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40 The Superior Colliculus and the Cognitive Control of Movement

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ABSTRACT Normal vision consists of an alternation of saccadic eye movements and periods of visual fixation. This is a relatively simple alternation of movement and nonmovement, but the control of this alternation involves many of the cognitive issues underlying the performance of more complex actions. The superior colliculus (SC) is a nexus in the brain system controlling these saccades and fixations, and recent experiments have shown how this structure and its cortical inputs contribute to their control. Activity on the SC movement map shows that the amplitude and direction of the impending saccade is most likely to be represented by the vector average across a large population of neurons. The control of the alternation of movement and nonmovement, which in the case of the saccadic system is the alternation of saccades and fixation, involves the balance of competition between SC neurons tonically active during visual fixation (fixation neurons) and those active long before saccades (buildup neurons). The preparation to move may be represented by the long lead or delay activity of buildup neurons because, as the probability that a saccade will be made increases, this delay activity increases. Whether the processing preceding movement initiation is discrete for each area or is distributed across areas can be answered by comparing neuronal activity in the SC with that of the neurons in frontal and parietal cortices that project to the SC. The processing is distributed with overlap between neuronal activity in cortex and SC and a shift in the SC toward movement preparation.

Rapid or saccadic eye movements shift our line of sight from one part of the visual field to another and allow us to direct the higher visual acuity provided by the fovea toward successive points in the field. We make these eye movements as frequently as two times per second during such tasks as scanning the visual scene or reading. Just as important as the saccades, however, is the lack of movement between them because it is in these periods of fixation when almost all vision occurs. Thus, saccades and fixations can be regarded as an integrated system that both moves the eye rapidly to the next target and then holds the eye steadily on that target. This alternation of saccades and fixation is particularly evident during reading (figure 40.1).

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Even given that saccades and fixation are necessary for normal vision, why should such a relatively mechanistic system be of any relevance to cognitive neuroscience? There are at least three reasons.

First, saccades are relatively simple movements, and they therefore offer the opportunity to understand a simple system within the brain for generating actions. The saccades involve rotation of each eye by the coordinated activation of only six muscles, have no variation of load—and therefore have no need for load compensation—and have no complexity introduced by the movement of joints (Robinson, 1968). These relatively simple movements do, however, involve most of the cognitive issues underlying the performance of more complex actions. For example, a saccadic eye movement to a visual stimulus requires shifting attention and selecting a target, transforming input from a sensory map to the output on a movement map, and then coordinating the appropriate muscles to execute the movement and hold the eye in the new position.

Second, we have a superb animal model of the human saccadic-fixation system in the old-world monkey, which allows us to study the brain mechanisms controlling the movement. Since the introduction of the now standard techniques for recording neuronal activity from awake behaving monkeys (Evarts, 1966), the accurate recording of eye movements (Fuchs and Robinson, 1966; Judge, Richmond, and Chu, 1980), the training of monkeys to control eye movements (Fuchs, 1967), and the visual stimuli that guide them (Wurtz, 1969), the regions of the monkey brain active before visually guided saccades have been identified. The visual pathway to the striate cortex, to extrastriate areas, and then to the regions of the parietal and frontal cortices have been identified and are included in figure 40.2A. Just as the elaboration of the changes in visual processing at each step in this pathway has contributed to our understanding of the visual input on which further cognitive processing is built, the extensive understanding of the brainstem oculomotor centers for saccade generation (Hepp et al., 1989; Moschovakis and Highstein, 1994) has led to greater understanding of what output is required of cognitive

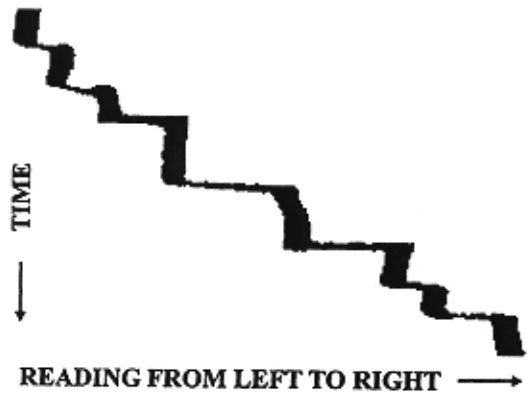

FIGURE 40.1 Interplay between rapid eye movements (saccades) and the pauses between these movements (fixation). In this example of the successive eye positions during the reading of a single line of text, saccades (vertical lines) move the eye across the page but are separated by periods of visual fixation (horizontal lines). Almost all of visual perception occurs during these periods of visual fixation. (Modified from Yarbus, 1967.)

processing for movement generation. Understanding the "spinal cord" for the saccadic system allows us to step backward gradually from the basic mechanics of movement to consider the control of these actions at higher levels.

Third, there is a nexus in the system through which much (although not all) of the information from the cortex flows to the brainstem oculomotor structures, the superior colliculus (SC). The SC receives inputs from the parietal and frontal areas of the cerebral cortex known to be related to saccade generation both directly and through the basal ganglia (Fries, 1984; Hikosaka and Wurtz, 1983) (figure 40.2A) and projects directly to the pontine and midbrain oculomotor areas. The SC can be viewed as a location for the coordination of the varied inputs from the forebrain and as the location for the transformation of these into outputs for the control of movement. It is the last step in the system in which both a visual and a motor map clearly are evident (Robinson, 1972; Schiller and Stryker, 1972; Wurtz and Goldberg, 1972).

In this chapter, we concentrate on several recent developments of our understanding of the saccadic/fixation system and its relation to the control of movement. We first describe how the neuronal elements in the SC are organized, including how only a few neuron types can be organized across a map to provide different output signals. Then we consider how the SC contributes to the alternating pattern of saccades and fixations. Finally we consider the "delay" activity between the neuronal response to the stimulus and the burst before a saccade and consider the transition leading to this activity in the cortical neurons that project to the SC.

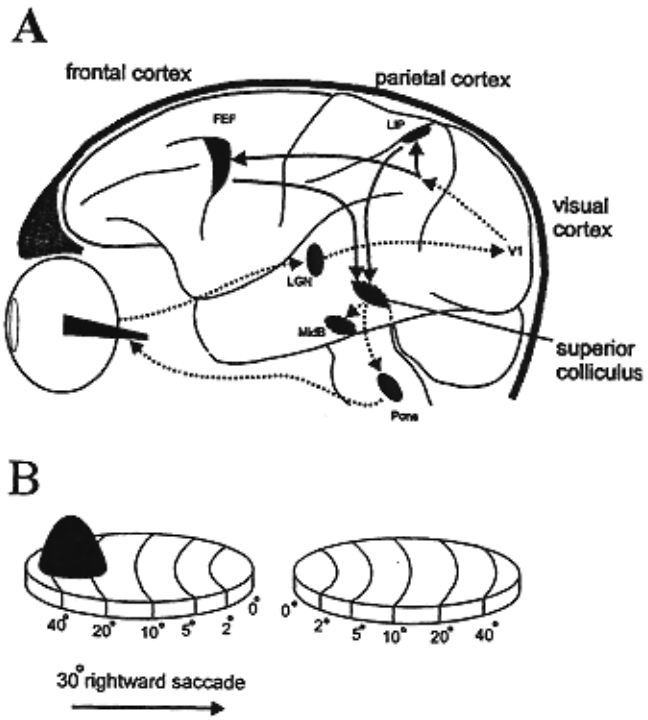

FIGURE 40.2 Brain systems for the generation of saccades and fixation. (A) Outline of the pathway from the visual input through cerebral cortex to oculomotor centers controlling eye muscles. The schematic drawing shows a lateral view of a monkey brain. The afferent visual pathway is indicated by the dotted line arrows passing from the retina to the lateral geniculate nucleus (*LGN*) of the thalamus to the striate cortex (*V1*), and then by a series of steps (not shown) to the parietal cortex (the lateral intraparietal [*LIP*] area) and the frontal cortex (the frontal eye field [*FEF*]). The projections from these areas to the superior colliculus (*SC*) are indicated by the solid line arrows. Omitted from the drawing are several areas (including the supplementary eye field in frontal cortex), reciprocal connections between areas, and the indirect pathway through the basal ganglia (caudate and substantia nigra pars reticulata). The output from the colliculus to the midbrain and pons oculomotor centers and then to the eye muscles also are indicated by dotted lines. (B) The schematic drawing of the organization of the SC on the right and left sides of the brain. Small saccades are represented in the rostral SC (0°) and large saccades are represented in the caudal SC (40°). The activity before a saccade is across a population of neurons, which is represented by a mound in the SC on the side of the brain opposite to the direction of the impending saccade.

Organization of the superior colliculus

The SC lies on the roof of the midbrain and consists of successive gray and white layers. Neurons in the superficial layers respond to visual stimuli and have receptive fields in the field contralateral to their location in the SC. Neurons in the intermediate layers often also respond to visual stimuli, but their most vigorous discharge is before the onset of saccadic eye movements.

We concentrate on these saccade-related neurons because it is in them that we see the transition from sensory to motor-related activity.

SUPERIOR COLLICULUS MAP FOR SACCADES Just as superficial-layer neurons have visual receptive fields, the saccade-related neurons in the intermediate layers have movement fields, that is, they increase their activity before saccades made only to one region of the visual field (Wurtz and Goldberg, 1972). There is a gradient of activity within each movement field, and a neuron's maximum discharge is associated with saccades of specific amplitude and direction–the neuron's optimal saccadic vector. Different neurons have different vectors, and the neurons are organized in a highly regular fashion to produce a neural map of saccadic vectors covering the contralateral visual field (figure 40.2B). On this map, large saccades are represented in the caudal portion, small are in the rostral region, upward are more medial, and downward are more lateral.

NEURON TYPES The activity of all the neurons within the intermediate layers is not identical, however, and to systematically study them, different types have been identified. The classification we use (Munoz and Wurtz, 1995a) is based largely on the activity of these neurons that comes *after* any initial visual response to the saccade target but *before* any burst of activity preceding saccade onset. This intervening activity is best revealed in two behavioral paradigms, the visual and memory-delayed saccade tasks. In these delay tasks, there is a period of active fixation during which we measure the resting discharge rate of the neuron. Then a visual target is presented in the center of the neuron's movement field, but the monkey is required to delay making a saccade to it (the delay period) until the fixation stimulus is extinguished (the cue to move). In the visual task, the target remains until the end of each trial, whereas in the memory task, it is only flashed and the monkey has to make a saccade to the remembered location.

Burst neurons (Munoz and Wurtz, 1995a) are the saccade-related neurons that have been most extensively studied in the SC and those that also have been referred to as saccade-related burst neurons (Sparks, 1978). In the delayed saccade tasks, these neurons frequently display a brief response time-locked to the onset of the visual stimulus, but their salient response is the vigorous burst of activity before a saccade made into their movement field (figure 40.3A). In contrast to other saccade-related neurons, they have next to no discharge during the delay period. The burst neurons are found throughout the rostral to caudal extent of the collicular map, and it is assumed that their activity is a signal to make a saccade with a vector coded by the position of that neuron on the SC map.

Buildup neurons continue to show sustained activity during the delay period in a delayed saccade task (figure 40.3B). In contrast to the burst neurons, these neurons do not necessarily show a discrete saccade-related burst in activity and certainly must include many, if not all, of the saccade-related neurons other than the burst neurons that have been described in many experiments, beginning with the earliest investigations (Mays and Sparks, 1980; Mohler and Wurtz, 1976; Sparks, 1978; Wurtz and Goldberg, 1972). Because of their delay activity, they originally were referred to as long lead neurons (Sparks, 1978). Munoz and Wurtz (1995a) more recently categorized these neurons as *buildup* neurons because their delay activity often increases gradually as the saccade onset approaches. Note that this neuronal class also may include neurons that were described differently by other groups, for example, quasi-visual neurons (Mays and Sparks, 1980) and prelude bursters (Glimcher and Sparks, 1992). Like the burst neurons, the buildup neurons are found throughout the collicular map. Our working hypothesis is that their buildup or delay activity represents the preparation to make a saccade, and their activation may facilitate saccade production.

Fixation neurons become active tonically when the animal fixates a visual stimulus and pause when a saccade occurs (figure 40.3C). These neurons originally were described in the cat SC (Munoz and Guitton, 1991) and then were identified in the monkey (Munoz and Wurtz, 1993a). In the monkey, the fixation neurons were shown further to sustain their discharge when the fixation stimulus was removed and the monkey continued to fixate, thereby ruling out the possibility that these neurons were simply visual neurons with a foveal receptive field excited by the fixation stimulus. The fixation neurons are found in the rostral region of the SC on both sides of the brain. Their discharge is related to the maintenance of fixation.

CONCLUSION: SUPERIOR COLLICULUS ORGANIZATION It is striking that there may be only a few neuron types within the SC intermediate layers although there may be more variation, particularly among the buildup neurons, than we consider here. The two points to be emphasized on the organization, however, are not new but rather have been recognized for a number of years. First, the map of saccadic vectors, which we have described (see review by Sparks and Hartwich-Young, 1989), conveys the vector for the *change* in eye position produced by the saccade. Second, a change in the activity of a *population* of neurons on the SC map has significant importance, which we

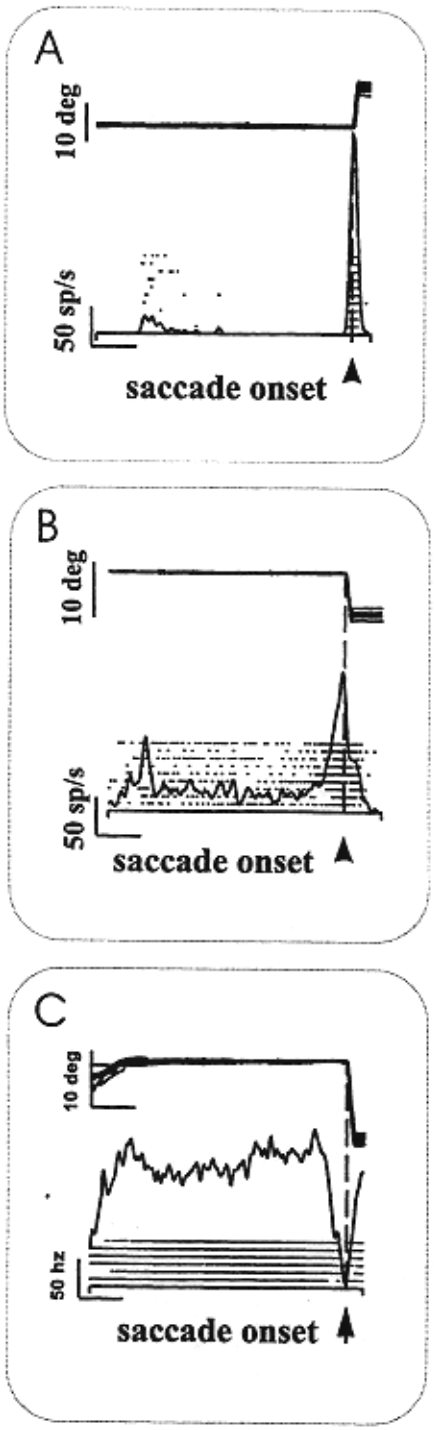

FIGURE 40.3 Classification of neurons in the intermediate layers of the SC into three categories: burst, buildup, and fixation. The top row of each example shows the eye position traces, and below these are rasters, with each tick in the raster representing a single action potential and each row of ticks representing a single trial. The superimposed spike density functions show the sum of the activity across the trials. All traces are aligned on the onset of the saccadic eye movement. (A) An example of a burst neuron with a clear response to the onset of the visual target (start of the line) and a burst of activity before the saccade (arrowhead under the line). (B) An example of a buildup neuron with a slight visual response, continuing activity, and a burst of activity before the saccade. Buildup neurons can be defined as those whose discharge rate during the delay period is significantly higher ($P < .01$) than the resting rate. While the buildup neurons clearly are different from the burst neurons, they may form a continuum with them; the buildup neurons also may be a more heterogeneous as suggested by the broad range of discharge behaviors exhibited in response to the visual stimulus and before the saccade. (C) An example of a fixation neuron with a high rate of discharge while the monkey is fixating on a target.

have not yet emphasized. Before the onset of a saccade, it is not just a few neurons that become active but roughly 25% of the entire population (Munoz and Wurtz, 1995b), and it is the vector average of these active neurons that appears to determine which saccade is made (Sparks et al., 1990), as represented by the mound of activity on the SC map in figure 40.2B.

Interaction of saccades and fixation

One of the striking observations on the SC is the identification of neurons that are related to both the generation of saccades and the maintenance of fixation.

CONTINUITY OF FIXATION AND BUILDUP NEURONS Fixation neurons have been described as a separate class of neurons because of their high discharge rate during fixation, and understanding how they are related to other SC neurons depends on determining what controls their high rate of discharge during active visual fixation. One possibility is that the discharge of these fixation neurons could be influenced by factors similar to those influencing the buildup neurons because although we have described the buildup and fixation neurons as behaving very differently (figure 40.3), they have at least two characteristics in common. First, they both lie somewhat deeper in the SC than the burst neurons (Munoz and Wurtz, 1995a). Second, they both have movement fields; most fixation neurons, like buildup neurons, were shown to have a burst of activity with small contralaterally directed saccades (Munoz and Wurtz, 1995a).

One possibility is that the fixation neurons have delay activity just as buildup neurons do, and that the increased activity during fixation essentially is this delay activity. To investigate this, Krauzlis and associates (1997) recorded from neurons in the rostral SC that had the characteristics of fixation neurons, including continued discharge when the monkey fixated in the absence

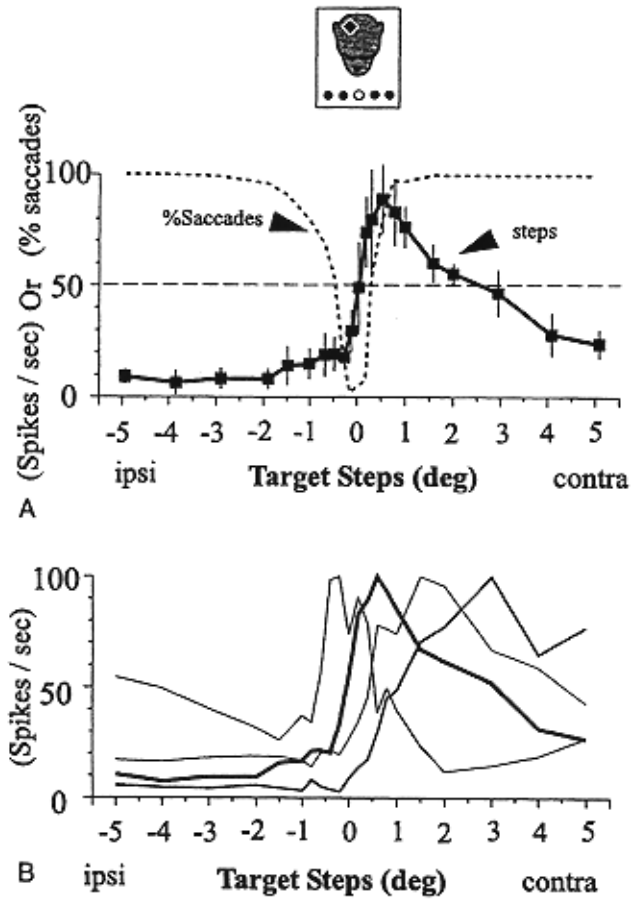

FIGURE 40.4 Fixation neuron activity with small changes in target position. (A) Changes in firing rate of a neuron after small steps of the fixation target into the visual field either ipsilateral or contralateral from the SC in which the neuron was recorded. Symbols show the mean firing rate over an interval beginning 100 ms after the target step and lasting for 100 ms or until 8 ms before the saccade onset. Error bars indicate ±1 SD for the 12 trials. The dotted line indicates the percentage of trials on which each target step elicited a saccade. Dashed line indicates the mean firing rate with no target steps. (B) Change in activity of four fixation neurons from one monkey after the same small steps. The four have been normalized so that the peaks of the curves are 100. The neuron with the darkened trace is the same as in (A). (From Krauzlis and associates, 1997.)

of a fixation target. They stepped the target on which the monkey was fixating to slightly eccentric locations and rewarded the monkey for making a saccade to the target. Fixation neurons showed the largest increases in firing rates with small steps into the contralateral visual field; the neuron in figure 40.4A showed the largest response for steps of approximately 0.5°. Different neurons showed the maximal response for contralateral steps of different sizes (figure 40.4B). Saccades to large target steps were accompanied by a decrease in activity for both ipsilateral and contralateral saccades, as had been reported previously (Munoz and Wurtz, 1993a). What is particularly relevant is that the increase always was for small steps and that the increase in activity occurred whether the step elicited a saccade or not. Krauzlis and colleagues (1997) concluded that the fixation neurons are tonically active during visual fixation, not because they are carrying a unique fixation signal but because they are indicating target locations very close to the fovea that usually do not elicit a saccade.

What these experiments show is that the activity of the fixation neurons during fixation has two striking similarities to the delay activity in buildup neurons. First, both the fixation and buildup neurons increase their activity for a target at some eccentricity in the contralateral visual field, the fixation neurons for very small target steps and the buildup neurons for larger steps. Second, the delay activity is present even if no saccade is made, as indicated for the fixation neurons by Krauzlis and associates (1997) and for the buildup neurons by Munoz and Wurtz (1995a). Thus, the fixation neurons might be regarded most parsimoniously as a rostral continuation of the buildup neurons. The hypothesis that emerges is that the activity of these fixation and buildup neurons indicates an error between where the eye is and where the target is, and the size of the error represented by the neuron depends on the location of the neuron on the SC movement map. This simplification eliminates the problem of deciding where in the rostral SC the fixation cells end and where buildup neurons begin. The different effects of activating these neurons depends on their interactions within the SC and probably their differential connections outside the SC, as we consider in the next section.

FIXATION AND SACCADE INTERACTION How does this proposed continuity of fixation and buildup neurons affect the original hypothesis (Munoz and Wurtz, 1993a, 1993b) that the activity of monkey fixation neurons suppresses the generation of saccades? We believe that the interaction between saccades and fixation still is controlled by a system within the brain that includes the SC but that the observations just described show how the neuronal elements are organized to carry out this interaction. The basic observations on the effects of activating or inactivating the rostral SC still hold. Saccades are suppressed by the activation of the SC fixation region by either electrical stimulation or injection of the chemical agent bicuculine, a gamma-aminobutyric acid (GABA) antagonist (Munoz and Wurtz, 1993b). Conversely, saccade production is facilitated by the inactivation of the fixation neurons by injection of the GABA agonist muscimol (Munoz and Wurtz, 1993). Both observations show that alteration of the rostral SC affects when saccades are generated.

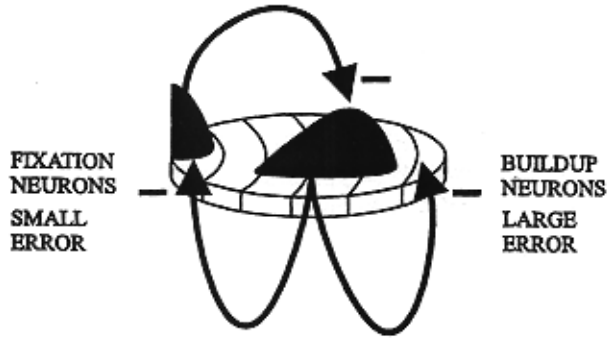

FIGURE 40.5 Mutual inhibition of fixation and buildup neurons. The arrows indicate inhibitory interactions. The fixation neurons in the rostral SC inhibit the saccade-related neurons in the caudal SC whereas the saccade-related neurons inhibit both other saccade neurons and the fixation neurons. Not shown are the inhibitory inputs from the basal ganglia or the presumed excitatory inputs from cerebral cortex.

This leads to a hypothesis of the interaction between fixation and saccades shown schematically in figure 40.5. The basic assumption is that when any population of SC neurons is active anywhere on the SC map, they suppress the activity of all other SC neurons. When buildup neurons in the caudal SC become active, they act to inhibit all other neurons in the SC, including the most rostral fixation-type buildup neurons. They also provide an error signal according to their position on the SC, indicating which saccadic vector would be required to bring the eye to the target to eliminate the error. The output of these neurons may be to adjacent burst neurons as well as directly to neurons in the pons and midbrain outside the SC. When the fixation neurons in the rostral SC become active, they also inhibit all other SC neurons. The fixation activity also indicates that a position error exists, but this error signal is so small that no eye movement is elicited–giving incidentally a physiological basis for the concept of a dead zone for saccade initiation (but considerably larger than that in humans) (Wyman and Steinman, 1973). The outcome of this competition for fixation or saccade (here and probably in other parts of the brain as well) determines the initiation of saccades. There may be differences in the strength of the inhibitory effects in the rostral and caudal regions of the SC, but the neural mechanisms can be regarded as fundamentally the same.

Several lines of evidence are consistent with the interactions between rostral and caudal SC, as outlined in figure 40.5. First, it has become clear that activity in the rostral SC does directly inhibit saccade-related neurons in the caudal SC. Initially, the saccade suppression effect caused by the activation of the SC fixation region logically could be explained by an interaction between fixation and saccade-related elements located downstream of the SC. Indeed, a pause in the activity of brainstem omnipause neurons is necessary to release the burst generator and produce a saccade (Hepp et al., 1989; Moschovakis and Highstein, 1994), and these neurons receive preferential projection from the rostral SC (Büttner-Ennever and Horn, 1994; Paré and Guitton, 1994). The SC fixation neurons therefore could alter the generation of the saccade as an influence onto the activity of omnipause neurons, that is, without acting directly on the SC saccade-related neurons. Munoz and colleagues (1996), however, showed that when electrical stimulation of the rostral SC interrupts saccades in mid-flight, there is a pause in the saccade-related activity of both burst and buildup neurons in caudal SC when the eyes stop momentarily. This indicates that the fixation neuron activity does act on the SC saccade-related activity.

The mutual inhibitory effect of activity in the rostral and caudal SC also has received support from both physiological and anatomic experiments. By electrically stimulating within the monkey SC, Munoz and Istvan (1998) established the inhibitory effect of fixation neurons on burst and buildup neurons and of these saccade-related neurons back onto fixation neurons. In addition, these experiments showed that stimulation of saccade regions also inhibits other remotely located saccade-related neurons. (The only exception to the mutual inhibition between collicular neurons is the connection between the fixation neurons in the two SCs: stimulation of one fixation region excites rather than inhibits the fixation neurons in the other SC.) The presence of GABAergic connections throughout the SC (Mize, 1992) and the recent experiments in the ferret (Meredith and Ramoa, 1998) showing that stimulation at any point in the SC, rostral or caudal, produces an initial inhibition at distant points in the SC, support the hypothesis of inhibitory interactions. Behan and Kime (1996) showed in the cat that biocytin injections label cells within 0.5 mm up to 5.0 mm from the injection site, which provides anatomic evidence for horizontal connections throughout the length of the SC. Thus, the mutual inhibition between different parts of the SC, consistent with the fixation/saccade interactions within the SC, are supported by what is known about the physiological and anatomic connections between these neurons.

EXPRESS SACCADES The interaction between fixation- and saccade-related activity has been explored in a recent series of experiments on an express saccade, a short latency saccade that is critically dependent on an intact SC (Schiller, Sandell, and Maunsell, 1987). These exper-

iments illustrate most clearly the interactions we have considered.

One of the most prominent ideas about the shift in fixation caused by a saccade, which usually is addressed with respect to the accompanying shift of attention, is that there must be an initial disengagement from the current fixation stimulus. This disengagement also was proposed to be critical in the reduction of saccadic reaction time in the gap saccade paradigm and the generation of express saccades (Fischer and Weber, 1993), the latencies of which (~80 ms) approach the conduction time from the retina to the eye muscles (Fischer and Boch, 1983). With the discovery of the SC fixation neurons, a neural correlate for the fixation disengagement was obvious: the disengagement occurs when the fixation neurons reduce their discharge after the disappearance of the fixation stimulus or pause before saccades. The increased incidence of express saccades following rostral SC inactivation (Munoz and Wurtz, 1991) provided experimental support for this hypothesis, as did the finding that a decrease in fixation activity during the gap task correlates with the reduction in saccade reaction time (Dorris, Paré, and Munoz, 1997).

Recording of the activity of SC burst neurons revealed that the saccade-related burst of activity is indistinguishable from the target-related responses of these neurons when an express saccade is produced (Dorris, Paré, and Munoz, 1997; Edelman and Keller, 1996). It was hypothesized that the release of fixation permits the target-related responses of SC neurons to be strong enough to trigger short-latency express saccades (Edelman and Keller, 1996; Sommer, 1994).

Fixation disengagement, however, can only be a part of the mechanism for express saccades in the monkey, as indicated by recent behavioral experiments by Paré and Munoz (1996). Using the gap saccade task, they trained monkeys to make express saccades after presenting the saccade target repeatedly in the same part of the visual field. After this training, express saccades were made only to a restricted region of the visual field centered on the location of the target used for training. The disappearance of the fixation stimulus before target presentation did not lead to express saccade production at the other target locations but simply reduced the mean saccadic reaction time. Overall, these results indicate that it is the neuronal activity associated with the preparation to make saccades to the training target that determines the occurrence of express saccades, not just the release of fixation afforded by the disappearance of the fixation stimulus. In subsequent physiological experiments, Dorris, Paré, and Munoz (1997) showed that fixation neurons decrease their discharge before express saccades, but that these changes are not predictive of express saccade production (figure 40.6A). In contrast, the increased level of discharge of buildup neurons before target presentation was found to be correlated with the occurrence of express saccades when these were made into the movement field of the neurons (figure 40.6B). This increase in the early activity of buildup neurons thus may facilitate the excitability of the corresponding saccade region and thereby allow the target-related responses of the SC neurons to trigger the short-latency express saccades. Additional evidence for this motor preparation hypothesis for express saccade generation comes from another behavioral study. Using a scanning saccade paradigm, Sommer (1997) compared the spatial attributes of saccades produced in response to suddenly appearing stimuli with those made while scanning to unchanging stimuli. He found that the occurrence of express saccades was related to the congruence between the location of the suddenly appearing target and the goal of voluntary saccade planning.

If we generalize the observations made on express saccade generation to all saccades, the decision to make a saccade can be reduced to the competitive interaction between the signals related to planning a saccade and those related to maintaining fixation. Each signal is necessary, but not sufficient, for determining when a saccade will be initiated.

CONCLUSION: SACCADE AND FIXATION INTERACTION The observations described emphasize several points. First, the interactions underlying the alternation of fixation and saccades all may depend on interactions between *one neuronal class* in the SC, what we have referred to as the buildup neuron. This illustrates a case in which one neuronal element can be used to produce quite different effects (fixation or saccades) depending on its relation to other neurons on a neuronal map. Second, the movement generation is a result of *the balance of competition* between the activity of the neurons signaling movement (the buildup neurons and probably the burst neurons as well) and those signaling nonmovement (the fixation neurons). This interaction is based on competing activity on two parts of the SC map (rostral fixation and caudal saccade), and the outcome determines when a saccade will be made.

Superior colliculus delay activity

The SC activity that we have concentrated on in considering fixation is the activity in the delay period that precedes the burst of activity accompanying saccades. Following, we consider factors that alter this delay activity.

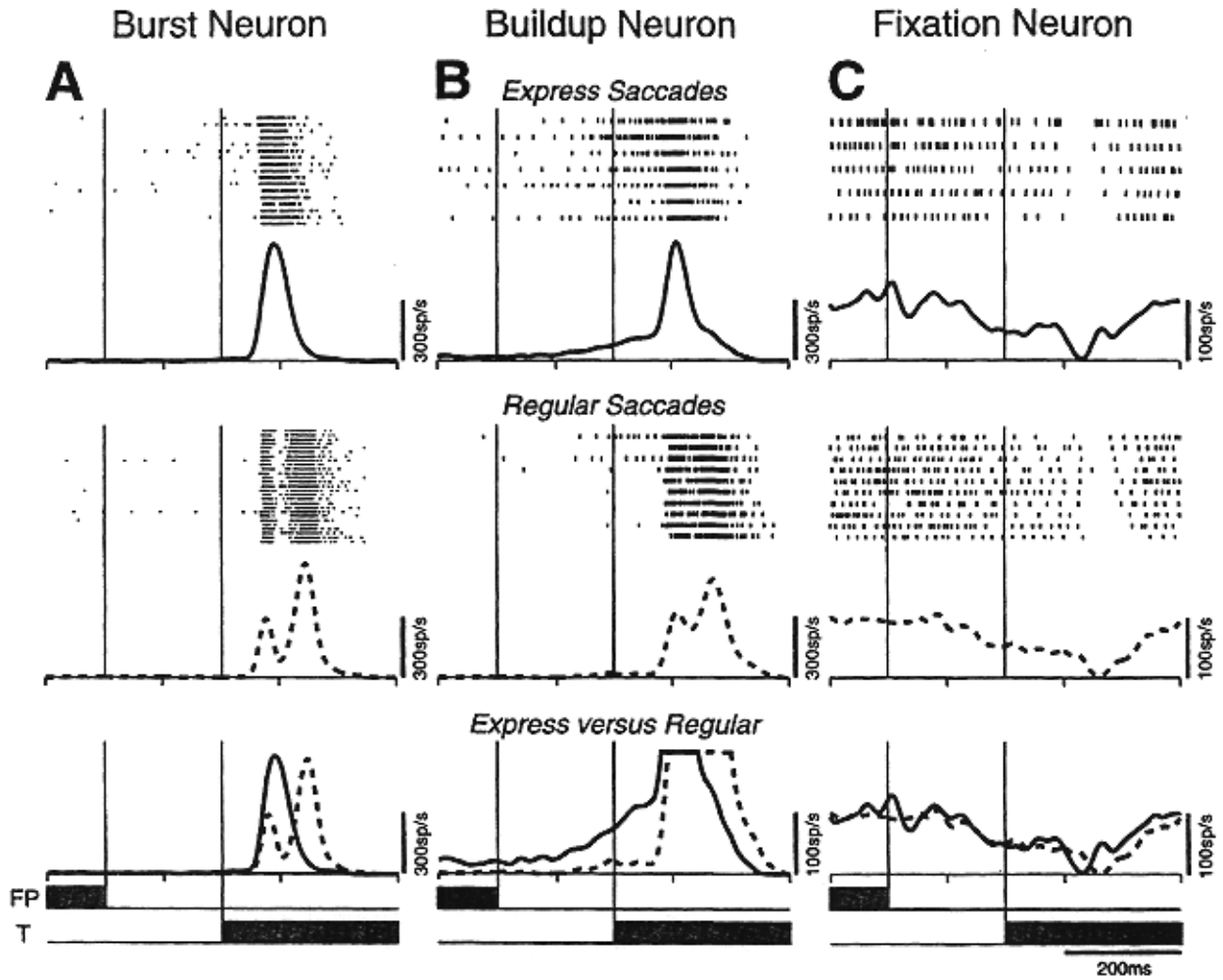

FIGURE 40.6 Neuronal activity of burst (A), buildup (B), and fixation (C) neurons during the generation of short-latency express saccades (top) and long-latency regular saccades (middle). The paradigm used was the gap saccade task in which the fixation stimulus (*FP*) is extinguished 200 ms before the target (*T*) presentation (as indicated by the bars along the bottom). Rasters and spike density functions are aligned on target onset. In the bottom panel, the spike density functions of the express (solid line) and the regular (dashed line) trials are superimposed. (A) The activity of burst neurons displayed two bursts before regular saccades: one time-locked to the target onset and a later one time-locked to the saccade onset. In contrast, express saccades were preceded by only one robust burst time-locked to both target and saccade onsets. (B) The discharge rate of buildup neurons during the gap period before the target onset was greater for express saccade trials compared with regular saccades. After the target presentation, the buildup neuron showed activity similar to the burst neuron. (C) The activity of fixation neurons decreased during the gap and paused just before either express or regular saccades. However, the level of activity associated with each type of saccades did not differ. Express saccades were defined as those with latencies between 70 to 120 ms, and regular saccades had latencies between 130 and 180 ms. (After Dorris, Paré, and Munoz, 1997.)

Because this delay period activity is not tied to the occurrence of the saccade, Munoz and Wurtz (1995a) proposed that it represents the preparation to make a saccade. Consistent with this motor preparation hypothesis, Dorris and colleagues (1997) demonstrated that the level of delay activity for a significant proportion of buildup neurons (41%) predicts motor performance. They showed that the level of buildup activity during a gap period (between the time the fixation stimulus was turned off but before presentation of the target) was inversely correlated to the reaction time of the saccade: the greater the discharge, the shorter the reaction time.

If these early neuronal changes are related to preparation to make a saccade, then such changes should be sensitive to the likelihood that a saccade will be made (Riehle and Requin, 1993). Two recent experiments tested whether manipulating the prior knowledge of the

monkey as to whether a saccade will be made alters the delay activity of the SC buildup neurons.

TARGET PROBABILITY PARADIGMS Basso and Wurtz (1997, 1998) used two paradigms that required monkeys to make a saccade to a peripheral stimulus while changing the probability that a given stimulus would be the saccade target.

In the first paradigm, varying the number of stimuli presented to the monkey changed the probability that the one located in the movement field of the neuron would become the target. One, two, four, or eight stimuli were presented, and then later the monkey was cued as to which one was the saccade target (figure 40.7). During the preselection period, when the monkey did not know which of the stimuli would be the target, increasing the number of possible targets from one to eight decreased the activity. When the target dimmed, the activity in each condition increased to the level present when only a single target was present. During the preselection period, the mean discharge rate of all buildup neurons studied decreased as target probability decreased.

In a second paradigm, the number of stimuli remained constant so that no changes related to visual interaction could account for the results. Presenting the eight targets in a series of blocked trials, in which the same one of the eight stimuli became the target, increased target probability. After a number of trials, the neurons developed a higher level of activity than in the case when any of the eight stimuli could become the target. Therefore, changes in target probability modulate the buildup activity during a delay period whether those changes result from varying the number of stimuli or varying the monkey's previous experience. In contrast, the burst of activity of burst neurons and the saccade-related burst activity of buildup neurons did not change in either probability experiment.

GO/NOGO PARADIGM In this experiment, the fixation stimulus provided information about whether a saccade would be required to a peripheral stimulus located in the movement field of the neuron. In the two types of trials used, the fixation stimulus first changed color, signaling to the monkey that the upcoming peripheral stimulus should be either ignored (Nogo trials) or should be taken as the target for a saccade (Go trials–see stimulus bars in figure 40.8). Most buildup neurons tested in this paradigm (61%) showed delay activity specific to the Go instruction, as exemplified in figure 40.8 (Sommer, Paré, and Wurtz, 1997). For these neurons, the buildup activity was much higher after target presentation if the Go instruction was provided, that is, if a saccade was to be executed. For the sample of buildup neurons ($n = 62$), the delay activity in Go trials was 2.4 times greater than that observed in Nogo trials. Other studies have demonstrated similar modulation of early low-frequency discharge in SC neurons in tasks designed to show activity changes with movement selection (Glimcher and Sparks, 1992) or shifts in attention (Kustov and Robinson, 1996).

CONCLUSION: DELAY ACTIVITY Both the target probability and the Go/Nogo experiments show that a greater probability that the monkey will be required to make a saccade on a particular trial leads to a higher level of delay or buildup activity. This increase is consistent with viewing the buildup neuron activity as part of the preparation to make a saccade that precedes the burst of activity at the time of a saccade. This increased activity that precedes the generation of a saccade represents an increase in activity on one part of the SC map, and this activity develops over time during the delay period. We believe that what we refer to loosely as "preparation to move" might best be regarded as the gradual specification over time of one part of the SC map as the center of activity for the next saccade. This localized activity specifies the vector for the next saccade, and the narrowing of activity over time is a narrowing of the selection of this vector.

Cortical input to superior colliculus

Many of the fixation, buildup, and burst neurons are likely to receive input from the cerebral cortex. This opens the possibility of seeing the transition in activity between the cerebral cortex and the superior colliculus. To explore this transition, the cortical neurons that project to the SC must be identified. It is not sufficient to compare the activity of any neuron in the cortical areas that project to the SC with the SC activity because many of the cortical neurons might not project to the SC. We need to study specifically those neurons that project to the SC and have used antidromic stimulation to do so. In this technique, a cortical neuron is identified as projecting to the SC if electrical stimulation of the SC produces spikes in the cortical neuron with short consistent latencies (along with other criteria, Lemon, 1984). We have concentrated on the neurons in the parietal and frontal cortex because neurons in these regions have well-established projections to the SC (Huerta, Krubitzer, and Kaas, 1986; Leichnetz and Goldberg, 1988; Lynch, Graybiel, and Lobeck, 1985).

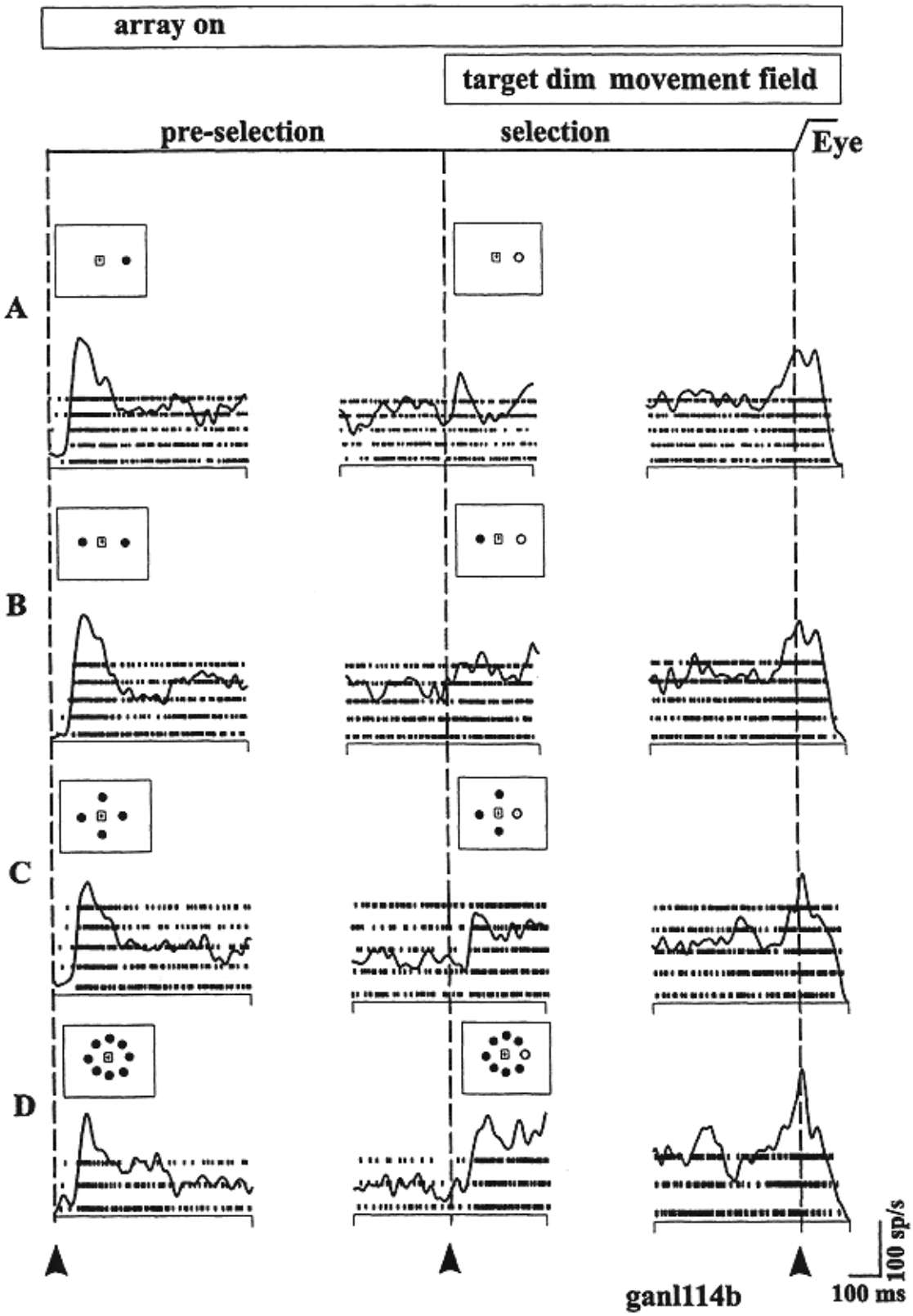
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FIGURE 40.7 Buildup neuron activity modulated by shifts in target probability. Increases in the number of potential targets for the impending saccade reduced buildup neuron activity. The events of the task are indicated by the labeled periods of time across the top, and the spatial arrangement is indicated by a schematic of the stimulus display in front of the monkey. The first column shows activity on five trials in the rasters and the mean of these in the superimposed spike density trace when one (A), two (B), four (C), or eight (D) targets were presented during the preselection. This example is taken from trials when the target was in the movement field of the neuron. The first column is aligned (vertical dashed lines) on the onset of the possible targets (preselection period), the second on the dimming of one of the targets (selection period), and the third on the onset of the saccade (eye movement period). There is an initial visual response and subsequent sustained or delay period activity. Both of these activities were reduced as the number of stimuli was increased. When the target dimmed, the activity increased. This neuron had a burst of action potentials associated with the onset of the saccade that did not differ substantially between the probability conditions.

PARIETAL CORTEX INPUTS TO SUPERIOR COLLICULUS The lateral intraparietal (LIP) area is the area in the inferior parietal lobule where saccade-related activity has been described by both the Andersen and Goldberg groups (Andersen and Gnadt, 1989; Andersen, Essick, and Siegel, 1987; Barash et al., 1991; Colby, Duhamel, and Goldberg, 1995; Colby, Duhamel, and Goldberg, 1996; Mazzoni et al., 1996). Paré and Wurtz (1997) first identified the LIP neurons that can be activated antidromically from electrical stimulation within the SC. The projection neurons act primarily on the intermediate layers of the SC because antidromic activation thresholds reached minimum values at depths where neurons showed saccade-related activity. The LIP projection to the SC appears to be organized topographically because the most efficient antidromic stimulation typically was obtained at SC sites with movement fields similar to those of the LIP neurons.

Approximately 75% of the identified LIP efferent neurons responded to the onset of the visual target, and approximately 60% of the neurons maintained their activity during the delay period of either the visual or memory-delayed saccade tasks. Several LIP neurons also exhibited a modest saccade-related increase in activity, but none had only a saccade-related burst of activity like the SC burst neurons or some frontal eye field (FEF) efferent neurons. Although the LIP neurons therefore were more similar to the SC buildup neurons than to the SC burst neurons, the LIP neurons differed from the SC neurons in the Go/Nogo task (figure 40.9A). Only about one third of the LIP efferent neurons had delay activity that occurred only after the Go instruction, and the mean

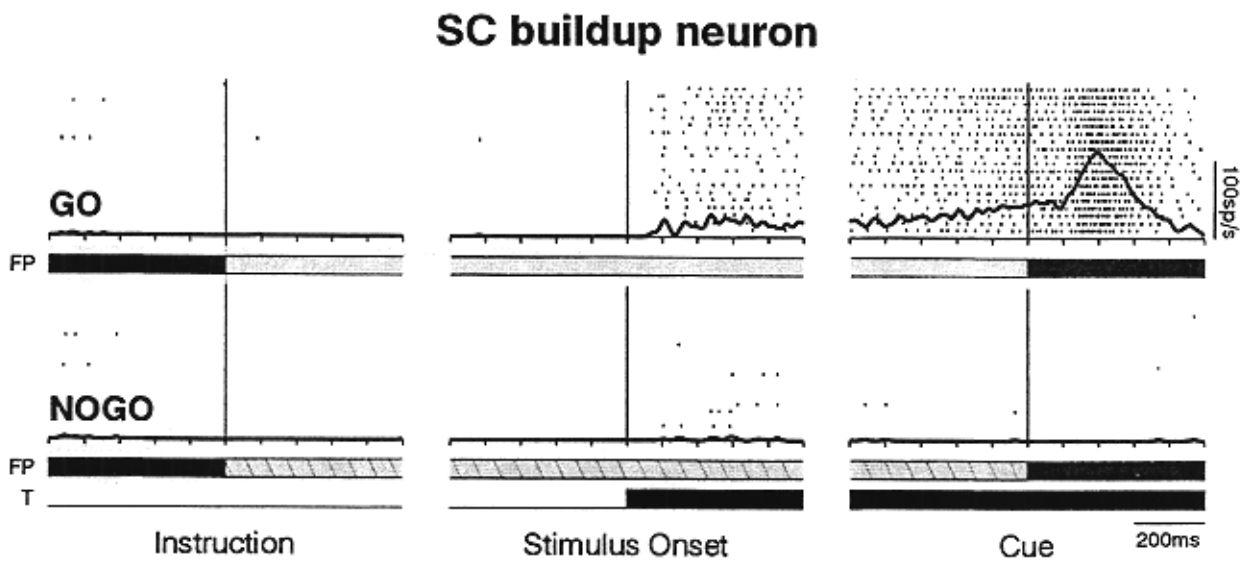

FIGURE 40.8 Neuronal activity of a buildup neuron in a Go/Nogo saccade paradigm. Instructions were given by a change in color of the fixation stimulus (*FP*) before the peripheral stimulus (*T*) appeared. A change from blue to red indicated a Nogo instruction, whereas a change to green indicated a Go instruction. After the peripheral stimulus was presented, there was a delay period. After this, the fixation stimulus returned to its original blue color, cueing the monkey to either maintain fixation for a prolonged duration (Nogo) or execute the saccade (Go). Rasters and spike density functions for Go (top) and Nogo (bottom) trials are aligned on the instruction (left), target (middle), and cue (right). This neuron started to discharge after the target appearance and exhibited delay activity only if the Go instruction had been given. The discharge peaked at the onset of the saccades made in response to the cue presentation. This neuron was virtually silent during Nogo trials. To quantify the level of delay activity, the discharge rate was measured during a 300-ms epoch ending at the time of the cue presentation. This neuron's delay activity was 33.3 sp/s and 0.2 sp/s in Go and Nogo trials, respectively.

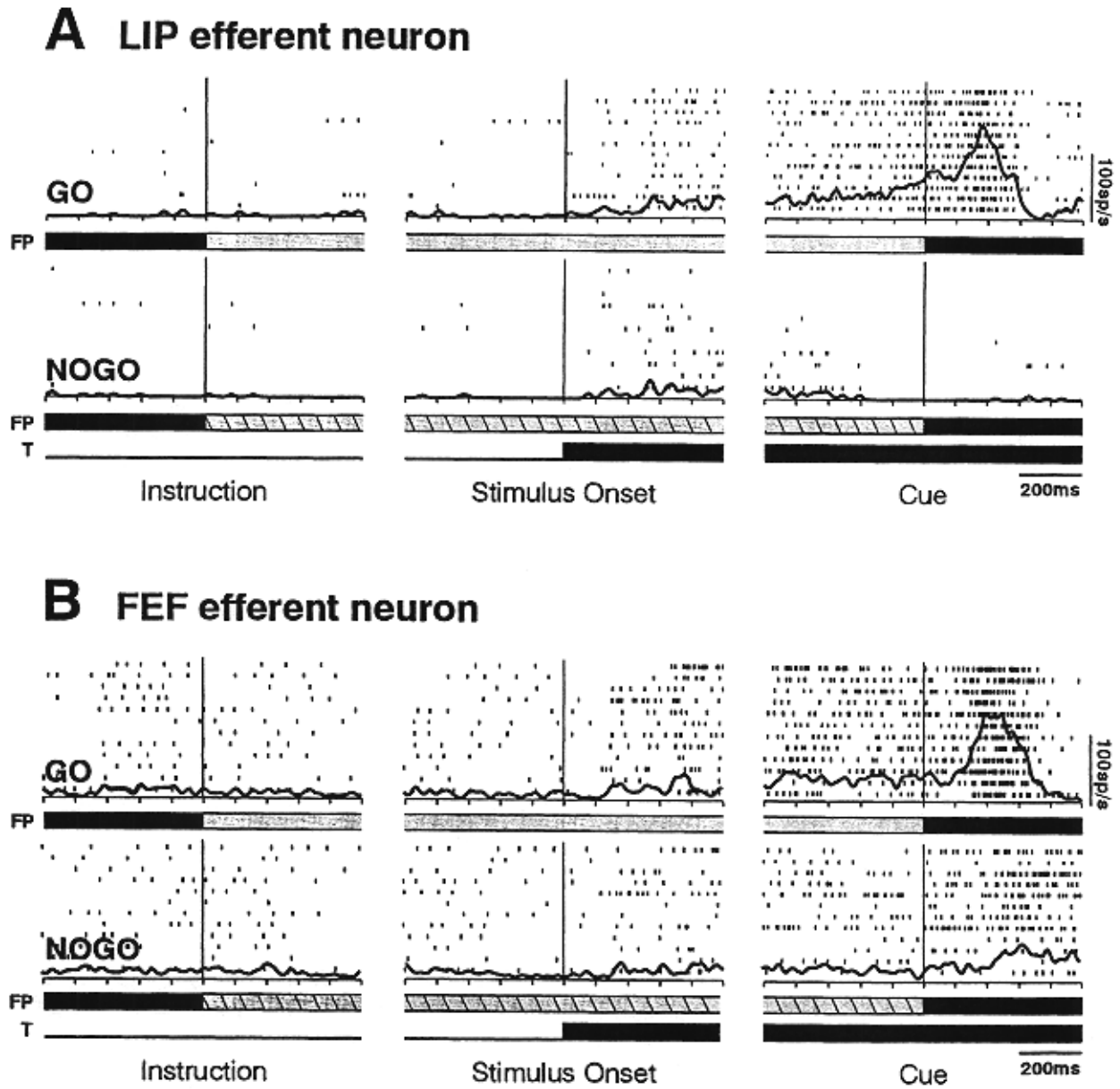

FIGURE 40.9 Neuronal activity of cortical efferent neurons in a Go/Nogo delayed saccade paradigm. Responses of lateral intraparietal (LIP) (top) and frontal eye field (FEF) (bottom) neurons that were activated antidromically by SC stimulation. In the Go trials, the neurons started discharging after the stimulus onset and maintained their discharge until the saccade was made in response to the cue. In the Nogo trials, less response was observed after the stimulus presentation. Same organization as in figure 40.8.

delay activity in Go trials was only 1.3 times greater than that in the Nogo trials. The delay activity of the LIP neurons started at the time of the visual target and continued for most neurons regardless of whether there was preparation to make the saccade. This is in contrast to the SC neurons, where activity in approximately two thirds of the SC neurons had activity only after the Go instruction, and across the sample there was 2.4 times greater activity on the Go trials. Thus, the SC neurons were substantially more related to the signal to move, and using our interpretation of the Go/Nogo task, were more closely related to the preparation to move whereas the LIP neurons were more dependent on the visual stimulus than the signal to make a saccade.

FRONTAL CORTEX INPUTS TO SUPERIOR COLLICULUS The FEF is the area in the frontal lobe that, together with the SC, is necessary for saccade generation (Schiller, True, and Conway, 1980). Segraves and Goldberg (1987) previously reported that FEF neurons that

are antidromically activated by SC stimulation carry a saccade-related burst signal that sometimes is combined with a response to visual stimulation. We have confirmed this observation and find that approximately 15% of the FEF neurons have only presaccadic bursts. Thus, in contrast to the LIP area, the FEF does have neurons projecting to the SC that have characteristics of the SC burst neurons. As was the case with the LIP region, the FEF projection to SC was topographically organized and directed to the intermediate layers.

In addition to these burst-like neurons, approximately one third of the FEF efferent neurons showed delay activity between the onset of the stimulus and the saccade, which made them similar to the SC buildup neurons. Like the SC buildup neurons, but in contrast to the LIP efferent neurons, nearly two thirds of the FEF efferent neurons with delay activity had activity in Go trails that was higher than in Nogo trials (figure 40.9B). This activity on the Go trials, although frequent, was small, only 1.5 times greater than that observed on Nogo trials (Wurtz and Sommer, 1998). Thus, although the FEF has buildup-like activity as frequently as do the SC neurons, the Go/Nogo effect is smaller, like that in the LIP. The FEF appears to be a more heterogeneous area than is the LIP region and includes both burst- and buildup-type neurons.

While investigating the frontal cortex inputs to the SC, there was an additional finding that raises questions about the unidirectional flow of information from cortex to brainstem outlined in figure 40.2A: electrical stimulation of the SC (Sommer and Wurtz, 1998) showed that many FEF neurons *receive* inputs from the SC, presumably by means of a thalamic synapse (Lynch, Hoover, and Strick, 1994). These FEF neurons always had a phasic visual response, and some also carried signals such as delay activity, a presaccadic burst, or fixation activity. These results suggest that the processes underlying saccade generation involve a bidirectional communication between the SC and cortical areas.

COMPARISON OF SUPERIOR COLLICULUS INPUTS The neurons in the LIP area and the FEF that project directly to the SC carry an amalgam of visual, delay, and saccade signals, although the ratio of these signals differs between the two areas. The efferents from the LIP area have not been found to show activity comparable to the burst neurons in the SC, whereas FEF efferents frequently do. The efferents of both the LIP area and the FEF show delay activity, but in the LIP area the activity is more pronounced. Thus, the delay activity is represented more heavily in LIP efferents than is the burst, and for the FEF, it is approximately

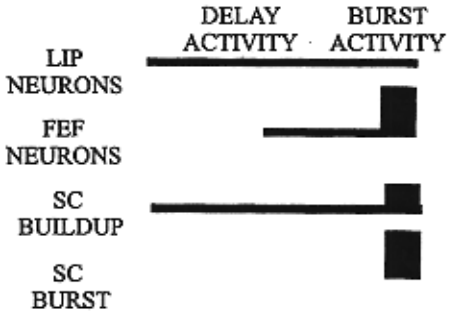

FIGURE 40.10 Summary of the overlap of activity between neurons in the cerebral cortex (LIP and FEF) that project to the SC and the neurons in the SC (burst and buildup). Activity in the delay period is represented by the horizontal line: the greater the activity, the longer the line. The burst is represented by the height of the vertical bar, the higher the bar the larger the burst. The delay activity is consistently present in LIP and SC buildup neurons, but there is little if any burst in the LIP neurons. The delay activity is also present in the FEF neurons, but these neurons also frequently have a burst of activity, as do the SC burst neurons. There are therefore both *differences* between the neurons in the two cortical areas projecting to the SC and *overlap* between the cortical activity and SC activity, the LIP having greater similarity to the SC buildup neurons than to the SC burst neurons and the FEF tending to have the reverse relationship.

the reverse. Figure 40.10 compares schematically this overlap of the activity in the LIP efferents, the FEF efferents, and the SC buildup and burst neurons. Whether this input is directed separately to the burst and buildup neurons in the SC remains to be determined. Because signals also are transmitted from the SC to cortex (and between the FEF and the LIP area as well), the development of saccade-related activity must result from the continuous interaction between multiple brain structures, including the FEF, the LIP area, and the SC.

CONCLUSION: DISTRIBUTED PROCESSING The shifts in processing from one area to the next in the brain logically could be of substantially different types (Miller, 1988). On the one hand, this processing could be discrete: the processing in one area is completed and the result is passed on to the next area, where a new stage of processing begins. Conversely, the processing might be distributed: the same type of processing in one area continues at some higher level in the next area. Comparison of the activity of cortical and SC neurons offers one of the few opportunities in sensorimotor systems to determine which type of transformation occurs. In the visual-oculomotor processing that we have considered, it is distributed processing. One of our most striking observations is that the same type of neuronal activity can be identified in both the cortex and

the SC and that there is simply a shift in the characteristics of the processing. For example, the processing conveyed by buildup neurons is carried on in both the parietal cortex and the SC, but analysis of the nature of the continuing activity shows that there is a shift in the dependence of that activity toward the preparation to make a saccade.

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