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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.

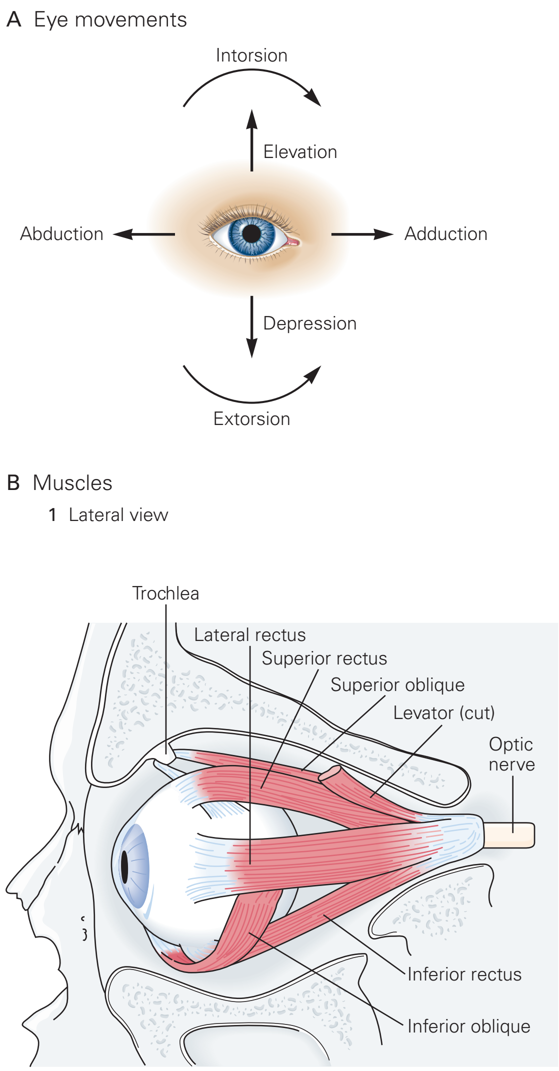

Figure 35–1 The different actions of eye movements and the muscles that control them.

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).

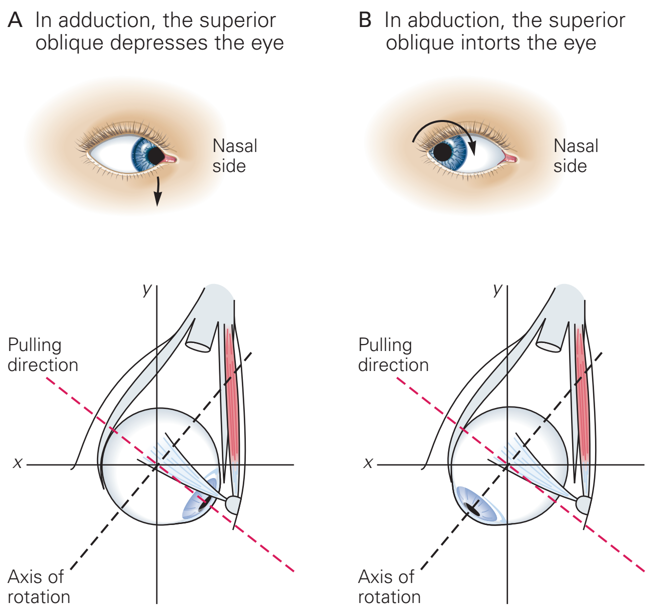

Figure 35–2 The effect of orbital position on the action of the superior oblique muscle.

A. When the eye is adducted (looking toward the nose), contraction of the superior oblique depresses the eye.

B. When the eye is abducted (looking away from the nose), contraction of the superior oblique intorts the eye.

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 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

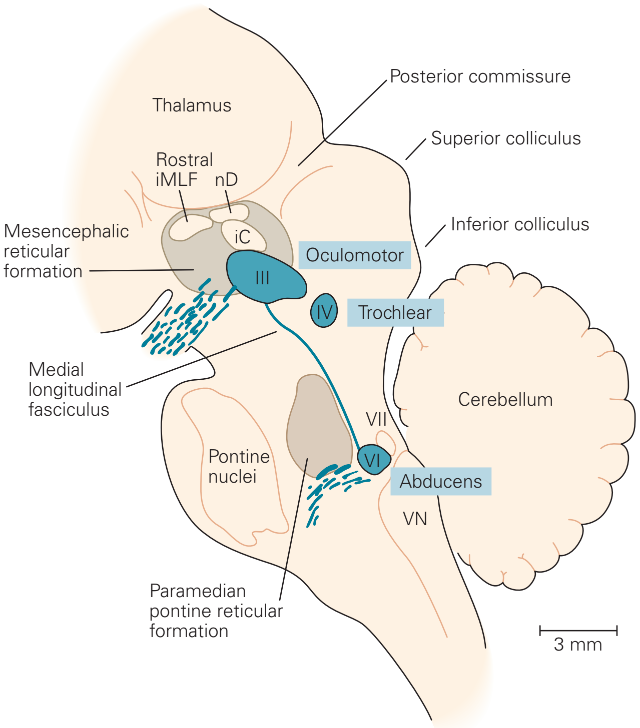

Figure 35–3 The oculomotor nuclei in the brain stem. The nuclei are shown in a parasagittal section through the thalamus, pons, midbrain, and cerebellum of a rhesus monkey. The oculomotor nucleus (cranial nerve III) lies in the midbrain at the level of the mesencephalic reticular formation; the trochlear 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 accomodation.

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.

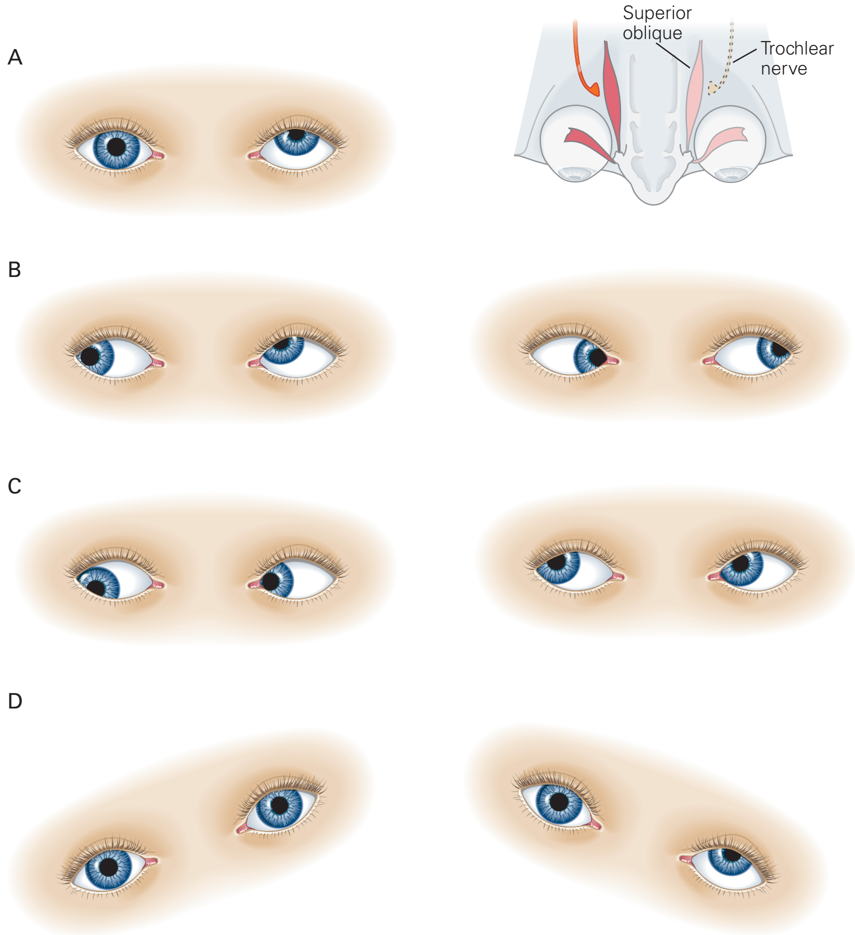

Figure 35–4 **Effect of a left trochlear nerve palsy.** The trochlear nerve innervates the superior oblique muscle, which inserts behind the equator of the eye. It depresses the eye when it is adducted and intorts the eye when it is abducted.

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 Landott 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

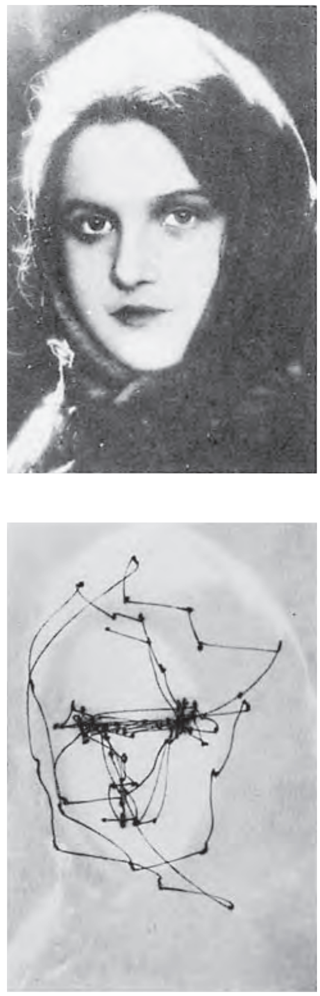

Figure 35–5 Eye movements track the outline of an object of attention. An observer looks at a picture of a woman for 1 minute. The resulting eye positions are then superimposed on the picture. As shown here, the observer concentrated on certain features of the face, lingering over the woman's eyes and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

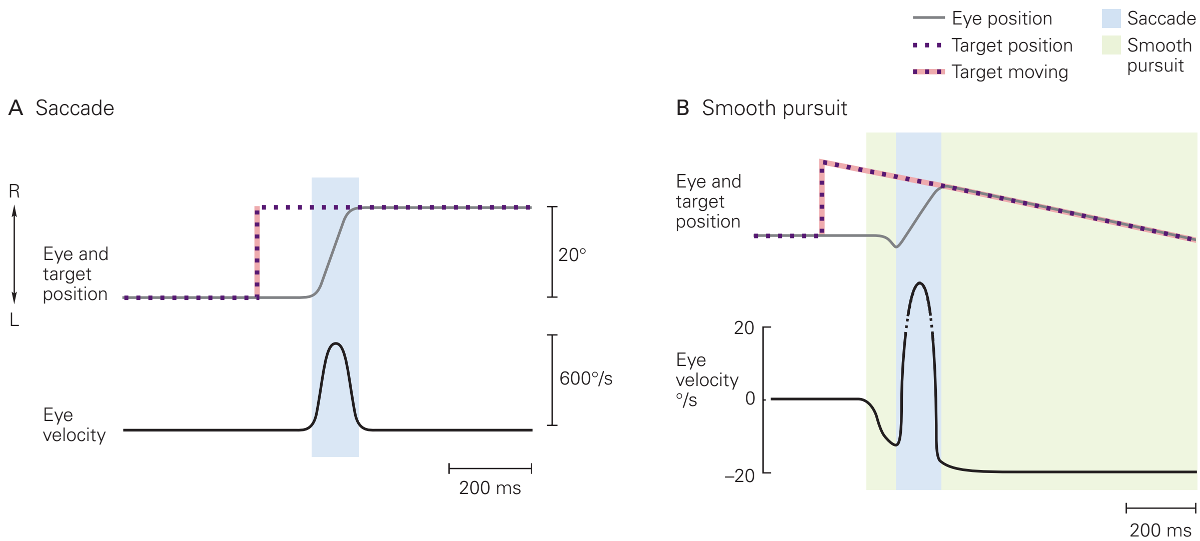

Figure 35–6 Saccadic and smooth-pursuit eye movements. Eye position, target position, and eye velocity are plotted against time.

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.)

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 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 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

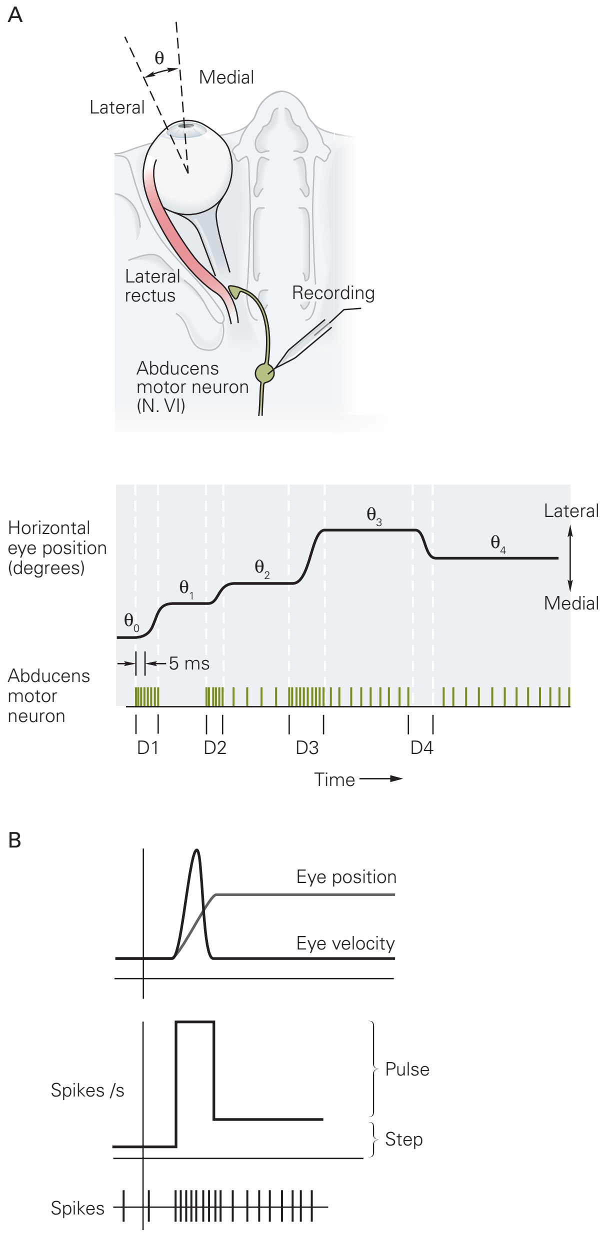

Figure 35–7 Oculomotor neurons signal eye position and velocity.

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.