

orientations within the plane of each saccular macula, but the maculae are oriented vertically in nearly parasagittal planes. The saccules are therefore especially sensitive to vertical accelerations. Certain saccular hair cells also respond to accelerations in the horizontal plane, in particular those along the anterior-posterior axis.

Central Vestibular Nuclei Integrate Vestibular, Visual, Proprioceptive, and Motor Signals

The vestibular nerve projects ipsilaterally from the vestibular ganglion mainly to four vestibular nuclei (medial, lateral, superior, and descending) in the dorsal part of the pons and medulla, in the floor of the fourth ventricle. Many vestibular nerve fibers also bifurcate, sending a direct projection to the fastigial nucleus, the nodulus and uvula, and the reticular formation (Figure 27–8A). These nuclei integrate signals from the vestibular organs with signals from the spinal cord, cerebellum, and visual system.

The vestibular nuclei project, in turn, to many central targets, including the oculomotor nuclei, reticular and spinal centers concerned with gaze and postural movement, and the thalamus (Figure 27–9). Many vestibular nuclei neurons have reciprocal connections with the cerebellum, primarily in the flocculo-nodular lobe, that form important regulatory mechanisms for eye movements, head movements, and posture (Figures 27–8 and 27–9). The vestibular nuclei receive inputs from the premotor cortex, the accessory optic system (nucleus of the optic tract), the neural integrator nuclei (nucleus prepositus hypoglossi and interstitial nucleus of Cajal), and the reticular formation (Figure 27–8). Further projections from the vestibular nuclei reach the rostral and caudal lateral medulla nuclei that are involved in regulation of blood pressure, heart rate, respiration, and bone remodeling, as well as the parabrachial nucleus for homeostasis modulation. Finally, there are projections from the vestibular nuclei to the medial geniculate (auditory) nuclei, as well as the supragenual nucleus and dorsal tegmental nucleus, which contribute to spatial orientation (Figure 27–9).

The superior and medial vestibular nuclei receive fibers predominantly from the semicircular canals in the medial regions and some otolith input in the lateral regions (Figure 27–8). They send fibers predominantly to the cerebellum, reticular formation, thalamus, oculomotor centers, and spinal cord (Figure 27–9). Oculomotor center outputs include the three oculomotor nuclei (abducens, oculomotor, trochlear), as well as the neural integrators for converting head velocity into head position signals in the nucleus hypoglossi

(horizontal eye movements) and interstitial nucleus of Cajal (vertical eye movements). These nuclei are described in some detail later.

Another major output pathway concerned with gaze control arises from the medial vestibular nucleus (as well as lesser projections from the descending and lateral vestibular nuclei) and projects bilaterally to the cervical spinal cord through the medial vestibulospinal tract (Figure 27–9; see Chapter 35). There are two categories of medial vestibulospinal fibers. Vestibulospinal neurons project only to the spinal cord to control neck musculature. Vestibulo-ocular neurons project to both the spinal cord and the oculomotor nuclei and are involved in coordinated eye and head movements to maintain gaze stability.

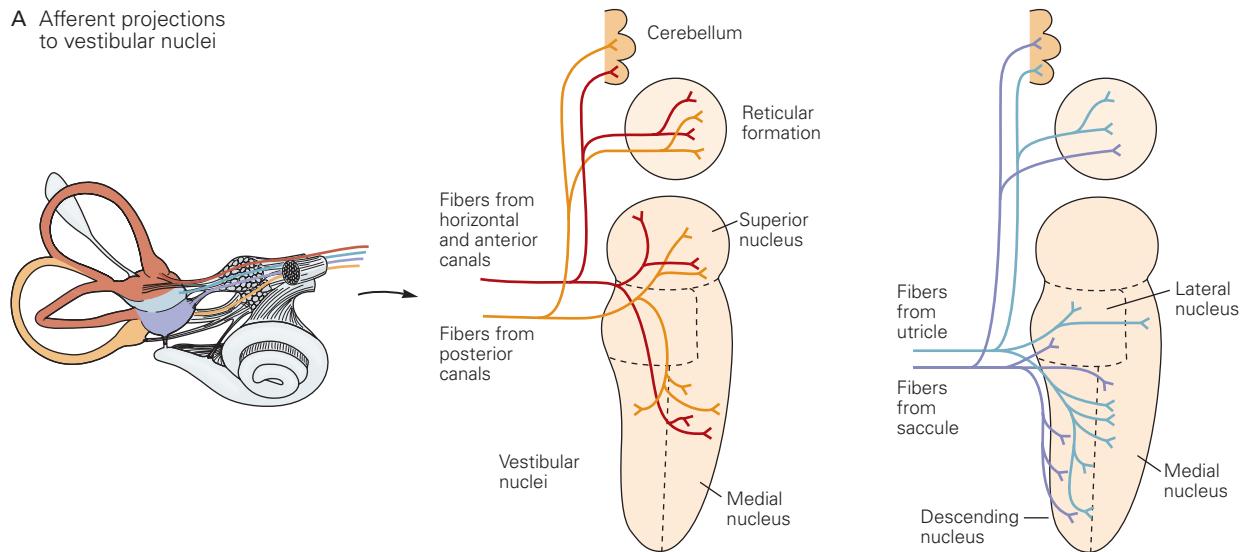
The lateral vestibular nucleus (Deiters' nucleus) receives fibers from the semicircular canals medially and the otolith organs laterally. There is a major output to all levels of the ipsilateral spinal cord through the lateral vestibulospinal tract that is concerned principally with postural reflexes through modulation of limb and axial musculature (Figure 27–9). Lateral vestibular nuclei neurons also project heavily to the reticular formation. The descending vestibular nucleus receives predominantly otolithic input, but also receives semicircular canal fibers medially, and projects to the cerebellum, reticular formation, and spinal cord (medial vestibulospinal tract). The primary neurotransmitters for excitatory vestibular nuclear projections include glutamate, whereas the inhibitory projections are either glycine or γ -aminobutyric acid (GABA). Vestibular projections to the spinal systems are discussed in more detail in Chapter 36.

The Vestibular Commissural System Communicates Bilateral Information

Many of these vestibular nuclei neurons receive convergent motion information from the opposite ear through an inhibitory commissural pathway that uses GABA as a neurotransmitter (Figure 27–8B). The commissural pathway is highly organized according to the type of receptor from which information is received. For example, cells receiving signals from the ipsilateral horizontal excitatory canal will also receive signals from the contralateral horizontal canal through an inhibitory interneuron. Due to the directional selectivity of the receptors in each ear, the contralateral horizontal canal input will always be decreased during an ipsilateral head turn, in effect "disinhibiting" the inhibitory input from the contralateral side.

The effect of the commissural system is to increase the response of the vestibular nuclei neuron and

A Afferent projections to vestibular nuclei



B Central projections to vestibular nuclei

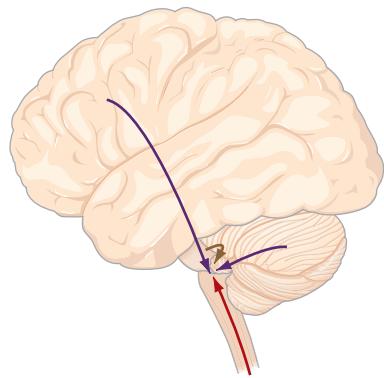
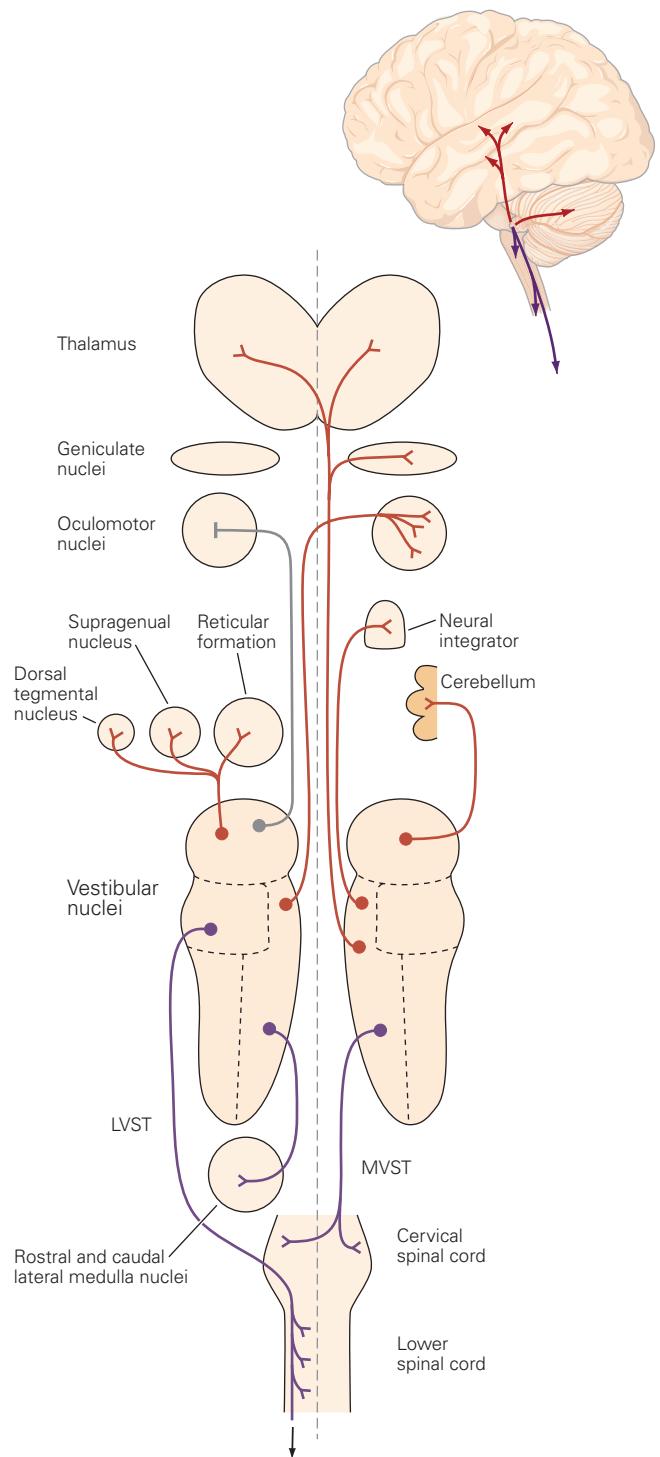


Figure 27–8 Afferent fiber and central projections to the vestibular nuclei.

A. Afferent fibers from vestibular receptors terminate in the brain stem and cerebellum. Fibers from semicircular canals project primarily to the medial portions of the superior and medial vestibular nuclei, the descending vestibular nucleus, the cerebellum (nodulus and uvula), and the reticular formation. Fibers from the otoliths primarily project to the lateral portions of

all vestibular nuclei, the nodulus and uvula, and the reticular formation. (Adapted, with permission, from Gacek and Lyon 1974.)

B. Central projections to the vestibular nuclei arise from a number of cortical, brain stem, and spinal cord regions. These include the premotor and multisensory cortices, accessory optic nuclei, cerebellum, neural integrator nuclei, reticular formation, spinal cord, and commissural fibers from the contralateral vestibular nuclei.



decrease noise from the incoming afferent signal, giving rise to a “push-pull” vestibular function. From an engineering point of view, the “push-pull” set point in the nuclei neurons constantly updates canal signals from the opposing ear to act as a comparator junction and can explain the relatively high spontaneous firing rate of canal afferents at nearly 100 spikes/s. For example, during a leftward head turn, left brain stem nuclei neurons receive high firing rate signals from the left horizontal canal and low firing rate signals from the right horizontal canal. The comparison of activity is interpreted as a left head turn (Figure 27–5). Similar comparisons between signals also occur for inputs from the anterior semicircular canal on one side and the posterior semicircular canal on the opposite ear side. Thus, for rotational motion in any head plane, the comparator is able to determine the direction of movement with great specificity.

Any disruption of the normal balance between left and right ear canal inputs (eg, from trauma or disease in the receptor organs or nerve) will be interpreted by the brain as a head rotation, even though the head is stationary. These effects often lead to illusions of spinning or rotating that can be quite upsetting and may produce nausea or vomiting. However, over time, the commissural fibers provide for vestibular compensation, a process by which the loss of unilateral vestibular receptor function is partially restored centrally and behavioral responses such as the vestibulo-ocular reflex mostly recover.

Combined Semicircular Canal and Otolith Signals Improve Inertial Sensing and Decrease Ambiguity of Translation Versus Tilt

In some instances, the vestibular input from a single receptor may be ambiguous. For example, Einstein (1908) showed that linear accelerations are equivalent whether they arise from translational motion or tilts of the head relative to gravity. The otolith receptors cannot

Figure 27–9 (Left) Output projections from the vestibular nuclei. The vestibular nuclei project to a number of brain regions below the cortical level. Two separate descending pathways project through the lateral and medial vestibulospinal tracts (LVST, MVST) to terminate in the spinal cord. The vestibular nuclei also project to the reticular formation and the lateral medullary nuclei in the brain stem. Ascending projections to the supragenual nucleus, the dorsal tegmental nucleus, the oculomotor nuclei (abducens, oculomotor, and trochlear), and the neural integrator nuclei are very prominent (red line, excitatory; gray line, inhibitory), as are projections to the cerebellum (nuclei, nodulus, and uvula). Other prominent vestibular projections terminate in the geniculate nuclei and the thalamus (ventral lateral, posterior, and intralaminar thalamic regions).

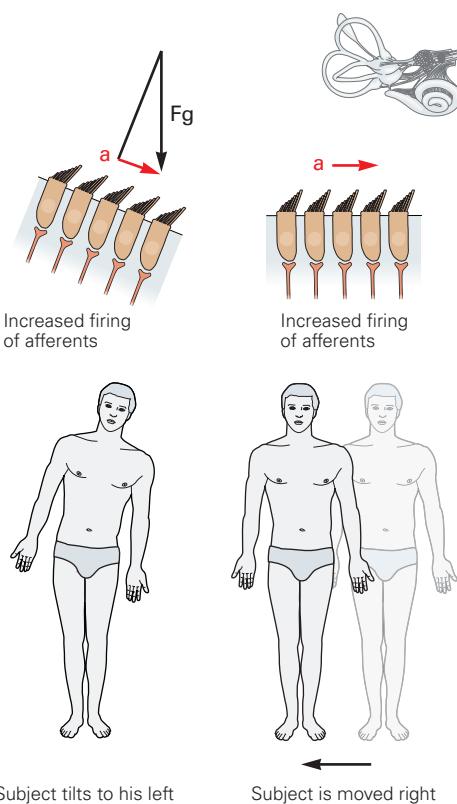


Figure 27–10 Vestibular inputs signaling body posture and motion can be ambiguous. The postural system cannot distinguish between tilt and linear acceleration of the body based on otolithic inputs alone. The same shearing force acting on vestibular hair cells can result from tilting of the head (left), which exposes the hair cells to a portion of the acceleration (a) owing to gravity (F_g), or from horizontal linear acceleration of the body (right).

discriminate between the two: So how is it that we can tell the difference between translating rightward and tilting leftward, where the linear acceleration signaled by the otolith afferents is the same (Figure 27–10)?

It is now well established that convergent vestibular nuclei and cerebellar neurons use combined signals from both the semicircular canals and the otolith receptors and some simple computations to discriminate between tilt and translation. As a result, some central vestibular and cerebellar cells encode head tilt, whereas other cells encode translational motion, which, as we will see, is extremely important for the control of head and eye movements.

Vestibular Signals Are a Critical Component of Head Movement Control

An important discovery is the differing responses in some vestibular nuclei neurons to actively versus

passively generated head movements. Specifically, in contrast to vestibular afferents, some neurons in the vestibular nuclei and cerebellum well known for responding to vestibular stimuli during passive movement lose or reduce their sensitivity during self-generated movement. The preferential response to passive motion, or to the passive components of combined active and passive motion, has been interpreted as sensory prediction error signals: The brain predicts how self-generated motion activates the vestibular organs and subtracts these predictions from afferent signals. Such error signals are important for the on-line control of head movement, as well as head movement estimation.

Computationally, these properties have been interpreted quantitatively using concepts common to all sensorimotor systems; that is, active and passive motion signals are processed by internal models of the motion sensor (ie, the canals, otolith organs, and neck proprioceptors). The brain uses an internal representation of the laws of physics and sensory dynamics (which can be elegantly modeled as forward internal models of the sensors) to process vestibular signals. Without such error signals, accurate self-motion estimation would be severely compromised. These computational insights suggest that, unlike early interpretations, vestibular signals remain critically important when coupled to self-motion estimation and head movement control during actively generated head movements.

Vestibulo-Ocular Reflexes Stabilize the Eyes When the Head Moves

In order to see clearly and maintain focus on visual objects during head motion, the eyes maintain foveal fixation through a series of vestibulo-ocular reflexes (VORs). If you shake your head back and forth while reading, you can still discern words because of the VORs. If instead you move the book at a similar speed while holding your head steady, you can no longer read the words.

In the latter instance, vision provides the brain with the only corrective feedback for stabilizing of the image on the retina, and visual processing in vertebrates is much slower (around 100 ms latency) and less effective than vestibular processing (around 10 ms) for image stabilization. The vestibular apparatus signals how fast the head is rotating, and the oculomotor system uses this information to stabilize the eyes to fix visual images on the retina.

There are two components of VORs. The *rotational VOR* compensates for head rotation and receives its input predominantly from the semicircular canals. The *translational VOR* compensates for linear head movement. These two VOR responses arise from connections from vestibular nuclei neurons to the abducens, oculomotor, and trochlear nuclei (Figure 27–9).

The Rotational Vestibulo-Ocular Reflex Compensates for Head Rotation

When the semicircular canals sense head rotation in one direction, the eyes rotate in the opposite direction at equal velocity in the orbits (Figure 27–11). This compensatory eye rotation is called the vestibular slow phase, although it is not necessarily slow: The eyes may reach speeds of more than 200 degrees per second if the head's rotation is fast. During fast head movements, the VOR must act quickly to maintain stable gaze. A trisynaptic pathway, the three-neuron arc, connects each semicircular canal to the appropriate eye muscle (Figure 27–11).

The rotational VOR represents a phylogenetically old reflex. Many invertebrates and all vertebrate species, from amphibians, reptiles, fish, and birds to non-human primates, have the ability to reflexively rotate their eyes opposite to the direction of head rotation, thus keeping the visual world stable on the retina. Primary afferents from the horizontal semicircular canals send excitatory signals through the vestibular nuclei and the medial longitudinal fasciculus to the contralateral abducens nucleus (Figure 27–11). Abducens motor neurons send impulses via cranial nerve VI to excite the ipsilateral lateral rectus muscle. At the same time, abducens interneurons send excitatory signals to motor neurons in the contralateral oculomotor nucleus, which innervates the medial rectus muscle (see Chapter 35 for details on other projections).

The three-synapse pathway illustrated in Figure 27–11 is not sufficient to elicit appropriate compensatory eye movements. This is because the afferent signal from the semicircular canals is proportional to head velocity, while the compensatory eye movement requires eye position changes. To convert velocity to position requires temporal integration (simple calculus) that occurs through neural networks in the brain stem nuclei for most head motion speeds. However, at high rotation frequencies, the viscoelastic properties of the eyeball, eye muscles, and surrounding tissues provide an additional integration step. Thus, the rotational VOR is thought to consist of two parallel processes.

The first process consists of the direct neural pathway known as the three-neuron arc (Figure 27–11).

The second neural integrator process consists of additional parallel pathways that ensure that the correct proportion of velocity and position commands are delivered to the oculomotor nuclei to move the eye appropriately (Figure 27–9 and see Chapter 35). Without this second indirect integrator pathway, the response to a head rotation would initially bring the eye to the correct position, but the eye would drift away from that position since the oculomotor neurons would lack the tonic input to compensate for the elastic restoring forces of the eyeball (Chapter 35). This is exactly what happens after lesions of brainstem and cerebellar structures that are thought to participate in this neural integration (eg, the prepositus hypoglossi and the interstitial nucleus of Cajal; Figure 27–9). It is generally thought that the integrator pathway is shared by all conjugate eye movement systems (saccades, smooth pursuit, and the VOR), although the direct pathway is at least partly segregated for different types of eye movements (ie, VOR, smooth pursuit, saccades).

With continued head rotation, the eyes eventually reach the limit of their orbital range and stop moving. To prevent this, a rapid saccade-like movement called a quick phase displaces the eyes to a new point of fixation in the direction of head rotation.

If rotation is prolonged, the eyes execute alternating slow and quick phases called *nystagmus* (Figure 27–12). Although the slow phase is the primary response of the rotational VOR, the direction of nystagmus is defined in clinical practice by the direction of its quick phase. Since prolonged rightward rotation excites the right horizontal canal and inhibits the left horizontal canal, leftward slow phases and a *right-beating nystagmus* result.

If the angular velocity of the head remains constant, the inertia of the endolymph is eventually overcome, as in the earlier coffee cup example. The cupula relaxes and vestibular nerve discharge returns to its baseline rate. As a consequence, slow-phase velocity decays and the nystagmus stops, although the head is still rotating.

In fact, the nystagmus lasts longer than would be expected based on cupular deflection. By a process called *velocity storage*, a brain stem network provides a velocity signal to the oculomotor system, although the vestibular nerve no longer signals head movement. Eventually, however, the nystagmus does decay and the sense of motion vanishes in darkness. Further, the same rotation in the presence of a visual surround activates the optokinetic reflex (Chapter 35) and elicits a steady-state nystagmus pattern that is sustained indefinitely. The interactions between canal and optokinetic

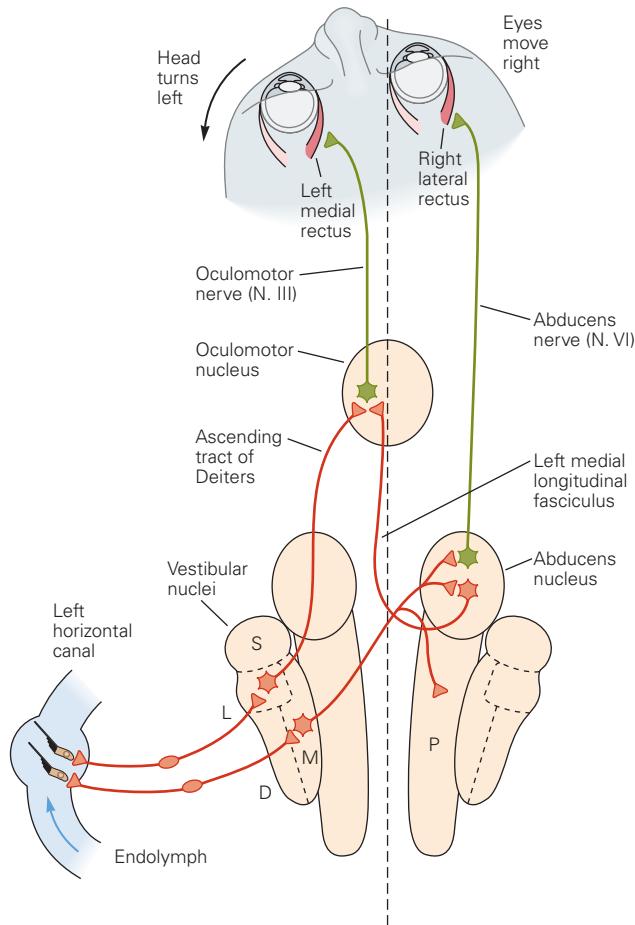
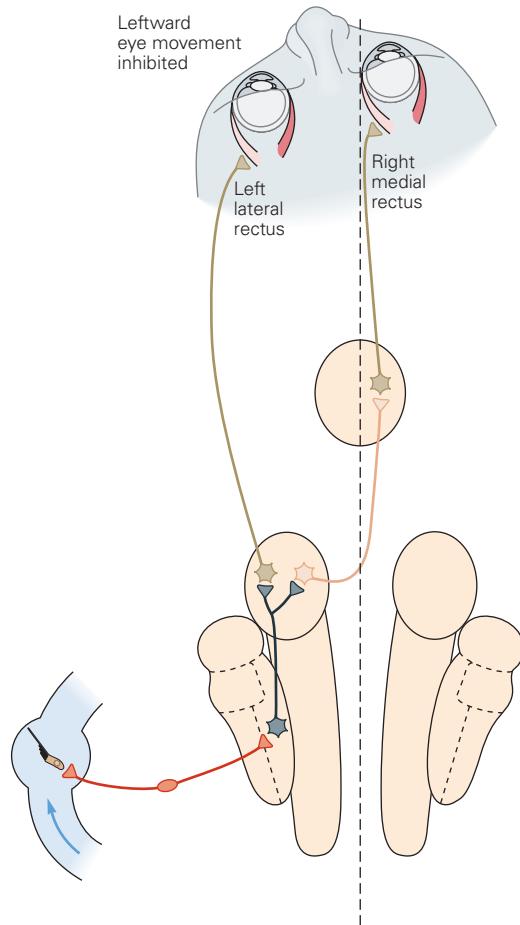
A Excitatory connections**B Inhibitory connections**

Figure 27–11 The horizontal vestibulo-ocular reflex. Similar pathways connect the anterior and posterior canals to the vertical recti and oblique muscles.

A. Leftward head rotation excites hair cells in the left horizontal canal, thus exciting neurons that evoke rightward eye movement. The vestibular nuclei include two populations of first-order neurons. One lies in the medial vestibular nucleus (M); its axons cross the midline and excite neurons in the right abducens nucleus and nucleus prepositus hypoglossi (P). The other population is in the lateral vestibular nucleus (L); its axons ascend ipsilaterally in the tract of Deiters and excite neurons in the left oculomotor nucleus, which project in the oculomotor nerve to the left medial rectus muscle.

The right abducens nucleus has two populations of neurons. A set of motor neurons projects in the abducens nerve and excites the right lateral rectus muscle. The axons of a set of interneurons cross the midline and ascend in the left medial longitudinal fasciculus to the oculomotor nucleus, where they

excite the neurons that project to the left medial rectus muscle. These connections facilitate the rightward horizontal eye movement that compensates for leftward head movement. Other nuclei shown are the superior (S) and descending (D) vestibular nuclei.

B. During counterclockwise head movement, leftward eye movement is inhibited by sensory fibers from the left horizontal canal. These afferent fibers excite neurons in the medial vestibular nucleus that inhibit motor neurons and interneurons in the left abducens nucleus. This action reduces the excitation of the motor neurons for the left lateral and right medial rectus muscles. The same head movement results in a decreased signal in the right horizontal canal (not shown), which has similar connections. The weakened signal results in decreased inhibition of the right lateral and left medial rectus muscles and decreased excitation of the left lateral and right medial rectus muscles. (Adapted from Sugiuchi et al. 2005.)

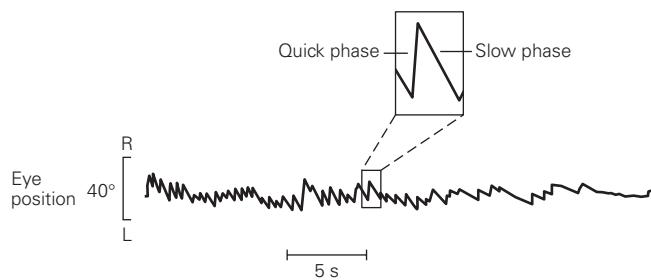


Figure 27–12 Vestibular nystagmus. The trace shows the eye position of a subject in a chair rotated counterclockwise at a constant rate in the dark. At the beginning of the trace, the eye moves slowly at the same speed as the chair (slow phase) and occasionally makes rapid resetting movements (quick phase). The speed of the slow phase gradually decreases until the eye no longer moves regularly. (Reproduced, with permission, from Leigh and Zee 2015.)

signals during rotation occurs through the velocity storage network.

If head rotation stops abruptly, the endolymph continues to be displaced in the same direction that the head had formerly rotated. With rightward rotation, this inhibits the right horizontal canal and excites the left horizontal canal, resulting in a sensation of leftward rotation and a corresponding left-beating nystagmus. However, this occurs only in darkness. In the light, optokinetic reflexes suppress postrotatory nystagmus since there is no visual motion stimulus.

The Translational Vestibulo-Ocular Reflex Compensates for Linear Motion and Head Tilts

When the head rotates, all images move with the same velocity on the retina. When the head moves sideways, however, the image of a close object moves more rapidly across the retina than does the image of a distant object. This can be understood easily by considering what happens when a person looks out the side window of a moving car. Objects near the side of the road move out of view almost with the speed of the car, whereas distant objects disappear more slowly. To compensate for linear head movement, the vestibular system must take into account the distance to the object being viewed—the more distant the object, the smaller the needed eye movement. During linear movements that do not involve head rotation, an appropriate translational VOR is elicited, driven by input from the otolith organs. Neurons in the vestibular nuclei, including some different from those providing the main drive to the rotational VOR, carry this signal to the extraocular motor neuron pools.

Side-to-side head movements result in a horizontal eye movement in a direction opposite to the head movement. Vertical displacements of the body, such as during walking or running, elicit oppositely directed vertical eye movements to stabilize gaze. However, in contrast to the rotational VOR where a head rotation is compensated by an equal but opposite eye rotation, horizontal displacement must be compensated by an eye rotation that depends on the viewed object distance, a nontrivial computation. For example, during a lateral head displacement, nearby objects move on the retina more rapidly than distant ones. So, in order to stabilize a nearby object on the retina, the eyes need to rotate by a larger amount than is needed for a distant object. Thus, the horizontal compensatory eye movements that are elicited during lateral motion scale with target distance; the closer the target, the larger is the compensatory eye movement. Similarly, as in the rotational VOR, compensatory responses to translation occur at relatively short latency (10–12 ms).

Fore-aft translations produce converging and diverging eye movements that bring the eyes together or move them apart. The amount of convergence or divergence is also dependent upon visual target distance, such that close visual objects produce large eye movements and distant visual objects produce little eye movements. Further, the amount of relative left and right eye movement is dependent upon visual object eccentricity relative to straight ahead. Unlike the rotational VOR that is a full-field image stabilization reflex, the goal of the translational VOR is to selectively stabilize visual objects on the fovea. In general, the two eyes move disjunctively, consisting of either a pure vergence movement or a combination of vergence and conjugate eye movements. In practice, although the direction of the evoked eye movement is typically consistent with geometrical predictions, the primate/human translational VOR typically undercompensates for near-target viewing, with gains of only about 0.5.

The translational VOR differs from the rotational VOR in the ability to generate compensatory eye movements during translation that optimize visual acuity on the central retina. These abilities appear to be specific to frontal-eyed animals, such as primates. Many lateral-eyed species, like the rabbit, do not generate eye movements that compensate for the visual consequences of translation during self-motion.

Because gravity exerts a constant linear acceleration force on the head, the otolith organs also sense the orientation of the head relative to gravity. When the head tilts away from the vertical in the roll plane—around the axis running from the occiput to the nose—the eyes rotate in the opposite direction to reduce the

tilt of the retinal image. This ocular counter-rolling reflex—the ability to use a gravity-sensing mechanism to maintain gaze relative to the horizon—is of paramount importance for lateral-eyed, afoveate species that typically lack a well-developed saccadic system. But such functional utility for these tilt responses has lost its advantage in the primate oculomotor system, where static ocular counter-rolling and counter-pitching in humans have a gain of less than 0.1.

Vestibulo-Ocular Reflexes Are Supplemented by Optokinetic Responses

The VORs compensate for head movement imperfectly. They are best at sensing the onset or abrupt change of motion; they compensate poorly for sustained motion at constant speed during translation or constant angular velocity during rotation. In addition, they are insensitive to very slow rotations or low-amplitude linear accelerations.

Thus, vestibular responses during prolonged motion in the light are supplemented by visual stabilization reflexes that maintain nystagmus when vestibular input ceases: optokinetic nystagmus, a full-field stabilization system, and ocular following, a foveal stabilization system. Although the two classes of reflexes are distinct, their pathways overlap.

The Cerebellum Adjusts the Vestibulo-Ocular Reflex

As we have seen, the VOR keeps the gaze constant when the head moves. There are times, however, when the reflex is inappropriate. For example, when you turn your head while walking, you want your gaze to follow. The rotational VOR, however, would prevent your eyes from turning with your head. To prevent this sort of biologically inappropriate response, the VOR is under the control of the cerebellum and cortex, which suppress the reflex during volitional head movements.

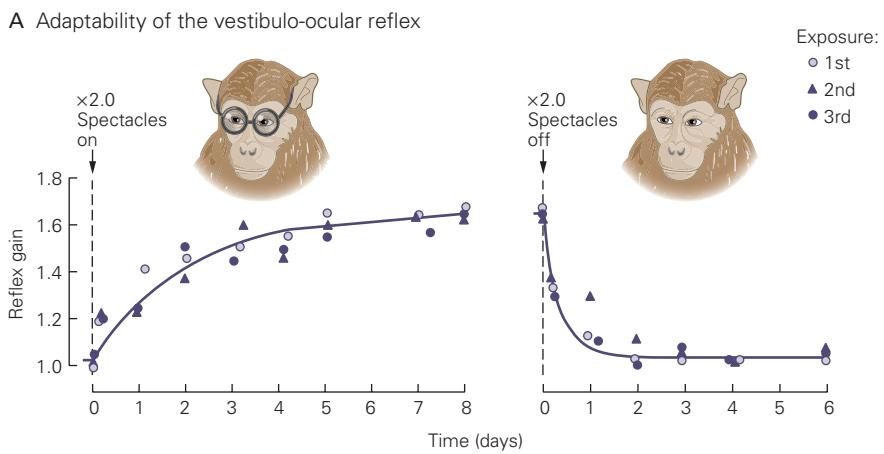
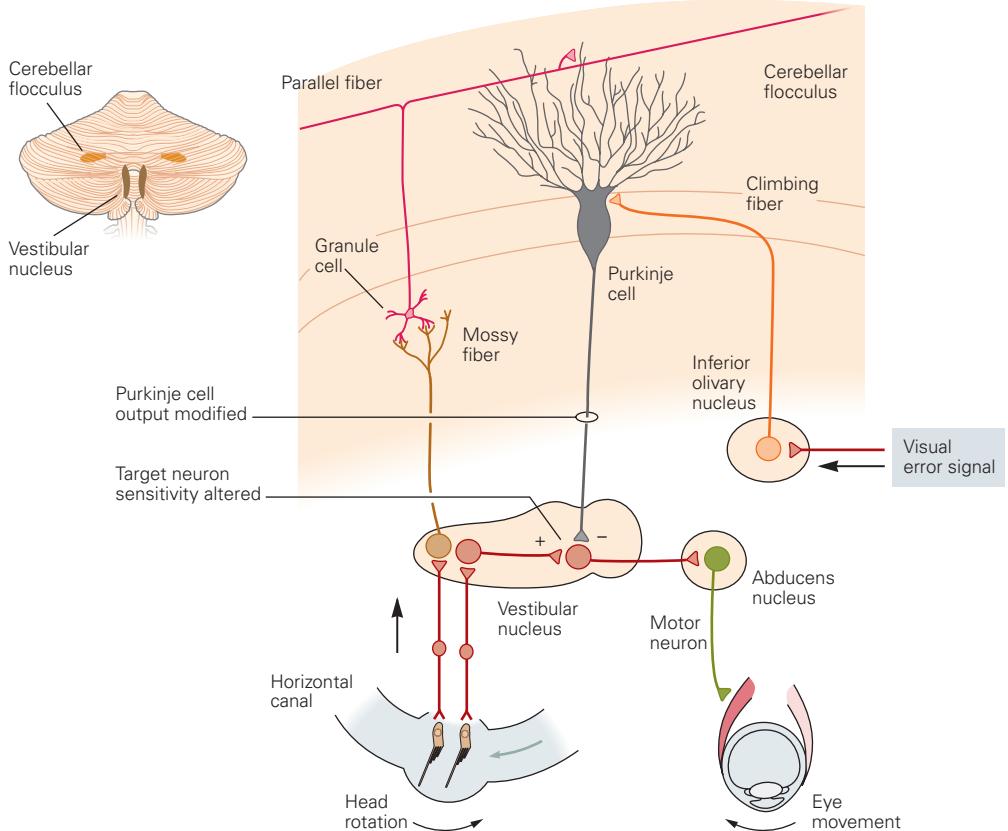
In addition, the VOR must be continuously calibrated to maintain its accuracy in the face of changes within the motor system (fatigue, injury to vestibular organs or pathways, eye-muscle weakness, or aging) and differing visual requirements (wearing corrective lenses). Indeed, the VOR is a highly modifiable reflex. The brain continuously monitors its performance by evaluating the clarity of vision during head movements. When head turns are consistently associated with image motion across the retina, the VOR undergoes gain changes in the direction appropriate to improve the compensatory ability of the reflex. For example, when viewing the world through spectacles

that magnify or miniaturize the visual scene, the rotational VOR gain (in darkness) increases or decreases accordingly. The reflex behavior can adapt over several minutes, hours, and days. This is accomplished by sensory feedback that modifies the motor output. If the reflex is not working properly, the image moves across the retina. The motor command to the eye muscles must be adjusted until the gaze is again stable, rotational retinal image motion is zero, and there is no error.

Anyone who wears eyeglasses depends on this plasticity of the VOR. Because lenses for nearsightedness shrink the visual image, a smaller eye rotation is needed to compensate for a given head rotation, and the gain of the VOR must be reduced. Conversely, glasses for farsightedness magnify the image, so the VOR gain must increase during their use. More complicated is the instance of bifocal or progressive spectacles, in which the reflex must use different gains for the different magnifications. In the laboratory, the reflex can be conditioned by altering the visual consequences of head motion. For example, if a subject is rotated for a period of time while wearing magnifying glasses, the reflex gain gradually increases (Figure 27–13A).

This process requires changes in synaptic transmission in both the cerebellum and the brain stem. If the flocculus and paraflocculus of the cerebellum are lesioned, the gain of the VOR can no longer be modulated. Mossy fibers carry vestibular, visual, and motor signals from the pontine and vestibular nuclei to the cerebellar cortex; the granule cells, with their parallel fiber axons, relay these signals to the Purkinje cells (Figure 27–13B). The synaptic efficacy of parallel fiber input to a Purkinje cell could be modified by the concurrent action of climbing fiber input. Indeed, the climbing fiber input to the cerebellum carries a retinal error signal, thought to serve as a “teaching” signal enabling the cerebellum to correct the error in the VOR. This adaptation requires long-term plasticity of multiple mechanisms through multiple sites (Chapter 37).

In addition to the Purkinje cell, plasticity is also found in the vestibular nuclei, in a particular class of neurons known as flocculus target neurons, which receive GABAergic inhibitory input from Purkinje cells in the flocculus as well as direct inputs from vestibular sensory fibers. During adaptation of the VOR, these neurons change their sensitivity to the vestibular inputs in the appropriate way, and after adaptation, they can maintain those changes without further input from the cerebellum. The importance of the cerebellum in calibrating eye movements is also evident in patients with cerebellar disease, who are often characterized by a VOR response of abnormal amplitude or direction.

**B Sites of adaptive learning****Figure 27–13** The vestibulo-ocular reflex is adaptable.

A. For several days, the monkey continuously wears magnifying spectacles that double the speed of the retinal-image motion evoked by head movement. Each day, the gain of the vestibulo-ocular reflex—the amount the eyes move for a given head movement—is tested in the dark so that the monkey cannot use retinal motion as a clue to modify the reflex. Over a period of 4 days, the gain increases gradually (*left*). It quickly returns to normal when the spectacles are removed (*right*). (Adapted, with permission, from Miles and Eighmy 1980.)

B. Adaptation of the vestibulo-ocular reflex occurs in cerebellar and brain stem circuits. A visual error signal, triggered by motion of the retinal image during head movement, reaches the inferior olivary nucleus. The climbing fiber transmits this error signal to the Purkinje cell, affecting the parallel fiber-Purkinje cell synapse. The Purkinje cell transmits changed information to the floccular target cell in the vestibular nucleus, changing its sensitivity to the vestibular input. After the reflex has been adapted, the Purkinje cell input is no longer necessary.