

Anatomy and Physiology of the Normal Vestibular System

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Purpose of the Vestibular System

The human vestibular system is made up of three components: a peripheral sensory apparatus, a central processor, and a mechanism for motor output (Fig. 1.1). The peripheral apparatus consists of a set of motion sensors that send *information to the central nervous system—specifically, the vestibular nucleus complex and the cerebellum—*about head angular velocity and linear acceleration. The central nervous system processes these signals and combines them with other sensory information to estimate

head and body orientation. The output of the central vestibular system goes to the ocular muscles and spinal cord to serve three important reflexes, the *vestibulo-ocular reflex (VOR)*, the *vestibulocollic reflex (VCR)*, and the *vestibulospinal reflex (VSR)*. The VOR generates eye movements that enable clear vision while the head is *in motion*. The VCR acts on the neck musculature to stabilize the head. The VSR generates compensatory body movement in order to maintain head and postural stability and thereby prevent falls. The performance of the VOR, VCR and VSR is monitored by the central nervous

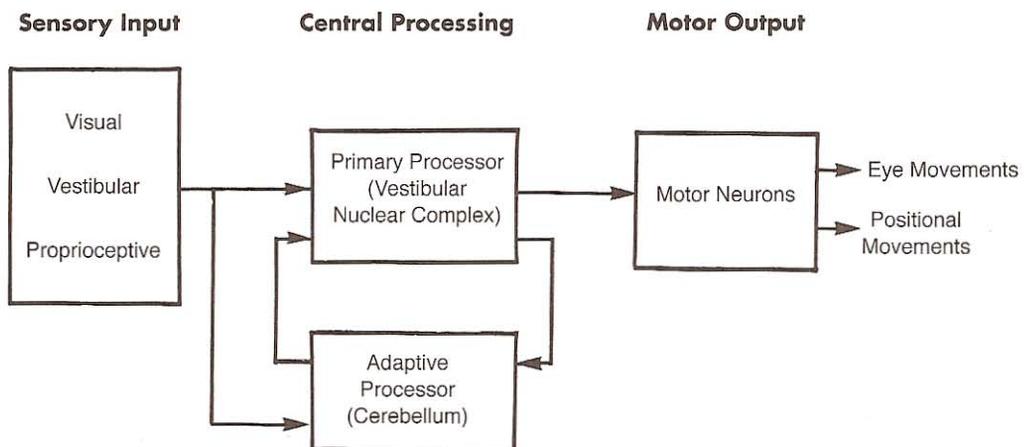


Figure 1.1 Block diagram illustrating the organization of the vestibular system.

system, is readjusted as necessary by the cerebellum, and is supplemented by slower but more capable higher cortical processes.

From a rehabilitation perspective, it is crucial to realize that because orientation in space is a critical function, multiple fail-safe mechanisms are closely integrated into vestibular responses. The capability for repair and adaptation is remarkable! Two years after removal of half of the peripheral vestibular system, such as by a unilateral vestibular nerve section, finding clinical evidence of vestibular dysfunction is often quite difficult. The ability of central mechanisms to use vision, proprioception, auditory input, tactile input, or knowledge about an impending movement allows vestibular responses to be based on a richly textured, multimodal sensory array.

With these general philosophical considerations kept in mind, the purpose of this chapter is to describe the anatomy and the physiologic responses of the vestibular system, with particular attention to aspects relevant to rehabilitation. We proceed from the peripheral structures to central structures and conclude with a discussion of “higher-level” problems in vestibular physiology that are relevant to rehabilitation.

The Peripheral Sensory Apparatus

Figure 1.2 illustrates the peripheral vestibular system in relation to the ear. The peripheral vestibular system

consists of the membranous and bony labyrinths as well as the motion sensors of the vestibular system, the hair cells. The peripheral vestibular system lies within the inner ear. Bordered laterally by the air-filled middle ear and medially by temporal bone, it is posterior to the cochlea.¹

Bony Labyrinth

The *bony labyrinth* consists of three semicircular canals (SCCs), the cochlea, and a central chamber called the *vestibule* (Fig. 1.3). The bony labyrinth is filled with perilymphatic fluid, which has a chemistry similar to that of cerebrospinal fluid (high Na:K ratio). Perilymphatic fluid communicates via the cochlear aqueduct with cerebrospinal fluid. Because of this communication, disorders that affect spinal fluid pressure (such as lumbar puncture) can also affect inner ear function.^[2]

Membranous Labyrinth

The *membranous labyrinth* is suspended within the bony labyrinth by perilymphatic fluid and supportive connective tissue. It contains five sensory organs: the membranous portions of the three SCCs and the two otolith organs, the *utricle* and *sacculle*. Note that one end of each SCC is widened in diameter to form an *ampulla*. This

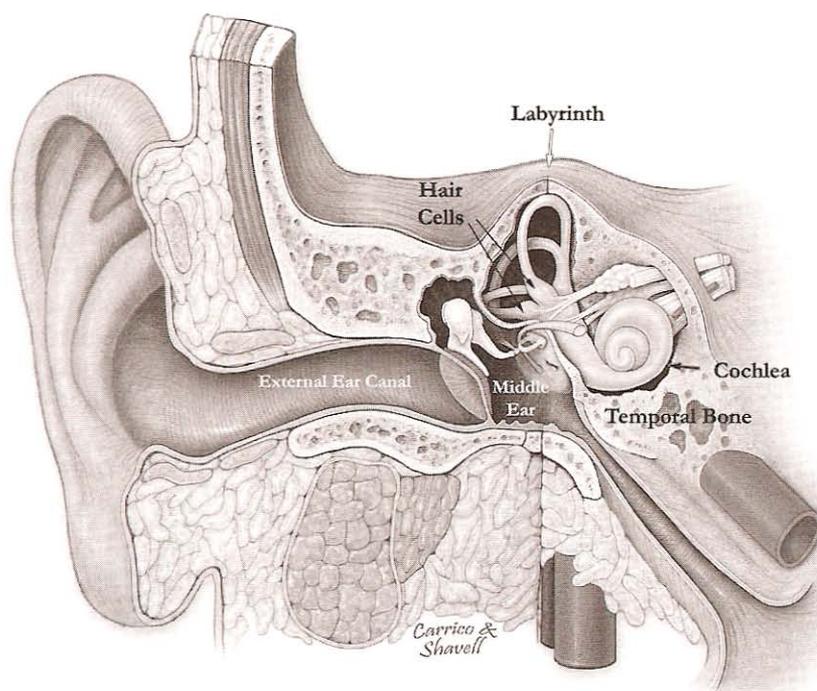


Figure 1.2 Anatomy of the peripheral vestibular system in relation to the ear. (Illustration adapted from <http://www.dizziness-and-balance.com/disorders/hearing/sensorineural.htm>, with permission.)

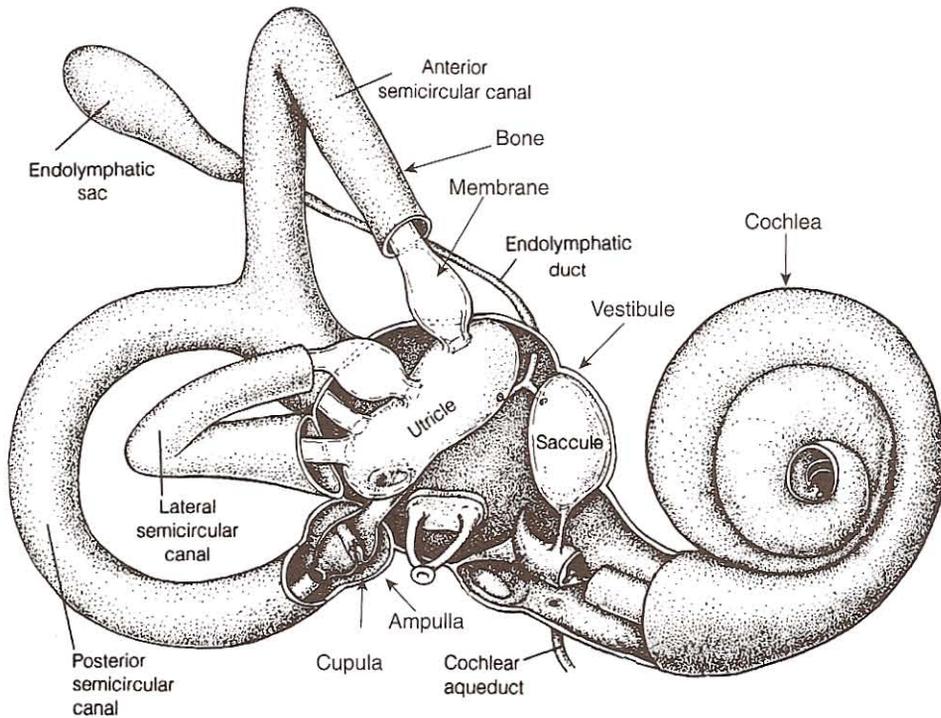


Figure 1.3 The membranous and bony labyrinths. The *inset* illustrates the perilymphatic and endolymphatic fluid compartments. (Adapted from an illustration by Mary Dersch; originally adapted from Pender, 1992.²)

widening is relevant to the understanding of a common vestibular condition, benign paroxysmal positional vertigo (see later).

The membranous labyrinth is filled with endolymphatic fluid (see Fig. 1.3). In contrast to perilymph, the endolymph resembles intracellular fluid in electrolyte composition (high K:Na ratio). Under normal circumstances, there is no direct communication between the endolymph and perilymph compartments.

Hair Cells

Specialized hair cells contained in each ampulla and otolith organ are biological sensors that convert displacement due to head motion into neural firing (Fig. 1.4). The hair cells of the ampullae rest on a tuft of blood vessels, nerve fibers, and supporting tissue called the *crista ampullaris*. The hair cells of the saccule and utricle, the *maculae*, are located on the medial wall of the saccule and the floor of the utricle. Each hair cell is innervated by an afferent neuron located in the vestibular (Scarpa's) ganglion, which is located close to the ampulla. When hairs are bent toward or away from the longest process of the hair cell, firing rate increases or decreases in the vestibular nerve (see Fig. 1.4A). A flexible, diaphrag-

matic membrane called the *cupula* overlies each crista and completely seals the ampulla from the adjacent vestibule. Associated with angular head motion, endolymphatic pressure differentials across the cupula cause the cupula to bend back and forth, stimulating the hair cells (Fig. 1.4B).³

The *otolithic membranes* are structures that are similar to the cupulae but they are also weighted. They contain calcium carbonate (limestone) crystals called *otoconia* and have substantially more mass than the cupulae (Fig. 1.5). The mass of the otolithic membrane causes the maculae to be sensitive to gravity and linear acceleration. In contrast, the cupulae normally have the same density as the surrounding endolymphatic fluid and are insensitive to gravity.⁴

Vascular Supply

The labyrinthine artery supplies the peripheral vestibular system (Fig. 1.6; see also Fig. 1.11). The labyrinthine artery has a variable origin. Most often it is a branch of the anterior-inferior cerebellar artery (AICA), but occasionally it is a direct branch of the basilar artery. Upon entering the inner ear, the labyrinthine artery divides into the anterior vestibular artery and the com-

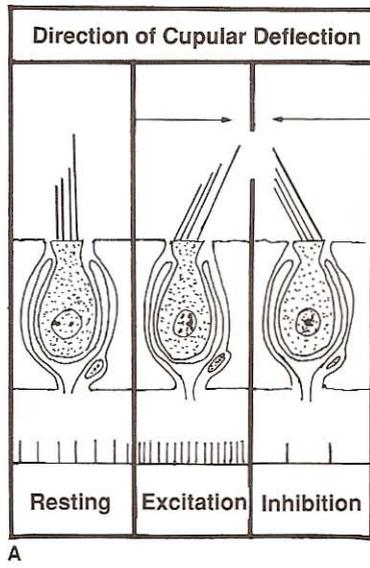
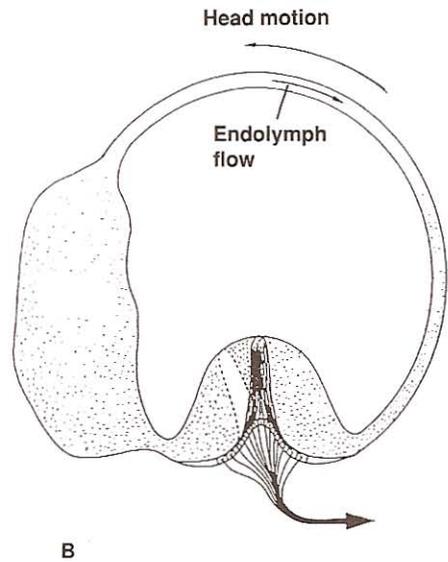


Figure 1.4 Effects of head rotation on the canals. (A) The direction from which hair cells are deflected determines whether or not hair-cell discharge frequency increases or decreases. (B) Cross section of the membranous labyrinth illustrating endolymphatic flow and cupular deflection in response to head motion. (From Bach-Y-Rita et al, 1971.³)



mon cochlear artery. The anterior vestibular artery supplies the vestibular nerve, most of the utricle, and the ampullae of the lateral and anterior SCCs. The common cochlear artery divides into a main branch, the main cochlear artery, and the vestibulocochlear artery. The main cochlear artery supplies the cochlea. The vestibulocochlear artery supplies part of the cochlea, the ampulla of the posterior semicircular canal, and the inferior part of the saccule.⁵

The labyrinth has no collateral anastomotic network and is highly susceptible to ischemia. Only 15 seconds of selective blood flow cessation is needed to abolish auditory nerve excitability.⁶

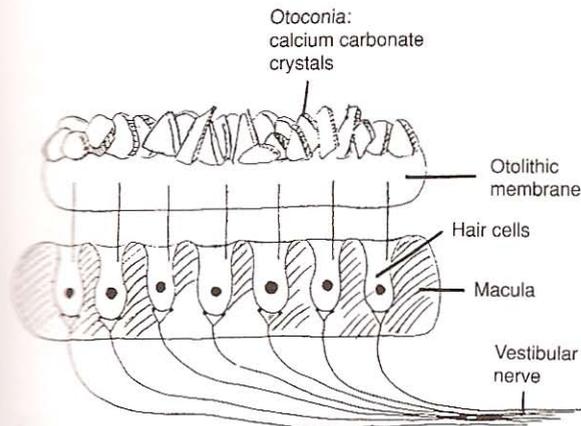


Figure 1.5 The otolithic macula and its overlying membrane. (From Baloh et al, 1990.⁴)

Physiology of the Periphery

The hair cells of the canals and otoliths convert the mechanical energy generated by head motion into neural discharges directed to specific areas of the brainstem and the cerebellum. By virtue of their orientation, the canals and otolith organs are able to respond selectively to head motion in particular directions. By virtue of differences in their fluid mechanics, the canals respond to angular velocity, and the otoliths to linear acceleration.

Semicircular Canals

The SCCs provide sensory input about head velocity, which enables the VOR to generate an eye movement that matches the velocity of the head movement. The desired result is that the eye remains still in space during head motion, enabling clear vision. Neural firing in the vestibular nerve is proportional to head velocity over the range of frequencies in which the head commonly moves (0.5–7 Hz). In engineering terms, the canals are “rate sensors.”

A second important dynamic characteristic of the canals has to do with their response to prolonged rotation at constant velocity. Instead of producing a signal proportional to velocity, as a perfect rate sensor should, the canals respond reasonably well only in the first second or so, because output decays exponentially with a time constant of about 7 seconds. This behavior is due to a spring-like action of the cupula that tends to restore it to its resting position.⁷

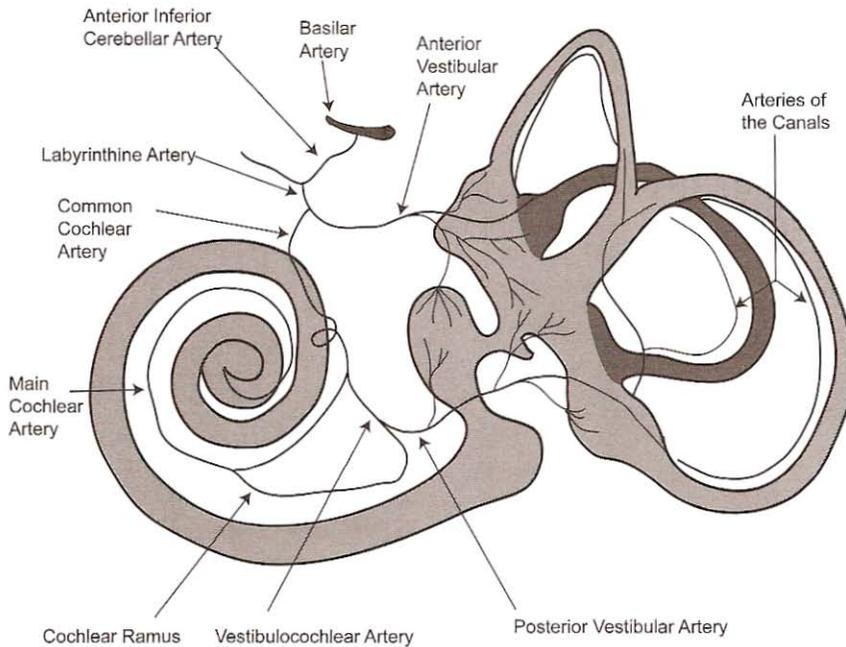


Figure 1.6 The arterial supply of the labyrinth. (Redrawn from Schuknecht, 1993.⁵)

Three important spatial arrangements characterize the alignment of the SCC's loops. First, each canal plane within each labyrinth is perpendicular to the other canal planes, analogous to the spatial relationship between two walls and the floor of a rectangular room (Fig. 1.7). Second, paired planes of the SCCs between the labyrinths conform very closely to each other. The six individual SCCs become the following three coplanar pairs: (1) right and left lateral, (2) left anterior and right posterior, and (3) left posterior and right anterior. Third, the planes of the canals are close to the planes of the extraocular muscles, thus allowing relatively simple connections between sensory neurons (related to individual canals), and motor output neurons (related to individual ocular muscles).

The coplanar pairing of canals is associated with a push-pull change in the quantity of SCC output. When angular head motion occurs within their shared plane, the endolymph of the coplanar pair is displaced in opposite directions with respect to their ampullae, and neural firing increases in one vestibular nerve and decreases on the other side. For the lateral canals, displacement of the cupula towards the ampulla (ampullopetal flow) is excitatory.

There are three advantages to the push-pull arrangement of coplanar pairing. First, pairing provides *sensory redundancy*. If disease or surgical intervention affects the SCC input from one member of a pair (e.g., as in vestibular neuritis, or canal plugging for benign paroxysmal positional vertigo), the central nervous system will

still receive vestibular information about head velocity within that plane from the contralateral member of the coplanar pair.

Second, such a pairing allows the brain to ignore changes in neural firing that occur on both sides simultaneously, such as might occur due to changes in body temperature or chemistry. These changes are not related to

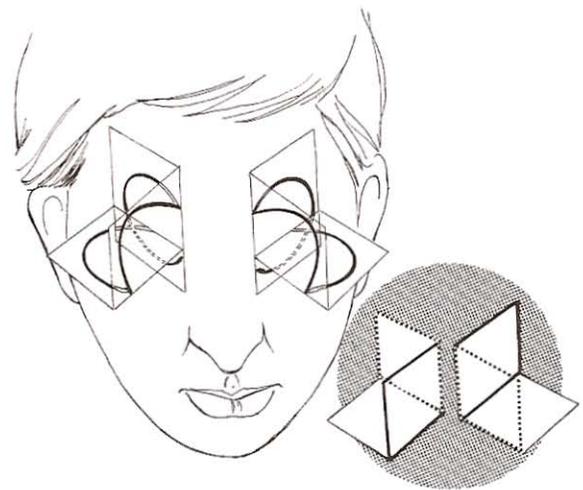


Figure 1.7 The spatial arrangement of the semicircular canals. The canals on each side are mutually perpendicular, are paired with conjugate canals on the opposite side of the head, and also are closely aligned with the optimal pulling directions of the extraocular muscles.

head motion and are “common-mode noise.” The engineering term for this desirable characteristic is “common-mode rejection.” Third, as discussed in a later section, a push-pull configuration assists in compensation for sensory overload.

Otoliths

The otoliths register forces related to linear acceleration (Fig. 1.8). They respond to both linear head motion and static tilt with respect to the gravitational axis. The function of the otoliths is illustrated by the situation of a passenger in a commercial jet. During flight at a constant velocity, he has no sense that he is traveling at 300 miles per hour. However, in the process of taking off and ascending to cruising altitude, he senses the change in velocity (acceleration) as well as the tilt of the plane on ascent. The otoliths therefore differ from the SCCs in two basic ways: They respond to linear motion instead of angular motion, and to acceleration rather than to velocity.⁷

The otoliths have a simpler task to perform than the canals. Unlike the canals, which must convert head velocity into displacement to properly activate the hair cells of

the cristae, the otoliths need no special hydrodynamic system. Exquisite sensitivity to gravity and linear acceleration is obtained by incorporation of the mass of the otoconia into the otolithic membrane (see Fig. 1.5). Force is equal to mass multiplied by acceleration, so with incorporation of a large mass, a given acceleration produces enough shearing force to make the otoliths extremely sensitive (*shearing force* refers to force that is directed perpendicularly to the processes of the hair cells).

Like the canals, the otoliths are arranged to enable them to respond to motion in all three dimensions (Fig. 1.9). However, unlike the canals, which have one sensory organ per axis of angular motion, the otoliths have only two sensory organs for three axes of linear motion. In an upright individual, the saccule is vertical (parasagittal), whereas the utricle is horizontally oriented (near the plane of the lateral SCCs). In this posture, the saccule can sense linear acceleration in its plane, which includes the acceleration oriented along the occipitocaudal axis as well as linear motion along the anterior-posterior axis. The utricle senses acceleration in its plane, which includes lateral accelerations along the interaural axis as well as anterior-posterior motion.⁸

The earth’s gravitational field is a linear acceleration field, so in a person on the earth, the otoliths register tilt. For example, as the head is tilted laterally (which is also called *roll*; see Fig. 1.8), shear force is exerted upon the utricle, causing excitation, but shear force is lessened upon the saccule. Similar changes occur when the head is tilted forwards or backwards (called *pitch*). Because linear acceleration can come from two sources—earth’s gravitational field as well as linear motion—there is a sensor ambiguity problem. We discuss strategies that the central nervous system might use to solve this problem later, in the section on higher-level vestibular processing.

In the otoliths, as in the canals, there is redundancy, with similar sensors on both sides of the head. Push-pull processing for the otoliths is also incorporated into the geometry of each of the otolithic membranes. Within each otolithic macula, a curving zone, the *striola*, separates the direction of hair-cell polarization on each side. Consequently, head tilt increases afferent discharge from one part of a macula while reducing the afferent discharge from another portion of the same macula. This extra level of redundancy in comparison with the SCCs probably makes the otoliths less vulnerable to unilateral vestibular lesions.

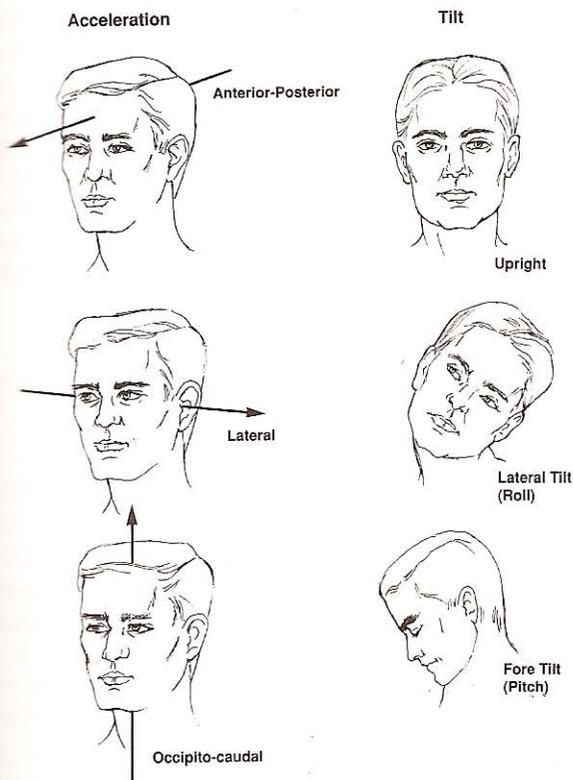


Figure 1.8 The otoliths register linear acceleration and static tilt.

The Vestibular Nerve

Vestibular nerve fibers are the afferent projections from the bipolar neurons of Scarpa’s (vestibular) ganglion. The

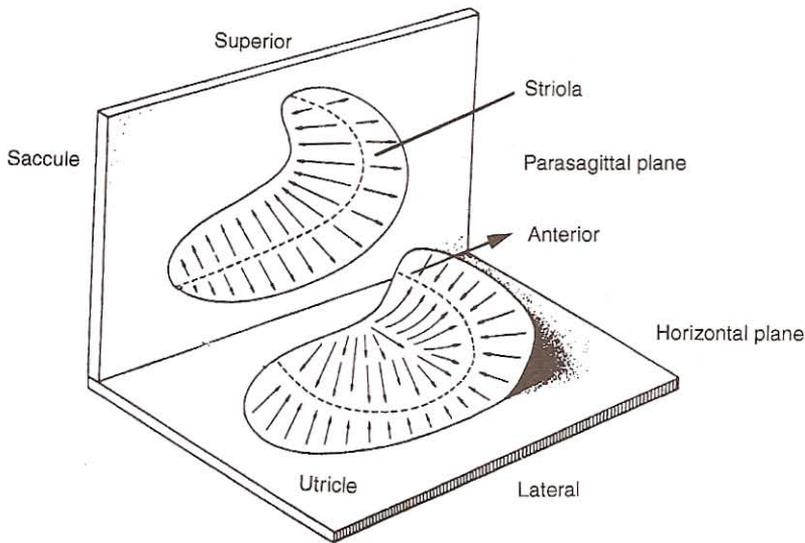


Figure 1.9 Geometry of the otoliths. (From Barber and Stockwell, 1976.)^[8]

vestibular nerve transmits afferent signals from the labyrinths along its course through the *internal auditory canal* (IAC). In addition to the vestibular nerve, the IAC contains the cochlear nerve (hearing), the facial nerve, the nervus intermedius (a branch of the facial nerve, which carries sensation), and the labyrinthine artery. The IAC travels through the petrous portion of the temporal bone to open into the posterior fossa at the level of the pons. The vestibular nerve enters the brainstem at the pontomedullary junction. Because the vestibular nerve is interposed between the labyrinth and the brainstem, some authorities consider this nerve a peripheral structure, whereas others consider it a central structure. We consider it a peripheral structure.

There are two patterns of firing in vestibular afferent neurons. Regular afferents usually have a tonic rate and little variability in interspike intervals. Irregular afferents often show no firing at rest and, when stimulated by head motion, develop highly variable interspike intervals.⁹ Regular afferents appear to be the most important type for the VOR, because in experimental animals irregular afferents can be ablated without much change in the VOR. However, irregular afferents may be important for the VSR and in coordinating responses between the otoliths and canals.

Regular afferents of the monkey have tonic firing rates of about 90 spikes per second and a sensitivity to head velocity of about 0.5 spike per degree per second.^{10,11} We can speculate about what happens immediately after a sudden change in head velocity. Humans can easily move their heads at velocities exceeding 300 degrees per second (deg/sec). As noted previously, the

SCCS are connected in a push-pull arrangement, so that one side is always being inhibited while the other is being excited. Given the sensitivity and tonic rate noted previously, the vestibular nerve, which is being inhibited, should be driven to a firing rate of 0 spikes per second, for head velocities of only 180 deg/sec! In other words, head velocities greater than 180 deg/sec may be unquantifiable by half of the vestibular system. This *cutoff behavior* has been advanced as the explanation for Ewald's second law, which says that responses to rotations that excite a canal are greater than those to rotations that inhibit a canal.^{12,13} Cutoff behavior explains why a patient with unilateral vestibular loss avoids head motion toward the side of the lesion. More is said about this issue in the later discussion of how the central nervous system may compensate for overload.

Central Processing of Vestibular Input

There are two main targets for vestibular input from primary afferents: the vestibular nuclear complex and the cerebellum (see Fig. 1.1). The vestibular nuclear complex is the primary processor of vestibular input and implements direct, fast connections between incoming afferent information and motor output neurons. The cerebellum is the adaptive processor; it monitors vestibular performance and readjusts central vestibular processing if necessary. At both locations, vestibular sensory input is processed in association with somatosensory and visual sensory input.

Vestibular Nucleus

The vestibular nuclear complex consists of four “major” nuclei (superior, medial, lateral, and descending) and at least seven “minor” nuclei (Fig. 1.10). This large structure, located primarily within the pons, also extends caudally into the medulla. The superior and medial vestibular nuclei are relays for the VOR. The medial vestibular nucleus is also involved in VSRs and coordinates head and eye movements that occur together. The lateral vestibular nucleus is the principal nucleus for the VSR. The descending nucleus is connected to all of the other nuclei and the cerebellum but has no primary outflow of its own. The vestibular nuclei between the two sides of the brainstem are laced together via a system of commissures that are mutually inhibitory. The commissures allow information to be shared between the two sides of the brainstem and implement the push-pull pairing of canals discussed earlier.¹⁴

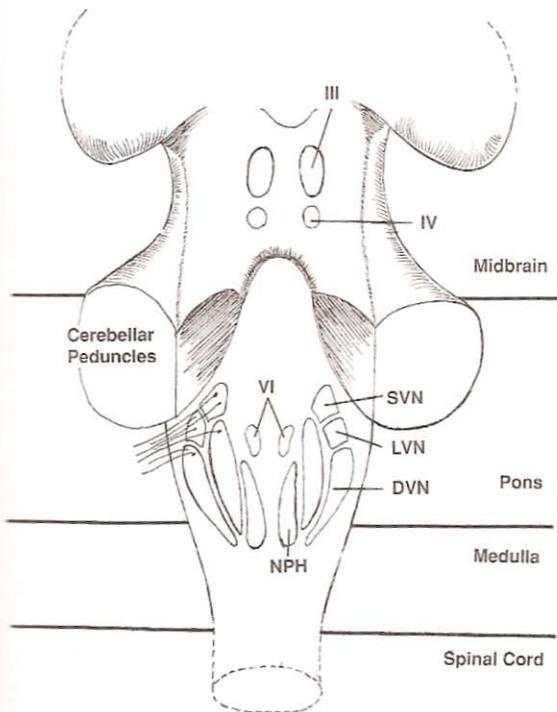


Figure 1.10 The vestibular nuclear complex. This section shows the brainstem with the cerebellum removed. DVN = descending vestibular nucleus; LVN = lateral vestibular nucleus; NPH = nucleus prepositus hypoglossi; III = oculomotor nucleus (inferior oblique muscle and medial, superior, and inferior rectus muscles); IV = trochlear nucleus (superior oblique muscle); VI = abducens nucleus (lateral rectus muscle). The medial vestibular nucleus (not labeled) lies between the NPH and the DVN. (From Brodal, 1981.¹⁴)

In the vestibular nuclear complex, processing of the vestibular sensory input occurs concurrently with the processing of extravestibular sensory information (proprioceptive, visual, tactile, and auditory). Extensive connections between the vestibular nuclear complex, cerebellum, ocular motor nuclei, and brainstem reticular activating systems are required to formulate appropriate efferent signals to the VOR and VSR effector organs, the extraocular and skeletal muscles.

Vascular Supply

The vertebral-basilar arterial system supplies blood to the peripheral and central vestibular system (Fig. 1.11). The

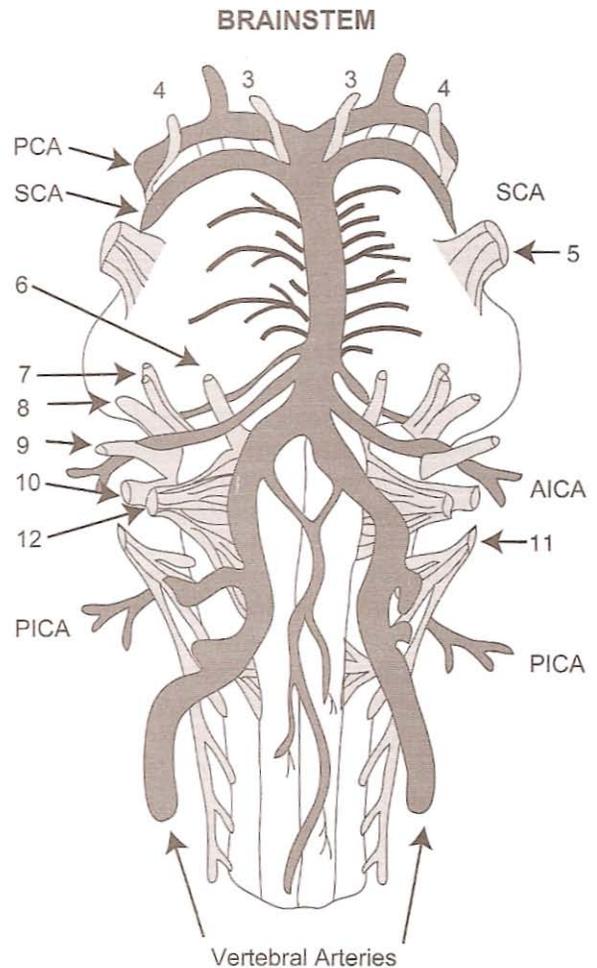


Figure 1.11 The vertebral-basilar system. AICA = anterior inferior cerebellar artery; PCA = posterior cerebellar artery; PICA = posterior inferior cerebellar artery; SCA = superior cerebellar artery. Numerals indicate individual cranial nerve roots (all nerves are paired, but for clarity, both sides are not always labeled here). (© Northwestern University, with permission.)

posterior-inferior cerebellar arteries (PICAs) branch off the vertebral arteries. The two PICAs are the most important arteries for the central vestibular system. They supply the surface of the inferior portions of the cerebellar hemispheres as well as the dorsolateral medulla, which includes the inferior aspects of the vestibular nuclear complex. The basilar artery is the principal artery of the pons. The basilar artery supplies central vestibular structures via perforator branches, which penetrate the medial pons, short circumferential branches, which supply the anterolateral aspect of the pons, and long circumferential branches, which supply the dorsolateral pons. The AICA is an important branch of the basilar artery because it is the sole blood supply for the peripheral vestibular system via the labyrinthine artery. The AICA also supplies blood to the ventrolateral cerebellum and the lateral tegmentum of the lower two-thirds of the pons. Recognizable clinical syndromes with vestibular components may appear after occlusions of the basilar artery, labyrinthine artery, AICA, and PICA.

Cerebellum

The cerebellum, a major recipient of outflow from the vestibular nucleus complex, is also a major source of input itself. Although the cerebellum is not required for vestibular reflexes, vestibular reflexes become uncalibrated and ineffective when this structure is removed. Originally, the “vestibulocerebellum” was defined as the portions of the cerebellum receiving direct input from the primary vestibular afferents. We now understand that most parts of the *cerebellar vermis* (midline) respond to vestibular stimulation. The cerebellar projections to the vestibular nuclear complex have an inhibitory influence on the vestibular nuclear complex.

The *cerebellar flocculus* adjusts and maintains the gain of the VOR.¹⁵ Lesions of the flocculus reduce the ability of experimental animals to adapt to disorders that reduce or increase the gain of the VOR. Patients with cerebellar degenerations or the Arnold-Chiari malformation typically have floccular disorders.

The *cerebellar nodulus* adjusts the duration of VOR responses and is also involved with processing of otolith input. Patients with lesions of the cerebellar nodulus, such as those with medulloblastoma, show gait ataxia and often have nystagmus, which is strongly affected by the position of the head with respect to the gravitational axis.

Lesions of the anterior-superior vermis of the cerebellum affect the VSR and cause a profound gait ataxia with truncal instability. Patients with such lesions are unable to use sensory input from their lower extremities

to stabilize their posture. The lesions are commonly related to excessive alcohol intake and thiamine deficiency.

Neural Integrator

Thus far we have discussed processing of velocity signals from the canals and acceleration signals from the otoliths. These signals are not suitable for driving the ocular motor neurons, which need a neural signal encoding eye position. The transformation of velocity to position is accomplished by a brainstem structure called the *neural integrator*. The nucleus prepositus hypoglossi, located just below the medial vestibular nucleus, appears to provide this function for the horizontal oculomotor system.¹⁵ Although a similar structure must exist for the vestibulospinal system, the location of the VSR neural integrator is currently unknown. Clinically, poor function of the oculomotor neural integrator causes gaze-evoked nystagmus.

Motor Output of the Vestibular System Neurons

Output for the Vestibulo-ocular Reflex

The output neurons of the VOR are the motor neurons of the ocular motor nuclei, which drive the extraocular muscles. The extraocular muscles are arranged in pairs, which are oriented in planes very close to those of the canals. This geometrical arrangement enables a single pair of canals to be connected predominantly to a single pair of extraocular muscles. The result is conjugate movements of the eyes in the same plane as head motion.

Two white matter tracts carry output from the vestibular nuclear complex to the ocular motor nuclei. The *ascending tract of Deiters* carries output from the vestibular nucleus to the ipsilateral abducens nucleus (lateral rectus) during the horizontal VOR. All other VOR-related output to the ocular motor nuclei is transmitted by the *medial longitudinal fasciculus* (MLF) (Fig. 1.12). Because the median longitudinal fasciculus is often injured in multiple sclerosis, this connection may account for central vestibular symptoms in patients with this disorder.¹⁴

Output for the Vestibulospinal Reflex

The output neurons of the VSR are the anterior horn cells of the spinal cord gray matter, which drive skeletal muscle. However, the connection between the vestibular nuclear complex and the motor neurons is more compli-

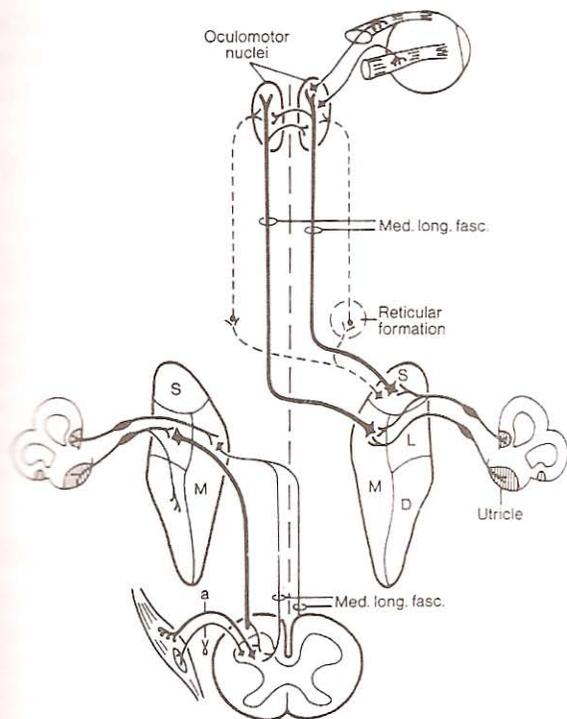


Figure 1.12 The vestibulo-ocular reflex (VOR) and vestibulospinal reflex (VSR) arcs. S, L, M, and D indicate the superior, lateral, medial, and descending vestibular nuclei, respectively. The lateral vestibulospinal and medial vestibulospinal tracts are shown as heavy lines and light lines, beginning in the lateral vestibular nucleus and medial vestibular nucleus, respectively. (From Brodal, 1981.¹⁴)

ated than that for the VOR. The VSR has a much more difficult task than the VOR, because multiple strategies involving entirely different motor synergies can be used to prevent falls. For example, when one is shoved from behind, one's center of gravity might become displaced anteriorly. In order to restore "balance," one might (1) plantar-flex at the ankles, (2) take a step, (3) grab for support, or (4) use some combination of all three activities. The VSR also has to adjust limb motion appropriately for the position of the head on the body (see the frame of reference problem discussed later in the section on higher-level problems in vestibular processing).

The VSR must also use otolith input, reflecting linear motion, to a greater extent than the VOR. Although the eyes can only rotate and thus can do little to compensate for linear motion, the body can both rotate and translate.

Three major white matter pathways connect the vestibular nucleus to the anterior horn cells of the spinal cord. The lateral vestibulospinal tract originates from the ipsilateral lateral vestibular nucleus, which receives the

majority of its input from the otoliths and the cerebellum (see Fig. 1.12). This pathway generates antigravity postural motor activity or protective extension, primarily in the lower extremities, in response to the head position changes that occur with respect to gravity. The medial vestibulospinal tract originates from the contralateral medial, superior, and descending vestibular nuclei (see Fig. 1.12) and mediates ongoing postural changes or head righting in response to SCC sensory input (angular head motion). The medial vestibulospinal tract descends only through the cervical spinal cord in the medial longitudinal fasciculus and activates cervical axial musculature.

The reticulospinal tract receives sensory input from all of the vestibular nuclei as well as from all of the other sensory and motor systems involved with maintaining balance. This projection has both crossed and uncrossed components and is very highly collateralized. As a result, the reticulospinal tract through the entire extent of the spinal cord is poorly defined, but it is probably involved in most balance reflex motor actions, including postural adjustments made to extravestibular sensory input (auditory, visual, and tactile stimuli).

Vestibular Reflexes

The sensory, central, and motor output components of the vestibular system have been described. We now discuss their integration into the VOR, VSR, and VCR. Additionally, we include brief descriptions of cervical, visual, and somatosensory reflexes. Although not directly mediated by the vestibular apparatus, these reflexes have a close interaction with vestibular reflexes.

The Vestibulo-ocular Reflex

The VOR normally acts to maintain stable vision during head motion. This reflex has two components. The angular VOR, mediated by the SCCs, compensates for rotation. The linear VOR, mediated by the otoliths, compensates for translation. The angular VOR is primarily responsible for gaze stabilization. The linear VOR is most important when near targets are being viewed and the head is being moved at relatively high frequencies. An example of how the horizontal canal VOR is orchestrated follows:

1. When the head turns to the right, endolymphatic flow deflects the cupulae to the left (see Fig. 1.4B).
2. The discharge rate from hair cells in the right crista increases in proportion to the velocity

of the head motion, whereas the discharge rate from hair cells in the left lateral crista decreases (see Fig. 1.4A).

3. These changes in firing rate are transmitted along the vestibular nerve and influence the discharge of the neurons of the medial and superior vestibular nuclei and cerebellum.
4. Excitatory impulses are transmitted via white matter tracts in the brainstem to the oculomotor nuclei, which activate the right (ipsilateral) medial rectus and the left (contralateral) lateral rectus. Inhibitory impulses are also transmitted to their antagonists.
5. Simultaneously, contraction of the left lateral rectus and right medial rectus muscles and relaxation of the left medial rectus and right lateral rectus muscles occur, resulting in lateral compensatory eye movements toward the left.
6. If the eye velocity is not adequate for the given head velocity and retina image motion is more than 2 deg/sec, the cerebellar projection to the vestibular nuclei will modify the firing rate of the neurons within the vestibular nuclei to reduce the error.

The Vestibulospinal Reflex

The purpose of the VSR is to stabilize the body. The VSR actually consists of an assemblage of several reflexes named according to the timing (dynamic vs. static or tonic) and sensory input (canal vs. otolith); these reflexes are discussed in more detail in Chapter 2. As an example of a VSR, let us examine the sequence of events involved in generating a labyrinthine reflex, as follows:

1. When the head is tilted to one side, both the canals and the otoliths are stimulated. Endolymphatic flow deflects the cupula, and shear force deflects hair cells within the otoliths.
2. The vestibular nerve and vestibular nucleus are activated.
3. Impulses are transmitted via the lateral and medial vestibulospinal tracts to the spinal cord.
4. Extensor activity is induced on the side to which the head is inclined, and flexor activity is induced on the opposite side. The head movement opposes the movement sensed by the motion sensors.

The Vestibulocollic Reflex

The VCR acts on the neck musculature to stabilize the head. The reflex head movement produced counters the

movement sensed by the otolithic or SCC organs. The precise pathways mediating this reflex have yet to be detailed.

Cervical Reflexes

The Cervico-ocular Reflex

The *cervico-ocular reflex* (COR) interacts with the VOR. The COR consists of eye movements driven by neck proprioceptors that can supplement the VOR under certain circumstances. Normally, the gain of the COR is very low.¹⁶ The COR is facilitated when the vestibular apparatus is injured.^{17,18} It is rare, however, for the COR to have any clinical significance.

The Cervicospinal Reflex

The *cervicospinal reflex* (CSR) is defined as changes in limb position driven by neck afferent activity. Analogous to the COR, which supplements the VOR under certain circumstances, the CSR can supplement the VSR by altering motor tone in the body. Like the VSR, the CSR consists of an assemblage of several reflexes. Two pathways are thought to mediate these reflex signals: an excitatory pathway from the lateral vestibular nucleus and an inhibitory pathway from the medial part of the medullary reticular formation.

When the body is rotated with head stable, neurons of the excitatory vestibulospinal system increase their rate of firing on the side to which the chin is pointed. At the same time, neurons thought to be in the inhibitory reticulospinal system show a reduced rate of firing. This activity leads to extension of the limb on the side to which the chin is pointed and flexion of the limb on the contralateral side. Vestibular receptors influence both of these systems by modulating the firing of medullary neurons in a pattern opposite to that elicited by neck receptors. With their interaction, the effects on the body of vestibular and neck inputs tend to cancel one another when the head moves freely on the body, so that posture remains stable.¹⁹

The Cervicocollic Reflex

The *cervicocollic reflex* (CCR) is a cervical reflex that stabilizes the head on the body. The afferent sensory changes caused by changes in neck position create opposition to that stretch by way of reflexive contractions of appropriate neck muscles. The reflex is thought to be primarily a monosynaptic one.¹⁶ The extent to which the CCR contributes to head stabilization in normal humans is cur-

remains uncertain, but it seems likely that the CCR is useful primarily to stabilize head movement in the vertical plane, and it may also be facilitated after labyrinthine loss.

Visual Reflexes

The visual system is a capable and sophisticated sensory system that influences vestibular central circuitry and drives visual after-responses (i.e., smooth pursuit) and postural reactions. Because of intrinsic delays in multisynaptic visual mechanisms, visual responses occur at a substantially longer latency and are much less suited to tracking at frequencies above about 0.5 Hz than vestibular responses. Visual tracking responses may be facilitated after vestibular loss.

Somatosensory Reflexes

Somatosensory mechanisms appear to be involved in postural stability as well. Bles and associates documented somatosensory-induced nystagmus (“stepping-around nystagmus”).²⁰ Interestingly, the subjects in their study with bilateral vestibular loss developed a more pronounced nystagmus than normal subjects. This finding implies that subjects with bilateral vestibular loss use somatosensory information to a greater extent than normal subjects.

Neurophysiology of Benign Paroxysmal Positional Vertigo

Although most vestibular disorders can be described in terms of imbalance between the ears or loss of function, *benign paroxysmal positional vertigo* (BPPV) has an entirely different mechanism. BPPV is caused by movement of detached otoconia within the inner ear (*canalithiasis*) or otoconia adherent to the cupula (*cupulolithiasis*) (Fig. 1.13). Great progress has now been made in our understanding of BPPV.

Figure 1.14, from Squires and colleagues,²¹ illustrates the fluid mechanics of BPPV. In this disorder, vertigo and nystagmus begin after a characteristic latency of about 5 seconds. The delay in onset of symptoms is caused by movement of detached otoconia through the ampulla, because pressure caused by moving otoconia is negligible until otoconia enter the narrow duct of the SCC. Figure 1.14 also shows that particle-wall interactions can account for variability in duration and latency of BPPV.²¹

Other results from fluid mechanics have direct bearing on our understanding of treatment maneuvers for BPPV. Under the influence of a full 1 g of gravity, typical otoconia move at a rate of 0.2 mm/sec, or only about 1% of the circumference of the canal each second. It follows that inertial effects of treatment maneuvers can

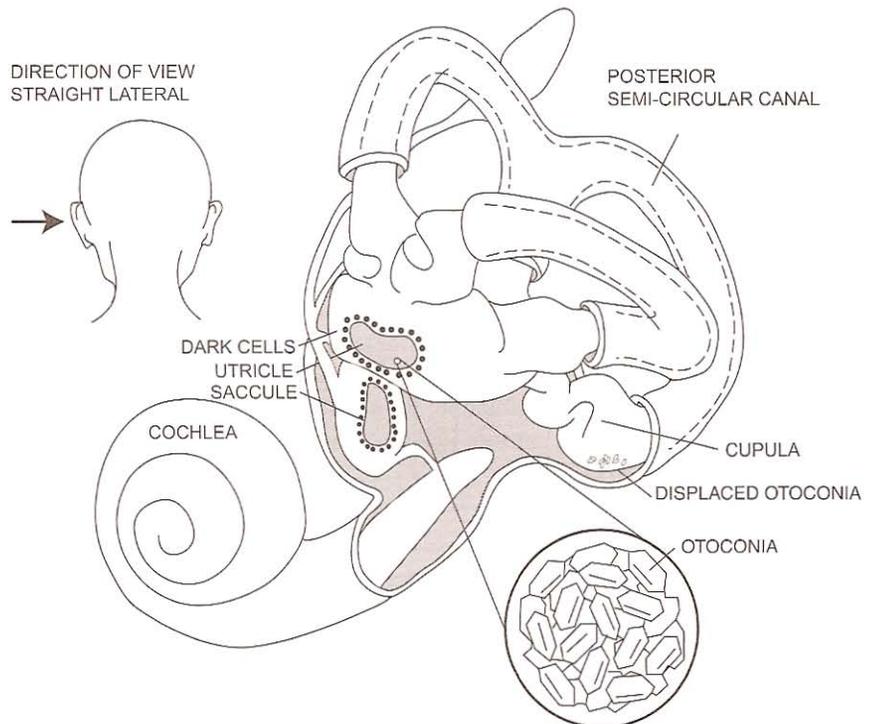


Figure 1.13 Physiology of benign paroxysmal positional vertigo. Otoconia become displaced from the utricle and relocate to the bottom of the posterior semicircular canal, which is the lowest part of the inner ear. (© Northwestern University, with permission.)

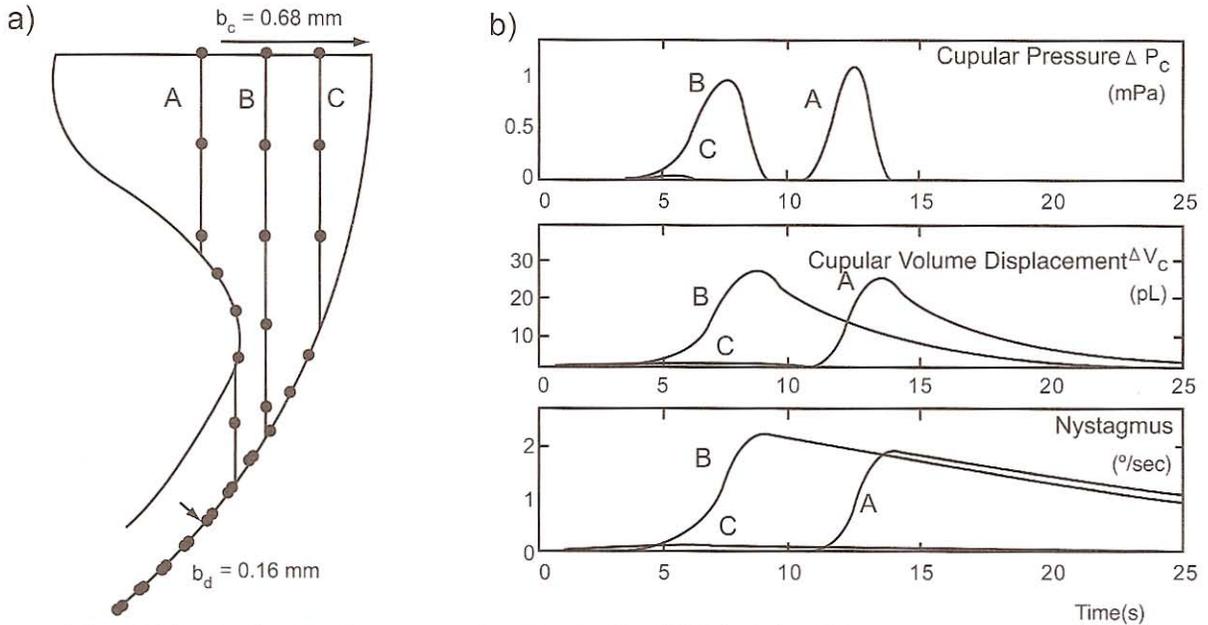


Figure 1.14 Fluid mechanics of benign paroxysmal positional vertigo. (A) Trajectories of three otoconia after a sudden change of head position that makes the posterior canal vertical. Otoconia begin close to the cupula, fall through the ampulla with radius b_c , and then enter the duct with radius b_d . (B) Simulated pressure, displacement, and nystagmus due to otoconia falling with the trajectories of A. (From Squires et al, 2004.)²¹

cause negligible movement of otoconia and that, practically, sudden jerks of the head or maneuvers that incorporate eccentric moments (such as the Semont maneuver) are unlikely to have a substantial additional effect in comparison with maneuvers that rely on gravity to accomplish canalith repositioning.²²

Higher-Level Vestibular Processing

In this section we identify some of the more sophisticated aspects of central vestibular processing, which are not reflexes but rather require much more processing, are generally much more accurate, and often are at least partially under conscious control. Because these mechanisms are more modifiable than vestibular reflexes, they are especially relevant to rehabilitation. Most of these mechanisms process multiple sensory inputs.

Velocity Storage

How good does the VOR have to be? In order to keep the eye still in space while the head is moving, the velocity of the eyes should be exactly opposite to that of head movement. When this happens, the ratio of eye move-

ment to head movement velocity, called the *gain*, equals -1.0 . In order to maintain normal vision, retinal image motion must be less than 2 deg/sec . In other words, for a head velocity of 100 deg/sec , which is easily produced by an ordinary head movement, the gain of the VOR must be 98% accurate, because any greater error would cause vision to be obscured.

The normal VOR can deliver this high standard of performance only for brief head movements. In other words, the VOR is compensatory for high-frequency head motion but not for low-frequency head motion. This fact can be most easily seen if one considers the response of the SCCs to a sustained head movement, which has a constant velocity. The canals respond by producing an exponentially decaying change in neural firing in the vestibular nerve. The *time constant* of the exponential is about 7 seconds; in other words, the firing rate decays to 32% of the initial amount in 7 seconds. Ideally, the time constant should be infinite, which would be associated with no response decline. Apparently, a time constant of 7 seconds is not long enough, because the central nervous system goes to the trouble to persevere the response, replacing the peripheral time constant of 7 seconds with a central time constant of about 20 seconds.

The perseveration is provided via a brainstem structure called the *velocity storage mechanism*.²³

The velocity storage mechanism is used as a repository for information about head velocity derived from several kinds of motion sensors. During rotation in the light, the vestibular nucleus is supplied with retinal slip information. *Retinal slip* is the difference between eye velocity and head velocity. Retinal slip can drive the velocity storage mechanism and keep vestibular-related responses going even after vestibular afferent information decays. The vestibular system also uses somatosensory and otolith information to drive the velocity storage mechanism.²⁴ The example discussed here shows how the vestibular system resolves multiple, partially redundant sensory inputs.

Estimation: Going Beyond Reflexes

Reflexes are by definition simple sensory processors that rapidly convert sensory input into motor outflow. What happens when sensory input is not available (such as when the eyes are closed) or inaccurate (such as when a person with positional vertigo tilts the head), or noisy (such as when a sensor has been damaged)? A mechanism that combines sensory inputs, weights them according to their relevance and reliability, and provides a

reasonable estimate of orientation in space, even without any recent sensory input, is needed. In engineering terms, we are discussing an “estimator.”

Navigating the space shuttle involves similar problems. The shuttle has dozens of sensors and motors. Some sensors respond quickly, and some slowly. They may differ in accuracy, scaling, coordinate frame, timing, and noise characteristics. No single sensor can provide a complete picture of the shuttle’s state. A mechanism is needed to integrate sensor output and to develop an internal estimate of the state of the system (i.e., position, velocity, acceleration) in order to keep the shuttle on the desired course and heading.

The engineering solution to this problem developed out of work performed by Kalman and is often called a *Kalman filter*. It is also commonly called an “optimal estimator” or an “internal model.” The essentials of the Kalman filter are shown in Figure 1.15. There is considerable evidence that mechanisms similar to Kalman filters are used for human sensorimotor processing.²⁵

The Kalman filter is far more powerful than a simple reflex. Several key concepts must be considered before one can understand how it is superior. First, internal models of sensors and motor output are used to develop an estimate of the current sensory and motor state. These internal models are adjusted according to experience and

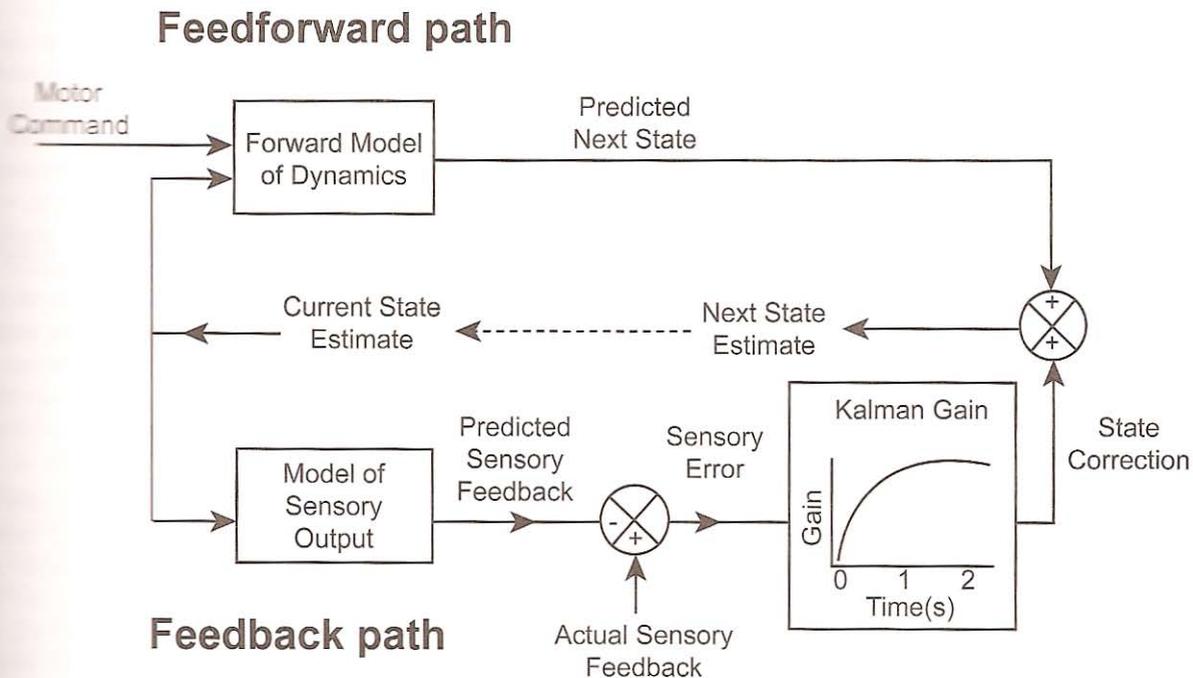


Figure 1.15 Block diagram showing a Kalman filter such as may be used by the body for sensorimotor integration. Sensory inflow and motor outflow are used to estimate the current state. (From Wolpert and Miall, 1997)^[25]

must track changes in bodily function. It seems likely that vestibular rehabilitation affects internal models.

Second, sensory input is not used to directly compute body state, but rather, the difference between sensory input and predicted sensory input is used to correct the current estimate of body state. This design allows the Kalman filter to easily combine multiple sensor inputs—from eyes, ears, and somatosensors. The Kalman filter continues to work even in the absence of a sensory input, because it uses its estimate when the sensor is missing. Both of these highly desirable features make the Kalman filter far superior to a simple assemblage of reflexes.

The *Kalman gain* weights the extent to which a sensory input affects the ongoing state estimate. This weighting provides a method of adjusting for the salience and reliability of sensory streams. It seems highly likely that vestibular rehabilitation adjusts the Kalman gain.

Overall, this sort of mechanism is clearly far superior to vestibular reflexes: Although not as fast, it can be far more accurate, it functions even in the absence of sensory input, and it is modifiable by experience and rehabilitation.

Higher-Level Problems of the Vestibular System

Compensation for Overload

Humans can easily move their heads at velocities exceeding 300 deg/sec. Consider, for example, driving a car. When one hears a horn to the side, one's head may rapidly rotate to visualize the problem and, potentially, avoid an impending collision. Similarly, during certain sports (e.g., racquetball), head velocity and acceleration reach high levels. One must be able to see during these sorts of activities, but the vestibular nerve is not well suited to transmission of high-velocity signals. The reason is the cutoff behavior discussed in the earlier section on motor output of the vestibular system. High-velocity head movement may cause the nerve on the inhibited side to be driven to a firing rate of 0.

In this instance, the vestibular system must depend on the excited side, which is arranged in “push-pull” configuration with the inhibited side. Whereas the inhibited side can be driven only to 0 spikes per second, the side being excited can be driven to much higher levels. Thus, the push-pull arrangement takes care of part of the overload problem. Note, however, that in patients with unilateral vestibular loss, this mechanism is not available to deal with the overload problem, and they are commonly disturbed by rapid head motion toward the side of the lesion.

Sensor Ambiguity

Sensory input from the otoliths is intrinsically ambiguous, because the same pattern of otolith activation can be produced by either a linear acceleration or a tilt. In other words, in the absence of other information, we have no method of deciding whether we are being whisked off along an axis or whether the whole room just tilted. Canal information may not be that useful in resolving the ambiguity, because one might be rotating *and* tilting at the same time. These sorts of problems are graphically demonstrated in subway cars and airplanes, which can both tilt and/or translate briskly.

Outside of moving vehicles, vision and tactile sensation can be used to decide what is happening, perhaps through the use of a Kalman filter as discussed previously. As long as one does not have to make a quick decision, these senses may be perfectly adequate. However, visual input takes 80 msec to get to the vestibular nucleus and tactile input must be considered in the context of joint position and of the intrinsic neural transmission delays between the point of contact and the vestibular nuclear complex.

Another strategy that the brain can use to separate tilt from linear acceleration is *filtering*. In most instances, tilts are prolonged but linear accelerations are brief. Neural filters that pass low or high frequencies can be used to tell one from the other.

Nevertheless, in humans, evolution apparently has decided that the ambiguity problem is not worth solving. Otolith-ocular reflexes appropriate to compensate for linear acceleration or tilt do exist in darkness but are extremely weak in normal humans.²⁶ Stronger otolith-ocular reflexes are generally seen only in the light, when vision is available to solve the ambiguity problem. Sensory ambiguity becomes most problematic for patients who have multiple sensory deficits, because they cannot use other senses to formulate appropriate vestibulospinal responses.

Motion Sickness

The phenomenon of motion sickness illustrates how the brain routinely processes multiple channels of sensory information simultaneously. The *motion sickness syndrome* consists of dizziness, nausea or emesis, and malaise after motion. It is thought to be caused by a conflict between movement information in related sensory channels, such as visual-vestibular conflict or conflict between an actual and an anticipated sensory input. For example, motion sickness is often triggered by reading a

think while riding in a car. In this instance, the vestibular and proprioceptive systems signal movement, but the visual system signals relative stability.

The vestibular apparatus provides partially redundant information, allowing for the possibility of intralabyrinthine conflict. Space motion sickness is thought to be caused by intralabyrinthine conflict. About 50% of space shuttle astronauts experience motion sickness during the initial 24 to 72 hours of orbital flight. It is currently thought that space motion sickness is due to a disturbance in "otolith-tilt translation."²⁷ The otoliths normally function in the context of a gravitational field, so that at any moment the total force acting on the otoliths is the vector sum of the force due to gravity and that due to linear acceleration of the head. The central nervous system expects linear acceleration to be mainly related to tilt, because linear acceleration due to gravity is usually much greater than that due to acceleration of the head. When one is outside earth's gravitational field, like astronauts in outer space, the only source of otolith stimulation is linear acceleration of the head. In susceptible individuals, the central nervous system continues to interpret linear acceleration as being primarily related to tilt, which is now untrue, causing the motion sickness syndrome.^{27,28}

Structures that appear to be essential for the production of motion sickness are (1) intact labyrinth and central vestibular connections, (2) cerebellar nodulus and uvula that coordinate labyrinthine stimuli, (3) the chemoreceptive trigger zone located in the area postrema, and (4) the medullary vomiting center.²⁹ Why some people are more prone to motion sickness than others is not completely understood.

Repair

Thus far, we have described some of the problems posed by the limitations of the vestibular sensory apparatus and the constraints of physics. In normal individuals, these problems can be satisfactorily resolved by relying on redundancy of sensory input and central signal processing. In addition to these intrinsic problems, there are extrinsic problems related to ongoing changes in sensory apparatus, central processing capabilities, and motor output channels. Because being able to see while one's head is moving and avoiding falls are so important to survival, the repair facility of the vestibular system must be regarded as an integral part of its physiology; for this reason, it is our final topic.

Adaptive plasticity for peripheral vestibular lesions is dealt with elsewhere in this volume. Suffice it to say here that repair is amazingly competent, even enabling

the vestibular system to adapt to peculiar sensory situations requiring a reversal of the VOR.³⁰ Adjustments of internal models and weighting of sensory inputs (e.g., Kalman gain) are probably at least as important as readjustment of reflexes, because internal models provide many important features that reflexes cannot provide (such as functioning in the absence of sensory input).

Although most people are capable of abstract thought and can generalize from one context to another, there is a high degree of *context dependency* to the repair of peripheral vestibular lesions. In other words, adaptations learned within one sensory context may not work within another. For example, a patient who can stabilize gaze on a target with the head upright may not be able to do so when making the same head movements from a supine posture. Experimentally, in the cat, VOR gain adaptations can be produced that depend on the orientation of the head.³¹ Similarly, when the VOR of cats is trained with the use of head movements of low frequency, no training effect is seen at high frequencies.³²

Another type of context dependency relates to the VSRs and has to do with the difference in reference frames between the head and body. Because the head can move on the body, information about how the head is moving may be rotated with respect to the body. For example, consider the situation in which the head is turned 90 degrees to the right. In this situation, the coronal plane of the head is aligned with the sagittal plane of the body, and motor synergies intended to prevent a fall for a given vestibular input must also be rotated by 90 degrees. For example, patients with vestibular impairment who undergo gait training in which all procedures are performed only in a particular head posture (such as upright) may show little improvement in natural situations in which the head assumes other postures, such as looking down at the feet. Little is understood about the physiology of context dependency.

Repair of central lesions is much more limited than that available for peripheral lesions; this is the "Achilles' heel" of the vestibular apparatus. Symptoms due to central lesions last much longer than symptoms due to peripheral vestibular problems. The reason for this vulnerability is not difficult to understand. To use a commonplace analogy, if your television breaks down you can take it to the repair shop and get it fixed. If, however, your television is broken and the repair shop is out of business, you have a much bigger problem. The cerebellum fulfills the role of the repair shop for the vestibular system. When there are cerebellar lesions or lesions in the pathways to and from the cerebellum, symptoms of vestibular dysfunction can be profound and permanent.

Clinicians use this reasoning when they attempt to separate peripheral from central vestibular lesions. A spontaneous nystagmus that persists over several weeks is generally due to a central lesion; a peripheral nystagmus can be repaired by an intact brainstem and cerebellum.

Summary

The vestibular system is an old and sophisticated human control system. Accurate processing of sensory input about rapid head and postural motion is difficult as well as critical to survival. Not surprisingly, the body uses multiple, partially redundant sensory inputs and motor outputs in combination with central state estimators and competent central repair. The system as a whole can withstand and adapt to major amounts of peripheral vestibular dysfunction. The weakness of the vestibular system is a relative inability to repair central vestibular dysfunction.

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