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The Control of Gaze

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Highlights

IN PRECEDING CHAPTERS, WE LEARNED about the motor systems that control the movements of the body in space. In this and the next chapter, we consider the motor systems that control our gaze, balance, and posture as we move through the world around us. In examining these motor systems, we will focus on three biological challenges that these systems resolve: 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?

In this chapter, we describe the oculomotor system and how it uses visual information to guide eye movements. It is one of the simplest motor systems, requiring the coordination of only the 12 evolutionarily old muscles that move the two eyes. In humans and other primates, the primary objective of the oculomotor system is to control the position of the fovea, the central point in the retina that has the highest density of photoreceptors and thus the sharpest vision. The fovea is less than 1 mm in diameter and covers less than 1% of the visual field. When we want to examine an object, we must move its image onto the fovea (Chapter 22).

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 where the eyes are looking.

Horizontal rotation of the eye away from the nose is called *abduction*, and rotation toward the nose is *adduction* (Figure 35–1A). Vertical movements are referred to as *elevation* (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).

Most eye movements are conjugate; that is, both eyes move in the same direction. These eye movements are called *version* movements. 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. When you change your gaze from far to near, the eyes move in opposite directions—both eyes adduct. These movements are called *vergence* movements.

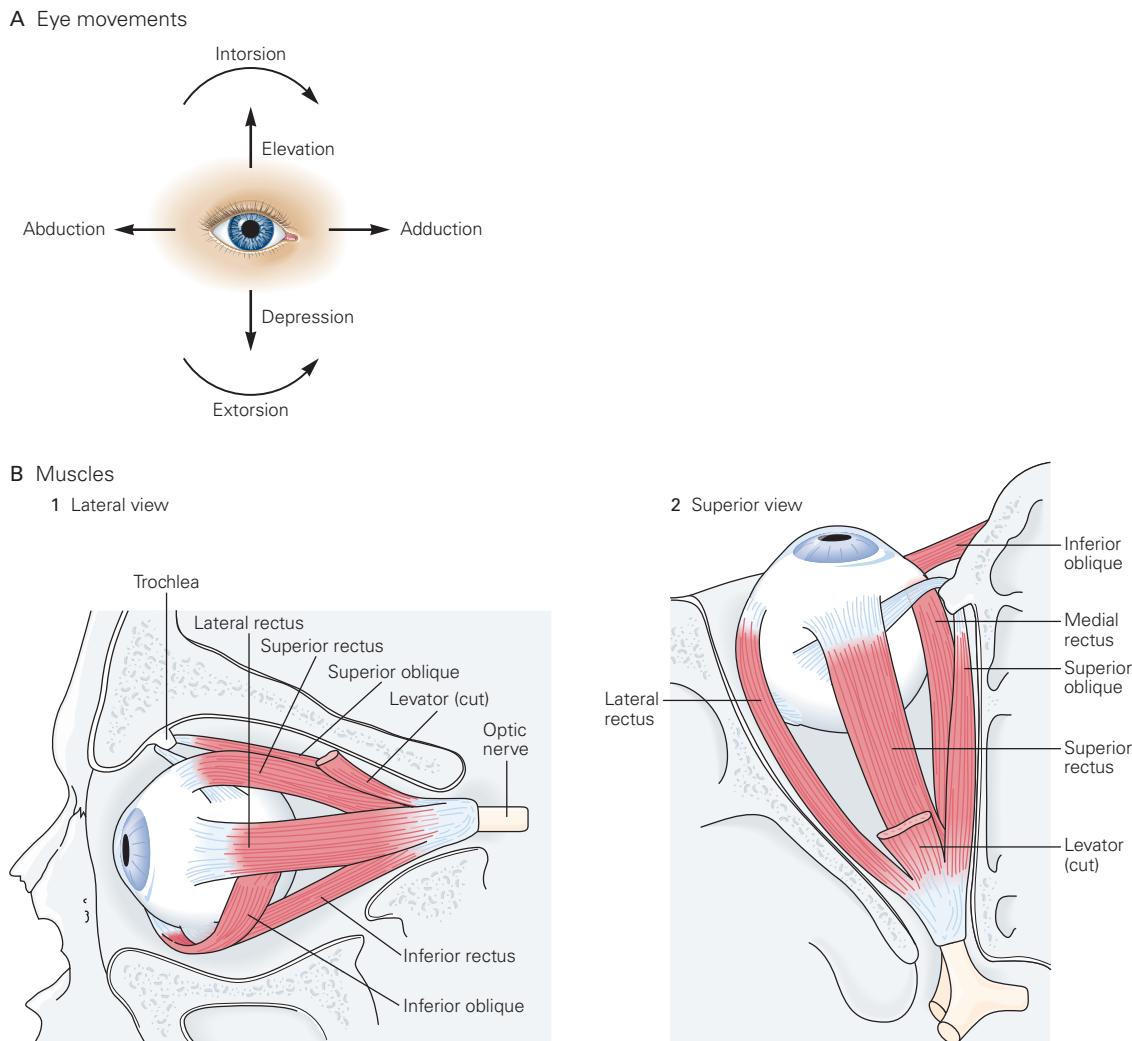


Figure 35–1 The different actions of eye movements and the muscles that control them.

A. View of the left eye and the three dimensions of eye movement.

B. 1. 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 tendon passes through the trochlea, a bony pulley on the nasal side of the orbit, before it inserts on the globe. The levator muscle of the upper eyelid raises the lid. 2. 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.

The Six Extraocular Muscles Form Three Agonist–Antagonist Pairs

Each eye is rotated by six extraocular muscles arranged in three agonist–antagonist pairs (Figure 35–1B). 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 center of the eye, so the superior rectus elevates the eye and the inferior rectus depresses it. 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 anteromedial wall of the orbit. The oblique muscles insert posterior to the center of the eye, so the superior oblique depresses the eye and the inferior oblique elevates it.

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 as a result of these pulleys.

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 superior and inferior recti and the obliques are often called the cyclovertical muscles because they produce both vertical and torsional eye rotation. The relative amounts of each rotation 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, while the oblique muscles exert their maximal vertical action when the eye is adducted (Figure 35–2).

Movements of the Two Eyes Are Coordinated

Humans and other frontal-eyed animals have binocular vision—the fields of vision of the two eyes overlap. 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

A In adduction, the superior oblique depresses the eye



B In abduction, the superior oblique intorts the eye

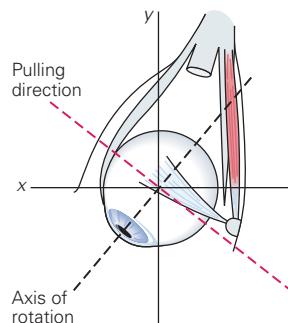
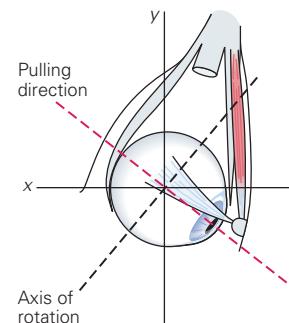


Figure 35–2 The effect of orbital position on the action of the superior oblique muscle.

- When the eye is adducted (looking toward the nose), contraction of the superior oblique depresses the eye.
- When the eye is abducted (looking away from the nose), contraction of the superior oblique intorts the eye.

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, while the right inferior rectus and the left superior oblique both move the eyes downward in right gaze (Table 35–1).

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 the three oculomotor nuclei in the brain stem (Figure 35–3).

Table 35–1 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

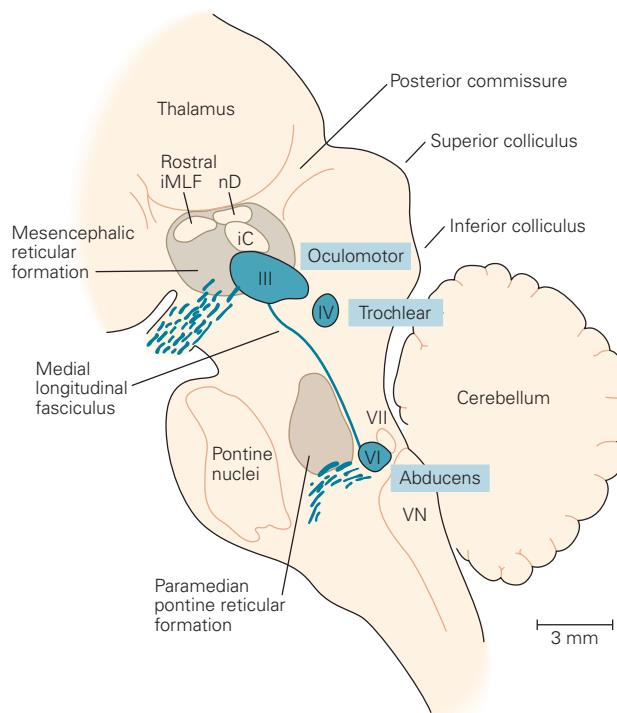


Figure 35–3 The oculomotor nuclei in the brain stem. The nuclei are shown in a parasagittal section through the thalamus, pons, midbrain, and cerebellum of a rhesus monkey. The oculomotor nucleus (cranial nerve III) lies in the midbrain at the level of the mesencephalic reticular formation; the trochlear 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 with Figure 40–5. (Abbreviations: iC, interstitial nucleus of Cajal; iMLF, interstitial nucleus of the medial longitudinal fasciculus; nD, nucleus of Darkshevich; VN, vestibular nuclei.) (Adapted from Henn, Hepp, and Büttner-Ennever 1982.)

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 contralateral 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.)

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. Superior rectus axons cross the midline and join the contralateral oculomotor nerve. Thus, both superior rectus and superior oblique motor neurons innervate their respective muscles on the opposite side. 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, which constricts the pupil, and the ciliary muscles that adjust the curvature of the lens to focus the eye during vergence movements from far to near, the process of 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 cavernous 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 35–1).

The force generated by an extraocular muscle is determined both by the firing rate of the motor neurons and the number of motor units recruited. Like the motor units for skeletal muscle (Chapter 31), eye motor units are recruited in a fixed sequence. For example, as the eye moves laterally, the number of active abducens neurons and their individual firing rates both increase, thereby increasing the strength of lateral rectus contraction.

Box 35-1 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 left trochlear nerve (IV) lesion affects both torsional and vertical eye movements by weakening the superior oblique muscle (Figure 35-4). Vertical misalignment in superior oblique paresis is also affected by the position of the head. A tilt to one side, such that the ear moves toward the shoulder, induces a small torsion of the eye in the opposite direction, known as ocular counter-roll. For example, 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 inferior rectus and right inferior oblique. In the left eye, the elevation action of the superior rectus is canceled by the depression action of the superior oblique, so the eye only rotates about the line of sight. When the head tilts to the right, the inferior oblique and inferior rectus extort the left eye and the superior oblique and the superior rectus relax.

With paresis of the left superior oblique, the elevating action of the superior rectus is unopposed when the head tilts to the left such that the left eye moves further upward. In contrast, tilting the head to the right relaxes

the superior rectus and superior oblique (Figure 35-4D). Thus, patients with trochlear nerve lesions often prefer to keep their heads tilted away from the affected eye because this reduces the misalignment and can eliminate diplopia.

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 cannot move medially or upward. Downward movement is also affected because the inferior rectus muscle is weak; because the eye is abducted, the primary action of the intact superior oblique is intorsion rather than depression.

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 leads to Horner syndrome, which includes 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.

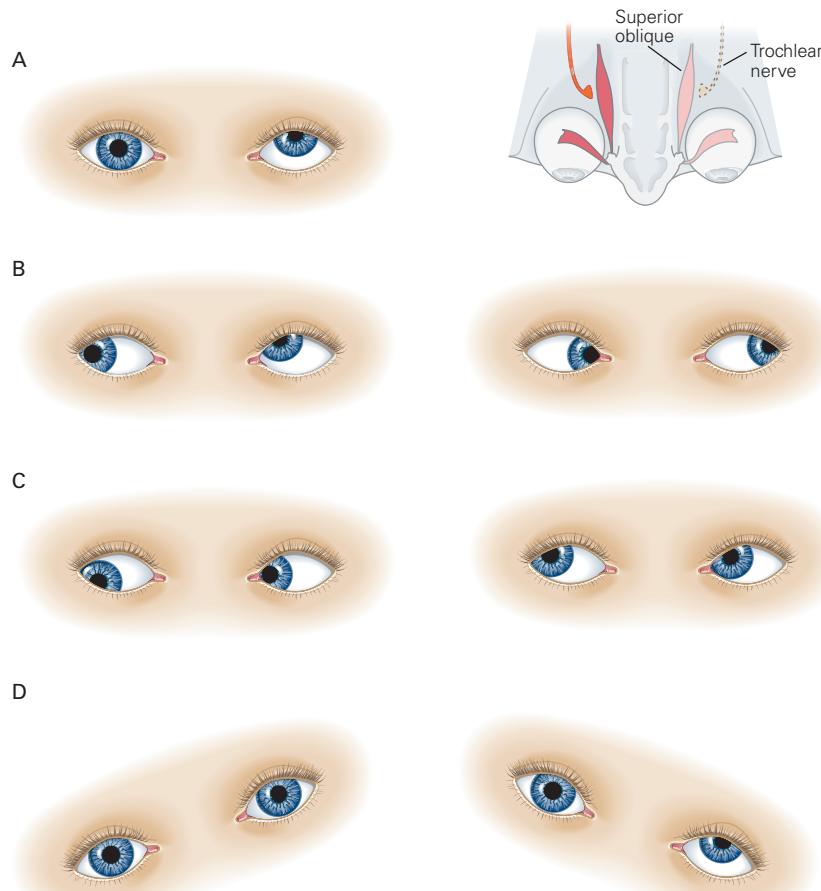


Figure 35-4 Effect of a left trochlear nerve palsy. The trochlear nerve innervates the superior oblique muscle, which inserts behind the equator of the eye. It depresses the eye when it is adducted and intorts the eye when it is abducted.

A. Hypertropia, a permanent upward deviation of the eye, can be seen when a patient is looking straight ahead. The right eye is in the center of the orbit, but the affected 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 (Chapter 27). 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.

Six Neuronal Control Systems Keep the Eyes on Target

The oculomotor nuclei are the final common targets for all types of eye movements generated by higher brain networks. Hermann Helmholtz and other 19th-century psychophysicists appreciated that analysis of eye movements was essential for understanding visual perception, but they assumed that all eye movements were smooth. In 1890, Edwin Landolt discovered that during reading the eyes do not move smoothly along a line of text but make fast intermittent movements called saccades (French, jerks), each followed by a short pause.

By 1902, Raymond Dodge outlined 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 oculomotor nuclei 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 of an object of interest is positioned on both foveae regardless of its distance.
- Vestibulo-ocular reflexes stabilize images on the retina during brief head movements.
- Optokinetic movements stabilize images 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 by actively suppressing eye movement. The optokinetic and vestibular systems are discussed in Chapter 27. We consider the other four systems here.

An Active Fixation System Holds 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, patients with irrepressible saccadic eye movements (opsoclonus)—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 (Chapter 25) (Figure 35–5). Saccades allow us to scan the environment quickly and to read. Highly stereotyped, they have a standard waveform with a single smooth increase and decrease of eye velocity. Saccades are also extremely fast, occurring within a fraction of a second at angular speeds up to 900° per second (Figure 35–6A). The velocity of a saccade is determined only by its size. We can voluntarily change

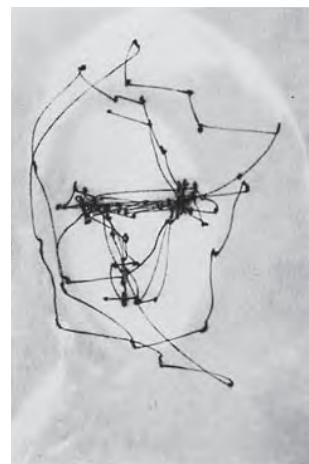


Figure 35–5 Eye movements track the outline of an object of attention. An observer looks at a picture of a woman for 1 minute. The resulting eye positions are then superimposed on the picture. As shown here, the observer concentrated on certain features of the face, lingering over the woman's eyes and mouth (*fixations*) and spending less time over intermediate positions. The rapid movements between fixation points are *saccades*. (Reproduced, with permission, from Yarbus 1967.)

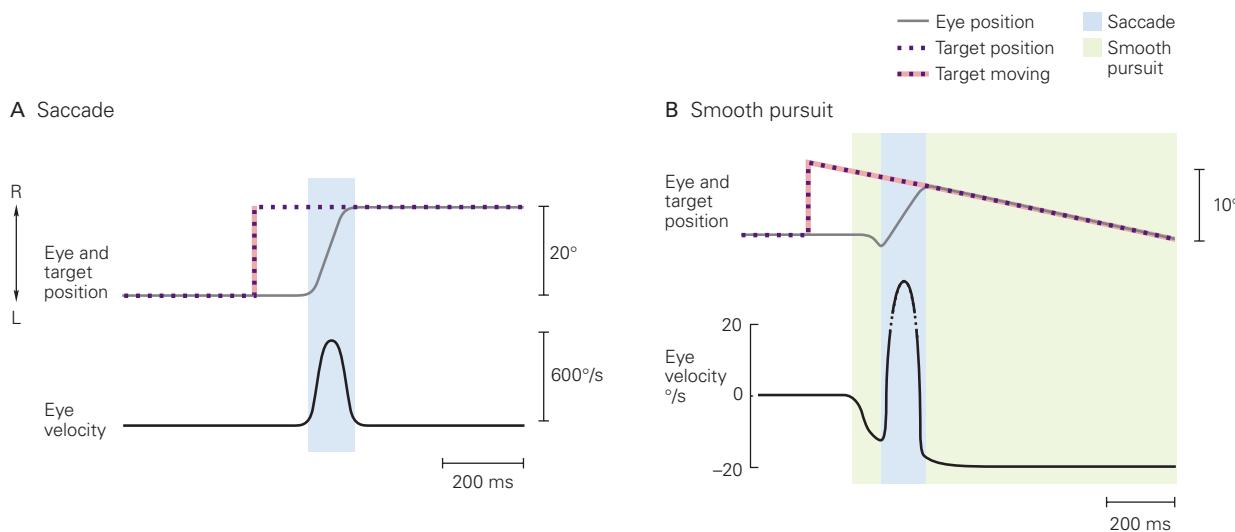


Figure 35-6 Saccadic and smooth-pursuit eye movements. Eye position, target position, and eye velocity are plotted against time.

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°. A saccade from one edge of the thumb to the other therefore traverses 1° of arc. (Abbreviations: L, left; R, right.)

the amplitude and direction of saccades but not their speed, although fatigue, drugs, or pathological states can slow saccades.

Ordinarily, there is no time for visual feedback to modify the course of a saccade as it is being made; instead, corrections to the direction and/or amplitude of movement are made over the course of successive 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 (eg, "look left").

When a saccade is made, the activity of neurons in higher brain centers that control gaze specify only a desired change in eye position (eg, 20° to the right of current gaze, usually based on a target location in the visual field). For the eye movement to be made, this location signal must be transformed into signals for the eye muscles that execute the desired velocity and change in eye position. We can illustrate how the gaze system generates eye movements by considering the activity of an oculomotor neuron during a saccade (Figure 35-7A). To move the eye quickly to a new position in the orbit and keep it there, two passive forces

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.

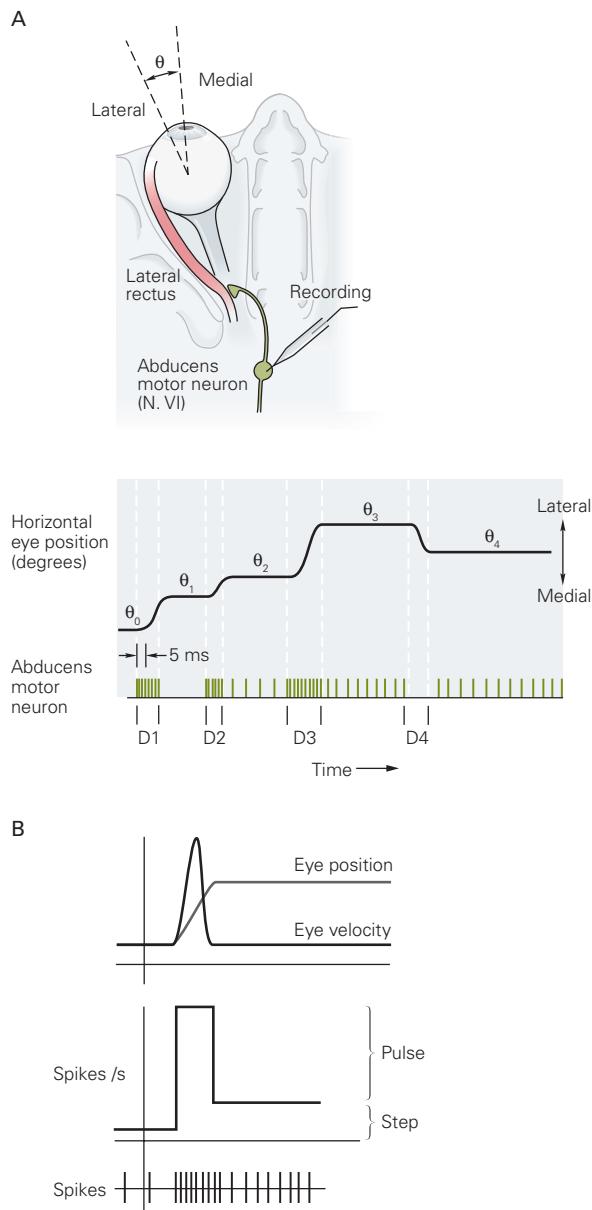
must be overcome: the elastic force of the orbital tissues, which tends to restore the eye to a central position, and a velocity-dependent viscous force that opposes rapid movement. Thus, the motor signal for an eye movement must include both a position component to counter the elastic force and a velocity component to overcome orbital viscosity and move the eye quickly to the new position.

This eye position and velocity information are coded by the discharge frequencies of oculomotor neurons. When a saccade is made, the firing rate of a neuron rises rapidly as eye velocity increases; this is called the *saccadic pulse* (Figure 35-7B). The frequency of this pulse determines the speed of the saccade, whereas the length of the pulse controls the duration of the saccade and thus its amplitude. When the saccade is completed and the eye has reached its goal, there must be a new level of tonic input to the eye muscles that is appropriate for the elastic restoring force at that orbital position. This difference in the tonic firing rate between before and after the saccade is called the *saccadic step* (Figure 35-7B). If the size of the step is not properly matched to

Figure 35–7 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_3). 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 (θ_4) associated with a tonic discharge. (Adapted from Fuchs and Luschei 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).



the pulse, then the eye drifts away from the target after the saccade. As described later, the pulse and step are generated by different brain stem structures.

The Motor Circuits for Saccades Lie in the Brain Stem

Horizontal Saccades Are Generated in the Pontine Reticular Formation

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

adjacent to the abducens nucleus to which it projects (Figure 35–8A). 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 (toward the same side as the discharging neurons), and their activity resembles the pulse component of oculomotor neuron discharge (Figure 35–7B).

There are several types of burst neurons (Figure 35–8B). Medium-lead excitatory burst neurons make direct excitatory connections to motor neurons and interneurons in the ipsilateral abducens nucleus.

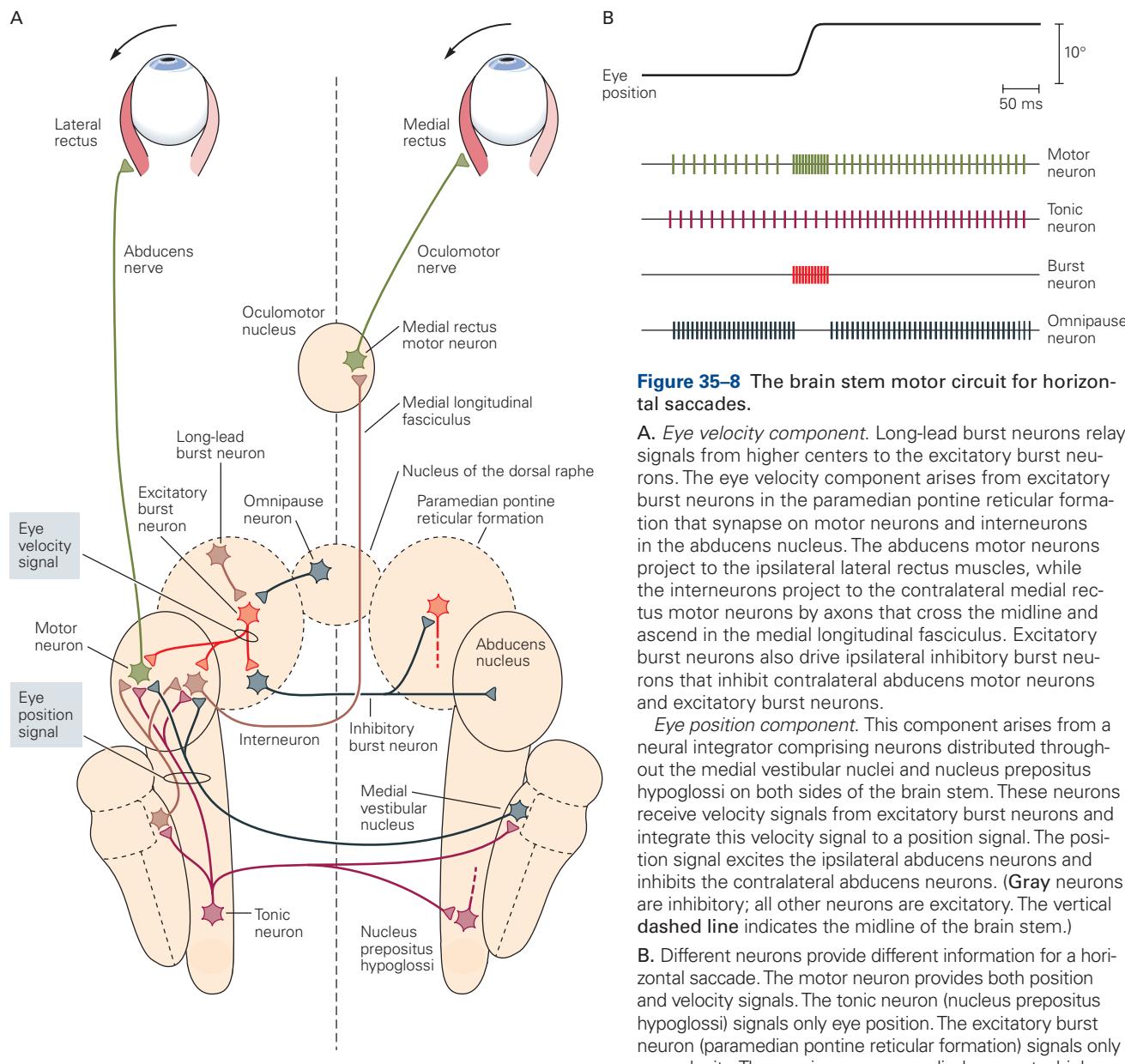


Figure 35–8 The brain stem motor circuit for horizontal saccades.

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, while 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 indicates 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.

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*, fires continuously except around the time of a saccade; firing ceases shortly before and during all saccades

(Figure 35–8B). Omnipause neurons are located in the nucleus of the dorsal raphe in the midline (Figure 35–8A). They are GABAergic (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 after a saccade, because there would be no new position signal to hold the eyes against elastic restorative forces. The appropriate tonic innervation is required to keep the eye at the new orbital position. This tonic position signal, the saccadic step, can be generated from 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 nucleus prepositus hypoglossi (Figure 35–8A) 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. Moreover, integration of the horizontal saccadic burst requires coordination of the bilateral nuclei prepositi hypoglossi and medial vestibular nuclei through commissural connections. Thus, a midline lesion of these connections also 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 35–8A).

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 susceptibility of the medial longitudinal fasciculus to strokes and multiple sclerosis make it clinically important.

Several cerebellar structures play an important role in the calibration of the saccade motor signal. First, the oculomotor portion of the dorsal vermis, acting through the caudal fastigial nucleus, controls the duration of the pulse and thus the accuracy of the saccade. The fastigial nucleus increases saccade velocity at the beginning of contraversive saccades and contributes to

braking ipsiversive saccades to end the saccade. Second, the flocculus and paraflocculus of the vestibulocerebellum calibrate the neural integrator to ensure that the step is properly matched to the pulse, in order to hold the eyes at the new position after each saccade.

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 (Figure 35–3). Vertical and torsional neural integration are performed in the nearby interstitial nucleus of Cajal. The pontine and mesencephalic systems participate together 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 via the posterior commissure. 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 contraversive and vertical saccades. A lesion of the abducens nucleus has a similar effect, as 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 35–8A). 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 typically 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.

A lesion of the cerebellar fastigial nucleus causes ipsiversive saccades to overshoot their targets (*hypermetric saccades*), due to failure of normal termination of the saccadic burst. Contraversive saccades undershoot their targets (*hypometric saccades*). Correspondingly, damage to the oculomotor vermis disinhibits the fastigial nucleus and causes hypometric ipsiversive saccades. This may be due to an additional failure to compensate for the position-dependent passive forces of the orbital tissues.

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 extraocular muscles for saccades. However, among higher

mammals, eye movements are ultimately driven by cognitive behavior. The decision of 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 35–9).

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

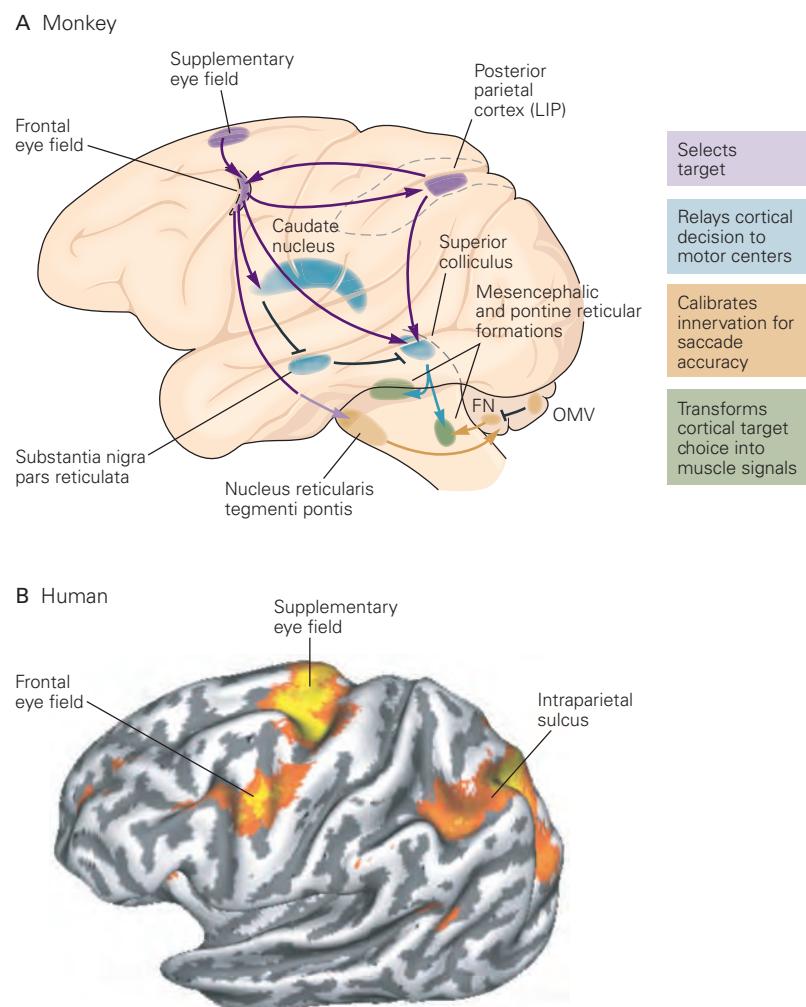
The superior colliculus in the midbrain is a major visuomotor integration region, the mammalian homolog of the optic tectum in nonmammalian vertebrates. It can be divided into two functional regions: the superficial layers and the intermediate and deep layers.

The three superficial layers receive both direct input from the retina and a projection from the striate

Figure 35–9 Cortical pathways for saccades.

A. In the monkey, the saccade generator in the brain stem receives a command from the superior colliculus. That command is relayed through the pontine and mesencephalic burst circuits, providing the motor signals that drive the extraocular muscles for saccades. 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. The oculomotor vermis (OMV) of the cerebellum, acting through the fastigial nucleus (FN), calibrates the burst to keep saccades accurate.

B. This lateral scan of a human brain shows areas of cortex activated during saccades. (Adapted from Curtis and Connolly 2008.)



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. Neurons in the superficial layers of the superior colliculus are functionally arranged in a retinotopic map of the visual field in which representation of the visual field closest to the fovea occupies the largest area (Figure 35–10).

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, active animals. Edward Evarts pioneered this approach in studies of the skeletomotor system, after which it was extended to the oculomotor 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 35–11A). 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. There is a map of movement fields in the intermediate layers that is in register with the map of visual receptive fields in the overlying superficial layers. Each movement neuron discharges before a saccade to the center of the overlying visual receptive field. A map of saccades evoked by electrical stimulation of the intermediate layers resembles the visual map.

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 many sensory systems (Chapter 17) and the skeletal motor system (Chapter 34).

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

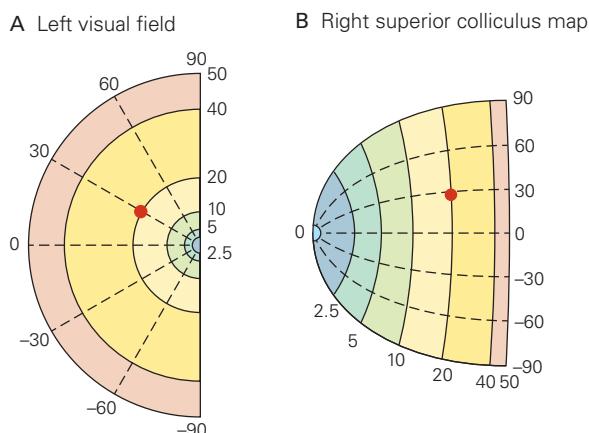


Figure 35–10 Neurons in the superior colliculus are organized in a retinotopic map.

- A. Map of the left visual field in polar coordinates. Dashed lines represent the angle and solid lines the eccentricity.
- B. Spatial map of neurons in the superior colliculus represented in polar coordinates of the visual field. In the nucleus, more neurons represent the part of the visual field close to the fovea and fewer neurons represent the periphery. For example, a stimulus appearing at 20° eccentricity and 30° elevation in the visual field (red dot) will excite neurons at the location of the red dot on the collicular map. (Reproduced, with permission, from Quaia et al. 1998.)

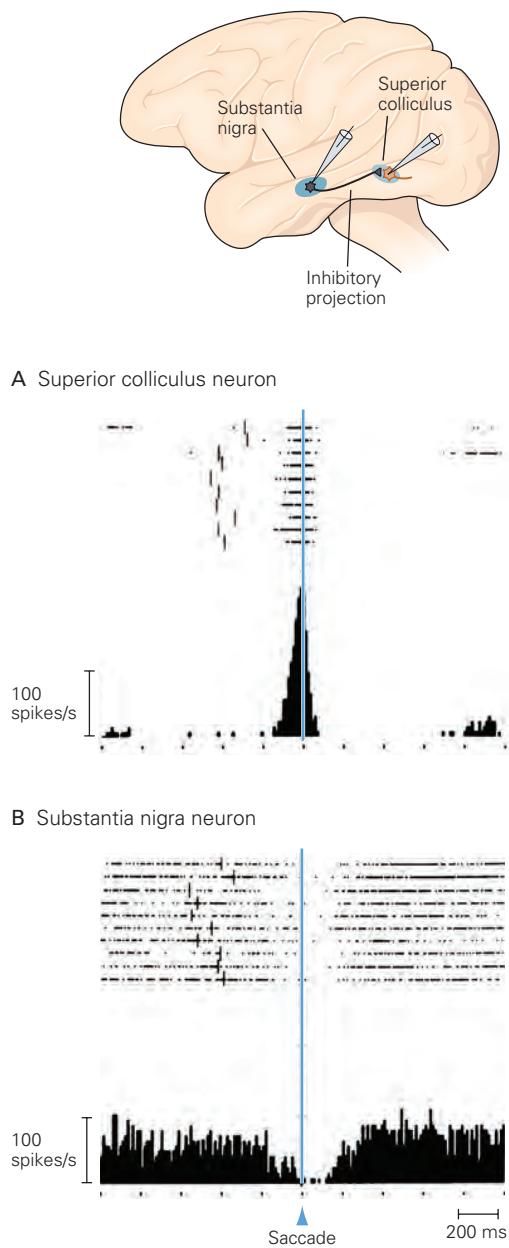


Figure 35–11 Neurons in the superior colliculus and substantia nigra are active around the time of a saccade. (Reproduced, with permission, from Hikosaka and Wurtz 1989.)
A. A neuron recorded from the region in the superior colliculus from which the neuron in B could be excited antidromically fires in a burst immediately before the saccade. Raster plots of activity in successive trials of the same task are summed to form the histogram below. The small vertical lines in the raster indicate target appearance. The trials are aligned at the beginning of the saccade (blue line).
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.

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 contraversive saccades, although with time, this ability is recovered.

The Rostral Superior Colliculus Facilitates Visual Fixation

The most rostral portion of the superior colliculus receives inputs from the fovea and the foveal representation in primary visual cortex (V1). 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 and Two Regions of Cerebral Cortex Control the Superior Colliculus

The superior colliculus receives a powerful GABAergic inhibitory projection from neurons in the substantia nigra, which fire spontaneously with high frequency. This discharge is suppressed at the time of voluntary eye movements to the contralateral visual field (Figure 35–11B) by inhibitory input from neurons in the caudate nucleus, which fire before saccades to the contralateral visual field.

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 of attended objects in the visual field is better than perception of unattended objects, 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 barely

noticeable. Saccadic eye movements and visual attention are closely intertwined (Figure 35–5).

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 former location of the vanished object. Neurons in the lateral intraparietal area respond from the moment the object appears and continue firing after the object has vanished and throughout the delay until the saccade begins (Figure 35–12A), 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 35–12B).

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 ipsilateral 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 (Chapter 59).

Patients with Balint syndrome, which is usually the result of bilateral lesions of the posterior parietal

A Neuron fires from appearance of target until saccade

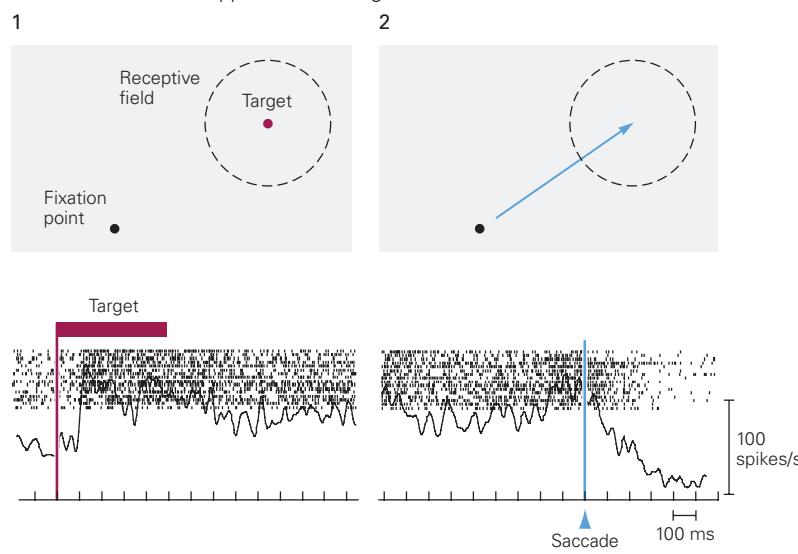
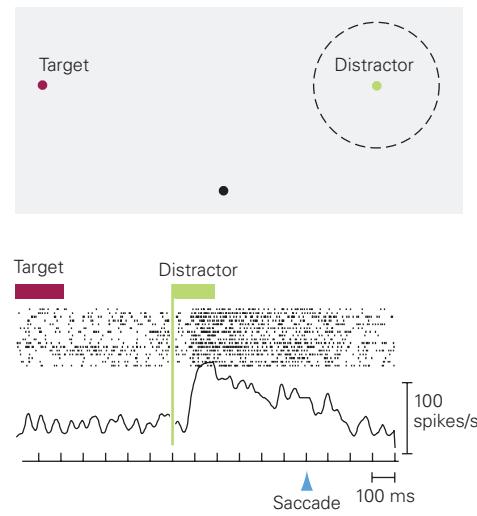


Figure 35–12 A parietal neuron is active before memory-guided saccades. Traces are aligned at events indicated by vertical lines. (Adapted, with permission, from Powell and Goldberg 2000.)

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).

B Neuron responds as powerfully to distractor in receptive field



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 initially to a distractor in the receptive field as strongly as it did to the target of a saccade.

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 35–13A). 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 saccades to their movement fields. 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 behavior (Figure 35–13B). 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.

Movement-related neurons in frontal eye field control the superior colliculus through two pathways. They excite the superior colliculus directly and they release it from the inhibitory influence of the substantia nigra by exciting the caudate nucleus, which in turn inhibits the nigra (Figure 35–9A). The frontal eye field also projects to the pontine and mesencephalic reticular formations, although not directly to the burst cells.

Two other cortical regions besides LIP that have 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 spatial information other than the direction of the desired eye movement. 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 the

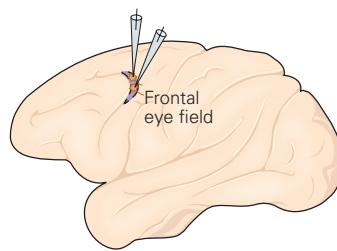
target. The dorsolateral prefrontal cortex has neurons that discharge when a monkey makes a saccade to a remembered target. The activity commences with the appearance of the stimulus and continues throughout the interval during which the monkey must remember the location of the target.

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.

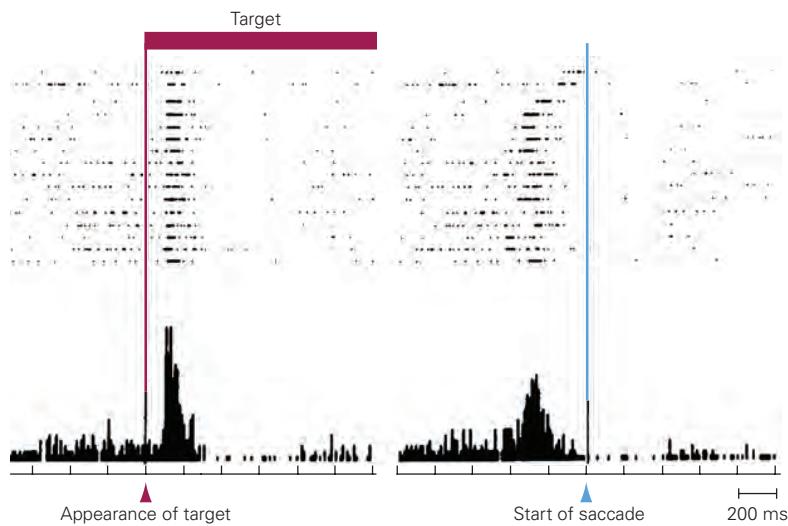
Damage to the frontal eye field alone causes more subtle deficits. Lesions of the frontal eye field in monkeys cause transient contralateral neglect and paresis of contraversive gaze, which recover rapidly. The latter deficit may reflect the loss of frontal eye field control of the substantia nigra; 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, the "anti-saccades task." For example, if a stimulus appears on the left, the subject should make a saccade of the same size to the right. To do this, the subject must attend to the stimulus, without turning the eyes toward it, and use its location to calculate the desired saccade to the opposite direction. Patients with frontal lesions have great difficulty suppressing the unwanted saccade to the stimulus.

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



A Visual neuron responds to the stimulus and not to movement



B Movement-related neuron responds before movement but not to stimulus

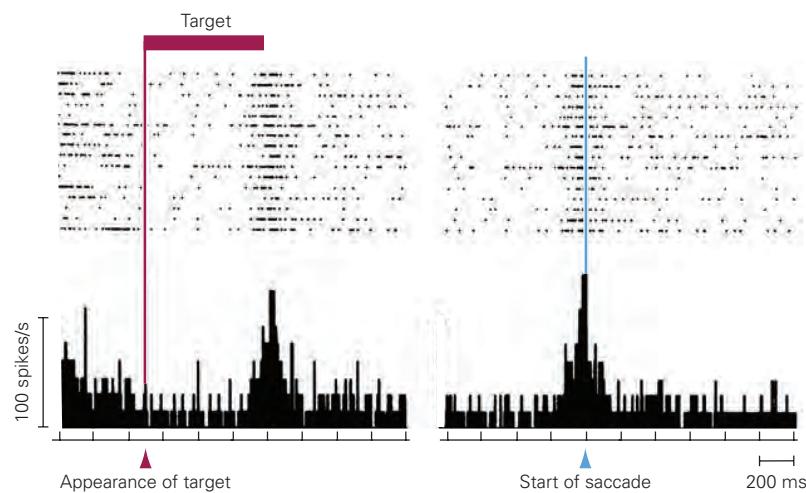


Figure 35–13 Visual and movement-related neurons in the frontal eye field. (Reproduced, with permission, from Bruce and Goldberg 1985.)

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*) but is active at the time of the saccade (*right*).

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 if disease damages an oculomotor nerve or causes an eye muscle to become weak, although the brain can compensate to some degree for such changes. Guntram Kommerell described a case that dramatically illustrates this point. A diabetic patient had an acute partial abducens nerve lesion affecting one eye and a retinal hemorrhage in the other. Because of the poor vision in the eye with a normal abducens nerve, he ordinarily used the eye with the newly weakened lateral rectus muscle. After a few days, the eye recovered the ability to make fairly accurate eye movements. When the weak eye was patched and the subject attempted to make a saccade with the visually poor eye, the saccade overshot the target. This implies that in order to compensate for the weakness of the visually normal eye the brain increased the neural signal to both eyes, resulting in too large a signal to the eye with normal motor input. This change in the motor response depends on the fastigial nucleus and vermis of the cerebellum (Figure 35–9A) and results from the visual system signaling that the preceding eye movement was inaccurate.

Some Rapid 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 35–14). 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 that of the head, the reflex must be temporarily suppressed.

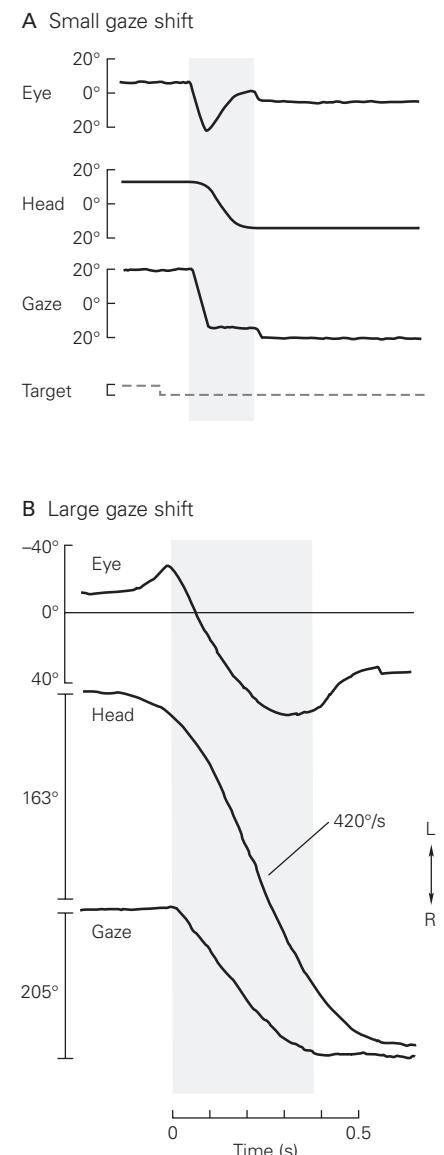


Figure 35–14 Directing the fovea to an object when the head is moving requires coordinated head and eye movements.

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. (Abbreviations: L, left; R, right.) (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 part A, and gaze becomes still. (Reproduced, with permission, from Lauritis and Robinson 1986.)

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° per second, much 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. 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. Therefore, as the target moves back toward the center of gaze, the smooth-pursuit system briefly moves the eye away from the target before the saccade is initiated (Figure 35–6B). The subsequent saccade then brings the eye to the target. Neurons that signal eye velocity for smooth pursuit are found in the medial vestibular nucleus and the nucleus prepositus hypoglossi. They receive projections from the flocculus of the cerebellum and project to the abducens nucleus as well as the oculomotor nuclei in the midbrain.

Neurons in both the flocculus and vermis transmit an eye-velocity signal that correlates with smooth pursuit. These areas receive signals from the cerebral cortex relayed by the dorsolateral pontine nucleus (Figure 35–15). Thus, lesions in the dorsolateral pons disrupt ipsiversive smooth pursuit.

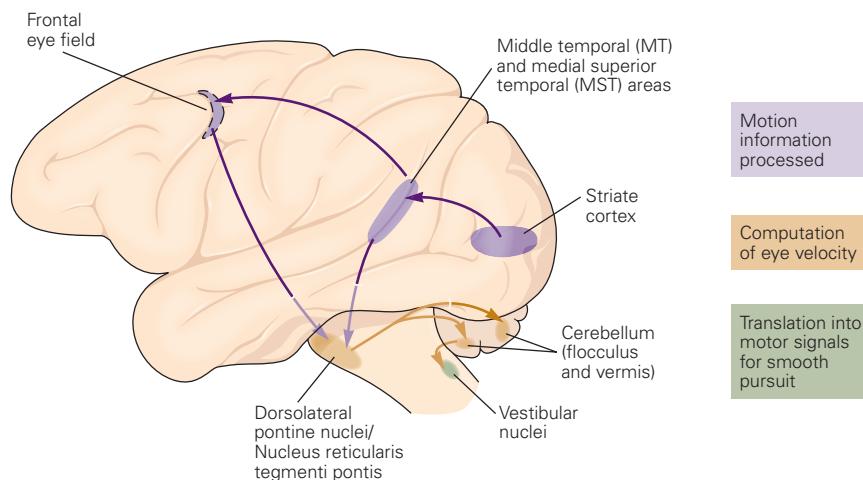
Figure 35–15 Cortical pathways for smooth-pursuit eye movements in the monkey. The cerebral cortex processes information about motion in the visual field and sends it to the oculomotor neurons via the dorsolateral pontine nuclei, the vermis and flocculus of the cerebellum, and the vestibular nuclei. The initiation signal for smooth pursuit may originate in part from the frontal eye field.

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.

The middle temporal and medial superior temporal areas were named because of their position in sulcus-free cortex of the owl monkey, a New World monkey. In humans and Old World monkeys, these areas lie in the superior temporal sulcus, at the junction between the occipital and parietal lobes. 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, whose activity signals 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 two motion-selective areas provide the sensory information to guide pursuit movements but may not be able to initiate them. Electrical stimulation of either area does not initiate smooth pursuit but can affect pursuit movement, accelerating ipsiversive pursuit and slowing contraversive pursuit. The frontal eye field may



be more important for initiating pursuit. This area has neurons that fire in association with ipsiversive smooth pursuit. Electrical stimulation of the frontal eye field initiates ipsiversive pursuit, whereas lesions of this area 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 (the 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.

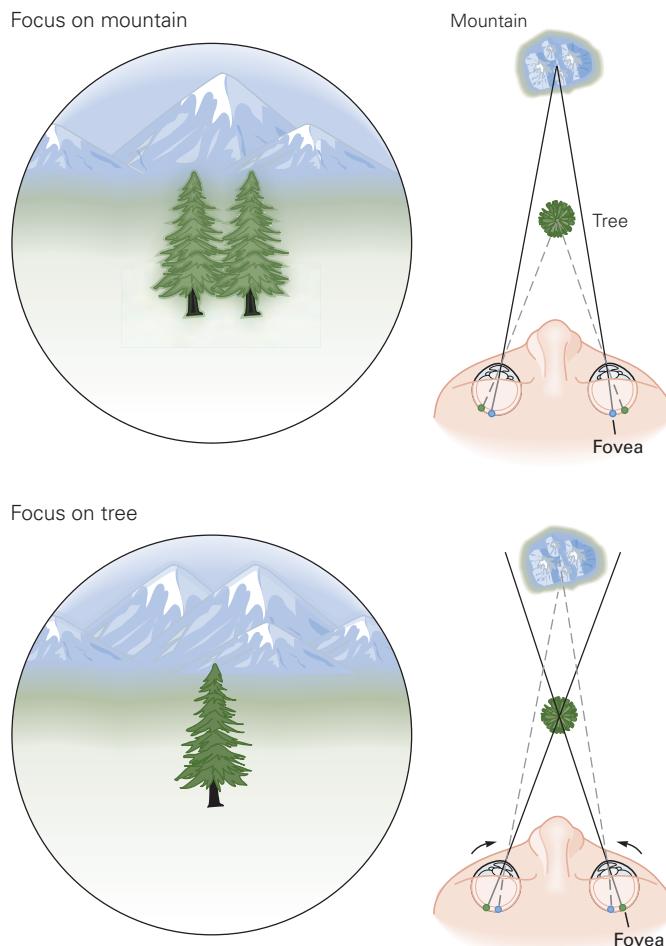
Patients with parietal deficits that include the motion-sensitive areas have two different types of deficit. The first is a directional deficit that resembles that of monkeys with lesions of the medial superior temporal area: targets moving toward the side of the lesion cannot be tracked. The second is a retinotopic deficit that resembles the deficit of monkeys

with lesions of the middle temporal area: There is an impairment of smooth pursuit of a stimulus limited to the visual hemifield opposite the lesion, regardless of the direction of motion.

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 *converge* or rotate toward each other; when we look at an object that is farther away, they *diverge* or rotate away from each other (Figure 35–16). 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

Figure 35–16 Vergence movements. When the eyes focus on a distant mountain, images of the mountain lie on the foveae, while those of the tree in the forefront occupy different retinal positions, yielding the percept of a double image. When the viewer looks instead at the tree, the vergence system must rotate each eye inward. Now the tree's image occupies similar positions on both foveae 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.)



sense of depth, vergence movements eliminate retinal disparity at the fovea.

Vergence is a function of the horizontal rectus muscles only, because the two eyes are horizontally, not vertically, displaced. Convergence of the eyes for near-field viewing is accomplished by simultaneously increasing the tone of the medial recti muscles and decreasing the tone of the lateral recti muscles to converge the eyes. Conversely, distance viewing is accomplished by reducing the tone of the medial rectus and increasing the tone of the lateral rectus.

At any given time, the entire visual field is not in focus on the retina. When we look at something nearby, 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*. With age, 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*. 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.

Highlights

1. The oculomotor system provides a valuable window into the nervous system for both the clinician and the scientist. Patients with oculomotor deficits may experience alarming symptoms such as double vision that quickly send 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.
2. The purpose of eye movements is to rotate the eye in the orbit in order to direct the fovea, the area of the retina with best acuity, to the point of greatest interest in the visual scene and then to keep the image steady.
3. Six muscles work together to move each eye. These eye muscles are yoked in three pairs. The lateral rectus abducts the eye horizontally, and the medial rectus adducts it. The cyclovertical eye muscles move the eye both vertically and torsionally.
4. Motor neurons for the extraocular muscles lie in three brainstem nuclei. The abducens nucleus in the pons contains the neurons for the lateral rectus. The other oculomotor neurons are in the midbrain: The trochlear nucleus contains superior oblique neurons, and the oculomotor nucleus has the motor neurons for the medial, superior, and inferior rectus muscles and the inferior oblique muscle. Neurons that constrict the pupil and those that elevate the eyelid also lie in the oculomotor nucleus.
5. There are six different types of eye movements, with different control systems: (1) Saccades shift the fovea rapidly to a new visual target. (2) Smooth-pursuit movements keep the image of a moving object on the fovea. (3) Vergence movements rotate the eyes in opposite directions so that the image of an object of interest is positioned on both foveae regardless of its distance. (4) Vestibulo-ocular reflexes hold images still on the retina during brief, rapid head movements. (5) Optokinetic movements hold images stationary during sustained or slow head movements. (6) Fixation is an active process that keeps the eye still during intent gaze when the head is not moving.
6. The firing pattern of eye muscle neurons combines independent signals that code eye position and velocity. The neurons that generate the velocity signal for horizontal saccades lie in the paramedian pontine reticular formation, and this velocity signal is integrated in the medial vestibular nucleus and nucleus prepositus hypoglossi to provide the position signal.
7. The mesencephalic reticular formation provides the position and velocity signals for vertical and torsional eye movements as well as vergence eye movements.
8. Presaccadic burst neurons in the superior colliculus project a desired displacement signal to the reticular formation. These neurons are inhibited by a GABAergic projection from the substantia nigra and excited by projections from the frontal eye field and the posterior parietal cortex. A motor signal from the frontal eye field excites the caudate nucleus, which then inhibits the substantia nigra, allowing a saccade to occur.
9. The posterior parietal cortex projects an attentional signal to the superior colliculus that does not distinguish between attention and movement.

10. Most large gaze shifts involve head movements as well as eye movements. Because the eye moves faster than the head, it typically reaches the target first. The vestibulo-ocular reflex maintains the eye on target by driving the eye with a velocity opposite to that of the head movement.
11. The cerebellum calibrates eye movements based on visual feedback and mediates the learning process that keeps them accurate over time.
12. Smooth pursuit is driven by a network that includes the medial vestibular nucleus, the flocculus of the cerebellum, the dorsolateral pontine nucleus, and two motion-selective areas that are found in the superior temporal sulcus of some monkeys—the middle temporal and medial superior temporal areas. Homologous areas in the human brain are located at the parieto-occipital junction. The pursuit area of the frontal eye fields initiates smooth-pursuit movements.
13. Although the motor programming of eye movements is well understood, the great bulk of physiological research in this field was done with a monkey making a directed saccade to a spot of light. The neural mechanisms underlying the free choice of saccade targets as we explore the visual world are poorly understood. This question, lying at the intersection of cognition and motor control, is one of the great unknowns in neuroscience and will be at the center of oculomotor research in the future.

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