39

The Control of Gaze

In preceding chapters we learned about the motor systems that control the movements of the body in space. In this and the next two chapters we consider the motor systems concerned with gaze, balance, and posture. As we explore the world around us, these motor systems act to stabilize our body, particularly our eyes. In examining these motor systems we shall be concerned with how these systems have resolved three biological challenges to knowing where we are in space: How do we visually explore our environment quickly and efficiently? How do we compensate for planned and unplanned movements of the head? How do we stay upright?

The gaze system stabilizes the image of an object on the retina when the object moves in the world or the head moves and keeps the eyes still when the image remains stationary. It has two components: the

oculomotor system and the head-movement system. The oculomotor system moves the eyes in the orbits; the head-movement system moves the eye sockets.

In this chapter we describe the oculomotor system and how visual information guides eye movements. It is one of the simplest motor systems, requiring the coordination of only the 12 muscles that move the two eyes. In humans and primates the main job of the oculomotor system is to control the position of the fovea, the central, most sensitive part of the retina. The fovea is less than 1 mm in diameter and covers a tiny fraction of the visual field. When we want to examine an object, we must move its image onto the fovea.

Six Neuronal Control Systems Keep the Eyes on Target

Hermann Helmholtz and other 19th-century psychophysicists who first studied visual perception systematically were particularly interested in eye movements. They appreciated that an analysis of eye movements was essential for understanding visual perception, but they did not realize that there is more than one kind of eye movement. In 1890 Edwin Landott discovered a second type of eye movement. When reading, the eyes do not move smoothly along a line of text but make fast, intermittent movements—saccades—each followed by a short pause (see Chapter 29).

By 1902 Raymond Dodge was able to outline five distinct types of eye movement that direct the fovea to a visual target and keep it there. All of these eye movements share an effector pathway originating in the three bilateral groups of oculomotor neurons in the brain stem.

- Saccadic eye movements shift the fovea rapidly to a new visual target.
- Smooth-pursuit movements keep the image of a moving target on the fovea.
- Vergence movements move the eyes in opposite directions so that the image is positioned on both foveae.
- Vestibulo-ocular reflexes hold images still on the retina during brief head movements.
- Optokinetic movements hold images stationary during sustained head rotation or translation.

A sixth system, the fixation system, holds the eye stationary during intent gaze when the head is not moving. This requires active suppression of eye movement. The optokinetic and vestibular systems are discussed in Chapter 40; we consider the remaining four systems here.

An Active Fixation System Keeps the Fovea on a Stationary Target

Vision is most accurate when the eyes are still. The gaze system actively prevents the eyes from moving when we examine an object of interest. It is not as active in suppressing movement when we are doing something that does not require vision, such as mental arithmetic. Patients with disorders of the fixation system—for example, some individuals with congenital nystagmus—have poor vision not because their visual acuity is deficient but because they cannot hold their eyes still enough for the visual system to work correctly.

The Saccadic System Points the Fovea Toward Objects of Interest

Our eyes explore the world in a series of very quick saccades that move the fovea from one fixation point to another (Figure 39–1). Saccades allow us to scan the environment quickly and to read. They are highly stereotyped; they have a standard waveform with a single smooth increase and decrease of eye velocity. They are also extremely fast, occurring within a fraction of a second at angular speeds up to 900 degrees per second (Figure 39–2A). The velocity of a saccade is determined by only the distance of the target from the fovea. We can change the amplitude and direction of saccades voluntarily but not their speed.

Ordinarily there is no time for visual feedback to modify the course of a saccade; corrections to the direction of movement are made in successive saccades. Only fatigue, drugs, or pathological states can slow saccades. Accurate saccades can be made not only to visual targets but also to sounds, tactile stimuli, memories of locations in space, and even verbal commands ("look left").

The Smooth-Pursuit System Keeps Moving Targets on the Fovea

The smooth-pursuit system holds the image of a moving target on the fovea by calculating how fast the target is moving and moving the eyes at the same speed. Smooth-pursuit movements have a maximum angular velocity of approximately 100 degrees per second, much

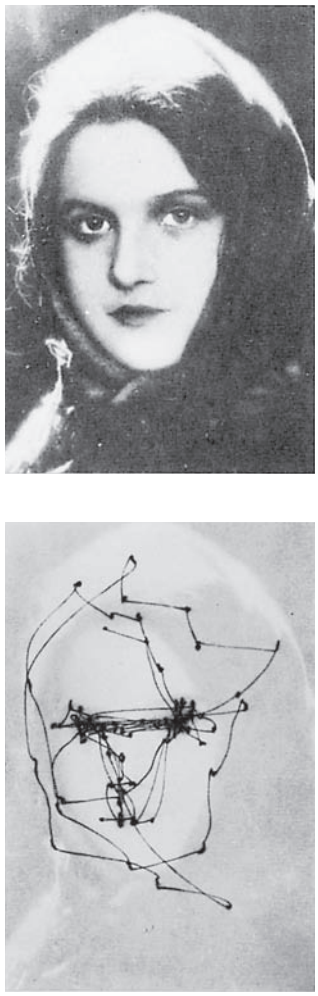


Figure 39–1 Eye movements track the outline of an object of attention. An observer looks at a picture of a woman for 1 minute. The resulting eye positions are then superimposed on the picture. As shown here, the observer concentrated on certain features of the face, lingering over the woman's eyes and mouth (*fixations*) and spending less time over intermediate positions. The rapid movements between fixation points are *saccades.* (Reproduced, with permission, from Yarbus 1967.)

slower than saccades. Drugs, fatigue, alcohol, and even distraction degrade the quality of these movements.

Smooth pursuit and saccades have very different central control systems. This is best seen when a target jumps away from the center of gaze and then slowly moves back toward it. A smooth-pursuit movement is initiated first because the smooth-pursuit system has a shorter latency and responds to target motion on the peripheral retina as well as on the fovea. As the target moves back toward the center of gaze, the eye briefly moves away from the target before the saccade is initiated (Figure 39–2B). The subsequent saccade then brings the eye to the target.

The Vergence System Aligns the Eyes to Look at Targets at Different Depths

The smooth-pursuit and saccade systems produce conjugate eye movements: Both eyes move in the same direction and at the same speed. In contrast, the vergence system produces disconjugate movements of the eyes. When we look at an object that is close to us, our eyes rotate toward each other, or *converge;* when we look at an object that is farther away, they rotate away from each other, or *diverge* (Figure 39–3). These disconjugate movements ensure that the image of the object falls on the foveae of both retinas. Whereas the visual system uses slight differences in left and right retinal positions, or *retinal disparity,* to create a sense of depth, the vergence system drives disconjugate movements to eliminate retinal disparity at the fovea.

Vergence is a function of the horizontal rectus muscles only. Near-field viewing is accomplished by simultaneously increasing the tone of the medial recti muscles and decreasing the tone of the lateral recti muscles. Distance viewing is accomplished by reducing medial-rectus tone and increasing lateral-rectus tone. Accommodation and vergence are controlled by midbrain neurons in the region of the oculomotor nucleus. Neurons in this region discharge during vergence, accommodation, or both.

At any given time the entire visual field is not in focus on the retina. When we look at something close by, distant objects are blurred. When we look at something far away, near objects are blurred. When we wish to focus on an object in a closer plane in the visual field, the oculomotor system contracts the ciliary muscle, thereby changing the radius of curvature of the lens. This process is called *accommodation.* In older individuals accommodation declines owing to increased rigidity of the lens; reading glasses are then needed to focus images at short distances.

Accommodation and vergence are linked. Accommodation is elicited by the blurring of an image, and whenever accommodation occurs the eyes also converge. Conversely, retinal disparity induces vergence, and whenever the eyes converge, accommodation also takes place. At the same time, the pupils transiently constrict to increase the depth of field of the focus. The linked phenomena of accommodation, vergence, and pupillary constriction comprise the *near response.*

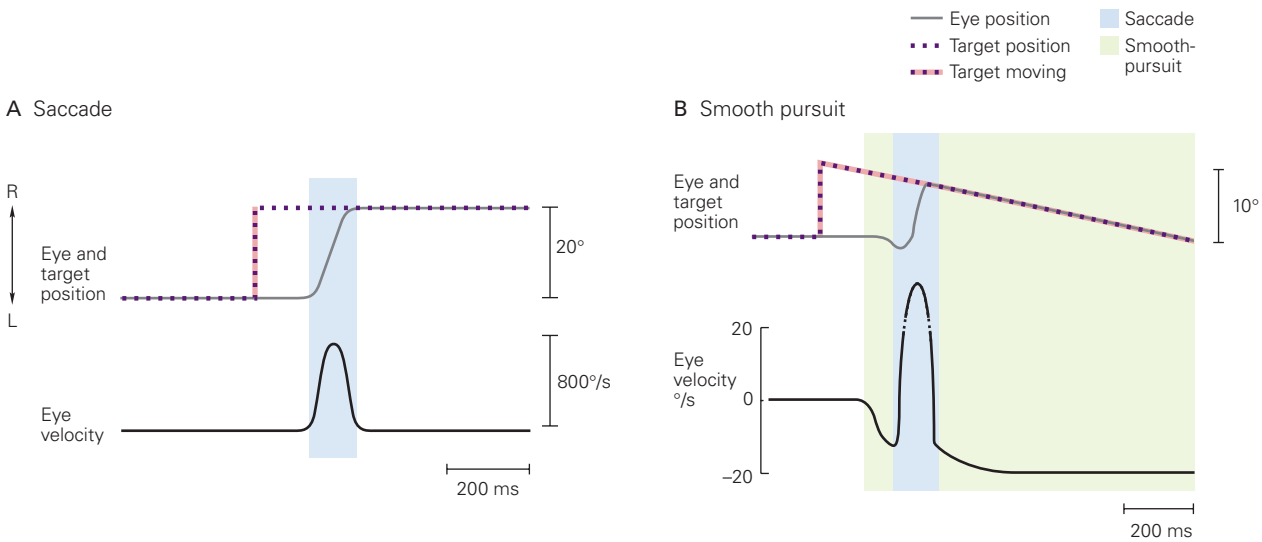


Figure 39–2 Saccadic and smooth-pursuit eye movements. Eye position, target position, and eye velocity are plotted against time.

A. The human saccade. At the beginning of the plot the eye is on the target (the traces representing eye and target positions are superimposed). Suddenly the target jumps to the right, and within 200 ms the eye moves to bring the target back to the fovea. Note the smooth, symmetric velocity profile. Because eye movements are rotations of the eye in the orbit, they are described by the angle of rotation. Similarly, objects in the visual field are described by the angle of arc they subtend at the eye. Viewed at arm's length, a thumb subtends an angle of approximately 1 degree. A saccade from one edge of the thumb to the other therefore traverses 1 degree of arc.

B. Human smooth pursuit. In this example the subject is asked to make a saccade to a target that jumps away from the center of gaze and then slowly moves back to center. The first movement seen in the position and velocity traces is a smooth-pursuit movement in the same direction as the target movement. The eye briefly moves *away* from the target before a saccade is initiated because the latency of the pursuit system is shorter than that of the saccade system. The smooth-pursuit system is activated by the target moving back toward the center of gaze, the saccade adjusts the eye's position to catch the target, and thereafter smooth pursuit keeps the eye on the target. The recording of saccade velocity is clipped so that the movement can be shown on the scale of the pursuit movement, an order of magnitude slower than the saccade.

The neural signal sent to each eye muscle has two components, one related to eye position and the other to eye velocity. Velocity and position signals are generated by different neural systems that converge on the motor neuron. In addition, horizontal and vertical eye movements are specified independently; vertical movements are generated in the mesencephalic reticular formation and horizontal movements in the pontine reticular formation.

Inhibitory neurons suppress unwanted eye movements. Omnipause neurons in the pontine reticular formation prevent excitatory neurons in the brain stem from stimulating the motor neurons. Fixation neurons in the rostral superior colliculus inhibit movement-related neurons in the colliculus while exciting omnipause neurons in the pons. Inhibitory neurons in the substantia nigra inhibit these same movement-related neurons from firing except during saccades.

The Eye Is Moved by the Six Extraocular Muscles

Eye Movements Rotate the Eye in the Orbit

To a good approximation, the eye is a sphere that sits in a socket, the orbit. Eye movements are simply rotations of the eye in the orbit. The eye's orientation can be defined by three axes of rotation—horizontal, vertical, and torsional—that intersect at the center of the eyeball, and eye movements are described as rotations around these axes. Horizontal and vertical eye movements change the line of sight by redirecting the fovea; torsional eye movements rotate the eye around the line of sight but do not change gaze.

Horizontal rotation of the eye away from the nose is called *abduction* and rotation toward the nose is *adduction.* Vertical movements are referred to as *elevation*

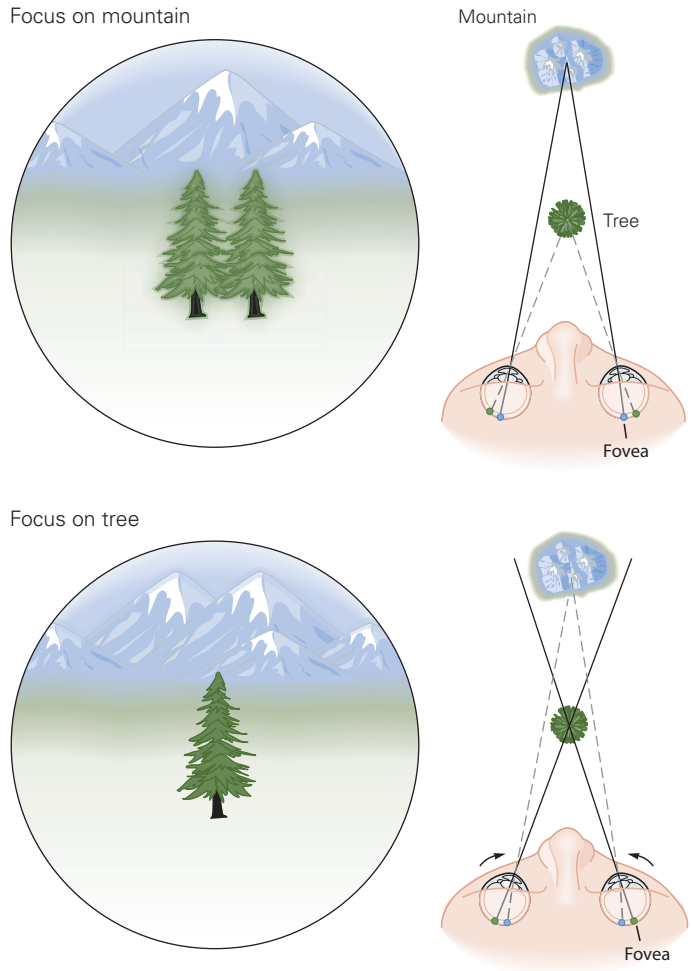


Figure 39–3 Vergence movements. When the eyes focus on a distant mountain, images of the mountain lie on the foveae, whereas those of the nearer tree occupy different retinal positions relative to the two foveae, yielding the percept of a double image. When the viewer looks instead at the tree (**below**), the vergence system must rotate each eye inward. Now the tree's image occupies similar positions on the foveae of both retinas and is seen as one object, but the mountain's images occupy different locations on the retinas and appear double. (Reproduced, with permission, from F. A. Miles.)

(upward rotation) and *depression* (downward rotation). Finally, torsional movements include *intorsion* (rotation of the top of the cornea toward the nose) and *extorsion* (rotation away from the nose).

Except for vergence, most eye movements are conjugate. For example, during gaze to the right the right eye abducts and the left eye adducts. Similarly, if the right eye extorts, the left eye intorts.

The Six Extraocular Muscles Form Three Agonist–Antagonist Pairs

Each eye is rotated by six extraocular muscles arranged in three agonist–antagonist pairs (Figure 39–4). The four rectus muscles (lateral, medial, superior, and inferior) share a common origin, the annulus of Zinn, at the apex of the orbit. They insert on the surface of the eye, or sclera, anterior to the eye's equator. The origin of the inferior oblique muscle is on the medial wall of the orbit; the superior oblique muscle's tendon passes through the trochlea, or pulley, before inserting on the globe, so that its effective origin is also on the medial wall of the orbit. The oblique muscles insert on the posterior globe.

Each muscle has a dual insertion. The part of the muscle farthest from the eye inserts on a soft-tissue pulley through which the rest of the muscle passes on its way to the eye. When the extraocular muscles contract, they not only rotate the eye but also change their pulling directions.

The actions of the extraocular muscles are determined by their geometry and by the position of the eye in the orbit. The medial and lateral recti rotate the eye horizontally; the medial rectus adducts, whereas the lateral rectus abducts. The superior and inferior recti and the obliques rotate the eye both vertically and

torsionally. The superior rectus and inferior oblique elevate the eye, and the inferior rectus and superior oblique depress it. The superior rectus and superior oblique intort the eye, whereas the inferior rectus and inferior oblique extort it.

The relative amounts of vertical and torsional rotation produced by the superior and inferior recti and the obliques depend on eye position. The superior and inferior recti exert their maximal vertical action when the eye is abducted, that is, when the line of sight is parallel to the muscles' pulling directions. Conversely, the oblique muscles exert their maximal vertical action when the eye is adducted (Figure 39–5).

Movements of the Two Eyes Are Coordinated

Humans and other animals with eyes in front have binocular vision. This facilitates stereopsis, the ability to perceive a visual scene in three dimensions, as well as depth perception. At the same time, binocular vision requires precise coordination of the movements of the two eyes so that both foveae are always directed at the target of interest. For most eye movements both eyes must move by the same amount and in the same direction. This is accomplished, in large part, through the pairing of eye muscles in the two eyes.

Just as each eye muscle is paired with its antagonist in the same orbit (eg, the medial and lateral recti), it is also paired with the muscle that moves the opposite eye in the same direction. For example, coupling of the left lateral rectus and right medial rectus moves both eyes to the left during a leftward saccade. The orientations of the vertical muscles are such that each pair consists of one rectus muscle and one oblique muscle. For example, the left superior rectus and the right inferior oblique both move the eyes upward in left gaze. The binocular muscle pairs are listed in Table 39–1.

The Extraocular Muscles Are Controlled by Three Cranial Nerves

The extraocular muscles are innervated by groups of motor neurons whose cell bodies are clustered in three

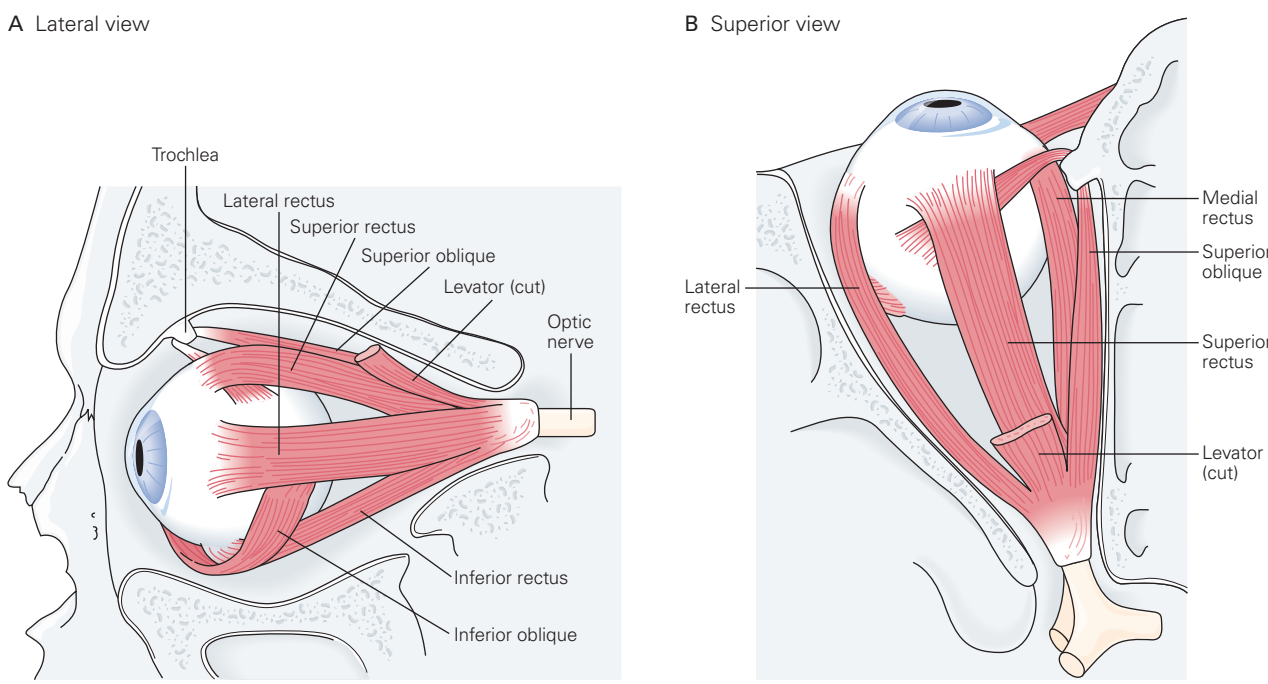


Figure 39–4 The origins and insertions of the extraocular muscles.

A. Lateral view of the left eye with the orbital wall cut away. Each rectus muscle inserts in front of the equator of the globe so that contraction rotates the cornea toward the muscle. Conversely, the oblique muscles insert behind the equator and contraction rotates the cornea away from the insertion. The superior oblique muscle passes through a bony pulley, the trochlea, before it inserts on the globe. The levator muscle of the upper eyelid raises the lid.

B. Superior view of the left eye with the roof of the orbit and the levator muscle cut away. The superior rectus passes over the superior oblique and inserts in front of it on the globe.

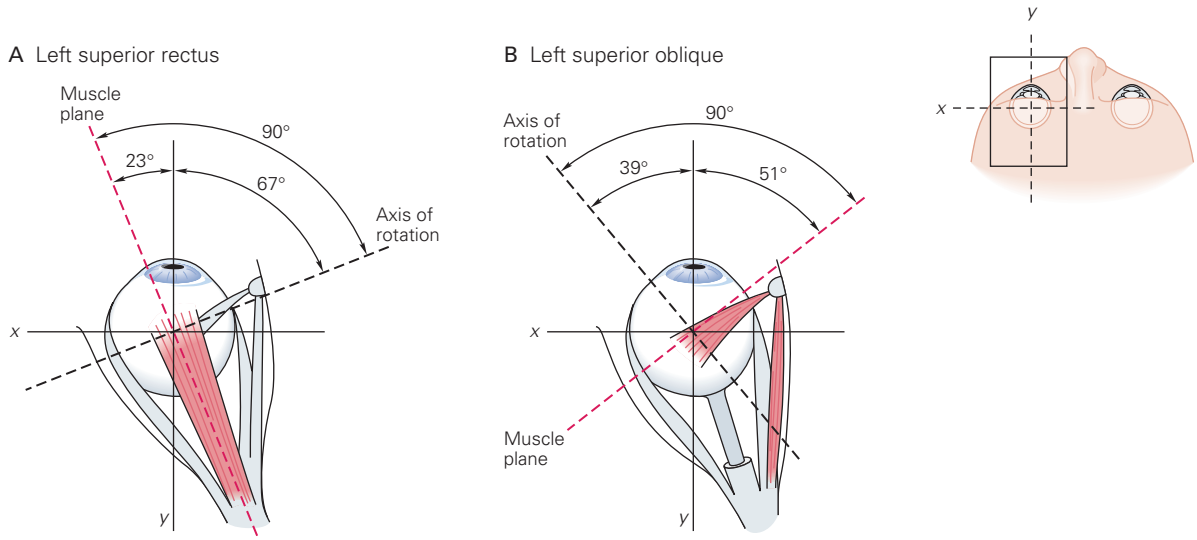


Figure 39–5 **Each superior rectus and oblique muscle has both torsional and elevational actions.** How much elevation and torsion each muscle provides depends on the position of the eye. (Adapted, with permission, from von Noorden 1980.)

A. When eye position is in the primary visual axis (the *y* axis in the diagram) or lateral to it (*abduction*), elevation is provided by the superior rectus and all of the intorsion is from the superior oblique muscle. When the eye is positioned completely medial to the visual axis (*adduction*), intorsion comes predominantly from the superior rectus and most of the elevation comes from the inferior oblique.

B. When the eye is positioned 16 degrees or more laterally from the primary visual axis, the superior oblique intorts the eye and depression comes from the inferior rectus. When it is completely medial to the visual axis, the superior oblique rotates the eye vertically downward and all of the intorsion comes from the superior rectus.

nuclei in the brain stem (Figure 39–6). The lateral rectus is innervated by the abducens nerve (cranial nerve VI), whose nucleus lies in the pons in the floor of the fourth ventricle. The superior oblique muscle is innervated by the trochlear nerve (cranial nerve IV), whose nucleus is located in the midbrain at the level of the inferior colliculus. (The trochlear nerve gets its name from the trochlea, the bony pulley through which the superior oblique muscle travels.)

Figure 39–6 Vertical Muscle Action in Adduction and Abduction

Muscle	Action in adduction	Action in abduction
Superior rectus	Intorsion	Elevation
Inferior rectus	Extorsion	Depression
Superior oblique	Depression	Intorsion
Inferior oblique	Elevation	Extorsion

All the other extraocular muscles—the medial, inferior, and superior recti and the inferior oblique—are innervated by the oculomotor nerve (cranial nerve III), whose nucleus lies in the midbrain at the level of the superior colliculus. The oculomotor nerve also contains fibers that innervate the levator muscle of the upper eyelid. Cell bodies of axons innervating both eyelids are located in the central caudal nucleus, a single midline structure within the oculomotor complex. Finally, traveling with the oculomotor nerve are parasympathetic fibers that innervate the iris sphincter muscle, the constrictor of the pupil, and the ciliary muscles that adjust the curvature of the lens to focus the eye during accommodation.

The pupil and eyelid also have sympathetic innervation, which originates in the intermediolateral cell column of the ipsilateral upper thoracic spinal cord. Fibers of these neurons synapse on cells in the superior cervical ganglion in the upper neck. Axons of these postganglionic cells travel along the carotid artery to the carotid sinus and then into the orbit. Sympathetic pupillary fibers innervate the iris dilator muscle, causing the pupil to dilate and thus providing the pupillary component of the so-called "fight or flight" response.

Sympathetic fibers also innervate Müller's muscle, a secondary elevator of the upper eyelid. The sympathetic control of pupillary dilatation and lid elevation is responsible for the "wide-eyed" look of excitement and sympathetic overload.

The best way to understand the actions of the extraocular muscles is to consider the eye movements that remain after a lesion of a specific nerve (Box 39–1).

Extraocular Motor Neurons Encode Eye Position and Velocity

We can illustrate how the gaze system generates eye movements by considering the activity of an oculomotor neuron during a saccade. To move the eye quickly to a new position in the orbit and keep it there, two passive forces must be overcome: the elastic force of the orbit, which tends to restore the eye to a central position in the orbit, and a velocity-dependent viscous force that opposes rapid movement. Thus the motor signal must include information about tonic position, which opposes the elastic force, and velocity, which overcomes orbital viscosity and moves the eye quickly to a new position.

Information about the position and velocity of the eye is conveyed by the discharge frequency of an oculomotor neuron (Figure 39–8). The firing rate of the neuron rises rapidly as the eye's velocity increases from 0 degrees to 900 degrees per second; this is called the *saccadic pulse.* The frequency of this pulse determines the speed of the saccade, whereas the duration of the pulse controls the duration of the saccade. The difference in the firing rates before and after the saccade is called the *saccadic step.* As described below, the pulse and step are generated by different brain stem structures.

Oculomotor neurons differ from skeletal motor neurons in several ways. Although the extraocular muscles are rich in sensors resembling the muscle spindles of skeletal muscles, there are no ocular stretch reflexes. Oculomotor neurons do not have recurrent inhibitory connections. All oculomotor neurons participate equally in all types of eye movements; no motor neurons are specialized for saccades or smooth pursuit.

However, like skeletal motor units, eye motor units are recruited in a fixed sequence (see Chapter 38). Regardless of the type of eye movement, the specific ocular motor neurons recruited depend on the position of the eye in the orbit and the desired eye velocity. For example, as the eye moves laterally the number of active abducens neurons increases, causing more muscle fibers in the lateral rectus to contract.

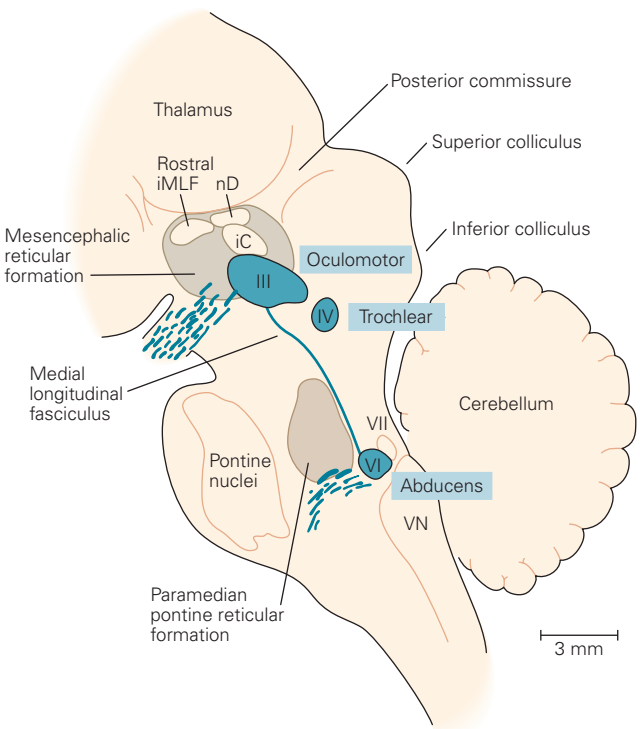


Figure 39–6 The ocular motor nuclei in the brain stem. The nuclei are shown in a parasagittal section through the thalamus, pons, midbrain, and cerebellum of a rhesus monkey. The oculomotor nucleus (cranial nerve III) lies in the midbrain at the level of the mesencephalic reticular formation. The trochlear nucleus (nerve IV) is slightly caudal, and the abducens nucleus (nerve VI) lies in the pons at the level of the paramedian pontine reticular formation, adjacent to the fasciculus of the facial nerve (VII). Compare Figure 45–5. (**iC**, interstitial nucleus of Cajal; **iMLF**, interstitial nucleus of the medial longitudinal fasciculus; **nD**, nucleus of Darkshevich; **VN**, vestibular nuclei.) (Adapted, with permission, from Henn et al. 1984.)

The Motor Circuits for Saccades Lie in the Brain Stem

How are the motor signals for velocity and position of the eye determined? The higher centers that control gaze specify only a desired change in eye position. The activity of neurons in these centers specifies a target location in the visual field. This location signal must be transformed into a motor signal that encodes the velocity and position of eye movement.

Horizontal Saccades Are Generated in the Pontine Reticular Formation

The signal for horizontal saccades originates in the paramedian pontine reticular formation, adjacent to the

Box 39–1 Extraocular Muscle or Nerve Lesions

Patients with lesions of the extraocular muscles or their nerves complain of double vision (diplopia) because the images of the object of gaze no longer fall on the corresponding retinal locations in both eyes. Lesions of each nerve produce characteristic symptoms that depend on which extraocular muscles are affected. In general, double vision increases when the patient tries to look in the direction of the weak muscle.

Abducens Nerve

A lesion of the abducens nerve (VI) causes weakness of the lateral rectus. When the lesion is complete the eye cannot abduct beyond the midline, such that a horizontal diplopia increases when the subject looks in the direction of the affected eye.

Trochlear Nerve

A lesion of the trochlear nerve (IV) affects both torsional and vertical eye movements. When the patient looks straight ahead, the affected eye is above the normal eye (Figure 39–7A). The difference increases when the patient looks to the right, adducting the eye with the weak muscle (Figure 39–7B left), and decreases when the patient looks to the left, abducting the eye (Figure 39–7B right), because the superior oblique predominantly depresses the eye in adduction.

The deficit is worse when patients attempt to depress and adduct the eye, but improves when they elevate the adducted eye (Figure 39–7C). Patients with superior oblique paresis often keep their heads tilted away from the affected eye. A tilt of the head to one side, such that one ear is pointed downward, induces a small torsion of the eye in the opposite direction, known as ocular counter-roll.

When the head tilts to the left, the left eye is ordinarily intorted by the left superior rectus and left superior oblique, while the right eye is extorted by the right superior rectus and right inferior oblique. The elevation action of the superior rectus is canceled by the depression action of the superior oblique, so the eye only rotates. When the head tilts to the right, the inferior oblique and inferior rectus extort the left eye and the superior oblique relaxes.

With paresis of the left superior oblique, when the head tilts to the left the elevation of the superior rectus is unopposed and the eye moves upward (Figure 39–7D right). The diplopia can be minimized by tilting the head to the right (Figure 39–7D left).

Oculomotor Nerve

A lesion of the oculomotor nerve (III) has complex effects because this nerve innervates multiple muscles. A complete lesion spares only the lateral rectus and superior oblique muscles. Thus the paretic eye is typically deviated downward and abducted at rest, and it cannot move medially or upward from a middle position. Downward movement is partially affected because the inferior rectus muscle is weak but the superior oblique is preserved.

Because the fibers that control lid elevation, accommodation, and pupillary constriction travel in the oculomotor nerve, damage to this nerve also results in drooping of the eyelid (ptosis), blurred vision for near objects, and pupillary dilation (mydriasis). Although sympathetic innervation is still intact with an oculomotor nerve lesion, the ptosis is essentially complete, since Müller's muscle contributes less to elevation of the upper eyelid than does the levator muscle of the upper eyelid.

Sympathetic Oculomotor Nerves

Sympathetic fibers innervating the eye arise from the thoracic spinal cord, traverse the apex of the lung, and ascend to the eye on the outside of the carotid artery.

Interruption of the sympathetic pathways to the eye yields Horner syndrome, whose characteristic features are a partial ipsilateral ptosis owing to weakness of Müller's muscle and a relative constriction (miosis) of the ipsilateral pupil. The pupillary asymmetry is most pronounced in low light because the normal pupil is able to dilate but the pupil affected by Horner syndrome is not.

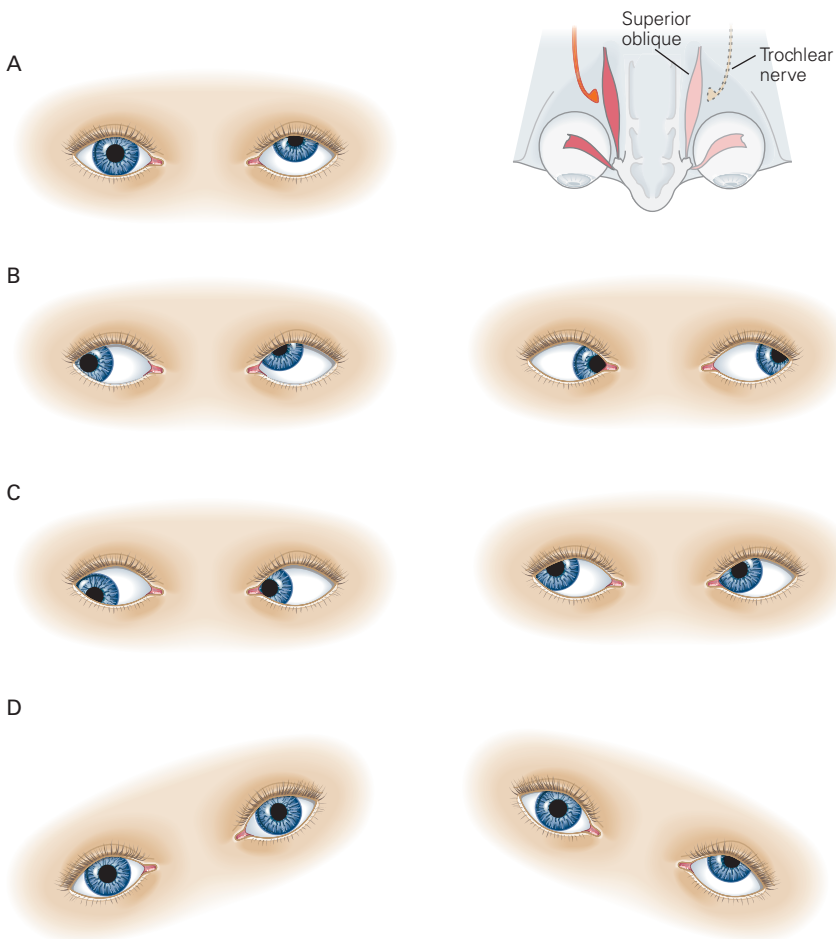


Figure 39–7 **Effect of a left trochlear nerve palsy.** The trochlear nerve innervates the superior oblique muscle, which inserts behind the equator of the eye. It depresses the eye when it is adducted and intorts the eye when it is abducted.

A. Hypertropia occurs when the eye is in the center of the orbit and the left eye is slightly above the right eye.

B. The hypertropia is worse when the eye is adducted because the unopposed inferior oblique pushes the eye higher (**left**). The condition is improved when the eye is abducted (**right**) because the superior oblique contributes less to depression than to intorsion.

C. When the patient looks to the right the hypertropia is worse on downward gaze (**left**) than it is on upward gaze (**right**).

D. The hypertropia is improved by head tilt to the right (**left**) and worsened by tilt to the left (**right**). The ocular counter-rolling reflex induces intorsion of the left eye on leftward head tilt, and extorsion of the eye on rightward head tilt (see Chapter 40). With leftward head tilt, intorsion requires increased activity of the superior rectus, whose elevating activity is unopposed by the weak superior oblique, causing increased hypertropia. With rightward head tilt and extorsion of the left eye, the unopposed superior rectus muscle is less active, and the hypertropia decreases.

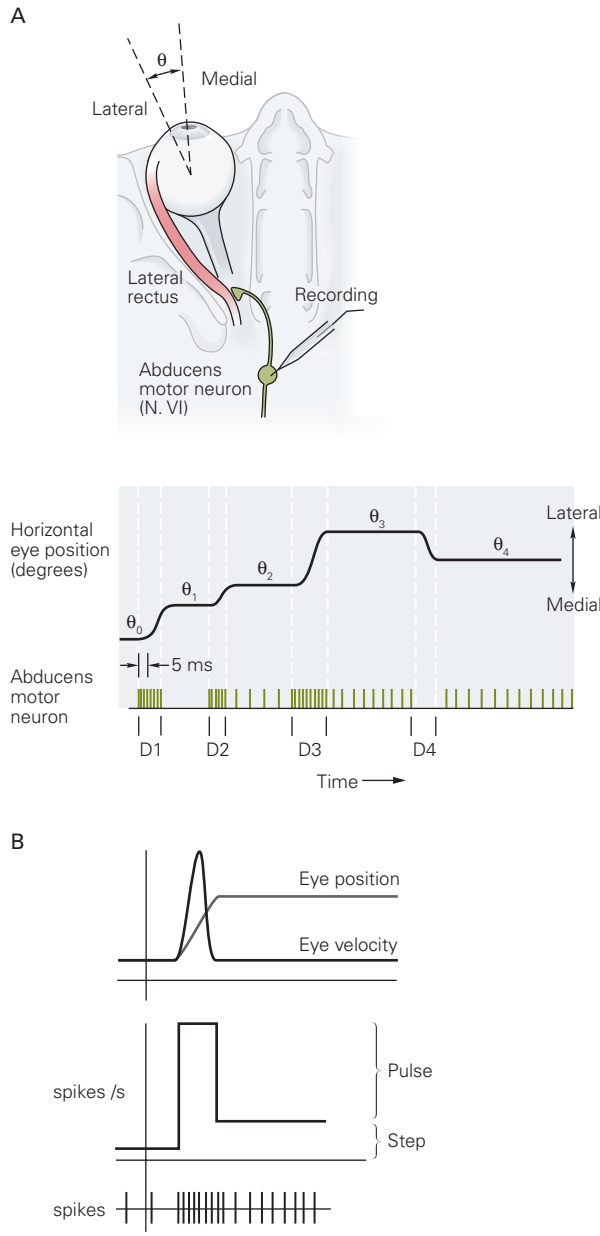


Figure 39–8 Oculomotor neurons signal eye position and velocity.

A. The record is from an abducens neuron of a monkey. When the eye is positioned in the medial side of the orbit the cell is silent (**position** θ_0). As the monkey makes a lateral saccade there is a burst of firing (**D1**), but in the new position (θ_1) the eye is still too far medial for the cell to discharge continually. During the next saccade there is a burst (**D2**), and at the new position (θ_2) there is a tonic position-related discharge. Before and during the next saccade (**D3**) there is again a pulse of activity and a higher tonic discharge when the eye is at the new position (θ_4). When the eye makes a medial movement there is a period of silence during the saccade (**D4**) even though the eye ends up at a position associated with a tonic discharge. (Adapted, with permission, from A. Fuchs 1970.)

B. Saccades are associated with a step of activity, which signals the change in eye position, and a pulse of activity, which signals eye velocity. The neural activity corresponding to eye position and velocity is illustrated both as a train of individual spikes and as an estimate of the instantaneous firing rate (spikes per second).

abducens nucleus to which it projects (Figure 39–9A). The paramedian pontine reticular formation contains a family of *burst neurons* that gives rise to the saccadic pulse. These cells fire at a high frequency just before and during ipsiversive saccades, and their activity resembles the pulse component of ocular motor neuron discharge (Figure 39–9B).

There are several types of burst neurons. Medium-lead burst neurons make direct excitatory connections to motor neurons and interneurons in the ipsilateral abducens nucleus. Long-lead burst neurons drive the medium-lead burst cells and receive excitatory input from higher centers. Inhibitory burst neurons suppress the activity of contralateral abducens neurons and contralateral excitatory burst neurons and are themselves excited by medium-lead burst neurons.

A second class of pontine cells, *omnipause neurons*, fire continuously except around the time of a saccade; firing ceases shortly before and during all saccades (Figure 39–9B). Omnipause neurons are located in

the nucleus of the dorsal raphe in the midline (Figure 39–9A). They are GABA-ergic (γ-aminobutyric acid) inhibitory neurons that project to contralateral pontine and mesencephalic burst neurons. Electrical stimulation of omnipause neurons arrests a saccade, which resumes when the stimulation stops. Making a saccade requires simultaneous excitation of burst neurons and inhibition of omnipause cells; this provides the system with additional stability, such that unwanted saccades are infrequent.

If the motor neurons received signals from only the burst cells, the eyes would drift back to the starting position because there would be no new position signal to hold the eyes against elastic restorative forces. David A. Robinson first pointed out that the tonic position signal, the saccadic step, can be generated from

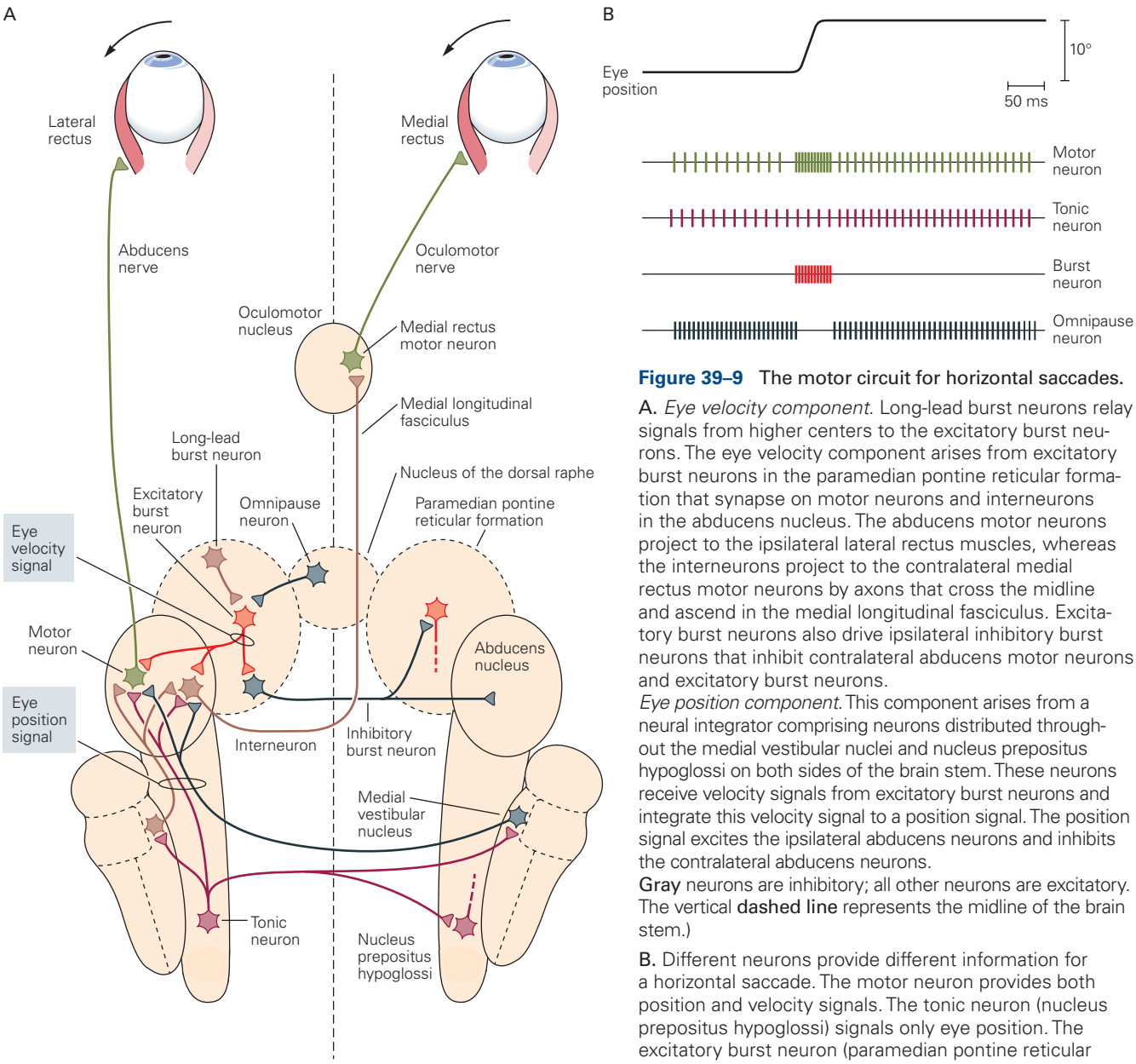


Figure 39–9 The motor circuit for horizontal saccades.

A. *Eye velocity component.* Long-lead burst neurons relay signals from higher centers to the excitatory burst neurons. The eye velocity component arises from excitatory burst neurons in the paramedian pontine reticular formation that synapse on motor neurons and interneurons in the abducens nucleus. The abducens motor neurons project to the ipsilateral lateral rectus muscles, whereas the interneurons project to the contralateral medial rectus motor neurons by axons that cross the midline and ascend in the medial longitudinal fasciculus. Excitatory burst neurons also drive ipsilateral inhibitory burst neurons that inhibit contralateral abducens motor neurons and excitatory burst neurons.
Eye position component. This component arises from a neural integrator comprising neurons distributed throughout the medial vestibular nuclei and nucleus prepositus hypoglossi on both sides of the brain stem. These neurons receive velocity signals from excitatory burst neurons and integrate this velocity signal to a position signal. The position signal excites the ipsilateral abducens neurons and inhibits the contralateral abducens neurons.
Gray neurons are inhibitory; all other neurons are excitatory. The vertical **dashed line** represents the midline of the brain stem.)

B. Different neurons provide different information for a horizontal saccade. The motor neuron provides both position and velocity signals. The tonic neuron (nucleus prepositus hypoglossi) signals only eye position. The excitatory burst neuron (paramedian pontine reticular formation) signals only eye velocity. The omnipause neuron discharges at a high rate except immediately before, during, and just after the saccade.

the velocity burst signal by the neural equivalent of the mathematical process of integration. Velocity can be computed by differentiating position with respect to time; conversely, position can be computed by integrating velocity with respect to time.

For horizontal eye movements, neural integration of the velocity signal is performed by the medial vestibular nucleus and the nucleus prepositus hypoglossi in conjunction with the flocculus of the cerebellum. As expected, animals with lesions of these areas make normal horizontal saccades but the eyes drift back to a middle position after a saccade. Integration of the horizontal saccadic burst requires coordination of the bilateral nuclei propositi hypoglossi and medial vestibular nuclei through commissural connections. A midline lesion of these connections causes failure of the neural integrator.

Medium-lead burst neurons in the paramedian pontine reticular formation and neurons of the medial vestibular nucleus and nucleus prepositus hypoglossi project to the ipsilateral abducens nucleus and deliver respectively the pulse and step components of the motor signal. Two populations of neurons in the abducens nucleus receive this signal. One is a group of motor neurons that innervate the ipsilateral lateral rectus muscle. The second group consists of interneurons whose axons cross the midline and ascend in the medial longitudinal fasciculus to the motor neurons for the contralateral medial rectus, which lie in the oculomotor nucleus (Figure 39–9A).

Thus, medial rectus motor neurons do not receive the pulse and step signals directly. This arrangement allows for precise coordination of corresponding movements of both eyes during horizontal saccades and other conjugate eye movements. The length of the medial longitudinal fasciculus and its vulnerability to demyelination and ischemia make it clinically important.

Vertical Saccades Are Generated in the Mesencephalic Reticular Formation

The burst neurons responsible for vertical saccades are found in the rostral interstitial nucleus of the medial longitudinal fasciculus in the mesencephalic reticular formation (see Figure 39–6). Vertical and torsional neural integration are performed in the nearby interstitial nucleus of Cajal. Both the pontine and mesencephalic systems participate in the generation of oblique saccades, which have both horizontal and vertical components.

Purely vertical saccades require activity on both sides of the mesencephalic reticular formation, and communication between the two sides occurs in the posterior commissure. In contrast, there are not separate omnipause neurons for horizontal and vertical saccades; pontine omnipause cells inhibit both pontine and mesencephalic burst neurons.

Brain Stem Lesions Result in Characteristic Deficits in Eye Movements

We can now understand how different brain stem lesions cause characteristic syndromes. Lesions that include the paramedian pontine reticular formation result in paralysis of ipsiversive horizontal gaze of both eyes but spare vertical saccades. A lesion of the abducens nucleus has a similar effect, for both abducens motor neurons and interneurons are affected. Lesions that include the midbrain gaze centers cause paralysis of vertical gaze. Certain neurological disorders cause degeneration of burst neurons and impair their function, leading to a progressive slowing of saccades.

Lesions of the medial longitudinal fasciculus disconnect the medial rectus motor neurons from the abducens interneurons (Figure 39–9A). Thus during conjugate horizontal eye movements, such as saccades and pursuit, the abducting eye moves normally but adduction of the other eye is impeded. Despite this paralysis in version movements, the medial rectus acts normally in vergence movements because the motor neurons for vergence lie in the midbrain, as will be discussed later. This syndrome, called an *internuclear ophthalmoplegia*, is a consequence of a brain stem stroke or demyelinating diseases such as multiple sclerosis.

Saccades Are Controlled by the Cerebral Cortex Through the Superior Colliculus

The pontine and mesencephalic burst circuits provide the motor signals necessary to drive the muscles for saccades. However, among higher mammals eye movements are ultimately driven by cognitive behavior. The decision when and where to make a saccade that is behaviorally important is usually made in the cerebral cortex. A network of cortical and subcortical areas controls the saccadic system through the superior colliculus (Figure 39–10).

The Superior Colliculus Integrates Visual and Motor Information into Oculomotor Signals to the Brain Stem

The superior colliculus in the midbrain is a major visuomotor integration region, the mammalian homolog

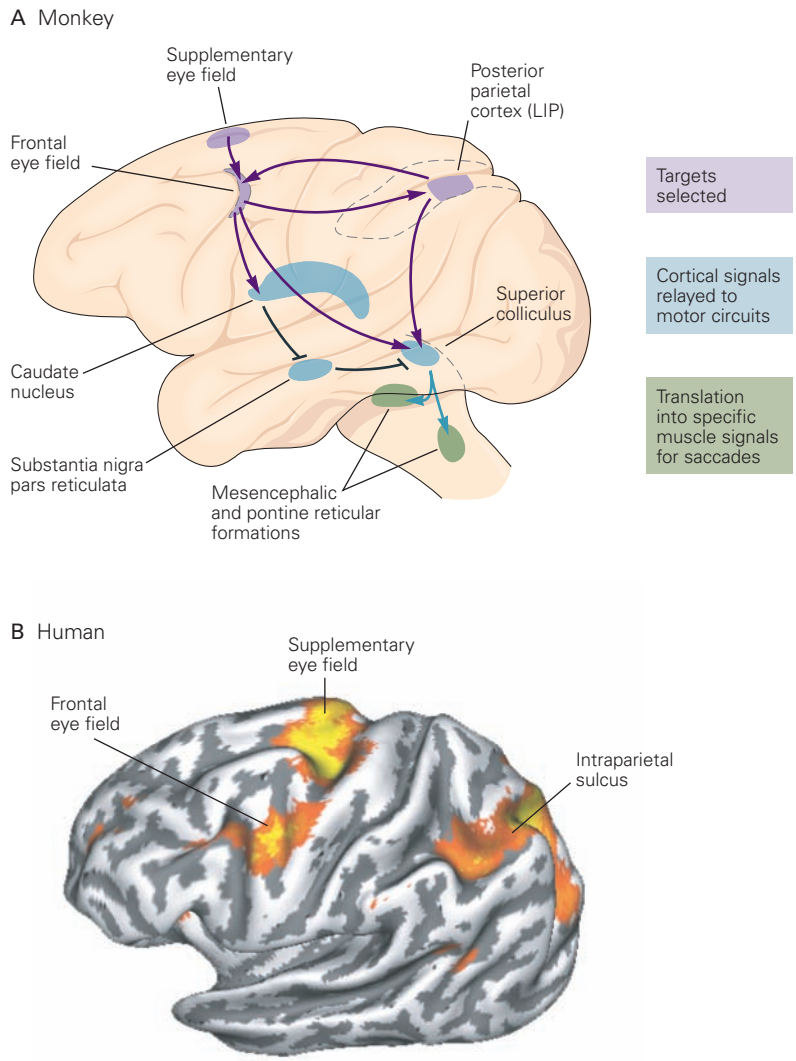


Figure 39–10 Cortical pathways for saccades. **A.** In the monkey the saccade generator in the brain stem receives a command from the superior colliculus. The colliculus receives direct excitatory projections from the frontal eye fields and the lateral intraparietal area (**LIP**) and an inhibitory projection from the substantia nigra. The substantia nigra is suppressed by the caudate nucleus, which in turn is excited by the frontal eye fields. Thus the frontal eye fields directly excite the colliculus and indirectly release it from suppression by the substantia nigra by exciting the caudate nucleus, which inhibits the substantia nigra. (Reproduced, with permission, from R. J. Krausliz.)

B. This lateral scan of a human brain shows areas of cortex activated during saccades. (Reproduced, with permission, from Curtis and Connelly 2010.)

of the optic tectum in nonmammalian vertebrates. It can be divided into two functional regions: the superficial layers and the intermediate and deep layers.

The three superficial layers receive both direct input from the retina and a projection from the striate cortex representing the entire contralateral visual hemifield. Neurons in the superficial layers respond to visual stimuli. In monkeys the responses of half of these vision-related neurons are quantitatively enhanced when an animal prepares to make a saccade to a stimulus in the cell's receptive field. This enhancement is specific for saccades. If the monkey attends to the stimulus without making a saccade to it—for example, by making a hand movement in response to a brightness change—the neuron's response is not augmented.

Neuronal activity in the two intermediate and deep layers is primarily related to oculomotor actions. The movement-related neurons in these layers receive visual information from the prestriate, middle temporal, and parietal cortices and motor information from the frontal eye field. The intermediate and deep layers also contain somatotopic, tonotopic, and retinotopic maps of sensory inputs, all in register with one another. For example, the image of a bird will excite a vision-related neuron, whereas the bird's chirp will excite an adjacent audition-related neuron, and both will excite a bimodal neuron. Polymodal spatial maps enable us to shift our eyes toward auditory or somatosensory stimuli as well as visual ones.

Much of the early research describing the sensory responsiveness of neurons in the intermediate layer

was done in anesthetized animals. To understand how the brain generates movement, however, the activity of neurons needs to be studied in alert animals while they behave normally. Edward Evarts pioneered this approach in studies of the skeletomotor system, after which it was extended to the ocular motor system.

One of the earliest cellular studies in active animals revealed that individual movement-related neurons in the superior colliculus selectively discharge before saccades of specific amplitudes and directions, just as individual vision-related neurons in the superior colliculus respond to stimuli at specific distances and directions from the fovea (Figure 39–11A). The movement-related neurons form a map of potential eye movements that is in register with the visuotopic and tonotopic arrays of sensory inputs, so that the neurons that control eye movements to a particular target are found in the same region as the cells excited by the sounds and image of that target. Each movement-related neuron in the superior colliculus has a *movement field*, a region of the visual field that is the target for saccades controlled by that neuron. Electrical stimulation of the intermediate layers of the superior colliculus evokes saccades into the movement fields of the stimulated neurons.

Movement fields are large, so each superior colliculus cell fires before a wide range of saccades, although each cell fires most intensely before saccades of a specific direction and amplitude. A large population of cells is thus active before each saccade, and eye movement is encoded by the entire ensemble of these broadly tuned cells. Because each cell makes only a small contribution to the direction and amplitude of the movement, any variability or noise in the discharge of a given cell is minimized. Similar population coding is found in the olfactory system (see Chapter 32) and skeletal motor system (see Chapter 37).

Activity in the superficial and intermediate layers of the superior colliculus can occur independently: Sensory activity in the superficial layers does not always lead to motor output, and motor output can occur without sensory activity in the superficial layers. In fact, the neurons in the superficial layers do not provide a large projection directly to the intermediate layers. Instead, their axons terminate on neurons in the pulvinar and lateral posterior nuclei of the thalamus, which relay the signals from the superficial layers of the superior colliculus to cortical regions that project back to the intermediate layers.

Lesions of a small part of the colliculus affect the latency, accuracy, and velocity of saccades. Destruction of the entire colliculus renders a monkey unable to make any contralateral saccades, although with time this ability is recovered.

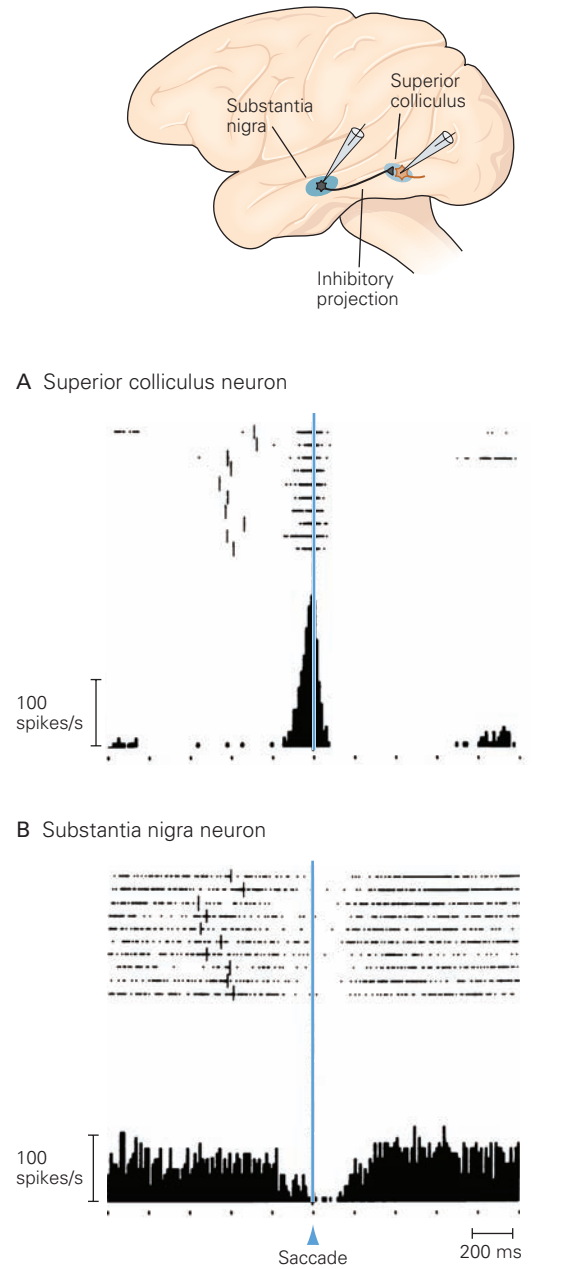


Figure 39–11 **Neurons in the superior colliculus and substantia nigra are active around the time of a saccade.** The two neurons were recorded simultaneously. (Reproduced, with permission, from Hikosaka and Wurtz 1989.)

A. A neuron in the superior colliculus fires in a burst immediately before the saccade. Raster plots of activity in successive trials of the same task are summed to form the histogram below.

B. A neuron in the substantia nigra pars reticulata is tonically active, becomes quiet just before the saccade, and resumes activity after the saccade. This type of neuron inhibits neurons in the intermediate layers of the superior colliculus.

The Rostral Superior Colliculus Facilitates Visual Fixation

The most rostral portion of the superior colliculus contains a representation of the fovea. Neurons in the intermediate layers in this region discharge strongly during active visual fixation and before small saccades to the contralateral visual field. Because the neurons are active during visual fixation, this area of the superior colliculus is often called the fixation zone.

Neurons here inhibit the movement-related neurons in the more caudal parts of the colliculus and also project directly to the nucleus of the dorsal raphe, where they inhibit saccade generation by exciting the omnipause neurons. With lesions in the fixation zone an animal is more likely to make saccades to distracting stimuli.

The Basal Ganglia Inhibit the Superior Colliculus

The substantia nigra pars reticulata sends a powerful GABAergic inhibitory projection to the superior colliculus. Neurons in the substantia nigra fire spontaneously with high frequency; this discharge is suppressed at the time of voluntary eye movements to the contralateral visual field (see Figure 39–11B). Suppression is mediated by inhibitory input from neurons in the caudate nucleus, which fire before saccades to the contralateral visual field.

Two Regions of Cerebral Cortex Control the Superior Colliculus

The superior colliculus is controlled by two regions of the cerebral cortex that have overlapping but distinct functions: the lateral intraparietal area of the posterior parietal cortex (part of Brodmann's area 7) and the frontal eye field (part of Brodmann's area 8). Each of these areas contributes to the generation of saccades and the control of visual attention.

Perception is better at an attended place in the visual field than at an unattended place, as measured either by a subject's reaction time to an object suddenly appearing in the visual field or by the subject's ability to perceive a stimulus that is just noticeable. Saccadic eye movements and visual attention are closely intertwined (see Figure 39–1).

The lateral intraparietal area in the monkey is important in the generation of both visual attention and saccades. The role of this area in the processing of eye movements is best illustrated by a memory-guided saccade. To demonstrate this saccade, a monkey first fixates a spot of light. An object (the stimulus) appears in the receptive field of a neuron and then disappears; then the spot of light is extinguished. After a delay the monkey must make a saccade to the location of the vanished stimulus. Neurons in the lateral intraparietal area respond at the onset of the stimulus and continue firing during the delay until the saccade begins (Figure 39–12A), but their activity can be also dissociated from saccade planning. If the monkey is planning a saccade to a target outside the receptive field of a neuron, and a distractor appears in the field during the delay period, the neuron responds as vigorously to the distractor as it does to the target of a saccade (Figure 39–12B).

Lesioning of a monkey's posterior parietal cortex, which includes the lateral intraparietal area, increases the latency of saccades and reduces their accuracy. Such a lesion also produces selective neglect: A monkey with a unilateral parietal lesion preferentially attends to stimuli in the contralateral visual hemifield. In humans as well, parietal lesions—especially right parietal lesions—initially cause dramatic attentional deficits. Patients act as if the objects in the neglected field do not exist, and they have difficulty making eye movements into that field (see Chapter 17).

Patients with Balint syndrome, which is usually the result of bilateral lesions of the posterior parietal and prestriate cortex, tend to see and describe only one object at a time in their visual environment. These patients make few saccades, as if they are unable to shift the focus of their attention from the fovea, and can therefore describe only a foveal target. Even after these patients have recovered from most of their deficits, their saccades are delayed and inaccurate.

Compared to the neurons in the parietal cortex, neurons in the frontal eye field are more closely associated with saccades. Three different types of neurons in the frontal eye field discharge before saccades.

Visual neurons respond to visual stimuli and half of these neurons respond more vigorously to stimuli that are the targets of saccades (Figure 39–13A). Activity in these cells is not enhanced when an animal responds to the stimulus without making a saccade to it. Likewise, these cells are not activated before saccades that are made without visual targets; monkeys can be trained to make saccades of a specific direction and amplitude in total darkness.

Movement-related neurons fire before and during all saccades to their movement fields, whether or not they are made to a visual target. These cells do not respond to stimuli in their movement fields that are not targets of saccades. Unlike the movement-related cells in the superior colliculus, which fire before all saccades, movement-related neurons of the frontal eye field fire only before saccades that are relevant to the monkey's

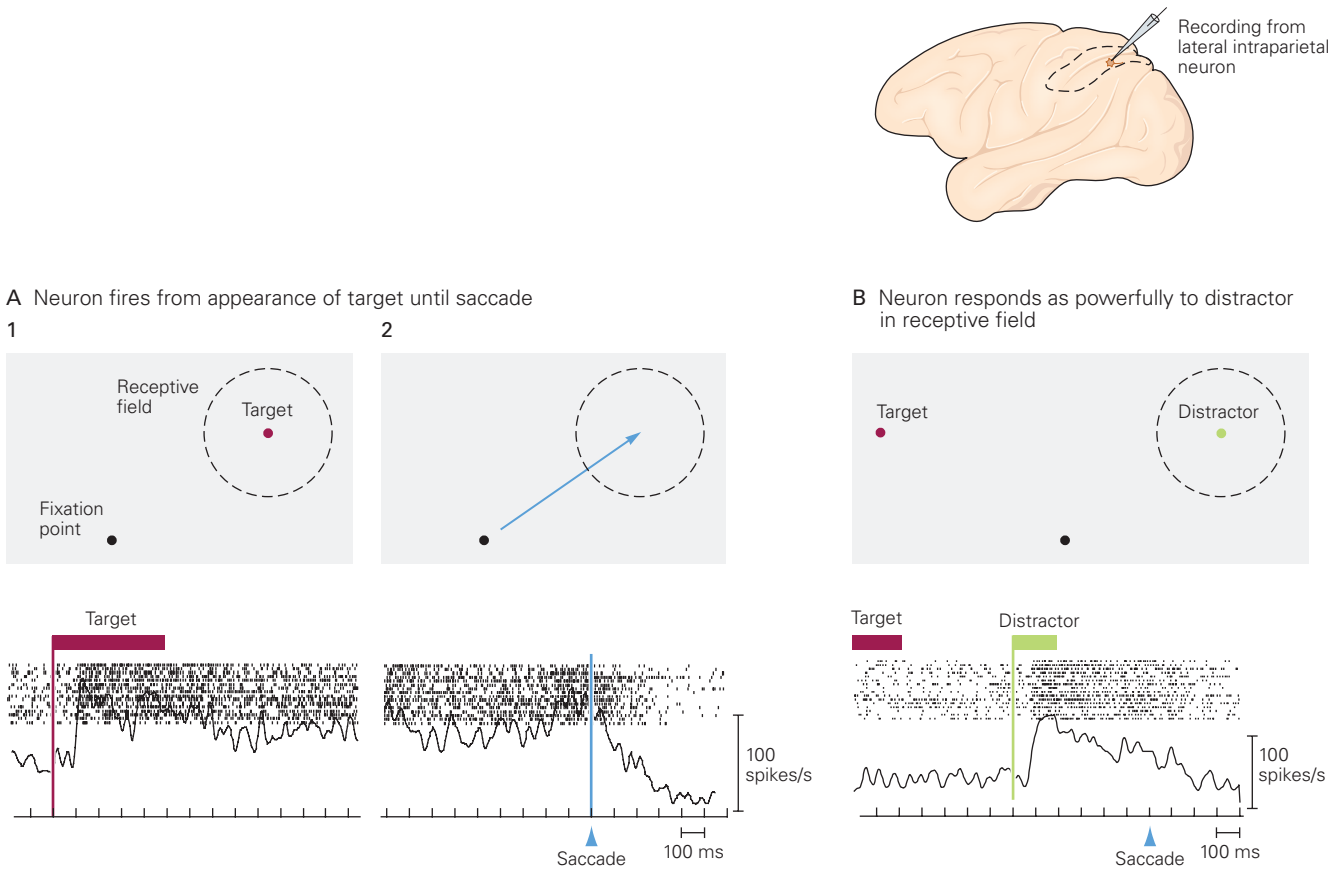


Figure 39–12 A parietal neuron is active before memory-guided saccades. Traces are aligned at events indicated by vertical lines. (Reproduced, with permission, from Powell and Goldberg 2000.)

A. The monkey plans a saccade from a fixation point to a target in the receptive field of a neuron in the lateral intraparietal cortex. The neuron responds to the appearance of the target (**1**). It continues to fire after the target has disappeared but before the signal to make the saccade, and stops firing after the onset of the saccade (**2**).

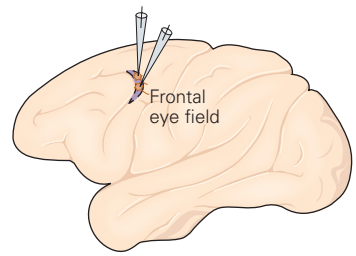
B. The monkey plans a saccade to a target outside the receptive field. The neuron responds to a distractor in the receptive field as strongly as it did to the target of a saccade.

behavior (Figure 39–13B). These neurons, especially those whose receptive fields lie in the visual periphery, project more strongly to the superior colliculus than do the visual neurons.

Visuomovement neurons have both visual and movement-related activity and discharge most strongly before visually guided saccades. Electrical stimulation of the frontal eye field evokes saccades to the movement fields of the stimulated cells. Bilateral stimulation of the frontal eye field evokes vertical saccades.

The frontal eye field controls the superior colliculus through two pathways (see Figure 39–10). First, the movement-related neurons project directly to the intermediate layers of the superior colliculus, exciting movement-related neurons there. Second, movement-related neurons form excitatory synapses on neurons in the caudate nucleus that inhibit the substantia nigra pars reticulata. Thus, activity of movement-related cells in the frontal eye field simultaneously excites the superior colliculus and releases it from the inhibitory influence of the substantia nigra. The frontal eye field also projects to the pontine and mesencephalic reticular formations, although not directly to the burst cells.

Two other cortical regions with inputs to the frontal eye field are thought to be important in the cognitive aspects of saccades. The *supplementary eye field* at the most rostral part of the supplementary motor area contains neurons that encode saccades in terms of spatial referents other than direction. For example, a neuron in the left supplementary eye field that ordinarily fires before rightward eye movements will fire before a leftward saccade if that saccade is to the right side of



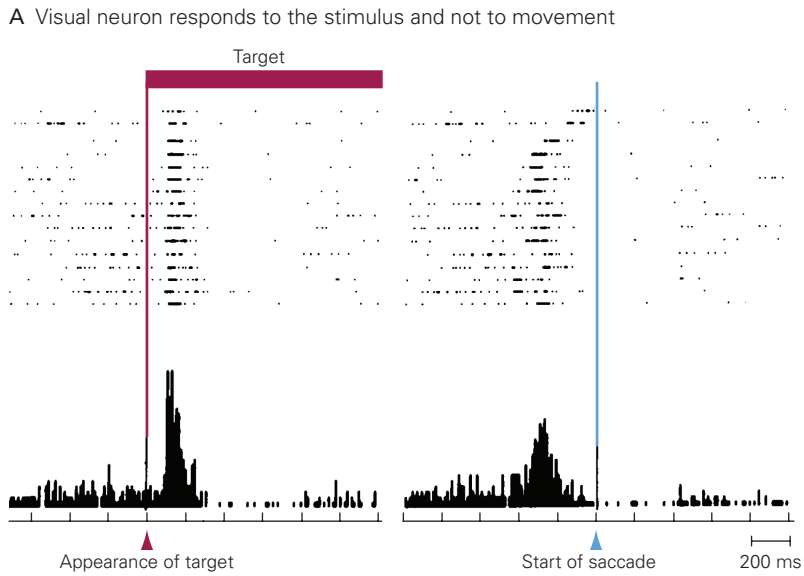


Figure 39–13 Visual and movement-related neurons in the frontal eye field. (Reproduced, with permission, from Bruce and Goldberg 1985.)

A. Activity of a visual neuron in the frontal eye field as a monkey makes a saccade to a target in its visual field. Raster plots of activity in successive trials of the same task are summed to form the histogram below. In the record on the left the individual trials are aligned at the appearance of the stimulus. A burst of firing is closely time-locked to the stimulus. In the record on the right the trials are aligned at the beginning of the saccade. Activity is not well aligned with the beginning of the saccade and stops before the saccade itself commences.

B. Activity of a movement-related neuron in the frontal eye field. The records of each trial are aligned as in part A. The cell does not respond to appearance of the saccade target (**left**). However, it is active at the time of the saccade (**right**).

the target. The *dorsolateral prefrontal cortex* has neurons that discharge when a monkey makes a saccade to a remembered target. The activity commences with the appearance of the stimulus and continues throughout the interval during which the monkey must remember the location of the target.

We can now understand the effects of lesions of these regions on the generation of saccades. Lesions of the superior colliculus in monkeys produce only transient damage to the saccade system because the projection from the frontal eye field to the brain stem remains intact. Animals can likewise recover from cortical lesions if the superior colliculus is intact. However, when both the frontal eye field and the colliculus are damaged, the ability to make saccades is permanently compromised. The predominant effect of a parietal lesion is an attentional deficit. After recovery, however, the system can function normally because the frontal eye field signals are sufficient to suppress the substantia nigra and stimulate the colliculus.

Damage to the frontal eye field alone causes more subtle deficits. Lesions of the frontal eye field in monkeys cause transient contralateral neglect and paresis of contralateral gaze that rapidly recover. The latter deficit may reflect the loss of frontal eye field control of the substantia nigra; this loss of control means that the constant inhibitory input from the substantia nigra to the colliculus does not get suppressed, and the colliculus is unable to generate any saccades. Eventually the system adapts, and the colliculus responds to the remaining parietal signal. After recovery the animals have no trouble producing saccades to targets in the visual field but have great difficulty with memory-guided saccades. Bilateral lesions of both the frontal eye fields and the superior colliculus render monkeys unable to make saccades at all.

Humans with lesions of the frontal cortex have difficulty suppressing unwanted saccades to attended stimuli. This is easily shown by asking subjects to make an eye movement away from a stimulus. When the stimulus appears the subject must attend to it, without turning the eyes toward it, and use its location to calculate the desired saccade. Patients with frontal lesions cannot suppress the saccade to the stimulus, even though they can make normal saccades to visual targets.

As we have seen, neurons in the lateral intraparietal area of monkeys are active when the animal attends to a visual stimulus whether or not the animal makes a saccade to the stimulus. In the absence of frontal eye field signals this undifferentiated signal is the only one to reach the superior colliculus. In humans the failure to suppress a saccade is therefore to be expected if the superior colliculus responds to a parietal signal that generates attention to the stimulus, without the frontal-nigral control that normally prevents saccades in response to parietal signals.

The Control of Saccades Can Be Modified by Experience

Quantitative study of the neural control of movement is possible because the discharge rate of a motor neuron has a predictable effect on a movement. For example, a certain frequency of firing in the abducens motor neuron has a predictable effect on eye position and velocity.

This relationship can change, however, if the muscle becomes weak through disease. The brain can compensate to some degree for such changes. For example, a diabetic patient may have an abducens-nerve lesion affecting one eye and a retinal hemorrhage in the other. He is forced to use the eye with the weak lateral rectus muscle because he experiences poor vision in the eye with the normal abducens nerve. If the latter eye is patched to prevent double vision, the influence of the weak eye increases, such that the weak eye is eventually able to make accurate saccades. The influence of the patched eye also increases, causing that eye to make excessively large saccades. This is of little importance to vision, however, because the patched eye does not contribute to vision. This change in the motor response depends on the fastigial nucleus and vermis of the cerebellum.

Smooth Pursuit Involves the Cerebral Cortex, Cerebellum, and Pons

The task of the smooth-pursuit system differs from that of the saccade system. Instead of driving the eyes as rapidly as possible to a point in space, it must match the velocity of the eyes to that of a target in space. Neurons that signal eye velocity for smooth pursuit are found in the medial vestibular nucleus and the nucleus prepositus hypoglossi. They project to the abducens nucleus as well as the ocular motor nuclei in the midbrain and receive projections from the flocculus of the cerebellum.

Neurons in both the vermis and flocculus transmit an eye-velocity signal that correlates with smooth pursuit (Figure 39–14). These areas receive signals from the cerebral cortex relayed by the dorsolateral pontine nucleus. Thus lesions in the dorsolateral pons disrupt ipsilateral smooth pursuit.

There are two major cortical inputs to the smooth-pursuit system in monkeys. One arises from motion-sensitive regions in the superior temporal sulcus and the middle temporal and medial superior temporal areas. The other arises from the frontal eye field.

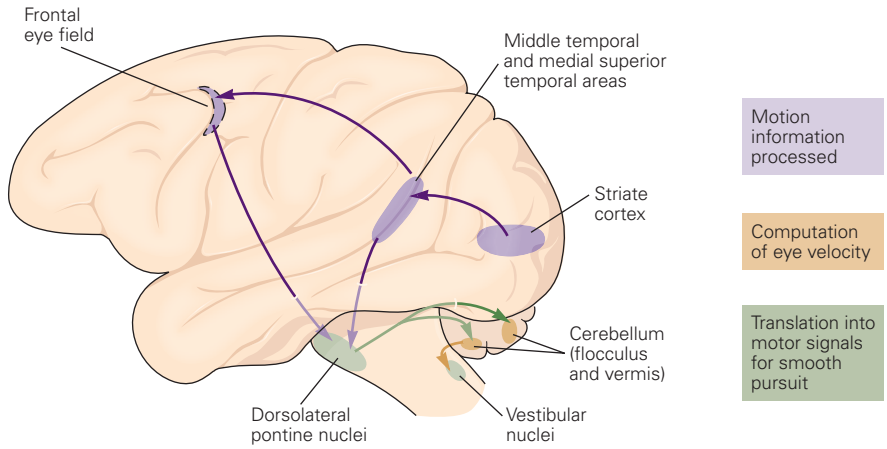


Figure 39–14 Pathways for smooth-pursuit eye movements in the monkey. The cerebral cortex processes information about motion in the visual field and sends it to the ocular motor neurons via the dorsolateral pontine nuclei, the vermis and flocculus of the cerebellum, and the vestibular nuclei. The initiation signal for smooth pursuit may originate in part from the frontal eye field. (Reproduced, with permission, from R. J. Krausliz.)

Neurons in both the middle temporal and medial superior temporal areas calculate the velocity of the target. When the eye accelerates to match the target's speed, the rate of the target's motion across the retina decreases. As the speed of the retinal image decreases, neurons in the middle temporal area, which describe retinal-image motion, stop firing, even though the target continues to move in space. Neurons in the medial superior temporal area continue to fire even if the target disappears briefly. These neurons have access to a process that adds the speeds of the moving eye and the target moving on the retina to compute the speed of the target in space.

Lesions of either the middle temporal or medial superior temporal area disrupt the ability of a subject to respond to targets moving in regions of the visual field represented in the damaged cortical area. Lesions of the latter area also diminish smooth-pursuit movements toward the side of the lesion, no matter where the target lies on the retina.

The temporal cortex provides the sensory information to guide pursuit movements but may not be able to initiate them. Electrical stimulation of either temporal area does not initiate smooth pursuit but can affect pursuit movement, accelerating ipsilateral pursuit and slowing contralateral pursuit. The frontal eye field may be more important for initiating pursuit. This area has neurons that fire in association with ipsilateral smooth pursuit. Electrical stimulation of the frontal eye field initiates ipsilateral pursuit, whereas lesions of the frontal eye field diminish, but do not eliminate, smooth pursuit.

In humans, disruption of the pursuit pathway anywhere along its course, including lesions at the level of cortical, cerebellar, and brain stem areas, prevents adequate smooth-pursuit eye movements. Instead, moving targets are tracked using a combination of defective smooth-pursuit movements, whose velocity is less than that of the target, and small saccades. Patients with brain stem and cerebellar lesions cannot pursue targets moving toward the side of the lesion.

Patients with parietal deficits have two different types of deficit. The first is a directional deficit that resembles that of monkeys with lesions of the middle superior temporal area: Targets moving toward the side of the lesion cannot be tracked. The second is a retinotopic deficit that resembles the deficit of monkeys with lesions of the medial temporal area. Normal subjects can generate smooth-pursuit eye velocity to match the velocity of a stimulus in the periphery (see Figure 39–2). Most patients cannot generate smooth pursuit of a stimulus limited to the visual hemifield opposite the lesion, regardless of the direction of motion.

Some Gaze Shifts Require Coordinated Head and Eye Movements

So far we have described how the eyes are moved when the head is still. When we look around, however, our head is moving as well. Head and eye movements must be coordinated to direct the fovea to a target.

Because the head has a much greater inertia than the eyes, a small shift in gaze drives the fovea to its target before the head begins to move. A small gaze shift usually consists of a saccade followed by a small head movement during which the vestibulo-ocular reflex moves the eyes back to the center of the orbit in the new head position (Figure 39–15). For larger gaze shifts, the eyes and the head move simultaneously in the same direction. Because the vestibulo-ocular reflex ordinarily moves the eyes in the direction opposite

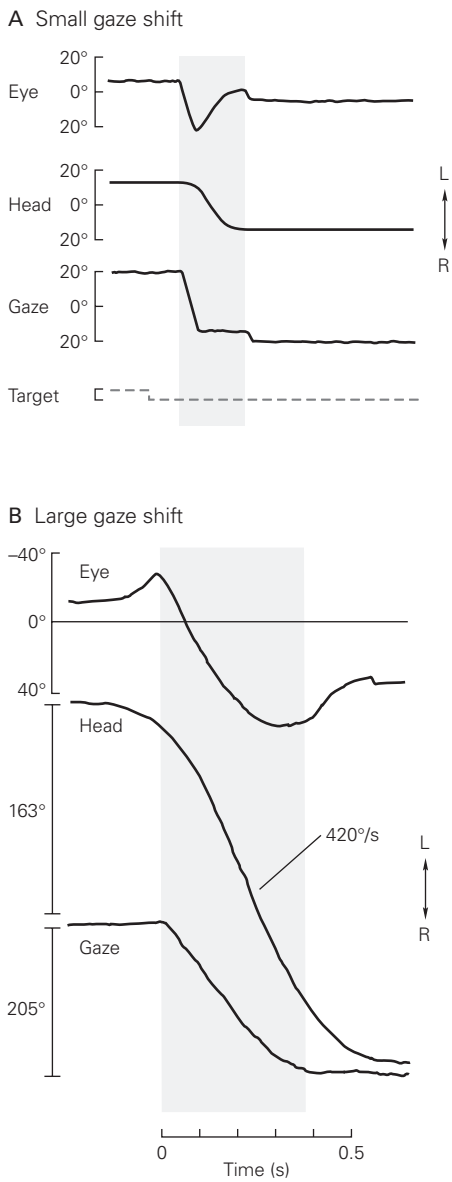


Figure 39–15 Directing the fovea to an object when the head is moving requires coordinated head and eye movements.

A. For a small gaze shift, the eye and head move in sequence. The eye begins to move 300 ms after the target appears. Near the end of the eye movement, the head begins to move as well. The eye then rotates back to the center of the orbit to compensate for the head movement. The gaze record is the sum of eye and head movements. (Reproduced, with permission, from Zee 1977.)

B. For a large gaze shift the eye and head move in the same direction simultaneously. Near the end of the gaze shift the vestibulo-ocular reflex returns, the eye begins to compensate for head movement as in A, and gaze becomes still. (Reproduced, with permission, from Laurutis and Robinson 1986.)

that of the head, the reflex must be temporarily suppressed for the eyes and head to move simultaneously.

Many of the neural centers that control simple saccades also control gaze shift. Electrical stimulation of the superior colliculus in a monkey with its head fixed evokes saccades, but stimulation of an animal whose head can move freely results in saccades combined with head movement. Neurons in the superior colliculus that carry eye-movement signals also project to neurons in the reticular formation that drive the neck muscles, presumably enabling a combined head and eye movement to position the fovea on an object of interest.

An Overall View

The oculomotor system provides a valuable window into the nervous system for both the clinician and the scientist. Patients with oculomotor deficits experience double vision, an alarming symptom that quickly sends them to seek medical help. A physician with a thorough knowledge of the oculomotor system can describe and diagnose most oculomotor deficits at the bedside and localize the site of the lesion within the brain based on the neuroanatomy and neurophysiology of eye movements. Much of our understanding of neural processes arises from our knowledge of the oculomotor system as a microcosm of human behavior.

The cerebral cortex chooses significant objects in the environment as targets for eye movements. Cortical signals are relayed to motor circuits in the brain stem by the superior colliculus. The cortical and collicular signals do not specify the contribution of each muscle to the movement. Instead, the motor programming for eye movements is performed in the brain stem, which translates the signals from higher centers into signals appropriate for each muscle. The cerebellum plays an important role in calibrating eye-muscle movement.

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Mark F. Walker

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