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15 Neural Basis of Saccadic Eye Movements in Primates

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> *The muscles were of necessitie provided and given to the eye, that so it might move on every side; for if the eye stoode faste and immoveable, we should be constrained to turne our head and necke (being all of one peece) for to see; but by these muscles it now moveth it selfe with such swiftness and nimbleness, without stirring of the head, as is almost incredible*... Andreas Laurentius (1599)

Introduction

When something attracts our attention, we direct our gaze to it. This is accomplished with a rapid eye movement, a saccade that aims the fovea of both eyes at the object of interest. The process of making a saccade can be broken down into a number of subsidiary processes. These include (a) noticing and identifying a target, (b) deciding to look at it, (c) combining the retinal coordinates of the target with the position of the eye in the orbit to localize the target in space in order to accurately direct the saccade, (d) releasing fixation of the present object, (e) converting the spatial code of the desired change in eye position into the temporal discharge pattern that moves the eyes the desired amount and (f) quickly and accurately moving the eyes. Once the object is fixated, the image is stabilized through a combination of optokinetic nystagmus, the vestibulo-ocular reflex and visually guided pursuit. A richly interconnected, widely distributed neuronal network has evolved to accomplish these processes. The purpose of this chapter is to review the network that is responsible for the saccadic eye movements. The optokinetic and pursuit systems will not be directly considered since a number of reviews of these systems are available (Eckmiller, 1987; Waspe and Henn, 1987; Lisberger *et al.*, 1987).

The oculomotor and visual systems are among the best understood of the brain. Several techniques have contributed to our knowledge of these systems. One of the earliest experimental approaches used in understanding brain function was electrical stimulation (Fritsch and Hitzig, 1870). This technique was used to demonstrate which regions of the brain elicit eye as well as other body movements when stimulated, and it is as instructive today as it was a century ago. Analysis of the effects of clinical and experimental lesions have also helped to show which regions are involved in saccade generation. That is, once a particular area is suspected of generating saccades, it is possible to test its role by removing or inactivating the area. Information of this sort has been derived from clinical studies in human patients and experimental studies in nonhuman primates. If an eye movement deficit occurs following the lesion, then the nature of the impairment reveals something about what the lesioned region contributed to normal gaze control. Both anatomical tracing studies and neurophysiological recordings have helped to further our understanding of the neural basis of eye movements. In particular, the connectivity between different regions, determined anatomically and physiologically, can substantiate or foretell functional relationships. Finally, microelectrode recordings from single neurones in the brains of awake, behaving monkeys, developed in the 1960s (Jasper *et al.*, 1960; Evarts, 1968), have provided a watershed of data about the oculomotor system.

The plan of this chapter is to work through each region of the brain that has been implicated in generating saccadic eye movements. The results of each of these techniques will be reviewed for each region. In an attempt to visualize the patterns of neuronal modulation that have been observed in each oculomotor area, stylized figures are presented to represent the activity of the different populations of neurones in each structure. Much work on the oculomotor system has been done in nonprimate species, especially in the cat, but insofar as possible only primate work will be cited. For more general information the reader is referred to some recent books devoted to the neuronal basis of gaze control (Leigh and Zee, 1983; Carpenter, 1988; Buttner-Ennever, 1989; Wurtz and Goldenberg, 1989).

While saccades are at issue in this chapter, the particular details of saccadic eye movements will only be mentioned incidentally. More detailed descriptions of saccades have also been presented (for example, Dodge, 1903; Robinson, 1964; Hallet, 1985). Also, it should be kept in mind that since the orienting response normally includes head and even body movements as well as eye movements to effect a change of gaze, it is somewhat artificial to consider eye movements isolated from head and body movements (see for example Bizzi, 1974; Tomlinson and Bahra, 1986a,b; Guitton and Volle, 1987). Finally, it is important to understand the intimate though not obligate relation between the focus of gaze and the focus of attention. Ordinarily we pay attention to what we are looking at, but this need not be so; we can see things 'out of the corner of our eye' (for example, Helmholtz, 1909/1962; Posner, 1980; Remington, 1980). Many of the areas that will be reviewed can be understood in terms of directing attention as well as in terms of directing gaze.

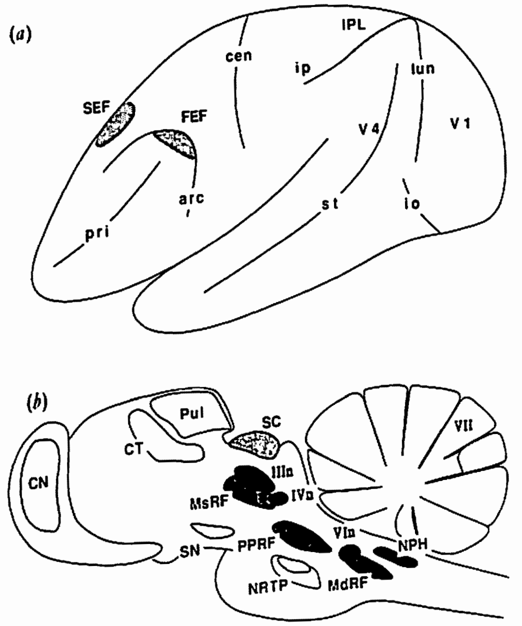

Fig 15.1 *Brain regions involved in generating visually guided saccades. (a) Cortical areas involved in saccadic eye movements are stippled. The density of the stipple is intended to indicate how close an area is to the oculomotor nuclei; the darker the stipple, the closer to the oculomotor nuclei. The major sulci ares are labelled in lower case: pri = principal, arc = arcuate, cen = central, ip = intraparietal, st = superior temporal, lun = lunate, io = inferior occipital. The different cortical areas are labelled in upper case: V1 = visual area 1, primary or striate visual cortex, V4 = visual area 4, also referred to as the prelunate area, IPL = inferior parietal lobule, including the lateral intraparietal area 7a and the medial superior temporal area, FEF = frontal eye field, SEF = supplementary eye field at the rostral end of the supplementary motor area. (b) Composite parasagittal section illustrating the location of subcortical regions involved in saccade generation. CN = caudate nucleus, CT = central thalamus, representing primarily the central and paracentral intralaminar nuclei, the mediodorsal nucleus, the ventrolateral nucleus and the lateral dorsal nucleus, Pul = pulvinar nucleus, SN = substantia nigra pars reticulata, SC = superior colliculus, NRTP = nucleus reticularis tegmenti pontis, VII = lobule VII of the cerebellar vermis, MsRF = mesencephalic reticular formation, PPRF = paramedial pontine reticular formation, MdRF = medullary reticular formation, NPH = nucleus prepositus hypoglossi, IIIn = oculomotor nucleus, IVn = trochlear nucleus, VIn = abducens nucleus.*

Brainstem

Ocular Motor Nuclei

The final process in a saccade is the actual conjugate movement of the eyes which consists of a pulse of force combined with a step in the maintained force that holds the eye at its final position (Robinson, 1964). The neurophysiological mechanisms underlying this stage are relatively well understood. Recent descriptions of the ocular motor nuclei in alert, behaving monkeys have been provided (Robinson, 1981; Henn, *et al.*, 1982; Fuchs and Kaneko, 1985; Fuchs *et al.*, 1985). Thus, this section will only briefly review the large body of data.

Final Common Pathway

The final common pathway consists of the three motor nuclei for the extrinsic eye muscles: the oculomotor (IIIn), the trochlear (IVn) and the abducens (VIn). The abducens nucleus innervates the lateral rectus muscle; the trochlear nucleus innervates the superior oblique muscle, and the different subdivisions of the oculomotor nucleus innervate the medial, superior and inferior recti and the inferior oblique muscles. Fig. 15.1 illustrates the locations of the motor nuclei; the oculomotor and trochlear nuclei lie in the mesencephalon, and the abducens nucleus is in the pons.

Physiological Properties

While there are different kinds of eye muscle fibres, it appears that all motorneurones are active during both slow and fast eye movements. Fig. 15.2 illustrates the typical pattern of modulation of an ocular motor neurone. For a movement in the direction that the innervated muscle pulls ocular motor neurones typically discharge transiently, and then maintain a higher tonic discharge rate as long as the eye remains in the final position (Fuchs and Luschei, 1970; Robinson, 1970; Schiller, 1970; Henn and Cohen, 1973). The discharge begins 5–8 ms before the saccade starts and decays 8–12 ms before the saccade ends. This burst provides the pulse of force that moves the eye;

the duration of the phasic burst is correlated with the amplitude of the saccade. The maintained elevation of activity keeps the eyes in the final position resulting from the saccade; this maintained activity during fixation is a linear function of gaze angle. Movements in the opposite direction are accompanied by a pause and subsequent reduction in activity. There is a degree of variation in the relative magnitudes of the phasic and tonic components in different ocular motor neurones with some cells being predominantly phasic while others, more exclusively tonic.

Connectivity

As mentioned, a motoneurone innervates one muscle pulling in one direction. A conjugate horizontal eye movement, for instance, requires coordinated excitation of the agonist lateral rectus and contralateral medial rectus combined with inhibition of the antagonist medial rectus and contralateral lateral rectus. Similar coordination is necessary for saccades of all directions, of course, but the details are more involved due to the geometry of the extrinsic eye muscles. So obviously there must be coordination between the motoneurones in the different nuclei to provide for conjugate movements, for if the two fovea are not aimed at the same point in the image, diplopia results. This coordination is achieved through various interconnections between the gaze centres in the brainstem through the medial longitudinal fasciculus.

One level of this organization is between the motor nuclei themselves. Not all of the neurones in the motor nuclei innervate the extrinsic eye muscles; there are interneurones that communicate with other ocular motor nuclei (Graybiel and Hartweig, 1974; Baker and Highstein, 1975; Maciewicz *et al.*, 1975; Graybiel, 1977). For example, interneurones in the abducens nucleus synapse on motor neurones in the contralateral oculomotor nucleus that innervate the medial rectus.

Effects of Lesions

Not surprisingly, lesions of the motor neurones or nerves cause severe eye movement deficits (reviewed for example by Henn *et al.*, 1982a; Miller, 1985; Bogousslavsky and Meienberg, 1987). Damage of the motor nuclei or nerves results in a specfic loss of movement of the eye innervated by the compromised nerve in the direction corresponding to the deafferented extrinsic eye muscle.

Effects of Electrical Stimulation

Stimulation of a motor nucleus or a nerve evokes an eye movement in the direction of the muscle's pull (Schiller and Stryker, 1972). In interpreting the results of microstimulation at least two parameters are considered, the minimum current intensity required to elicit an eye movement and the latency of the resulting eye movement. The lower the minimum intensity and the shorter the latency, the closer the stimulated site can be considered to the oculomotor nuclei. Thus, the threshold of current required to evoke an eye movement from the ocular motor nuclei is less than 10 μA, and the latency of the eye movement is no more than 10 ms. Stimulation of a motor neurone does not evoke a saccade; rather the amplitude of the eye movement increases with the duration or the frequency of stimulation.

Supranuclear Regions

The neurones that provide direct input to the ocular motor neurones occupy an extensive longitudinal region in the brainstem reticular formation (Fig. 15.1(b)). These neurones are responsible for generating the burst-tonic pattern of activity in the motoneurones. Horizontal gaze changes are served by the paramedian pontine reticular formation (PPRF) and medullary reticular formation (MdRF) that surround the abducens nucleus. Vertical gaze changes are mediated by the rostral mesencephalic reticular formation (MsRF) which includes the interstitial nucleus of Cajal, the rostral interstitial nucleus of the medial longitudinal fasciculus and also the posterior commissure. The mesencephalic and pontine oculomotor centres are interconnected via the medial longitudinal fasciculus. The nucleus prepositus hypoglossi has also been shown to play a fundamental role in gaze control. Several excellent reviews devoted to this region of the oculomotor system have appeared (Keller, 1981; Robinson, 1981; Henn *et al.*, 1982b; Fuchs *et al.*, 1985; Fuchs and Kaneko, 1985).

Reticular Formation

Single unit recordings in these premotor regions in alert, behaving monkeys have revealed three general neuronal types. Most of the data is derived from the pontine areas subserving horizontal eye movements (Luschei and Fuchs, 1972; Keller, 1974; Henn and Cohen, 1976; van Gisbergen *et al.*, 1981; Hepp and Henn, 1983; Strassman *et al.*, 1986a,b; Scudder *et al.*, 1988), but work on the mesencephalic region has shown comparable properties (Buttner-Ennever and Buttner, 1978; King and Fuchs, 1979; King *et al.*, 1981). Parenthetically, the coordination of these two systems in the generation of oblique saccades is an active area of research (Van Gisbergen, *et al.*, 1985; King, *et al.*, 1986).

Fig. 15.2 illustrates the patterns of activity of the supranuclear units. Burst neurones discharge immediately before ipsilaterally directed saccades. Two groups of presaccadic burst neurones have been described based on the time of onset of their activity. Medium-lead burst neurones begin to discharge 10–15 ms before the saccade; the phrase medium-lead is used to contrast these cells from the short-lead bursting ocular motor neurones. Long-lead burst cells begin up to 100 ms or more before the saccade. This subdivision is somewhat arbitrary, however, since in actuality the onset times form a continuum. Another distinguishing feature, though, is that the onset of activity in medium-lead burst neurones is more abrupt than that in the long-lead which show a gradual prelude of activation. Medium-lead and long-lead burst neurones are not segregated but are found intermingled in the brainstem.

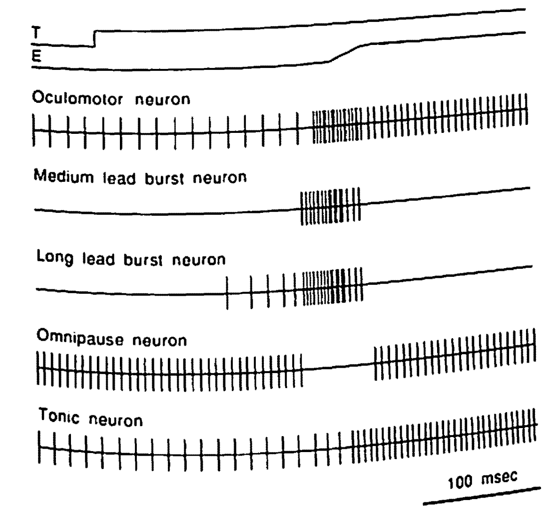

Fig. 15.2 *Stylized representations of the neuronal activity in the brainstem associated with saccades. The appearance of a visual target is indicated by the line marked T. The position of the eye is shown by the line marked E. The time scale of 100 ms is indicated at the bottom. The activity of the different labelled units is shown schematically. Further description is in the text.*

Anatomical connection studies show that the medium-lead burst cells project to the ocular motor nuclei (Buttner-Ennever and Henn, 1976; Highstein *et al.*, 1976; Hikosaka *et al.*, 1978; Yoshida *et al.*, 1981; Strassman *et al.*, 1986; Scudder *et al.*, 1988). It has been suggested that the long-lead and medium-lead burst cells are in a hierarchical relationship with the long-lead burst neurones providing excitatory input to the medium-lead burst cells (Luschei and Fuchs, 1972; Sasaki and Shimazu, 1981; Hepp and Henn, 1983). Consistent with this interpretation is the observation that the long-lead burst cells provide a route for signals from higher centres like the superior colliculus to influence the supranuclear brainstem oculomotor network (Raybourn and Keller, 1977).

The medium- and long-lead burst neurones can be further distinguished into two populations; one group lies rostral to the abducens nucleus in the dorsal PPRF and provides excitatory input to the ipsilateral abducens neurones and, via the medial longitudinal fasciculus, to the contralateral medial rectus neurones. The other group is found in the dorsomedial MdRF, caudal and ventromedial to the abducens nucleus; these provide inhibitory input to the contralateral abducens motor neurones (Strassman *et al.*, 1986; Scudder *et al.*, 1988). These two populations of medium-lead burst neurones are responsible for generating the coordinated phasic burst of activity in the agonist motor neurones and inhibition in the antagonist motor neurones.

The parameters of the medium-lead burst cell activation are well correlated with saccade metrics. In particular, the size of the discharge corresponds to saccade amplitude; the duration of the discharge specifies the duration of the saccade, and the peak firing rate of medium-lead burst cells determines saccade velocity. In addition, the burst cells are tuned for saccade direction with the majority surrounding the abducens nucleus responding best for horizontal saccades.

Another population of neurones in the supranuclear brainstem gaze centres displays a complementary pattern of modulation. Omnipause neurones have a high maintained level of activity while the monkey fixates or pursues a visual target; immediately before a saccade these cells exhibit a pause or suppression of activity. The pause begins 10–15 ms before the saccade, and the duration of the pause is correlated with the duration of the saccade. The omnipause neurones are located rostral to the abducens nucleus in a thin, dorsoventrally elongated nucleus on either side of the midline, the nucelus raphe interpositus (Buttner-Ennever *et al.*, 1988). Evidence has been presented in the cat that omnipause neurones inhibit the medium-lead burst neurones (Nakao *et al.*, 1980; Curthoys *et al.*, 1984; Langer and Kaneko, 1984). Moreover, electrical stimulation in the vicinity of these omnipause neurones prevents the execution of saccades (Keller, 1974). Furthermore, it has been shown that individual omnipause neurones send their axons across the midline and arborize rostral and caudal to the abducens nucleus, in the regions where excitatory and inhibitory burst neurones are found (Ohgaki *et al.*, 1987). One source of afferents to the omnipause neurones is the deep layers of the superior colliculus (Raybourn and Keller, 1977; Langer and Kaneko, 1984). This input seems to be responsible for indirectly triggering the initiation of a saccade since stimulation of the superior colliculus inhibits omnipause neurones (Raybourn and Keller, 1977). Omnipause neurones also receive input from inhibitory medium-lead burst cells (Nakao *et al.*, 1980; Langer and Kaneko, 1984); this connection appears to regulate saccade duration.

The third group of units found in the supranuclear oculomotor regions exhibits a maintained tonic discharge that varies systematically with the position of the eye in the orbit. The activity of these tonic neurones does not change with saccades; i.e. there is no phasic burst or suppression associated with saccades. It is thought that these cells provide to ocular motor neurones the step of maintained activity that holds the eye at the final position, but because the tonic neurones are so widely distributed it has not been possible to test this connection directly. To derive their eye position signal, it appears that the tonic neurones integrate input from the medium-lead burst cells (Yoshida *et al.*, 1981; Strassman *et al.*, 1986a,b).

Nucleus Prepositus Hypoglossi

One brainstem location in the caudal medulla containing a high density of eye position neurones is the nucleus prepositus hypoglossi (NPH). Single unit recordings have been accomplished in this region in the cat (Baker *et al.*, 1976; Gresty and Baker, 1976; Lopez-Barneo *et al.*, 1982). The units discharge with all types of eye movements in all directions; although most cells are responsive in relation to horizontal eye movements. Some of the units encode eye position; other units encode both eye position and velocity. Also, some neurones in the NPH are visually responsible; they have large, binocular receptive fields, respond well to moving stimuli and are directional. The original of these visual responses would seem to be the superior colliculus. It is not presently known whether similar visual responses are present in the NPH of the monkey, although it seems likely since it receives afferents from the inferior parietal lobule (Faugier-Grimaud and Ventre, 1989) and frontal eye fields (Stanton *et al.*, 1988b). Stimulation of the NPH with currents around 20 μA results in ipsilateral nystagmus (Cannon and Robinson, 1987). The NPH receives input from the vestibular nuclei, the cerebellum and the reticular gaze centres (Walberg, 1961; Angaut and Brodal, 1967; Carpenter *et al.*, 1970; Kotchabhakdi *et al.*, 1978) and projects directly to the ocular motor nuclei (Graybiel and Hartweig, 1974; Baker and Berthoz, 1975; Baker *et al.*, 1977; Graybiel, 1977). The NPH also sends efferents to the cerebellum (Brodal, 1952; Torvik and Brodal, 1954; Alley *et al.*, 1975). Selective ablation of the NPH in both cat (Cheron *et al.*, 1986a,b) and monkey (Cannon and Robinson 1987) results from a loss in the step component of the saccade, i.e. the eye drifts back from the eccentric position following a saccade. Based on these results, it appears that the NPH serves to integrate the eye velocity signal of the medium-lead burst cells to encode eye position.

Effects of Electrical Stimulation

Stimulation of the PPRF elicits ipsilateral eye movements (Cohen and Komatsuzaki, 1972; Keller, 1974). The minimum current required to elicit an eye movement from the PPRF is around 5–10 μA, and the latency of the eye movement is no more than 10 ms. The evoked eye movement is not a saccade; instead the amplitude of the eye movement increases with stimulation frequency and duration. The amplitude and direction of the evoked eye movement do not vary with the position of the eye in the orbit.

A great deal of information can be derived from the results of stimulating supranuclear pontine centres in behaving monkeys while they are preparing saccadic eye movements (Sparks *et al.*, 1987). In such experiments monkeys are trained to saccade to briefly flashed visual targets. As mentioned, when the PPRF in the vicinity of the burst cells is electrically stimulated, the eyes are driven horizontally. When such stimulation is delivered after the target is flashed but before the saccade is initiated, the monkeys can compensate for the perturbation in the eye position and accurately fixate the target. This has been observed following stimulation of many but not all sites in PPRF; what distinguishes the two types of sites has yet to be determined. These results indicate that information about eye position is available downstream from those sites in PPRF at which the monkeys could compensate for the stimulus elicited eye perturbation.

In these same experiments the pontine stimulation occasionally triggered premature saccades. The direction and amplitude of this premature eye movement depended not only on the site of stimulation but also on the location of the visual stimulus, and the amplitude of the premature saccade increased with the delay between visual target presentation and electrical stimulation. This result suggests that the input to the saccade generator takes time to build up which is consistent with the idea that the input to the saccade generation network arrives via the long-lead burst cells within the PPRF from presaccadic cells in the superior colliculus and cortex (see below).

Effects of Lesions

Not surprisingly, lesions of the supranuclear oculomotor centres cause permanent and severe eye movement deficits (reviewed by Cohen and Henn, 1972; Miller, 1985; Bogousslavsky and Meienberg, 1987). Unilateral PPRF lesions result in a permanent paralysis of ipsilateral conjugate gaze. Bilateral lesions of the rostral MRF result in a vertical gaze palsy. Interruption of the medial longitudinal fasciculus causes internuclear ophthalmoplegia, a condition in which one eye fails to adduct in a conjugate eye movement. The basis of this effect is the loss of input to the medial rectus motor neurones in the oculomotor nucleus. Finally, large bilateral lesions of the pons that include the midline impair both horizontal and vertical eye movements. Due to the density and complexity of the brainstem neuropil, different constellations of deficits can occur depending on the exact location of the lesion. In fact, a testimony to how well this part of the oculomotor system is understood is the fact that the nature of the eye movement deficit is used to diagnose the location of brainstem lesions. As should be evident from the foregoing description, however, horizontal gaze deficits are more readily and successfully diagnosed.

Model of Saccade Generation

The information about the response properties and connectivity of the neurones in the brainstem has provided for detailed models to account for the generation of a saccade (Robinson, 1975; Becker and Jurgens, 1979; Keller, 1981; van Gisbergen, 1981; Hepp and Henn, 1982; Fuchs *et al.*, 1985; Tweed and Vilis, 1985; Scudder, 1988). It is believed that the various elements in the brainstem comprise a local feedback circuit that is responsible for generating the saccade. During fixation or pursuit, when saccades are undesired, the medium-lead burst cells are inhibited by the omnipause neurones. A saccade is initiated when input from higher centres (e.g. superior colliculus and/or cortex, see below) asserts the location of the target and inhibits the omnipause neurones. The release of inhibition from the omnipause neurones allows the medium-lead burst cells to discharge that in turn excite the motor neurones that drive the eyes in a saccade. The burst cells will discharge until the motor error signal is reduced. The current eye position is derived in another neural network that integrates the burst cell discharge. This eye position signal goes to the motoneurones to provide the step which will ultimately hold the eye at its final position, and the eye position signal is also fed back onto the burst cells as one component of the motor error. Thus, the excitatory burst neurones are inhibited by the signal of the current eye position so that when the fovea reaches the target and the dynamic motor error is reduced to zero, the inhibition cancels the excitation and the motoneurones cease to be driven and the eye stops moving. The decay of activity in the inhibitory burst neurones releases their inhibition on the omnipause neurones so that they reinstate their high discharge rate and in so doing inhibit the burst neurones until the next saccade.

While it is clear that the output of this system is an eye movement, the input is less clear. The input could be the position of the target on the retina. It could also be the location of the target in space by taking into account both the retinal location of the target and the position of the eye. The input might also be considered to be the desired change in eye position, i.e. the initial motor error. Another input to the circuit is the signal to trigger the saccade. The remainder of this chapter considers the various sources of this input.

Nucleus Reticularis Tegmenti Pontis

The precerebellar nucleus reticularis tegmenti pontis (NRTP) occupies a large region dorsal to the basilar pontine nuclei (Fig. 15.1). Single unit recordings indicate that a dorsomedial section of it is involved in visuomotor behaviour (Kato *et al.*, 1982; Crandall and Keller, 1985). The NRTP receives afferents from the accessory optic system and the nucleus of the optic tract (Precht *et al.*, 1982; Maekawa *et al.*, 1984), two regions that are known to play a fundamental role in generating pursuit eye movements (Grasse and Cynader, chapter 5). NRTP lesions in cats impair optokinetic nystagmus (Kato *et al.*, 1982). This section will, however, consider the role of NRTP in generating saccadic eye movements.

Physiological Properties

A small group of units discharge following the appearance of a visual stimulus, that is the target for a saccade. The latency of response is approximately 70 ms, and the units have well defined visual receptive fields. The visually responsive NRTP cells respond to moving stimuli and are also active during pursuit (Keller and Crandall, 1983). A larger population of neurones discharge in relation to both the appearance of a visual stimulus and the subsequent saccade. The visual response of all of these cells is enhanced if the stimulus is the target for a saccade; this enhancement is spatially selective, i.e. the visual stimulus must fall within the cell's receptive field. The cells with both visual and motor activity resemble a particular class of neurone recorded in the superior colliculus called quasi-visual cells (see below). In short, while this class of neurone does respond to a visual stimulus, it will also fire when the monkey must make a saccade into the region of the receptive field even though no stimulus is present.

The presaccadic burst cells in NRTP discharge on average 30 ms before saccades that are directed at a particular angle and amplitude into the ipsilateral hemifield. The amplitude and direction of saccades for which a given cell is active defines that cell's movement field. Half of the presaccadic burst cells have relatively localized movement fields, and the other half have expansive movement fields occupying up to a hemifield. These units also discharge in association with spontaneous saccades in the dark. The movement fields of most of the presaccadic burst units are retinotopically organized, but the activity of almost a quarter of the cells varies with the initial gaze angle. In other words, eye position is encoded to some extent in the NRTP. Another small group of units discharge during fixation of a visual stimulus. These units pause during saccades, but the onset and offset of modulation is not tightly linked to either the appearance of the visual stimulus or the refixation.

Connectivity

The origin and perhaps the functional role of the visuomotor neuronal activity in the NRTP can be surmised from its anatomical relations. The dorsomedial sector of the NRTP receives descending projections from the frontal and supplementary eye fields (Kunzle and Akert, 1977; Leichnetz *et al.*, 1984b; Huerta *et al.*, 1986; Huerta and Kaas, 1988; Stanton *et al.*, 1988b). It also receives a substantial input from the superior colliculus (Harting, 1977). The responses of the neurones in the NRTP resemble what is observed in these cortical regions or in the superior colliculus (reviewed below). The NRTP does not project directly to the PPRF (Buttner-Ennever and Henn, 1976), but there is evidence for a projection to the abducens nucleus (Langer *et al.*, 1986). Primarily, however, it is a major mossy fibre afferent source to a specific region of the vermis and flocculus of the cerebellum. Thus, it appears that the collicular and cortical visuomotor regions involved in saccade generation have access to the cerebellum via the NRTP. We are accordingly led to review what role the cerebellum plays in gaze control.

Cerebellum

Effects of Lesions

Once a saccade is in flight, it must stop accurately. That is, the pulse and step of activity must be balanced to stop the saccade on target and then keep the eye at rest. The cerebellum appears to play a key role in adjusting the gain of the pulse and step that move the eyes. Evidence to support this claim is derived from ablation studies (Aschof and Cohen, 1971; Ritchie, 1976; Optican and Robinson, 1980; Zee *et al.*, 1981). It appears that the regulation of the pulse and step gain are spatially segregated in the cerebellum. In the first place, ablation of the entire cerebellum results in saccades that are too long, and the eye drifts from its final position; in other words the gain of neither the step nor the pulse are appropriate. In contrast, ablation of the midline cerebellar vermis results in saccades that are too long, but the eye does not drift. In other words, the control of the pulse gain is lost, but the step gain control is preserved. Finally, bilateral ablation of the flocculus results in excessive drift of the eyes after a saccade, i.e. loss of control of the step gain.

Effects of Electrical Stimulation

Several classical studies have demonstrated that electrical stimulation of the cerebellum elicits eye movements (Hitzig, 1874; Ferrier, 1876; Mussen, 1927; Dow, 1935; Magoun *et al.*, 1935; Hare *et al.*, 1936, 1937; Hampson *et al.*, 1950; Cohen *et al.*, 1965). More recent investigations using alert monkeys have localized the effects of microstimulation. Different types of eye movements are elicited by stimulation of different regions of the cerebellum. Saccades can be elicited from the posterior cerebellar vermis; saccades and smooth eye movements are elicited from crus I and II and lobulus simplex of the hemispheres, and nystagmus follows stimulation of the flocculus, nodulus and uvula (Ron and Robinson, 1973). Low intensity (less than 10 µA) stimulation of lobule VII of the vermis (Fig. 15.1(b)) elicits ipsilaterally directed saccades with latencies of approximately 10 ms (Fujikado and Noda, 1987). The lowest thresholds are required in the fibre layers and not in the cellular layers of the cerebellar cortex (McElligott and Keller, 1984; Fujikado and Noda, 1987). Even so, there is evidence that Purkinje cells are necessary to mediate the stimulus elicited saccades; kainic acid destruction of Purkinje cells in a restricted region of the oculomotor vermis eliminates saccades evoked by stimulation of that region (Noda and Fujikado, 1987a).

There appears to be a topographic map of saccadic direction in the vermis (Ron and Robinson, 1973; McElligott and Keller, 1984; Noda and Fujikado, 1987b). Vertically directed saccades are elicited from the medial vermis, and the horizontal component of the saccade increases as more lateral areas are stimulated. As the stimulating electrode is advanced through the cerebellum, the elicited saccades change from upward to downward.

The saccades elicited by microstimulation of the cerebellum tend to have curved trajectories. Unlike stimulation of superior colliculus or frontal eye fields (see below), increasing the intensity of the current in the cerebellum changes the amplitude and the direction of the saccade. This would seem to be due to current spread in the dense cerebellar neuropil. Another very interesting property of saccades evoked by cerebellar stimulation is that the direction and amplitude of the saccade varies with initial eye position (Ron and Robinson, 1973; McElligott and Keller, 1984); this is in contrast to superior colliculus or frontal eye fields (see below) and indicates that the cerebellar vermis encodes the position of the eye in the orbit. Stimulation of the vermis during a saccade in flight changes the eye's trajectory in a specific fashion; contralaterally directed saccades are foreshortened, but ipsilaterally directed saccades are not affected (Keller *et al.*,1983).

Physiological Properties

Vermis

There is abundant saccade-related neuronal activity in the vermis (Llinas and Wolfe, 1977, Kase *et al.*, 1980; McElligott and Keller, 1982) as well as activity related to smooth pursuit (Kase *et al.*, 1979; Suzuki *et al.*, 1981). Mossy fibres can be divided into three patterns of activity related to saccadic eye movements (Kase *et al.*, 1980). As shown in Fig. 15.3, these are long-lead burst, short-lead burst and burst-tonic units. The activity of long-lead burst mossy fibres begins a prelude on average 160 ms before the saccade, and they burst about 16 ms before the saccade. The short-lead burst mossy fibres begin to discharge on average 7 ms before the saccade. The discharge of burst-tonic mossy fibres begins an average of 0.2 ms before the saccade. Some short-lead burst mossy fibres display a directional preference.

Purkinje cells in the vermis also show a variety of patterns of modulation. A few pause for all directions of saccades. Others show bursts that begin with the onset of the saccade (average of 0.6 ms before the saccade is initiated) and last for the duration of the saccade. Other Purkinje cells burst after saccade onset and continued to discharge until after the saccade is completed. The onset of activity in this latter population of units averages 40 ms before the end of the saccade, and the offset is 70 ms after saccade termination. The baseline activity of many mossy fibres and Purkinje cells varies with the angle of gaze. This final result is consistent with the observation, described above, that the eye movements elicited by microstimulation of the vermis vary with eye position.

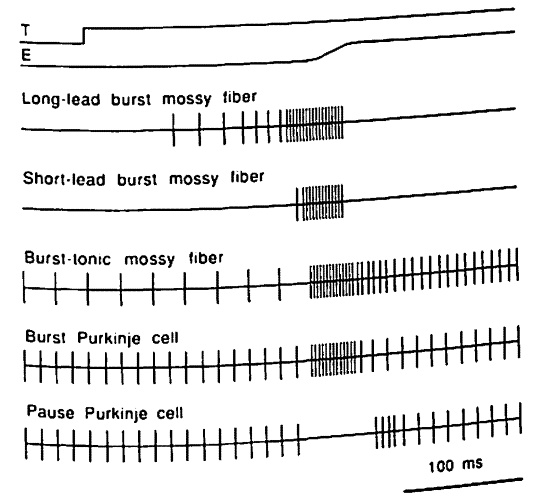

Fig. 15.3 *Neuronal activity in cerebellum associated with saccades. Conventions are as in Fig. 15.2.*

Flocculus

Purkinje cells in the flocculus also discharge in relation to saccades (Noda and Suzuki, 1979a). In contrast to the vermis, most Purkinje cells in this region exhibit a pause in activity associated with saccades; the duration of the pause is correlated with the length of the saccade, beginning on average 10 ms before the saccade. Other Purkinje cells exhibit bursts of activity during saccades; the burst precedes the saccade by an average of 4 ms. In a minority of Purkinje cells a burst is associated with saccades in one direction and a pause with saccades in the opposite direction. Some Purkinje cells are tuned for the direction of saccade, and others are not. The tonic activity of Purkinje cells in this region of the cerebellum also varies with eye position (Noda and Suzuki, 1979b).

Mossy fibres and non-Purkinje cell elements in the flocculus exhibit patterns of saccade-related modulation similar to those seen in the vermis (Noda and Suzuki, 1979c). Long-lead burst cells begin to discharge on average 113 ms before saccades; these units display amplitude and directional tuning. Short-lead burst units become active 7 ms before saccades and also are directionally tuned to different degrees. Burst-tonic units exhibit a saccade-related burst and gaze angle related tonic activity during fixation. Finally, tonic units are characterized only by their maintained activity that varies with eye position.

Connectivity

The connections of the oculomotor cerebellar flocculus (Langer *et al.*, 1985) and vermis (Yamada and Noda, 1987) have been studied in monkeys. The major source of input to cerebellar oculomotor vermis and flocculus, as reviewed, is a dorsomedial region of NRTP (Lafleur *et al.*, 1974; Langer *et al.*, 1985; Yamada and Noda, 1987). Visuomotor signals have other routes of access to the cerebellar vermis too. The dorsal and dorsal-lateral pontine nuclei also provide afferents to the vermis (Hoddevik *et al.*, 1977; Brodal, 1979). These pontine nuclei receive cortical afferents from striate cortex, a number of extrastriate visual areas, the lateral interparietal area, and the prearcuate region of frontal cortex (Kunzle and Akert, 1977; Brodal, 1978, 1979; Glickstein *et al.*, 1980; Galletti *et al.*, 1982; Maunsell and Van Essen, 1983; Ungerleider *et al.*, 1984; Glickstein, 1985; May and Andersen, 1986) as well as the superior colliculus (Harting, 1977). Moreover, recent studies have shown that the dorsolateral pontine nuclei carry signals important for smooth pursuit (Keller and Crandall, 1983; Suzuki and Keller, 1984; Mustari *et al.*, 1988; Thier *et al.*, 1988).

The efferents of the oculomotor cerebellum reveal the various points at which it can influence saccade generation. It appears that the ocular motor nuclei themselves receive weak if any direct input from the cerebellum (Carpenter and Strominger, 1964; Graybiel and Hartweig, 1974; Graybiel, 1977; Batton *et al.*, 1977; Langer *et al.*, 1986; Gonzalo-Ruiz *et al.*, 1988). The Purkinje cells of the oculomotor vermis terminate in specific regions of the ipsilateral fastigial nucleus (Yamada and Noda, 1987). Consistent with this projection, low intensity microstimulation of the fastigial nucleus also elicits saccadic eye movements (Noda *et al.*, 1988) and saccade-related neuronal activity has been recorded in the fastigial nucleus (Hepp *et al.*, 1982). The fastigial nucleus projects to supranuclear oculomotor sites including the PPRF (Gonzola-Ruiz *et al.*, 1988), the NRTP (Asanuma *et al.*, 1983a), the region of omnipause neurones in the brainstem (Langer and Kaneko, 1984), the deep layers of the superior colliculus (Huerta and Harting, 1984a,b; Gonzalo-Ruiz *et al.*, 1988), the mediodorsal, intralaminar, ventrolateral and ventroposterior lateral thalamic nuclei (Hendry *et al.*, 1979; Thach and Jones, 1979; Kalil, 1981; Asanuma *et al.*,1983c; Gonzalo-Ruiz *et al.*, 1988). Some fastigial nucleus neurones in the rat send collateral branches to both superior colliculus and the PPRF or to both PPRF and the paraoculomotor region (Gonzala-Ruiz and Leichnetz, 1987).

Summary

The results of lesion, stimulation and recording studies in the cerebellum converge on the interpretation that the cerebellum serves to calibrate the saccade generation system through signalling the position of the eye in the orbit. The eye position signal in the cerebellum may be derived from eye muscle proprioceptive afferents (Fuchs and Kornhuber, 1969; Wolfe, 1971; Baker *et al.*, 1972; Schwarz and Tomlinson, 1977; Steinbach, 1987) as well as a corollary discharge represented by the presaccadic and postsaccadic cells that appear to encode the position of the eye in the orbit. The representation of eye position is verified by the microstimulation results. The effects of stimulation during saccades also reveals the point in the process of generating a saccade at which the cerebellum exerts its effects. That is, the fact that stimulation of the vermis during a saccade slows and shortens the eye movement indicates that the motor error signal is reduced. One way for the cerebellar vermis to effectively reduce the motor error signal is by changing the eye position signal. This would require that the cerebellum exert its influence prior to the supranuclear medium-lead burst cells. Thus, it appears that the contribution of the cerebellum to the motor error signal may be an internal representation of eye position.

Superior Colliculus

The superior colliculus is a laminated structure that crowns the midbrain (Fig. 15.1(b)). While it has been the focus of many experiments over the years (starting with Adamuk, 1870), as recently as a decade ago it was possible for one reviewer to write '... there is doubt whether the colliculus plays an important role in conjugate eye movements.' (Bender, 1980, p.50). Further studies of this structure have revealed its fundamental role in the generation and guidance of saccades. A number of reviews of superior colliculus function have already been presented (Wurtz and Albano, 1980; Sparks and Mays, 1981; Schiller, 1984; Sparks, 1986, 1988), but work on this structure certainly has not slowed (Stein and Meredith, chapter 4).

Connectivity of Upper Layers

As a first approximation, the superior colliculus can be considered in two subdivisions. The superficial division is visual, and the deeper division (also referred to as the intermediate and deep layers) is oculomotor. The upper division receives afferents from the retina (Wilson and Toyne, 1970; Hendrickson *et al.*, 1970; Tigges and Tigges, 1970; Lund, 1972a,b; Tigges and O'Steen, 1974; Hubel *et al.*, 1975; Schiller and Malpeli, 1977; Perry and Cowey, 1984). The retinotectal projection arises from no more than 10% of all the ganglion cells; just a few of these ganglion cells are in the P-α/A/broad-band class, and the remainder have small somas and sparsely branched dendritic trees corresponding to the heterogeneous W cell class (see Kaplan, chapter 2). The upper layers of the superior colliculus also receives major afferents from the striate and extrastriate cortex (Garey *et al.*, 1968; Abplanalp, 1970; Harting and Noback, 1971; Kadoya *et al.*, 1971; Lund, 1972b; Lund *et al.*, 1975; Finlay *et al.*, 1976), the frontal eye fields (Kuypers and Lawrence, 1967; Astruc, 1971; Kunzle and Akert, 1974; Kunzle *et al.*, 1976; Leichnetz *et al.*, 1981; Fries *et al.*, 1974; Huerta *et al.*, 1986; Stanton *et al.*, 1988b). The superficial layers of the superior colliculus project to the dorsal lateral geniculate nucleus, the pregeniculate nucleus, and the inferior and lateral pulvinar (Mathers, 1971; Harting *et al.*, 1973; Benevento and Fallon, 1975; Trojanowski and Jacobson, 1975b; Harting *et al.*, 1978). They are also interconnected with the pretectum (Harting *et al.*, 1973; Benevento *et al.*, 1977).

Physiological Properties of Upper Layers

The pattern of modulation of units in the superior colliculus is illustrated in Fig. 15.4. As expected, neurones in the upper layers of the superior colliculus are visually responsive (Humphrey, 1968; Schiller and Koerner, 1971; Goldberg and Wurtz, 1972a; Updyke, 1974; Marrocco and Li, 1977). The response latency ranges from 40–80 ms with most cells responding after 40–50 ms; visual latency increases with depth in the colliculus. The responses in the monkey superior colliculus typically do not depend on the form, colour or direction of motion of the stimulus, but they do respond well to small flashing or moving spots. Directionally tuned responses can be observed for stimuli moving relative to a drifting textured background, but this directional tuning is more pronounced in the deeper layers (Bender and Davidson, 1986). The visual cells in the superior colliculus receive an extraretinal signal that allows them to respond specifically to stimuli moving in the world and not to stationary stimuli across which the eye moves (Robinson and Wurtz, 1976; Richmond and Wurtz, 1980). Spatiotemporal interactions in these cells also appears to modulate their responsiveness to stimuli during saccades (Wurtz *et al.*, 1980). There is an orderly retinotopic map in the superior colliculus with the fovea represented rostrally, and the upper visual field represented medially (Lane *et al.*, 1971; Cynader and Berman, 1972; Goldberg and Wurtz, 1972a; Kaas *et al.*, 1974).

The activity of approximately half of these superficial layer visual cells is modulated in relation to saccades; that is, when the stimulus in their receptive field is the target for a saccade, the visual response is enhanced (Goldberg and Wurtz, 1972b; Wurtz and Mohler, 1976a). This enhancement, it will be seen, is a characteristic property of higher visuomotor centres. The enhancement is not observed in the most superficial sublamina of the colliculus where the retinal afferents terminate, so cortical influences are necessary. The enhancement is expressed as either a more vigorous initial response or an elevated discharge rate after the initial visually evoked response. Since it only occurs for stimuli falling in the cell's receptive field, the enhancement is not due to some general arousal process. The enhanced response does not require the abrupt appearance of the stimulus but can occur when a continuously visible stimulus is cued as the target for a saccade. Also, the enhancement begins 200–300 ms before the saccade and intensifies as the stimulus is flashed nearer the time of saccade initiation. Finally, the visual responses are not enhanced if the monkey responds with an arm and not an eye movement. These results indicate that this enhancement of the visual response of upper layer superior colliculus units may be interpreted as reflecting the internal process of selective attention associated with initiation of a visually guided saccadic eye movement.

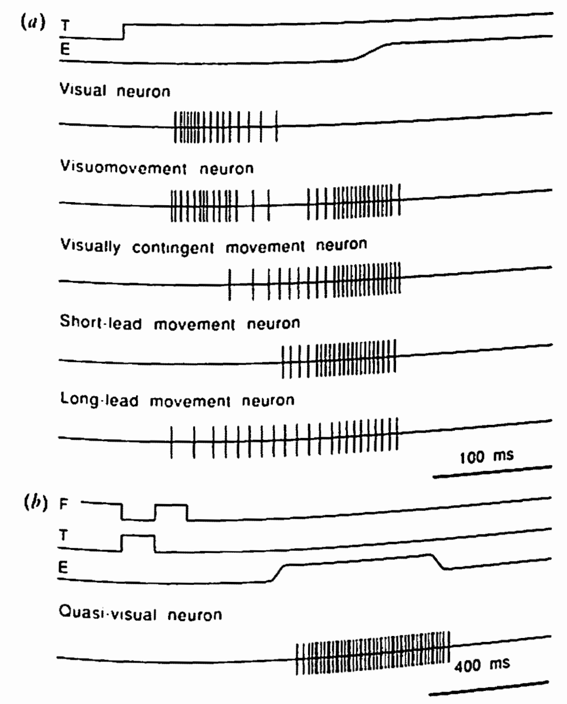

Fig. 15.4 *Neuronal activity in the superior colliculus associated with visually guided saccades. (a) Conventions are as in Fig. 15.2. (b) Double saccade task. The monkey fixates a central spot (F). When the fixation spot disappears, a peripheral target (T) is flashed followed immediately by a flash of the central spot (F). The monkey is required to saccade to the position of the first flash and then to the position of the second flash. Note the longer time scale of 400 ms.*

Physiological Properties of Deeper Layers

Visually responsive units are also recorded in the deeper subdivision of the primate superior colliculus (Schiller and Koerner, 1971; Cynader and Berman, 1972; Updyke, 1974). The visual response of these deeper collicular neurones arrives from the visual cortex (Schiller *et al.*, 1974) and is derived from the broad-band, magnocellular geniculostriate pathway (Schiller *et al.*, 1979). Interestingly, the visual response enhancement observed in the upper layer cells is not present in the deep division (Goldberg and Wurtz, 1972b; Mohler and Wurtz, 1976).

Another interesting type of visually responsive unit has been called quasi-visual (Mays and Sparks, 1979). These units discharge following the appearance of a target within their receptive field and maintain their activity until a saccade to the target. The defining characteristic of these cells, however, is that to be activated, they do not require the actual appearance of the target in their receptive field. The task in which this is observed is illustrated in Fig. 15.4(b). A monkey is presented two targets that he must fixate sequentially, but both targets are presented before the first saccade is made. The arrangement of the targets is such that following the first saccade, the location of the second target would fall in the cell's receptive field. Following the first saccade, as soon as the location of the second target falls in the cell's receptive field, the quasi-visual neurone begins to fire until the second saccade is executed. The modulation of this population of neurones has been argued to signal the initial motor error between the desired and present eye position; however, it is not presently known where this population of neurones projects in order to interpret how this signal is utilized. Actually, this pattern of modulation has been observed also in the basal ganglia, posterior parietal cortex and frontal cortex (see below).

In the deeper division of the superior colliculus are found many neurones that discharge in association with saccadic eye movements (Schiller and Koerner, 1971; Wurtz and Goldberg, 1972a; Sparks, 1975; Mohler and Wurtz, 1976; Sparks *et al.*, 1976; Sparks, 1978; Mays and Sparks, 1980; Nagata and Kase, 1983; Sparks and Porter, 1983; Waitzman *et al.*, 1988). This discharge is related to saccadic eye movements and not associated head orienting movements (Robinson and Jarvis, 1974). The onset of activity in the population of presaccadic units ranges from 50–200 ms. While it is not possible to clearly distinguish short-lead from long-lead presaccadic cells, the deeper the unit in the superior colliculus, the earlier the onset. Still, certain of these presaccadic cells resemble the supranuclear medium-lead burst cells in that they exhibit a distinct burst 20–50 ms before the saccade. There is a high correlation between the onset of activity of these presaccadic burst cells and the time of initiation of the saccade

(Sparks, 1978) which indicates that this population most likely projects to the supranuclear brainstem saccade generator.

The response of these units is described by a particular movement field given by the direction and amplitude of the saccades for which the cell responds. The movement fields of collicular presaccadic units are fairly broadly tuned, a point to which we will return momentarily. The movement fields are arranged in a retinotopic map that corresponds to that represented by the visual cells in the superficial layers, and the size of the movement field increases with the eccentricity represented and also with depth in the colliculus. The saccade-related discharge of these units does not vary with the position of the eye in the orbit; in other words, the movement fields are represented in a retinal rather than a spatial coordinate system.

The presaccadic movement neurones described so far discharge in relation to the fast phase of the vestibulo-ocular reflex or optokinetic nystagmus as well as to visually guided saccades. In contrast, a specific sub-population of the movement neurones in the superior colliculus discharge only in association with visually triggered saccades; these cells do not fire for saccades made in the dark (Mohler and Wurtz, 1976). When no saccade is executed, a degree of visual responsiveness is evident in some members of this class. Other members of the group can discharge even in the occasional trial when the monkey fails to make a saccade. Further, these units discharge earlier and more vigorously when the monkey anticipates making a saccade. This combined contingency of the presaccadic discharge on the presence of a visual target and the monkey's motivational state is a key element found in higher centres (discussed below).

Many other cells with saccadic discharge have associated visual responses (Schiller and Koerner, 1971; Wurtz and Goldberg, 1972a; Mays and Sparks, 1980). When a visual stimulus is presented, the units discharge even if no saccade is ultimately required. When the stimulus is the target for a saccade, this group of neurones shows a burst synchronized with the visual stimulus and another with the saccade when the saccade is delayed relative to the presentation of the target. When the monkey is required to generate a saccade into the cell's receptive/movement field without a visual target, then the first sensory burst is absent, but the saccade burst is expressed. This pattern of two bursts, one visual and another presaccadic is also observed in parietal and frontal cortex (see below).

It may seem self-evident that the visual responses in the colliculus participate in the selection of the target for a saccade. This has been tested experimentally by presenting monkeys with a choice of targets. In such an experiment, subjects are presented with two stimuli that are distinguished by colour (red and green), one of which is defined as the target. If the stimuli lie close to one another and the subject makes short latency saccades, the saccades tend to be directed to a point in between the two stimuli; only if the saccade is delayed is it correctly directed to the target (Ottes *et al.*, 1985). Single units were recorded in the superior colliculus of monkeys performing this task (Ottes *et al.*, 1987). Since, as mentioned, visually responsive collicular neurones are not colour selective, it is of interest to determine how they behave when the selection of the saccade target requires a colour discrimination. The results reveal that the visual component of the response of visuomovement cells has the same latency and magnitude regardless of whether the stimulus falling in the receptive/movement field is the target or the nontarget. Furthermore, there is no substantial difference in the saccade-related component of the response associated with saccades to a single target versus saccades to a target distinguished by colour. These results indicate that, at least in this task, superior colliculus cells do not reflect this discrimination and that higher cortical centres provide the signal that specifies the target of the saccade.

Connectivity of Deeper Layers

The input to and output of the deep subdivision of the superior colliculus is so complicated that it is difficult to fully rationalize the origin and impact of the different signals described. The deep layers of the superior colliculus in primates receive input from a variety of cortical areas including primary visual cortex, extrastriate visual areas, posterior parietal cortex, temporal cortex, the supplementary and frontal eye fields and prefrontal cortex (Kuypers and Lawrence, 1967; Garey *et al.*, 1968; Astruc, 1971; Kunzle and Akert, 1974; Kunzle *et al.*, 1976; Goldman and Nauta, 1976; Benevento and Davis, 1977; Jones and Wise, 1977; Hartmann-von Monakow *et al.*, 1979; Leichnetz *et al.*, 1981; Fries, 1984; Weber and Yin, 1984; Komatsu and Suzuki, 1985; Lynch *et al.*, 1985; Huerta *et al.*, 1986; Huerta and Kaas, 1988; Stanton *et al.*, 1988b). Subcortical afferents to the deep division of the superior colliculus have been studied more in cat than in primates. Briefly, the deep layers of the superior colliculus receive afferents from the diencephalon, notably the reticular nucleus and the pregeniculate nucleus; the pretectum; various midbrain structures including in particular the substantia nigra pars reticularis; various pontine and medullar nuclei, including the NRTP; certain cerebellar nuclei and finally the cervical spinal cord (reviewed by Huerta and Harting, 1984a,b).

The efferents of the deep division of the superior colliculus are just as widespread. Ascending projections travel to the ventroanterior, ventrolateral, mediodorsal, central lateral, pulvinar, lateral dorsal and reticular thalamic nuclei (Harting *et al.*, 1973; Benevento and Fallon, 1975; Harting *et al.*, 1980). The descending projections end in the

ipsilateral substantia nigra pars reticulata, mesencephalic reticular formation, pons, medulla and spinal cord and in the contralateral pons, including PPRF, medulla and spinal cord (Harting *et al.*, 1973; Frankfurter *et al.*, 1976; Harting, 1977).

Effects of Lesions

Given all this visual and oculomotor activity in the superior colliculus, it may not be an overstatement to say that the effects on saccade generation of ablating the superior colliculus are hardly devastating (Ferrier and Turner, 1901; Bender *et al.*, 1957; Pasik *et al.*, 1966; but see Denny-Brown, 1962). Monkeys can still execute reasonably accurate saccades, but they do so less frequently and with an increase in latency and a transient reduction of velocity; they also exhibit ipsilateral gaze deviation (Rosvold *et al.*, 1958; Pasik *et al.*, 1966; Wurtz and Goldberg, 1972b; Mohler and Wurtz, 1977; Kurtz and Butter, 1980; Albano and Wurtz, 1982; Albano *et al.*, 1982). Similar though perhaps more severe deficits are observed following lesions involving the pretectum and surrounding thalamic areas (Albano and Wurtz, 1982a; Keating *et al.*, 1986). Collicular ablation also causes contralateral neglect syndromes (Denny-Brown, 1962; Sprague and Meikle, 1965; Anderson and Symmes, 1969; Keating, 1974; Milner *et al.*, 1978; Kurtz and Butter, 1980), and monkeys with ablations of the superior colliculus do suffer impairments in performing visual search (Bender and Butter, 1987). This indicates that even as the colliculus plays a role in generating saccadic shifts of gaze, it also is important for shifts of attention.

The fact that saccades can still be generated following ablation of the superior colliculus indicates that at least one other area of the brain is responsible for generating visually guided saccades and that the superior colliculus, while certainly important, is not necessary for the execution of saccades. More recent work, though, has revealed one type of saccade that the superior colliculus is uniquely responsible for (Schiller *et al.*, 1987); ablation of the colliculus permanently prevents express saccades, which are short latency (70 ms) saccades to predictable visual targets (Fischer and Boch, 1983; Boch *et al.*, 1984; Boch and Fischer, 1986; Fischer and Ramsperger, 1986; Mayfrank *et al.*, 1986).

As will be reviewed below, electrical stimulation of different areas of the cerebral cortex also elicits saccadic eye movements. It is possible to dissect the functional relationships between the superior colliculus and the cortical visuomotor areas by ablating the colliculus and ascertaining whether cortical stimulation still evokes eye movements or by determining the effects of combined ablation of the superior colliculus and different cortical areas. This research strategy has been used for a number of years, and as techniques improve, the results are refined. The results of the original studies (reviewed by Pasik and Pasik, 1964) indicated that ablation of the superior colliculus does not eliminate saccades elicited by surface electrical stimulation of the occipital or frontal cortex. However, more recently it has been shown that collicular ablation does indeed cause a loss of saccades evoked by the more cultivated intracortical microstimulation of occipital (striate and prestriate) and parietal cortex but not of the frontal eye fields (Schiller, 1977; Keating *et al.*, 1983). Still more recent versions of the collicular ablation experiment demonstrate that when the lesion is restricted to the superior colliculus and spares corticofugal fibres passing to the brainstem, stimulation of occipital and parietal cortex can still elicit saccades (Keating and Gooley, 1988). This result indicates that occipital and parietal cortex communicate with the saccade generator either directly or via frontal cortex as well as via the superior colliculus. These results are taken as evidence that two parallel pathways mediate visually guided eye movements, one depending on the frontal eye fields and the other on the superior colliculus (reviewed by Schiller, 1985).

The frontal eye field pathway depends on input via the striate cortex (Mohler and Wurtz, 1977). What appeared to be definitive evidence that the superior colliculus and frontal eye fields are the only areas necessary to generate saccadic eye movements was derived from the results of ablating both the frontal eye fields and the superior colliculus together (Schiller *et al.*, 1979). Combined ablation of these areas eliminated essentially all visually guided eye movements. This view may require refinement, however, because evidence is accumulating that shows that at least two other regions of the cerebral cortex (reviewed below), the lateral interparietal area and the supplementary eye fields, are involved in generating saccades and have independent access to the brainstem saccade generator.

Effects of Reversible Inactivation

More sophisticated means of inactivation provide even more information about how the superior colliculus functions to generate saccades. Localized injections of the GABA agonist, muscimol, prevent saccades into the movement field of the injection site. By contrast, injections of the GABA antagonist, bicuculline, result in repeated saccades into the movement field of the injection site (Hikosaka and Wurtz, 1985a; Schiller *et al.*, 1987). These results show that the presaccadic neurones of the superior colliculus are under the influence of GABAergic inhibition; the source of which will be discussed below.

The results of these local inactivation experiments have provided important information about a fundamental issue concerning the role of the superior colliculus in saccade generation. As reviewed above, the presaccadic units in the superior colliculus do not fire for saccades of just one angle and amplitude; rather, they have tuned movement fields. Accordingly, since any single unit in the colliculus discharges for saccades of varying directions and amplitudes, how can accurate saccades be specified? It has been suggested that the activity of many colliculus cells is pooled or averaged in some fashion (Sparks *et al.*, 1976; van Gisbergen *et al.*, 1987). In general, the direction and amplitude of a saccade is suggested to be given by a vector sum or average of the activity of the population of superior colliculus cells that include the saccade in their movement fields.

Direct evidence supporting the vector summation idea comes from collicular inactivation experiments. Localized pharmacological inactivation of a small region of the deep layers of the superior colliculus with lidocaine affects the trajectory and velocity of saccades directed around the movement field represented by the inactivated area (Hikosaka and Wurtz, 1986). Specifically, saccades aimed at a target located beyond the movement field represented by the inactivated area are too long, and by contrast, saccades aimed at a target located before the movement field are too short (Lee *et al.*, 1988).

Effects of Electrical Stimulation

Further evidence for a vector summation model is provided by electrical stimulation of the superior colliculus. To review, stimulation of the deep layers of the superior colliculus elicits saccades (Robinson, 1972; Schiller and Stryker, 1972). The currents required are as low as 20 µA, and the latency of the evoked saccades averages 40–50 ms with near threshold stimulation and can be as low as 20 - 25 ms with suprathreshold currents. The low intensity and short latency are consistent with the direct impact the superior colliculus has on the saccade generator. Indeed, the shortest latency observed for stimulation evoked saccades corresponds to the onset time of the collicular short-lead presaccadic burst neurones. The direction and amplitude of the saccade evoked by collicular stimulation varies with the site of stimulation and corresponds to the movement fields of the cells recorded through the stimulating electrode. Unlike the saccades evoked by stimulation of the cerebellum, the vector of the saccade elicited by collicular stimulation does not vary with the initial position of the eye in the orbit. Whereas prolonged stimulation of the motor nuclei results in larger eye movements, prolonged stimulation of the superior colliculus results in a series of saccades all of the same amplitude and direction (Schiller and Stryker, 1972).

To return to the question at hand, the evidence for vector summation of collicular output is provided by simultaneous stimulation of two sites in the superior colliculus. Such combined stimulation results in a saccade that is the vector sum of the saccades that are elicited by stimulation of the two sites independently (Robinson, 1972). If the intensity of stimulation at the two sites is varied, then the direction and amplitude of the resultant saccade changes according to the two vectors weighted by the intensity of the stimulation.

Sensorimotor Transformation

Given, then, that the combined activity of a subpopulation of neurones in the deep layers of the superior colliculus provides input to the saccade generator for a specific saccade, then the next question is what activates the collicular cells? How is the visually evoked activity registering the target transmitted to the movement cells. These questions have received considerable experimental attention. Since the retinotopic map of the upper layers matches the movement fields of the deeper layers, it was thought that the superior colliculus encodes saccades in a retinocentric coordinate system (Pitts and McCulloch, 1947; Schiller and Koerner, 1971). In other words, when a visual target appears, cells in the upper layers respond. This activation, it was imagined, is transmitted to the deep layers that trigger a saccade of the appropriate direction and amplitude to foveate the stimulus.

As attractive as this conception is, a number of lines of evidence indicate that it is untenable. Physiological experiments have shown that neuronal activity in the upper and lower layers of the superior colliculus can be dissociated; that is, visual neurones in the upper layers can discharge without triggering a saccade, and conversely, a saccade can be generated by activity in the deeper layers without there having been activity in the upper layers (Mays and Sparks, 1980). If the sensory and motor representations are dissociated, then for what purpose?

Psychophysical experiments have also shown that the position of the eye in the orbit is taken into account in generating a saccade. When a visual stimulus is briefly presented while a saccade is in flight, it can be foveated by a subsequent saccade (Hallet and Lightstone, 1976). Thus, an eye position signal must be combined with the retinal error signal to direct saccades accurately; or in other words, the location of the target on the retina is not sufficient, instead the change in eye position must be signalled. Physiological evidence for such a combination has been observed. If a monkey's eyes are diverted by stimulation of the superior colliculus (Sparks and Mays, 1983) or, as reviewed above, the PPRF (Sparks *et al.*, 1987) prior to the initiation of a visually guided saccade, the monkey is able to compensate for the perturbation and make a saccade directly to the location of the visual target. Furthermore, neurones in the superior colliculus discharge for the compensatory saccade that was not directly elicited by a visual stimulus (Sparks and Porter, 1983). These results may be interpreted to indicate that the output of the deep division of the colliculus to the saccade generator is a motor error signal.

Other evidence that supports this view is derived from the fact that certain units in the deeper subdivision of the superior colliculus respond to somatosensory and auditory stimuli (see Stein and Meredith, Chapter 4). Typically the maps of these different sensory systems appear to be in register in the colliculus. The integration of auditory and visual input in the deeper layers of the superior colliculus in generating saccades has been investigated (Jay and Sparks, 1987a,b). First, visuomovement cells do not exhibit their first burst when an auditory target for a saccade is presented; however, the second burst associated with the saccade does typically occur. At the same time, there are other visuomovement neurones which do not show either the sensory- or the saccade-related burst for saccades to auditory targets; they are modality specific. What is more (and this is the point for the question under consideration) the receptive fields of auditory responsive cells shift with eye position; the auditory map in the deep layers of the superior colliculus is not in strict register with the retinal visual map but changes according to the direction of gaze. This combination of results indicates that visual and auditory signals, which are encoded in different coordinate systems, are combined in some but not all collicular neurones to be expressed in a common motor output. Moreover, the sensory maps appear to be in motor rather than in sensory coordinates.

Finally, it has been demonstrated that the discharge of some burst cells in the intermediate layers of the superior colliculus peaks immediately before the saccade and declines while the saccade is in flight (Waitzman *et al.*, 1988). These results indicate that at least some cells in the superior colliculus signal not only the initial motor error but the dynamic motor error signal.

Summary

It appears that at least some of the neurones in the superior colliculus encode the motor error of a saccade. The visual input from the retina and visual cortex would seem to be the source of the retinal error signal. As reviewed above, the eye position signal might be derived from the cerebellum. The superior colliculus can deliver the motor error signal by its direct projection onto long-lead burst neurones in the supranuclear saccade generator (Buttner *et al.*, 1977; Raybourne and Keller, 1977). Besides providing a signal that specifies where to direct a saccade, it has been hypothetized that the superior colliculus also provides a trigger signal to the brainstem saccade generator. The superior colliculus projects to the omnipause neurones in the brainstem, and, as described, omnipause neurones are inhibited by stimulation of the superior colliculus. Conceivably, it is via this pathway that the superior colliculus can initiate a saccade. However, if the visual activity in the upper layers does not necessarily trigger the activity of the motor cells in the deeper layers, what structure does initiate their activity?

Basal Ganglia

Overview

In the foregoing sections we have considered how the brainstem performs the task of generating saccadic eye movements. The next question to consider is what structures are responsible for actually initiating the eye movement once a target is decided upon, for even though we may identify something to look at, we do not always look. This issue takes the review into the basal ganglia, a collection of structures long known to be important for movement initiation (reviewed by Graybiel and Ragsdale, 1979; DeLong and Georgopoulos, 1981; Alexander *et al.*, 1986).

It is presently thought that a number of functionally distinct circuits pass through the basal ganglia. The general scheme appears to be organized along these lines: A number of cortical areas project to partially overlapping regions of the caudate or putamen. The striatum projects to the globus pallidus and/or the substantia nigra that in turn project to specific thalamic nuclei that provide the major afferents to a particular cortical area. The manifestation of this scheme of interest with respect to eye movements can be described as follows: The body and head of the caudate receives afferents from a large expanse of cortex, mainly frontal and specifically for our interests, the frontal and supplementary eye fields; this region of the caudate in turn projects to the caudal-dorsomedial section of the internal segment of the globus pallidus and the lateral portion of the substantia nigra pars reticulata. These pallidal/nigral regions project to the magnocellular portion of the ventral anterior nucleus and the paralaminar, parvicellular and densocelluluar subdivisions of the mediodorsal nucleus of the thalamus (Ilinsky *et al.*, 1985). Completing the cycle, these thalamic nuclei provide input to the frontal and supplementary eye fields. The substantia nigra pars reticulata also sends afferents to the superior colliculus (Jayaraman *et al.*, 1977; Beckstead *et al.*, 1981), and this pathway will be seen to play a pivotal role in saccade initiation. The substantia nigra pars reticulata itself receives input from the PPRF (Graybiel, 1977).

Diseases of the basal ganglia, besides other well known clinical manifestations, result in eye movement deficits. For example, Huntington's disease, which causes degeneration in the caudate nucleus, results in impaired initiation of saccades and reduced saccade velocity (Starr, 1967; Avanzini *et al.*, 1979; Leigh *et al.*, 1983; Lasker *et al.*, 1987, 1988). Also, Parkinson's disease, which causes degeneration of the nigrostriatal projection, results in increased saccade latency, reduced saccade velocity and hypometric saccades (Slatt *et al.*, 1966; Chaco, 1971; Corin *et al.*, 1972; DeJong and Melvill Jones, 1971; Melvill Jones and DeJong, 1971; Shibasaki *et al.*, 1979; Shimizu *et al.*, 1981; Teravainen and Calne, 1980; White *et al.*, 1983). Similar deficits are suffered by MPTP-treated monkeys (Brooks *et al.*, 1986). Furthermore, administration of L-DOPA to Parkinson's patients or MPTP patients improves saccadic performance (Highstein *et al.*, 1969; Hotson *et al.*, 1986).

Substantia Nigra

Physiological Properties

A variety of neuronal responses have been recorded in the substantia nigra pars reticulata (Fig. 15.1(b)) of monkeys performing a variety of visually guided eye movement tasks. The patterns of modulation observed in the substantia nigra are illustrated in Fig. 15.5. Most of the substantia nigra neurones have a high resting discharge rate and display suppressed activity associated with visual stimuli or saccades (Hikosaka and Wurtz, 1983a). One class of neurone is identified by a visual response. The visual response latency is long (70–167 ms with an average of 120 ms) which indicates that the visual signals reach the substantia nigra by a quite indirect route. Consistent with most visual centres, the receptive fields of substantia nigra visual cells are located in the contralateral hemifield but can extend into the ipsilateral, and neighbouring neurones tend to have overlapping receptive fields. The responses of the visual neurones do not vary with gaze angle. The visual response of some substantia nigra neurones is enhanced if and only if the stimulus falls in a cell's receptive field and is the target for a saccade. Finally, some neurones respond specifically to auditory stimuli with a mean latency of 65 ms when they are saccade targets.

Another population of units is suppressed in relation to saccades to visual targets. The suppression of activity in these units begins about 20 ms before the saccade and continues until approximately 200 ms after. These units are not modulated in association with spontaneous saccades but require a visual target. Still other neurones display both a visual response and a saccade related response.

The responses of other units in the substantia nigra varies according to whether or not the monkey is fixating another stimulus (Fig. 15.5 (b), (c)) (Hikosaka and Wurtz, 1983b). One population of units is suppressed at the appearance of a light stimulus but only if the monkey is not already fixating another stimulus. The average latency of this response is 126 ms, and the receptive fields revealed under these conditions are very large, centred on the fovea and extending into both the contra- and ipsilateral hemifields. The modulation of these units tends to habituate with shorter intertrial intervals. A second pattern of modulation is a response to the offset of a spot of a light that the monkey is fixating. This occurs with an average latency of 131 ms. If another stimulus is present when the fixation spot disappears, this fixation contingent off response is attenuated. This pattern of modulation is observed in units that display other sensory or oculomotor responses. Since these two patterns of modulation are evident only when a light that is being fixated disappears or when a light appears that will be fixated, it seems reasonable to suppose that they provide a signal related to changing fixation, i.e. initiating saccades.

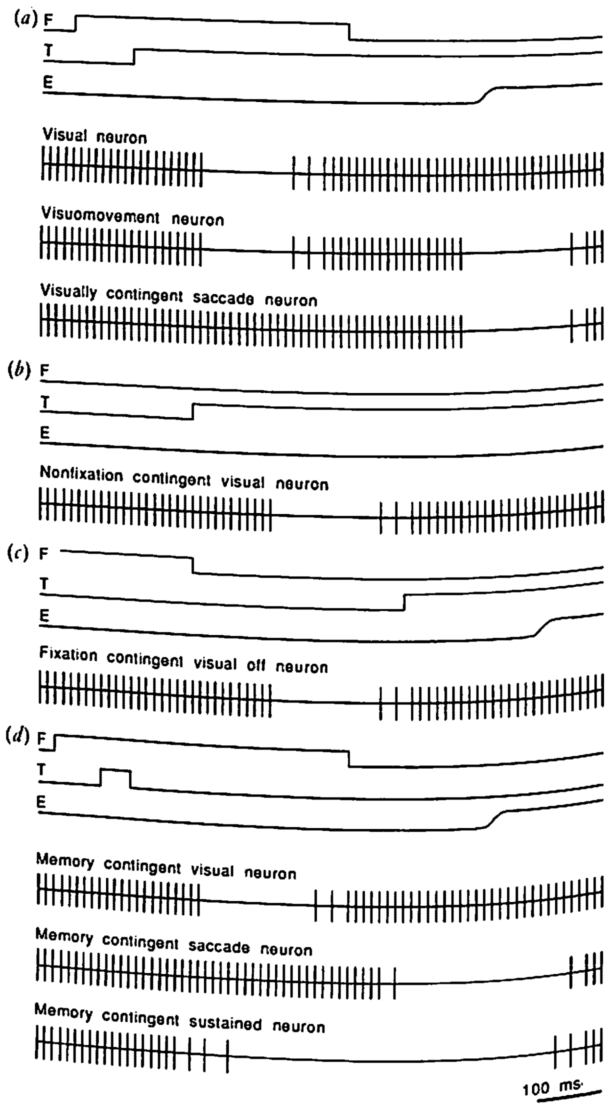

Fig. 15.5 *Neuronal activity in substantia nigra associated with saccades. Several tasks are represented. (a) In the first task a spot appears (F) that the monkey fixates. After some time a peripheral target (T) appears. After another delay elapses, the fixation spot disappears, cueing the saccade. (b) Initially no stimulus is on, and the monkey is now fixating nowhere in particular. A stimulus (T) appears, but the monkey does not fixate it. (c) The monkey's gaze is on the fixation spot (F) which disappears followed later by the presentation of a target (T). (d) While the monkey is fixating one spot (F), the saccade target (T) is presented briefly. The monkey is allowed to saccade to the position of the target only after the fixation spot disappears, so he has to remember the location of the target.*

When the monkey is required to make a saccade to a briefly flashed target that has disappeared, three types of memory-contingent responses are observed (Fig. 15.6(d)) (Hikosaka and Wurtz, 1983c). The first pattern of modulation is a response to a visual stimulus that will have to be remembered as a target for a saccade. In fact, if a saccade is made directly to the stimulus while it is still visible, these units do not respond as well. The latency (100–120 ms) and receptive field properties of these cells are similar to the more typical visual neurones. The second type of response is a decrease in discharge rate associated with saccades to remembered targets; these units are not as well modulated for saccades that are made to a visible stimulus, neither are they modulated in association with spontaneous saccades. The onset of modulation of these memory-contingent saccade neurones ranges from 70–280 ms before the saccade, and the movement fields of these units emphasize the contralateral hemifield. The third group of these units exhibits a sustained suppression following the appearance of a briefly flashed stimulus until the execution of the saccade to the remembered target. Other neurones display separate periods of modulation in association with the appearance of the target to be remembered and the saccade to the remembered location. These latter cells appear to be functionally similar to the quasi-visual cells described in the superior colliculus.

This body of work indicates that the substantia nigra pars reticulata contains neuronal elements that would appear to be required to trigger saccades in a variety of circumstances. The visual and visually contingent saccade cells can trigger saccades made to stimuli that are still in view, and the memory-contingent cells can trigger saccades made to the location of visual stimuli that have disappeared. A further indication of its participation in visuomotor behaviour is the neglect syndrome that follows lesions of the substantia nigra (Feeney and Wier, 1979).

Relation to Superior Colliculus

Two lines of evidence indicate that the substantia nigra exerts a significant influence on the oculomotor system via its projection to the superior colliculus. Although it should not be overlooked that the substantia nigra does project to the supranuclear reticular gaze regions in the brainstem (Schneider *et al.*, 1985). Neurones in the substantia nigra that display visuomotor activity can be antidromically activated by microstimulation of the superior colliculus (Hikosaka and Wurtz, 1983d). The stimulation is most effective if the receptive fields of the substantia nigra and superior colliculus cells are overlapping. This suggests that the projection of the substantia nigra to the superior colliculus is topographically organized which is consistent with the presence of spatially tuned cells in the substantia nigra. The lowest threshold for microstimulation is found in the deeper layers of the superior colliculus where saccade related neurones are recorded. Furthermore, a functional correlation is observed between the neuronal activity in regions of superior colliculus and substantia nigra that are interconnected. Specifically, when a substantia nigra cell is suppressed in relation to a given saccade, the superior colliculus cell receiving afferents from it discharges at the same time.

The projection from the substantia nigra to the superior colliculus appears to be inhibitory, and the inhibition appears to be mediated by GABA. As reviewed above, injection of the GABA agonist, muscimol into the superior colliculus suppresses the execution of saccades into the movement field represented by the region receiving the injection (Hikosaka and Wurtz, 1985a). In contrast, injection of bicuculline, a GABA antagonist, into the superior colliculus results in facilitated saccade initiation. The effect of injecting muscimol into the substantia nigra corresponds to the effect of injecting bicuculline into the superior colliculus (Hikosaka and Wurtz, 1985b). Specifically, following the increase of inhibition of the substantia nigra neurones, the monkey initiated unnecessary, undesirable saccades that interfered with performance of the visually guided or memory guided saccades. This result indicates that the substantia nigra pars reticulata imposes a sustained GABAergic inhibition on the superior colliculus.

Thus, it appears that the substantia nigra can initiate saccades by releasing their inhibition on presaccadic collicular neurones. It should be mentioned, however, that the function of the nigrothalamic projections in relation to saccadic eye movements is not understood. Given the evidence that the substantia nigra triggers the superior colliculus, the question of saccade initiation is hence pushed back to the structures that provide input to the substantia nigra.

Striatum

Physiological Properties

Besides its anatomical relations, two lines of evidence implicate the caudate nucleus in visuomotor behaviour. First, lesions of the striatum result in a contralateral neglect syndrome similar to that observed following ablation of parietal or frontal cortex (Bowen, 1969). Second, single unit recordings reveal a number of neurones discharging in relation to visually-guided or memory-guided saccades.

Single unit recordings in the head and body of the caudate nucleus in behaving monkeys have revealed a variety of visual and saccade related neurones (Hikosaka *et al.*, 1989a,b,c). The visually responsive cells have large receptive fields that are located primarily in the contralateral hemifield. The response of some of the cells is uninfluenced by whether the stimulus is the target for a saccade; in contrast, other visual neurones show the spatially specific saccade-related enhancement. Still other visual neurones are selectively activated by briefly flashed stimuli that are used as the target for a later, memory-guided saccade. A final subgroup of the visually responsive cells discharge following the appearance of a stimulus that is presented when the monkey is not already fixating another spot. The visual response latency ranges from 75–300 ms with an average of approximately 150 ms. Interestingly, the latency of the memory selective visual response is around 50 ms longer than that of the other groups. These visual response latencies are longer than those observed in other visuomotor brain regions which indicates that they reflect substantial processing. Other cells in the caudate nucleus respond to auditory stimuli, but none of these are bimodal.

In tasks in which the monkey is required to make a delayed saccade to a visible or remembered target, other cells are specifically activated. One population of caudate neurone is active after the monkey fixates one spot and waits for the presentation of the stimulus that will be the target for a saccade. These cells apparently reflect the internal expectation of the trained monkey. Indeed, some units do not even require that the stimulus appears, only that the monkey ultimately makes the appropriate saccade.

Other neurones exhibit a maintained elevation from the appearance of the target until the cue to move. Some of these units discharging in the delay interval between presentation of the target and the cue to move are more active when the target is briefly flashed, requiring the monkey to remember its location. These delay neurones are more active in association with contralaterally directed saccades.

When the monkey makes a saccade to the remembered location and waits for the appearance of a visual stimulus at that location another population of neurones in the caudate is activated. These cells begin to discharge after the goal-directed saccade and continue until the target reappears. Some of these units display a burst following the reappearance of the target.

A distinct population of units in the caudate are active specifically prior to goal-directed saccades; these cells discharge less if at all in relation to spontaneous, self-generated saccades. One variety discharges preferentially in relation to visually guided saccades while another is active mainly for memory-guided saccades, and yet another is active for saccades made under both circumstances. The presaccadic burst of these units begins up to 300 ms (on average approximately 100 ms) before the saccade. The movement fields of these presaccadic caudate neurones are fairly large and emphasize the contralateral hemifield. Another distinct subpopulation of the presaccadic neurones shows a much longer prelude of activation in trials that demanded a long (over 2000 ms) response delay, beginning 300–400 ms after target presentation and peaking after the cue to saccade. Yet another population of units encountered in the caudate nucleus discharge during fixation, and the activity of some of these fixation cells varies with the position of the eye in the orbit.

There was no apparent segregation among the different types of cells recorded in caudate. This issue is especially interesting given the neurochemical diversity and segregation observed in the striatum (Graybiel and Ragsdale, 1978; Goldman-Rakic, 1982; reviewed by Graybiel, 1982; Graybiel and Ragsdale, 1983) and will likely be an active area of future research.

Connectivity

This work indicates that the caudate nucleus can provide signals to the substantia nigra pars reticulata that result in a saccade. As alluded to, the substantia nigra pars reticulata is a major recipient of afferents from the caudate (Grofova, 1975; Feger and Crossman, 1984; Parent *et al.*, 1984), and the projection is inhibitory, GABAergic (Precht and Yoshida, 1971; Yoshida and Precht, 1971; Feger and Ohye, 1975; Fonnum *et al.*, 1978; DiChiara *et al.*, 1980; Fisher *et al.*, 1986; Pan *et al.*, 1983). A reasonable scenario for saccade generation to this point, then, goes like this: in preparing to execute a saccade, caudate units become active. Their activation in turn inhibits neurones in the substantia nigra that project to the superior colliculus. This release of the nigral inhibition on the superior colliculus finally allows a saccade to be generated.

We are now confronted with the problem of the origin of the visuomotor activity in the caudate nucleus. As mentioned above, the head of the caudate nucleus receives afferents from cortical areas that have been implicated in gaze control (reviewed below) including the frontal eye field, the supplementary eye field and inferior parietal lobule (Kunzle and Akert, 1977; Yeterian and Van Hoesen, 1978; Jurgens, 1984; Selemon and Goldman-Rakic, 1985; Arikuni and Kubota, 1986; Stanton *et al.*, 1988a). Parenthetically, these cortical areas also project to the putamen, so it will be instructive to learn whether visuomotor neuronal activity is present there too. The caudate also receives substantial input from the intralaminar thalamic nuclei (Parent *et al.*, 1983a) that also contain visuomotor neurones (see below). The responses of these caudate nucleus neurones resemble in most respects those of the cortical and thalamic neurones that probably provide their input. The long visual response latency observed in the caudate is consistent with it being derived from the long latency visual responses of frontal cortical neurones. Also the saccade-related responses are similar to those observed in these cortical areas. Before reviewing the work in the cortex, however, it is necessary to consider the thalamic nuclei that provide afferents to the visuomotor cortex.

Thalamus

Moving the eyes is only half of the problem, where to look, what is interesting and worthwhile to direct our gaze to is the other half. Prior to initiating a saccade, a target must be selected. Considering this part of the saccade generation process moves the discussion toward the input end of the system. A number of thalamic nuclei have been shown to play key roles in gaze control; this section will progress from the more sensory to the more oculomotor.

Lateral Geniculate and Pregeniculate Nuclei

As reviewed in chapters 9 (Henry) and 3 (Casagrande and Norton), the dorsal lateral geniculate nucleus (dLGN) is the relay station for retinal input to striate cortex. Investigations in the cat dLGN showed modulation related to eye movements during sleep or vestibular nystagmus (Bizzi, 1966; Kawamura and Marchiafava, 1966; Jeannerod and Putkomen, 1971; see also Lal and Friedlander, 1989), and an early study in the monkey showed that a monophasic negative field potential can be recorded throughout the dLGN associated with saccades in the dark (Feldman and Cohen, 1968). However, single unit recordings in the dLGN of alert monkeys reveal essentially no saccade-related modulation (Buttner and Fuchs, 1973; Duffy and Burchfiel, 1975).

The pregeniculate nucleus is a relatively thin layer of cells positioned above to the dLGN. In subprimate species this structure is called the ventral lateral geniculate nucleus. By whatever name, it is considered to be associated with the thalamic reticular nucleus, a veil of cells surrounding the thalamus. Units recorded in the pregeniculate nucleus are modulated in association with saccades (Buttner and Fuchs, 1973; Magnin and Fuchs, 1977); although, the modulation is unimpressive compared to what we have reviewed. The neurones either burst or are suppressed 80–500 ms after a saccade. Usually, it makes no difference whether the saccade is visually guided or occurs in the dark. In addition, some of the pregeniculate cells are visually responsive with a latency of around 25 ms.

Saccade-related modulation is consistent with the fact that the pregeniculate nucleus receives afferents from the superior colliculus (Benevento and Fallon, 1975; Harting *et al.*, 1978; Weber *et al.*, 1983), certain pontine nuclei (Graybiel, 1974; Weber *et al.*, 1983) as well as from the pulvinar (Trojanowski and Jacobson, 1976). The pregeniculate nucleus also receives retinal and visual cortical afferents (Spatz *et al.*, 1970; Spatz and Tigges, 1973; Ogren and Hendrickson, 1976; Maunsell and Van Essen, 1983; Asanuma *et al.*, 1985). The pregeniculate nucleus neurones send axons to only subcortical sites, including the ipsilateral pretectum, the deep subdivision of the superior colliculus, the pontine nuclei and the contralateral pregeniculate nucleus (Hendrickson, 1973; Graybiel, 1974; Edwards *et al.*, 1974; Swanson *et al.*, 1974).

Despite these somewhat inauspicious results, this 'eyebrow to the lateral geniculate nucleus' (Magnin and Fuchs, 1977) may be rescued from obscurity by a hypothesis recently put forward to explain the neuronal mechanisms underlying directed visual attention (Crick, 1984). Succinctly, it is proposed that the reticular thalamic nucleus in general and the pregeniculate nucleus in particular serve to coordinate the activity of specific ensembles of thalamic relay neurones providing inputs to particular cortical areas. This coordination is conjectured to underlie the roving spotlight of attention. One thalamic nucleus may be especially subject to such coordination – the pulvinar.

Pulvinar

The pulvinar consists of a collection of four nuclei—medial, lateral, inferior and anterior—that are distinguished on the basis of their connectivity and functional properties (Fig. 15.6). The increase in size of the pulvinar in primates parallels that of the extrastriate visual cortex. The role of the pulvinar in visuomotor behaviour has been reviewed (Cooper *et al.*, 1974; Chalupa, 1977 and chapter 6).

Connectivity

The inferior pulvinar receives input from retinal ganglion cells (Campos-Ortega *et al.*, 1970; Mizuno *et al.*, 1982; Itaya and Van Hoesen, 1983; Nakagawa and Tanaka, 1984) as well as from the ipsilateral superior colliculus and pretectum (Benevento and Fallon, 1975, Trojanowski and Jacobson, 1975b; Benevento and Rezak, 1976; Partlow *et al.*, 1977; Lin and Kaas, 1979; Harting *et al.*, 1980; Benevento and Standage, 1983; Marrocco *et al.*, 1981, Huerta and Harting, 1983). The inferior division of the pulvinar projects mainly to striate but also to extrastriate visual cortex, and these projections are reciprocated (Ferrier and Turner, 1897; Le Gros Clark and Northfield, 1937; Chow, 1950; Siqueira, 1971; Campos-Ortega and

Hayhow, 1972; Hollander, 1974; Benevento and Rezak, 1975, 1976; Ogren and Hendrickson, 1976, 1977, 1979b; Trojanowski and Jacobson, 1976, 1977; Benevento and Davis, 1977; Graham *et al.*, 1979; Lin and Kaas, 1979, 1980; Rezak and Benevento, 1979; Lund *et al.*, 1981; Graham, 1982; Maunsell and Van Essen, 1983; Ungerleider *et al.*, 1983). It is not presently clear whether the collicular and cortical afferents are strictly segregated. The lateral nucleus of the pulvinar receives input from the superior colliculus and pretectum (Benevento and Fallon, 1975; Trojanowski and Jacobson, 1975; Harting *et al.*, 1980; Benevento and Standage, 1983) and is interconnected primarily with extrastriate visual areas including the inferior parietal lobule (Chow, 1950; Campos-Ortega and Hayhow, 1972; Hollander, 1974; Benevento and Rezak, 1975, 1976; Trojanowski and Jacobson, 1975, 1976; Curcio and Harting, 1978; Ogren and Hendrickson, 1976, 1977, 1979; Benevento and Davis, 1977; Graham *et al.*, Graham, 1982; Maunsell and Van Essen, 1983; Ungerleider *et al.*, 1983; Weber and Yin, 1984; Asanuma *et al.*, 1985).

While the inferior and lateral pulvinar nuclei are intimate with the visual pathways, the medial nucleus of the pulvinar forms different associations. It receives light input from the superior colliculus (mainly the deep layers) and pretectum (Benevento and Fallon, 1975; Trojanowski and Jacobson, 1975; Partlow *et al.*, 1977; Harting *et al.*, 1980; Benevento and Standage, 1983). The medial nucleus is interconnected with the superior temporal gyrus, the inferior parietal lobule, cingulate cortex and insular cortex (Siqueira 1965, 1971; Campos-Ortega and Hayhow, 1972; Burton and Jones, 1976; Baleydier and Mauguiere, 1977, 1980, 1985, 1987; Divac *et al.*, 1977; Mesulam *et al.*, 1977; Stanton *et al.*, 1977; Kasdon and Jacobson, 1978; Pearson *et al.*, 1978; De Vito, 1978; Mufson and Mesulam, 1984; Weber and Yin, 1984; Asanuma *et al.*, 1985; Yeterian and Pandya, 1985). The medial pulvinar has also been reported to be interconnected with the frontal cortex including the prearcuate gyrus in the vicinity of the frontal eye fields as well as the supplementary eye fields (Bos and Benevento, 1975; Trojanowski and Jacobson, 1974, 1976; Kunzle *et al.*, 1976; Kievit and Kuypers, 1977; Barbas and Mesulam, 1981; Leichnetz, 1982; Huerta *et al.*, 1986; Stanton *et al.*, 1988a). It has been argued, to the contrary, that the purported frontal cortical interconnection with the medial pulvinar is more rightly assigned to a diffuse part of the central lateral nucleus that is inserted into the medial pulvinar (Jones, 1985). Whether this makes any functional difference has yet to be worked out.

Effects of Lesions

The effects of lesions of the pulvinar have yielded conflicting results. In some cases ablation of the pulvinar results in no behavioural deficits (Chow, 1954; Cooper *et al.*, 1974; Riklan *et al.*, 1974; Ungerleider *et al.*, 1977; Leiby *et al.*, 1982; Bender and Butter, 1987). At the same time, lesions of the pulvinar have also been shown to result in visual neglect, a reduced number of eye movements with prolonged fixations, impairment in visual search and an impairment in learning visual discriminations (Chalupa *et al.*, 1976; Ungerleider and Christensen, 1977, 1979; Zihl and von Cramon, 1979; Ogren *et al.*, 1984). But these effects may be due to inadvertently damaging fibres passing to the superior colliculus (Nagel-Leiby *et al.*, 1984).

Physiological Properties

The patterns of neuronal modulation recorded in the pulvinar are illustrated in Fig. 15.6. Consistent with their connections, most cells in the inferior and lateral subdivisions are visually responsive; visual responsiveness is less prominent in the medial subdivision and is essentially absent in the anterior subdivision (Gattass *et al.*, 1978, 1979; Bender, 1981; Benevento and Miller, 1981; Petersen *et al.*, 1985). In the inferior and lateral segments the latency of the visual response is approximately 65 ms, but in the medial pulvinar the response latency is somewhat longer and more variable, averaging 84 ms. As expected from the topographic afferents from the retina, superior colliculus and visual cortex, the cells in the inferior and lateral divisions have receptive fields that are well localized and retinotopically organized. In contrast, the medial division is not topographically organized and the cells have larger receptive fields. Neurones in each region are broadly tuned for the orientation or direction of motion of visual stimuli. Some neurones in the inferior and lateral pulvinar that respond to moving stimuli while the monkey fixates do not respond to the same stimulus when it is stationary and the monkey makes an eye movement over it (Robinson and Petersen, 1985). This indicates that the pulvinar neurones receive an extraretinal signal modulating their activity relative to movements of the eyes. The visual responses of these cells, however, do not change with eye position. Lesions of the visual cortex but not the superior colliculus reduce the visual responsiveness of inferior pulvinar neurones (Bender, 1983).

The units in all three regions respond more when a visual stimulus is the target for a saccade. Furthermore, cells in the medial division show enhanced responses to visual stimuli that the monkey must attend to without shifting gaze. The cells that show enhanced responses for stimuli that are targets for a saccade are not modulated in relation to the saccade itself, so they do not provide a motor signal. Units in the inferior and lateral segments that are enhanced if a saccade is executed do not show spatial selectivity; that is, the cell's response is enhanced for stimuli even outside of its receptive field. In contrast, enhanced cells in the medial pulvinar are spatially selective.

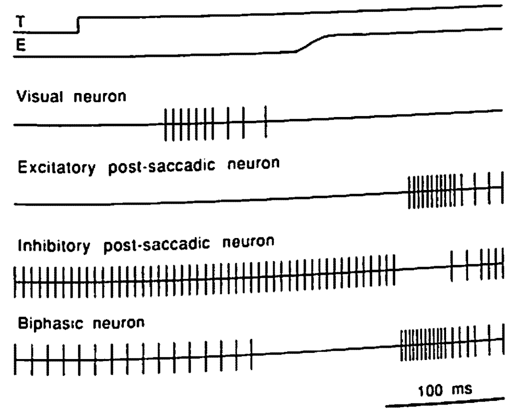

Fig. 15.6 *Neuronal activity in the pulvinar associated with saccades. Conventions are as in Fig 15.2.*

Approximately half of the cells in the inferior and medial divisions of the pulvinar are modulated in relation to saccades (Perryman *et al.*, 1980; Robinson *et al.*, 1986). Three major patterns of discharge are observed. Most of these units discharge after the saccade is initiated with a delay of on average 72 ms. Other cells are suppressed on average 58 ms after the saccade. The third group of neurones display a pause in activity that begins around 44 ms before the saccade followed by a burst after the saccade. Some of these units (predominantly the biphasic) discharge in association with saccades made to visual stimuli as well as to spontaneous saccades made in the dark. These are most common in the inferior and medial divisions. Another population of cells discharges in association with both the appearance of the target and the conclusion of the saccade. All of these neurones appear to have large movement fields and are not influenced by the position of the eye in the orbit. Such units apparently signal the occurrence of a saccade.

The patterns of connectivity and neuronal modulation in the different subdivisions of the pulvinar indicate that they subserve different functions. The inferior and lateral pulvinar nuclei seem to signal the occurrence of visual stimuli and of saccades. The various cortical areas responsible for visual processing ought to be informed of the execution of a saccade so that their processing can be halted or modified while the rapid eye movement is degrading the retinal image (Latour, 1962; Volkmann, 1962, 1968; MacKay, 1970; Matin, 1974; Campbell and Wurtz, 1978; Volkman *et al.* 1978). In fact, evidence will be presented that neurones in striate cortex show a degree of suppression associated with saccadic eye movements, and we have mentioned the post-saccadic modulation of the pregeniculate nucleus. One issue to contend with in this interpretation, however, is the observation that the saccadic suppression begins around 80–100 ms before the saccade, peaks during the movement and persists for 50–150 ms after the saccade. This observation would require that presaccadic units must play a role in modulating visual processing.

Effects of Pharmacological Manipulation

The medial division appears to be involved in selective visual attention. Experimental support for this has been obtained by injecting a GABA agonist or antagonist into the medial pulvinar of monkeys performing a task requiring shifts of visual attention (Petersen *et al.*, 1987). Monkeys were trained to respond to the appearance of a visual stimulus that appeared on either the left or right. The location of the target could be cued by a prior stimulus. This task has been designed to investigate spatial shifts of visual attention in humans (for example, Posner, 1980). If the target appears at the cued location, the reaction time is faster than if the target appears elsewhere. This difference, it is argued, reflects the shift of an attentional process anticipating the appearance of the target at the cued location. Increasing GABAergic inhibition in the medial pulvinar with the agonist, muscimol, increases the reaction times which suggests a slowing or other impairment of the shift of attention. In contrast, decreasing GABAergic inhibition with the antagonist, bicuculline, facilitates the shift of attention in this task as reflected in a reduction in reaction times.

Intralaminar Nuclei

The internal medullary lamina containing the intralaminar nuclei lies primarily between the mediodorsal and the ventrolateral nuclei in the central thalamus (Fig. 15.1). Rostrally, it consists of the central medial, paracentral and central lateral nuclei. Caudally it is comprised of the centre median and parafascicular nuclei. Early thought held that this region of the thalamus subserved a nonspecific arousal function; this was based on the widespread cortical recruiting response that was obtained by electrical stimulation in the intralaminar nuclei (Jasper, 1960).

Effects of Lesions and Electrical Stimulation

More recently evidence has accumulated for the participation of the rostral intralaminar nuclei in visuomotor behaviour. In the first place, lesions in the vicinity of the intralaminar nuclei result in a contralateral visual neglect (Watson *et al.*, 1978; Watson and Heilman, 1979). Furthermore, microstimulation elicits saccades in cat (Schlag and Schlag-Rey, 1971; Maldonado *et al.*, 1980) and monkey (Schlag-Rey and Schlag, 1984). In the monkey the minimum current required is 30 μA and the latency of the saccade is around 40 ms. Stimulation of dorsolateral sites in the intralaminar nuclei elicit saccades that converge on a point in the orbit, while stimulation ventromedially evoked saccades that do not vary with eye position (Schlag-Rey *et al.*, 1987).

Physiological Properties

Single unit recordings in this region of alert, behaving monkeys reveal a variety of response properties (Fig. 15.7). Some units display a visual response with a latency of 80–120 ms (Schlag and Schlag-Rey, 1984). These cells have large receptive fields that include the fovea and emphasize the contralateral hemifield. Apparently, the visual responses do not vary with eye position. The activity of some of the visual cells is enhanced when the stimulus is the target of a saccade. A subpopulation of the visually responsive cells expressed this saccade contingency to a greater degree, being active only when the visual stimulus was the target for a saccade.

Other intralaminar units display a sustained elevation or suppression of activity while the monkey fixates a visual stimulus. The latency of the response following the appearance of the target to be fixated is approximately 100 ms. The origin of this activity is not only visual, however. This is revealed if the monkey saccades away from and back to the target, in which case such units stop discharging before the saccade and reinstate their activity as soon as the target is refixated with no latency. This pattern of modulation during saccades indicates that these fixation units also receive an extraretinal signal. Such activity is evident even when the monkey pursues a moving stimulus. The activity of these units does not vary systematically with gaze angle. The specific relation of these cells to the act of fixation indicates that they provide a signal related to the maintenance of fixation; although, it is not presently clear whether such a signal is a command or a correlate.

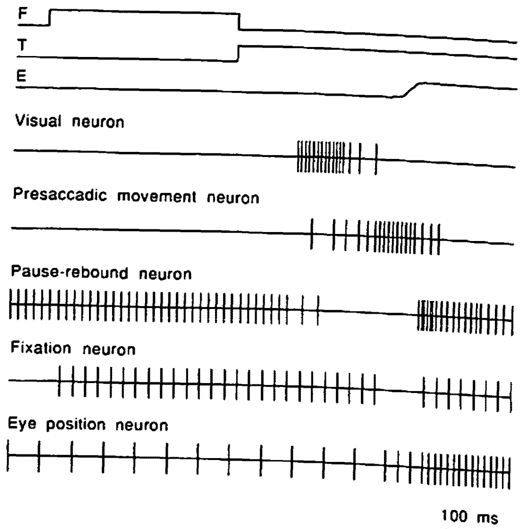

Fig. 15.7 *Neuronal activity in the central thalamus associated with saccades. The fixation spot appears which the monkeys fixates. The fixation spot disappears when a peripheral target appears and a saccade is executed.*

Another population of the intralaminar thalamic neurones exhibits activity related to saccades (Schlag-Rey and Schlag, 1984). Some of these cells discharge prior to saccades, firing on average approximately 100–150 ms and even more than 400 ms before self-initiated saccades; the presaccadic unit discharge occurred after the appearance of the target for visually guided saccades. The presaccadic burst cells tend to display well defined movement fields that are concentrated in the contralateral hemifield. The activity of some presaccadic movement neurones is related to gaze angle.

A different population of cells in the intralaminar nuclei exhibit a pause in activity during saccades and a rebound burst after the saccade. There is variation in the degree of pause and rebound modulation across cells in this population. In many of these units the pause began up to 100 ms before the saccade, while the burst can begin from 25–300 ms after the saccade. A few members of this population exhibit both the pause and rebound postsaccadically. The movement fields of pause-rebound cells are less well defined than are those for presaccadic movement neurones. The modulation of most pause-rebound cells does not vary in light or darkness, but the rebound bursts of a few units are gradually attenuated when the monkey makes saccades in the dark. It has been hypothesized that these units provide a signal to visual neurones to synchronize their processing with the ongoing saccadic eye movements (Schlag and Schlag-Rey, 1983) and in so doing may play a significant role in the development of visual cortical response properties (Singer, 1982; Singer and Rauschecker, 1982).

In the central thalamus other units are modulated according to the position of the eye in the orbit. The maintained activity of these cells is not dependent on visual stimulation and thus reflects an oculomotor signal. The modulation of these eye position cells is not correlated with a particular angle of gaze but is rather accounted for by a gaze axis. In other words, these units discharge whenever the eyes assume an angle of gaze that falls along a particular line projected into the visual field. The preferred axis of gaze of this population of cells tends to lie in the contralateral hemifield. Furthermore, the discharge rate of a cell in relation to a particular eye position is different if the eye moves to that position from different directions.

In these investigations the visual and saccade related neurones were found interspersed in the rostral intralaminar nuclei (primarily the central lateral and paracentral nuclei). Overall, the different visual and eye movement neurones were found intermingled, although visual neurones tend to be more rostral than eye movement cells. The only significant grouping is observed in the eye position units; they constitute essentially the only cell type recorded in the lateral dorsal nucleus.

Connectivity

The lateral dorsal nucleus apparently does not project to the supplementary or frontal eye fields but it does to the posterolateral portion of the inferior parietal lobule (Faugier-Grimaud and Ventre, 1989). The rostral intralaminar nuclei are interconnected with visual and oculomotor regions. The bulk of these anatomical data were compiled in subprimate species. Subcortical afferents to the rostral intralaminar nuclei include but are not limited to the ascending reticular activating formation (Nauta and Kuypers, 1958), the cerebellum (Hendry *et al.*, 1979; Thach and Jones, 1979; Kalil, 1981; Asanuma *et al.*, 1983c; Gonzalo-Ruiz *et al.*, 1988), the pontine reticular formation (Buttner-Ennever and Henn, 1976; Graybiel, 1977; Robertson and Feiner, 1982), the deep subdivision of the superior colliculus (Benevento and Fallon, 1975; Graham, 1977; Partlow *et al.*, 1977; Harting *et al.*, 1980; Graham and Berman, 1981; Yamasaki *et al.*, 1986), the pretectum (Benevento *et al.*, 1977) and weakly if at all from the substantia nigra pars reticulata (Carpenter *et al.*, 1976; Hendry *et al.*, 1979; Ilinsky *et al.*, 1985). It should be admitted that the apparent organization of the response properties of the neurones in this part of the thalamus would not be predicted from the apparently disorganized and diffuse fashion in which the multitude of afferents arborize.

The cortical connections with the intralaminar nuclei are organized and reciprocal. Interestingly, it has been shown that some of the cortical cells send branches to both the intralaminar nuclei and the striatum (Royce, 1983); the converse of branching axons from the thalamic neurones has not been described. The intralaminar nuclei project to striate and extrastriate visual cortical areas, the inferior parietal lobule, the frontal eye fields, the supplementary eye fields and prefrontal cortex (Jacobson *et al.*, 1978; Barbas and Mesulam, 1981; Tigges *et al.*, 1982; Asanuma *et al.*, 1985; Huerta *et al.*, 1986; Schlag-Rey *et al.*, 1987; Stanton *et al.*, 1988b; Huerta and Kaas, 1988). The intralaminar nuclei also provide a major projection to the striatum (Jones and Leavitt, 1974; Kalil, 1978).

Other Nuclei

Other thalamic nuclei are mentioned in connection with various visuomotor brain centres. For example, the lateral portion of the mediodorsal nucleus and the medial portion of the ventrolateral or ventroanterior nuclei are reciprocally connected with the frontal and supplementary eye fields (see Huerta *et al.*, 1986; Huerta and Kaas, 1988; Stanton *et al.*, 1988a). These thalamic regions also receive substantial input from the deep layers of the superior colliculus (Benevento and Fallon, 1975; Harting *et al.*, 1980) and the lateral substantia nigra pars reticulata (Carpenter *et al.*, 1976; Ilinsky *et al.*, 1985). In fact, some cells in the substantia nigra send branching collaterals to both the ventral thalamic nuclei and the superior colliculus (Parent *et al.*, 1983b). The ventrolateral nucleus also receives input from the fastigial nucleus (Asanuma *et al.*, 1983b). This portion of the thalamus also receives input from the supranuclear eye movement brainstem sites (Graybiel, 1977). Some visuomotor units have been recorded in these thalamic nuclei adjacent to the intralaminar nuclei (Schlag and Schlag-Rey, 1984), but further experimental work is required to more fully characterize the neuronal activity related to visually guided saccades in these nuclei and distinguish it from what is observed in the pregeniculate, the pulvinar and the intralaminar nuclei.

The parafascicular nucleus also deserves more attention to determine its role in visuomotor behaviour. It receives input from the frontal eye fields (Huerta *et al.*, 1986; Stanton *et al.*, 1988a) and the inferior parietal lobule (Divac *et al.*, 1977; Mesulam *et al.*, 1977; Kasdon and Jacobson, 1978 but see Asanuma *et al.*, 1985; Yeterian and Pandya, 1985) and the deep layers of the superior colliculus (Benevento and Fallon, 1975; Harting *et al.*, 1980) and also the PPRF (Graybiel, 1977). In the cat the parafascicular nucleus projects to the ventral lateral geniculate nucleus (pregeniculate nucleus) (Royce and Mourey, 1985).

Cortex

While many saccadic eye movements are reflexive, such as the fast phases of the vestibulo-ocular reflex or of optokinetic nystagmus or the orienting response to a startling stimulus, the most interesting use of fast eye movements is in rapidly and economically scanning the complex scenes we confront during natural vision (e.g. Monty and Senders, 1976; Groner and Fraisse, 1982). This scanning process is clearly under voluntary control; indeed, we can saccade on verbal command with no target in sight. The cerebral cortex is required for these saccades. Unilateral ablation of the entire cerebral cortex in monkeys results in severe oculomotor deficits (Pasik *et al.*, 1960; Tusa *et al.*, 1986); goal directed, visually guided saccades into the affected hemifield are eliminated while spontaneous saccades and the quick phases of nystagmus are preserved.

The standard textbook treatment of the cortical control of eye movements declares that there is a frontal eye field responsible for voluntary scanning movements and an occipital eye field responsible for visually guided eye movements. Considerable work in recent years allows us to adopt a more sophisticated view. A surge of progress in understanding the role of the cerebral cortex in the generation of eye movements followed the recognition that goal-directed saccades by motivated monkeys are operationally different from spontaneous, self-generated saccades. Several areas of the cerebral cortex participate in gaze control, and – what is by now an old theme – different areas play different roles. Some areas, like striate cortex (also known as area 17 or visual area V1), provide input to the system, while other areas, like the frontal eye fields, are stationed toward the output end. The role of the cortex in the generation and control of saccades is an active area of research with additional areas being implicated.

Striate Cortex

As reviewed in chapters 8, 9, 10, 11 and 12, striate cortex is the first station in the cortical visual pathway (Fig. 15.1(a)). There are two ways to approach the relation of striate cortex to saccadic eye movements. One is to consider what impact the primary visual area has on the oculomotor system, and the other is to consider what impact the oculomotor system has on striate cortex. These two will be taken in turn.

Effects of Electrical Stimulation

Electrical stimulation of the occipital cortex evokes contraversive conjugate eye movements (Schaeffer, 1888; Walker and Weaver, 1940; Wagman *et al.*, 1958; Schiller, 1977; Keating *et al.*, 1983). The threshold for intracortical microstimulation, however, is relatively high, 100 μA, and the latencies are relatively long, 60–80 ms. Thus, the access of striate cortex to the brainstem saccade generator must be indirect.

Effects of Lesions

Ablation of striate cortex does not eliminate eye movements in monkeys (Pasik and Pasik, 1964; Segraves *et al.*, 1987) or humans (Poppel *et al.*, 1973; Perenin and Jeannerod, 1975; Weiskrantz *et al.*, 1974). Partial lesions of V1 temporarily prevent monkeys from making saccades to targets that lie in the affected area of the visual field (Mohler and Wurtz, 1977). However, as soon as the afflicted monkeys can detect the presence of a stationary visual stimulus in the affected region, they can execute an accurate saccade to it. In contrast, saccades to stimuli moving in the affected region are permanently inaccurate (Segraves *et al.*, 1987). These results indicate that striate cortex is required for the estimation of the velocity of a moving saccade target. Consistent with this interpretation, monkeys suffering striate cortex lesions are also unable to pursue a target moving in the affected region of the visual field (Segraves *et al.*, 1987).

Physiological Properties

Neurones in V1 respond to the visual stimuli that constitute the targets for saccades with latencies ranging from 30–70 ms. Unlike their counterparts in higher cortical areas, most neurones in area 17 do not have enhanced activity if the stimulus is a target for a saccade (Wurtz and Mohler, 1976b; Robinson *et al.*, 1980). A minority of units, though, do show a slight enhancement for a stimulus that is the target for a saccade, but this enhancement is not spatially selective, i.e. the stimulus need not fall in the cell's receptive field for the enhancement to occur. This indicates that the variation in activity has more to do with general arousal than with any selective attention process.

The results of experiments demonstrating a perisaccadic suppression of the spontaneous activity of striate cortex neurones manifests the impact of the oculomotor system on visual processing (Duffy and Burchfield, 1975). This neural suppression, which is observed even during eye movements in the dark, begins on average 20–30 ms after the saccade is initiated and lasts for around 200 ms. Most cells are suppressed specifically for saccades into one quadrant. This modulation resembles the suppression seen in the pregeniculate nucleus and may be a correlate of saccadic suppression, perhaps imposed by the pause-rebound neurones described above.

It has also been possible to demonstrate the effects of saccadic eye movements on the visual responsiveness of striate cortex neurones (Wurtz, 1969a,b; Barlett *et al.*, 1976; Judge *et al.*, 1980). When presented with a stimulus during a saccade, most striate cortex neurones exhibit an attenuated response or no change from the response observed when the eye is at rest. When the stimulus is moved across the receptive field at the same speed as that achieved during a saccade, the cells' particular response is no different. In other words, these results indicate that striate cortex cells do not receive an extraretinal signal informing them of eye movements.

There is evidence for modulation of neuronal activity in area 17 associated with spatial shifts of attention (Boch, 1986). These results were obtained in monkeys performing a task in which they were required to attend to a peripheral stimulus while fixating another. With a behaviourally relevant stimulus present in its receptive field, following the disappearance of the fixation spot, half of the units recorded in area 17 show an elevation of activity with a latency of approximately 200 ms. This reactivation is spatially selective, requiring the stimulus to lie within a cell's receptive field.

Occipital Extrastriate Visual Cortical Areas

V2

Single unit recordings in the posterior bank of the lunate sulcus (area V2) of monkeys making visually guided saccades reveal properties similar to those observed in striate cortex (Robinson *et al.*, 1980). While the visual response of many of the units does not change if the stimulus is the target for a saccade, a larger percentage of the units than that observed in striate cortex exhibit the nonspatially specific presaccadic enhancement.

V3A

As explained, in the generation of a saccade it is necessary to register the position of the eye in the orbit. Recent work has demonstrated that the visual responses of neurones in the extrastriate visual area V3A vary with the angle of gaze (Galletti and Battaglini, 1989). The cells discharge more in response to a visual stimulus when the monkey's gaze is directed to the contralateral hemifield. There is no correlation of gaze angle modulation with the other receptive field properties, like orientation or direction selectivity. It appears that the gaze-angle dependent cells may be segregated from the non-gaze-angle tuned cells. The specific subcortical afferents of V3A are not presently known. It is of interest, however, that the inferior parietal lobule receives indirect input from V3A (see Andersen, 1987).

V4

In contrast to most cells of area V1, neurones in the prelunate gyrus (visual area V4) (Fig. 15.1(a)) display an enhanced response when a saccade is made to the visual stimulus but only if the stimulus lies within the cell's receptive field (Fischer and Boch, 1981). The latency of response is approximately 80 ms. When the saccade is delayed after the appearance of the target there is a second elevation of activity associated with the saccade (Boch and Fischer, 1983). This second elevation is synchronized more to the disappearance of the fixation spot that serves as the cue to move than to the execution of the saccade. In a different task that requires a monkey to shift attention to a peripheral stimulus without making a saccade to it, most cells in V4 display an elevation approximately 220 ms after the cue to shift attention (Fischer and Boch, 1985). In another study monkeys were presented with several stimuli simultaneously and required to identify the one different from the rest by making a saccade. A few cells in the prelunate gyrus exhibit a specific enhancement associated with the selection of a particular stimulus for the saccade (Haenny *et al.*, 1988).

Mention was made in the introduction that it is important to consider shifts of attention along with shifts of gaze. A number of recent experiments have demonstrated the specific effects of directed visual attention on the activity of neurones in V4. The results of these experiments are based on recordings in alert monkeys performing visual discrimination or matching tasks. Neurones in V4 give an enhanced response when the monkey is attentive during a task (Mountcastle *et al.*, 1987). The response of most units in V4 to their optimal stimulus is attenuated when the monkey is not attending to that stimulus but is instead attending to an adjacent non-optimal stimulus (Moran and Desimone, 1985); however, it is necessary that the two stimuli both fall in the V4 unit's receptive field. Perhaps not unexpectedly, such attention-related modulation is not observed in striate cortex. Other experiments show that when the monkey is required to perform a visual discrimination in a number of circumstances, V4 neurones show amplified activity and refined selectivity (Haenny and Schiller, 1988; Haenny *et al.*, 1988; Spitzer *et al.*, 1988).

This prelunate cortical area receives a substantial thalamic input from the inferior and lateral pulvinar and projects back to the lateral, inferior and medial pulvinar (Chow, 1950; Campos-Ortega and Hayhow, 1972; Hollander, 1974; Benevento and Rezak, 1975, 1976; Ogren and Hendrickson, 1976, 1977; Benevento and Davis, 1977; Trojanowski and Jacobson, 1975, 1976; Curcio and Harting, 1978; Graham *et al.*, 1979; Ogren and Hendrickson, 1979; Graham, 1982; Ungerleider *et al.*, 1983; Weber and Yin, 1984; Asanuma *et al.*, 1985). V4 projects to other visuomotor cortical areas including a particular subregion of the inferior parietal lobule, the lateral interparietal area (Seltzer and Pandya, 1980; Andersen *et al.*, 1985). V4 also projects in the vicinity of, if not directly to the frontal eye fields (Kunzle and Akert, 1977; Huerta *et al.*, 1987).

Superior Temporal Polysensory Area

There is preliminary evidence that the superior temporal polysensory area (Bruce *et al.*, 1982) may participate in directing gaze and attention. This area receives thalamic input from the medial pulvinar (Burton and Jones, 1976) and is interconnected with the various visuomotor areas including the inferior parietal lobule and the frontal eye field (e.g. Seltzer and Pandya, 1984; Huerta *et al.*, 1987). It also projects to the deep division of the superior colliculus (Fries, 1984). During visually guided saccades, visual, visuomotor and presaccadic motor cells have been recorded (Colby and Miller, 1986). Finally, ablation of this area results in contralateral neglect (Luh *et al.*, 1986).

Inferior Parietal Lobule

A number of reviews of parietal lobe function have appeared (Lynch, 1980; Wurtz *et al.*, 1980; Mesulam, 1981; Hyvarinen, 1982; Mountcastle *et al.*, 1984; Andersen, 1987). The inferior parietal lobule (IPL) in macaque monkeys is located at the posterior limit of the parietal lobe, adjacent to the occipital extrastriate visual cortical areas (Fig. 15.1(a)). This region can be subdivided anatomically and functionally; in fact, progress in understanding this part of the brain has been correlated with the degree of parcellation. The area that appears to participate in gaze control occupies the medial aspect of the IPL and is referred to as area 7a (nomenclature of Vogt and Vogt, 1919) or area PG (nomenclature of von Bonin and Baily, 1947); the lateral region, area 7b or PF, subserves the somatosensory system (Hyvarinen and Shelepin, 1979; Hyvarinen, 1981). The region originally referred to as 7a has itself been subdivided into at least three fields. First, on the anterior bank of the superior temporal sulcus is an area referred to as the medial superior temporal (MST) area; this is an extrastriate visual area comprised of units that are responsive to moving stimuli (see Maunsell and Newsome, 1987; Motter, chapter 14). Second, an area on the lateral bank of the intraparietal sulcus is referred to as area POa or the lateral interparietal area (LIP). Third, the crown of the gyrus is still referred to as area PG or 7a.

Connectivity

The IPL receives thalamic afferents from the pulvinar and intralaminar nuclei (Petras, 1971; Burton and Jones, 1976; DeVito and Simmons, 1976; Divac *et al.*, 1977; Mesulam *et al.*, 1977; Stanton *et al.*, 1977; Kasdon and Jacobson, 1978; Pearson *et al.*, 1978; Weber and Yin, 1984; Asanuma *et al.*, 1985; Baleydier and Mauguiere, 1987). The crown of the IPL, area 7a, receives thalamic input mainly from the medial pulvinar nucleus as well as from the lateral posterior nucleus, the central medial, paracentral and parafascicular intralaminar nuclei, the rostral thalamus around the mamillothalamic tract, the anterior medial nucleus, and the ventral anterior nucleus (Divac *et al.*, 1977; Mesulam *et al.*, 1977; Stanton *et al.*, 1977; Kasdon and Jacobson, 1978; Asanuma *et al.*, 1985; Baleydier and Mauguiere, 1987). LIP's thalamic afferents arise primarily in the lateral pulvinar (Asanuma *et al.*, 1985). The posterolateral bank of the IPL, including MST receives thalamic input from the inferior portion of the ventral posterior nucleus, the magnocellular part of medial geniculate nucleus, the central lateral intralaminar nucleus, and the lateral dorsal nucleus (Faugier-Grimaud and Ventre, 1989). These thalamocortical projections tend to be topographic and reciprocal. The neurones in the medial pulvinar that project to 7a do not branch to cingulate (Baleydier and Mauguiere, 1987) or frontal cortex (Asanuma *et al.*, 1985). Area 7a also receives subcortical afferents from the nucleus basalis of the substantia innominata, the claustrum, the pretectum and the locus coeruleus (Mesulam *et al.*, 1977).

The cortical afferents also distinguish 7a from LIP. 7a receives input from occipital, cingulate, parietal, temporal and prefrontal (except the frontal eye fields) (Divac *et al.*, 1977; Mesulam *et al.*, 1977; Seltzer and Pandya, 1978, 1984; Baleydier and Mauguiere, 1987). In distinction to 7a, LIP receives afferents from the prelunate gyrus (V4), the rostral, somatosensory portion of the IPL (PF) and the frontal eye fields (Seltzer and Pandya, 1980; Andersen *et al.*, 1985).

The efferent projections of the IPL also distinguish the different subareas. The posterolateral bank of the IPL, besides reciprocating the previously mentioned thalamic projections, sends efferents to the deep division of the superior colliculus, the pontine nuclei including the dorsolateral, the vestibular nuclei and the nucleus prepositus hypoglossi (Faugier-Grimaud and Ventre, 1989). LIP projects to the dorsal pontine nuclei (May and Andersen, 1986), to the deep division of the superior colliculus overlapping frontal eye field afferents (Fries, 1984; Asanuma *et al.*, 1985; Lynch *et al.*, 1985) to the pregeniculate nucleus, zona incerta and pretectum (Asanuma *et al.*, 1985). Area 7a sends reciprocal thalamic projections, as well as providing input to the dorsolateral, lateral and ventral pontine nuclei, the striatum, the intralaminar thalamic nuclei, the pretectum, the superior colliculus, the reticular thalamic nucleus, the suprageniculate nucleus and the claustrum (Peele, 1942; Petras, 1971; Brodal, 1978; Wiesendanger *et al.*, 1979; Glickstein *et al.*, 1980; Weber and Yin, 1984; May and Andersen, 1986). The parietal input to the frontal lobe, including the frontal eye fields is more prominent from LIP than 7a (Barbas and Mesulam, 1981; Andersen *et al.*, 1985; Huerta *et al.*, 1987). The IPL also projects to the superior temporal sulcus (Seltzer and Pandya, 1984) as well as exchanging prominent connections with the limbic system (Seltzer and Van Hoesen, 1979; Seltzer and Pandya, 1984).

Effects of Electrical Stimulation

Electrical stimulation of the IPL elicits saccades (Ferrier, 1875; Fleming and Crosby, 1955; Wagman, 1964; Keating, 1983; Shibutani *et al.*, 1984). The threshold for intracortical microstimulation averages 85–90 µA, but in LIP the threshold is lower, approximately 40 µA, and the latencies are shorter, 30–50 ms. There is some evidence for a columnar organization of saccade direction; that is neighbouring sites tend to elicit saccades of similar directions. At most sites the vector of the saccade does not vary with eye position, but at a few sites in the posterolateral IPL the direction of the evoked saccade varies with eye position. Ablation of either the superior colliculus or frontal eye fields alone does not prevent stimulus elicited saccades from the parietal cortex; so the IPL has access to the saccade generator through either of these structures (Keating and Gooley, 1988).

Effects of Lesions

A lesion of posterior parietal cortex in humans results in a variety of visual attention, localization and orientation deficits (see, for example, Critchley, 1953; Mesulam, 1981). This extensive literature cannot be reviewed here; instead we will concentrate on the visuomotor deficits (Balint, 1909; Holmes, 1918; Paterson and Zangwill, 1944; Cogan and Adams, 1953; Carmichael *et al.*, 1954; Hecaen and de Ajuriaguerra, 1954; Cogan, 1965; Godwin-Austen, 1965; Allison *et al.*, 1969; Sundqvist, 1979; Baloh *et al.*, 1980; Hausser *et al.*, 1980; Montero *et al.*, 1982; Pierrot-Deseilligny *et al.*, 1986). Experimental parietal lesions in monkeys appear to produce similar impairments (Lynch and McLaren, 1982, 1983, 1989). In general, there are fewer saccades into the affected hemifield and the saccades have increased latency. Once fixated, there are difficulties in maintaining sustained fixation or in pursuing the stimulus if it moves. Combined ablation of the posterior parietal cortex and the frontal eye fields in both monkey (Lynch *et al.*, 1986) and man (Hecaen and de Ajuriaguerra, 1954; Hausser *et al.*, 1980) results in a much more severe gaze deficit.

A hallmark deficit resulting from parietal lesions is contralateral neglect (failure to respond to stimuli presented in the affected hemifield) or extinction (perpetual selection of the stimulus in the unaffected hemifield when two stimuli are presented together). Experimental investigations of patients suffering parietal lobe lesions indicate that the basis of the neglect or extinction is not sensory but can be attributed to the attention system (Riddoch and Humphreys, 1983; Posner *et al.*, 1984, 1987; Baynes *et al.*, 1986). Indeed, it may be that the deficit is as specific as an impairment in the particular operation of disengaging attention from the present object (Posner *et al.*, 1984).

Physiological Properties

Quite a variety of neurones related to different visuomotor behaviours have been recorded in the IPL (Fig. 15.8). One population of units is visually responsive with a latency of approximately 90 ms. The response of these cells is enhanced if the monkey is attentive or if the stimulus is the target for a saccade (Yin and Mountcastle, 1977; Robinson *et al.*, 1978; Motter and Mountcastle, 1981; Mountcastle *et al.*, 1981; Andersen *et al.*, 1987). The enhancement of the visual response is spatially specific; it does not require execution of a saccade but instead a covert shift of attention (Bushnell *et al.*, 1981). In other words, if a monkey is required to direct attention to a peripheral stimulus while continuing to fixate a central stimulus, parietal neurones show enhanced responses to the appearance of the peripheral stimulus. It is important to note that this attention-specific enhancement does not originate in area 7a; it is present in the medial pulvinar (see above). Consistent with this is the demonstration that the activity of these visually responsive cells is facilitated by electrical stimulation of the pulvinar (Blum,1985). The sensitivity of visual cells also varies with the angle of gaze (Andersen and Mountcastle, 1983; Andersen *et al.*, 1985b). The receptive field properties of these units are more sophisticated than those of cells recorded from lower cortical visual areas; for example, many of the units in the IPL have large receptive fields that spare the fovea and respond selectively to stimuli moving radially with respect to the fovea in different parts of the receptive field (Motter *et al.*, 1987; Steinmetz *et al.*, 1987). The cells in the posterolateral IPL, i.e. the medial superior temporal (MST) visual area respond specifically to expansion, contraction or rotation of the image and integrate motion over a large part of the visual field (Sakata *et al.*, 1983; Saito *et al.*, 1986; Tanaka *et al.*, 1986).

Establishing whether nonvisual neurones in the IPL discharge specifically before saccades has proven to be a less than straightforward endeavour. In early recordings, units were found that appeared to discharge in relation to visually guided and not spontaneous saccades (Lynch *et al.*, 1977). Subsequent investigations, however, demonstrated that units that discharged with saccades also had visual responses and that the modulation associated with the saccade actually seemed to be triggered by the visual stimulus

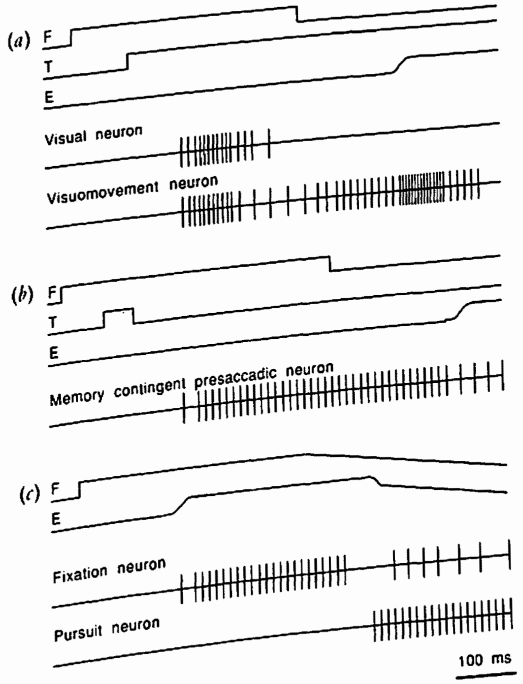

Fig. 15.8 *Neuronal activity in the inferior parietal lobule associated with saccades and smooth pursuit. (a) Same visual tracking task as that illustrated in Fig. 15.5(a). (b) Same as 15.5(d). (e) The monkey fixates a spot (F); when the spot begins to move, the monkey pursues it.*

(Robinson *et al.*, 1978). Some of this disagreement may have been a result of recording in different subdivisions within the IPL. Recent work indicates that there is saccade-related activity in the LIP and less in 7a. In a task that separates in time the appearance of the target and the subsequent saccade, parietal units are found that respond for both the visual stimulus and the saccade (Andersen *et al.*, 1987). The visual response component shows a latency of around 75 ms, and the presaccadic discharge begins 100–150 ms before the saccade (Barash *et al.*, 1988). These visuomovement cells display movement fields that can range from 20° in width to the entire hemifield. Also the activity of many of the saccade-related cells in LIP varies with eye position (Andersen *et al.*, 1988).

In a task that requires a saccade to the remembered location of a briefly flashed target, another population of neurones in LIP can be distinguished (Gnadt and Andersen, 1988). These units exhibit the same pattern of modulation as that of the quasi-visual cells recorded in the superior colliculus. They begin to discharge following the appearance of the target that must be remembered, and they maintain an elevated rate of discharge until the saccade is executed. These units are tuned for the direction and amplitude of the saccade. Furthermore, in a double step task in which two saccades are required, such cells begin to discharge prior to a saccade of the appropriate direction and amplitude even if no visual stimulus falls in the cell's receptive field.

Another population of neurones recorded in the IPL is active during fixation or pursuit (Mountcastle *et al.*, 1975; Lynch *et al.*, 1977; Robinson *et al.*, 1978). The activity is modulated by the angle of gaze and is interrupted by saccades (Lynch *et al.*, 1977; Sakata *et al.*, 1980; Andersen *et al.*, 1987). Some fixation cells have gaze fields that are fairly restricted, while the remainder have expansive gaze fields. Many of the fixation neurones have visual responses (Robinson *et al.*, 1978; Komatsu and Wurtz, 1988a,b), but other neurones discharge during smooth pursuit and not during stable fixation; they also discharge continuously during pursuit even when the target is briefly turned off (Mountcaste *et al.*, 1975; Lynch *et al.*,1977; Sakata *et al.*, 1980; Newsome *et al.*, 1988). The activity of these cells indicates that they receive both visual and extraretinal pursuit signals. Furthermore, visual tracking cells are also active when combined eye and head movements are employed (Kawano *et al.*, 1984). These units related to fixation and pursuit tend to be recorded in the posterolateral IPL, i.e. in MST. Interestingly, this is the region in which the saccades evoked by microstimulation vary with eye position (Shibutani *et al.*, 1984). This region also receives thalamic afferents from the lateral dorsal nucleus (Faugier-Grimaud and Ventre, 1989), the region where a high density of eye position cells are recorded (Schlag-Rey and Schlag, 1984).

Summary

It appears that the IPL should be considered a collection of functionally distinct but related areas. One portion seems to play an important role in directing visual attention. The evidence from anatomical, lesion and single-unit recording experiments indicates that the crown of the intraparietal gyrus, area 7a subserves this function. The fact that the discharges of many of the neurones in the IPL is modulated by eye position suggests that the IPL may be important for spatial localization (see Skavenski and Hansen, 1978; Matin, 1985). Finally, as reviewed, certain investigators have argued that the posterior parietal cortex serves to command movements, and recent evidence for presaccadic neurones in the LIP and pursuit-related activity in MST is consistent with such a role. In this capacity as a saccade command centre, however, the IPL must be coordinated with the frontal cortex.

Frontal Eye Fields

Effects of Electrical Stimulation

Ferrier (1875) demonstrated that electrical stimulation of a large part of frontal cortex elicits eye movements. Subsequent workers further delineated the regions of frontal cortex from which eye movements could be elicited in a variety of primate species including human (Beevor and Horsley, 1888; Horsley and Schaefer, 1888; Mott and Schaefer, 1890; Sherrington, 1893; Risien Russell, 1894; Grunbaum and Sherrington, 1901; Jolly and Simpson, 1907; Vogt and Vogt, 1907, 1919; Leyton and Sherrington, 1917; Foerster, 1931; Penfield and Boldrey, 1937; Smith, 1940, 1949; Walker, 1940; Rasmussen and Penfield, 1948; Penfield and Rasmussen, 1950; Crosby *et al.*, 1952; Lemmen *et al.*, 1959; Wagman *et al.*, 1961; Robinson and Fuchs, 1969; Marrocco, 1978; Schiller *et al.*, 1979; Blum *et al.*, 1982; Bruce *et al.*, 1985). The results of these studies converged on the region that lies in the rostral bank of the arcuate sulcus in rhesus monkeys (Fig. 15.1), called the frontal eye field (FEF). A recent review of FEF function has appeared (Crowne, 1983).

The results of the more recent of these investigations show that electrical microstimulation of the FEF results in saccades whose direction and amplitude do not vary with gaze angle but rather vary with location in the FEF. The minimum current required is less than 50 μA, and the latency of the saccade is 30–45 ms. In single penetrations the direction of the evoked saccade varies gradually which indicates that saccade direction may be arranged in a columnar organization in the FEF. There is a measure of topography in that smaller saccades are represented laterally, and longer saccades, medially. Also, stimulation in the region representing short saccades can result in smooth pursuit eye movements. The threshold for eliciting saccades from a particular site depends on the state of the monkey. Saccades are evoked with the lowest current levels when the monkey is alert but not fixating intently. Electrical thresholds are elevated if the monkey is actively fixating or pursuing a target (Marrocco, 1978; Goldberg *et al.*, 1986) or if the monkey is drowsy or pharmacologically depressed (Robinson and Fuchs, 1969).

A number of other interesting effects of FEF stimulation have been observed. First, prolonged stimulation (over 200 ms) results in multiple saccades, all of the same direction and amplitude. Also, simultaneous stimulation of the FEF and the superior colliculus elicits a saccade whose amplitude and direction are the vector sum of the individual saccades represented at the stimulation sites in either structure weighted by the stimulus current (Schiller *et al.*, 1979). This result indicates that there is a high degree of organization in the mapping of these two structures relative to one another. Finally, monkeys can compensate for deflections of the eye caused by FEF stimulation prior to saccade initiation, and this compensation is not eliminated by lesions of the superior colliculus (Schiller and Sandell, 1983).

Connectivity

The FEF can influence the oculomotor system at a number of points. The FEF projects directly to both mesencephalic and pontine brainstem oculomotor regions (Astruc, 1971; Kunzle and Akert, 1977; Leichnetz, 1982b; Leichnetz *et al.*, 1984a,b; Schnyder *et al.*, 1985; Huerta *et al.*, 1986; Stanton *et al.*, 1988b). The deep layers of the superior colliculus also receive FEF afferents (Astruc, 1971; Kunzle *et al.*, 1976; Kunzle and Akert, 1977; Leichnetz *et al.*, 1981; Distel and Fries, 1982; Fries, 1984; Komatsu and Suzuki, 1985; Huerta *et al.*, 1986; Stanton *et al.*, 1988b).

Another major output of the FEF is the striatum (Kunzle and Akert, 1977; Yeterian and Van Hoesen, 1978; Selemon and Goldman-Rakic, 1985; Stanton *et al.*, 1988a). The termination in the striatum is topographically organized; the medial aspects of the FEF project to the central part of the head and body of the caudate and dorsomedial putamen while the lateral portion of the FEF terminates ventrolaterally in the caudate and ventromedially in the putamen (Stanton *et al.*, 1988). It appears that the FEF efferents terminate in precisely the region where visuomotor activity is recorded in the caudate (Hikosaka *et al.*, 1989a,b,c). The FEF also projects to the pretectum (Astruc, 1971; Kunzle and Akert, 1977; Leichnetz, 1982a). There is evidence in the rat that single cells in the FEF may branch and send collaterals to the PPRF and oculomotor nucleus or the superior colliculus and oculomotor nucleus (Leichnetz and Gonzalo-Ruiz, 1987).

The FEF is reciprocally connected to a number of thalamic nuclei; however, a consensus on the details has not been reached. The nuclei mentioned include the mediodorsal, ventroanterior, medial pulvinar, central lateral, paracentral, parafascicular, ventrolateral including area X, limitans and suprageniculate (Pribram *et al.*, 1953; Scollo-Lavizzari and Akert, 1963; Astruc, 1971; Trojanowski and Jacobson, 1974; Box and Benevento, 1975; Tobias, 1975; Kievet and Kuypers, 1977; Barbas and Mesulam, 1981; Ilinsky *et al.*, 1985; Huerta *et al.*, 1986; Stanton *et al.*, 1988a). Notably absent is any connection with the lateral dorsal nucleus. These connections are topographically organized (Stanton *et al.*, 1988a). The functional role of the various thalamocortical relations of the FEF is an area deserving further study.

The FEF are also reciprocally connected with a number of cortical regions including the supplementary eye field, peri-principal prefrontal, postarcuate premotor, caudal superior temporal sulcus (areas MT and MST), the IPL (area LIP) and cingulate (Pandya and Kuypers, 1969; Jones and Powell, 1970; Chavis and Pandya, 1976; Mesulam *et al.*, 1977; Maioli *et al.*, 1983; Godschalk *et al.*, 1984; Petrides and Pandya, 1984, 1988; Anderson *et al.*, 1985; Barbas and Mesulam, 1981, 1985; Ungerleider and Desimone, 1986; Huerta *et al.*, 1987).

It is worthwhile to pause and consider what each of these cortical projections implies about FEF function. The connection with the supplementary eye fields and the IPL is probably involved in coordinating saccades as reviewed in this chapter. The connections with prefrontal cortex are probably important for the more complex planning and regulation of visual behaviour (see Fuster, 1980; Goldman-Rakic, 1987). The post-arcuate premotor area is a higher level skeletomotor area (reviewed by Wise 1984, 1985); this connection indicates that FEF participate in the coordination of eye, head and body movements (see for example Bizzi, 1974; Herman *et al.*, 1981; Gielen *et al.*, 1984; Fisk and Goodale, 1985; Bock, 1986, 1987; Fischer and Rogal, 1986; Tomlinson and Bahra, 1986a,b; Guitton and Volle, 1987; Baedeker and Wolf, 1987). The neural basis of this coordination has only begun to be explored (Bizzi and Schiller, 1970; Van der Steen *et al.*, 1986). Afferents from the extrastriate visual areas in parietal and temporal lobes provide visual input that has undergone considerable processing (Maunsell and Newsome, 1987; Motter, chapter 14) and is certainly important for specifying the target for a visually-guided saccade. The feedback projections of the FEF onto these same visual cortical areas must also be performing an important function in modulating the visual processing in synchrony with the moving eyes.

Physiological Properties

The first single unit recordings in the FEF of alert monkeys found a small proportion (less than 10%) of neurones that discharged in relation to saccadic eye movements (Bizzi, 1968; Bizzi and Schiller, 1970). One population of these units fired in relation to spontaneous saccades as well as to the fast phase of nystagmus in untrained monkeys, but the discharge began after the saccade, i.e. was postsaccadic. The other identified type of neurone displayed a maintained level of activity that varied with the angle of gaze; these cells were also active during pursuit and the slow phase of nystagmus. Neither of these classes of units changes their pattern of modulation in the dark, so their activity is not determined by visual afferents.

The fact that the saccade-related cells discharged only after the saccade had been initiated was difficult to reconcile with the numerous studies demonstrating that saccades could be elicited by stimulating the FEF. Furthermore, it is possible to record an evoked potential over frontal cortex as well as the posterior parietal cortex prior to voluntary saccades (Kurtzberg and Vaughan, 1973). Adding to the riddle was the observation that lesions of the FEF result in transient but clear eye movement deficits (Ferrier and Yeo, 1884; Bianchi, 1895; Jacobson, 1936; Kennard, 1939; Clark and Lashley, 1947; Welch and Stuteville, 1958). Finally, the fact that 90% of the units recorded in these original studies were not modulated consistently in the untrained monkeys indicates that something more was required to elicit activity in these neurones and learn how the FEF participate in gaze control.

Subsequent single unit recordings in the FEF of monkeys trained in visual tracking tasks revealed that nearly half of the cells have visual responses (Fig. 15.9) (Mohler *et al.*, 1973; Wurtz and Mohler, 1976b; Pigarev *et al.*, 1979; Kubota *et al.*, 1980; Suzuki and Azuma, 1977, 1983; Goldberg and Bushnell, 1981; Bruce and Goldberg, 1985; Schall, 1989a). The latency of response is 60–100 ms. The time to peak activation is 45–50 ms. The receptive fields are large, emphasizing the contralateral hemifield but often extending into the ipsilateral. There is a rough map of the visual field in the prearcuate cortex with smaller, more central receptive fields represented laterally and larger, more peripheral receptive fields represented medially. This map corresponds to the length of the saccades elicited by microstimulation in the different regions of the FEF. The response of the visual cells is enhanced if the stimulus is the target for a saccade (Wurtz and Mohler, 1976b; Goldberg and Bushnell, 1981). This enhancement is not observed if the stimulus falls outside the cells' receptive field or if the monkey is not required to make a saccade. Finally, the visual neurones tend not to respond to auditory stimuli.

While it seems beyond doubt that the visually responsive neurones in the FEF participate in selecting the target for a saccade, this role has only recently been investigated experimentally (Schall, 1989c). In the physiology experiments described throughout this chapter a single, unambiguous target is presented, but monkeys can be trained to perform visual search tasks in which multiple stimuli are presented among which they must discriminate the target. The saccade latency during visual search is longer than that in a typical detection task; presumably the extra time reflects the additional processing required to select the target. The preliminary results of single-unit recordings in the FEF of a monkey performing visual search indicate that while the activity of most visually responsive cells is no different during search or detection, there are examples of neurones with either longer response latency or prolonged activation during search. Other visually responsive cells displayed a spatial tuning according to the position of the correct target relative to the receptive field even though in all trials the same distractor stimulus fell in the receptive field. These preliminary results suggest that some neuronal activity in the FEF may reflect the internal processing of saccade target selection during visual search.

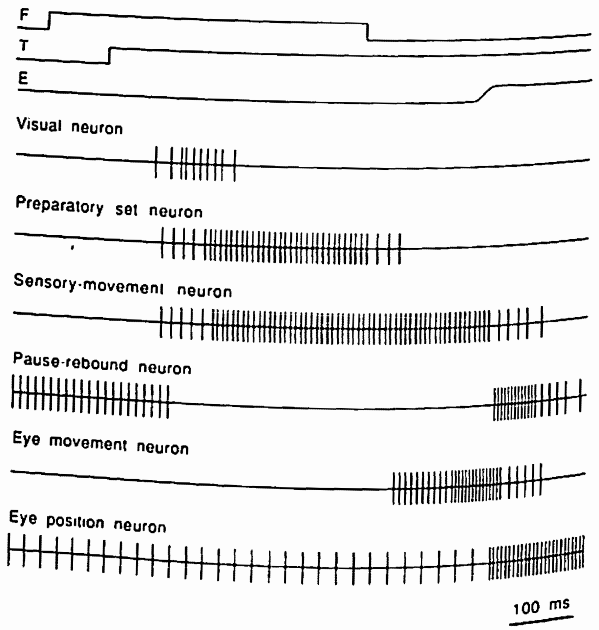

Fig. 15.9 *Neuronal activity in the frontal eye field associated with saccades. Same task as that illustrated in 15.5(a).*

Subsequent recordings in monkeys trained in a delayed response task reveal further patterns of modulation since the visual and saccade components of the response are separated in time (Bruce and Goldberg, 1985; Schall, 1989a). Besides the visually reponsive cells, other units in the FEF are found that discharge in a more prolonged fashion before visually guided saccades. One population of units discharges following the appearance of the target until the presentation of the cue to move. These have been called preparatory set neurones since they are active during the period in which the monkey is preparing but not executing the movement. In other words, these units are discharging specifically during the period in which visual attention is shifting to the location of the target (e.g. Posner, 1980). A few set cells begin to discharge in anticipation of the target, but excluding this, the typical response latency is 90–95 ms, slightly longer than the latency of the visual cells. The time to reach peak activation averages 120 ms which is longer than the rise time of the visual cells. After the cue to execute the saccade is given, the activity of the set cells decays within 150 ms, before saccade initiation.

Another population of units in the FEF discharges in relation to both presentation of the stimulus and the subsequent saccade; these are referred to as visuomovement cells. Most members of this population of units display two discrete bursts associated with the target and the saccade. A subgroup of the visuomovement cells displays a sustained elevation following the presentation of the target until performance of the saccade. Some of these sustained visuomovement cells discharge throughout a delay period when the target is no longer visible (Bruce and Goldberg, 1985; Funahashi *et al.*, 1989). Actually, these two examples represent ends of a continuum, for there are many visuomovement cells in the FEF that show both the visual and saccade-related bursts as well as a degree of sustained elevation in the intervening delay period. The visuomovement neurones have receptive/movement fields that are predominantly in the contralateral hemifield. The two subgroups of visuomovement cells may be further distinguished by their visual response latency; that of the double-bursting visuomovement cells averages 65–70 ms while the response latency of the sustained visuomovement cells is slightly longer, 95–100 ms. Also the time to peak activation of the double-bursting cells is shorter (60 ms) than that of the sustained visuomovement cells (100 ms). Like their counterparts in the superior colliculus, the double-burst visuomovement cells exhibit the saccade component but not the sensory component of their response when monkeys make a saccade to auditory targets. Examples of sustained auditory-saccade cells have also been observed. These results indicate that the sensory component of these cells is not bimodal, but the saccade component is. It will be very interesting to determine whether the auditory receptive fields in FEF shift with eye position like their counterparts in the superior colliculus.

A distinct group of units in the FEF discharge in association with goal-directed saccades. This presaccadic activity is recorded only for saccades that are made by monkeys motivated by the expectation of a reward whether or not they are visually guided; it is not associated with spontaneous, self-generated saccades. This contingency accounts for why these presaccadic units were not found in the initial recordings from the FEF in untrained monkeys. The movement fields of these cells tend to be larger than the receptive fields of visual cells, but they are also largely restricted to the contralateral hemifield. The onset time of the presaccadic eye movement cells averages 120–150 ms prior to saccade initiation; the onset of the presaccadic burst in the double-burst visuomovement cells is in the same range. These presaccadic neurones comprise the output to the saccade generator since microstimulation at the sites where these cells are recorded elicits saccades with currents less than 50 μA, but microstimulation at the sites of other classes of units requires currents above 100 μA (Bruce *et al.*, 1985).

A significant proportion of the units in the FEF discharge specifically after saccades. Some of these units exhibit presaccadic activity in one direction and postsaccadic activity in the opposite direction. Such postsaccadic activity may be a corollary discharge used to register the execution of the saccade. The post-saccadic cells tend to be tuned for contralateral saccades, so they can signal the occurrence of a saccade of a specific direction.

In a go/no-go delayed response task in which a monkey must execute or withhold a saccade to a target based on a cue, a few FEF cells show specific modulation relative to the no-go cue. This modulation can be seen in visuomovement or movement cells and consists of either an elevation or suppression following the cue to withhold a saccade.

Finally, a small percentage of units in FEF is modulated by the position of the eye in the orbit. Since stimulation of the FEF never elicits saccades that bring the eyes to a particular location in the orbit, it could be argued that this population of eye position cells is largely ineffective. Another small population of units in the FEF discharge in relation to smooth pursuit. These are found mainly in the foveal representation.

Relation to Superior Colliculus

By simultaneously recording in the FEF and stimulating the superior colliculus, it is possible to determine the functional properties of corticotectal neurones through antidromic activation (Segraves and Goldberg, 1987). Half of the corticotectal cells observed in this experiment were presaccadic movement cells, and a quarter of the corticotectal cells were visually responsive with foveal receptive fields. The remainder of the corticotectal units were a heterogeneous group including some postsaccadic cells. Further evidence for a topographic projection from the FEF to the superior colliculus is provided by the fact that the lowest thresholds for antidromic activation were found at sites which represented the same saccade vector as that recorded in the FEF. This is consistent with the previously mentioned observation that paired stimulation of the FEF and superior colliculus evokes saccades of the resultant vector. The antidromic activation latency for the presaccadic cells is significantly shorter than that of the foveal visual cells. Since there is diversity in the morphology of the FEF neurones sending axons to the superior colliculus (Fries, 1984); it will be very interesting to determine the morphological correlates of these different populations of cells.

Effects of Lesions

We have already alluded to the oculomotor deficits that follow FEF ablation, a transient ipsilateral deviation of the eyes and a lack of contralaterally directed saccades (Ferrier and Yeo, 1884; Bianchi, 1895; Jacobson, 1936; Kennard, 1939; Silberpfennig, 1941; Clark and Lashley, 1947; Latto and Cowey, 1971a,b; Rizzalatti *et al.*, 1983). Following frontal cortex damage, contralaterally directed saccades are also slower and less accurate than normal (Sharpe, 1986). Interestingly, Alzheimer's disease, which is known to involve frontal lobe dysfunction, has associated eye movement deficits; saccades show increased latency, hypometria, and are reflexive (Pirozzolo and Hansch, 1981; Fletcher and Sharpe, 1986). Monkeys suffer a loss in predictive saccadic eye movements (Bruce and Borden, 1986), an inability to generate saccades to remembered locations (Deng *et al.*, 1986), and an impairment of visual pursuit (Lynch, 1987) following FEF ablation.

Deficits are also observed in other visuomotor behaviours. Following frontal cortex damage patients exhibit difficulties in making saccades on verbal command (Holmes, 1938) and in instructed visual scanning (Luria *et al.*, 1966). They are also unable to direct a saccade in the direction opposite that of a flashed target (Guitton *et al.*, 1985); this indicates that the FEF plays some role in inhibiting the more reflexive eye movements. Lesions of the FEF and surrounding cortex impair visual search in monkeys (Latto, 1978a; Collin *et al.*, 1982; but see Schiller *et al.*, 1987) and man (Teuber *et al.*, 1949; Chedru *et al.*, 1973; Karpov *et al.*, 1968); even though monkeys suffer no deficit in learning a visual search task following FEF ablation (Latto, 1978b). FEF ablation does not affect the discrimination of real world movement *vs* self-generated movement (Collin and Cowey, 1980), but it does impair visual spatial discrimination (Latto, 1986). Contralateral neglect and extinction has also been observed following unilateral frontal cortex lesions (Kennard and Ectors, 1938; Silberpfennig, 1941; Welch and Stuteville, 1958; Heilman and Valenstein, 1972; Damasio *et al.*, 1980; Crowne *et al.*, 1981; Rizzolatti *et al.*, 1983).

The results of these ablation studies indicate that subjects ultimately can still generate saccades following FEF lesions. Other experiments have demonstrated that the superior colliculus is primarily responsible for this ability. Combined, bilateral lesions of the FEF and superior colliculi result in a permanent loss of saccades (Schiller *et al.*, 1980). This work indicates that two parallel pathways are normally involved in the generation of saccades: the subcortical pathway depends on the superior colliculus, and the cortical pathway is headed by the FEF. A reasonable working hypothesis is that the subcortical pathway is responsible for reflexive, orienting saccades, and the cortical pathway is responsible for the more voluntary, visually guided saccades. This is borne out by the effect of FEF and superior colliculus lesions on saccade latency; ablation of the superior colliculus but not FEF results in a loss of the short latency, express saccades (Schiller *et al.*, 1987).

Summary

The evidence now seems incontrovertible that the FEF plays a fundamental role in generating saccadic eye movements. In clarifying this it has become apparent that saccades are generated under a number of different conditions. But it is also clear that FEF participates in directing visual attention. Surely Ferrier would be impressed by the progress that has been made in understanding the function of the FEF in generating saccadic eye movements. At the same time, would he not wonder whether the remainder of the frontal cortex from which he elicited eye movements is also involved? If this chapter had been written even just five years ago, we would have had no answer for him.

Supplementary Eye Fields

Supplementary Motor Area

During voluntary, conjugate eye movements two distinct loci of elevated cerebral blood flow can be distinguished in frontal cortex of humans (Melamed and Larsen, 1979; Orgogozo and Larsen, 1979; Fox *et al.*, 1985). The lateral locus corresponds to the FEF, and the dorsomedial locus corresponds to the supplementary motor area. The supplementary motor area is a second motor area; apparently one step above the primary motor area, it was discovered first in humans (Penfield and Welch, 1949, 1951) and later in macaque monkeys (Woolsey *et al.*, 1952). It occupies dorsomedial area 6, rostral to area 4. This cortical area has attracted considerable attention recently because of evidence that it plays a significant role in generating movements. Three lines of research have prompted recent enthusiasm about the supplementary motor area.

First, regional cerebral blood flow studies in human subjects performing a variety of movements have demonstrated that the supplementary motor area is activated during complex, volitional movements but not during sustained muscular contraction or simple, repetitive movements (Orgozo and Larsen, 1979; Roland *et al.*, 1980; but see Fox *et al.*, 1985). Moreover, blood flow in the supplementary motor area is also elevated when a subject just imagines a complex movement sequence without actually executing it (Ingvar and Philipson, 1977; Roland *et al.*, 1980).

Second, prior to the execution of a movement a 'readiness potential' can be observed in the EEG recorded over the supplementary motor area (Deecke and Kornhuber, 1978; Deecke *et al.*, 1985). This readiness potential appears when a subject plans a movement that is subsequently withheld (Libet *et al.*, 1983b). Also, the readiness potential recorded prior to pre-planned, externally cued movements is qualitatively different from that recorded prior to spontaneous movements (Libet *et al.*, 1982). Possibly the most dramatic result is that the readiness potential begins before the subject reports being aware of the intention to act (Libet *et al.*, 1983a; see also Libet, 1985).

Third, single unit recordings from the supplementary motor area in behaving monkeys have demonstrated a variety of pre-movement neuronal activity. Sensory cells responding to visual, auditory and tactile stimuli are recorded (Brinkman and Porter, 1979; Wise and Tanji, 1981; Tanji and Kurata, 1982; Okana and Tanji, 1987; Romo and Schultz, 1987; Hummelsheim *et al.*, 1988). Other units discharge in relation to limb movements (Tanji and Tanaguchi, 1978; Sakai, 1978; Brinkman and Porter, 1979; Smith, 1979; Tanji and Kurata, 1979, 1981, 1982; Tanji *et al.*, 1980; Wise and Tanji, 1981; Okano and Tanji, 1987; Romo and Schultz, 1987). Still other neurones in the supplementary motor area are neither sensory nor motor, but discharge while the movement is being prepared, or in response to different cues and instructions during a task (Sakai, 1978; Tanji *et al.*, 1980; Kurata and Tanji, 1985; Tanki and Kurata, 1985).

Effects of Electrical Stimulation

In the original explorations of the supplementary motor area through electrical stimulation (Penfield and Welch, 1949, 1951; Woolsey *et al.*, 1952), a rough somatotopic map was described; caudally the hindlimb is represented, more rostral is the forelimb and most rostral is the face. Furthermore, stimulation at the rostral end of the supplementary motor area elicited eye movements. Evidence will be reviewed that indicates that the rostral supplementary motor area may rightly be called a supplementary eye field (SEF) (Fig. 15.1(a)).

Intracortical microstimulation of the SEF evokes saccades (Mitz and Wise, 1987; Schlag and Schlag-Rey, 1987). The parameters of stimulation are comparable with those in FEF with a threshold less than 50 μA and a latency of 30–50 ms. Preliminary evidence with intracortical microstimulation indicates that Ferrier may have been correct; saccades can be elicited with low intensity stimulation at sites extending from the prearcuate FEF to the dorsomedial SEF (Mitz and Godschalk, 1987). Unlike the saccades evoked by stimulation of the superior colliculus or FEF, the direction and amplitude of the saccade elicited by stimulation of many but not all sites in SEF varies with eye position. In other words, stimulation of most sites in the SEF tends to bring the eye to a specific position in the orbit, and if the eye is already at that position, then no saccade is evoked. In further contrast to the superior colliculus and FEF, prolonged stimulation of the SEF does not result in multiple saccades, but rather the eye is moved to and then stays at the specific position (Schall and Schiller, unpublished observation).

Effects of Lesions

The effects of inactivating or ablating the SEF on eye movements have not been systematically investigated. There have been some hints of contraversive gaze disorders following SEF ablations (Laplane *et al.*, 1977).

Connectivity

The connectivity of the SEF is distinct from that of the remainder of the supplementary motor area. The SEF is interconnected with the other visuomoter cortical areas including the FEF, the IPL, the superior temporal sulcus as well as the cingulate, prefrontal, postarcuate premotor area and the supplementary motor area (Jeffers *et al.*, 1987; Huerta and Kaas, 1988). The SEF also projects to the striatum (Jurgens, 1984; Selemon and Goldman-Rakic, 1985; Arikuni and Kubota, 1986) and is reciprocally connected with the ventroanterior, ventrolateral, nucleus X, mediodorsal and intralaminar nuclei (Trojanowski and Jacobson, 1974; Kievit and Kuypers, 1977; Miyata and Sasaki, 1984; Ilinsky *et al.*, 1985; Wiesendanger and Wiesendanger, 1985; Schlag-Rey *et al.*, 1987). Interestingly, the thalamic nuclei providing the major input to the SEF themselves receive input from the dentate and not the fastigial nucleus (Wiesendanger and Wiesendanger, 1985) in contrast to the caudal supplementary motor area which receives thalamic input from regions dominated by pallidal afferents (Schell and Strick, 1984). Also, there is preliminary evidence that the connectivity between the intralaminar nuclei and SEF is highly organized (Schlag-Rey *et al.*, 1987). SEF regions from which eye-position dependent saccades are elicited are connected with the caudolateral portion of the intralaminar nuclei from which eye-position dependent saccades are also evoked. In contrast, SEF sites from which fixed vector saccades are evoked are connected with the rostromedial portion of the intralaminar nuclei.

The superior colliculus receives afferents from the rostral supplementary motor area, and it appears that the SEF synapses deeper in the superior colliculus than the FEF (Leichnetz *et al.*, 1981; Fries, 1984; Huerta and Kaas, 1988). Projections have also been observed from the SEF to the brainstem oculomotor regions such as the NRTP, mesencephalic reticular formation and pontine reticular formation as well as certain pontine nuclei although these projections do not appear to be as dense as those from the FEF (Wiesendanger *et al.*, 1979; Leichnetz *et al.*, 1984a,b; Schnyder *et al.*, 1985; Schlag-Rey *et al.*, 1987; Huerta and Kaas, 1988; Shook *et al.*, 1988).

Physiological Properties

A variety of neuronal responses associated with visually guided saccades have been observed in the SEF (Fig. 15.10) (Brinkman and Porter, 1979; Schlag and Schlag-Rey, 1987; Schall, 1989b). Sensory cells responding to visual and/or auditory stimuli have been reported in the SEF. The latency of response averages around 90 ms, and the time to reach peak activation is 85 ms. While the response latency is not significantly different from that observed in the FEF, the rise time is longer. This reflects the fact that the visually responsive cells in the SEF respond in a less robust and consistent fashion than their counterparts in the FEF. This difference is probably due to the fact that the visual input to the FEF is stronger than that to the SEF. The visual cells have large receptive fields that tend to lie in the contralateral hemifield. The receptive fields of a large proportion of these units include the fovea or are restricted to central retina. No topographic organization is observed, however. Specific tests of whether the visual responses are enhanced if the stimulus is or is not the target of a saccade and/or of an attention shift have not been performed, but it seems likely that the enhancement will be present.

Neurones that are specifically active during the period between the presentation of the target and the subsequent cue to execute a saccade are also observed in the SEF (Schall, 1989b). Such a pattern of modulation has also been observed in the caudate nucleus (Hikosaka *et al.*, 1989c), and supplementary motor area (Tanji *et al.*, 1980; Tanji and Kurata, 1985), primary motor cortex (Tanji and Evarts, 1976), the postarcuate premotor region that lies lateral to SMA (Godschalk and Lemon, 1983; Weinrich and Wise, 1982; Weinrich *et al.*, 1984; Wise and Mauritz, 1985) and prefrontal cortex (Kubota and Funahashi, 1982). Because they discharge during the period in which the movement is being prepared, this group of neurones has been called preparatory set cells (see Evarts *et al.*, 1984). It is possible that these preparatory set cells also play an important role in saccade initiation since their offset is correlated with the beginning of the long-lead presaccadic activation. The visual response latency of these units is around 100 ms, and the time to peak activation is over 150 ms. After the cue is presented, the activity in this population of units decays after approximately 135 ms, before the saccade is initiated (the typical saccade latency in these tasks averages 200 ms). The preparatory set cells also responded preferentially in association with contralateral targets.

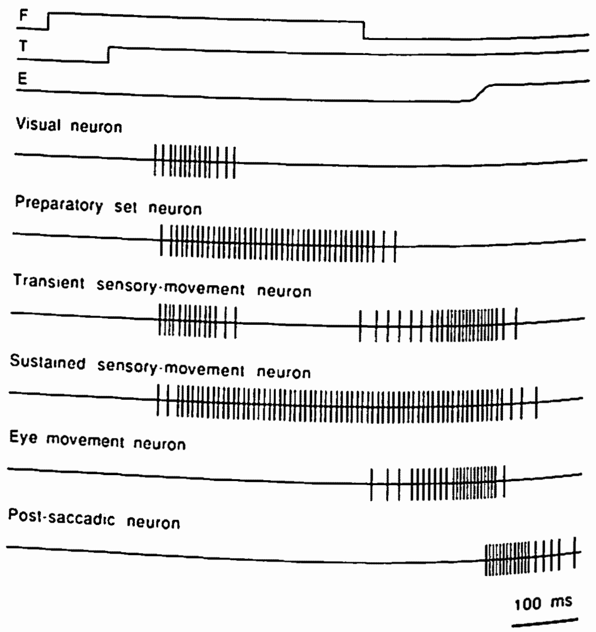

Fig. 15.10 *Neuronal activity in the supplementary eye field associated with saccades. Same task as that illustrated in 15.5(a).*

Other neurones in the SEF display a more prolonged discharge, starting after the appearance of a target and lasting until a saccade is executed (Schall, 1989b). These may be referred to as visuomovement cells, but they differ in certain respects from their counterparts in the FEF, IPL and superior colliculus. The visuomovement cells in these latter named structures display distinct bursts associated with the presentation of the target and the execution of the saccade, even if there may be a measure of elevation between the bursts. In distinction, the visuomovement cells in the SEF do not show discrete bursts but instead a sustained elevation of activity. In this respect these units resemble the quasi-visual cells of the superior colliculus and IPL; however, they have not been tested in monkeys making saccades to remembered locations. The visuomovement cells of the SEF display movement fields that are predominantly in the contralateral hemifield. The average latency of response to the visual stimulus is 116 ms; which is significantly longer than the response latency of the sensory or preparatory set cells. Also, a number of the visuomovement cells become active prior to the appearance of the target when the monkey could anticipate it. The time to peak activation, 220 ms, is also longer than that of sensory or set cells. These visuomovement cells are inactive within 95 ms following saccade initiation. Most of the visuomovement cells in the SEF also respond to auditory stimuli that are targets for saccades; however, visual and auditory specific examples are also encountered. These sensory-movement cells were recorded in monkeys performing a go/no-go task in which they either executed or withheld a saccade based on a cue (Schall, 1989b). Following the cue to withhold the saccade, the activity of most visuomovement cells decayed sooner and more rapidly than it would have otherwise. A few of the sensory-movement cells, however, gave a brief discharge following the no-go cue.

Another unusual type of neuronal modulation observed in the SEF is seen in cells that have a moderate level of spontaneous activity. When the target appears, the activity of these cells is suppressed until the saccade at which point there is a rebound burst discharge. The latency of the pause averages 12 ms after presentation of the target, which indicates that these cells can express some anticipation. The burst begins around 40 ms before the saccade. These cells resemble the pause-rebound cells that are observed in the central intralaminar thalamus (Schlag-Rey and Schlag, 1984) and pulvinar (Robinson *et al.*, 1986). It has been speculated that these units provide feedback to visual processing centres, giving a signal to inhibit visual neurones when a saccade is disrupting the image.

Neurones that discharge before saccades have also been observed in the SEF (Brinkman and Porter, 1979; Schlag and Schlag-Rey, 1987; Schall, 1989b). Like their counterparts in the FEF and IPL and the visually triggered saccade cells of the superior colliculus, these units are not active for spontaneous saccades in untrained or unmotivated monkeys. These units begin to discharge on average 144 ms before visually guided saccades and earlier before self-generated saccades. This onset time is not significantly different from the presaccadic burst seen in the FEF or lateral interparietal area. They have broad movement fields that emphasize the contralateral hemifield, but the contralateral tendency is less pronounced in the SEF than in the FEF. Units that are active specifically postsaccadically are not as common in the SEF as they are in the FEF. It is not clear why this should be so; perhaps the SEF does not make use of a corollary discharge in its function.

Other neurones recorded in the SEF display a tonic discharge that varies systematically with the position of the eye in the orbit (Schlag and Schlag-Rey, 1985; Schall, 1989b). Unfortunately, this population of units has not been investigated in detail; such a study may reveal one of the fundamental differences between the frontal and the SEF, that the FEF encodes saccades in a retinotopic coordinate frame while the SEF encode saccades in a spatial or head-centred coordinate frame. In the go/no-go task alluded to above, a number of neurones were found in the SEF that were specifically activated following the no-go cue, when the monkey is required to withhold a saccade (Schall, 1989b). The average latency is around 120 ms and the modulation lasts at least 250 ms. Similar no-go specific modulation has also been observed in the supplementary motor area (Tanji and Kurata, 1985) and prefrontal cortex (Sasaki and Gemba, 1986; Watanabe, 1986).

Comparison with Frontal Eye Fields

The existence of two frontal cortical fields involved in saccade generation raises questions about the role of each area. First, are they in a hierarchical or parallel relationship relative to one another? Second, what functional properties distinguish the two areas, and what unique contributions does each make? Third, are both regions necessary and sufficient for saccade generation? Fourth, how does each area relate to LIP and other visuomotor regions of the brain?

Several pieces of evidence indicate that the SEF and FEF operate in parallel during the generation of motivated, visually guided saccades. First, the visual response latencies of cells in the two areas are not markedly different; although the FEF reaches its peak activation sooner than the SEF. Second, the latency of saccades elicited by microstimulation are not significantly different. Third, both frontal cortical areas share reciprocal projections as well as direct projections to brainstem oculomotor centres.

The presence of the medial SEF and lateral FEF for the oculomotor system is paralleled by the medial supplementary motor area and lateral postarcuate premotor area for the skeletomotor system. The idea has been proposed that the lateral premotor area is mainly involved in externally triggered movements while the supplementary motor area is responsible for self-generated movements (see Goldberg, 1985). It may be useful to think about SEF and FEF according to a similar dichotomy. Unfortunately, neuronal recordings in both the supplementary motor area and the lateral premotor area in monkeys performing both self-generated and externally-triggered movements indicate that the differentiation by this criterion is perhaps partial but not decisive (Okano and Tanji, 1987; Romo and Schultz, 1987; Kurata and Wise, 1988).

This distinction between self-generated and externally-cued movements introduces one purported difference between the supplementary and FEF, and that is that cells in the SEF are active before 'spontaneous' saccades but cells in the FEF are not. The evidence that led to this interpretation (Schlag and Schlag-Rey, 1987) was derived from recordings in monkeys that were rewarded for making saccades to stimuli that appeared at unanticipated locations and times. In this situation the saccades are derived from a more motivated state than are the saccades that are made by an untrained monkey. In other recordings from the FEF and SEF (Bruce and Goldberg, 1985; Schall, 1980a,b) monkeys performed more structured tasks that required a single goal-directed saccade to earn a reward. Saccades made in the intertrial interval were never rewarded and so would be less motivated. In this situation presaccadic activity was associated with the task-related saccade but not the eye movements in the intertrial interval. Taken together, then, these results suggest that neurones in both SEF and FEF discharge specifically before visually guided, goal-directed saccades executed by motivated subjects. This issue, however, warrants further examination.

The most pronounced functional difference between FEF and SEF observed so far appears to be that an eye position signal is more pronounced in the SEF. Presently, this is indicated more by the results of microstimulation than by single unit recordings. Single units whose activity is related to eye position in the orbit have been recorded in FEF too (Bizzi, 1968; Bizzi and Schiller, 1970; Bruce and Goldberg, 1985). Further experimental work is underway to determine whether a greater proportion of units in SEF are tuned for the position of the eye in the orbit (Tehovnik *et al.*, 1989).

The fact that bilateral ablation of both the FEF and superior colliculi results in a complete loss of saccades (Schiller *et al.*, 1980) indicates that the SEF alone is not sufficient to generate saccades. Still, it would be interesting to repeat the ablation of both the FEF and superior colliculus to determine whether stimulation of the SEF can evoke eye movements. Preliminary work (Schall *et al.*, 1987) indicates that saccades can be elicited by microstimulation of the SEF following either FEF or superior colliculus ablation.

Prefrontal Cortex

Preliminary evidence indicates that a region rostral to the FEF, around the principal sulcus is active during the generation of visually guided saccades. Units recorded from the prefrontal cortex in the banks of the principal sulcus (area 46 or FD) are visually responsive (Suzuki and Azumi, 1977, 1983; Mikami *et al.*, 1982b; Boch and Goldberg, 1989). The latency of the response averages 80–120 ms. The receptive fields of the cells tend to emphasize the contralateral hemifield, and receptive fields that include the fovea are smaller than their peripheral counterparts. Some but not all visually responsive neurones in this area give a spatially specific enhanced response if the stimulus is the target for a saccade (Boch and Goldberg, 1989). There is also some evidence that the visual responses are enhanced when the monkey directs attention without making a saccade to the peripheral target (Mikami *et al.*, 1982a). In the delayed saccade task certain units exhibit two bursts of activity, one following the presentation of the target and the second immediately prior to the saccade; the presaccadic burst occurs up to 160 ms before the saccade with an average of around 50 ms (Boch and Goldberg, 1989). Other neurones are specifically active after a visually guided saccade. While these results indicate some role of prefrontal cortex in directing attention and generating saccades, low intensity microstimulation of this cortical area does not elicit saccades. During a delayed-response visual tracking task, many neurones in prefrontal cortex are active following the brief presentation of the target until the cue to move, throughout the delay (up to 6000 ms) until the saccade (Funahashi *et al.*, 1989). Similar visuomovement activity has also been recorded dorsal to the principal sulcus (Joseph and Barone, 1987).

Discussion

The data reviewed in this chapter are summarized in Tables 15.1, 15.2 and 15.3. Fig. 15.11 indicates the complex connectivity between the various areas that are involved in generating saccades. Table 15.1 reviews the results of microstimulation of the various brain regions on evoking saccadic eye movements. Two points are evident. First, the effects of microstimulation of many of the regions are not known. Second, based on the threshold and latency it is possible to define three levels distant from the ocular motor nuclei. The first, motor tier consists of the brainstem motor neurones and supranuclear regions. The second, visuomotor tier consits of the superior colliculus, the intralaminar thalamic nuclei, the lateral interparietal area, and the frontal and supplementary eye fields. The third, visual tier is represented by striate cortex and probably the extrastriate visual cortical areas.

Table 15.2 summarizes the visual response latencies in the different brain regions. The increase in latency, of course, reflects how distant a region is from the retina and reveals the progression of processing culminating in a saccade. Also the regions in which the visual response is enhanced when the stimulus is a target for a saccade are evident. The enhancement is observed in subcortical as well as cortical structures. In some areas the enhancement is observed only when a saccade is made, while in others the visual response is enhanced if selective attention is shifted without an overt eye movement. The source of the enhancement is not presently known, but it is likely to be derived from ascending projections from the mesencephalic reticular formation.

Table 15.3 presents a summary of the time relative to a saccade at which the movement cells in the different regions become active. In comparing Table 15.3 with Tables 15.1 and 15.2, it is clear that presaccadic activity is found only in those areas from which saccades could be elicited with less than 50 μA. The position of the eye in the orbit is signalled in a number of areas. This is indicated by variation in the saccade elicited by microstimulation as noted in Table 15.1, by modulation of the visual response according to the position of the eye in the orbit as noted in Table 15.2, and by modulation of the eye movement activity noted in Table 15.3. The signal of the position of the eye in the orbit can be derived from two sources: a continually updated internal representation and proprioceptive afferents. This signal appears to originate in the brainstem and is passed up to cerebellum as well as to certain cortical areas via the intralaminar and lateral dorsal thalamic nuclei.

While such summaries demonstrate the remarkable progress that has been made toward understanding the neural basis of saccadic eye movements, a number of very interesting questions remain open. For example, readers of this volume will be familiar with the remarkable functional transformations that occur in the geniculostriate projection; orientation and direction selectivity appear; binocular interactions are used to provide an analysis of

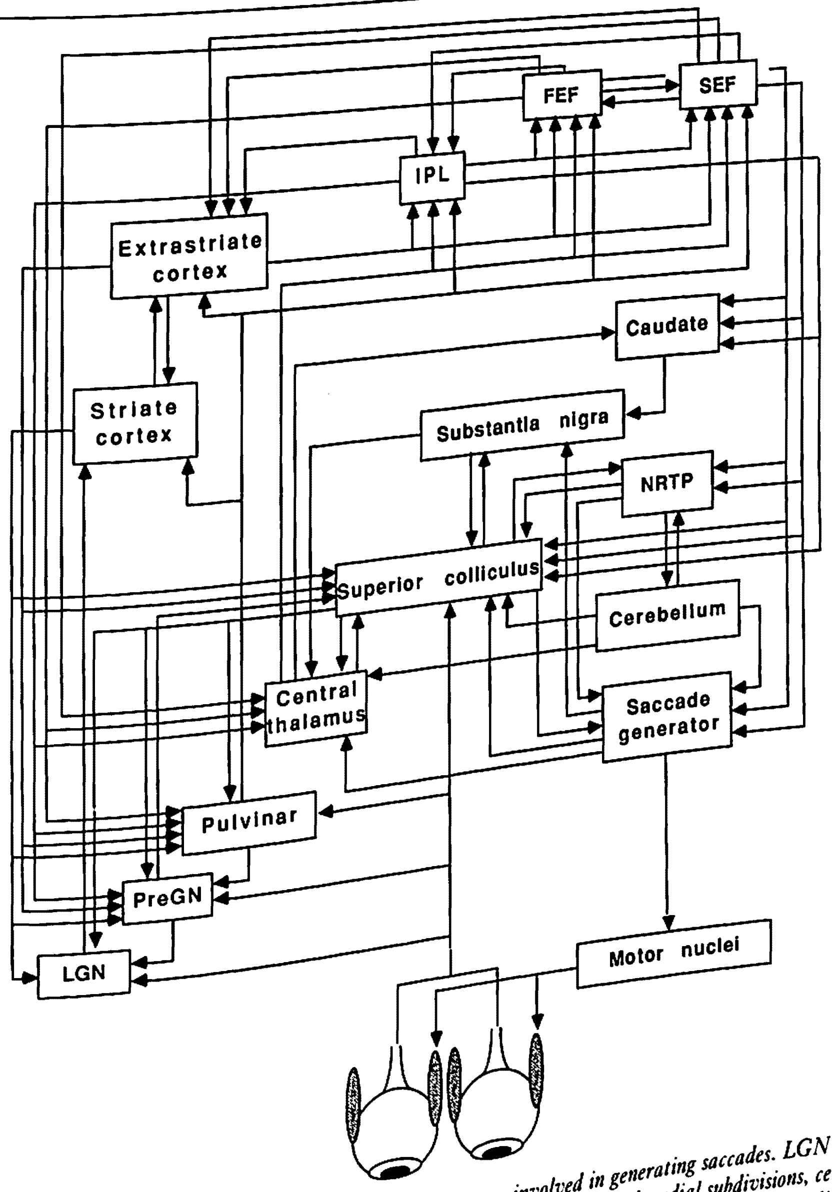

Fig. 15.11 *Schematic diagram of the connections between the various regions involved in generating saccades. LGN – dorsal lateral geniculate nucleus, preGN – pregeniculate nucleus, pulvinar – represents inferior, lateral and medial subdivisions, central thalamus – represents the centrolateral and paracentral intralaminar nuclei as well as the lateral dorsal, ventrolateral and mediodorsal nuclei, superior colliculus – represents both upper and lower subdivisions, striate cortex – area 17 or V1, extrastriate cortex – represents V3A and V4, IPL – inferior parietal lobule, FEF – frontal eye field, SEF – supplementary eye field, caudate – represents head of caudate nucleus, substantia nigra – represents pars reticulata portion, NRTP – nucleus reticularis tegmenti pontis, cerebellum – represents lobule VII of the vermis and the flocculus, saccade generator – represents the mesencephalic and pontine reticular formation, the nucleus prepositus hypoglossi and the ocular motor nuclei.*

depth, and so on. Based on the present data, however, it is not possible to conclude what comparable transformations occur in the visuomotor cortical areas. This implies that the most interesting experiments have yet to be done.

Another question, which is fundamental to all movement systems, is what is the source of the command to move (see for example Kupfermann and Weiss, 1978; Llinas and Bunge, 1978; Davis and Kovac, 1985; Eaton and DiDomenico, 1985). Neurones have been described in a number of regions that discharge before saccades (Table 15.3); the activation of these neurones serves to ultimately drive the eyes. It appears that in addition to the superior colliculus, three areas of the cerebral cortex are involved in generating saccades. Traditionally, movement has been thought of as requiring three levels or subsystems responsible for generation, initiation and coordination. The current view, however, is evolving away from such a hierarchical scheme. It is presently thought that neuronal motor systems consist of a collection of reciprocal loops in which individual neurones may serve in each of the three functions. This is especially evident in the saccade generating system; indeed as illustrated in Fig. 15.11, the phrase parallel distributed network is nowhere appropriate if not here. Now the challenge is to delineate the manner in which the various loops and areas are coordinated and what unique contribution each makes.

Another exciting area of current research is at the sensory-motor interface. In the 'higher' visuomotor centres there are a number of neurones which are neither sensory nor motor. Moreover, many of the sensory cells in these areas do not require the actual appearance of the stimulus to be activated, and many of the motor cells discharge for only particular movements. Continued progress in understanding these areas requires confronting a number of issues. For example, in what way are these non-sensory/non-motor cells most appropriately characterized? What transformations occur in the different visuomotor cortical areas? One problem of particular interest which has not received a great deal of attention is the process by which the target for a saccade is selected. Also, once a target is identified in retinotopic coordinates, where and how is the eye position signal combined to provide a representation of the target in space?

Table 15.1 *Summary of microstimulation results.*

Structure	Threshold (μA)	Latency (ms)	Properties 1	2	3
Motor nuclei	< 10	< 10	Y	N	N
Supranuclear	< 10	10	Y	N	N
Cerebellum	< 10	10	N	Y	Y
NRTP	?	?	?	?	?
Superior colliculus	< 10	30	N	Y	N
Substantia nigra	?	?	?	?	?
Caudate nucleus	?	?	?	?	?
Thalamus					
Pulvinar	?	?	?	?	?
Intralaminar	30	40	N	Y	Y
Cortex					
V1	100	70	N	Y	N
V4	?	?	?	?	?
IPL					
7a	80	50	N	Y	N
LIP	< 50	40	N	Y	N
MST	80	50	N	N	Y
FEF	< 50	35	N	Y	N
SEF	< 50	40	N	Y	Y
Prefrontal	None	–	–	–	–

The different columns labelled 'Properties' indicate the particular attributes of the eye movements evoked by stimulation of the structure according to the following code: 1) amplitude increases with frequency and duration of stimulation, 2) evoked saccade does not change with eye position, 3) evoked saccade does change with eye position. Entries are Y for a positive result, N for a negative result, ? for an unknown result and – for the cases in which no results are obtained.

Table 15.2 *Summary of visual responses.*

Structure	Latency (ms)	Properties 1	2	3	4	5	6
Motor nuclei	None	–	–	–	–	–	–
Supranuclear	None	–	–	–	–	–	–
NRTP	70	Y	?	Y	N	N	?
Cerebellum	?	?	?	?	?	?	?
Superior colliculus							
upper	45	Y	N	Y	N	Y	N
lower	55	N	N	N	N	Y	N
Substantia nigra	120	Y	N	Y	N	Y	N
Caudate nucleus	150	Y	?	Y	N	Y	?
Thalamus							
LGN	30	N	N	N	N	Y	N
PreGN	30	N	N	N	N	Y	?
Pulvinar							
Inf/lat	65	Y	N	N	Y	Y	N
Medial	85	Y	Y	Y	N	Y	N
Intralaminar	100	Y	?	?	?	Y	N
Cortex							
V1	30	Y	Y	N	Y	Y	N
V2	40	Y	?	N	Y	Y	?
V3A	60	?	?	?	?	?	Y
V4	80	Y	Y	Y	N	Y	N
IPL							
7a	90	Y	Y	Y	N	Y	Y
LIP	75	?	?	?	?	?	Y
MST	80	?	?	?	?	?	?
FEF	80	Y	N	Y	N	Y	N
SEF	90	?	?	?	?	?	?
Prefrontal	200	Y	Y	Y	N	Y	?

Conventions are as in Table 15.1. The properties are as follows: 1) saccade-related enhancement, 2) attention-related enhancement, 3) spatially restricted enhancement, 4) not spatially restricted enhancement, 5) no saccade-related enhancement, 6) visual response varies with gaze angle. In some structures different cells may exhibited different properties.

Table 15.3 *Summary of saccade-related responses.*

Structure	Onset (ms)	Properties 1	2	3	4
Motor nuclei	6	Y	N	Y	N
Supranuclear					
burst	10, > 100	Y	N	N	Y
omnipause	10	Y	N	N	Y
tonic	–	Y	N	Y	N
NRTP	30	Y	N	Y	Y
Cerebellum					
vermis					
mossy fibre	7160	Y	N	Y	Y
Purkinje cell	< 1, + 40	Y	N	Y	Y
flocculus					
mossy fibre	7113	Y	N	Y	Y
Purkinje cell	6	Y	N	Y	Y
Superior colliculus					
upper	none	–	–	–	–
lower	20–200	Y	Y	N	Y
Substantia nigra	20	N	Y	?	Y
Caudate nucleus	100–200	N	Y	?	Y
Thalamus					
LGN	None	–	–	–	–
PreGN	None	–	–	–	–
Pulvinar					
Inf/Med	44, + 65	Y	Y	N	Y
Lat	None	–	–	–	–
Intralaminar	120	?	?	Y	Y
Cortex					
V1	None	–	–	–	–
V3A	None	–	–	–	–
V4	None	–	–	–	–
IPL					
7a	None	–	–	–	–
LIP	120	N	Y	Y	?
FEF	130, + 40	N	Y	N	Y
SEF	140, + 20	N	Y	?	?
Prefrontal	None	–	–	–	–

Conventions as in Table 15.1. The properties are as follows: 1) obligatory, i.e. discharges for every saccade including the fast phases of nystagmus, 2) contingent, i.e. discharges only for visually guided or other goal-directed saccades made by a motivated monkey, 3) activity varies with eye position, 4) activity does not vary with eye position. In some structures different cells may exhibit different properties. Also, the onset times of different subpopulations of cells are separated by a comma; positive values indicate postsaccadic onset.

Conclusion

The act of making a saccade is the end product of a number of subsidiary processes. The evidence reviewed suggests that different parts of the brain are responsible for the different processes, but it is not possible at present to state what the precise contribution of each area is. Indeed, the further a region lies from the sensory and motor apparatus of the eye, the more speculative statements about its role become. It seems reasonable to suggest that all of the areas that exhibit visual activity – superior colliculus, substantia nigra, caudate nucleus, pulvinar, central thalamus, visual cortex, inferior parietal lobule and frontal and supplementary eye fields – may be involved in noticing and identifying a target for a saccade. It appears that the superior colliculus acts as more of an event detector and does not analyze the features of the visual stimuli; thus, saccades are elicited in a more rapid and automatic fashion as a result of superior colliculus function. The complexity of the striate and extrastriate visual cortical areas seems to be designed for a more elaborate analysis of the visual world. These areas may operate to discriminate a particular stimulus among a variety of others based on differences in, for example, size, shape, colour or motion. These stages of analyses are also filtered by attention. By the time the visual signal reaches the frontal cortex, any stimulus specificity is lost, but presumably the signal reflects the processing that has occurred.

Prior to making a saccade, fixation of the present target must be released. The areas that appear to be responsible for the decision to release fixation and make a saccade include the frontal and supplementary eye field, the caudate nucleus and the substantia nigra. It is via this pathway that a signal appears to be generated that releases inhibition on the superior colliculus, allowing a saccade to be performed. The execution of the saccade begins when the omnipause neurones in the brainstem reticular formation are inhibited, thus releasing their inhibition on the burst neurones which drive the ocular motoneurones.

To make an accurate saccade, the retinal coordinates of the target must be combined with the position of the eye in the orbit. It is not presently clear where and how this is accomplished. It appears that the motor error, the difference between where the eye is looking and where it is supposed to be looking, is represented at least at the level of the superior colliculus. The source of the retinal error is clear; the visual input reviewed above provides this. The source of the eye position signal may be localized to the nucleus prepositus hypoglossi, but proprioceptive information should not be overlooked. This eye position signal is widely distributed in the saccade network, having been recorded in the nucleus reticularis tegmenti pontis, cerebellum, central thalamic nuclei, extrastriate area V3A, inferior parietal lobe and supplementary eye fields.

The spatial code of the motor error must be converted into a temporal code that is required to move the eye the desired amount. This conversion appears to be accomplished in the mesencephalic reticular formation, paramedian pontine reticular formation and medullary reticular formation. These sites are responsible for generating the appropriate activity in the ocular motor neurones that results in the desired eye movement.

While the foregoing summary seems to adequately describe the current view of the mechanisms underlying saccadic eye movements, work continues. Undoubtedly our ideas will have to be modified as more data become available for consideration. Nevertheless, for now we must be impressed with the progress that has been made since Laurentius offered his explanation.

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Glossary

This glossary is provided to explain to the reader, the terminology used by different contributors. It is not an exhaustive or definitive listing. The number at the end of an entry refers to the chapter from which the definition has been extracted. Multiple entries for a single term indicate small but often critically different uses of that term in different chapters.

Accessory optic system (AOS)
In most mammals this consists of a bilateral network of three terminal nuclei located in the anterior midbrain near the mesodiencephalic border. The currently accepted nomenclature used to identify the sub-nuclei was originally introduced by Hayhow (1959) and is based upon the relative position of each nucleus within the midbrain: the **medial terminal nucleus (MTN)** is situated at the basal surface of the midbrain ventral to the red nucleus and slightly rostral to the emergence of the third cranial nerve; the **lateral terminal nucleus (LTN)** is found posterior and dorsal to the MTN, occupying a position ventro-medial to the medial geniculate body along the lateral surface of the brain; the **dorsal terminal nucleus (DTN)** is located at the anterior-lateral edge of the superior colliculus in close proximity to dorso-lateral pretectum. (5)

Acetylcholine (ACh)
A transmitter used by several non-retinal afferents to the LGN. ACh has both excitatory and inhibitory influences on LGN cells. (3)
Neuro-transmitter which, in the cortex, appears to modulate the level of excitability. (9)

Acetylcholinesterase (AChE)
Enzyme responsible for the hydrolysis of ACh. (9)

Activity profile
More general term than sensitivity profile since it includes both excitatory and inhibitory effects. To disclose the latter, the use of two stimuli is generally required. These profiles can be measured along the length and width of the RF. (8)

Afferent and efferent motion perception
Motion perception with eyes stationary and during pursuit respectively. (8)

γ-Aminobutyric acid
See GABA

AMLS
Anterior medial lateral suprasylvian visual area of cats. (3) (13)

Analogous
Used in the context of evolution, analogous refers to structures that have a common function. (3)

Anisotropic mapping
Representation of the visual field in which the magnification factor is different in different directions. (11)

Apical dendrite
Extended receiving process arising from the apex of a cortical pyramidal cell. (9)

Architectonics
The study of the systematic design or topography of neural areas. (9)

Area 17
Striate cortex. (3) Synonym for primary visual cortex (or V1). (7) (8) (9) (10) (11)

Area 18
In non-primates, the secondary visual area (also called V2) immediately bordering area 17. In primates, Brodmann's cytoarchitectonic area 18 includes several visual areas, but the term is sometimes used to refer specifically to V2. (7) (8) (11)

Area centralis
Central area of retina in animals such as cats. The region contains the greatest density of ganglion cells. (2)

***l*-Aspartate**
Laevo-rotatory form of the salt of aspartic acid, uptake of which is assessed with the *d*-form of the salt, which is not metabolized. (9)

Attentive fixation
Active behavioural state of visual fixation of gaze that is differentiated from others, e.g. spontaneous fixations, by the expectation of a behaviourally relevant change in the foveated target. (14)

Axo-axonal contacts
Synapses made by the axon terminals of one neurone with the axon of another. (9)

Axonal collaterals
Locally distributed branches arising from the axons of cortical neurones. (9)