Direction Constancy in the Oculomotor System

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Whenever a visual image moves across the retina, the brain is faced with a dilemma: Did the image move because an object in the environment (or the environment itself) changed location or because the eyes changed location with respect to a stable environment? If the brain compares the retinal positions of individual objects within the visual image and finds that only one moved with respect to the others, then it can reasonably assume that the retinal motion was caused by motion of the object and not of the eyes. (This comparison of an object's position with the positions of other objects is known as *exocentric*, or allocentric, localization.) But how does the brain react to this dilemma when the entire visual image moves, whether the image is a fully structured scene or merely a single object in otherwise complete darkness? Under these conditions, exocentric localization techniques fail, and the brain must rely on its ability to localize the objects with respect to itself, the observer. For this *egocentric* localization, the brain must combine information concerning the retinal location of the visual image with knowledge of where the eyes are pointing (i.e., the internal representation of eye position).

Although direction constancy, or the brain's ability to use egocentric and exocentric cues to compensate for the image motion caused by eye movements, is most often considered in the realm of perception (i.e., why doesn't the world appear to jump about as I move my eyes?), it is equally a motor phenomenon. Whereas eye movements will affect the retinal location of a stationary object, the brain's representation of that object's location within the appropriate motor coordinate system must remain constant to ensure the accuracy of a targeting movement. Because the neuronal activity underlying the trajectory of a targeting movement can be more easily investigated than that underlying a subjective report, the motor aspect of direction constancy is much more amenable to neurophysiological investigation than the perceptual aspect. Until recently, however, most neurophysiologists have ignored the details of where and how a retinotopic signal (i.e., expressed in retinal coordinates) is converted into a signal that is stable in spite of intervening eye movements. Our laboratory has undertaken several lines of research to elucidate the mechanisms underlying the phenomenon of direction constancy in the oculomotor system and to identify the brain structures involved.

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JUST HOW CONSTANT IS OCULOMOTOR DIRECTION CONSTANCY?

When investigating the accuracy of the egocentric localization abilities of the oculomotor system, one must consider the individual contributions of the signals that encode retinal image location and eye position, as well as the fidelity of the mechanism used to combine these signals to achieve direction constancy. The accuracy of the retinal location signal is based largely on the amount of detail provided by the topographic representations of the retina and early visual structures. However, temporal factors do seem to affect the precision of the retinal signal at some stage of saccadic programming: Short-duration flashes evoke saccades of greater variability in direction and amplitude than do long-duration flashes.

Much less is known about the accuracy of the internal representation of eye position, and even less about the brain's ability to combine this signal with the signal of retinal position. To investigate these accuracies, we presented to human and monkey subjects a series of three visual stimuli in otherwise complete darkness (a typical trial is shown in Fig. 1a).[1] Each trial began when the subject fixated the first stimulus (F in Fig. 1a). After 750 ms, this fixation point was replaced by a 5-ms *initial flash* (I, located 20° right of fixation), which served as the goal for a saccadic eye movement (the *initial saccade*). A subsequent *target flash* (T, 2-ms duration) was presented in one of five possible locations aligned 10° above the fixation point and initial flash, with an onset time randomly selected to occur before, during, or after the initial saccade. The subject completed the task by making a *targeting saccade* to the location of the target flash.

Because the initial saccade often intervened between the occurrence of the target flash and the subsequent targeting saccade, the attainment of true egocentric direction constancy would require the oculomotor system to faithfully combine a neural signal accurately encoding eye position with one accurately en-

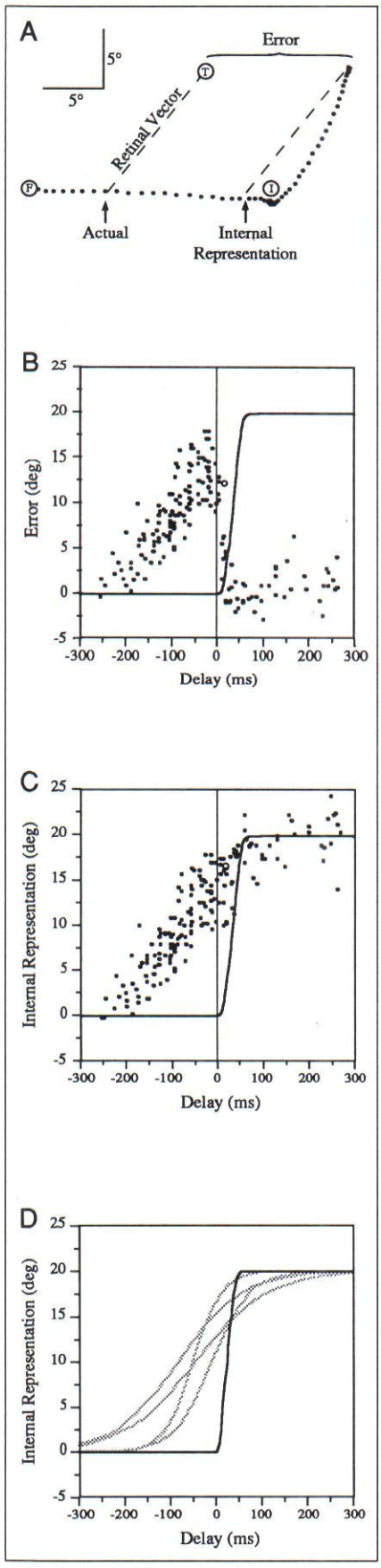

Fig. 1. Mislocalization of flashes presented around the time of an ongoing saccade. (a) Eye movement trajectories in *xy* coordinates for a single experimental trial. The points represent the eye's position at 2-ms intervals. The initial saccade is from the fixation point (F) to the initial flash (I); the oblique targeting saccade is the subject's subsequent attempt to localize the target flash (T, presented when the eye was at the position marked "Actual"), resulting in an error of approximately 12°. Graphically subtracting the retinal vector (dashed line) from the end point of the targeting saccade gives an indication of the value of the internal representation of eye position used to convert the retinal vector into the saccade goal. (b) Magnitude of the localization errors from a single subject, plotted with respect to the delay between presentation of the target flash and onset of the initial saccade. Positive error values indicate localization errors in the same direction as the initial saccade (i.e., to the right); positive delay values indicate trials in which the target flash was presented after initial movement onset (0 ms). The solid curve depicts the time course of a typical initial saccade. The open circle denotes the datum point for the trial shown in (a). (c) Internal representation of eye position derived as stated in the text and in (a). Other annotations as in (b). (d) Best-fit sigmoidal curves showing the internal representation of eye position from 4 human subjects (stippled curves). Other annotations as in (c).

coding the retinal location of the target flash.[2] Any imperfection in these signals or in their combination will be reflected in the pattern of errors made by the subject. Figure 1b shows the magnitude of the localization errors made by a human subject, plotted with respect to the time delay between the onset of the initial saccade and the target flash. For targets presented well before or well after the initial movement, localization was reasonably good. However, target flashes presented just before the initial movement were consistently mislocalized; these errors peaked with flashes presented at movement onset and then sharply declined throughout the duration of the initial movement.

What might account for this pattern of errors? The random variability (seen as the scatter of data points at individual delay times) is probably due to the difficulty in localizing short-duration flashes, as mentioned earlier. This variability cannot explain, however, the errors that are highly correlated with flash delay; these must be the result of a misrepresentation of eye position. In the trial depicted in Figure 1a, the target flash was presented 18 ms after the onset of the initial saccade (i.e., when the eye was at the position marked "Actual"). Because egocentric localization is based on the vector addition of the internal representation of eye position and the target's retinal vector (in Fig. 1a, the dashed line that connects the target location with the eye's actual position), the inverse—subtraction of the retinal vector from the end point of the corresponding targeting saccade—yields the internal representation of eye position. In Figure 1a, then, the oculomotor system mistakenly "thought" the eye had moved 18° before the target was presented; in fact, it had moved only 6°.

Figure 1c shows the location of the internal representation for each trial, plotted with respect to the time delay between the onset of the initial saccade and the target flash. The resulting picture of the internal representation has a time course much different from that of the actual initial saccade—in this particular subject, it starts changing approximately 200 ms before the saccade and has a much slower velocity. This general finding was verified in 3 other human subjects (Fig. 1d) and 1 monkey trained in the same task (Fig. 2, light stippled curve), although the onset times and velocities differed among subjects. The use of a similarly damped representation of eye position has been noted in perceptual localization since the work of Matin and his colleagues,[3] but motor localization abilities have been thought to be based on an accurate representation.[4] Our findings and

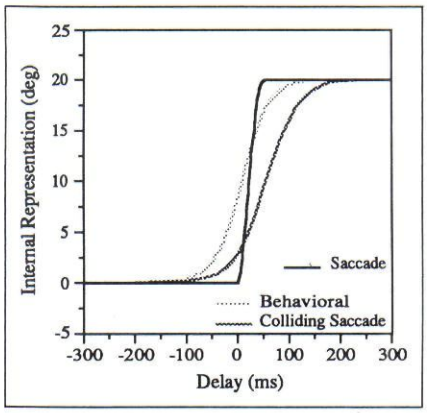

Fig. 2. Best-fit sigmoidal curves showing the time course of the internal representation of eye position from a single monkey as derived by behavioral (light stippled curve) and colliding-saccade (dark stippled curve) paradigms. The solid curve depicts, for comparison, the time course of a typical initial saccade in the behavioral experiment.

those of other recent studies indicate that this is not the case; around the time of a saccade, there appears to be a discrepancy between where the eyes are and where the oculomotor system "thinks" the eyes are.[5] As a result, egocentric localization abilities break down for a short time surrounding a saccadic eye movement.

(Although we have implicated a damped representation of eye position as the cause of the mislocalizations shown in Fig. 1a, it is possible that an accurate representation is combined with the retinal location signal by a mechanism that itself has a slow time constant. These two possible explanations are behaviorally indistinct; for simplicity, however, we will continue to refer to the internal representation as being damped.)

In the experiment described, all possible exocentric cues were removed (or at least minimized) by performing the experiment in complete darkness and by separating the target flash in time from the fixation point and initial flash (i.e., a dark period of 45 to 495 ms elapsed between offset of the initial flash and onset of the target). In everyday experience, however, exocentric cues are abundant. To test their effects in a controlled manner, we performed a second experiment in which we removed the gap of darkness between the initial and target flashes by increasing the duration of the initial flash.[6] The resulting temporal proximity might allow the subjects to compare the retinal locations of the separate flashes more easily. Indeed, with this minor modification to the task, there was a significant improvement in the subjects' ability to localize the target flash, especially for the target locations that were in close spatial proximity to the initial flash. (A similar role of spatial proximity, described as the "adjacency principle" by Gogel, has been noted in the use of exocentric cues in many perceptual tasks.[7]) Although the presence of exocentric cues did not, in our subjects, completely eliminate the errors associated with egocentric localization, it appears that the oculomotor system is capable of using these cues to assist in programming a more appropriate targeting saccade. This aspect of oculomotor control seems very important, but has thus far been ignored by oculomotor neurophysiologists and modelers.

WHAT BRAIN STRUCTURE IS RESPONSIBLE FOR OCULOMOTOR DIRECTION CONSTANCY?

What oculomotor structure is responsible for achieving direction constancy? Traditionally, answering this question meant recording from single neurons in a monkey while the animal performed a task similar to the one described earlier. When the target flash is presented before the initial saccade, the retinal location of the target is dissociated from the saccade vector necessary to acquire it. This should allow the neurophysiologist to determine whether the cells in a given structure respond in a direction-constant manner: Cells that respond best to the retinal vector of a target flash should presumably lie before the oculomotor processing stage in which direction constancy is achieved, whereas those that respond best to the saccade vector should lie after.

To date, three oculomotor structures have been tested with this general paradigm (superior colliculus, SC; frontal eye field, FEF; and lateral intraparietal area).[8] For each structure, the results were mixed: Two cell populations were found, one group responding best to the retinal vector, the other to the saccade vector. Given these results, it seems that each structure either is partly (or fully) responsible for the achievement of oculomotor direction constancy or is a recipient of projections from the structure responsible. This similarity of unit response has disappointed investigators seeking to distinguish the separate roles of these structures. Furthermore, none of these studies completely excluded exocentric cues: Although each was performed in complete darkness, some exocentric cues were available from the sequentially presented stimuli, as they were in the second of our behavioral tasks described in the preceding section. It is therefore uncertain whether the direction-constant response seen in each structure was due to egocentric or exocentric processing.

Can microstimulation be used as a tool to help determine which structure or structures in the brain are responsible for egocentric direction constancy? Imagine a hypothetical situation in which, instead of presenting a visual stimulus to a subject, the investigator were to electrically stimulate the retina to evoke a brief phosphene that would be mistaken for a real flash. The subject's brain should interpret the incoming signals as it would any other visual stimulus (i.e., the signal would be combined with the internal representation of eye position to achieve direction constancy), and the even-

tual targeting saccade would compensate for any intervening eye movements. The same should hold true if the optic nerve were stimulated rather than the retina. As the investigator continued to stimulate different structures along the pathways involved in oculomotor generation, moving downstream from the retina, he or she should eventually find an oculomotor structure in which stimulation evokes a movement that is no longer compensatory. Theoretically, one can use this *colliding-saccade paradigm* of microstimulation, in monkeys, to determine the location in the brain where a retinotopic signal is converted into a direction-constant one.[9]

We have used this paradigm to test thoroughly three oculomotor structures: SC, thalamic internal medullary laminar complex (IMLc), and FEF. Previous studies had shown that at most sites in these structures, microstimulation—applied when the monkey's eyes are stationary—evokes a saccade with a direction and amplitude characteristic of the particular stimulation site (a so-called fixed-vector saccade). Is the trajectory of the electrically evoked movement affected if stimulation is applied during an ongoing saccade? The answer, we found, was dependent on the particular location tested.

In deep SC layers, where unit activity is predominantly motor in nature (i.e., time-locked to saccades in a preferred direction), the electrically evoked movements were unaffected by the presence of the intervening saccade.[10] Thus, it seems clear that this portion of SC lies either downstream from the oculomotor site responsible for egocentric direction constancy or on a pathway unconcerned with direction constancy.

In contrast, compensation was seen at all stimulation sites in the superficial, visual layers of SC and at all sites in thalamic IMLc and FEF. The amount of compensation was quantified in each case by graphically subtracting the vector of the fixed-vector saccade from the end point of each compensatory colliding saccade (as was demonstrated with the behavioral data in Fig. 1a). From these measurements, an important distinction emerged between the effects of microstimulation in superficial SC and those in thalamic IMLc and FEF.

In the case of the superficial SC, the electrically evoked saccades compensated for the portion of the ongoing movement that occurred during the latent period between stimulation onset and the start of the evoked saccade.[10] At first glance, it appears that this type of compensation would lead to a means of achieving perfect direction constancy. In fact, this is quite untrue, because of an often-ignored limitation of the visual system: It takes many milliseconds for the neural activity generated in the rods and cones of the retina to propagate into the brain proper. Because of this afferent processing delay, perfect direction constancy would require the oculomotor system to compensate for any eye movements that occur after the onset of the visual target, not simply for movements that occur after the onset of the corresponding neural activity in the brain. In essence, the brain would need access to a delayed representation of where the eyes *were* when a visual target was presented. Use of an accurate but undelayed representation of where the eyes currently *are* would lead to localization errors even larger than those that are associated with the damped representation of eye position (Fig. 1b).[11] The superficial layers of SC, therefore, do not appear to lie on the neural pathway used by the oculomotor system in its attempt to achieve egocentric direction constancy.

With microstimulation of thalamic IMLc[12] or FEF,[13] the evoked movements compensated not only for the change in eye position that occurred after stimulation onset, but also for a portion of the change that preceded it. The dark stippled curve in Figure 2 shows the internal representation of eye position as derived in the FEF colliding-saccade experiments. The shape of this curve closely matches that of the curve derived in the behavioral experiment performed in the same monkey (light stippled curve), but a timing mismatch of approximately 50 ms does exist. The timing difference between these two views of the internal representation of eye position can be attributed to the afferent processing delay of the retina and early visual structures; when microstimulation is applied to an oculomotor structure in the brain, this delay is bypassed. Indeed, the mismatch of 50 ms between the colliding-saccade and behavioral views of the eye position signal very closely matches the latency of FEF visual activity measured with single-unit recordings. The colliding saccades evoked from thalamic IMLc seem to be similarly based on this damped representation of eye position. From these results, FEF and IMLc can be inferred to fall before the stage of oculomotor processing responsible for egocentric direction constancy.

Because FEF is thought to be the region of cortex closest to the final common pathway for saccade generation, our findings suggest that oculomotor direction constancy is achieved in some subcortical structure that receives input, directly or indirectly, from FEF. It is well known that FEF projects to the motor-related layers of SC, but we have evidence, derived from an experiment in which we recorded from single neurons in SC while stimulating FEF, that the compensation for intervening movements does not occur on this pathway.[14] Most likely, then, this compensation occurs in one of the brain stem structures that receives FEF input. It remains unclear why our conclusions, based on mi-

crostimulation studies, differ from those of the single-unit investigations of SC, FEF, and lateral intraparietal area, as described earlier.[8]

CONCLUSIONS

The findings from the behavioral and microstimulation studies presented here contradict the widely held notion that egocentric direction constancy in the oculomotor system is accurate. Instead, it seems that the brain's use of a damped representation of eye position leads to oculomotor localization errors similar to those previously reported in perceptual localization tasks.[3] Further studies are necessary to determine if oculomotor and perceptual direction constancies are achieved by the same mechanism in a single brain structure, or merely by similar mechanisms in separate structures.

Although the colliding-saccade paradigm has yet to pinpoint the oculomotor structure responsible for egocentric direction constancy, it has provided a better understanding of where this structure may lie in the cascade of oculomotor programming steps. Perhaps future colliding-saccade studies of the many untested oculomotor areas, in conjunction with investigations of the associated single-unit responses, will yield a precise identification of the responsible structure. Furthermore, the oculomotor system's as-yet-overlooked ability to use exocentric location cues, when examined at psychophysical and neurophysiological levels, is certain to be a fertile area in which to develop insight into the many ways that the brain processes visual spatial information.

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Notes

1. P. Dassonville, J. Schlag, and M. Schlag-Rey, Oculomotor localization relies on a damped representation of saccadic eye displacement in human and nonhuman primates, *Visual Neuroscience, 9,* 261–269 (1992).

2. Although we refer to the oculomotor system's attempt to achieve egocentric constancy—with the implication that target localization is performed in a head- or body-centered coordinate system [D.A. Robinson, Oculomotor control signals, in *Basic Mechanisms of Ocular Motility and Their Clinical Implications,* G. Lennerstrand and P. Bach-y-Rita, Eds. (Pergamon Press, Oxford, 1975)]—some modelers of the oculomotor system have advocated the use of a coordinate system not centered on the head or body. Instead, the retinotopic signal of target position is updated with each saccade by subtracting a representation of the change in eye position or velocity [e.g., R. Jürgens, W. Becker, and H.H. Kornhuber, Natural and drug-induced variations of velocity and duration of human saccadic eye movements: Evidence for a control of the neural pulse generator by local feedback, *Biological Cybernetics, 39,* 87–96 (1981)]. Although the results described in the present review can be applied equally to either model, our discussion refers only to the former for simplicity.

3. L. Matin, Eye movements and perceived visual direction, in *Handbook of Sensory Physiology,* Vol. VII/4, D. Jameson and L. Hurvich, Eds. (Springer-Verlag, Heidelberg, 1972); see also S. Mateeff, Saccadic eye movements and localization of visual stimuli, *Perception & Psychophysics, 24,* 215–224 (1978).

4. P.E. Hallett and A.D. Lightstone, Saccadic eye movements toward stimuli triggered by prior saccades, *Vision Research, 16,* 99–106 (1976); R.M. Hansen and A.A. Skavenski, Accuracy of spatial localizations near the time of saccadic eye movements, *Vision Research, 25,* 1077–1082 (1985).

5. See also H. Honda, Eye movements to a visual stimulus flashed before, during, or after a saccade, in *Attention and Performance XIII: Motor Representation and Control,* M. Jeannerod, Ed. (Erlbaum, Hillsdale, NJ, 1990); J.M. Miller, Egocentric localization of a brief perisaccadic flash by manual pointing, *Society for Neuroscience Abstracts, 18,* 215 (1992). In the latter study, subjects were required to localize a perisaccadic target with a pointing movement of the arm; although the representation of eye position for each subject was found to be damped in a manner similar to what is described here, the representation did not begin to change until the eye itself began to move.

6. P. Dassonville, J. Schlag, and M. Schlag-Rey, Human oculomotor system uses both exo- and egocentric cues in the localization of successive targets, *Society for Neuroscience Abstracts, 17,* 860 (1991); see also M. Hayhoe, J. Lachter, and P. Møller, Spatial memory and integration across saccadic eye movements, in *Eye Movements and Visual Cognition,* K. Rayner, Ed. (Springer-Verlag, New York, 1992).

7. W.C. Gogel, The adjacency principle in visual perception, *Scientific American, 238,* 126–139 (1978).

8. Superior colliculus: L.E. Mays and D.L. Sparks, Dissociation of visual and saccade-related responses in superior colliculus neurons, *Journal of Neurophysiology, 43,* 207–232 (1980); frontal eye field: M.E. Goldberg and C.J. Bruce, Primate frontal eye fields: III. Maintenance of a spatially accurate saccade signal, *Journal of Neurophysiology, 64,* 489–508 (1990); lateral intraparietal area: S. Barash, R.M. Bracewell, L. Fogassi, J.W. Gnadt, and R.A. Andersen, Saccade-related activity in the lateral intraparietal area: II. Spatial properties, *Journal of Neurophysiology, 66,* 1109–1124 (1991); J.-R. Duhamel, C.L. Colby, and M.E. Goldberg, The updating of the representation of visual space in parietal cortex by intended eye movements, *Science, 255,* 90–92 (1992).

9. J. Schlag and M. Schlag-Rey, Colliding saccades may reveal the secret of their marching orders, *Trends in Neurosciences, 13,* 410–415 (1990).

10. M. Schlag-Rey, J. Schlag, and B. Shook, Interactions between natural and electrically evoked saccades: I. Differences between sites carrying retinal error and motor command signals in monkey superior colliculus, *Experimental Brain Research, 76,* 537–547 (1989).

11. J. Schlag, M. Schlag-Rey, and P. Dassonville, Spatial programming of eye movements, in *Brain and Space,* J. Paillard, Ed. (Oxford University Press, Oxford, 1991).

12. J. Schlag, M. Schlag-Rey, and P. Dassonville, Interactions between natural and electrically evoked saccades: II. At what time is eye position sampled as a reference for the localization of a target? *Experimental Brain Research, 76,* 548–558 (1989).

13. P. Dassonville, J. Schlag, and M. Schlag-Rey, The frontal eye field provides the goal of saccadic eye movement, *Experimental Brain Research, 89,* 300–310 (1992); P. Dassonville, J. Schlag, and M. Schlag-Rey, Time course of internal representation of eye position as derived from microstimulation experiments, *Investigative Ophthalmology and Visual Science, 31*(Supplement), 84 (1990).

14. M. Schlag-Rey, J. Schlag, and P. Dassonville, How the frontal eye field can impose a saccade goal on superior colliculus neurons, *Journal of Neurophysiology, 67,* 1003–1005 (1992).