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CHAPTER 1

Vying for dominance: dynamic interactions control visual fixation and saccadic initiation in the superior colliculus

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Abstract: Visual fixation and saccadic initiation are the tools that we use to explore the visual environment. In the superior colliculi, these behaviors may be viewed as independent motor programs that compete for control. Here, we provide a model describing the superior colliculi's involvement in visual fixation and saccadic initiation, in which extrinsic signals (sensory, motor, and cognitive) are shaped into competing motor programs through its local circuitry. In addition to providing evidence in support of this model, we demonstrate that it can explain the differences in the timing of saccadic initiation that have been observed experimentally.

Introduction

By the time you finish reading this sentence, your eyes will have made a series of rapid movements, separated by short intervals in which they are still (e.g., McConkie and Rayner, 1975 and Yang and McConkie, 2001). This alternating pattern of saccadic initiation and visual fixation, respectively, is not limited to activities such as reading text, but occurs whenever the high acuity foveae of the eyes are directed to visual objects of interest (e.g., Yarbus, 1967), which occurs, on average, hundreds of thousands of times a day.

A complex collection of brain areas controls these behaviors including regions of the parietal and frontal cortices, basal ganglia, thalamus, superior colliculus, cerebellum, and brainstem reticular formation (Fig. 1; for recent reviews see: Moschovakis et al., 1996; Leigh and Zee, 1999; Schall and Thompson, 1999; Hikosaka et al., 2000; Munoz et al., 2000; Glimcher, 2001; Scudder et al., 2002). Considering the number of brain areas that are involved, fully understanding the physiological basis of visual fixation and saccadic initiation will not be a simple feat. Nonetheless, significant progress has been made in understanding the basis of these behaviors in the superior colliculi, wherein visual fixation and saccadic initiation can be viewed as independent motor plans that compete for expression. In this chapter, we describe the neurophysiological mechanisms responsible for shaping competing motor plans and how these processes evolve across time. In addition, we will show that differences in the timing of saccadic initiation may be explained through these mechanisms.

Dynamic reciprocal interactions shape visual fixation and saccadic initiation

At the most basic level, the relationship between visual fixation and saccadic initiation can be viewed as a game between two competing processes. Understanding the game requires careful consideration of the field upon which these competing processes hap-

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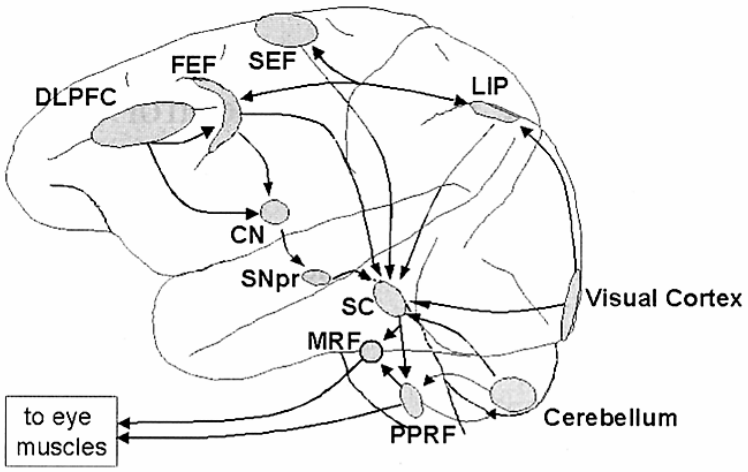

Fig. 1. Lateral view of monkey brain illustrating major areas that are involved in visual fixation and saccadic initiation. CN, caudate nucleus; DLPFC, dorsolateral prefrontal cortex; FEF, frontal eye fields; LIP, lateral intraparietal area; MRF, mesencephalic reticular formation; SC, superior colliculus; SEF, supplementary eye fields; SNpr, substantia nigra pars reticulata.

pen, the players involved, the mechanisms, or rules, governing these interactions, and watching the game unfold.

The intermediate layers of the superior colliculi are the fields upon which these interactions occur. At this depth, neurons are organized into a retinotopically coded motor map that specifies saccades into the contralateral visual field (e.g., Robinson, 1972). As illustrated in Fig. 2A, the amplitude of the saccade increases systematically along the rostral to caudal axis and the direction of the saccade shifts from upwards to downwards along the medial to lateral axis. Extrinsic input from sensory, motor, and cognitive areas modulates the activity of this map (Sparks and Hartwich-Young, 1989), which is shaped into independent motor plans, in part, through local inhibitory interneurons (Mize et al., 1991; Behan and Kime, 1996; Meredith and Ramoa, 1998; Munoz and Istvan, 1998; Olivier et al., 1999).

Broadly speaking, the competition is between two classes of neurons (Fig. 2B). Fixation neurons, located in the rostrolateral pole of the superior colliculus, produce tonic activity during visual fixation and pause during saccades (Munoz and Guitton, 1991; Munoz and Wurtz, 1993a). Saccadic neurons, located throughout the rest of the intermediate layers, produce bursts of action potentials prior to and during the execution of saccades made to their response field (Wurtz and Goldberg, 1972; Sparks et al., 1976; Munoz and Wurtz, 1995a). For ease of communication, the terms 'fixation' and 'saccadic' will be used throughout the rest of this chapter to describe neurons in and regions of the superior colliculus. It is important to realize, however, that this distinction is misleading for two reasons. First, rather than forming two distinct classes of neurons, fixation and saccadic neurons may consist of one class of neurons that encode actions (Munoz and Guitton, 1991; Munoz and Wurtz, 1995b; Krauzlis et al., 1997, 2000). The different characteristics expressed by these neurons arise from their position on the motor map. Fixation neurons respond to foveal and parafoveal input and encode actions specific to that location, maintaining gaze and initiating small ($<2°$) saccades. Saccadic neurons respond to more eccentric regions in the contralateral visual field and encode actions specific to that location, initiating saccades. Second, although the interactions between fixation and saccadic neurons will be focused upon in this review, it should be realized that competing motor plans can be formed anywhere across the motor map (Basso and Wurtz, 1997; Munoz and Istvan, 1998).

Two rules govern the interactions between the neurons in the intermediate layers. The first rule is that

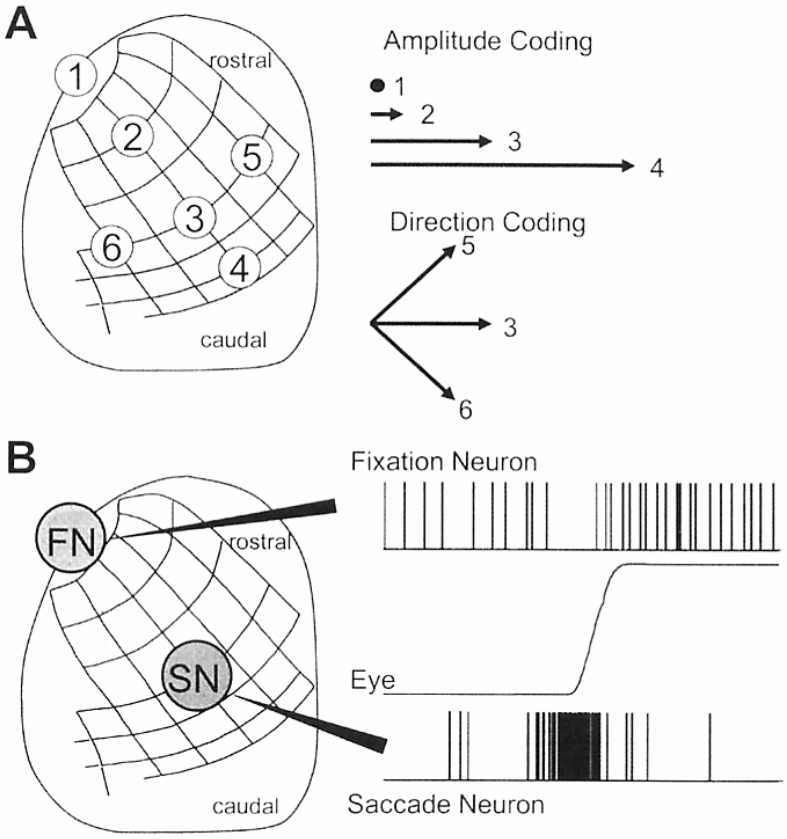

Fig. 2. Superior colliculus' retinotopic motor map. (A) Representation of amplitude (sites 1–4) and direction (sites 3, 5, 6) coding of rightward saccadic vectors in the left superior colliculus. (B) Typical discharge characteristics of a fixation neuron (FN) and a saccadic neuron (SN) in the left superior colliculus when monkey initiates a rightward saccade.

the activity of one region, or node, facilitates nodes that are nearby and inhibits nodes that are more distant, as illustrated in Fig. 3. The second rule is that the amount of activity expressed in the intermediate layers remains reasonably constant; with only the distribution of this activity changing. Therefore, if the activity of one node is strong, then the inhibition of distant nodes will be strong (solid line). If the activity of one node is weak, then the inhibition of distant nodes will be weak (dotted line).

'Watching' the game can be imagined with a simple example. The participant's job is to initiate saccades to visual targets that appear to the left side or to the right side of fixation. At the beginning of each trial, the participant maintains gaze at center and waits for the target to appear. Actively maintaining gaze constitutes a motor plan that is correlated with bilateral tonic activity in the fixation region in the rostral colliculi (Fig. 4A). Shortly after, a visual target appears to the right side. This sensory input enhances a point location in the left colliculus and changes the balance of activity across the intermediate layers: nearby nodes are facilitated and distant nodes are inhibited (Fig. 4B). Ultimately, the saccadic plan is initiated, which is correlated with a strong burst of action potentials in a caudal region of the left colliculus that shuts down the rest of the map in a winner-takes-all fashion (Fig. 4C).

If this dynamic interactions model is accurate, then altering the balance of activity in the interme-

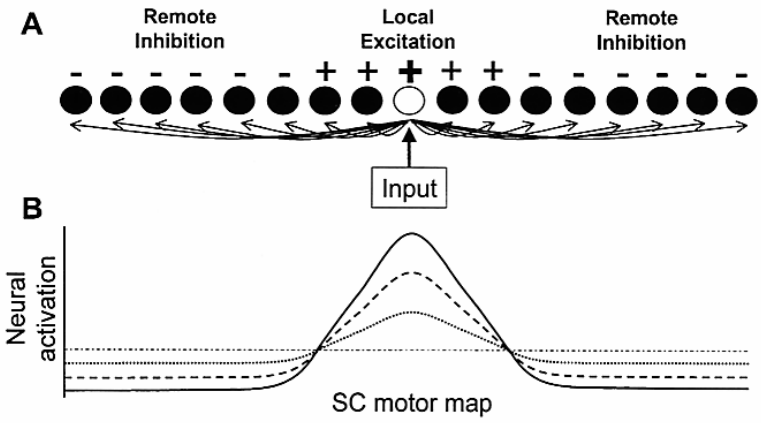

Fig. 3. Schematic representation of the dynamic interactions across the intermediate layers. (A) Local and distant interactions. Each circle represents an individual node. Extrinsic input to one node excites local nodes (plus signs) and inhibits distant nodes (minus signs). (B) Relative distribution of activity. The intensity of the extrinsic input alters the relative distribution of activity across the intermediate layers. See text for further details.

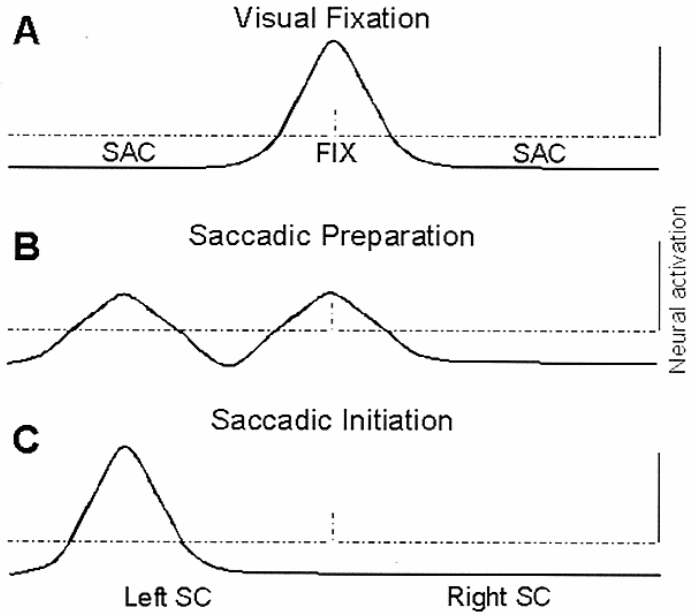

Fig. 4. Dynamic interactions unfold across time. Schematic representation of the distribution of activity across the intermediate layers of both colliculi during (A) visual fixation, (B) saccadic preparation, and (C) saccadic initiation.

diate layers, artificially, will have predictable consequences on neuronal excitability and behavior. This idea has been tested in two ways: (1) by facilitating confined regions of the intermediate layers with microstimulation and monitoring its influence on fixation and saccadic neurons located across the motor

map (Munoz and Istvan, 1998), and (2) by facilitating and inhibiting regions of the intermediate layers with different chemical compounds and observing the consequences on behavior (Hikosaka and Wurtz, 1985; Munoz and Wurtz, 1993b).

Munoz and Istvan (1998) recorded the activity of fixation and saccadic neurons while monkeys initiated saccades to eccentric targets. On a selected proportion of trials, different regions of the intermediate layers were stimulated electrically and the ensuing effects on fixation and saccadic neurons were observed. The timing of microstimulation depended on the class of neurons that was being monitored. When fixation neurons were monitored, stimulation was applied when the monkey was actively fixating the central fixation marker (e.g., Fig. 4A). When saccadic neurons were monitored, stimulation was applied when a saccade was being initiated into the neuron's response field (e.g., Fig. 4C). Four regions of the superior colliculus were stimulated relative to the recording site; fixation regions or saccadic regions were stimulated on the same side as the recording electrode or on the opposite side as the recording electrode.

The findings from this study were consistent with the dynamic interactions model. Stimulating saccadic regions inhibited both fixation (Fig. 5A) and saccadic (not shown) neurons that were distant from the stimulation locus at very short latencies. Stimulating fixation regions inhibited saccadic neurons at very short latencies (Fig. 5B). In both instances, the inhibition was more potent and was evident earlier when the ipsilateral side was stimulated compared to the contralateral side. Importantly, stimulating one fixation region excited fixation neurons on the contralateral side (Munoz and Istvan, 1998). Based on these data and complementary studies in anaesthetized animals (McIlwain, 1982) and reduced preparations (Meredith and Ramoa, 1998), artificially enhancing specific regions of the intermediate layers with microstimulation, inhibits nodes that are distant and facilitates nodes that are, functionally, nearby.

Modifying the distribution of activity across the intermediate layers with pharmacological manipulations also has predictable consequences on behavior. Increasing the activity of one region should facilitate actions that are supported by that region and inhibit actions made elsewhere, whereas decreasing the activity of one region should inhibit actions that are supported by that region and facilitate actions made elsewhere. These predictions have been confirmed. Facilitating the fixation region with bicuculline (black bars in Fig. 6A), a $GABA_A$ antagonist, increased the reaction time of saccades (Munoz and Wurtz, 1993b). Inhibiting the fixation region with a microinjection of muscimol (gray bars in Fig. 6A), a $GABA_A$ agonist, decreased saccadic reaction times (Munoz and Wurtz, 1993b). As illustrated in Fig. 6B, the opposite pattern was obtained when the excitability of saccadic neurons was altered. Microinjection of bicuculline into a saccadic region shortened saccadic reaction times, while injecting muscimol increased saccadic reaction times (Hikosaka and Wurtz, 1985). Thus, modifying the activity of specific regions of the motor map affects behavior in a manner that is consistent with the dynamic interactions model.

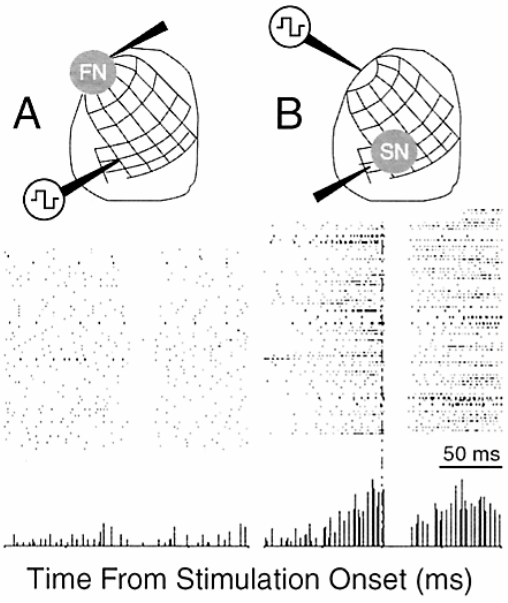

Fig. 5. Neurophysiological evidence supporting dynamic interactions model. (A) Stimulating saccadic regions produces potent, short-latency inhibition of fixation neurons (FN). (B). Stimulating fixation regions produces potent, short-latency inhibition of saccadic neurons (SN). Rasters and histograms are aligned on stimulation onset (dashed vertical line). Adapted from Munoz and Istvan (1998).

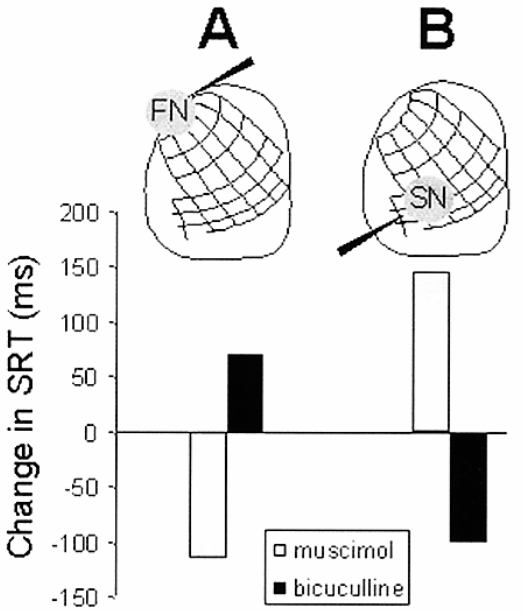

Fig. 6. Pharmacological manipulations support dynamic interactions model. (A) Modulating activity of fixation regions with muscimol (gray bars) decreases saccadic reaction times, whereas bicuculline (black bars) increases saccadic reaction times. (B) Modulating activity of saccadic regions with muscimol increases saccadic reaction times, whereas bicuculline decreases saccadic reaction times. Data adapted from Munoz and Wurtz (1993b) and Hikosaka and Wurtz (1985).

Up to this point, the evidence that we have described speaks to distant interactions across the intermediate layers, in most instances. Yet, the dynamic interactions model also makes specific predictions for local interactions. Using a different technique, we have probed both local and distant interactions across the intermediate layers. Behavioral evidence has demonstrated that presenting an irrelevant distractor influences responding to a target and its influence depends on the relative distance between target and distractor. If the distractor appears close to the target, then saccadic reaction time is reduced. If the distractor appears far from the target, then saccadic reaction time is increased (Walker et al., 1997; Olivier et al., 1999). These behavioral observations match the dynamic interactions model perfectly. Presenting a distractor will excite neighboring nodes in the intermediate layers, enhancing their responsiveness to a nearby target. Presenting a distractor will inhibit distant nodes, impairing their responsiveness to a remote target.

The activity of saccadic neurons was monitored as monkeys performed the distractor task that was described above (Olivier et al., 1999). As illustrated in Fig. 7, the results from this study provided compelling support for the dynamic interactions model. Compared to the control condition, in which no distractor was presented (Fig. 7A), presenting a distractor close to the target caused a saccade to be initiated immediately following presentation of the target (see arrow Fig. 7B), whereas presenting a distractor far from the target inhibited the low frequency activity of the neuron, making it more difficult for the neuron to reach threshold (see arrow Fig. 7C).

All told, a picture emerges in which the alternating pattern of visual fixation and saccadic initiation arises, at least in part, from dynamic interactions across the intermediate layers of the superior colliculi. Extrinsic input selectively enhances confined regions of the intermediate layers that changes the distribution of activity elsewhere in the map in a push–pull fashion: nearby nodes are facilitated and distant nodes are inhibited (see Fig. 3). These dynamic interactions shape competing motor plans across the intermediate layers. When one of these plans reaches threshold, it produces a strong burst of activity and inhibits other regions in a 'winner-take-all' fashion, allowing a saccade to be initiated (see Trappenberg et al., 2001 for implementation of the model). Enhancing or inhibiting specific regions of the intermediate layers has provided compelling support for this model.

Evidence supporting dynamic reciprocal interactions: behavioral anomalies explained

As a model of motor planning, saccadic eye movements are an ideal action to study because they are very stereotyped. However, this characteristic also makes it more difficult to push the boundaries of the dynamic interactions model to see if it can explain the exception to the rule, as well as the rule itself. In this section, we describe several instances in which the timing of saccadic initiation has been altered, allowing us to probe the predictions of the dynamic interactions model in these boundary conditions.

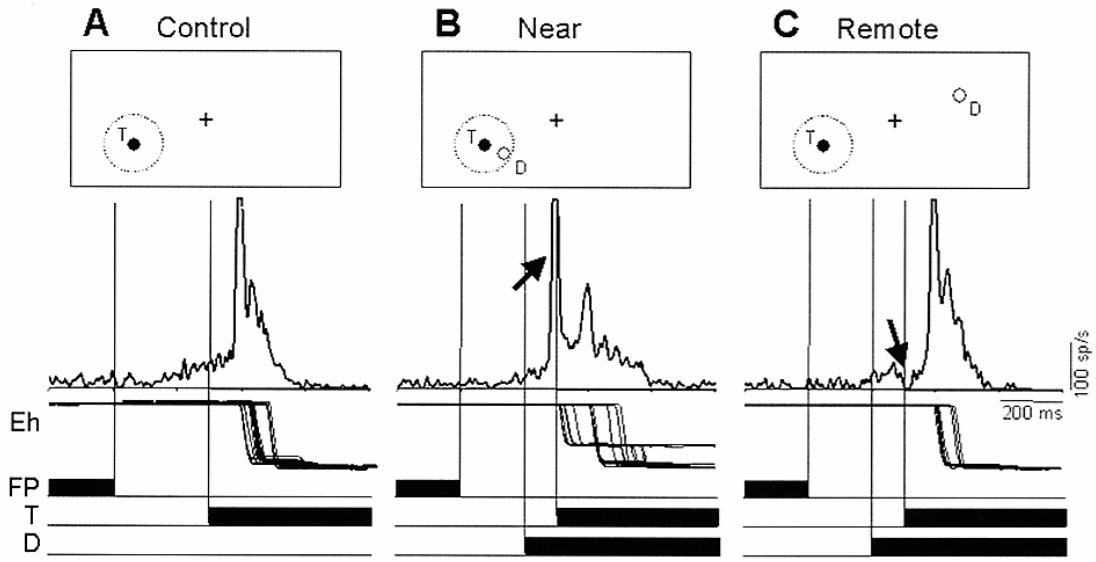

Fig. 7. Distractor effects on pretarget activity of saccadic neurons support dynamic interactions model. (A) Control trials reveal buildup of pretarget activity that precedes target- and saccade-related bursts after the target appears (black circle) in the neuron's response field (dotted circle). (B) Presentation of near distractor (gray circle) 100 ms before target appearance facilitates neuron (marked by black arrow) and often triggers express saccades to the distractor. (C) Remote distractor inhibits neurons (marked by black arrow) and delays the onset of the saccade. Illustrated from top to bottom in each panel are a schematic of target and distractor locations, the spike density waveform, horizontal eye position (Eh), and traces depicting the timing of fixation point (FP) disappearance, target (T) and distractor (D) appearance.

Before doing so, a brief reminder of the basic saccadic task will be provided because minor modifications to this task change the timing of saccadic initiation. In this task (Fig. 4), each trial begins with the monkey fixating a central marker. A target then appears to an eccentric position in the visual field to which the monkey initiates a saccade. In the following subsections, the modifications made to this paradigm, the corresponding behavioral and neurophysiological consequences, and the interpretation of these changes, with regard to the dynamic interactions model, are described.

The gap effect

The 'gap effect' refers to the observation that inserting a short temporal gap in between the disappearance of the fixation point and the appearance of a target reduces saccadic reaction times (Saslow, 1967). Two, not mutually exclusive, theories have been provided to explain this effect. First, the gap may act as a non-localized warning signal, increasing the observer's readiness to respond to the target (e.g., Kingstone and Klein, 1993; Paré and Munoz, 1996). Second, the gap may disengage attention, or oculomotor fixation, which reduces saccadic reaction times because one step involved in initiating the saccade has been eliminated (Mayfrank et al., 1986; Fischer and Weber, 1993).

Within the dynamic interactions framework, readiness and oculomotor disengagement have straightforward neurophysiological consequences. 'Readiness' may be evidenced as non-localized extrinsic input that modulates the excitability of the intermediate layers. 'Oculomotor disengagement' may be evidenced as decreased activity of fixation neurons.

Indeed, such neurophysiological correlates have been observed in the intermediate layers when monkeys perform the gap saccade task. Before we describe these effects fully, we will first provide additional detail about the characteristics of fixation and saccadic neurons because understanding these char-

acteristics helps reveal the consequences of 'readiness' and 'oculomotor disengagement'.

Up to now, two 'classes' of neurons have been described in the intermediate layers, fixation neurons that represent foveal and parafoveal regions and saccadic neurons that represent more eccentric regions of the motor map. This simplistic representation is somewhat misleading because saccadic neurons have additional characteristics that can be used to divide them into different categories, including (1) the presence or absence of long-lead activity prior to saccadic initiation, (2) the shape of their movement fields, and (3) the depth below the dorsal surface of the superior colliculus (Munoz and Wurtz, 1995a,b) [1]. Burst neurons produce brief, high frequency discharges for saccades that fall within a small range of amplitudes and directions (i.e., closed movement field) and tend to reside more superficially in the intermediate layers. Buildup neurons often begin firing well before saccadic initiation, discharge for all saccades whose amplitudes are equal to or greater than their optimal (i.e., open-ended movement fields) and tend to reside deeper in the intermediate layers. Fixation neurons, like buildup neurons, are located in deeper parts of the intermediate layers and have open-ended movement fields.

The characteristic firing pattern of each neuron type is illustrated in Fig. 8 for the gap saccade task. Before the target appears, fixation neurons (Fig. 8B) are tonically active when the fixation marker is visible (left side of left vertical line) and this activity continues during the gap period, albeit at a reduced rate (right side of left vertical line); burst neurons (Fig. 8C) are silent during this interval, and buildup neurons (Fig. 8D) begin to discharge at a low frequency tonic rate during the gap period. After the target appears (right side of right vertical line), a saccade is initiated. At the time of saccadic initiation, fixation neurons pause and burst and buildup neurons produce a high frequency burst of action potentials. Owing to the shared characteristics of buildup and fixation neurons, we have proposed that these neurons participate in the dynamic interactions across the intermediate layers, whereas burst neurons amplify the saccade-related signals (Wurtz and Munoz, 1995; Trappenberg et al., 2001). We mention this difference because the neutal correlates of readiness and oculomotor disengagement are manifest in the low frequency, tonic activity of fixation and buildup neurons.

We explored the neurophysiological consequences of the gap effect on the dynamic interactions between fixation and buildup neurons (Dorris and Munoz, 1995; Dorris et al., 1997). If fixation and buildup neurons do interact in a reciprocal manner, then removing the fixation marker should decrease the activity of fixation neurons and increase the excitability of buildup neurons. As illustrated in Fig. 9A, this exact pattern was observed. From a relatively stable rate of discharge during visual fixation, the activity of fixation neurons began to decline about 100 ms after the fixation marker disappeared and reached a nadir 200–300 ms into the gap before rising toward a level slightly less than that observed during visual fixation. Correspondingly, the activity of buildup neurons increased during the gap starting ~100 ms after the fixation marker disappeared and reached a maximum rate about 250 ms into the gap period. As illustrated in Fig. 9B, the mean saccadic reaction times measured from trials with different gap durations followed the same pattern as the activity recorded from fixation and buildup neurons for long (≥600 ms) gap duration trials. That is, mean saccadic reaction times were lowest for gap durations of 200–300 ms, when the pretarget fixation activity was at its minimum and the buildup activity was at its maximum.

In summary, the gap effect can be interpreted easily within our model. The gap has two interactive influences; it decreases the tonic activity of fixation neurons and increases the low frequency, pretarget activity of buildup neurons. These reciprocal interactions in the intermediate layers produce the shorter saccadic reaction times that are observed in behavior.

Motor preparation

One shortcoming of the 'readiness' explanation is that the low frequency activity of buildup neurons can be localized in some circumstances (Glimcher and Sparks, 1992). Thus, rather than being an unlo-

[1] Like fixation and saccadic neurons, burst and buildup neurons may represent opposite ends of one continuum of neurons rather than two distinct classes of neurons.

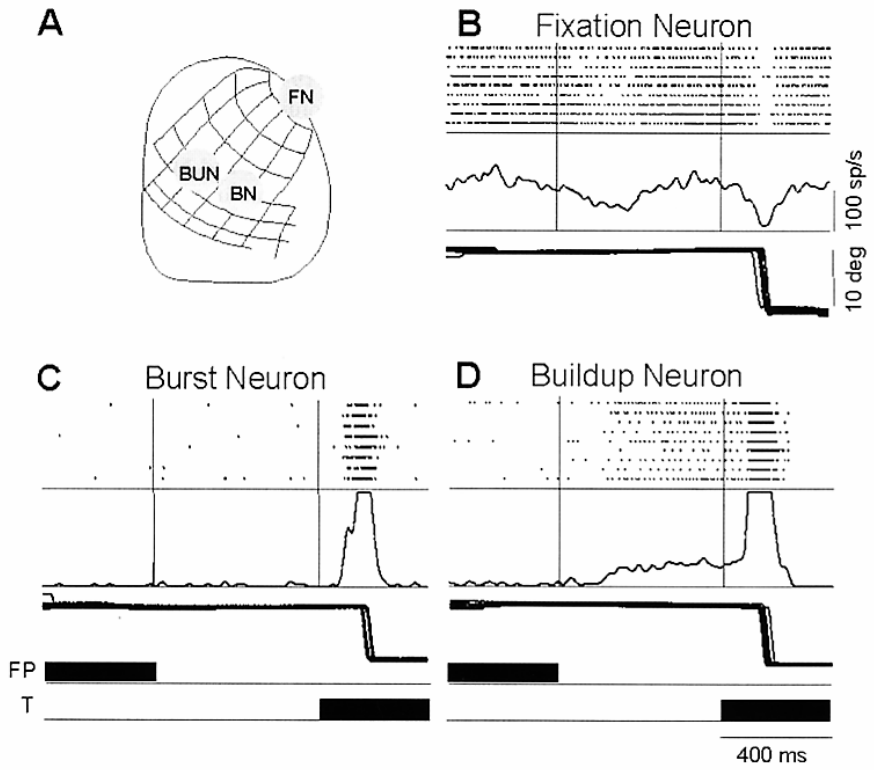

Fig. 8. Characteristic discharge patterns of a fixation neuron (B), a burst neuron (C), and a buildup neuron (D) during the gap saccade task. In this example, the fixation point (FP) disappears (left vertical line) 600 ms before the target (T) appears (right vertical line).

calized 'readiness' signal, this low frequency activity may signify 'motor preparation' that is specific to potential target locations. Considering that only two target locations were used in the gap saccade task (Dorris et al., 1997) and that multiple motor plans can be programmed concurrently (Basso and Wurtz, 1997; McPeek and Keller, 2002), this notion has substantial merit. We tested this question by manipulating the probable location of the target in the gap saccade task, which could appear in two locations equally often, or in one location 100% of the time (Dorris and Munoz, 1998). If the low frequency pretarget activity is a sign of 'readiness', then it should be similar in all three conditions. If, however, the low frequency activity is a sign of 'motor preparation', then the buildup activity should be greatest when the target appears in the neuron's response field 100% of the time, intermediate when the target appears in the neuron's response field 50% of the time, and virtually absent when the target never appears in the neuron's response field.

As illustrated in Fig. 10A, the findings from this study were entirely consistent with a motor preparation explanation. In addition, this finding supports the dynamic interactions model because the level of pretarget activity in the 100% in response field condition was approximately double of that observed in the 50% in response field condition, suggesting that the level of activity is being divided between two locations in the latter case (Fig. 10B).

Indeed, if the activity codes motor preparation, then greater pretarget activity should be correlated with shorter saccadic reaction times. This is precisely what was observed when pretarget activity of buildup neurons and saccadic reaction times were compared on a trial-by-trial basis (Dorris and Munoz, 1998).

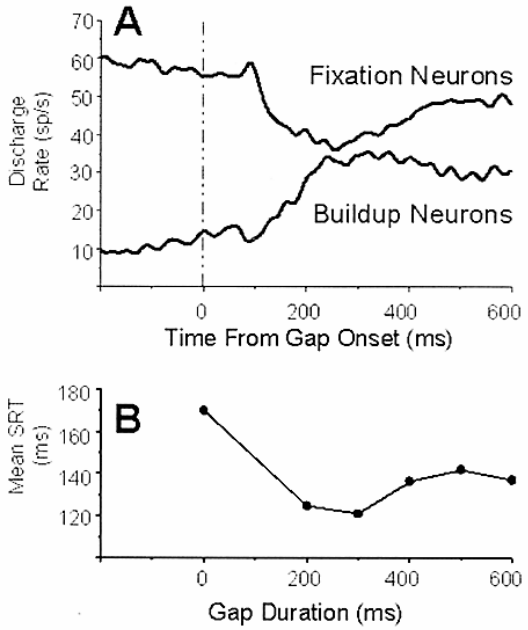

Fig. 9. Gap effect on pretarget activity and saccadic reaction times. (A) Population responses of fixation and buildup neurons in the gap saccade task with a gap period 600 ms. (B) Mean saccadic reaction times of trials with gap periods of 0, 200, 300, 400, 500, and 600 ms.

Thus, pretarget activity represents an early motor preparation signal that prepares the oculomotor system for an impending saccade.

Express saccades

Combining a gap period with a probability manipulation has the additional consequence of creating a bimodal distribution of saccadic reaction times, as illustrated in Fig. 11A. Regular latency saccades (>150 ms) exceed the minimum afferent and efferent conduction times that are needed for visual input to initiate a saccade (Carpenter, 1981). Express saccades (~100 ms), on the other hand, approach the minimal time required for sensory–motor transformation to occur (Fischer and Weber, 1993).

The gap effect describes the speeding up of regular saccades whenever a gap is introduced (Saslow, 1967). Express saccades, alternatively, require additional factors to be obtained, including a predictable target location and temporal regularity of its appearance (Fischer et al., 1984; Boch and Fischer, 1986; Rohrer and Sparks, 1993; Sommer, 1994; Paré and Munoz, 1996). Accordingly, we have hypothesized that express saccades require advanced motor preparation to be obtained (Paré and Munoz, 1996). Put into context of the dynamic interactions model, extrinsic preparatory input to a specific region of the motor map combined with increased excitability of saccadic regions (owing to decreased activity of fixation neurons) will produce express saccades when an abrupt visual transient appears at the location being prepared (Paré and Munoz, 1996).

Regular and express saccades have markedly different neurophysiological signatures (Edelman and Keller, 1996; Dorris et al., 1997). Regular saccades are associated with two bursts of action potentials, one in response to the target's appearance and one in response to the motor action (Fig. 11C). Express saccades, on the other hand, are associated with one burst of action potentials, a combined visuomotor burst (Fig. 11B).

We wondered if differences in pretarget activity might account for the differences between regular and express saccades. This appears to be the case (Dorris et al., 1997). Regular saccades were generated when pretarget activity was low. Express saccades were generated when pretarget activity was high. In all likelihood, pretarget activity may determine how close the system is to achieving threshold for saccadic initiation (Fig. 12). When pretarget activity is low, the target-related response does not exceed the saccadic threshold and a later signal is required to trigger a saccade. When pretarget activity is high, the target-related response exceeds saccadic threshold and triggers a saccade at express latency.

In summary, express saccades are a special instance of saccadic initiation, in which localized input to a particular region of the motor map elevates pretarget activity to a high level, allowing the appearance of a visual target to elicit a saccade immediately. Thus, express saccades depend on a unique combination of dynamic interactions that permit the immediate initiation of a saccade to a visual target.

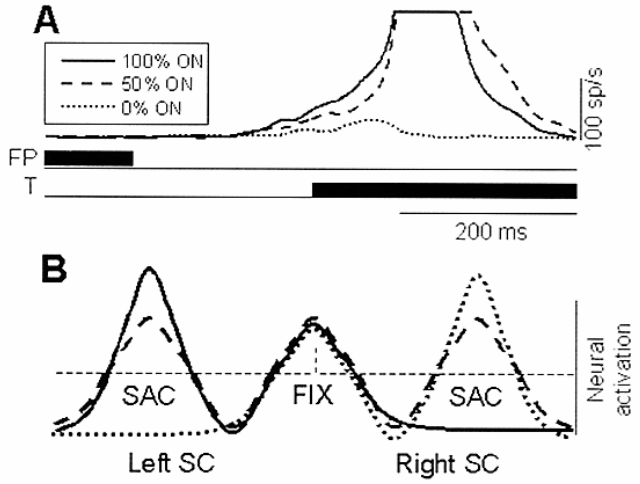

Fig. 10. Probability effects on pretarget activity of buildup neurons. (A) Spike density waveform of a buildup neuron when the target appeared in the neuron's response field 100% (solid line) 50% (dashed line) or 0% (dotted line) of the time. (B) Schematic representation of pretarget activity across intermediate layers when the probability of the target location is manipulated within a block of trials: 100% right (solid line), 100% left (dotted line), 50% right and 50% left (dashed line). Adapted from Dorris and Munoz (1998).

Antisaccades

Up to now, all of the tasks that have been described share one common feature, i.e., saccades were directed to a visual target. However, the relationship between visual targets and saccades can be more flexible than this. Indeed, a saccade may be initiated to any region of the visual field, even when a competing visual target is presented at the same time. The antisaccade task specifically assesses this ability. In this task, participants must (1) suppress the urge to initiate a saccade to a visual target, and (2) initiate a saccade to the target's mirror location (Hallett, 1978). Antisaccades provide a novel way of exploring the dynamic interactions across the intermediate layers because sensory and motor signals are dissociated and, therefore, are being represented in competing regions of the motor map in opposite colliculi.

We explored this possibility by monitoring fixation and saccadic neurons in the intermediate layers, while monkeys initiated prosaccades (saccade to target) and antisaccades (saccade away from target) in the same block of trials (Everling et al., 1998, 1999; Bell et al., 2000). The color of the fixation marker indicated the response that was required on each trial. In addition, a 200 ms gap was presented on half of the trials to further change the dynamics of the intermediate layers during this task.

Saccadic reaction times were longer and the number of errors was greater for antisaccades than for prosaccades, as illustrated in Fig. 13. Introducing a gap enhanced this difference, by increasing the number of direction errors that were made on antisaccade trials.

The neural correlates of prosaccades and antisaccades were compared in several different epochs (Everling et al., 1998, 1999). Understanding the purpose of each epoch requires breaking down a single trial into its basic components. The appearance of the fixation marker has two consequences: (1) it instructs the response (i.e., prosaccade or antisaccade) that is required on each trial and (2) it warns the participant that a target will appear soon. Thus, the fixation epoch induces a cognitive set for the upcoming trial. Then, the target appears or the fixation marker is removed for 200 ms before the target appears. As has been noted above, presenting or not presenting a gap modifies the excitability of saccadic regions in the intermediate layers. The target's position specifies the location to which a saccade must be initiated and then the saccadic plan is generated.

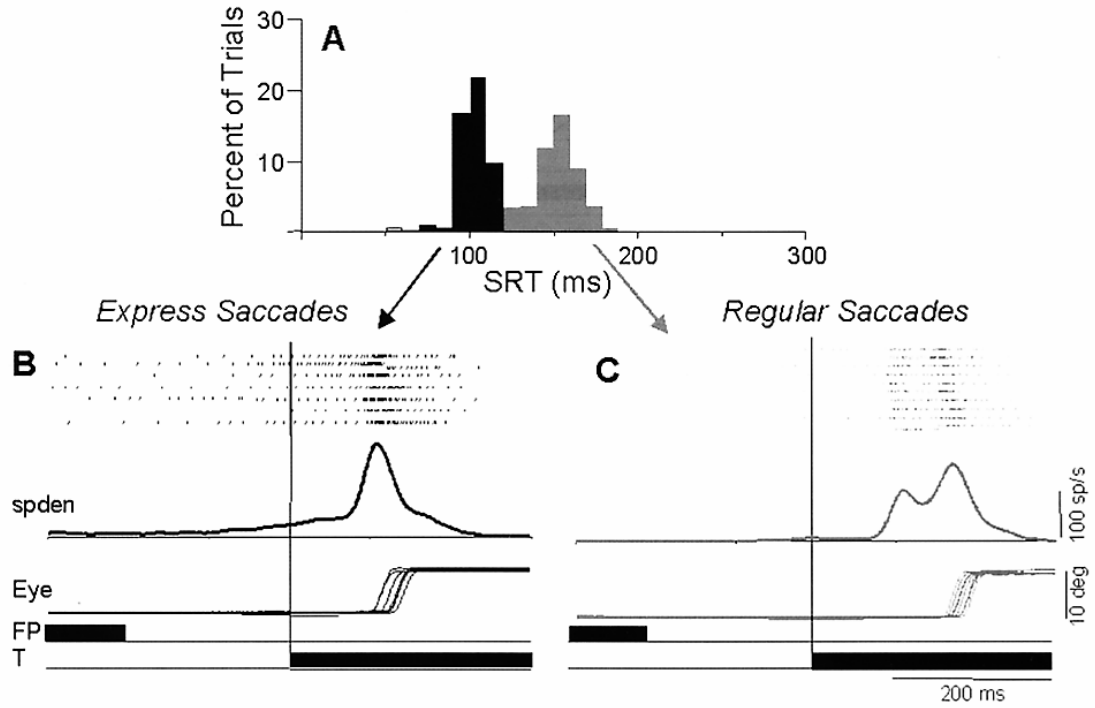

Fig. 11. Neurophysiological correlate of distribution of saccadic reaction times. (A) Bimodal distribution of saccadic reaction times in the gap saccade task. Neurophysiological signatures of (B) express saccades and (C) regular saccades recorded from a buildup neuron. Adapted from Dorris et al. (1997).

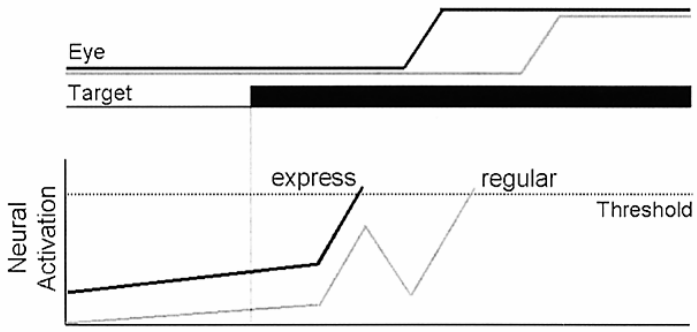

Fig. 12. Schematic representation of neural activity as it accumulates towards saccadic threshold. For express saccades, a high level of pretarget activity is combined with a target-related response and threshold for a saccade is met. For regular saccades, lower pretarget activity does not allow the target-related response to reach saccadic threshold; and a later saccade-related signal is required.

Fig. 14 illustrates the activity of a fixation neuron during prosaccade and antisaccade trials in the overlap (Fig. 14A) and in the gap (Fig. 14B) conditions. The activity of the fixation neuron changed depending on the trial that was being performed (Everling et al., 1999). Upon presentation of the fixation marker,

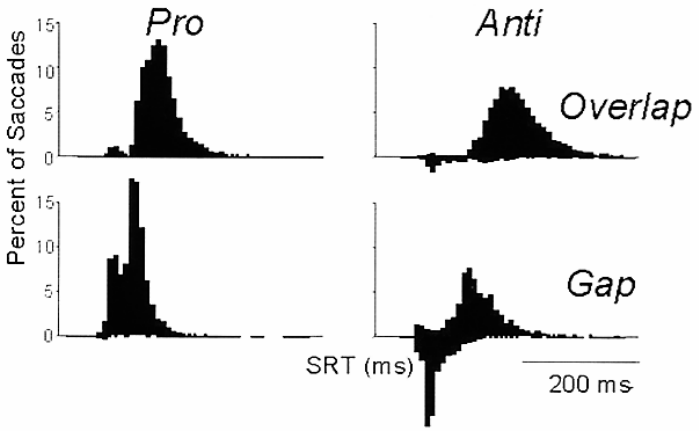

Fig. 13. Distribution of saccadic reaction times in the combined pro-/anti-saccade task. Values above *x*-axis represent correct responses, values below *x*-axis represent direction errors. Adapted from Bell et al. (2000).

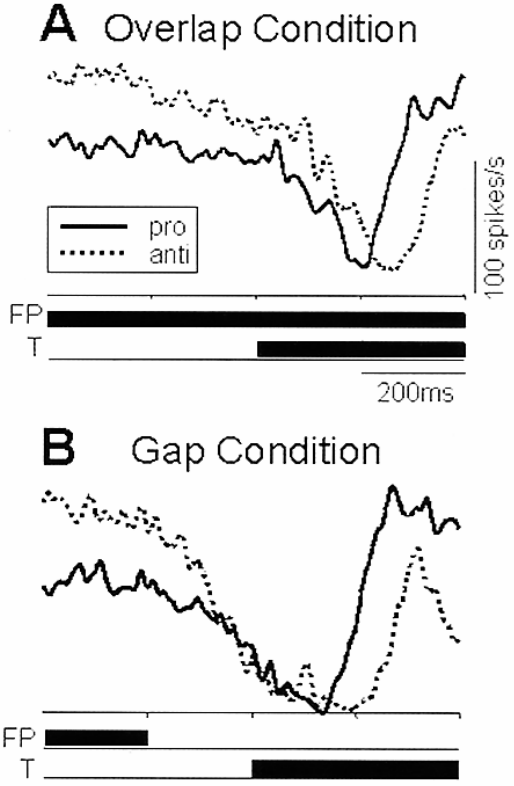

Fig. 14. Discharge of a fixation neuron in the combined pro-/anti-saccade task for overlap (A) and gap (B) conditions. Adapted from Everling et al. (1999).

the fixation neuron elicited more activity for antisaccade trials than for prosaccade trials. In the overlap condition (fixation marker illuminated throughout entire trial), this heightened level of tonic activity was maintained into the target epoch (Fig. 14A). In the gap condition, the tonic activity of the fixation neuron decreased during the gap period to the same rate in prosaccade and antisaccade trials (Fig. 14B). The fixation neuron then paused during the saccade in all conditions (Fig. 14).

Fig. 15 illustrates the activity of a buildup neuron during prosaccade and antisaccade trials in the gap condition when the target appeared in the neuron's response field (Fig. 15A) or the saccade was directed into the neuron's response field (Fig. 15B). The activity of the buildup neuron was greater for prosaccade trials than for antisaccade trials (Everling et al., 1999), which was particularly notable for target-related and saccade-related discharges. In addition, less pretarget activity was observed on antisaccade trials. A similar pattern of findings was obtained for the overlap condition (not shown).

These findings are consistent with the dynamic interactions model. For prosaccades, the target appeared in and the saccade was initiated to the same location. As a consequence, there was no disparity between the two signals and strong bursts of action potentials were elicited for both. For antisaccades, however, the target appeared in and the saccade was generated to opposite fields. As a consequence, the

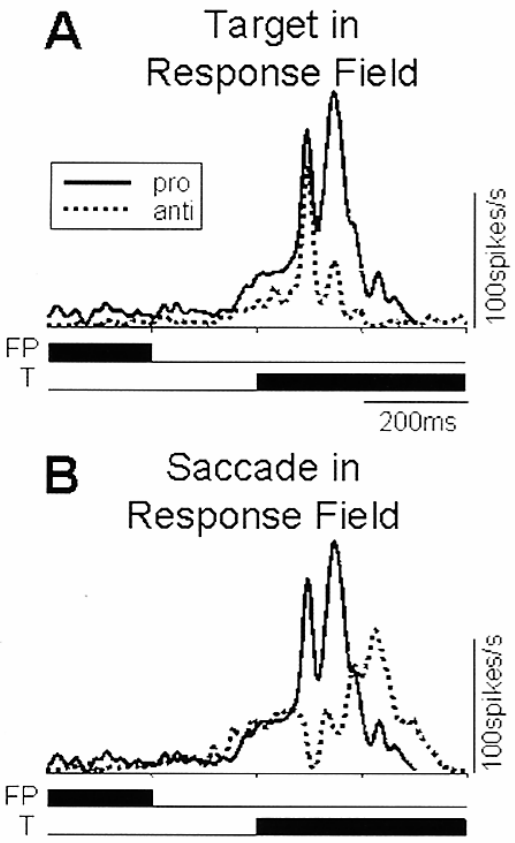

Fig. 15. Discharge of a buildup neuron in the combined pro-/anti-saccade task in the gap condition for trials in which the target (A) or the saccade (B) was directed into the neuron's response field. Adapted from Everling et al. (1999).

target-related and saccade-related signals were in competing regions of the motor map. These competitive interactions were responsible for the delay in saccadic reaction times for antisaccade trials (see Fig. 13). Also consider that pretarget activity may be detrimental for generation of correct antisaccade responses because, when the target-related response is added, this combined activity could exceed saccadic threshold (see Fig. 12) and a saccade would be generated to the target's location. For antisaccades initiating a saccade to the target is an error.

If this explanation is accurate, then comparing correctly executed and incorrectly executed responses

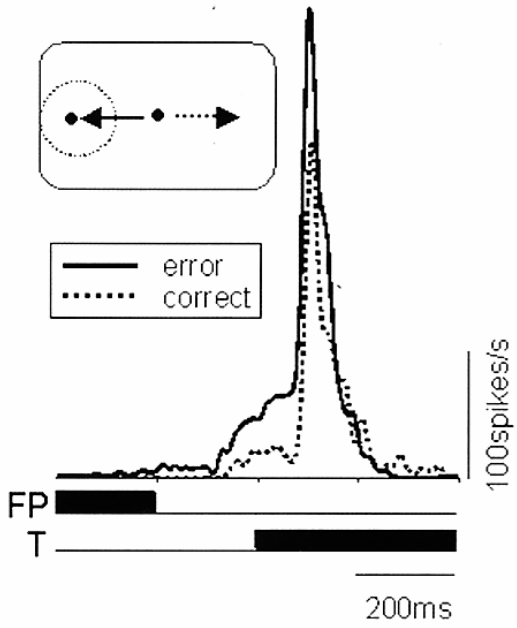

Fig. 16. Discharge of a buildup neuron for correct and error trials initiated in the anti-gap condition in which the target was located in the neuron's response field and the correct response was to look away. Adapted from Everling et al. (1998).

should reveal greater pretarget activity when an error is made. As illustrated in Fig. 16, this expected pattern was obtained (Everling et al., 1998). More pretarget activity was observed for incorrectly executed trials. In many instances, the elevated pretarget activity was sufficient to initiate a saccade at express latency.

In summary, antisaccades have a special influence on the dynamic interactions across the intermediate layers because fixation neurons, saccadic neurons that encode the visual target, and saccadic neurons that encode the motor action act as competing motor plans. Fig. 17 depicts the dynamic interactions across the intermediate layers for prosaccade and antisaccade responses. During visual fixation (Fig. 17A), fixation neurons are more active on antisaccade trials, leading to greater inhibition of saccadic neurons. In this case, greater fixation-related activity is beneficial because it makes it harder for the visual target to trigger a saccade, allowing a saccade to be executed in the opposite direction. When the target appears to

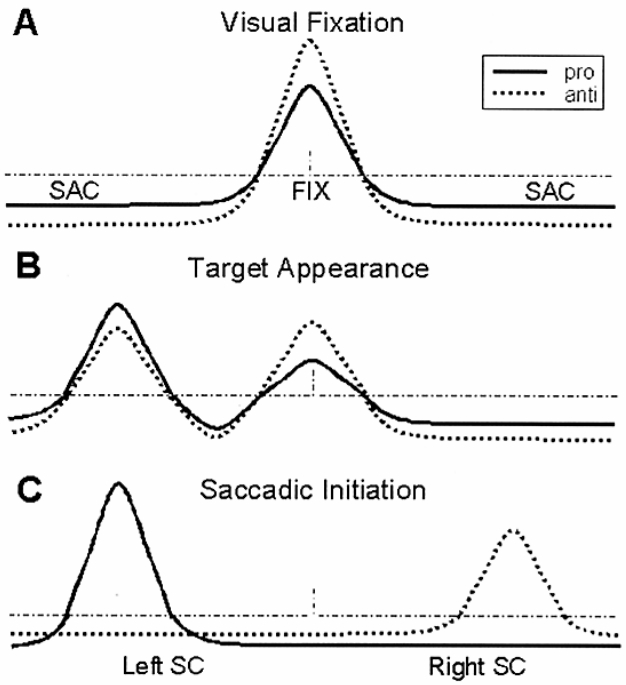

Fig. 17. Schematic representation contrasting activity on collicular motor map for prosaccade (solid lines) and antisaccade (dotted lines) trials.

the right side (Fig. 17B), it elicits a burst of action potentials in the left colliculus. This target-related activity is attenuated for antisaccade trials because the fixation-related activity is still exerting a strong influence on the interactions across the intermediate layers. Eventually a saccade is planned and this plan is initiated. For prosaccade trials, a large burst of action potentials is obtained (Fig. 17C). However, for antisaccade trials, a saccade is planned to the left side (right colliculus) that competes with the target-related activity in the left colliculus and the residual fixation-related activity at the fovea. These competing signals attenuate the saccade-related activity (Fig. 17C) and delay the development of the saccadic burst activity, leading to longer reaction times for antisaccades.

Summary

By the time you have reached this point, your daily count of alternating saccades and fixations will have increased considerably. So too will have your understanding of the dynamic interactions model.

In the superior colliculi, visual fixation and saccadic initiation may be viewed as independent motor plans that compete for dominance across the intermediate layers. Extrinsic input modifies a point location on the retinotopic motor map that is shaped into a motor plan through the intrinsic circuitry of the superior colliculi. Independent motor plans compete for selection in a push–pull fashion and when a saccadic plan ultimately reaches threshold, it produces a strong burst of action potentials that shuts down the remaining regions of the intermediate layers.

Modifying the activity of the intermediate layers changes these dynamic interactions in predictable ways. Enhancing the activity of one region facilitates nearby locations and inhibits distant locations. Diminishing the activity of one region inhibits nearby locations and facilitates distant locations. Such effects have been demonstrated in the neurophysiological activity of single cells (Munoz and Istvan, 1998;

Olivier et al., 1999) and in behavior (Hikosaka and Wurtz, 1985; Munoz and Wurtz, 1993b).

In addition to explaining visual fixation and saccadic initiation during basic saccadic tasks, the dynamic interactions model can explain changes in the timing of saccadic initiation that are observed when this task is modified. Namely, the gap effect, or decreased saccadic reaction times as a consequence of a gap period, occurs because removing fixation decreases the activity of fixation regions and, correspondingly, increases the excitability of saccadic regions. Express saccades, are a special instance of such dynamic interactions, in which decreased fixation activity and heightened motor preparation signals cause the target-related activity to be translated into a saccadic signal immediately. Finally, the slowing of saccadic initiation for antisaccades, can be interpreted as the consequence of multiple competing signals across the intermediate layers.

It should be emphasized that the dynamic interactions that we have described in this chapter are not limited to the superior colliculi. On the contrary, similar interactions take place at many levels of the neuraxis (Moschovakis et al., 1996; Leigh and Zee, 1999; Schall and Thompson, 1999; Hikosaka et al., 2000; Munoz et al., 2000; Glimcher, 2001; Scudder et al., 2002). At this juncture, however, the dynamic interactions involved in producing visual fixation and saccadic initiation are better understood in the superior colliculi because of its well-organized motor map and its well-characterized neuronal elements. Although we are a long way from understanding how the brain controls visual fixation and saccadic initiation, we have made substantial progress in understanding these behaviors in the superior colliculi.

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