CHAPTER

36

Eye Movements

As we learned in Chapter 28, the photoreceptor mosaic of the vertebrate retina transduces light energy in the form of photons into neural activity, ultimately in the form of action potentials. The spatial resolution of this transduction system is limited by the resolution of the photoreceptor mosaic, but only if the eye can be kept stationary with regard to the objects in the external world that are the subjects of visual analysis. Thus, stabilizing the retina with regard to the outside world and aligning the retina with moving or stationary targets is a critical challenge to effective vision. Evolutionary pressures have shaped the eye movement systems of all animals to meet this challenge in ways that are tailored to the visual structures and environmental needs of each species. In this chapter we will examine the neural and behavioral systems vertebrates use to achieve effective retinal stabilization. These systems employ two principal classes of mechanisms, one for *gaze stabilization* and one for *gaze shifting.* The former is found in all animals with visual systems, but the latter is found only in animals with retinal specializations, such as the primate fovea, that can be used to examine a limited region of visual space with higher acuity.

GAZE-STABILIZATION MECHANISMS

A completely stationary animal, whose photoreceptors were anchored to the earth, would always be able to resolve stationary stimuli at the resolution limits of its retina. When an animal moves, however, it risks degrading its visual acuity. Rotating the line of sight by moving the head, for example, will cause a point of light fixed in the environment to streak across the retina, appearing as a line or curve to the visual system. Gaze-stabilization mechanisms are movement systems that counteract this effect of self-motion on visual acuity. These mechanisms coordinate movements of the eye that precisely compensate for self-motion, thus stabilizing the visual world on the retina.

Gaze-stabilization mechanisms fall into two subclasses: the **vestibuloocula** system and the **optokinetic** system. Vestibuloocular system relies on the semicircular canals to determine the precise rate at which the head is rotated in any direction. The optokinetic system relies on information from the photoreceptors themselves to compute the speed and direction at which the visual world is shifting across the retina. As we will learn, the vestibuloocular system is most efficient at higher speeds of rotation, where the vestibular apparatus most accurately measures head velocity. In contrast, the optokinetic system operates most efficiently at low speeds of rotation, where the photoreceptors can be used to accurately determine the speed and direction of image motion. Both systems compensate for rotation by activating the **extraocular muscles** to produce a perfectly matched counterrotation of the eyes. The result is that the line of sight remains constant with respect to the environment, despite movements of the head. Thus, these sensorimotor systems use different types of sensory data to activate a common muscular system, stabilizing retinal images during self-motion over a wide range of speeds.

GAZE-SHIFTING MECHANISMS

Versional Movements Shift the Line of Gaze with Respect to the Visual World

While essentially all vertebrates have gaze-stabilization systems, several groups of vertebrates have

evolved specialized retinas that can be effectively employed only if the direction of gaze can be shifted. Primates, for example, have a highly specialized central region of the retina known as the fovea. The fovea can gather visual information from only 1° of the visual world, but the photoreceptors in the fovea are packed at high density, permitting high resolution. High resolution would be useless, however, unless the fovea could be specifically directed to areas of interest in the visual world and stabilized with respect to those stimuli. To accomplish this, essentially all vertebrates with retinal subregions specialized for higher acuity have evolved gaze-shifting systems that employ the extraocular muscles and whose neural components are probably evolutionarily derived from gaze-stabilization mechanisms. Most gaze-shifting mechanisms can be divided into two main groups: the **saccadic system,** which rapidly shifts gaze from one point to another, and the **smooth pursuit system,** which allows the fovea to track a moving target as it slides across a stationary background. The saccadic and smooth pursuit systems together are often referred to as **versional systems.** It should come as no surprise that the smooth pursuit system is believed to have evolved from the optokinetic system, both systems move the eyes to limit the velocity with which visual stimuli move across the retina. In a similar manner, the saccadic system appears to have evolved from a behavioral mechanism shared by the optokinetic and vestibuloocular systems, which will be discussed later.

Vergence Movements Shift the Lines-of-Gaze of the Two Eyes with Respect to Each Other

A third class of gaze-shifting eye movements have evolved in an even smaller subset of vertebrates, those with both fovealike retinal subregions and binocular vision. These animals have the ability to scrutinize a single visual target with both eyes and have evolved a special eye movement system to control the angle formed by the lines of gaze of the two eyes. When the eyes focus on an infinitely distant target, the lines of gaze projecting from the two foveas are parallel. As the target moves closer, however, the lines of gaze must converge. Thus, animals with binocular vision, must have an eye movement system that can keep both eyes aligned with visual targets as those targets vary in distance from the eyes. Binocular convergence is accomplished by adding a gaze control signal, which is different for each eye, to the shared saccadic or pursuit signal. This mechanism of producing binocular convergence is known as **Hering's law of equal innervation.**

Summary

All eye movements belong to one or more of these five classes: the vestibuloocular, optokinetic, saccadic, smooth pursuit, and vergence systems. These classes account for all types of eye movements in vertebrates. While each of these systems is a largely distinct neural entity, they all engage a common set of motor neurons and thus a common set of muscles. This shared motor circuitry imposes some interesting commonalities on the systems. For this reason, we will first discuss the common muscular system before we examine the five eye movement systems.

THE OCULOMOTOR NUCLEI AND THE EXTRAOCULAR MUSCLES

The oculomotor system, in contrast to the somotomotor system, is said to be relatively simple. The motions of the single ball-in-socket eye joint do not face the complexities of coordinating somatic multiple joints. There are only six muscles that move each eye. The muscles are controlled by three cranial nerves, the actions of which are rather simply coordinated by the medial longitudinal fasciculus. Control mechanisms within the brainstem compute and mix together the dynamic and static signals to move and hold the eye.

Six Muscles Move Each Eye

In primates, all eye movements are produced by the contraction or relaxation of six extraocular muscles. These muscles surround each eye and can rotate the eye in any direction. As Fig. 36.1 illustrates, the muscles are arranged in three antagonistic pairs, much like the pairing that occurs in the skeletomuscular system. The **medial** and **lateral rectus** muscles form an antagonistic pair that controls the horizontal position of each eye. Contraction of the lateral rectus, coupled with commensurate relaxation of the medial rectus, causes the eye to rotate outward, shifting the direction of gaze laterally (Fig. 36.2). Because the two eyes move together, contraction of the medial rectus of one eye is accompanied by contraction of the lateral rectus of the other eye, thus similarly rotating both eyes. The **superior** and **inferior recti** control the up-and-down rotation of the eye, while the **superior** and **inferior oblique** muscles control torsion, the rotation of the eye about the line of sight. The obliques also make a small contribution to pulling the eye up or down. Because the superior and inferior recti also generate some torsion, the obliques are particularly important for guar-

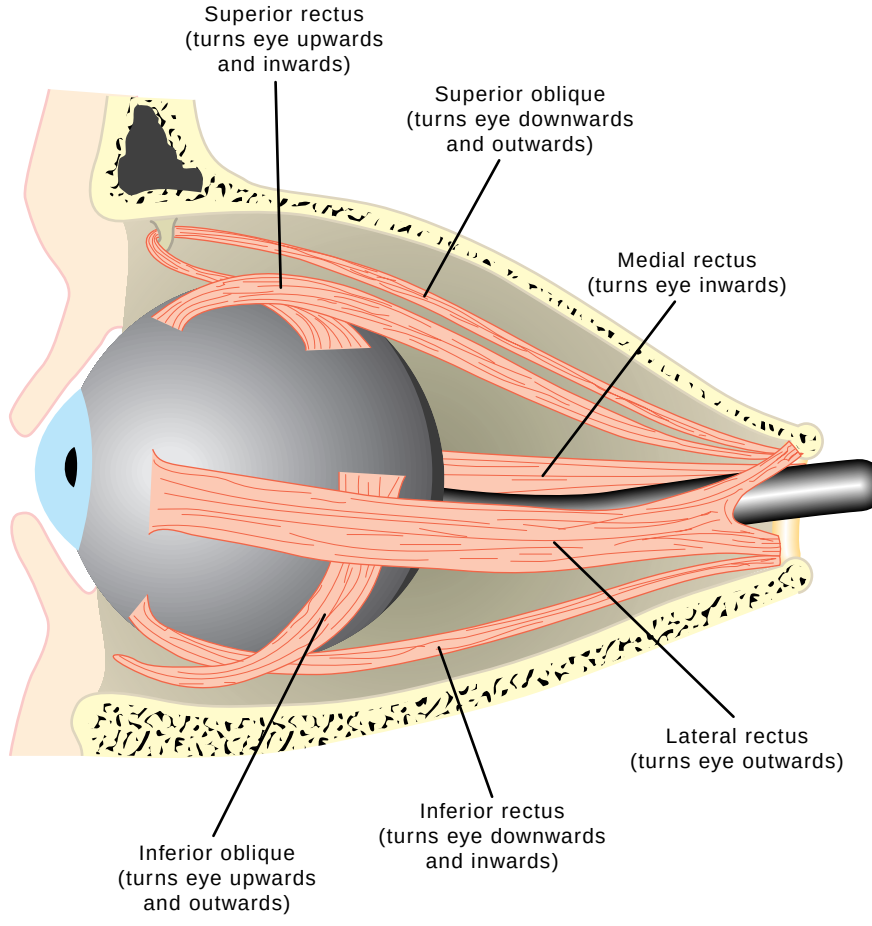


FIGURE 36.1 Muscles of the eye. Eye movements are controlled by six extraocular muscles arranged in three pairs, shown here in a cutaway view of the eye in its socket, or orbit.

anteeing that the eye maintains the same horizontal orientation as it moves around the orbit.

Three Cranial Nerves Control the Extraocular Muscles

The six extraocular muscles are innervated by three of the bilaterally paired cranial nerves discussed in Chapter 2. The oculomotor nerve (cranial nerve III) innervates the medial, superior, and inferior recti and the inferior oblique; the trochlear nerve (IV) innervates the superior oblique; and the abducens nerve (VI) innervates the lateral rectus. Thus, the somata of the oculomotor motor neurons are located in the third, fourth, and sixth cranial nuclei. These three nuclei are heavily interconnected by a pathway called the **medial longitudinal fasciculus.** This interconnection facilitates the coordination of extraocular muscle activity that is necessary for precise control of eye movements.

Eye Movements Are Produced by a Combination of Static and Dynamic Forces

To understand how muscle forces control the position and movement of the eyes, we must first understand how the tissues and muscles of the orbit produce resistance to movement. In its simplest form, the eye in the orbit can be thought of as a sphere held in place by a system of springs that tend to draw the eye into a central position. To hold the eye at an eccentric position, the muscles must produce a *static* force adequate to counteract the spring tensions. To move the eyes from one eccentric position to another requires an additional, *dynamic*, force that can overcome the resistance of the orbit to motion and accelerate the eye. Whereas the static force must be maintained as long as the eye is stationary, the dynamic force need be applied only during the actual movement of the eye.

Oculomotor muscle force is controlled directly by

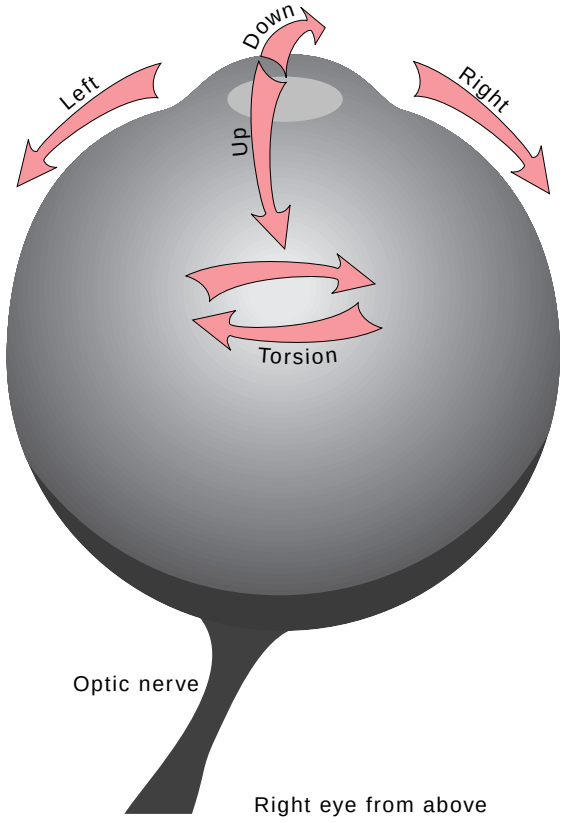


FIGURE 36.2 Axes of eye rotations. The extraocular muscles can rotate each eye along the horizontal, vertical, and torsional axes.

the firing rates of the oculomotor motor neurons. Therefore, when studying the oculomotor motor neurons, one can largely separate the static and dynamic forces by investigating motor neuron firing rates when the eye is either stationary or in motion.[2,3] Such investigations reveal that eye position is a linear function of firing rate while the eye is stationary (Fig. 36.3). Each motor neuron has a recruitment point (an eye position at which the neuron begins to fire) and a characteristic slope (change in firing frequency for a 1° shift of the eye toward the pulling direction of the innervated muscle.) Oculomotor motor neurons also show a high-frequency *pulse* of activity during much of the high-velocity phase of eye movements. This pulse is followed by a sustained firing rate associated with the new static position of the eye. From these data we can conclude that the high-frequency pulse of activity generates the dynamic force produced during an eye movement, whereas the sustained firing generates the static force.

The preceding discussion describes the behavior of oculomotor motor neurons when their muscles are pulling the eye. For every eye movement, however, one muscle of each antagonistic pair must relax when the other contracts. During movements in which the muscle under study relaxes, the motor neurons innervating that muscle pause during the movement and then resume firing at a reduced rate appropriate for the new orbital position of the eye.

Perhaps the most interesting aspect of these observations is that all oculomotor motor neurons exhibit this pattern of behavior, encoding in their rate *both* the static and the dynamic components of the force structure of each movement.[4] This finding has two important implications. First, it means that all oculomotor muscle fibers contribute to both movement and position holding (because all motor neurons do so.) Second, it means that the combined pulse / step structure of all eye movements is computed at or before the level of the oculomotor motor neurons. As we will see in the following sections, the principal task of the oculomotor system is to compute these pulse / step muscle force patterns. The challenge faced by the system is to achieve either gaze stabilization or gaze shifting by converting sensory information from many modalities—visual, vestibular, auditory, and somato-

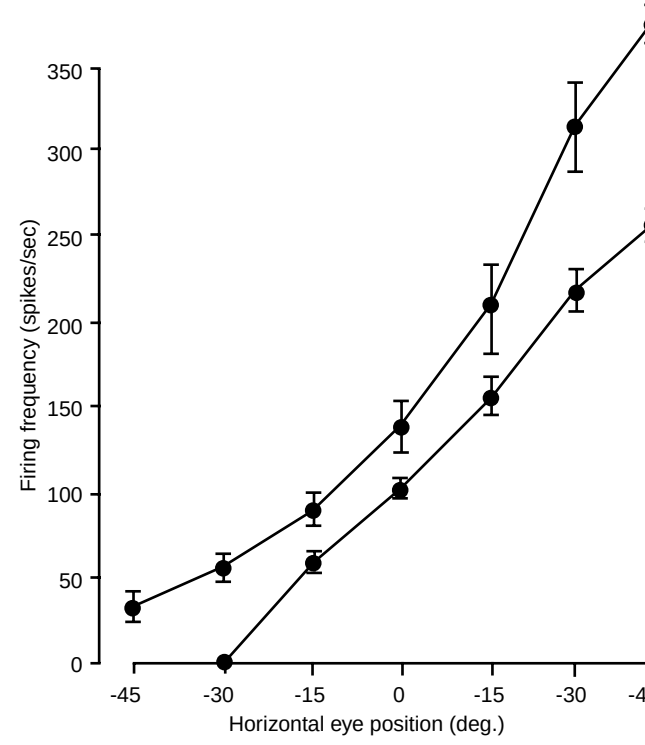


FIGURE 36.3 Rate-position curves for the abducens motor neurons. Such plots of motor neuron firing rate as a function of eye position when the eye is stationary demonstrate that motor neuron firing rate is linearly related to static eye position. (From Fuchs and Leishei.[2])

sensory—into the common language of pulse / step muscle forces.

Summary

The simplicity of the oculomotor system derives from the simplicity of its mechanics, the simplicity of its muscular and neural control, and the compactness of the brainstem circuitry that computes the fundamental neural signals required to drive it.

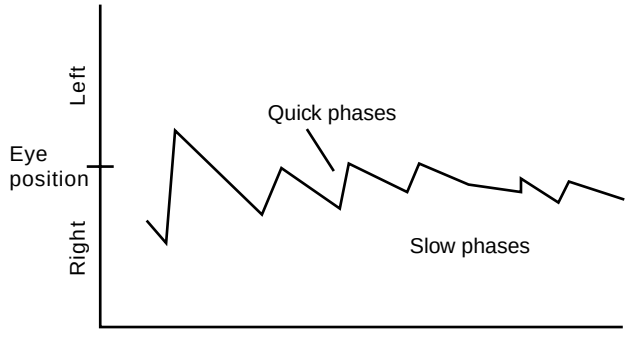
THE VESTIBULOOCULAR REFLEX

The **vestibuloocular reflex** (VOR) is the neural system by which rotations of the head are detected by the semicircular canals of the vestibular organs, and the eyes are counterrotated in their sockets an equal amount in the opposite direction to stabilize the line of sight. This reflex is in constant use. Whenever you walk, for example, the VOR is engaged, compensating for the small visually disruptive movements of the head that are produced during locomotion. The VOR is also highly precise. If you rotate your head from left to right while reading this page, you will find that you can move your head quite quickly before the text becomes unreadable.

To compensate for head rotation, the VOR rotates the eyes. If the head continues to rotate, of course, the eyes cannot continue to counterrotate without being pointed backward in the orbit. To overcome this limitation, the eye is often reset to a central position in the orbit during a vestibular eye movement. After this reseting is complete, the compensatory counterrotation resumes (Fig. 36.4). The comparatively slow compensatory movements and the quick resetting movements constitute the two phases of the VOR. This characteristic pattern of alternating quick and slow phases during sustained rotation is called **nystagmus.** (A leftward nystagmus is one in which the quick phases shift gaze to the left.) Note that only the slow phase compensates for head rotation, the quick phase simply returns the eye to the center of the orbit. In the following discussion we will focus on how inputs from the semicircular canals structure the compensatory slow phase of the VOR.

The Semicircular Canals Measure Angular Velocity of the Head in Space

Each member of the bilaterally symmetrical pair of vestibular organs contains three semi-circular canals oriented at roughly 90° to one another (Fig. 36.5). Each canal consists of a very thin bony tube filled with fluid. As the tube rotates, the fluid inside lags behinds due to its inertia. The rotational motion of the tube can therefore be detected by comparing the relative rates at which the fluid and the tube move. In the vestibular organ, this comparison is performed by the **cupula,** a thin, elastic membrane that stretches across the canal. Rotation of a canal deflects the cupula, and this deflection is monitored by hair cells like those in the cochlea (Chapter 27). Recall that the neural output of a hair cell is proportional to the deflection of its kinocilium. In the semicircular canals, the kinocilia are mechanically coupled to the cupula, so that the output of the hair cell serves as an accurate measure of the angular velocity of canal rotation.[5]



Obviously, a system of this type is most sensitive to rotations aligned precisely in the plane of the canal. Put more exactly, a given canal can measure only that portion of the rotational velocity that lies in its plane. To completely measure the rotational velocity of the head in three dimensions, it is thus necessary to use three separate canals, each oriented in a different plane. In fact, vertebrates have six canals—three on each side—arranged in coplanar pairs. Rotation of the head to the left, for example, activates the two canals that lie in the horizontal plane. The hair cells in one of these canals will be depolarized by this rotation, as the cupula deflects their kinocilia in their preferred direction. The hair cells in the other canal will be hyperpolarized, as that canal's cupula, moving in the opposite direction, deflects their kinocilia in the opposite direction. Single-unit recordings from canal afferent fibers during rotations (Fig. 36.6) show that this system does accurately code rotational velocity to the nervous system.[6,7]

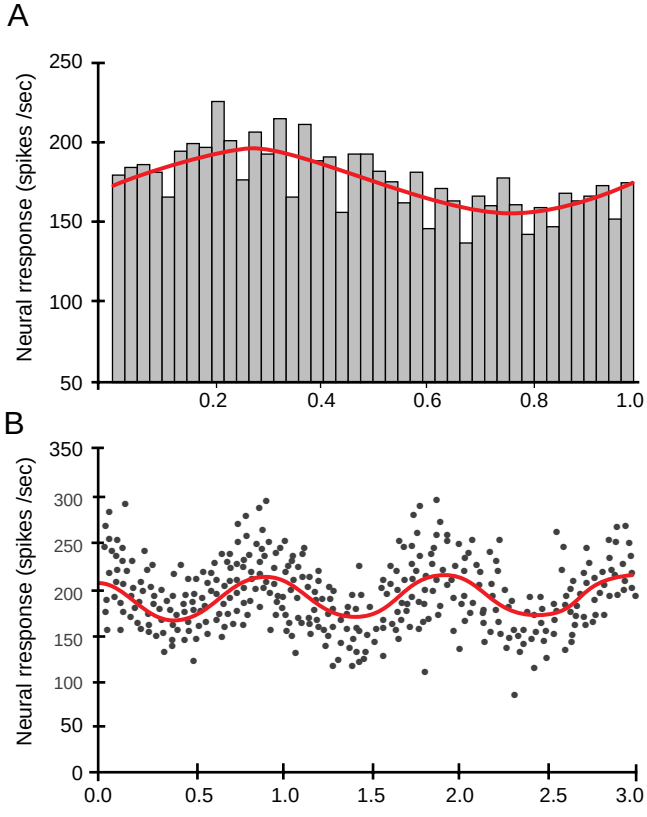


FIGURE 36.6 Response of a semicircular canal afferent to movement of the cupula. (A) The average firing frequency of the afferent nerve varies sinusoidally as the cupula is deflected in and then out in a single cycle. (B) The instantaneous firing frequency of the afferent is plotted over three consecutive cycles. (From Dickman and Correia.[7])

The Vestibular Nucleii Add Static Eye-Holding Signals to the Dynamic Eye-Moving Signals

Canal afferents synapse on neurons of the vestibular nuclei, many of which have a firing rate that is proportional to rotational velocity in one of the canal planes (Fig. 36.7).[8] How is this sensory measure of the rotational velocity of each canal used to compute a matched velocity of eye rotation? Because each of the canal pairs is roughly aligned with one of the extraocular muscle pairs, the velocity signal associated with one canal pair could, in principle, be used to control the eye velocity governed by the aligned pair of muscles. The vestibular nuclei do make some direct projections to the oculomotor nuclei,[9] suggesting that head velocity signals could directly regulate eye velocity. The canal-derived velocity signal could therefore account for the velocity of eye movements, the *dynamic* component of the VOR.

Once the velocity of the head dropped to zero, we might expect the eye to return to its initial, pre-VOR position, because the static force necessary to hold the eye at a given position in the orbit must be changed each time the velocity of the eye carries it to a new position. As explained in the preceding section, it is the static, or step, force that must persist after each eye movement is complete. If the sensory signals in the vestibular nucleus are the source of the dynamic phase of each VOR eye movement, what is the source of the accompanying change in static force? Because velocity is the first derivative of position, the static force could be computed by taking the mathematical integral of the velocity signal supplied by the vestibular nuclei. One model proposed to explain this process is diagramed in Fig. 36.8.[10] In this model, signals proportional to velocity are generated by the semicircular canal afferents and transmitted to the vestibular nucleus. From there, the signal is relayed to the oculomotor nuclei and to a neural integrator that adds any change in step intensity to the pre-VOR step intensity. The output from the integrator is also passed to the oculomotor nuclei. In this way, a simple sensory signal encoding velocity could be transformed into a static and dynamic force pair tailored to the needs of the extraocular muscle system.

The VOR Is Plastic

In the preceding section, we described how the VOR appears to operate in general. To function well, this system must relate given levels of canal activation to given velocities of eye rotation. Changes in the strength or efficiency of the extraocular muscles, for example,

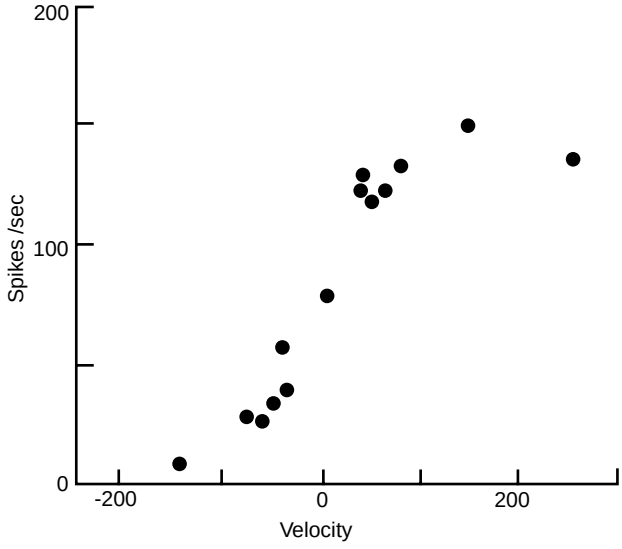
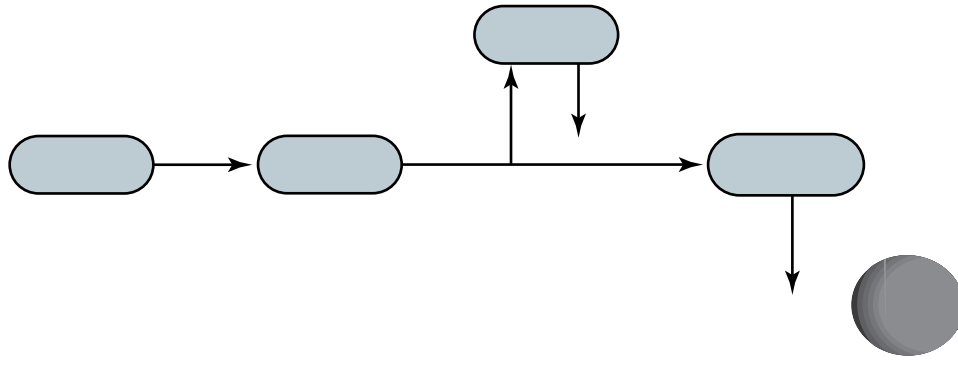


FIGURE 36.7 The firing rate of semicircular canal afferents codes rotational velocity. Over a range of almost 300%, firing rate is a linear function of velocity. (From Groen *et al.*[8])

would require adjustments in the strength of the linkage between the vestibular signal and the muscle contraction. The strength of this linkage, usually referred to as its *gain,* is equal to the magnitude of the induced eye rotation divided by the magnitude of the vestibular rotation. Thus, a gain of 1 describes a situation in which eye and head rotations are perfectly matched, a gain of 0.5 describes one in which the eye undercompensates for head rotation by half, and a gain of 2 describes one in which the eye rotates twice as far as it should. To examine the flexibility, or **plasticity,** of VOR gain, investigators instructed human subjects to wear magnifying lenses. Such lenses expand the view of a small region of visual space, causing eye movements to produce smaller displacements of the retinal image. For example, 3× lenses would require a subject to make an eye movement three times as large to compensate for a given head rotation. In one experiment, a subject who wore 2× magnifying lenses had a VOR gain that changed to 1.8 after a few days.[11] This and similar experiments demonstrated that the VOR can adapt. Although the cerebellum is necessary for adaptation to occur, it can be removed after adaptation is complete without changing the gain of the VOR.[12–14] This observation led to a revision of the neural integrator model of the VOR. According to the revised model, the cerebellum uses visual information (the slippage of an image on the retina during head movement) to determine whether the current VOR gain effectively cancels out the effects of the head movement. If the effects are not canceled out, the cerebellum generates an *error signal* that can be used to increase or decrease the gain of the VOR.[15]

The VOR Is Frequency-Limited

Many researchers studying the VOR have analyzed its gain during sinusoidal rotations of humans and animals. In these experiments, subjects are rotated back and forth at various frequencies. The gain of the VOR is calculated at each frequency and then plotted as a function of frequency. These rotations are performed in the dark so that the optokinetic system, which is discussed in the next section, cannot be responsible for eye movements. The rationale for this approach is that for some types of systems, those referred to as *linear,* it is possible to calculate the gain (or response) of the system to any stimulus if the response of the system



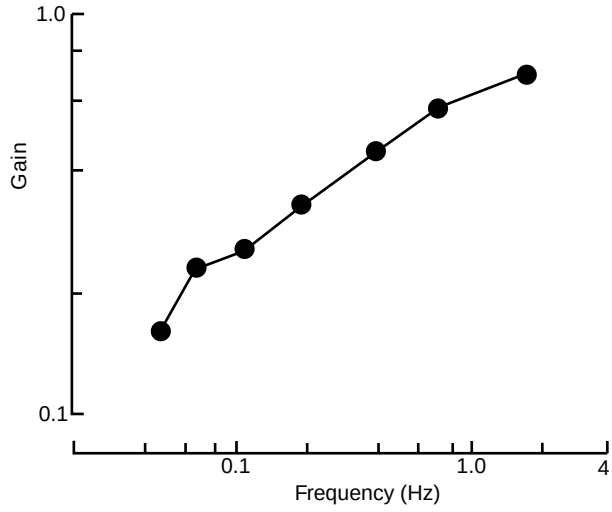


FIGURE 36.9 Gain of the vestibuloocular reflex as a function of rotation frequency. (From Baarsma and Collewijn.[16])

to all cases of single-frequency stimulation is known. As Fig. 36.9 indicates,[16] the VOR counterrotates the eye almost perfectly except at very low frequencies (around 0.1 Hz and lower). The lower limit on the VOR is of particular interest, because at low frequencies the visual system can be used to stabilize gaze. As we will see in the next section, the optokinetic system does just that, efficiently compensating for very-low-speed movements of the head.

Summary

The vestibuloocular reflex is a good paradigm for understanding motor control in general. The behavioral goals are clear-cut, the mechanics of the movements and the neuromuscular controls are straightforward, and all of the computation needed for holding and moving signals is performed by a compact and relatively well described circuitry within the brainstem, all driven by input signals from the semicircular canals.

THE OPTOKINETIC SYSTEM

Like the VOR, the optokinetic system activates the oculomotor musculature to stabilize gaze during rotations of the head. Unlike the VOR, however, the optokinetic system uses visual information, extracting from the global pattern of visual stimulation a measure of how fast and in what direction the visual world is moving across the retina. This movement of the visual world, often called **retinal slip,** is used to generate an eye movement equal in speed and opposite in direction to the retinal slippage, thus stabilizing the visual world on the retina. Like the VOR, the optokinetic system produces nystagmus: a slow phase, during which the eyes compensate for movement of the visual world, is followed by a quick phase that moves the eyes back from the limits of their orbital rotation to a more central position. To study optokinetic nystagmus, scientists typically present subjects with a display of vertical stripes or randomly arranged dots that rotate uniformly around the center of the subject's head. Because the subjects are stationary, we know that the vestibular system cannot be responsible for generating the nystagmus, which must therefore be produced by the movement of the visual stimulus.

Midbrain Circuits Translate Visual Signals into Velocity Signals

In humans and most other vertebrates, the retina projects directly to a midbrain area just rostral to the superior colliculus called the **pretectum.** Many neurons in this area become active when the visual world slips in a particular direction, and their firing rate increases as the velocity of slippage increases (up to a point). Thus, pretectal neurons encode the velocity and direction of retinal slip.[17] They project directly to the vestibular nuclei via pontine and medullary relays. In fact, many vestibular neurons can be activated by both vestibular and visual stimuli,[18] suggesting that the two types of stimuli access a common circuitry for eye velocity control. This common motor circuitry may also integrate the optokinetic velocity signals to calculate the static force necessary to hold the eye at its new position.

The Optokinetic Response Has Gain

Like the VOR, the optokinetic response has a gain that can be measured. In this case, gain is the ratio of eye rotational speed to visual world rotational speed. A gain of 1 indicates that the eye rotates perfectly, completely stabilizing the visual world on the retina. A gain of less than 1 indicates that the eye lags behind the visual world, only partly compensating for retinal slip. Measurements of gain as a function of frequency have been made for the optokinetic response just as they have for the VOR. To measure optokinetic gain, however, researchers rotate an animal back and forth in front of an illuminated and stationary visual environment after surgically removing the semicircular canals.[16] Notice in Fig. 36.10 that the gain of the optokinetic response falls off at higher frequencies of rotation. Comparison of Figs. 36.9 and 36.10 reveals that the vestibuloocular and optokinetic systems, which trans-

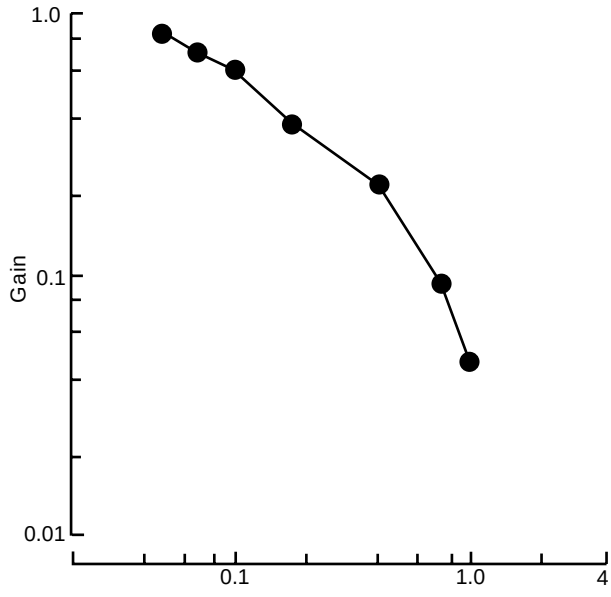


FIGURE 36.10 Gain of the optokinetic response as a function of rotation frequency. (From Baarsma and Collewijn.[16])

lates two types of sensory signals into a common motor framework, can effectively stabilize gaze over a very wide range of image / head velocities.

Summary

The optokinetic system complements the vestibuloocular reflex. Both maintain stability of gaze despite head movement, and both employ simple and shared control components, the vestibular working best at high frequency and the optokinetic working best at low frequency head movements.

THE SACCADIC SYSTEM

Saccades are gaze-shifting responses that can rotate the eyes as quickly as 800°-s^{-1}. The circuitry that controls these movements is perhaps the most successfully studied in the mammalian motor system. Like the gaze-holding systems we have already examined, the saccadic system must generate a dynamic force pulse that accelerates the eye to a high velocity as well as an increment in the static force that keeps the eye at its new position. As we will see, the saccadic system uses visual, somatosensory, and auditory information to compute the eye rotation necessary to align the line of sight (often called the line of gaze) with a visual target. The magnitude and direction of the desired change in eye position—the motor error—are relayed to a set of brainstem control circuits that calculate the static and dynamic forces necessary for the selected eye rotation.

The Brainstem Contains Circuits that Control Saccades

As we learned in an earlier section, the extraocular muscles are controlled by three bilaterally symmetrical pairs of cranial nerve nuclei. Lying between these nuclei, straddling the midline of the brainstem, is the **paramedian pontine reticular formation** (PPRF). Recordings from the PPRF in awake, behaving monkeys demonstrated the existence of a class of neurons called **burst neurons,** which fire a vigorous burst of action potentials beginning about 8–12 ms before each eye movement.[19,20] Interestingly the number of spikes in these bursts is linearly related to the horizontal amplitude of the saccade. A large upward movement that involves only a small rotation of the eyes to the left is preceded by a small burst in the left-preferring burst neurons, whereas a larger rotation directly to the left is preceded by a larger burst in the same neurons. These bursts, which code horizontal (in this case, leftward) motor error, could be used to generate the dynamic force pulse needed to accelerate the eye during a saccade. Supporting this hypothesis is the observation that these neurons often project directly into the oculomotor nuclei.[21,22] A group of neurons that seem to code vertical motor error has also been discovered just rostral to the oculomotor nuclei.

These findings suggest that the dynamic phase of saccade is coded by pontine burst neuron activity. If the dynamic phase were generated alone, however, the viscoelastic ''springs'' that pull the eye back toward the center of the orbit would slowly return the eye to its presaccadic position. This is exactly what happens when a lesion is placed in the prepositus nucleus of the hypoglossal nerve,[23] suggesting that this nucleus may contain a neural integrator that calculates the static force needed to hold the eye at its new position.

Lying just rostral and ventral to the burst neurons is a group of tonically active neurons called omnipause neurons, which stop firing action potentials shortly before the burst neurons become active.[20] The duration of this pause in firing is tightly correlated with the duration of the saccade. Furthermore, electrical stimulation of the omnipause region prevents saccades from occurring. These observations suggest that the cessation of firing in omnipause neurons may be critical for triggering a saccade, whereas the amplitude and duration of the saccade would be specified by the particular motor error signals.

Where does the oculomotor brainstem receive the motor error signals that control saccades? Two princi-

pal structures appear to serve that role: the superior colliculus and the frontal eye fields. In fact, either structure seems to be capable of generating these signals, since lesions of one or the other can be compensated for but lesions of both abolish all saccades.[24] We will begin our discussion of the generation of saccadic motor error signals by examining the superior colliculus.

The Superior Colliculus Is a Laminated Structure Lying above the Aqueduct in the Midbrain

At the end of the 19th century, researchers discovered that electrical stimulation of the superior colliculus produced high-velocity movements of the eyes similar to saccades. However, it was not until the early 1970s that researchers began to reexamine the superior colliculus and provide fundamental insights into how eye movements are controlled. In one series of experiments[25] an electrode was lowered into the colliculus and a small stimulating current was delivered through the electrode tip. About 20 ms after the onset of stimulation, a saccade began. The saccade had a characteristic amplitude and direction, and once the eye had moved that amount, it stopped. If the stimulation was maintained, a second saccade of identical amplitude and direction was eventually made from the end point of the first saccade. Moving the electrode to a new location in the colliculus yielded a similar pattern of results, but the movement amplitude and direction were different. These experiments allowed investigators to draw three important conclusions. First, stimulation of the colliculus does not simply cause the eye to move; rather, it specifies a precise eye movement specific for the location that is stimulated. Second, stimulation produces movements of a particular amplitude and direction regardless of the starting place of the eye in the orbit. Therefore, like pontine burst neurons, the colliculus appears to encode saccades with respect to a rotation of the eye, using what we have been referring to as motor error coordinates. Finally, the colliculus contains a topographic map of eye rotations: adjacent sites in the colliculus, when stimulated, produce saccades that shift gaze to adjacent points in visual space. The topographic map of the colliculus, which is now often referred to as a motor map, encodes movements in motor error coordinates much as visual area VI encodes visual stimuli with regard to their site of activation on the retina.

In another series of experiments, single-unit recordings were made in the superior colliculi of awake monkeys while the animals made saccades to fixate visual targets.[26] These recordings revealed a group of neurons, called collicular saccade-related burst neurons, that fire a high-frequency burst of impulses beginning about 20 ms before the saccade. Each neuron fires strongly before some saccades but only weakly or not at all before others. Although a given neuron is most active before a movement of a particular amplitude and direction (termed its *best movement*), it is also active for many similar movements. Thus, individual burst neurons are very broadly tuned. Collicular burst neurons are also arranged topographically, as cells with similar best movements are located near each other in the colliculus. These neurons encode motor error rather than information about the absolute position of the eye.

Vector Averaging across Collicular Neuron Populations May Code for Eye Movement

The collicular stimulation and recording experiments gave rise to an obvious hypothesis: collicular burst neurons generate a motor error signal that is used by the pontine oculomotor circuitry to control saccades. One problem with this hypothesis is that collicular burst neurons are very broadly tuned, whereas saccades are highly precise. How could the amplitude and direction of a saccade be coded accurately by such inaccurate elements? One possible solution to this problem is that the output of the colliculus may be *vector-averaged*.[27,28] According to this proposal, each collicular burst neuron ''votes'' for its best direction, but the strength of its vote is described by its rate of firing. Thus, a cell with a 10° upward best movement would vote strongly for a 10° upward movement and more weakly for movements 10° upward and slightly to the right or left. The vector average of these votes could specify the amplitude and direction of the movement with tremendous precision. As we saw in Chapter 33, the same proposal has been adopted to describe the way in which neurons in the motor cortex may code movement direction with precision.

The idea that vector averaging is used in the superior colliculus was tested by anesthetizing a small, circular portion of the colliculus with lidocaine injected from a micropipette.[29] Saccades corresponding to the best movements of the cells at the center of the anesthetized region were unaffected by the anesthesia, presumably because the cells around this region voted weakly, but equally, for movements in that direction. However, when the animal was directed to make a saccade that placed the anesthetized region to one side of the active region, its movements were systematically biased away from the direction specified by the anesthetized region (Fig. 36.11). These experiments established that a vector average of a population code is

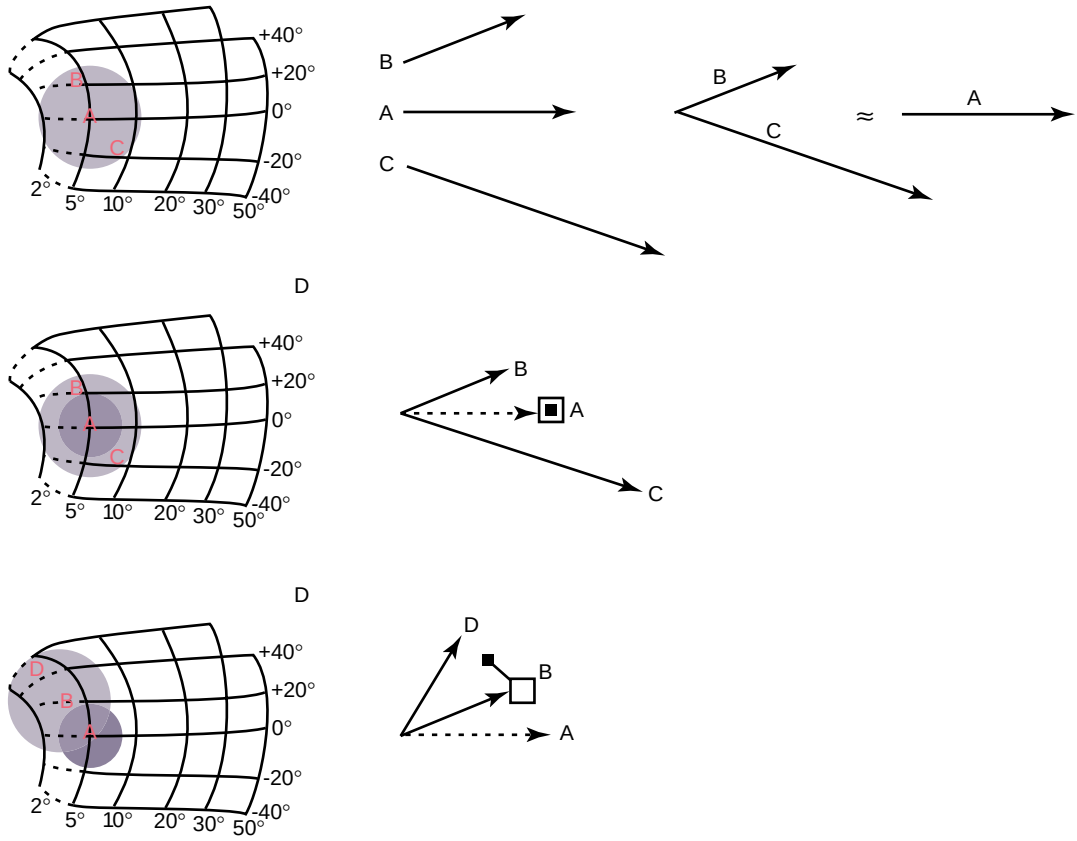


FIGURE 36.11 Vector averaging in the superior colliculus. *Top left:* The stippled area represents the location of neurons in the superior colliculus that are active before a 5° rightward saccade. *Top middle:* Cells at locations A, B, and C fire most vigorously for movements in the directions shown by the arrows. *Top right:* The weighted average of activity at points B and C yields the same movement as activity at A. *Bottom Panels:* The dark stippled area represents a pharmacologically deactivated collicular region. The regions of the colliculus activated during saccades A and B are plotted in the left and right panels, respectively. Saccades targeting A are normal, while saccades targeting B deviate toward D. (From Lee *et al.*[29])

used to specify the desired motor error for a saccade. They provided the first example of vector averaging in a mammalian motor system.

Sensory Signals from Many Modalities Guide Saccade Planning

So far we have described how motor error is coded in the superior colliculus and how pontine circuitry uses that information to govern the rotation of the eye. We have not yet addressed the sources of those motor error signals. Primates and other foveate animals can shift their gaze to fixate visual, auditory, and somatosensory targets. This means that if a visual stimulus is presented in the peripheral visual field, the eye can be rotated to align the fovea with that stimulus. If a sound is presented or a point on the body surface is touched, the eye can be directed so that the line of gaze intersects with the sound source or that point on the body surface. Each of these events involves what is known as a sensorimotor transformation. Information encoded by each sensory system according to its own topographic map must be translated into motor error signals.

The simplest of these sensorimotor transformations involves the use of visual information to derive motor error. The simplicity is due to the fact that the retina, like the oculomotor muscles, is anchored to the eye. Thus, the location on the retina activated by a visual stimulus specifies the eye rotation required to foveate the stimulus. For example, if a visual stimulus activates the retina at a location 10° to the right of fixation, then a 10° rightward saccade will bring the fovea in line with the stimulus. In fact, the most superficial laminae of the superior colliculus receive a direct retinal projection that is topographically aligned with the collicular

motor map.[30] Moreover, most of the extrastriate visual areas described in Chapter 28 also project directly to the colliculus, providing another route by which retinally sampled visual stimuli can be transformed into motor error signals.

The generation of saccades to auditory targets presents a larger problem for sensorimotor transformation. Auditory targets are localized by comparing information received from the two ears. However, knowing that the target lies straight ahead does not specify what rotation of the eye will fixate it. To solve that problem, the current line of gaze must also be known. If, for example, the eyes are directed leftward, then a straight-ahead target requires a rightward movement. If the eyes are directed rightward, a leftward eye movement is required. Recordings from collicular neurons have provided some insight into how the brain solves this problem.[31] These recordings show that collicular saccade-related burst neurons encode the motor error of impending saccades to auditory targets just as they do for visual targets, irrespective of where the eyes are directed before the saccades begin. Thus, the rotation required to foveate an auditory target—not the location of the target with respect to the ears—is supplied to the pontine saccadic circuitry by the collicular burst neurons (Fig. 36.12).[32] Another class of neurons found in the deepest laminae of the colliculus become active when an auditory target is presented in their receptive field. However, these neurons are activated only when the eyes are aligned so that the auditory target has the foveal motor error associated with each cell's location on the collicular motor map. This means that sensory stimuli received by the ears and used to compute the locations of targets relative to the ears must be transformed into motor error coordinates *before* they activate these collicular neurons. These studies helped establish that a *sensorimotor transformation* is a process by which stimuli form the many different sensory systems are translated into a common framework, one appropriate for activating the musculature.

Somatosensory stimuli are also transformed to guide saccadic eye movements. However, unlike auditory stimuli, for which only the position of the eyes in the orbit is necessary to transform location data,

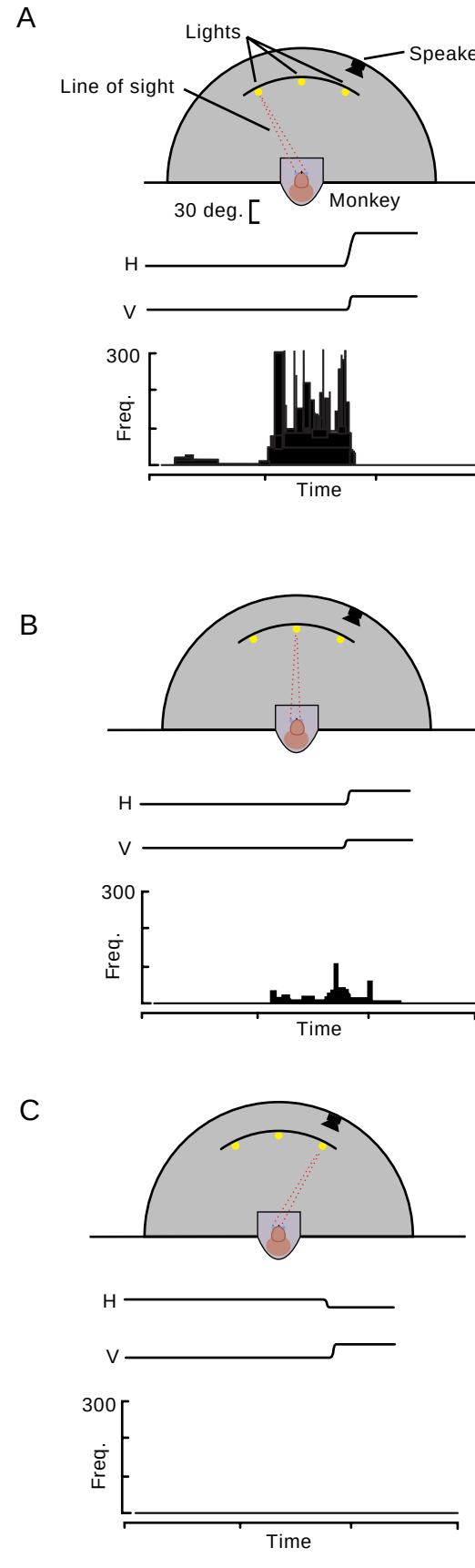


FIGURE 36.12 Shifts in auditory response fields produced by eye movements. All three panels plot the response of a single collicular neuron to an acoustic stimulus fixed 15° to the right of the monkey. (A) When the animal is fixating a leftward target, the neuron fires vigorously when the stimulus is presented. (B) When the animal fixates straight ahead, the neuron fires less vigorously. (C) When the animal fixates the speaker, the neuron is silent to the same acoustic stimulus. (From Jay and Sparks.[32])

BOX 36.1

CLINICAL SYNDROMES OF THE FRONTAL EYE FIELDS

Damage to the frontal eye field in one hemisphere causes impairment in the ability to shift gaze to the opposite side. At rest, gaze may be tonically shifted to the side damaged, due to unopposed normal tonic activity from the frontal eye field in the opposite hemisphere. In contrast, epileptic convulsive (''seizure'') discharge in one frontal eye field causes forced gaze shift and / or saccades away from that hemisphere. Tests of even greater sensitivity depend on the fact that the frontal eye fields control voluntary gaze shifts and saccades. Thus, when a visual stimulus is presented in one visual field, the normal tendency is to look at it using visual targeting mechanisms in parietal lobes and superior colliculus in addition to those in the frontal eye fields. However, the frontal eye fields are exclusive in their ability to override voluntarily these visual attraction ''reflexlike'' mechanisms. Thus, the instruction to the subject is to ''look away from the visual stimulus when it appears.'' An impaired ability to do this, for example, to look to the left away from a right visual field stimulus, indicates damage (e.g., tumor) of the right frontal eye field.

somatosensory stimuli of the limbs, for example, must be corrected both for the position of the eyes in the head and for the location of the stimulated limb with respect to the head. If neurons in the deeper layers of the colliculus code somatosensory stimuli in motor error coordinates, then their receptive fields on the body surface must depend on both eye position and body surface position. Recordings from these neurons confirm this prediction. In fact, if a monkey is made to shift its initial eye position, a collicular neuron's receptive field can be moved from one hand to the other! This remarkable demonstration of sensorimotor transformation makes it clear how important it is for the nervous system to take signals from disparate sensory systems and combine them in a single coordinate framework.

Higher-Level Saccadic Systems Include Cerebral Cortex and the Basal Ganglia

The sources of the collicular saccadic control signals are only vaguely understood. On anatomical and physiological grounds, we do know that collicular saccade-related information is provided by the frontal eye fields, the parietal cortex, and the basal ganglia. All of these areas project directly to the colliculus and have patterns of unit activity that indicate they play a role in activating collicular burst neurons. The most important of these areas is believed to be the frontal eye fields. See Box 36.1. Unlike the parietal cortex and the basal ganglia, the frontal eye fields project directly to the pontine eye movement control circuitry.[33] After lesions of the colliculus, the frontal eye fields alone can generate saccades.[24] This cortical area must therefore serve a role similar to that of the colliculus. Many of the collicular properties we have described above, like the transformation of auditory targets into saccadic motor error coordinates, are also evident in the frontal eye fields. Ongoing research seeks to understand how all of these antecedent structures give rise to the oculomotor control signals that guide collicular, pontine, and cortical eye movement control structures.

Summary

The saccadic system brings in higher brainstem and cerebral mechanisms to identify a target and move gaze to it. The eye movements themselves (saccade means ''jump'') are very fast, and they minimize the time lost in visual contact while gaze is shifting to a new target.

SMOOTH PURSUIT

The optokinetic system moves the eyes at a velocity that compensates for movement of the visual field. We considered this a gaze-stabilization mechanism and pointed out that essentially all animals have this phylogenetically ancient system. Animals with foveas can also use saccades to direct the fovea to scrutinize targets throughout visual space. However, these animals face a special challenge when they make a saccade to examine a moving target. The saccade may bring the fovea briefly into alignment with the target, but unless the eye can be made to move with the same velocity as the target, the target image will not be stabilized on the retina. Matching the velocity of the eye to that of

BOX 36.2

CLINICAL SYNDROMES OF THE MEDIAN LONGITUDINAL FASCICULUS

The MLF carries signals that coordinate actions of the lateral rectus of one eye with the medial rectus of the other eye for lateral movement in the VOR, smooth pursuit, saccades, and maintained gaze. Thus, damage (typically a small infarct caused by occlusion of a small artery) of the right MLF (after crossing midline) will impair the coordinate adduction of the right eye that naturally accompanies abduction of the left eye on attempted left gaze. Right gaze (abduction of right eye, adduction of left) will be normal. Damage to the MLF on both sides (typically a bilateral lesion in multiple sclerosis) causes failure of adduction of both eyes—that is, the right eye on attempted left gaze, the left eye on attempted right gaze. To be sure that it is indeed the MLF that is involved and not the third nerve itself (diabetic infarct) or the neuromuscular junction (myasthenia gravis), the clinician must show that adduction is normal in *vergence,* which does not critically involve the MLF. In contrast, damage to the midbrain convergence center (tumor of the pineal body with pressure from above) will impair adduction of both eyes in attempted convergence, but not adduction or lateral gaze.

the visual stimulus when the visual stimulus is the entire visual world is, of course, the optokinetic response. When the visual stimulus is only a tiny portion of the visual world, eye movements need to be driven by only a small portion of the retina. The eye movement system that minimizes the retinal slip of a small visual target while producing an increased retinal slip for the rest of the visual world is known as smooth pursuit. Although smooth pursuit might be viewed as a specialized form of the optokinetic response, these two systems involve at least partially distinct neural architectures.

Compared to what is known about the saccadic system, little is known about the brainstem circuits that compute the velocities of pursuit-related motor error. In general, neurobiologists have tended to view the pursuit system as similar to the optokinetic system: retinal slip, in this case restricted to a selected portion of the visual world, is minimized by adjusting eye velocity until slip is reduced or eliminated. Models of the pursuit system are thus quite similar to those of the optokinetic system. Knowledge about the speed and direction of the target's motion across the retina and the eye's current movement is used to compute a desired speed and direction of eye movement. The eye velocity signal is then passed via the brainstem oculomotor nuclei to the extraocular muscles to control the dynamic movement of the eye. This dynamic signal, like the dynamic signals for other eye movements, is presumed to be integrated to compute the static signal necessary to maintain the eye in a fixed position should the eye stop. This integrator is thought to reside in the prepositus nucleus of the hypoglossal nerve. Two hypotheses have been proposed to explain the source of the dynamic signal. One hypothesis states that the speed and direction of slip are the only visually derived inputs used by the smooth pursuit system.[34] The other hypothesis states that information about the position, speed, and acceleration of the target is combined to compute a dynamic force structure optimized for the properties of the visual stimulus.[35] The first hypothesis can thus be thought of as muscle-driven, in the sense that it uses a minimum of visual information and structures the dynamic force based entirely on the properties of the motor system. The second hypothesis can be thought of as sensory-driven, structuring the dynamic force by analyzing the visual target. As we examine the neural structures that generate pursuit movements, we will see that these two hypotheses place the computational burden on different brain structures: the muscle-driven hypothesis places this burden in the oculomotor brainstem, where dynamic forces are structured for other types of eye movements, whereas the sensory-driven hypothesis places it in the largely cortical area that compute information related to target motion.

The Cerebellum Plays a Key Role in Smooth Pursuit

The dorsolateral pontine nucleus (DLPN) is a critical link in the smooth pursuit system, bridging between the cortical motion-processing systems discussed in Chapter 33 and the oculomotor portions of the cerebellum and brainstem. The DLPN contains neurons that encode the direction and speed of pursuit, the direction and speed of target motion, or both.[36] Thus, the DLPN could, in principle, process information about both the

sensory representations of targets and the motor error signals required to drive the eyes. The output of the DLPN passes to the cerebellum (the flocculus, paraflocculus, and vermis), where there are neurons whose firing rate is tightly coupled to the velocity of eye rotation specifically during smooth pursuit. These neurons in turn project to the vestibular nuclei, where their motor error signals are thought to be integrated into the vestibulopontine oculomotor systems.[37,38]

Cortical Areas MT and MST Are Sources of Visual Target Motion Signals

The identified brainstem pursuit pathways form a portion of the complete pursuit system. Signals that indicate target motion are critical to their function. This information appears to be provided to the pursuit system by the cortical motion system, which is composed in part of areas MT and MST (described in Chapter 33). These areas compute the direction and speed of moving stimuli throughout the visual field and pass this information to the DLPN, directly and via the posterior parietal cortex and the frontal eye fields.

Summary

The smooth pursuit system provides insights into both brainstem structures for oculomotor control and into cortical structures for sensory processing. Pursuit thus may be unique; it combines high-level moving visual signals and pontine oculomotor circuits.

VERGENCE

The function of the vergence system is to converge or diverge the lines of gaze projecting from both eyes so that they meet at the target of foveal vision (Fig. 36.13). Of course, animals without binocular foveal vision do not need to align a single retinal region on both eyes with a single visual target. Therefore, only a limited number of vertebrates, including primates, generate vergence movements. The eye movements we have considered so far produce shifts in gaze as if a single line of sight were being redirected. In fact, this is exactly how the saccadic and smooth pursuit systems appear to compute gaze shifts. How then are movements of the two eyes coordinated so that both eyes, which are separated by several centimeters, fixate a single target? The German physiologist Ewald Hering proposed that an entirely separate system computes the appropriate vergence ''correction'' for each eye during gaze shifts and adds that signal to the binocular saccadic or smooth pursuit motor error command. In this section we will examine the sources of the vergence motor error signal and describe the physiological mechanisms that compute the dynamic and static muscle forces necessary to produce vergence movements.

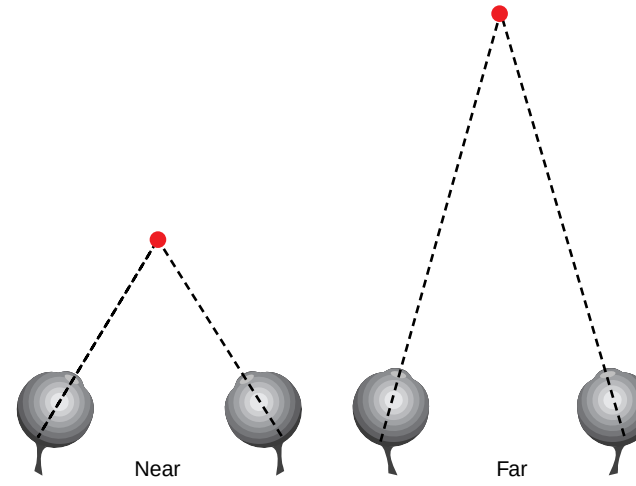


FIGURE 36.13 Convergence angle depends on target distance.

There Are Four Sources of Vergence Motor Error Signals

The brain generates four experimentally separable classes of vergence motor error commands. The first and most obvious is related to **binocular disparity,** which occurs when a visual stimulus is presented to both eyes but appears at different locations on each retina. The second class deals with accommodation. Apparently, the accommodative state of both lenses is monitored, and this information is used to compute the distance to the target currently in focus. The third class, usually referred to as tonic, represents the default state of convergence for the animal in total darkness. In humans it is equal to about 3° of convergence. The final class of vergence motor error commands uses monocular (or cognitive) depth cues, such as linear perspective, to infer the distance to targets. This class is usually referred to as **proximal vergence.**

The Vergence Brainstem Is Similar to the Saccadic Brainstem

Physiological studies of the oculomotor brainstem[39] have revealed that a small group of neurons lying in the rostral midbrain reticular formation appear to form a control center for vergence much like the pontine horizontal gaze-control center for saccades. Two types of neurons have been identified in this area. **Vergence burst neurons** fire a high-frequency burst of impulses

before the onset of a vergence movement, and their firing frequency is related to the velocity of the movement. Both convergence-specific and divergence-specific burst neurons have been identified. The number of impulses in a burst is related to the amplitude of the movement. **Vergence burst-tonic neurons** appear to combine the dynamic and static responses of the vergence system. By analogy with the saccadic system, it is tempting to presume that during a vergence eye movement, the output of the vergence burst neurons is integrated and reflected in the activity of the vergence burst-tonic neurons.

Most Gaze Shifts Involve Both Versional and Vergence Movements

As shown in Fig. 36.14, most gaze shifts for binocular, foveate animals involve both a coordinated movement of the two eyes together (version) and a separate movement of each eye to adjust for differences in the distance to the target (vergence). Thus, the point of gaze (the location at which the lines of gaze from each eye meet) must move both across and in or out. If we were to plot the location of the point of gaze during a saccade at successive 2-ms intervals, it would follow the trajectory plotted in Fig. 36.14. The observed eye movement has three phases. First, the eyes begin to converge quite slowly before the saccade begins. This constitutes the beginning of the vergence movement. After a delay, the high-velocity saccade begins; the point of gaze shifts across the visual field, but it also shifts in depth much more rapidly. Finally, the high-velocity saccade and the terminal, slow portion of the vergence movement are completed.

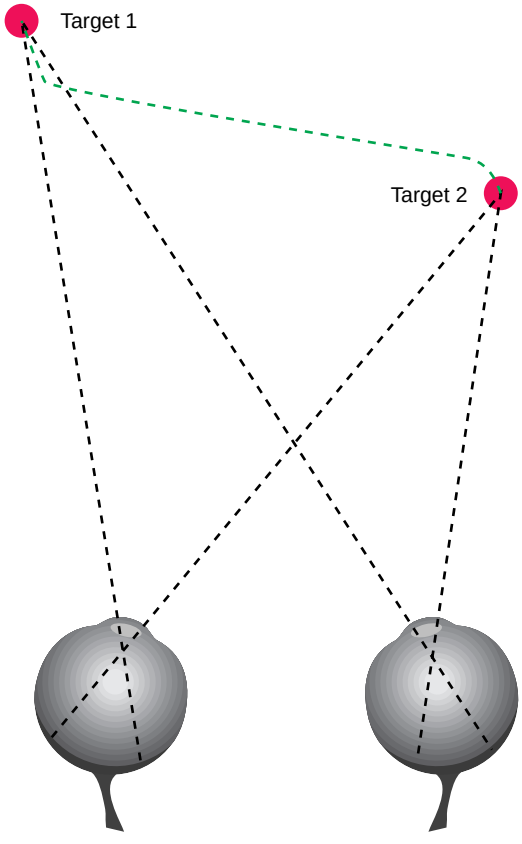


FIGURE 36.14 Trajectory of a saccade, viewed from above, that shifts the point of gaze laterally and in depth. (A) Vergence mechanisms begin to converge the eyes prior to the saccade. (B) The saccade then begins, and the rate of vergence accelerates. (C) After the saccade, the vergence mechanisms continue to converge the eyes until both foveas are aligned with the target.

Simultaneous studies of the vergence and saccadic systems[40] suggest that the vergence burst neurons are normally inhibited by the saccadic omnipause neurons, and that this inhibition limits the frequency of the vergence burster neurons' firing rate and hence the velocity of the vergence movement. The onmipause neurons, which act as the saccade trigger, are silent during saccades, disinhibiting the vergence burst neurons and thereby accelerating vergence while the saccade is in progress. After the saccade is completed, the omnipause neurons become active again, reinhibiting the vergence burst neurons and reducing the velocity of the remaining portion of the vergence movement. These studies reveal how the separate oculomotor systems interact during normal eye movements.

Summary

The effect of eye movements is twofold. First, eye movements stabilize the line of gaze while animals move in their environment. Unpredictable high-speed rotations of the head, which are produced whenever an animal moves, shift the line of gaze and smear the optical image, reducing the resolution of the visual system. These rotations are compensated for by counterrotations of the eyes generated by the vestibuloocular system. Working in tandem with the vestibuloocular system is the optokinetic system, which compensates for slower movements of the head. Second, in animals with retinas that have a small region of high resolution, a specialized set of eye movements—saccades, smooth pursuit, and vergence—align that region with visual targets of interest. Together, these sets of movements permit the efficient gathering of visual information by the retina.

As we have observed, eye movements involve generating at least two classes of signals: those involved in moving the eye to a new position and those involved in holding the eye at that position. The oculomotor system appears to combine these signals at or before

the level of the motor neurons for all classes of movements. The oculomotor system also appears to employ a critical shortcut, computing explicitly only the dynamic signals and then integrating those signals to produce the static signals.

Many classes of sensory signals—vestibular, visual, auditory, and somatosensory—are used to guide eye movements. In order for a single common output pathway to make use of this wide variety of input signals, each of these inputs must be transformed into a motor error signal. This process of sensorimotor transformation, which has been studied so extensively in the oculomotor system, may well prove to be a general feature of all or most vertebrate motor systems.

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