JOURNAL OF NEUROPHYSIOLOGY
Vol. 49, No. 1, January 1983. *Printed in U.S.A.*

Spatial Localization of Saccade Targets. I. Compensation for Stimulation-Induced Perturbations in Eye Position

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SUMMARY AND CONCLUSIONS

1. Monkeys were trained to look to brief visual targets presented in a completely darkened room. On some trials, after the visual target disappeared but before a saccade to the target could be initiated, the eyes were driven to another position in the orbit by electrical stimulation of the superior colliculus. Retinocentric models of the saccadic system predict that a saccade with a predetermined distance and direction based entirely on retinal error will occur. If this were the case, gaze would miss the target location by a distance and direction equal to the vector of the stimulation-induced movement. Spatial models assume that the retinal error signal will be combined with information about the change in eye position produced by stimulation and predict that the animal will look to the position of the target in space.

2. Results confirm the predictions of spatial models. Animals compensated for the stimulation-induced perturbation by looking to the position of the target in space. The result predicted by retinocentric models—a saccade with a direction and amplitude based on retinal error alone—was never observed.

3. The eye movement that compensated for the change in eye position produced by stimulation was a saccade, not a passive, low-velocity movement to an orbital position of mechanical equilibrium established by a tonic pattern of motoneuron activation specified by the visual target. This indicates that a new saccade command, based on stored information about the location of the retinal image and information about the new position of the eyes, had been issued. Computation of the vector of the compensatory saccade does not necessarily increase the latency of target acquisition. The interval between the end of a stimulation-induced saccade and the beginning of the compensatory saccade was frequently 20 ms or less, permitting the animal to acquire the target with a normal latency.

4. Compensatory movements to the remembered location of the target were less accurate than movements to a continuously illuminated target. The reduction in accuracy is attributed to nonveridical spatial memory and eye-position signals, but precise estimates of the magnitude of the error attributable to each of these sources were not obtained.

5. Fixation of a visual target significantly increased the threshold for producing saccades by collicular stimulation. Also, the amplitude of saccades evoked by suprathreshold currents was reduced if stimulation occurred during fixation. This indicates that fixation is more than the mere absence of saccade commands and that an active process prevents saccades during fixation.

6. If stimulation occurred prior to a saccade to a visual target, the vector of the stimulation-induced saccade was affected by the position of the visual target. Changes in the horizontal position of the target affected the horizontal component of the stimulation-induced saccade, and changes in the vertical position of the target affected the vertical component. The magnitude of the interaction effect was a function of the interval between target onset and stimulation onset.

7. Results are consistent with existing models of the saccadic system that assume that saccades are controlled by a local feedback circuit located in the pontine reticular formation.

INTRODUCTION

Many neurophysiological studies are concerned with the broad question of how the neural events initiated by physical stimuli are transformed into the signals needed to control specific movements. To this end, acute microelectrode studies have described the quantitative relationship between the physical dimensions of sensory stimuli and the pattern of neuronal activity observed at successive synaptic relays in the afferent pathways. More recently, the relationship between neuronal activity and motor events such as the contraction of a specific muscle or certain movement patterns have been examined using chronic microelectrode-recording techniques. With these methods, much has been learned about the coding of sensory information, the signals carried by supranuclear neurons serving as inputs to motoneurons, and the types of motoneuron activity that precede specific movements (see, for example, Ref. 25).

A general problem that has not been solved is how signals in sensory coordinates are translated into commands in a motor frame of reference. A complex transformation of sensory data is usually required, since the spatial coordinates of sensory signals (e.g., the activation of a specific region of the retina, or binaural differences in the phase, or intensity of auditory cues) are usually different from the spatial coordinates of the movements they guide (e.g., the direction, velocity, and amplitude of hand or limb movements). Visually guided eye movements may represent one type of sensorimotor interaction that avoids this complexity. Since the receptors move with each rotation of the eye, the necessary transformation could be relatively simple. A retinal error signal (the distance and direction of the retinal image of the target from the fovea) could specify, directly, the vector of the saccade required to bring the image of the target onto the fovea. In this scheme, the position of the eyes in the orbit is unimportant, since there is a one-to-one mapping of retinal loci to saccade vectors. Indeed, early models of the saccadic eye movement system (23, 42) assumed that the oculomotor controller generated command signals based solely on retinal error. Saccades were assumed to be preprogrammed or ballistic, since the vector of the movement was presumed to be determined at saccade onset. The major argument for retinocentric models, other than their apparent simplicity, is the functional organization of the superior colliculus (SC). The superficial layers of the SC receive retinotopically arranged visual afferents (2, 5, 8, 10, 19, 20, 29), while the underlying layers have a corresponding saccadic motor map (22, 27, 32, 37, 41). The alignment of these maps suggests that a simple point-to-point activation of motor cells by overlying visual cells could provide the sensorimotor transformation required by a retinocentrically coded system (23, 27).

Retinocentric models of the saccadic system do not address the problem of how saccades are directed by stimuli from other sensory modalities that are localized in nonretinal coordinates. For example, saccades made to auditory cues (40, 43), localized in a head frame of reference, must be mediated by a separate motor system or else visual and auditory signals must be transformed into a common coordinate system permitting the use of the same motor circuitry. The possibility that visual and auditory cues are localized in a common, spatial frame of reference is supported by the observation that visual targets are not localized, perceptually, on the basis of retinal information alone. Since the eyes and head are not always stationary, there is not a one-to-one correspondence between the direction of a visual stimulus with respect to the head and the location of its image on the retina. Therefore, the perception of visual direction must be based on a combination of retinal, eye position, and other information (see, for example, Refs. 7, 9, 15, 30, 31). Furthermore, recent experiments have shown that (with the head stationary) saccade targets can be localized using a combination of retinal and eye-position signals (6, 17), and spatial models of the saccadic system consistent with these and other data have been developed (24, 44).

The demonstration that saccade targets can be localized using retinal and eye-position information under certain experimental conditions does not provide evidence that saccade targets are normally localized in a spatial frame of reference. The major purpose of the present study was to test directly the predictions of retinocentric and spatial models of the saccadic system. After a visual target was briefly flashed but before a saccade to the target could be initiated, the position of the eyes was changed by electrical stimulation of the SC. As predicted by spatial models, subjects compensated for this perturbation by making a saccade that directed the foveae toward the location of the target in space. Computation of the vector of the compensatory saccade required that information about the retinal location of the target image be combined with information about the stimulation-induced change in eye position. Preliminary reports of these findings have appeared elsewhere (16, 18).

METHODS

Surgical, recording, and stimulation methods

Five macaque monkeys (three *Macaca mulatta* and two *Macaca nemestrina*) served as subjects. Under sodium pentobarbital anesthesia, each animal underwent three sterile surgical procedures. First, four stainless steel bolts were implanted in the skull. A lightweight aluminum frame, attached to the bolts, was used to immobilize the head during training and data-collection sessions. In the second procedure, a coil of fine wire was implanted beneath the insertions of the four rectus muscles to be used as a search coil for eye movement recordings. In two animals, eye coils were implanted in both eyes. After 4–8 wk of behavioral training, a stainless steel receptacle for a microdrive was positioned over a 15-mm-diameter opening centered on the midline at stereotaxic 0 (anterior-posterior).

Eye-position signals were obtained with a sensitivity of at least 0.25°, using the implanted search coil. Exposure of the animal to two alternating magnetic fields in spatial and phase quadrature generated signals in the coil that could be phase detected to produce two voltages—one proportional to horizontal eye position and one proportional to vertical eye position (4).

During data-collection sessions, a 21-gauge stainless steel cannula penetrated the dura and a commercially prepared, parylene-coated, tungsten microelectrode (Bak Electronics) was advanced through the cannula into the target brain area by means of a remotely operated hydraulic microdrive. The microelectrode was used for electrical stimulation and monitoring of extracellular spike potentials. Spike potentials were amplified by an amplifier with high-input impedance and filtered below 300 Hz and above 6 kHz to reduce contamination by the 26-kHz signals of the magnetic fields. Electrical stimulation consisted of a train of 0.2-ms cathodal pulses. Current, pulse frequency, and train duration were varied in the range of 10–50 μA, 300–700 Hz, and 40–60 ms, respectively. Electrode placements and stimulation sites were verified histologically.

Behavioral training

Monkeys were kept on a 23-h water-deprivation schedule for 6 days each week. Before each training or data-collection session, the monkey was put into a primate chair and placed in an electrostatically shielded, sound-attenuated chamber. The head-restraint device was clamped to a rigid bar attached to the frame holding the magnetic field coils. This permitted the monkey's head to be fixed in the same position in the magnetic field each day and simplified eye-position calibration. Each monkey was trained to fixate and follow small (0.1°) visual targets presented either on a large-screen oscilloscope or on a board containing an array of light-emitting diodes (LEDs). The oscilloscope (Hewlett-Packard 1321 with P47 phosphor decaying to 10% in less than 80 ns) provided a viewing area of at least 50° (horizontal) by 40° (vertical) when placed 35 cm from the monkey. The LED array consisted of 33 lights arranged radially with eight arms at 45° intervals. Each arm had four lights equally spaced from 5 to 20° from a center light. A PDP-8/A computer controlled the target lights and delivered reinforcement for appropriate tracking of the targets. Details of the computer system and training procedure have been previously described (36, 37).

Experimental design

The basic experiment consisted of two trial types (control trials and stimulation trials) presented in random sequence with a ratio of approximately three control trials for each stimulation trial.

CONTROL TRIALS. On control trials, the monkey was required to maintain fixation of a center target (represented by the intersection of the axes in Fig. 1) for a variable period (1–2 s). Then the center target was turned off and another target (T) was presented briefly (10–150 ms) at an eccentricity of 5–25°. The monkey was required to

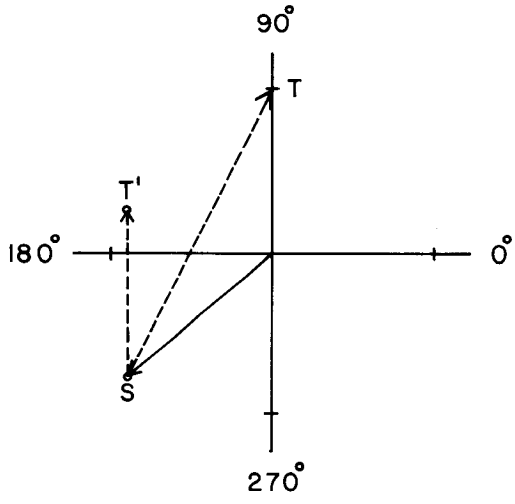

FIG. 1. Schematic representation of stimulation trials and results predicted by retinocentric and spatial models of the saccadic system. On control trials while the monkey was fixating a center target (intersection of the axes), the fixation light was turned off and another target (T) appeared briefly (10–150 ms). On stimulation trials, after target T was turned off but before the saccade to T began, eyes were driven to another position (S) by electrical stimulation of the superior colliculus. If the animal attempts to acquire target T, retinocentric models predict that the animal would look to position T'. Spatial models predict a saccade from S to T. See text for further details.

look to the location of the flashed target T within 400 ms and to maintain that position for 500 ms to obtain a reward (0.1 ml of water). Usually, the location of the eccentric target was randomly selected from 8 to 12 predetermined directions and amplitudes. The criterion for being "on target" was varied by the experimenter over a range of ±2 to ±8°, depending on the duration and eccentricity of the target and on the accuracy with which a particular monkey was able to look to the position of a brief target.

STIMULATION TRIALS. Stimulation trials were the same as control trials except that after target T was extinguished but before a saccade was begun, electrical stimulation drove the eyes to another position (S) in the orbit. If the animal attempts to look to target T, a retinocentric model would predict that a saccade with a predetermined distance and direction (based entirely on retinal error) would occur; i.e., the animal would look to position T'. A spatial model assumes that the retinal-error signal will be combined with information about the change in eye position produced by brain stimulation and predicts that the animal will look to the position of the target in space (position T). The interval between target T onset and the onset of electrical stimulation was systematically varied from 50 to 125 ms.

Two important features of the experimental design should be emphasized. First, except for the fixation target and the briefly flashed target, the task was performed in total darkness. Thus, the targets could not be localized using visual background cues as an external frame of reference. Second, the target at position T was extinguished before the stimulation-induced saccade began. Since the target was not present either during or after stimulation-induced saccades, compensation for the stimulation-induced perturbation could not be based on a visual update of target position.

Other trial types

INTERRUPTED SACCADES. The onset of the saccade to target T was detected by a computer subroutine which, after a specified delay, triggered electrical stimulation. On these trials, a stimulation-induced movement could interrupt the visually elicited movement if the delay was short.

STIMULATION DURING FIXATION. Electrical stimulation was presented during fixation of the center target. Threshold current and the amplitude of the evoked saccade were compared with similar measurements taken for saccades evoked by electrical stimulation during intertrial intervals.

Data collection and analyses

During data-collection sessions, animals were required to track target displacements that varied systematically in radius and angle from an initial center fixation point until the SC had been localized by extracellular unit recording. Then, in total darkness, a series of data-collection trials was presented. Since the general arousal level of the animals gradually declined when in darkness and since some animals developed slow horizontal or vertical drifts of the eyes in the dark, blocks of data-collection trials were intermixed with blocks of trials presented in the light.

Digital codes representing the following experimental parameters were stored on digital magnetic tape: target onset, offset, and duration; rate, duration, and current of the stimulation train; horizontal and vertical eye-position signals (sampled at 500 Hz); time of target acquisition; and time of reinforcement. The sequence of target, stimulation, and eye movement events could be reconstructed with a resolution of 2 ms.

During data-collection trials, two on-line displays were monitored by the experimenter. The trajectories of each eye movement and representations of the visual targets were displayed on an X-Y oscilloscope. At the end of each trial, horizontal and vertical eye-position signals, target onset and offset, and stimulation onset and offset

were displayed as a function of time on a Tektronix 613 storage oscilloscope. These displays were used to modify electrical stimulation and target parameters interactively.

Off-line analyses of the data were conducted using the Vision Research Center (CORE) computer facility. The trajectories and time course of saccades occurring on control and stimulation trials for each stimulation site were reviewed by examining *x-y* or time plots of horizontal and vertical eye positions on a graphics terminal. Programs were written that provided measurements of *1*) the accuracy and peak velocity of visually induced saccades on control trials, *2*) the average vector of stimulation-induced saccades, *3*) the peak velocity of eye movements compensating for the stimulation-induced perturbation, and *4*) the error (difference between target and eye position) remaining at the end of the compensatory movement. The peak velocity of compensatory saccades (which were usually oblique movements) was computed using the equation $V_p = \sqrt{V_h^2 + V_v^2}$, where V_p = peak velocity of the oblique saccade, V_h = peak velocity of the horizontal component, and V_v = peak velocity of the vertical component.

RESULTS

Basic observations

Typical eye movements occurring on stimulation trials for one stimulation site are illustrated in Fig. 2. Each dot represents both

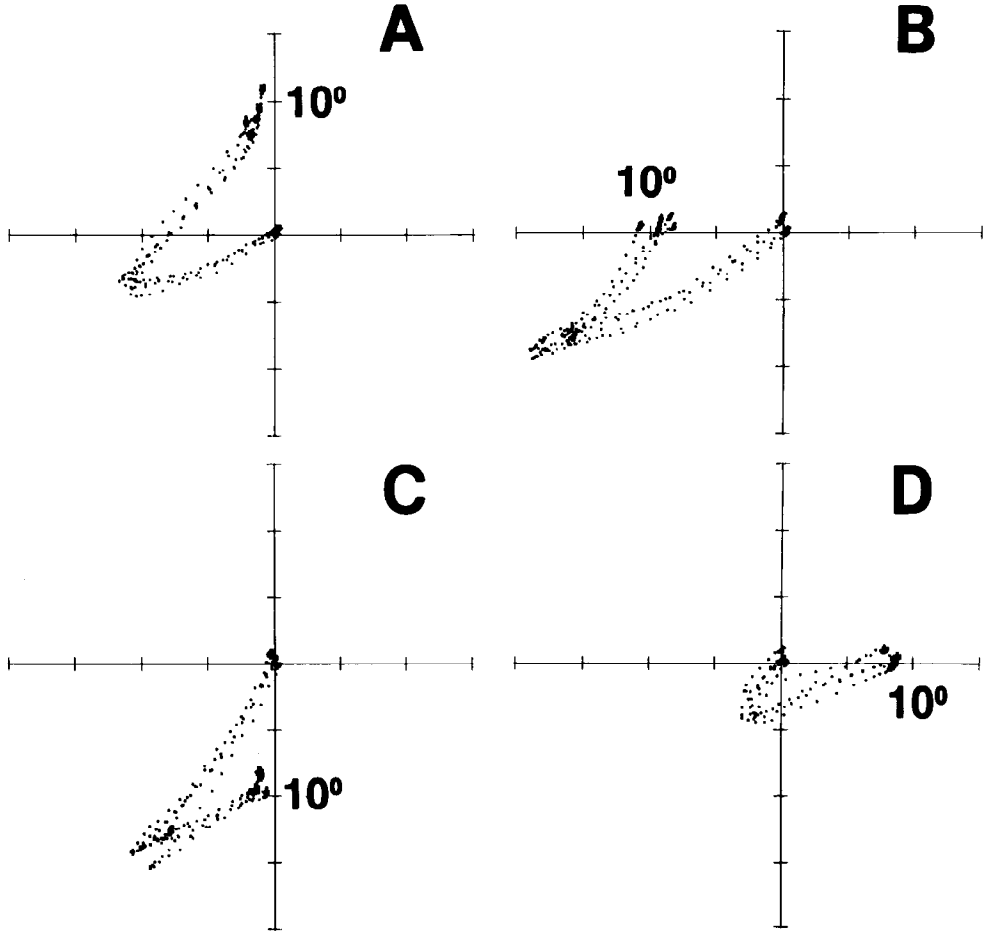

FIG. 2. Trajectories of typical eye movements occurring on stimulation trials. The target was presented 10° above (*A*), to the left (*B*), below (*C*), or to the right (*D*) of fixation. Trials were presented in random order and subsequently sorted according to target position. For all trials illustrated in this figure, the same collicular site was stimulated with identical stimulation parameters. For trials with a specific target position, little variability was observed in the vector of the stimulation-induced saccade or in the vector of the compensatory movement. See text for further details.

the horizontal and vertical position of the eye sampled at 2-ms intervals. The trajectories of eye movements occurring on five trials are superimposed on each *x-y* plot. Referring to Fig. 2*A*, electrical stimulation of the SC occurred after the offset of the brief visual target flash (10° above the fixation point) but before the animal initiated a saccade to the target position. In this case, stimulation of the SC produced a downward and leftward movement of the eyes. After a brief delay, a compensatory eye movement was made to the approximate position of the target. Comparable data using the same stimulation site but with the target appearing 10° to the left, 10° below, and 10° to the right of the fixation target are shown in *B*, *C*, and *D*, respectively. In each case the monkey made a compensatory movement to within a few degrees of the actual target position.

Results obtained using a different stimulation site are illustrated in Fig. 3. The *x-y* plots of the eye movements occurring on five stimulation trials are superimposed for target

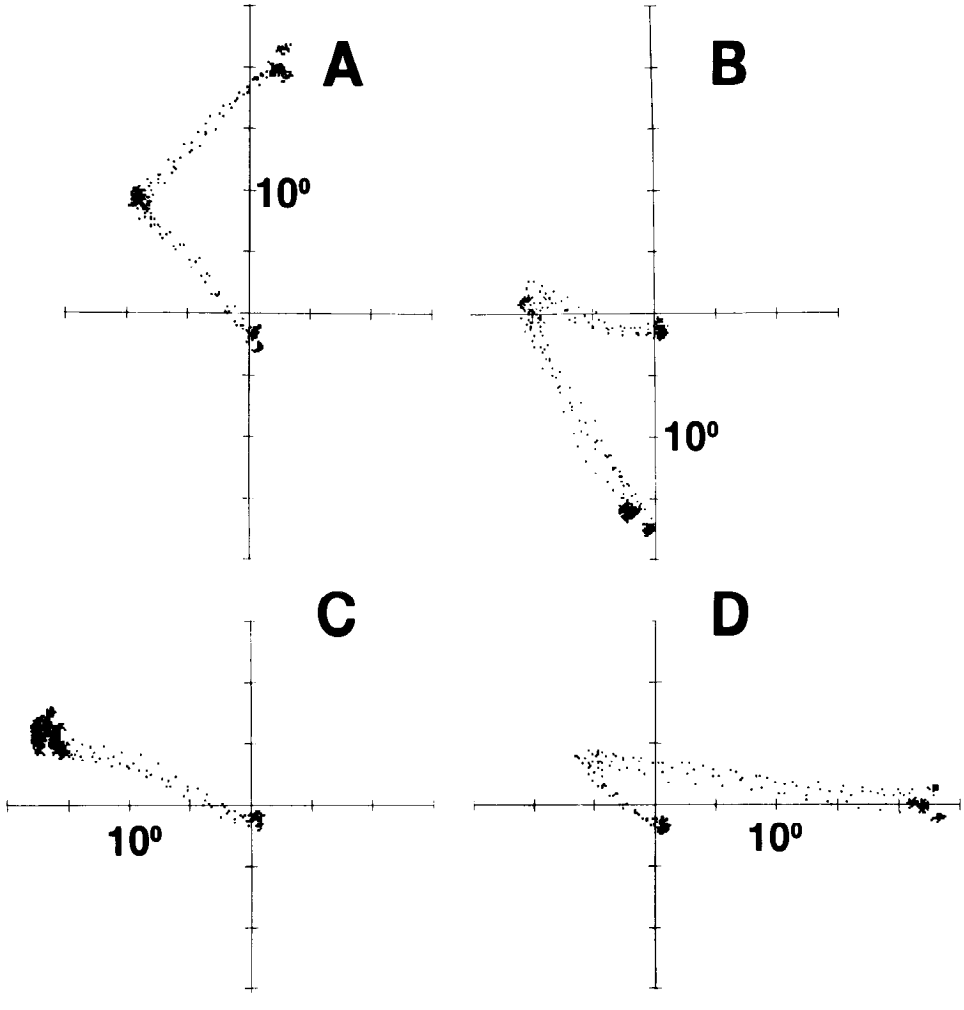

FIG. 3. Trajectories of stimulation-induced and compensatory eye movements on stimulation trials using a different collicular-stimulation site. Brief targets appeared 20° above (*A*), below (*B*), to the left (*C*), and right (*D*) of fixation. Five trials are superimposed in each panel. In *C*, collicular stimulation drove the eyes to the approximate position of the target and a compensatory movement failed to occur. Note that (as in Fig. 2) even though the same collicular site was stimulated using identical stimulation parameters, the vector of the stimulation-induced movement varied depending on the location of the brief target.

positions 20° above (*A*), 20° below (*B*), 20° to the left (*C*), and 20° to the right (*D*) of fixation. Stimulation of the SC produced a leftward and upward movement. For the trials shown in Fig. 3*A*, *B*, and *D*, a compensatory movement to the position of the briefly flashed target followed the stimulation-induced saccade. As illustrated in Fig. 3*C*, if stimulation drove the eyes to the approximate position of the target, a compensatory movement did not occur.

Results similar to those illustrated in Figs. 2 and 3 were obtained from 65 stimulation sites in five monkeys. In general, regardless of the position of the target in space or the vector of the saccade required to look at the target following the stimulation-induced movement, a compensatory saccade occurred that directed gaze to the approximate position of the target in space. We never observed the result predicted by retinocentric models—a saccade with a direction and amplitude based on retinal error alone.

Two trials in which a stimulation-induced saccade interrupted a visually triggered saccade are illustrated in Fig. 4. The *x*-*y* plots (*A*, *B*) display the trajectories of the saccades, and the time course of the horizontal and vertical components of the movements are shown below. Even though the visually elicited saccades were interrupted in midflight at the end of the stimulation-induced saccade, a saccade to the approximate position of the target was initiated after delays of 80 ms (*A*) or 20–30 ms (*B*).

Accuracy of compensatory movements

The difference between eye position and target position was computed at the end of the visually triggered saccade (control trials) or at the end of the compensatory saccade (stimulation trials). This error (expressed in degrees of visual angle) was used as a measurement of the accuracy of target acquisition. Since accuracy is affected by many variables, including target duration and location, criteria used for reinforcement, and level of motivation, the difference in accuracy observed on control and stimulation trials, not absolute accuracy, was chosen for analysis.

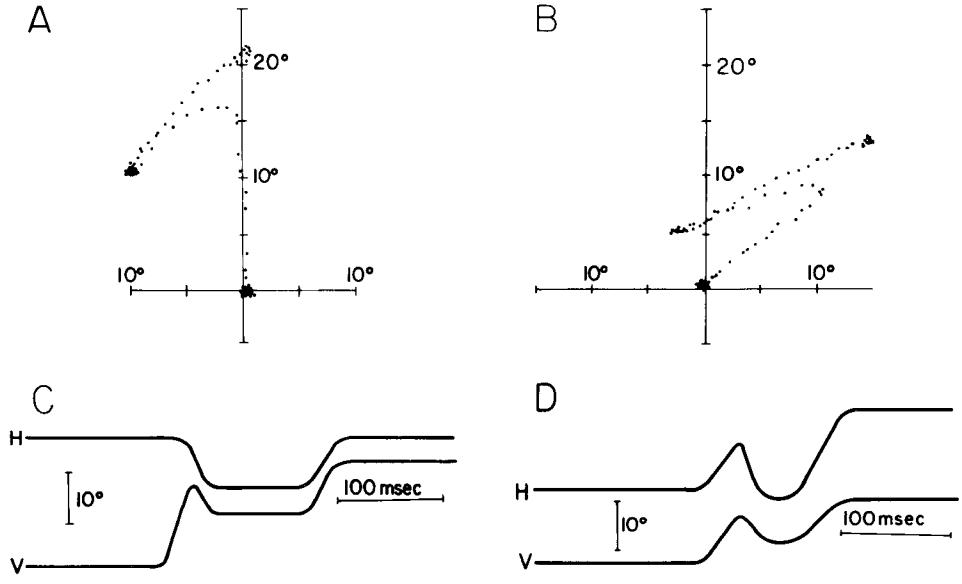

FIG. 4. Target acquisition on trials in which the stimulation-induced saccade interrupted a visually triggered saccade. *A*: a target was flashed 20° above fixation for 100 ms. After a normal-latency interval, the animal initiated a saccade toward the target position but this visually triggered saccade was interrupted by a stimulation-induced movement, which drove the eyes downward and to the left. Nevertheless, after approximately 80 ms, the animal made a saccade that compensated for both the interrupted visually triggered saccade and the stimulation-induced movement and directed gaze to the approximate position of the target in space. *B*: similar to *A* except the target appeared 20° from fixation at an angle of 45°. *C*: separate plots of horizontal (H) and vertical (V) eye position for the trial illustrated in *A*. *D*: horizontal and vertical eye position as a function of time for the trial shown in *B*.

Accuracy data for four target positions and five stimulation sites are presented in Table 1. In general, target acquisition was less accurate on stimulation trials than on control trials. Note, however, that the amplitude of the compensatory saccade on stimulation trials, in many cases, was greater than the amplitude of the acquisition saccade on control trials (see Fig. 2). Furthermore, if the stimulation-induced movement occurred late in the reaction-time period, the interval between the offset of the visual target and the onset of the movement to acquire the target was greater than on nonstimulation trials. Thus, a reduction in accuracy on stimulation trials could reflect increases in error related to the amplitude of the required movement and/or a degradation in the short-term spatial memory of target location. Additionally, some combinations of stimulation-induced saccades and target positions resulted in more error than others. For example, when the stimulation-induced movement drove the eyes to a position near the target, compensatory movements failed to occur, resulting in a greater error.

Although the differences in accuracy observed on control and stimulation trials are statistically significant except for trials in which compensatory movements failed to occur, the magnitude of the differences was small. Moreover, the compensatory saccades directed gaze remarkably near the previous position of the target, considering an unanticipated displacement of the eyes necessitated a large-amplitude saccade to the remembered target location.

Velocity of compensatory movements

Following stimulation, the compensatory eye movement to the target position had a velocity within the range of normal saccades. Often it was possible to find within a single session several pairs of visually triggered and compensatory eye movements with the same trajectory. For these matched pairs of movements, plots of the velocity of the horizontal and vertical components of the movements were, in most cases, identical. A scatter diagram and least-squares line of best fit of saccade amplitude and peak velocity for visually triggered saccades are plotted in Fig. 5*A*. Similar data for compensatory movements are shown in Fig. 5*B*. It is clear from the similarity of the slope and y intercept constants that compensatory movements have normal saccadic velocities.

TABLE 1. *Accuracy of target acquisition: comparison of brief target and stimulation trials*

Stimulation Site	Target Eccentricity, deg	Trial Type	Target Position: Right	Left	Up	Down
M97-1-68	10	Stimulation	1.38 ± 0.5 (33)	2.80 ± 0.38 (13)	1.29 ± 0.70 (22)	2.02 ± 0.80 (22)
		Brief target	1.07 ± 0.42 (13)	1.93 ± 0.40 (17)	0.91 ± 0.51 (19)	1.43 ± 0.52 (23)
		Diff	0.31	0.87	0.38	0.59
M91-1-72	10	Stimulation	3.50 ± 1.59 (20)	2.01 ± 1.20 (22)	1.70 ± 0.86 (35)	3.60 ± 1.61 (23)
		Brief target	0.90 ± 0.39 (25)	0.84 ± 0.39 (38)	2.01 ± 1.19 (34)	1.26 ± 1.62 (39)
		Diff	2.60	1.17	−0.31	2.34
M91-1-70	20	Stimulation	2.18 ± 0.90 (33)	8.17* ± 1.0 (23)	2.73 ± 1.3 (29)	4.48 ± 1.4 (25)
		Brief target	4.23 ± 1.1 (21)	4.62 ± 0.9 (11)	3.12 ± 1.0 (24)	2.11 ± 0.9 (17)
		Diff	−2.05	3.55	−0.39	2.37
M91-1-75	10	Stimulation	4.33 ± 0.96 (25)	7.57* ± 0.93 (25)	1.47 ± 0.67 (28)	6.35 ± 1.29 (22)
		Brief target	1.63 ± 0.33 (9)	1.45 ± 0.57 (15)	2.07 ± 0.64 (10)	3.63 ± 1.05 (13)
		Diff	2.70	6.12	−0.60	2.72
M247-1	15	Stimulation	4.35 ± 0.95 (19)	6.42 ± 0.95 (19)	1.89 ± 0.79 (25)	5.46 ± 1.05 (17)
		Brief target	2.02 ± 0.46 (29)	0.56 ± 0.28 (26)	1.5 ± 0.5 (35)	0.74 ± 0.5 (22)
		Diff	2.33	5.86	0.39	4.72

Values are average errors in degrees ± SE. Values in parentheses are numbers of tests. * No compensatory saccade.

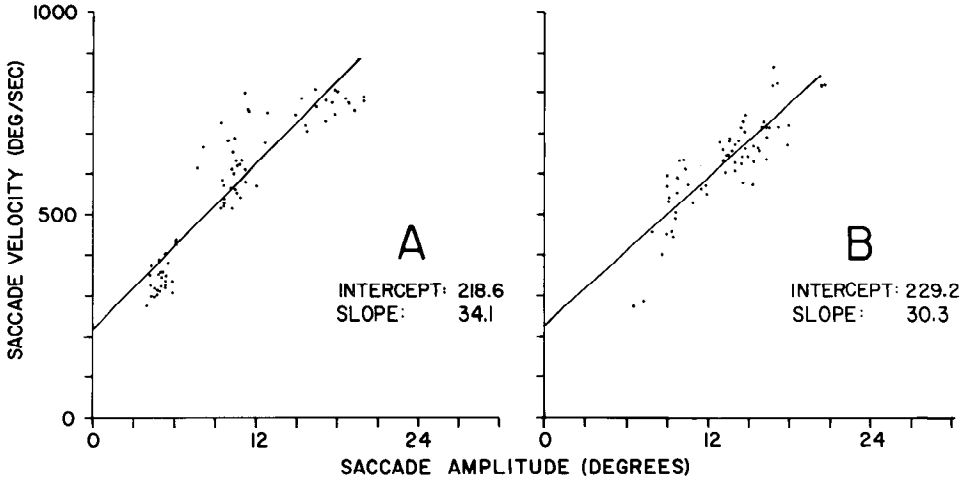

FIG. 5. Velocity of visually triggered and compensatory eye movements. *A*: scatter diagram and least-squares line of best fit of saccade amplitude and peak velocity for visually triggered saccades. *B*: scatter diagram and least-squares line of best fit of saccade amplitude and peak velocity for movements compensating for the stimulation-induced saccade. These data were obtained from trials presented in random sequence within the same data-collection session.

Timing of compensatory saccades

This section describes the general pattern of temporal interactions observed between stimulation-induced and visually triggered saccades. When stimulation occurred early in the reaction-time interval (20–30 ms after target onset), following the stimulation-induced movement the animal looked back to the position of the (now absent) fixation target. Later the animal made a second saccade to the remembered location of the eccentric target. When stimulation followed target onset by 50–100 ms, a return saccade to the location of the fixation target was not observed. The eyes remained at the position reached after stimulation and a compensatory saccade to the target location occurred with a normal latency (see Fig. 6*B*). The occurrence of the stimulation-induced saccade did not increase the time required to acquire the target (compare Fig. 6*A* and *B*). With greater delays in stimulation onset the interval between the stimulation-induced and visually triggered saccades was shortened, with the compensatory saccade occurring at the time the visually triggered saccade was normally observed. However, when stimulation onset occurred near the onset of a visually triggered saccade, the latency of target acquisition was increased (Fig. 6*C*). This pattern of interaction depends on the motivational and attentive states of the monkey. Often, after 600–700 trials, the reaction time of the animal increased by 20–50 ms and corresponding delays in stimulation onset were required to produce results comparable to those depicted in Fig. 6.

Effect of fixation on probability and amplitude of stimulation-induced saccades

Fixation of a visual target reduced the probability of evoking an eye movement by stimulation of the SC. The probability of evoking a stimulation-induced movement is plotted as a function of stimulation current for two stimulation sites in Fig. 7. The threshold current was lower when stimulation occurred randomly between trials (open circles) than when stimulation occurred during fixation of a visual target (filled circles). Similar effects of fixation on threshold current were observed at all stimulation sites within the SC.

The amplitude of saccades evoked by SC stimulation are also affected by fixation. Figure 8 plots the amplitude of stimulation-induced saccades as a function of stimulation current for the same stimulation sites shown in Fig. 7. At each current level the amplitude

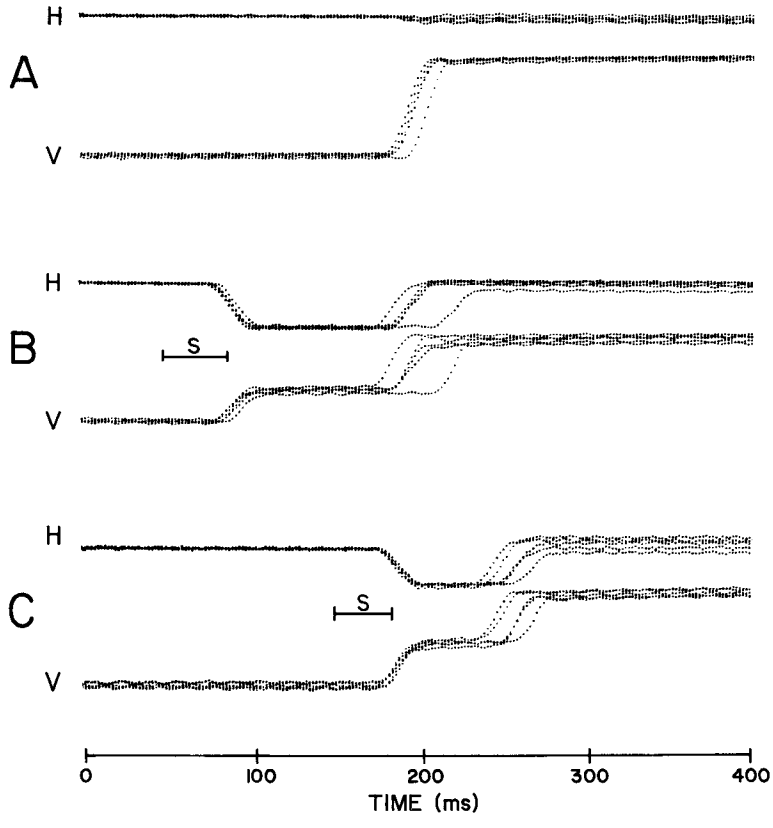

FIG. 6. Temporal interaction of stimulation-induced and visually triggered saccades. *A*: plots of five control (initial target to target A; 0–A) saccades to a target 20° above the fixation point. H, horizontal eye position; V, vertical eye position. *B*: plots of five trials in which stimulation began 50 ms after target onset. Stimulation (S) drove eyes upward and to the left but usually the occurrence of the stimulation-induced movement did not increase target-acquisition time. *C*: plots of five trials in which stimulation (S) began 150 ms after target onset. See text for further details.

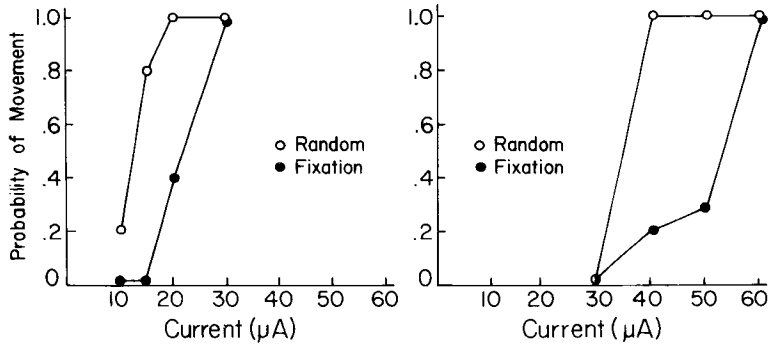

FIG. 7. Effect of fixation on the probability of evoking a saccade by collicular stimulation. At stimulation site 1 (left), a 40-ms train of 0.1 ms duration pulses delivered at 500 Hz and a current of 20 μA produced a saccade on 100% of the trials if stimulation occurred randomly between trials (open circles). Stimulation of the same site with identical stimulation parameters produced a saccade on only 40% of the trials if stimulation occurred while the animal was fixating a visual target (filled circles). Comparable results obtained from a stimulation site with a higher threshold are also shown (right). The probability values are based on at least 10 trials for each point.

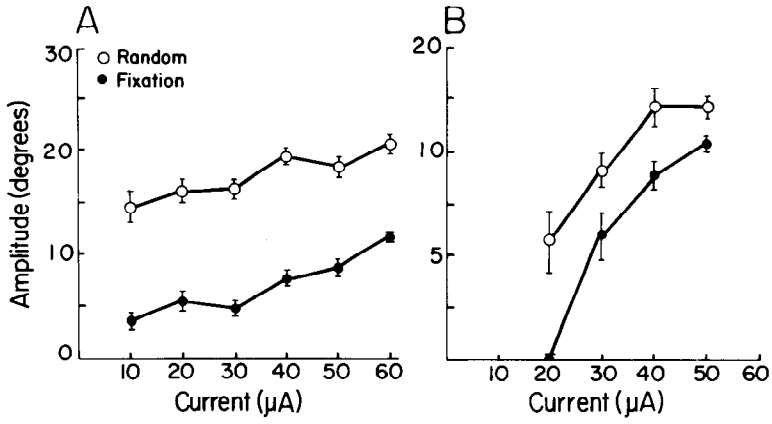

FIG. 8. Effect of fixation on the amplitude of stimulation-induced saccades. Data were obtained from the same two stimulation sites used in Fig. 8. The amplitude of the stimulation-evoked saccade is plotted as a function of stimulation current for site 1 (*A*) and site 2 (*B*). Saccades evoked by collicular stimulation during fixation (filled circles) are reduced in amplitude when compared to saccades evoked by identical stimulation during an intertrial interval (open circles). Vertical bars represent ± 1 SE of the mean.

of the stimulation-induced saccade is smaller when stimulation is delivered during a fixation interval (filled circles) than when stimulation is delivered between trials (open circles).

Effect of target location on stimulation-induced movements

The direction and amplitude of stimulation-induced saccades varied depending on

TABLE 2. *Effect of target position on horizontal and vertical components of stimulation-induced saccade*

Stimulation Site	Target Position	*n*	Horizontal Amplitude	Vertical Amplitude
TO 72	Left 10°	21	20.3 ± 1.5	10.8 ± 1.0
	Right 10°	13	15.4 ± 1.2	11.6 ± 1.1
	Diff		4.9	−0.8
	Up 10°	26	17.0 ± 0.89	8.1 ± 0.66
	Down 10°	28	15.9 ± 0.89	16.4 ± 0.92
	Diff		1.1	−8.3
TO 70	Left 20°	32	16.0 ± 0.35	6.0 ± 0.27
	Right 20°	17	9.9 ± 0.64	7.5 ± 0.77
	Diff		6.1	1.5
	Up 20°	18	9.6 ± 0.4	11.8 ± 1.1
	Down 20°	12	10.2 ± 0.17	2.4 ± 0.32
	Diff		−0.6	9.4
TO 75	Left 10°	25	11.32 ± 0.20	6.73 ± 1.0
	Right 10°	25	8.64 ± 0.29	7.66 ± 0.28
	Diff		2.68	−0.93
	Up 10°	28	9.7 ± 0.14	5.04 ± 0.17
	Down 10°	20	9.2 ± 0.14	8.84 ± 0.28
	Diff		0.5	−3.80

Values are means ± SE.

target location. The amplitude of the up component of the stimulation-induced saccade was greater when the visual target appeared above the fixation target than when the target appeared below the fixation point (Figs. 2 and 3). Similarly, the amplitude of the leftward horizontal component was larger when the visual target appeared to the left and smaller when the target appeared to the right. Table 2 presents the average amplitude of the horizontal and vertical components of the stimulation-induced saccade for four target positions (left, right, up, down) at three stimulation sites. The amplitude of the hor-

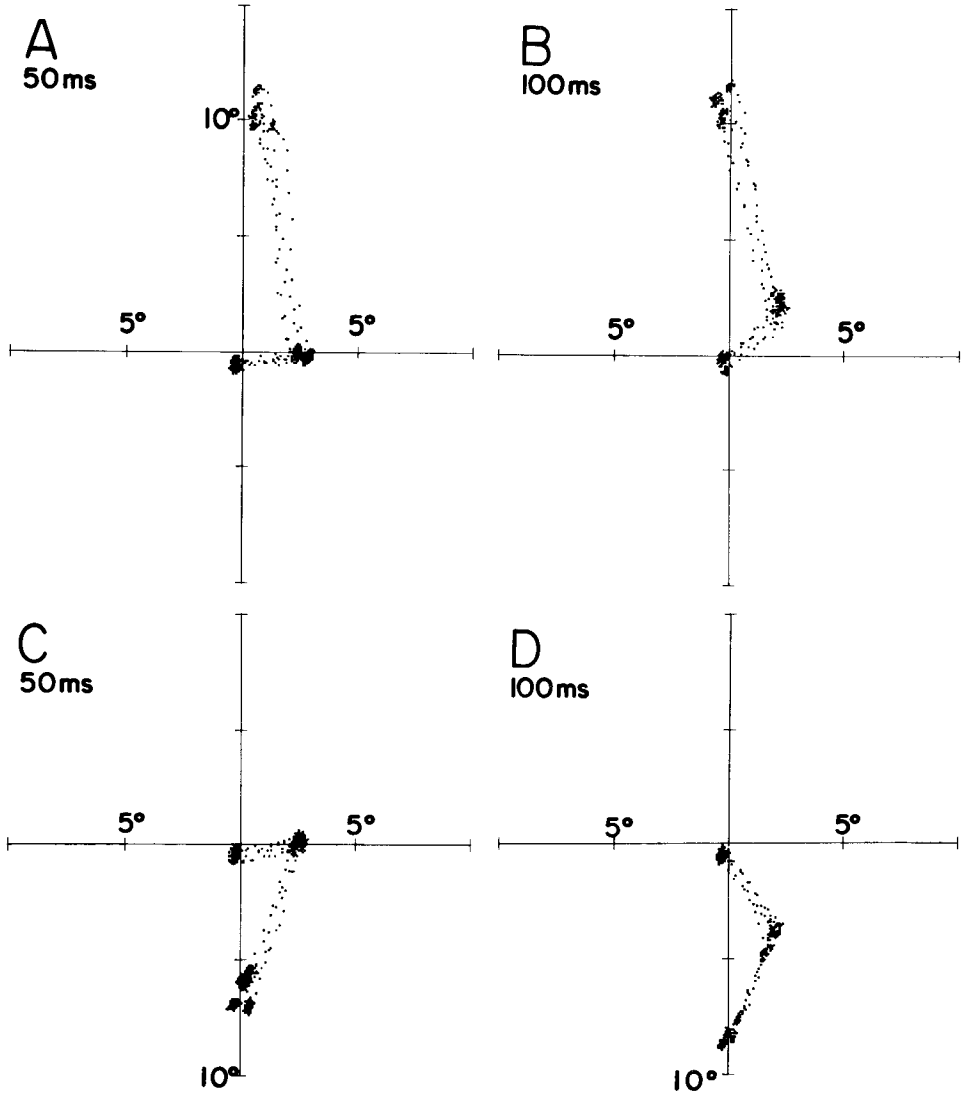

FIG. 9. Effect of target position and duration on the direction of saccades evoked by collicular stimulation. Trajectories of stimulation-induced and compensatory saccades for five trials are superimposed in each panel. The target appeared 10° above fixation (*A*, *B*) or 10° below fixation (*C*, *D*) for 50 ms (*A*, *C*) or 100 ms (*B*, *D*). Target offset was followed immediately by a 40 ms duration stimulation train. The direction of the stimulation-induced movement was unaffected by target position if stimulation followed a 50-ms target flash (*A*, *C*) and a 3° amplitude rightward saccade was evoked. If stimulation followed a 100-ms target flash, the stimulation-induced movement had an upward component if the target appeared above fixation (*B*) or a downward component if the target appeared below fixation (*D*).

izontal (but not vertical) component was affected by the horizontal position of the target. The amplitude of the vertical (but not horizontal) component was affected by the vertical position of the target.

There was also a temporal component to the effects of target position on the trajectory of the stimulation-induced saccade, as illustrated in Fig. 9. The trajectories of the stimulation-induced saccade and the compensatory saccade occurring on five trials are superimposed in each section of the figure. On the trials illustrated in *A* and *C*, target duration was 50 ms and for the trials shown in *B* and *D*, target duration was 100 ms. There was no delay between target offset and stim-

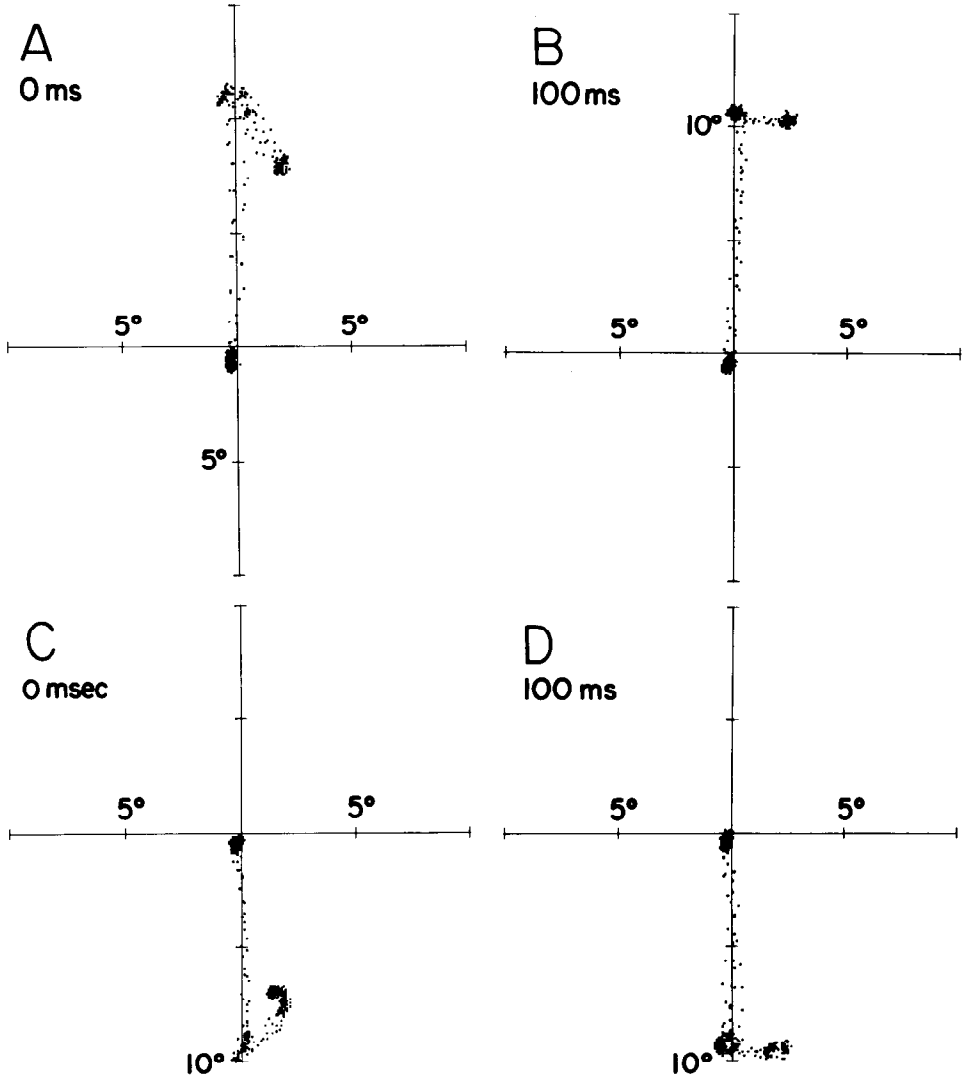

FIG. 10. Effect of prior visually triggered saccades on the vector of stimulation-induced movements. Trajectories of eye movements occurring on four trials are superimposed in each panel. Stimulation during intertrial intervals produced a 5° rightward saccade. When collicular stimulation occurred immediately after a 10° upward visually triggered saccade (*A*), a movement with a downward component occurred. Stimulation immediately following a 10° downward visually triggered saccade (*C*) produced a saccade with an upward component. The vertical components of the stimulation-induced saccades were not observed if stimulation was delayed for 50–100 ms after the termination of the visually triggered saccade (*B*, *D*).

ulation onset. With 50-ms targets, the vector of the stimulation-induced saccade was identical to the vector of saccades produced by random stimulation. With 100-ms targets, the stimulation-induced saccade had an upward component if the visual target appeared above the fixation target (*B*) and a downward component if the target appeared below the fixation target (*D*).

The vector of a stimulation-induced saccade could also be influenced by a prior visually triggered saccade. Figure 10 displays trials in which collicular stimulation was delivered following a visually triggered 10° saccade. Stimulation of this site between trials produced a 5° rightward movement. However, stimulation immediately following an upward saccade produced a movement with a downward component (*A*). If stimulation was delayed 50–100 ms after the termination of the visually triggered saccade (*B*), the downward component was not observed. Similarly, if stimulation was delivered immediately after a downward saccade (*C*), the stimulation-elicited movement had an upward component. The upward component was not present when stimulation was delayed for 50–100 ms (*D*).

Trochlear nerve stimulation

As illustrated in Fig. 11*A*, stimulation of the trochlear nerve (after decussation) produces a depression and abduction of the ipsilateral eye. After stimulation offset, the eye returns to the original position with an exponential time course. The trochlear nerve was systematically stimulated in three animals, before or during saccades, to briefly flashed targets. Typical results are shown in Fig. 11*B* and *C*. While fixating the center target, a second target was flashed for 100 ms, 10° to the right of fixation. Immediately

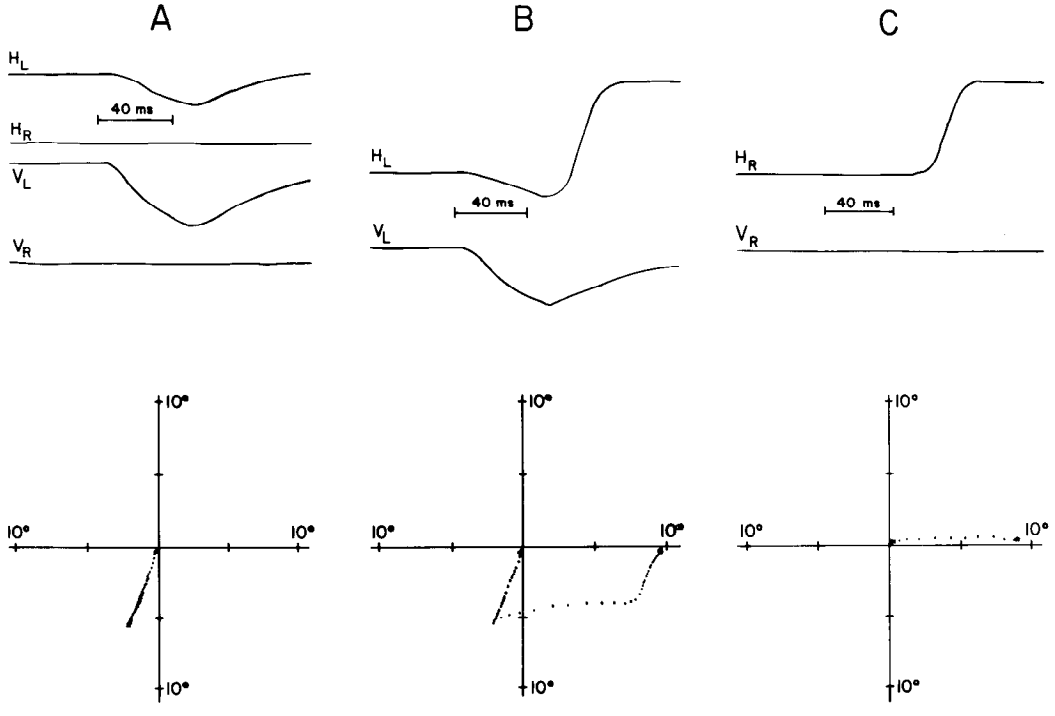

FIG. 11. Failure to compensate for eye movements produced by trochlear nerve stimulation. *A*: stimulation of trochlear nerve during fixation. While fixating the center target the left trochlear nerve was stimulated for 40 ms (horizontal bar), driving the left eye downward and to the left. Top: eye-position signals for both eyes. H_L, horizontal position of the left eye; H_R, horizontal position of the right eye; V_L, vertical position of the left eye; V_R, vertical position of the right eye. Note that with trochlear nerve stimulation, the velocity of the stimulation-induced movement is considerably less than the velocity of saccades with the same amplitude, and the eye returns to the prestimulation position with an exponential time course. *B* and *C*: trajectories of eye movements of the left (*B*) and right (*C*) eyes during a stimulation trial. See text for details.

after target offset, trochlear nerve stimulation moved the left eye downward and to the left (*B*) but had no effect on the right eye (*C*). On cessation of the stimulation, the left eye begins to return to the original eye position with a slow, exponential time course. During the slow return movement, a conjugate rightward saccade, 10° in amplitude, occurs (*B*, *C*) and is superimposed on the slow-velocity return movement (*B*). Compensation for the movement produced by trochlear nerve stimulation would require recomputation of the vector of the acquisition saccade. Since the saccade command is a conjugate signal, modification of a saccade vector should be observable in the trajectories of both eyes. There was no modification of the trajectory of the right eye and the trajectory of the movement of the left eye was a simple algebraic combination of the trochlear nerve stimulation-induced movement and a 10° rightward saccade. We found no evidence in these experiments of compensation for displacements of the eye produced by trochlear nerve stimulation.

DISCUSSION

Localization of saccade targets

In the present experiment, after a visual target was briefly flashed but before a saccade to the target could be initiated, the eyes were driven to another position in the orbit by electrical stimulation of the SC. According to retinocentric models (23, 42), if the animal attempts to look to the visual target, a saccade with an amplitude and direction based solely on a retinal-error signal will occur. In that case, gaze will miss the target location by a distance and direction equal to the vector of the stimulation-induced movement (see Fig. 1). Spatial models (24, 39, 44) assume that the retinal-error signal will be combined with information about the stimulation-induced change in eye position to produce a saccade that moves the foveal projection to the position of the target in space. Our results confirm the predictions of spatial models. Animals compensated for the stimulation-induced perturbation by looking to the position of the target in space.

The possibility that the compensatory movement is merely a passive rotation to an orbital position of mechanical equilibrium established by the tonic pattern of motoneuron innervation that was specified by the visual target must be considered (P. Grobstein and R. Schmidt, personal communications). If this were the case, the compensatory movement would be a slow movement with a velocity comparable to movements observed during the return of the eye to its original position after a passive mechanical displacement (26). Instead, compensatory movements have normal saccadic velocities and, like visually triggered saccades, require a brief pulse of innervation to the motoneurons to overcome the viscous impedance of the orbital tissues and generate a high-velocity movement (24). Thus, compensatory movements must be produced by a pulse and a step of innervation to the motoneurons appropriate for the vector of the compensatory movement—not simply a tonic rate of activity specified by the visual target before the stimulation-induced perturbation.

The motor command for the compensatory saccade could not be based on retinal information alone, since the target was not present either during or after the stimulation-induced saccade. Nor could the target be localized using background cues as an external frame of reference since the task was performed in total darkness. The occurrence of stimulation trials was completely unpredictable so that compensation for the stimulation-induced perturbation could not have been predetermined. Thus, the vector of the compensatory saccade must have been computed based on information about the stimulation-induced change in eye position and stored information about the site of retinal activation. Computation of the vector of the compensatory saccade must occur quite rapidly. The interval between the end of a stimulation-induced saccade and the beginning of a compensatory saccade was usually brief (see Figs. 4*D* and 6), with intersaccadic intervals of 20 ms or less frequently being observed.

Accuracy of compensatory saccades

Compensatory movements to the remembered location of the target were less accurate than movements to a continuously illuminated target. Since the target was no longer present at the end of the stimulation-induced saccade, eye-position signals were combined

with a stored representation of target coordinates. The accuracy of the compensatory movements depends, therefore, on both the fidelity of spatial memory and the resolution of the eye-position signals. Comparison of the accuracy of compensatory movements with the accuracy of control (brief target) trials suggests that both spatial memory and eye-position signals contribute to the reduction in accuracy. However, it was not possible in this experiment to obtain precise estimates of the magnitude of the error attributable to each of these sources.

Sources of eye-position signals

Two findings are pertinent to the question of the source of the eye-position signal used in the spatial localization of saccade targets. First, failure to observe compensation for perturbations in eye position produced by trochlear nerve stimulation supports the hypothesis that an internal copy of the efferent command ("efference copy") provides eye-position information (39, 44). Stimulation of the trochlear nerve produces an eye movement that should activate proprioceptors in the extraocular muscles without activation of the normal premotor and motoneuron circuitry presumed to be the source of an efference copy signal. Thus, if the eye-position signal is based on activation of receptors in extraocular muscles, a compensatory movement should be observed. If, however, the eye-position signal is based on efference copy, compensation would fail to occur. The failure to observe compensatory movements following trochlear nerve stimulation indirectly supports suggestions that efference copy is the source of the eye-position signal. However, this negative finding must be interpreted cautiously. The pattern of muscle receptor activation following trochlear nerve stimulation may differ from that occurring during voluntary movements, particularly if the gain of the receptor response is dependent on γ-efferent signals. A reduced or altered proprioceptive signal could account for this negative finding. Furthermore, since trochlear nerve stimulation produces movement of only one eye, it is possible that the eye-position signal is derived from proprioceptive signals originating from the unperturbed eye. Second, the ability of the animal to compensate for interrupted saccades is informative. Assume that the eye-position signal used to compute the vector of the compensatory saccade is based on a corollary discharge of the motor-command signal. Two types of corollary discharges are available. The burst of collicular or pontine long-lead burst neurons could provide a "discrete" signal of the vector of the impending saccade. Alternatively, the activity of neurons with tonic firing rates proportional to eye position provide a "continuous" signal of eye position. Accurate compensation for stimulation-induced perturbations of ongoing saccades indicates that the vector of the compensatory saccade is based on a continuous rather than a discrete eye-position signal.

Effect of target location on stimulation-induced saccades

Previous studies (22, 28) indicate that the direction and amplitude of saccades evoked by stimulation of the SC are determined by the site of stimulation within the colliculus and are independent of stimulation parameters. We found that if stimulation occurs prior to an impending saccade to a visual target, the vector of the stimulation-induced saccade may be affected by the position of the visual target. Changes in the horizontal position of the target affect, primarily, the horizontal component of the stimulation-induced movement. Changes in the vertical position of the target affect, primarily, the vertical component of the saccade. The magnitude of the interaction effect depends on the interval between target onset and stimulation onset, suggesting that signals specifying the amplitude of the horizontal and vertical components of the impending visually triggered saccade gradually build up during the reaction-time interval.

We interpreted this interaction effect in the following manner. Since separate brain stem circuits have been identified for the control of horizontal and vertical saccades (see below), separate signals specifying the horizontal and vertical components of a saccade are required. When stimulation of the SC follows the appearance of a visual target, the site of stimulation specifies a change in horizontal eye position (horizontal motor error, HME) that is combined with a signal of the horizontal component of the movement required to look to the visual target. The vertical mo-

tor error (VME) specified by the stimulation site and the VME specified by the visual target are combined in a similar fashion. Stimulation of the SC also produces, after a delay, a trigger signal that initiates a saccade with horizontal and vertical components dependent on the contents of the HME and VME storage sites at the time the trigger signal arrives. Since the visual specification of HME and VME occurs gradually, not instantaneously, the vector of the observed saccade depends on the interval between target onset and stimulation onset.

Effect of fixation on stimulation-induced movements

We found that fixation of a visual target significantly increases the threshold for producing saccades by stimulation of the SC. Also, the amplitude of saccades evoked by suprathreshold currents was reduced if stimulation occurred during fixation. Thus, fixation is not merely the absence of saccade commands but an interval during which saccades are actively suppressed. This allows time for extraction of detailed information about the visual target and provides the time required for computing the horizontal and vertical coordinates of the next saccade. Neurons have been observed in the SC (38) and paramedian pontine reticular formation (PPRF) (21, 35) that display marked increases in firing during fixation of a visual target regardless of the location of the target in the visual field. These neurons could provide an excitatory input into the pause cells (as suggested in Fig. 12), increasing the inhibition exerted on medium-lead burst (MLB) cells and, thereby, increasing the threshold for eliciting saccades by collicular stimulation. Alterations in the amplitude of saccades evoked by stimulation during fixation may represent a special case of the interaction between the location of a visual target and the vector of stimulation-induced saccades. Following stimulation, the HME and VME specified by the stimulation site are combined with the HME and VME specified by the visual target. Since during fixation HME and VME are specified as zero, a reduction in the horizontal and vertical components of the stimulation-induced movement would be observed.

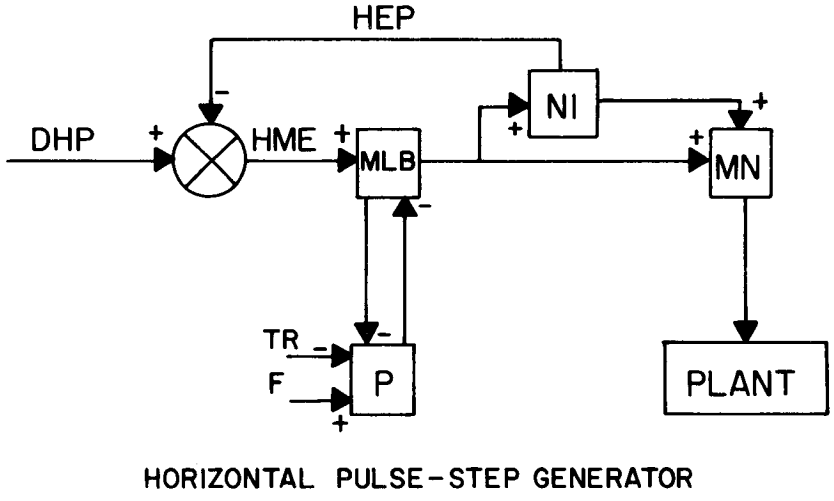

FIG. 12. Model of the horizontal pulse-step generator (adapted from D. A. Robinson, Ref. 23). DHP, desired horizontal position of the eye in the orbit; HEP, current horizontal eye position; HME, horizontal motor error, the difference between DHP and HEP; NI, neural integrator; MN, motoneuron; MLB, medium-lead burst neuron; P, pause neuron; TR, trigger signal. The duration of the MLB neuron discharge is assumed to be controlled by pause cells, since microstimulation of the pontine region containing pause neurons exerts a powerful and selective inhibitory effect on the saccadic system (3, 11, 12). Saccades are initiated by a trigger signal (TR), which inhibits the pause cells briefly, permitting the MLB neurons to begin to discharge. MLB cell activity through the appropriate motoneurons moves the eyes, reducing motor error, and the discharge of MLB neurons continues until motor error becomes zero. At this point MLB cell discharge ceases and P cells resume their tonic discharge, thereby inhibiting the system until another trigger signal arrives. A similar circuit related to vertical saccades is thought to reside in the midbrain reticular formation (1, 13).

Role of PPRF and SC in compensatory saccades

Compensation for perturbations in eye position produced by collicular stimulation can be accounted for by current models (24, 39) of the oculomotor system, which assume that saccades are controlled by a local feedback circuit located in the pons. These models are based on microstimulation and chronic unit-recording data (11, 14, 34) indicating that the PPRF contains the supranuclear neuronal networks controlling all types of conjugate horizontal eye movement. The key elements of these models are shown in Fig. 12. The input to this circuit is the desired horizontal position of the eye in the orbit (DHP). Once triggered, the circuit drives the eye at a high velocity until actual eye position matches desired eye position; the eye automatically stops on target. Saccades resulting from collicular stimulation are initiated, presumably, by the activity of MLB neurons. The eye-position signal observed at the output of the neural integrator (NI) will reflect this perturbation and the comparison of the altered eye-position signal with DHP results in an updated motor-error signal. On arrival of a trigger signal, a saccade that compensates for the stimulation-induced perturbation in eye position will be produced automatically. Thus, after the desired position of the eye in the orbit has been specified, there is no apparent need for neural activity in brain areas (such as the SC) providing inputs to the pontine circuit to be modified.

However, a recent model of SC function (17) predicts that stimulation-induced perturbations in eye position will produce a shift in the site of active neurons in the SC. The discharge of one class of collicular neuron (quasi-visual or QV neuron) is thought to reflect motor error (the difference between current and desired eye position) and hold this information in spatial register until a saccade occurs or is canceled (17). If the eyes move after a brief target has disappeared, the site of QV cell activity changes to a location that represents the new motor-error signal. This finding indicates that visual and eye-position signals are combined at or before the level of the SC. In the present experiment, saccades made to compensate for stimulation-induced perturbations of eye position must be based on a combination of visual and eye-position information. If these signals are combined at or before the level of the SC, then saccade-related activity should be observed in the SC before compensatory movements. The experiment described in the following paper (33) was designed as an explicit test of this prediction.

ACKNOWLEDGMENTS

We thank Robert Deich and Claude Horton for technical assistance. Data acquisition and analysis programs were developed by Richard Sheetz and Kathy Pearson.

This research was supported by National Institutes of Health Grants R01EY01189, R01EY02293, and P30EY03039.

Received 4 February 1982; accepted in final form 7 July 1982.

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