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A neural model of the saccade generator in the reticular formation

Gregory Gancarz[a,b], Stephen Grossberg[a,b,*]

[a]*Department of Cognitive and Neural Systems, Boston University, 677 Beacon Street, Boston, MA 02215, USA*
[b]*Center for Adaptive Systems, Boston University, 677 Beacon Street, Boston, MA 02215, USA*

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Abstract

A neural model is developed of the neural circuitry in the reticular formation that is used to generate saccadic eye movements. The model simulates the behavior of identified cell types — such as long-lead burst neurons, short-lead excitatory and inhibitory burst neurons, omnipause neurons, and tonic neurons — under many experimental conditions. Simulated phenomena include: saccade staircases, duration and amplitude of cell discharges for saccades of variable amplitude, component stretching to achieve straight oblique saccades, saturation of saccade velocity after saturation of saccade amplitude in response to high stimulation frequencies, tradeoffs between saccade velocity and duration to generate constant saccade amplitude, conservation of saccade amplitude in response to sufficiently brief stimulation of omnipause neurons, and high velocity smooth eye movements evoked by high levels of electrical stimulation of the superior colliculus. Previous saccade generator models have not explained this range of data. These models have also invoked mechanisms for which no neurophysiological evidence has been forthcoming, such as resetable integrators, perfect integrators, or target position movement commands. The present model utilizes only known reticular formation neurons. It suggests that a key part of the feedback loop within the saccade generator is realized by inhibitory feedback from short-lead to long-lead burst neurons, in response to excitatory feedforward signals from long-lead to short-lead burst neurons. When this property is combined with opponent interactions between agonist and antagonist muscle-controlling neurons, and motor error, or vector, inputs from the superior colliculus and other saccade-controlling brain regions, all of the above data can be explained. Taken together, these components generate a saccade reset cycle whereby activation of long-lead burst neurons inhibits omnipause neurons and thereby disinhibits short-lead excitatory burst neurons. The excitatory short-lead burst neurons can then respond to excitatory inputs from the long-lead burst neurons. Outputs from the excitatory short-lead burst neurons are integrated by the tonic cells while they also inhibit the long-lead burst neurons via inhibitory burst interneurons. When this inhibition is complete, the omnipause neurons are disinhibited. The omnipause neurons can then, once again, inhibit the short-lead burst neurons, whose inhibition of the long-lead burst neurons is thereby removed. The saccadic cycle can then begin again. In response to sustained electrical input, this cycle generates a staircase of identical saccades whose properties match the data much better than the staircases proposed by alternative models. A comparative analysis of the hypotheses and predictive capabilities of other saccade generator models is provided. © 1998 Elsevier Science Ltd. All rights reserved.

Keywords: Eye movements; Saccade generator; Reticular formation; Burst neurons; Saccade staircase; Neural network

1. Introduction

Saccades are rapid eye movements which enable the fovea to quickly scan a visual scene. Cells in the reticular formation represent the final common pathway in oculomotor control. The reticular formation consists of a number of saccade-related areas which provide direct input to the oculomotor neurons. These areas contain a number of functionally distinct cell types, including tonic neurons (TN), excitatory and inhibitory burst neurons (EBN and IBN) some of which display a long-lead prelude of activity (LLBN), and omnipause neurons (OPN) (Luschei and Fuchs, 1972; Keller, 1974) (Fig. 1). These cells appear to be connected in such a manner as to produce a local feedback loop which controls the eye.

The precise functional organization of this circuit, known as the saccade generator (SG), is still a topic of debate. This is a part of the brain that invites, indeed requires, models in order to be understood, and many models (e.g. Robinson, 1975; Jurgens et al., 1981; Grossberg and Kuperstein, 1986; Scudder, 1988; Dominey and Arbib, 1991; Moschovakis, 1994; Breznen and Gnadt, 1997) have been proposed about how it works. These models can be broadly classified into two basic types: *position models* in which the quantity to be canceled by feedback represents a position of the eye

* Corresponding author. Tel.: +1-617-353-7857; Fax: +1-617-353-7755; E-mail: steve@cns.bu.edu

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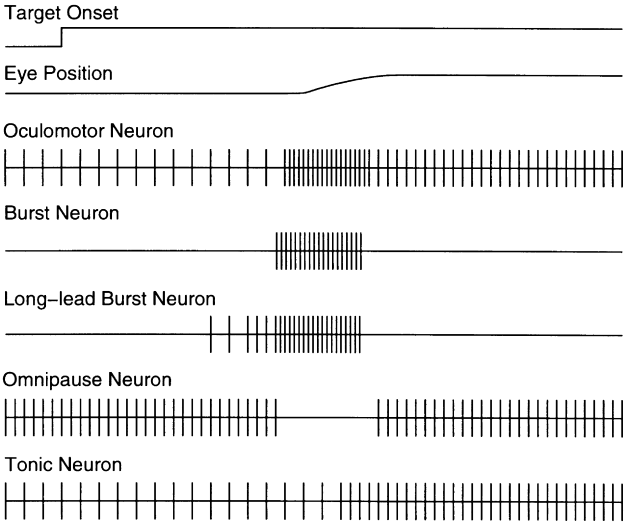

Fig. 1. Typical discharge patterns of SG cells during saccades. Eye muscles are controlled by oculomotor neurons which show a pulse-step pattern of activity. Burst neurons produce a high frequency burst of activity during saccades, but are silent during fixation. Long-lead burst neurons follow a pattern of low-frequency discharge, followed by a high frequency burst during the saccade. Omnipause neurons discharge at high rates during fixation, but are completely silent during saccades. Tonic neurons display a maintained discharge that is proportional to eye position over much of the ocular range. Adapted with permission from Schall (1991).

in the orbit (Robinson, 1975; Grossberg and Kuperstein, 1986), and *vector models* in which the quantity to be canceled is a motor error that can represent both the direction and length of a movement (Jurgens et al., 1981; Scudder, 1988; Moschovakis, 1994; Breznen and Gnadt, 1997). In the position models, it is dimensionally consistent to have feedback from a cell representing eye position, while in the vector models the motor error can be canceled by a quantity representing the displacement of the eye.

Evidence appears to favor the vector type models. The superior colliculus (SC) and frontal eye fields (FEF), which are the primary inputs to the SG, encode a displacement signal, and not the desired absolute position of the eye (Sparks and Mays, 1980; Scudder, 1988). Further support for vector models comes from recent results by Nichols and Sparks (1995), who found that the size of saccades evoked by electrical stimulation of the SC are influenced by prior visually guided saccades in a way that is consistent with vector processing.

The vector models proposed to date have, however, made a number of unrealistic assumptions. One common assumption is the inclusion of a resetable integrator (RI) cell to provide a displacement feedback signal to cancel the motor error movement signal (Jurgens et al., 1981; Moschovakis, 1994). The activity profile of such a cell is typically a nearly linear increase in firing rate until the saccade ends, at which time the integrator is actively reset, thereby causing its activity to drop to zero. The activity profile of a RI cell does not resemble that of typical reticular neurons. Scudder (1988) was able to avoid the use of a resetable integrator by proposing that the LLBNs act as perfect integrators. In the Scudder (1988) model, the LLBNs integrate the excitatory desired displacement input from the SC, as well as the inhibitory feedback from the burst neurons. However, evidence does not indicate that LLBNs are perfect or near perfect integrators (Raybourn and Keller, 1977). The Scudder (1988) model made other unrealistic assumptions. For example, the trigger connection from the SC to the OPN is inhibitory in the model, while it has been experimentally shown that the direct connections from the SC to the OPNs are excitatory (Raybourn and Keller, 1977; Gandhi and Keller, 1997). The Scudder model has difficulty producing large saccades. For large saccades, the EBN's burst shape takes on an unrealistic appearance, as the peak activity occurs late in the burst. The Scudder model also does not produce realistic saccadic staircases. The model produces repetitive saccades in response to sustained input, but these saccades are small and vary over a much smaller range of amplitude than found in actual data (Scudder, 1988; Breznen and Gnadt, 1997).

This paper describes a feedback opponent vector architecture (FOVEATE) which eliminates the unrealistic assumptions made by prior models. FOVEATE derives its name from the fact that it combines vector and opponent cell interactions with an internal feedback circuit — from EBN neurons via IBN neurons to LLBN neurons — that uses only known SG neurons. Because of these circuit interactions, EBNs function as a resetable integrator, and we propose that a separate resetable integrator cell type is unnecessary. Further, the model's LLBNs are not perfect or near perfect integrators. Simulations show that FOVEATE can explain a range of phenomena unmatched by prior SG models (Table 1). These data include: saccade staircases, duration and amplitude of cell discharges for saccades of variable amplitude, component stretching to

Table 1
Comparison of SG models

Model	Accurate interrupted saccades	Velocity duration tradeoff	Staircase	Smooth staircase	Straight oblique staircase
Ro:75	M	M	N	N	N
JuBeKo:81	M	M	N	N	N
GrKu:86	M	M	M	N	M
Sc:88	Y	M	N	N	N
DoAr:91	M	M	M	N	M
Mo:94	Y	M	Y	N	M
BrGn:97	M	M	Y	Y	M
GaGr:98	Y	Y	Y	Y	Y

Ro:75 = Robinson (1975); JuBeKo:81 = Jurgens et al. (1981); GrKu:86 = Grossberg and Kuperstein (1986); Sc:88 = Scudder (1988), DoAr:91 = Dominey and Arbib (1991); Mo:94 = Moschovakis (1994); BrGn:97 = Breznen and Gnadt (1997); GaGr:98 = Gancarz and Grossberg (1998) (FOVEATE).

Y stands for 'Yes, simulations show that the model can explain the data', M stands for 'Might be able to explain the data, but this has not been simulated', and finally, N stands for 'No, unable to explain the data'.

achieve straight oblique saccades, saturation of saccade velocity after saturation of saccade amplitude in response to high stimulation frequencies, tradeoffs between saccade velocity and duration to generate constant saccade amplitude, conservation of saccade amplitude in response to sufficiently brief stimulation of omnipause neurons, and high velocity smooth eye movements evoked by high levels of electrical stimulation of the superior colliculus (Schiller and Stryker, 1972; Evinger et al., 1981; Breznen et al., 1996; Missal et al., 1996; Stanford et al., 1996).

2. Methods

The subset of FOVEATE that controls right and left movements is shown in Fig. 2. The model LLBNs receive inputs from the SC, the FEF, and the cerebellar nuclei. The LLBN activity codes motor error. Suppose, for example, that a movement input is received from the left movement channel. Then its target LLBN population excites the corresponding EBN population, which in turn excites the TN population. The TN integrates the EBN's signal. The EBN also excites inhibitory burst neurons (IBN), which have inhibitory connections to the LLBNs. This closes a feedback loop within the SG. The EBNs are strongly inhibited by the OPN. The OPN is tonically active due to an arousal input (A), and is turned off by inhibition from the LLBN. Inhibition from an agonist EBN on the antagonist TN ensures that as the agonist TN activity increases, the antagonist TN activity decreases, or vice-versa.

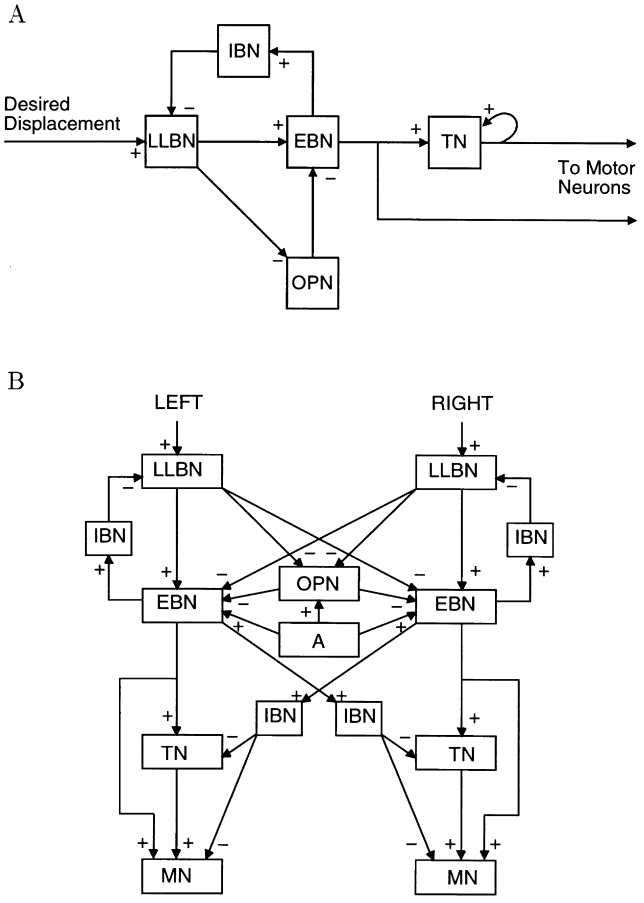

Fig. 2. (a) FOVEATE model of the saccade generator for control of a single extraocular muscle; and (b) FOVEATE model for control of an antagonistic pair of extraocular muscles. Long-lead burst neurons (LLBN), excitatory burst neurons (EBN), inhibitory burst neurons (IBN), omnipause neurons (OPN), arousal signal (A), tonic neurons (TN), motorneurons (MN).

FOVEATE draws upon many of the favorable aspects of earlier models of SG function (that are more thoroughly compared in Section 4), while eliminating those hypotheses not supported in the data. First, the Robinson (1975) hypothesis of a feedback loop is retained, as it is supported by evidence such as saccadic staircases and accurate interrupted saccades (Schiller and Stryker, 1972; Keller and Edelman, 1994). We also retain the Robinson (1975) idea that the EBNs and TNs both excite MNs, leading to their pulse step behavior (Keller, 1981), and the opponent and cellular organization of the Grossberg and Kuperstein (1986) model. In contrast to the Robinson (1975) and Grossberg and Kuperstein (1986) models, we assume the total input to the LLBN is desired eye displacement, similar to Scudder (1988). We thus eliminate the eye position input of the Grossberg and Kuperstein (1986) model. The LLBNs in the present SG model are not assumed to be perfect or near perfect integrators, as in the Scudder model, since this assumption is not supported in the data (Raybourn and Keller, 1977). We also do not need to hypothesize a separate resetable integrator cell type, as in Jurgens et al. (1981), since in the FOVEATE model, the EBN functions as a resetable integrator that is reset by the OPN. In contrast to the Dominey and Arbib (1991) model, which requires two types of LLBN cells to separately code the velocity and size of saccades, FOVEATE requires only a single vector (speed and size) LLBN cell type.

Simulations of the model illustrate that FOVEATE addresses many of the problems of previous models. For example, while the Scudder (1988) model had difficulty producing saccadic staircases, FOVEATE is able to produce realistic staircases. This property derives from the model's *reset cycle*. This reset cycle refers to how the EBN to LLBN inhibitory feedback can shut down the LLBN and how that, in turn, can disinhibit OPN activity, thereby shutting down the EBN and disinhibiting the LLBN, which initiates the cycle again. This reset cycle is discussed further in Section 3.

The model does not include all known connections, since these are not rate-limiting in simulating the targeted data. For example, Chimoto et al. (1996) have reported that the superior colliculus projects directly to the EBNs. This could only help model performance by acting as an additional task-sensitive arousal input. Gancarz and Grossberg (1997, 1998) show how FOVEATE can be embedded in a larger architecture that includes aspects of superior colliculus, parietal and prefrontal cortex, and cerebellum.

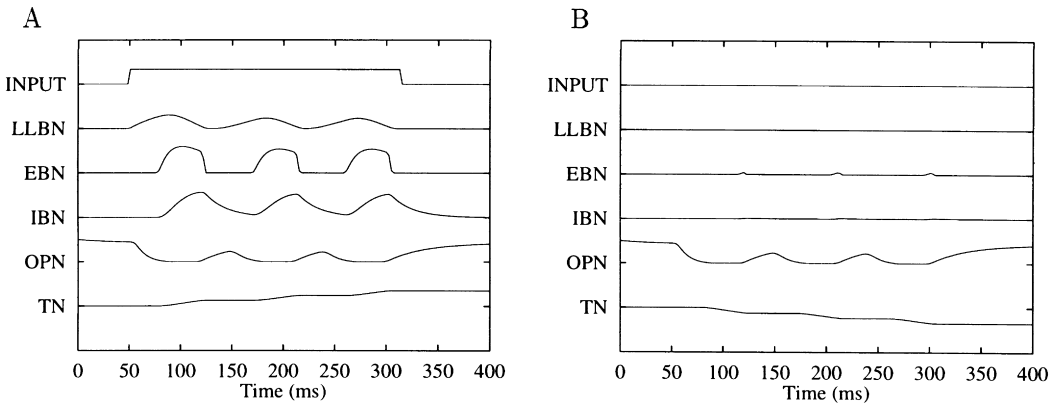

Fig. 3. Activity profiles produced in response to sustained input to the left side of the SG: (a) activity in the left side of the SG; and (b) activity in the right side of the SG.

3. Results

3.1. Cell activity profiles and saccadic staircases

Fig. 3 shows the evolution of SG cell activity in response to constant input I_l to the left side that is on for 265 ms. The model cell activation profiles resemble those found in the reticular formation (see Fig. 1). The model LLBNs show a prelude of activity, which is characteristic of the cell type (Van Gisbergen et al., 1981). The EBN burst begins after the onset of LLBN activity (Luschei and Fuchs, 1972). The antagonist EBN produces a small burst at the end of a saccade. This antagonistic rebound has been found experimentally (Van Gisbergen et al., 1981; Brown and Day, 1997), and may function as a braking pulse to decelerate the eye at the end of a saccade. Due to inhibition from one side of the SG on the other, as the activity of one TN increases, the activity of the other decreases by a similar amount. The OPN ceases firing completely during a saccade, as found experimentally (Raybourn and Keller, 1977).

The model produces saccadic staircases in response to sustained input as seen in Fig. 3. Saccadic staircases are a series of saccades of similar amplitude, caused by continuous stimulation of the SC (Schiller and Stryker, 1972). The model produces staircases as a result of interactions between the LLBN, the EBN and the OPN, as illustrated in Fig. 4. The IBN has been left out of the Fig. 4 since IBN activity follows the EBN activity.

During fixation, the model is in the rest phase. As shown in Fig. 4(a), the OPNs are active, inhibiting the EBNs, and

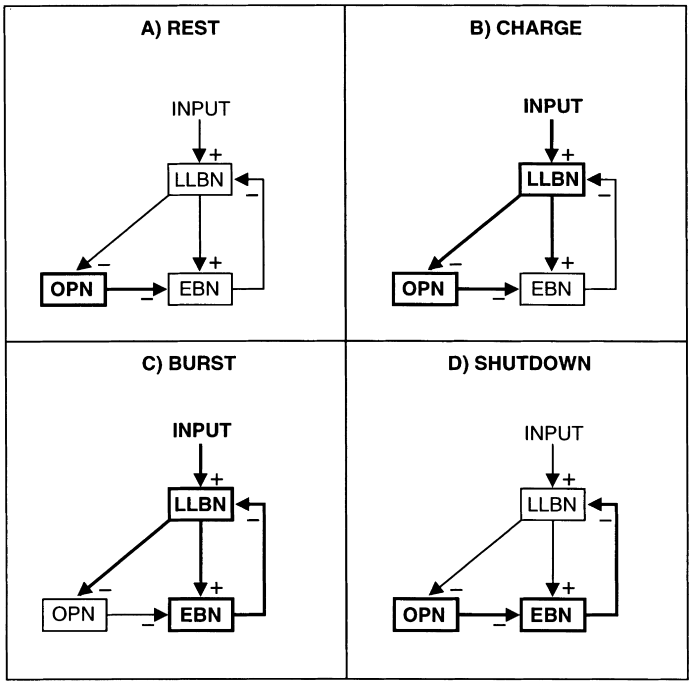

Fig. 4. FOVEATE model reset cycle. Bold lines and text indicate activity. Model reset cycle involves four phases: rest, charge, burst, and shutdown. Only the agonist side of the model SG is shown.

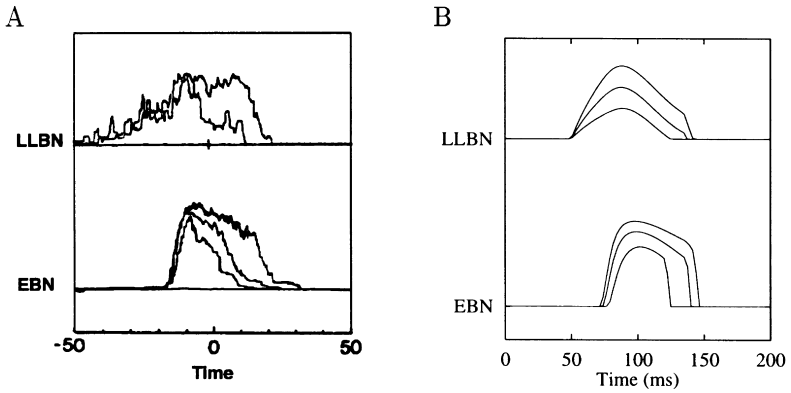

Fig. 5. (a) Cell activity profiles in the reticular formation of monkey. LLBN discharge rate for 5 and 22 degree saccade, and EBN discharge rate for 5, 10, and 20 degree saccades. Cells burst at greater levels and increased duration for larger saccades. Data replotted with permission from Van Gisbergen et al. (1981). (b) Simulation. Increased input strength results in larger LLBN and EBN burst size.

thus suppressing saccades. When the input to the SG is turned on, the LLBN's activity begins to build. This is known as the charge phase [Fig. 4(b)]. The LLBN inhibits the OPN. Once the LLBN has successfully turned the OPN off, the EBN is free to burst due to excitation from the LLBN, and the model enters the burst phase [Fig. 4(c)]. During the burst phase, negative feedback from the EBN to the LLBN (through the IBN) causes the LLBN activity to decay. During this phase the EBN activity also decays, since the excitatory input from the LLBN to the EBN is decreasing. Once the EBN burst has turned off the LLBN through the negative feedback loop, the model enters the shutdown phase [Fig. 4(d)]. With the LLBN off, inhibition on the OPN is removed, and the OPN begins to fire again. The OPN activity strongly inhibits the EBN, in effect 'resetting' it. The model is now once again at the rest phase, and the reset cycle is complete. If the input to the SG is left on, then the LLBN again begins to charge in response to the sustained input, and the saccadic cycle continues. In this manner, a series of saccades can be produced.

3.2. Saccadic amplitude and duration to varying input levels

A SG model needs to be able to produce saccades of varying amplitude. FOVEATE responds to increased input levels with larger saccades. As the input level is increased, the LLBN and EBN burst amplitude and duration increases, as illustrated in Fig. 5(b). These larger bursts result in increased saccadic amplitudes.

The model LLBN and EBN activity profiles resemble those recorded from the reticular formation shown in Fig. 5(a). In fact, model EBN burst shape represents the four phases of the model reset cycle. At rest, both the LLBN and the EBN are silent. When input to the SG is turned on, the LLBN begins to charge and its activity increases. While the LLBN is charging, the EBN is silent due to OPN inhibition. Once the LLBN turns the OPN off, the EBN is free to burst, and its activity rapidly reaches a maximum. This is why EBN onset is delayed relative to LLBN onset in the model. As the burst phase continues, EBN activity slowly decays since the LLBN activity is decreasing. Finally, during shutdown, the LLBN activity has been sufficiently reduced to allow the OPN to once again begin firing. OPN reactivation results in termination of the EBN burst.

The recorded LLBN burst shape shows a longer buildup than in our model. This happens in the model simulations because the input to the LLBN used a step function, for simplicity, and also because the simulations lacked a complete SC circuit, which includes burst, buildup, and fixation neurons (Munoz and Wurtz, 1995a, b; Grossberg et al., 1997). Since SC fixation cells excite the OPNs (Gandhi and Keller, 1997), and the fixation neurons take time to turn off (Munoz and Wurtz, 1993), this would further delay the OPN pause, and thus delay the saccade. However, during the period when the fixation cell activity is decreasing, the LLBN would receive excitatory input from the SC burst and buildup cells, whose activity is increasing. In this manner, there would be a longer buildup of activity in the LLBNs. Simulations connecting the SG to a full SC model have found a longer LLBN buildup, as in the data (Gancarz and Grossberg, 1997, 1998).

3.3. Straight oblique saccades

Oblique saccades typically display straight trajectories (Evinger et al., 1981). By joining two FOVEATE models together, the complete model produces straight oblique saccades. One SG controls horizontal eye movement (the horizontal movement component), while the other controls vertical movement (vertical component). Both share a single OPN, as in Scudder, (1988). However, unlike the Scudder (1988) model, FOVEATE controls the OPN using the LLBNs, which represent motor error, and this allows FOVEATE to produce realistic saccadic staircases, which the Scudder model cannot (see Section 4). Fig. 6(b) shows five separate eye movements produced by FOVEATE that vary in direction and amplitude. The

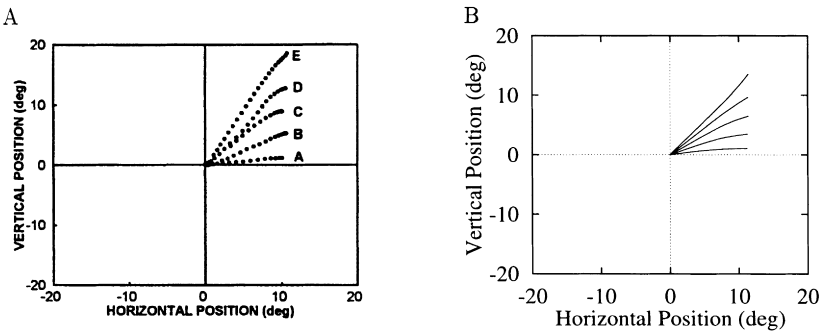

Fig. 6. (a) Visually guided saccades in monkey. Reprinted from Nichols and Sparks (1996) with permission. (b) The model produces fairly straight oblique saccades.

saccades remain fairly straight for all five directions, and resemble those produced by monkeys, including a slight tendency to curve, as shown in Fig. 6(a) (Nichols and Sparks, 1996).

It has been shown that subsequent saccades in a staircase continue in the same direction as the initial saccade (Schiller and Stryker, 1972; Breznen et al., 1996). FOVEATE also displays this property. Fig. 7 shows three simulated saccades in a staircase produced by constant input to both the horizontal and vertical SG. The saccades are of equal length, as in the data (compare with Fig. 2 in Schiller and Stryker (1972)). Eventually, the saccades become shorter, if only because of cell saturation and approach to the edge of the workspace.

FOVEATE produces straight oblique saccades because the smaller saccade component is stretched. This stretching occurs because the larger component shuts off the OPN for a longer period of time than if the smaller component was acting alone. This increases the small component's EBN burst duration, since in the model, OPN reactivation is largely responsible for terminating a EBN burst. Thus, the small component burst is stretched to match the large component burst. This results in straight saccades. Thus straight oblique saccades follow naturally from the assumptions that the EBNs inhibit the LLBNs, and that both vertical and horizontal LLBNs can shut off a shared set of OPNs.

Quaia and Optican (1997) have recently suggested that component stretching during oblique saccades occurs as a result of the fairly broad Gaussian tuning curves of EBNs and the finding that some EBNs have on-directions which are not purely horizontal or vertical. In their model, EBNs with rightward on-directions excite rightward MNs, while EBNs with leftward on-directions inhibit rightward MNs. A similar type of arrangement is hypothesized for leftward, upward, and downward MNs. In the case of a purely horizontal rightward saccade, there will be substantial activity in the EBNs with rightward on-directions, and minimal activity in the leftward EBNs. As a vertical component is added to this saccade, the leftward EBNs become substantially active due to their broad tuning curves. This activity inhibits the rightward MN, reducing its activity. This reduces the rate the horizontal motor error is canceled by feedback, and thus stretches the smaller component.

Some problems with the Quaia and Optican (1997) model are its lack of specificity with regard to the feedback loop and the neural mechanisms underlying the proposed calculations in the model. In the Quaia and Optican (1997) model, the EBN activities were calculated by comparing the direction of the current motor error vector to the on-direction of the EBN. How this calculation would occur biologically is not specified in the model. Nor is the source of the feedback signal, or the method for where and how the current motor error vector is calculated and represented. Also, Scudder et al. (1988) noted that the on-direction of EBNs tend to cluster near the axes, while Quaia and Optican (1997) used a uniform distribution of on-directions. The finding that on-directions tend to cluster near the axes supports lumping the EBNs together, as in FOVEATE and many other SG models. Also, even though in FOVEATE the EBNs were lumped together, this does not mean the tuning curves of model EBNs are circular. Fig. 8 shows the EBN tuning curve for FOVEATE. The arousal input to the FOVEATE EBNs, coupled with the opponent nature of the model results in a cardioid tuning curve like that found in the data (Scudder et al., 1988). Still, it may be

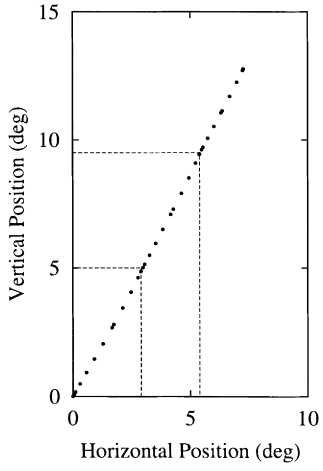

Fig. 7. Saccades in a staircase continue in the same direction as the initial saccade. Three saccades are shown in this figure. Eye position was sampled at regular time intervals.

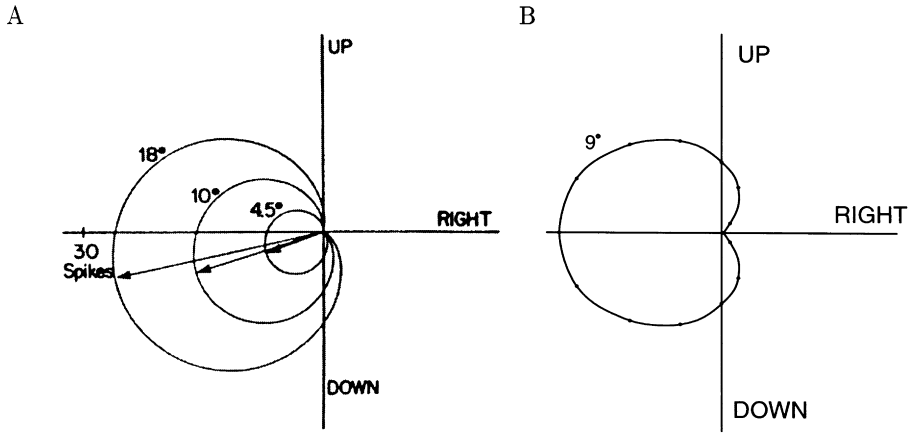

Fig. 8. (a) Tuning curve for a short-lead burst neuron. Reprinted from Scudder et al. (1988) with permission. (b) FOVEATE EBN neuron has a cardioid-like tuning curve.

the case that having a distribution of EBN on-directions assists in producing straight oblique saccades, as suggested by Quaia and Optican (1997).

3.4. Velocity saturating after amplitude

Stanford et al. (1996) varied the frequency of electrical stimulation to the SC and found that the amplitude of a saccade evoked from a particular point on the SC is not only a function of stimulation location. Rather, the stereotypical saccade amplitude for a particular site is obtained only with sufficiently high stimulation frequency. Below this level, saccades of smaller amplitude are produced. Stanford et al. (1996) also found that the function relating saccade amplitude to stimulation frequency saturated before the function relating saccade velocity to stimulation frequency (i.e. velocity continued to increase after amplitude had peaked). The higher velocity saccades had shorter durations than the slower velocity saccades of similar amplitude. These findings are summarized in Fig. 9(a)–(c) which shows saccade amplitude, duration, and peak velocity as a function of stimulation frequency.

In FOVEATE, velocity also saturates after amplitude. To demonstrate how this happens in the model, simple assumptions about the effect of electrical stimulation of the SC, and how this signal gets relayed to the reticular formation, must

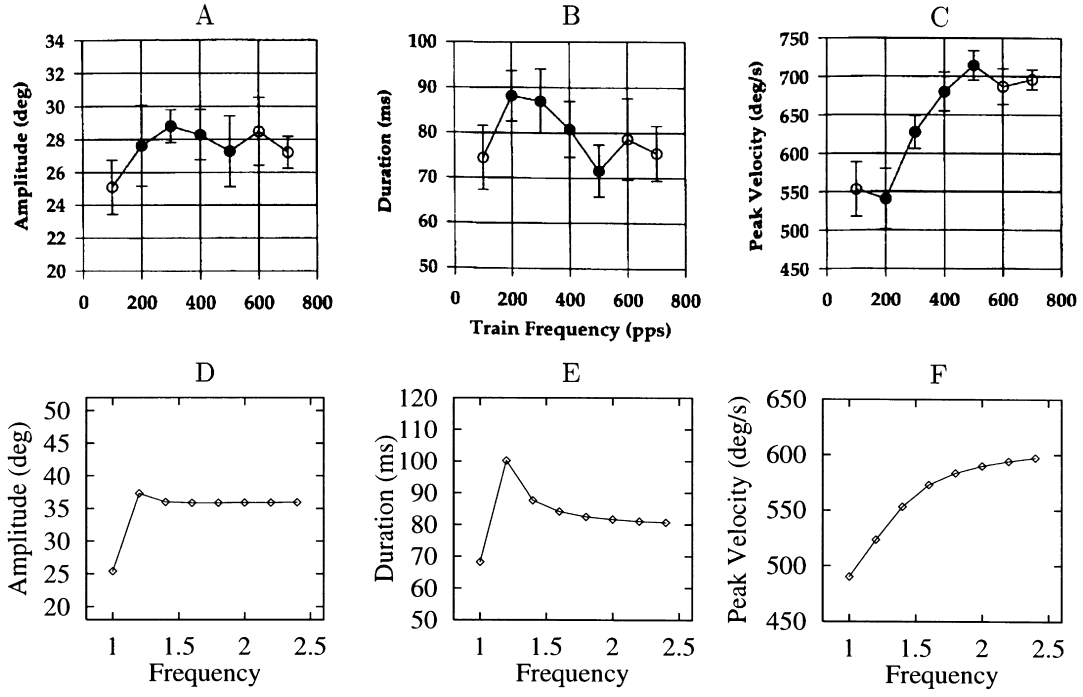

Fig. 9. Effect of stimulation frequency on: (a) saccadic amplitude; (b) duration; and (c) peak velocity in monkey. Range over which amplitude and duration vary is highlighted by filled symbols. Reprinted from Stanford et al. (1996) with permission. Effect of stimulation frequency on: (d) model saccadic amplitude; (e) duration; and (f) peak velocity.

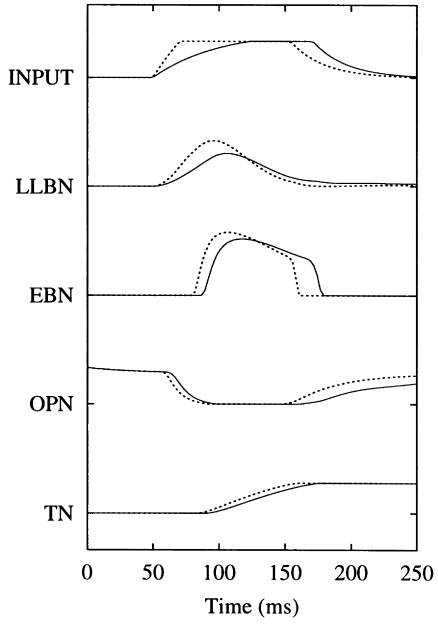

Fig. 10. Velocity and duration can be traded, keeping amplitude constant. Only the shape of the input signal to the model was varied. In this Figure, and Figs. 3, and 12, the tonic cell (TN) output approximates saccade shape, since the motor plant was not modeled.

first be made. These assumptions are consistent with models of the deeper SC layers (Gancarz and Grossberg, 1997; Grossberg et al., 1997).

For any given region of the brain, current strength is a primary determinant of the size of the population of cells that is activated directly by microstimulation (Stanford et al., 1996). Since current was not varied in the Stanford et al. (1996) study, it seems reasonable that for a given stimulation site, the effective current spread remained relatively constant. The frequency of stimulation, however, was varied. Increased stimulation frequency causes a neuron's firing rate to increase, until the maximum firing rate of the cell is reached.

Since the size of the population remains fairly constant as frequency is increased, once the cells in that population cause the signal function between the SC and LLBN to saturate, the amplitude of the input signal reaching the SG will reach a maximum. How rapidly this output reaches the maximum, however, depends on the frequency of stimulation. This can be seen by looking at the input traces in Fig. 10, which show the input to the SG for two values of stimulation frequency (F). The value of F was set to 1.3, and 3, corresponding to the solid and dotted traces in the Figure. The input to the SG reaches the same level, but more rapidly in the higher stimulation frequency case. With higher frequency stimulation, the model produces a EBN burst of higher amplitude, but shorter duration. Note that in both cases, the amplitude of the eye movement (TN activity) is the same.

Fig. 9 (d)–(f) shows the relationship between stimulation frequency and saccade amplitude, peak velocity, and duration for the model. As frequency is increased, saccade amplitude initially increases, reaches a maximum, and then slightly declines. Velocity, however, continues to increase with higher stimulation, even after amplitude has saturated. As velocity increases and amplitude levels off, the saccade duration decreases. This occurs because the EBN burst grows more quickly and to a greater peak activity, while its duration decreases. Thus the TN and MN cells integrate faster, yielding higher peak speeds, but the total amplitude that they integrate is approximately constant. This property, too, derives from EBN-to-LLBN inhibitory feedback.

3.5. Smooth staircase eye movements

Breznen et al. (1996) and Missal et al. (1996) have found that when the SC is electrically stimulated for a prolonged duration at high levels, the resulting saccadic staircase degenerates. An initial saccade is produced, followed by smooth eye movement as shown in Fig. 11(a). By analyzing the velocity trace, these smooth movements were found to often consist of a series of accelerations and decelerations, as if a series of small saccades were being made, each of which starts before the previous one ends.

FOVEATE responds similarly to a high level of sustained input, as seen in Fig. 11(b). An initial EBN burst is followed

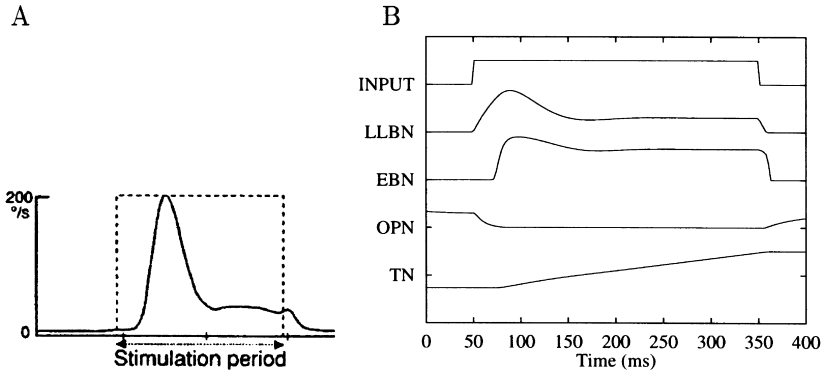

Fig. 11. (a) Smooth eye movements evoked by electrical stimulation of SC in cat. Plot shows eye velocity as a function of time. Reprinted from Missal et al. (1996) with permission from Springer. (b) Simulation: smooth eye movement produced in response to high sustained input to the left side of the model. Input was three times stronger than that of Fig. 3. Compare model EBN discharge with eye velocity data on left plot.

by a lower sustained activity in the EBN firing rate. This occurs because, when the LLBN is receiving very strong excitatory input, feedback from the EBN–IBN burst is not sufficiently strong to drive the LLBN to zero. Thus, the LLBN stays partly active, and the OPN remains significantly inhibited, resulting in continuous EBN activity at a lower amplitude, which results in a smooth eye movement.

3.6. Interrupted saccades

When the omnipause region of the brainstem is stimulated by an electrical pulse during a saccadic eye movement, the saccade abruptly slows or stops, depending on the strength of stimulation (Keller, 1977; King and Fuchs, 1977). If stimulation is removed rapidly enough, the eye resumes movement, landing very close to the desired (uninterrupted) displacement (Keller and Edelman, 1994). The interrupted saccades are typically accurate if the stimulation pulse is applied near the beginning or middle of the saccade (Scudder, 1988).

To simulate stimulation of the OPN region, an excitatory input term (J) was added to the model equation that governs OPN activity in the model [see Appendix A, Eq. (8)]. Fig. 12 shows activity in the model when J is set to 1.8 for 5 ms, in the middle of the saccadic burst. The OPN begins to fire again, cutting short the EBN burst, and leaving the LLBN active. When OPN stimulation is removed, the saccade continues. The interrupted saccade has the same amplitude as the uninterrupted saccade, as can be seen by looking at the plot of TN activity for an uninterrupted saccade shown by the dotted line in the Figure. The duration of the interrupted saccade is longer than that of the uninterrupted one. The interrupted model saccades are accurate since the LLBN activity (which codes motor error) remains fairly constant during OPN stimulation. Thus, when stimulation is removed, the remaining LLBN activity again shuts off the OPN and a saccade is produced which zeros the remaining motor error. In this manner, the interrupted saccade lands near the endpoint of the uninterrupted saccade.

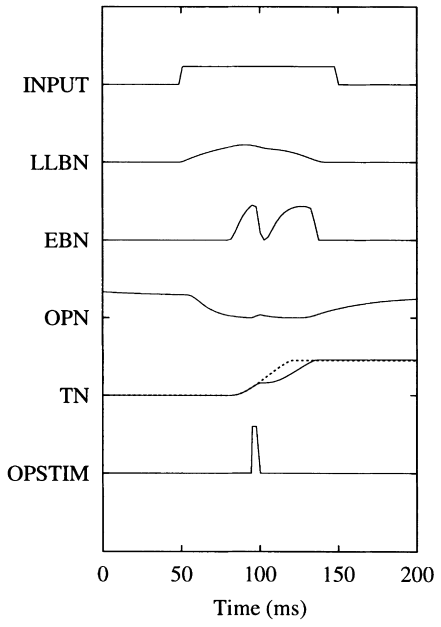

Fig. 12. OPN stimulation results in an interrupted saccade that remains accurate. Dotted line shows amplitude of uninterrupted saccade for comparison.

When a full superior colliculus is used to input to the saccade generator, longer interruptions may be compensated, since other modeling work has suggested how the spreading wave at the SC buildup cells may remain active until feedback from the tonic cells matches the desired foveation point (Grossberg et al., 1997).

4. Discussion

The simulations presented above show that a feedback opponent vector architecture (FOVEATE) can reproduce a large range of psychophysical and neurophysiological data about saccades without making the unrealistic assumptions of earlier models. FOVEATE instead predicts that EBN neurons can inhibit LLBN neurons via IBN neurons. This prediction does not yet seem to have been proved or disproved. Table 1 compares FOVEATE to other models of reticular function with respect to explaining a variety of experimental data. In the table, Y stands for ‘Yes, simulations show that the model can explain the data’, M stands for ‘Might be able to explain the data, but this has not been simulated’, and finally, N stands for ‘No, unable to explain the data’. As seen in the Table, all of the above models can in principle produce accurate interrupted saccades. This is because each of the above models, whether vector or position type, contains a feedback loop which compares desired displacement or position with current displacement or position. After an interruption, feedback allows computation of the remaining motor error, which can trigger a second saccade which foveates the target.

The above models can also likely explain the recent data of Stanford et al. (1996). Their experiments illustrated that by varying the frequency of SC stimulation, saccade velocity and duration can be traded, while keeping amplitude constant. As far as we are aware, our simulations are the first to explain these data. The key to understanding this data is the assumption of a saturating signal function between the SC and the SG. The frequency of stimulation changes the rate at which the input to the SG reaches the final displacement, but not the end value. That the amplitude of the saccades remains constant independent of stimulation frequency is inherent to a local feedback loop due to self-termination upon integration to a desired displacement (Robinson, 1975; Grossberg and Kuperstein, 1986). However, the saccade velocity depends on the frequency of stimulation, since more or less signal is injected into the feedback loop. Thus, when coupled with a saturating input, all models with a feedback loop should be able to

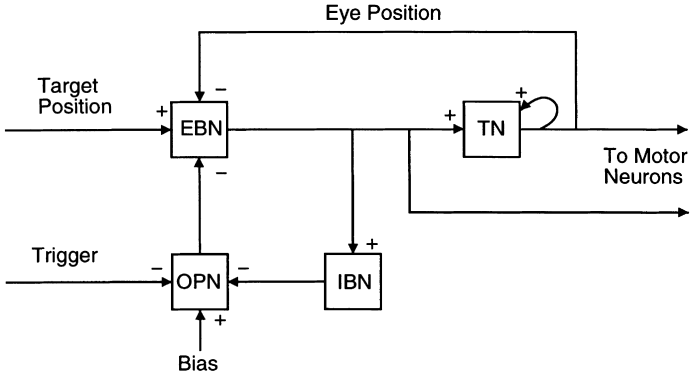

Fig. 13. Robinson (1975) model of the saccade generator. The input to the SG is the target position relative to the head. This target position input excites the excitatory burst neuron (EBN). An inhibitory trigger signal turns off the omnipause neuron (OPN), thus releasing the EBN from strong OPN inhibition. The EBN burst excites the tonic neuron (TN), which integrates its input. The EBN continues to burst until inhibitory feedback from the tonic neuron cancels the target position input. At this point, the EBN ceases firing, as does the inhibitory burst neuron (IBN), and this allows the OPN to once again begin to fire due to a steady excitatory input bias. The motor neurons receive input from both the EBN and the TN, thus showing the characteristic pulse step behavior found experimentally.

capture this data. We have found the basic effect to be robust for a range of parameter choices.

Saccade staircases provide a better test to differentiate between the models. Recall that saccadic staircases are a series of saccades of similar amplitude, caused by continuous stimulation of the SC or the FEF (Schiller and Stryker, 1972). The Table notes that the Robinson (1975) model (Fig. 13) cannot produce saccadic staircases. Sustained electrical stimulation of the SC or FEF most likely produces a constant input to the SG. In the Robinson (1975) model, once the TN has cancelled the position input to the SG, no further saccades will be produced. Thus, it is unable to explain staircases, nor smooth staircases, nor straight oblique staircases.

The Jurgens et al. (1981) model (Fig. 14) is also unable to produce saccadic staircases. This is because the model does not include any mechanism to reset its RI after a saccade.

The Grossberg and Kuperstein (1986) model (Fig. 15) is able to explain staircases because, after the first saccade, the initial eye position signal to the LLBN is updated. This increases the input to the LLBN by an amount corresponding to the length of the prior saccade. Thus, this extra input now triggers a second saccade equal in amplitude to the initial saccade. This process can repeat, producing a staircase. However, the model cannot explain smooth staircase data, since within the ocular range the TN can always cancel the desired position signal. Also, the initial eye position signal of the Grossberg and Kuperstein (1986) model has not been found in the brainstem. In fact, FOVEATE is the same as the Grossberg and Kuperstein (1986) model with the initial eye position signal removed and the inhibitory feedback to the LLBNs moved from the TN to the EBN–IBN. This comparison illustrates the predictive power of FOVEATE's new feedback hypothesis.

The Scudder (1988) model (Fig. 16) does not produce realistic saccadic staircases (Scudder, 1988; Breznen and Gnadt, 1997). The model produces repetitive saccades in response to sustained input, but these saccades are small and vary over a much smaller range of amplitude than found in actual data (Scudder, 1988).

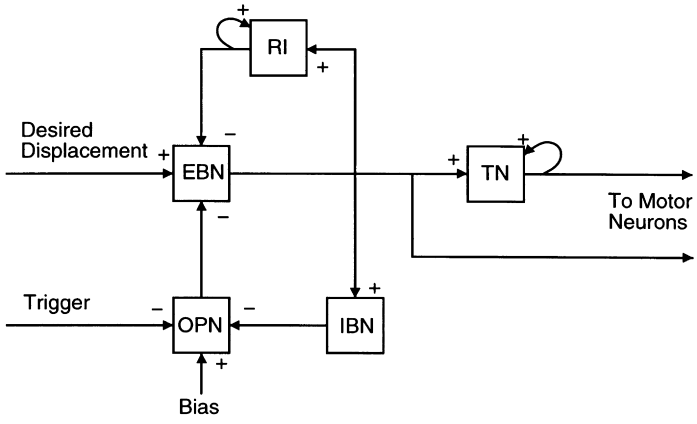

Fig. 14. The Jurgens et al. (1981) model of the SG modifies the Robinson model to address the concern that the SC and FEF output is a displacement signal. Input to the model is the desired displacement of the eyes, and not the absolute position of the eye in the head. Local feedback originates from a resetable leaky integrator (RI) instead of from a representation of eye position, as in the Robinson model. The RI integrates the EBN burst, and is reset to zero before each saccade. Since the EBN burst has the dimension of velocity, the RI codes displacement.

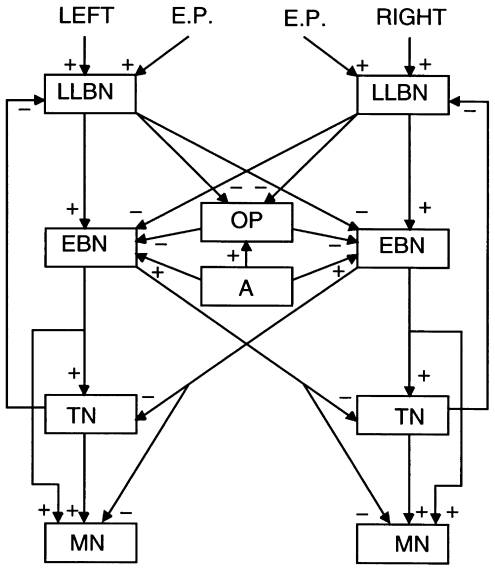

Fig. 15. Grossberg and Kuperstein (1986) model of the saccade generator. The LLBN codes the desired spatial position of the eye in the head. The SC is assumed to send a signal to the LLBN coding the desired eye displacement. By combining this signal with an eye position signal which codes eye position prior to a saccadic movement, the LLBN can code desired eye position. The eye position input to the LLBN is only updated after the movement is completed. Feedback to the LLBN comes from the TN, which codes eye position. When this feedback has quenched the LLBN activity, the OPN is released from inhibition by the LLBN. The OPN begins to fire, and inhibits the EBN, thus terminating the eye movement.

Of the three models which have simulated saccadic staircases (Moschovakis (1994); Breznen and Gnadt (1997); and FOVEATE), we believe the FOVEATE mechanism is most consistent with data on staircases, and computationally most robust. Saccadic staircases are produced in FOVEATE as a result of interactions between the LLBNs, EBNs and OPNs, and the saccades composing the staircase are of equal amplitude and direction. This results from the model reset cycle, in which the model returns to a rest state after each saccade. In addition, at high input levels, FOVEATE staircase degenerates into smooth eye movement, as found by Breznen et al. (1996) and Missal et al. (1996). This again occurs in the model because of its reset cycle since, when the LLBN is receiving very strong excitatory input, the EBN–IBN feedback burst is not sufficiently strong to drive the LLBN to zero.

Breznen and Gnadt (1997) have recently shown that a modified version of the Jurgens et al. (1981) model can reproduce some aspects of staircase saccades. In the original Jurgens et al. (1981) model, input to the model is the desired displacement of the eyes, and local feedback to the EBN originates from a resetable leaky integrator (RI). The RI integrates the EBN burst, and is reset to zero before each saccade. Since the EBN burst has the dimension of velocity, the RI codes displacement. Breznen and Gnadt (1997) replaced the RI with a leaky integrator. They then showed that a step input to their modified model produces an oscillatory response which consists of an initial large EBN burst followed by a series of smaller EBN bursts. At higher stimulation levels, they produced smooth eye movement consisting of a series of accelerations and decelerations.

Both FOVEATE and the Breznen and Gnadt (1997) model reproduce the finding that at high SC stimulation levels, smooth eye movements are produced. In both models, at these high stimulation levels, the OPNs remain inactive throughout the duration of the stimulus train, as found in the data (Reusser et al., 1996). However, at lower stimulation levels, the Breznen and Gnadt (1997) model produces a large saccade, followed by very small saccades, while FOVEATE produces staircases which contain saccades of equal amplitude and direction. Typically, all the saccades in a staircase are of the same amplitude (Schiller and Stryker, 1972; Schiller, 1977).

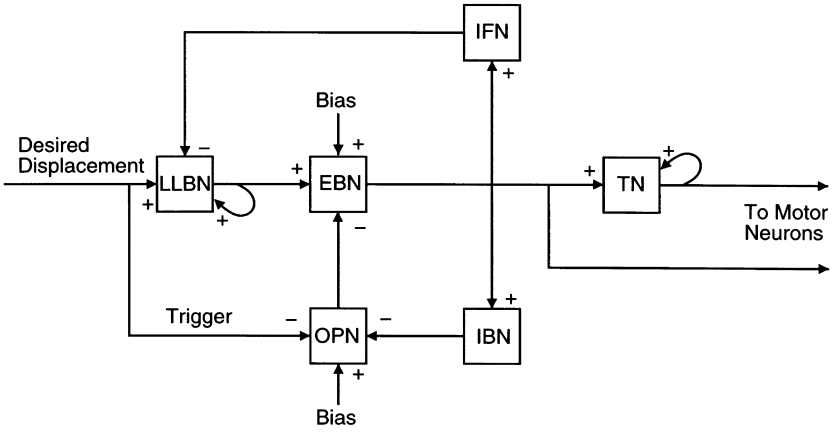

Fig. 16. Scudder (1988) model of the saccade generator. The SC output corresponds to a desired displacement of the eye, and not the desired position of the eye in its orbit, as in the Robinson model. Since the same desired displacement command can be issued from any number of initial eye positions, the model cannot use the TN as a source of feedback as they are not dimensionally consistent. Instead, the LLBNs receive inhibitory feedback from an inhibitory feedback neuron (IFN) which receives input from the EBN. The LLBN integrates the excitatory desired displacement input, as well as the inhibitory input from the IFN. Output from the LLBN goes to the EBN. The model's EBN is designed to fire at a particular rate in the absence of any input. This 'bias' toward activity can be represented by a tonically active excitatory input signal. The EBN output is integrated by the TN. The motorneurons receive input from the EBN and the TN, as in the Robinson model.

This difference derives in part from how the OPNs are controlled in the two models. In FOVEATE, the OPN is inhibited by the LLBN, which codes motor error. After the first saccade in a staircase, the LLBN motor error is quenched by the feedback loop, and the OPN partially reactivates due to its arousal input. This reactivation shuts off the EBN (which also codes motor error), and in turn, the IBN. At this point, the model reset cycle has reached its rest phase, and the activities of all the cells are approximately the same as before the initial saccade. Thus, each subsequent saccade begins from the same initial conditions, and thus has approximately the same amplitude. In contrast, in the Breznen and Gnadt (1997) model, the OPN is assumed to be strongly inhibited by SC activity. This assumption is not supported, as it has been experimentally shown that the direct connections from the SC to the OPNs are excitatory (Raybourn and Keller, 1977; Gandhi and Keller, 1997). Their model OPN remained completely inactive during the stimulation period, since the SC remains active. Thus, after the first saccade, the OPN remains off, allowing the EBN to burst too rapidly, before the leaky integrator (LI) has decayed sufficiently. Thus, subsequent EBN bursts are small.

FOVEATE is also supported by the simulation of Fig. 7 which showed that model oblique staircases continue in the same direction as the initial saccade, consistent with the data. However, it is unclear whether the Breznen and Gnadt (1997) model would share this property. FOVEATE cell activity profiles are also more realistic than those of the Breznen and Gnadt (1997) model. For example, the FOVEATE EBN burst shown in Fig. 5 peaks toward the beginning of the burst, while the Breznen and Gnadt (1997) model EBN peaks in the middle of the burst.

Another model which is able to produce saccadic staircases is the Moschovakis (1994) saccade generator model, illustrated in Fig. 17. The Moschovakis (1994) model is able to produce saccadic staircases in the following manner: electrical stimulation of the SC excites the latch cell, causing latch cell activity to build, and this activity inhibits the OPN. Once the OPN activity has ceased, the inhibition on the RI and LLBN is removed, allowing a saccade to begin. Excitation from the LLBN causes the EBN to burst, producing a saccade. Once the inhibitory feedback from the RI has quenched LLBN activity, the EBN activity decays since the excitatory input from the LLBN has been removed. The latch activity also decays, since the excitatory input from the EBN has been removed. Since stimulation to the SC continues, the latch cell cannot decay completely, as it is excited by SC activity. However, the latch cell decays sufficiently to allow the OPN activity to rise slightly as a result of the excitatory bias on the OPN. The slight rise in OPN activity resets the RI, and the first saccade terminates. Continued SC stimulation again excites the latch cell, turning off the OPN, beginning a second saccade, and thus producing a staircase.

As with the Breznen and Gnadt (1997) model, the important difference with regards to producing saccadic staircase is how FOVEATE and the Moschovakis (1994) model control the OPN. In FOVEATE, the OPN is controlled by the LLBN, which represents motor error. When the feedback loop has zeroed the motor error, inhibition from the OPN is removed, allowing the OPN to fire. In the Moschovakis (1994) model, the OPN is controlled by a latch cell, which in turn is controlled by the EBN and the SC. Latch cell activity inhibits the OPN allowing a saccade to begin. Note that without the connection from the SC to the latch cell in the Moschovakis (1994) model, saccades could not begin at all. This is because the OPN strongly inhibits the EBN. Thus if the OPN is on, for example before a saccade, the EBN cannot become activated, and thus the latch cell cannot become activated. Only by having a connection from the SC to the latch cell can the saccadic process by initiated in the Moschovakis (1994) model.

It is this extra connection from the SC, which is not needed in FOVEATE, which is one of our primary criticisms of the Moschovakis (1994) model. As shown in the Moschovakis (1994) model diagram, there is a weight between the SC and the LLBN, which controls the size of a saccade. This weight is not present in the SC-to-latch-cell connection. This is because the Moschovakis (1994) model can only produce staircases if the SC-to-latch-cell connection strength is tightly controlled. With too strong a connection, the latch cell will stay strongly activated throughout SC stimulation, and thus the OPN will never be reactivated. Thus, only a single saccade would be produced since the RI would not be reset. If the SC-to-latch-cell connection is too

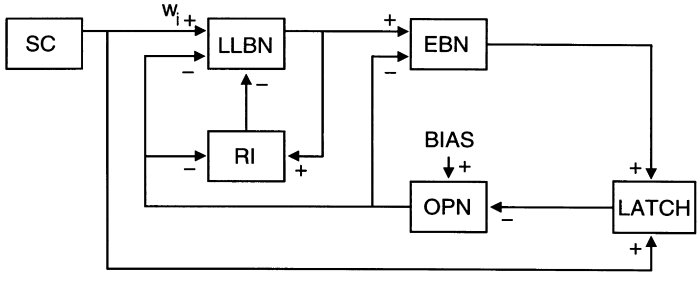

Fig. 17. Moschovakis (1994) model of the saccade generator. The Moschovakis (1994) model is a vector type model in which the LLBN receives input from the SC, and is inhibited by a resetable integrator (RI). This RI cell is strongly inhibited by the OPN in the model, which has an excitatory bias. The OPN is inhibited by a latch cell. This latch cell is strongly excited by the EBN, and weakly excited by the SC.

weak, then the latch cell may not become sufficiently active to inhibit the OPN, or saccades will be produced at unnaturally long latencies. For this reason, there cannot be a weight between the SC and the latch cell in the Moschovakis (1994) model, since for large saccades, the latch cell would get too strong an input, but for small saccades too weak an input. Thus, the Moschovakis (1994) model needs a second, extra connection from the SC which must have a precise connection strength. Further, other areas which can control the SG in the absence of the SC, such as the FEF, must also have this added connection.

In FOVEATE, the latch cell, the secondary connection from the SC, and the resetable integrator — none of which has been experimentally found — are unnecessary. This is because the FOVEATE OPN is controlled by the LLBN, which occurs functionally before the EBNs, which are inhibited by the OPN. Input from the SC to the LLBN causes LLBN activity to increase, even if the OPN is initially fully active. Once LLBN activity increases, it inhibits the OPN, allowing a saccade to begin. As long as the feedback loop is able to zero the motor error, the OPN will be reactivated, independent of the size of the saccade. Thus, in addition to requiring fewer connections and cell types than the Moschovakis (1994) model, FOVEATE is also functionally more robust.

Although this article restricts its modeling to the cell types that exist within the saccade-controlling circuits of the reticular formation, we have recently embedded the FOVEATE circuit into a larger system for saccadic control, which linked the FOVEATE circuit to the superior colliculus, visual cortex, parietal and prefrontal cortex, and the cerebellum (Gancarz and Grossberg, 1997, 1998). This larger study demonstrates that the present saccade generator circuit is compatible with a wide range of additional data about the selection, planning, execution, and learning of saccadic eye movements. With regard to connections between the SC and the SG, Everling et al. (1998) have recently concluded that there are differences in the discharge properties of superior colliculus fixation neurons (SCFN) and OPNs that are irreconcilable with the hypothesis that the discharge pattern of OPNs reflects simply the excitatory input from SCFNs. They concluded that there are likely additional excitatory inputs to the OPNs. This result gives indirect support for the FOVEATE model hypothesis of arousal cells that keep the OPNs on even when the SCFNs shut down. It also shows that there must be other cells that shut the OPNs off; they are not just 'winding down' when SCFN input is removed. FOVEATE proposes that the LLBNs are these cells.

All vector models face a problem that must also be solved by position models; namely, after the tonic cells integrate signals from the motor error burst cells, how does the brain ensure that the saccade actually moves the fovea to the target? In Grossberg and Kuperstein (1986) and Gancarz and Grossberg (1997, 1998), it is shown how cerebellar learning can use the visual error signals that are caused by inaccurate saccades to adaptively modify the feedforward gains that project to the long-lead bursters, and thereby lead to accurate saccades.

In summary, we have shown that FOVEATE is consistent with data on saccadic staircases, interrupted saccades, straight oblique saccades, saccade velocity–duration tradeoffs, and high velocity smooth eye movements evoked by high level, prolonged SC stimulation. The model is able to explain these data without making any of the experimentally unsupported assumptions of earlier models.

Acknowledgements

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Appendix A Model equations

FOVEATE is described by differential equations for the membrane potentials of the different cell types in the SG. Each neuron equation was based on a classical membrane equation (Hodgkin, 1964; Grossberg, 1973, 1982):

$$C_{\mathrm{m}}\frac{\mathrm{d}V(t)}{\mathrm{d}t} = -[V(t)-E_{\mathrm{excit}}]g_{\mathrm{excit}}(t) - [V(t)-E_{\mathrm{inhib}}]g_{\mathrm{inhib}}(t) - [V(t)-E_{\mathrm{leak}}]g_{\mathrm{leak}} \quad \text{(A1)}$$

where the parameters E represent reversal potentials, g_{leak} is a constant leakage conductance, and the time varying conductances $g_{\mathrm{excit}}(t)$ and $g_{\mathrm{inhib}}(t)$ represent the total excitatory and inhibitory inputs to the cell. The $V(t)$ terms that multiply these conductances in Eq. (A1) represent *shunting* interactions that realize automatic gain control properties of the cell.

Eq. (A1) can be rescaled to have a zero passive equilibrium point, instead of E_{leak}, by writing it in terms of a new variable $W(t) = V(t) - E_{\mathrm{leak}}$. Thus E_{leak} can be set equal to zero in Eq. (A1) without loss of generality.

In cells where $V(t)$ does not get too close to the reversal potentials E_{excit} and E_{inhib}, the automatic gain control terms can be ignored, leading to an *additive* equation that uses a sum of excitatory input, inhibitory input, and passive decay terms instead of the right-hand side of Eq. (A1). All equations in FOVEATE use either the additive or shunting versions of Eq. (A1).

The equations were numerically integrated using fourth order Runge–Kutta with a fixed step size of 0.001.

Equations are given for horizontal saccade control. The simulated model also included a second circuit for control of vertical movement. The equations and parameters for the vertical circuit are the same as those for the horizontal circuit given below and are thus omitted. Both the horizontal and the vertical circuits shared a single OPN. The OPN equation given shows how both models are joined. In the following equations, the subscripts l and r refer to the left and right side of the horizontal circuit, respectively. Activations were bounded from below at zero. A unit interval of simulation time was set equal to 50 ms of real world time. Parameters were chosen to best fit the data. However, the basic model properties are robust to parameter choice.

Appendix A.1 Long-lead burst neurons

Long-lead burst neuron (LLBN) activity for the left and right side of the SG is represented by the variables L_{l} and L_{r}. LLBNs receive excitatory input I. They receive inhibitory feedback from the ipsilateral IBNs (B). LLBNs are leaky integrators with a passive decay rate of 1.3 (the $-1.3L$ term):

$$\frac{\mathrm{d}L_{\mathrm{l}}}{\mathrm{d}t} = -1.3L_{\mathrm{l}} + I_{\mathrm{l}} - 2B_{\mathrm{l}} \tag{A2}$$

and

$$\frac{\mathrm{d}L_{\mathrm{r}}}{\mathrm{d}t} = -1.3L_{\mathrm{r}} + I_{\mathrm{r}} - 2B_{\mathrm{r}} \tag{A3}$$

Appendix A.2 Excitatory burst neurons

The EBNs (E) receive excitatory input from the ipsilateral LLBNs, and from an arousal signal equal to 1. They are inhibited by the contralateral LLBN, and by the OPN (P). They are leaky integrators with a passive decay rate of 3.5:

$$\frac{\mathrm{d}E_l}{\mathrm{d}t} = -3.5E_{\mathrm{l}} + (2 - E_{\mathrm{l}})(5L_{\mathrm{l}} + 1) - (E_{\mathrm{l}} + 1)(10L_{\mathrm{r}} + 20g(P)) \tag{A4}$$

and

$$\frac{\mathrm{d}E_{\mathrm{r}}}{\mathrm{d}t} = -3.5E_{\mathrm{r}} + (2 - E_{\mathrm{r}})(5L_{\mathrm{r}} + 1) - (E_{\mathrm{r}} + 1)(10L_{\mathrm{l}} + 20g(P)) \tag{A5}$$

Appendix A.3 Inhibitory burst neurons

Inhibitory burst neurons (B) are excited by the ipsilateral EBNs, and are leaky integrators that decay at a rate of 2.4. They send inhibitory feedback to the ipsilateral LLBNs:

$$\frac{\mathrm{d}B_l}{\mathrm{d}t} = -2.4B_{\mathrm{l}} + 3E_{\mathrm{l}} \tag{A6}$$

and

$$\frac{\mathrm{d}B_{\mathrm{r}}}{\mathrm{d}t} = -2.4B_{\mathrm{r}} + 3E_{\mathrm{r}} \tag{A7}$$

Appendix A.4 Omnipause neurons

The OPNs (P) are tonically on, except when inhibited by LLBN activity. The OPNs are leaky integrators, with a decay rate of 0.2, that are activated by a tonic excitatory arousal input of 1.2 which, due to the shunting term $(1 - P)$, drives it close to saturation. Inhibition from the LLBNs passes through a sigmoid signal function $g()$, which is calibrated so that a single active inhibitory $g()$ term from the LLBNs can silence the OPN. The inhibitory effect of LLBNs from the vertical circuit on the OPN is shown by the terms L_{vu} (LLBN vertical up) and L_{vd} (LLBN vertical down). The excitatory term J represents external electrical stimulation where such occurs in an experiment (see Section 3.6):

$$\frac{\mathrm{d}P}{\mathrm{d}t} = -0.2P + (1 - P)(1.2 + J) - 3.5(P + 0.4)(g(L_{\mathrm{l}}) + g(L_{\mathrm{r}}) + g(L_{\mathrm{vu}}) + g(L_{\mathrm{vd}})) \tag{A8}$$

Appendix A.5 Tonic neurons

The TNs (T) integrate their input at a rate of 0.1. They are excited by ipsilateral EBNs, and inhibited by contralateral EBNs via IBN interneurons:

$$\frac{\mathrm{d}T_{\mathrm{l}}}{\mathrm{d}t} = 0.1(E_{\mathrm{l}} - E_{\mathrm{r}}) \tag{A9}$$

and

$$\frac{\mathrm{d}T_{\mathrm{r}}}{\mathrm{d}t} = 0.1(E_{\mathrm{r}} - E_{\mathrm{l}}) \tag{A10}$$

Appendix A.6 Signal function

A sigmoid signal function was used of the form

$$g(x) = \frac{x^4}{0.1^4 + x^4} \tag{A11}$$

The parameter 0.1 determines the value of x at which the function $g(x)$ equals 0.5, and the exponent 4 controls the sharpness of the sigmoid. The function is 0 for x equal to 0, and approaches 1 for large values of x.

Appendix A.7 Eye position

Horizontal eye position (θ) was assumed to be proportional to tonic cell activity. The value 0.5 represents tonic cell activity when the eye is in the center of its range:

$$\theta = 260(T_{\mathrm{r}} - 0.5) \tag{A12}$$

Modeling the eye muscle plant was not necessary to simulate the data reported herein.

Appendix A.8 SC activity

Activity A of an idealized SC cell has a decay rate of 1,

and is excited by electrical stimulation frequency F:

$$\frac{dA}{dt} = -A + F \tag{A13}$$

Appendix A.9 Signal function between SC and SG

SC cell activities lead to saturating output signals $f(x)$:

$$f(x) = \begin{cases} 0 & : \quad \text{if } x < 0 \\ x & : \quad \text{if } 0 < x < 1 \\ 1 & : \quad \text{otherwise.} \end{cases} \tag{A14}$$

Appendix A.10 Input to SG

The output signal $f(A)$ is multiplied by a weight (W) which scales the input (I) to the SG, and thus the amplitude of the saccades:

$$I = Wf(A) \tag{A15}$$

In the saccadic staircase simulation (Fig. 3), input I to the left side of the SG was set equal to 1 for 265 ms. For the amplitude and duration simulation (Fig. 5), input I was set equal to 1, 1.75, and 2.5; in each case for 85 ms. In the straight oblique simulation (Fig. 6), inputs I to the horizontal and vertical circuits were: (0.67, 0.08), (0.7, 0.22), (0.74, 0.4), (0.75, 0.6), (0.7, 0.9). These inputs were left on for 75 ms. In the oblique staircase simulation (Fig. 7), inputs I to the horizontal and vertical circuits were held at (0.2, 0.33) for 250 ms. In the cardioid simulation (Fig. 8), net EBN burst activity was calculated during saccades in response to the following inputs to the horizontal and vertical circuits: (0, 0, 0.7, 0), (0, 0.45, 0, 0.45), (0, 0, 0, 0.7), (0.45, 0, 0, 0.45), (0.7, 0, 0, 0), (0.2, 0, 0, 0.63), (0.63, 0, 0, 0.2), (0, 0.2, 0, 0.63). The input was on for 50 ms. The EBN activity sums were normalized for plotting. To produce the velocity saturating after amplitude simulation (Fig. 9), SC stimulation frequency F was varied between 1 and 2.4 at increments of 0.2. The weight W was set equal to 2, and stimulation duration was 125 ms. For the simulation of Fig. 10, W was set equal to 2. For the high velocity trial (dotted line), stimulation frequency F was 3, and stimulation duration was 82 ms. For the slower velocity trial (solid line), F was 1.3, and stimulation duration was 117 ms. In the smooth staircase simulation (Fig. 11), I was set to 3 for 300 ms. In the interrupted saccade simulation (Fig. 12, solid line), I was set to 0.7 for 100 ms. OPN stimulation J was set to 1.8 for 5 ms. For the uninterrupted saccade (dotted line), I was set to 0.7 for 100 ms.

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