

Commentary: Saccadic eye movements: overview of neural circuitry

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Abstract: Recent neuroanatomical, neurophysiological, clinical, and brain imaging studies have generated a wealth of data describing the neural control of saccadic eye movements and visual fixation. These studies have identified many of the cortical and subcortical structures involved in controlling the behavior. Critical nodes in the network include regions of the parietal and frontal cortices, basal ganglia, thalamus, superior colliculus, cerebellum, and brainstem reticular formation. Specific functions are likely not localized to only one brain area, but rather, they may be distributed across multiple areas. This commentary is used to review briefly the neural circuitry controlling saccadic eye movements and visual fixation.

Introduction

One of the most important functions of the central nervous system is the generation of movement in response to sensory stimulation. The visual guidance of saccadic eye movements represents one form of sensory-to-motor transformation that has provided significant insight into our understanding of motor control and sensorimotor processing. The eyes have a simple and well defined repertoire of movements and the neural circuitry regulating the production of saccadic eye movements is now understood at a level that is sufficient to link cortical and subcortical areas together. The contributions to this section of the book by Munoz and Fecteau, Enderle, Zambarbieri, Tinsley and Everling, and Stampe and Reingold address aspects of saccadic processing at various stages of the neuraxis from the brainstem reticular formation, the superior colliculus, the cerebellum, to the

cerebral cortex. This commentary will therefore be used to review briefly the neural circuitry controlling saccadic eye movements.

Before reviewing the details of the neural circuitry it is first important to recognize why saccadic eye movements have evolved and what they are used for. The primate retina has a specialized region in its center, called the fovea, which serves the central 1° of the visual field and provides the greatest visual acuity (Perry and Cowey, 1985). In most cortical and subcortical visual areas, the fovea has the greatest representation, emphasizing the importance of foveal vision in most aspects of visual processing and visually guided behavior (Dow et al., 1981; Van Essen et al., 1984). In order to maximize the efficiency of foveal vision, we must have the ability to align the fovea rapidly upon novel targets that may appear unexpectedly in the visual world and then keep the fovea aligned upon these targets for a sufficient period of time for the visual system to perform a comprehensive analysis of the image. Thus, we have the ability to both move the eyes from one target of interest to another and we also have the ability to suppress eye movements to irrelevant stimuli or locations and maintain foveal vision at a specific location

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as demanded by the task. Thus, saccades are used to redirect the fovea from one target of interest to another and a fixation mechanism is used to keep the fovea aligned on the target during subsequent image analysis. This alternating behavior of saccade–fixate is repeated several hundred thousand times a day and is critical for complex acts such as visual search, reading, or driving an automobile.

Overview of brain areas

Experiments in both humans and animals have contributed extensively to our understanding of the neural circuitry controlling visual fixation and saccade generation. Human experiments typically involve either studying patient groups with discrete lesions to a specific part of the brain that is involved in controlling some aspect of the behavior, or more recently, employing various functional imaging techniques to correlate changes in blood flow or oxygen metabolism in specific brain areas to aspects of the behavior. Additional techniques can be employed in animal experiments such as single cell recording, artificial activation via electrical microstimulation, reversible ac-

tivation or deactivation of neurons with microinjection of transmitter substance, or irreversible lesions.

These different techniques have provided a wealth of data describing the role of various brain areas in the control of visual fixation and saccade generation. Fig. 1 highlights some of the areas that have been identified in non-human primates and analogous areas that have been identified in the human brain. Critical nodes in the network include regions of the parietal and frontal cortices, basal ganglia, thalamus, superior colliculus, cerebellum, and brainstem reticular formation (see Wurtz and Goldberg, 1989; Leigh and Zee, 1991; Moschovakis et al., 1996; Schall and Thompson, 1999; Hikosaka et al., 2000; Munoz et al., 2000; Scudder et al., 2002 for detailed review of aspects of the circuitry). Because these brain areas span almost the entire neuraxis, neurological immaturity, degeneration or malfunction may influence the ability of a subject to maintain visual fixation and generate accurate saccades. Indeed, many neurological and psychiatric disorders are accompanied by disturbances in eye movements and visual fixation, which have become important cues in the identification of the affected brain regions.

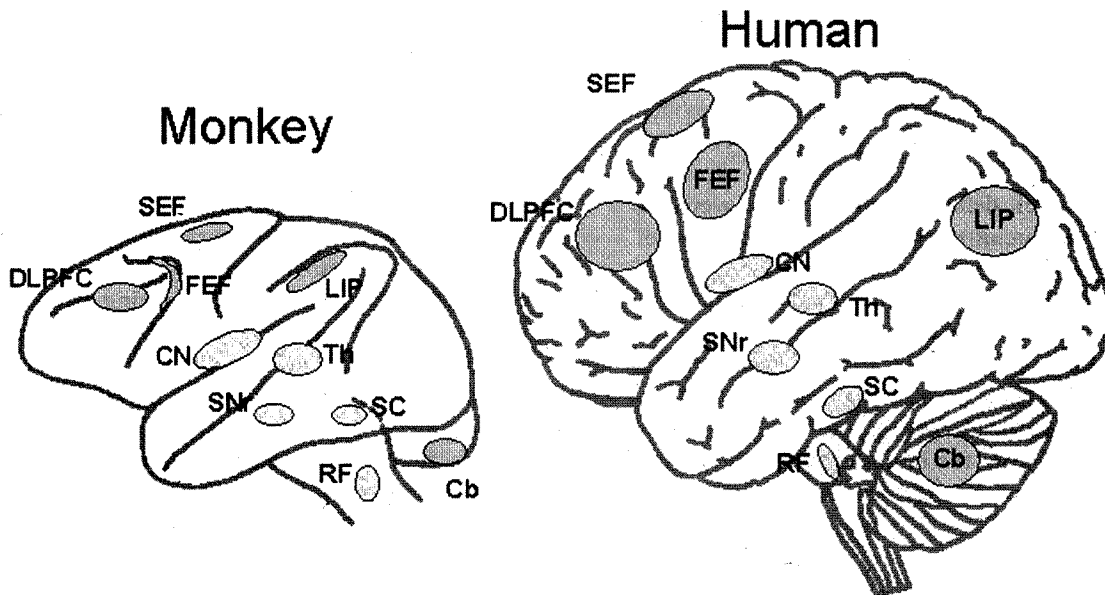


Fig. 1. Brain areas contributing to the control of saccadic eye movements in human and non-human primates. Abbreviations: Cb, cerebellum; CN, caudate nucleus; DLPFC, dorsolateral prefrontal; FEF, frontal eye field; LIP, lateral intraparietal area; RF, reticular formation; SC, superior colliculus; SEF, supplementary eye field; SNr, substantia nigra pars reticulata; Th, thalamus.

Brainstem saccadic burst generator

Many studies involving neuronal recording in awake animals and pathway tracing between areas have provided detailed information regarding the premotor circuitry for the control of saccadic eye movements (Fig. 2). Eye movements are controlled by the synergistic action of six extraocular muscles that are organized into three orthogonal pairs (lateral and medial rectus; inferior rectus and superior oblique; superior rectus and inferior oblique). The motoneurons (MN) that innervate the extraocular muscles are located in cranial nerve nuclei III, IV, and VI in the brainstem (for details see Leigh and Zee, 1991). The MN have a pulse-step discharge during saccadic eye movements. The MN discharge a burst of action potentials (the pulse) for saccades in the on-direction

and have pause in their discharge for saccades in the off-direction. The frequency of this burst is correlated to eye velocity and the number of spikes in the burst scales with amplitude. In addition, there is a tonic component of the discharge of MN (the step) that is correlated to eye position and is present following a saccade to maintain the eye at an eccentric position in the orbit.

The reciprocal pattern of discharge (on-direction burst, off-direction pause) among the MN to create the saccade is generated by premotor neurons located in the mesencephalic, pontine and medullary regions of the reticular formation. The burst component of the MN discharge is generated by the saccadic burst generator circuit in the brainstem reticular formation (see Moschovakis et al., 1996; Scudder et al., 2002 for detailed review). Excitatory and inhibitory

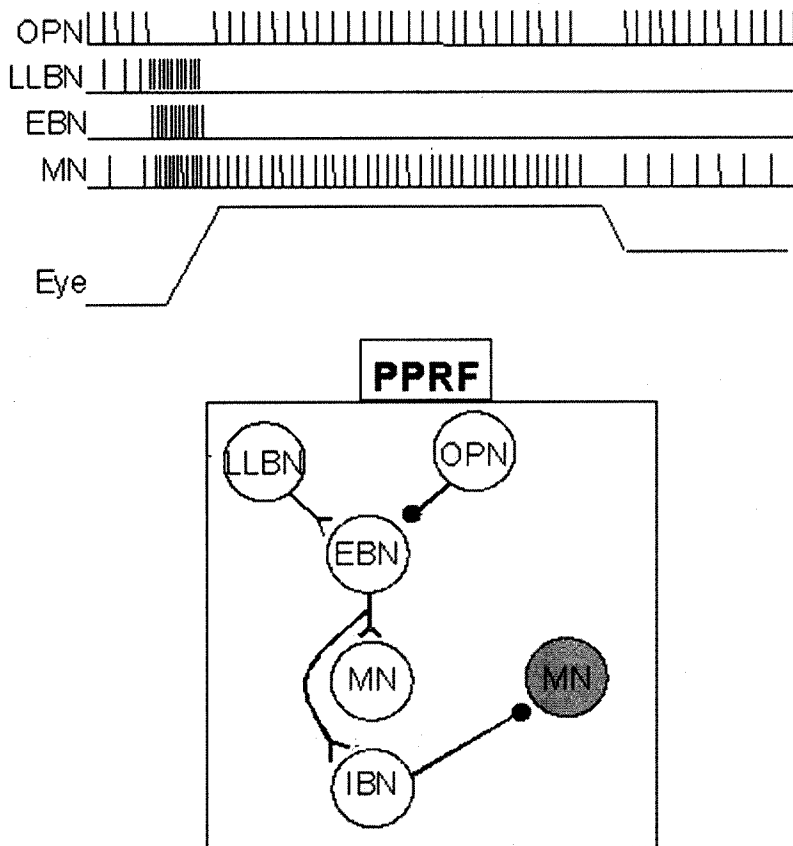


Fig. 2. Identification of elements in the brainstem premotor circuit controlling saccade generation. Agonist motoneuron is white and antagonist motoneuron is shaded gray. Abbreviations: EBN, excitatory burst neuron; IBN, inhibitory burst neuron; LLBN, long-lead burst neuron; MN, motoneuron; OPN, omnipause neuron.

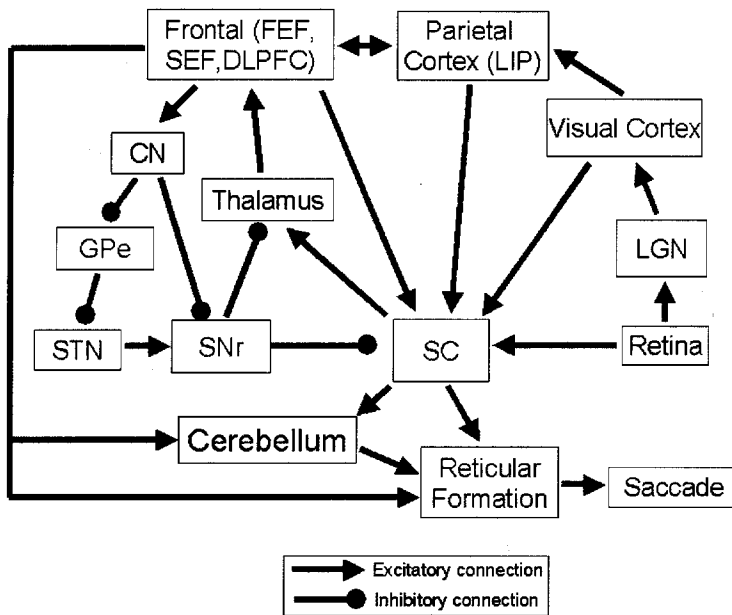


Fig. 3. Circuitry connecting brain areas involved in controlling saccadic eye movements. Abbreviations: CN, caudate nucleus; DLPFC, dorsolateral prefrontal; FEF, frontal eye field; GPe, globus pallidus external; LGN, lateral geniculate nucleus; LIP, lateral intraparietal area; SC, superior colliculus; SEF, supplementary eye field; SNr, substantia nigra pars reticulata; STN, subthalamic nucleus.

burst neurons (EBN and IBN) are silent during fixation and discharge bursts of action potentials for saccades in the on-direction. The EBN excite the on-direction MN monosynaptically and also excite the IBN, which in turn inhibit the antagonist MN. The EBN and IBN for horizontal saccades are located in the paramedian pontine reticular formation (PPRF); the EBN and IBN for vertical saccades are located in the rostral interstitial nucleus of the medial longitudinal fasciculus in the mesencephalon.

Other neurons in the PPRF are believed to control the discharge of EBN and IBN (see Moschovakis et al., 1996; Scudder et al., 2002 for review). Long-lead burst neurons (LLBN), also located in the brainstem reticular formation, discharge a high frequency burst for saccades into the contralateral hemifield. In addition to the burst, these cells also have a low frequency buildup of activity before the burst. It is believed that LLBN project to the EBN and IBN to provide the burst input. The EBN and IBN for horizontal and vertical systems are subject to potent inhibition from omnipause neurons (OPN), also located in the PPRF, which discharge tonically during all fixations and pause for saccades in all directions. Thus,

in order to generate a saccade, OPN must be silenced and the LLBN then activate the appropriate pools of EBN and IBN to produce the saccade command that is sent to the MN. Following completion of the saccade, OPN are reactivated to inhibit the EBN and IBN. The tonic activity of OPN ensures that any early, presaccadic activity among other premotor elements cannot lead to spurious activity among EBN and IBN, which would disrupt fixation.

It has been hypothesized that the step response that is carried by the MN is produced by integration of the pulse (Robinson, 1975). This eye position signal arises from different populations of brainstem neurons (see Scudder et al., 2002 for detailed review). A major source of the horizontal eye position signal to the MN is provided by neurons in the nucleus prepositus hypoglossi, located caudal to abducens nucleus. Neurons in the interstitial nucleus of Cajal in the mesencephalon produce the tonic, step response for the vertical saccadic system. Many neurons in these nuclei have combined burst-tonic discharges: they burst for on-direction saccades and produce tonic discharges that are correlated to eye position. Neural integration of the burst is believed

to be achieved via a distributed network that includes these nuclei and elements in the vestibular nuclei.

Inputs to the brainstem premotor circuitry arise from several structures including the frontal cortex, superior colliculus and cerebellum (Fig. 3). Although our understanding of how these inputs are coordinated to control the actions of the burst generator circuit are incomplete, significant progress has been made in recent years (see Scudder et al., 2002 for detailed review).

Superior colliculus inputs to the saccadic burst generator

The superior colliculus (SC) plays a critical role in the control of visual fixation and saccadic eye movements. The superficial layers of the SC contain neurons that receive direct retinal inputs as well as inputs from other visual areas (Robinson and McClurkin, 1989). These visual neurons are organized into a visual map of the contralateral visual hemifield.

The intermediate layers of the SC contain neurons whose discharges are modulated by saccadic eye movements and visual fixation (see Munoz et al., 2000; Scudder et al., 2002 for review). These neurons are organized into a retinotopically coded motor map specifying saccades into the contralateral visual field. Neurons that increase their discharge before and during saccades, referred to as saccadic neurons, are distributed throughout the intermediate layers of the SC. Neurons that are tonically active during visual fixation and pause during saccades, referred to as fixation neurons, are located in the rostromedial pole of the SC where the fovea is represented. These saccadic and fixation neurons in the SC project to the premotor circuitry in the brainstem reticular formation to influence behavior.

Munoz and Fecteau (2002, this volume) describe characteristics of a dynamic interactions model in which signals related to visual fixation and saccadic initiation interact across the motor map within the superior colliculus. Local interconnections are used as the substrate for motor programs to compete. The outputs from these interactions are then passed to the brainstem premotor circuitry to help guide behavior. Thus, the superior colliculus provides signals to the brainstem premotor circuitry that specify where and when a saccade will occur.

The superior colliculus is a site of multisensory convergence. That is, visual, auditory, and somatosensory information converges onto single neurons in the intermediate layers where it is integrated to guide behavior (see Stein and Meredith, 1993 for review). Such integration is critical for dealing with real world situations in which targets or objects of interest to the saccadic system may not be only visual. This multisensory convergence however leads to a fundamental problem because each of the sensory systems (visual, auditory, and somatosensory) uses very different coordinate systems. Visual inputs are coded retinotopically, auditory inputs are coded craniotopically, and tactile inputs are coded somatotopically. Despite the very different coding systems on the sensory side, the same motor system is used to localize a target. Zambbarbieri (2002, this volume) addresses this issue. She provides a review of auditory saccade characteristics that provide novel insight into the behavior of saccadic mechanisms.

Cerebellar inputs to the saccadic burst generator

There are also important inputs to the brainstem saccadic burst generator that arise from the cerebellum (see Scudder et al., 2002 for review). The superior colliculus and oculomotor areas in the frontal cortex project to the nucleus reticularis tegmenti pontis and other pontine nuclei. Neurons in these nuclei then project to the cerebellar vermis. Neurons in the oculomotor vermis discharge in relation to saccades and microstimulation in this region of the cerebellum can elicit saccades. Purkinje cells in the oculomotor vermis project to the caudal fastigial nucleus. Neurons in the oculomotor region of the caudal fastigial nucleus then project back to the brainstem burst generator circuit to terminate directly on the EBN and IBN. It has been hypothesized that these cerebellar inputs to the premotor circuitry are critical for maintaining saccadic accuracy.

Early models of the saccadic generating system focused only upon the neural elements in the PPRF (Robinson, 1975; Scudder, 1988). In these models, the superior colliculus was believed to provide the input to the burst generator circuit. However, more recently, models have included the superior colliculus, cerebellum, and elements within the PPRF (e.g., Quaia et al., 1999). Enderle (2002, this volume)

describes in considerable detail aspects of a new model of saccade generation that includes inputs to the burst generator from the superior colliculus and cerebellum. In this model, saccades are initiated by inputs to the burst generator circuitry from the superior colliculus and the saccades are then terminated by inputs from the caudal fastigial nucleus. In addition, he provides a novel mechanism for the control of the EBN which provides a large component of the excitatory drive to the MN during saccade execution. Specifically, Enderle uses a Hodgkin–Huxley model of an EBN and proposes that EBN are driven at least initially by a post-inhibitory rebound. In other words, he proposes that when EBN are released from the OPN inhibition, they will discharge at a high frequency automatically without stimulation.

Higher centers involved in presaccadic processing

Inputs to the superior colliculus, cerebellum, and brainstem reticular formation from posterior parietal and frontal cortices and basal ganglia (see Fig. 3) play a critical role in selection of potential saccadic targets to ultimately influence behavior. Visual inputs that are important for maintaining visual fixation or generating saccades are sent initially via the retina and lateral geniculate nucleus to the visual cortex. Projections via the dorsal stream of extrastriate cortex proceed to regions of posterior parietal cortex involved in sensory–motor transformations and attentional processing (see Andersen et al., 1997; Colby and Goldberg, 1999; Glimcher, 2001 for detailed review). One area in particular that lies at the sensory–motor interface and may play an important role in decision making is the lateral intraparietal area (LIP). Visual inputs important for fixation and saccade control can be directed from visual cortex and LIP directly to the superior colliculus to influence premotor processing and ultimately influence behavior.

Frontal cortical oculomotor areas include the frontal eye fields (FEF), the supplementary eye fields (SEF), and the dorsolateral prefrontal cortex (DLPFC). Area LIP projects directly to the FEF and SEF (see Schall, 1997; Schall and Thompson, 1999 for review). The FEF, SEF, and DLPFC all project to the superior colliculus, the cerebellum, and brainstem

reticular formation. In addition, these frontal oculomotor areas are also interconnected. Tinsley and Everling (2002, this volume) describe a role for neurons in the DLPFC in fixation control. In their chapter, they demonstrate that many neurons in DLPFC have discharges that are modulated by the gap period of a gap saccade task. Such modulations are similar to those described previously in the FEF and superior colliculus.

There are several pathways from the frontal cortex through the basal ganglia (see Fig. 3) that participate in presaccadic processing (for detailed review see Hikosaka et al., 2000). These pathways through the basal ganglia allow for the integration of motivation and reward information with saccade planning. There is a *direct pathway* in which the frontal areas project to the caudate nucleus to excite GABAergic neurons which in turn project directly to the substantia nigra pars reticulata. The neurons in the substantia nigra pars reticulata form the major output of the basal ganglia. They are GABAergic and they project to the intermediate layers of the superior colliculus and the thalamus. The thalamus then projects back to frontal and parietal cortices. Via this direct pathway through the basal ganglia, activation of cortical inputs will lead to disinhibition of the superior colliculus and thalamus because the signals pass through two inhibitory synapses.

There is also an *indirect pathway* through the basal ganglia in which a separate set of GABAergic neurons in the caudate nucleus project to the external segment of the globus pallidus. Neurons in external segment of the globus pallidus are GABAergic and project to the subthalamic nucleus. Neurons in the subthalamic nucleus then send excitatory projections to the substantia nigra pars reticulata, which in turn projects to the superior colliculus and thalamus. Thus, the indirect pathway travels through three inhibitory synapses and activation of cortical input will serve to inhibit the superior colliculus and thalamus.

Reflexive versus voluntary saccade control

Saccades can be generated reflexively, to stimuli that appear suddenly in the periphery, or voluntarily, in the absence of any overt sensory stimuli. In addition, the brain has evolved mechanisms to suppress unwanted or reflexive saccades during periods of

visual fixation. There are many cortical areas that participate in the control of saccadic eye movements (Fig. 3). Evidence suggests that different classes of saccades are mediated by different cortical areas (Pierrot-Deseilligny et al., 1991; Gaymard et al., 1998). Pathways from visual and posterior parietal cortices may provide the dominant input to the superior colliculus for the execution of visually triggered saccades. Lesions of posterior parietal cortex increase reaction time of visually guided saccades (Heide and Kompf, 1998).

Regions of frontal cortex that project directly, and indirectly via the basal ganglia, to the superior colliculus play a more critical role in suppressing reflexive saccades and generating voluntary saccades. Lesions of the frontal eye fields (FEF) have only a modest effect on visually guided saccades, but they produce significant impairment in the generation of voluntary or memory-guided saccades (Gaymard et al., 1998, 1999). These movements have increased reaction times and reduced saccadic velocities. Lesions of the DLPFC reduce the ability of subjects to suppress reflexive pro-saccades in the anti-saccade task (Guitton et al., 1985; Pierrot-Deseilligny et al., 1991). Thus, a critical function of the DLPFC may be the voluntary suppression of unwanted or reflexive saccades.

Indeed saccadic inhibition is a very important concept and the neural mechanisms of this phenomenon are only beginning to be understood. In addition to voluntary saccadic suppression, evidence is emerging that there may be a bottom-up mechanism for saccadic suppression in which sudden changes in the visual environment may transiently inhibit the generation of saccadic eye movements. Stampe and Reingold (2002, this volume) describe such mechanisms in their chapter. They show that when subjects perform a visual search task, a sudden change in the visual display will produce a sharp reduction in saccadic frequency within 70 ms. They speculate that such a mechanism may be important to suppress saccades when new visual information is available that may be important to guide behavior.

Conclusions

Fixation and saccadic signals are distributed across a network of brain areas that extends from the pari-

etal and frontal cortices, through the basal ganglia and thalamus, to the superior colliculus, cerebellum, and brainstem reticular formation. Evidence is accumulating to show that these competing signals may interact at multiple levels of the neuraxis. Thus, it is likely that specific functions are not localized to only one brain area. Rather, they may be distributed across multiple areas. The chapters in this section provide compelling evidence for the distributed nature of the neural mechanisms that control saccadic eye movements.

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