Neural control of saccadic eye movements

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Recent experiments report that localization of brief targets presented during an ongoing saccade is not accurate. Because interpretations of these findings challenge an important tenet of existing oculomotor models, we examine the methodological and logical bases of these conclusions. Also, we review recent research related to the roles of the frontal eye fields and cerebellum in the control of saccadic eye movements. Pathways by which neurons in the frontal eye fields control the initiation and metrics of saccades have been clarified by studying the functional properties of neurons in the frontal eye fields that project to oculomotor regions of the pons, and the discovery of a short-latency pathway that enables relatively direct control of saccade initiation. We review the puzzling literature on the role of vermal lobules VIc, VII and the fastigial nuclei in the control of saccadic eye movements and suggest a testable hypothesis about how the fastigial projection to inhibitory burst neurons could modify saccade metrics.

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Introduction

Three topics were selected from the burgeoning literature on the neural control of saccadic eye movements for review and comment. (Saccades are quick, high velocity eye movements usually made in an attempt to bring the image of an eccentric target onto the fovea, the region of the retina with the greatest density of photoreceptors). We review recent research related to the roles of the frontal eye fields and cerebellum in the control of saccadic eye movements, summarize and critique results of recent studies estimating the accuracy of extraretinal eye position signals used for the localization of visual targets, and comment on the use of the term 'retinocentric' in the oculomotor literature.

Accuracy of extraretinal eye position signals

In 1976 Hallett and Lightstone [1] reported that subjects could make accurate saccades to targets flashed (brief target) during an ongoing saccade (perturbing saccade). This implied that an accurate saccade to the location of the brief target could not be based solely upon information about the location of its retinal image because the eye continued to move after it was presented (see Fig. 1). Hallett and Lightstone [1] concluded that saccadic localization of the brief target was based upon information about the site of retinal stimulation and information about the change in eye position that occurred after the flash. This finding provided an impetus for developing models of the saccadic system in which it is postulated that gaze is directed to a predetermined position in space, rather than being driven a certain distance and direction.

Four recent attempts [2,3,4•,5•] to replicate the findings of Hallett and Lightstone provide general support for the hypothesis that saccade targets are localized using a combination of retinal and eye position signals. All four studies, however, report that localization of a brief target presented during a saccade is not accurate. Moreover, they claim that the brief target is also mislocalized when it is presented just before, or even after, a saccade.

Dassonville *et al.* [4•] conclude that the oculomotor system uses a temporally distorted eye position signal when computing target location. According to their estimates, the eye position signal begins to change at least 100 ms before the onset of a saccade and does not reach a veridical value until about 50 ms after a saccade ends. This causes targets presented just before or after each saccade to be mislocalized. Using a similar experimental paradigm, Gellman and Fletcher [5•] reach even more far-reaching conclusions. Because of the high variability and low fidelity of the authors' estimate of the eye position signal, they conclude that the saccadic system must normally operate in retinal coordinates; localizing targets in spatial coordinates requires the use of an accurate eye position signal, a signal they believe is unavailable.

Abbreviations

EBN—excitatory burst neuron; **FEF**—frontal eye field; **IBN**—inhibitory burst neuron; **PEF**—posterior eye field; **SRBN**—saccade-related burst neuron.

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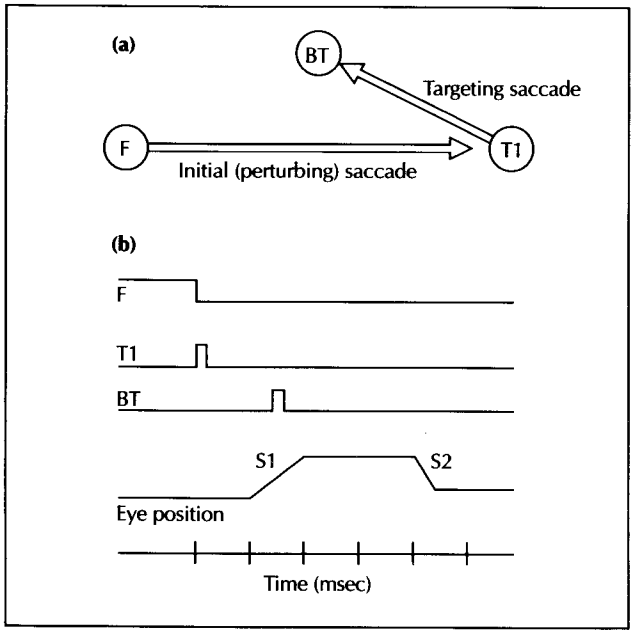

Fig. 1. Schematic diagram of a single target localization trial. **(a)** Representation of saccade trajectories and target locations. F—fixation point; T1—target for initial saccade; BT—brief target. **(b)** Timing of stimulus presentations. T1 appears when the fixation stimulus (F) is extinguished. BT is presented after a variable delay and, as illustrated, often appears during the perturbing eye movement. S1—initial (perturbing) saccade; S2—saccade directed toward the location of the BT (modified from [4•]).

Because the conclusions of Dassonville *et al.* [4•] and Gellman and Fletcher [5•] challenge an important tenet of existing oculomotor models, the methodological and logical bases of these conclusions should be critically assessed. In a related study, Miller [6•] used manual pointing to measure the localization of visual stimuli flashed during a saccade. He notes that in earlier studies estimates of the accuracy and time course of an eye position signal could be confounded by two other potential sources of information about the direction and amplitude of the perturbing saccade. First, the relative locations of the retinal images of the initial fixation target and the target for the perturbing saccade provide information about the direction and amplitude of the impending saccade. Second, in earlier studies, the target for the perturbing saccade was always located in the same position and an estimate of the location of the brief target could be based upon an anticipated change in eye position. Miller [6•] eliminated these sources of information about the metrics of the perturbing saccade by using an auditory stimulus as a fixation target and randomizing the location of the acoustic target that specified the goal of the perturbing saccade. Under these conditions, Miller [6•] obtained an estimate of the eye position signal that began to change 2 ms after the eye moved and reached a stable post-saccadic value with a time constant of about 70 ms. To what extent the different estimates of the time course of the eye position signal obtained from gaze pointing and manual-pointing tasks can be attributed to these procedural differences must be addressed in future experiments.

In these studies, all of the mislocalization errors were attributed to inaccuracies in an eye position signal. But other sources almost certainly contribute to the errors. Because of retinal processing time and afferent delays, the neural signals encoding the retinal location of the target may not reach cortical or subcortical areas until 50–100 ms after the stimulus appears. An eye movement may occur in this interval. To compensate, retinal signals could be referred to a delayed eye position signal [7], but localization errors would still occur because afferent delays are variable and depend upon the retinal eccentricity of the stimulus as well as stimulus intensity (see [6•] for references). Based upon these considerations, an alternative interpretation of the mislocalization data can be formulated: that saccadic mislocalization of visual targets does not occur because of a temporally distorted eye position signal that begins to change up to 100 ms before the onset of a saccade, but rather, that localization errors emerge when retinal signals with variable processing and afferent delays are combined with a temporally undistorted eye position signal, which may also have a variable delay [7]. Currently, there is no way to determine which, if either, of these extreme interpretations of the mislocalization data is correct. But both hypotheses should be considered when designing experiments to study the neural mechanisms for coding the location of a saccade target.

Gellman and Fletcher's conclusion [5•] that the oculomotor system works in retinal coordinates seems premature. In a natural environment, localization of visual targets that are present only during a saccade is rarely required. Usually, a target is continuously present, but the retinal representation of the target may be masked, as images of other objects in the textured environment sweep across the retina during the movement. Accordingly, an active fixation mechanism may inhibit the initiation of a saccade until an accurate signal of target position in space is obtained using a more stable retinal image and a veridical eye position signal. In this context, estimates of the accuracy and time course of eye position signals obtained when visual targets are presented while the eye is stationary become important. Such estimates can be obtained by measuring the accuracy of saccades that compensate for perturbations in eye position produced by unpredictable electrical stimulation of the superior colliculus [8]. Available evidence indicates that these perturbations can be compensated for quickly and accurately. This would not occur if the only available eye position signal were "highly variable and of low fidelity" [5•] or had a high degree of temporal distortion.

Signals and connections of the frontal eye fields

Important new findings concerning the role of frontal eye field (FEF) neurons in the control of saccadic

eye movements emerged from lesion [9•] and electrophysiological [10•,11••–13••] experiments. Pathways by which neurons in the FEF control the initiation and metrics of saccades have been clarified by studying the functional properties of neurons in the FEF that project to oculomotor regions of the pons [11••]. Corticopontine cells discharge in association with saccadic eye movements, are responsive to visual stimulation of the fovea, or have firing rates related to eye position. In addition, Segraves [11••] made the important observation that high-intensity, short-duration electrical stimulation of the FEF causes omnipause neurons in the pons to stop firing with a latency of 5 ms or less. The pontine omnipause neurons tonically inhibit pontine burst cells until a few milliseconds before saccade onset. Thus, this short-latency pathway enables relatively direct control of saccade initiation by FEF neurons.

Electrical stimulation of FEF neurons during ongoing spontaneous or visually guided saccades produces movements indicating that FEF neurons specify a change in eye position with reference to an earlier position of the eye rather than to the position of the eye when the stimulation train became effective [12••]. Depending on the timing and trajectories of the ongoing movement, saccades of almost any direction and amplitude can be evoked from a single stimulation site. These findings strongly suggest that, unlike the deeper layers of the superior colliculus where microstimulation always produces a movement with a particular direction and amplitude, FEF stimulation specifies a saccadic goal rather than a particular movement.

A recent experiment by Schlag-Rey *et al.* [13••] emphasizes the precise point-to-point anatomical projections from the FEF to the superior colliculus. When the eyes are stationary, the saccadic goal specified by FEF stimulation can be achieved by producing a movement with a particular direction and amplitude, a movement that could be initiated by excitation of the proper subset of collicular neurons. Indeed, if the eyes are stationary and a 5° rightward saccade is evoked by suprathreshold stimulation of a FEF site, an increase in the activity of saccade-related burst neurons (SRBNs) in the superior colliculus, normally active before the 5° rightward saccade, is observed [13••]. All other SRBNs display a decrease in activity. The same subset of collicular neurons will be activated, however, when the same FEF site is stimulated during a spontaneous or visually guided saccade, even if the stimulation-induced movement has a direction and amplitude different from the 5° rightward movement encoded by the site of collicular activity. A similar dissociation between the activity of SRBNs and the metrics of the movement that actually occurs has recently been observed by Stanford and Sparks [14••]. Collectively, these findings have important implications for models of oculomotor control. In most models, the collicular signal, serving as a primary input to the control circuitry in the pons and medulla, is the major determinant of the direction and amplitude of the ensuing saccade.

Because the collicular command signal can be dissociated from the metrics of the saccade that actually occurs, new models must recognize that the metrics of the movements may be modified by signals introduced after collicular commands are issued.

A comment on the use of the term 'retinocentric'

The term 'retinocentric' is used in the oculomotor literature in two ways: either to indicate that neural signals are tied to the retinal locus of a visual stimulus, or to indicate that the signal is in eye-centered coordinates rather than head-centered or environment-centered. Because of the dual usage of the term, the exact meaning is often ambiguous and may be interpreted incorrectly by readers. For example, cells in the FEFs are often said to carry retinotopic or retinocentric signals when "the critical signal from the frontal eye fields is neither the retinal location of a saccade target, nor its spatial location, but rather the saccadic movement the target will evoke" [15].

We suggest restricting the use of the term retinocentric to cases in which cells discharge if, and only if, a specific region of the retina is activated. Especially when discussing sensorimotor integration, retinocentric implies that visual signals are prepotent or dominant. It seems incorrect to use the term retinocentric to describe the activity of a cell before a saccade to an auditory target.

The terms 'oculocentric' or 'eye-centered' are more general, do not imply dominance of a particular sensory modality, and can be used to describe signals or events that may be related to the position of the eye, but not necessarily tied to activation of a specific region of the retina.

The role of the cerebellum in the generation of saccades

Recording and stimulation studies have demonstrated that the vermal lobules VIc, VII and the fastigial nuclei are involved in saccadic eye movements [16,17•,18,19]. An understanding of their contribution to saccade generation would provide insight into the cerebellar control of movement, in general, and, more specifically, into the distributed oculomotor system. As is apparent in this brief review, the existing data shed little light on the exact function of these regions, but do spawn more questions, some of which are posed below.

Lesions in these regions produce a saccadic dysmetria that is markedly modulated by initial orbital position [20,21], suggesting that the cerebellum participates in compensating for the viscous and elastic properties of the eye. Recordings in the posterior vermis have re-

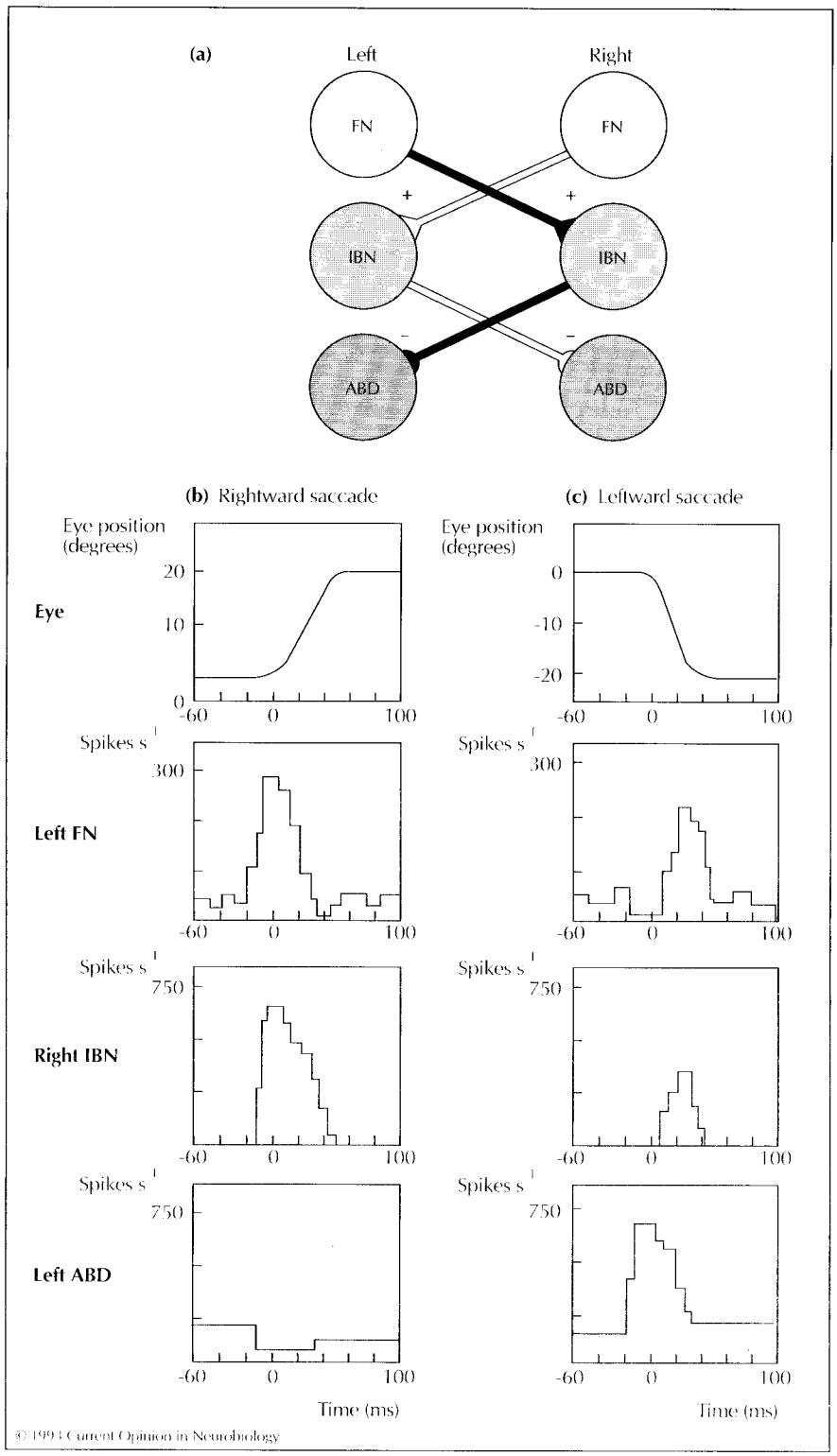

Fig. 2. Possible mechanism for 'braking' saccades without antagonist motoneuron excitation. **(a)** Schematic of fastigial nucleus (FN)–inhibitory burst neuron (IBN)–abducens motoneuron (ABD) connections. Excitatory connections are shown with triangular synapses and inhibitory ones with circular synapses. **(b)** and **(c)** Hypothetical activity associated with a rightward and a leftward saccade. For rightward movements, the left FN will fire early, exciting the right IBN, and, therefore, influencing the pause in the left ABD. **(c)** For leftward movements, the left FN will exhibit the late burst (associated with saccade offset), causing the right IBN to fire late, which will then alter the rate of decrease of ABD activity. The opposite will happen in the right FN–left IBN–right ABD. The firing rate of the IBN in the off-direction **(c)** is shown as much smaller than in the on-direction **(b)** as was reported by Scudder *et al.* [28].

vealed cells whose activity during saccades is modulated by the initial position of the eye [22]. Although the primary output of this region is the caudal fastigial nuclei [23], the single study that recorded caudal fastigial activity during both centripetal and centrifugal saccades showed no modulation of firing rate that depended upon initial eye position [16]. These paradoxical results beg for further studies concerning the influence of orbital position on fastigial activity.

Stimulation of the Purkinje cells in lobules VIc, VII produces ipsilateral saccades [18,24••]. That the inhibitory Purkinje output results in an excitatory burst in the brainstem saccadic-generating circuits is puzzling. It

suggests that inhibition and/or disinhibition of tonic activity is sufficient to trigger the burst in the excitatory burst neurons (EBNs).

Most fastigial and Purkinje cells burst for both ipsilateral and contralateral saccades [16,19]. Ohtsuka and Noda [25•] have shown that the mossy fiber inputs to the vermis burst to either contralateral or ipsilateral saccades, but not both; hence, the bidirectional Purkinje bursts appear to result from converging mossy fiber inputs. The timing of the mossy fiber burst offset is correlated with the movement offset, regardless of movement direction [25•]. The vermal circuitry transforms the mossy fiber signal so that for ipsilateral movements, the Purkinje cell burst offset, is correlated with movement completion, but for contralateral movements, the Purkinje cell burst onset (not offset) is correlated with movement completion [25•].

The fastigial neurons that Ohtsuka and Noda studied [16] display a late burst for ipsilateral movements. This burst onset is associated with movement completion, but the duration of the burst is not correlated with saccade duration. For contralateral movements, however, the burst precedes movement onset, and burst duration is correlated with saccade duration. These disparate activities suggest that two different functions are performed for contralateral as opposed to ipsilateral saccades. The association of burst onset with the completion of ipsilateral saccades suggests that the cerebellum is involved in coding motor error cessation. It seems unlikely, however, that this late burst of fastigial neurons actively brakes the eye by applying an opposing force to the antagonist circuit, as there is no clear evidence of an active brake [26].

Where is the bidirectional burst reflected in the brainstem nuclei? It does not appear at the level of the EBNs, which receive a fastigial projection [27]. The inhibitory burst neurons (IBNs), which lie immediately caudal to the abducens nuclei and which inhibit the contralateral abducens, receive a predominantly contralateral fastigial projection [27]. A few of the cells studied in this region demonstrate a bidirectional burst [28]. The timing of these bursts, with respect to movement offset, was not examined, leaving it unclear whether this activity resembles the fastigial activity. As illustrated in Fig. 2, the fastigial projection to the IBNs could predict and arrest potentially hypermetric saccades without involving antagonist motoneuron action.

The fastigial nuclei can be functionally divided into a rostral and caudal part [29]. The rostral region comprises cells that respond to vestibular and optokinetic stimuli, but do not exhibit any eye movement sensitivity. The caudal region comprises cells that respond during saccadic movements, intermixed with cells that have both a vestibular and a smooth pursuit response. Because vestibular and saccadic cells are largely segregated in the vermis [19], the interspersion of cell types in the caudal region implies a convergence of Purkinje fibers. Both the caudal and the rostral regions share a common characteristic: some cells display a strikingly inconsistent firing rate for identical stimuli. A subset of caudal fastigial neurons displays marked response variability, even to the point of exhibiting a vigorous burst on one trial and no response at all for a similar saccade on the next trial (EJ Barton, DL Sparks, unpublished data). Similarly, rostral cells often exhibit fluctuations in response to identical vestibular or optokinetic stimuli, and some rostral vestibular cells will temporarily stop firing to stimuli that previously elicited vigorous responses [29]. This lability across the different cell types suggests a common operation is being performed for the disparate movements. As error correction is one putative function of cerebellum processing, this inconsistent firing rate could reflect the inconsistent internal error of the saccadic generator.

Conclusions

Although recent experiments report that the localization of brief targets presented during an ongoing saccade is not accurate, we argue that it would be premature to conclude that an inaccurate or damped eye position signal is always used to compute the location of saccade targets. Additional studies incorporating the control procedures suggested by Miller [6•] are needed and the accuracy of the eye position signals used to compute target location when the eye is stationary (the usual situation) must be considered.

Recent studies of the FEF and superior colliculus report the surprising finding that the commands issued by the superior colliculus can be dissociated from the metrics of the movements that actually occur [13••,14••]. Experiments clarifying the neural bases of this dissociation will provide new insights into the overall organization of the saccadic system.

How do the patterns of saccade-related activity observed in the cerebellar vermis and fastigial nucleus influence the signals reaching the extraocular muscles? A review of recent work indicates that this question is still unresolved. Recent findings do add new pieces to the puzzle and form the bases of hypotheses requiring additional experiments.

References and recommended reading

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Subjects were asked to look at the location of a visual target flashed before, during, or after a visually guided saccade. Saccades to the perisaccadic flash were not accurate. These data are interpreted as supporting the hypothesis that the oculomotor system uses a damped eye position signal when computing target location.

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