CHAPTER 20

Eye Movements and Sensorimotor Integration

Overview

EYE MOVEMENTS ARE EASIER TO STUDY than movements of other parts of the body. This fact arises in part from the relative simplicity of muscle actions on the eyeball. There are only six extraocular muscles, each of which has a specific role in adjusting eye position. Moreover, there is a limited set of stereotyped eye movements, and the central circuits governing each one are partially distinct. Eye movements have therefore been a useful model for understanding the mechanisms of motor control. Indeed, much of what is known about the regulation of movements by the vestibular system, basal ganglia, and cerebellum has come from the study of eye movements (see Chapters 14, 18, and 19). In this chapter, the major features of eye movement control are used to illustrate principles of sensorimotor integration that also apply to more complex motor behaviors.

What Eye Movements Accomplish

Eye movements are important in humans because high visual acuity is restricted to the fovea, the small circular region (about 1.2 mm in diameter) in the central retina that is densely packed with cone photoreceptors (see Chapter 11). Eye movements can direct the fovea to new objects of interest in the visual field—a process called **foveation**—or compensate for disturbances that cause the fovea to be displaced from an object already foveated.

Several decades ago, the Russian physiologist Alfred Yarbus demonstrated that eye movements reveal a good deal about the strategies used to inspect a scene. Yarbus used contact lenses with small mirrors on them to document (by the position of a reflected beam on photosensitive paper) the pattern of eye movements made while individuals examined a variety of objects and scenes. Figure 20.1 shows the changes in the direction of an individual's gaze while viewing a photograph. The thin, straight lines represent the quick, ballistic eye movements (**saccades**) used to align the foveae with particular parts of the scene. Little or no visual perception occurs during a saccade, which occupies only a few tens of milliseconds. The denser spots along these lines represent points of fixation where the observer paused for a variable period to take in visual information from the area of interest. These results obtained by Yarbus, and subsequently by many others, showed that vision is an active process in which eye movements typically shift the view several times each second to direct the foveae toward selected parts of the scene to examine especially interesting or informative features. The selection of areas of interest as targets of the saccades indicates that non-foveal areas of the retina have sufficient resolution to guide the foveae toward these areas for closer examination. In the figure, the spatial distribution of the fixation points is not random, and indicates that the individual spent much

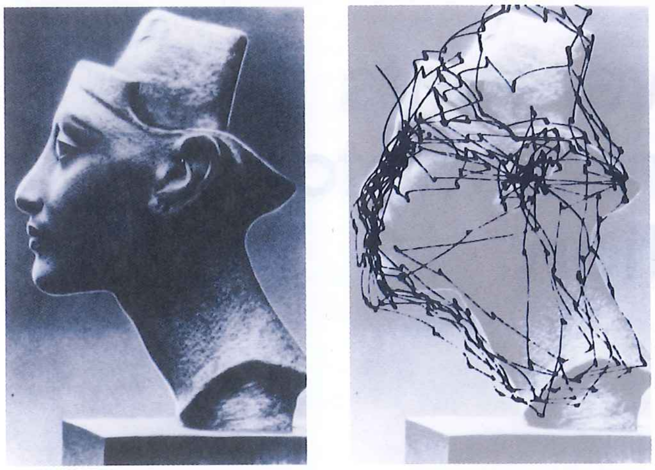


FIGURE 20.1 Eye movements of an individual viewing a photograph. The individual was shown this photograph (left) of the famous bust of Queen Nefertiti. The diagram on the right shows the individual's eye movements over a 2-min viewing period. (From Yarbus, 1967.)

more time scrutinizing Nefertiti's eye, nose, mouth, and ear than examining the middle of her cheek or neck. Thus, eye movements allow us to scan the visual field, pausing to focus attention on the portions of the scene that convey the most significant information. It follows from Figure 20.1 that tracking eye movements can be used to determine which aspects of a scene are particularly arresting; in fact, today's corporate advertisers can use modern versions of Yarbus's method to determine which pictures and scene arrangements will best sell their products.

The importance of eye movements for visual perception has also been demonstrated by experiments in which a visual image is stabilized on the retina, either by paralyzing the extraocular eye muscles or by moving a scene in exact register with eye movements so that the different features of the image always fall on exactly the same parts of the retina (Box 20A). Such stabilized visual images rapidly disappear, for reasons that remain poorly understood. Nonetheless, observations on motionless images make it plain that eye movements are essential for visual perception.

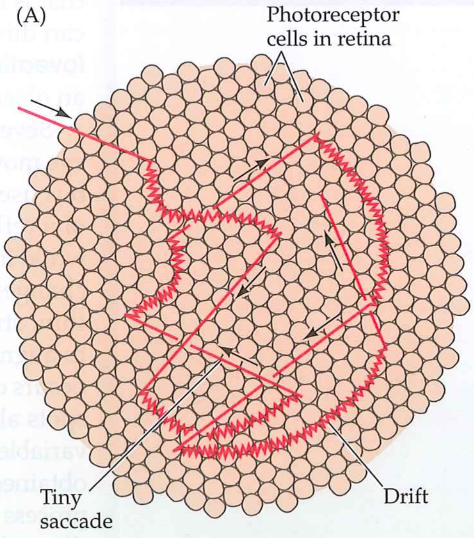
Actions and Innervation of Extraocular Muscles

Three antagonistic pairs of muscles control eye movements: the **lateral** and **medial rectus muscles**; the **superior** and **inferior rectus muscles**; and the **superior** and **inferior oblique muscles**. These muscles are responsible for movements of the eye along three different axes: *horizontal*, either toward the nose (adduction) or away from the nose (abduction); *vertical*, either elevation or depression; and *torsional*, movements that bring the top of the eye toward the nose (intorsion) or away from the nose (extorsion). Horizontal movements are controlled entirely by the medial

BOX 20A ■ The Perception of Stabilized Retinal Images

Visual perception depends critically on frequent changes of scene. Normally, our view of the world is changed by saccades, and tiny saccades that continue to move the eyes abruptly over a fraction of a degree of visual arc occur even when the observer stares intently at an object of interest. Moreover, continual drift of the eyes during fixation progressively shifts the image onto a nearby but different set of photoreceptors. As a consequence of these several sorts of eye movements (Figure A), our point of view changes more or less continually.

The importance of a continually changing scene for normal vision is dramatically revealed when the retinal image is stabilized. If a small mirror is attached to the eye by means of a contact lens and an image is reflected off the mirror onto a screen, then the individual necessarily sees the same thing, whatever the position of the eye—every time the eye moves, the projected image moves by exactly the same amount (Figure B). Under these circumstances, the stabilized image actually disappears from perception within a few seconds!



(A) Diagram of the types of eye movements that continually change the retinal stimulus during fixation. The straight lines indicate microsaccades, and the zigzag lines drift; the structures in the background are photoreceptor cells drawn approximately to scale. The normal scanning movements of the eyes (saccades) are much too large to be shown here but obviously contribute to the changes of view that we continually experience, as do slow tracking eye movements (although the fovea tracks a particular object, the scene nonetheless changes). (After Pritchard, 1961.)

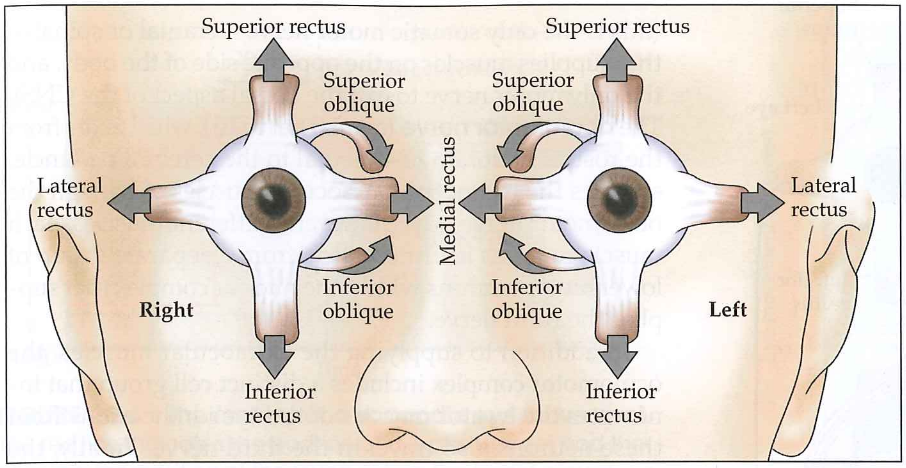


FIGURE 20.2 Extraocular muscles contribute to vertical and horizontal eye movements. Horizontal movements are mediated by the medial and lateral rectus muscles, while vertical movements are mediated by the superior and inferior rectus and the superior and inferior oblique muscles.

and lateral rectus muscles; the medial rectus muscle is responsible for adduction, the lateral rectus muscle for abduction (Figure 20.2). Vertical movements require the coordinated action of the superior and inferior rectus muscles, as well as the oblique muscles. The relative contributions of the rectus and oblique muscles depend on the horizontal position of the eye. In the primary position (eyes straight ahead), both of these muscle groups contribute to vertical movements. Elevation is due to the action of the superior rectus and inferior oblique muscles, while depression is due to the action of the inferior rectus and superior oblique muscles. When the eye is abducted, the rectus muscles are the prime vertical movers; elevation is due to the action of the superior rectus, and depression is due to the action of the inferior rectus. When the eye is adducted, the oblique muscles are the prime vertical movers. In this position, elevation is due to the action of the inferior oblique muscle, while depression is due to the action of the superior oblique muscle. The oblique muscles are also primarily responsible for torsional movements.

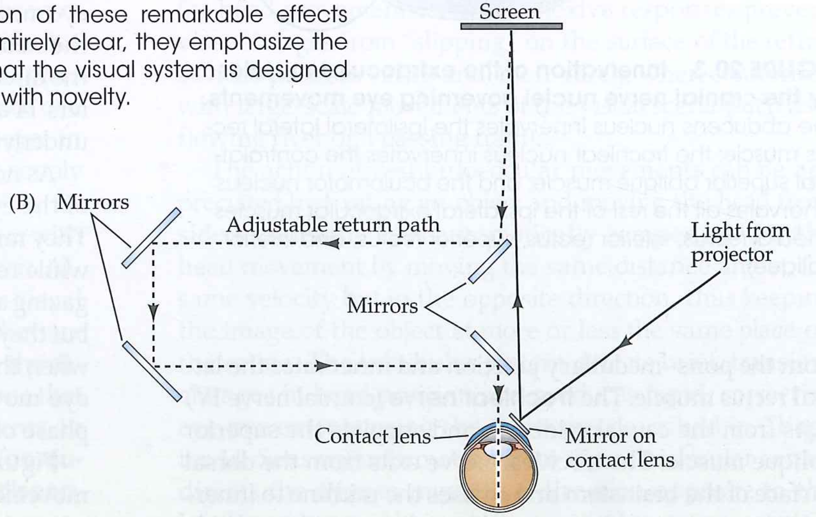
The extraocular muscles are innervated by lower motor neurons whose axons form three cranial nerves: the abducens, the trochlear, and the oculomotor (Figure 20.3). The **abducens nerve** (cranial nerve VI) exits the brainstem

BOX 20A ■ *(continued)*

A simple way to demonstrate the rapid disappearance of a stabilized retinal image is to visualize one's own retinal blood vessels. The blood vessels, which lie in front of the photoreceptor layer, cast a shadow on the underlying receptors. Although normally invisible, the vascular shadows can be seen by moving a source of light across the eye, a phenomenon first noted by J. E. Purkinje more than 150 years ago. This perception can be elicited with an ordinary penlight pressed gently against the lateral side of the closed eyelid. When the light is wiggled vigorously, a rich network of black blood vessel shadows (called a "Purkinje tree") appears against an orange background. (The vessels appear black because they are shadows.) By starting and stopping the movement, it is readily apparent that the image of the blood vessel shadows disappears within a fraction of a second after the light source is stilled.

The conventional interpretation of the rapid disappearance of stabilized images is retinal adaptation. In fact, the phenomenon is at least partly of central origin. Stabilizing the retinal image in one eye, for example, diminishes perception through the other eye, an effect known as interocular transfer. Although the explanation of these remarkable effects is not entirely clear, they emphasize the point that the visual system is designed to deal with novelty.

(B) One means of producing stabilized retinal images. By attaching a small mirror to the eye, the scene projected onto the screen will always fall on the same set of retinal points, no matter how the eye is moved. (After Riggs et al., 1953.)



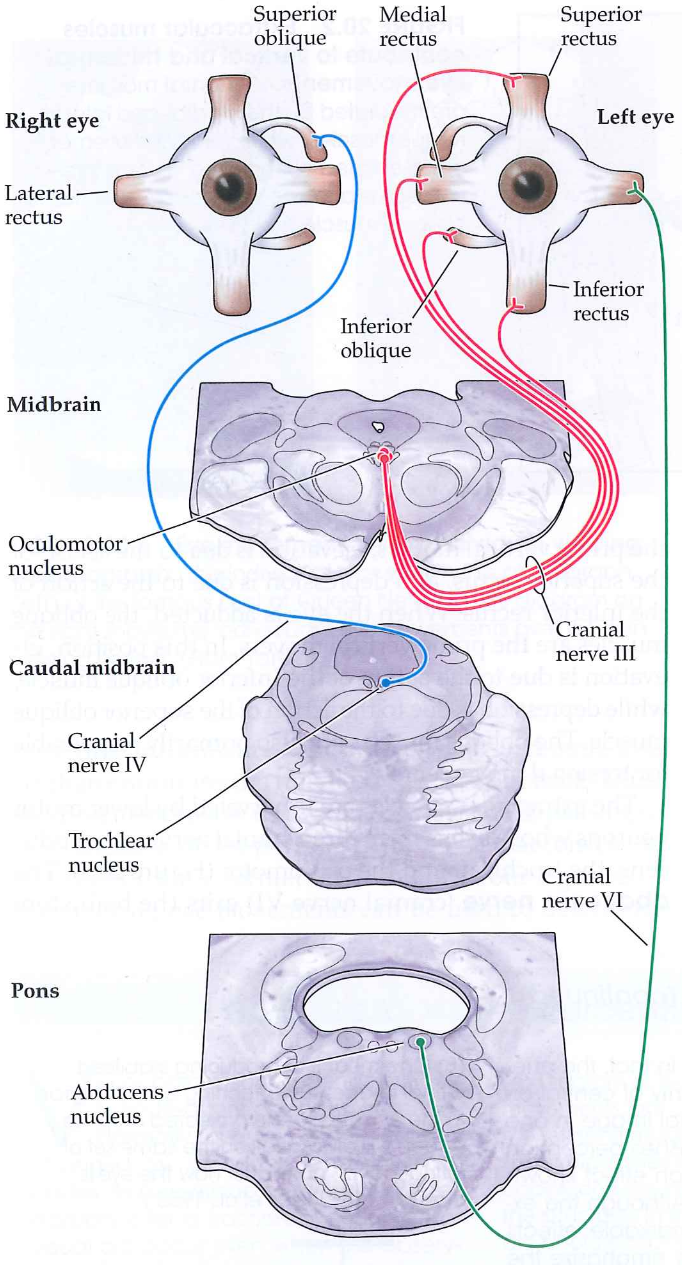


FIGURE 20.3 Innervation of the extraocular muscles by the cranial nerve nuclei governing eye movements. The abducens nucleus innervates the ipsilateral lateral rectus muscle; the trochlear nucleus innervates the contralateral superior oblique muscle; and the oculomotor nucleus innervates all the rest of the ipsilateral extraocular muscles (medial rectus, inferior rectus, superior rectus, and inferior oblique).

from the pons–medullary junction and innervates the lateral rectus muscle. The **trochlear nerve** (cranial nerve IV) exits from the caudal midbrain and supplies the superior oblique muscle. The trochlear nerve exits from the dorsal surface of the brainstem and crosses the midline to innervate the superior oblique muscle on the contralateral side (this is the only somatic motor nerve—cranial or spinal—that supplies muscles on the opposite side of the body, and the only motor nerve to exit the dorsal aspect of the CNS). The **oculomotor nerve** (cranial nerve III), which exits from the rostral midbrain just medial to the cerebral peduncle, supplies the rest of the extraocular muscles. Although the oculomotor nerve governs several different muscles, each muscle receives its innervation from a separate group of lower motor neurons within the nuclear complex that supplies the third nerve.

In addition to supplying the extraocular muscles, the oculomotor complex includes a distinct cell group that innervates the levator muscles of the eyelid; the axons from these neurons also travel in the third nerve. Finally, the third nerve carries preganglionic parasympathetic axons that are responsible for pupillary constriction (see Chapters 12 and 21) from the nearby Edinger–Westphal nucleus. Thus, damage to the third nerve results in three characteristic deficits: impairment of eye movements, drooping of the eyelid (a clinical sign called **ptosis**), and pupillary dilation, due to the unopposed action of sympathetic inputs to the dilator muscles of the iris.

Types of Eye Movements and Their Functions

The five basic types of eye movements can be grouped into two functional categories: those that serve to *shift* the direction of gaze, and those that serve to *stabilize* gaze. Shifts in eye position are necessary to foveate new targets and to follow foveated targets as they move in visual space. Stabilizing movements of the eyes are used to maintain foveation when the head moves and when there are large-scale movements of the visual field. Thus, saccades, smooth pursuit movements, and vergence movements shift the direction of gaze, and vestibulo-ocular and optokinetic movements stabilize gaze. The functions of each type of eye movement are introduced here; in subsequent sections, the neural circuitry responsible for movements that shift the direction of gaze is presented in more detail (see Chapters 14 and 19 for further discussion of the neural circuitry underlying gaze-stabilizing movements).

As noted earlier, saccades are rapid, ballistic movements of the eyes that abruptly change the direction of fixation. They range in amplitude from the small movements made while reading to the much larger movements made while gazing around a room. Saccades can be elicited voluntarily, but they occur reflexively whenever the eyes are open, even when they are fixated on a target (see Box 20A). The rapid eye movements (REM) that occur during an important phase of sleep (see Chapter 28) also are saccades.

Figure 20.4 shows the time course of a saccadic eye movement. After the onset of a target for a saccade (in this example, the stimulus was the movement of an already

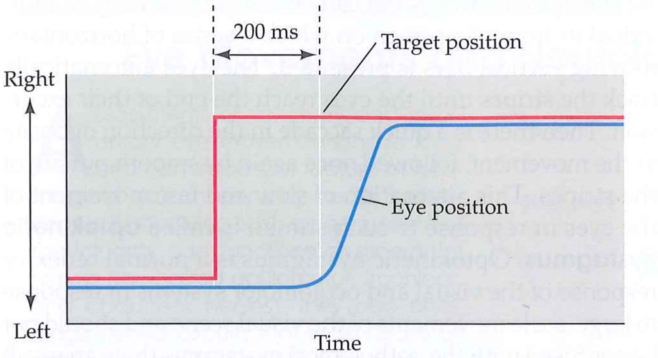


FIGURE 20.4 The metrics of a saccadic eye movement. The red line indicates the position of a fixation target and the blue line the position of the fovea. When the target moves suddenly to the right, there is a delay of about 200 ms before the eye begins to move to the new target position. (After Fuchs, 1967.)

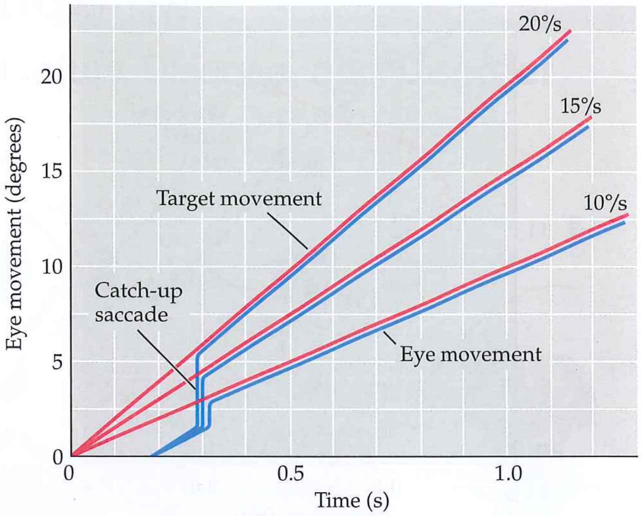


FIGURE 20.5 The metrics of smooth pursuit eye movements. These traces show eye movements (blue lines) tracking a stimulus moving at one of three different velocities (red lines). After a quick saccade to foveate the target, the eye movement attains a velocity that matches the velocity of the target. (After Fuchs, 1967.)

fixated target), it takes about 200 ms for eye movement to begin. During this delay, the position of the target with respect to the fovea (that is, how far the eye has to move) is computed, and the difference between the initial and intended position is converted into a motor command that activates the extraocular muscles to move the eyes the correct distance in the appropriate direction. Saccadic eye movements are said to be ballistic because the saccade-generating system usually does not respond to subsequent changes in the position of the target during the course of the eye movement. If the target moves again during this time (which is on the order of 15 to 100 ms), the saccade will miss the target, and a second saccade must be made to correct the error.

Smooth pursuit movements are much slower tracking movements of the eyes designed to keep a moving stimulus on the fovea once foveation is achieved. Such movements are under voluntary control in the sense that the observer can choose whether or not to track a moving stimulus (Figure 20.5). Surprisingly, only highly trained observers can make a smooth pursuit movement in the absence of a moving target. Most people who try to move their eyes in a smooth fashion without a moving target to track simply make saccades.

Vergence movements align the fovea of each eye with targets located at different distances from the observer. Although vergence movements are required to track a visual target that may be moving closer or farther away, they are more commonly employed when abruptly shifting the direction of gaze, for example, from a near object to one that is more distant. Unlike other types of eye movements, in which the two eyes move in the same direction (**conjugate eye movements**), vergence movements are **disconjugate** (or **disjunctive**); they involve either a convergence or divergence of the lines of sight of each eye to foveate an object that is nearer or farther away. Convergence is one of the three reflexive visual responses elicited together to shift gaze from a distant to a near object. The other components of the so-called **near reflex triad** are accommodation of the lens, which by increasing the curvature of the lens brings the close object into focus, and pupillary constriction, which by reducing spherical aberration increases the depth of field and sharpens the image on the retina (see Chapter 11).

Vestibulo-ocular movements and **optokinetic eye movements** operate together to move the eyes and stabilize gaze relative to the external world, thus compensating for head movements. These reflexive responses prevent visual images from "slipping" on the surface of the retina as head position varies and, more rarely, when confronted with large-scale movements of the visual scene (such as a flowing river or a passing train).

The action of vestibulo-ocular movements can be appreciated by fixating an object and moving the head from side to side; the eyes automatically compensate for the head movement by moving the same distance and at the same velocity but in the opposite direction, thus keeping the image of the object at more or less the same place on the retina. The vestibular system detects brief, transient changes in head position and produces rapid, corrective eye movements using the pathways described in Chapter 14. Sensory information from the semicircular canals directs the eyes to move in a direction opposite to the head movement. Although the vestibular system operates

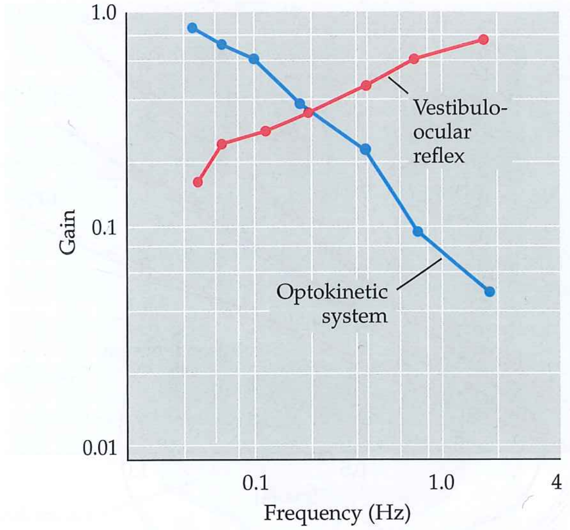


FIGURE 20.6 Operational ranges of the vestibulo-ocular and optokinetic systems. Functions of the vestibulo-ocular and optokinetic systems were assessed independently in rabbits by rotating the animals with their eyes closed (to isolate the vestibulo-ocular reflex) or following recovery from bilateral labyrinthectomy (to isolate the optokinetic system). At low frequencies of movement (below 1 Hz or one back-and-forth cycle of stimulation per second), the gain of the vestibulo-ocular reflex (the ratio of eye movement to head movement) diminishes below unity. However, the gain of the optokinetic system (the ratio of eye movement to retinal slip) approaches unity at such low frequencies of stimulation. Thus, the vestibulo-ocular and optokinetic systems act in a complementary, frequency-dependent fashion to stabilize gaze over a broad range of stimulation frequencies. (After Baarsma and Collewijn, 1974.)

effectively to counteract rapid movements of the head, it is relatively insensitive to slow movements (below 1 Hz) or to persistent rotation of the head. For example, if the vestibulo-ocular reflex is tested with continuous rotation of an individual and without visual cues about the movement of the image (i.e., with eyes closed or in the dark), the compensatory eye movements cease after only about 30 seconds of rotation. However, if the same test is performed with visual cues, eye movements persist. The compensatory eye movements in this case are due to the activation of another system that relies not on vestibular information, but on visual cues indicating motion of the visual field. This optokinetic system is especially sensitive to slow movements (below 1 Hz) of large areas of the visual field, and its response builds up slowly. These features complement the properties of the vestibulo-ocular reflex, especially as head movements slow down and vestibular signals decay (Figure 20.6). Thus, should a visual image slowly "slip" across the retina, the optokinetic system will respond by inducing compensatory movements of the eyes at the same speed and in the opposite direction.

The optokinetic system can be tested by seating an individual in front of a screen on which a series of horizontally moving vertical bars is presented. The eyes automatically track the stripes until the eyes reach the end of their excursion. Then there is a quick saccade in the direction opposite to the movement, followed once again by smooth pursuit of the stripes. This alternation of slow and fast movement of the eyes in response to such stimuli is called **optokinetic nystagmus**. Optokinetic nystagmus is a normal reflexive response of the visual and oculomotor systems in response to large-scale movements of the visual scene and should not be confused with the pathological nystagmus that can result from certain kinds of brain injury (for example, damage to the vestibular system or the cerebellum; see Chapters 14 and 19, respectively). Indeed, clinicians have long regarded eye movements to be key indicators of neurological function and dysfunction (Clinical Applications).

Neural Control of Saccadic Eye Movements

The problem of moving the eyes to fixate a new target in space (or indeed any other movement) involves two separate tasks: controlling the *amplitude* of movement (how far), and controlling the *direction* of the movement (which way). The amplitude of a saccadic eye movement is encoded by the duration of neuronal activity in the lower motor neurons of the oculomotor nuclei. For instance, as shown in Figure 20.7, neurons in the abducens nucleus fire a burst of action potentials just prior to abducting the eye (by causing the lateral rectus muscle to contract) and are silent when the eye is adducted. The amplitude of the movement is correlated with the duration of the burst of action potentials in abducens neurons. Following each saccade, abducens neurons reach a new baseline level of discharge that is correlated with the position of the eye in the orbit. The steady baseline level of firing generates the muscle force needed to hold the eye in its new position.

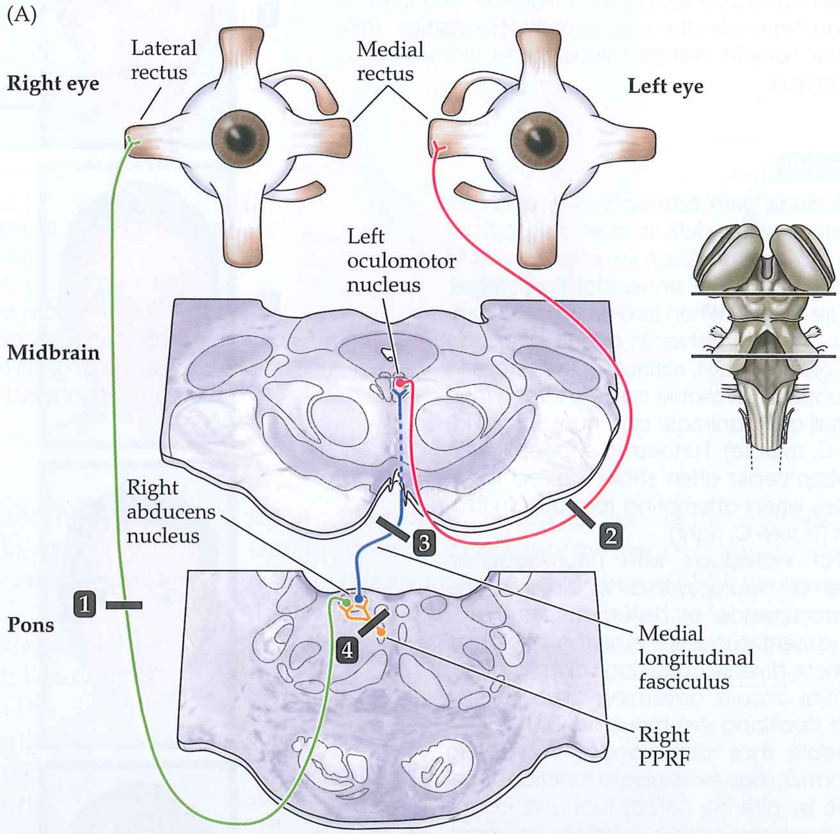
The direction of the movement is determined by which eye muscles are activated. Although in principle any given direction of movement could be specified by independently adjusting the activity of individual eye muscles, the complexity of the task would be overwhelming. Instead, the direction of eye movement is controlled by the local circuit neurons in two **gaze centers** in the reticular formation (see Box 17C), each of which is responsible for generating movements along a particular axis. The **paramedian pontine reticular formation** (**PPRF**), also called the horizontal gaze center, is a collection of local circuit neurons near the midline in the pons. These neurons are responsible for generating horizontal eye movements. The **rostral interstitial nucleus**, or vertical gaze center, is located in the rostral part of the midbrain reticular formation and is responsible for vertical movements. Activation of each gaze center separately results

CLINICAL APPLICATIONS

Eye Movements and Neurological Injury, Disease, and Disorder

Assessments of voluntary and involuntary eye movements have long been an important feature of neurological examinations by health professionals. Not only do such assessments test the integrity and functions of six cranial nerves (the paired oculomotor, trochlear, and abducens nerves), they also challenge central circuits that span nearly all major divisions of the CNS, except for the spinal cord (although spinal cord neurons do play a role in the sympathetic regulation of pupil diameter; see Chapter 21).

Just as any voluntary movement can be analyzed in terms of lower and upper motor neuronal control, so can the voluntary activity of the extraocular muscles that shift and stabilize visual gaze. Injuries that affect cranial nerves III, IV, or VI lead to lower motor neuronal signs and symptoms associated with the affected extraocular muscles and to predictable deficits in conjugate gaze (Figures A and B). A person afflicted with such an injury would experience *diplopia* (double vision), especially when gaze is cast in the direction of action for the affected muscle. For example, a sixth nerve palsy on the right (see lesion 1 in Figures A and B) or a third nerve palsy on the left (see lesion 2 in Figures A and B) would lead to severe diplopia with rightward gaze. (Complete injury to the oculomotor nerve would also impair the levator muscle of the upper eyelid; the ipsilateral eye would therefore be closed and would need to be opened manually to facilitate vision and assessment of oculomotor function.) Damage to the fibers passing through the medial longitudinal fasciculus in the left side of the pons would likewise produce diplopia with rightward gaze (due to insufficient contraction of the left medial rectus muscle), with the added complication of nystagmus in the right eye (a condition that is not well explained, termed *internuclear ophthalmoplegia*; see lesion 3 in Figures A and B). Injury to the paramedian tegmentum of the pons may damage the horizontal gaze center (PPRF) and prevent conjugate gaze into the ipsilateral (right, in this case) visual field (see lesion 4 in Figures A and B). Similar deficits in conjugate gaze, but toward the contralateral visual field, are characteristic of damage to upper motor neurons in the frontal eye fields and superior colliculus, as described in the main text.



(A) Injuries to lower motor neurons and local brainstem circuits impair conjugate gaze and produce diplopia. Lesion 1 damages the right abducens nerve. Lesion 2 injures fibers in the left oculomotor nerve that innervate the left medial rectus muscle. Lesion 3 damages the left medial longitudinal fasciculus. Lesion 4 injures the local circuit neurons connecting the right paramedian pontine reticular formation (PPRF, or horizontal gaze center) to the right abducens nucleus. (See Figure 20.8 legend for additional detail.)

Given how much central circuitry in the cerebral hemispheres, cerebellum, and brainstem governs eye movements in natural viewing (see Figure 20.13), it is not surprising that eye movements may be disturbed in a variety of neurological and neuropsychiatric conditions that involve neurodegeneration or atypical neurodevelopment. For example, individuals with idiopathic Parkinson's disease may show mild impairments of smooth pursuit eye movements and decreased gain of voluntary saccades, while individuals with Huntington's disease may show difficulty initiating saccades in response to verbal instruction, with increased saccade latency and saccade slowing, especially in the vertical plane (see Chapter 18). Patients with Alzheimer's disease or frontotemporal dementia typically show increases in errors when challenged with anti-saccade and saccade suppression tasks, with gaze-fixation instability and prolonged latency of voluntary and reflexive saccades. Other patients with spinocerebellar ataxias (see Chapter 19) may show a variety of disturbances in voluntary eye movements, reflecting the degeneration of cerebellar circuits that help regulate the gain of brainstem circuits that govern conjugate eye movements, including the vestibulo-ocular reflex (see Chapter 14).

Finally, it is interesting to note how eye movements are often atypical in

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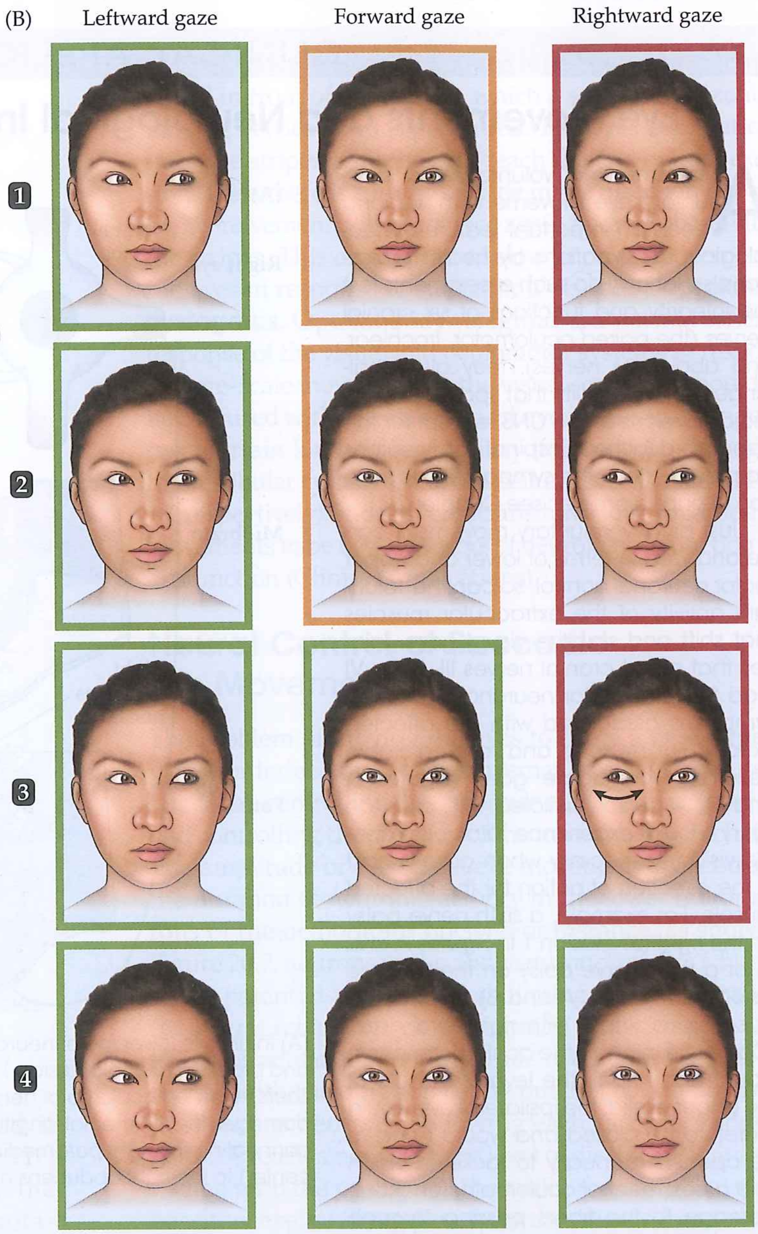
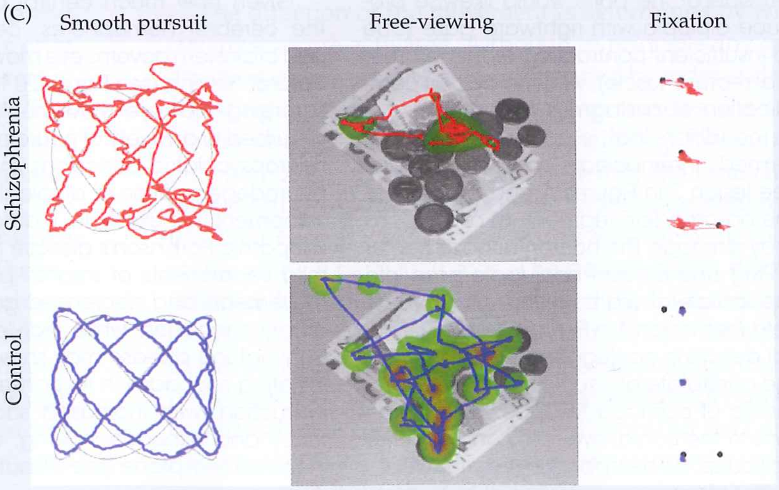
CLINICAL APPLICATIONS (continued)



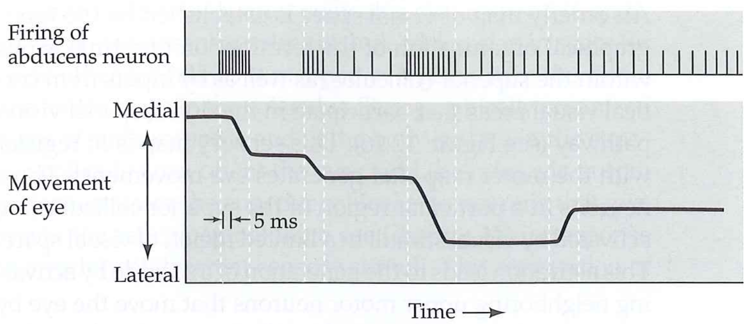
(B) Depiction of deficits in conjugate gaze associated with the four lesions shown in Figure (A). Each of these lesions impairs a person's ability to make conjugate shifts of gaze toward the right. Lesion 3 typically results in internuclear ophthalmoplegia and nystagmus of the contralateral eye (double-headed curved arrow). The frame color in each panel indicates the severity of diplopia: green, minimal diplopia; orange, mild diplopia; red, severe diplopia.

individuals with schizophrenia. Such individuals show deficits in smooth pursuit eye movements (such as when following stimuli moving in sinusoidal trajectories; Figure C, left). When free-viewing, individuals with schizophrenia often undersample visual scenes, restricting their gaze to a subset of available targets, even when stimuli are inanimate and nonsocial (Figure C, middle). Furthermore, people with schizophrenia often show marked instabilities when attempting to maintain fixation (Figure C, right).

For individuals with neurodegenerative or neuropsychiatric disease, the co-occurrence of disturbances in eye movement suggests that the etiologies of these diverse conditions directly affect central circuits governing gaze shifting and stabilizing eye movements. It is also possible that disturbances in viewing behavior may exacerbate functional decline by altering perceptual and cognitive experience of the visual environment. In either case, it has become clear that clinical assessments of voluntary and involuntary eye movements are providing valuable diagnostic criteria and promising biomarkers of disease severity, progression, or regression.



(C) Assessments of smooth pursuit, free-viewing, and fixation stability in individuals with schizophrenia (red traces) and in typical individuals (blue traces). Left: Smooth pursuit eye movements superimposed on sinusoidal patterns in two dimentions. Note the irregularities that degrade smooth pursuit in individuals with schizophrenia. Middle: Free-viewing reveals constrained patterns of visual sampling in individuals with schizophrenia (top) compared with typical individuals. Right: Assessment of fixation stability in three conditions: near distractor (top), single fixation target (center), and far distractor (bottom). Note the fixation instabilities in individuals with schizophrenia compared with typical individuals. (After Benson et al., 2012.)



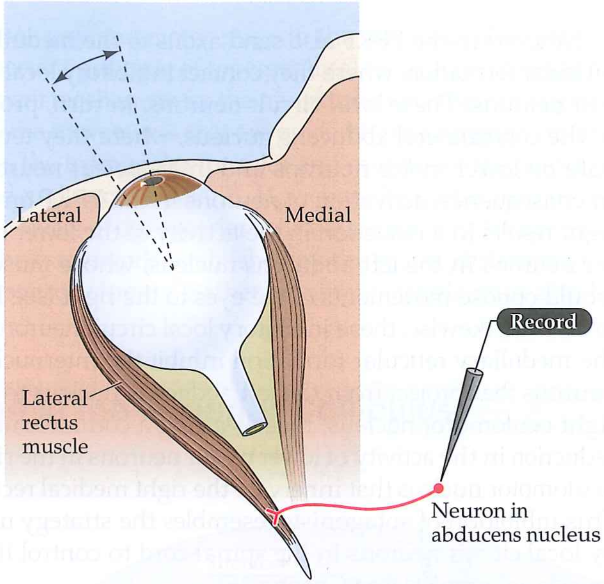


FIGURE 20.7 Motor neuron activity in relation to saccadic eye movements. The experimental setup is shown on the right. In this example, an abducens lower motor neuron fires a burst of activity (upper trace) that precedes and extends throughout the movement (solid line). An increase in the tonic level of firing is associated with more lateral displacement of the eye. Note also the decline in firing rate during a saccade in the opposite direction. (After Fuchs and Luschei, 1970.)

in movements of the eyes along a single axis, either horizontal or vertical. Activation of the gaze centers in concert results in oblique movements whose trajectories are specified by the relative contribution of each center.

An example of how the PPRF works with the abducens and oculomotor nuclei to generate a horizontal saccade to the right is shown in Figure 20.8. Neurons in the PPRF innervate cells in the abducens nucleus on the same side of the brain. The abducens nucleus contains two types of neurons. One type comprises the lower motor neurons that innervate the lateral rectus muscle on the same side. The other type, called internuclear neurons, send their axons across the midline. These axons ascend in a fiber tract called the **medial longitudinal fasciculus** and terminate in the portion of the oculomotor nucleus that contains lower motor neurons that innervate the medial rectus muscle. As a result of this arrangement, activation of PPRF neurons in the right side of the brainstem causes horizontal movements of both eyes to the right; likewise, activation of PPRF neurons in the left half of the brainstem induces horizontal movements of both eyes to the left.

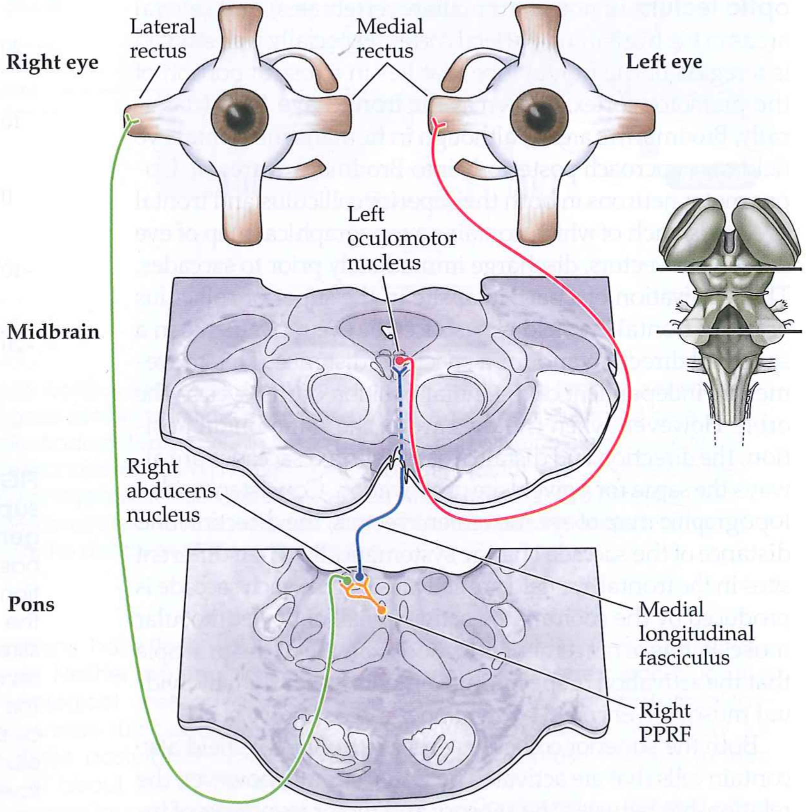


FIGURE 20.8 Synaptic circuitry responsible for horizontal movements of the eyes to the right. This simplified diagram depicts how activation of local circuit neurons in the right horizontal gaze center (the right PPRF; orange) leads to increased activity of lower motor neurons and internuclear neurons in the right abducens nucleus. The lower motor neurons (green) innervate the lateral rectus muscle of the right eye. The internuclear neurons (blue) project via the medial longitudinal fasciculus to the contralateral oculomotor nucleus, where they activate lower motor neurons (red) that in turn innervate the medial rectus muscle of the left eye. The coordinated action of the right lateral rectus and left medial rectus muscles rotates the eyes to the right. Inhibitory local circuit neurons in the medullary reticular formation (not illustrated) inhibit activity in the left abducens nucleus, which has the effect of decreasing tone in the antagonistic muscles.

Neurons in the PPRF also send axons to the medullary reticular formation, where they contact inhibitory local circuit neurons. These local circuit neurons, in turn, project to the contralateral abducens nucleus, where they terminate on lower motor neurons and internuclear neurons. In consequence, activation of neurons in the PPRF on the right results in a reduction in the activity of the lower motor neurons in the left abducens nucleus, whose muscles would oppose movements of the eyes to the right (see Figure 20.7). Likewise, these inhibitory local circuit neurons in the medullary reticular formation inhibit the internuclear neurons that project from the left abducens nucleus to the right oculomotor nucleus, thus assuring a commensurate reduction in the activity of lower motor neurons in the right oculomotor nucleus that innervate the right medical rectus. This inhibition of antagonists resembles the strategy used by local circuit neurons in the spinal cord to control limb muscle antagonists (see Chapter 16).

Although saccades can occur in complete darkness, they are often elicited when something in the visual field attracts attention and the observer directs the foveae toward the object of interest for more detailed examination. How, then, is sensory information about the location of a salient target in space transformed into an appropriate pattern of activity in the horizontal and vertical gaze centers? Two regions of the brain that project to the gaze centers are demonstrably important for the initiation and accurate targeting of saccadic eye movements: the superior colliculus of the midbrain (called the **optic tectum** in non-mammalian vertebrates) and several areas in the frontal and parietal cortex. Especially well studied is a region of the frontal lobe that lies in a rostral portion of the premotor cortex, known as the **frontal eye field** (classically, Brodmann's area 8, although in humans the frontal eye field may encroach posteriorly into Brodmann's area 6). Upper motor neurons in both the superior colliculus and frontal eye fields, each of which contains a topographical map of eye movement vectors, discharge immediately prior to saccades. Thus, activation of a particular site in the superior colliculus or in the frontal eye field elicits saccadic eye movements in a specified direction and for a specified distance. This movement is independent of the initial position of the eyes in the orbit. However, when the eyes are in the same initial position, the direction and distance of the elicited saccades are always the same for a given site of activation. Consistent with a topographic map of eye movement vectors, the direction and distance of the saccade change systematically when different sites in the frontal eye field are activated. Since each saccade is produced by the coordinated activity of all of the extraocular muscles, this arrangement is a good example of the principle that the activation of specific movements rather than individual muscles is encoded by the upper motor neurons.

Both the superior colliculus and the frontal eye field also contain cells that are activated by visual stimuli; however, the relationship between the sensory and motor responses of individual cells is better understood for the superior colliculus. An orderly map of visual space is established by the topographical organization of the termination of retinal axons within the superior colliculus, as well as by inputs from cortical visual areas that participate in the dorsal spatial vision pathway (see Figure 12.16). This sensory map is in register with the motor map that generates eye movements. Thus, neurons in a particular region of the superior colliculus are activated by visual stimuli in a limited region of visual space. This activation leads to the generation of a saccade by activating neighboring upper motor neurons that move the eye by an amount just sufficient to align the foveae with the region of visual space that provided the stimulation (Figure 20.9).

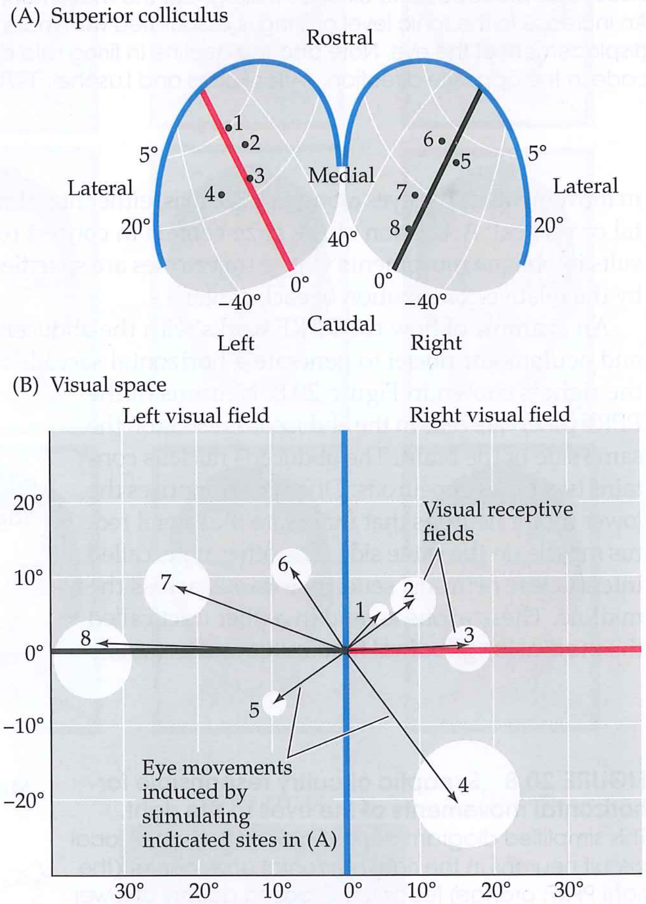
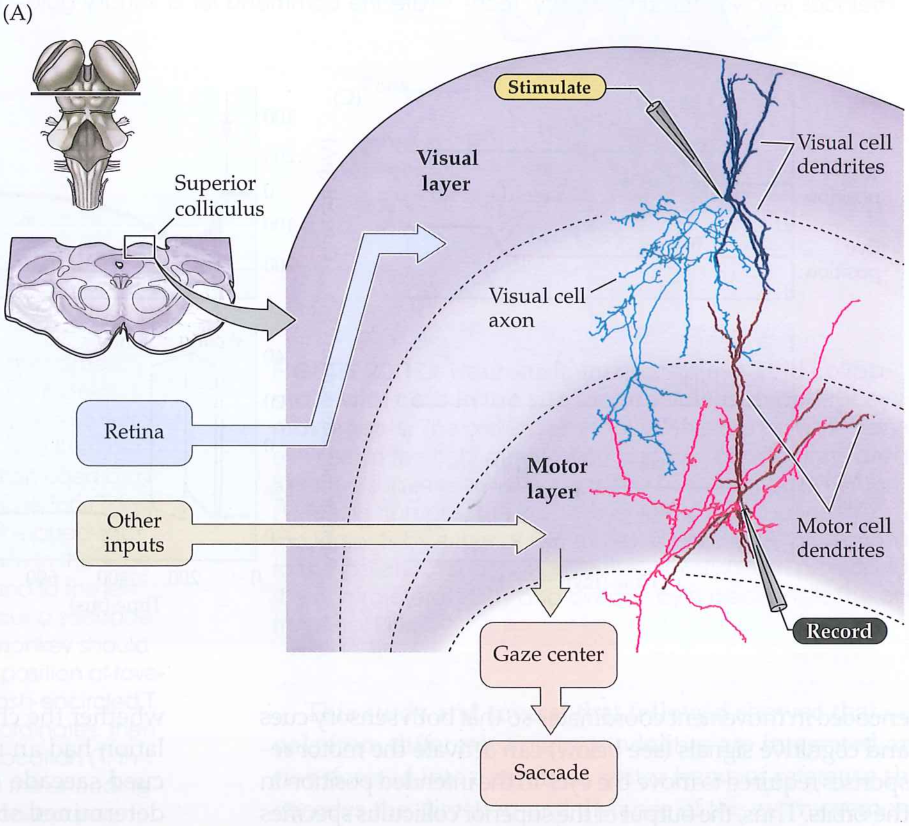


FIGURE 20.9 The sensory map of visual space in the superior colliculus is in register with the motor map that generates eye movements. Evidence for this registration has been obtained from electrical recording and stimulation. (A) Surface views of the superior colliculus illustrating the location of eight separate electrode recording and stimulation sites. (B) Map of visual space showing the visual receptive field location of the sites in (A) (white circles), and the amplitude and direction of the eye movements elicited by stimulating these sites electrically (arrows). In each case, electrical stimulation results in eye movements that align the foveae with a region of visual space that corresponds to the visual receptive fields of neurons at that site. (After Schiller and Stryker, 1972.)

Neurons in the superior colliculus also respond to auditory and somatosensory stimuli. Indeed, location in space for these other modalities is mapped in register with the visual and motor maps in the colliculus. Topographically organized maps of auditory space and of the body surface in the superior colliculus can orient the eyes (and the head, via output projections from the superior colliculus to neurons that give rise to the reticulospinal tract; see Chapter 17) in response to a variety of different sensory stimuli. This registration of the sensory and motor maps in the colliculus illustrates an important principle of topographical maps: They provide an efficient mechanism for the transformation of sensory signals into the movements that are guided by these signals (in this case, the extraocular muscles and muscles of the posterior head and neck; Box 20B). However, the motor map in the deep layers of the superior colliculus is not simply organized in the framework established by the spatial distribution of the sensory inputs. Rather, the input signals must also be

BOX 20B ■ Sensorimotor Integration in the Superior Colliculus



(A) The superior colliculus receives visual input from the retina and sends a command signal to the gaze centers to initiate a saccade. In the experiment illustrated here, a stimulating electrode activates cells in the visual layer, and a patch clamp pipette records the response evoked in a neuron in the subjacent motor layer. The cells in the visual and motor layers were subsequently labeled with a tracer called biocytin. This experiment demonstrates that the terminals of the visual neuron are located in the same region as the dendrites of the motor neuron.

The superior colliculus is a laminated structure in which differences between the layers provide clues about how sensory and motor maps interact to produce appropriate movements. As discussed in the text, the superficial or "visual" layer of the colliculus receives input from retinal axons that form a topographic map. Thus, each site in the superficial layer is activated maximally by the presence of a stimulus at a particular point of visual space. In contrast, neurons in the deeper or "motor" layers generate bursts of action potentials that command saccades, effectively generating a motor map; thus, activation of different sites generates saccades having different vectors (see Figure 20.9). The visual and motor maps are *in register*, so that visual cells responding to a stimulus in a specific region of visual space are located directly above the motor cells that command eye movements toward that same region (Figure A).

The registration of the visual and motor maps suggests a simple strategy for how the eyes might be guided toward an object of interest in the visual field. When an object appears at a particular location in the visual field, it activates neurons in the corresponding part of the visual map. As a result, bursts of action potentials are generated by the subjacent motor cells to command a saccade that rotates the two eyes just the right amount to direct the foveae toward that same location in the visual field. This behavior is called "visual grasp" because successful sensorimotor integration results in the accurate foveation of a visual target.

This seemingly simple model, formulated in the early 1970s when the collicular maps were first found, assumes point-to-point connections between the visual and motor maps. In practice, however, these connections have been difficult to demonstrate. Neither the anatomical nor the physiological methods available at the time were sufficiently precise to establish these postulated synaptic connections. At about the same time, motor neurons were found to command saccades to nonvisual stimuli; moreover, spontaneous saccades occur in the dark. Thus, it was clear that visual-layer activity is not always necessary for saccades. To confuse matters further,

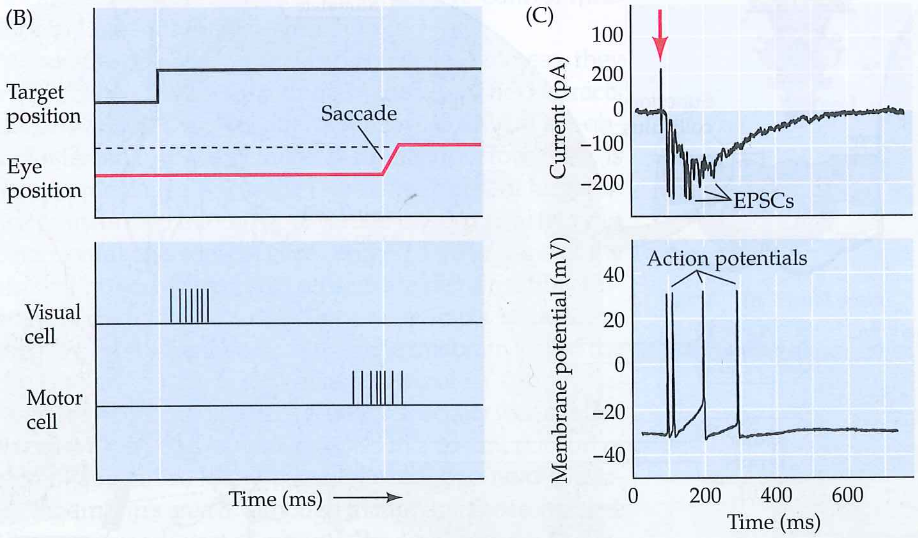
Continued on the next page

BOX 20B ■ *(continued)*

animals could be trained *not* to make a saccade when an object appeared in the visual field, showing that the activation of visual neurons is sometimes insufficient to command saccades. The fact that activity of neurons in the visual map is *neither necessary nor sufficient* for eliciting saccades led investigators away from the simple model of direct connections between corresponding regions of the two maps, toward models that linked the layers indirectly through pathways that detoured through the cortex.

Eventually, however, new and better methods resolved this uncertainty. Techniques for filling single cells with axonal tracers showed an overlap between descending visual-layer axons and ascending motor-layer dendrites, in accord with direct anatomical connections between corresponding regions of the maps. At the same time, in vitro whole-cell patch clamp recording (see Box 4A) permitted more discriminating functional studies that distinguished excitatory and inhibitory inputs to the motor cells. These experiments showed that the visual and motor layers do indeed have the functional connections required to initiate the command for a visually guided saccadic eye movement. A single brief electrical stimulus delivered to the superficial layer generates a prolonged burst of action potentials that resembles the command bursts that normally occur just before a saccade (Figures B and C).

These direct connections presumably provide the substrate for the very short-latency, reflex-like express saccades that are unaffected by destruction of the frontal eye fields. Other visual and nonvisual inputs to the deep layers probably explain why activation of the retina is neither necessary nor sufficient for the production of saccades.



(B) The onset of a target in the visual field (top trace) is followed, after a short interval, by a saccade to foveate the target (second trace). In the superior colliculus, the visual cell responds shortly after the onset of the target, while the motor cell responds later, just before the onset of the saccade. (C) Bursts of excitatory postsynaptic currents (EPSCs) recorded from a motor-layer neuron in response to a brief (0.5 ms) current stimulus applied via a steel wire electrode in the visual layer (top; see arrow). These synaptic currents generate bursts of action potentials in the same cell (bottom). (B after Wurtz and Albano, 1980; C after Ozen et al., 2000.)

encoded in movement coordinates so that both sensory cues and cognitive signals (see below) can activate the motor responses required to move the eyes to the intended position in the orbits. Thus, the output of the superior colliculus specifies movement intention rather than movements to fixed positions in external space or on the body surface.

The organizing framework of this motor map was demonstrated in an ingenious series of studies performed by David Sparks and his colleagues at the University of Alabama. They showed that retinal error signals (i.e., the distance and direction of the retinal projection of the target from the fovea) in retinotopic coordinates are often not sufficient to localize saccade targets. Using trained monkeys, the investigators cued a voluntary saccade with a brief flash of light, but before the saccade could be initiated, they stimulated a site in the deep layers of the superior colliculus that induced a saccade away from the point of fixation. They recorded eye movements to determine whether the change in eye position induced by the stimulation had an impact on the direction and distance of the cued saccade (Figure 20.10). If the saccade vectors were determined simply by the retinotopic coordinates of the target, then the monkey would be expected to make a saccade of the cued direction and distance (about 10° in the upward direction in this example). However, because of the deviated starting position, the saccade should systematically miss the target position by the amount of the stimulation-induced deviation (indicated by the dashed arrow pointing upward to the dash-encircled T on the left side of Figure 20.10A). The results consistently showed, however, that this was not the case. The animals compensated for the stimulation-induced shift by performing a compensatory saccade (a saccade indicated by the oblique, black dashed arrow to the T that appears within a black circle—the actual target location—in Figure 20.10A). This compensatory action was based on stored information about

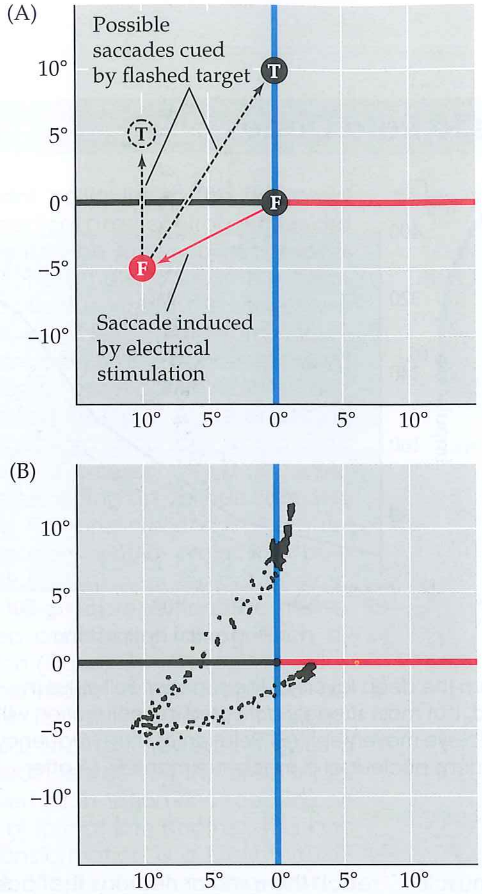


FIGURE 20.10 Saccades are encoded in movement coordinates, not retinotopic coordinates. (A) Map of visual space illustrating experimental design. Monkeys were trained to fixate a central location (F, in black) and then perform a saccade to a remembered target location cued by a brief flash at a location 10° above the starting position (T, in black). After cueing, but before expression of the cued saccade, an electrical stimulus was applied to a site in the superior colliculus that induced a saccade down and to the left (to the location marked by the F in red). If the cued saccade was encoded in retinotopic coordinates, the monkey should move its eyes 10° above the stimulus-induced position of foveation (F, in red) to a location marked by the dash-encircled T. If the saccade was encoded in movement coordinates, then a compensatory saccade to the cued target location (T, in black) would be expected. (B) Consistent with the encoding of saccades in movement coordinates, the monkeys performed compensatory saccades upward and to the right, toward the location of the cued target. Dots represent eye movements sampled at 500 Hz. (After Sparks and Mays, 1983.)

the location of the retinal image and current information about the position of the eyes in the orbit. The upper motor neurons that initiate the compensatory saccade are located at the expected site in the motor map of saccade vectors, but their activation depends on information in addition to the retinotopic location of the target. This information may be provided by circuits in the cerebral cortex that integrate this information and, in turn, activate the site in the superior colliculus that initiates the compensatory saccade (see Figure 20.10B).

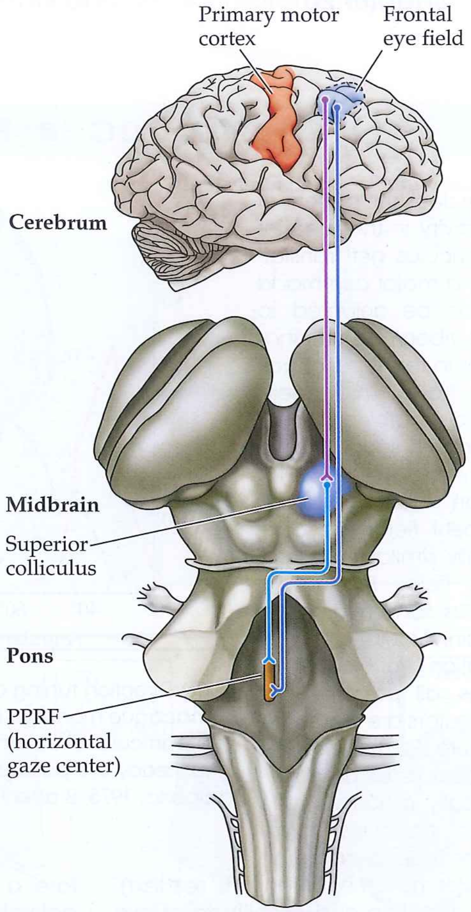
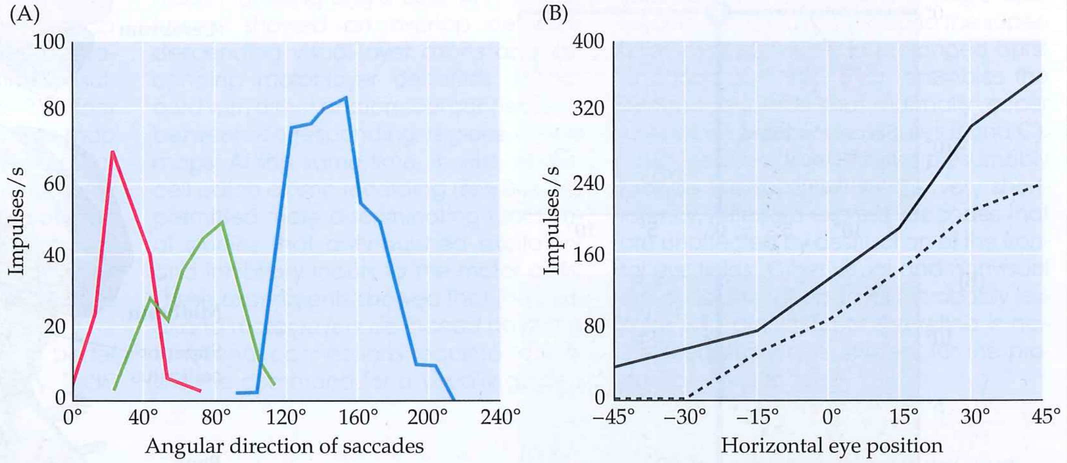


FIGURE 20.11 Neurons in the frontal eye field collaborate with cells in the superior colliculus to control eye movements. The projections shown here are from the frontal eye field in the right cerebral hemisphere (Brodmann's area 8) to the superior colliculus and the horizontal gaze center (PPRF). In humans, the frontal eye field can influence eye movements by either of two routes: indirectly, by projections to the ipsilateral superior colliculus, which in turn projects to the contralateral PPRF; and directly, by projections to the contralateral PPRF.

This study and several that followed showed that signals from different sensory modalities are integrated and transformed into a common motor frame of reference that encodes the direction and distance of the eye movements necessary to foveate an intended target. This "place code" for intended eye position generated in the upper motor neurons of the superior colliculus is then translated into a "rate code" by downstream gaze centers in the reticular formation that can then direct the activity of lower motor neurons in the ocular motor nuclei (Box 20C).

The eye movement regions of the cerebral cortex collaborate with the superior colliculus in controlling saccades. Thus, the frontal eye field projects to the superior colliculus, and the superior colliculus projects to the PPRF on the contralateral side (Figure 20.11). (The superior colliculus also projects to the vertical gaze center, but for simplicity the discussion here is limited to the PPRF.) The frontal eye field can thus control eye movements by

BOX 20C ■ From Place Codes to Rate Codes



(A) Direction tuning of three neurons recorded from the deep layers of the superior colliculus in macaque monkeys. Each neuron is broadly tuned, but most strongly activated in conjunction with a particular direction (and amplitude) of saccadic eye movement. (B) Relation of firing frequency to steady eye position in two neurons in the abducens nucleus of a macaque monkey. (A after Sparks, 1975; B after Fuchs and Luschei, 1970.)

How does the pattern of activity in the superior colliculus get translated into a motor command that can be delivered to muscle fibers? Recall that neurons in the superior colliculus have "movement fields," discharging in conjunction with saccadic eye movements of a particular direction and amplitude. Movement fields are conceptually similar to the receptive fields that occur in various sensory areas of the brain. Across the entire population of collicular neurons, all possible saccade vectors are represented (Figure A). Because the movement fields are topographically organized, the superior colliculus forms a *motor map* of saccade vectors (or movement intentions; see text).

The direction and amplitude of eye movements are encoded quite differently by the extraocular muscles (Figure B). *Direction* is controlled by the ratio of activation of the different muscles, and *amplitude* is controlled by the magnitude of the activation of those muscles. In other words, to make a saccade go farther, the muscle pulling the eye must pull harder and longer than it would for a shorter saccade. Amplitude is therefore a *monotonic* function of muscle activation.

The pattern of activity must be transformed from a code in which collicular neurons are tuned for particular saccade amplitudes to a code in which most or all α motor neurons respond, regardless of saccade amplitude, but the level or duration of their activity varies monotonically with saccade amplitude. This transformation occurs before signals from the superior colliculus reach the α motor neurons that activate the extraocular muscles.

Various models have been proposed to explain this transformation. The basic idea, shared by all models, is that the saccade vector, as signaled by the locus of activity in the superior colliculus, is decomposed into two monotonic amplitude signals corresponding rough ly to the horizontal and vertical components of the saccade vector. The weights of the projections from the

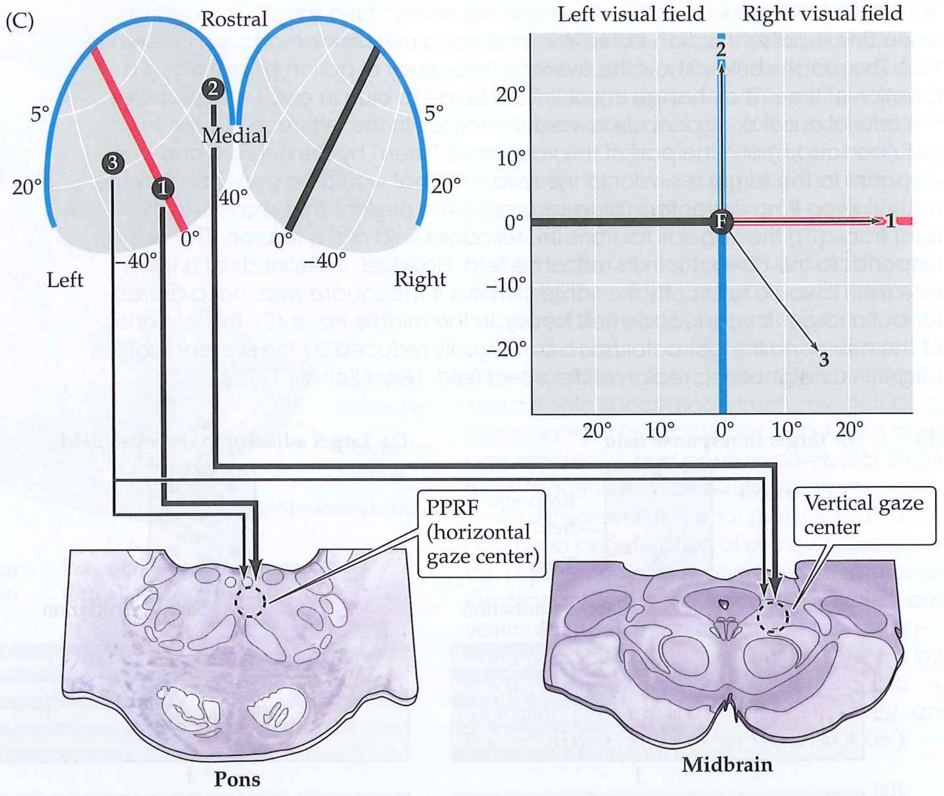
activating selected populations of upper motor neurons in the superior colliculus. This cortical area also projects directly to the contralateral PPRF; as a result, the frontal eye field can also control eye movements independently of the superior colliculus. The parallel inputs to the PPRF from the frontal eye field and superior colliculus are reflected in the different deficits that result from damage to these structures. Injury to the frontal eye field results in an inability to make saccades to the contralateral side and a deviation of the eyes to the side of the lesion. These effects are transient, however; in monkeys with experimentally induced lesions of this cortical region, recovery is virtually complete in 2 to 4 weeks. Lesions of the superior colliculus increase the latency and decrease the accuracy, frequency, and velocity of saccades; yet saccades still occur, and the deficits also improve with time. These results suggest that the frontal eye fields and the superior colliculus provide complementary pathways for the control of saccades. Moreover, one of these structures appears to be able to compensate (at least partially) for the loss of the other. In support of this interpretation, combined lesions of the frontal eye field and the superior colliculus produce a dramatic and permanent loss in the ability to make saccadic eye movements.

These observations do not, however, imply that the frontal eye fields and the superior colliculus have the same functions. Superior colliculus lesions produce a permanent deficit in the ability to perform very short-latency, reflex-like eye movements called **express saccades**. Express saccades are evidently mediated by direct pathways to the superior colliculus from the retina or visual cortex that can access the upper motor neurons in the colliculus

BOX 20C ■ *(continued)*

superior colliculus to the horizontal and vertical gaze control centers are thought to be tuned to accomplish this. For example, a site in the superior colliculus where the movement fields encode 5° rightward movements would project to the rightward horizontal gaze control center with a modest strength. A site encoding 10° rightward saccades would send a stronger projection to that center. A site encoding an oblique saccade with a 10° horizontal and a 5° vertical component would project to both the horizontal and vertical centers, with weights proportional to the required contribution along each direction (Figure C).

This model is too simple to account for all the relevant experimental findings. However, it gives a general idea of how the brain might convert information encoded in one kind of format into another. This kind of transformation is a likely requirement of sensorimotor integration in many behavioral contexts where sensory cues guide movement.

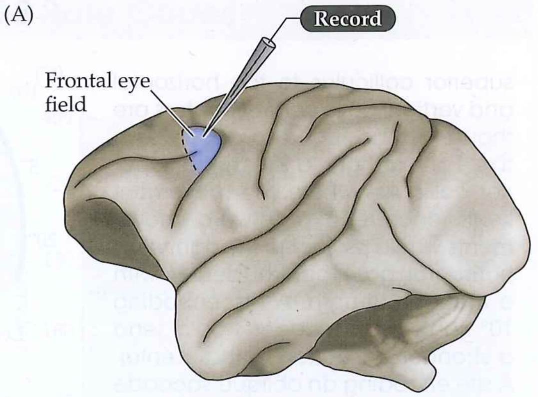


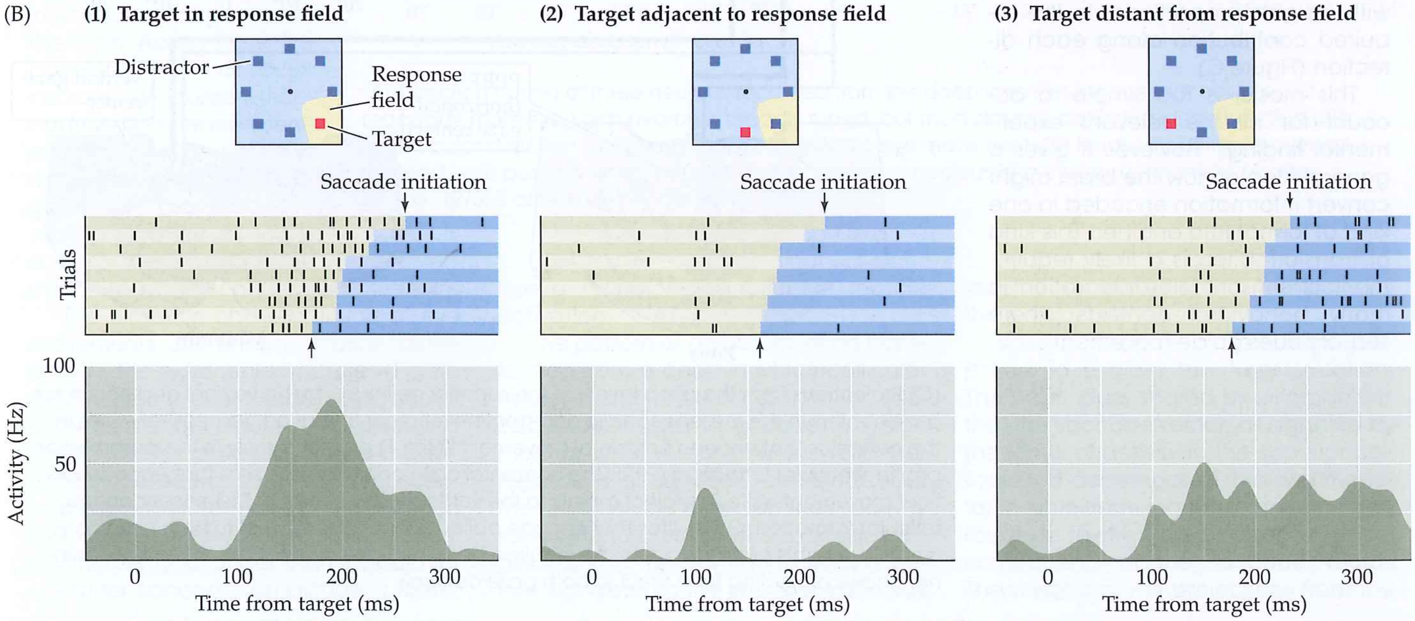
(C) Projections from the deep layers of the superior colliculus to the vertical and horizontal gaze centers in the mesencephalic and pontine reticular formation, respectively. Sites in the colliculus that encode horizontal movements (site 1) project mainly to the paramedian pontine reticular formation (PPRF, the horizontal gaze center), while sites that encode vertical movements (site 2) project mainly to the vertical gaze center in the mesencephalic reticular formation. Other sites that encode oblique saccades project to both gaze centers with weights proportional to the required horizontal and vertical displacements (thinner arrows projecting from site 3 to both gaze centers).

without extensive, and more time-consuming, processing in the frontal cortex (see Box 20B). In contrast, frontal eye field lesions produce permanent deficits in the ability to make saccades that are not guided by an external target. For example, people (or monkeys) with a lesion in the frontal eye field cannot voluntarily direct their eyes *away* from a stimulus in the visual field; this type of eye movement is called an "anti-saccade." Such lesions also eliminate the ability to make a saccade to the remembered location of a target that is no longer visible.

Finally, the frontal eye fields are essential for systematically scanning the visual field to locate an object of interest within an array of distracting objects (see Figure 20.1). Figure 20.12 shows the responses of a frontal eye field neuron during a visual task in which a monkey was required to foveate a target located within an array of distracting objects. This frontal eye field neuron discharges at different levels to the same stimulus, depending on whether the stimulus is the target of the saccade or a "distractor," and on the location of the distractor relative to the actual target. For example, the differences between the middle and the left and right traces in Figure 20.12 demonstrate that the response to the distractor is much reduced if it is located close to the target in the visual field. Results such as these suggest that lateral interactions within the frontal eye fields enhance the neuronal responses to stimuli that will be selected as saccade targets. They also suggest that such interactions suppress the responses to uninteresting and potentially distracting stimuli. These sorts of interactions presumably reduce the occurrence of unwanted saccades to distracting stimuli in the visual field.

FIGURE 20.12 Responses of neurons in the frontal eye fields. (A) Locus of the left frontal eye field on a lateral view of the rhesus monkey brain. (B) Activation of a frontal eye field neuron during visual search for a target. The vertical tick marks represent action potentials, and each row of tick marks is a different trial. The graphs below show the average frequency of action potentials as a function of time. The change in color from beige to blue in each row indicates the time of onset of a saccade toward the target. In the left trace (1), the target (red square) is in the part of the visual field "seen" by the neuron, and the response to the target is similar to the response that would be generated by the neuron even if no distractors (blue squares) were present (not shown). In the right trace (3), the target is far from the response field of the neuron. The neuron responds to the distractor in its response field. However, it responds at a lower rate than it would to exactly the same stimulus if the square were not a distractor but a target for a saccade (left trace). In the middle trace (2), the response of the neuron to the distractor has been sharply reduced by the presence of the target in a neighboring region of the visual field. (After Schall, 1995.)





Neural Control of Smooth Pursuit Movements

Until recently, smooth pursuit and saccades were considered to be mediated by different structures, but studies such as those carried out by Richard Krauzlis at the Salk Institute for Biological Studies indicate that these two types of eye movements involve many of the same structures. Not only are smooth pursuit movements mediated by neurons in the PPRF, they also are under the influence of motor control centers in the rostral superior colliculus and subareas within the frontal eye fields, both of which receive sensory input from the dorsal spatial vision pathway in the parietal and temporal lobes. The exact routes by which visual information reaches the PPRF to generate smooth pursuit movements are not known, but pathways from the cortex to the superior colliculus and PPRF similar to those that mediate saccades may play a role; an indirect pathway through the cerebellum also has been suggested (Figure 20.13). It is clear, however, that neurons in the striate and extrastriate visual areas provide sensory information that is essential for the initiation and accurate guidance of smooth pursuit movements. In monkeys, neurons in the middle temporal area (which is largely concerned with the perception of moving stimuli; see Chapter 12) respond selectively to targets moving in a specific direction, and damage to this area disrupts smooth pursuit movements. In humans, damage of comparable areas in the parietal and occipital lobes also results in abnormalities of smooth pursuit movements. Finally, a pathway from the retina that detects movements of the visual stimulus on the retina (retinal drift) terminates in the cerebellum after relays in the pretectum and inferior olive (see Chapter 19), and adjusts the gain of this system to ensure that the velocity of the eye movements matches that of the movement of the visual target.

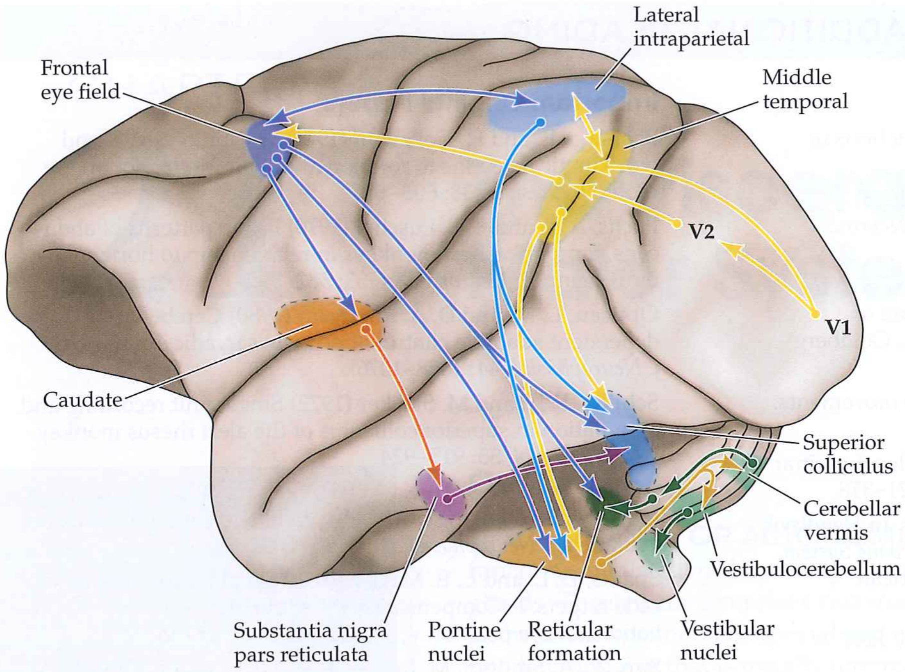


FIGURE 20.13 Sensory and motor structures and the connections that govern saccadic and smooth pursuit eye movements. This illustration summarizes data from studies on the rhesus macaque brain. Although these two types of eye movements were once thought to be controlled by separate circuits in the forebrain and brainstem, it is now recognized that they are governed by similar networks of cortical and subcortical structures. Visual signals are processed by the dorsal spatial vision pathway, including the middle temporal and lateral intraparietal areas. Sensory and attentional signals then guide motor planning areas in the frontal eye field. These cortical areas interact with subcortical structures, including basal ganglia (caudate and substantia nigra pars reticulata) and pontine-cerebellar structures (pontine nuclei, cerebellar vermis, and vestibulocerebellum), that modulate the initiation and coordination of eye movements by the superior colliculus and downstream oculomotor centers in the reticular formation and vestibular nuclei. The eye movements regulated by this complex circuitry are guided by a variety of sensory and cognitive signals, including perception, attention, memory, and reward expectation. (After Krauzlis, 2005.)

Neural Control of Vergence Movements

When a person wishes to look from one object to another object that is located at a different distance from the eyes, a saccade is made that shifts the direction of gaze toward the new object, and the eyes either diverge or converge until the object falls on the fovea of each eye. The structures and pathways responsible for mediating such vergence movements are not well understood, but they appear to include several extrastriate areas in the occipital lobe. Information about the location of retinal activity is relayed through the two lateral geniculate nuclei to the cortex, where the information from the two eyes is integrated. The appropriate command to diverge or converge the eyes, which is based largely on information from the two eyes about the amount of binocular disparity (see Chapter 12), is then sent from the occipital cortex to "vergence centers" in the brainstem. One such center is a population of local circuit neurons located in the midbrain near the oculomotor nucleus. These neurons generate a burst of action potentials that initiate a vergence movement, and the frequency of the burst determines its velocity. There is a division of labor within the vergence center, so that some neurons command convergence movements while others command divergence movements. These neurons also coordinate vergence movements of the eyes with accommodation of the lens and pupillary constriction to maximize the clarity of images formed on the retina, as discussed in Chapter 11.

Summary

Despite their specialized function, the systems that control eye movements have much in common with the motor systems that govern movements of other parts of the body. Just as the spinal cord provides the basic circuitry for coordinating the actions of muscles around a joint, the reticular formation of the pons and midbrain provides the basic circuitry that mediates movements of the eyes. Descending projections from upper motor neurons in the superior colliculus and the frontal eye field innervate gaze centers in the brainstem, providing a basis for integrating eye movements with sensory information that indicates the location of objects in space. The superior colliculus and the frontal eye field are organized in a parallel as well as a hierarchical fashion, enabling one of these structures to compensate for the loss of the other. Eye movements, like other movements, are also under the control of the basal ganglia and cerebellum; this control ensures the proper initiation and successful execution of these relatively simple motor behaviors, thus allowing observers to interact efficiently with the visual environment.

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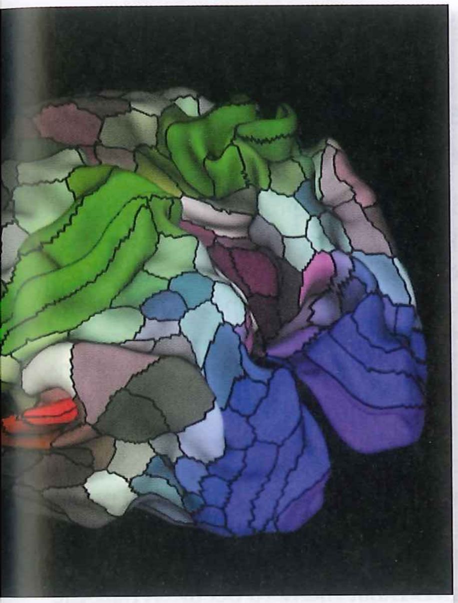
CHAPTER

29

Attention



Overview

ATTENTION IS THE CONSCIOUS OR UNCONSCIOUS FUNCTION that focuses on some external or internal stimulus, presumably at the expense of fully processing other information. This idea can be traced back to William James, who stated:

> *Everyone knows what attention is. It is the taking possession by the mind, in clear and vivid form, of one out of what seem several simultaneously possible objects or trains of thought. … It implies withdrawal from some things in order to deal effectively with others…*
>
> James, 1890 (*The Principles of Psychology*, pp. 403–404)

A problem, however, is the implication of a subjective "I" that decides to attend to one thing or another, as in "I paid attention to what he said." Who or what "I" might be raises broad philosophical as well as key neurobiological questions. To complicate matters further, attention must be distinguished from other terms that are related to it but not synonymous, for example, the difference between attention and other forms of arousal. As described in Chapter 28, wakefulness is a continuum of brain states that range from inattentiveness to a fully alert and aroused state when one is specifically paying attention to something. Studies of attention consider how and why we focus on particular aspects of the flood of information available in the internal and external environments. Although such studies were initially limited to the behavioral measures such as reaction time and processing accuracy, or the behavioral consequences of brain damage, over the last few decades neuroscientists have directly measured the influence of attention on brain activity during pertinent tasks. The goal of this chapter is to review the phenomenonology of attention, its implications, and the puzzles that it entails.

Attention as "Selective" Processing

Attention as "selective" refers to the allocation of neural resources to the analysis of particular information at the expense of resources that might have been allocated to other concurrent information that is in principle available. The quotation marks around "selective" are needed because it is unclear who or what the implied agent is, a quandary considered later in the chapter.

The usual example demonstrating selective attention is the *cocktail party effect*, wherein a listener can attend to one voice in a noisy conversation and "tune out" other simultaneous sound signals (Figure 29.1). An analysis of this effect was carried out in the 1950s by psychologist Colin Cherry who presented different dialogues to

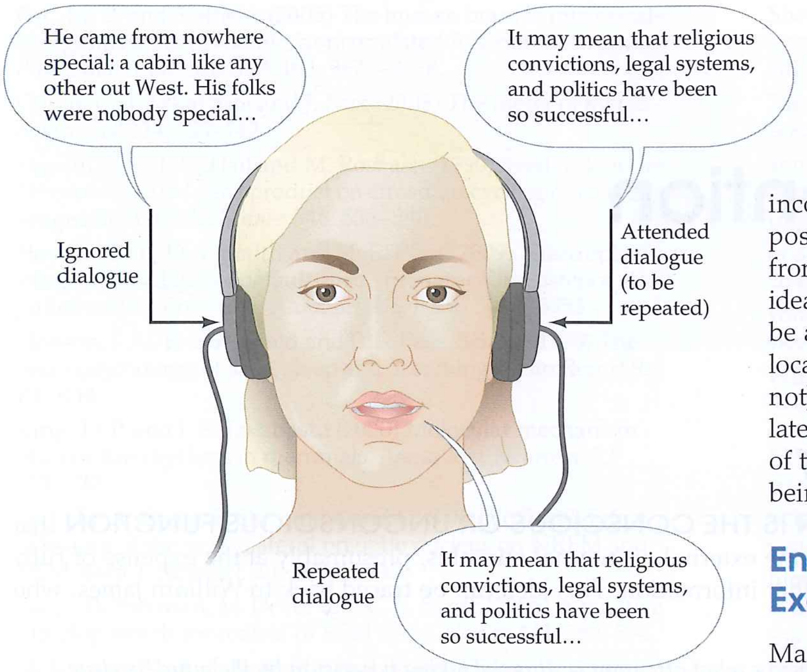


FIGURE 29.1 Attention as selective filtering. Colin Cherry's experiment using two voices speaking different dialogues presented separately to the left and right ears. See text for explanation.

each ear at the same time. Subjects were instructed to attend to only one of these inputs, and to immediately repeat the content to ensure that they were indeed attending to that stream of speech. Cherry then tested subjects' ability to report the content of the other, unattended input stream. Whereas the subjects could accurately report the content of the attended channel, they were unable to provide more than rudimentary information about the unattended stream. Cherry concluded that an attentional mechanism was filtering out unattended information at a relatively low level in sensory processing.

Although the idea that early attentional processing actively determines the information passed on to other parts of the nervous system for further analysis seems plausible, it soon became clear that at least some information in an unattended channel was being processed. A familiar example is attending to one's name when it is mentioned in otherwise unattended conversations. Consistent with this finding, other investigators proposed a "late-selection model" in which information filtering occurs relatively late in sensory processing pathways. According to this theory, only after higher-level processing is complete does an attentional mechanism determine what input information enters consciousness or influences behavior. At about the same time, psychologist Anne Treisman suggested a filtering system that could attenuate the inputs from concurrent sensory channels in a flexible manner. In an unattended channel, only especially salient or relevant information (e.g., one's name) would reach threshold for further processing and ultimately entry into consciousness. Psychological theories of attention were revised still further by incorporating the influence of the "perceptual load" imposed by a task—that is, the difficulty of a task arising from stimulus complexity and/or presentation brevity. The idea was that when the load is low, more processing would be available for other inputs. Thus, depending on the allocation of neural resources, information would (or would not) be allowed to reach higher processing levels where late-selection mechanisms presumably operate. The merits of these various concepts of attentional filtering are still being studied today.

Endogenous versus Exogenous Attention

Many behavioral studies, like those of Cherry, entail *voluntary* attentional tasks. Following an experimenter's instruction (or subjectively fulfilling a self-generated desire), subjects consciously direct attention to a particular aspect of the environment, such as an individual voice or a location in visual space. This type of attention is called **endogenous attention**. A particularly useful paradigm for studying endogenous attention was developed by Michael Posner and colleagues in the late 1970s (Figure 29.2A). While subjects maintain visual fixation on a central point, a trial begins with a centrally presented cue, such as an arrow pointing to the left or right, indicating where an upcoming target stimulus is most likely to occur. When the target appears, regardless of whether it occurs in the cued location, the subject must perform a discrimination task, such as indicating whether the target is a circle or an oval. In most of the trials the target is presented at the cued location, but sometimes it is presented at another location. Figure 29.2B shows the typical behavioral results: subjects respond faster to targets appearing at the cued location ("valid") than those appearing away from the cued location ("invalid"). An advantage of this and related paradigms is quantification of attentional effects.

In contrast, stimuli arising from events or conditions in the environment that attract attention automatically trigger **exogenous attention**. Exogenous (or involuntary) attention thus refers to the situation in which an unexpected noise, flash of light, movement, or other salient stimulus causes a shift of focus, trumping whatever else one happens to be attending at that moment. In conjunction with this shift in attention, the unexpected stimulus also facilitates the processing of information in that region, at the same time diminishing the efficacy of processing elsewhere. Like endogenous attention, exogenous attention has been

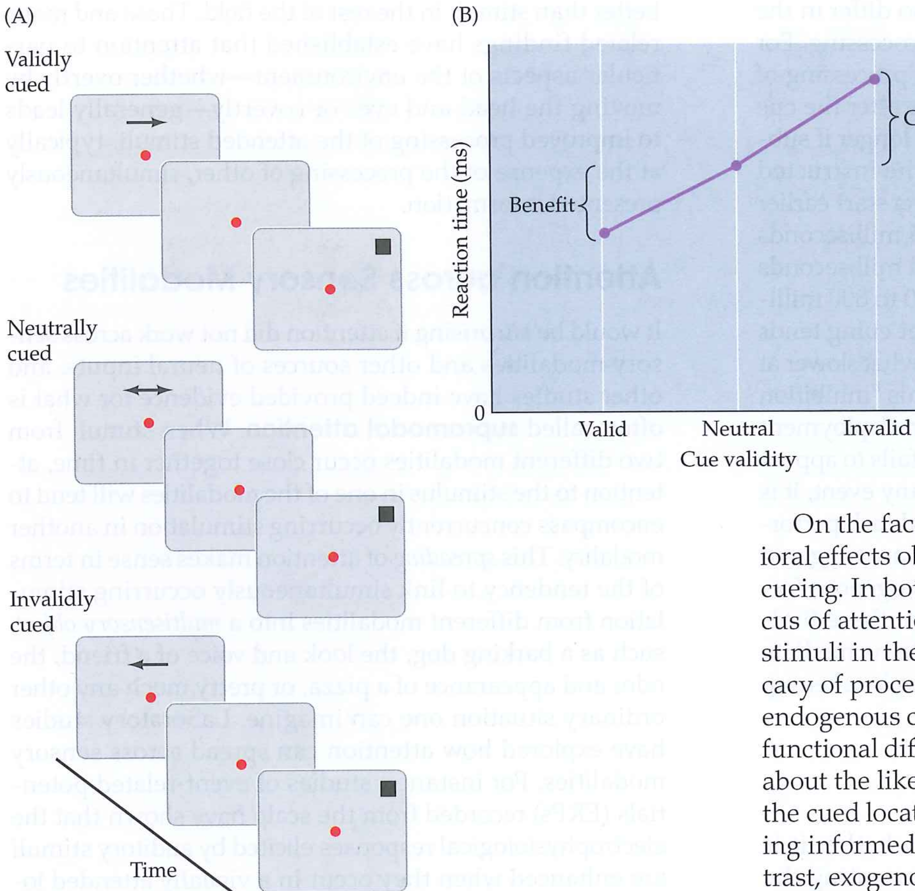


FIGURE 29.2 A cueing paradigm for studying endogenous visual spatial attention. (A) In this paradigm, a centrally presented instructional cue indicates where a target will most likely be presented (validly cued), where it will be less likely to be presented (invalidly cued), and where the cue provides no information as to the likely target location (neutrally cued). (B) Typical results show the benefits and costs in the reaction time for target detection after valid and invalid cueing, relative to the neutral-cue condition. (After Posner et al., 1980.)

studied in a variety of behavioral experiments. One approach has used trial-by-trial cueing in which a sensory cue such as a flash of light is presented at a particular location shortly before a target stimulus is presented either in that location or elsewhere (Figure 29.3). In such circumstances, subjects are again quicker to respond to a target presented in the cued location compared with an uncued location.

On the face of it, this sort of paradigm and the behavioral effects observed are similar to studies of endogenous cueing. In both instances, a cue induces a shift in the focus of attention that facilitates the sensory processing of stimuli in the attended region, and diminishes the efficacy of processing elsewhere. Despite these similarities, endogenous cueing and exogenous cueing have important functional differences. In endogenous cueing, information about the likelihood that the target stimulus will occur in the cued location is provided by prior knowledge (e.g., being informed where the target is likely to occur). In contrast, exogenous attention is not driven by any explicit information about a likely target location. That is, even if an exogenous cue (e.g., a flash) is presented randomly in the two possible locations from trial to trial, and thus has no predictive value about where a target will occur, processing in the cued location is nonetheless facilitated, presumably because the flash preceding the appearance of a target automatically draws attention to that location.

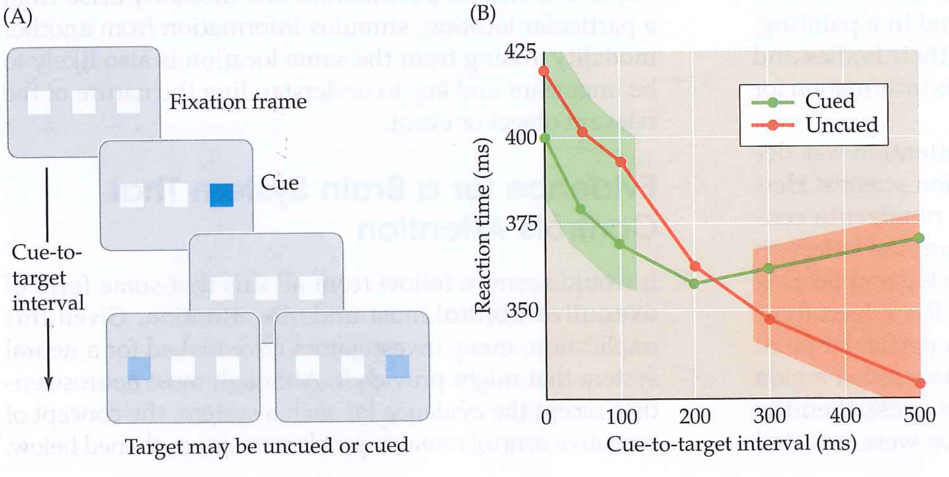


FIGURE 29.3 Exogenously triggered attention. (A) In this paradigm a brief flash is presented in one of two possible target locations, serving as an exogenous cue for a target that might follow at that location or at the other location. The occurrence of a target at the cued versus uncued location is random, with the probability at each location being 50%. (B) Shortly after the exogenous cue (green-shaded time period), stimulus processing at that location is facilitated, as indicated by faster response times to cued relative to uncued targets. At longer intervals (orange-shaded time period), however, there is a decrement in performance for the cued targets, known as *inhibition of return*. (From Klein, 2000; data from Posner and Cohen, 1984.)

Endogenous and exogenous attention also differ in the time courses of their influence on target processing. For endogenously cued attention, the improved processing of a cued target begins about 300 milliseconds after the cue and can last for some seconds afterward, or longer if subjects maintain their focus of attention on the instructed location. In contrast, exogenous cueing effects start earlier and are short-lived, beginning as early as 75 milliseconds after the cue and lasting only a few hundred milliseconds or so. Moreover, at still longer intervals (~400 to 800 milliseconds after the cue), the effect of the target cuing tends to reverse, with subjects actually being somewhat slower at responding to targets in the cued location. This "inhibition of return" probably reflects the reasonable redeployment of attention to other locations when a target fails to appear within a short time at the cued location. In any event, it is clear that the pattern of effects on behavioral task performance differs between attentional shifts that are triggered endogenously and those that are driven by exogenous factors. One important question emerging from these findings is whether or not different neural systems mediate exogenous and endogenous attention—a topic taken up in more detail below.

Covert Attention

Another way of categorizing attention is whether it is overt or covert. **Overt attention** involves orienting the head and eyes to a stimulus, thereby aligning visual (and auditory) processing with it and improving perception. **Covert attention** involves somehow directing attention to a stimulus without moving the head or eyes. The Russian psychologist and cyberneticist Alfred Yarbus first quantified overt attention by measuring subjects' patterns of gaze in response to viewing paintings and sculptures, using an ingenious system of small mirrors glued to the eyes of participants which redirected light to photo-tracing paper. He found that, in the absence of any instructions, participants tended to look at the faces and eyes of individuals in the artwork. But when instructed to ascertain the ages or wealth of individuals depicted in a painting, their gaze patterns shifted to focus on their bodies and clothing, reflecting the importance of this information for solving the task.

An experimental example of covert attention was described by the German physicist and vision scientist Hermann von Helmholtz at the end of the nineteenth century. When Helmholtz briefly flashed arrays of letters on a screen and asked subjects to report the letter appearing at a particular location, he observed that if a subject (typically himself) steadily fixated gaze on a particular point in the visual field but directed attention to another region of the field (that is, without moving the eyes), then the stimuli presented in the attended location were reported better than stimuli in the rest of the field. These and many related findings have established that attention to particular aspects of the environment—whether overtly by moving the head and eyes or covertly—generally leads to improved processing of the attended stimuli, typically at the expense of the processing of other, simultaneously presented information.

Attention across Sensory Modalities

It would be surprising if attention did not work across sensory modalities and other sources of neural inputs, and other studies have indeed provided evidence for what is often called **supramodal attention**. When stimuli from two different modalities occur close together in time, attention to the stimulus in one of the modalities will tend to encompass concurrently occurring stimulation in another modality. This *spreading* of attention makes sense in terms of the tendency to link simultaneously occurring stimulation from different modalities into a *multisensory object*, such as a barking dog, the look and voice of a friend, the odor and appearance of a pizza, or pretty much any other ordinary situation one can imagine. Laboratory studies have explored how attention can spread across sensory modalities. For instance, studies of event-related potentials (ERPs) recorded from the scalp have shown that the electrophysiological responses elicited by auditory stimuli are enhanced when they occur in a visually attended location, even when they are task-irrelevant. Correspondingly, ERP responses to task-irrelevant visual stimuli are enhanced when they occur in a location being attended for auditory stimuli. Similar results are observed between the visual and tactile modalities, as well as between tactile and auditory modalities. Complementary studies using fMRI have indicated that these enhanced responses to stimuli in the task-irrelevant modality include increased activity at relatively low-level processing areas in the sensory cortices. The biological value of this supramodal linkage during spatial attention is easy to understand: if important stimuli pertinent to one modality arise from a particular location, stimulus information from another modality arising from the same location is also likely to be important and key to understanding the nature of the relevant object or event.

Evidence for a Brain System That Controls Attention

It would seem to follow from all this that some form of **executive control** must underlie attention. Given this implication, many investigators have looked for a neural system that might provide it. Although most neuroscientists accept the evidence for such a system, the concept of executive control remains problematic, as explained below.

Notably, there is a strong overlap in the circuits that control attention and those that govern movements of the head and eyes. This overlay suggests attentional control builds upon systems that originally evolved to orient organisms to objects and events in the world that were crucial for successful behavior, belying the notion of a privileged central executive in control of attention.

Evidence from Neurological Patients: Hemispatial Neglect Syndrome

The earliest evidence for an anatomically definable system of attentional control came from neurological patients. A relatively common lesion is injury to the right inferior parietal lobe and adjacent regions (Figure 29.4). As introduced in Chapter 27, such lesions, first described in the 1940s, cause deficits in attention to the left side of personal and extrapersonal space (i.e., the side contralateral to the lesion). Right parietal, right superior temporal, or right frontal brain damage can all lead to difficulty attending to the left side of visual space and/or the left side of objects.

Since objects in the left visual field stimulate the visual system normally in such patients, the primary problem is attentional, not sensory. Depending on the extent and severity of the lesion, when an object in the left visual field is specifically pointed out or the object is made particularly salient (for example, by presenting food to a hungry individual), patients then tend to report being able to see it. Thus, these impairments are quite different from the visual deficit that follows a lesion in the visual cortex (see Chapter 12). Patients with visual cortical lesions are effectively blind in specific corresponding parts of the contralateral visual field. In contrast, the underlying problem with lesions to right parietal cortex appears to be an attentional deficit, not a sensory one; the patients can apparently *see* stimuli in the left visual field, but they tend not to notice them or be able to orient their attention to them effectively. Although these deficits are often most obvious in vision, they are evident in other sensory modalities. For instance, many patients with right hemisphere damage are less able to attend to the left side of their own body, as shown by the tendency to shave or apply makeup on only the right side of the face, or to dress only the right side of the body. This constellation of signs and symptoms is called **hemispatial** (or **contralateral**) **neglect syndrome**.

A possible reason why attentional deficits are most often associated with right parietal lesions is that this region influences mechanisms of attention in *both* hemispheres, whereas the corresponding left parietal area influences mainly those on the right. There are, however, alternative explanations, and the anatomy is a good deal more complex than implied here. For instance, some investigators have suggested that the relatively greater importance of right parietal cortex in neglect syndromes reflects right hemispheric lateralization for vigilance or alertness. Interestingly, in non-human primates damage to the inferior parietal lobe on either the left or right side induces neglect of contralateral space.

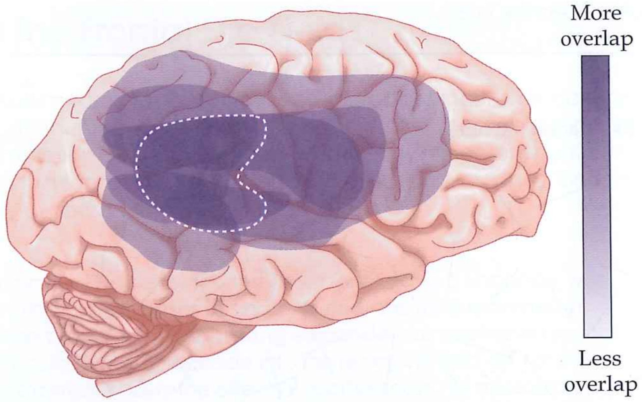


FIGURE 29.4 Cortical lesions leading to left hemispatial neglect syndrome. This composite diagram shows the distribution of right-hemisphere damage in eight patients with left hemispatial neglect. The degree of overlap of damaged brain areas across patients is indicated by shading level. Although some of the lesions include parietal and frontal lobes, as well as parts of the temporal lobe, the region most commonly affected is in the right inferior parietal lobe (dashed line). (After Heilman and Valenstein, 1985.)

As shown in Figure 29.5, the left-sided neglect evident in these patients can be demonstrated clinically by one of several simple tests. In the single-line bisection test (see Figure 29.5A), patients are asked to mark the center of a horizontal line. Patients with neglect tend to ignore the left side of the line and thus their estimate of the center is displaced to the right. In the line cancellation test (see Figure 29.5B), patients are asked to draw a line through each of several lines scattered across a page; in this test, patients cancel lines mainly on the right side of the page. In addition, the left-sided neglect is not limited to ignoring objects in the left hemispace; patients with this syndrome also tend to ignore the left sides of objects *wherever* they are in visual space. For example, if asked to draw a copy of an object, these patients tend to draw only its right side (see Figure 29.5C). Such patients even tend to ignore the left side of their visual imagery and memory. So if asked to draw a clock from memory, they are likely to draw half a clock, sometimes remembering to include all 12 numbers in the drawing, but placing all of the numbers on the right (see Figure 29.5D).

Assessments of hemispatial neglect patients also elicit *extinction*. This phenomenon is revealed when the neurologist stands in front of the patient with arms outstretched and moves a finger on either the right or the left hand. If

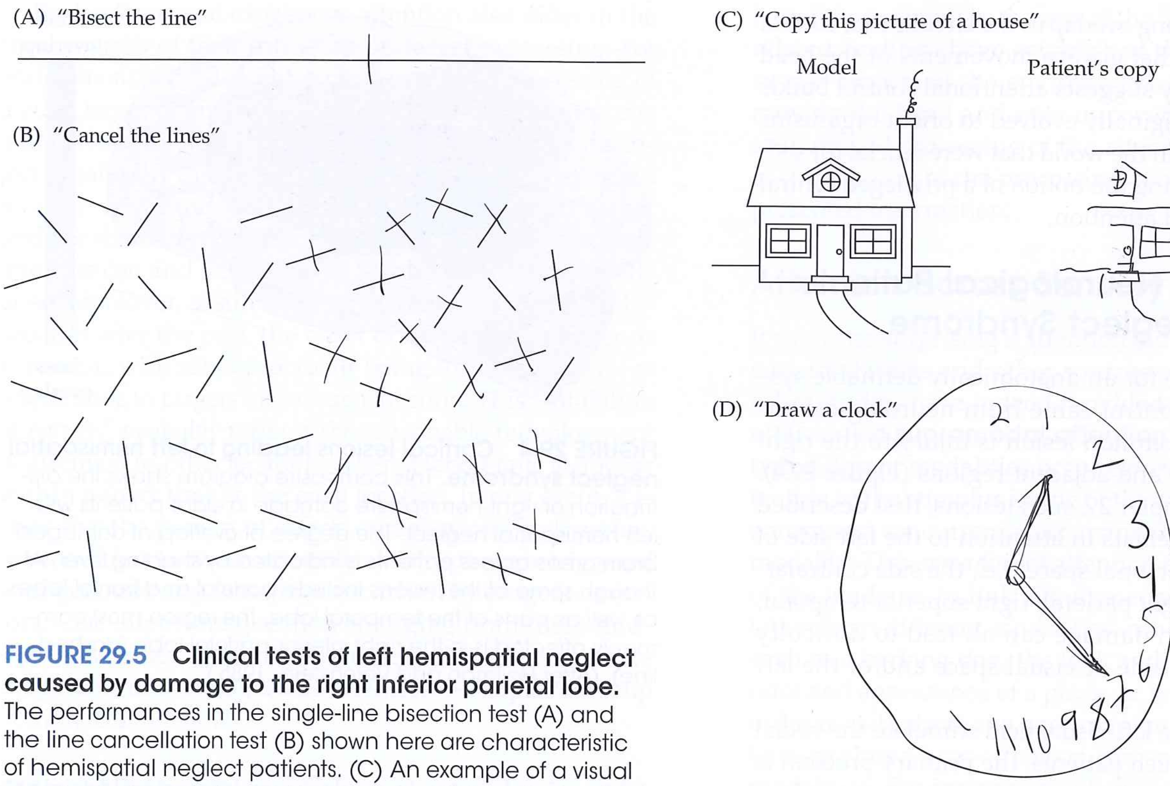


FIGURE 29.5 Clinical tests of left hemispatial neglect caused by damage to the right inferior parietal lobe. The performances in the single-line bisection test (A) and the line cancellation test (B) shown here are characteristic of hemispatial neglect patients. (C) An example of a visual copying task as performed by a hemispatial neglect patient. (D) A drawing of a clock face from memory by a hemispatial neglect patient. (A,C after Posner and Raichle, 1994; B after Blumenfeld, 2010; D after Grabowecky et al., 1993.)

a finger on either side is moved by itself, the patient generally reports the presence of the moving finger correctly, presumably because a moving stimulus is a particularly strong attractor of attention, even for these patients. If both fingers are moved at the same time, however, the patient typically reports seeing only the one on the right. This test suggests that the normal competition between the stimulus inputs from the two sides is now dominated by the right visual field, which "extinguishes" the input from the left. Extinction emphasizes again that the underlying problem is an attentional deficit, not a sensory one.

Other Brain Regions That Affect Attention

Lesions in parts of the frontal cortex that are connected to the parietal cortex can also cause attentional deficits. In particular, lesions to the **frontal eye fields** disrupt the ability both to initiate eye movements to targets in the contralateral visual field and to direct attention toward that side (Box 29A). Moreover, lesions in these frontal regions can interfere with attentional functions such as task switching and ignoring irrelevant information. As a rule, unilateral frontal lesions tend to have a greater effect on motor-related aspects of attention, compromising the ability to initiate or direct eye or limb movements toward contralateral space.

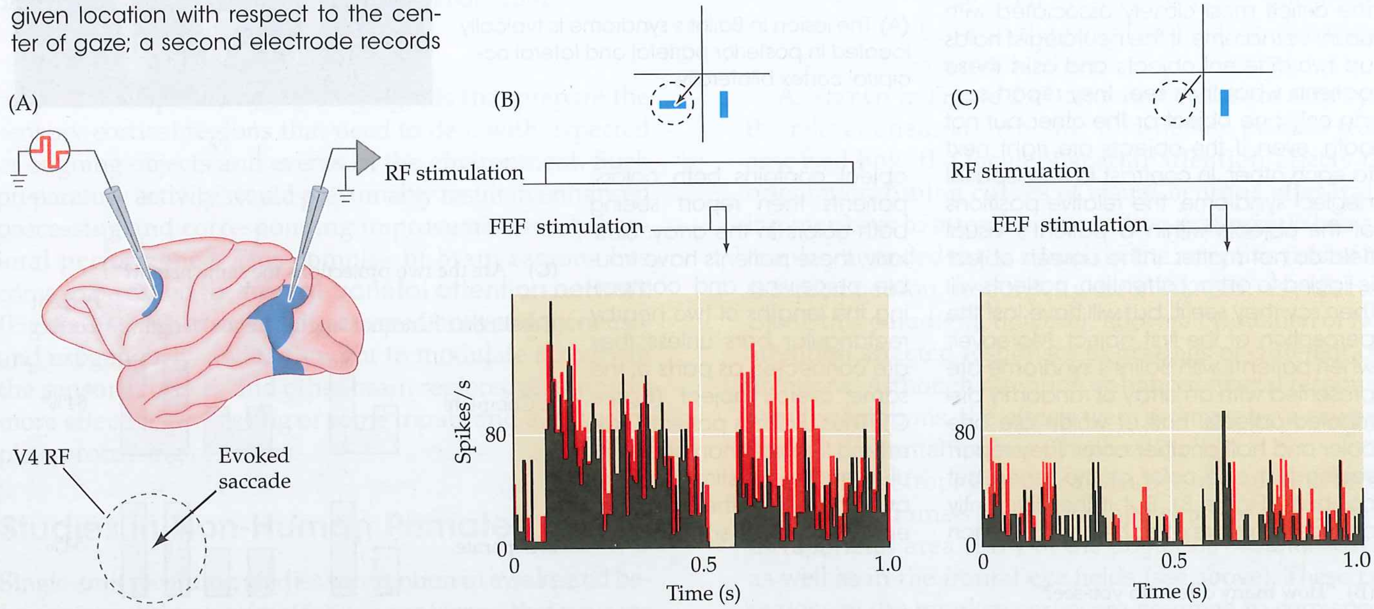
Brainstem lesions can also affect attentional control. The interactions between the superior colliculi and the parietal cortex are apparent in the so-called **Sprague effect**, in which the hemispatial neglect induced by a parietal lesion can be mostly compensated by a lesion of the superior colliculus on the other side. The proposed explanation is that the parietal lesion-induced neglect results not from the cortical damage itself, but from an imbalance of activity between the two parietal lobes in attentional control. According to this theory, a lesion of the contralateral colliculus helps to restore the appropriate balance, because of its connections to the parietal lobe on the same side. Regardless, this effect underscores an important role for the superior colliculus in attentional control, possibly via functional interactions with parietal cortex. Experimental studies in monkeys have demonstrated that these interactions are mediated by the pulvinar, the thalamic relay that connects the parietal cortex and the superior colliculus.

In sum, clinical evidence indicates that damage to a variety of brain areas can lead to deficits of attention with different characteristics and possible mechanisms (Clinical Applications). These findings are generally inconsistent

BOX 29A ■ Attention and the Frontal Eye Fields

One of the main issues in understanding attention is demonstrating the effect of one brain region on another as a way of supporting the concept of a specific attention network. In the example here, the effect of frontal eye field (FEF) stimulation on the response of a single neuron in the extrastriate visual cortex of a monkey is evaluated. The rationale of the experiment is that the normal function of the FEF is to generate saccadic eye movements to locations in visual space that warrant attention. Thus, to test the presence of a link and the effect of attention, a stimulating electrode is placed in the FEF in a locus that would evoke saccades to a given location with respect to the center of gaze; a second electrode records the activity of a visual cortical neuron responsive to the same location in visual space. As shown, FEF stimulation while the monkey attended the fixation point caused a saccade to the expected location and increased neuronal activity at the recoding site. The implication is that the FEF and this region of visual cortex (V4) comprise part of an attention network.

Microstimulation of sites within the FEF, below the threshold for eliciting a saccade, was carried out while the visual stimulus responses of single V4 neurons were recorded in monkeys performing a fixation task. (A) The stimulating electrode was positioned so that suprathreshold stimulation would evoke a saccade into the receptive field (RF) of the V4 cell under study. (B) This example shows the effect of subthreshold FEF microstimulation on the response of a single V4 neuron to an oriented bar presented in the cell's receptive field. The mean response during control trials is shown in black; the enhanced response arising from the FEF microstimulation is shown in red. (C) On trials in which the visual stimulus was presented outside the receptive field of the V4 neuron, no enhancement is seen. (After Moore et al., 2003.)



with the idea of a specific brain region devoted to attention and its control, and instead suggest that attention depends on a large number of brain regions working together to determine what we attend to.

Evidence from Normal Subjects

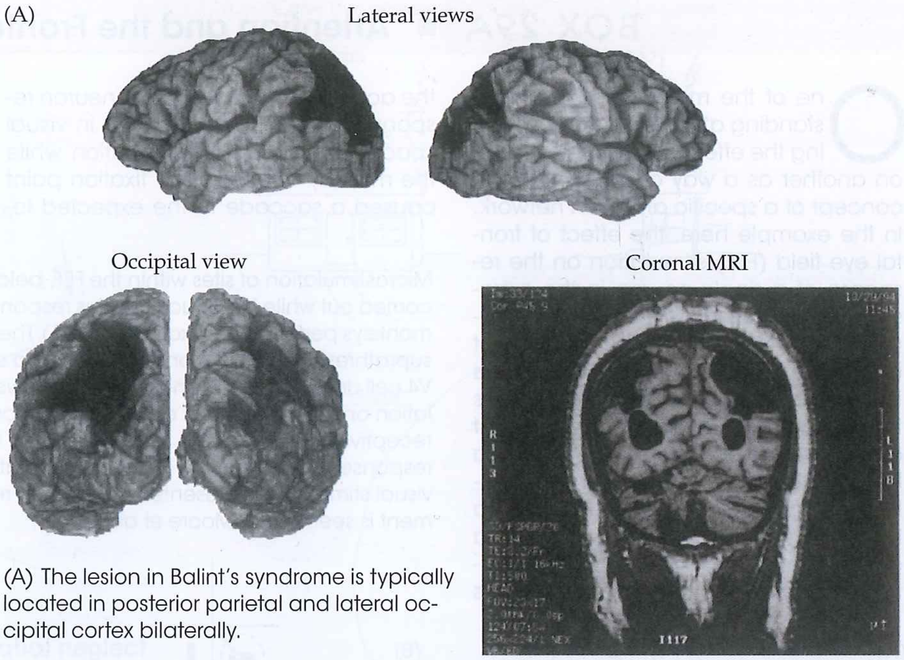
The advent of noninvasive brain-imaging methods has complemented clinical observations with studies of attention in normal participants. In accord with studies of neglect patients, tasks involving attention reliably activate a set of brain regions in the dorsal parietal and dorsolateral frontal cortices. Much the same regions are activated by attending voluntarily, be it to spatial locations or nonspatial features like color or orientation. Involuntary shifts in attention elicited by unexpected events are associated with additional activity in ventral areas of the parietal cortex in the vicinity of the right temporal–parietal junction, which in turn activates more dorsal regions of the frontal cortex.

Based on this and other evidence, Maurizio Corbetta and Gordon Shulman have proposed that attention is mediated by two interacting systems that carry out different functions. In this interpretation, one system consisting of regions of the intraparietal cortex and superior frontal cortex serves endogenous attention; the other system, consisting mainly of the cortex near the temporal–parietal junction and ventral frontal cortex (mainly in the right hemisphere), is specialized for the detection of unexpected or salient exogenous stimuli. Both systems are

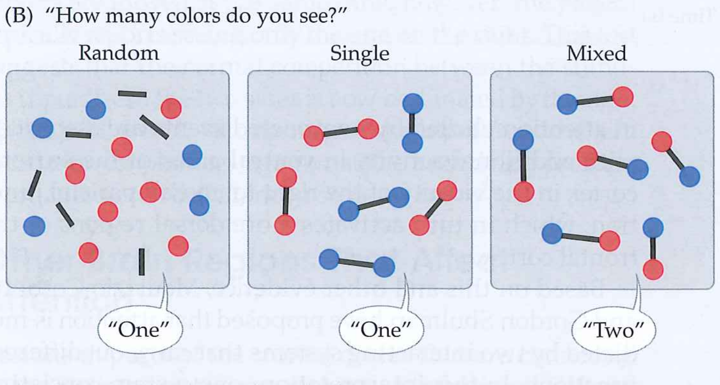
CLINICAL APPLICATIONS

Balint's Syndrome

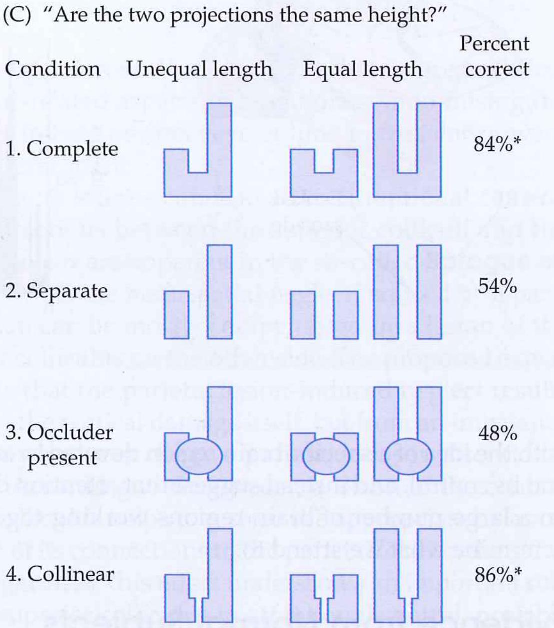
Another brain lesion that has striking effects on attention is bilateral damage to the dorsal posterior parietal and lateral occipital cortex, leading to a disorder known as **Balint's syndrome** (Figure A). Damage of this sort, however, presents quite a different clinical picture and an even more debilitating deficit. First characterized by the Hungarian physician Rezsö Bálint, the signature of this syndrome has three characteristics: (1) simultanagnosia, the inability to attend to and/or perceive more than one visual object at a time; (2) optic ataxia, the impaired ability to reach for or point to an object in space under visual guidance; and (3) oculomotor apraxia, difficulty voluntarily directing the eye gaze toward objects in the visual field with a saccade. Simultanagnosia is the deficit most closely associated with Balint's syndrome. If the neurologist holds up two different objects and asks these patients what they see, they report seeing only one object or the other but not both, even if the objects are right next to each other. In contrast to hemispatial neglect syndrome, the relative positions of the objects within a patient's visual field do not matter. If the unseen object is jiggled to attract attention, patients will then say they see it, but will have lost the perception of the first object. Moreover, when patients with Balint's syndrome are presented with an array of randomly distributed objects, half of which are one color and half another color, they report seeing just one color or the other, but not both (Figure B). But if the differently colored items are attached so that each object contains both colors, patients then report seeing both colors in the array. Similarly, these patients have trouble perceiving and comparing the lengths of two nearby rectangular bars unless they are connected as parts of the same overall object (Figure C). Thus, Balint's patients can attend to more than one stimulus quality or stimulus part, but only when the parts are embodied in the same object.



(A) The lesion in Balint's syndrome is typically located in posterior parietal and lateral occipital cortex bilaterally.



(B) The inability of Balint's patients to perceive or attend to more than one object at a time (simultanagnosia) prevents them from noticing more than one color in the displays shown in the left and middle panels. However, if the blue and red circles are connected to form single objects (right panel), the patient is able to report both colors, indicating that the deficit lies in the inability to attend to multiple objects rather than in a failure to attend to multiple qualities.



(C) Similarly, if patients are asked to compare the lengths of two nearby rectangles, their performance is near chance (50% correct) if the objects are separated, whereas they perform much better when the components are connected as part of the same object. The asterisk denotes performance statistically better than chance. (A from Friedman-Hill et al., 1995; B after Humphreys and Riddoch, 1993; C after Cooper and Humphreys, 2000.)

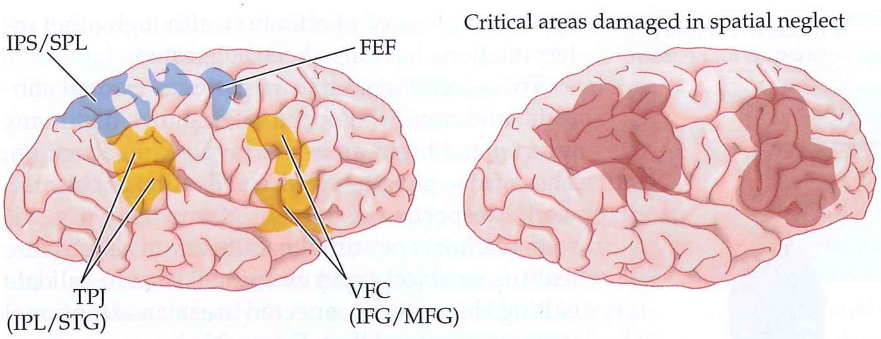


FIGURE 29.6 A postulated attentional control network, illustrated in the right hemisphere. The areas in blue indicate the dorsal frontal-parietal regions that tend to be activated by endogenous stimuli; the areas in yellow indicate the more ventral regions that tend to be activated during reorienting, and by exogenous stimuli. IPS/SPL = interparietal sulcus/superior parietal lobule; FEF = frontal eye fields; TPJ = temporal-parietal junction; IPL/STG = inferior parietal lobule/superior temporal gyrus; VFC = ventral frontal cortex; IFG/MFG = inferior frontal gyrus/middle frontal gyrus. (After Corbetta and Shulman, 2002.)

proposed to operate by sending signals that prepare the sensory cortical regions that need to deal with expected or ongoing objects and events in the environment. Such preparatory activity would presumably result in enhanced processing and corresponding improvements in behavioral performance. This complex of brain regions has come to be called the **frontal-parietal attention network** (Figure 29.6). The network is activated both endogenously and exogenously, and is thought to modulate activity in the sensory cortices and other brain regions, resulting in more effective processing of some inputs and a less complete processing of others.

Studies in Non-Human Primates

Single-unit recording studies of attention in awake and behaving non-human primates have confirmed that neurons in parietal and frontal regions homologous to the putative attention network in humans tend to be active when animals are attending to a task. These findings, mostly in macaque monkeys, have allowed researchers to study attention and the possible mechanisms of attentional selection at the neuronal level, most often using visual attention as the experimental paradigm.

In general, visual cortical neurons respond strongly only if a stimulus is presented within the cell's receptive field. The firing rate signals the optimal stimulus for that neuron (i.e., the characteristics to which the cell is tuned, such as a particular orientation, direction of movement, color, etc.). Once a cortical neuron is located and its receptive field characterized, the animal's attention can be manipulated to investigate its effects on neuronal responsiveness. When effective and ineffective stimuli (i.e., stimuli that matched or did not match the neuron's tuning curve) were presented together within a neuron's receptive field in visual area V4, the cell fired strongly only when the effective stimulus was being attended. When the monkey attended the ineffective stimulus, the neuron responded weakly, even though the stimulus had not changed (Figure 29.7). These observations indicate that neuronal responses depend on the locus of attention within a neuron's receptive field, at least for cells in these areas of cortex. In the later stages of visual processing—that is, in the ventral pathway leading to the inferior temporal cortex—the observed pattern was different. At this level, attention modulated the neuronal responses even if the ignored stimulus was relatively far away from the attended one, presumably because the receptive fields in this region are much larger.

As shown in Figure 29.7, attention enhances activity of the relevant neurons. Another non-human primate study assessed how the locus of spatial attention affects the orientation tuning curves of visual neurons after training monkeys to attend to one of two gratings. When the monkey attended to the stimuli in the receptive field of the recorded neuron, responses were enhanced, as expected. Using this paradigm, however, allowed evaluation of how attention affected responses to gratings of different orientations. Although attention enhanced neural responses at all orientations, the effects were stronger for a neuron's preferred orientation (see Chapter 12).

Other electrophysiological studies of attention in experimental animals have examined neurons in the lateral intraparietal area (LIP) of the posterior parietal cortex, as well as in the frontal eye fields (see above). These two regions of the monkey cortex are assumed to correspond with the parietal and frontal areas in humans where damage causes neglect syndromes, and where neuroimaging studies have shown activity related to attentional control. One interest in these particular areas is the possibility of "integrating centers" within a broader set of brain areas involved in attention. For instance, the firing rates of LIP neurons in response to a stimulus in their receptive field are greater when the task is to make a saccade to a target in the receptive field rather than simple fixation. Neuronal responses are also enhanced when a monkey attends to the stimulus in the receptive field but does not make a saccade, or when the saccade is delayed. These results suggest that enhanced neuronal responsiveness is not due to saccade preparation per se, but to the allocation of attention to the spatial location of the target in the neuron's receptive field, which occurs when the monkey plans to shift his gaze there.

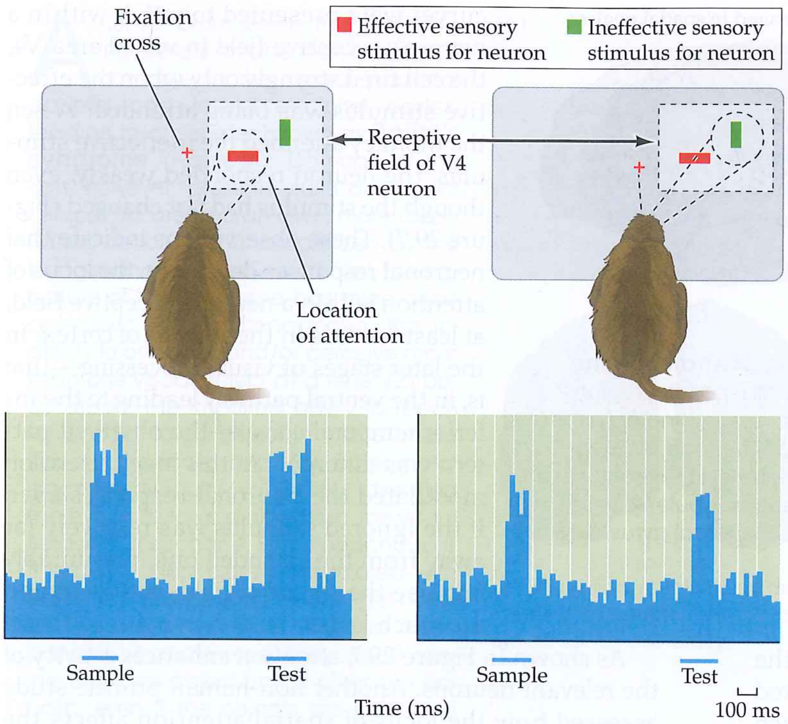


FIGURE 29.7 Effects of attention on the firing rates of single neurons in the visual cortex. At the attended location (circled), two stimuli—sample and test—were presented sequentially; the monkey had to discriminate whether they were the same or different. Irrelevant stimuli were presented simultaneously with the sample and test but at a separate location in the receptive field. Stimuli could either be effective stimuli for the neuron (red bars in this example) or ineffective stimuli (green bars). When both an effective stimulus and an ineffective stimulus were presented within the receptive field and the monkey attended to the effective stimulus, the neural responses were robust. When the monkey attended to the ineffective stimulus, however, the responses were much reduced, despite the presence of an effective stimulus in the receptive field. In short, the locus of attention has a clear effect on the activity the relevant neurons. (After Moran and Desimone 1985.)

A related question in experimental animals is how activity in these regions could lead to enhanced stimulus processing in the sensory cortices. Some relevant information had already been provided by fMRI studies in humans. When participants direct sustained attention to a particular visual-field location expecting the onset of a visual stimulus there, increased activity is elicited not only in the frontal and parietal cortices but in extrastriate cortex as well. The implication is that the increased activity in visual cortex in the absence of visual stimulation reflects preparatory signals from the frontal-parietal network that favors the attended location. In accord with this idea, microstimulation of the frontal eye fields in monkeys improves performance in attentional tasks and simultaneously increases the activity of neurons in V4 with receptive aligned with the retinotopic locus of stimulation. Saccade-related activity in the frontal eye fields has also supported this "premotor theory" of attention, although other interpretations have also been suggested.

This wealth of studies in patients, normal subjects, and non-human primates shows that many interrelated brain areas are involved in attention. These facts, plus the reliable activation of this network irrespective of the sensory modality (e.g., visual, auditory) or stimulus category (e.g., location, feature, or object type) examined, *seem* to validate calling these interconnected areas an attentional system or network (see Figure 29.6).

Problems with the Concept of Attention as Executive Control

Many aspects of the brain can be understood in terms of systems and subsystems; the visual system, the auditory system, the skeletal motor system, and the visceral motor system, to name a few, are anatomically and functionally specialized regions that carry out relatively specific tasks. Attention, however, does not fit easily into this category. The anatomical regions of association cortex illustrated in Figure 29.6 participate in many different neural functions and have no defining characteristic other than their enhanced activity when human subjects or other animals are focused on something. Although the putative attention network could be interpreted in functional rather than anatomical terms, this concept would still entail a network charged with assessing what is required for any given task, selecting the most relevant components of concurrent stimulus information and directing their flow. Thus, even a functional characterization of attention leaves the concept of executive control on center stage.

At a subjective level, the notion of executive control as the foundation of attention obviously has its uses. For example, we routinely make statements such as "I decided to pay attention" and assume a personal "I" that makes decisions and directs the focus of endogenous attention. But in terms of neuroscience, the concept of an executive controller that monitors the rest of the brain and makes decisions about its operation is hard to credit. An executive system would have to receive input from all the systems and subsystems the nervous system uses to sense the external and internal environment, take into account relevant information from memory, and integrate the affective and motivational state of the individual. To complicate matters further, the system would have to direct processing resources to any location in space, to any specifiable feature of an object or a scene, and to any point in time. In short, the neural processes that an attentional system would need to have to monitor and direct include the full gamut of information-processing circuitry entailed in normal brain function.

In response, advocates of the dedicated attentional system shown in Figure 29.6 would point out that these brain regions are indeed widely connected to many other cortical regions, as well as many subcortical brain structures (e.g., the subcortical components of the limbic system, the amygdala, the subthalamic nucleus, the superior colliculus, and the striatum, to name just some). But the fact remains that the only "system" fully capable of integrating the range of external and internal information needed to generate optimal behavior at any moment is the entire brain and the rest of the nervous system.

Alternative Interpretations of Attention

Most researchers in the field recognize these problems with the traditional conception of attention and have sought to deal with them in one way or another. One variation on idea of attentional control is that a variety of brain regions—including those responsible for short-term memory, emotional state, motivation, motor planning, and other areas—issue *biasing signals* to specialized nodal areas (e.g., the LIP and frontal eye fields) where signal integration and control occur. Although these biasing signals are not themselves taken to be attentional, they would nonetheless give rise to the privileged processing of some information at the expense of other input determined by the output of these nodes. So construed, the integration of biasing signals in specialized nodes behaves *as if* there were attentional control, but without an overarching executive.

Despite the appeal of this sort of explanation, the decentralization of attentional control among a set of integrating nodes assumes much the same theoretical burden as the traditional concept of attention. Unless one understands how and in what sense input acting on nodes can privilege some information, the problem of attentional selection has only been pushed back a step. Instead of attributing selection to a single executive, selection is now carried out by a plurality of minor executives, each charged with monitoring, winnowing, and redirecting incoming information. Although abolishing the idea of a dedicated executive system seems a step in the right direction, replacing it with a set of distributed executives does not resolve the basic problem inherent in the traditional concept of attention.

Another proposal for understanding attentional control is the idea of **saliency maps**. The interpretation in this case is that cortical areas such as the LIP and frontal eye fields—as well as subcortical structures such as the superior colliculus—contain maps defining corresponding regions of space or some other key quality. When incoming signals activate different locations within these maps, that region of space (or its equivalent) is given processing priority. Thus, at any given moment the highest level of activity in the combination of postulated saliency maps creates a de facto priority map that determines what we attend to. The activity in saliency maps is imagined to be affected by biasing signals from sensory input, the relevance of objects and locations for current goals, motor planning, differing motivations to respond to objects and locations, the emotional valence of stimuli, and so on. As a result, attention in one domain (e.g., visual) can be influenced by a range of additional (e.g., nonvisual) factors. While the idea of replacing a dedicated attentional system with saliency maps is also attractive, this approach suffers from a reliance on preparatory signals that arise in a manner that is not well defined in terms of locus or mechanism. Rather than resolving the flaws inherent in the traditional concept of attention, saliency maps may also be reformulating the basic problem in the concept of executive control, albeit in a manner consistent with newer evidence from brain-imaging and other studies.

Other conceptions of how attention could operate without a dedicated executive are based on computational principles. For example, redirection of attention could arise from attractor states found in dynamic systems (the brain being one such system). Thus, when an experimenter or naturally occurring stimuli "cue attention," the rules governing "attractor states" would redirect processing. Attention would thus be construed as an emergent phenomenon determined by the computational demands of efficient neural processing.

Another way that some investigators in engineering and computer science have thought about brain operations without executive controllers is based on robotics. Instead of programming a robotic system with fixed behavioral capabilities directed by a central executive, "decisions" are made in a non-representational manner based on information accumulated and instantiated in the robot's circuitry by trial and error. The behavior that ensues from this approach provides the appearance of central control when in fact there is none. Although the performance of such mobile robots is still in its infancy, the framework they provide applies to attention since the behaviors elicited from these design principles could be construed in attentional terms.

Thus a more radical possibility is that attention is generated automatically (reflexively) according to the full range of internal and external stimuli that affect the activity in the nervous system's circuitry at any given time. The responses would, like the robot, have been generated by trial and learning over evolutionary and individual time. As a result there would no executive system, no overarching control, and no selectivity in the usual sense of the word. In these terms, whatever is attended to is simply the result of preexisting neural circuitry shaped empirically by the mechanisms of natural selection and individual learning that favor responses that promoted successful behavior in the past. From this perspective, the term *attention* would simply be a useful colloquialism that expresses a subjective sense of what are, in fact, predetermined responses based on existing neural circuitry. The idea that the internal mental processes that we think of as voluntary outcomes determined be a neural "I" would be no different in kind from the involuntary shifts of attention elicited by a flash of light or a novel sound.

Summary

Attention research seeks to understand how processing resources are directed to deal effectively with ever-changing internal and external environments. Endogenous attention refers to the ability to voluntarily direct attention based on one's goals, expectations, or knowledge. Exogenous attention refers to involuntary shifts of attention triggered by salient stimuli in the environment. Both lead to enhanced processing of the information to which attention has been directed. Insight into both the psychological and the neural mechanisms of attention has been greatly advanced in recent years by combining older behavioral approaches with EEG and fMRI that can evaluate brain activity while humans or other animals are engaged in attentional tasks. The widely accepted idea based on these studies of a dedicated attentional system that monitors brain activity and makes decisions about the allocation of neural resources is nonetheless problematic. Many attention researchers recognize this problem and have sought to provide other ways of conceptualizing attention and interpreting the relevant experimental results, but so far without any general agreement among them.

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