal stimulus and the saccade needed to fixate that stimulus—but the remapping may be performed in different amounts for different target distances. It would be difficult to distinguish this possibility from a true gain change on the basis of an evaluation of saccade amplitude only. Semmlow et al. (1989) addressed part of this problem by varying both the starting position and the target location for saccades after adaptation, but many issues remain unresolved.[3]

Another reason to question the conclusion that adaptation is due to a gain change involves the absence of any transfer of adaptation to targets on the opposite side of fixation from the initial adapting stimulus (Deubel, 1987; Miller et al., 1981; Semmlow et al., 1989; Wolf et al., 1984). This is because a global change in gain might be expected to affect all saccades, regardless of their direction. Such a possibility is supported by the findings of Abrams and Jonides (1988), who showed that advanced knowledge of the amplitude of an upcoming saccade can facilitate preparation of the movement even without prior knowledge of its direction. Nevertheless, some models of saccade production propose considerable independence between the mechanisms that underlie saccades to the right and to the left (e.g., Becker & Jurgens, 1979), and the transfer of adaptation would not necessarily be expected even if the adaptation was caused by a gain change.

Finally, it is possible that the changes observed in earlier studies are due at least in part to a conscious correction on the part of the subject. This is possible because the sizes of the target jumps during the saccades were often quite large (50% of the initial target step in the McLaughlin et al., 1968, study; 25% or 50% in the Miller et al., 1981, study; and 27%–40% in Wolf et al.'s, 1984, study), and subjects may easily have noticed them. A conscious correction might be expected to be used most completely for saccades that were similar in amplitude to the adapting stimulus, which is consistent with results that have been reported (Semmlow et al., 1989; Wolf et al., 1984). The failure to find transfer of adaptation to the opposite side is also consistent with the possible contribution of a conscious correction: Subjects would be unlikely to apply such a correction to movements that were never inaccurate.

Kinematic Features of Saccadic Eye Movements

Given the importance of the possible conclusions from the results of adaptation studies and the implications that they could have for the mechanisms underlying saccades, we sought to obtain more definitive data by using a new approach. Prior conclusions about saccade adaptation have been based on analyses of saccade amplitudes only. To learn more about the saccadic system, we reasoned that insight into adaptation could be obtained by studying detailed kinematic features of the saccades in addition to their amplitudes.

Saccades arise from muscle activity that consists of a phasic and a tonic component, the so-called pulse step, or pulse slide

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[1] Our use of the term *map* assumes only that each location in space somehow has associated with it a set of motor commands that will generate a saccade to that location. By *remapping* we simply mean that the commands normally associated with a given spatial location before adaptation may be associated with some other spatial location after adaptation. Whether the change occurs at an early peripheral level (e.g., the subject might perceive targets differently after adaptation) or at a more central motor level (e.g., perception might be veridical, but the mapping between perceived locations and motor commands may be rearranged) is an issue that we do not address.

[2] This is true only for adaptation that results in a shortening of saccades (i.e., in which the target for the saccade moves closer to fixation during eye movements in the training trials). Semmlow, Gauthier, and Vercher (1989) concluded that adaptation that results in an increase in saccade amplitude involves a remapping of target locations. We consider only the former type of adaptive modification.

[3] Some of Semmlow, Gauthier, and Vercher's (1989) subjects revealed adaptation whose magnitude depended on the amplitudes of the test saccades but not on the specific starting and ending locations of the saccades, suggesting that those subjects were not remapping saccade motor commands. Nevertheless, part of their analysis depends on the absence of certain correlations between saccade target locations and the size of the adaptive change, and even some of those correlations were marginally significant. As will be seen, our approach allows us to distinguish between the various alternatives on the basis of kinematic differences in saccades before and after adaptation.

step (Sparks & Mays, 1990). Initially, a brief pulse of high force is applied to the eye to propel it from its starting position toward the target. After a fairly rapid decline in force (the slide), the eye muscles arrive at a new steady-state tension sufficient to hold the eye in its new position (the step). Occasionally the pulse and the step are not appropriately matched, and a slow gliding movement of the eye known as a *glissade* occurs after the saccade has ended. When the pulse and step are appropriately matched, the net forces operating on the eye during the saccade can be described by considering properties of the phasic component only. In this case, the movement of the eye can be characterized reasonably well by using only two parameters: the magnitude and duration of the force pulse. Attempts to model details of saccade kinematics with this approach have met with some success (Abrams et al., 1989).

For saccades smaller than about 10°, larger saccades typically require both greater force magnitudes and increased force durations. Furthermore, kinematic features of saccades such as peak velocity, peak force, and duration are usually very tightly coupled to saccade amplitude (Robinson, 1964). Nevertheless, there are several unusual situations in which saccades of a given amplitude may have force-time functions that are not normal (e.g., Becker & Fuchs, 1969; Henriksson, Pyykko, Schalen, & Wennmo, 1980; Zee, Optican, Cook, Robinson, & Engel, 1976). Thus, it is possible that some changes in saccades could be produced by parametric adaptation that would not be evident in their amplitudes. The existing evidence is not definitive: Fitzgibbon, Goldberg, and Segraves (1986) did find some small changes in kinematic features of saccades in the monkey, but Albano and King (1989) reported that adaptation had no effect on kinematics of fairly large (15°–30°) saccades in humans. Thus, it is still not known to what extent saccade kinematics might change after adaptation for moderate-size saccades in humans. There are some good reasons to believe that adaptation would affect saccade kinematics, and some potentially significant conclusions could be made if it did.

One reason to expect a possible change in saccade kinematics after parametric adaptation comes from a recent study on aimed limb movements (Abrams, Meyer, & Kornblum, 1990). They showed that manipulations of the perceived location of a limb-movement target may cause limb movements to be rescaled in force but not time. Given the similarities between saccades and rapid limb movements (Abrams et al., 1989), it would not be surprising if similar changes could occur in force pulses for saccades as well. Additionally, the ample evidence of irregularities in saccadic force-time functions mentioned earlier suggests that the magnitudes and durations of saccades may not be as closely linked as previously believed.

Evaluation of saccade kinematics may help to answer several unresolved questions about saccade adaptation. In particular, if kinematic features of saccades after adaptation are different from those before adaptation (other than changes related to differences in amplitude), then it would be possible to rule out a strategy in which subjects consciously modify their saccades to compensate for the movement of the target. This is because production of an intentional undershoot would be expected to yield a saccade that was indistinguishable from a normometric one that was the same size. The Appendix includes a report of the results of an experiment that confirms that saccades that intentionally undershoot a target are indistinguishable from target-directed saccades of the same size.

If kinematic differences exist in saccades as a result of adaptation, then it would be possible to eliminate the hypothesis that adaptation involves a remapping of target locations. Such a mechanism would require the saccades produced after adaptation to be normal, differing from saccades before adaptation only in amplitude (and amplitude-dependent kinematic features). The reason for this is that "remapping" implies that subjects will emit normal saccades after adaptation; however, they would produce saccades that normally would be associated with different target locations.

Additional details of any kinematic differences in the saccades could provide new insights into saccade mechanisms and motor control in general. In particular, for small saccades, the amplitude is normally adjusted by modulation of both a force parameter (i.e., the magnitude of the force pulse) and a time parameter (i.e., the duration of the pulse). Nonnormal kinematics would imply that a change occurred in one parameter without a corresponding change in the value of the other. Although it is difficult to predict whether the force parameter or the time parameter will be more susceptible to the effects of adaptive modification, whichever parameter is more sensitive will be revealing about the computation of force and time for saccades, and aimed movements in general.

Overview of Present Experiments

In the present experiments, we addressed these issues by examining kinematic features of saccades before and after adaptive modification. In Experiment 1, we explored questions about adaptive transfer by using stimuli that were judged to be more likely to yield transfer than those used previously. An analysis of kinematic features of the saccades suggested that saccades after adaptive modification may differ from those before adaptation in ways other than their amplitudes. Experiment 2 served to rule out some alternative explanations of the source of the changes observed. Finally, in Experiment 3 we pursued details of saccade kinematics and directly compared kinematic features of adapted saccades with those of equal-size normal saccades.

Experiment 1

In this experiment, we used an adaptation paradigm similar to that used previously by others (e.g., Miller et al., 1981; Semmlow et al., 1989) but with several modifications designed to enhance the amount of adaptation and the opportunity to observe transfer to other targets. Subjects made saccades to both the left and right to targets that were 8° and 10° away from fixation. Saccades were produced in three phases of the experiment: a baseline phase, an adaptation phase, and a test phase. Saccades in the baseline and test phases were always to targets that disappeared at the onset of eye movement.

Thus, subjects received no retinal feedback at all regarding the accuracy of these movements, and as a result they might have been less likely to use a strategy involving conscious corrections. In the adaptation phase, all movements were to the 10° target on the right, and the target was displaced 2° closer to fixation during the saccade. The fairly small step size (20% of the initial target displacement) reduced the possibility that subjects would notice the step (Bridgeman, Hendry, & Stark, 1975).

Method

Subjects

Twelve Washington University (St. Louis, MO) students each served in two 40-min sessions on separate days. They were naive with respect to the hypotheses under investigation. Each subject was paid a base rate of $3.50 per session plus bonuses on the basis of performance.

Apparatus

An IBM PC-AT computer controlled the presentation of stimuli and the acquisition of data throughout the experiment. Subjects were seated at a table in a dark room with their eyes 38 cm from a cathode-ray-tube (CRT) display. The subject's head was steadied by means of a dental-impression plate. The left eye was occluded, and the position of the right eye was monitored with a scleral-reflectance device (Gulf & Western, Model 200) mounted on a spectacles frame. The analog output from the eye movement monitor was digitized at a rate of 1000 Hz with a resolution of 0.05°. To calibrate the monitor, samples were taken while the subject fixated at each of 11 evenly spaced points across the CRT. Eye position was computed by using piecewise linear interpolation of the calibration points. Calibration was performed at the beginning of each session and was verified before each trial.

Procedure

At the beginning of each trial, subjects viewed a display that consisted of a small fixation cross at 0° (straight ahead) and a dot that served as the saccade target. The target was either 8° or 10° to the right or left of the fixation cross. (Positions to the left of fixation are denoted by negative values.) Subjects fixated the cross, and then 1 s later the cross changed to a dot, indicating successful fixation and serving as a warning that the imperative signal was about to be presented. One second later, the fixation point disappeared, signaling the subject to make an eye movement to the saccade target. Subjects were advised to fixate the target as accurately as possible with a single eye movement, but they were not pressured to minimize their latencies.

Saccades were obtained in three different phases of the experiment: baseline, adaptation, and test. The sequence of events on each of these types of trials is shown in Figure 1. In the baseline and test phases, the target for the saccade disappeared at the onset of eye movement. All four targets were studied during these phases. During the adaptation phase, subjects looked only to the 10° target (a positive value indicates to the right of fixation). The target jumped from 10° to 8° at the first moment at which the position of the eye differed by more than 1.5° from its position during the first 10 ms after the offset of the fixation point (very close to the onset of eye movement), and it remained illuminated for 1 s.

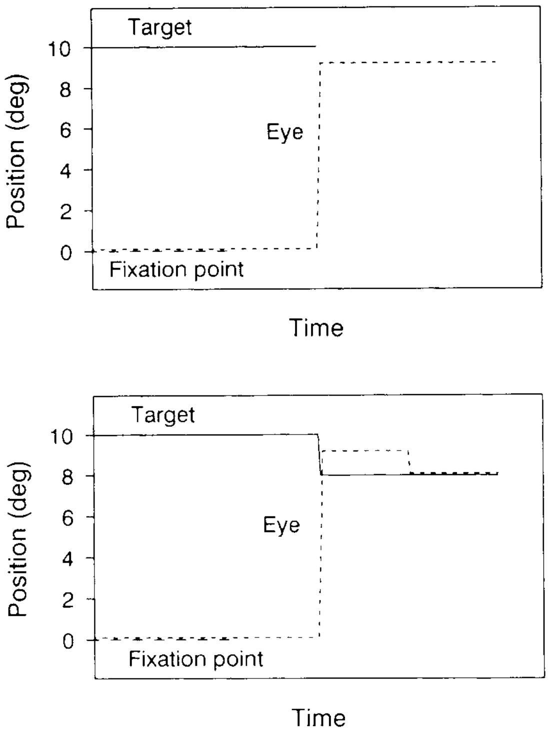

Figure 1. Sequence of events on trials in Experiment 1. (Upper panel: Events on the baseline and test trials [the target was located either 8° or 10° to the right or left of fixation]. Lower panel: Events on adaptation trials [the target always jumped from 10° to 8° at the onset of eye movement]. See text for further explanation.)

Eye Movement Analysis

To identify the eye movements for kinematic analyses, the signal obtained from the eye movement monitor on each trial was differentiated and filtered with a low-pass digital filter with an 80-Hz cutoff. The resulting velocity profiles were analyzed to detect the occurrence of saccades. The beginning of a saccade was defined to be at the first moment that the velocity of the eye exceeded 10°/s and remained above that value continuously for at least 10 ms while subsequently exceeding 35°/s. The end of the saccade was defined to be at the first moment afterward that the velocity of the eye fell below 10°/s. With this algorithm, we were able to detect reliably the occurrence of saccades larger than about 0.7° of visual angle.

The velocity profiles were also used to identify important kinematic features of the saccades such as the time and magnitude of peak velocity. Further differentiation of the velocity profiles produced acceleration profiles that were used to identify the moments of peak positive and negative acceleration and their magnitudes. These kinematic features directly reflect the force and time parameters for the saccades (i.e., force magnitude and force duration): Increases or decreases in force magnitude are reflected by increases or decreases in the peak positive acceleration; changes in force duration affect temporal features of the saccades such as the time to peak velocity or the overall saccade duration (Abrams et al., 1989; Bahill & Stark, 1975a).

Design

In each session, subjects first completed a practice block of 12 trials, 3 to each of the four targets. Data from these trials are not included in the analysis. Subjects then completed a baseline block of 24 trials, followed by two sequences each consisting of 24 adaptation trials and then 12 test trials. The order of saccade targets was randomized within each baseline and test block.

Results

Saccade Amplitude

Figure 2 shows the mean saccade amplitudes from the baseline and test blocks (before and after adaptation, respectively) to each of the four targets studied. As expected, saccades to 8° targets were shorter than those to 10° targets, $F(1, 11) = 1{,}149.9$, $MS_e = 0.07$, $p < .0001$. Additionally, saccades were shorter after adaptation than before, $F(1, 11) = 12.4$, $MS_e = 0.75$, $p < .005$. Finally, the adaptation affected saccades directed to the right only, yielding an interaction between the effects of adaptation and target direction, $F(1, 11) = 10.8$, $MS_e = 0.41$, $p < .01$. For rightward saccades, the mean adaptive change was 1.05°, or 53% of the 2° target jumps, a value that is comparable to those reported by others (Miller et al., 1981; Semmlow et al., 1989). Subjects reported noticing very few if any of the saccade-contingent target steps.

Saccade Kinematics

Further details regarding the adaptive processes are revealed by analysis of kinematic features of the saccades. Figure 3 shows phase planes (velocity vs. position) for the rightward baseline and test saccades measured at five moments in time (movement onset, peak positive acceleration, peak velocity, peak negative acceleration, and movement end). Overall, test saccades were shorter than the baseline saccades (i.e., at the end of the saccade), $F(1, 11) = 24.5$, $MS_e = 0.54$, $p < .001$. Additionally, the position of the eye at the moments of peak positive acceleration, peak velocity, and peak negative acceleration was shorter for test (adapted) saccades than for baseline saccades: Peak positive acceleration, $F(1, 11) = 18.9$, $MS_e = 0.01$, $p < .005$; peak velocity, $F(1, 11) = 54.8$, $MS_e = 0.10$, $p < .0005$; peak negative acceleration, $F(1, 11) = 29.0$, $MS_e = 0.53$, $p < .0005$. Finally, the magnitude of the peak velocity during test saccades was lower than for the baseline saccades, $F(1, 11) = 16.1$, $MS_e = 956.6$, $p < .005$.

Although saccades after adaptation were much shorter in amplitude than those before adaptation, there were no differences in temporal features of saccades produced before and after adaptation. This can be see in Figure 4, which shows mean acceleration-time (force-time) curves for the saccades from Figure 3. As seen in the figure, temporal features of the saccades were unaffected by the adaptation: The times of occurrence of peak positive acceleration, peak velocity, peak negative acceleration, and overall movement duration were the same before and after adaptation: Overall duration, $F(1, 11) < 1$, $MS_e = 8.2$; peak positive acceleration and peak negative acceleration, $Fs(1,11) < 2.1$, $ps > .15$; for peak velocity, $F(1, 11) = 4.1$, $MS_e = 2.9$, $p > .05$. The figure also shows that the values of peak positive and negative acceleration were smaller for the test saccades, although only the latter difference was reliable: Peak positive acceleration, $F(1, 11) = 2.8$, $MS_e = 10.87 \times 10^6$, $p < .15$; peak negative acceleration, $F(1, 11) = 18.5$, $MS_e = 4.53 \times 10^6$, $p < .005$. These differences in the forces for the eye movements were sufficiently large to cause saccades after adaptation to be much shorter in amplitude than those before adaptation, even though the durations of the saccades did not change.

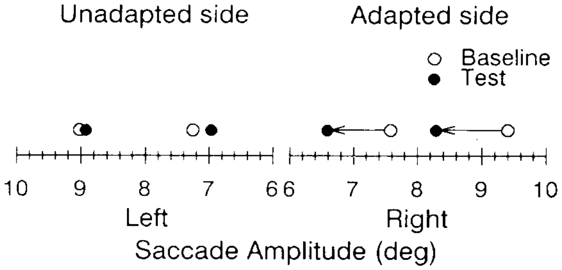

Figure 2. Mean saccade amplitudes from baseline (before adaptation) and test (after adaptation) trials in Experiment 1.

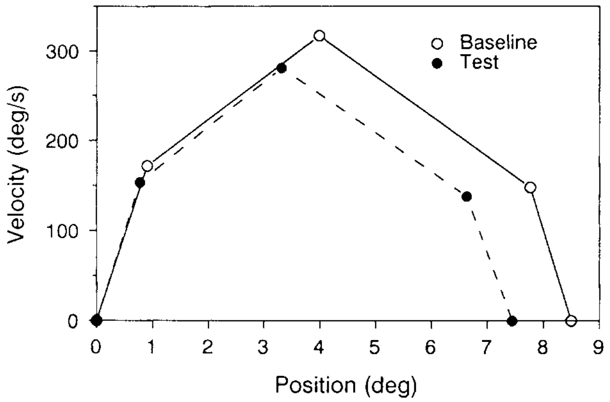

Figure 3. Phase planes for saccades in the baseline and test trials: Experiment 1.

Discussion

The analysis of saccade amplitudes revealed that adaptive modification affected saccades in the direction of the initial adaptation movements only, which is consistent with other investigators' reports (e.g., Deubel, 1987; Miller et al., 1981). Saccades in the opposite direction were unaffected. Analysis of saccade amplitudes, however, is not definitive with respect to the mechanism underlying the adaptation. Analysis of kinematic features of the saccades showed that saccades before and after adaptation had similar temporal features, although saccades after adaptation had smaller forces and thus traveled less far. This suggests that the adaptation arose from a change in some general parameter of the saccade motor program: a parameter related to force (i.e., force magnitude) but not timing (i.e., force duration). This result may yield important insights into oculomotor-control mechanisms because saccades of different amplitudes typically arise as a result of modulation of both force magnitude and duration (considering only saccades less than about 10°; Abrams et al., 1989; Bahill, Clark, & Stark, 1975b). This conclusion, however,

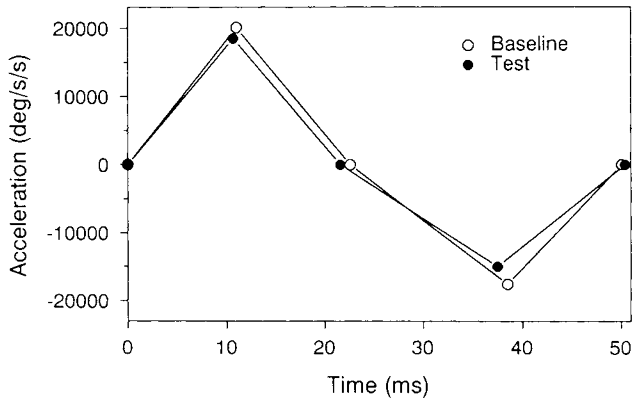

Figure 4. Acceleration-time curves for saccades in the baseline and test trials: Experiment 1.

must be made with caution because it is based on the failure to find differences between temporal parameters of the baseline and test saccades. Experiment 3 pursues this issue with a more powerful design.

Experiment 2

Before continuing our investigation of saccade kinematics, we should consider an alternative interpretation of the results from Experiment 1. In Experiment 1, the baseline and test phases were completely confounded with time, a problem that is common to most adaptation paradigms. Thus, subjects may merely have become fatigued with time in the experiment, and that might be why their eye movements were shorter; there may have been no true adaptation at all. This possibility is supported by the observation that saccade amplitudes are sometimes affected by fatigue (Bahill & Stark, 1975b). Furthermore, because all saccades during the adaptation phase were to the right, rightward saccades would be expected to be most affected by fatigue, which is consistent with the results. To test this possibility, we had subjects produce saccades in the same manner as in Experiment 1, except that the target remained fixed at 8° on every trial in the adaptation phase. Thus, no true adaptation could occur here. If the changes observed in Experiment 1 were due to adaptation, then they should not occur in the present experiment. But if the results of Experiment 1 could be attributed to fatigue, then the same pattern should be observed here.

Method

Subjects

Six Washington University students each served in two 40-min sessions on separate days. They were naive with respect to the hypotheses under investigation. Each subject was paid a base rate of $3.50 per session plus bonuses on the basis of performance.

Apparatus, Procedure, and Design

All aspects of this experiment were identical to Experiment 1 with only one exception: During the adaptation trials, the target did not jump from 10° to 8° as it had in Experiment 1. Instead, the target was initially presented and remained visible at 8°. Thus, subjects produced the same number of eye movements to the same targets and in the same order as they had in Experiment 1, but with no possibility of true adaptation.

Results

Mean saccade amplitudes to each of the four targets in the baseline and test phases are shown in Table 1. As can be seen, there were no differences in the saccades between the baseline and test phases, $F(1, 5) < 1$, $MS_e = 0.86$. But as expected, saccades to the 8° targets were reliably shorter than those to 10° targets, $F(1, 5) = 411.1$, $MS_e = 0.10$, $p < .001$. An analysis between Experiments 1 and 2 confirmed that the effect of adaptation interacted with that of experiment: The adaptation observed in Experiment 1 was not present in Experiment 2, $F(1, 16) = 7.39$, $MS_e = 0.61$, $p < .02$.

Discussion

This experiment shows that the changes observed in the saccades of Experiment 1 did not arise simply as a function of time spent in the experimental session. Instead, they were a direct result of the saccade-contingent target steps that were used in Experiment 1, presumably reflecting adaptation to the modified relation between the retinal location of the target and the eye movement needed to fixate it.

Experiment 3

Experiment 3 was designed to further pursue questions about saccade kinematics that were raised earlier. In particular, the results of Experiment 1 showed that saccades after adaptation differed from those before adaptation as a result of a change in the magnitude of the force pulse used to move the eye but not a change in the duration of the pulse. Such a result may be informative about oculomotor-control mechanisms because for saccades less than about 10°, saccades of different amplitudes are usually produced by modulation of both force magnitude and duration (Abrams et al., 1989; Bahill et al., 1975b). If subjects can modulate the two parameters separately, then the pattern typically observed in normal saccades may reflect important constraints imposed by the "software" of the oculomotor system and not physical limitations of the system.

To examine these issues, in Experiment 3 we compared adapted saccades with normal saccades having the same amplitudes. Such a comparison was not possible in Experiment 1 because there we obtained only normal (baseline) saccades

Table 1
Mean Amplitudes of Saccades (in Degrees) From Experiment 2

	Target amplitude			
Trial block	−10°	−8°	8°	10°
Baseline	8.8	7.2	6.8	8.8
Test	8.7	7.0	6.8	8.8

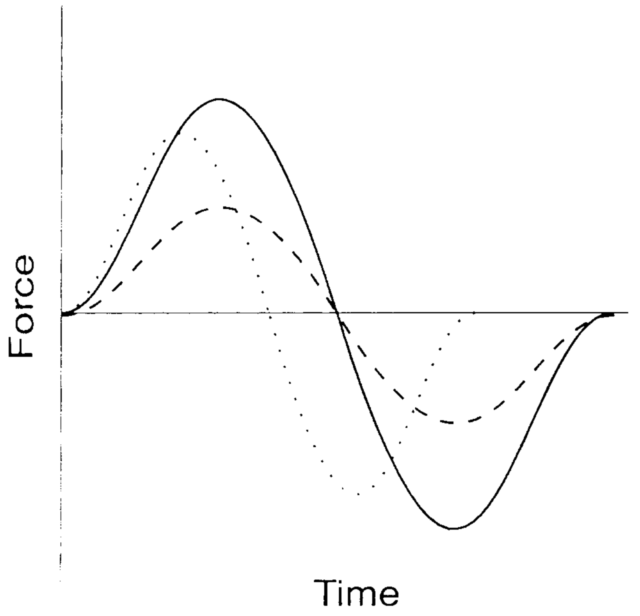

Figure 5. Force-time curves for three hypothetical saccades. (The solid curve represents a large, normal [baseline] saccade. The dotted and dashed curves represent smaller saccades. The dotted curve shows a saccade with smaller force magnitudes and durations compared with the baseline [solid] saccade, whereas the dashed curve shows a movement with the same force duration as the baseline saccade but a lower force magnitude. The dotted and dashed curves could arise from two saccades with the same amplitude but with different kinematic features. See text for additional discussion.)

and shorter adapted saccades. If the adaptation arises because of a change in the magnitude of the force pulse but not the duration of the pulse, then adapted saccades should have durations that are longer and forces that are smaller than equal-size normal saccades. These predictions are illustrated in Figure 5, which shows hypothetical force-time (acceleration-time) curves from three saccades. (Recall that in theory, larger saccades could be produced by either an increase in the magnitude of force applied to the eye, an increase in the duration of force application, or both.) The solid curve represents a large, normal baseline saccade, perhaps to a 10° target. The dotted curve is rescaled from the baseline saccade in both force magnitude and duration, much like normal 8° saccades would be (Abrams et al., 1989). If adapted saccades have normal kinematics, then the adapted saccades would have force-time curves that are indistinguishable from those of equal-size normal saccades, much like the dotted curve in Figure 5. (The procedure we used, described in the following, was designed to ensure that the adapted saccades and the normal saccades would be the same size.) If, however, adapted saccades were produced by a reduction in the magnitude of the force for the saccade but not a corresponding reduction in the duration of the force pulse, as suggested by the results of Experiment 1, then adapted saccades would have a force-time curve more like the dashed curve shown in Figure 5. The dashed curve shows a saccade with the same force-pulse duration as the larger baseline saccade but with a lower force magnitude. As a result, the adapted saccade (dashed curve) has a duration that is longer and forces that are smaller than the equal-size normal saccade (dotted curve). Such a result is exactly what would be expected if adaptation caused subjects to reduce the magnitude of the force pulse for the saccade but not the duration of the pulse. Such a finding would answer several questions about parametric adaptation of saccades and provide new insights into oculomotor-control mechanisms.

To test these possibilities, we obtained saccades after adaptive modification in a manner similar to that used in Experiment 1, and we also obtained equal-size normal saccades from the same subjects.

Method

Subjects

Nine subjects served in the present experiment. Seven were naive with respect to the hypotheses under investigation, and the others were 2 of the authors (Dobkin and Abrams). Each subject served in 8–10 30-min sessions (2 sessions per day). The naive subjects were paid $5 per session.

Apparatus, Procedure, and Design

The apparatus was the same as that used in Experiments 1 and 2. Exceptions to the procedure and design are described in the following. Each subject served in two sessions on a given day. In the first session (adaptation), saccade-contingent target displacements were used to elicit adapted saccades. After a rest period of at least 1 hr, the second session (control) was run. The purpose of the control session was to obtain normal (i.e., nonadapted) saccades of the same size as the adapted saccades from each subject's earlier adaptation session.

Adaptation session. Each adaptation session began with a warm-up block consisting of 10 saccades to a stationary target located at 10°. After the warm-up, subjects produced 24 saccades to a target at 10° that disappeared at the onset of eye movement. These served as baseline trials. Subjects then served in a block of 76 trials that began with 20 adaptation trials followed by 4 test trials. The remainder of the block consisted of sequences of eight adaptation trials followed by four test trials. (The 2 authors who served as subjects received 12 adaptation trials, not 20, at the beginning of this block.) Each adaptation trial began with a target located at 10° that was moved to 8° at the onset of the saccade. One second after presentation of the imperative signal (approximately 700 ms after the end of the saccade), the target (then at 8°) drifted smoothly back to the fixation location. This was done to minimize the need for subjects to produce unnecessary additional saccades. Such saccades, if produced in the presence of fixed visible objects, might attenuate the amount of adaptation. The test trials consisted of targets presented at 10° that disappeared at the onset of eye movement, as in the baseline block.[4]

Control session. At least 1 hr after the end of the adaptation session, subjects served in a control session. All aspects of this session were identical to those in the adaptation session, with only two exceptions. First, on trials corresponding to the adaptation trials in the earlier session, the target was not displaced; it remained visible at 10°. Thus no true adaptation should have occurred during the control

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[4] The 2 authors who served as subjects and 1 naive subject participated with targets located at 8° and 6° (as opposed to 10° and 8°). The pattern of results for each of the target sets was the same, and all results are pooled across the two target sets.

sessions. Second, on trials corresponding to the earlier test trials, the target location was computed by an algorithm designed to yield saccade amplitudes that converged on the mean amplitude obtained from the test trials in the adaptation session. This was accomplished by increasing or decreasing the target amplitude by 0.1° each time two consecutive trials yielded saccades that were both shorter or both longer than the desired amplitude, respectively. We refer to the test trials in the control session as the *control saccades.*

Results

We conducted preliminary analyses to identify the 3 days with the greatest amount of adaptation for each subject. We then subjected data from these days to the additional analyses described in the following.

Saccade Amplitude and Duration

We first compared the baseline and test saccades from the adaptation sessions to determine the presence of adaptation. Saccades on the test trials were significantly shorter in amplitude than those from the baseline trials, $F(1, 8) = 65.7$, $MS_e = 0.273$, $p < .001$ (mean difference = 1.15°). The amount of adaptation was 58% of the 2° target displacement (similar to the 53% observed in Experiment 1). The durations of the test saccades were slightly shorter than those for the baseline saccades, $F(1, 8) = 6.85$, $MS_e = 1.53$, $p < .05$ (mean difference = 0.88 ms).

Next, we compared control saccades (the normal test trials from the control session) with the test (adapted) saccades. As expected, there were no differences in amplitude between the control and test saccades (test mean = 7.76°, control mean = 7.71°), $F(1, 8) < 1$, $MS_e = 0.04$, but the control saccades were significantly shorter in duration than the test saccades, $F(1, 8) = 7.75$, $MS_e = 6.6$, $p < .05$ (mean difference = 1.9 ms).

Saccade Kinematics

Acceleration-time curves from the baseline, test, and control saccades are shown in Figure 6. First, consider the differences between baseline and test saccades. As expected, test saccades had smaller peak positive and negative accelerations than the baseline saccades: Positive acceleration, $F(1, 8) = 30.0$, $MS_e = 1.26 \times 10^6$, $p < .001$; negative acceleration, $F(1, 8) = 16.6$, $MS_e = 3.51 \times 10^6$, $p < .005$. The time of occurrence of peak velocity was also earlier for the test saccades compared with the baseline saccades, $F(1, 8) = 11.6$, $MS_e = 1.9$, $p < .01$.

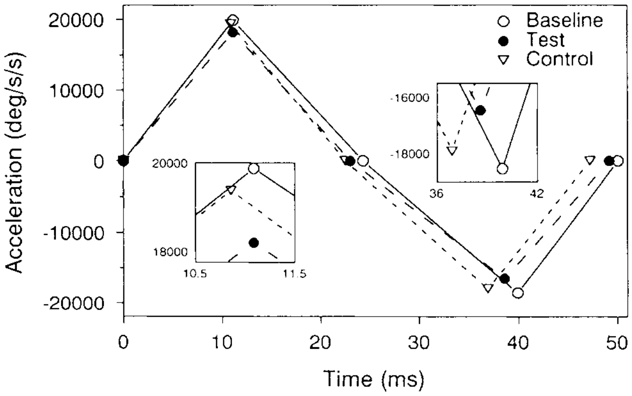

Figure 6. Acceleration-time curves for saccades in the baseline (before adaptation), test (after adaptation), and control (normal, unadapted) trials: Experiment 3. (Insets show details at the peak positive acceleration [left] and peak negative acceleration [right inset].)

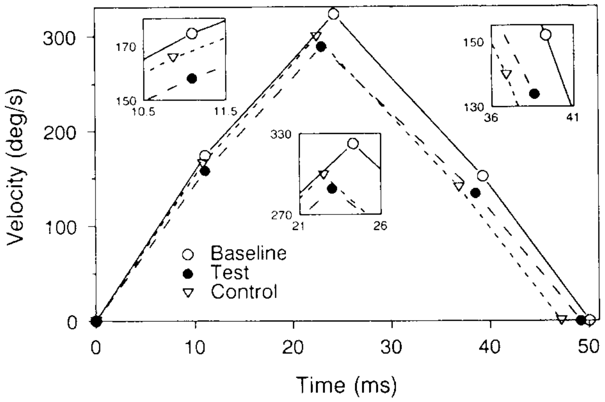

Figure 7. Velocity-time curves for saccades in the baseline, test, and control trials: Experiment 3. (Insets show details at the peak positive acceleration, peak velocity, and peak negative acceleration [left to right], respectively.)

The most important results involve differences between the test saccades and the equal-size control saccades. As seen in Figure 6, test saccades had smaller peak positive and negative accelerations than the control saccades: Positive acceleration, $F(1, 8) = 18.0$, $MS_e = 1.04 \times 10^6$, $p < .005$; negative acceleration, $F(1, 8) = 6.94$, $MS_e = 4.12 \times 10^6$, $p < .05$. Additionally, the peak velocity and peak negative acceleration occurred later in the test saccades: Peak velocity, $F(1, 8) = 6.4$, $MS_e = 0.69$, $p < .05$; peak negative acceleration, $F(1, 8) = 5.3$, $MS_e = 7.1$, $p = .05$. Note that the test saccades had longer durations than the control saccades, as reported earlier.

The acceleration-time curves shown in Figure 6 are very similar to the hypothetical force-time curves from Figure 5. Most important, the test (adapted) and control saccades do not differ in amplitude, but their force-time curves do differ, which implies that the test saccades do not have normal kinematics. In particular, the test saccades have longer durations and smaller force magnitudes than the equal-size control saccades.

Velocity profiles for the baseline, test, and control eye movements are shown in Figure 7. Not surprisingly, velocities of the test saccades at the three points measured were lower than those for the larger baseline saccades, $Fs(1, 8) > 34.0$, $ps < .001$. Differences in instantaneous velocities were also apparent between the equal-size control and test saccades: Test saccades traveled slower than the control saccades at the peak positive acceleration and at peak velocity even though the times were somewhat later in relation to saccade onset: Peak positive acceleration, $F(1, 8) = 14.3$, $MS_e = 53.3$, $p < .01$; peak velocity, $F(1, 8) = 9.73$, $MS_e = 134.6$, $p < .025$.

We also compared the eye movements of the naive subjects with those of the authors to determine the extent to which the overall pattern of results could be attributed to either group. The authors showed differences between control and test saccades in times, velocities, and forces that were in the same direction as those from the naive subjects but with smaller magnitudes. Thus, the sizes of the effects reported before are not inflated by the data from the 2 authors who served as subjects.

Postsaccade Changes in Eye Position

We also analyzed the position of the eye 50 ms and 100 ms after the nominal end of the saccade. We conducted this analysis because it was possible that our saccade-detection algorithm missed some important activity around the end of the eye movement. For example, dynamic overshoot and undershoot (Bahill, Clark, & Stark, 1975a) and glissadic overshoot and undershoot (Weber & Daroff, 1972) might produce some changes in eye position that were not detected by our methods. If so, then it was possible that differences could exist between saccades in the different conditions other than those that we reported earlier. The analysis showed that some small changes in eye position did occur after the end of the saccade. The change in position, however, was less than 2% of the total saccade amplitude. Furthermore, there were no differences in the amount of change that occurred in the 50 ms or 100 ms following the saccade when comparing the baseline and test movements, $ts(8) < 1.5$, *ns*. Similarly, changes in eye position after the saccade were the same for test and control movements, $ts(8) < 1$, *ns*. Thus, we could be confident that the kinematic analysis reported previously captured the important differences between the eye movements in the various conditions.

Discussion

The present results replicate and extend the findings of Experiment 1. Saccade adaptation primarily affected the magnitude of the force pulse used to move the eye, not the duration of the pulse. As a result, the adapted saccades were inappropriately long in duration, and they had forces that were too small compared with normal saccades of the same size.

The conclusion that the adaptation arose from a change primarily in force magnitude and not duration follows from a consideration of the changes that must take place in the force pulses between the normal baseline saccades and the smaller but also normal control saccades. To produce the smaller control saccades, it is necessary to reduce both the magnitude of the force pulse applied to the eye and the duration of the pulse (in relation to the magnitude and duration of the baseline saccades; see Figure 5). If the test saccades had normal kinematics, they too would involve the same reductions in force magnitude and duration as the control saccades (because the test and control saccades are the same size). Only very small differences in force durations exist between the baseline and test saccades, however; temporal differences between the control and baseline saccades far exceed the temporal differences between the test and baseline saccades. On the other hand, fairly large reductions in force magnitude did take place during the adaptive modification, causing the test saccades to have force magnitudes that were even smaller than those for the control saccades (much like the hypothetical force-time curves shown in Figure 5). Thus, the adaptation caused the test saccades to have smaller forces and longer durations than the equal-size control saccades. Because the test saccades arose from adaptive modification of the longer baseline saccades, we conclude that the adaptation took place primarily as a result of a reduction in the magnitude of the force that moves the eye.

The present results allow us to rule out two possible mechanisms that might underlie the adaptive behavior. First, the presence of kinematic differences between the equal-size test and control saccades suggests that the adaptation does not arise solely from a conscious correction on the part of the subject. Any conscious attempt to produce shorter saccades would be expected to yield saccades with normal kinematics (see the Appendix for a demonstration of this). Second, it is also possible to rule out a mechanism that remaps motor commands for saccades onto different targets to correct for the retinal error introduced by the saccade-contingent target displacements. This is because remapped saccades would be expected to have the same kinematics as the normal control saccades of the same size. Thus, the results suggest that the adaptation arises as a result of a change in the gain of the saccadic system caused primarily by the adjustment of a parameter related to the magnitude of the force pulse for the saccade.

The changes observed in the saccadic force pulses caused by adaptation were not exclusively in the magnitude of the force. Rather, we observed differences in some temporal features between the baseline and test saccades, and these temporal differences may themselves be informative. For example, peak velocity occurred somewhat earlier in the test saccades compared with the baseline saccades, and the overall duration of the test saccades was slightly shorter. This could have occurred for several reasons. First, some portion of the adaptive change in amplitude may have arisen from either a conscious correction, from a remapping of target locations, or both. Either of these possibilities would cause adapted saccades to differ from baseline saccades in both force magnitude and duration. Second, it is possible that there is a limited range in which force magnitude and duration can be separately modulated. Any attempt to reduce force magnitude beyond this limit may produce changes in the duration of the force pulse. Support for this possibility was provided by Keele, Ivry, and Pokorny (1987), who showed interactions between the control of force and timing for limb movements. Regardless of these possibilities, comparison of baseline, control, and test saccades shows that the greater part of the adaptive change is accomplished by changes in force magnitude.

General Discussion

Taken together, the results of the present experiments answer several previously unresolved questions about saccade adaptation and oculomotor control. Experiment 1 showed that parametric adaptation elicits shorter saccades in the direction of the initial adaptation trials but has no effect on saccades in the opposite direction (confirming findings of other investigators). Experiments 1 and 3 showed that the effect of the adaptation is primarily on the magnitude of the force pulse used to produce the eye movement; the duration of the pulse is less sensitive to the adaptive modification studied here. These results suggest that the mechanisms underlying adaptation involve a general change in the gain of the saccadic system and not a remapping of target locations.

There was one difference between the results of Experiments 1 and 3 that is worth noting. Experiment 1 revealed no differences in any temporal features between the baseline saccades and the shorter test saccades. But there were some temporal differences between the baseline and test saccades in Experiment 3. This may be due to the slightly larger amount of adaptation present in Experiment 3. Indeed, there are differences in temporal features of the saccades from Experiment 1 that are in the same direction as those observed in Experiment 3, but these differences were not reliable. Given this pattern of results, we can tentatively conclude that temporal changes do indeed take place during adaptive modification, but they are considerably smaller than the changes in force magnitudes.

Implications for Models of Saccade Programming

The present results support the view that saccades are prepared by specifying values of individual parameters such as direction, force magnitude, and force duration (Abrams & Jonides, 1988; Abrams et al., 1989; Becker & Jurgens, 1979; Hou & Fender, 1979). If saccades were programmed solely through a specification of the desired end location of the eye, then kinematic differences would not be expected between adapted saccades and normal saccades of the same size. Some additional conclusions are possible with regard to details of the computation of force and time parameters for saccades. Although kinematic features of saccades such as duration and peak force are usually tightly coupled to saccade amplitude, the present findings show that the computation of the magnitude and duration of the force pulse for saccades (i.e., the force and time parameters; Abrams et al., 1989) are not inextricably linked. Instead, there are situations in which it is possible to modulate the force and time parameters separately.[5] Similar results have been reported for the computation of force and timing for repetitive limb movements (Keele et al., 1987). The fact that force magnitude and duration can be adjusted separately raises the question of why the oculomotor system does not typically do so. One possibility is that the combinations of force magnitude and duration used for normal saccades may be optimal in the sense that they are most likely to yield movements with the briefest possible durations but still an acceptably small amount of spatial error. Such optimization of movement parameters has been observed for rapid limb movements (Meyer, Abrams, Kornblum, Wright, & Smith, 1988).

A question that we cannot yet answer is why the magnitude of the force was more affected by the adaptation than the duration of the force. One possibility is that the timing of oculomotor activity is governed by a mechanism that is accessible to several different perceptual and motor systems (such a purpose is believed to be served by the cerebellum; Kornhuber, 1975). Ideally, such a central timing generator might be expected to be relatively unaffected by a potentially peripheral disturbance in only a single system (e.g., the disturbance introduced by the present method). Thus, force magnitude would be more likely to be modified than force duration when inappropriate movements are detected, which is consistent with the present results. Additional results consistent with this interpretation have been reported in studies of limb movements: Abrams et al. (1990) found that manipulation of the perceived location of a limb-movement target caused subjects to adjust the magnitude of the force applied to the limb but not the duration of the force. Taken together, those results and the present findings suggest that common principles may underlie the computation of force magnitude and force duration for movements of both the eyes and the limbs (see also Abrams et al., 1989).

Implications for Perceptual Processes

The present results may provide insight into perceptual processes. According to Coren (1986), changes in the eye movements that people produce should affect the perception of objects that are scanned with those eye movements. Thus, we might expect the perception of objects scanned while the eye was adapted to differ from that when the eye was in an unadapted state. The present results, however, show that the adapted saccades arise from changes in force magnitude, not force duration. Thus, the effect that adaptation has on perception depends on the extent to which the perceptual system monitors eye movements by evaluating either the magnitude or the duration of the force for the eye movements. If the latter, then perception might be unaffected by adaptation, despite the changes in saccade amplitude produced by the adaptation. Results consistent with this possibility have been reported (McLaughlin et al., 1968). More work is needed to resolve this issue, but the answer may provide considerable insight into the processes involved in monitoring changes in the position of the eyes.

Conclusions

Evaluation of saccade kinematics revealed that subjects can adjust separately the magnitudes and durations of the force pulses used to produce saccadic eye movements. When the usual relation between a movement and its perceptual consequences is altered, subjects modify primarily the magnitude of the force pulse, not its duration. As a result, saccades that are shortened in amplitude have durations that are inappropriately long and forces that are smaller than normal.

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[5] We mean to suggest only that a saccade of a given amplitude can be produced by more than one combination of force parameter and time parameter. Given a desired saccade amplitude, once a force parameter has been selected, the time parameter is necessarily constrained.

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Appendix

We now describe a simple experiment that we conducted to examine kinematic features of saccades that were intentional undershoots of a visible target. Because such intentional undershoots are consciously produced, one might assume that they would have kinematic features that are indistinguishable from normal target-directed saccades of the same size. It is possible, however, that the attempt to produce these unusual saccades would yield movements that differed from normal saccades. The present experiment was undertaken to determine whether kinematic differences exist between intentional undershoots and equal-size normal saccades.

Method

Subjects

Ten subjects naive to the purpose of the experiment each participated in one 45-min session. They were paid $5 for participating.

Apparatus and Procedure

The apparatus was the same as that used in Experiment 3, and the procedure was similar. There were three types of trials: normal, undershoot, and control. On normal trials, subjects made saccades to a target located 10° to the right of fixation. On undershoot trials, subjects were instructed to produce saccades that undershot the 10° target. And on control trials, subjects made saccades directly to a target whose position was controlled by an algorithm designed to yield saccades of the same size as those in the undershoot trials.

Each type of trial began with the appearance of a fixation cross and a target (dot) to the right of fixation. After 500 ms, the fixation cross changed to a warning symbol (described in the following). Eye position was monitored to ensure that subjects were fixating on the warning symbol. The warning symbol remained on the display for 1 s, at which time it disappeared, signaling the subject to make a saccade. On trials in which the subject was required to look directly at the target (normal and control trials), the warning symbol was a dot. On trials in which the subject was to undershoot the target location (undershoot trials), the warning symbol was an asterisk. The screen was blanked 1 s after the movement signal, and the next trial began after a short delay.

Design

Subjects first completed a block of 10 normal trials and then a block of 10 undershoot trials that served as practice. They then served in three blocks of 20 trials each in which the condition alternated between normal and undershoot from trial to trial. Subjects then took a 10-min break, during which the undershoot trials were analyzed. After the break, subjects completed one more warm-up block of 10 trials. They then served in a block of 10 control trials, followed by two blocks of 15 control trials each. Only the last two blocks of control trials were analyzed.

Results and Discussion

Table A1 shows means of several kinematic features of the normal, undershoot, and control saccades. As can be seen, undershoots differ from normal saccades on all dimensions shown, but the undershoots are indistinguishable from the equal-size control saccades. The present results provide no support for the hypothesis that consciously shortened saccades have kinematic features that differ from those of normal saccades of the same size. Instead, we can conclude that conscious adjustments to saccade amplitude yield saccades with normal kinematics. Thus, if subjects had consciously corrected their saccades in Experiments 1 or 3, this would not have caused the adapted saccades in those experiments to differ from the equal-size control saccades.

Table A1
Kinematic Features of Normal, Undershoot, and Control Saccades

Dependent measure	Normal	Undershoot	Control
Amplitude (deg)	9.38**	7.35	7.53
Duration (ms)	50.08*	48.07	47.82
Peak positive acceleration (°/s/s)	22,617.5**	19,205.8	20,658.7
Peak velocity (°/s)	360.04**	299.26	303.39
Peak negative acceleration (°/s/s)	−19,331.0*	−16,545.6	−17,841.2

Note. Normal and undershoot saccade pairs differed significantly, using a t test with 9 df. There were no differences between undershoot and control saccades.
* $p < .05$. ** $p < .0005$.

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