14 The Modular Design of the Oculomotor System in Monkeys

P. Dominey, M. Arbib, and A. Alexander

14.1 Introduction

In this model we examine the modular design methodology as it applies to the design of both cortical and subcortical regions in the monkey. We will examine the topographic relations between the Posterior Parietal Cortex (PP), the Frontal-Eye Field (FEF), the Basal Ganglia (BG), the Superior Colliculus (SC), and the Mediodorsal thalamus (MD) as they work together to control the oculomotor regions of the brainstem (BS). We will also describe several experiments that can be performed on the model that demonstrate the modulation of eye movement "motor error maps", sustained potentiation (memory), and dynamic remapping of spatial targets within the "motor error maps". Although, the experiments were originally documented in Cerebral Cortex in 1992 (Dominey and Arbib), we have modified the model to make it easier to understand and to take advantage of the new features in NSL.

This work was initially motivated by data on the double saccade by Mays and Sparks, 1980 and 1983. In their testing, they found that monkeys could perform the double saccade task (as described below), though their accuracy was considerably affected by the delay between the retinal error input and the representation of eye position. Also, single unit recording studies of the Frontal-Eye Field, the Superior Colliculus, and the Lateral Inter Parietal (LIP) during visual and memory guided saccades indicate that cells in these regions code saccades in terms of direction and amplitude rather than head-centric spatial locations (Sparks 1986, Segraves and Goldberg 1987, Anderson et al. 1990, and Barash et al. 1991). We will attempt to duplicate their findings by examining two saccade paradigms in which retinotopic coding alone is inadequate to explain the spatial accuracy of the saccade. The five catagories of saccade experiments that we will be looking at are the simple saccade task, the double saccade task, the memory guided saccade task, the lesioning of FEF or SC, and the compensatory (or stimulated) saccade task.

14.2 Model Description

In the simple saccade task, a monkey is seated in a primate chair with its head fixed and eyes free to move. An illuminated point appears in the center of a grid of lights in front of him. We call this the fixation point. The fixation point disappears and a single light is illuminated. To get his reward, the monkey must saccade to this target. The timing diagram of these sequences and the resultant saccade are shown in figure 14.1.
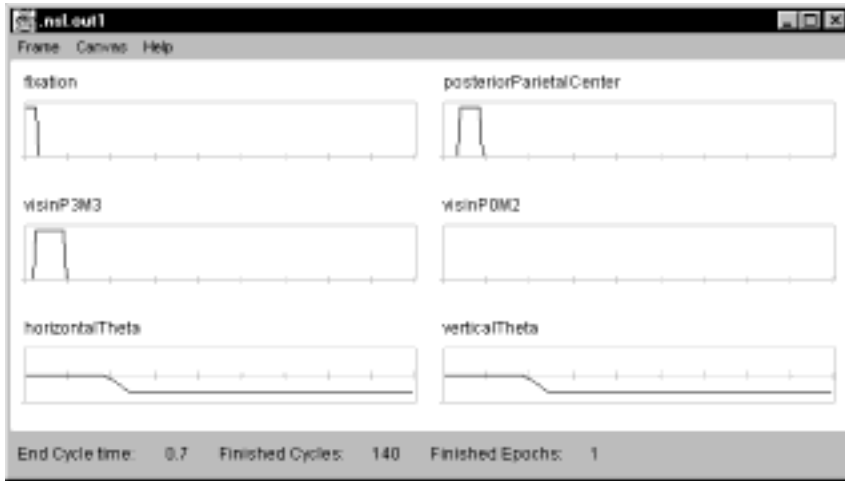
After performing several simple saccade experiments, it became clear that the longer the saccade, the more likely the error in acquiring the target became.

In the double saccade task, an illuminated point appears in the center of a grid of lights in front of him (figure 14.2). The illumination point disappears and two different lights are illuminated in rapid succession. To get his reward, the monkey must saccade to the first target and then to the second. The total duration of the two targets presentation is less than the time it takes to saccade to the first target. Because there are two targets, the representation of the second target, visinM3P3, in the motor error map would move as visinM3P3 itself would move across the retina during the saccade to the first target, visinM3P0.
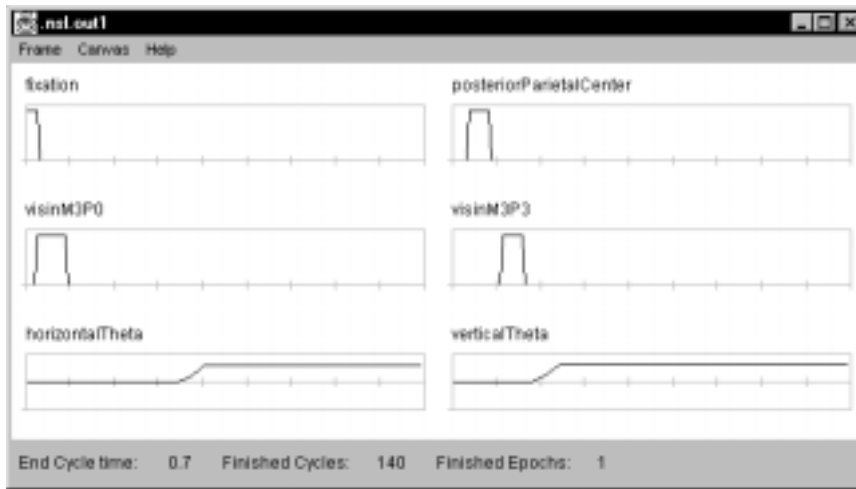
In the single, memory saccade task, an illuminated fixation point appears, then a target is while the fixation point is still illuminated (figure 14.3.) Once the fixation point is un-illuminated, the monkey is free to move his eye to the target. Thus, he has to remember where this target was to saccade to it.
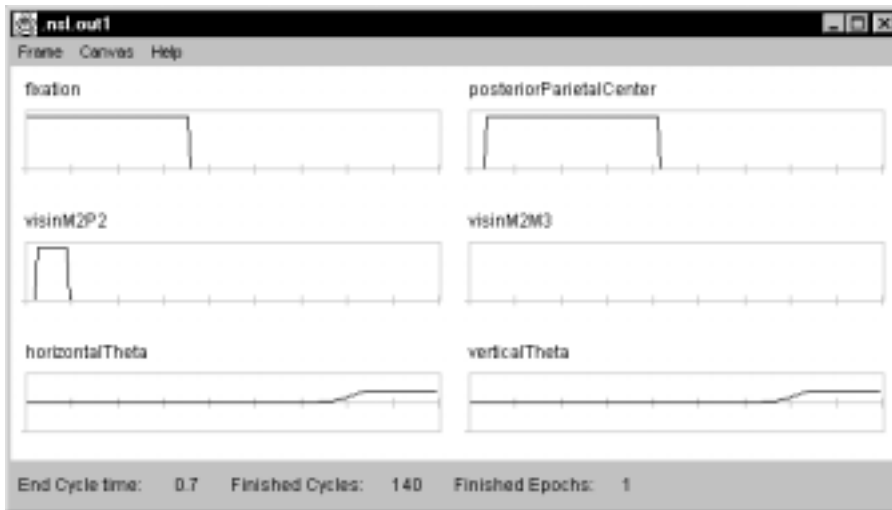
In the lesioning experiments we simply disable the output of the FEF (actually the fefsac variable) when we lesion FEF, and when we lesion SC we disable the output of the SC (actually the "supcol" variable). The lesioning experiments we describe here are different than the lesioning experiments we will talk about later where we combine the lesioning with stimulation. In these experiments the Long Lead Burst Neurons (LLBN) are strengthened due to the lesioning; however, it is not the case when we combine lesioning with the stimulation.
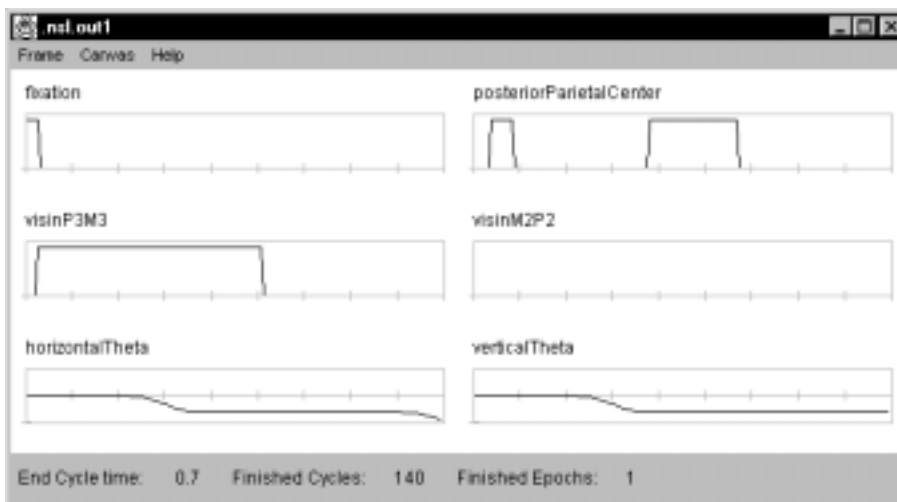
In the compensatory experiments we first describe two experiments where we stimulate the SC, and then we describe two experiments where we stimulated the FEF. Finally we describe an experiment where we stimulate the SC but lesion the FEF, and then describe an experiment where we stimulate the FEF but lesion the SC. When performing the stimulate and lesion experiment (Schiller and Sandell, 1983; Keating and Gooley, 1988) a visual target is briefly presented and removed before a saccade can begin. Before the visual saccade can begin, an electrical stimulus is applied to either the FEF or SC. The monkey will first saccade to the stimulated location and then to the real target (Dassaonville, Schlag, Schlag-Rey, 1990) even though timewise the real target appeared first. This is due to the fact that the visual signal takes much longer to get from the retina to either the FEF or SC. After performing either of these experiments we will see that

when either the FEF or SC is externally stimulated during an ongoing saccade that the brain compensates for different components of the ongoing movement.
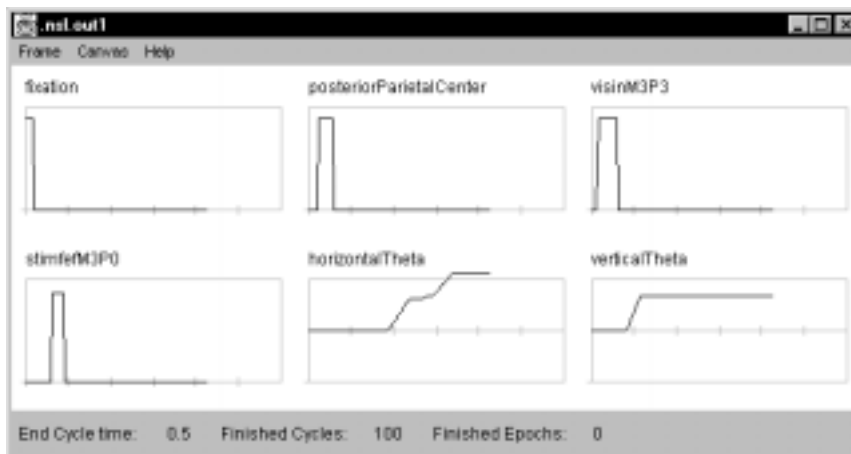
The simple saccade experiment allows us to study the topographic relations between sensory and motor areas, including inhibitory projections that manage motor field activities during the saccade. The double saccade experiment allows us to study the dynamic remapping of the target representation to compensate for intervening movement. The memory saccade experiment allows us to study the cortical and subcortical activity that sustains spatial memory. The lesioning experiments allow us to study the affects of lesioning. And the compensatory experiments allow us to study both the affects of stimulation to the FEF and of stimulation to the SC.

In the model, (figure 14.6) we have tried to localize the mechanisms that allow the monkey to accurately attain its target when an intervening saccade takes the eyes away from the location where the target was illuminated. The problem is that in many oculomotor structures, saccades are coded as a displacement from a given eye position, rather than as a final location. This means that the displacement code is only valid if it is updated almost continuously to account for the intervening changes in eye position, or if the saccade begins from the eye position at which the target was specified. Some experimental results indicate that the updating of the displacement code occurs before the signal reaches the FEF while other experiments indicate that this transformation occurs downstream from the FEF. In our model, we have chosen to represent this remapping in the lateral intra parietal (LIP) (part of the PP) before the signal reaches the FEF. Gnadt and Andersen (1988) found cells in the LIP that appear to code for future eye movement and show quasi-visual (QV) cell behavior. In a double saccade task they found cells that code for the second eye movement, though a visual target never falls in these cells' receptive fields. They proposed that PP might receive corollary feedback activity from saccades, suggesting that PP has access to eye position information that could be used to generate the QV shift. And since PP projects to both FEF and SC, it is likely that the PP is the origin of the QV activity seen in those two areas.

Looking at figure 14.6 we see that the dynamic re-mapping of spatial information contributes to the second saccade via multiple routes:

> LIP/PP to SC to the Brain Stem
> LIP/PP to FEF to the Brain Stem
> LIP/PP to FEF to SC to the Brain Stem

These multiple routes also contribute to the monkey's ability to saccade to the a target even though the SC or the FEF has been lesioned.

14.3 Model Implementation

The eye movement or saccade model as portrayed in figure 14.5 has been a useful system for studying how spatial information is transformed in the brain from a retinotopic coordinate system to the appropriate temporal discharge pattern of the motor plant. In this schematic we see the images transmitted to the Retina, and then to the Visual Cortex. From the Visual Cortex the image is transferred to the Posterior Parietal Cortex (PP) and specifically the Lateral Intra Parietal (LIP) within the PP. The Quasi Visual (QV) cells of the LIP/PP project to the intermediate layers of the SC (Lynch et al. 1985) and to the FEF (Petrides and Pandya 1984). Lesion studies have demonstrated that either the frontal eye field (FEF) or the superior colliculus (SC) is sufficient for commanding the execution of saccades. (Schiller and Sandell 1983; Keating and Gooley 1988). Also in figure 14.5, we see the basic mechanisms for calculating spatial accuracy. Mechanism A performs a dynamic memory remapping that updates motor error via an efferent velocity signal that is approximated by the damped change in eye position (DCEP).
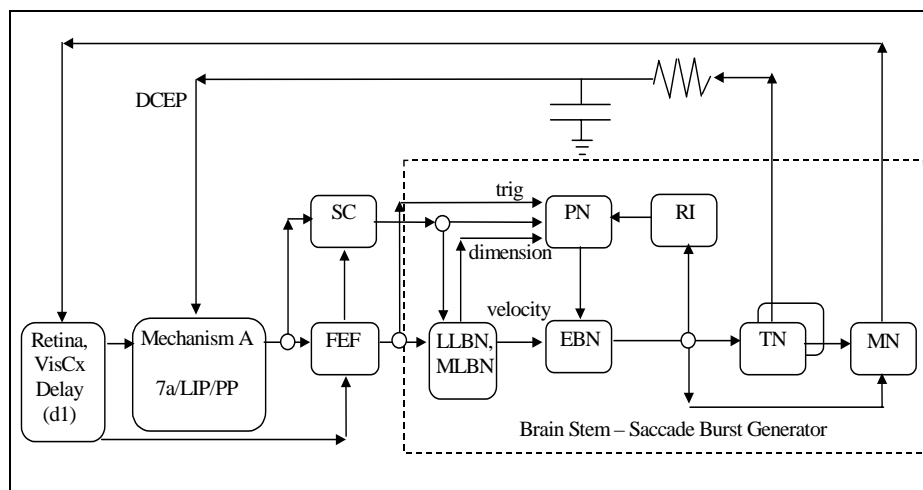

Figure 14.6

Spacial Accuracy within the Saccade Model. DCEP-Damped Change in Eye Position, 7a/LIP-Oculomotor Region of Posterior Parietal Cortex, FEF-Frontal Eye Field,SCd-Deep, Motor Layer in Superior Colliculus, LLBN-Long Lead Burst Neurons, MLBN-Medium Lead Burst Neurons, EBN-Excitatory Burst Neurons,PN-Omni-Pause Neurons, RI-Resettable Integrator, TN-Tonic Neurons, MN-Oculomotor Neurons

Also in figure 14.6 we see the Saccade Burst Generator. The saccade burst generator (SBG) performs the spatiotemporal transformation from the motor error maps of the SC and FEF to generate eye movements as a function of activity in tonic position cells and excitatory burst neurons (Robinson 1970, 1972).

The two neural areas we do not see in the figure 14.6 but which are included in our model in figure 14.7 are the mediodorsal thalamus (THAL) and the basal ganglia (BG). The thalamus with the FEF provides a reciprocal connection that implements the spatial memory loop. The basal ganglia (BG), on the other hand, provides a mechanism for the initiation of cortico-thalamic interactions via the removal of inhibition from the basal ganglia's substantia nigra pars reticulata (Snr) on the mediodorsal thalamus. The BG also plays a role in the disinhibition of SC and THAL for saccades requiring spatial memory (Fuster and Alexander 1973; Hikosaka and Wurtz 1983; Ilinsky et al 1985; Goldman-Rakic 1987).

The computer model emulates the above system as closely as possible. In figure 14.7 below, we see the exact schematic that is used to generate the code for the top level of the model, DomineyTop. DomineyTop contains many of the same components as in figure 14.6 above; however, we have encapsulated the Burst Saccade Generator (BSG) as part of the brainstem into one module. We have also changed the names of the neural areas to conform to the NSLM naming conventions (names begin with lower case letters for instances of objects).
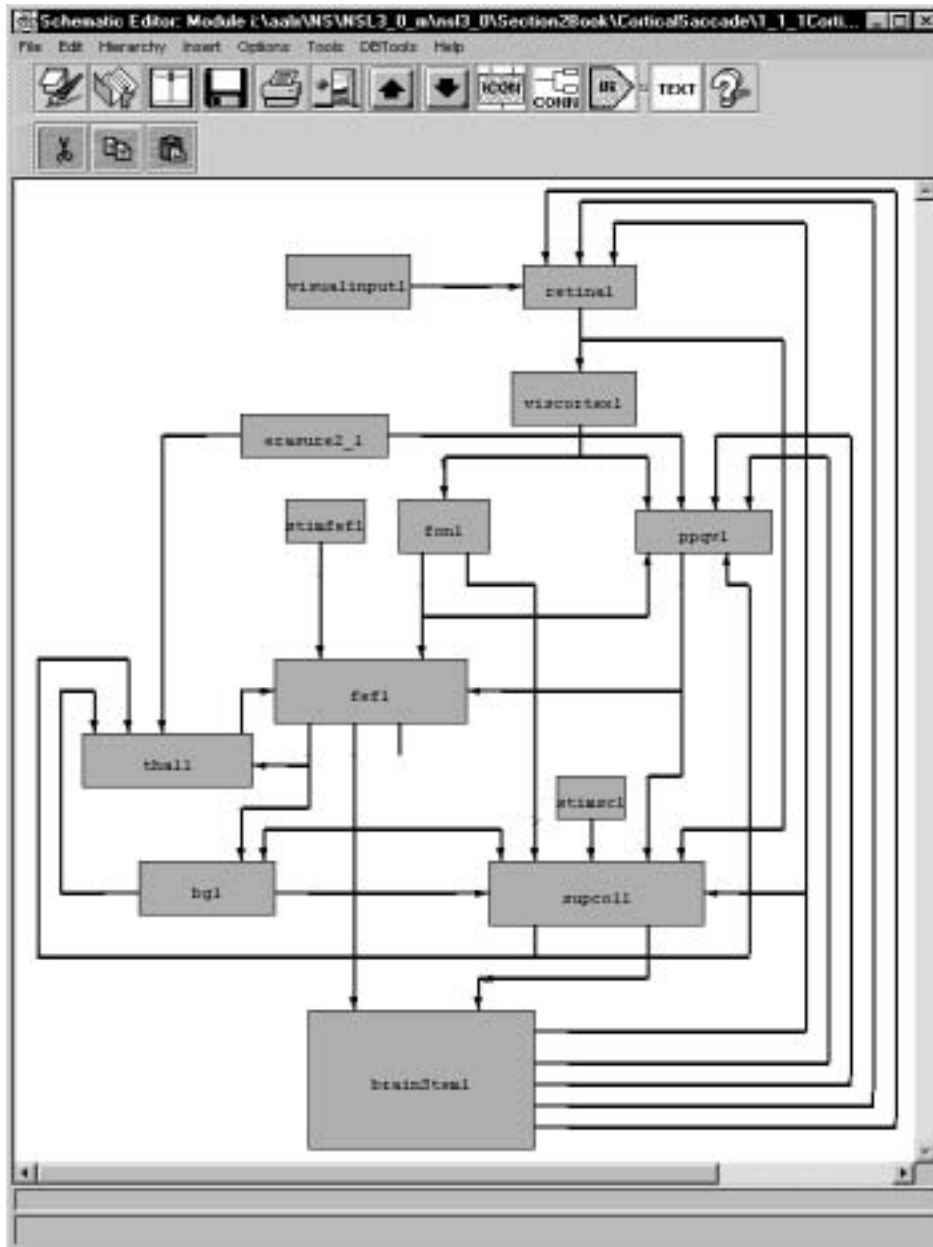
    In figure 14.7, we see the complete Dominey Model for all of the saccade experiments we will be performing. The external world is represented by a 27 by 27 neural element array, called visualinput. The fixation point and targets are specified on this array via the user interface or by using the default parameters. The visual input is remapped on to the retina, in retinotopic coordinates and then the visual image travels through the Visual Cortex (viscortex) to the Posterior Parietal. At this point, we connect the PP to both the FEF and to the Lateral Inter Parietal within the Posterior Parietal (ppqv1). In PPQV, we have implemented a variation of the Dynamic Memory algorithm by Droulez and Berthoz, 1991, for shifting targets on a motor error map, (what we called Mechanism A in figure 14.6.) This algorithm for the dynamic shifting of targets is called from PPQV but located in a library class we call DomineyLib. By providing special functions in library classes instead of buried in the model, we make it easier for other modelers to reuse these functions. This library is also used in the case of the Superior Colliculus, SC, which uses the "WinnerTakeAll" algrorithm to compute the tonic quasi-visual property

seen in the superior colliculus (Mays and Sparks 1980). We call the variable that represents these cells scqv. SC also contains cells that receive direct input from the retinal ganglion cells, and are active in generating reflexive saccades to visual targets. We call these cells scsup. If a fixation target is not present, then these cells will drive SC, generating short latency saccades (Braun and Breitmeyer 1988) via the transcollicular pathway (Sparks 1986). Note that since scsup cells are connected to both the retinal input and the fovea on cells, FOn, these cells will not fire until the FOn turns off. Also SC contains cells that generate presaccadic bursts before voluntary saccades. We call these cells scsac. Experimental data indicates that SC receives an excitatory topographic projection from presaccadic cells in FEF (Segraves and Goldberg 1987). This is accomplished via fefsac in our model.

The FEF module has three classes of saccadic cells. Visual cells (fefvis) respond to all visual stimulus (Bruce and Goldberg 1984). We have grouped these with our Quasi-visual like cells. Coding for the second saccade in the double saccade, these QV cells demonstrate the *right movement field* and the *wrong receptive field* responses, characteristic of QV cells, and are referred to as right-MF/wrong-RF cells (Goldberg and Bruce 1990) or fefvis within our model. Memory cells (fefmem) sustain activity during the delay period in memory experiments (Funahashi et all 1989) while movement cells, fefsac, discharge before all voluntary saccades corresponding to the cell's preferred dimensions (Segraves and Goldberg 1987).

In figure 14.7 , we see the FON module which contains cells distinguished in the FEF that have an on or off response to visual stimulation in the fovea. These foveal cells are not localized to a particular location of the topographic map of FEF, and they project to a wide range of locations within the SC (Seagraves and Goldberg 1987). We model the Foveal On cells, FOn, projecting the center element of the PP layer (the fovea) to a standard size array, that is used to provide inhibition to the elements of the caudate (CD in BG). In each of the following experiments we note that the removal of the illumination of the fixation point signals the monkey that he is free to move his eyes; thus, when FOn is off, the monkey is free to perform the saccades.

Also in figure 14.7 we see several darkened modules. These are the input modules where the user can change the defaults. The visual input module is used for specifying the targets and fixation point. The stimulation modules are used to provide stimulus to the FEF or SC in the compensatory saccade cases. The user is also free to dynamically change these NSL type variables at run-time using the NSLS scripting language. (However, note that variables must be writable to change them from the scripting language. Currently we have declared the variables "visualinput1/visualinput", "stimfef1/stimFEF", "stimsc1/stimSC", "brainstem1/llbn1/llbnPot_k1", "brainstem1-/llbn1/llbnPot_k3", "fef1/fefmemPot_k2", "fef1/fefsac", "supcol1/supcol", "supcol1/supcol_k3", and "ppqv1/qvMask_k1" as writable variables since their values change depending on the protocol used.)
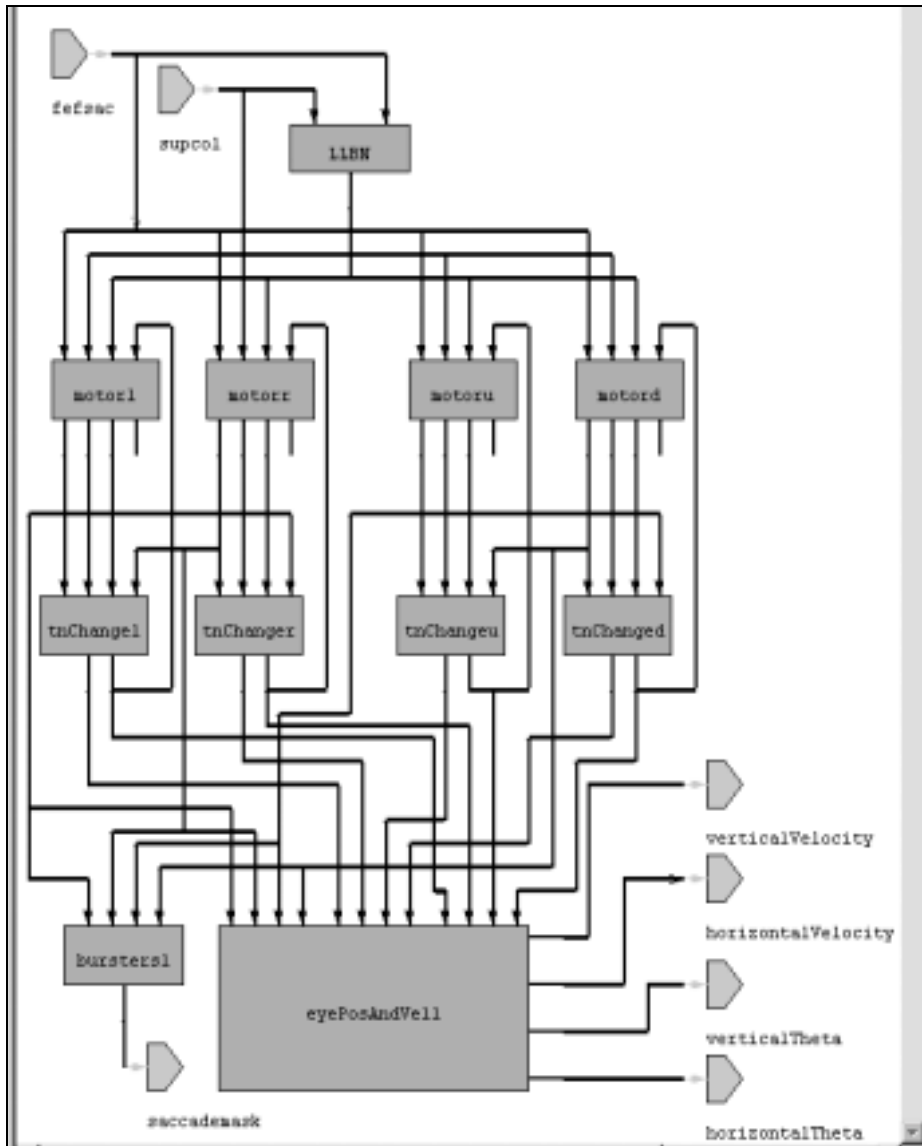
Figure 14.8
Saccade Burst Generator or
BrainStem Schematic

As stated in the earlier, the saccade burst generator (SBG) or the BrainStem Saccade Burst Generator (figure 14.8) performs the spatiotemporal transformations from the motor error maps of the SC and FEF (Robinson 1970, 1972). This SBG is based on one by Scudder (1988). Two of the properties of this model are that it will yield a saccade with the topographically coded metrics in response to either FEF or SC stimulation or both, and it accurately emulates the FEF and SC in that increased firing at a given point will increase the velocity and decrease the latency without changing the metrics of the saccade.

Also in figure 14.8 we see the Tonic Neuron modules. The tonic neurons (TN) provide corollary discharge signals lefttn, righttn, uptn, downtn, which, together with a delayed version of these signals, provide the input used by our model of the PP for the dynamic remapping function which underlies successful completion of the double saccade task (Mechanism A).

The main module in the BrainStem is the Motor Schema shown and described in figure 14.9.
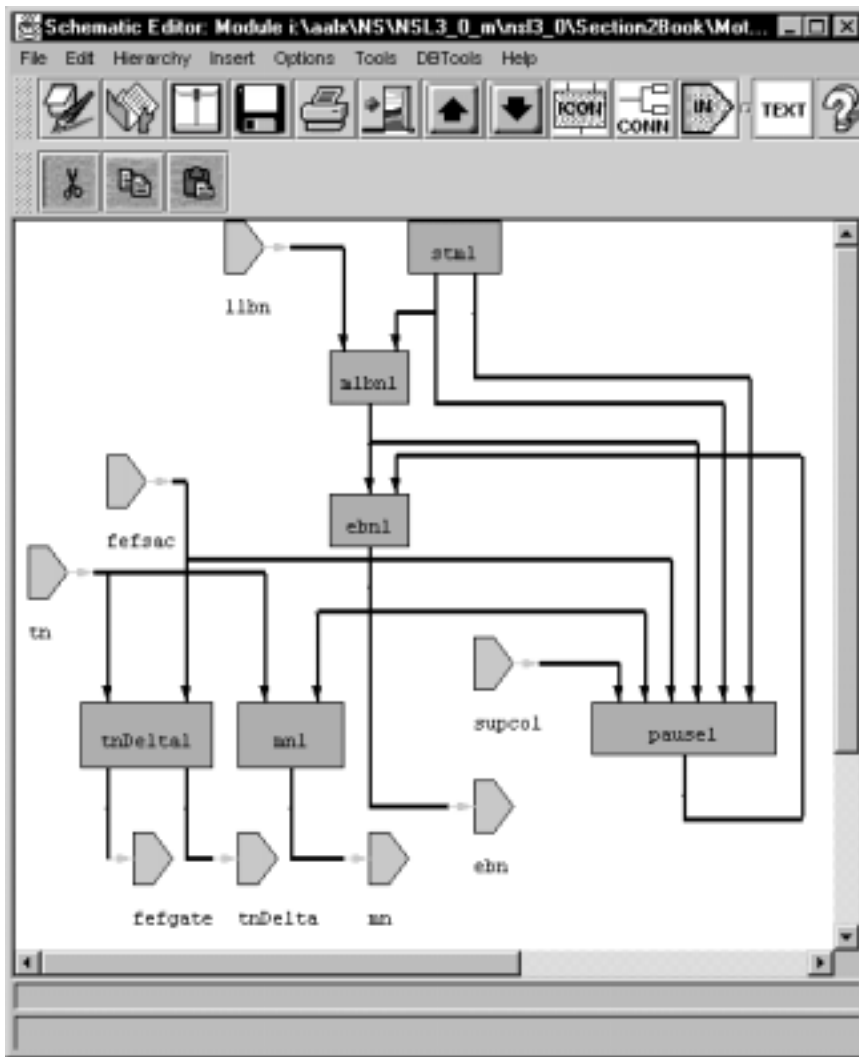
Figure 14.9
The Motor Module's
Schematic

In figure 14.9 we see the Motor Schema. This schematic represents the bulk of the Saccade Burst Generator (SBG). We can compare this to the schematic given by Dominey and Arbib 1992 describing the shared and cooperating spatial accuracy mechanisms. (See figure 14.6.) The Medium Lead Burst Neurons (mlbn) provide input to the Excitatory Burst Neurons (ebn). And the ebn neurons provide inputs to the tonic neurons, the resetable integrator within the Pause module, and the motor neurons, MN. The Pause module contains the resetable integrator, trigger cells, and the Omni-Pause Neurons. The TNDelta module gates the tonic neuron response. The oculer motor neurons (MN) move the eyes, and the STM module calculates spatio temporal transformations.
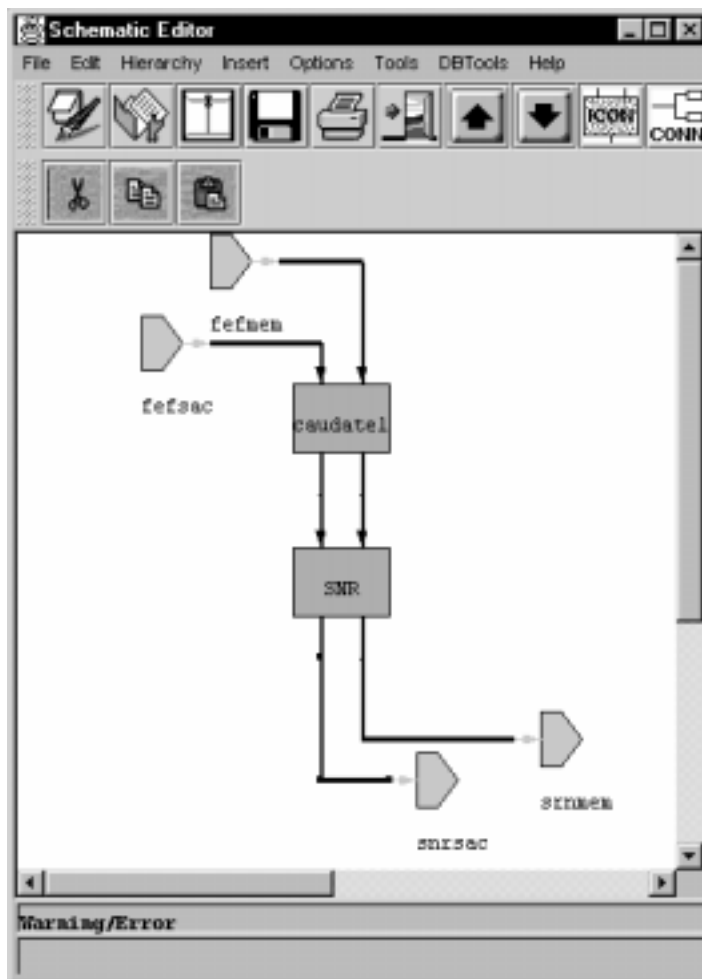
In figure 14.10, we see two inhibitory nuclei of the basal ganglia (BG), caudate (CD) and substantia nigra pars reticulata (SNr). They provide an additional, indirect link between FEF and SC (Chevalier et al 1985). This link allows FEF to selectively modulate the tonic inhibition of the SNr on the SC and the thalamus (Deniau and Chevalier, 1985; Alexander et al. 1986) through the caudate nucleus.

Originally this model was written in NSL2.1 as a series of functions which were executed sequentially. We have re-written the model to make it more object-oriented and modular. The first thing we did was to stop representing neural areas as functions and start representing them as classes. This allowed us the ability to set the initialization parameters in each class/neural area as well as provide other functionality that was local to the neural area.

Next, we instantiated those classes where practical in the model. For instance, in the Saccade Burst Generator (figure 14.5) we instantiate the Motor Schema module four times: once for each direction (left, right, up, down). This of course reduced the amount of code we needed to write for the saccade burst generator in the brain stem: we only had to write MLBN, EBN, Pause, TNDelta, and MN once, instead of four times: one for each direction of right, left, up, and down.

We also made the model hierarchical, grouping together neural areas by schema. Thus the MLBN, EBN, Pause, RI, TN, and MN are all part of Motor which is part of the Brainstem which is part of the top level model. Other schemas include the Memory System, and the Vision System.

Once we had grouped the modules together, and added ports to connect the different modules, it was clear that we had created the same circuit as that documented in Dominey and Arbib (1992) (see figure 14.6). Since the model was now in the Schematic Capture System (SCS), we could automatically generates the structural part of the model code (see figure 14.7).

Another aspect of the new model, is ability to change the dimension of the internal layer sizes (arrays) by changing just one variable. In the original model, we needed to improve the spatial resolution and thus changed the model from using a visual input layer of 27 by 27 neurons to 57 by 57 neurons (Dominey 1991); which meant that the retina and the rest of the neural arrays were 19 by 19. Since there are over 80 of these structures, being able to change 80 structures by changing just one variable is a big improvement over the old model.

Some of the more interesting uses of the NSLM code appeared in the SC and FEF modules. In both modules we have called custom routines from the library we created called "DomineyLib". Code Segment 14.1 shows the code for the SC, and code segment 14.2 shows the code for the FEF.

```
public void simRun() {
    scsupPot=nslDiff(scsupPot,scsupPot_tm, - scsupPot -
        scsupPot_k1*fon + scsupPot_k2*retina);
    ppqv_winner = DomineyLib.winnerTakeAll
        (ppqv,nWTAThreshold.get(),stdsz);
    scqvPot=nslDiff(scqvPot,scqvPot_tm,-scqvPot + ppqv_winner);
    scsacPot=nslDiff(scsacPot,scsacPot_tm, -scsacPot
        +scsacPot_k1*fefsac -
        scsacPot_k2*snrsac);
    supcolPot=nslDiff(supcolPot,supcolPot_tm, -supcolPot +
        supcolPot_k2*scsac +
        supcolPot_k3*scqv -
        supcolPot_k4*fon +
        supcolPot_k6*scsup -
        supcolPot_k1*scDelay); // this is zero.
    supcolPot[center][center] = 0; // no saccades to where we
        already are!
    sc_winner = DomineyLib.winnerTakeAll(supcolPot,
    nWTAThreshold.get(),stdsz);
    scsup = nslSigmoid
        (scsupPot,scsup_x1,scsup_x2,scsup_y1,scsup_y2);
    scqv = (saccademask^scqvPot);
    scsac = nslSigmoid
        (scsacPot,scsac_x1,scsac_x2,scsac_y1,scsac_y2);
    //aa: from the 92 paper equation 15 is set to zero if
        lesioning SC
    if ((protocolNum==6)|| (protocolNum==13)) { // lesion SC
        supcol=0;
    } else {
        supcol = nslSigmoid(sc_winner,supcol_
            x1,supcol_x2,supcol_y1,supcol_y2);
        supcol = supcol + (supcol_k3*stimulation);
    }
    scDelay=nslDiff(scDelay,scDelay_tm, -scDelay + supcol);
}
```

Code Segment 14.1
Code Segment from SC

```
public void simRun() {
    fefvisPot=nslDiff(fefvisPot,fefvisPot_tm,
        (- fefvisPot + ppqv));
    fefmemPot=nslDiff(fefmemPot,fefmemPot_tm,(- fefmemPot +
        fefmemPot_k4*thmem + fefmemPot_k2*fefvis -
            fefmemPot_k1*fon));
    fefsacPot=nslDiff(fefsacPot,fefsacPot_tm,( - fefsacPot +
        fefsacPot_k1*fefvis +fefsacPot_k2*fefmem -
            fefsacPot_k3*fon));
    fefsacPot[center][center] = 0;
    fefvis = nslSigmoid
        (fefvisPot,fefvis_x1,fefvis_x2,fefvis_y1,fefvis_y2);
    fefmem = nslSigmoid
        (fefmemPot,fefmem_x1,fefmem_x2,fefmem_y1,fefmem_y2);
    fefsactmp = nslSigmoid(fefsacPot,
        fefsac_x1,fefsac_x2,fefsac_y1,fefsac_y2);
    fefsac = fefsactmp + (fefsac_k1*stimulation);
    if ((protocolNum==7)||(protocolNum==14)) {//lesion fef
        fefsac=0;
    }
}
```

Code Segment 14.2
Code Segment from FEF

14.4 Simulation Results[1]

We now report the simulation results. We note that the neural populations we model carry information in terms of their discharge frequencies, the durations of discharge, and the latencies both between stimulus and firing, and between neural events in connected regions. All of the experiments can be found on line at our website, at http://www-hbp.usc.edu/~nsl/unprotected. The World Wide Web applet for this model and these experiments can also be found at the same location, as well as, example experimental results. We will note here that the run delta was set to 0.005, or 5 milli seconds and the run end-time was set to 0.7 seconds for each of the following 15 experiments/protocols.

The Single Location Saccade Experiment
In the single saccade experiment a light illuminates the fixation point which for the monkey is in the center of a screen, but for us is in the center of an array called visualinput. After .02 seconds the light goes away and a target appears someplace on the screen. For the monkey to get his reward he must saccade to the target location.

> **Protocol 1: single saccade**
> Fixation 0-0.02 at [(i=center,j=center)]
> Target A 0.02-0.07 at [(i=center+3,j=(center-3)]

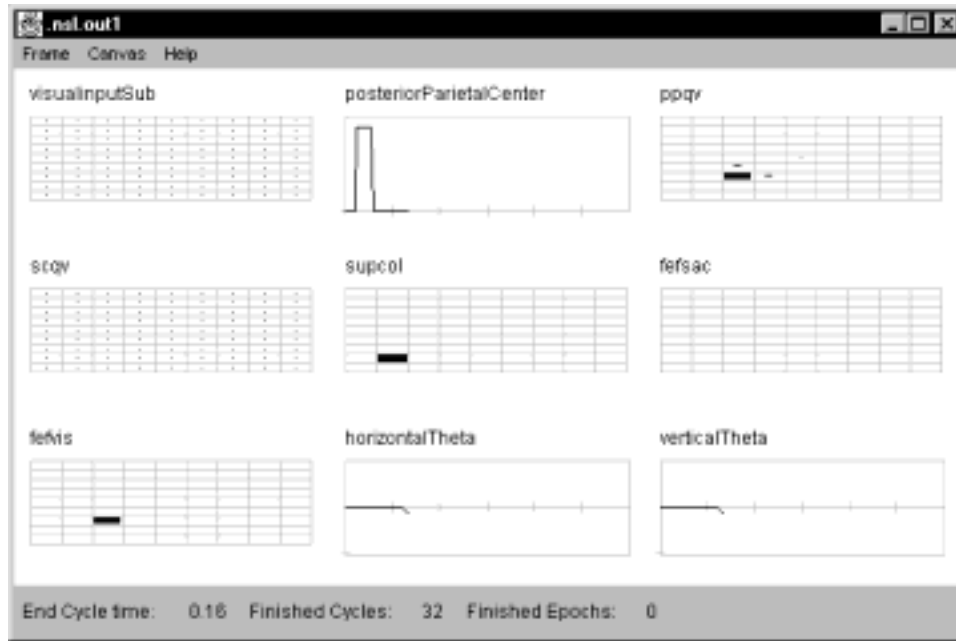Figure 14.11 displays the system at time equal 0.16 seconds.

At 0.16 seconds the saccade is under way and we can see activity in several of the major brain areas. After the fixation has been removed and the target appears, the center of the Posterior Parietal (PP) is off, and the FOn signal is off, which releases the inhibition on the caudate. The caudate after receiving the fovea off signal and the target information from the FEF, then projects an inhibitory signal to the SNR which releases the inhibition on the SC which contains the information of where the target is. Target information contained in SC and FEF output drive the brainstem to reposition the eye such that the target is in the center of the fovea. If the target is still lit, then this causes the center element of the PP to be on. If the target is not still lit, then the center of PP will be off, as in figure 14.12.
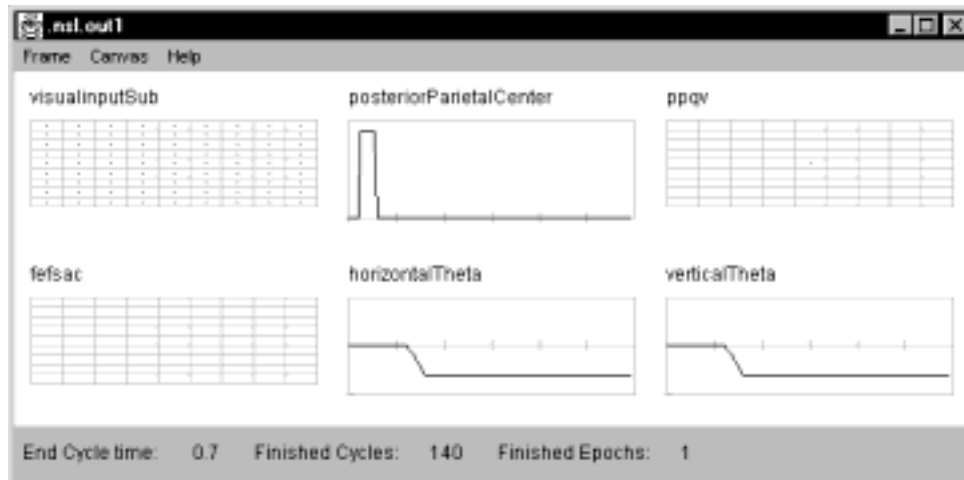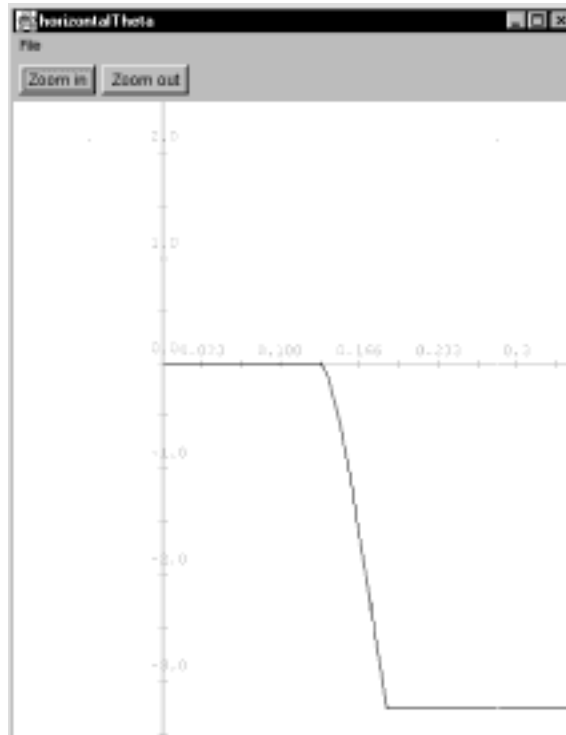
The Single Location Saccade Experiments Using Memory

The memory saccade experiment requires the thalamus to store spatial locations in memories via a reciprocal excitatory connections with FEF. When the target disappears, it is held in FEF memory (fefmem) by the reciprocal excitatory connection between MD of the thalamus and FEF. The removal of the fixation point causes the FOn signal to be reduced which allows the fefsac to fire and removes a source of inhibition from the SC. The combination of these events allows the stored spatial memory to command a saccade. The effect of the spatial memory is to keep the target position in the FEF active after it is extinguished.

> **Protocol 2: memorySingleI saccade.**
> Fixation 0-0.28 at [(i=center),(center)]
> Target A & fixation 0.02 - 0.07 at [(i=center-2),(j=center+2)]
> Note: Fixation off at off at 0.28
>
> **Protocol 3: memorySingleI saccade.**
> Fixation 0-0.28 at [(i=center),(center)]
> Target A & fixation 0.02 - 0.07 at [(i=center-2),(j=center-3)]
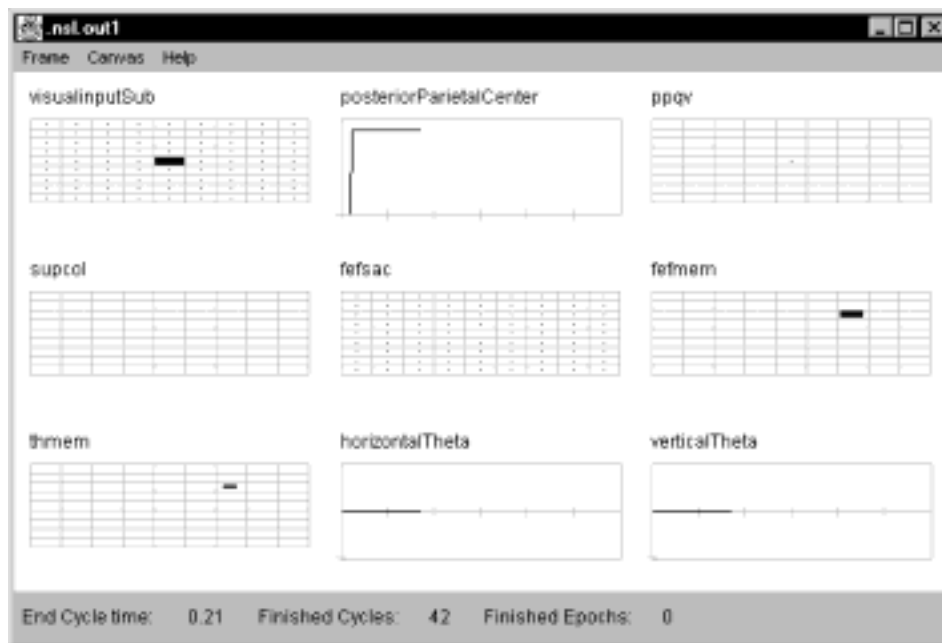> Note: Fixation off at off at 0.28

In figure 14.14, we see that at time equal 0.21 seconds no saccade has happened yet, but we can see that the location of the saccade is stored in the Thalmus (thmem) and the FEF (fefmem). As long as the fixation point remains on, the Thalamus and FEF will maintain the memory loop.

The Double Location Saccade Experiments

When performing the double saccade task, the motor error representation of the second target is dynamically remapped to com-pensate for the intervening movement. By using the ppqv layer as input to the fefvis layer, the FEF can contribute to the correct specification of both saccades in the double step task.

> **Protocol 4. doubleI saccade.**
> Fixation 0-0.02 at [(i=center),(j=center)]
> Target A 0.02 - 0.07 at [(i=center-3),j=(center)]
> Target B 0.09 - 0.13 at [(i=center-3),j=(center+3)]
>
> **Protocol 4. doubleII saccade.**
> Setup: delta = 0.005 = 5 msec; end-time 0.7 = .7 sec
> Fixation 0-0.02 at [(i=center),(j=center)]
> Target A 0.02 - 0.07 at [(i=center),j=(center-2)]
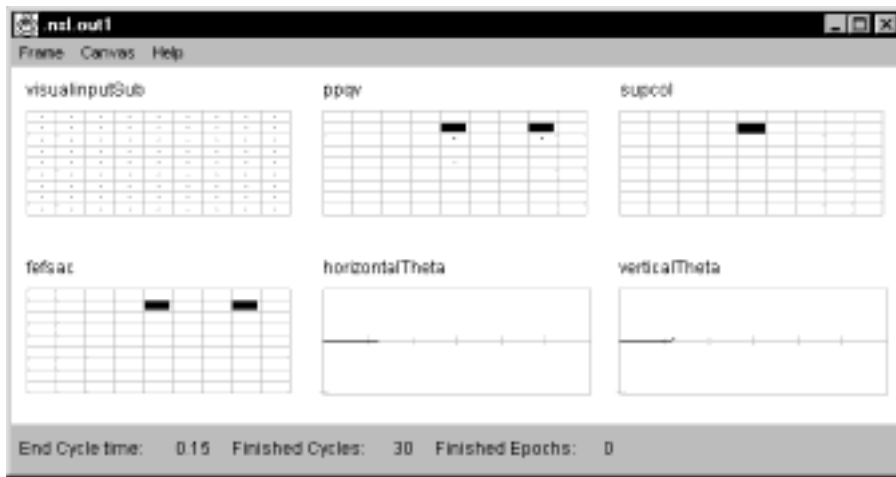> Target B 0.085 - 0.125 at [(i=center-3),j=(center)]

Figure 14.15
Double Saccade Experiment – Protocol 4 at time=.15 seconds.
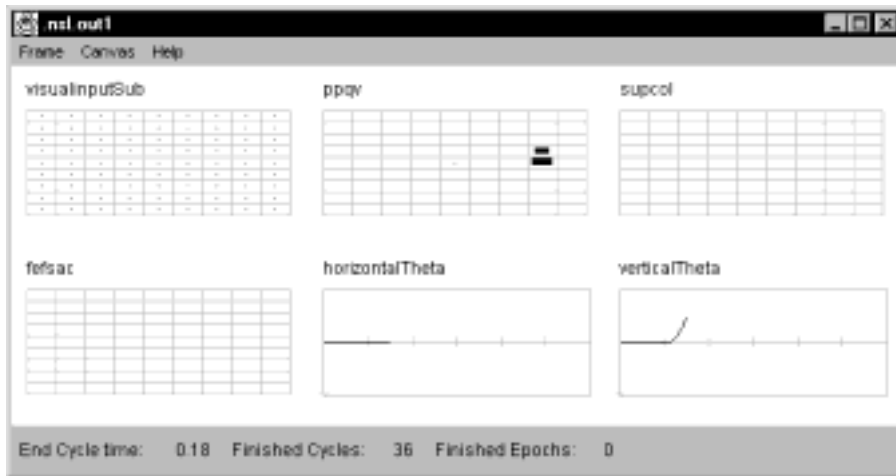

Figure 14.16
Double Saccade Experiment – Protocol 4 at time=.18 seconds

In figure 14.15 we see the activity in the ppqv before the dynamic remapping of the target takes place. The quasi-visual (QV) convolution mask (qvmask) is generated from the temporal offset of the horizontal and vertical eye position. When convolved with ppqv, the qvmask represents interactions thought to implement the quasi-visual shifting seen in the parietal cortex. The initial upward saccade along with the qvmask causes the cells on the upper part of ppqv to be excited, while the cells on the bottom part of ppqv are in inhibited, which causes the second target contained within ppqv to be shifted three cells down (the opposite direction). Or in otherwords, as the first target moves into the fovea or center of ppqv, the distance between the first and second target must be maintained, and thus it looks like the second target is moving away from its original location (see figure 14.16).

The Lesioning of SC or FEF Experiments
In the lesioning experiments either the SC or the FEF is lesioned. The protocol is the same as that for the simple saccade experiment except for the lesion.

> **Protocol 6 - lesionSC saccade.**
> Fixation 0-0.02 at [(i=center,j=center)]
> Target A 0.02-0.4 at [(i=center+3,j=(center-3)]
>
> **Protocol 7 - lesionFEF saccade.**
> Fixation 0-0.02 at [(i=center,j=center)]
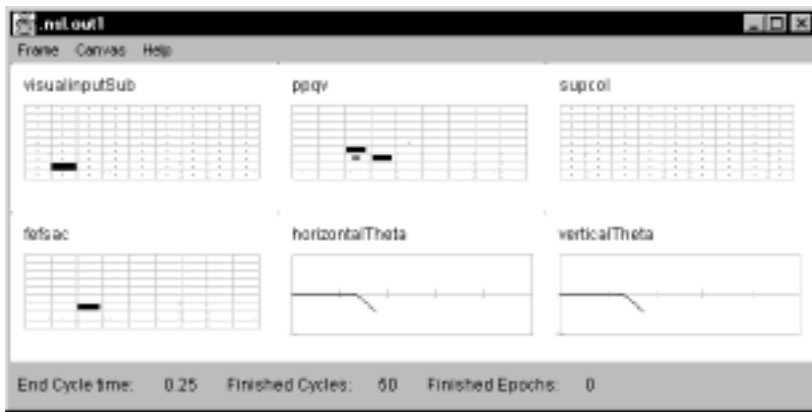> Target A 0.02-0.4 at [(i=center+3,j=(center-3)]

Figure 14.17
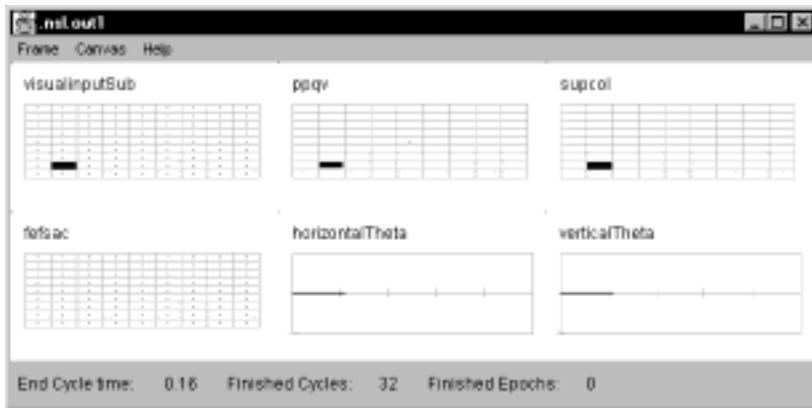Lesion SC experiment –
Protocol 6 at time=0.25
seconds

Figure 14.18
Lesion FEF experiment –
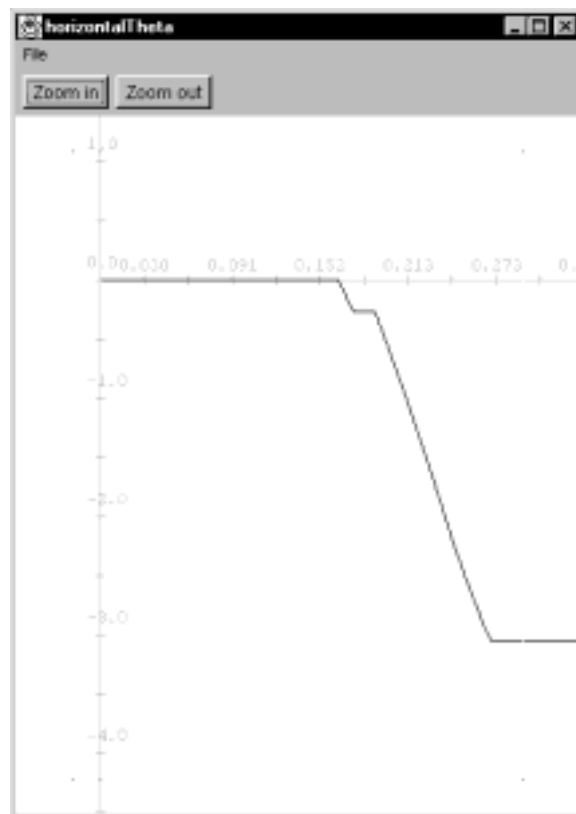Protocol 7 at time=0.16

Figure 14.19
Lesion SC experiment –
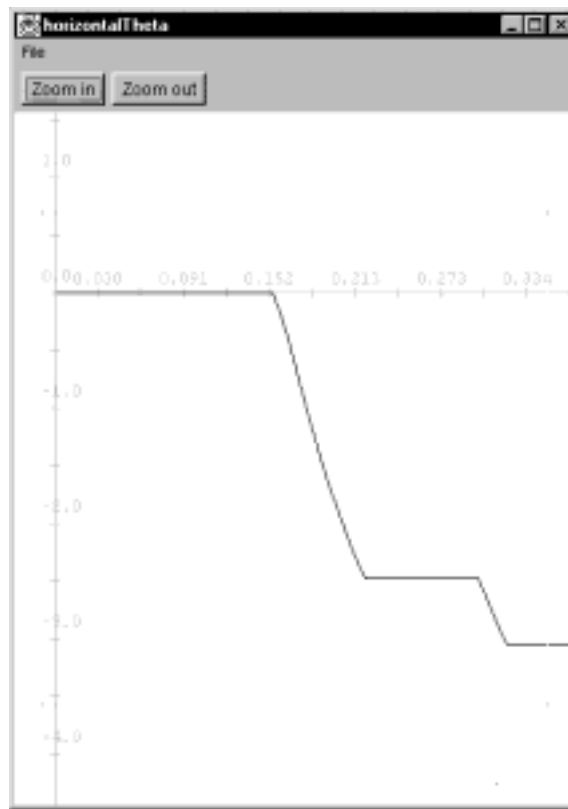Protocol 6 at time=0.4
seconds

Figure 14.20
Lesion FEF experiment –
Protocol 7 at time=0.4

Note that in figure 14.17 we see that when we lesion the SC, there is no activity in "supcol". In figure 14.18 we see that when we lesion FEF, there is no activity in "fefsac". In figure 14.19 we see that lesioning the SC causes about a 0.34 second delay (.167-.133) and shortens the amplitude by a small amount when compared to the simple saccade of figure 14.12. In figure 14.20 we see that lesioning the FEF causes a 0.19 second delay in the saccade when compared to the simple saccade of figure 14.12. and shortens the amplitude by a small amount but then tries to correct for the mistake. When the amplitude of the saccade is reduced, the eyes do not move as far, and it can take several saccades to acquire the target in the center of the fovea.

The Double Location Memory Saccade Experiments
In the double location memory saccade experiments the quasi-visual field shifting is used to reposition the location of the second target in "ppqv" as the eyes moves to the first location. In addition, when the first target location goes on again, fixation re-occurs and the memory elements within the FEF and Thalamus are activated, causing the location of the second target to be stored until the fixation (or in this case the illumination of the first target) goes off. Thus this experiment combines attributes of the double saccade experiment with the memory saccade experiment. The timing on these types of experiments is very critical. If the second saccade happens too late it will not be shifted in ppqv. If the second saccade happens too early, the location will not get stored in the FEF and Thalamus memory loop.

> **Protocol 8 - memoryDouble saccade**
> Fixation 0-0.02 at [(i=center),(j=center)]
> Target A 0.02 - 0.05 at [(i=center-3),(j=center)]
> Target B 0.095 - 0.11 at [(i=center-3),(j=center+3)]
> Target A 0.165 - 0.5 at [(i=center-3),(j=center)]

Protocol 15 - memoryDouble2 saccade
Fixation 0-0.02 at [(i=center),(j=center)]
Target A 0.02 - 0.07 at [(i=center),(j=center-2)]
Target B 0.09 - 0.105 at [(i=center-3),(j=center)]
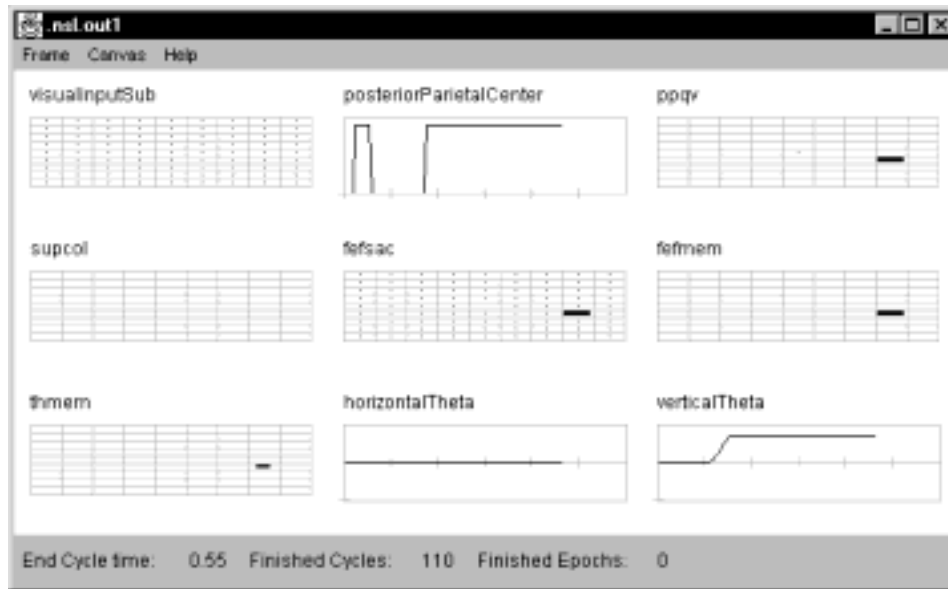Target A 0.17 - 0.5 at [(i=center),(j=center-2)]

Just as in the single memory saccade we can see in figure 14.21 some activity in fefmem and thmem for storing memory. Also, we see that the remapped location of the second target is off by a couple of degrees. This is probably due to the fact that the saccade falls short of the target and thus retains some of the information needed to acquire the first target. However, the second double memory saccade experiment (protocol 15) does not have this problem since it acquires the first target without overshooting. Its first saccade is also a shorter saccade than protocol 8's first saccade.

The Compensatory Saccade Experiments
In the first four compensatory saccade experiments, no lesioning is involved. Only stimulation of the indicated areas.

Protocol 9 - stimulated SC CompensatoryI saccade.
Fixation 0-0.02 at [(i=center,center)]
Target A 0.02 - 0.07 at [(i=center-3),(j=center+3)]
Stimulation 0.07 - .11 at [(i=center-3),(j=center)]
Note: Reduced target error due to location - only going 1 direction

Protocol 10 - stimulated SC CompensatoryII saccade.
Fixation 0-0.02 at [(i=center,center)]
Target A 0.02 - 0.07 at [(i=center-3),(j=center)]
Stimulation 0.07 - 0.11 at [(i=center),(j=center-2)]
Note: Increased target error due to location - must go two directions

Protocol 11 - stimulated FEF CompensatoryI saccade.
Fixation 0-0.02 at [(i=center,center)]
Target A 0.02 - 0.07 at [(i=center-3),(j=center+3)]
Stimulation 0.07 - 0.11 at [(i=center-3),(j=center)]

Protocol 12. stimulated FEF Compensatory II saccade.
Fixation 0-0.02 at [(i=center,center)]
Target A 0.02 - 0.07 at [(i=center-3),(j=center)]
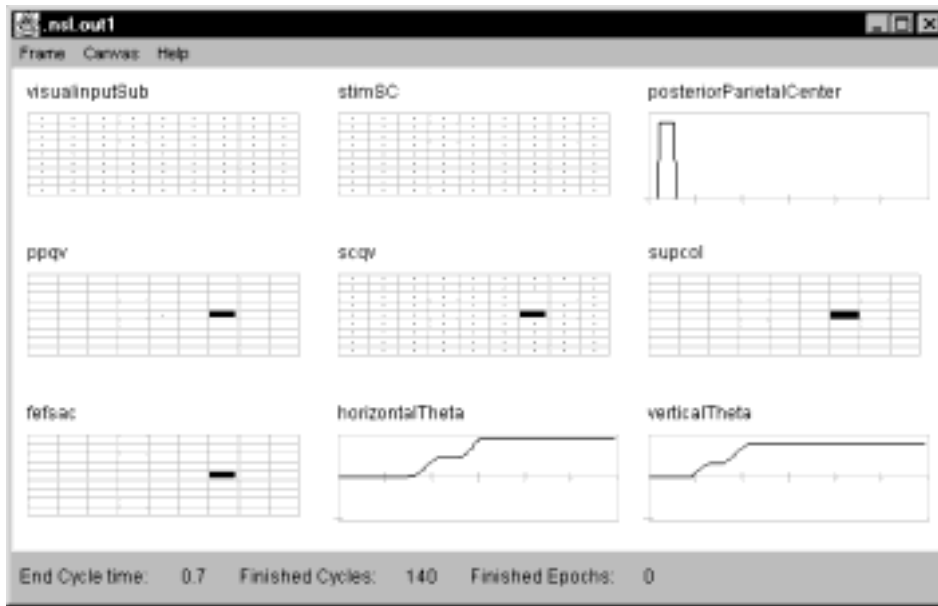Stimulation 0.07 - 0.11 at [(i=center),(j=center-2)]

As can be seen in the figure 14.22, the activity within "ppqv" is very similar to that as shown for the double saccade experiment. However, we can also see that the stimulated target is acquired first even though the stimulus was applied after the visual input cue was illuminated. Again this is due to the long path between the retina and the SC.

In the compensatory saccade experiment with lesioning simulated the electrical stimulation of both FEF and SC (figure 14.6), as described in Schiller and Sandell (1983) in which one of the two neural area was lesioned and the other was stimulated. For FEF stimulation we set the k1 parameter to 1.58, and applied electrical stimulation at 175 Hz for 40ms to fefsac. For SC stimulation we set the k1 parameter to 2.9, and applied electrical stimulation at 175 Hz for 40 ms to various locations in SC. The timing and movement data for these trials are summarized below and the results for protocol 14 are shown in figure 14.21.

Protocol 13 - stimulated FEF LesionSC I saccade. - no SC
Setup: delta = 0.005 = 5 msec; end-time 0.7 = .7 sec
Fixation 0-0.02 at [(i=center,center)]
Target A 0.02 - 0.07 at [(i=center-3),(j=center+3)]
Stimulation 0.07 - 0.11 at [(i=center-3),(j=center)]

Protocol 14 - stimulated SC LesionFEF I saccade. no FEF
Setup: delta = 0.005 = 5 msec; end-time 0.7 = .7 sec
Fixation 0-0.02 at [(i=center,center)]
Target A 0.02 - 0.07 at [(i=center-3),(j=center+3)]
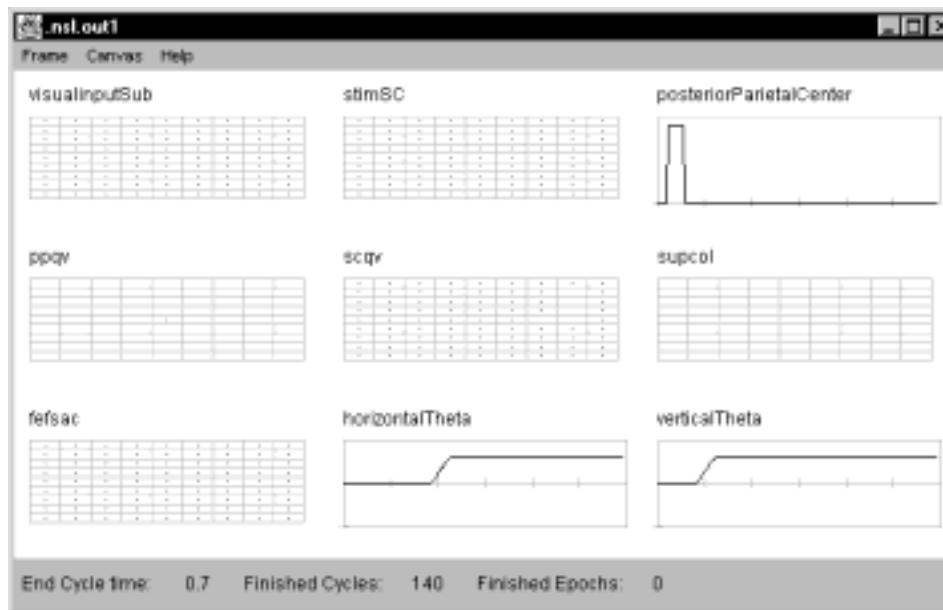Stimulation 0.07 - 0.11 at [(i=center-3),(j=center)]

As can be seen in figure 14.23, the stimulation of the SC causes the second target to be acquired first; while the lesioning of FEF causes some delay in making the saccade. However, in this group of experiments we also increase the strength of the projections from either the SC or the FEF (which ever was not lesioned) to the long lead burst neurons. The changes corresponding to the postoperative adaptation of the system reported by Schiller and Sandell (1983).

14.5 Summary

We have discussed the basis of our computer model and how NSL 3.0 has made it easier to represent and understand. In 1992 Dominey compared his simulated results as far as timing and output amplitude with the corresponding literature and found that they compared well. The original model demonstrates that:

1. The inhibitory projection from BG to SC allows selective cortical control of remembered target locations.
2. The topographic position codes in motor error maps of future saccades can be dynamically updated to account for ongoing eye movement (ppqv).
3. Saccades can be driven by memory that is hosted in reciprocal connections between FEF and the Thalamus.
4. Either the projection from SCS to the LLBNs or the projection from FEF to the LLBNs can trigger a saccade but in degraded mode.

This new model demonstrates the same concepts but at the same time does it in a more user friendly fashion. We have demonstrated that with the new NSL 3.0, one can represent neural areas in a more natural fashion, treating neural areas as objects or classes incorporating all of the features of one neural area into one section of the code instead of strewn about the code. We have added the "protocol" interface which allows us to switch from one protocol to another without leaving the simulator or loading a nsl script file. We have also added the ability to see the temporal plots with their X and Y markings within the Zoom Window. We have also added the ability to add new plots dynamically at run time for debugging the model dynamically. Also, not shown here, but another plot feature that can be used is color for encapsulating more information in one plot. Finally, we would like to offer that the ability to see the structure of the model before performing

an experimentis a very valuable tool since it allows the experimenter to better understand the model so that he/she does not waste time before performing possibly computer intensive and time consuming experiments with the model.

Notes

1. The Oculomotor model was implemented and tested under NSLJ.