Case Report

Physical Therapy Management of Peripheral Vestibular Dysfunction: Two Clinical Case Reports

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We describe the treatment of two patients with peripheral vestibular dysfunction using a novel, staged exercise program. Response to treatment was documented. The first patient, a 62-year-old woman with unilateral vestibular dysfunction (UVD) and a 6-month history of disequilibrium following herpes zoster oticus resulting in damage to the right inner ear, was treated with an 8-week course of vestibular physical therapy. During the 8 weeks, the patient attended weekly physical therapy sessions and was trained to perform vestibular adaptation exercises on a daily basis at home. The second patient, a 53-year-old woman with progressive disequilibrium secondary to profound bilateral vestibular hypofunction (BVH), was treated with a 16-week course of vestibular physical therapy. During the first 8 weeks, the patient attended weekly physical therapy sessions and was trained to perform vestibular adaptation and substitution exercises on a daily basis at home. During the second 8 weeks, the patient continued performing vestibular physical therapy exercises at home independently. Vestibular function (sinusoidal vertical axis rotation testing), postural control (clinical tests and posturography), stability during the performance of selected activities of daily living (ADLs), and self-perception of symptoms and handicap were measured prior to and at the conclusion of treatment for both patients and at the midpoint of treatment for the patient with BVH. After 8 weeks of treatment, both patients reported improvements in self-perception of symptoms and handicap and demonstrated objective improvements in clinical balance tests, posturography, and several kinematic indicators of stability during the performance of selected ADLs. Further improvements were noted in the patient with BVH after 16 weeks of treatment. Improvements in postural control were noted after 8 weeks of treatment for the patient with UVD and after 16 weeks for the patient with BVH. Vestibular function improved during the course of treatment for the patient with UVD only. These case reports describe two different individualized treatment programs and document self-reported and laboratory-measured functional improvements in two patients with vestibular deficients—one with unilateral damage and one with bilateral damage. [Gill-Body KM, Krebs DE, Parker SW, Riley PO. Physical therapy management of peripheral vestibular dysfunction: two clinical case reports. Phys Ther. 1994;74:129–142.]

Key Words: *Balance, Balance rehabilitation, Postural control, Vestibular rehabilitation.*

Many individuals with peripheral vestibular lesions undergo spontaneous resolution of their signs and symptoms due to central nervous system compensation.[1] Some patients, however, have persistent problems of dizziness and disequilibrium. The symptoms associated with unilateral vestibular dysfunction (UVD) include vertigo, dizziness, nausea, and postural instability; the symptoms associated with bilateral vestibular hypofunction (BVH) also include gait ataxia, blurred vision, difficulty reading, and oscillopsia.[2] The sensations of disequilibrium and poor balance may reflect the disparity between abnormal vestibular information and visual and proprioceptive information.[3] Secondary problems related to a reduced activity level, including

muscle weakness, limited endurance, and loss of flexibility, can further impair postural responses.[4] For patients with persistent symptoms, pharmacologic management appears to benefit only a small number[5] and surgical management is appropriate for even fewer patients.[6] Because the individual's ability to move in the environment without experiencing dizziness or loss of balance is severely impaired, overall functional abilities and quality of life are compromised.

Vestibular physical therapy is an exercise approach that has developed over the past several years to help manage the persistent functional problems associated with peripheral vestibular dysfunction.[7-10] Exercise programs, aimed at remediating the problems of dizziness, gaze instability, and balance dysfunction, are designed for each patient based on the patient's signs, symptoms, and functional limitations. A different type of treatment is utilized based on whether the patient exhibits absent versus reduced vestibular function.[7]

For patients with residual vestibular function, the treatment program, denoted the adaptation approach, is similar to that described by Cawthorne in the 1940s for patients with persistent symptoms from vestibular dysfunction.[11] The brain's ability to adapt to changes in demand or changes in sensory information received is key to this treatment approach.[12] By providing stimuli that induce adaptation of the vestibular system, such as combining movement of an image across the retina with head movement, compensation within the central nervous system is thought to be promoted.[7] For patients with unilaterally reduced or abnormal vestibular function, such as the first patient reported here, the adaptation approach is utilized.

For patients with no remaining vestibular function, a substitution approach is used. In this approach, the patient is encouraged to rely on visual and proprioceptive information to stabilize gaze and maintain postural stability in place of vestibular information.[3] For patients with bilaterally reduced (but not absent) vestibular function, such as the second patient reported here, a combined approach (incorporating exercises to foster both adaptation and substitution) is utilized, as it cannot be determined whether central nervous system adaptation to the maximum possible extent has already occurred. Vestibular physical therapy, as described in this case report, is currently offered in various centers across the United States, and some general information is available regarding patient response to the treatment approach in terms of subjective symptoms and self-rated disability.[13]

Horak et al[14] recently reported preliminary results regarding the relative effectiveness of vestibular rehabilitation, general conditioning exercises, and vestibular suppressant medication on reducing dizziness and imbalance. Their results suggest that although all three treatment approaches reduce dizziness, only vestibular rehabilitation improves balance (as measured by duration of unilateral stance and posturography).[14] To date, however, no one has both described a specific treatment program in detail and reported data regarding patient response to treatment in terms of whole-body movement analysis, clinical balance testing, posturography, symptoms of dizziness and disequilibrium, and perceived level of disability for an individual patient or series of patients with vestibular dysfunction.

The medical workup for each patient just prior to referral to physical therapy consisted of a neurological examination by a neurologist, vestibular testing including an electronystagmogram with caloric stimulation, sinusoidal vertical axis rotation, visual vestibular interaction rotation, and posturography testing utilizing the Equitest™ system.*[15,16] Each patient also underwent a three-dimensional movement analysis in our biomotion laboratory. A full-body kinematic and kinetic analysis of key activities of daily living (ADLs) (standing, free and paced gait, walking in place, ascending steps, and rising from a chair) was completed.[17,18] The motion analysis system is described in detail elsewhere.[17-19] This system consists of an 11-segment, 66-degree-of-freedom, full-body (head, arms, trunk, pelvis, thighs, shanks, and feet) kinematic model; two force plates; and software to integrate the kinematic and kinetic data.[18] SELSPOT II hardware† and a TRACK kinematic data-analysis software package‡ are used to acquire and analyze the three-dimensional full-body kinematic data. Floor reaction forces are acquired from two Kistler platforms§ and processed on

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§Type 9281A, Kistler Instruments AG, Winterthur, Switzerland.

the same computer as the kinematic data. Kinematic and kinetic data are sampled at 153 Hz and digitally filtered. Dynamic stability is quantified using the kinematics of the center of gravity (COG) and center of pressure (COP) as well as standard time-distance parameters (ie, double support time, average velocity, and so forth). The COG-COP moment arm, the horizontal separation between the COG and COP, is used to quantify the dynamics of each activity.[18,20]

Unilateral Vestibular Dysfunction

History

A 62-year-old right-handed woman was referred to physical therapy with a diagnosis of right-sided vestibular damage due to a herpes zoster oticus. The patient history included an initial onset of a right posterior headache followed by right facial paralysis the next day. On the day after onset of paralysis, the patient was extremely unsteady, had difficulty walking, and was hospitalized. After 5 days, lesions of herpes zoster were noted in the right ear and the patient was treated with acyclovir and prednisone. Two weeks later, at the time of discharge from the hospital, she was still unable to walk without assistance. Over the next 5 months, the patient noted some improvements in her facial palsy and balance ability. At the time the patient was seen at our hospital (6 months after the initial onset of symptoms), she reported persistent unsteadiness; difficulty walking out-of-doors; some ringing and buzzing in the right ear; and the inability to return to work as a city tour guide due to her inability to stand on a moving bus, walk in a straight line, or maintain balance in busy environments. These problems had been unchanged for the preceding 3 months.

Prerehabilitation Findings

Examination by the neurologist revealed full extraocular movements with no spontaneous or gaze nystagmus. The patient reported that an audiogram performed 4 months earlier had shown high-frequency hearing loss in the right ear. Hearing was intact to watch tick bilaterally, and a repeat audiogram was not performed. Facial sensation was intact, and there was a mild right facial droop. Patellar and Achilles tendon reflexes were brisk and symmetrical. Coordination and strength (tested by performing resisted isometric contractions of the shoulder flexors, elbow flexors and extensors, finger flexors, hip flexors, knee extensors, and ankle dorsiflexors) were intact in all extremities. A Romberg test was negative with sway. The patient was able to stand on foam with eyes closed, but appeared very unsteady and repeatedly fell backward. Gait appeared stable, with normal-sized steps and good arm swing. Tandem gait appeared moderately unsteady, with a tendency to fall to either side after a few steps. Positional testing (Hallpike maneuver both with and without head turning) revealed no nystagmus or dizziness. There was no spontaneous, gaze, post–head-shaking, or positional nystagmus behind Frenzel lenses.

An electronystagmogram revealed an 87% reduced right caloric response (total peak slow-phase velocity after warm and cool stimulation of the right and left ears was 2° and 29°/s, respectively) and left-beating positional nystagmus (eye movements with a fast component to the left and a slow component to the right). Sinusoidal vertical-axis rotation (SVAR) with a peak velocity of 50°/s at frequencies from 0.01 to 1.0 Hz revealed mildly decreased gains (slow-phase eye velocity/chair velocity[21]) of the vestibular ocular reflex in the lower frequencies of rotation to the following extent: between 2.0 and 2.5 standard deviations below the mean at 0.01 Hz and between 1.0 and 2.0 standard deviations below the mean at 0.02 to 0.05 and 0.1 Hz (as compared with healthy subjects) (Fig. 1). The SVAR also revealed increased phase leads (the relationship between the onset of the rotation-induced nystagmus and the angular velocity of the chair movement; an increased phase lead is seen with damage to the vestibular system) of greater than 2.5 standard deviations from the mean at 0.01, 0.02, and 0.05 Hz. Finally, there was a left preponderance (ie, more left than right) of rotation-induced nystagmus of greater than 2.5 standard deviations at 0.01, 0.02, 0.05, 0.10, 0.20, 0.50, and 1.0 Hz (ie, at all frequencies of rotation). There was good fixation suppression on visual vestibular interaction testing, and optokinetic nystagmus was normal. Details regarding SVAR testing and interpretation of the raw data obtained to compute gain, phase, and symmetry are described elsewhere.[22] These test results, combined with the examination and symptoms, are consistent with a poorly compensated unilateral vestibular lesion on the right side.

Posturography testing revealed excessive sway and falls on a sway-referenced platform with eyes closed and moderate sway with one fall on a sway-referenced platform with eyes fixed on a sway-referenced visual surround (Fig. 2). To achieve sway-referencing, the patient's anterior-posterior body sway is used to produce a rotation of the platform or the visual surround in a plane collinear with the patient's ankles at a gain of 1.0 (ie, at a speed that matches the patient's body sway); these maneuvers are intended to alter joint sensory information (proprioception) by keeping the angle between the foot and the leg constant when the platform is sway-referenced, or to provide conflicting visual information when the visual surround is sway-referenced.

During the initial physical therapy examination, the patient's sensation for light touch (of the lower leg and foot) and proprioception (of the knees, ankles, and toes) were intact. Proprioception was tested by having the patient identify the position of each joint as it was moved in different directions while her eyes were closed. Deep tendon reflexes were brisk and symmetrical. Manual muscle testing (of the shoulder girdle; hip extensors and abductors; knee flexors and extensors; and ankle dorsiflexors, plantar flexors, invertors, and ever-

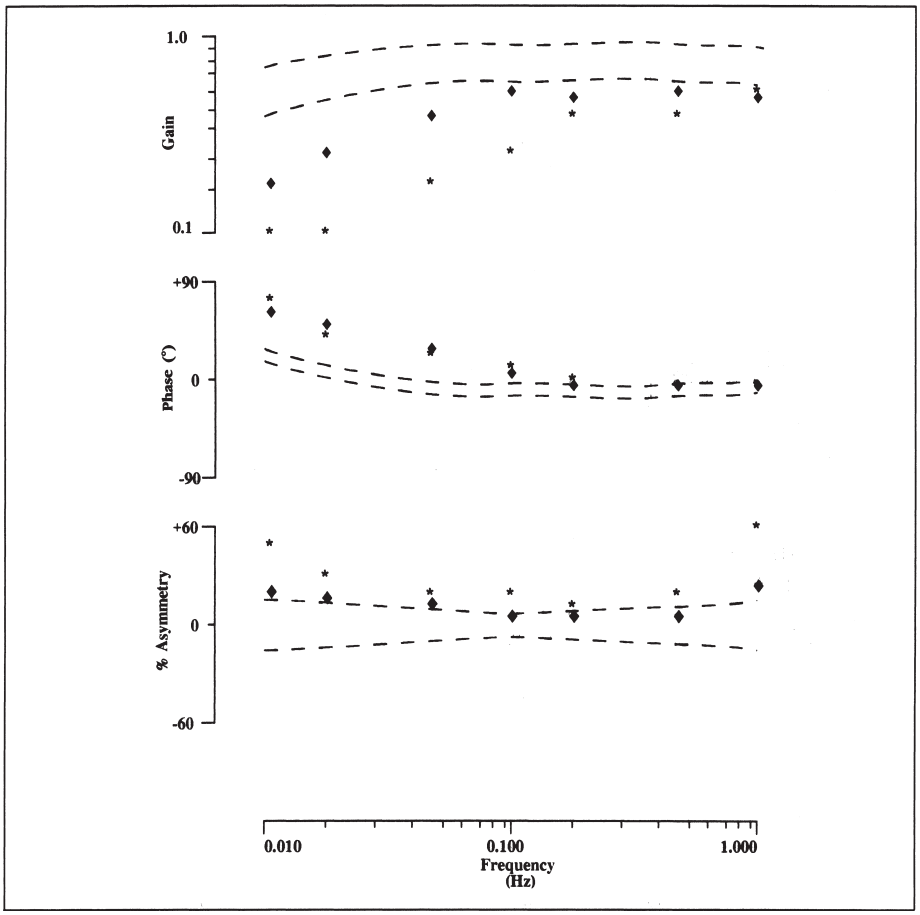

Figure 1. *Sinusoidal vertical-axis rotation test results for the patient with unilateral vestibular dysfunction before (*) and after (♦) treatment. Gain (the ratio of peak slow-phase eye velocity to peak chair velocity), phase (the interval between the stimulus and the response), and asymmetry (the percentage of difference between the peak slow-component eye velocities to the right and left) are displayed. Testing was performed at seven frequencies of rotation (0.01, 0.02, 0.05, 0.10, 0.20, 0.50, and 1.00 Hz). The dashed lines represent the mean values (±1 standard deviation) across all frequencies of rotation for healthy subjects. This patient's test results demonstrated decreased low-frequency gains and increased low-frequency phase leads both before and after treatment. Asymmetry was reduced following treatment, consistent with central nervous system adaptation to vestibular damage.*

tors) showed Normal (5/5) strength in all muscle groups except for the right-sided facial musculature, in which weakness was evidenced by minimal muscle contractions during attempts to smile, grimace, and purse lips. Heel-to-shin movements, finger-to-nose movements, and rapid alternating movements of the forearm were performed quickly, accurately, and smoothly, bilaterally. Active range of motion of the spine, shoulders, hips, knees, and ankles was full except for rotation and lateral flexion at the cervical spine, which was reduced by half. The patient reported a pulling sensation in the posterior cervical and upper trapezius muscle regions bilaterally during active cervical lateral flexion and rotation movements. Pursuit and saccadic eye movements could be performed fully and without symptoms. The patient reported that she had slightly blurred vision in the right eye. She reported not having vertigo or dizziness. She also reported the sensation of disequilibrium frequently (more than once per month but not continuously) at an intensity of 4/10 (10 being the highest level of intensity imaginable).

The patient scored 14 out of a possible 100 on the Dizziness Handicap Inventory (DHI)[23] (100 = the highest level of handicap), reporting problems in three of the nine items related to functional activities and in four of the seven items related to physical activities. The DHI, originally devised to measure perception of handicap in individuals with benign paroxysmal positional vertigo, is used in our clinic for all patients with vestibular dysfunction to objectively document perception of handicap related to dizziness or balance problems.

Clinical balance assessment revealed that the patient could stand with her eyes closed and her feet together for 60 seconds (measured with a digital stopwatch), but it demonstrated a significantly increased ankle sway (sway was observed and not measured). Unilateral stance with eyes open could be performed for 7 seconds, and the patient was unable to perform unilateral stance with her eyes closed. The patient could perform tandem stance for 60 seconds with her eyes open and for 5 seconds with her eyes closed. Stance on foam with eyes open could be performed for 60 seconds without difficulty; with eyes closed, stance on foam could be performed for 20 seconds with a marked ankle sway. Tandem gait with eyes open could be performed for a maximum of five steps. The patient adopted a forward-bent posture during standing and walking activities. Gait was wide based and characterized by an immobile trunk, no arm swing, and gaze fixation on the floor. The patient was unable to ambulate in a straight line at any speed; rather, she moved in a side-to-side path (to both sides) as she moved forward. Turns (90°) were performed slowly and with multiple small steps. During attempts to ambulate while rotating her head from side to side, the patient's speed of gait decreased and she crossed one foot over the other repeatedly.

Rehabilitation

The vestibular adaptation treatment program that the patient received consisted of exercises and activities outlined under phase 1 through

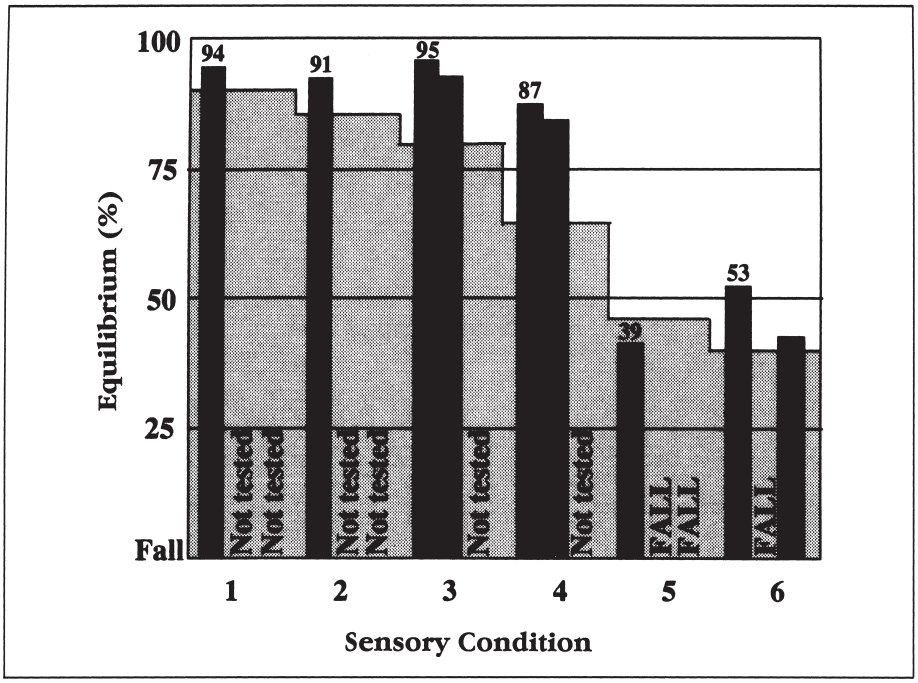

Figure 2. *Baseline (week 0) posturography (sensory organization) test results for patient with unilateral vestibular dysfunction. (Note: Equitest™ system was used.) Equilibrium score represents the angular difference between the patient's calculated maximum anterior-posterior center of gravity and the theoretical maximum displacement of 12.5 degrees; the result is expressed as a percentage between 0% and 100%, with 0% indicating sway exceeding the limits of stability and 100% indicating perfect stability. Numerical equilibrium scores for the best performance for each trial are indicated above the vertical bars. Shaded areas represent norms for performance; scores above the shaded areas represent normal test scores. Sensory conditions: 1=fixed platform, eyes open/fixed visual surround; 2=fixed platform, eyes closed; 3=fixed platform, eyes open/sway-referenced visual surround; 4=sway-referenced platform, eyes open/fixed visual surround; 5=sway-referenced platform, eyes closed; 6=sway-referenced platform, eyes open/sway-referenced visual surround. Not tested=repeat trials not performed. Standard testing procedures are to perform only one trial for sensory conditions 1 through 3, unless the patient is observed to have a large amount of sway or falls, and to perform three trials for sensory conditions 4 through 6, unless patient appears to have successfully completed the test in fewer trials. Fall=patient fell during trial.*

phase 3 in Table 1. The patient performed phase 1 of the program during the first 2 weeks of treatment, phase 2 during weeks 3 through 6, and phase 3 during weeks 7 and 8. The patient progressed to each new phase during weekly visits to the physical therapy clinic as her performance on the previous phase improved to the point at which the individual exercises could be performed easily and without an increase in the perception of disequilibrium. Correct posture and alignment were emphasized during all of standing and walking activities. A brief explanation of the rationale for each activity in the treatment program is included in Table 1 and is based primarily on the work of Herdman[7] and Shumway-Cook and Horak.[8,10] The patient was instructed to perform the exercises at least one time per day at home and to note daily the exercises she performed, number of repetitions, and any difficulties she experienced using a standard patient compliance tool in use at our clinic. The patient completed daily exercise logs indicating that she was compliant with the daily home program (ie, she performed the exercises 6–7 days per week over the course of the entire treatment period). The patient reported completing the daily exercise program once per day during weeks 1 through 3 and twice per day during weeks 4 through 8. After 8 weeks of treatment, the patient was reevaluated in the clinic and in the biomotion laboratory utilizing the same measures as those used during the initial assessment. Repeat SVAR testing and posturography were also performed.

The vestibular adaptation treatment program (Tab. 1) designed for this patient was based on the following interpretation of the patient's condition:

1. The patient's decreased cervical range of motion could be related to her voluntarily holding her head still during gait and other functional activities; decreased cervical range of motion and alignment could impair postural responses[10] and were therefore worth addressing in treatment.

2. The patient's primary problem of impaired postural stability was related to her vestibular hypofunction on the right side, as supported by the posturography test results of difficulty with sensory conditions 5 and 6 (Fig. 2).

3. The patient clearly demonstrated some ability to utilize vestibular information for postural control in situations in which accurate visual and proprioceptive information were not as available (ie, sensory conditions 5 and 6 could be partially performed on some trials).

Postrehabilitation Findings

At the conclusion of the 8-week period of treatment, the patient reported a slight decrease in the intensity of her sensation of disequilibrium (3/10) and improvements in four physical and two functional activities previously reported to be problematic on the DHI (Tab. 2). No items on the DHI were reported to be worse. Clinical balance assessment revealed no change in ability to perform unilateral stance with eyes open or eyes closed. Tandem stance with eyes closed could be performed for 20 seconds (improved from 5 seconds). Stance with eyes closed and with feet together on the floor and on foam could be per-

Table 1. *Vestibular Rehabilitation Treatment Program and Its Rationale for Patient With Unilateral Vestibular Dysfunction*

Rationale	Treatment Activity[a]
Phase 1	
1. Encourage active extraocular movements	Extraocular movements, self-selected speed
2. Enhance vestibular adaptation	Visual fixation, EO, stationary target, slow head movements, near targets
3. Encourage resetting of VOR gain	Imaginary visual fixation, EC, small head movements, self-selected speed
4. Promote utilization of somatosensory and vestibular inputs for postural control	Static stance, EO and EC, feet together, arms outstretched, book on head
5. Improve dynamic postural control utilizing all sensory inputs	Gait with narrowed base of support, EO
6. Improve dynamic postural control utilizing all sensory inputs	March in place slowly, EO
7. Decrease cervical musculature tightness	Active neck range of motion, all directions, slow movements
Phase 2	
1. Promote use of VOR at various speed head movements	Visual fixation, EO, stationary target, fast and slow movements, near targets
2. Enhance vestibular adaptation by inducing retinal slip	Visual fixation, EO, moving target in opposite direction, slow head movements
3. Promote utilization of somatosensory and vestibular inputs for postural control	Static stance, semitandem, EO and EC, arms close to body, book on head
4. Promote use of somatosensory inputs for postural control	Static stance on foam surface, EO, book on head
5. Improve dynamic postural control utilizing somatosensory inputs	Gait with narrowed base of support, EO, book on head
6. Improve dynamic postural control utilizing somatosensory inputs	Gait with normal base of support, EO, book on head
7. Decrease cervical muscle tightness	Active neck range of motion, all directions, slow movements
Phase 3	
1. Enhance vestibular adaptation	Visual fixation, EO, stationary target, fast and slow head movements, near and far targets
2. Enhance vestibular adaptation	Visual fixation, EO, moving target, slow and fast head movements
3. Promote use of somatosensory and vestibular inputs for postural control	Static stance on foam surface, EC, with and without book on head
4. Improve dynamic postural trol utilizing vestibular and somatosensory inputs	Gait with narrowed base of support, EC, with and without book on head
5. Improve dynamic postural control when head is moving utilizing all sensory inputs	Gait with normal base of support, fast head movements
6. Improve dynamic postural control utilizing somatosensory and vestibular inputs	March in place slowly, EO and EC, with and without book on head
7. Decrease cervical muscle tightness	Active neck range of motion, all directions, slow movements

[a]EO=eyes open; EC=eyes closed; VOR=vestibular ocular reflex.

Table 2. *Self-Reported Changes on the Dizziness Handicap Inventory[23] for Patient With Unilateral Vestibular Dysfunction[a]*

Physical/Functional Factors (0- to 8-Week Changes)	
+	Fewer restrictions on travel for business or pleasure
+	Ability to move head quickly
+	Ability to do job and household responsibilities
++	Ability to walk down the aisle of a supermarket
++	Ability to walk down a sidewalk
+	Ability to bend over

[a]+=item rated as "a little improved"; ++=item rated as "much improved."

formed without any observable increase in sway for 60 seconds. Tandem gait with eyes open could be performed for seven steps (improved from five steps). The patient's active cervical range of motion was full, and she no longer reported any feelings of muscular pulling during active cervical movements. Posture was improved, as noted by a more erect stance and only a minimally displaced forward head. During observation of gait, her base of support appeared normal and she demonstrated some arm swing and trunk rotation. She was able to walk in a straight line without difficulty but continued to stagger to the side if she attempted to turn quickly. She was able to ambulate in a straight path without slowing her speed of gait while rotating her head from side to side with only occasional cross steps.

Repeat sinusoidal vertical-axis rotation testing revealed less asymmetry of rotation-induced nystagmus (values were 2.5 standard deviations below the norm only at two frequencies of rotation—0.01 and 0.02 Hz), consistent with some central adaptation to the damage to the right inner ear (Fig. 1). Repeat posturography testing revealed an improvement in the patient's ability to stand on a sway-referenced platform with eyes closed

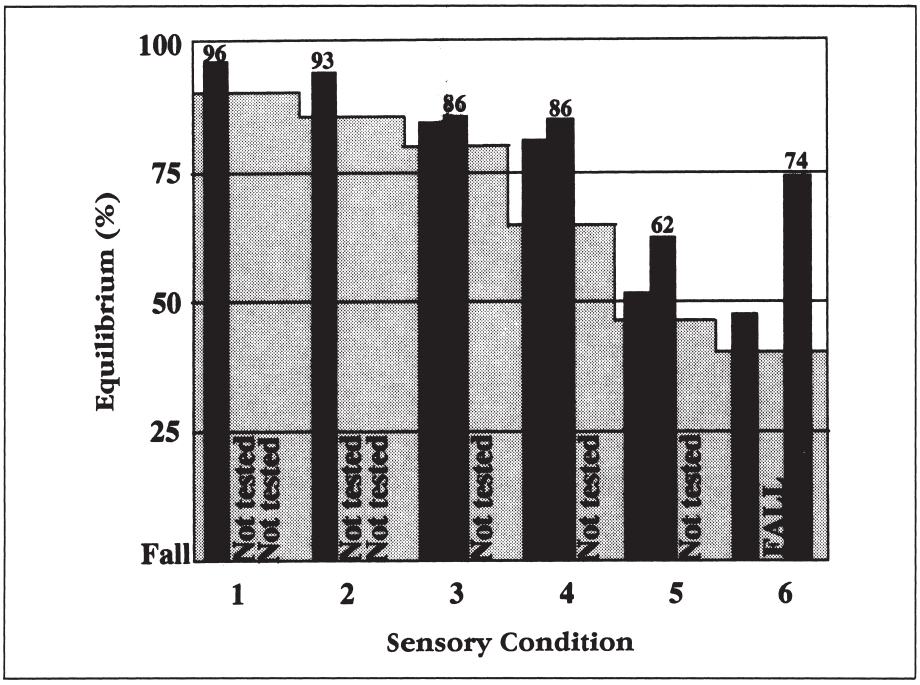

Figure 3. *Posttreatment (week 8) posturography (sensory organization) test results for patient with unilateral vestibular dysfunction.*

and with eyes fixed on a sway-referenced visual surround (Fig. 3). With eyes closed (sensory condition 5), the improvement consisted of no falls and less sway. With eyes open and fixed on a sway-referenced visual surround (sensory condition 6), the improvement consisted of less sway. Comparing the best performance on each trial, there was a 59% improvement (ie, less sway) on sensory condition 5 and a 28% improvement on sensory condition 6, as compared with the pretreatment posturography test.

The three-dimensional movement analysis revealed several changes indicative of improved postural stability (Tab. 3). The patient demonstrated reduced COG displacements during quiet standing with feet together and with eyes open and closed, which is a marker of improved balance control.[18] During preferred pace (or free) gait, velocity increased and double support time decreased, indicating that the patient spent more time in single-limb stance and moved more quickly during gait. The maximum COG-COP moment arm increased during free gait and preferred pace chair rise, indicating that the patient was able to increase the distance between the body's COP and COG—another marker of a higher level of stability.[18] Reduced medial-lateral COG excursion occurred along with reduced head displacements (relative to the trunk) in all directions and reduced head accelerations in the flexion-extension and lateral flexion directions, indicating that the patient walked in a more stable manner even when the speed of gait was controlled.

Bilateral Vestibular Hypofunction

History

A 53-year-old woman was referred to physical therapy with a diagnosis of BVH for a trial of physical therapy to help improve her balance, function, and activity level. The patient history, obtained through self-report, included 20 years of intermittent vertigo and the recent onset of worsening disequilibrium. The patient reported brief episodes of dizziness intermittently over the preceding 6 years, which usually followed a cold or flying in an airplane and would last a few days at a time. Four months prior to evaluation, the patient noted the onset of a persistent "floating" sensation made worse by head movements and following two upper respiratory infections. The patient complained of feeling unsteady while walking and of being nauseated and fatigued. All of these symptoms had been unchanged for 4 months prior to referral to physical therapy. The etiology of vestibular damage was unclear but believed to be due to either sequential unilateral damage (such as might be seen with viral labrynthitis) or a degenerative process involving only the vestibular portion of the inner ear or eighth cranial nerve.

Prerehabilitation Findings

Examination by a neurologist revealed full extraocular movements with no spontaneous or gaze nystagmus. The patient's hearing was normal bilaterally. There was normal strength (tested via isometric resisted contractions in the same muscle groups that were tested in the patient with UVD) in all extremities and no limb ataxia. Vibration and position sensation were present at the toes. A Romberg test was negative. Tandem gait was done well while the patient was walking forward and backward with eyes open. Positional testing revealed no nystagmus and only momentary dizziness in left and right ear down positions. There was no spontaneous, gaze, post–head-shaking, or positional nystagmus behind Frenzel lenses.

An audiogram was normal. The only abnormality on the electronystagmogram was very small (nearly absent) caloric responses bilaterally. Sinusoidal vertical-axis rotation with a peak velocity of 50°/s revealed extremely low gains (more than 3 standard deviations below normal) of the vestibular-ocular reflex at frequencies of 0.01, 0.02, 0.05, and 0.1 Hz, with values below 0.1 (Fig. 4). At higher frequencies of rotation, there was some remaining function, with gains only slightly below normal. Visual

Table 3. *Percentage of Improvement From Pretreatment in Kinematic Indicators of Stability[a] During Locomotor Performance for Patient With Unilateral Vestibular Dysfunction*

	Improvement (%)[b]
Feet together, eyes open	
ML COG displacement[c]	32
Feet together, eyes closed	
AP COG displacement[c]	41
ML COG displacement[c]	62
Free gait	
Double support time	20
Velocity	15
COG-COP maximum moment arm[d]	38
Paced gait	
ML COG excursion[c]	51
Head flexion displacement[c]	51
Head abduction displacement[c]	23
Head rotation displacement[c]	59
Head flexion acceleration[c]	70
Head lateral flexion acceleration[c]	84
Free chair rise	
COG-COP maximum moment arm	35

[a]ML=medial-lateral; AP=anterior-posterior; COG=center of gravity; COP=center of pressure.

[b]Improvement corresponds to a decrease in the value for all kinematic indicators of stability except velocity and COG-COP maximum moment arm; for these items, improvement reflects an increase in these values.

[c]Measures are relative to the room.

[d]COG-COP maximum moment arm is the difference between the body's COP and COG. A larger moment arm indicates that a state of less biomechanical stability is allowed to occur during the activity; that the patient allows the moment arm to get larger during an activity (and does not fall) signifies a higher level of overall balance control.[17]

vestibular interaction testing showed that the gain of the vestibular ocular reflex was below normal limits, a finding consistent with BVH. There was no evidence of brain-stem or cerebellar dysfunction.

Posturography showed excessive sway on one trial on a fixed platform with eyes closed, excessive sway on one trial with eyes open on a sway-referenced platform, and the inability to stand on a sway-referenced platform with eyes closed or eyes fixed on a sway-referenced visual surround (Fig. 5).

During the initial physical therapy examination, the patient's sensation for light touch was intact and proprioception was intact as noted by the patient's ability to accurately describe the joint position at the knees, ankles, and toes. The joint was repositioned with the patient's eyes closed. Patellar and Achilles deep tendon reflexes were intact. Manual muscle testing revealed Normal (5/5) strength in the following muscle groups: shoulder girdle; hip extensors and abductors; knee flexors and extensors; and ankle dorsiflexors, plantar flexors, invertors, and evertors. Heel-to-shin movements, finger-to-nose movements, and rapid alternating movements of the forearms were performed quickly, accurately, and smoothly, bilaterally. Extraocular movements were full. Saccadic eye movements could be performed fully but caused nausea. The patient reported blurry vision, a continuous sensation of dizziness at an intensity of 7/10, and the perception of frequent disequilibrium at a severe intensity (10/10). The patient scored 74 out of a maximum of 100 on the DHI,[23] reporting problems in six of the seven items related to physical activities, in eight of the nine items related to functional activities, and in eight of the nine items related to emotional health. Clinical balance assessment showed that the patient could perform tandem stance with eyes open for 60 seconds, was unable to perform tandem stance with eyes closed, and was able to perform unilateral stance with eyes open for 22 seconds and with eyes closed for 3 seconds. Stance on foam with eyes open could be performed for 20 seconds; it could not be performed with eyes closed. Tandem gait could be done for six steps with eyes open, but the patient was unable to walk in a straight line while rotating her head from side to side.

Rehabilitation

The combined vestibular adaptation/substitution treatment program that the patient received during the first 8 weeks consisted of exercises and activities outlined under phases 1 through 3 in Table 4. The patient performed phase 1 of the program during weeks 1 through 3, phase 2 during weeks 4 through 6, and phase 3 during weeks 7 and 8. The patient progressed to each new phase as her performance on the previous phase improved, as noted by her ability to perform the exercises easily and without an increase in the perception of dizziness or disequilibrium. The rationale for each activity included in the treatment program is briefly listed in Table 4 and is based of the work of Herdman.[3] The patient noted daily which exercises she performed at home and any particular difficulties she experienced utilizing the standard patient compliance record. During the second 8 weeks of treatment, the patient continued to perform independently at home the phase 3 activities that were difficult for her (which included all those listed). Weekly

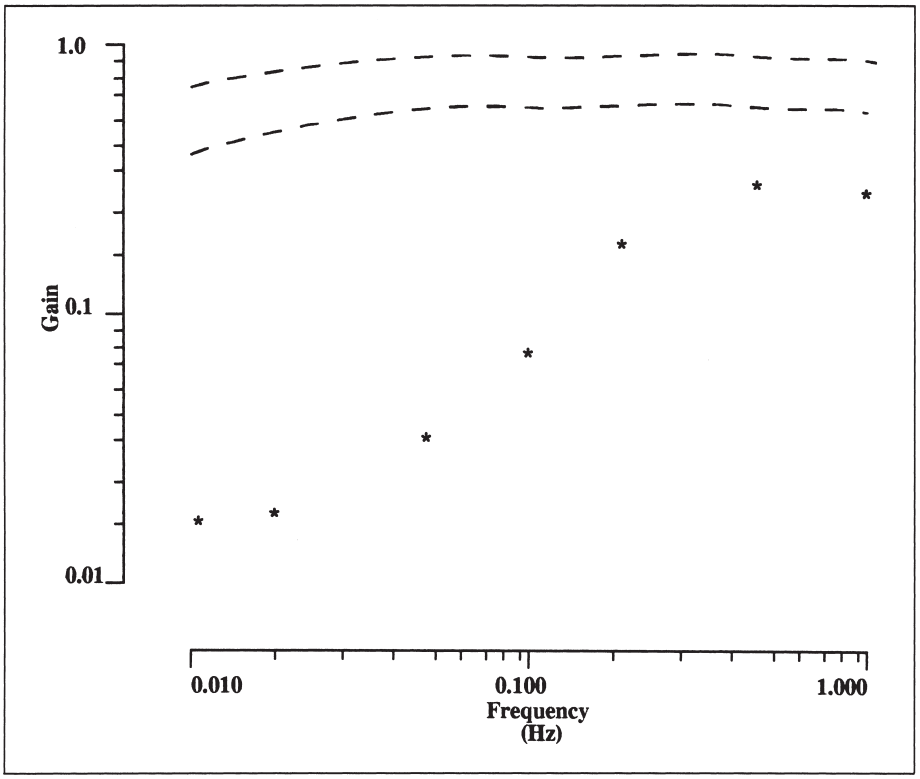

Figure 4. *Sinusoidal vertical-axis rotation test results for patient with bilateral vestibular hypofunction before treatment (*), demonstrating reduced gains at all frequencies of rotation with markedly reduced gains at the lower frequencies of rotation. Phase and asymmetry test results could not be interpreted accurately due to the low gains. No change of gain occurred following treatment.*

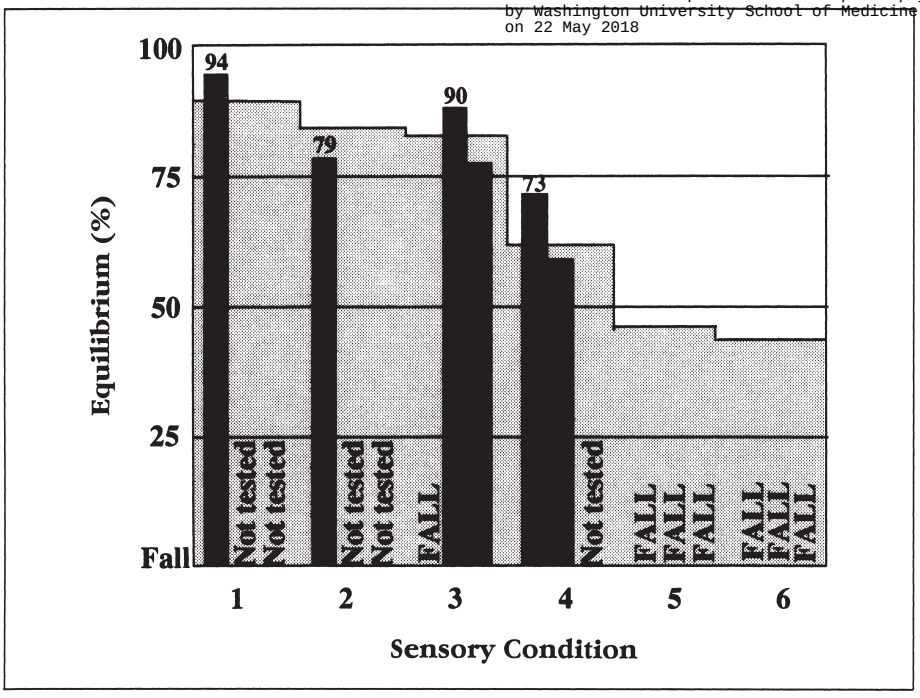

Figure 5. *Baseline (week 0) posturography (sensory organization) test results for patient with bilateral vestibular hypofunction.*

phone calls were made to the patient during weeks 9 through 15 to monitor progress and compliance. Over the course of the 16-week treatment period, the patient reported performing the exercises twice daily five or six times per week. After 8 and 16 weeks of treatment, the patient was re-evaluated in the clinic and in the biomotion laboratory utilizing the same measures as those used during the initial assessment. Repeat SVAR testing and posturography were performed after 8 and 16 weeks of treatment.

The treatment program designed for this patient (Tab. 4) was based on the following interpretation of the patient's condition:

1. The patient's primary problem of impaired postural stability was related to her markedly reduced (but not absent) BVH, as supported by a primary pattern of vestibular dysfunction on posturography testing[24,25] (Fig. 5).

2. The patient showed little evidence of effectively utilizing vestibular information for postural control, as supported by her inability to successfully complete any trial on sensory conditions 5 and 6.

3. The patient demonstrated effective use of both visual and somatosensory information for postural control when it was available, but she appeared to have difficulty switching from primarily utilizing one sensory modality to another (as evidenced by excessive sway on the first trials on sensory conditions 3 and 4 followed by improvements on successive trials). The patient's performance on posturography testing (sensory conditions 3, 5, and 6) and clinical balance testing suggest a possible preferential use of visual information for postural control.[24,25]

Postrehabilitation Findings

At the conclusion of the first 8-week period of treatment, the patient reported a slight decrease in the inten-

Table 4. *Vestibular Rehabilitation Treatment Program and Its Rationale for Patient With Bilateral Vestibular Hypofunction*

Rationale	Treatment Activity[a]
Phase 1	
1. Promote use of VOR and COR for gaze stability	Visual fixation, EO, stationary target, slow head movements
2. Promote use of saccadic eye movements for gaze stability	Active eye and head movements between stationary targets, sitting
3. Improve ability to utilize somatosensory and vestibular inputs for postural control	Static stance, EO and EC, feet together, arms outstretched
4. Improve ability to utilize vestibular and visual inputs for postural control	Static stance on foam surface, EO, arms outstretched
5. Improve dynamic postural control utilizing all sensory inputs	Gait with narrowed base of support, EO
6. Improve dynamic postural control utilizing all sensory inputs	March in place, EO
Phase 2	
1. Promote use of VOR and COR for gaze stability at various speeds of head movement	Visual fixation, EO, stationary target, fast head movements
2. Encourage vestibular adaptation by inducing retinal slip	Visual fixation, EO, moving target, slow head movements
3. Promote use of saccadic eye movements for gaze stability	Active eye-head movements between stationary targets in sitting and standing, fast and slow speeds, EO
4. Encourage resetting of VOR gain	Imaginary visual fixation, EC, small head movements, self-selected speeds
5. Improve ability to utilize somatosensory and vestibular inputs for postural control	Static stance, semitandem, EO and EC, arms close to body
6. Improve ability to utilize vestibular inputs for postural	Static stance on foam support, EO and EC, arms close to body control
7. Improve dynamic postural control utilizing vestibular inputs	Gait with narrowed base of support, EO
8. Improve dynamic postural control with head moving utilizing all sensory inputs	Gait with normal base of support, slow head movements, EO
9. Improve dynamic postural control utilizing all sensory inputs	Gait with wide turns to right and left, EO
10. Improve ability to utilize somatosensory inputs during ankle strategy postural movements and lateral body movements	Balance board practice, EO
Phase 3	
1. Encourage vestibular adaptation by inducing retinal error at various head speeds	Visual fixation on a moving target, fast head movements, EO
2. Promote use of VOR and COR for gaze stability	Active eye-head movements between stationary targets in standing, EO
3. Encourage resetting of VOR gain at various head speeds	Imaginary visual fixation, EC, small head movements, fast speed
4. Improve ability to utilize somatosensory and vestibular inputs for postural control	Static stance, semitandem, EO and EC, arms across chest
5. Improve ability to utilize vestibular inputs for postural control	Static stance on foam support, EO and EC, arms across chest
6. Improve dynamic postural control with head moving utilizing all sensory inputs	Gait with normal base of support, fast head movements, EO
7. Improve dynamic postural control utilizing all sensory inputs	Gait with sharp turns to right and left, EO

[a]EO=eyes open; EC=eyes closed; VOR=vestibular ocular reflex; COR=cervical ocular reflex.

sity of her dizziness (6/10) and a marked improvement in her perception of disequilibrium (reduced to an intensity of 5/10). She reported improvements in three physical activities and one functional activity previously reported to be problematic on the DHI (Tab. 5). In addition, she reported worsening of one emotional health component (depression) and one functional activity (participating in social activities), both of which she attributed to an increase in fatigue related to returning to a part-time office job. There was no change in the patient's static balance, as measured by ability to perform unilateral stance or stance on a foam support. On clinical testing of dynamic postural control, the only change noted was that the patient could walk in a straight line for 6.1 m (20 ft) with only one cross step while rotating her head from side to side.

Sinusoidal vertical-axis rotation testing demonstrated no change in the profoundly reduced gains of the vestibular-ocular reflex. Posturography showed a persistence of the patient's inability to stand on a sway-referenced platform with eyes closed or fixed on a sway-referenced visual surround and a slight decrease in sway when standing on a fixed platform with eyes closed.

The biomotion laboratory data revealed several improvements indicative of improved postural stability (Tab. 6). Specifically, the patient demonstrated reduced COG excursion in the anterior-posterior and medial-lateral directions during quiet standing with feet together and with eyes open and closed—markers of improved balance control.[18] During free gait, the patient's forward velocity increased, her medial-lateral COG excursion decreased, and her head movement accelerations (in the flexion/extension direction) decreased, indicating a more stable gait occurred (less body and head movements) at a faster speed. The acceleration of head lateral flexion movements increased, however, during free gait. During paced 1-Hz gait, the patient had a shorter double support time and

Table 5. *Self-Reported Changes on Dizziness Handicap Inventory[23] for Patient With Bilateral Vestibular Hypofunction*[a]

Physical/Functional Factors		
0- to 8-Week Changes	**9- to 16-Week Changes**	**Emotional Factors**
+ Ability to look up	+ Ability to look up	+ Less feeling of embarrassment in front of others
+ Ability to move head quickly	+ Ability to move head quickly	+ Less fear that others think individual is intoxicated
++ Ability to walk down aisle of supermarket	+ Ability to walk down aisle of supermarket	− Ability to participate in social activities
++ Ability to do job	+ Ability to walk down a sidewalk	− Feelings of depression

[a] +=item rated as "a little improved"; ++=item rated as "much improved"; −item rated as "a little worse."

stance duration time combined with an increased velocity of gait, indicating that she took longer strides. Reduced medial-lateral COG excursion occurred along with a variety of changes in head displacement (relative to the room) and reduced head accelerations in all directions. The patient's head displacements increased in some directions (flexion-extension and rotation), whereas a marked decrease was seen in the lateral direction. Finally, COG-COP maximum moment arm increased during paced chair rise.

Findings at Follow-up

At the conclusion of the 16-week treatment period, the patient reported no symptoms of dizziness and only occasional perceptions of disequilibrium at an intensity of 5/10. On the DHI, she reported further improvements (since the 8-week period) in four physical activities and in two items related to emotional health (Tab. 5). No items were reported as worsened since the 8-week test period. The only change in static balance noted was an improvement in her ability to stand on a foam support (60 seconds) with eyes open. Dynamic balance had improved in that tandem gait could be performed consistently for 10 consecutive steps without difficulty. Walking in a straight line for 6.1 m with head rotation from side to side could be performed without any cross steps.

There was no change in SVAR testing from the initial test or the 8-week test, indicating that no change occurred in this patient's baseline vestibular function. On posturography testing, the patient demonstrated less sway when standing on a sway-referenced platform with eyes open and closed and with eyes fixed on a sway-referenced visual surround (Fig. 6). Comparing the best performance among all trials for each sensory condition from pretreatment to posttreatment at 16 weeks, an 8% improvement (ie, less sway) was noted in sensory condition 4, a 39% improvement was noted in sensory condition 5, and a 65% improvement was noted in sensory condition 6. In addition, the patient demonstrated consistent performance and normal sway when standing on a fixed platform with eyes open looking at a sway-referenced visual surround (sensory condition 3).

On three-dimensional movement analysis of ADL tasks, the patient demonstrated continued or further changes in performance (with a few exceptions), indicative of further improvements in postural stability (Tab. 6). Improvements in performance were only partially sustained during the 9- to 16-week period of treatment for anterior-posterior COG excursion with feet together and eyes closed, medial-lateral COG excursion during free gait, double support and stance duration during paced gait, and COG-COP maximum moment arm during paced chair rise. Performance was still, however, improved over baseline for these factors. Head displacements during paced gait in the flexion-extension and lateral flexion directions were reduced, whereas head displacements in the rotation direction were slightly higher. Head accelerations during paced gait were reduced in all directions at the 16-week test period. These results indicate that the patient moved her head less distance (overall) and less frequently during gait activities in which the speed of gait was controlled.

Discussion

Both patients demonstrated improvements in postural stability and reported improvements in the perception of disequilibrium and handicap following 8-week individually designed programs of vestibular physical therapy. The patient with BVH also reported a decrease in the perception of dizziness. For the patient with BVH, improvements in performance noted after an initial 8-week period of treatment continued to occur during the second 8-week period when the patient continued exercises at home with telephone supervision.

Although there were some similarities in the patients' response to treatment, there were important differences between these two patients. The patient with UVD was 6 months poston-set of her vestibular damage and reported persistent and stable symptoms for 3 months prior to beginning vestibular physical therapy. Although most of the spontaneous resolution of symptoms associated with UVD occur within 3 to 6 months,[7,14] recovery can also be delayed or incomplete.[7,26] That this patient demonstrated documented improvements in SVAR testing from pretreatment to posttreatment

Table 6. *Percentage of Improvement From Pretreatment in Kinematic Indicators of Stability[a] During Activities of Daily Living and Locomotor Performance for Patient With Bilateral Vestibular Hypofunction*

	Improvement (%)[b]	
	Change From 0 to 8 Weeks	Overall Change From 0 to 16 Weeks
Feet together, eyes open		
AP COG excursion[c]	60	66
ML COG excursion[c]	27	48
Feet together, eyes closed		
AP COG excursion[c]	87	76
ML COG excursion[c]	45	91
Free gait		
Average velocity	13	36
ML COG excursion[c]	59	44
Head flex/ext acceleration[c]	12	60
Head lateral flex acceleration[c]	−8[d]	20
Paced gait		
Average velocity	11	19
ML COG excursion[c]	46	57
Double support time	20	8
Stance duration time	17	3
Head flex/ext displacement[c]	−46[d]	31
Head lateral flex displacement[c]	328	146
Head rotation displacement[c]	−58[d]	−3[d]
Head flex/ext acceleration[c]	7	52
Head lateral flex acceleration[c]	20	50
Head rotation acceleration[c]	7	50
Paced chair rise		
COG-COG maximum moment arm[e]	14	9

[a]AP=anterior-posterior; ML=medial-lateral; COG=center of gravity; COP=center of pressure, flex=flexion; ext=extension.

[b]Improvement corresponds to a decrease in the values for all kinematic indicators of stability except velocity and COG-COP maximum moment arm; for these items, improvement reflects an increase in these values.

[c]Measurements are relative to the room.

[d]These values represent a decline in kinematic indicators of stability.

[e]COG-COP maximum movement arm is the difference between the body's COP and COG.

suggests that central adaptation to her right-sided vestibular damage occurred during the course of treatment. At least two possibilities exist to explain this finding: (1) that spontaneous recovery, perhaps delayed in this patient, occurred and could have occurred regardless of her participation in treatment and (2) that the vestibular adaptation program was effective in promoting compensation in this patient with an incomplete vestibular deficit.[7] In contrast, the patient with BVH experienced no change in baseline vestibular function (as tested by SVAR) during the course of the 16-week treatment period. This patient had some remaining vestibular function as evidenced by SVAR testing at higher frequencies of rotation (0.2, 0.5, and 1.0 Hz). For this reason, a treatment program that focused on both the adaptative capability of the central nervous system and the substitution of alternative sensory information for postural control and gait was used. This patient demonstrated the ability to progress from less difficult (phase 1) to more difficult (phase 3) exercises during the course of treatment and also demonstrated improvements in performance—both factors that support the use of this treatment approach as a logical one.

If the treatment program for the patient with UVD were related to the improvements that were made, why might they have occurred? One possibility is that the patient was able to "train" her central nervous system, through the repetition of the various activities performed, to reinterpret the available vestibular information so that it more accurately represented what was truly occurring (ie, the interpretation of the vestibular input was more in agreement with that of the somatosensory and visual inputs in situations in which sensory conflict was absent).[7,10] Another possibility is that the patient's increased range of cervical mobility, combined with the experience of performing activities and movements that she had been avoiding since her initial onset, allowed her central nervous system to "realize" that she indeed had some postural control that was present and available for use on a daily basis.[10] A third possibility is that the patient, through the repetition of the various activities performed, was trained to more efficiently use any available sensory input (visual, somatosensory, or vestibular) for postural control and to choose among the various sensory information available as a situation required.[7] Finally, a combination of these factors may be operative.

If the improvements demonstrated by the patient with BVH were related to the treatment intervention, why might these have occurred? Clearly an improvement (or recovery) of vestibular function did not occur. One possibility is that the patient was trained to better use somatosensory and visual inputs for postural control in situations in which this sensory information was available (eg, the ADL tasks).[7] Another possibility is that the patient

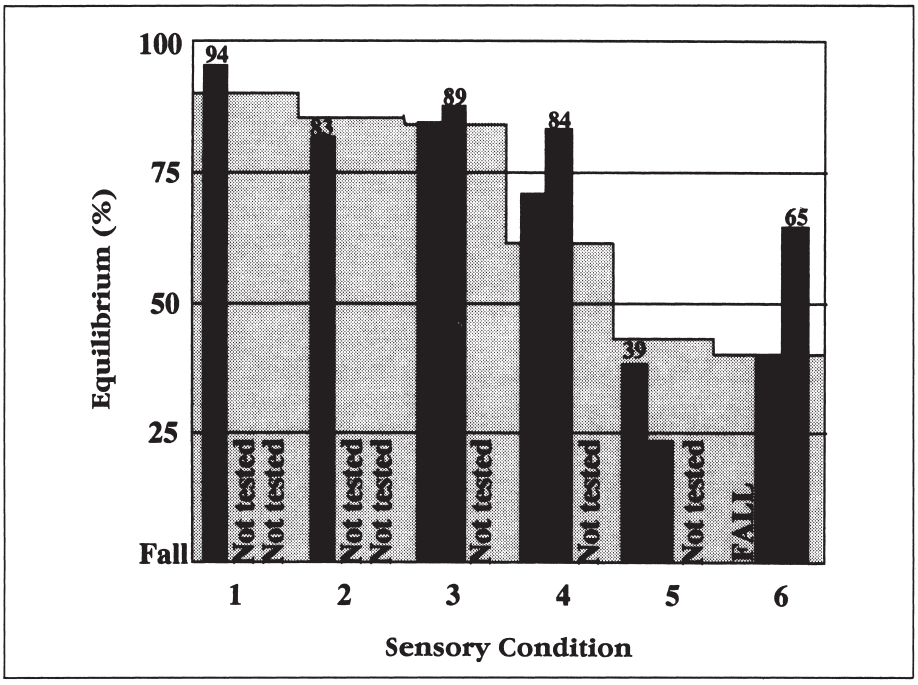

Figure 6. *Posttreatment (week 16) posturography (sensory organization) test results for patient with bilateral vestibular hypofunction.*

was able to maximize the use of the reduced vestibular information that was available to her.[7]

Both patients demonstrated improvements in clinical balance testing, although the patient with UVD made more improvements than the patient with BVH during the first 8 weeks of treatment. This may be related to the greater capacity of the patient with UVD to undergo adaptation or spontaneous recovery due to the lesser severity of damage to the vestibular system. At initial testing, the patient with BVH actually performed better than the patient with UVD on unilateral stance testing. This finding may be explained by the possibility that the patient with BVH may already have switched reliance on sensory information used in selected situations, such as in unilateral stance or during the single-limb phase of gait, to preferentially utilize proprioceptive inputs for postural control due to the severity and duration of her vestibular damage. Perhaps the patient with UVD had not undergone such a change.

How might the posturography results be explained for each patient? For both patients, the improvements seen in sensory conditions 5 and 6 indicate a definite improvement in the ability to utilize vestibular information for postural control in situations where both visual and proprioceptive inputs were reduced or distorted (Figs. 2, 3, 5, 6). For the patient with UVD, this change could be explained by either spontaneous recovery or perhaps the effects of the vestibular adaptation treatment program. For the patient with BVH, other factors must be considered, During sensory condition 5 (sway-referenced platform, eyes closed), only distorted somatosensory information and no visual information were available. Perhaps, however, enough somatosensory information was still available for the patient to successfully maintain postural control during the task. Another explanation is that enough vestibular information remained for the patient to use to prevent a fall, perhaps in combination with available somatosensory information. A similar explanation could be offered to explain the improvements made in sensory condition 6, in which the patient demonstrated improved ability to maintain postural control in a situation in which there was conflicting sensory information—a task believed to be mediated through the vestibular system.

Overall, the changes measured during the performance of key ADL tasks (in the biomotion laboratory) for both patients were related to minimizing excessive motion—changes that can be interpreted as evidence of improved postural stability. Of interest to note are the changes related to head displacement and acceleration for these patients. When the speed of gait was controlled (a testing situation used to control for speed-related changes in performance), both patients demonstrated less overall head movement and acceleration at the completion of treatment. The patient with UVD initially had decreased cervical spine mobility; yet, as active range of motion improved over the course of treatment, head stability during paced gait increased. The patient with BVH, for whom gaze stability function was compromised due to the bilateral damage,[7] initially (ie, at the 8-week period) showed an increase in head displacements in some directions during paced gait even though head accelerations decreased in all directions. At the 16-week period, all head displacement and acceleration movements were reduced in range or frequency except that of head rotation displacement. These findings suggest that this patient had improved head stability also at the 16-week period, but underwent a different course of improvement over a longer period of time. Perhaps the patient with BVH used head rotation displacement in some way to help compensate for gaze instability during whole-body movement (such as gait), in which case no marked change in this aspect of head movement could signify an effective substitution strategy as outlined by Herdman.[7]

Summary

After participation in individually designed programs of vestibular phys-

ical therapy, two patients with distinctly different vestibular pathology demonstrated improvements in postural stability, as noted by self-report, posturography testing, and three-dimensional movement analysis of ADL tests. One patient demonstrated changes in baseline vestibular function during the course of treatment, whereas the other patient showed no change, suggesting the possibility that different explanations need to be considered to understand why these changes might have occurred. Improvements in performance noted cannot be attributed to practice of the tasks, as the three-dimensional movement analysis of the ADL tasks included activities not performed as part of the treatment program. There were similarities in the two patients' responses to treatment, even though the treatment programs differed. Further research is necessary with a similar, but larger, patient population to continue to investigate the effectiveness of this treatment approach for patients with peripheral vestibular dysfunction.

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