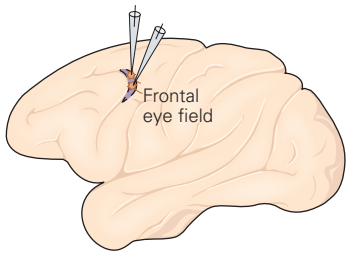
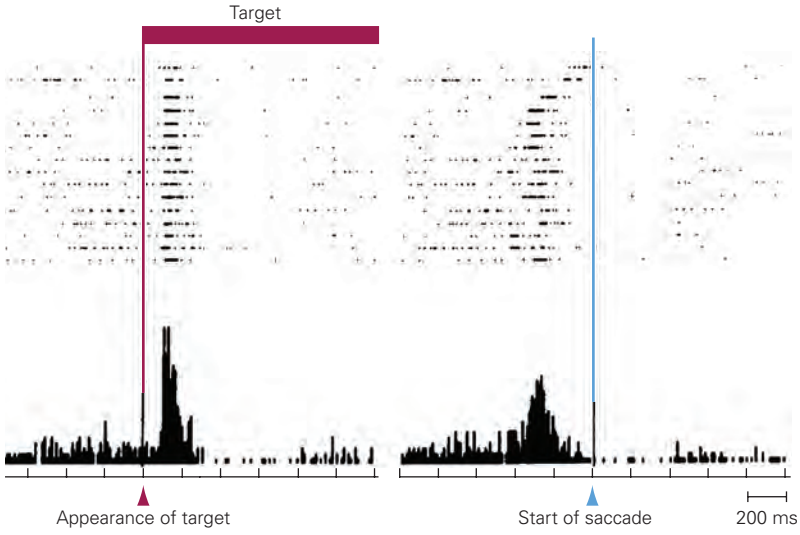


A Visual neuron responds to the stimulus and not to movement



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

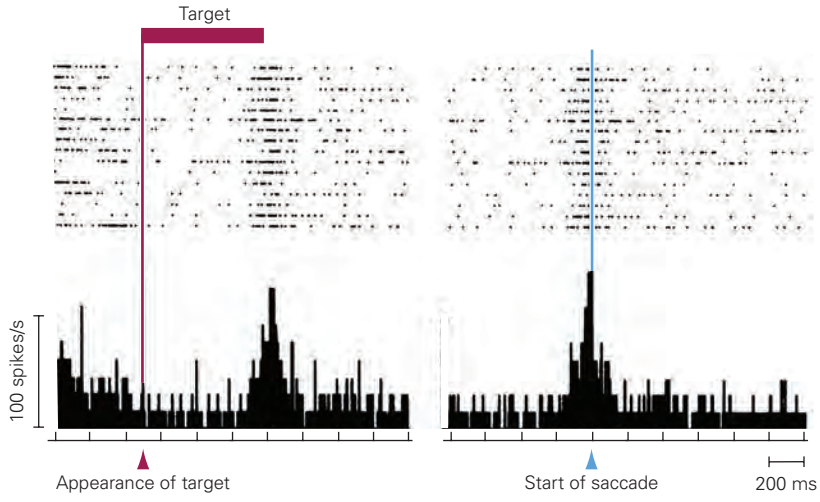


Figure 35–13 Visual and movement-related neurons in the frontal eye field. (Reproduced, with permission, from Bruce and Goldberg 1985.)

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

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

if the superior colliculus responds to a parietal signal that generates attention to the stimulus without the frontal-nigral control that normally prevents saccades in response to parietal signals.

The Control of Saccades Can Be Modified by Experience

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

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

Some Rapid Gaze Shifts Require Coordinated Head and Eye Movements

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

Because the head has a much greater inertia than the eyes, a small shift in gaze drives the fovea to its target before the head begins to move. A small gaze shift usually consists of a saccade followed by a small head movement during which the vestibulo-ocular reflex moves the eyes back to the center of the orbit in the new head position (Figure 35–14). For larger gaze shifts, the eyes and the head move simultaneously in the same direction. Because the vestibulo-ocular reflex ordinarily moves the eyes in the direction opposite that of the head, the reflex must be temporarily suppressed.

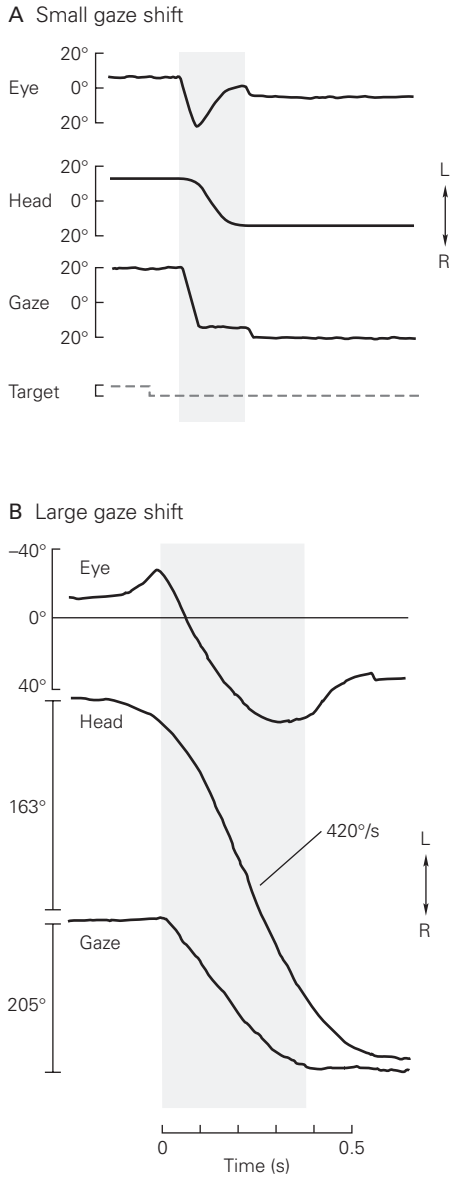


Figure 35–14 Directing the fovea to an object when the head is moving requires coordinated head and eye movements.

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

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

The Smooth-Pursuit System Keeps Moving Targets on the Fovea

The smooth-pursuit system holds the image of a moving target on the fovea by calculating how fast the target is moving and moving the eyes at the same speed. Smooth-pursuit movements have a maximum angular velocity of approximately 100° per second, much slower than saccades. Drugs, fatigue, alcohol, and even distraction degrade the quality of these movements.

Smooth pursuit and saccades have very different central control systems. This is best seen when a target jumps away from the center of gaze and then slowly moves back toward it. A smooth-pursuit movement is initiated first because the smooth-pursuit system has a shorter latency and responds to target motion on the peripheral retina as well as on the fovea. The task of the smooth-pursuit system differs from that of the saccade system. Instead of driving the eyes as rapidly as possible to a point in space, it must match the velocity of the eyes to that of a target in space. Therefore, as the target moves back toward the center of gaze, the smooth-pursuit system briefly moves the eye away from the target before the saccade is initiated (Figure 35–6B). The subsequent saccade then brings the eye to the target. Neurons that signal eye velocity for smooth pursuit are found in the medial vestibular nucleus and the nucleus prepositus hypoglossi. They receive projections from the flocculus of the cerebellum and project to the abducens nucleus as well as the oculomotor nuclei in the midbrain.

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

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

The middle temporal and medial superior temporal areas were named because of their position in sulcus-free cortex of the owl monkey, a New World monkey. In humans and Old World monkeys, these areas lie in the superior temporal sulcus, at the junction between the occipital and parietal lobes. Neurons in both the middle temporal and medial superior temporal areas calculate the velocity of the target. When the eye accelerates to match the target's speed, the rate of the target's motion across the retina decreases. As the speed of the retinal image decreases, neurons in the middle temporal area, whose activity signals retinal-image motion, stop firing, even though the target continues to move in space. Neurons in the medial superior temporal area continue to fire even if the target disappears briefly. These neurons have access to a process that adds the speeds of the moving eye and the target moving on the retina to compute the speed of the target in space.

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

The two motion-selective areas provide the sensory information to guide pursuit movements but may not be able to initiate them. Electrical stimulation of either area does not initiate smooth pursuit but can affect pursuit movement, accelerating ipsiversive pursuit and slowing contraversive pursuit. The frontal eye field may

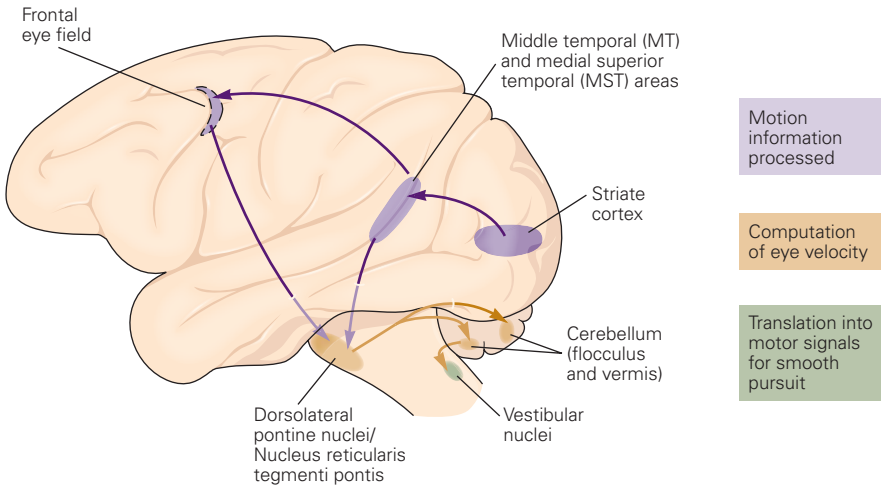


Figure 35–15 Cortical pathways for smooth-pursuit eye movements in the monkey. The cerebral cortex processes information about motion in the visual field and sends it to the oculomotor neurons via the dorsolateral pontine nuclei, the vermis and flocculus of the cerebellum, and the vestibular nuclei.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

be more important for initiating pursuit. This area has neurons that fire in association with ipsiversive smooth pursuit. Electrical stimulation of the frontal eye field initiates ipsiversive pursuit, whereas lesions of this area diminish but do not eliminate smooth pursuit.

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

Patients with parietal deficits that include the motion-sensitive areas have two different types of deficit. The first is a directional deficit that resembles that of monkeys with lesions of the medial superior temporal area: targets moving toward the side of the lesion cannot be tracked. The second is a retinotopic deficit that resembles the deficit of monkeys with lesions of the middle temporal area: There is an impairment of smooth pursuit of a stimulus limited to the visual hemifield opposite the lesion, regardless of the direction of motion.

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

The smooth-pursuit and saccade systems produce conjugate eye movements: Both eyes move in the same direction and at the same speed. In contrast, the vergence system produces disconjugate movements of the eyes. When we look at an object that is close to us, our eyes *converge* or rotate toward each other; when we look at an object that is farther away, they *diverge* or rotate away from each other (Figure 35–16). These disconjugate movements ensure that the image of the object falls on the foveae of both retinas. Whereas the visual system uses slight differences in left and right retinal positions, or *retinal disparity*, to create a

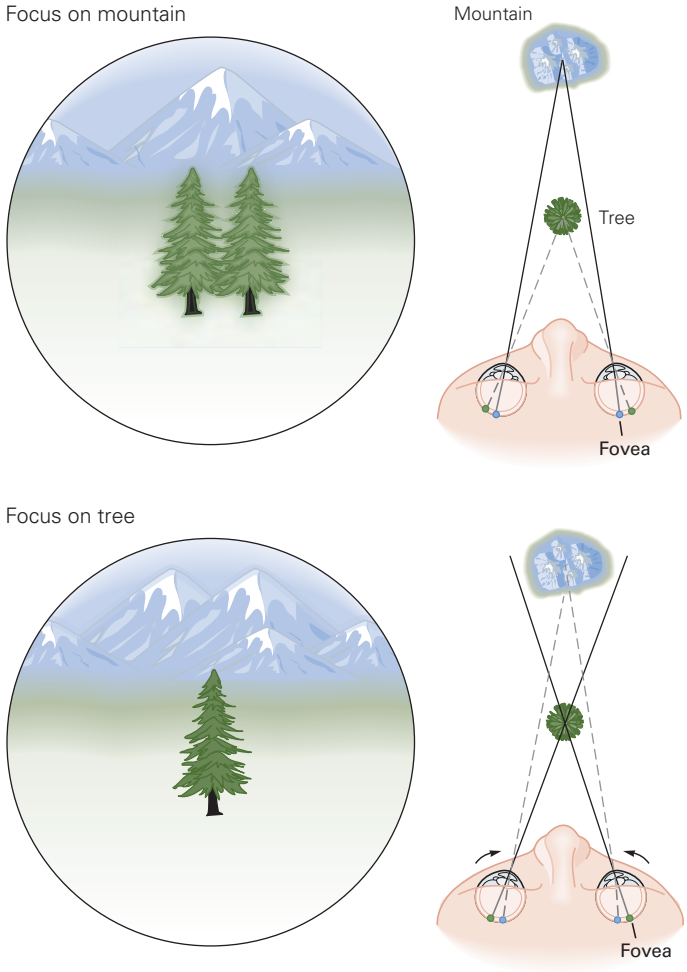


Figure 35–16 Vergence movements. When the eyes focus on a distant mountain, images of the mountain lie on the foveae, while those of the tree in the forefront occupy different retinal positions, yielding the percept of a double image. When the viewer looks instead at the tree, the vergence system must rotate each eye inward. Now the tree's image occupies similar positions on both foveae and is seen as one object, but the mountain's images occupy different locations on the retinas and appear double. (Reproduced, with permission, from F.A. Miles.)

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

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

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

Accommodation and vergence are linked. Accommodation is elicited by the blurring of an image, and whenever accommodation occurs, the eyes also converge. Conversely, retinal disparity induces vergence, and whenever the eyes converge, accommodation also takes place. At the same time, the pupils transiently constrict to increase the depth of field of the focus. The linked phenomena of accommodation, vergence, and pupillary constriction comprise the *near response.* Accommodation and vergence are controlled by midbrain neurons in the region of the oculomotor nucleus. Neurons in this region discharge during vergence, accommodation, or both.

Highlights

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

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

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36

Posture

The control of posture involves two interrelated goals, equilibrium (balance) and orientation, crucial for most tasks of daily living. Balance control maintains the body in stable equilibrium to avoid falls. Orientation aligns the body segments with respect to each other and to the world, such as maintaining the head vertical. Both balance and orientation use several different types of control: automatic postural responses, anticipatory postural adjustments, postural sway in stance, sensory integration for a body schema, orientation to vertical, and dynamic stability during gait.

To appreciate the complexity of maintaining balance and orientation, imagine that you are waiting tables on a tour boat. You have a tray full of drinks to be delivered to a table on the other side of the rolling deck. Even as your mind is occupied with remembering customer orders, unconscious but complex sensorimotor processes for controlling postural orientation and balance allow you to move about in an efficient and coordinated manner without falling. As you cross the rolling deck, your brain rapidly integrates and interprets sensory information and adjusts motor output to maintain your balance and the upright orientation of your head and trunk, as well as stabilize the arm supporting the tray of full glasses. Sudden

unexpected motions of the boat evoke automatic postural responses that prevent falls. Before you reach out to place a glass on the table, your nervous system makes anticipatory postural adjustments to maintain your balance.

Somatosensory, vestibular, and visual information are integrated to provide a coherent sense of the position and velocity of the body in space with respect to the support surface, gravity, and visual environment. Since the surface is unstable and vision is not providing earth-stable information, your dependence on vestibular information is greater than usual. Your head is kept stable while your trunk motions and walking pattern adjust for disequilibrium caused by the moving surface. You notice that both your voluntary tasks and your balance control deteriorate when trying to attend to both goals.

Equilibrium and Orientation Underlie Posture Control

Postural equilibrium refers to the ability to actively stabilize the upper body by resisting external forces acting on the body. Although the dominant external force affecting equilibrium on earth is gravity, other inertial forces and external perturbations must also be resisted. Depending on the particular task or behavior, different sets of muscles are activated in response to or in anticipation of disturbance to equilibrium.

Postural orientation refers to the ability to actively align body segments, such as the trunk and head, with respect to each other and to the environment. Depending on the particular task or behavior, body segments may be aligned with respect to gravitational vertical, visual vertical, or the support surface. For example, when skiing downhill, the head may be oriented to gravitational and inertial vertical, but not to the visual or support surface references that are inclined.

The biomechanical requirements of postural control depend on anatomy and postural orientation and thus vary among species. Nevertheless, in a variety of species, the control mechanisms for postural equilibrium and orientation have many common features. The sensorimotor mechanisms for postural control are quite similar in humans and quadrupedal mammals even though their habitual stance is different.

Postural Equilibrium Controls the Body's Center of Mass

With many segments linked by joints, the body is mechanically unstable. To maintain balance, the nervous system must control the position and motion of the body's *center of mass* as well as the body's rotation about it. The center of mass is a point that represents the average position of the body's total mass. In the standing adult, for example, the center of mass is located about 2 cm in front of the second lumbar vertebra; in a young child, it is higher. The location of the center of mass is not fixed but depends on postural orientation. For example, when you flex at the hips while standing, the center of mass moves from a location inside the body to a position outside the body.

Although gravity pulls on all body segments, the net effect on body equilibrium acts through the body's center of mass. The force due to gravity is opposed by the forces between the feet and the ground. Each point on the surface will generate a force on the foot. All the forces acting between the foot and the ground can be summed to yield a single force vector termed the *ground reaction force*. This origin of the ground reaction force vector on the surface is the point at which the rotational effect of all the forces on the feet are balanced and is termed the *center of pressure* (Box 36–1).

Maintaining balance while standing requires keeping the downward projection of the center of mass within the base of support, an imaginary area defined by those parts of the body in contact with the environment. For example, the two feet or one foot of a standing human define a *base of support* (Box 36–1). However, when a standing person leans against a wall or is supported by crutches, the base of support extends from the ground under the feet to the contact point between the body and the wall or crutches. Because the body is always in motion, even during stable stance, the body's center of mass continually moves about with respect to the base of support. Postural instability is determined by how fast the center of mass is accelerating toward and beyond the boundary of its base of support and how close the downward projection of the body's center of mass is to the boundary.

Upright stance requires two actions: (1) maintaining support against gravity by keeping the center of mass at some height and joints stable and (2) maintaining balance by controlling the trajectory of the center of mass in the horizontal plane. Balance and antigravity support are controlled separately by the nervous system and may be differentially affected in certain pathological conditions. For example, antigravity support can be excessive when spasticity is present after a stroke or insufficient in the hypotonia of cerebral palsy, although balance control may be preserved. Alternatively, in vestibular disorders, antigravity support can be normal, although balance control is disordered.