CHAPTER 7
SACCADE GENERATOR AND SACCADE RESET

7.1. Saccade Generator

In Section 2.5, we described learning requirements that suggest the need for a saccade generator (SG). An SG must be capable of shutting off its own output signal, thereby terminating a saccadic movement, while its input signal remains on. In this chapter, we will describe some implications of the existence of an SG. Robinson and his colleagues (Keller, 1981; Raybourn and Keller, 1977; Robinson, 1981) have contributed substantially to this problem. We will discuss how general properties of the SG impose new design constraints on networks that input to the SG. A detailed SG design must be also developed, however, in order to clearly state the properties of these input sources and how they may interact with SG cells.

7.2. Converting an Intensity Code into a Duration Code

In order for the SG output to shut off before its input shuts off, some measure of output size must accumulate and act as an inhibitory signal that counteracts the input. The net SG output must, moreover, change as a systematic function of SG input, not only to generate saccades in response to unconditioned inputs, but also to alter a saccade as the conditioned input corresponding to a fixed unconditioned input changes due to learning. In our discussions of both unconditioned inputs (Chapter 2) and conditioned inputs (Chapter 3), larger SG inputs are required to generate larger saccadic movements.

At first glance, it would appear that two qualitatively different types of circuits could, in principle, accomplish this goal (Grossberg, 1970, Section 3). In one circuit, a feedforward inhibitory interneuron v_1 accumulates the activity that counteracts the input at v_2 (Figure 7.1a). In the other circuit, a feedback inhibitory interneuron v_2 accumulates the activity that counteracts the input at v_1 (Figure 7.1b). These two designs lead to significantly different predictions about the input circuits that activate the SG. A feedforward inhibitory interneuron cannot, however, effectively carry out the required task. This is because the range of output sizes that a feedforward inhibitory interneuron can accomodate is insufficient for SG purposes. Either smaller inputs could not be shut off at all, which is unacceptable, or the total range of output sizes would be much too restricted. To mathematically prove these assertions herein would take us too far from our charted course. They are implied by Theorem 1 in Grossberg (1970, p.297). We therefore consider the simplest version of a feedback inhibitory interneuron (Figure 7.1b).

In such a circuit, some measure of output size accumulates and feeds back as an inhibitory signal that counteracts the input. Since the functional role of the SG output is to move the eye to its target position, the simplest accumulating measure of output size is the outflow signal itself.

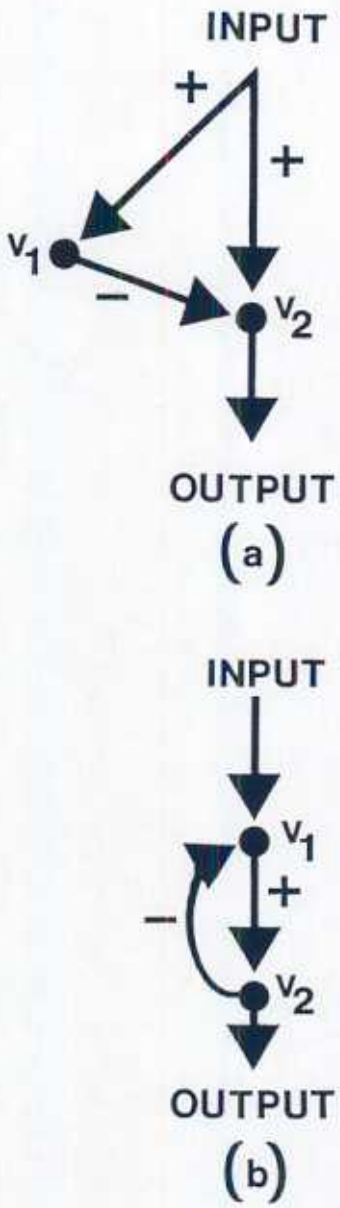


Figure 7.1. (a) A feedforward inhibitory interneuron v_1 inhibits the excitatory effect of the input at v_2; (b) When v_2 is excited by v_1, it feeds inhibitory signals back to v_1, thereby tending to inhibit the excitatory effect of the input upon v_1.

This argument suggests an agonist interaction like that depicted in Figure 7.2. In Figure 7.2, an input to burst (B) cells generates signals to the tonic (T) cells. The T cells integrate this input through time. This integrated T cell activity generates outflow signals to the motoneurons (MN), thereby causing them to move. In addition, the T cells generate an inhibitory feedback signal to the B cells and an excitatory feedforward signal to the MN cells. As this inhibitory signal builds up due to the integrative action of the T cells, it progressively weakens the excitatory effect of the input on the B cells. When the inhibitory T cell feedback completely cancels the input, the B cells shut off (hence their name), thereby terminating SG input to the T cells and allowing the saccade to end.

To see how this kind of network can work, consider the simple model

$$\frac{d}{dt}x = -Ax + I - By \tag{7.1}$$

and

$$\frac{d}{dt}y = f(x) \tag{7.2}$$

where $x(t)$ is the activity of the model B cells, $y(t)$ is the activity of the model T cells, and I is the input size. Suppose that $f(x)$ is a nonnegative increasing function such that $f(0) = 0$. Also suppose that $f(x)$ has a finite maximum, say 1, and that most inputs I which perturb $x(t)$ are large enough to drive $f(x)$ close to its maximum. Then when I turns on, $x(t)$ is activated and starts to grow. Function $f(x)$ rapidly grows to 1. By (7.2), $y(t)$ integrates the value 1 through time, hence grows (approximately) linearly with t. Thus $y(t) \cong t$. The net input $I - By$ to the B cells in (7.1) therefore satisfies

$$I - BY \cong I - Bt. \tag{7.3}$$

Thus the net input becomes zero at (approximately) time

$$t_I = \frac{I}{B}. \tag{7.4}$$

Thereafter the decay rate $-A$ of $x(t)$ and the negativity of $I - By$ drive $x(t)$ to zero.

The critical property of this circuit is that $y(t)$ integrates the (approximate) input 1 for a time interval of (approximately) I/B in duration, before $x(t)$ shuts off its input to $y(t)$. Thus the tonic outflow signal increases at an (approximately) constant rate for a duration that increases (approximately) linearly with the input intensity I. These linear properties break down at the small input sizes I such that $f(x)$ does not equal 1, and at the transitional times when $x(t)$ is increasing from equilibrium and decreasing back to equilibrium. Despite these caveats, it is clear that

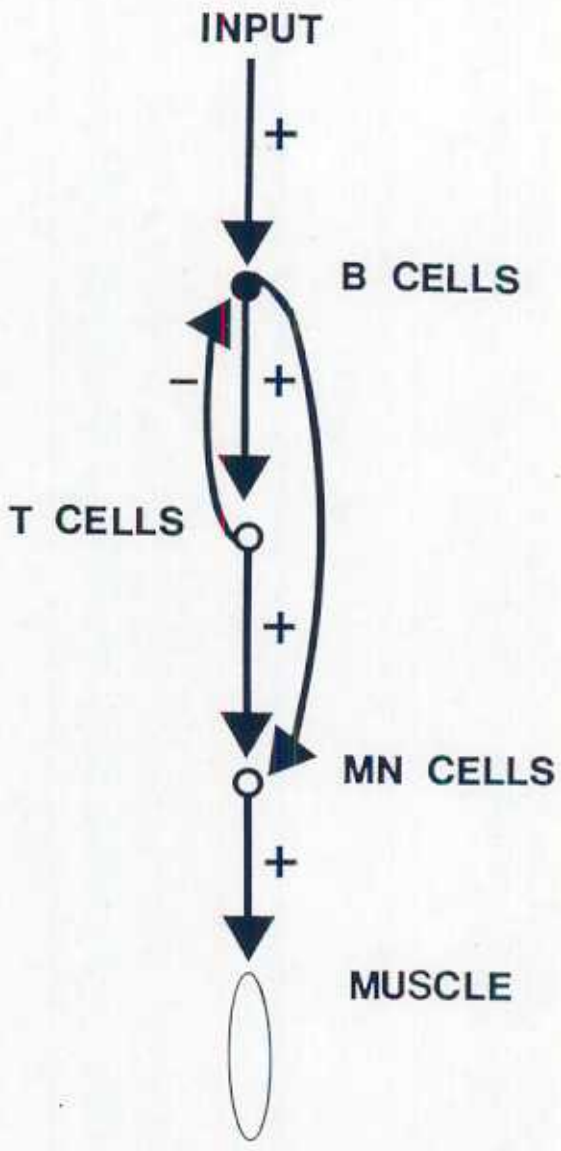


Figure 7.2. A simple saccade generator (SG) circuit: The input excites burst cells (B) which, in turn, excite tonic cells (T) and motoneurons (MN). The T cells inhibit the B cells. In such a circuit, the duration of B cell activity increases with the intensity of the input. (Filled circles are phasically active cells. Open circles are tonically active cells.)

such a system can generate movement signals that expand and contract in a regular way as the input increases and decreases over an unlimited range. In other words, such a system can convert an *intensity* code (I) into a code for temporal *duration* (t_I). Using such a code, the duration of a saccade should covary with the duration of burst cell activity. This is also true in the data (Luschei and Fuchs, 1972).

7.3. Summation of Retinotopic and Initial Eye Position Signals to the Saccade Generator

We can now draw several important conclusions about the inputs to the SG. By Chapter 5, T cell outputs generate the outflow signals that determine the position of the eyes in the head. Thus, after an accurate saccade terminates, the T cell feedback to the SG encodes the target position of the eyes. Since this T cell feedback signal cancels the total input to the SG, the total input to the SG also encodes the target position of the eyes.

We concluded in Chapters 3 and 4, by contrast, that the movement commands to the SG are computed in retinotopic coordinates. Retinotopic commands from the visually reactive movement system are derived from a retinotopic map (RM) that is activated by retinal lights (Chapter 3). Retinotopic commands from the intentional and predictive movement systems are derived from an RM that is activated by difference vectors (Chapter 6). We are hereby led to articulate the following important design problem: How can retinotopic commands be converted into target position commands before activating the SG?

The following conclusion now seems inevitable: at the SG, an RM adds its movement signal to a signal from an eye position map (EPM). In order for the sum of RM and EPM signals to encode the target position of the eye, the EPM signal must encode the initial position of the eye before the saccade begins.

7.4. The Eye Position Update Network

The primary functional role of the EPM map is to form part of a network that updates initial eye position signals to the SG after each saccade terminates. We call this network the *Eye Position Update Network* (EPUN). The existence of an EPUN is implied by the fact that T cell feedback shuts off the SG: After a correct saccade terminates, the T cell feedback cancels the total SG input. In order to prepare for the next saccade, the RM command that encoded the previous saccade command must be shut off, so that a new RM command can replace it. By the time that the new RM command is instated, a new EPM command must also be activated, so that the total RM + EPM command can encode the new target position. The new EPM command encodes, as always, the initial eye position. However, this new *initial* eye position is the *target* eye position of the previous saccade.

This summary suggests that the following sequence of events occurs between two successive saccades:

$$RM_1 + EPM_1 \rightarrow EPM_1 \rightarrow EPM_2 \rightarrow RM_2 + EPM_2. \qquad (7.5)$$

In (7.5), $RM_1 + EPM_1$ stands for the total SG input that caused the first saccade. After the saccade is over, the RM_1 input shuts off. This event does not cause a new saccade to occur because the T cell feedback, having cancelled the total input $RM_1 + EPM_1$, can also cancel the EPM_1 input alone. Then the eye position input is updated by the new initial position input EPM_2. The input EPM_2 approximately matches the T cell feedback, hence does not cause a new saccade. Finally, a new RM_2 input is activated. If the T cell feedback cancels the EPM_2 signal, then the RM_2 input generates a burst within the SG whose size depends upon the RM_2 command alone.

Some variation on the timing of the reset events in (7.5) is also consistent with functional requirements. For example, both RM_1 and EPM_1 may be simultaneously reset. It is not, however, permissible for EPM_1 to be reset before RM_1 is reset. Then the new total command $RM_1 + EPM_2$, which approximately equals $2RM_1 + EPM_1$, could elicit a spurious saccade in response to the increment RM_1.

7.5. Two Types of Initial Position Compensation: Eye Position Update and Muscle Linearization

This discussion suggests that both the RM and the EPM activate conditionable pathways through the adaptive gain (AG) stage that contribute to the total SG input. We already know that the RM must activate such a conditionable pathway (Chapter 3). The conclusion that the EPM also controls a conditionable pathway follows from the possibility that the size scale of the EPM signals is initially either much too large or much too small to balance the T cell feedback that is registered before and after saccades. Unless this calibration problem is solved, the conditionable RM pathway would have to compensate both for initial position errors of the unbalanced EPM and T cell inputs and for saccadic foveation errors. By contrast, if both the RM and the EPM control conditionable pathways, then they can cooperate to correct initial position errors and saccadic foveation errors.

One of the major lessons of Chapter 3 was to show how multiple sampling maps can cooperate to generate the total adaptive gain needed to correct saccadic foveation errors. Since both the RM and the EPM can activate conditionable pathways, they could, in principle, share the adaptive load to correct saccadic foveation errors, as in equations (3.34) and (3.35). Under normal circumstances, the MLN obviates much of the need for such cooperation by directly linearizing the response of the muscle plant, thereby enabling the RM to absorb most of the adaptive load needed to correct saccadic errors, as in equations (3.25) and (3.26).

The analysis in Chapter 5 suggests experimental tests of whether the EPM controls a conditionable pathway. Suppose, for example, that certain pathways from cerebellar direct response cells to MN cells carry the conditioned signals that linearize muscle responses (Section 5.3). If these pathways of the MLN were cut, then dysmetria should be caused due to the subsequent nonlinear muscle responses. As a result, the corollary discharges to the HMI would inaccurately encode the actual positions of the eyes. Any movement commands based upon HMI vectors would consequently lead to saccadic errors for two reasons: The vectors themselves would be incorrectly calibrated, and the total input to the SG would not compensate for muscle nonlinearity. By contrast, visually reactive saccades due to retinal lights would lead to saccadic errors for only one reason: They would not be erroneous due to incorrectly calibrated vectors. They would be erroneous only due to the nonlinearity of the muscle plant.

If such visually reactive saccades could be evoked sufficiently often, the adaptive cooperation of RM and EPM signals at the SG should substantially correct the dysmetria. By contrast, adaptation to vector-based RM commands might show less correction, because the corollary discharges from which the vectors are computed would continue to be miscalibrated. Ablating the frontal eye fields can cause only visually reactive saccades to occur, whereas ablating the superior colliculus can cause only intentional and predictive saccades to occur (Schiller, Sandell, and Maunsell, 1984). Such lesions, in concert with lesions of the pathway from cerebellar direct response cells to MN cells, may therefore be useful to test whether the EPM input to the SG is conditionable.

Another test of EPUN properties can be made by using paradigms in which saccade staircases are generated.

7.6. Saccade Staircases

The EPUN helps to explain saccade staircases of the type found by Schiller and Stryker (1972). In their experiments, a sustained electrode input to the deep layers of the superior colliculus caused a series of saccades of equal size. The EPUN design explains the existence of saccade staircases as follows.

The electrode input initially adds an RM input to the total SG input. A saccade is hereby generated. This RM input does not turn off, because the electrode does not turn off. On the other hand, after the saccade is over, the EPM input is updated, as in (7.5). The EPM input hereby increases by an amount corresponding to the length of the saccade. This new EPM input cancels the new level of T cell feedback. Hence the extra RM input due to the sustained electrode input can cause a second saccade. This argument can clearly be iterated to conclude that a saccade staircase will occur.

An intriguing feature of this explanation emerges when we ask why all the saccades in the saccade staircase are of approximately equal size. In Section 4.9 we noted that one factor is the *absence* of initial eye position compensation from the HMI. We have just noted that the staircase *per se* is

due to the *presence* of initial eye position compensation from the EPUN. Further argument is needed to explain the equal sizes of the saccades. Equal increments in total input to the SG do not imply equal saccade sizes if the muscle plant is nonlinear. However, the muscle plant is functionally linear due to the action of the MLN. A series of equal saccades is the result

This explanation shows that appropriate properties of three adaptive circuits (VCN, EPUN, and MLN) are needed to reconcile the properties of saccadic staircases with the greater body of saccadic data. Suitable manipulations in any of these circuits should therefore yield predictable changes in the properties of saccadic staircases. For example, cutting the cerebellar direct response pathways that are assumed to linearize muscle response in the MLN is predicted to elicit saccades of unequal sizes in a saccade staircase. Preventing the reset of the neurons that are assumed to update the EPM in the EPUN is predicted to cause just one saccade to be generated. This latter property can be used to discover what brain region houses this EPM.

Our explanation of saccadic staircases implies that during normal adult saccades, the RM input is shut off after the saccade is over. Any mechanism that prevents this reset event can elicit saccadic staircases. Perhaps saccadic staircases in infants (Salapatek *et al.*, 1980) are due to the fact that (cortical) mechanisms have not yet developed their ability to reset stored retinotopic commands after a nonfoveated target is shut off. Since the sizes of the saccades in these staircases are approximately equal, the more primitive MLN has presumably already developed by this developmental stage.

7.7. Circuit Design of the Eye Position Update Network

The EPUN is built up by using many of the same design principles as the RCN (Chapter 3), VCN (Chapter 4), and the MLN (Chapter 5). This fact illustrates that, once a network module has been synthesized by the evolutionary process, it may be specialized in many ways by hooking it up to different input and output pathways. The EPUN network is designed as follows.

As in the MLN, T cell activity is mapped into an EPM which sends sampling signals to the AG stage (Figure 7.3). Instead of using the OII-generated error signals of the MLN at the AG stage, the EPUN uses the second light error signals of the RCN and VCN. As in the RCN and VCN, the conditionable pathways from the AG stage project to the SG.

Both retinal lights and HMI vectors are encoded within an RM before a saccade begins and are stored in STM there until after the saccade is over. In this way second light error signals can correct foveation errors due to the RM command (Chapter 2). The hypothesis that RM + EPM signals cooperate at the SG leads to the suggestion that the EPM is activated using similar temporal rules: The T cell input to the EPM is encoded and stored in STM before a saccade begins, and is not reset until after the saccade is over. The EPM thus represents *initial* eye position right after the saccade ends, and the total RM + EPM input represents target position.

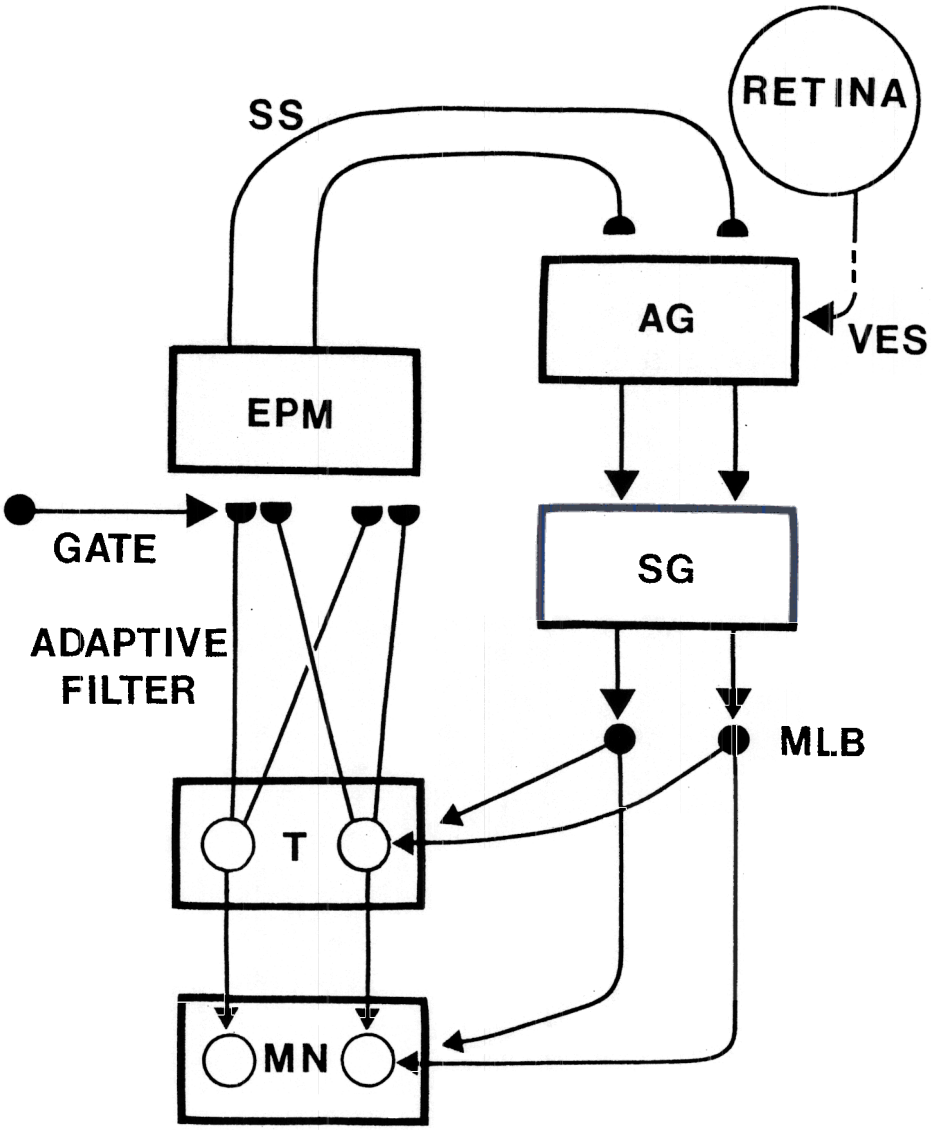


Figure 7.3. An eye position update network (EPUN): This network updates the present eye position signal to the saccade generator (SG) after a saccade is over. Due to this network, the retinotopically coded saccadic commands computed by the retinotopic command network (RCN) can activate the SG by the correct amount to foveate a target light. Abbreviations: T = tonic cells, MN = motoneurons, EPM = eye position map, AG = adaptive gain stage, VES = visual error signals, MLB = medium lead bursters.

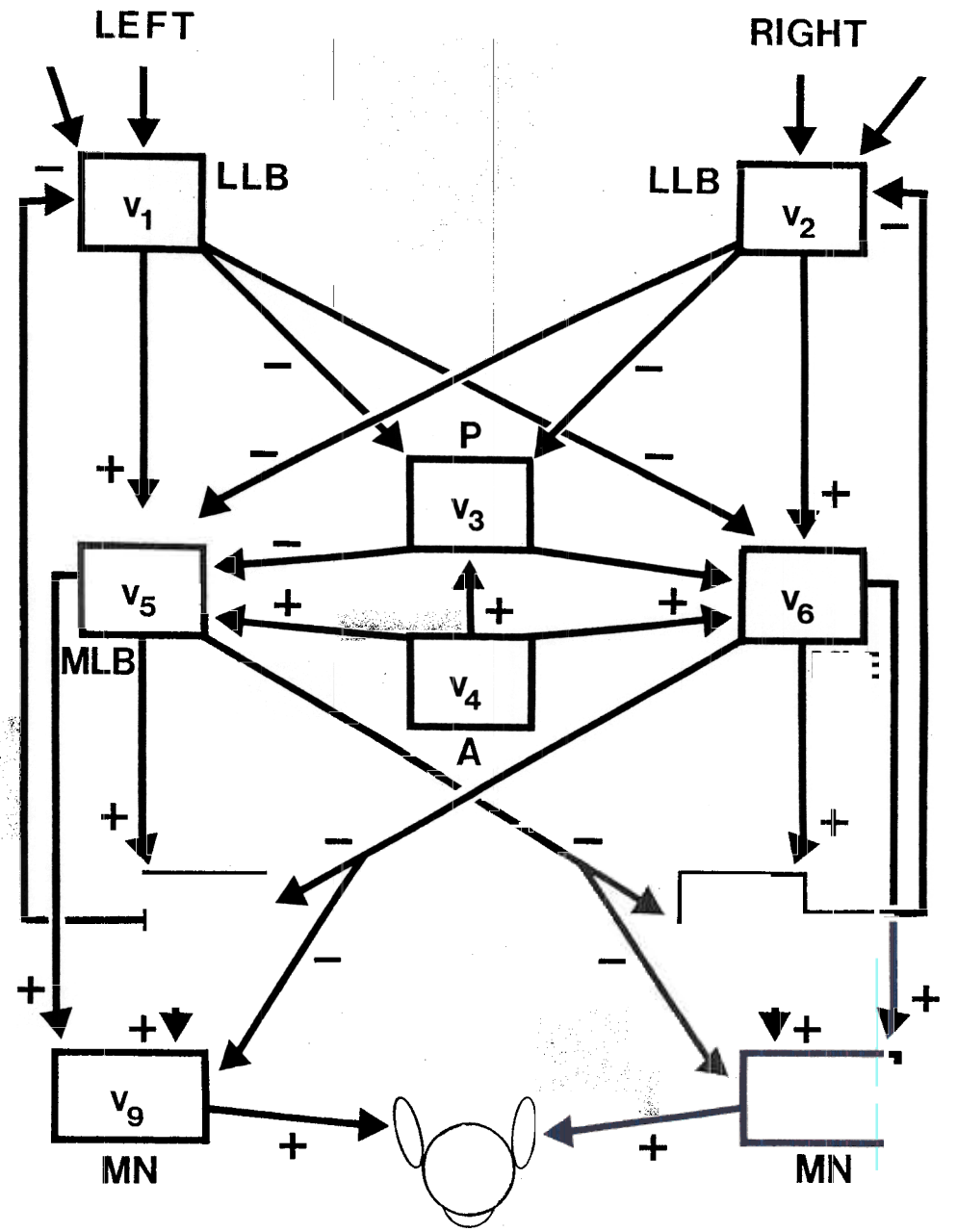
Thus any second light error signal that the saccade might generate can correct the cooperating conditionable pathways of this composite target position command.

The discrete sampling of T cell signals by the EPM is controlled by some type of gating action, because the T cells are tonically active and are thus always on. Several functional variations on this gating concept are readily imagined, and await future data to select among them. For example, a pause cell, or cell that is on except during a saccade (Section 7.8), could provide an excitatory gating action either at the synaptic knobs of T cell pathways to the EPM (Figure 7.3) or at the axon hillocks of these pathways. In the former case, the gating action could also modulate the plasticity of the adaptive filter which maps T cell outputs into EPM positions (Chapter 6). Alternatively, a burst cell, or cell that is on only during a saccade, could provide an inhibitory gating action at synaptic knobs or axon hillocks. Neural data which may provide interpretations of EPUN cells are reserved until Chapter 11, where we will also make several predictions about movement and postural subsystems.

7.8. A Saccade Generator Circuit

In this section, we describe a more complex SG model than the simple feedback inhibitory circuit of Figure 7.2. This circuit utilizes cell types such as pause cells, two types of burst cells, tonic cells, and motoneurons that will play a role in our later discussions. Data concerning such cell types were described by Luschei and Fuchs (1972). The circuit illustrates how agonist and antagonist movement signals can work together in a push-pull fashion. In particular, antagonist bursts can occur near the end of a saccade and isometric coactivation of medium lead bursters can occur when a saccade moves in a direction perpendicular to the preferred axis of the bursters (van Gisbergen, Robinson, and Gielen, 1981). The circuit also has the property that its accuracy can vary with the network's level of arousal, notably that saccadic undershoots can occur in a fatigued or insufficiently aroused state, as in the data of Bahill and Stark (1979). In our SG circuit, the last three properties are all explained by the same mechanism.

Figure 7.4 depicts how such a model SG circuit controls a left-right pair of mutually antagonistic muscles. To describe this circuit's properties, we consider how cells that contract the right muscle interact with each other and with the other cells of the network. A total RM + EPM input is received by the long-lead burster (LLB) population v_2. Population v_2 sends excitatory signals to the medium-lead burster (MLB) population v_6. Population v_6 also receives powerful inhibitory signals from the pauser (P) population v_3. The LLB cells cannot supraliminally activate the MLB cells until the P cells shut off. The inputs to the LLB cells can build up gradually through time and over spatially dispersed populations that can receive inputs from several sources of RM and EPM inputs. When the total LLB input reaches a critical size, the LLB population can directly inhibit the P cells v_3, and thereby indirectly disinhibit the MLB cells v_6.


LEFT
RIGHT
LLB
LLB
v_1
v_2
P
v_3
v_5
v_6
MLB
v_4
A
v_9
MN
MN

The gradual and spatially dispersed build-up of input to the LLB cells is hereby translated into a rapid and focussed onset of activity at the MLB cells.

The MLB cells activate their tonic (T) cells v_8 and their motoneuron (MN) cells v_{10}, also called burst-tonic cells. The T cells v_8 integrate their inputs, and relay this integrated activity to the MN cells v_{10}. Thus the MN cells receive integrated MLB input from the T cells, as well as direct MLB inputs. The MN cells, in turn, contract the right muscle. The direct MLB inputs enable their MN cells to generate a large initial signal to the eye muscles. This initial burst can overcome muscle inertia and any changes in total muscle input that may occur during the transition from posture to movement (Chapter 8). The T cell input, on the other hand, helps the eye to attain its final position and to maintain this position after the saccade is over. The T cells also generate the inhibitory feedback signals to the LLB cells which terminate the saccade, as in Figure 7.2.

Due to inhibitory feedback from the T cells, recording from the LLB cells will not easily disclose the existence of inputs from an eye position map (EPM), as in equation (7.5). This is because the T cell feedback tends to cancel the EPM input to the LLB cells. Inhibition of the T cell input should unmask the EPM input contribution to the LLB cells in such a circuit.

Activation of the right muscle circuitry also affects the left muscle circuitry. In order to understand how this happens in the circuit of Figure 7.4, we must first explain how the arousal (A) cells v_4 work. These A cells control the generalized "excitability" of the entire circuit. In particular, population v_4 determines the tonic activation level of the P cells v_3. The A cells also generate inputs to the MLB cell populations v_5 and v_6. The balance between A cell excitation and P cell inhibition keeps the MLB cells off between saccades. When the P cells v_3 are turned off by inputs from the LLB cells v_2, the unmasked input from the A cells v_4 combines with LLB input at v_6 to provide a strong onset of MLB activation. The primary function of the A cell population is thus to bring the size of MLB activation by unconditioned LLB inputs into a reasonable range at an early developmental stage, such that later conditioned changes in LLB inputs can correct any remaining errors of saccade length and direction.

When an input to the LLB cells v_2 initiates a saccade, the v_2 cells also inhibit the MLB cells v_5 of the left muscle. This inhibitory signal to v_5 cancels the excitatory signal from the A cells v_4 after the P cells v_3 are shut off. Thus the LLB cells v_2 simultaneously inhibit the P cells v_3 and the antagonistic MLB cells v_5 using broadly distributed off-surround pathways.

This hypothesis has important experimental implications. The antagonist MLB cells v_5 are shut off at the beginning of a saccade by the agonist LLB cells v_2. However, as the saccade progresses, the inhibitory feedback from the agonist T cells v_8 gradually shuts off the LLB cells v_2. As a result, LLB inhibition of the MLB cells v_5 gets progressively smaller, until the v_5 cells receive a net excitatory input from the A cells v_4. In other

words, the MLB cells v_5 experience an *antagonistic burst* towards the end of a saccade, as also occurs *in vivo* (van Gisbergen, Robinson, and Gielen, 1981).

The hypothesis that an antagonistic burst may be due to disinhibition of a tonic arousal signal has not been made in other SG models. Should it prove to be false, the A cells could be removed without destroying most of the circuit's properties. An antagonistic burst could also, for example, be due to weak coactivation of antagonist LLB cells during an agonist saccade. This hypothesis can be tested in several ways. For example, the circuit in Figure 7.4 predicts that the maximal size of this antagonistic burst in v_5 varies monotonically with the maximal size of the agonist burst in v_6, and that both maximal sizes should decrease during drowsy states that cause saccadic undershoots.

In order to achieve a push-pull effect at each antagonistic muscle pair, the MLB cells v_6 inhibit the antagonist T cells v_7 and the MN cells v_9 when they excite their own T cells v_8 and MN cells v_{10}. This type of push-pull effect has been reported at abducens MN cells by van Gisbergen, Robinson, and Gielen (1981). These authors also reported that burst neuron discharge rate closely followed the difference between target position and eye position signals during a saccade, thereby providing strong evidence for their hypothesis that T cell inhibition shuts off the saccade-generating burst.

7.9. Computer Simulations of a Saccade Generator Model

Figure 7.5 describes computer simulations of a model circuit of the type depicted in Figure 7.4. For completeness, we also summarize the differential equations that were used in this simulation. In order to simplify the model and to minimize the number of free parameters, we chose parameters equal to 1 and linear signal functions wherever possible.

Long Lead Bursters

$$\frac{d}{dt}x_1 = -x_1 + I_1 - x_7 + x_7(0) \tag{7.6}$$

$$\frac{d}{dt}x_2 = -x_2 + I_2 - x_8 + x_8(0) \tag{7.7}$$

Pausers

$$\frac{d}{dt}x_3 = -x_3 + x_4 - f(x_1) - f(x_2) \tag{7.8}$$

Arousal Cells

$$x_4 = \text{constant}$$

Medium Lead Bursters

$$\frac{d}{dt}x_5 = -x_5 + x_1 + x_4 - g(x_2) - g(x_3) \tag{7.9}$$

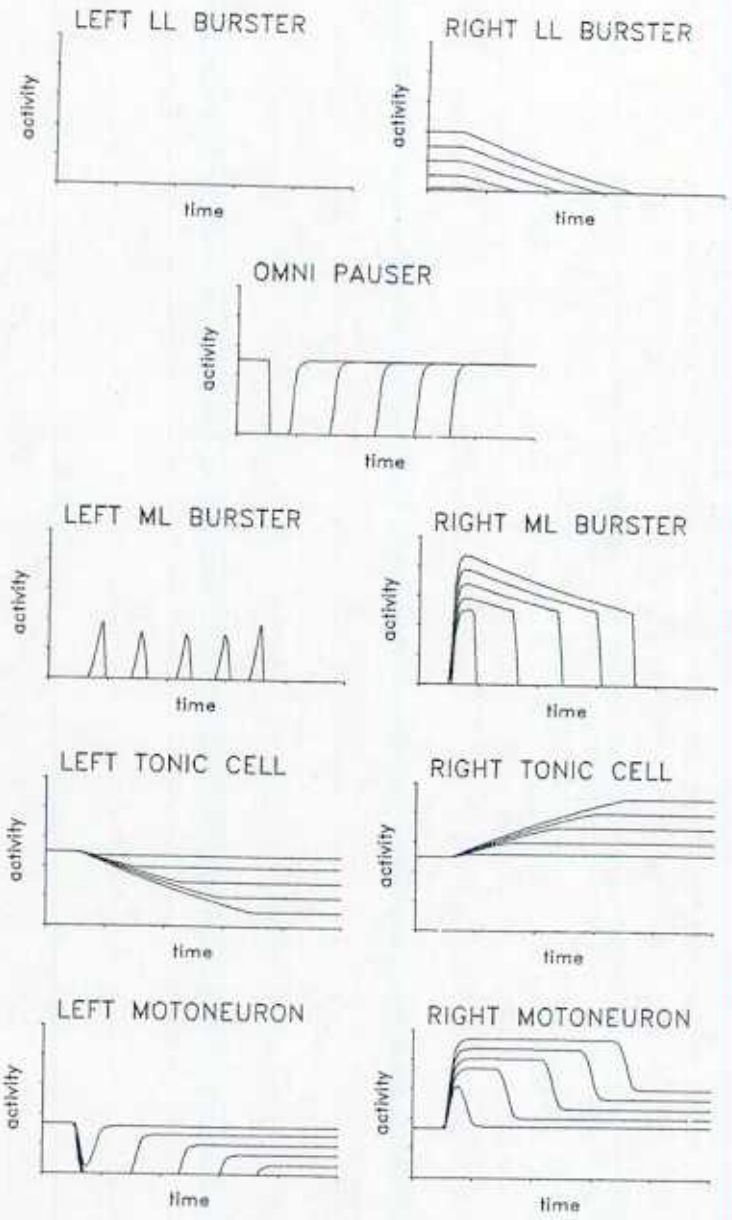


Figure 7.5. Computer simulation of a saccade generator (SG) circuit: The graphs depict the time evolution of cell activity in response to a series of constant inputs of increasing intensity to the right long lead burster population.

$$\frac{d}{dt}x_6 = -x_6 + x_2 + x_4 - g(x_1) - g(x_3) \tag{7.10}$$

Tonic Cells

$$\frac{d}{dt}x_7 = C(x_5 - x_6) \tag{7.11}$$

$$\frac{d}{dt}x_8 = C(x_6 - x_5) \tag{7.12}$$

Motoneurons

$$\frac{d}{dt}x_9 = -x_9 + x_5 - x_6 + x_7 \tag{7.13}$$

$$\frac{d}{dt}x_{10} = -x_{10} + x_6 - x_5 + x_8 \tag{7.14}$$

Initial data were chosen as follows:

$$x_1(0) = x_2(0) = x_5(0) = x_6(0) = 0 \tag{7.15}$$

$$x_3(0) = x_4(0) = x_7(0) = x_8(0) = x_9(0) = x_{10}(0) = \frac{1}{2} \tag{7.16}$$

Parameters, signal functions, and inputs were chosen as follows:

$$C = .01, \tag{7.17}$$

$$f(w) = \frac{w}{.001 + w}, \tag{7.18}$$

$$g(w) = \frac{w}{.02 + w}, \tag{7.19}$$

$$I_1 \equiv 0$$

$$I_2 = .02,\ .1,\ .2,\ .3,\ .4 \text{ on trials } 1 - 5.$$

In equation (7.6), term $x_7(0)$ is used to encode the EPM input corresponding to the target position attained by the previous saccade, which is hypothesized to equal the T cell inhibitory feedback from v_7 at the end of this saccade. Term $x_8(0)$ in equation (7.7) has a similar interpretation.

7.10. Comparison of Computer Simulations with Neural Data

Properties of model SG cells are similar to data on the response characteristics of various cell types in the deep layers of the superior colliculus, peripontine reticular formation, and oculomotor nuclei. Figure 7.6 schematizes the neural responses leading to a saccade. It highlights the main properties of the various cell types found in unit recordings by Luschei and Fuchs (1972) and Raybourn and Keller (1977).

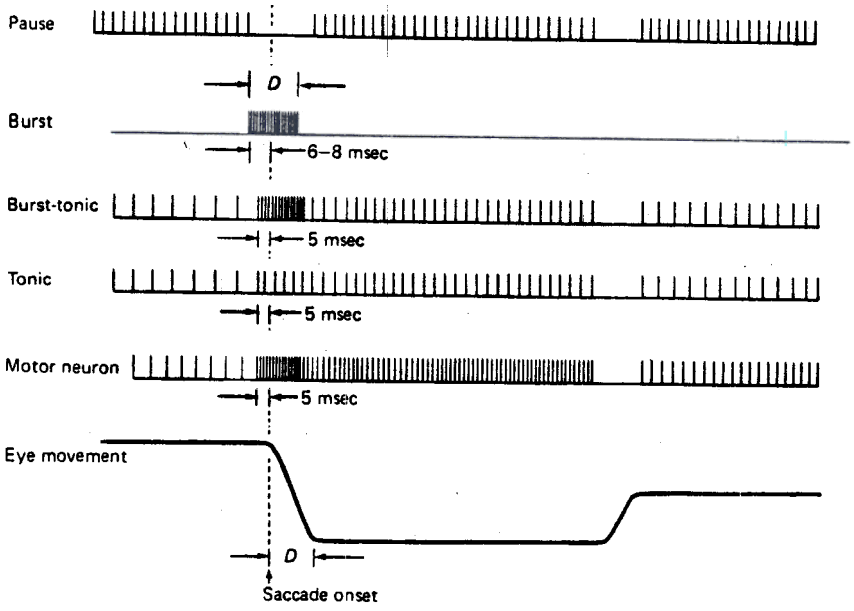


Figure 7.6. A schematic representation of the electrical activity recorded from cells in a saccade generator. Reprinted by permission of the publisher from "Oculomotor System" by P. Gouras, **Principles of Neural Science**, E.R. Kandel and J.H. Schwartz (Eds.), p.402. Copyright 1981 by Elsevier Science Publishing Co., Inc.

Agonist medium lead bursters (MLB) tend to burst to near saturating levels during saccades. The burst duration is proportional to the saccade length in the MLB's direction of action. Antagonist MLB cells burst slightly shortly before the saccade ends. Both MLB's have been found to burst equally to saccades orthogonal to the MLB's direction of action (van Gisbergen *et al.*, 1981). In the model, this occurs when a saccadic command shuts off the pausers v_3. These pausers are assumed to be omnipausers which are shut off by every saccadic command. If the saccade is perpendicular to an antagonist pair of LLB populations, then no other inputs perturb the corresponding circuit. Consequently both agonist and antagonist MLB populations are equally activated during the saccade.

The duration of pauser inactivity due to a saccadic command is proportional to saccade length. The tonic cell activity is proportional to present eye position. The motoneuron, or burst-tonic cell, activity is also proportional to eye position between saccades. In addition, motoneurons burst during saccades with a burst duration similar to that of the MLBs.

All of these properties of the experimental data appear in our model. Moreover, our simulations use the same neuron model for all cell types. The varied response characteristics seen in our simulations depend entirely on the connections, input and output signal functions, and parameter choices between the model cells. These cellular characteristics are thus emergent properties due to choices of network interactions and reaction rates. This type of model provides an alternative to control theory models whose transfer functions may not correspond to neural mechanisms. In particular, we have suggested that antagonist bursts near the end of a saccade, isometric coactivation during perpendicular saccades, and undershoot during a fatigued state may all be due to a common neural mechanism. This is not the type of explanation that is easily made by a control theory model.