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Key Words: eye movements • reflex, postural • space perception (visual) • visual perception

The peripheral vestibular apparatus—the vestibular labyrinths—detects head acceleration. The labyrinths integrate the input so that the signal ascending the vestibular nerve to the vestibular nuclear complex (VNC) is equivalent to head velocity (Keshner & Cohen, 1989). At the VNC, this information is combined with input from other sources, including visual input. Signals descending from the VNC to the spinal cord are involved in postural control and eye–head coordination; signals ascending from the VNC are involved in oculomotor control and some aspects of spatial orientation.

Visual/Vestibular Interactions
Oculomotor Function

The rehabilitation literature often refers to observations of conjugate eye movements as measures of the integrity of the vestibular system (e.g., DeGangi, Berk, & Larsen, 1980; Weeks, 1979). Conjugate eye movements are those in which both eyes move in parallel fashion. Conjugate eye movements involve the vestibuloocular reflex (VOR), the optokinetic response (OKR), pursuit movements, and saccades. Eye movements indicate vestibular function because the mechanisms generating some eye movements include reciprocal connections with the VNC. Other brain stem centers and the cerebellum contribute to oculomotor control as well.

Responding to head movements, the VOR generates compensatory eye movements to maintain gaze stability. For example, to look straight ahead, if the head rotates to the right at 10 degrees/s, the VOR produces a compensatory rotation of the eyes to the left at 10 degrees/s. Thus, the effective direction of gaze is straight ahead. The pure VOR is active only in darkness. In light, the VOR combines with other eye movements to scan the environment with combined eye–head movements. The VOR is also important for compensation of the normal physiological tremor of the stationary head. During combined eye–head movements, as are used to track a moving object, vestibular information may be used as negative feedback to the motor commands driving the eyes. For example, to shift gaze to a new location, the eyes and the head move. The eyes reach the final location first. Then, as the head continues to move toward the target, the eyes make a compensatory rotation in the opposite direction to maintain the direction of gaze (Lanman, Bizzi, & Allum, 1978). For gaze changes through small angles, the VOR may be suppressed during the initial phase of the head movement (Bizzi, 1981). As the head continues to move, however, the eyes are followed by the head, with a compensatory readjustment of final eye position to maintain gaze stability. For example, to lean forward to reach for an...
object, the neck may extend, which tilts the head with reference to the body to maintain the head in an upright position with reference to gravity. To compensate for that upward head movement, the VOR may generate a downward eye movement to maintain a stable gaze. The adequate stimulus for the VOR is head acceleration. It is active within the normal range of frequencies (0.01-7.0 Hz) for voluntary head movement (Büttner, Büttner, & Henn, 1978).

The optokinetic system generates the OKR in response to movement of the entire visual surround. The OKR maintains gaze stability at frequencies below 1 Hz (Robinson, 1981). For example, the OKR is used to watch the moving pictures at a cinema with a large curved screen. The adequate stimulus for the OKR is the velocity at which the entire visual image moves across the retina, called the retinal image slip velocity. The OKR interacts with the VOR to extend the range of sensitivity to whole-field movement in the visual world. That is, the visual system is sensitive to movement of the entire visual image across the retina, regardless of the way this movement was initiated (i.e., head acceleration, retinal image slip velocity, or both).

Avoeate mammals have only the VOR and OKR; higher mammals, including humans, have pursuit movements and saccades as well. Pursuit movements are the smooth tracking movements used to follow a discrete moving stimulus. Retinal image slip velocity is the adequate stimulus. Unlike the OKR, however, pursuit movements are stimulated by movement of a discrete part of the visual scene, rather than the entire visual surround. For example, pursuit movements are used to watch a moving wheelchair. Saccades are the rapid eye movements used to scan a stationary visual array. You are using saccades to read this page. Saccades are also used for corrective eye movements. For example, to watch a target that is moving too quickly to follow with just pursuit movements, such as an airplane, saccades may be used. The adequate stimulus for the saccadic system is the position of the visual image on the retina (Robinson, 1981).

The conjugate eye movements may be combined for more complex oculomotor behaviors such as nystagmus. Normal nystagmus is a combination of slow and fast phases (Baloh & Honrubia, 1979). Vestibular nystagmus is generated during rapid, continuous self-movement in the dark. Slow phases, in the opposite direction from the stimulus, are generated by the VOR to maintain gaze stability. Quick phases, in the same direction as the stimulus, are generated by the saccadic system to reset eye position before the next slow phase (Ron, Robinson, & Skavenski, 1972).

Optokinetic nystagmus differs from vestibular nystagmus. Unlike the VOR, optokinetic nystagmus is generated during continuous movement of the entire visual field while the observer is stationary (e.g., while the observer is watching a moving train from the adjacent platform). Slow phases are in the same direction as the stimulus and are generated by the optokinetic system to maintain gaze stability. Quick phases, in the opposite direction from the stimulus, are generated by the saccadic system to reset eye position. Thus, under different situations both vestibular and optokinetic nystagmus effectively stabilize the position of gaze in space.

A subject spinning around in a lighted room with eyes open generates a combination of vestibular and optokinetic nystagmus. The Southern California Postrotary Nystagmus Test (SCPNT), used by some therapists as a measure of vestibular function, tests this combined vestibular/optokinetic nystagmus. However, test results can be confounded by several inaccuracies in test administration. For example, the use of a hand-held stopwatch can lead to inaccurate measurements due to a lag in the therapist's reaction time. This lag introduces an error of at least one reaction time, 150-200 ms, which is the minimum reaction time (Marteniuk, 1976). Observation by the unaided eye to determine onset and cessation of eye movements can also introduce measurement error. Furthermore, subjects are unlikely to be able to maintain head stability in the designated position without rotations in any other plane; this changes the stimulus. Finally, both postrotatory nystagmus duration and amplitude vary in normal subjects over trials (Jager & Henn, 1981). The neural basis for this normal decrement in nystagmus duration on repeated testing is discussed later in this paper.

Apparent deficits on the SCPNT or on similar tests may indicate pathology in any of several centers or may be artifacts of the test variability. Duration and amplitude of the VOR may be controlled by the cerebellum (Lisberger, Miles, & Zee, 1984; Waespe, Cohen, & Raphan, 1985). Lesions in the cerebellar centers or in any of the other areas involved in generation of the VOR and OKR may cause decrements in the reflex. The SCPNT and similar tests should not, therefore, be used as a measure of pure vestibular function. Better tests of vestibular function include electrooculography and caloric testing. Simple methods do exist to test some components of the VOR and OKR at the patient's bedside. For example, the VOR and OKR may be examined in the light with use of a hand-held optokinetic drum (Zee, 1977). This test is reliable for indicating disorder, without specifying the locus of the deficit. It tests amplitude of the response reliably (Simmons & Büttner, 1985). Apparent deficits found in some adults, however, may indicate the changes associated with normal aging. For example, eye velocity during the OKR and pursuit
decreases with age in adults (Simmons & Büttnert). Tests of positional nystagmus may indicate peripheral or central deficits (Katsarkas, 1987).

Oculomotor Neuroanatomy and Physiology
Conjugate eye movements are generated by signals from the vestibular nuclei and related centers in the brain stem and cerebellum. Some pathways, such as those to cranial nerves III, IV, and VI, are used for all eye movements, and some are used under more limited circumstances. For example, primary vestibular signals are required for generation of the VOR but are not needed to produce saccades. Visual input from the optic tract is relayed to the VNC and to cerebellar Purkinje cells. Vestibular input is relayed directly and indirectly to the brain stem nuclei and cerebellum for visual/vestibular convergence at those centers. Vestibuloocular pathways include a loop through the flocculonodular lobe of the cerebellum, where vestibular and visual signals converge. The cerebellum is important for adaptation of the VOR to relatively permanent changes in input. For example, floccular lesions impair adaptation to the changes in visual input induced by the use of magnifying or reversing lenses (Lisberger et al., 1984; Robinson, 1976). Lesions of the nodulus and uvula impair habituation to repeated optokinetic or vestibular stimulation (Cohen, Cohen, & Raphan, 1987; Waespe et al., 1985). A complete description of these pathways can be found elsewhere (Miles & Lisberger, 1981; Robinson, 1981).

Models
The input to the VOR is head velocity, but the output of the reflex is eye position. Some central processing must occur to account for this change from velocity to position. Velocity can be transformed to position by mathematically summing, or integrating, velocity over time. The neural circuitry that performs this mathematical operation is called an integrator.

The brain stem oculomotor pathways may contain a neural integrator to transform the vestibular velocity signal to the eye position signal (Robinson, 1981). Because the duration of the VOR is significantly longer than the duration of the vestibular input, Raphan, Matsuo, and Cohen (1979) proposed a second neural integrator that holds, or stores, the velocity information for a period of time. This neural mechanism is called a velocity storage integrator (Raphan & Cohen, 1980). The velocity storage integrator discharges its stored information when the stimulation has ceased, which causes poststimulus eye movements. Furthermore, the velocity storage integrator has a switching mechanism that allows it to discharge, or dump, stored velocity information in the presence of a conflicting signal. For example, if a subject is given rotatory vestibular stimulation in the dark, he or she has perrotatory nystagmus in the opposite direction. When the rotation ceases, the labyrinthine hair cells are deflected in the opposite direction due to the deceleration, or change in velocity. This causes postrotatory nystagmus in the opposite direction. The postrotatory nystagmus stops when the lights are turned on. The nystagmus may stop because the velocity storage integrator dumps its stored velocity information in response to the new visual input to the system. The new visual information tells the nervous system that the subject has stopped moving, because the stationary visual surround does not appear to move in the direction opposite to that of the subject, as it would if the subject were still in motion. In other words, the velocity storage integrator has information about the subject's movement through space; it signals continued movement as well as the transient changes in acceleration registered by the vestibular end-organs. In this way the velocity storage integrator can act as a multisensory detector for motion perception (Raphan & Cohen, 1985).

Clinical Significance
Loss or impairment of the labyrinthine signal leads to incorrect calculations by the neural integrator. If the neural integrator makes incorrect calculations, the resulting behavioral dysfunction is manifested by loss of the VOR or incorrect stabilization and compensation for eye-head position.

Loss of the VOR can result in blurred vision during head and body movement (e.g., during locomotion). Such blurring could cause disorientation and an inability to distinguish environmental cues, such as signs and curbs. At first, the system is unable to compensate for loss of the VOR. Patients with VOR loss report blurred vision with any head movement and illusory movement of the world on head rotation (Atkin & Bender, 1968; Crawford, 1952). These symptoms seem to abate over time with development of several compensatory strategies: (a) increased use of the cervicoocular reflex, (b) centrally programmed compulsatory eye movements, and (c) compulsatory saccades (Dichgans, Bizzi, Morasso, & Tagliasco, 1974). People with chronic bilateral labyrinthine loss demonstrate stronger cervicoocular and somatosen-sory-ocular responses than do people without such loss (Bles, de Jong, & Rasmussens, 1984). Exercise is beneficial in the treatment of acute vestibular loss. Even generalized experience in moving through space facilitates development of adaptive strategies (Mathog & Peppard, 1982). More specifically, treatment activities that require use of these responses can be beneficial in the development of compensatory strategies. For example, a patient could be given activ-
ities that require visual pursuit of rapidly moving objects, the speed and trajectory of which are unpredictable. Such tasks should include visual images that cover a large part of the visual field, to induce combined eye–head movements. Equipment such as the Youville Visual Tracking Machine (Bergmann, 1977) can be used. Postural instability in patients with labyrinth deficits increases in the absence of vision (Bles et al., 1984). Therefore, treatment strategies for these patients should include activities in both well-lighted and dark environments.

Normal people adapt rapidly to changes in the usual level of vestibular input. For example, sailors at sea are able to walk about the ship and maintain a stable gaze. Also, many normal people wear magnifying or minimizing spectacles to correct their vision. They adapt rapidly to changes in the magnification of the visual image, such as those caused by a change in the eyeglass prescription (Collewijn, Martins, & Steinman, 1983). Cerebellar mechanisms are probably involved in such adaptation. Lesions of the nodulus and uvula, parts of the vestibulocerebellum, cause an inability to habituate the VOR and optokinetic nystagmus time constants and an inability to dump nystagmus when tilted (Cohen et al., 1987; Hain, Zee, & Maria, 1987; Waespe et al., 1985). Therefore, patients with lesions of these parts of the cerebellum may have difficulty maintaining a stable gaze. Conversely, since the nodulus and uvula are involved in habituation of the VOR time constant, vestibular nystagmus of short duration or short time constant does not necessarily indicate dysfunction. More likely, people with short time constants have been exposed to vestibular stimulation repeatedly. Such people include ballet dancers and children with mental retardation who spend much time rocking or spinning.

**Vection Illusions**

Vection illusions, or illusions of self-movement, result from the visual and vestibular inputs to the motor system that are mediated by the VNC. These illusions are normal events. In a vection illusion, visual stimuli produce the illusion of self-motion rather than of motion of the external visual world. For example, a subway rider, while staring out the window, might sense that the train has suddenly started to move. She would adjust her posture accordingly. On shifting her gaze, however, she would realize that the train that moved was the train she watched rather than the train she is on. Similarly, Helmholtz (1925) described the experience of leaning backward while watching a waterfall, to compensate for the sensation of falling forward toward the water. In both cases the illusion of body motion is induced by movement of the entire visual scene (Dichgans & Brandt, 1978).

The neural substrates for vection illusions involve sites with visual/vestibular convergence and connections to higher centers (Zee, Yee, & Robinson, 1976). Within the VNC, some neurons have direction-specific responses to vestibular stimulation, or head movement (Keller & Precht, 1978; Waespe & Henn, 1979). The same neurons also respond in the same way to optokinetic stimulation, or movement of the visual surround, in the opposite direction. In both cases the direction of nystagmus is the same. The vestibular and optokinetic stimuli, however, are in opposite directions. For example, in a study by Waespe and Henn, when a monkey turned his head to the left, which resulted in apparent motion of the visual world to the right, specific neurons responded. When the monkey was stationary and the visual world actually did turn to the right, the same neurons responded. These data suggest that neurons in the vestibular nuclei treat the converging visual and vestibular signals as equivalent. In this way, the VOR and OKR complement each other, which extends the range within which the system is sensitive to movement of the visual world. (See the description of the conjugate eye movements on p. 331.)

This same visual/vestibular interaction probably mediates the visual influence on posture. The postural adjustments made by infants and adults in response to vection illusions are those that would be appropriate compensatory responses if the subjects had moved rather than the visual surround (Butterworth & Hicks, 1977; Helmholtz, 1925; Lee & Lishman, 1975). For example, Lee and Lishman had subjects stand in a room with walls that moved in an anterior-posterior direction. When the room moved anteriorly, the wall facing the subject moved away. The subjects leaned forward as if compensating for having leaned backward. Thus, movement of the entire visual surround was interpreted as movement of the subject.

A relationship between changes in muscle tone and changes in labyrinthine signals has been suggested in the rehabilitation literature. For example, the Bobaths observed apparent changes in tone with changes of the position of the head in space (Bobath & Bobath, 1964, 1972). Labyrinthine signals do influence muscle tone, but they do so by interacting with proprioceptive signals (Kim & Partridge, 1969). The common clinical finding of increased extensor tone following rapid, whole-body linear or rotatory movement is often ascribed to the influence of labyrinthine stimulation. Instead, some of this effect can be attributed to short-term labyrinthine influences on spinal motoneurons via the vestibulospinal tracts, as discussed in the previous paper by Keshner and Cohen. Although increased extensor tone is sometimes considered evidence of the efficacy of vestibular stimula-
tion as a treatment modality, this may not be true. This short-term increase in extensor tone may be caused, at least in part, by the VNC-mediated visual influence on the postural system. In other words, the observed behavior may be partly a vection response—a postural response to observed movement of the entire visual scene. Because therapy is usually given in a well-lighted, structured visual environment, a patient receiving rapid vestibular stimulation in one direction also receives rapid, whole-field visual stimulation in the opposite direction as well as kinesthetic, somatosensory, and probably auditory input. Therapists can take advantage of this effect by manipulating the visual field to encourage various postural responses. Future research might find a way to separate these complex factors. To date, no empirical evidence supports the theory that these sensory modalities have any long-term effect on extensor tone changes.

Spatial Orientation

Spatial orientation strategies are divided into two categories: allocentric and egocentric. Allocentric orientation defines spatial location with reference to some external point; egocentric orientation defines spatial location with reference to the observer’s position. Vestibular information is used for egocentric orientation under both stationary and moving conditions (Potegal, 1982; Schone, 1984). Under stationary conditions, gravitational information is important; under moving conditions, acceleration information is more significant.

Neuroanatomy and Physiology

The vestibular nuclei project to the thalamus along several pathways (Abraham, Copack, & Gilman, 1977; Maciewecz, Phieps, & Highstein, 1982) and terminate at several sites within the thalamus (Blum, Abraham, & Gilman, 1979; Hawrylyshyn, Rubin, Tasker, Organ, & Fredrickson, 1978). Most thalamic neurons with direction-specific responses to vestibular stimulation also respond to proprioceptive stimuli or display direction-specific responses to optokinetic stimulation from the direction opposite to that of the vestibular stimulation (Büttner & Henn, 1976; Deecke, Schwarz, & Fredrickson, 1977; Lang, Büttner-Ennever, & Büttner, 1979).

From the thalamus, some fibers project to the cerebral cortex and other fibers project to an area in the head of the caudate nucleus, which also receives auditory projections (Wilson, Hull, & Buchwald, 1985). The caudate projection is independent of the vestibulocortical projections. The vestibulocortical projections in most animals are small. In cats and monkeys, at least two vestibulocortical projection areas have been found. Walzl and Mountcastle (1949) first identified a small area near the auditory and somatosensory areas in the cat. Their finding has been replicated and extended to include a second area (Sans, Raymond, & Marty, 1970). More recently, a third vestibulocortical area was found in the monkey (Grusser, Pause, & Schreiter, 1983). Only one vestibulocortical area has been found in humans (Penfield, 1957), but that may be because these data are difficult to collect. In rodents, cats, and monkeys, at least one vestibular projection lies within the forelimb somatosensory area. Neurons in this forelimb region, which have direction-specific responses to vestibular stimulation, also respond to proprioceptive and optokinetic stimulation (Grusser et al., 1983; Mergner, 1979).

Unlike the classical sensory cortical projections, which are relatively large and modality-specific, the vestibulocortical projections are small and also respond to signals that are not labyrinthine in origin. Thus, at all levels of the vestibular pathway beyond the primary vestibular afferents, vestibular signals are mixed with information from other sensory modalities.

Maintained Position in Space

The vestibular system provides information on the location of the gravitational vertical and horizontal planes for stationary or moving observers. The importance of this input may be appreciated in cases of loss or conflict. Patients with acute bilateral peripheral vestibular loss cannot regain the upright position in the absence of vision (Nashner, Black, & Wall, 1982). Furthermore, patients with bilateral peripheral vestibular deficits, who were tested on a rotating platform, were initially capable of maintaining an upright position with their eyes closed. During a series of consecutive dorsiflexion rotations, however, the patients began to fall backward (Keshner & Allum, 1986). Patients with acute unilateral peripheral vestibular loss, who are unaware that their eyes are deviated to one side, may overshoot or undershoot the distance when reaching for a target with their eyes open (Horsten, 1979). Normal subjects who experienced the visual /vestibular conflict of vection illusions also indicated a shift in the apparent vertical (Held, Dichgans, & Bauer, 1975), which demonstrates the influence of visual information on the established environmental reference.

Translations of the Body Through Space

Observers using an egocentric orientation strategy must locate themselves with reference to the target. As they move toward the target, they must update their position relative to the target in much the same way that the navigator of a ship keeps track of the
ship's position at sea. In both cases, the initial position, the velocity, and the direction of movement are known. The new position can be derived from that information (see Barlow, 1964, for a more formal analysis). Potegal (1982) suggested that moving observers can use vestibular information to monitor the distance and direction from the starting point to update their internal representation of their position in space.

Evidence for this hypothesis comes primarily from work that uses a passive-transport-and-return (PTR) paradigm. Typically, a subject with occluded vision is transported away from the starting point along a complex trajectory. When released, the subject must return to the starting point. Performance of this task requires use of the updating process mentioned earlier. Beritoff (1966) found that normal cats, dogs, and humans could perform such a task, either with or without vision, but labyrinthine-deficient humans could not. Barlow (1970) replicated Beritoff's findings with labyrinthine-deficient humans. In contrast, Worchel (1952) found that labyrinthine-deficient humans could perform a PTR task. He suggested that kinesthetic cues might have been used.

Studies of laboratory rats have resolved the controversy. Miller, Barnett, and Potegal (1978) used a PTR task in which the rat was enclosed in a toy truck in a square, black room. Evenly spaced around the walls of the room were eight identical water spouts. The rat was transported away from each of the spouts, in pseudorandomized order, along successively longer right-angle trajectories. The rat was rewarded if he returned to the spout from which he had been pulled without going to any other spout in the room. Sighted and blinded rats could perform the task. Sighted and blinded rats with VNC lesions, however, were significantly impaired on performance of the task. Sighted rats with lesions of the kinesthetic centers were somewhat impaired (Miller, Potegal, & Abraham, 1981, 1983). Rats with selective lesions of the peripheral vestibular apparatus were significantly impaired on postoperative retention of the task, although they were unimpaired on postoperative acquisition of a relative orientation task (Cohen & Potegal, 1983).

The vestibular role in navigation seems to be limited to short distances. Sighted and blind humans performing a PTR task were observed to make successively greater errors as the trajectory lengths increased (Worchel, 1951). Rats made similar errors (Cohen, 1986). This limitation to short-distance navigation may be due to the lack of an error detection device intrinsic to the neural integrator that transforms the velocity signal to a position signal. Although the cerebellum may be involved in error detection for the oculomotor system (Miles & Lisberger, 1981), no evidence exists to suggest a similar cerebellar role in the proposed vestibular updating system. Consequently, small calculation errors that accumulate during the updating process can produce large behavioral errors.

The significance of these data to impaired populations is not yet known. Patients with multiple handicaps, such as those with multiple sensory loss and movement disorders, may use vestibular information to orient themselves when transported over short distances, such as across the ward from sleeping to therapy areas. It is also possible that blind patients can use this kind of information to supplement other spatial systems, as Worchel's (1951, 1952) data suggested. Normal adults might use this system when navigating in the dark through a limited distance, as when one wakes up in the middle of the night to close a window. No data exist to confirm or deny these theories; therefore, these ideas remain speculative.

Summary

At all levels of the system beyond the primary vestibular afferents, vestibular signals are combined with signals from other sources. This combined output is involved with postural and oculomotor control and spatial orientation.

The brain stem pathways are involved with generation of the conjugate eye movements. Labyrinthine signals are required for the VOR and contribute to eye-hand coordination with all eye movements. Oculomotor control is affected by vestibular deficits. For example, loss of labyrinthine signals causes loss of the VOR. Because the VOR stabilizes gaze during head movement, patients with vestibular deficits may have blurred vision with head movement, including postural transitions, or translations of the entire body through space when ambulating. In the absence of vestibular feedback, patients may also have facilitation of the VNC-mediated visual influence on posture. Likewise, vestibular loss may impair spatial orientation when meaningful visual information is not available. These deficits may be manifested by the patient's difficulty in performing such tasks as reaching for an object, placing body parts when transferring from a bed to a chair, or navigating short distances in the dark.

Epilogue

The vestibular system may be regarded as a spatial orientation system. The vestibular postural reflexes help the animal, or person, to maintain the upright posture—in other words, to orient to the gravitational vertical. These postural mechanisms operate in response to motion, as when the head swings during locomotion or when the body is pushed over by an
obstacle or by a strong gust of wind. Similarly, these reflexes are used in recovery from a fall or from a sudden position change. Knowledge of the location of the gravitational vertical helps the observer to define the earth-horizontal axis, which is useful to look about the world.

The vestibular oculomotor reflexes enable the moving observer to maintain a stable gaze when watching a stationary or moving object. This function could be particularly useful for an animal moving toward its prey or a human moving toward the refrigerator. The vestibular spatial mechanism aids in remembering the location of the starting point. So, having chased, fought, and captured its prey, the hungry animal can return to its home nest with its dinner, using vestibularly guided navigation to locate the nest. Likewise, sleepy humans, having satisfied their hunger with a late-night snack, can return to bed for a good night’s sleep.

Acknowledgments

We would like to thank Dr. Bernard Cohen, Mount Sinai School of Medicine, and Dr. Victor Matsuo, Northwestern University Medical School, for their helpful comments on this paper.

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The American Journal of Occupational Therapy

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