Neurophysiology of Vestibular Rehabilitation

Timothy C. Hain, M.D.
Professor of Otolaryngology and Physical Therapy
Northwestern University
645 N. Michigan, Suite 410
Chicago Il 60611

ABSTRACT

The vestibular system is a sophisticated human control system. Accurate processing of sensory input about rapid head and postural motion is critical. Not surprisingly, the body uses multiple, partially redundant sensory inputs and motor outputs, combined with a very competent central repair capability. The system as a whole can adapt to substantial peripheral vestibular dysfunction. The Achilles' heel of the vestibular system is a relative inability to repair central vestibular dysfunction.

INTRODUCTION:

The peripheral vestibular system senses rotational and linear motion of the head. This information is used to stabilize the eyes so as to maintain gaze on targets of interest in spite of any head movement, as well as to maintain one's desired orientation to one's environment, often an upright posture.

Figure 1 about here

The peripheral vestibular system does not act in isolation. It is an important but not completely essential part of an orientation system mainly localized in the brainstem that integrates together information from that of other senses (e.g. vision, somatosensation), motor efference, and expected head and body orientation. The composite signal is used to estimate body motion and stabilize eye and body movement.

After an acute loss of peripheral vestibular function, postural and oculomotor deficits appear. With the head still, spontaneous jumping of the eyes (nystagmus) and tilting of the body away from upright appears. When the head is moved, vision and balance is further impaired. Associated with these deficits are behavioral changes aimed at minimizing the risk of disorientation by avoiding visual input, minimizing the risk of falling by adopting a more cautious and stable stance, and a reduced propensity to move the head.

Recovery from vestibular lesions has been studied on for over 100 years (Von Bechterew, 1883). Orientation in space and being able to walk upright are critical functions. It is understandable then that the vestibular system is supported by multiple vestibular repair mechanisms. The capability for repair and adaptation is remarkable ! Plasticity consists of neural adjustments that restore original function. This supplemented by substitution of other sensory input or internal estimates. Finally, one may change one's behavior to "work around" problems presented by a vestibular lesion.

Given sufficient time, persons with up to approximately 50% loss of vestibular function adapt so well that a casual observer may find them indistinguishable from someone without a vestibular lesion. Nevertheless, such persons can rarely attain the same degree of performance as normal, and a sophisticated clinician can nearly always detect this situation.

Our purpose in the following text is to describe the neurophysiology of the vestibular system, paying particular attention to aspects relevant to rehabilitation. We will proceed from the peripheral to central structures, and conclude with a discussion of "higher-level" problems in vestibular neurophysiology, which are relevant to rehabilitation.

THE PERIPHERAL SENSORY APPARATUS

Figure 2 illustrates the peripheral vestibular system, which lies within the inner ear. The periphery includes the membranous and bony labyrinths, and hair cells, which are the motion receptors of the vestibular system. It is bordered laterally by the air-filled middle ear, medially by temporal bone, and it is posterior to the cochlea.

Bony Labyrinth

The bony labyrinth consists of three semicircular canals (SCC), the cochlea, and a central chamber, called the vestibule (see Fig. 3). The bony labyrinth is filled with perilymphatic fluid, which has chemistry similar to cerebrospinal fluid (high Na:K ratio). Perilymphatic fluid communicates via the cochlear aqueduct (not shown) with cerebrospinal fluid. Because of this communication, disorders that affect spinal fluid pressure (such as lumbar puncture), can also affect inner ear function.

Membranous Labyrinth

The membranous labyrinth is suspended within the bony labyrinth by perilymphatic fluid and supportive connective tissue. It contains five sensory organs, namely the membranous portions of the three semicircular canals and the two otolith organs, the utricle and saccule. Note that one end of each semicircular canal is widened in diameter to form an ampulla. This widening will be relevant when we later discuss a common vestibular condition, benign paroxysmal positional vertigo.

The membranous labyrinth is filled with endolymphatic fluid (Fig. 3). In contrast to perilymph, the electrolyte composition of endolymph resembles intracellular fluid (high K:Na ratio). Under normal circumstances, no direct communication exists between the endolymph and perilymph compartments.

Hair Cells

Hair cells contained in each ampulla and otolith organ convert displacement due to head motion into neural firing (Fig. 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 ampullae. 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. 4A). A flexible, diaphragmatic 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 and causes the cupula to bend back and forth which stimulates the hair cells (Fig. 4B).

The otolithic membranes are structures similar to the cupulae, but are weighted membrane. They contain calcium carbonate (limestone) crystals called otoconia and have substantially more mass than the cupulae (Fig. 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.

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. Due to differences in their fluid mechanics, the canals respond to angular velocity, and the otoliths to linear - acceleration.

Semicircular Canals

The semicircular canals 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 to 7 Hz).

This fact poses a significant problem: How do the hair cells of the semicircular canals, which are activated by displacement, produce sensory input proportional to velocity? The labyrinth must have a method of converting head velocity into displacement. Biophysical properties of the semicircular canals loops accomplish the conversion. (Wilson and Jones 1979) The membranous canal loops have very thin walls and a small lumen diameter relative to the radius of the loop curvature. These characteristics make viscous drag on the endolymph very powerful. Viscosity, or in other words fluidic friction, slows down endolymph flow in a way similar to how honey slowly runs down the side of a jar. In a frictionless system with a freely moving cupula, for a step of constant rotational velocity, endolymph displacement would be proportional to velocity times time, or rotational position. The viscosity creates resistance to endolymph movement so that transcupular pressure and displacement become more closely proportional to head velocity. Because of these considerations, over the usual frequencies of head movement, endolymph displacement is proportional to angular head velocity, and the semicircular canals transmit a velocity signal to the brain.

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, 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. (Wilson and Jones 1979)

Three important spatial arrangements characterize the alignment of the semicircular canals 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. 6). Second, paired planes of the semicircular canals between the labyrinths conform very closely to each other. The six individual semicircular canals become 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 semicircular canals 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 opposite side. For the lateral canals, displacement of the cupula towards the ampulla (ampullopetal flow) is excitatory, whereas for the vertical canals, displacement of the cupula away from the ampulla (ampullofugal flow) is excitatory.

There are three advantages to the push-pull arrangement of coplanar pairing. First, pairing provides sensory redundancy. If disease affects the semicircular canals input from one member of a pair (e.g., as in vestibular neuritis), 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 head motion and are common-mode noise. The engineering term for this desirable characteristic is **common-mode rejection**. Third, as will be discussed in a later section, a push-pull configuration assists in compensation for overload.

Otoliths

The otoliths register forces related to linear acceleration (Fig. 7). 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, we have no sense that we are traveling at 300 miles per hour. However, in the process of taking off and ascending to cruising altitude, we sense the change in velocity (acceleration) as well as the tilt of the plane on ascent. The otoliths therefore differ from the semicircular canals in two basic ways: they respond to linear motion instead of angular motion and they respond to acceleration rather than to velocity. (Wilson and Jones 1979)

The otoliths have a simpler task to perform than the canals. Unlike the canals, that convert head velocity into cupular displacement through hydrodynamic viscosity, the otoliths need no special hydrodynamic system to do their job. Exquisite sensitivity to gravity and linear acceleration is obtained by incorporating the mass of the otoconia into the otolithic membrane (see Fig. 5). As force is equal to mass times acceleration, by incorporating 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. 8). However, unlike the canals, which have one sensory organ per axis of angular motion, there are only two sensory organs for three axes of linear motion. In an upright individual, the saccule is vertical (parasagittal), while the utricle is horizontally oriented (near the plane of the lateral semicircular canals). In this posture, the saccule can sense linear acceleration in its plane, which includes the acceleration oriented along the occipitocaudal axis and also 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 again, anterior-posterior motion.

Because earth's gravitational field is a linear acceleration field, on earth, the otoliths register tilt. For example, as the head is tilted laterally (which is also called roll; see Fig. 7), shear force is exerted upon the utricle, causing excitation, while 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—earths gravitational field as well as from linear motion—there is an ambiguity problem. We will discuss strategies that the central nervous system might use to solve this problem in our section on higher-level vestibular processing.

For the otoliths, as in the canals, there is redundancy as there are organs similar in orientation and function located 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 results in increased afferent discharge from one part of a macula, while reducing the afferent discharge from another portion of the same macula. This extra redundancy compared to the semicircular canals, probably makes the otoliths less vulnerable to unilateral vestibular lesions.

The Vestibular Nerve

Vestibular nerve fibers are the afferent projections from the bipolar neurons of Scarpa's (vestibular) ganglion. The vestibular nerve transmits afferent signals from the labyrinths along its course through the internal auditory canal (IAC).

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. (Goldberg and Fernandez 1971) Regular afferents appear to be the most important type for the VOR, because irregular afferents can be turned off with electrical stimulation without much change in the VOR of monkeys (Minor and Goldberg 1991). 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 sensitivity to head velocity of about 0.5 spikes per degree per second. (Fernandez and Goldberg 1971; Miles and Braitman 1980) We can speculate about what happens immediately after a sudden change in head velocity. Humans can easily move their heads at velocities exceeding 300° per second. As noted previously, the semicircular canals are connected in push-pull, so that one side is always being inhibited while the other is being excited. Given the sensitivity and tonic rate noted above, 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° per second! In other words, head velocities greater than 180° per second 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 for rotation that inhibits a canal. (Ewald 1892; Baloh, Honrubia et al. 1977) Cutoff behavior explains why patients with unilateral vestibular loss avoid head motion towards the side of their lesion. More will be said about this when we discuss 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). 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 main 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. 9). 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 vestibulospinal reflexes, and coordinates head and eye movements that occur together. The lateral vestibular nucleus is the principal nucleus for the vestibulospinal reflex. 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, which 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.

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). This process, sometimes called sensorimotor integration, must also communicate with the internal estimation process discussed later under the heading of higher level vestibular processing . 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.

Cerebellum

The cerebellum is a major recipient of outflow from the vestibular nucleus complex, and is also a major source of input itself. Although not required for vestibular reflexes, vestibular reflexes become uncalibrated and ineffective when the cerebellum is removed.

The cerebellar flocculus adjusts and maintains the gain of the VOR. Lesions of the flocculus reduce the ability of experimental animals to adapt to disorders, which reduce or increase the gain of the VOR. Patients with cerebellar degenerations or the Arnold-Chiari malformation typically have flocculus 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 patients 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. The nodulus is required for motion sickness (Bard, Woolsey et al. 1947).

Lesions of the anterior-superior vermis of the cerebellum affect the VSR, and cause a profound gait ataxia with truncal instability. These patients are unable to use sensory input from their lower extremities to stabilize their posture. These 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 or 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 (Cannon and Robinson 1987). Although a similar structure must exist for the connections with the vestibulospinal system (Ezure, Sasaki et al. 1978), the location of the VSR neural integrator is presently unknown.

MOTOR OUTPUT OF THE VESTIBULAR SYSTEM NEURONS

Output for the Vestibulo-Ocular Reflex

Figure 9 about here

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 same plane as head motion.

There are two white matter tracts that 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. 9). As the MLF is often injured in multiple sclerosis, this connection may account for central vestibular symptoms in these patients.

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 complicated than for the VOR. The VSR has a much more difficult task than the VOR, because there are multiple strategies that can be used to prevent falls, which involve entirely different motor synergies. For example, when 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 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. While the eyes can only rotate and thus can do little to compensate for linear motion, the body can both rotate and translate.

There are three major white matter pathways that 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. 9). This pathway generates antigravity postural motor activity or protective extension, primarily in the lower extremities, in response to the head position changes, which occur with respect to gravity. The medial vestibulospinal tract originates from the contralateral medial, superior, and descending vestibular nuclei (see Fig. 9) and mediates ongoing postural changes or head righting in response to semicircular canal 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 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 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 will now discuss their integration into reflexes called 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. The VOR has two components. The angular VOR, mediated by the semicircular canals, 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 in situations where 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 is given below:

1. When the head turns to the right, endolymphatic flow deflects the cupulae to the left (see Fig. 4B).
2. The discharge rate from hair cells in the right crista increases in proportion to the velocity of the head motion, while the discharge rate from hair cells in the left lateral crista decreases (see Fig. 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. Simultaneous contraction of the left lateral rectus and right medial rectus muscles, and relaxation of the left medial rectus and right lateral rectus occurs, 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 >2°per second, 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). As an example of a vestibulospinal reflex, let us examine the sequence of events involved in generating a labyrinthine reflex.

1. When the head is tilted to one side, both the canals and 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 registered by the vestibular system.

The Vestibulocollic Reflex

The vestibulocollic reflex (VCR) acts on the neck musculature to stabilize the head. The reflex head movement produced counters the movement sensed by the otolithic or semicircular canal 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. (Peterson 1988) The COR is facilitated when the vestibular apparatus is injured (Kasai and Zee 1978; Botros 1979). It is rare, however, that the COR has 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 chain 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. The interaction between 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. (Pompeiano 1988)

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. (Peterson 1988) The degree to which the CCR contributes to head stabilization in normal humans is presently uncertain, but it seems likely that it 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 drive visual following responses (i.e., smooth pursuit) and postural reactions. Because of intrinsic delays in multisynaptic visual mechanisms, the earliest visual responses occur at a substantially longer latency (about 80 msec) compared to vestibular responses (about 14 msec) and are much less suited to tracking at frequencies above about 0.5 Hz than vestibular responses (Lisberger 1990). Visual tracking responses may be facilitated after vestibular loss.

SOMATOSENSORY REFLEXES

Somatosensory mechanisms are involved in postural stability as well. Bles and associates documented somatosensory induced nystagmus ("stepping around nystagmus"). (Bles, de Jong et al. 1984) Interestingly, the subjects with bilateral vestibular loss developed a more pronounced nystagmus than did normal subjects. This implies that subjects with bilateral vestibular loss use somatosensory information to a greater extent than normal subjects.

HIGHER-LEVEL VESTIBULAR PROCESSING

In this section we will discuss 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 eyes should be exactly opposite to head movement. When this happens, the ratio of eye movement to head movement velocity, called the gain, equals - 1.0. In order to maintain normal vision, retinal image motion must be less than 2° per second. In other words, for a head velocity of 100° per second, which is easily produced by an ordinary head movement, the gain of the VOR must be 98 percent accurate, as 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 is not compensatory for low-frequency head motion. This fact can be most easily seen by considering the response of the semicircular canals 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, or in other words, the firing rate decays to 32 percent 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 perseverate the response, and replace 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. (Raphan, Matsuo et al. 1979)

The velocity storage mechanism is used as a repository for information about head velocity derived from several kinds of motion receptors. 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 otolithic information to drive the velocity storage mechanism. (Hain 1986) This example shows how the vestibular system resolves multiple, partially redundant sensory inputs.

Internal Estimation – going beyond reflexes

Reflexes are 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 their head), or noisy ?   A mechanism that combines sensory inputs, weights them according to their relevance, 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. There are 100's of transducers and many actuators. Some transducers respond quickly, and some slowly. They may differ in accuracy, scaling, coordinate frame, timing, and noise characteristics. A mechanism is needed to integrate transducer output and develop an internal estimate of the state of the system (i.e. position, velocity, acceleration), to keep the shuttle from crashing.

FIGURE 10 about here (Kalman filter -- optimal estimator).

The engineering solution to this problem developed out of work done by Kalman at MIT, and is often called a "Kalman filter". It is also called an "optimal estimator", or an "internal model". We will use the term "optimal estimator". The essentials of an optimal estimator are shown in figure10. There is very strong evidence that mechanisms similar to optimal estimators are used for human sensorimotor processing (Wolpert and Miall 1996) and vestibular processing (Oman 1990).

Estimators are far more powerful than simple reflexes. There are several key concepts that need to be introduced to understand how it is different. First, internal models of transducers and motor output are used to develop an estimate of the current state. These internal models are adjusted according to experience, and track changes in bodily function. It seems highly likely that vestibular rehabilitation improves internal models.

Second, body state is not computed from sensory input directly, but rather the *difference* between sensory input and predicted sensory input is used to update an *estimate* of body state. This design makes it possible for the estimator to continue to work even in the absence of sensory input, which is clearly a desirable feature.

Third, a "Kalman Gain" weights the degree to which a sensory input affects the ongoing state estimate. This provides a method of adjusting for the salience and reliability of sensory streams. Again, it seems highly likely that vestibular rehabilitation adjusts something similar to the Kalman gain to teach persons with vestibular deficits to compensate for unreliable sensory streams.

Overall, estimators are fundamentally superior to vestibular reflexes –they are intrinsically far more accurate. Estimators function even in the absence of sensory input. Estimators are modifiable to a greater extent than reflexes by experience. Estimators are just not as fast as reflexes as they are not "hard wired".

HIGHER LEVEL PROBLEMS OF THE VESTIBULAR SYSTEM

Compensation for Overload

Humans can easily move their heads at velocities exceeding 300° per second. For example, while driving in the car, when one hears a horn to the side, the head may rapidly rotate to visualize the problem and potentially to 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 section regarding the 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 wired in "push-pull" with the inhibited side. Whereas the inhibited side can only be driven 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 patients with unilateral vestibular loss do not have this mechanism available to deal with the overload problem, and are commonly disturbed by rapid head motion toward the side of their lesion.

Vestibular Ambiguity

Sensory input from the otoliths is intrinsically ambiguous, as 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 if 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 using an optimal estimator as discussed above. As long as one does not have to make a quick decision, these senses may be perfectly adequate. However, remember that visual input takes 80 ms to get to the vestibular nucleus, and that tactile input must be considered in the context of joint position, and 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, while linear accelerations are brief. Neural filters that pass low and 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. (Israel and Berthoz 1989) Stronger otolith-ocular reflexes are generally only seen 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

An instructive illustration of how the brain routinely processes multiple channels of sensory information simultaneously is found in the motion sickness syndrome. The motion sickness syndrome consists of dizziness, nausea or emesis, and malaise following motion. It is thought to be caused by a conflict between movement information in related sensory channels, such as visual-vestibular conflicts or conflict between an actual and an anticipated sensory input. For example, motion sickness is often triggered by reading a book 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, and this allows for the possibility of intralabyrinthine conflict. Space motion sickness is thought to be caused by intralabyrinthine conflict. About 50 percent 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." (Parker, Reschke et al. 1985) 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 that 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 outside of earth's gravitational field, such as is the situation for 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. (Parker, Reschke et al. 1985; Oman, Lichtenberg et al. 1986)

Structures that are generally required for motion sickness include: (1) intact labyrinth and central vestibular connections, (2) cerebellar nodulus and uvula that coordinate labyrinthine stimuli(Bard, Woolsey et al. 1947), (3) the chemoreceptive trigger zone located in the area postrema, and (4) the medullary vomiting center. (Harm 1990) Why certain subjects 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 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 also extrinsic problems that are 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 is so important to survival, the repair facility of the vestibular system must be considered as an integral part of its physiology, and for this reason, it is our final topic.

Adaptive plasticity for peripheral vestibular lesions is amazingly competent, even enabling the vestibular system to adapt to peculiar sensory situations requiring a reversal of the VOR. (Gonshor and Melvill-Jones 1976) Adjustments of internal models and weighting of sensory inputs (e.g. Kalman gain, see above) is likely at least as important as readjustment of reflexes, as 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. (Baker, Wickland et al. 1987) Similarly, when the VOR of cats is trained using head movements of low frequency, no training effect is seen at high frequencies. (Godaux, Halleux et al. 1983)

Another type of context dependency relates to the vestibulospinal reflexes 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 when the head is turned 90° 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°. 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 where the head assumes other postures, such as looking down at one's 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, both your television and the repair shop are broken, 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, which 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, combined with a competent central repair capability. The system as a whole can withstand and adapt to major amounts of peripheral vestibular dysfunction. The Achilles' heel of the vestibular system is a relative inability to repair central vestibular dysfunction.

FIGURE 1. Block diagram illustrating the organization of the vestibular system. (Copyright Timothy C. Hain, M.D)

FIGURE 2. Anatomy of the peripheral vestibular system. (Copyright Northwestern University)

FIGURE 3. The membranous and bony labyrinths. Adapted from (Pender 1992).

FIGURE 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 endolymph flow and cupular deflection in response to head motion. Adapted from (Bach-Y-Rita and al 1971)

FIGURE 5. The otolithic macula and its overlying membrane. Reprinted from Baloh RW, Honrubia V, 1990, with permission of the publisher, F. A. Davis.

FIGURE 6. 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.

FIGURE 7. The otoliths register linear acceleration and static tilt.

FIGURE 8. Geometry of the otoliths. Reprinted from Baloh RW, Honrubia V, 1990, with permission of the publisher, F. A. Davis. (Baloh and Honrubia 1990)

FIGURE 9. The VOR and VSR reflex 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 and light lines, beginning in the lateral and medial vestibular nucleus, respectively. From (Brodal 1981), with permission of the publisher, Oxford University Press.

FIGURE: 10. An internal model like this may be used for sensorimotor integration. Sensory inflow and motor outflow are used to estimate the current state. From (Wolpert, 1997) with permission of the publisher, CellPress.

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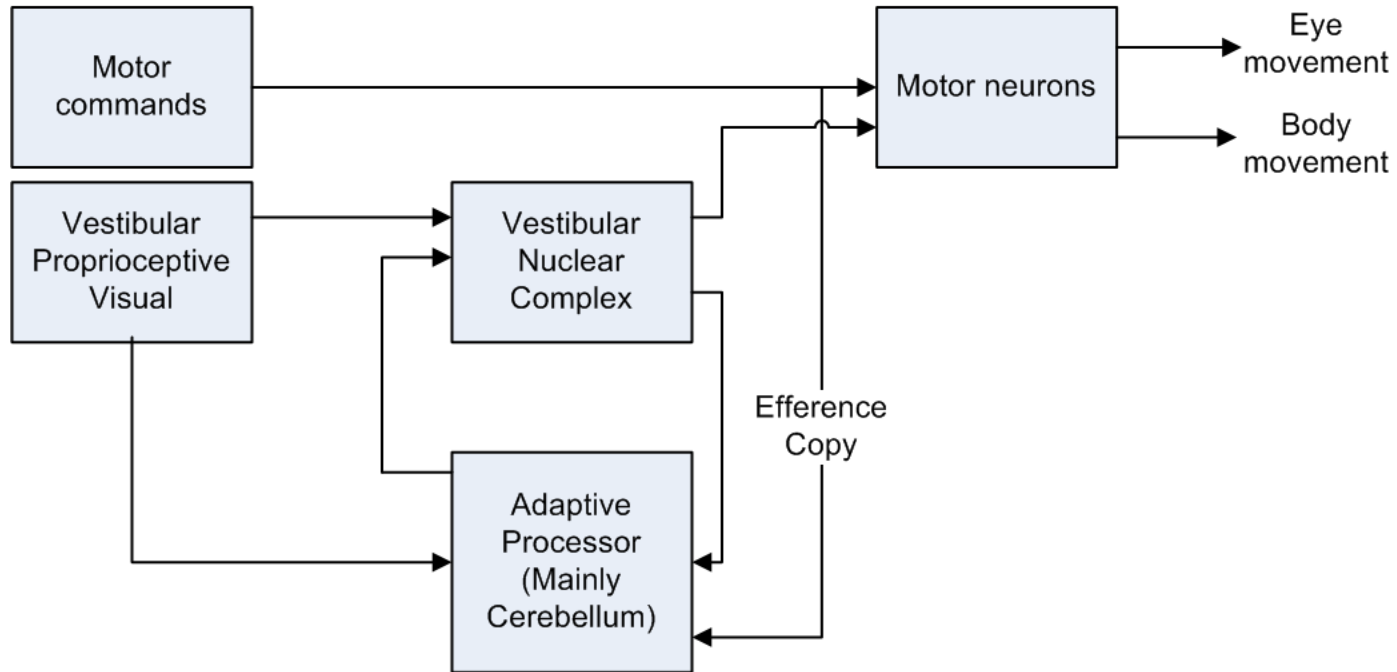
Figure 1: Block diagram illustrating the organization of the vestibular system. (copyright Timothy C. Hain, M.D)
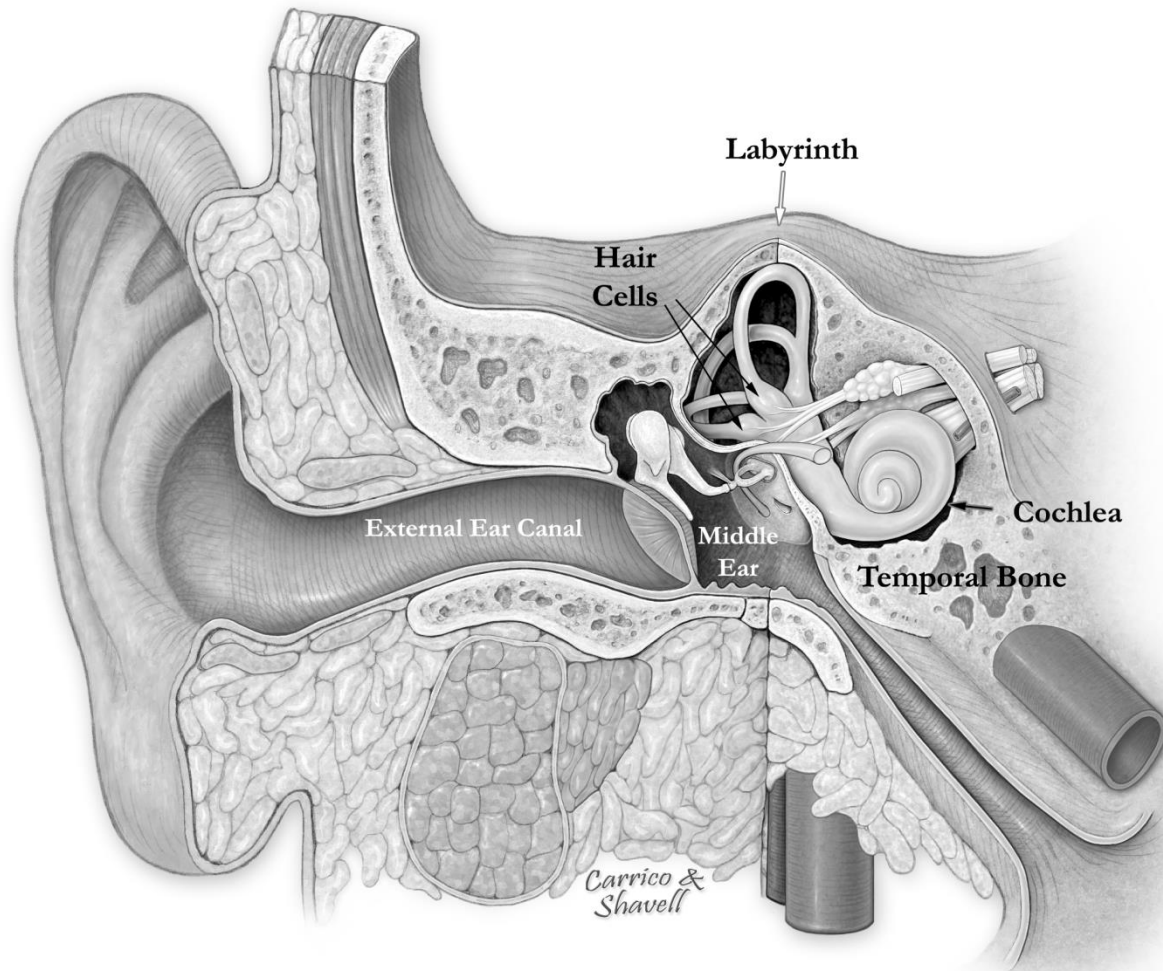
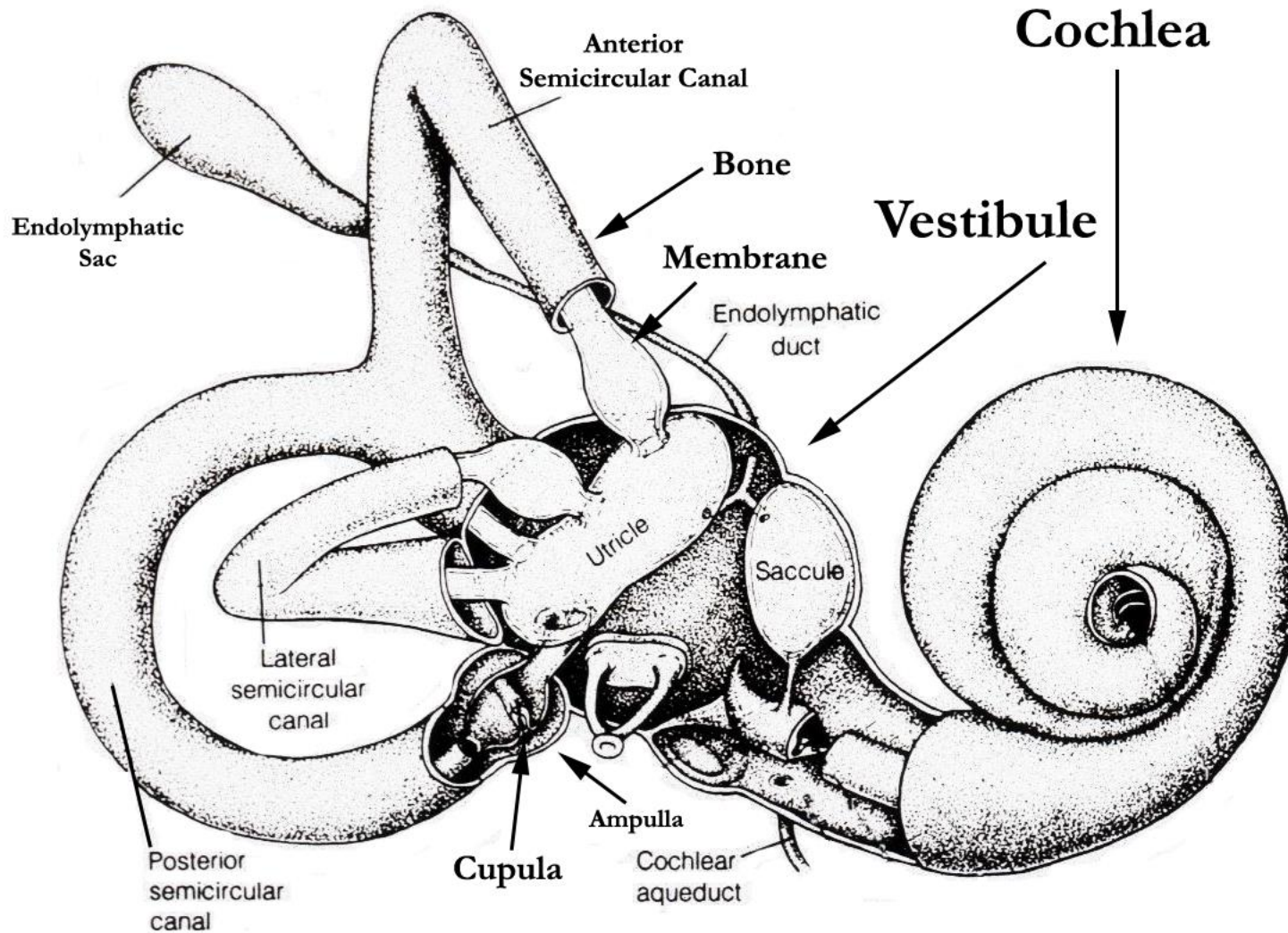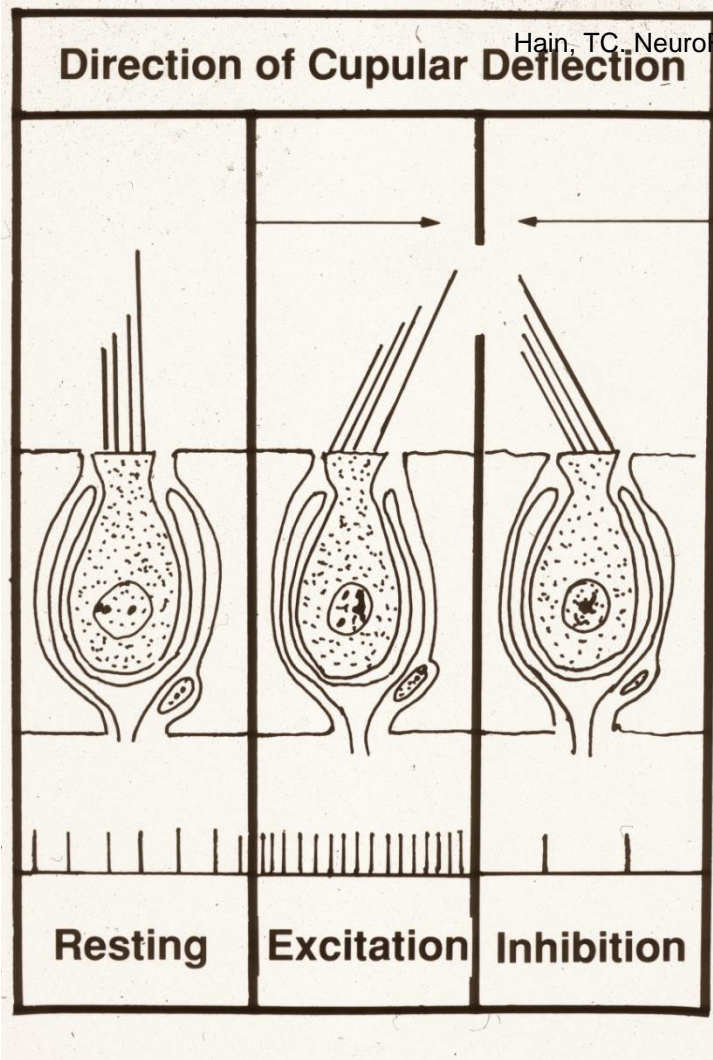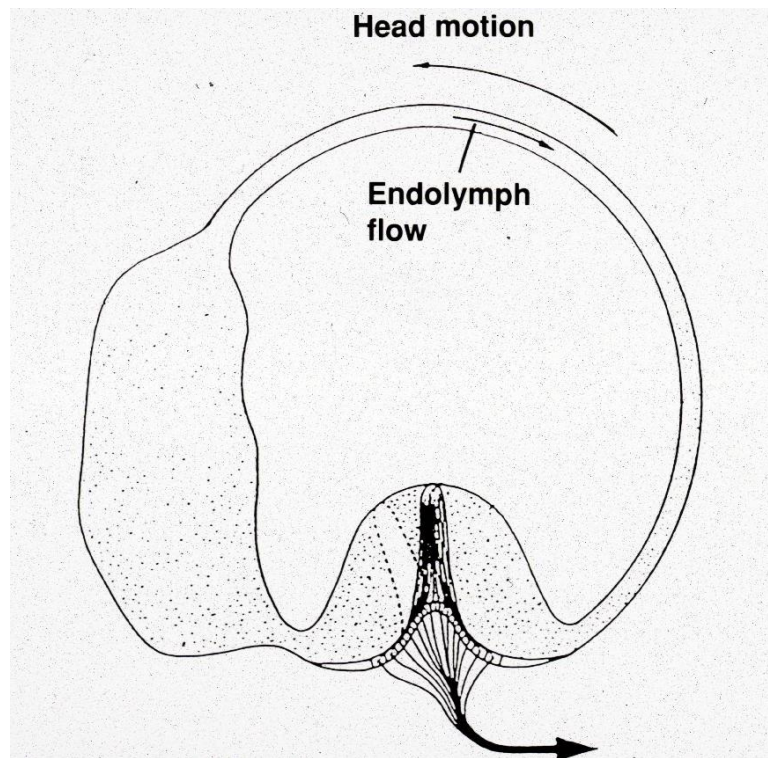Figure 2: Anatomy of the peripheral vestibular system. (copyright Northwestern University)

Figure 3: The membranous and bony labyrinths. Adapted from
(Pender 1992).

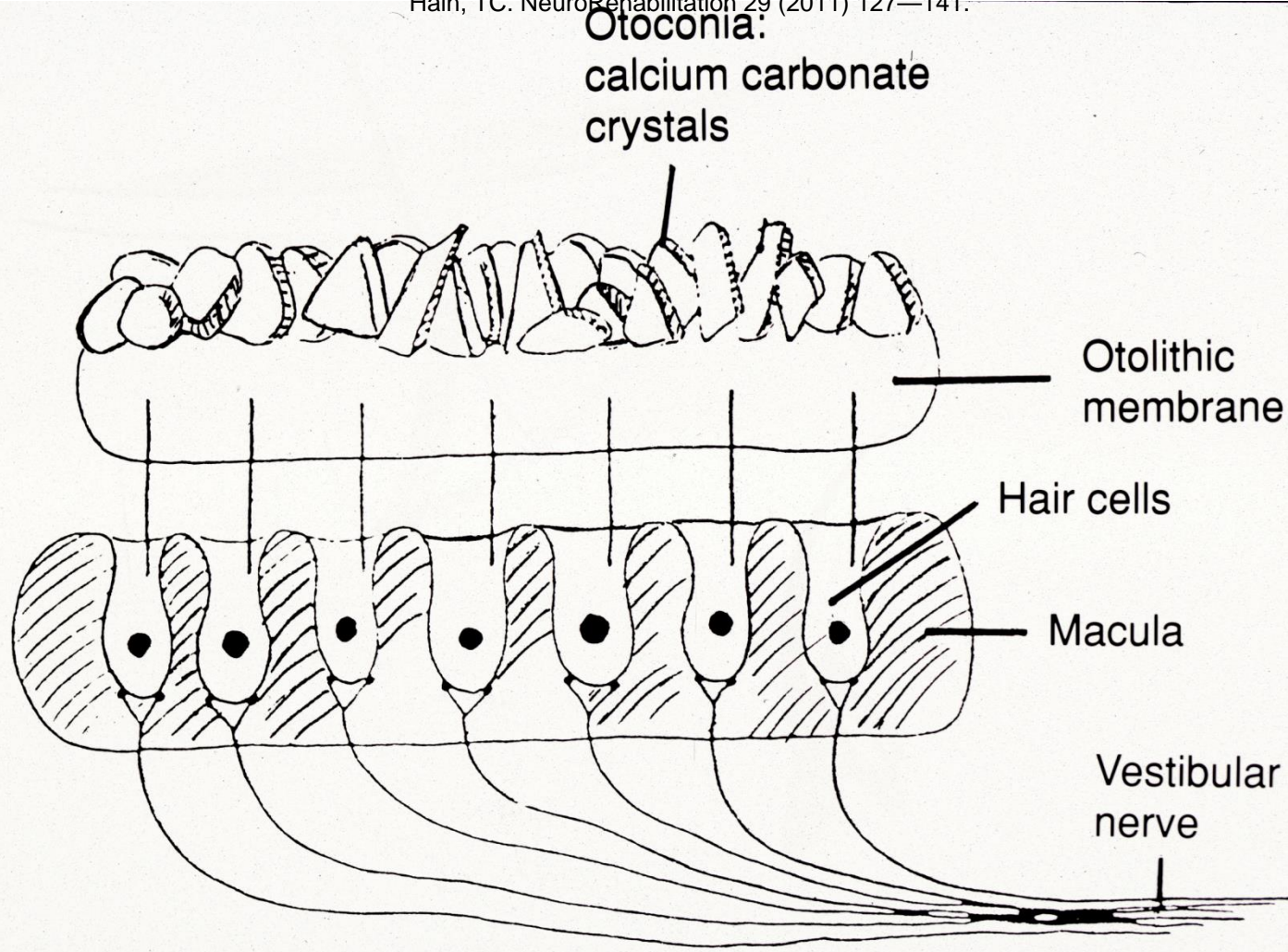Figure 4: Effects of head rotation on the canals. B. Adapted from (Bach-Y-Rita and al 1971)

Otoconia:
calcium carbonate
crystals

Otolithic membrane

Hair cells

Macula

Vestibular nerve

Figure 5: The otolithic macula and its overlying membrane. Reprinted from Baloh RW, Honrubia V, 1990, with permission of the publisher, F. A. Davis.
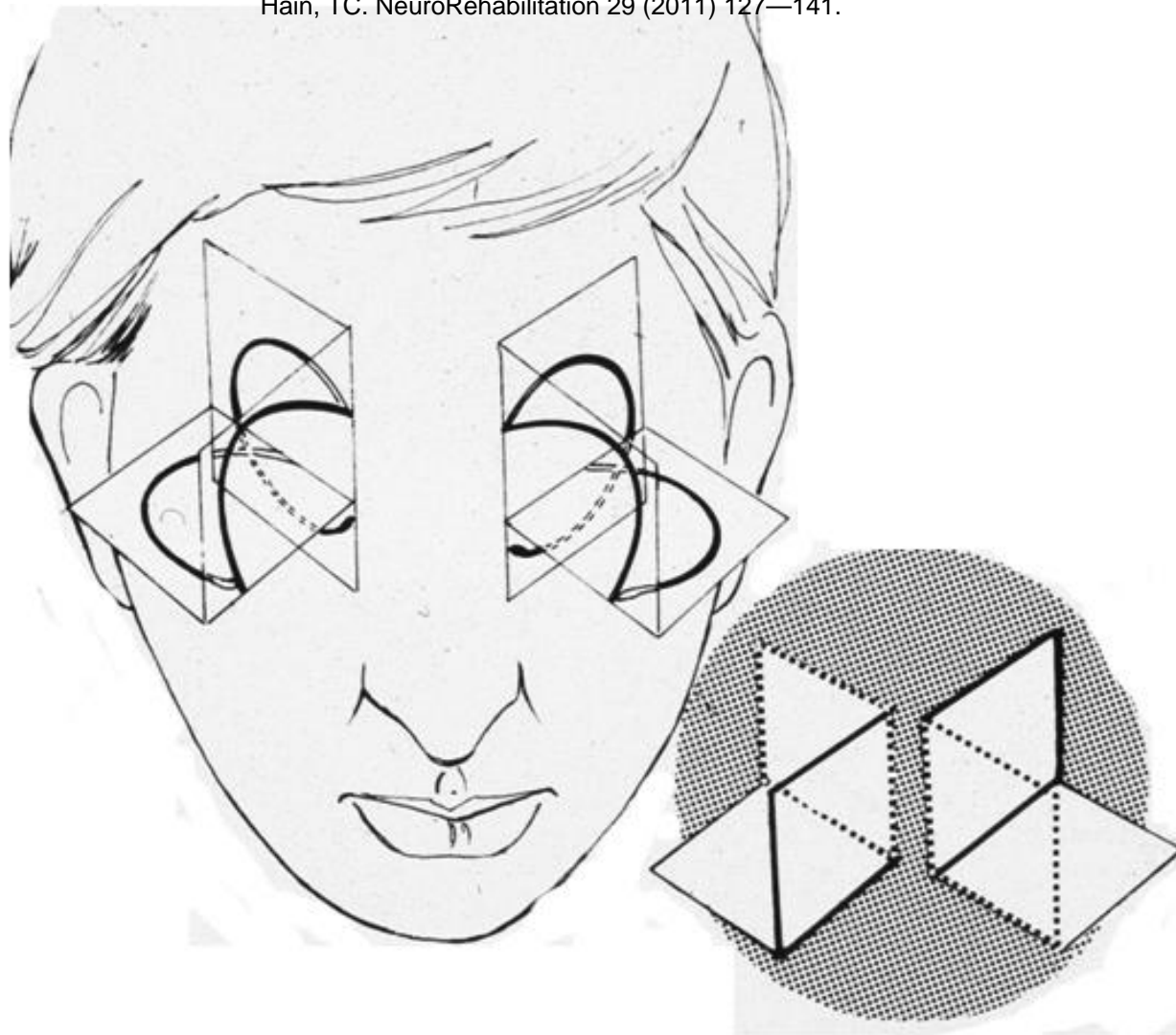
Figure 6: The spatial arrangement
of the semicircular canals
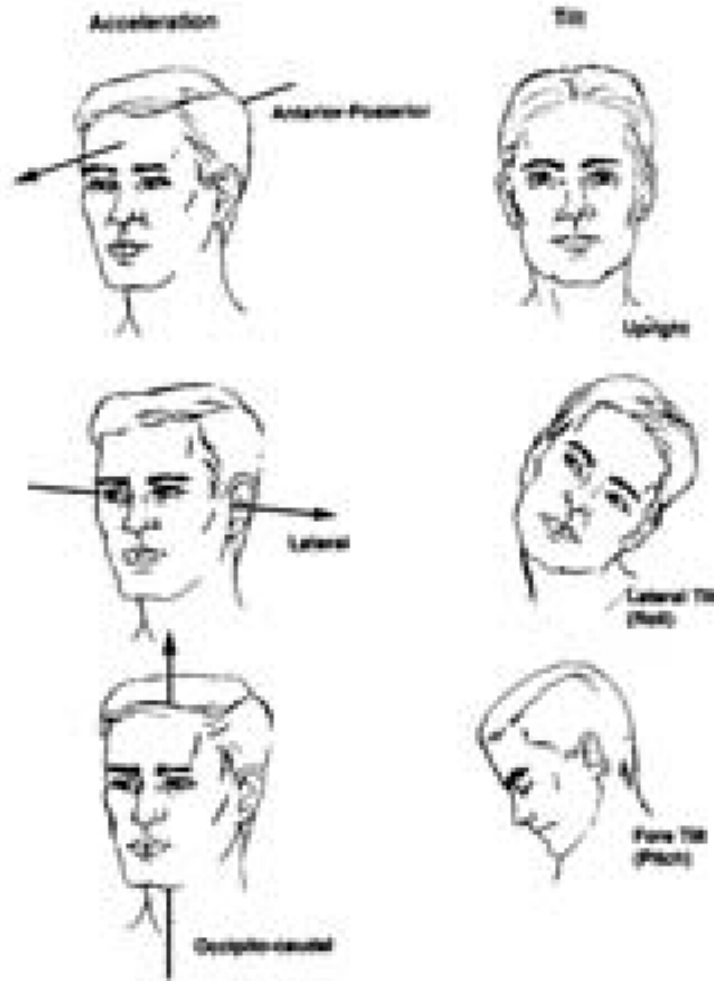
FIGURE 1-8. The otoliths register linear acceleration and static tilt.

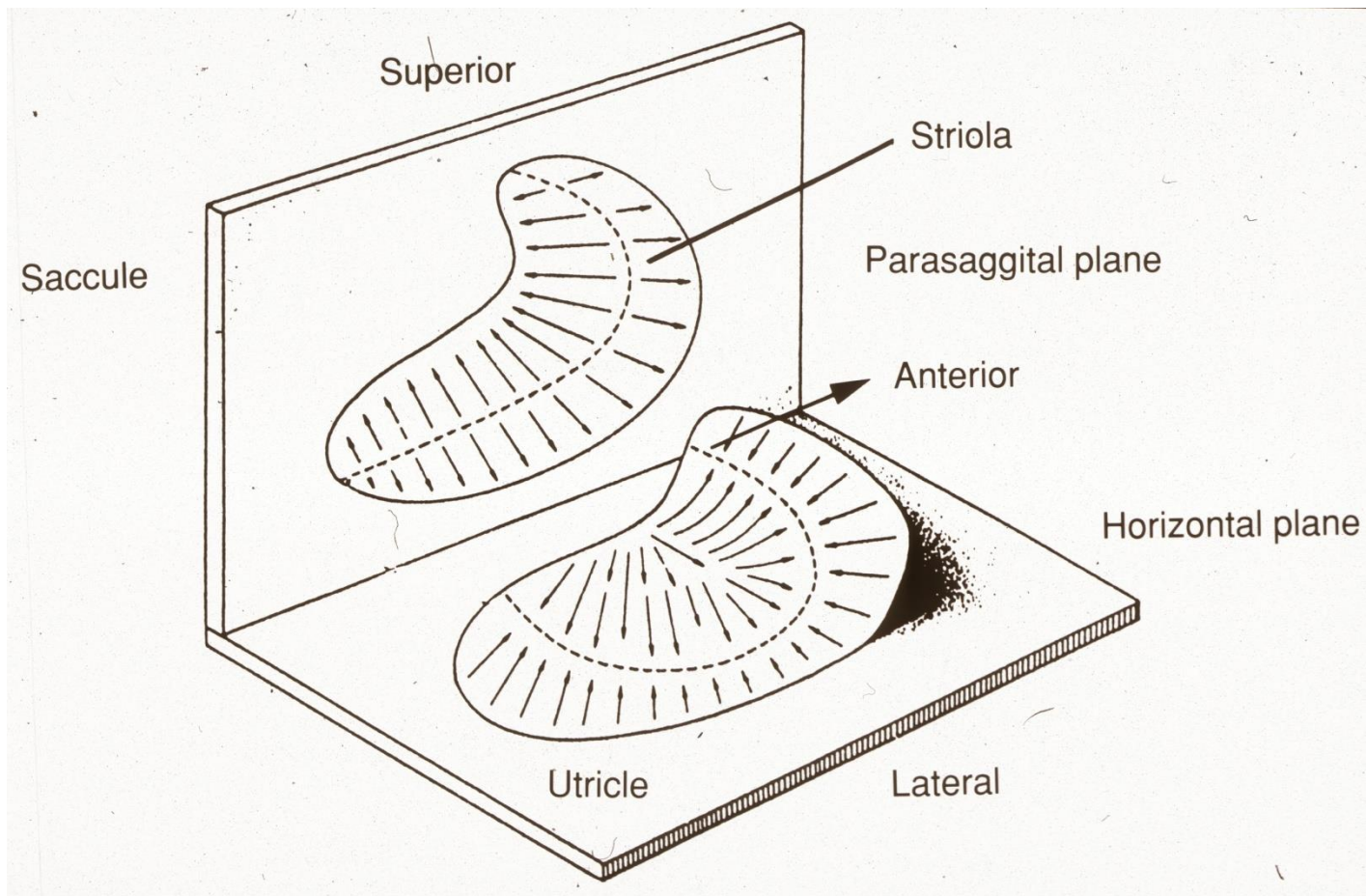FIGURE 7. The otoliths register linear acceleration and static tilt.
(Copyright Timothy C. Hain, M.)

FIGURE 8. Geometry of the otoliths. Reprinted from Baloh RW, Honrubia V, 1990, with permission of the publisher, F. A. Davis. (Baloh and Honrubia 1990)
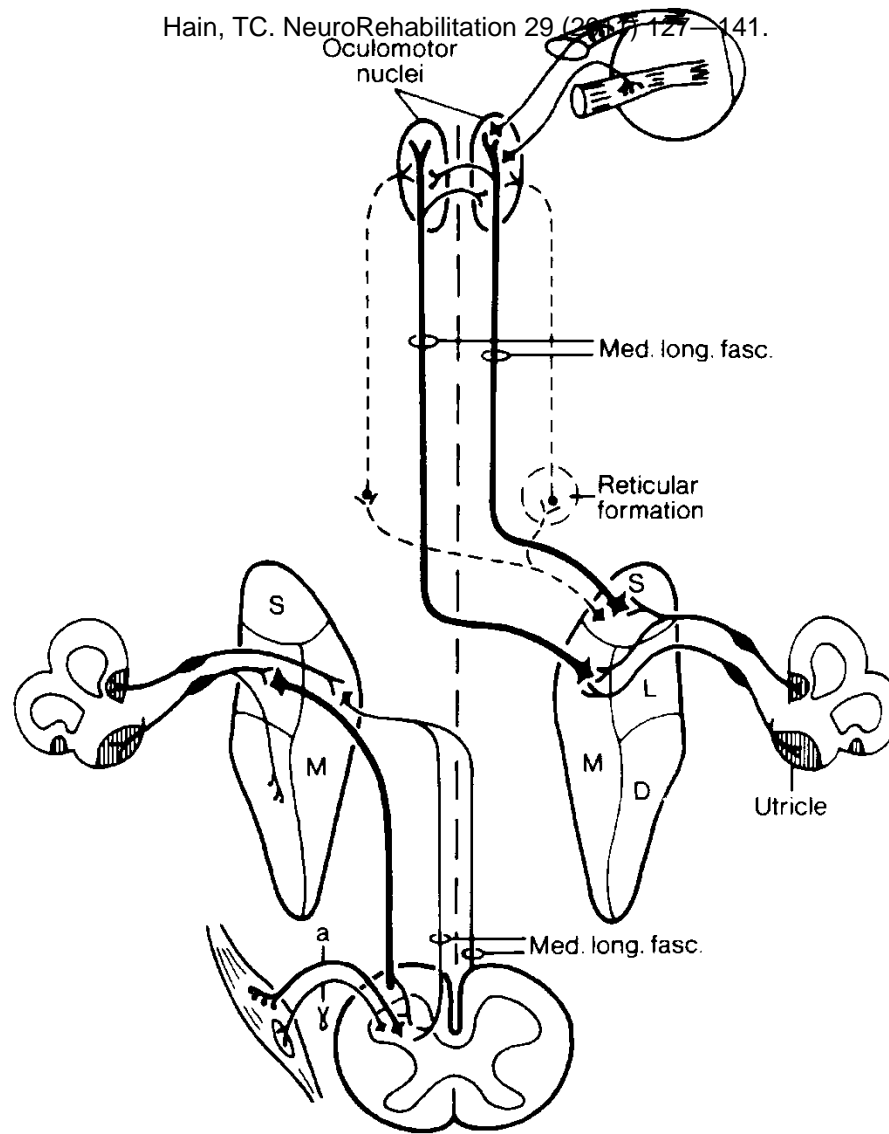
FIGURE 9. The VOR and VSR reflex arcs. From (Brodal 1981), with permission of the publisher, Oxford University Press.
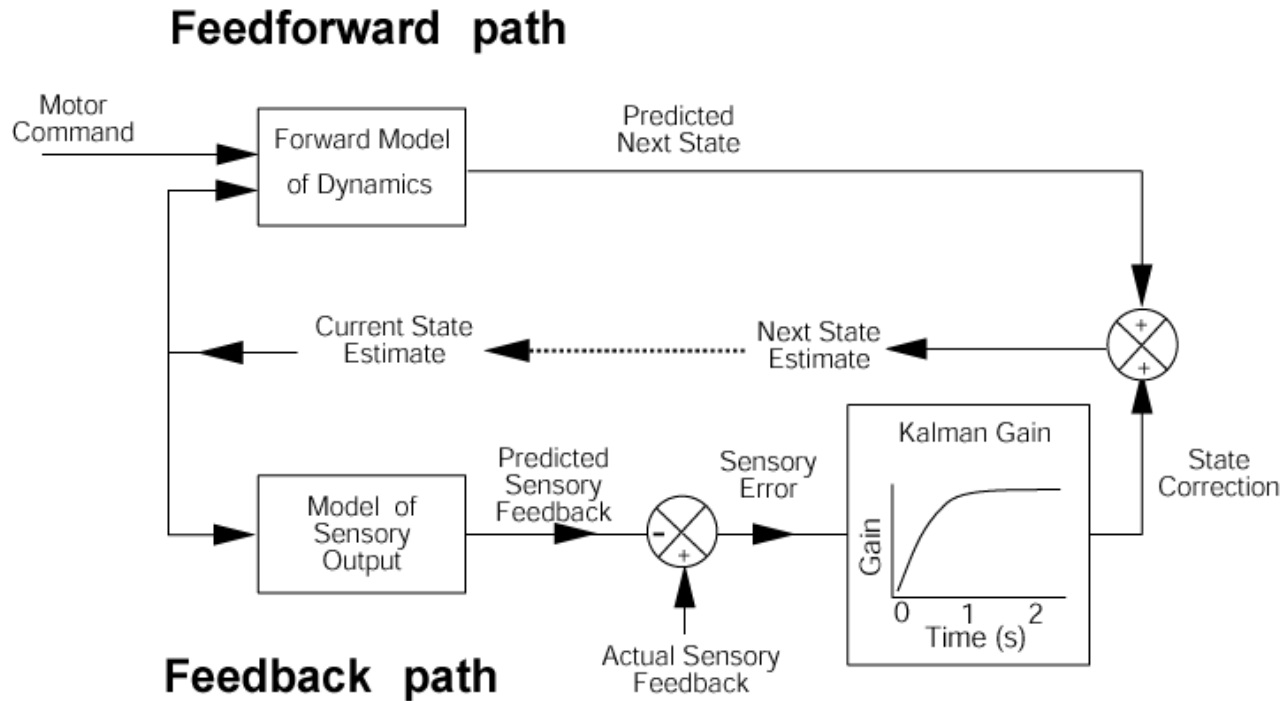
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Figure 10. An internal model like this may be used for sensorimotor integration. Sensory inflow and motor outflow are used to estimate the current state. From (Wolpert, 1997) with permission of the publisher, CellPress.