Advances in the Treatment of Vestibular Disorders

This article discusses the pathophysiology, evidence of treatment efficacy, and factors that contribute to improved treatment outcome in three different vestibular disorders. In patients with unilateral and bilateral vestibular loss, recent research suggests that customized, supervised exercises facilitate recovery of postural stability. These exercises are based on knowledge of normal vestibular function as well as on our understanding of the various compensatory mechanisms that can contribute to recovery. Recognizing the limitations of these compensatory mechanisms as substitutes for lost vestibular function is important in establishing treatment goals. Treatment of patients with benign paroxysmal positional vertigo (BPPV) is based on the identification of the specific canal involved and the anatomy of the labyrinth. Although patients with BPPV primarily experience brief episodes of vertigo, this disorder is also associated with postural instability, which may not resolve with remission of the positional vertigo. [Herdman SJ. Advances in the treatment of vestibular disorders. *Phys Ther.* 1997;77:602–618.]

Key Words: *Balance, Vestibular system.*

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The use of exercises in the rehabilitation of patients with vestibular disorders, a relatively old treatment approach dating to the 1940s, has for many years been based on anecdotal evidence of improved function with treatment. Recent studies[1–12] have led to a refinement of the treatments used and have documented treatment efficacy in a variety of vestibular disorders. This article will explore these advances in treatment of patients with the two most common vestibular problems who are referred for rehabilitation: (1) vestibular paresis or loss and (2) benign paroxysmal positional vertigo (BPPV).

Vestibular Paresis and Loss

The term "vestibular paresis" implies a loss of vestibular hair cells or vestibular neurons and therefore a decrease in the vestibular system's response to head movement. Unilateral and bilateral vestibular deficits both result in postural instability, disequilibrium, and oscillopsia, although these problems are usually more severe in patients with bilateral vestibular loss. Acute unilateral deficits also result in vertigo, spontaneous nystagmus, and skew deviation (vertical malalignment of the eyes due to an abnormal otolith input), which are the result of the asymmetry in the tonic firing of the vestibular neurons. This asymmetry recovers spontaneously, usually within a few days of onset, and exercises do not affect the course of recovery in patients with this condition.[13] Patients with vestibular loss usually do not have a history of vertigo or nystagmus because in most cases the vestibular loss is symmetrical. Postural instability, disequilibrium, and oscillopsia are due to the decreased gain of the vestibular response to head movement. *Gain* refers to the relationship of the input signal (in this case, head movement) to the output (the eye movement generated or postural stability). Ideally, the gain of the vestibular system would be "1." Recovery of postural stability and of vestibulo-ocular reflex (VOR) gain following vestibular loss requires both visual inputs and movement, and there is evidence that if visuomotor experience is delayed, the recovery period will be more prolonged.[13,14] This recovery may occur through an increase in the gain of the remaining vestibular response, but the substitution of other sensory and motor strategies is a major part of recovery in these patients. This recovery can be facilitated through the use of exercises.[1–3,15–23]

Treatment goals, I believe, should be specific to the patient's problems and should reflect both the direct effect of the vestibular paresis or loss and the indirect effects of the inactivity that accompanies these vestibular problems. These goals may include decreasing complaints of disequilibrium, improving postural stability in stance and during ambulation and other functional activities, improving gaze stability during head movements, and improving tolerance for activity (endurance). Effective treatment of people with vestibular deficits has recently been demonstrated in several controlled studies.[1–5]

Evidence That Exercise Facilitates Recovery Following Vestibular Loss

Although there is considerable anecdotal support that exercises are important in the rehabilitation of patients with vestibular problems,[17–23] only recently have prospective, controlled studies provided evidence that vestibular rehabilitation techniques are beneficial for patients with unilateral or bilateral vestibular losses.[1–3] These studies have primarily emphasized changes in postural stability, in disability, and in patient complaints as a result of these interventions. Horak et al[1] reported that patients with chronic, unilateral vestibular deficits had improved postural stability after a 6-week course of

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Table 1.
Factors Influencing Recovery Following Vestibular Paresis or Loss

Positive Influences	Negative Influences
Customized,[a] supervised exercises	Generic, unsupervised exercises
Stable unilateral vestibular deficits	Fluctuating disorders (eg, Ménière's disease)
Less severe initial disability	Head injury
Recent onset	Mixed central and peripheral lesion
	Vestibular suppressant medications

[a] Customized based on problems identified in the clinical examination.

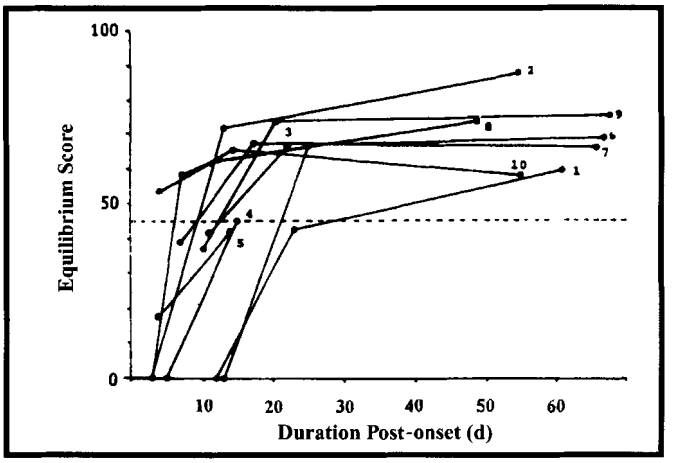

Figure 1.
Recovery of postural stability following unilateral vestibular loss in 10 patients as measured using computerized dynamic posturography. The equilibrium scores reflect anteroposterior sway, with 100 indicating perfect stability and 0 indicating loss of balance. The scores shown here are the averages from tests in which both visual and somatosensory cues were altered (tests 5 and 6; three trials each). Patients were tested as early as the third day after onset of the unilateral vestibular loss. Most patients recovered to within normal limits (above dotted line) by the third week from onset. (Adapted from Fetter et al[31] with permission.)

vestibular exercises compared with a group of patients performing general conditioning exercises. The exercises were customized for each patient and included balance and gait exercises as well as exercises incorporating combinations of head and eye movements. Krebs et al[2] studied the effectiveness of vestibular exercises on postural stability during functional activities in patients with chronic bilateral vestibular deficits. They used a placebo-controlled trial and found that patients performing customized exercises had better stability while walking and during stair climbing and were able to walk faster than patients performing isometric and conditioning exercises. Their vestibular exercise program also consisted of both balance and gait training and combinations of head and eye movements. Vestibular adaptation exercises have also been shown to produce a more rapid recovery during the acute stage following unilateral vestibular loss.[3] Adaptation exercises initiated on the third day following resection of acoustic neuroma resulted in improved postural stability in stance and during ambulation and in a decrease in the perception of disequilibrium when compared with the findings for a control group of patients.[3]

Factors influencing treatment efficacy. Several factors have been identified that affect the potential for recovery in patients with vestibular deficits (Tab. 1). The exercises used today in the treatment of patients with vestibular loss should be customized to the patient based on the results of the examination. Customized, supervised exercises for patients with vestibular disorders result in more patients achieving complete remission of symptoms (85%) compared with a generic, unsupervised exercise program that patients perform at home (64%).[4,24] Szturm et al[5] reported similar findings in a comparison of vestibular adaptation exercises with Cawthorne-Cooksey exercises,[25] which were not customized to the individual patient.

Studies suggest that patients with stable unilateral vestibular deficits (as opposed to fluctuating disorders such as Ménière's disease), symptoms provoked by movement,[4] less severe initial disability, or a more recent time of onset will have a better recovery. The effect of concurrent head injury on recovery of function is not clear. Shepard et al[26] have shown that patients with head injury tend to have a poorer prognosis, perhaps because the structures involved with central adaptation and compensation may be impaired, although many patients show a decrease in symptoms with treatment. Shepard et al[27] found that a more prolonged period of therapy was necessary if the patient had a mixed central and peripheral lesion, used vestibular suppressant medications, or had increased long-latency responses to sudden perturbations of the support surface. Shepard et al did not discuss the importance of the increased long-latency responses, but these responses may be related to the presence of central nervous system lesions in some patients. Keim et al,[28] however, reported little difference in recovery between patients with central vestibular deficits and patients with peripheral vestibular deficits. The effect of age on the rate and final level of recovery also is not clear, although studies[27,29] indicate that improvement occurs in elderly patients.

Mechanisms of Compensation Following Vestibular Loss

Understanding compensatory mechanisms and their limitations in improving postural and gaze stability should lead to more effective treatment of these patients and to a better understanding of the potential for functional recovery. The primary mechanisms of recovery of postural stability appear to be improved vestibular responses[3,30,31] and increased reliance on visual[32,33] and somatosensory cues.[30,34,35] A variety of mechanisms contribute to the recovery of gaze stability following unilat-

eral and bilateral vestibular loss. These mechanisms include recovery of the VOR itself,[36,37] alterations in saccadic amplitude and direction,[38,39] potentiation of the cervico-ocular reflex (COR),[39-42] central preprogramming,[39,43] visual tracking mechanisms,[39] and limiting of head movement and activity.[41]

Postural Stability

Postural stability is maintained through complex interactions among sensory inputs, biomechanical constraints, and voluntary motor control. Three systems—visual, vestibular, and somatosensory—provide the main inputs to the automatic postural reflexes and contribute to voluntary postural control. Although there is considerable redundancy in the contributions of the different sensory cues to postural stability, each sensory input appears to have an optimal stimulus frequency at which it acts to stabilize balance. The vestibular system functions across both low and high frequencies of input; however, neither visual nor somatosensory cues stabilize balance at the high frequencies.[44-46]

Recovery of vestibular function. Recovery of the vestibulospinal system is difficult to measure because it is difficult to isolate that system from systems associated with postural control. Several researchers[3,30,31] have noted the recovery of the ability of patients with unilateral vestibular loss to maintain their balance when both visual and somatosensory cues are altered (Fig. 1). This finding may indicate an improved ability of patients to use remaining vestibular signals to maintain their balance, or it may be due to recovery of some vestibular neurons or to adaptation of the vestibular system.

Adaptation implies a long-term change in how the vestibular system responds to head movement. It is important to note that although adaptive capabilities decrease with aging, older individuals have considerable abilities to modify the gain of their vestibular responses.[47] Most studies of vestibular adaptation have been performed using the VOR as the outcome measure, in part because it is far easier to measure the VOR than to measure the vestibulo-spinal reflex and because the "error signal" that induces a change in the gain of the vestibular response appears to be retinal slip, that is, movement of an image across the retina.[48-50] More recent studies indicate that eye movements are important as well.[51] This error signal is processed through the cerebellum and the vestibular nuclei and changes the output of the vestibular system (Fig. 2).[52]

Several concepts about adaptation of the vestibular system are particularly important. First, adaptation is context specific. This concept was demonstrated several years ago for frequency of head movement.[53] When attempts are made to artificially increase VOR gain by having subjects move their head while wearing magnifying glasses, the greatest changes in gain occur at the training frequency, with smaller changes in gain occurring at other frequencies.[53] These results suggest that vestibular adaptation exercises should be performed across a wide range of frequencies to be most effective. More recently, orientation of the head during training has been shown to be a factor, presumably because the otolith input influences the effect of training.[54]

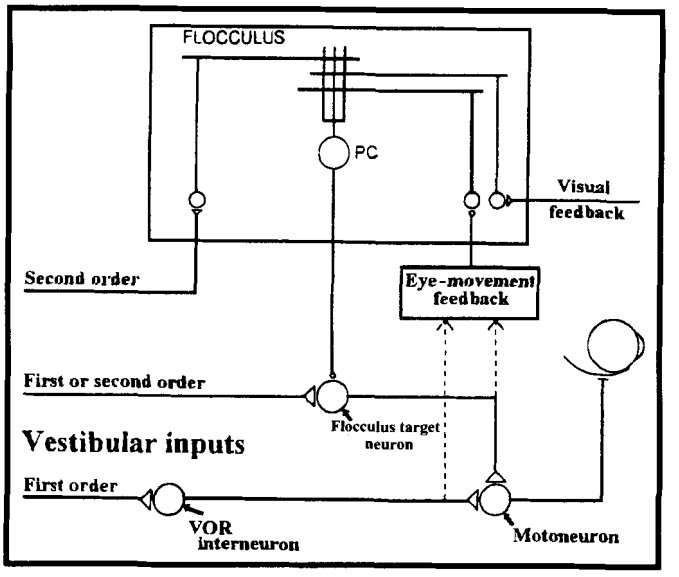

Figure 2.
Feedback loop that provides flocculus with the error signal necessary to produce a change in the gain of the vestibulo-ocular reflex (VOR). Lisberger[52] has suggested that this mechanism involves the flocculus and flocculus target neurons in the vestibular nucleus. The Purkinje cells (PC) in the flocculus receive visual, eye movement, and vestibular signals. (Adapted from Lisberger[52] with permission.)

Second, although VOR gain changes occur within minutes, it takes time to induce persistent changes in VOR gain.[55] This finding has been aptly demonstrated by subjects without vestibular disorders wearing reversing prism glasses.[55] When the head turns, the reversing prisms cause the visual environment to appear to move in the same direction as the head movement instead of opposite to the direction of head movement. Within a few minutes after wearing the reversing prisms, VOR gain begins to decrease to reduce this effect. If the glasses are then removed, VOR gain rapidly returns to normal. The longer the exposure to the altered visual input, however, the longer the VOR gain changes are retained.

Third, not all head movements appear to result in adaptation. Changes in VOR gain have been demonstrated for horizontal (yaw) and vertical (pitch) head movements in humans, but there is little adaptive capability associated with head movements in the roll plane (Leigh RJ, personal communication). Although the

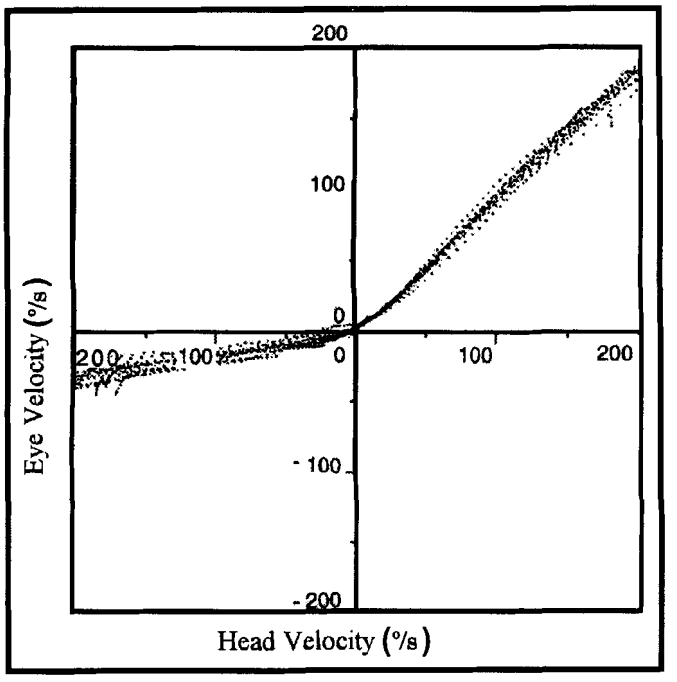

Figure 3.
Asymmetry in vestibulo-ocular reflex gain in patient with unilateral vestibular deficit becomes apparent at high speeds. Eye speed (Y-axis) is plotted against head speed (X-axis) for head rotations horizontally at speeds of up to 200°/s toward (positive numbers) and away from (negative numbers) the normal side. (Adapted from Halmagyi et al[66] with permission.)

cliché "If it makes you dizzy, it's good for you" is often used as a criterion for vestibular exercises, this may not always be appropriate; repeated head movements in the roll plane certainly make patients as well as subjects without vestibular disorders "dizzy" but will not substantially alter their long-term vestibular responses. Therapists need to take these factors into consideration when developing exercise programs for their patients.

Substitution of other mechanisms. Somatosensory cues: Black et al[30] showed that in the acute stage following resection of acoustic neuromas, subjects had decreased postural stability when somatosensory feedback from the lower extremities was distorted. This finding suggests that patients with unilateral vestibular loss may rely on somatosensory cues during that early stage of recovery. Bles and colleagues[34,35] have shown that during the course of recovery, patients with complete bilateral vestibular loss change how they rely on sensory cues for stability. Initially, they rely on visual cues as a substitute for the loss of vestibular cues, but over a 2-year period, these patients increase their reliance on somatosensory cues.

Visual cues: Visual inputs provide several different cues that affect postural stability. As a person sways, even in quiet stance, retinal slip information is used to determine body movement relative to environmental movement.[56] Changes in image size and retinal disparity, which would occur with fore-aft sway, are additional cues. Visual stabilization of balance appears to be primarily dependent on foveal vision. Some studies[32,33] indicate that patients with unilateral vestibular loss become less stable when visual cues are removed. Patients with bilateral vestibular loss also initially rely on visual cues for stability; however, this would not seem to be a particularly successful strategy during walking. Without the vestibular system, the eyes are not stable the during the head movement that occurs during ambulation and visual acuity degrades. Even at a visual acuity of 20/40, postural stability is decreased.[56,57] Patients may attempt to resolve this problem by decreasing head movements during activities such as walking.

Limitations of substitution. The stimulus frequency ranges at which somatosensory and visual cues contribute to postural stability are known only for stance and not for ambulation. Somatosensory cues appear to contribute postural stability between 1 and 3 Hz[44] and visual cues between 0.1 and 1 Hz.[45,46] The vestibular system operates over a wide range, with otolith signals contributing to postural stability at lower frequencies (<1 Hz) and the semicircular canals contributing to postural stability at higher frequencies (up to 5 Hz). Somatosensory and visual inputs, because they do not operate across the same frequencies as vestibular signals, would be only partially successful at substituting for lost vestibular cues.

When the body is relatively stable, such as while sitting or standing quietly, there is little head movement and visual and somatosensory cues are sufficient to maintain postural stability in patients with vestibular loss.[58] During locomotion, however, the frequency of head movements exceeds the compensatory ability of these systems. The dominant frequency of head movements in subjects without vestibular impairments walking in place ranges from 0.7 to 1.2 Hz for horizontal head movements and from 0.9 to 5.1 Hz for vertical head movements.[59] During running in place, the frequency of horizontal head movements increases to as high as 1.9 Hz and the frequency of vertical head movements increases to 5.8 Hz, well within the frequency range of vestibular function.[59–61] Similarly, the velocity of head movements during ambulation usually is well within the velocity range in which the normal vestibular system works (<300°/s).

There seems to a natural course as to which sensory cues are used to maintain postural stability at different stages following vestibular loss. Although visual cues become increasingly important, it is probably not optimal to foster visual dependency (eg, by teaching patients to fixate on a stationary object and to decrease their head

movements while walking) because that may limit the patients' ability to learn to use remaining vestibular function and somatosensory cues. Additionally, patients may voluntarily restrict head movements. Ultimately, restricting head movements would result in decreased tolerance for functional activities and still would not provide a mechanism for postural stability during head movements. Central preprogramming of postural responses would be an effective strategy only when the required movement can be anticipated.

Gaze Stability

Gaze stability refers to the stabilization of the eye in space in order to see clearly. The purpose of the VOR is to maintain gaze stability during head movements. If the VOR and other mechanisms cannot produce an appropriate compensatory eye movement, movement of the head will cause substantial retinal slip and therefore degradation of visual acuity. In reality, compensatory eye movements do not have to match head movements perfectly because 2° to 4°/s of retinal slip can be tolerated without degradation of visual acuity.[60,62–64] The consequences of poor gaze stability are visual blurring (poor visual acuity during head movements) and also poor postural stability because the contribution of visual cues to postural stability would decrease with decrements in visual acuity.[56,57] Exercises that improve gaze stability, therefore, may help to improve postural stability by improving the patient's ability to use visual cues for balance.

Recovery of vestibular function. During the acute stage following unilateral vestibular loss, VOR gain is as low as 0.25 and 0.5 for rotation of the head toward and away from the involved side[36,37] (normal VOR gain is usually between 0.5 and 0.8). The gain of the horizontal VOR recovers quickly and is within normal limits in 1 to 3 months for slow head rotations.[36,37] The gain of the vertical VOR to slow head movements is reduced symmetrically by approximately 66%. There is some evidence that vertical VOR recovers more slowly than does horizontal VOR.[65] When patients are tested using rapid head thrusts or unpredictable head movements, however, there is no improvement in VOR gain for head movements toward the involved side, even 1 year after onset.[66] The poor compensation for rapid head thrusts toward the deficit is predicted by Ewald's Second Law, which states that the response of the horizontal canals is less efficient for ampullofugal (contralateral) head rotation than for ampullopetal (ipsilateral) head rotation.[55] This horizontal canal response is due to the discharge properties of the vestibular nerve, which can be increased above its resting rate more than it can be decreased. (The firing rate of neurons can only be decreased to zero.) In patients with unilateral vestibular deficits, a marked asymmetry in VOR gain persists to head movements exceeding 100°/s (Fig. 3). The VOR gain improves in some patients with bilateral vestibular loss but, again, only to relatively low-speed head movements.[67] As with the recovery of postural stability, the recovery of VOR gain may be due to the recovery of some of the vestibular hair cells or neurons themselves or to the adaptive capability of the remaining vestibular system.

Table 2.
Frequency of Complaints in 100 Patients With Benign Paroxysmal Positional Vertigo[a]

Complaint	Frequency (%)
Poor balance	57
Sense of rotation (vertigo)	53
Trouble walking	48
Light-headedness	42
Nausea	35
Queasiness	29
Spinning inside head	29
Sense of tilt	24
Sweating	22
Sense of floating	22
Blurred vision	15
Jumping vision	13

[a] Patients could indicate more than one complaint. (Unpublished data; RJ Tusa, MD, Dizziness and Eye Movement Center, University of Miami, Miami, Fla.)

Substitution of other mechanisms in gaze stability. Modification of saccades: Patients with bilateral vestibular deficits may decrease the amplitude of saccadic eye movements and make both slow-phase (<60°/s) and saccadic eye movements in the same direction during combined eye and head movements.[39] Both of these strategies assist in moving the eye to the target when the head moves. Similarly, patients with unilateral vestibular loss may make saccadic eye movements in the same direction as slow-phase eye movements to augment an inadequate VOR.[68] Although this strategy may enable patients to visually "capture" a target once the saccade is completed, they still would not be able to see during the saccadic eye movement.

Cervico-ocular reflex: Somatosensory receptors in ligaments and joints in the upper cervical region project to the contralateral vestibular nuclei and can produce compensatory eye movements (ie, CORs) that parallel the VOR.[69] In individuals without vestibular disorders, the gain of the COR is variable and often negligible.[43,70,71] When the COR is present in these persons, it operates only at low frequencies (<0.1). The gain of the COR in patients with bilateral vestibular loss can be as great as 0.25, and the COR produces compensatory eye movements across a wider range of frequencies (up to 0.3 Hz).[39,40] There is little evidence that the COR is altered in patients with unilateral vestibular loss.

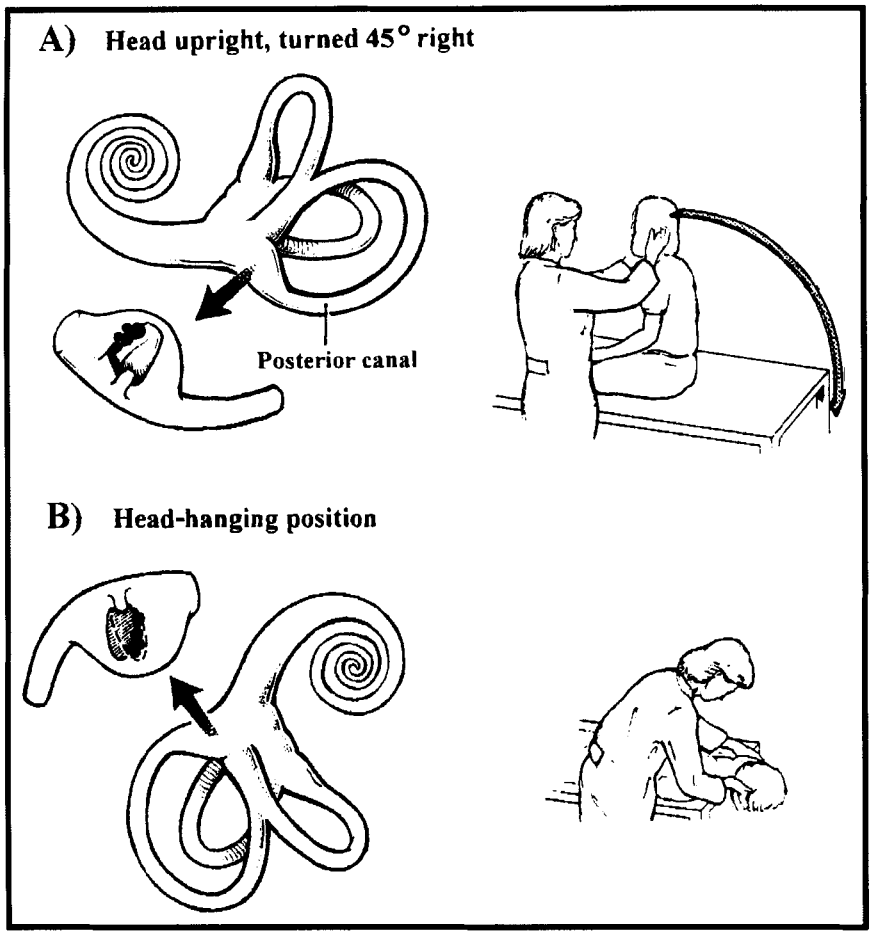

Figure 4.
Debris adhering to the cupula of the semicircular canal (shown here for posterior canal) will cause the cupula to be deflected when the person is moved from the sitting position (A) to the head-hanging position (B) in the Hallpike-Dix maneuver.

Central preprogramming: Central preprogramming of compensatory eye movements to improve gaze stability has been demonstrated primarily by comparing the gains of the compensatory eye movements during active and passive head movements.[39,43,70] Central preprogramming is effective in subjects without vestibular disorders and in people with unilateral and bilateral vestibular loss who have no central nervous system lesion.

Visual tracking: Visual tracking does not appear to be an important compensatory mechanism in the recovery of gaze stability. Kasai and Zee[39] reported that the smooth pursuit system works at the upper end of the normal range in patients with bilateral vestibular loss, that is, a gain of 1 at frequencies of less than 1 Hz and at speeds of 20° to 30°/s.

Decreased head movements: Some patients limit head movements in order to minimize oscillopsia.[41] This "learned disuse" would not be a particularly useful strategy for improving gaze stability because it would limit everyday activities and still would not provide a mechanism for seeing clearly during head movements.

Limitations of substitution. When the body is relatively stable, smooth-pursuit eye movements and the COR are sufficient to maintain gaze stability in patients with vestibular loss.[72–74] During locomotion, however, the speed and frequency of head movements exceed the compensatory ability of smooth-pursuit eye movements (<60°/s and <1 Hz) and the COR (0.3 Hz).[39,59,69] Therefore, neither tracking eye movements nor the COR will substitute completely for the lost vestibular function. Additionally, during ambulation, head movements do not occur in a predictable manner but are random. This "randomness" is an important factor because under these conditions, predictive eye movements (central preprogramming) will not help to stabilize gaze and a degradation of visual acuity would be expected in patients with vestibular loss.

Benign Paroxysmal Positional Vertigo

Benign paroxysmal positional vertigo is a biomechanical problem in which one or more of the semicircular canals is inappropriately excited, resulting in brief episodes of vertigo and in disequilibrium.[75] This disorder occurs in adults of all ages, although it is more common among older individuals, and accounts for 160,000 new cases of dizziness each year.[76,77] It is the most common cause of vertigo in patients with peripheral vestibular dysfunction and accounts for 20% to 30% of all patients seen for vertigo.[77] This disorder, therefore, represents a widespread problem, one that is, fortunately, easily treated. For patients with BPPV, the goals would be to achieve complete remission from vertigo and to improve postural stability.

Patients with BPPV experience a brief period of vertigo and nystagmus when the head is moved into particular positions. Diagnosis is based on characteristic findings, including: (1) a latency of 1 or more seconds after the head is moved into the provoking position before the onset of the vertigo and nystagmus, (2) a gradual reduction in the vertigo and nystagmus, with a duration of less than 60 seconds, (3) characteristic nystagmus, (4) reversal of the nystagmus, and a recurrence of vertigo, when the person returns to a sitting position,

and (5) decreased intensity of the vertigo with repeated movement of the person into the provoking position.[75] Although in some patients there is a preceding episode of vestibular neuronitis (15%) or a history of recent head injury (18%), in most patients with BPPV, the onset is inexplicable.[75]

In addition to complaints of vertigo, many patients with BPPV identify postural instability as a major problem (Tab. 2).[78,79] Patients have reported generalized disequilibrium, unsteady gait, sensitivity to head movements and to linear accelerations, and falls.[6,80] Black and Nashner[78] reported that patients with BPPV have increased postural sway when visual feedback is altered but that they have normal postural sway when visual feedback is absent. The authors suggested that these findings indicate that patients with BPPV have developed an inability to correctly weigh which sensory cue they should rely on when maintaining balance. Voorhees,[79] however, failed to find the same postural problems and instead found that patients with BPPV had difficulty maintaining their balance when somatosensory cues were altered and visual cues were either altered or absent. Voorhees concluded that patients with BPPV have difficulty maintaining their balance using vestibular cues. One explanation for the differences in the findings of these two studies is the incidence of patients with head injury in the study by Black and Nashner[78] but not in the study by Voorhees.[79] In patients with head injuries, central nervous system damage or horizontal canal involvement secondary to the head injury could contribute to the postural instability.

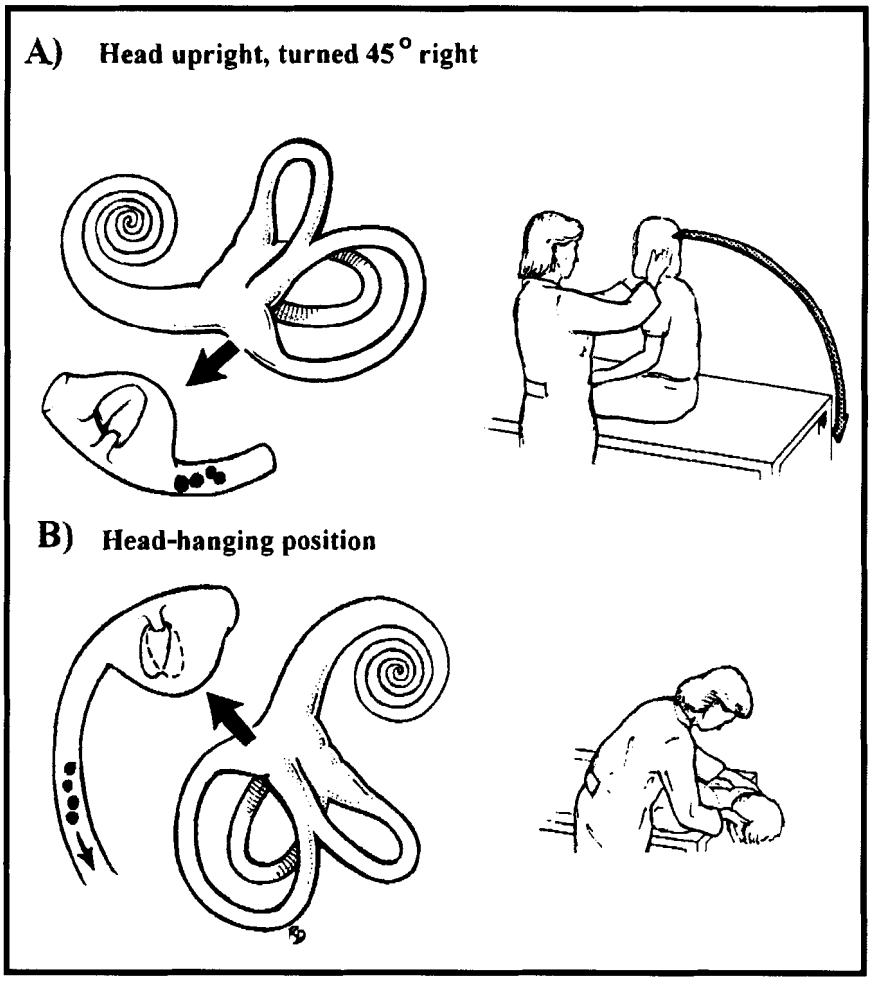

Figure 5.
Debris floating freely in the long arm of the semicircular canal (shown here for posterior canal) will drift to the most dependent portion of the canal when the person is moved from the sitting position (A) to the head-hanging position (B) in the Hallpike-Dix maneuver and therefore will cause the cupula to be deflected.

Postural instability may also occur if the BPPV is due to disruption of the anterior vestibular artery flow. The utricle's anterior and horizontal canals are supplied by the anterior vestibular artery and the saccule, and its posterior canal is supplied by the posterior vestibular artery. Disruption of the anterior vestibular artery would result in degeneration of the utricle, and therefore of the otoconia, and in horizontal canal hypofunction, but the posterior canal would still function. Cellular debris, probably otoconia, could float from the utricle into the still-functioning posterior canal, producing the symptoms of BPPV. Postural instability in patients with BPPV may be due to the abnormal signal from the semicircular canal or to an asymmetry in the signals from the utricles due to the loss of otoconia.[80,81] Identification of the underlying cause of the balance problems and appropriate treatment are critical to the successful management of these patients. In cases where the instability is due to an abnormal signal from the affected semicircular canal, new developments in our understanding of the pathophysiology of BPPV and new treatment approaches should result in more effective treatment.

Pathophysiological Basis of BPPV

Two different mechanisms have been proposed to explain the signs and symptoms of BPPV. One mechanism, "cupulolithiasis," refers to debris adhering to the cupula of the affected canal.[82] With changes in head position, gravity will cause the weighted cupula to be displaced (Fig. 4), resulting in nystagmus and vertigo. Several misgivings have been raised concerning this proposed mechanism. First, although debris has been

Table 3.
Considerations in Choice of Treatment

Treatment	Type of Benign Paroxysmal Positional Vertigo	Appropriate Canal	Consideration
Brandt-Daroff habituation	Cupulolithiasis	Posterior	Patient adherence
Liberatory maneuver	Cupulolithiasis or canalithiasis	Posterior, horizontal[a]	Neck extension, difficulty with rapid movement
Canalith repositioning maneuver	Canalithiasis	Posterior, anterior, horizontal[a]	Conversion to different canal, neck extension

[a] Modified maneuvers.

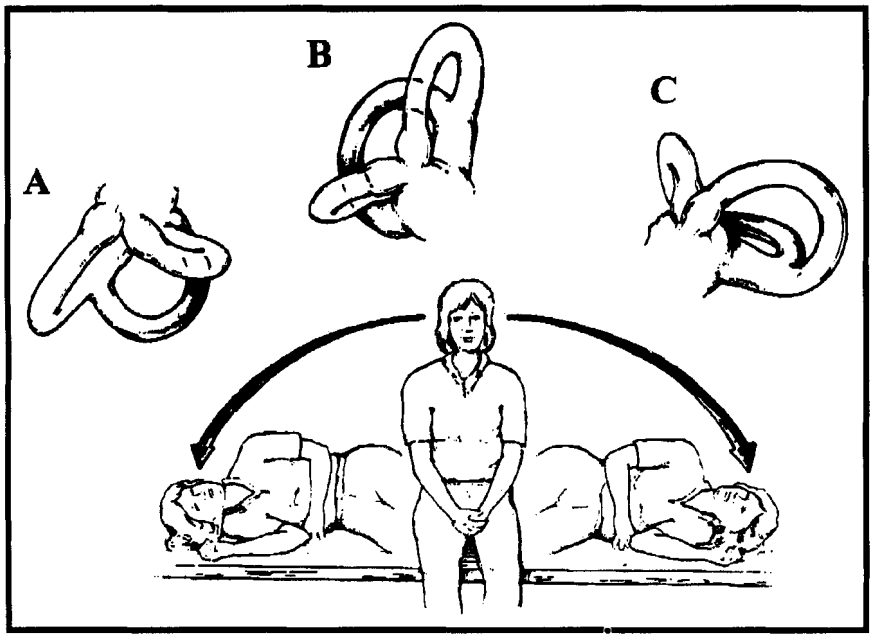

Figure 6.
Brandt-Daroff habituation exercises: The patient is first positioned sitting and then rapidly moves into the side-lying position (A). Torsional nystagmus may occur with the onset of the vertigo. The severity of the vertigo will be directly related to how rapidly the patient moves into the provoking position. The patient stays in that position until the vertigo stops, waits 30 seconds, and then sits up (B). Moving to the sitting position will usually result in vertigo, although this "rebound effect" will be less severe and of a shorter duration. Nystagmus, if it reoccurs, will be in the opposite direction. The patient remains in the upright position for 30 seconds and then moves rapidly into the mirror-image position on the other side (C), stays there for 30 seconds, and then sits up. The patient then repeats the entire maneuver 5 to 20 times, depending on the tolerance of the patient for vertigo and any accompanying nausea, or until the vertigo no longer occurs. The entire sequence is repeated three times a day until the patient has 2 consecutive days without vertigo. (Adapted from Brandt and Daroff.[86])

found adhering to the cupula in persons with a history of BPPV, similar deposits have been noted in persons without a history of positional vertigo.[83] Second, if the debris were adhering to the cupula, the nystagmus should occur as soon as the person is in the provoking position, but often there is a delay of onset of several seconds or more. Of even greater concern is that the deflection of the cupula should result in nystagmus that persists for as long as the person is kept in the provoking position, although some decrement of the nystagmus intensity may occur due to central adaptation.[84] One of the characteristics of BPPV is that the duration of the nystagmus, and of the vertigo, is typically brief, lasting less than 60 seconds.

The second mechanism, "canalithiasis," refers to debris floating within the endolymph of the semicircular canal.[7,85] When the head is moved into a position in the plane of the affected semicircular canal, the debris will move into the most dependent portion of the canal. This movement will cause the endolymph, and therefore the cupula, to move, producing vertigo and nystagmus (Fig. 5). The brief delay before the onset of the nystagmus and vertigo may be accounted for by the time it takes to overcome the inertia of the cupula. Once the debris has stopped moving, the cupula will return to its normal position within the ampulla and the nystagmus and vertigo will stop, accounting for the brief duration of the provoked signs and symptoms of BPPV. Support for this theory comes from the direct observation of debris in the affected canal of persons with BPPV.[8]

Several factors should be taken into consideration in choosing the appropriate treatment for patients with BPPV. These factors include whether the person is likely to have cupulolithiasis or canalithiasis, which canal is involved, comorbid problems, and the ability of the patient to adhere to the requirements of the treatment (Tab. 3).

Evidence That Treatment Facilitates Recovery in BPPV

Treatments based on cupulolithiasis. The Brandt-Daroff exercises were developed based on the theory that the signs and symptoms of BPPV are due to cupulolithiasis and that the posterior canal is affected (Fig. 6).[86] These exercises originally were believed to produce habituation of the vertigo. Brandt and Daroff,[86] however, noted that the response to treatment occurred immediately in some patients, and they suggested that the debris was physi-

cally dislodged from the cupula. Given the limitations of the Brandt-Daroff exercises, it is surprising that this treatment was shown to be effective for 95% of patients with BPPV within 2 weeks.[86] One possibility is that all patients in the study had cupulolithiasis of the posterior canal. This explanation, however, would seem to be unlikely. A second possibility is that some remissions were spontaneous and were not related to the treatment. Spontaneous recovery is common in patients with BPPV,[9,87] and this factor was not controlled for in the study by Brandt and Daroff.[86]

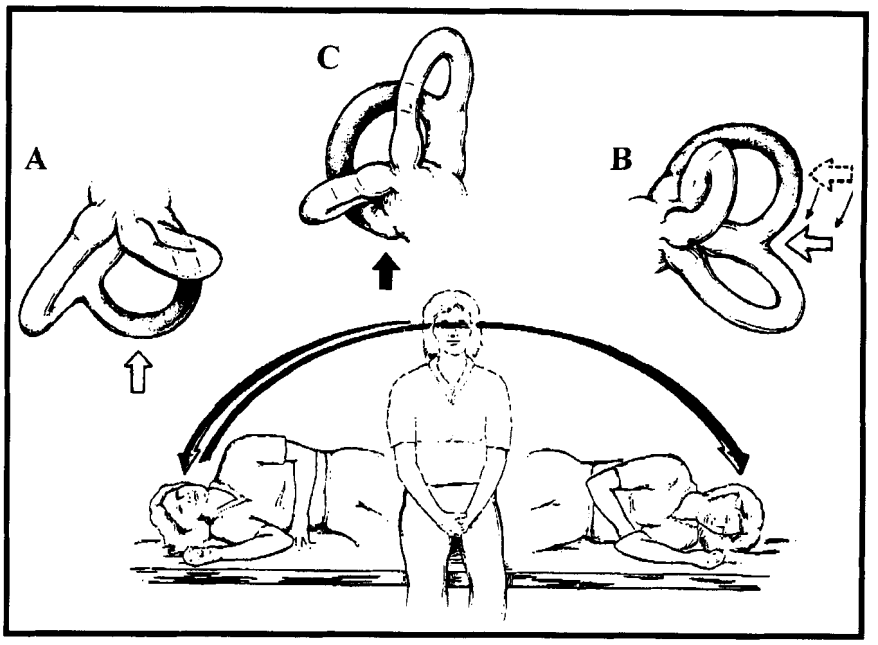

Figure 7.
Liberatory maneuver: The patient is quickly moved into the provoking side-lying position with the head turned into the plane of the posterior canal and is kept in that position for 2 to 3 minutes (A). The patient is then rapidly moved up through the sitting position and down into the opposite side-lying position, with the therapist maintaining the alignment of the neck and head on the body (B). (The face is then angled down toward the bed.) Typically, nystagmus and vertigo reappear in this second position. If the patient does not experience vertigo in this second position, the head is abruptly jostled once or twice using a small amplitude of movement, which often will provoke vertigo and nystagmus, presumably by freeing the debris. The patient stays in this position for several minutes. The patient is then slowly taken into a seated position (C). The maneuver can be repeated if the patient is symptomatic and has nystagmus when returned to the sitting position. The patient must keep the head upright for 48 hours (including while sleeping) and must avoid the provoking position for 1 week following the treatment. Although this treatment was developed based on the theory of cupulolithiasis, it may be effective in patients with canalithiasis as well. The arrows (B) indicate that the debris in the long arm of the posterior canal may move toward the common crus when the patient is moved into the opposite side-lying position. (Adapted from Herdman et al.[11])

A second treatment, also based on cupulolithiasis, is the Liberatory maneuver (Fig. 7).[9,10] This treatment is presumed to dislodge debris from the cupula of the posterior canal in patients with cupulolithiasis. The treatment may also cause debris to move through the long arm of the posterior canal, into the common crus, and into the vestibule, thus also relieving symptoms. Semont et al[9] and other authors[10,11] have reported a remission rate following this treatment of between 70% and 95%. Unfortunately, most studies have not used a control group. One innovative study[10] examined the effectiveness of the Liberatory maneuver on a series of 10 patients using the patients as their own control. The patients were first treated with the Liberatory maneuver, but on the *unaffected* side. None of the patients had any relief from their vertigo. The patients were then treated using only the post-Liberatory maneuver instructions that they were to keep the head upright for 48 hours, including sleeping in a sitting position. Again, at the end of a week, all patients were symptomatic. The patients were then treated using the Liberatory maneuver on the affected side. At the end of 1 week, all patients were symptom-free. Although the number of subjects in this study was small, the results suggest that this maneuver may be an effective treatment.

Treatment based on canalithiasis. In patients in whom the BPPV is due to canalithiasis, the most appropriate treatment appears to be the canalith repositioning maneuver (Fig. 8).[7] Some controversy exists as to the efficacy of this treatment. In the original studies,[7,11] in which 85% to 95% remissions of symptoms were reported, there were no control groups. At least some of the effects attributed to the maneuver, therefore, could have been due to spontaneous recovery. More recently, in two studies,[88,89] treatment effects were compared using untreated control groups. One research group[88] concluded that there was no difference between the remission rates of patients treated for 1 month using the canalith repositioning maneuver (n=16) and the control group (n=22). This study has been criticized because the researchers did not use the maneuver as it is commonly performed and because treatment effects were assessed 1 month after treatment, by which time recurrence of symptoms (which can occur in 10%–20% of all subjects) may have been a factor. Furthermore, advocates of the canalith repositioning maneuver suggest that one of the benefits of this treatment is the *rapid* relief of symptoms. In a different study using the canalith repositioning maneuver as originally proposed, Li[89] found that 70% of the treated group (n=27) had no nystagmus when evaluated 1 week after treatment compared with none of the untreated control group (n=23).

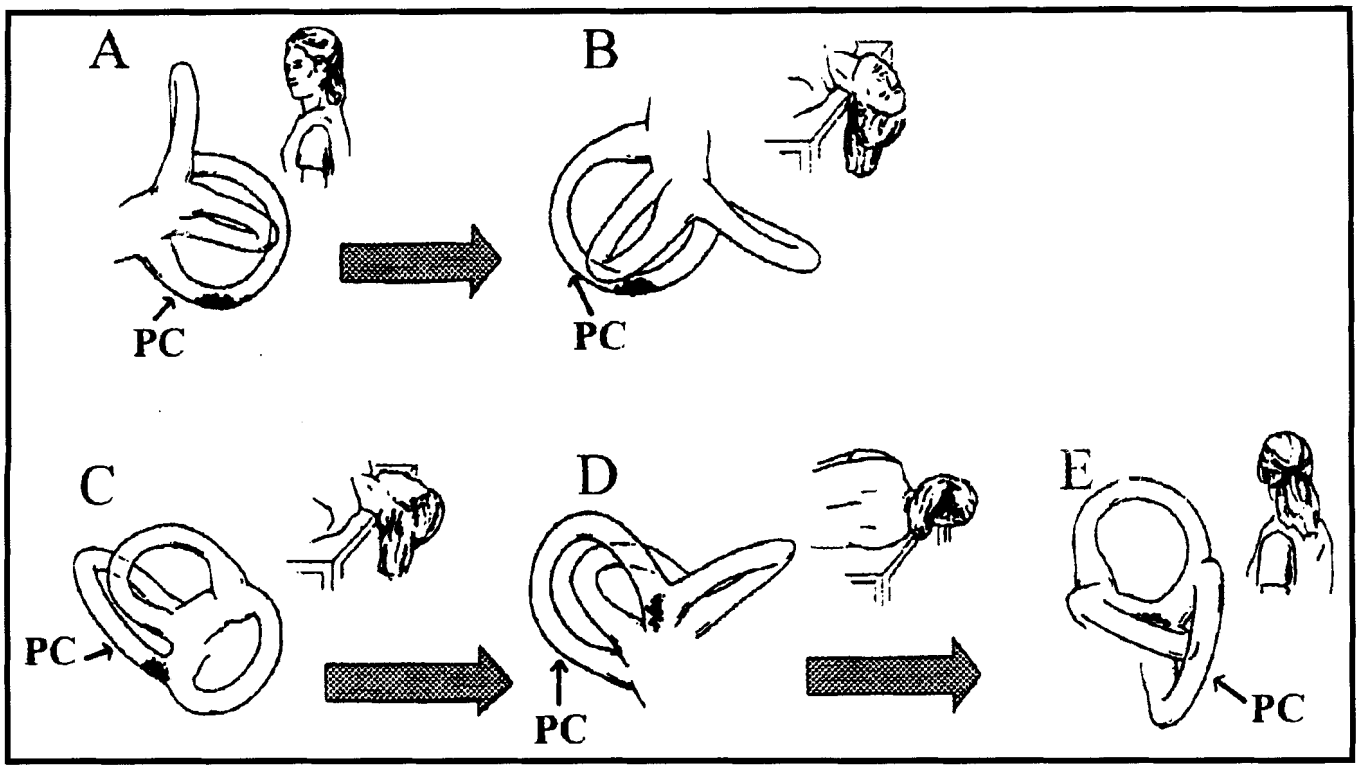

Figure 8.
Canalith repositioning maneuver for benign paroxysmal positional vertigo affecting the posterior semicircular canal on the left side. The location of debris within the posterior canal is indicated by the arrows. Note that the debris should always move away from the ampulla of the posterior canal, therefore resulting in nystagmus that always beats in the same direction. The patient is positioned sitting, and the head is turned 45 degrees toward the affected side (A) and then is quickly moved into the Hallpike-Dix position with the affected ear down (B). The patient is kept in that position for 2 to 3 minutes. The patient's head is then slowly rotated through extension until the opposite ear is down (C). Next, the patient is turned onto the side with the head rotated down 45 degrees (D). The patient remains in that position for 2 to 3 minutes and then slowly returns to the sitting position (E). Epley[7] suggests that the maneuver should be repeated until no nystagmus is observed when returning to a sitting position. The maneuver should be repeated if the direction of the nystagmus reverses. Immediately after the treatment, the patient is fitted with a soft collar and advised not to lie down, bend over, or move the head vertically for the next 48 hours. The patient is to avoid lying on the affected side for the subsequent 5 days. PC=Purkinje cells. (Adapted from Herdman and Tusa.[12])

Table 4.
Identification of Canal Involvement Based on Direction of Nystagmus During Hallpike-Dix Test[a]

Canal	Eye Muscle (Excited)	Right Hallpike-Dix Position	Reversal Phase	Return to Sitting Position
Right posterior	Ipsilateral superior oblique, contralateral inferior rectus	Upbeat, counterclockwise	Down and clockwise	Down and clockwise
Right anterior	Ipsilateral superior rectus, contralateral inferior oblique	Downbeat, counterclockwise	Up (and clockwise)	Up (and clockwise)
Left anterior	Ipsilateral superior rectus, contralateral inferior oblique	Downbeat, clockwise	Up (and counter-clockwise)	Up (and counter-clockwise)
Right horizontal	Ipsilateral medial rectus, contralateral lateral rectus	Horizontal[b]	Horizontal	Horizontal
Left horizontal	Ipsilateral medial rectus, contralateral lateral rectus	Horizontal[b]	Horizontal	Horizontal

[a] Direction of fast-phase eye movement of nystagmus generated by excitation of different canals (1) when patient is moved into the right Hallpike-Dix position, (2) during the reversal phase, and (3) after the patient returns to the sitting position. "Clockwise" and "counterclockwise" refer to direction of movement of the superior pole of the eye.
[b] Ageotropic if cupulolithiasis, geotropic if canalithiasis; Hallpike-Dix is not best provoking position; affected side is determined by intensity of symptoms.

The difference in the findings of these two studies may be due to the precise maneuver used.

Herdman et al[11] found that if patients with posterior-canal BPPV (n=30) were moved from the original provoking position (Fig. 8B) to the contralateral Hallpike-Dix position (Fig. 8C) and then returned to a sitting position, the remission rate was 50%. In comparison, the remission rate was 83% in a similar group of patients (n=30) who were rolled onto the contralateral side with the head turned 45 degrees toward the floor (Fig. 8B–D) before sitting up. This position facilitates the movement of the debris into the common crus. Li also advocated the use of a vibratory stimulus applied to the mastoid of the affected ear to presumably facilitate the movement of the debris through the canal during the treatment. The remission rate after one treatment, however, is no different from that reported by other researchers who did not use mastoid vibration during the maneuver.[87,89–91] Another variable in these studies may be which canal was involved, as the studies do not identify the direction of the nystagmus as part of the inclusion criteria.

One of the complications of the canalith repositioning maneuver is the possibility of conversion of BPPV of the posterior canal to BPPV involving the anterior or horizontal canal.[12] In a study of 85 consecutive patients with posterior-canal BPPV who were treated with the canalith repositioning maneuver, 5 patients had anterior-canal positional vertigo (n=2) or horizontal-canal positional vertigo (n=3) after undergoing the treatment.[12] The authors suggest that although movement of the debris into a different canal may occur during the treatment, it may also occur when the patient first lies down following the treatment. Observation of the direction of the nystagmus during treatment will ensure that the debris moves in the appropriate direction during the actual maneuver. For example, in posterior-canal BPPV, if the debris moves away from the cupula and toward the common crus, the nystagmus should always be in the same direction; a reversal of the nystagmus would indicate that the debris has moved toward the cupula or into the anterior canal. In patients who do not respond to treatment, careful observation of the direction of the nystagmus during reexamination is necessary to correctly identify which canal is involved.

Canal Involvement

Benign paroxysmal positional vertigo was originally thought to be a disorder of the posterior semicircular canal.[92,93] This belief was based on the direction of the nystagmus observed when the patient was moved into the provoking position (Tab. 4).[92,93] Signals from the posterior semicircular canal go to the ipsilateral superior oblique and contralateral inferior rectus muscles. Excitation of the receptors of the posterior canal results in a slow downward movement of the eyes, with a slow movement of the superior pole of the eye away from the affected side. These movements are followed by a quick resetting eye movement in the opposite direction. Thus, the direction of the nystagmus (which is always named by the direction of the fast phase) is "upbeating" and torsional, fast-phase beating toward the affected ("down-side") ear.

More recently, BPPV involving the anterior and horizontal canals has been reported.[6,94,95] As with BPPV involving the posterior semicircular canal, the direction of the nystagmus occurring when the person is moved into a provoking position is the basis for identifying the particular canal involved (Tab. 4). The anterior canal projects to the ipsilateral superior rectus muscle and to the contralateral inferior oblique muscle; the nystagmus, therefore, is "downbeating" and torsional. If the down-side ear is affected, the direction of the torsional component will be the same as in posterior-canal BPPV. That is, the superior pole will beat toward the down-side ear. The differentiation between anterior- and posterior-canal BPPV, therefore, must be made based on the direction of the vertical component of the nystagmus. If the nystagmus is downbeating and torsional, with the fast phase of the torsional component beating toward the "up-side" ear, it suggests that the affected anterior canal is in the up-side ear. The horizontal canal excites the ipsilateral medial and contralateral lateral rectus muscles, and in horizontal-canal BPPV, the nystagmus is horizontal when the patient is moved into the provoking position.[95] The best position is side-lying, not the Hallpike-Dix position, because of the alignment of the horizontal canal with respect to the pull of gravity. The direction of the nystagmus will depend on whether the debris is adhering to the cupula or is floating freely in the endolymph of the long arm of the canal (Fig. 9).

The identification of which of the semicircular canals is involved is most easily made by observing the direction of the nystagmus when the patient is first moved into the provoking position. In some patients, the nystagmus observed when the patient is first moved into the provoking position will reverse ("secondary nystagmus"). In addition, some patients will develop nystagmus when they return from the provoking position to a sitting position. The direction of the secondary nystagmus occurring in the provoking position and of the nystagmus that occurs when the patient returns to the sitting position can also be used to identify canal involvement (Tab. 4).[6] The secondary phase of nystagmus probably reflects the discharge of the velocity storage system. Velocity storage (storage of the velocity signal of the eye movement in the brain stem) for torsional nystagmus is poor. For this reason, in posterior- and anterior-canal

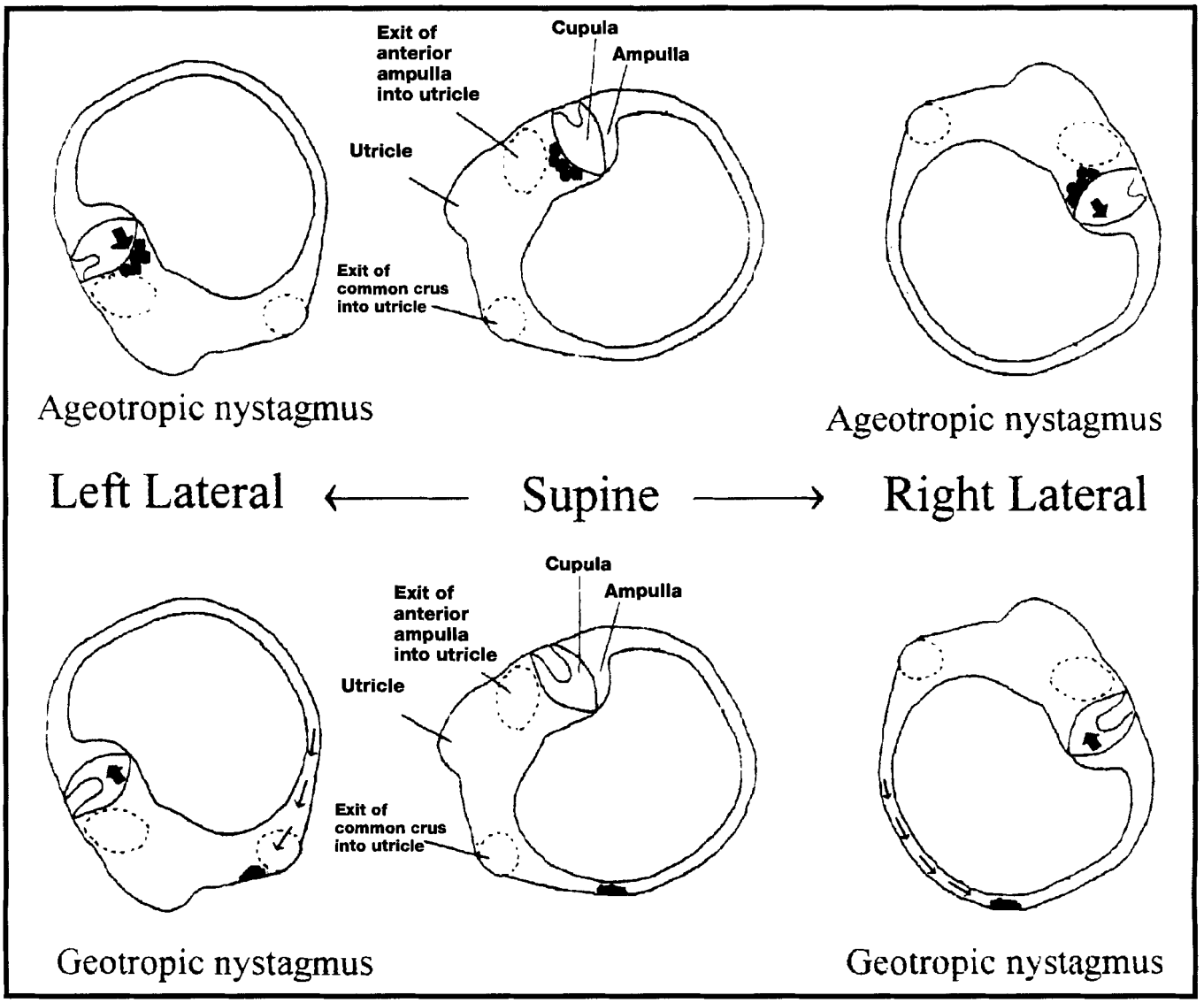

Figure 9.
The best position to provoke vertigo and nystagmus in horizontal-canal benign paroxysmal positional vertigo is to move person from the sitting to the side-lying position. The direction of the nystagmus will depend on whether the debris is adhering to the cupula (ageotropic nystagmus) or is floating freely in the endolymph of the long arm of the canal (geotropic nystagmus). (Adapted from Baloh et al.[95])

BPPV, the secondary phase is typically vertical. Nystagmus occurring when the person returns to a sitting position is due to movement of the cupula in the opposite direction; for the vertical canals, this nystagmus can have both vertical and torsional components. Posterior-canal involvement appears to be most common in patients with BPPV, occurring in more than 63% of all patients, with horizontal-canal BPPV being relatively uncommon (Tab. 5).[6] In that series of 77 consecutive patients with BPPV, however, the particular canal involved could not always be determined (24%). In most of the patients, the nystagmus was torsional, suggesting vertical-canal involvement, but because there was no vertical component, the differentiation between posterior canal and anterior canal could not be made. Additionally, some patients closed their eyes, and although nystagmus could be detected through the eyelids, the direction was not clear.

Because of the orientation of the canals, and because of the potential for movement of debris within each canal in canalithiasis, Brandt-Daroff exercises are unlikely to be appropriate for anterior-canal BPPV, although a modification of the exercises has been suggested for horizontal-canal cupulolithiasis. In this modification, the position changes are performed with the head at neutral on the body rather than turned 45 degrees away from the affected side.

The canalith repositioning maneuver has also been adapted for the treatment of patients with anterior- and horizontal-canal BPPV. In anterior-canal BPPV, the

maneuver would be the same as for posterior-canal BPPV (Fig. 8), but for horizontal-canal BPPV, a modification of the maneuver must be used, designed to move the person's head in the plane of the horizontal canal (Fig. 10).[81] The efficacy of this maneuver is not known because horizontal-canal BPPV is relatively unusual and no studies have been reported. More recently, a modification of the Liberatory maneuver has been proposed for the treatment of patients with horizontal-canal BPPV due to canalithiasis.[96] De la Meilleure et al[96] described six patients in whom successful remission of symptoms was achieved with one treatment. For this treatment, with the patients positioned supine with the head flexed 30 degrees, the head was first turned toward the affected side (again, based on intensity of vertigo and nystagmus). The head was kept in this position for 5 minutes and then rapidly turned 180 degrees to the opposite side (keeping the neck flexed 30° at all times). After 5 minutes, the patients sat up. The researchers asked the patients to avoid lying down for the subsequent 48 hours and to avoid shaking the head.

Summary

Significant changes in the use of vestibular exercises have been made in the last 5 years based on the direct outcome of controlled studies on the use of exercises in the treatment of vestibular loss and of BPPV. Furthermore, the exercises used in these treatments have become more sophisticated, reflecting an increased knowledge of the physiology and anatomy of the vestibular system and the mechanisms of recovery and compensation following vestibular dysfunction. The limitations of the mechanisms that substitute for the vestibular system in maintaining postural and gaze stability indicate that exercises to improve remaining vestibular function should be emphasized in the rehabilitation process.

Table 5.
Percentage of Canal Involvement in Benign Paroxysmal Positional Vertigo[85]

Canal Involved	Percentage
Posterior	63
Anterior	12
Horizontal	2
Unknown (vertical?)	23

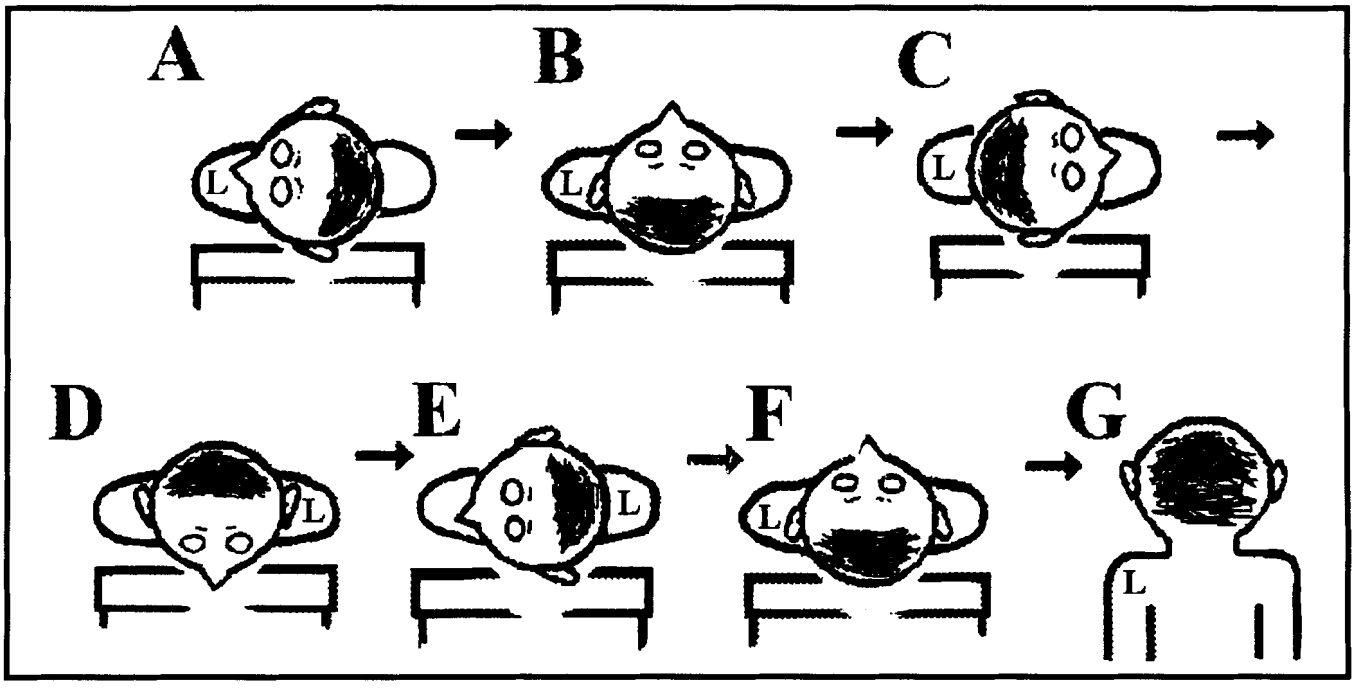

Figure 10.
Canalith repositioning maneuver for benign paroxysmal positional vertigo (BPPV) affecting the horizontal semicircular canal on the left side. The patient turns the head toward the affected side and moves quickly into the supine position (note that the head should be flexed slightly in order to position the horizontal canal in parallel with the pull of gravity) (A). After the nystagmus and vertigo stop, the head is turned to the right (B, C) and the patient is rolled toward the right and into a prone position (D) (if the patient experiences vertigo, the movement should be stopped until the vertigo stops). The head is then turned to the patient's right (E), and the patient is rolled again to a supine position (F). The patient then sits up (G). As with the canalith repositioning maneuver for posterior-canal BPPV, the patient then should keep the head upright for 48 hours. (Reprinted with permission from Herdman SJ. Physical therapy in the treatment of patients with benign paroxysmal positional vertigo. *Neurology Report.* 1996;20(3):46–53.)

References

1 Horak FB, Jones-Rycewicz C, Black FO, Shumway-Cook A. Effects of vestibular rehabilitation on dizziness and imbalance. *Otolaryngol Head Neck Surg.* 1992;106:175–180.

2 Krebs DE, Gill-Body KM, Riley PO, Parker SW. Double-blind, placebo-controlled trial of rehabilitation for bilateral vestibular hypofunction: preliminary report. *Otolaryngol Head Neck Surg.* 1993;109:735–741.

3 Herdman SJ, Clendaniel RA, Mattox DE, et al. Vestibular adaptation exercises and recovery: acute stage after acoustic neuroma resection. *Otolaryngol Head Neck Surg.* 1995;113:77–87.

4 Shepard NT, Telian SA. Programmatic vestibular rehabilitation. *Otolaryngol Head Neck Surg.* 1995;112:173–182.

5 Szturm T, Ireland DJ, Lessing-Turner M. Comparison of different exercise programs in the rehabilitation of patients with chronic peripheral vestibular dysfunction. *J Vestib Res.* 1994;4:461–479.

6 Herdman SJ, Tusa RJ, Clendaniel RA. Eye movement signs in vertical canal benign paroxysmal positional vertigo. In: Fuchs AF, Brandt T, Buttner U, Zee D, eds. *Contemporary Ocular Motor and Vestibular Research: A Tribute to David A Robinson.* Stuttgart, Federal Republic of Germany: Georg Thieme Verlag; 1994:385–387.

7 Epley JM. The canalith repositioning procedure: for treatment of benign paroxysmal positional vertigo. *Otolaryngol Head Neck Surg.* 1992;107:399–404.

8 Parnes LS, Price-Jones RG. Particle repositioning maneuver for benign paroxysmal positional vertigo. *Ann Otol Rhinol Laryngol.* 1993; 102:325–331.

9 Semont A, Freyss G, Vitte E. Curing the BPPV with a Liberatory maneuver. *Adv Otorhinolaryngol.* 1988;42:290–293.

10 Ireland D. The Semont maneuver. In: *Proceedings of the XVIIth Bárány Society Meeting.* Prague, Czechoslovakia: Bárány Society; 1994:367–370.

11 Herdman SJ, Tusa RJ, Zee DS, et al. Single treatment approaches to benign paroxysmal positional vertigo. *Arch Otolaryngol Head Neck Surg.* 1993;119:450–454.

12 Herdman SJ, Tusa RJ. Complications of the canalith repositioning procedure. *Arch Otolaryngol Head Neck Surg.* 1996;122:281–286.

13 Fetter M, Zee DS. Recovery from unilateral labyrinthectomy in rhesus monkeys. *J Neurophysiol.* 1988;59:370–393.

14 LaCour M, Roll JP, Appaix M. Modifications and development of spinal reflexes in the alert baboon (*Papio papio*) following an unilateral vestibular neurectomy. *Brain Res.* 1976;113:255–269.

15 Igarashi M, Levy JK, O-Uchi T, et al. Further study of physical exercise and locomotor balance compensation after unilateral labyrinthectomy in squirrel monkeys. *Acta Otolaryngol.* 1981;92:101–105.

16 Igarashi M, Ishikawa K, Ishii M, et al. Physical exercise and balance compensation after total ablation of vestibular organs. In: Pompeiano O, Allum JHJ, eds. *Progress in Brain Research.* Amsterdam, the Netherlands: Elsevier Science Publishers BV; 1988:395–401.

17 McCabe BF. Labyrinthine exercises in the treatment if disease characterized by vertigo: their physiologic basis and methodology. *Laryngoscope.* 1970;80:1429–1433.

18 Hecker HC, Haug CO, Herndon JW. Treatment of the vertiginous patient using Cawthorne's vestibular exercises. *Laryngoscope.* 1974;84: 2065–2072.

19 Dix MR. The rationale and technique of head exercises in the treatment of vertigo. *Acta Otorhinolaryngol Belg.* 1979;33:370–384.

20 Zee DS. Vertigo. In Johnson R, ed. *Current Therapy in Neurological Disease.* St Louis, Mo: CV Mosby Co; 1985:8–13.

21 Herdman SJ. Exercise strategies for vestibular disorders. *Ear Nose Throat J.* 1989;68:961–964.

22 Shumway-Cook A, Horak FB. Vestibular rehabilitation: an exercise approach to managing symptoms of vestibular dysfunction. *Seminars in Hearing.* 1989;10:196–209.

23 Telian SA, Shepard NT, Smith-Wheelock M, Kemink JL. Habituation therapy for chronic vestibular dysfunction: preliminary results. *Otolaryngol Head Neck Surg.* 1990;103:89–95.

24 Cass SP, Borello-France D, Furman JM. Functional outcome of vestibular rehabilitation in patients with abnormal sensory-organization testing. *Am J Otol.* 1996;17:581–594.

25 Cawthorne T. Vestibular injuries. *Proc R Soc Med.* 1946;39:270–272.

26 Shepard NT, Telian SA, Smith-Wheelock M. Habituation and balance retraining therapy. *Neurol Clin.* 1990;5:459–476.

27 Shepard NT, Telian SA, Smith-Wheelock M, Raj A. Vestibular and balance rehabilitation therapy. *Ann Otol Rhinol Laryngol.* 1993;102:198–205.

28 Keim RJ, Cook M, Martini D. Balance rehabilitation therapy. *Laryngoscope.* 1992;102:1302–1307.

29 Norre ME, Beckers A. Vestibular habituation training for positional vertigo in elderly patients. *J Am Geriatr Soc.* 1988;36:425–429.

30 Black FO, Shupert CL, Peterka RJ, Nashner LM. Effects of unilateral loss of vestibular function on the vestibulo-ocular reflex and postural control. *Ann Otol Rhino Laryngol.* 1989;98:884–889.

31 Fetter M, Diener HC, Dichgans J. Recovery of postural control after an acute unilateral vestibular lesion in humans. *J Vestib Res.* 1991;1:373–383.

32 Takemori S, Ida M, Umegu H. Vestibular training after sudden loss of vestibular function. *ORL J Otorhinolaryngol Relat Spec.* 1985;47:76–83.

33 Norre ME, Beckers A. Vestibular habituation training: exercise treatment for vertigo based on habituation effect. *Otolaryngol Head Neck Surg.* 1989;101:14–19.

34 Bles W, Vianney de Jong JMB, de Wit G. Compensation for labyrinthine defects examined by use of a tilting room. *Acta Otolaryngol (Stockh).* 1983;l95:576–579.

35 Bles W, Vianney de Jong JMB, Rasmussens JJ. Postural and oculomotor signs in labyrinthine-defective subjects. *Acta Otolaryngol (Stockh).* 1984;406:101–104.

36 Allum JHJ, Yamane M, Pfaltz CR. Long-term modification of vertical and horizontal vestibulo-ocular reflex dynamics in man. *Acta Otolaryngol (Stockh).* 1988;105:328–337.

37 Paige GP. Nonlinearity and asymmetry in the human vestibulo-ocular reflex. *Acta Otolaryngol (Stockh).* 1989;108:1–8.

38 Dichgans J, Bizzi E, Morasso P, Tagliasco V. Mechanisms underlying recovery of eye-head coordination following bilateral labyrinthectomy in monkeys. *Exp Brain Res.* 1973;18:548–562.

39 Kasai T, Zee DS. Eye-head coordination in labyrinthine-defective human beings. *Brain Res.* 1978;144:123–141.

40 Barnes GR. Head-eye coordination in normals and patients with vestibular disorders. *Adv Otorhinolaryngol.* 1979;25:197–201.

41 Chambers BR, Mai M, Barber HO. Bilateral vestibular loss, oscillopsia, and the cervico-ocular reflex. *Otolaryngol Head Neck Surg.* 1985;93: 403–407.

42 Gresty MA, Hess K, Leech J. Disorders of the vestibulo-ocular reflex producing oscillopsia and mechanisms compensating for loss of labyrinthine function. *Brain.* 1977;100:693–716.

43 Bronstein AM, Hood JD. The cervico-ocular reflex in normal subjects and patients with absent vestibular function. *Brain Res.* 1986; 373:399–408.

44 Diener HC, Dichgans J, Guschlbauer B, Mau H. The significance of proprioception on postural stabilization as assessed by ischemia. *Brain Res.* 1984;296:103–109.

45 Diener HC, Dichgans J, Bruzek W, Selinka H. Stabilization of human posture during induced oscillations of the body. *Exp Brain Res.* 1982;45:126–132.

46 Dichgans J, Brandt T. Visuo-vestibular interactions: effects on self-motion perception and postural control. In: Held R, Leibowitz HW, Teuber HL, eds. *Handbook of Sensory Physiology.* Berlin, Federal Republic of Germany: Springer; 1978:755–804.

47 Paige GD. Senescence of human visual-vestibular interactions: smooth pursuit, optokinetic, and vestibular control of eye movements with aging. *Exp Brain Res.* 1994;98:355–372.

48 Gauthier GM, Robinson DA. Adaptation of the human vestibulo-ocular reflex to magnifying lenses. *Brain Res.* 1975;92:331–335.

49 Miles FA, Eighmy BB. Long-term adaptive changes in primate vestibulo-ocular reflex, I: behavioral observation. *J Neurophysiol.* 1980; 43:1406–1425.

50 Shelhamer M, Tiliket C, Roberts D, et al. Short-term vestibulo-ocular reflex adaptation in humans, II: error signals. *Exp Brain Res.* 1994;100:328–336.

51 Tiliket C, Shelhamer M, Roberts D, Zee DS. Short-term vestibulo-ocular reflex adaptation in humans, I: effect on the ocular motor velocity-to-position neural integrator. *Exp Brain Res.* 1994;100:316–327.

52 Lisberger SG. The neural basis for learning of simple motor skills. *Science.* 1988;242:728–735.

53 Lisberger SG, Miles FA, Optican LM. Frequency-selective adaptation: evidence for channels in the vestibulo-ocular reflex. *J Neurosci.* 1983;3:1234–1244.

54 Tiliket C, Shelhamer M, Tan S, Zee DS. Adaptation of the vestibulo-ocular reflex with the head indifferent orientations and positions relative to the axis of the body. *J Vestib Res.* 1993;3:181.

55 Leigh RJ, Zee DS. *The Neurology of Eye Movements.* Philadelphia, Pa: FA Davis Co; 1991.

56 Brandt T, Paulus WM, Straube A. Visual acuity, visual field, and visual scene characteristics affect postural balance. In: Igarashi M, Black FO, eds. *Vestibular and Visual Control of Posture and Locomotion Equilibrium.* Basel, Switzerland: S Karger AG, Medical and Scientific Publishers; 1985:93–98.

57 Paulus WM, Straube A, Brandt T. Visual stabilization of posture: physiological stimulus characteristics and clinical aspects. *Brain.* 1984; 107:1143–1163.

58 Herdman SJ, Sandusky AL, Hain TC, et al. Characteristics of postural stability in patients with aminogycoside toxicity. *J Vestib Res.* 1994;4:71–80.

59 Grossman GE, Leigh RJ, Bruce EN, et al. Performance of the human vestibulo-ocular reflex during locomotion. *J Neurophysiol.* 1989; 62:264–272.

60 Demer JL, Goldberg J, Porter FI. Effect of telescopic spectacles on head stability in normal and low vision. *J Vestib Res.* 1991;1:109–122.

61 Saito A, Okada Y, Yoshida A. Recovery of gaze disturbance in bilateral vestibular loss. *ORL J Otorhinolaryngol Relat Spec.* 1989;51:305–310.

62 Westheimer G, McKee SP. Visual acuity in the presence of retinal-image motion. *J Opt Soc Am.* 1975;65:847–850.

63 Demer JL, Amjadi F. Dynamic visual acuity of normal subjects during vertical optotype and head motion. *Invest Ophthalmol Vis Sci.* 1993;34:1894–1906.

64 Demer JL, Honrubia V, Baloh RW. Dynamic visual acuity: a test for oscillopsia and vestibulo-ocular reflex function. *Am J Otol.* 1994;15: 340–347.

65 Yamane M, Allum JHJ, Pfaltz CR. Vertical and horizontal vestibuloocular reflex dynamics following unilateral vestibular deficit. In: Graham MD, Kemink JL, eds. *The Vestibular System: Neurophysiologic and Clinical Research.* New York, NY: Raven Press; 1987:557–564.

66 Halmagyi GM, Curthoys IS, Cremer PD, et al. Head impulses after unilateral vestibular deafferentiation validate Ewald's Second Law. *J Vestib Res.* 1990/1991;1:187–197.

67 Black FO, Shuppert CL, Peterka RJ, Nashner LM. Effects of unilateral loss of vestibular function on the vestibulo-ocular reflex and postural control. *Ann Otol Rhinol Laryngol.* 1989;98:884–889.

68 Segal B, Katsarkas A. Long-term deficits of goal-directed vestibulo-ocular function following total unilateral loss of peripheral vestibular function. *Acta Otolaryngol (Stockh).* 1988;106:102–110.

69 Hikosaka O, Maeda M. Cervical effects on abducens motoneurons and their interaction with vestibulo-ocular reflex. *Exp Brain Res.* 1973;18:512–530.

70 Barnes GR. Visual-vestibular interaction in the control of head and eye movement: the role of visual feedback and predictive mechanisms. *Prog Neurobiol.* 1993;41:435–472.

71 Sawyer RN, Thurston SE, Becker KR, et al. The cervico-ocular reflex of normal human subjects in response to transient and sinusoidal trunk rotations. *J Vestib Res.* 1994;4:245–249.

72 Leigh RJ, Brandt T. A reevaluation of the vestibulo-ocular reflex: new ideas of its purpose, properties, neural substrate, and disorders. *Neurology.* 1993;43:1288–1295.

73 Leigh RJ, Sharpe JA, Ranalli PJ, et al. Comparison of smooth pursuit and combined eye-head tracking in human subjects with deficient labyrinthine function. *Exp Brain Res.* 1987;66:458–464.

74 Demer JL. Evaluation of vestibular and visual oculomotor function. *Otolaryngol Head Neck Surg.* 1995;112:16–35.

75 Baloh RW, Honrubia V, Jacobson K. Benign positional vertigo: clinical and oculographic features in 240 cases. *Neurology.* 1987;37:371–378.

76 Froehling DA, Silverton MD, Mohr DN, et al. Benign positional vertigo: incidence and prognosis in a population-based study on Olmsted County, Minnesota. *Mayo Clin Proc.* 1991;66:596–601.

77 Bloom J, Katsarkas A. Paroxysmal positional vertigo in the elderly. *J Otolaryngol.* 1989;18:96–98.

78 Black FO, Nashner LM. Postural disturbances in patients with benign paroxysmal positional nystagmus. *Ann Otol Rhinol Laryngol.* 1984;93:595–599.

79 Voorhees RL. The role of dynamic posturography in neurotologic diagnosis. *Laryngoscope.* 1989;99:995–1001.

80 Buchele W, Brandt T. Benign paroxysmal positional vertigo and posture. In: Bless W, Brandt T, eds. *Disorders of Posture and Gait.* Amsterdam, the Netherlands: Elsevier Science Publishers BV; 1986: 101–111.

81 Epley JM. New dimensions of benign paroxysmal positional vertigo. *Otolaryngol Head Neck Surg.* 1980;88:599–605.

82 Schuknecht HF. Cupulolithiasis. *Arch Otolaryngol.* 1969;90:765–778.

83 Moriarty B, Rutka J, Hawke M. The incidence and distribution of cupular deposits in the labyrinth. *Laryngoscope.* 1992;102:56–59.

84 Boumans LJJM, Rodenburg M, Maas AJJ. Gain of the adaptation mechanism in the human vestibulo-ocular reflex system. *ORL J Otorhinolaryngol Relat Spec.* 1988;50:319–329.

85 Hall SF, Ruby RR, McClure J. The mechanisms of benign paroxysmal vertigo. *J Otolaryngol.* 1979;8:151–158.

86 Brandt T, Daroff RB. Physical therapy for benign paroxysmal positional vertigo. *Arch Otolaryngol.* 1980;106:484–485.

87 Gyko K. Benign paroxysmal positional vertigo as a complication of bedrest. *Laryngoscope.* 1988;98:332–333.

88 Blakley BW. A randomized, controlled assessment of the canalith repositioning maneuver. *Otolaryngol Head Neck Surg.* 1994;110:391–396.

89 Li JC. Mastoid oscillation: a critical factor for success in the canalith repositioning procedure. *Otolaryngol Head Neck Surg.* 1995;112:670–675.

90 Harvey SA, Hain TC, Adamiec LC. Modified Liberatory maneuver: effective treatment for benign paroxysmal positional vertigo. *Laryngoscope.* 1994;104:1206–1212.

91 Welling DB, Barnes DE. Particle repositioning maneuver for benign paroxysmal positional vertigo. *Laryngoscope.* 1994;104:946–949.

92 Bárány R. Diagnose von krankheitserscheinungen im bereiche des otollithebapparates. *Acta Otolaryngol (Stockholm).* 1921;2:434–437.

93 Dix MR, Hallpike CS. Pathology, symptomatology, and diagnosis of certain disorders of the vestibular system. *Proc R Soc Med.* 1952;45:341–354.

94 McClure J. Horizontal canal BPV. *J Otolaryngol. 1985;14:30–35.*

95 Baloh RW, Jacobson K, Honrubia V. Horizontal semicircular canal variant of benign positional vertigo. *Neurology.* 1993;43:2542–2549.

96 De la Meilleure G, Dehaene I, Depondt M, et al. Benign paroxysmal positional vertigo of the horizontal canal. *J Neurol Neurosurg Psychiatry.* 1996;60:68–71.