# Model of the Control of Saccades by Superior Colliculus and Cerebellum

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**Quaia, Christian, Philippe Lefèvre, and Lance M. Optican.** Model of the control of saccades by superior colliculus and cerebellum. *J. Neurophysiol*. 82: 999–1018, 1999. Experimental evidence indicates that the superior colliculus (SC) is important but neither necessary nor sufficient to produce accurate saccadic eye movements. Furthermore both clinical and experimental evidence points to the cerebellum as an indispensable component of the saccadic system. Accordingly, we have devised a new model of the saccadic system in which the characteristics of saccades are determined by the cooperation of two pathways, one through the SC and the other through the cerebellum. Both pathways are influenced by feedback information: the feedback determines the decay of activity for collicular neurons and the timing of the activation for cerebellar neurons. We have modeled three types of cells (burst, buildup, and fixation neurons) found in the intermediate layers of the superior colliculus. We propose that, from the point of view of motor execution, the burst neurons and the buildup neurons are not functionally distinct with both providing a directional drive to the brain stem circuitry. The fixation neurons determine the onset of the saccade by disfacilitating the omnipause neurons in the brain stem. Excluding noise-related variations, the ratio of the horizontal to the vertical components of the collicular drive is fixed throughout the saccade (i.e., its direction is fixed); the duration of the drive is such that it always would produce hypermetric movements. The cerebellum plays three roles: first, it provides an additional directional drive, which improves the acceleration of the eyes; second, it keeps track of the progress of the saccade toward the target; and third, it ends the saccade by choking off the collicular drive. The drive provided by the cerebellum can be adjusted in direction to exert a directional control over the saccadic trajectory. We propose here a control mechanism that incorporates a spatial displacement integrator in the cerebellum; under such conditions, we show that a partial directional control arises automatically. Our scheme preserves the advantages of several previous models of the saccadic system (e.g., the lack of a spatial-to-temporal transformation between the SC and the brain stem; the use of efference copy feedback to control the saccade), without incurring many of their drawbacks, and it accounts for a large amount of experimental data.

## INTRODUCTION

The saccadic system (i.e., the neural system that controls the rapid eye movements called saccades) has attracted the attention of many investigators during the last 40 years. Thanks to the combined efforts of so many researchers, a great deal of data are now available about the pattern of neural activity, the anatomy of functional connections, and the effects of lesions and electrical stimulation in several brain areas involved in controlling saccades. The availability of such a large database and the relative simplicity of the mechanical system to be controlled (the eye plant), has prompted models of the saccadic system to spring up like mushrooms on a damp forest floor.

In 1975, a milestone in the history of saccadic modeling, the Robinson model, was published (Robinson 1975; Zee et al. 1976). The central idea of that model, inherited by almost all subsequent models of the saccadic system, was that saccades are controlled by a local feedback loop, which in Robinson's model was used to compare the desired position of the eyes with an internal estimate of their actual position, thus producing an estimate of the instantaneous (or dynamic) motor error. This model, as well as others derived from it, was mainly conceptual, and many of its building blocks were not closely associated with anatomic structures. However, the growth of anatomic and physiological knowledge, due to the large number of experiments carried out after 1975 (largely prompted by the many predictions of Robinson's model), impelled modelers to identify the different parts of their models with specific regions of the brain.

### *Why a new model of the saccadic system?*

Although the concept that several brain structures cooperate to produce fast and accurate saccadic eye movements has long been widely accepted, models necessarily concentrate on a restricted subset of these structures. Initially models included only the brain stem circuitry, but soon the great amount of data available about the superior colliculus (SC) made it essential to find a role for this midbrain structure. Accordingly, models focused on the role played by the SC in controlling saccades and in determining the firing pattern observed in brain stem neurons. However, during the last 10 years, new experimental evidence has induced modelers to attribute an increasing importance to the SC. This trend has lead to the development of a fairly large family of models that impute to the SC a dominant role in determining saccade metrics, and that thus could be dubbed ''colliculocentric'' (Arai et al. 1994; Droulez and Berthoz 1988; Lefèvre and Galiana 1992; Optican 1994; Van Opstal and Kappen 1993; Waitzman et al. 1991).

One of the major problems with colliculocentric models is that they have difficulties in explaining why lesions of the SC do not result in large and enduring deficits. In particular, it is

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well known (Schiller et al. 1980) that collicular ablations impair the ability to make saccades only for a brief time. Furthermore even in the acute phase of a collicular lesion, the trajectory and speed of saccades can be affected without a striking loss of accuracy (Aizawa and Wurtz 1998; Quaia et al. 1998a). Conversely, it has been shown that cerebellar lesions (e.g., Optican and Robinson 1980) induce permanent deficits, affecting dramatically the accuracy and consistency of saccades. Thus we feel that a model is needed that gives less import to the SC and gives more relevance to the role of the cerebellum in controlling saccades.

### *What is the role of the cerebellum in controlling saccades?*

For decades, the role attributed to the cerebellum by the few models of the saccadic system that considered it (e.g., Dean et al. 1994; Grossberg and Kuperstein 1989; Optican 1986; Optican and Miles 1985) has been to compensate for alterations of the oculomotor plant due to age or injury and to adjust the saccadic command as a function of the orbital position, compensating for plant nonlinearities. Such an approach was justified on the basis that cerebellar lesions impair the ability of the system to compensate for changes in the oculomotor plant (Optican and Robinson 1980) and induce saccadic dysmetria (e.g., Optican and Robinson 1980; Ritchie 1976; Robinson et al. 1993; Sato and Noda 1992b; Takagi et al. 1998), often as a function of orbital position. In all those schemes, the assumption was made (implicitly or explicitly) that the extracerebellar pathway generates, using a feedback loop controller, a command that is a fixed function of the desired displacement of the eyes; that command then is supplemented by a fixed (but adaptable over the long term) command produced by the cerebellum. Thus in those schemes the extracerebellar pathway guarantees the consistency of saccades, whereas the cerebellum is responsible for their accuracy. The major failure of this scheme is that it does not account for one of the most striking effects of cerebellar lesions: the increased variability of saccades. In fact, after cerebellar impairment, saccades not only loose their characteristic accuracy, becoming dysmetric (hypermetric or hypometric depending on the cerebellar areas affected by the lesion), but they also become subject to a conspicuous trial-to-trial variability, affecting both amplitude and direction (e.g., Robinson 1995; Robinson et al. 1993; Takagi et al. 1998).

This last observation, which has been reported after both permanent and temporary lesions, clearly is inconsistent with the cerebellar output being simply an adaptive function of the starting orbital position and the desired displacement of the eyes. Accordingly, we propose that the cerebellar contribution is carefully tailored during each saccade to compensate for both the characteristics of the oculomotor plant and the variability present in the rest of the saccadic system during the preparation and execution of the movement. In our model the cerebellar output is tailored in flight, because it is part of a feedback loop, functionally similar to that proposed by Robinson as the core component of the saccadic system. Thus in our scheme the cerebellum is responsible for both the accuracy and the consistency of saccades. The increased variability observed after cerebellar lesions is simply due to the unmasking of variability inherently present in the rest of the saccadic system. Unmasking occurs because the mechanism (i.e., the feedback loop) that normally compensates (at least partially) for the variability is itself impaired. Previous models did not include the cerebellum in the feedback path and thus could not account for the increased variability.

We will show here how the presence of two separate pathways, one through the superior colliculus and the other through the cerebellum, can account for many of the properties of the saccadic system and for a great deal of anatomic and physiological data as well as for the effects of lesions and electrical stimulation. In another paper (Lefèvre et al. 1998), we presented a distributed implementation of the model described here. In that paper, we used simulations to demonstrate that this model *1*) produces normal saccades that lie on the so-called main sequence (Bahill et al. 1975), *2*) guarantees the accuracy of saccades regardless of their speed, *3*) replicates the patterns of activation observed in collicular burst, buildup, and fixation neurons as well as in fastigial oculomotor region (FOR) neurons, *4*) exerts a partial trajectory control, and *5*) replicates the effects of sustained electrical stimulation of the SC (i.e., it generates staircases of saccades). The decision to present the distributed implementation of the model as a separate paper was motivated by our desire to focus here on the neurophysiological basis of the model in a paper of reasonable length. Nonetheless, we will present here some additional simulations of that model, mainly to show how it can account for the effects of collicular and cerebellar lesions. We also will discuss the implications and the advantages that this organization has for controlling eye movements, as well as its limits and possible extensions. Finally particular care will be devoted to illustrating the predictions of the model and describing experimental tests that could corroborate or refute it.

Earlier accounts of this model appeared in abstract form (Lefèvre et al. 1996; Optican et al. 1996; Quaia et al. 1996).

## BACKGROUND

To justify our choices in attributing roles to the large number of cell types and anatomic interconnections that we are modeling, we now briefly describe a subset of the relevant literature, pointing out inconsistencies in the data, some of the previous modeling studies, and alternative interpretations. Because this analysis is not a complete review of the pertinent literature, we will refer to existing reviews for all the topics on which there is general agreement, concentrating our efforts on the most controversial or least explored subjects.

### *Intermediate layers of the SC*

Since the early 1970s, single-unit recordings (Schiller and Stryker 1972; Wurtz and Goldberg 1971, 1972) and electrical stimulation experiments (Robinson 1972; Schiller and Stryker 1972) indicated that the intermediate layers of the SC must play an important role in producing saccades. Cells in the SC (from now on we always refer implicitly to the intermediate layers of the SC) are characterized by fairly large movement fields (i.e., the range of movements associated with activation of a neuron) (Sparks et al. 1976), which are organized topographically (i.e., cells close together have similar movement fields). Neurons that discharge in correspondence with small saccades are located rostrally, whereas large movements are encoded in more caudal sites. Accordingly, electrical stimula-

tion at rostral sites results in small saccades, whereas at more caudal sites larger saccades are evoked. These results indicate that the saccadic (or target) vector is spatially, and not temporally, encoded on the SC; movements toward targets in the left visual hemifield are encoded in the right SC and vice-versa (for a review, see Guitton 1991; Sparks and Hartwich-Young 1989; Wurtz 1996).

Recently saccade-related neurons in the SC have been divided into three classes according to their pattern of activity and location: burst, buildup, and fixation neurons (Munoz and Wurtz 1992, 1993a, 1995a; Wurtz and Optican 1994). The burst neurons, as classified by Munoz and Wurtz (1995a), are characterized by a brisk discharge synchronized with saccade onset, have a closed movement field (i.e., they discharge only for saccades around an optimal vector), and are probably the same cells described by Sparks and colleagues as saccade-related burst neurons (SRBNs) (Sparks 1978; Sparks and Mays 1980). Fixation neurons, located in the rostral pole of the SC, behave in an opposite manner, i.e., they discharge during active fixation and pause during saccades in any direction (except sometimes they do not pause, or even burst, for small, contraversive saccades). These cells pause immediately before the onset of a saccade and resume firing at the time of saccade termination (Munoz and Wurtz 1993a). The third class of cells is represented by the so-called buildup neurons (located among and just below the burst neurons), which are characterized by a small buildup of activity preceding saccades (hence their name) and have an open movement field (i.e., they discharge, albeit with different intensities, for all saccades in one direction larger than a certain amplitude). Some, but not all, buildup cells are characterized by a burst occurring at saccade onset, similar to that of the burst cells. In the majority of buildup cells, this burst component has a closed movement field, similar to that of the burst neurons (see Munoz and Wurtz 1995a, their Figs. 7*B* and 8). One striking characteristic of the buildup neurons is that some of the activity (but not the burst component) in the buildup layer seems to spread rostrally across the SC during a saccade (Munoz and Wurtz 1995b). This observation, based on the analysis of the time course of cells' discharge during saccades of different amplitude, is reminiscent of the finding that in the cat the locus of collicular activation appears to move rostrally during a saccade (Munoz et al. 1991a), possibly encoding instantaneous gaze error spatially (Guitton et al. 1993; Munoz et al. 1991b).

### *Role of the SC in current models of the saccadic system*

The function classically attributed to the SC is to provide the desired displacement signal to the brain stem circuitry (e.g., Grossberg and Kuperstein 1989; Scudder 1988; Tweed and Vilis 1990). Thus in these schemes, the SC is outside the local feedback loop that has been postulated to control saccades. In many of these models, the collicular output is processed by a spatial-to-temporal transformation (STT, a process or mechanism used to transform information from a spatial encoding to a temporal encoding), which converts the location of the activated locus on the collicular map into a temporal signal encoding the desired displacement of the eyes.

Recently the finding that there is a fairly good correlation between the level of activity of some collicular neurons and the residual motor error prompted the development of a model (Waitzman et al. 1991) in which the burst neurons encode motor error with their temporal discharge. In this case, as well as in similar models (Arai et al. 1994; Van Opstal and Kappen 1993), the SC becomes part of the local feedback loop. One of the major advantages of these schemes is that they do not require an STT because the information that is encoded spatially on the SC (i.e., the desired displacement) is never converted into a temporal code and the dynamic motor error is encoded temporally in the brain stem as well as in the SC. The lack of an STT, which is a feature of several other models as well as the model presented here (see following text), is very important, because it simplifies considerably the connectivity from the SC to the brain stem (Quaia and Optican 1997).

Unfortunately there are some major problems with the scheme proposed by Waitzman and colleagues: first of all, because it posits that only the level of collicular activation, but not its spatial distribution, is under feedback control, it cannot account for the purposeful curvature of saccades [which is such that when the eyes are not headed in the correct direction they are brought back toward the target (Becker and Jürgens 1990; Erkelens and Sloot 1995; Erkelens and Vogels 1995). This behavior is particularly prominent after collicular reversible inactivation (Aizawa and Wurtz 1998)]. Another problem with this scheme is that it does not explain why sustained electrical stimulation of the colliculus produces movements the amplitude of which is a function of the rostrocaudal position of the electrode on the SC map (Paré et al. 1994; Robinson 1972; Stanford et al. 1996).

Because of these problems, we think it is unlikely that the collicular burst neurons are part of a feedback loop used to tightly control saccade amplitude. Nonetheless we think that the correlation between burst neuron discharge and dynamic motor error is not just an epiphenomenon. In fact, when saccades are interrupted in midflight by electrical stimulation of the region containing omnipause neurons (OPNs), the burst neurons' activity goes temporarily to zero (supposedly because of antidromic stimulation of collicular fixation neurons) and then resumes a level of activity that is again compatible with the encoding of dynamic motor error (Keller and Edelman 1994). This last finding makes the hypothesis that the burst neurons' discharge simply is preprogrammed very unlikely.

The peculiar characteristics of the buildup neurons' discharge, and particularly the rostral spread of activity during a saccade, makes it tempting to ascribe to this class of neurons a distinct function (e.g., Wurtz and Optican 1994). In particular, it has been proposed (Optican 1994) that the displacement of the center of activity on the buildup layer could represent an internal estimate of the progress of the saccade toward the target (i.e., functionally represent the output of a displacement integrator). This role for the spread of activity is similar to the role attributed to the SC by models based on cat data (Droulez and Berthoz 1988; Lefèvre and Galiana 1992).

Unfortunately, a close inspection of the pattern of activity of monkey buildup neurons reveals that an interpretation of the spread of activity as functionally important in controlling the movement is problematic. For example, to have a significant effect, the change of spatial distribution of activity during a saccade should be quite dramatic. However, the activity that spreads across the buildup layer during a saccade is only a small fraction of the activity that is produced at the site corresponding to the target (often characterized by a burst, see

preceding text). Thus the center of gravity of the activated area in the buildup layer does not change much during the movement (Anderson et al. 1998). One could argue that the spread of activity over the SC map could have an effect by inducing a timely reactivation of the fixation neurons, contributing to stopping the movement. However, under this hypothesis, lesions of the rostral pole of the colliculus are expected to induce dysmetria (in particular hypermetria), whereas such lesions do not seem to affect saccade amplitude (Munoz and Wurtz 1993b). Thus even though it is certainly possible that the reactivation of the fixation zone plays a role in stabilizing the system, we think that it is unwarranted to attribute to it a dominant role in the determination of saccade amplitude. Finally it should be noted that this spread of activity begins well before saccade onset (e.g., during a 50° saccade the 3° buildup cell gets activated between 100 and 50 ms before saccade onset and reaches its maximal activation $\geq$20 ms before saccade onset) (Munoz and Wurtz 1995b, Fig. 3). This observation makes the hypothesis that the spread is controlled by feedback information tightly related to the movement pretty unlikely even though it does not rule out less tight feedback schemes.

One final problem common to all colliculocentric models is that they cannot easily account for some recent findings that suggest a dissociation between saccade metrics and the collicular locus activated. For example, it has been shown that the collicular movement fields are different when comparing visually guided movements with saccades to remembered targets (Stanford and Sparks 1994). Analogous results have been obtained using the averaging saccade task (Edelman and Keller 1998), after adaptation induced with the double step paradigm (Frens and Van Opstal 1997; Goldberg et al. 1993), and when saccades to moving targets are considered (Keller et al. 1996). In all these cases, the collicular locus activated appears to be a function of the location of the target and not of the movement evoked. As will become clear later, these results, which challenge the various colliculocentric models, are perfectly compatible with our model; in fact, in our scheme the actual displacement of the eyes is determined by the cerebellum, which decides when to stop the movement.

### *Cerebellum*

A great deal of evidence points toward lobuli VIc and VII of the cerebellar vermis as being involved in the control of saccadic eye movements. First of all, only very small currents are needed to evoke saccades from this region (Noda and Fujikado 1987), whereas much higher currents are needed to evoke saccades from nearby lobuli (Keller et al. 1983; Ron and Robinson 1973). Second, ablations of this area result in dysmetric movements (Ritchie 1976; Takagi et al. 1998). Finally, neurons in this area present saccade-related activity (Helmchen and Buttner 1995; Ohtsuka and Noda 1995; Sato and Noda 1992a), whereas activity in neurons belonging to other vermal lobuli is not modulated during saccades (Sato and Noda 1992a). Unfortunately, there is not much agreement regarding the pattern of saccade-related activity of these neurons. Whereas Ohtsuka and Noda (1995) reported that neurons in the oculomotor vermis produce an early burst for ipsilateral saccades and a late burst for contralateral movements, Helmchen and Büttner (1995) reported that the preferred direction (i.e., the direction associated with the early burst) is ipsilateral for half the cells and contralateral for the other half.

In turn the oculomotor vermis projects to an ellipsoidal region in the caudal fastigial nucleus (Yamada and Noda 1987), the so-called FOR. These projections are strictly ipsilateral and topographically organized (Carpenter and Batton 1982; Courville and Diakiw 1976; Noda et al. 1990). Because the vermis does not project directly outside the cerebellum, the signals present in the FOR determine the effect of the cerebellar vermis on saccades. Consequently any model that is concerned with the control of saccades by the cerebellum has to give strong import to the saccade-related discharge of the FOR neurons. Fortunately there is general agreement on the pattern of discharge of these neurons (Fuchs et al. 1993; Helmchen et al. 1994; Ohtsuka and Noda 1990, 1991). They produce an early burst of activity for movements in one direction (preferred direction) and a late burst, time-locked with the end of the movement, for saccades in the opposite direction. The preferred direction always has a contralateral horizontal component.

### *Model*

In this section, we describe our model in detail. We first outline the structure of the model to provide a general idea of the role that the various areas play in the overall picture. To avoid any misunderstanding, we stress that all the connections and patterns of activity described hereafter refer to our model, and we will indicate, by means of citations to the relevant literature, when they are supported by experimental findings. Similarly, when we make assertions relative to the role played by brain areas in controlling saccades, we refer to our model of the saccadic system not to the saccadic system itself, even when this is not explicitly stated.

### *Overall structure of the model*

In designing this model, we gave primary significance to the patterns of saccade-related activity recorded from single cells in the SC, in the cerebellum (especially the fastigial nuclei, which contain the cerebellar neurons that project to the brain stem saccadic circuitry), and in the brain stem. Using many of the known anatomic connections between these different areas, we have created a model in which the metric and dynamic characteristics of saccades are determined by the cooperation of two parallel pathways (Fig. 1). The first pathway (collicular pathway) involves the cerebral cortex (which provides the target location in retinotopic coordinates), the SC, the premotor medium-lead burst neurons (MLBNs) [which are divided into excitatory (EBN) and inhibitory (IBN) burst neurons] and the motoneurons (MNs) that innervate the extraocular muscles. The core structure of this pathway is the SC, which plays two roles: first, it determines the onset of the saccade, by releasing the excitation provided to the OPNs, which tonically inhibit (*gate*) the MLBNs in between saccades. Second, it drives the eyes toward the target. Thus this pathway provides a *go* signal and what we call a *directional drive.*

The second pathway (cerebellar pathway) involves the cerebral cortex, the SC (which just relays the target information), the cerebellum (vermis lobuli VIc and VII and FOR), MLBNs, and MNs. The cerebellum, which is the central structure of this

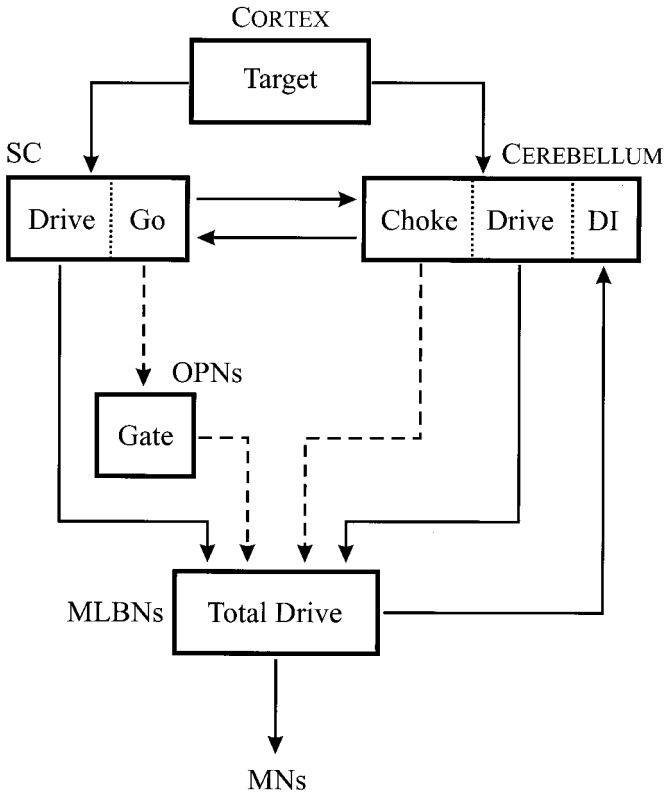


FIG. 1. Overview of model structure. There are 2 major pathways, one through the superior colliculus (SC) and the other through the cerebellum. SC performs 2 functions: it determines the onset of the saccade (Go) by causing the omnipause neurons (OPNs) to release their inhibitory action (Gate) on the medium lead burst neurons (MLBNs). SC also provides an excitatory input (Drive) to the MLBNs. Cerebellum performs 3 functions: it provides an additional drive to the MLBNs, it monitors the progress of the saccade by acting as a displacement integrator (DI), and it chokes off the drive to the MLBNs, ending the movement. Sum of the 2 drives (unless modulated by the choke) is passed on to the motoneurons (MNs) and determines the velocity of the eyes. —, excitatory signals; - - -, inhibitory signals.

second pathway, plays three roles: *1*) it provides an additional directional drive, *2*) it monitors the progress of the saccade toward the target (acting as a displacement integrator, DI), adjusting its output to compensate for directional errors, and, when the eyes approach the target, and *3*) it chokes off the drive provided by these two pathways to the motoneurons, ending the saccade. Thus this pathway also provides two signals to the brain stem circuitry: a *directional drive* and a *choke* signal.

As will become clear further on, there is a fundamental difference in our model between the collicular and the cerebellar drives: whereas the first cannot change direction during a saccade (i.e., the ratio between the horizontal and vertical components of the collicular drive is fixed throughout the movement), the second is adjustable in direction.

### *Brain stem circuitry*

The brain stem network that we use in our model is supported by a great deal of experimental evidence and is essentially identical to that used in several other models. Thus here we just briefly describe its fundamental aspects. Several reviews describing the evidence for the connections we use have been published (e.g., Büttner-Ennever and Büttner 1988; Fuchs et al. 1985; Hepp et al. 1989; Moschovakis et al. 1991).

The basic structure of the horizontal channel of the brain stem circuitry implemented in our model is represented in Fig. 2. The muscles innervated to move the eyes in the horizontal plane (i.e., to rotate the eye ball around the vertical axis) are the lateral recti (LR), which rotate the left eye to the left and the right eye to the right (i.e., they rotate the eyes temporally), and the medial recti (MR), which exert opposite effects (i.e., they rotate the eyes nasally). When a conjugate movement of the eyes is produced, the LR of one eye and the MR of the other eye act as agonists (i.e., their tension is increased), whereas the other two muscles act as antagonists (i.e., their tension is decreased). The innervation to the lateral recti is provided by motoneurons (MN) located in the ipsilateral abducens (VI) nucleus; intermixed with these motoneurons are interneurons (IN), which presumably receive the same inputs and project to the motoneurons of the contralateral MR, located in the contralateral oculomotor (III) nucleus. We modeled the eye plant as a second-order system, with time constants of 0.15 and 0.005 s (Keller and Robinson 1972; Robinson 1973), and

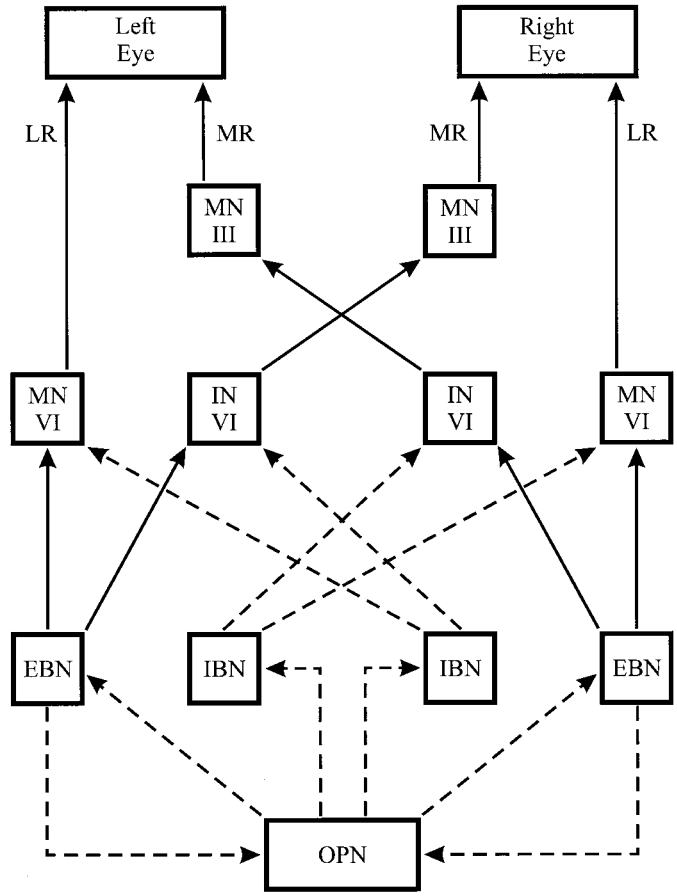


FIG. 2. Brain stem circuitry for the generation of horizontal saccades. OPNs tonically inhibit excitatory (EBN) and inhibitory (IBN) burst neurons between saccades. In turn, EBNs inhibit OPNs (through an inhibitory interneuron, not shown), keeping them off during saccades. EBNs excite directly the MNs of the ipsilateral lateral rectus (LR) muscle and, indirectly through interneurons (IN), the MNs of the contralateral medial rectus (MR) muscle. Conversely the IBNs inhibit directly the MNs of the contralateral LR and, indirectly through INs, the MNs of the ipsilateral MR. Drive to each population of MNs is determined by the difference between the activity of EBNs of one side and IBNs on the other side of the brain stem.

because the tension exerted by a pair of muscles is a linear function of the difference in innervation between the agonist and the antagonist (Haustein 1989), we lumped the two muscles into one equivalent muscle.

Each side of the brain stem contains two populations of MLBNs, one (EBNs) that excites the ipsilateral MNs and INs and another (IBNs) that inhibits contralateral MNs and INs. These populations of MLBNs are inhibited by OPNs (located across the midline), which fire tonically during periods of fixation and pause during saccades, thus acting as a gate. In turn, MLBNs inhibit the OPNs, helping to keep them inactive during saccades. Because no direct projections from the IBNs to the OPNs have been found (Büttner-Ennever and Büttner 1988), we assume that the EBNs inhibit the OPNs through an interneuron.

The difference between the signal carried by the ipsilateral EBNs and that carried by the contralateral IBNs determines the velocity of the horizontal component of the movement. This velocity signal then is integrated by neurons located in the nucleus prepositus hypoglossi and in the vestibular nuclei (for clarity this pathway has been omitted in Fig. 2); the output of this neural integrator, which is fed to the motoneurons, is used to hold the eyes in an eccentric position at the end of the saccade.

The scheme for the vertical channel is organized similarly (e.g., Crawford and Vilis 1992), even though two pairs of muscles for each eye (vertical recti and obliques) are activated during vertical movements. For the sake of simplicity and because we consider only movements in Listing's plane, we modeled the vertical channel in the same way as the horizontal channel (which is reasonable under the hypothesis described in Quaia and Optican 1998).

### *Superior colliculus*

INPUTS TO BURST NEURONS. We have modeled four inputs to the collicular burst neurons: the first input comes from the frontal eye fields (FEF), and it encodes the location of the target for the impending saccade in retinotopic coordinates (*saccadic command*) by providing a topographically organized excitatory input to the SC. Each input fiber discharges maximally for one saccade vector; its discharge decreases following a Gaussian function as the direction of the movement deviates from the preferred vector and following a log-Gaussian function as the amplitude of the movement deviates from the preferred vector. This is in agreement with recordings from movement cells in FEF (Bruce and Goldberg 1985). The width of the FEF movement fields is larger than that of collicular burst neurons, and we assume that they are narrowed by intracollicular on-center-off-surround connections (Grossberg 1973, 1988), which determine the size of the burst neurons' movement fields.

Similarly, we modeled the temporal characteristics of this signal as being less brisk than those of the collicular burst neurons; in particular, the FEF activity rises earlier compared with saccade onset, the activation outlasts the saccadic movement, and the activity does not decay much during the saccade (Fig. 3*A*). Such characteristics are compatible with recordings from cortico-tectal neurons in FEF (Segraves and Park 1993), which probably are the movement cells studied by Bruce and Goldberg (1985).

The second input to the burst neurons (*fixation command*) is

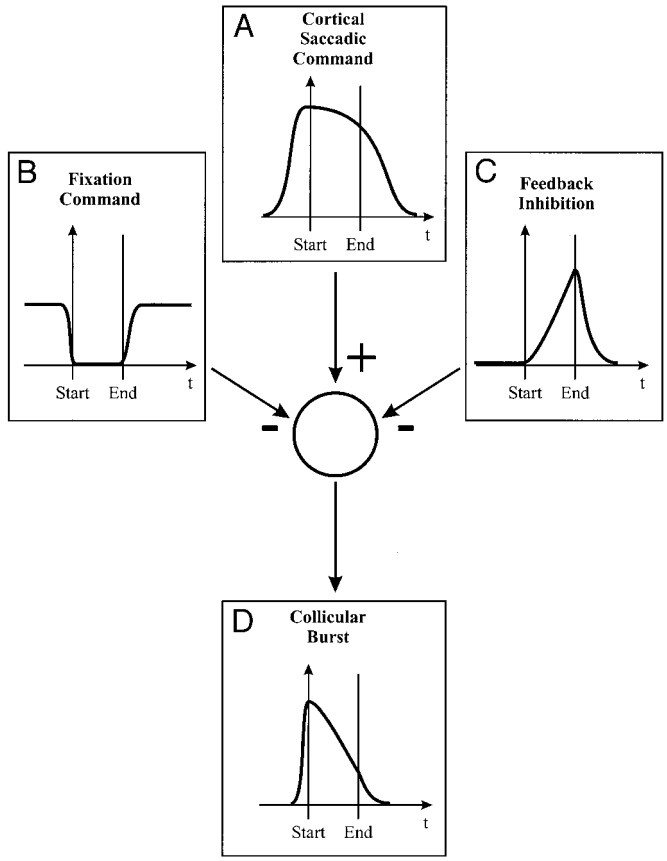


FIG. 3. Schematic of the temporal characteristics of the inputs to model collicular burst neurons. *A*: cortical saccadic command is a phasic excitatory input that starts firing before saccade onset and outlasts the movement. *B*: fixation command is a tonic inhibitory input that is switched off at the beginning of the movement and is reactivated after the saccade is over. *C*: feedback inhibition input approximately encodes the progress of the saccade toward the target. *D*: output of collicular burst neurons is determined by the sum of the 3 signals described above. It is a burst that starts just before saccade onset and is almost over by saccade end.

provided by the collicular fixation neurons, which provide inhibition until just before saccade onset, when they turn off, allowing the burst neurons to start discharging (Fig. 3*B*). These neurons then are reactivated around the end of the saccade. This is compatible with recordings in the rostral pole of the SC (Everling et al. 1998; Munoz and Wurtz 1993a). The relative weight of these first two inputs determines, in our model, the onset of burst neurons' discharge, and thus the latency of the movement.

The third input to the burst neurons encodes, in a relatively sloppy way, the magnitude of the displacement since the beginning of the saccade. This signal, which we call *feedback inhibition*, inhibits the burst neurons, thus determining the observed decay of activity as a function of dynamic motor error (Fig. 3*C*); as we will explain at length later, it does not need to be particularly accurate. Such an extracollicular signal is necessary in our model to reproduce the results of Keller and Edelman (1994) and Waitzman et al. (1991), as also pointed out by Keller and colleagues (Anderson et al. 1998), but there is no direct experimental evidence for (or against) the existence of a feedback inhibition signal.

The fourth and final input to the collicular burst neurons also comes from the cortex, but, because it is weak and has minimal effect on burst neurons' discharge, we will describe it later. For

now it suffices to say that this fourth input is, in our model, the source of the early activity observed in buildup cells. It will be made clear later why we propose that the burst neurons receive this input as well.

ACTIVITY OF SC BURST NEURONS. We modeled the output of the burst neurons as a burst of activity that starts just before the beginning of the saccade and is almost over by the end of the saccade (Fig. 3*D*). Thus in our model, the burst neurons are only partially clipped, i.e., the neurons are still active at the end of the movement, even though at a fairly low level (~20% of maximum activation). The choice of keeping this residual activity at the end of the movement is due to the experimental finding that, even though some burst cells are clipped (i.e., the activity is over by saccade end), most burst cells (probably as many as 70%) are only partially clipped (Munoz and Wurtz 1995a; Waitzman et al. 1991). The presence of unclipped activity is not a problem because, as stated above, in our model the collicular output does not encode dynamic motor error, which has to be zero at the end of the saccade. In fact, later on it will become clear that the presence of unclipped activity is an indispensable feature of the model.

It is important to point out that in our model the spatial characteristics of the first three inputs described in the preceding text (which essentially determine the activity of burst neurons, because the fourth input is very weak) are not under feedback control and, except for noise-related variations, do *not* change during a saccade. Consequently, in our scheme the activity in the burst layer maintains its spatial distribution throughout the saccade, and it is modulated only in intensity by feedback signals. Accordingly, only the magnitude of the output of the burst cells changes during the saccade, and thus in our model, the purposeful curvature of saccades (which reflects a feedback-driven directional control) cannot be due to this collicular output.

OUTPUTS OF BURST NEURONS. In our model, the burst cells excite the contralateral MLBNs (both EBNs and IBNs) (see Chimoto et al. 1996), with weights that are a function of the position of the cell on the collicular map (caudal sites have stronger projections than rostral sites), as originally proposed by Edwards and Henkel (1978). Cells in the lateral and medial part of the SC project preferentially to vertical MLBNs, whereas cells along the central meridian project preferentially to horizontal MLBNs (see Grantyn et al. 1997). However, the input provided by the SC to the MLBNs is a directional drive signal and no spatial-to-temporal transformation (see *Role of the SC in current models of the saccadic system*) is performed. Thus the input provided to the MLBNs by the SC can be the same even if two different collicular loci are activated at different levels (e.g., a 20° locus weakly activated compared with a 10° locus strongly activated). In contrast, by definition the output of an STT always must be different when different loci are activated regardless of the level of activity.

Thus in our scheme, the SC burst cells provide a signal that only drives the eyes approximately in the right direction. The direction of the movement is determined by the lateromedial location of the collicular site activated, whereas its speed depends on (but is not strictly encoded by) the level of activation of the burst neurons and the rostrocaudal location of the active site. This last aspect is in agreement with results from single unit recordings (Berthoz et al. 1986), collicular lesions (Aizawa and Wurtz 1998; Hikosaka and Wurtz 1985, 1986; Lee et al. 1988; Quaia et al. 1998a), and electrical stimulation of the SC (Paré et al. 1994; Stanford et al. 1996).

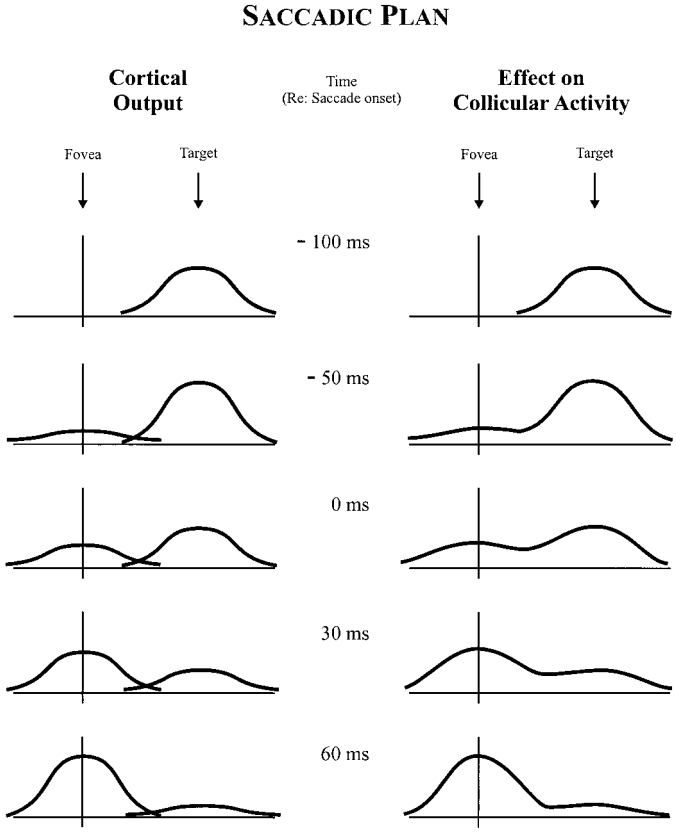


FIG. 4. Schematic outlining the effect of a cortical predictive remapping of the saccadic plan input. If the cortical activity is remapped from the locus corresponding to the position of the target to the foveal zone, starting ~80 ms before saccade onset, the effect on collicular buildup neurons is a pattern of activation that resembles a spread of activity toward the rostral pole of the SC. Note that this figure does not account for the other inputs to collicular neurons, which were shown in Fig. 3.

In the model, the burst neurons also provide a topographically organized input to the nucleus reticularis tegmenti pontis (NRTP) and to the pontine nuclei (see Thielert and Thier 1993), which in turn project heavily to the cerebellum. As we will describe later, we propose that the function of these projections is to relay to the cerebellum information regarding the target location, retaining the spatial code and thus avoiding the need for an STT. Finally, the burst neurons inhibit the fixation neurons, thus helping to keep them off during the saccade.

INPUTS TO BUILDUP NEURONS. In our model, the second cortical input to the SC, which we call the *saccadic plan* input and briefly introduced in the previous section, is the source of the early activation and of the rostral spread of activity in buildup neurons. We call this signal the saccadic plan because it indicates the presence and location of an area of interest in the visual scene. Any such location is a potential target for a saccade, but a saccade to it is not necessarily generated. In our model, this signal starts exciting buildup neurons soon after the target has been designated and is characterized by a perisaccadic spread (i.e., a particular input fiber is activated later for larger saccades in one direction). Recordings from lateral intraparietal cortex (LIP) neurons projecting to the SC (Paré and Wurtz 1997) revealed the presence of a signal that could be

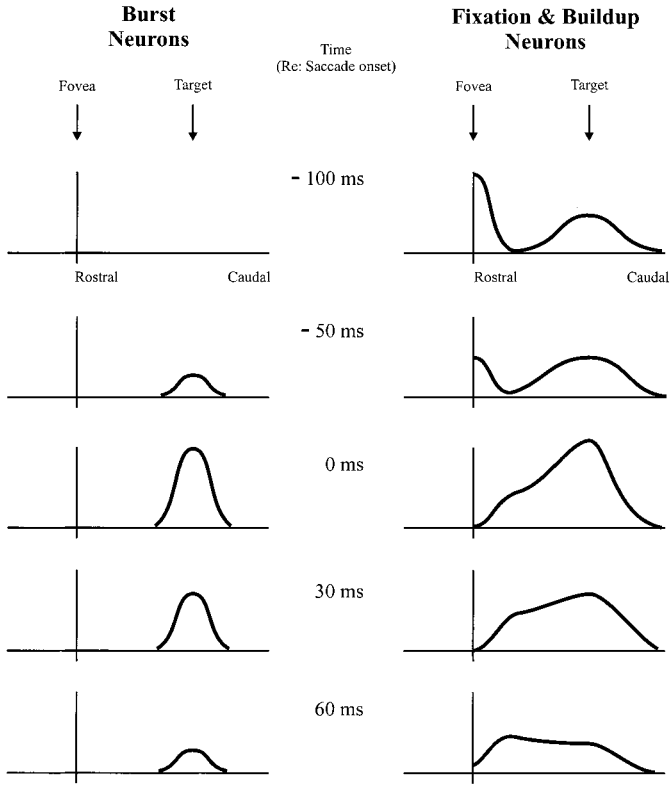


FIG. 5. Pattern of activation of collicular neurons (schematic). Spatial distribution of activity in collicular burst neurons (*left*) is shown at different times before and during a horizontal saccade (saccade onset = 0 ms, duration = 60 ms). Only the activity in the row of neurons corresponding to horizontal saccades/targets is shown. *Right*: activity of fixation (around the vertical line indicating the rostral pole of the SC) and buildup neurons during the same period is illustrated.

compatible with these requirements. Actually, because of the breadth of the cortical movement fields, there is no need for the input to spread: all that is needed is a step-like remapping of the target from its initial eccentric position to a foveal position (Fig. 4, *left*).

In Fig. 4 (*right*), we show the effect of such a remapping of the saccadic plan input on collicular buildup neurons (this must not be confused with the actual pattern of activation of buildup neurons, shown in Fig. 5, which also is determined by other inputs). One characteristic of this spread/remapping is that to start before saccade onset (see BACKGROUND), it must be predictive and cannot depend on feedback information regarding an ongoing movement. The presence of such a signal is supported by the findings of predictive target remapping in LIP by Goldberg and colleagues (Duhamel et al. 1992; Goldberg and Bruce 1990; Goldberg et al. 1990; Quaia et al. 1998b). The onset of such remapping (80 ms before saccade onset) is consistent with the timing of the spread observed in the SC. It also should be noted that such remapping has been reported in FEFs only in visual neurons (Umeno and Goldberg 1997) and not in movement neurons, which in our model carry the saccadic command input to the SC (and thus cannot show remapping).

Besides the saccadic plan input, in our model the buildup neurons receive three other inputs, described in a previous section: the saccadic command, the fixation command, and the feedback inhibition (Fig. 3).

ACTIVITY OF THE BUILDUP NEURONS. In Fig. 5 we show how, in our scheme, the spatial distribution of neuronal activity across the SC changes before and during the movement. Here the case of a horizontal saccade having a duration of ~60 ms is illustrated. As already pointed out, only the burst neurons around the optimal vector are activated during a saccade (Fig. 5, *left*). The activation starts just before, and it peaks around, saccade onset (see also Fig. 3*D*); no change in the spatial distribution of the activity occurs. The fixation neurons (Fig. 5, *right*, rostral neurons) are inactive during the saccade and are otherwise firing tonically (see also Fig. 3*B*). Buildup neurons are instead characterized by the superposition of the burst and of the input described in Fig. 4, which produces a pattern of activation that resembles a rostrally directed spread of activity. It is important to note that because, in our model, feedback information controls the strength of the burst input but not the spread (or remapping) of activity toward the rostral pole, the buildup neurons cannot contribute to the goal-directed curvature of saccades (i.e., even if there is a change in spatial distribution, it does not depend on the trajectory of the eyes and thus is not part of a trajectory control mechanism).

OUTPUTS OF THE BUILDUP NEURONS. In our scheme, the buildup neurons project to the same recipients as the burst neurons. Thus they provide an excitatory input to MLBNs (directional drive), an inhibitory input to the collicular fixation neurons, and topographically organized inputs to NRTP and pontine nuclei. Thus we propose that, as far as movement execution is concerned, buildup neurons are not functionally different from burst neurons.

FIXATION NEURONS. In our model, the fixation neurons receive five inputs: an excitatory visual input from targets on the fovea, an excitatory input that is related to the desire to keep the eyes steady (active fixation), an excitatory input from the caudal fastigial nucleus, an inhibitory input from the ipsilateral caudal SC (burst and buildup neurons), and an excitatory input from the contralateral rostral pole of the SC. Several investigators have provided experimental evidence that supports this scheme (e.g., May et al. 1990; Munoz and Istvan 1998; Munoz and Wurtz 1993a).

The role of the fixation neurons is to provide a *go* signal for the saccade. They carry out this role by turning off just before the beginning of each saccade, thus reducing the excitatory input of the OPNs and allowing the MLBNs to turn on and start the saccade. In our model, the role of this gate circuitry is twofold: first, it stabilizes the circuit during periods of fixation, avoiding the onset of oscillations (Robinson 1975; Van Gisbergen et al. 1981). Second, the presence of a gating mechanism allows the collicular signal to rise to its maximum just before saccade onset, thus providing the MLBNs with the strongest possible drive, which in turn results in the maximum acceleration of the eyes (Scudder 1988).

Furthermore in our scheme, the fixation neurons are reactivated after the end of the saccade to help maintain fixation. We have shown elsewhere (Lefèvre et al. 1998) that the diminished activation of the burst neurons and the increased overall activation of the FOR at the end of the saccade is sufficient to induce a timely reactivation of the fixation neurons.

In our model, the fixation neurons project to both the OPNs and to the collicular burst/buildup neurons; both these connections are supported by experimental evidence (Büttner-Ennever

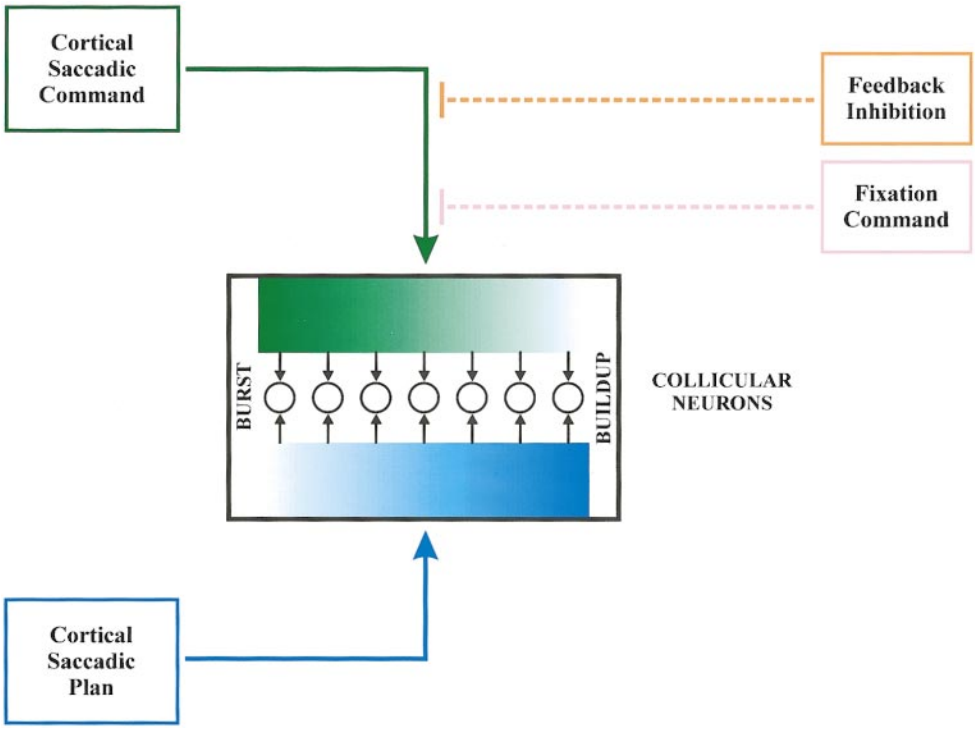


FIG. 6. Classification of collicular neurons. In our scheme, burst and buildup neurons are extremes of a continuum of neurons. The stronger the connections with the saccadic command input (e.g., from cortical frontal eye fields), the stronger the burst; the stronger the connections with the saccadic plan input (e.g., from parietal cortex), the stronger the early activation and the spread of activity. For this scheme to work, the inhibitory inputs should act at the dendritic level to shunt the saccadic command input.

and Horn 1994; Gandhi and Keller 1997; Munoz and Istvan 1998; Paré and Guitton 1994). It must be noted that because the activity of the fixation neurons is determined by the activity of burst and buildup neurons, the onset time of the saccade is not under direct voluntary control (even though it is possible to voluntarily prevent the execution of a saccade).

DIFFERENCE BETWEEN BURST AND BUILDUP NEURONS. Physiological recordings indicate that the early activity observed in collicular neurons can vary, in the same cell, from a significant level to essentially zero activity, depending on the experimental conditions, such as likelihood of appearance of a target in the response field or initial eye position (Basso and Wurtz 1997; Dorris et al. 1997; Paré and Munoz 1996). Thus if, as we propose here, this same low level component confers to the buildup neurons their open-movement field characteristics, the same neuron could be classified as burst or buildup depending on the conditions under which it is observed.

To account for these observations, in our model burst and buildup neurons share the same inputs and constitute a single class of neurons. Neurons that receive a strong cortical saccadic command $\Delta E$ show a strong burst of activity, whereas neurons that receive a weaker $\Delta E$ input produce a smaller burst or no burst at all. Similarly the stronger the cortical saccadic plan input, the larger the buildup (Fig. 6). The characteristics of individual neurons, which form a continuum, are then just the result of the different relative contribution of the four inputs shown in Fig. 6.

INHIBITORY CONNECTIONS IN OUR MODEL OF THE SC. It must be noted (see Fig. 6) that in our scheme the inhibition from the fixation neurons acts on the saccadic command input at the dendritic level, shunting that signal, and not (or only weakly) on the soma of the burst/buildup neurons. This arrangement allows our buildup neurons to be active long before the saccade (when the fixation neurons are active) and to have a burst closely synchronized with the saccade. Such connections have not been shown experimentally, but under these conditions, it should be possible to find a frequency of stimulation in the fixation zone that would prevent the occurrence of the burst but not the early activity in the buildup cells. Lower frequencies would not be sufficient to prevent the occurrence of the burst, and higher frequencies also might inhibit the early activity if a fraction of the inhibition acts at the level of the soma. In fact, such a finding has been reported recently (Munoz and Istvan 1998).

The same consideration holds for the intracollicular excitation-inhibition that narrows the movement field of the collicular burst; in our scheme these connections act at the level of the burst input, otherwise it would not be possible to have a narrow movement field for the burst and a large movement field for the buildup. Finally for the same reasons, in our scheme the feedback inhibition also acts at the dendritic level (Fig. 6).

An alternative scheme (Grossberg et al. 1997; Optican 1994) posits that only the buildup neurons receive the saccadic plan input and that the burst neurons generate the burst from the buildup activity using a winner-take-all network. The burst then is imposed on the buildup neurons by the burst neurons, and there is resonant feedback between the two layers. In these schemes, inhibition from the fixation neurons is provided only to the burst neurons and can be applied directly to the soma. Currently no experimental evidence conclusively differentiates between these two schemes. Nonetheless both schemes are compatible with the rest of our model and in particular with the function exerted by the cerebellum in controlling saccades.

### *Cerebellum*

INPUTS. To keep track of how far the eyes have turned since the beginning of the saccade, the cerebellum needs accurate information about eye movements. In our model, the cerebellum obtains this information by monitoring the output of the

MLBNs (i.e., velocity efference copy). In support of this hypothesis, bilateral projections from regions containing MLBNs to the cerebellum have been reported (Noda et al. 1990; Thielert and Thier 1993; Yamada and Noda 1987), and MLBN-like activity has been recorded in mossy fibers (Kase et al. 1980; Ohtsuka and Noda 1992). However, in one study no direct projections from the MLBNs to the cerebellum have been reported (Strassman et al. 1986a,b), thus an alternative would be to extract the velocity signal from the burst-tonic signal provided (presumably by the nucleus prepositus hypoglossi) to the cerebellum, which also has been documented (Kase et al. 1980).

The signals just described enable the cerebellum to act as a displacement integrator (DI, Fig. 1); however, to generate the choke signal at the appropriate time, the cerebellum also needs to know the desired amplitude of the movement. In our scheme, this information is provided by the NRTP [where the desired displacement is spatially coded (Crandall and Keller 1985)], which we propose sends topographically organized projections to the cerebellum. In support of this hypothesis, recordings in mossy fibers (Kase et al. 1980; Ohtsuka and Noda 1992) revealed the presence of signals similar to those reported by Crandall and Keller in NRTP. Alternatively such signals could be provided by the pontine nuclei [in particular the dorsomedial pontine nuclei (DMPN), which receive strong projections from the FEF and project heavily to the cerebellum (Noda et al. 1990)].

As we will describe in detail below, we propose that the cerebellum uses these two signals (eye velocity and desired displacement) to keep track of the residual motor error, enabling it to issue the choke signal at the appropriate time.

ACTIVITY. The discharge characteristics of fastigial neurons have played a significant role in guiding our modeling effort. In our model, each fastigial neuron produces an early burst for saccades in one direction (having a contralateral horizontal component) and a late burst for saccades in the opposite direction. The early burst occurring in the contralateral FOR provides, through crossing connections from the FOR to the MLBNs, an additional directional drive. Thus the sum of the FOR and the collicular inputs to MLBNs determines the initial direction and speed of the saccade (Fig. 7). However, because of the relatively mild effects on initial acceleration of muscimol injections in the FOR (Robinson et al. 1993), we posit that, at the very beginning of the saccade, the cerebellar contribution to the overall directional drive is not very intense (~20–30% of the total drive). Accordingly, in our model the collicular pathway is stronger than the cerebellar pathway.

In contrast to the early burst observed for saccades in the preferred direction, a late burst is produced in correspondence with saccades in the opposite direction. This burst occurs later and later for larger and larger saccades (see Fuchs et al. 1993; Helmchen et al. 1994; Ohtsuka and Noda 1990, 1991); it had been proposed that such a signal contributes to the deceleration of the eyes at the end of the movement (Fuchs et al. 1993; Noda 1991; Robinson 1995). In our model, this signal exerts a more fundamental role: we propose that this late burst is generated by the cerebellum to actually end the saccade when the eyes are approaching the target, similar to the proposal by Sparks and Barton (1993); this function is performed in our model by activating the IBNs contralateral to the movement (Fig. 7).

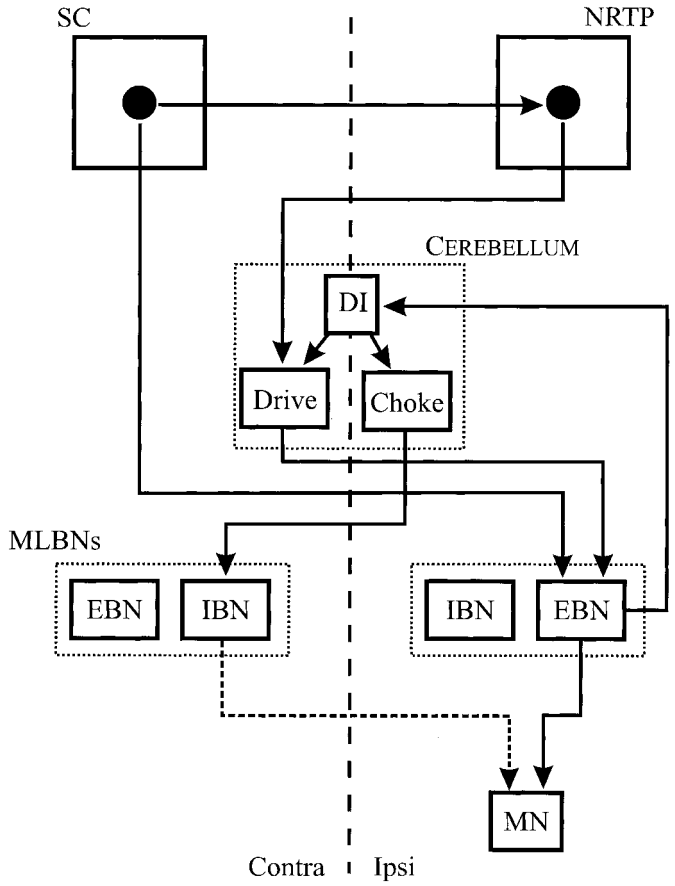


FIG. 7. Contributions to the saccadic drive. At the beginning of the movement, on the side contralateral to the movement (e.g., left for a rightward saccade), both the SC and the cerebellum excite the EBNs that contact the MNs (both ipsilateral to the movement) of the agonist muscle. During the saccade, the cerebellum integrates (DI) the efference copy of the drive signal, and when the eyes approach the target, the ipsilateral fastigial neurons produce a choke signal through the contralateral IBNs that inhibits the MNs of the agonist muscle.

An important novel aspect of our scheme is that the early and late bursts are not two distinct bursts, but a single burst that spreads from the contralateral to the ipsilateral FOR during horizontal saccades and within each FOR during vertical movements. The major consequence of this spreading mechanism is that if the speed of the spread (which in our model is controlled by the vermis) is an appropriate function of the velocity of the movement, the FOR acts as a spatial displacement integrator that keeps track of the residual motor error. The integration of the velocity signal is carried out by the cerebellum in the spatial as opposed to temporal, domain. To perform a spatial integration of the velocity signal, some sort of topographic organization has to exist (Optican 1995); accordingly in our model, the FOR is organized topographically. Under this assumption, there are regions of the FOR that project preferentially to vertical bursters and others that project more heavily to horizontal bursters; furthermore the preferred directions of neurons spans the whole contralateral hemifield. In fact, recordings in the FOR appear to be compatible with this scheme (Fuchs et al. 1993; Ohtsuka and Noda 1991).

Furthermore thanks to this topographical organization of the

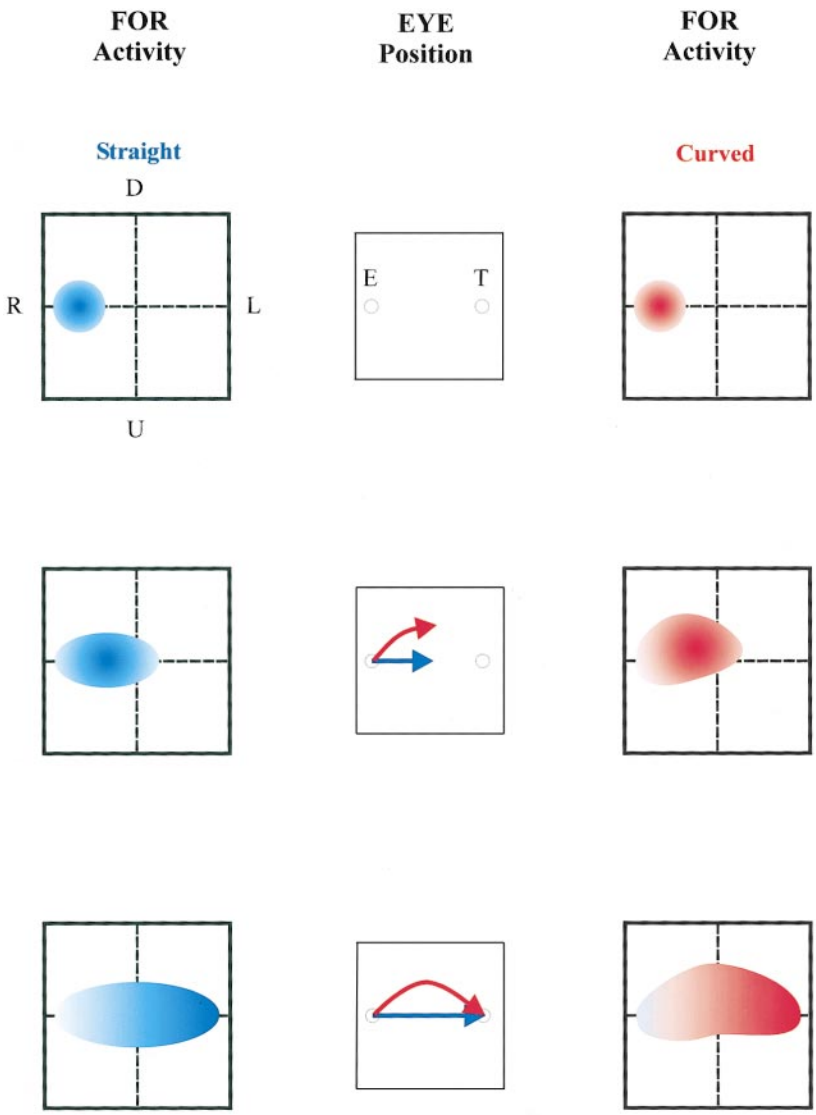


FIG. 8. Directional control by the fastigial oculomotor region (FOR). Here the 2 fastigial nuclei are represented as a single map. Neurons in the left half of the map drive the eyes toward the right (R), neurons in the top half of the map drive the eyes downward (D), etc. Pattern of activation of the fastigial nuclei during 2 saccades to the same target is represented schematically. *Middle column*: trajectory of the eyes is plotted for a straight (blue) and for a curved (red) saccade. E, initial eye position; T, target position. *Left*: FOR activity during the straight saccade. *Right*: FOR activity during the curved saccade. Initially the activity is the same in the 2 cases (as it depends only on the desired displacement of the eyes), but as the saccade progresses, the pattern of activation reflects the movement of the eyes so that when the eyes deviate upward the locus of activity spreads upward, increasing the downward component of the FOR output and reducing the upward component.

FOR, a directional control over the saccade automatically arises. When a horizontal saccade starts, the activated area is in the contralateral FOR at a location proportional to the amplitude of the movement (Fig. 8, *top*), and thus its contribution is collinear with the collicular drive. As the saccade progresses, the activity spreads across the map; if the eyes are moving straight toward the target (Fig. 8, *middle*, blue arrow), the FOR activity spreads into an area having the same amount of projections to the upward and downward MLBNs (Fig. 8, *left*). However, if the saccade is bending away from a straight trajectory, going for example upward (Fig. 8, *middle*, red arrow), the activity spreads toward an area that projects more heavily to the downward MLBNs (Fig. 8, *right*), compensating for the directional error. Thus in our model the FOR exerts a directional control over the saccade, redirecting the eyes toward the target, and even though the output of the collicular pathway is unidimensional, saccades can be curved purposefully. As the eyes approach the target, the activity reaches the other side of the FOR, and the choke signal is applied to the brain stem circuitry (Fig. 7). Because the collicular drive to the EBNs is choked off by the cerebellar input acting on the contralateral IBNs, and not on OPNs, the two components of a saccade can terminate at different times as occasionally observed (Bahill and Stark 1977; Becker and Jürgens 1990; King et al. 1986). Note that the spread of activity in the FOR is very different from the spread of activity in the buildup layer of the SC, which in our model begins before the saccade and is not under feedback control.

OUTPUTS. As indicated in the previous section, in our model the FOR projects to the contralateral MLBNs, stronger to the IBNs than to the EBNs. Experimental evidence supports this hypothesis (Gonzalo-Ruiz et al. 1988; Noda et al. 1990).

At the beginning of horizontal saccades, the FOR contralateral to the direction of the saccade produces a burst, exciting the MLBNs ipsilateral to the saccade and thus supplying an additional drive. In contrast, toward the end of the movement the FOR ipsilateral to the direction of the saccade bursts, thus exciting the MLBNs contralateral to the saccade. The activity induced in the contralateral EBNs is canceled out, at the level of the MNs, by the activity still present in the ipsilateral IBNs because of the collicular drive. At the same time the ipsilateral FOR also excites the contralateral IBNs, with stronger weights (as supported by anatomic studies, see preceding text); this late

activity in the contralateral IBNs cancels out, at the level of the MNs, the activity present in the ipsilateral EBNs because of the collicular pathway and of the contralateral FOR, thus stopping the saccade. In other words, the late excitation of the contralateral IBNs is used to choke off the activity still present in the ipsilateral EBNs. We call this a choke and not a brake because the saccade is terminated by removing the pulse component of the drive to the agonist muscle, and not by activating the antagonist. Thus no cocontraction of the agonist-antagonist pair of muscles is produced. The same line of reasoning can be applied to vertical and oblique saccades; however, in those cases, the concepts of ipsilateral and contralateral are lost, and it is useful to visualize the two FORs as a single map.

It now becomes clear why we said earlier that the presence of unclipped activity in the SC is an indispensable feature in our model: if the collicular drive was over at, or before, the end of the saccade there would be nothing left for the cerebellar pathway to choke off. Furthermore the lack of activity in the caudal SC would cause the reactivation of the collicular fixation neurons, which in turn would reactivate the OPNs, opening the gate and making the positive drive produced by the contralateral FOR useless. When this happens, the accuracy of the saccade cannot be controlled by the cerebellum. Thus the collicular pathway always must supply a drive that would produce hypermetric saccades so that the cerebellum can turn them into normometric movements by choking off the collicular drive at the appropriate time. After the saccade has been stopped in this way, the OPNs reactivate, stabilizing the saccadic circuit. Nevertheless in our model, neither the removal of excitatory input to the ipsilateral EBNs nor the reactivation of the OPNs is necessary to stop the movement.

### *Action of the vermis*

As we already pointed out, in our scheme the desired displacement signal is delivered to the cerebellum by connections from the NRTP, which is characterized by a retinotopic organization (i.e., cells have retinotopic response fields) (Crandall and Keller 1985). So, the earliest burst on the FOR is imposed by topographic inputs from NRTP (or from DMPN). However, in our scheme, the connections from NRTP (or from DMPN) to the FOR need to be bilateral; this aspect, which is supported by experimental evidence (Noda et al. 1990), is extremely important. In fact during small saccades, there is no time for the ipsilateral burst to be generated by making the contralateral burst spread across the FOR under the effect of velocity feedback. Thus in these conditions, the ipsilateral FOR, which in our model provides the choke, should start discharging before the onset of the saccade (this is in agreement with experimental findings) (see Fuchs et al. 1993, their Fig. 1).

Another reason for having bilateral projections from the NRTP to the FOR is related to the fact that the vermis, which in our model controls the spread, can only disinhibit the FOR neurons. So it is conceivable that the burst of the FOR neurons is determined by a widespread excitatory input from the NRTP, controlled by a selective inhibition of FOR cells by the vermis (Fig. 9). At the beginning of the movement, the activity is localized in the contralateral FOR (Fig. 9, solid annulus), whereas by the end of the movement the activity has spread to the other FOR (Fig. 9, dashed annulus). The only relationship to be learned to produce accurate saccades is the relationship

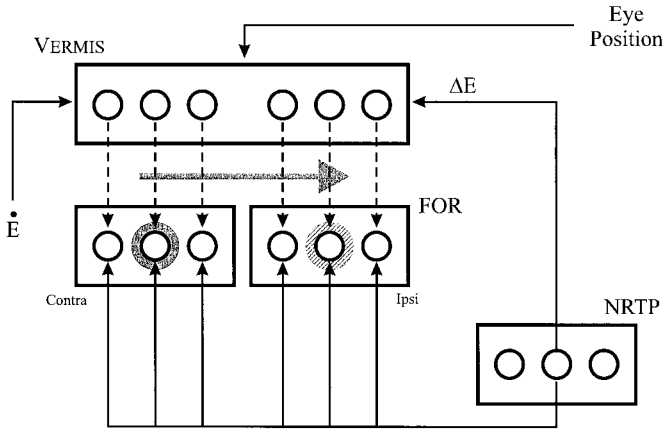


FIG. 9. Hypothetical mechanism for producing a spread of activity in the FOR. Nucleus reticularis tegmenti pontis (NRTP) could provide a widely distributed input to the FOR, and the vermis then could sequentially disinhibit the FOR. Inhibition is released initially in the contralateral FOR (solid annulus), whereas at the end of the movement the other FOR gets activated (dashed annulus). Location of the solid annulus is a function of the desired displacement ΔE and eye position, whereas the speed of the spread is a function of velocity feedback ($\dot{E}$) and eye position.

between the velocity of the movement [which is a function of the output of the MLBNs and the orbital position of the eyes (Collins 1975)] and the speed and direction of the spread. It also should be noted that because in our model the SC encodes (spatially) the location of the target and not the desired displacement, eye position information also could be used by the cerebellum to implement the visuomotor transformation needed to convert a target location from retinotopic coordinates into the displacement of the eyes required to foveate it (Klier and Crawford 1998).

### *Simulations*

As pointed out in the INTRODUCTION, in another paper (Lefèvre et al. 1998) we presented a distributed implementation of the model described here. Now we briefly indicate how sensitive the model is to changes in its various parameters, and we present some additional simulations, showing how it can account for the effects of collicular and cerebellar lesions. The simulations reported here were performed using MATLAB/SIMULINK (The Mathworks, Natick, MA) running on a Challenge-L computer (Silicon Graphics, Mountain View, CA). All the details of the implementation are presented elsewhere (Lefèvre et al. 1998); unless the contrary is stated explicitly, the simulations in both papers have been obtained using the same values for the various parameters of the model.

### *Sensitivity of the model to changes in its parameters*

As expected, our model is very sensitive to the relationship between the MLBNs' activity, which determines the speed of the eyes, and the speed of the spread of activity in the FOR. If this relationship is not precise, saccades will not be accurate. A second important factor is the mapping from NRTP/DMPN to the cerebellum. This mapping determines the area of the FOR that bursts at the beginning of the movement; the location of this area is also very important to ensure the accuracy of the movement.

On the other hand, the model's saccade accuracy turned out

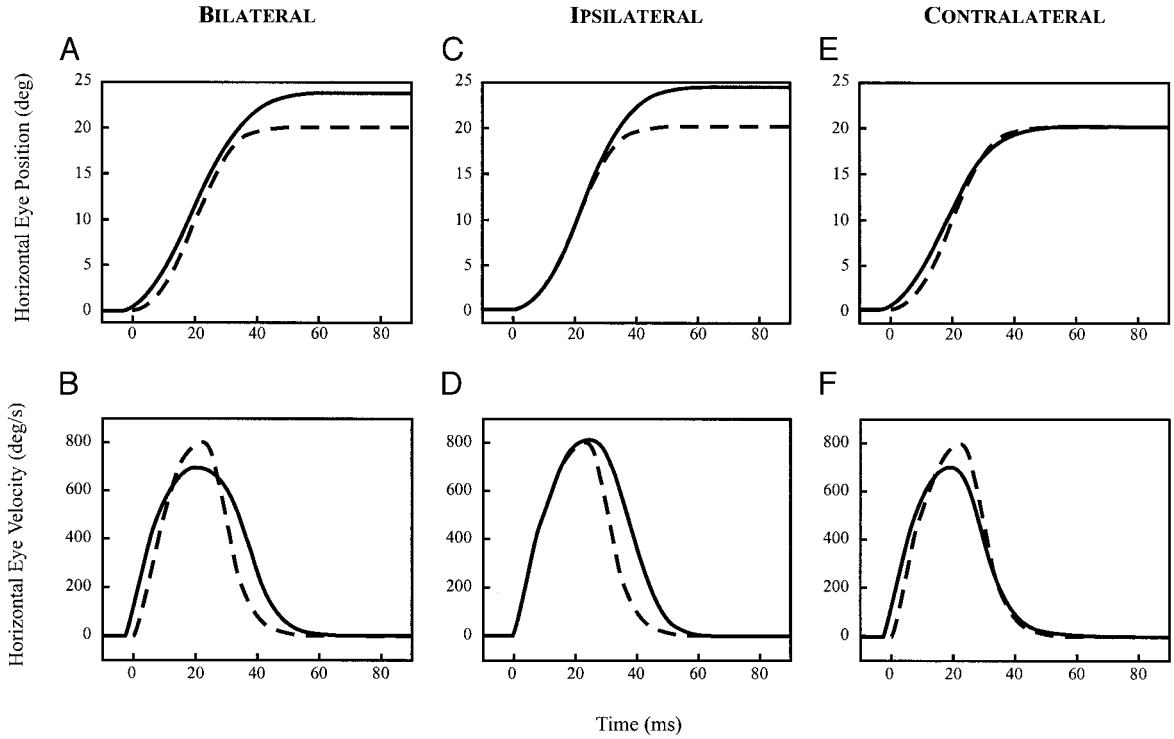


FIG. 10. Simulation of the effects of FOR lesions on a 20° rightward saccade. *A* and *B*: eye position and velocity before (- - -) and after (—) a bilateral FOR lesion. In agreement with what has been observed experimentally, after the lesion saccades are slower and larger than normal. *C* and *D*: effects of a lesion of the right FOR. Saccades ipsilateral to the lesion are bigger and faster than normal, as reported experimentally. *E* and *F*: effects of a lesion of the left FOR. Only the decrease in speed, but not the hypometria, observed experimentally is replicated by our model (see text for comments). All results obtained by reducing the output of the FOR by 60% (see text).

to be fairly insensitive to changes in the speed of the movement and thus to the weight of the connections between the SC and the MLBNs; furthermore altering the feedback inhibition signal to the SC has little effect on the metric of saccades. Similarly the weight of the connections between the FOR and the MLBNs is not very important as long as the input to the IBNs (the choke) is strong enough to overcome the collicular input to the EBNs (otherwise the movement would not stop).

Finally the OPNs deserve a special note: we have noticed that even though under normal circumstances they play essentially no role in determining the characteristics of saccades, they can become very important when abnormal conditions are considered. For example, they can have important effects after lesions or during electrical stimulation. Thus we suggest that it would be interesting to study their behavior under these conditions or, for example, to study how a lesion of the OPNs effects electrically evoked saccades.

### *Effects of cerebellar lesions*

Lesions of the oculomotor cerebellum have a large impact on the characteristics of saccades. Because in our implementation we have focused on the role of the FOR and we have not directly addressed the issue of how the cerebellar cortex carries out its function, we will describe here simulations of lesions of the FOR. All the simulations we show refer to the effects of FOR lesions on a saccade to a target located 20° to the right of the center. In all figures the prelesion (control) saccades are indicated with a dashed line, whereas the postlesion saccades are indicated with a solid line.

It has been shown (Robinson et al. 1993) that when the fastigial nuclei are lesioned bilaterally saccades become hypermetric regardless of their direction. Furthermore their speed is lower than expected for saccades of their size and even lower than the speed of normal (i.e., prelesion) saccades to the same target. To simulate these conditions with our model, we have assumed that the effect of a lesion of the FOR is to attenuate its output (because some of the FOR possibly is spared). For example, when we impose an attenuation of 60%, we obtain saccades that are hypermetric (Fig. 10*A*) and slower (Fig. 10*B*) than normal just as reported in the literature. Effects on latency by actual lesions seem to be very inconsistent; in our simulations, we observe a very small latency decrease due to a decrease in the excitatory drive provided by the FOR to the collicular fixation neurons.

With unilateral lesions of the FOR, it is possible to evoke a much larger range of effects (Ohtsuka et al. 1994; Robinson et al. 1993). First of all, ipsilateral saccades become hypermetric, while their velocity (at least for 20° saccades) slightly increases. Our simulations (performed by attenuating by 60% the output of the right FOR for a 20° rightward movement) are in agreement with such findings (Fig. 10, *C* and *D*). Conversely after contralateral lesions, saccades become hypometric and slower. However, when we simulate this condition with our model (using the same attenuation as before), we can reproduce the slowing down,but not the hypometria (Fig. 10, *E* and *F*). This is due to the fact that we are assuming that altering the activity in the contralateral FOR (the one that is active at the beginning of the movement) does not affect in any way

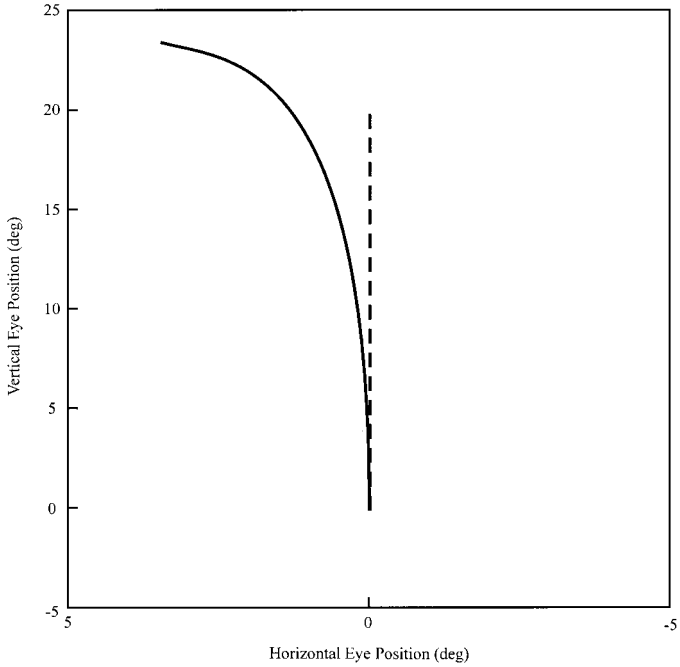


FIG. 11. Simulation of the effects of a lesion of the left FOR on the trajectory of a 20° upward saccade. Late deviation from the normal trajectory and the hypermetria are in agreement with experimental results.

the functioning of the spatial integrator. Thus even though the saccade starts slower, the choking signal supplied by the ipsilateral FOR is delivered later and the eyes land on target. However, it should be noted that the FOR projects to the NRTP (Noda et al. 1990), which in turn projects to the vermis, possibly disrupting the mechanism underlying the spatial integration of the velocity signal and inducing an early activation of the choke (in our simulations, we only attenuated the output of the cells). To clarify this issue, a better understanding of the NRTP-vermis interaction is needed.

Unilateral lesions of the FOR also affect vertical saccades, which become slightly hypermetric and bend toward the side of the injection (Robinson et al. 1993). Because of a large edge effect (due to the need to activate both collicular maps), the current implementation of our model is not very well suited to simulate vertical saccades. However, because of our model's structure, the effects of a lesion of the left FOR on an upward saccade are equivalent to the effects of a lesion of the upper half of the FOR (see Fig. 8) on a rightward saccade. This allows a vertical saccade to be simulated by interchanging of horizontal and vertical in our model. The results of such a simulation (Fig. 11) are very similar to what has been reported in the literature (see Robinson et al. 1993, their Fig. 2). In particular, note that the saccade starts in the correct direction and then starts bending away from the target. Furthermore the saccade is also slower (not shown), as reported by Robinson and colleagues (1993).

Another study of unilateral injections of muscimol in the fastigial nuclei of the head-free cat (Goffart and Pelisson 1994) showed that ipsilateral saccadic deficits were compatible with a remapping of the target rather than with a generalized hypermetria. In contrast, contralateral saccades were hypometric, as expected. Our model, in its present form, does not predict such results; this could be due to the disruption of some additional mechanism (perhaps related to the removal of the tonic level of activity that is normally present in fastigial neurons, which we have not modeled here). Nonetheless it should be noted that the effect of unilateral FOR lesions on vertical saccades, which is reproduced very well by our model, would be very difficult to explain with a theory that posits a role for the FOR in specifying the target.

Finally we previously pointed out that when the FOR is lesioned the variability of saccades is considerably increased, both in amplitude and in direction. As pointed out in the preceding text, this increased variability is incompatible with classic models of cerebellar contribution that use only long-term adapted control signals. On the other hand, the increased variability is compatible with our model, where the cerebellum is the structure that accounts for both the accuracy and consistency of saccades. Because noise sources have not been included in this implementation of our model, we did not use simulations to demonstrate this property. However, because without a cerebellum our model of the saccadic system would simply be a feed-forward controller, the results are obvious.

### *Effects of collicular lesions*

Even though the SC is not necessary to produce saccadic eye movements (Schiller et al. 1980), it is well known that its partial chemical inactivation causes, at least in the acute phase of the lesion, changes in all saccadic parameters. Typical effects of reversible partial deactivation of the SC are increased latency, decreased peak velocity, and dysmetria of the movements (Aizawa and Wurtz 1998; Hikosaka and Wurtz 1985, 1986; Lee et al. 1988; Quaia et al. 1998a). Furthermore it has been reported recently that the trajectory (Aizawa and Wurtz 1998) and the initial velocity and direction (Quaia et al. 1998a) also can be affected systematically.

We have simulated a collicular lesion by attenuating the output of a region of the SC. We have reduced the activity of one cell by 70%, of its 8 neighbors by 60%, and of the successive 12 neighbors by 50% (both buildup and burst neurons were affected in the same way) with the central cell corresponding to a 15° saccade at 45° of elevation. Then we have looked at the effect of this lesion on a 10° and a 20° saccade, both at 45° of elevation. In another paper (Quaia et al. 1998a), we suggested that the effects of SC lesions on the initial direction of saccades can be accounted for if it is assumed that the lesion always causes a change in the horizontal drive larger than what would be expected given the location of the lesion. To include this assumption in our simulations, we also have reduced the drive of the SC to the horizontal MLBNs by 30%.

When, under the above mentioned conditions, a saccade to a 20° target is simulated (Fig. 12, *A* and *B*), the eyes deviate upward and then curve back toward the target. However, the compensation is only partial so that the saccade falls short of the target. The speed (both initial and peak) of the movement is considerably lower than in the control situation, even though the amplitude of the movement is not much different. When a 10° saccade is simulated (Fig. 12, *C* and *D*), a similar pattern of curvature is observed, and again both peak and initial speed are considerably affected. However, in this case the eyes fall considerably short of the target.

All these characteristics are in agreement (at least qualita-

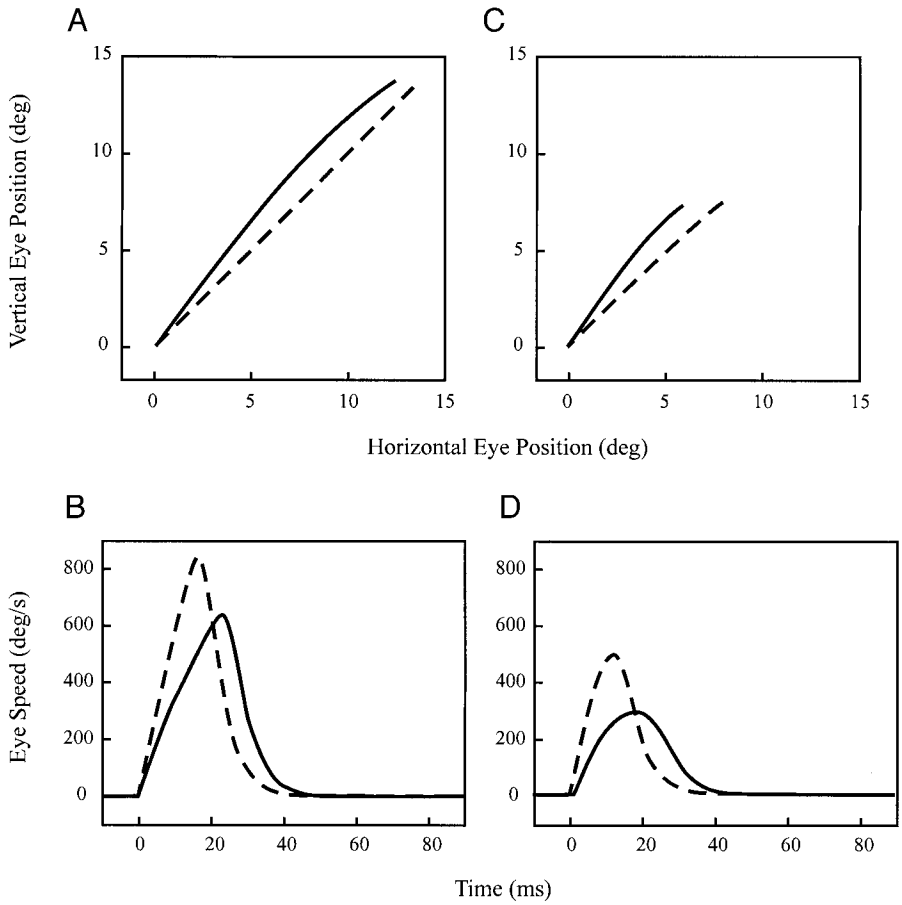


FIG. 12. Simulation of the effects of a collicular lesion, centered at 15° of amplitude and 45° of elevation, on trajectory and eye velocity. *A* and *B*: effect on a 20° oblique saccade. *C* and *D*: effect on a 10° oblique saccade. In both cases, the results (decreased initial and peak velocity, increased curvature, dysmetria) are in qualitative agreement with experimental data. See text for details about the parameters used to simulate the lesion.

tively) with the results presented by Aizawa and Wurtz (1998); however, our simulations clearly fail to show the large change in latency which is a trademark of collicular lesions. This failure is due principally to the fact that the cortical fixation input provided to the collicular fixation neurons (see preceding text) is, in our current implementation, removed abruptly and not gradually. A more gradual removal of this input would make the timing of the saccade onset more sensitive to the balance between the activity of the burst/buildup neurons and that of the fixation neurons, thus allowing a much larger spread of latencies.

## DISCUSSION

We have presented a model in which saccades are generated by the cooperation of two pathways, both influenced by feedback information. In this sense, our model departs from the Robinsonian scheme that has dominated saccadic modeling for the last 20 years, where the saccadic drive was generated by a single feedback loop. The main concepts that characterize our scheme are as follows: *1*) the saccade ends not because the MNs run out of drive from the EBNs but because that drive is actively choked off; *2*) only one part of the drive can be controlled in direction; *3*) the cerebellar contribution depends on feedback information, and it is tailored carefully for each movement; *4*) no classical spatial-to-temporal transformation (which would produce a temporally coded dynamic motor error) is performed; and *5*) the displacement integrator is implemented in the spatial domain in the cerebellum.

Even though we have left out many other structures, both cortical and subcortical, which certainly are involved in controlling saccadic eye movement, we think that the areas we have modeled are sufficient to reproduce at least the simplest saccadic behavior. In the next few sections, we will compare the model presented here with other models recently proposed; because in previous sections we already have described at length other models of the collicular involvement in saccadic control, we will now focus on the role of the cerebellum. Finally we will indicate some experiments that could be used to test our scheme and to shed further light on the saccadic system.

### *Types of cerebellar models*

Models of cerebellar function can be divided into two groups: those that are inspired by theories of learning in neural networks and those that are inspired by principles of control theory. Models of the first group stem from the early work by Marr (1969), Grossberg (1969), and Albus (1971); two of the most influential theories in this group are those of Houk, Barto, and colleagues (Barto et al. 1998; Berthier et al. 1993; Houk 1989; Houk et al. 1996) and of Grossberg and colleagues (Contreras-Vidal et al. 1997; Grossberg and Kuperstein 1989). On the other hand, cerebellar models inspired by principles of control theory propose that the cerebellum functions as a model of the controlled system and usually do not deal with adaptation and learning in the cerebellum. Some of these models (e.g., Jordan and Rumelhart 1992; Miall and Wolpert 1996; Miall et al. 1993) suggest that the cerebellum computes an

estimate of the effect that an outgoing motor signal will have on the controlled plant (direct, or forward, models), whereas others (e.g., Gomi and Kawato 1992; Kawato and Gomi 1992) estimate the motor outputs needed to generate a desired movement (inverse models).

Even though we took special care in reproducing the pattern of activation of FOR neurons, our model is clearly more inspired by principles of control theory than by theories of network learning. More precisely, we think that our scheme could be regarded as a forward model because the spatial integration process (which we hypothesize takes place in the cerebellum) is used to predict when the eyes are approaching the target, given the efference copy of the motor command. However, this signal is not fed back to the main controller, and thus it is different from classical control schemes. In this sense, it is closer to models like the one proposed by Grossberg and Kuperstein (1989), where the cerebellum is embedded in a side loop. One important advantage of learning theory models is that, especially in their most recent versions (Barto et al. 1998; Contreras-Vidal et al. 1997), they make testable predictions about the pattern of activities in Purkinje cells and interneurons (especially basket cells) in the cerebellar cortex. This is certainly a most desirable feature; unfortunately at this stage, our model lacks this. However, we think that our model has certain advantages (which will be outlined next) over existing models that justify our decision to take another approach and to propose a different model.

### *Comparison with other models of cerebellar involvement in saccadic control*

Recently some models that address the role of the cerebellum in the in-flight control of saccades have appeared; however, in only one of those models (Houk et al. 1992, 1996) is the cerebellum part of the feedback loop. The theory proposed by Houk and colleagues posits that the Purkinje cells in the cerebellar cortex are trained to recognize particular configurations of the proprioceptive inputs (carried by the mossy fibers), and when these patterns occur, they fire to stop an ongoing movement. Thus one of the roles that we propose here for the cerebellum is similar to the one proposed by Houk and colleagues (i.e., to terminate the saccade when the eyes approach the target), even though the mechanism used by the cerebellum to achieve this goal is very different in the two models. The pattern-recognition mechanism proposed by Houk and colleagues works well to control limb movement, where the delays in the system are shorter than the duration of the movement and proprioceptive feedback can be used to track (and even predict) the ongoing movement (Barto et al. 1998). However, we think there are some fundamental problems in extending their model to the control of saccadic eye movements. First of all in Houk's model, the movement is interrupted when a given final position, and not displacement, is attained. Thus the cerebellar cortex should work in head coordinates; however, it has been shown recently that saccadic adaptation, which almost certainly is controlled by the cerebellum (Goldberg et al. 1993; Optican and Robinson 1980), occurs in oculocentric coordinates (Frens and van Opstal 1994). Furthermore it is known that proprioceptive feedback plays no role in the in-flight control of saccades (Guthrie et al. 1983); one could argue that an internal estimate of the position of the eyes could be used instead, but no signal encoding the position of the eyes during saccades has been found in the mossy fibers [in our model (Fig. 9), the eye position signal is used only before the movement to determine the displacement of the eyes required to foveate the target]. One could overcome these problems by postulating the presence of a displacement integrator in the brain stem, whose output then could be fed to the cerebellum. However, to the best of our knowledge, such a signal has not been observed in the mossy fibers. Finally in its present form, the scheme proposed by Houk predicts a pattern of activity for the FOR that mirrors the activity in the SC, i.e., a burst of activity only for saccades in one direction, that is not compatible with what is reported in the cerebellar literature. For these reasons, we think that even though Houk's scheme is consistent with data on limb control, it is at odds with some crucial data regarding the saccadic system.

Another theory of cerebellar function is the one proposed by Grossberg and colleagues, both for saccadic (Grossberg and Kuperstein 1989) and limb control (Contreras-Vidal et al. 1997). One of the major differences between Houk's and Grossberg's models is that Grossberg proposes an extracerebellar loop to compute the residual motor error and to generate a desired velocity signal, which then is fed to the cerebellum. Thus the cerebellum is part of a side loop, and it works with velocity (as opposed to position) signals. We believe that our model has two main advantages over the one proposed by Grossberg and colleagues: first, it does not require a spatial-to-temporal transformation, which is part of the extracerebellar loop in Grossberg's model. Second, our model can account more readily for the large increase in variability observed after cerebellar lesions.

Recently, Dean (1995) proposed a model of the saccadic system that deals with the role played by the fastigial nuclei in on-line control of saccades, taking particular care in reproducing the pattern of FOR activation. There are several similarities between Dean's models and ours, including the connectivity between the FOR and the MLBNs. The role attributed by Dean to the FOR is to ensure saccadic accuracy; because of the timing of the FOR bursts, this is achieved by contributing to the acceleration of the eyes at the beginning of saccades and to their deceleration at the end of the movement. This role, which also has been proposed in other studies (Fuchs et al. 1993; Helmchen et al. 1994; Noda 1991; Robinson 1995; Sparks and Barton 1993), is similar to the one that we propose here, i.e., to provide a directional drive and to choke off the collicular output at the end of the movement. However, in our model, there are not two distinct bursts, one in the ipsilateral and the other in the contralateral FOR, but one single burst that spreads across the fastigial nuclei. Accordingly the FOR does not play a role only at the beginning and at the end of the saccade but also during the saccade, exerting a trajectory control. Furthermore the role played in our scheme by the late part of the burst is not just to slow the movement but to stop it when the eyes reach the target. Importantly in our scheme the cerebellum determines when the movement should end. In Dean's model, the cerebellum only makes a preprogrammed contribution to a saccade the end of which is controlled by the local feedback loop in the brain stem.

Another, fundamental, difference between Dean's model and ours is that in his scheme the brain stem circuit (extracerebellar pathway) consists of a feedback loop with a gain lower than one.

Consequently, Dean's model does not predict the increased variability in saccades observed after cerebellar lesions because it is the brain stem that guarantees the consistency of saccades. In contrast, in our model the loop is closed through the cerebellum, which is the structure that guarantees both the consistency and accuracy of saccades. Nonetheless, the two schemes would be in good agreement if the feedback loop proposed by Dean was affected by large and unpredictable changes in gain due to an unreliable feedback integrator. However, it is not clear whether the presence of an unreliable integrator would affect the accuracy of the saccades produced by Dean's scheme even when the cerebellum is working properly.

### *Predictions and experimental tests*

FOR NEURONS' ACTIVITY. We have conjectured that the FOR is organized topographically and that a spatial integration is performed in the vermis and represented on the fastigial map. We have shown that all the input/output connections needed are in place; furthermore this hypothesis makes some testable predictions about the pattern of activity in FOR neurons.

First, the burst should occur later and later for larger and larger ipsilateral saccades. Second, for contralateral saccades, the timing of the burst should depend on both the saccadic vector and the location of the cell on the fastigial map. Third, there should be cells that burst only for contralateral saccades larger than a given amplitude. Fourth, adaptive alteration of saccadic size should alter the time of occurrence of the ipsilateral burst, which should remain time-locked to the end of the movement. Finally, in analogy with what has been done in the SC (Keller and Edelman 1994), it would be very interesting to observe how the activity of fastigial neurons changes during interrupted saccades. Our model predicts that, under those conditions, the contralateral burst would be prolonged and the ipsilateral burst would be delayed to preserve its timing relative to the end of the movement.

Because of the short-duration of saccades, to test these predictions, FOR cells' activity should be observed during saccades of very different amplitude. Unfortunately the majority of the studies on FOR activity dealt principally with saccades smaller that 20°; nonetheless some evidence in support of the first (e.g., Ohtsuka and Noda 1991), third (Fuchs et al. 1993), and fourth (Scudder 1998) predictions is already available.

MLBN ACTIVITY. The discharge and connectivity of FOR neurons raise some expectations regarding the activity present in MLBNs during saccades. More specifically, the late burst present in the ipsilateral FOR should induce, toward the end of a saccade, a discharge in the contralateral EBNs and IBNs. In fact evidence for a late burst in at least some EBNs for contralateral movements has been reported (Keller 1974; Strassman et al. 1986a; Van Gisbergen et al. 1981). This burst is pretty weak, but that is in line with our prediction: we do not expect these neurons to discharge $>$200 spikes/s, and for no more than $\sim$20 ms [because of the reactivation of the OPNs (Everling et al. 1998; Fuchs et al. 1991; Paré and Guitton 1998)]. Thus only three or four spikes are expected.

This late discharge is exhibited also by a sizable subset of the IBNs (Scudder et al. 1988; Strassman et al. 1986b), and appears to be stronger, as predicted by our model. Unfortunately even though it is clear that IBNs are activated later for contralateral than for ipsilateral saccades, it has not been ascertained whether the burst for contralateral movements is time-locked with the end of the saccade (i.e., it lags saccade onset more and more for larger and larger saccades). Thus although further exploration is needed for a definitive answer, experimental recordings in these regions support (or at least are compatible with) our interpretation.

Another prediction of the model regards the effects of collicular electrical stimulation combined with complete FOR lesions: given the mechanism for the reactivation of the OPN that we have implemented in our model, the removal of the choke and the lack of a sizable decay of the collicular output (because of the sustained stimulation) should suppress the generation of staircases, and the eyes should keep turning as long as the electrical stimulation is applied (up to the oculomotor limit). A final prediction of our model is that lesions of the cerebellum should cause the disruption of the directional control of saccades. Note that this does not mean that after cerebellar lesions saccades should be straight but only that the curvature should not indicate a systematic redirection of the eyes toward the target. Unfortunately no systematic study on the curvature of saccades after cerebellar lesions has been carried out, but the data appears to be consistent with the lack of a directional control (for example, see Robinson et al. 1993, their Fig. 10; Vilis and Hore 1981, their Fig. 7).

### *Conclusions*

We have presented a model that, using two parallel pathways, preserves the advantages of many previous models (e.g., the lack of a spatial-to-temporal transformation between the SC and the brain stem, and thus a much simplified connectivity) without incurring many of their drawbacks.

In our model, the SC plays a lesser role than in many recent models; we propose that the SC helps determine the target and provides a directional drive that moves the eyes approximately in the right direction. It is up to the cerebellum to guarantee that the overall drive is appropriate to accurately foveate the target. Moreover, we propose that the burst and buildup neurons are, as far as movement execution is concerned, functionally indistinguishable [but it is possible that they exert different roles for other aspects of eye movements, like target selection (Optican 1994), learning of consistent maps for different modalities (Grossberg et al. 1997) and determination of reaction time (Dorris et al. 1997)].

One of the most important innovations of the model that we presented here is that in this scheme the cerebellum carries out the function that in previous models was ascribed to the displacement integrator and feedback summing junction, i.e., monitoring the dynamic motor error. Here the cerebellum plays a pivotal role in guaranteeing both the accuracy and the consistency of saccades. This role is accomplished by choking off the collicular drive at the appropriate time and by compensating for directional errors by providing an appropriate directional drive to the brain stem circuitry. Thus the signal provided by the cerebellum is subject not only to long-term adaptation, as often suggested, but is adjusted during each saccade to compensate for the instantaneous behavior of the rest of the system.

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