Review Article

Vestibular Rehabilitation Therapy for the Dizzy Patient

LH Tee,[1] *Bphty, Mphty (Neurology)*, NWC Chee,[2] *MBBS, FRCS (Edin & Glasg), FAMS (ORL)*

Abstract

A customised vestibular rehabilitation therapy (VRT) programme is an important treatment modality in patients with vestibular dysfunction resulting in motion-provoked vertigo, oscillopsia (gaze instability), disequilibrium and gait disturbances. We discuss in this paper the patient selection criteria for VRT, rehabilitation strategies for unilateral and bilateral vestibular deficits, and some of the compelling evidence to support the use of VRT in treating such patients.

Ann Acad Med Singapore 2005;34:289-94

Key words: Disequilibrium, Gaze stability, Oscillopsia, Vertigo, Vestibular exercises

Introduction

The use of vestibular exercises to treat patients with persistent symptoms of positional vertigo and disequilibrium has been around for many years. The Cawthorne and Cooksey exercises, developed in the 1940s, consist of a series of eye, head and body movements aimed at treating patients with unilateral vestibular paresis and post-concussion syndrome. The rationale for the exercises, which originated from the observation that patients who were active recovered faster, was based on the supposition that the head movements that provoke the patient's dizziness play an important role in hastening the recovery process.

With a better understanding of the function and adaptation of the vestibular system, a customised vestibular rehabilitation therapy (VRT) programme adapted to suit the specific needs of the individual became widely accepted in the 1990s and has become the primary modality of treatment for a large number of patients with dizziness.

VRT involves the active participation of the patient. A trained vestibular therapist would initially assess the patient to establish the individual's main impairments and problems. Realistic treatment goals will be set with the patient. Treatment can be delivered in the form of supervised outpatient exercises or home exercise programmes. Vestibular exercises are supervised and progressed by the therapist. Appropriate outcome measures to measure dynamic visual acuity (DVA), balance and gait are adopted to measure the patient's progression. The average duration of a VRT programme ranges from 4 to 10 weeks.[1-8]

Which Types of Patients are Suitable for Vestibular Rehabilitation Therapy?

VRT is most effective in individuals with a stable unilateral peripheral vestibular deficit with incomplete central compensation.

Following an acute vestibular dysfunction such as in vestibular neuritis, sign and symptoms of skew deviation, nystagmus, vertigo, oscillopsia, disequilibrium, nausea and vomiting will typically resolve when acute compensation of the central nervous system (CNS) is achieved within the first 24 to 72 hours. Acute compensation occurs at the level of the vestibular nuclei under the influence of the cerebellum.[2] The system has to release the cerebellar inhibition to establish symmetrical tonic firing rates in the second-order neurons originating in the vestibular nuclei. Dynamic compensation to enable accurate vestibular responses to head movement is a slower process that takes several weeks and occurs by reorganisation of the brainstem and cerebellar pathways. While some patients may recover rapidly and spontaneously, patients who have an incomplete compensation or decompensation of the CNS to the vestibular system after the acute lesion will complain of residual motion-provoked vertigo, oscillopsia and disequilibrium. Such patients are good candidates for VRT and will benefit from a period of customised VRT supervised by a trained vestibular therapist.

VRT is also indicated in patients with stable bilateral vestibular deficits. The treatment strategies differ for

[1] Department of Physiotherapy
[2] Department of Otorhinolaryngology
Tan Tock Seng Hospital, Singapore
Address for Reprints: Dr Nelson Chee, Department of Otorhinolaryngology, Tan Tock Seng Hospital, 11 Jalan Tan Tock Seng, Singapore 308433.

unilateral and bilateral vestibular dysfunction and will be discussed in more detail below.

VRT is not suitable for patients whose symptoms occur in spontaneous episodes, such as in Meniere's disease, and when postural changes or body movements do not reliably provoke the sensation of dizziness. Alternative medical or surgical treatment strategies should be considered in such cases.

Unilateral Vestibular Lesion (UVL)

Patients with UVL will present with one or more of the following physical impairments:

1. Gaze instability or oscillopsia i.e, blurring of vision associated with head movements due to decreased vestibular-ocular reflex (VOR) gain (ideal VOR gain = 1).[4] Individuals will complain of gaze instability with head movements or disequilibrium. They often have difficulties watching television or reading due to blurring of vision. Road signs become blurred when these individuals try to read as they walk down the streets.
2. Motion-provoked vertigo is dizziness induced by specific head movements or activities/positions. Individuals tend to avoid head movements or certain activities due to hypersensitivity to movements.
3. Postural instability: Patients with UVL may present with disequilibrium either during static or dynamic activities. Disequilibrium increases when the individual is required to incorporate head movements or perform the task at a faster speed. Take for instance, crossing a busy street while turning the head sideways to look out for oncoming vehicles or running after a bus.
4. Sensitivity to motion in their environment: Individuals may complain of disequilibrium or dizziness induced by movement in their environment. This is due to a failure to differentiate between exocentric and egocentric motion. Individuals may experience imbalance or dizziness when standing or walking through crowds or visually busy environments, which worsens when they walk against the crowds. Challenging environments may include places such as supermarkets, shopping malls and train stations.
5: Physical de-conditioning: With prolonged avoidance of activities in the fear of reproducing symptoms and falls, individuals with UVL tend to develop physical de-conditioning.

Treatment for Patients with UVL

The main aims of VRT are to: 1) Increase gaze stability i.e., DVA during both static and dynamic activities of daily living (ADLs). ADLs, such as engaging in a conversation, nodding one's head, and walking along the street, require the individual to move his head. 2) Reduce the symptoms of dizziness. 3) Improve postural and gait stability. 4) Improve independence in ADLs. 5) Increase cardiovascular conditioning.

1. Treatment for Gaze Stability

The best stimulus for vestibular adaptation is by inducing a movement of the visual image across the retina known as a retinal slip during head movement. This retinal slip generates an error signal, and the CNS compensates to decrease the error signal by increasing the VOR gain.[9]

VOR X1 and X2 viewing exercises as described by Herdman[10,11] enhance vestibular adaptation by improving VOR gain and thus improve gaze stabilisation. In the VOR X1 viewing exercise, the patient views a stationary object while turning his head back and forth to the point where oscillopsia is induced. In the VOR X2 viewing exercise, the target moves in the opposite direction to the head rotation, again to the point where oscillopsia occurs. Both horizontal and vertical head rotations and target movements are performed.

Adaptation exercises need to be context- and environment-specific. The exercises should be designed such that they reflect the various environments in which the individual performs his or her normal ADLs.

In addition, the exercise programme can be designed to be performed at different head positions or frequencies for optimal effects (Fig. 1). This is because the VOR, adapted at a single frequency of head rotation, will only have the greatest VOR gain at that particular frequency and less gain at other frequencies.[12] VOR exercises are often incorporated with other balance exercises as appropriate.

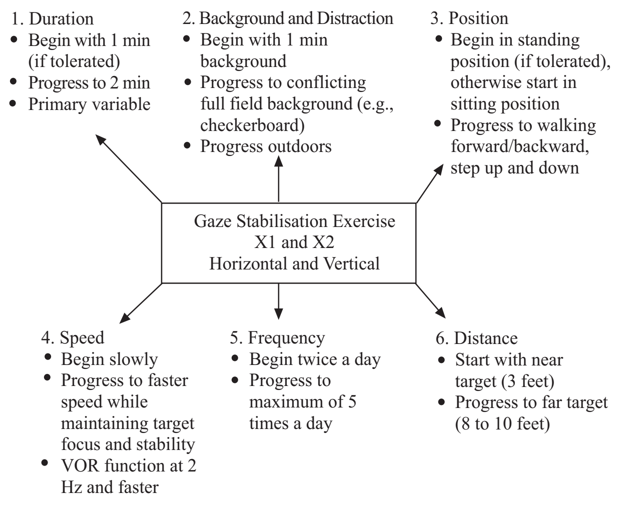

Fig. 1. Treatment progression for gaze stabilisation exercises (X1 and X2 viewing).

2. Treatment for Motion-provoked Vertigo

Habituation exercise reduces the symptoms of dizziness by repetitive exposure to the specific movement that provokes dizziness. It is the learned suppression of vertigo via repetitive exposure to movements. During habituation, there is a reduction in the amplitude of synaptic potentials (excitatory postsynaptic potential/EPSP) produced by the sensory neuron on the interneuron and motor neuron. The short-term changes of decreased ESPS amplitude during habituation exercises result in initial temporary reduction of vertigo. With persistent exposure to the provoking movements, the individual will experience gradual and permanent decline in dizziness across sessions. Reduction of dizziness is due to the long-term structural changes in the sensory cells, resulting in a decrease in the number of synaptic connections between the sensory neuron, interneurons and motor neurons. In addition, the number of active transmitting zones within the existing connections decreases. Habituation needs to persist for weeks to months to allow the structural changes of the sensory cells to take place so that the individual can experience permanent decline in the intensity of dizziness. [13]

The vestibular therapist will first identify specific positions or movements that provoked the patient's vertigo. The Motion Sensitivity Quotient Test developed by Shepard and Telian[14] can be used as a reference to assess and establish an individualised habituation exercise programme for patients with chronic UVL. The therapist needs to be creative in assessing provoking movements that may be experienced by the patients.

The therapist can choose up to 4 movements that produce mild to moderate dizziness for the individual to practise repeatedly. Each movement is performed as quickly as possible to produce mild to moderate symptoms, with rest between each movement to allow symptoms to subside and an additional 30-second wait before the next repetition. Three to 5 sets of exercises are performed once or twice daily. As habituation sets in, movements can be performed more rapidly in different positions or activities.

3. Treatment for Postural Instability

Treatment of postural instability for an individual with vestibular dysfunction requires an exercise programme that includes a variety of sensory environments involving alteration of the visual, somatosensory and vestibular inputs. Treatment aims to "force-use" the remaining vestibular inputs and recruiting substitution of the somatosensory and visual inputs to maintain postural control.

Alteration of visual inputs can include keeping the eyes closed, introducing a full visual field background (e.g., conflicting checkerboard), incorporating head movement, decreasing the lighting, and introducing a busy, conflicting visual environment or moving stimuli (e.g., a train moving pass the train platform). Some ways to alter the somatosensory inputs involve varying the supporting surfaces i.e., introducing conforming, uneven or moving surfaces or by decreasing the base of support. Balance can be challenged by standing or balancing on conforming surfaces (e.g., foam, wobble board, trampoline or balance beam) and walking on uneven surfaces (e.g., grass, ramps or treadmill). Incorporating head movement in various orientations during exercises can alter vestibular inputs. Take for instance, moving the head sideways, vertically and diagonally while standing, and subsequently, while walking; tossing a tennis ball sideways, up and down while standing, and subsequently, while walking, will also incorporate head movement. Habituation is facilitated if the individual also has motion-provoked dizziness induced by head movement. Eye tracking of the tennis ball as it is being tossed sideways or up and down also helps to train the smooth pursuit of the eyes.

Postural stability training involves the alteration of one or both of the visual, and somatosensory and vestibular environments. Balance training should begin with altering a single sensory modality, followed by involving multiple sensory modalities as the individual's balance improves.[9] Both the visual and somatosensory inputs can be altered as one's balance improves by practicing standing on conforming support surfaces (foam, wobble board, grass, mat) with the eyes closed or with head movement incorporated. More challenges can be introduced by tossing a tennis ball or bean bags while walking or doing a tandem walk on a thick carpet, a mat or grass, or walking on a treadmill and performing head movements. Subsequently, patients are encouraged to do these exercises outdoors (e.g., in the garden or a park).

4. Treatment to Decrease Sensitivity to Motion in the Environment

Individuals with increased sensitivity to motion in the environment can be gradually trained to become less sensitive by initially getting them to sit in a visually busy environment such as a shopping mall or supermarket. Subsequently, the individual may first push a trolley in the supermarket or shopping mall and then progress to walking independently, initially with the crowd, then against it. Head movements (horizontal, vertical or diagonal) can be incorporated as appropriate to increase the difficulty of the exercise.

5. Treatment for Physical Conditioning

A physical conditioning programme consisting of thrice weekly, 15- to 20-minute sessions should be included in a vestibular rehabilitation programme as appropriate to prevent or maintain cardiovascular endurance. These exercises can include brisk walking, stair climbing, walking on the treadmill, riding a stationary bicycle or swimming.

Bilateral Vestibular Lesion (BVL)

Patients with BVL will present with one or more of the following physical impairments, which are quite similar to that for UVL:

1. Gaze instability or oscillopsia associated with head movements is recognised as a hallmark of BVL.[15] Approximately one-third of the patients with BVL reported oscillopsia. The oscillopsia is worse when patients walk in dim light.
2. Postural instability and gait disturbances: Disequilibrium and gait disturbances are common problems in patients with BVL. Symptoms manifest most clearly in the dark. This is because individuals with BVL tend to rely on visual cues for maintaining balance. Difficulty with balance increases when either visual or somatosensory input is decreased. Individuals with BVL also tend to ambulate with a big base of support to compensate for postural instability. They have difficulty changing directions while walking as turning often results in disequilibrium.
   Herdman et al[16] reported that the incidence of falls in subjects with BVL was significantly higher compared to subjects with UVL. The study suggested that the increased risk for falling in patients with vestibular loss is related to the degree of vestibular impairment as well as increasing age.
3. Physical de-conditioning: With gaze instability associated with head movements and the increased risk for falling, individuals with BVL tend to decrease their level of activity and mobility. With time, decreased activity can lead to cardiovascular de-conditioning.

Treatment for Patients with BVL

Rehabilitation for individuals with BVL would involve exercises to enhance remaining vestibular function, as well as substitution strategies to replace the lost vestibular function and to improve gaze stability and postural stability.

1. Treatment for Gaze Stability

Strategies to improve gaze stability include:

(i) Potentiation of the cervical-ocular reflex (COR), where receptors in the muscles and joint facets in the upper cervical spine region project to the contralateral vestibular nucleus, producing a compensatory slow phase eye movement. This compensatory eye movement is opposite to the direction of head movement during low-frequency, brief head movements and complements the VOR.

(ii) Modification of saccades either by decreasing the amplitude of saccades or the use of corrective saccades during combined eye and head movement to maintain gaze stability on a visual target.

(iii) Increased smooth pursuit eye movement.

(iv) Central preprogramming of eye movement. This is an effective compensatory eye movement strategy to maintain gaze stability if the task is predictable.[12] Central programming of eye movements is based on the prediction or anticipation of intended motor behaviour. Herdman et al,[17] in their study to determine the role of central preprogramming in dynamic visual acuity with vestibular loss, suggested that both healthy subjects and subjects with UVL and BVL used central preprogramming to maintain gaze stability during predictable head movements. All the above substitution strategies are, however, only effective for low-frequency head movement (<1 Hz).

(v) Restriction of head movements to fixate gaze.

Some exercises that can be performed to improve gaze stability are as follows:

(i) VOR X1 and X2 viewing exercises to improve the remaining vestibular function and central pre-programming of eye movements.

(ii) The exercise involving active eye-head movements between 2 targets, as described by Herdman,[11] helps to facilitate the use of saccadic or smooth pursuit strategies and central pre-programming.

(iii) Imaginary target exercise helps to facilitate the central preprogramming of eye movements.[11]

2. Treatment for Postural Instability and Gait Disturbances

To improve postural stability, substitution strategies include: (i) the utilisation of visual and somatosensory cues; and (ii) central preprogramming of movement, which is also an effective compensatory strategy for postural stability when making an anticipated movement.[12]

Treatment progression for postural imbalance is similar to that of UVL. In patients with BVL, the focus is on training with a decreased step width to tandem ambulation, i.e., gradually decreasing the base of support as tolerated. Balance training could also include an obstacle course or functional tasks that require positional changes or head movements.

Behavioural and environmental modifications should also be considered, such as ensuring adequate lighting, non-skid floors, non-obstructed walkways, avoiding rushed movements, planning of movement before moving in crowded places and working in a sitting position to reduce the risk of falls. The therapist may need to consider prescribing walking assistive aids for individuals who cannot safely walk independently.

3. Treatment for Physical De-conditioning

The treatment for physical de-conditioning for BVL is similar to that for UVL.

Effectiveness of Vestibular Rehabilitation

VRT has become one of the main treatment modalities for patients suffering from vestibular dysfunctions. Patients are referred for VRT with the aim of helping to relieve them of symptoms of dizziness, gaze instability, disequilibrium and to improve their functional status. Studies have been done to evaluate the efficacy of VRT. To date, there have been a few randomised controlled studies conducted.

One early study by Horak et al[3] aimed to investigate the effectiveness of an individualised VRT programme compared with general conditioning exercise and vestibular suppressant medication in reducing dizziness and imbalance in patients with chronic UVL. Twenty-five subjects were randomly allocated to 1 of 3 treatment groups (VRT, general conditioning exercise and medication). The customised VRT programme consisted of habituation, gaze stability, eye-head coordination, balance and general conditioning exercises. A computerised dynamic posturography test was used to measure static balance function. The subject's self-rating of the intensity of dizziness and a dizziness questionnaire was used to document changes in self-perceived dizziness. The researchers demonstrated that after 6 weeks of treatment, all 3 treatment approaches reduced the symptom of dizziness, but only VRT improved balance.

Another study by Herdman et al[18] aimed to determine whether VRT facilitated the onset and rate of recovery of patients with UVL post-acoustic neuroma resection. Twenty-one subjects were randomised into experimental and control groups. The experimental group performed gaze stability exercises to improve vestibular gain and ambulation on postoperative day 3. Subjects in the control group did only smooth pursuit eye movement exercises and ambulation, also starting on postoperative day 3. Posturography test was used to evaluate the balance function. Patient's self-perceived vertigo and disequilibrium were reported on a visual analogue scale of 0 to10. The authors reported greater improvement in postural stability and self-perceived disequilibrium with the subjects in the experimental group.

A third experimental study by Krebs et al[4] evaluated the effects of VRT in 8 subjects with BVL. The subjects were randomly assigned to 2 groups. The experimental group received 8 weeks of outpatient VRT and 8 weeks of home VRT exercises. The control group received 8 weeks of control treatment (isometric strengthening exercises) followed by 8 weeks of outpatient VRT exercises. The VRT programme consisted of a combination of vestibular adaptation and substitution exercises, depending on the subject's rate of progress. Unlike the previous 2 studies mentioned earlier, gait and stairs locomotion were used as outcome measures to determine the effectiveness of intervention. In addition, the Dizziness Handicap Inventory was used to document the subject's self-perceived dizziness in the psychosocial and functional aspects. The results indicated that at the end of 8 weeks, subjects who underwent VRT walked faster and had better stability during walking and stair climbing compared to the control group during control treatment, even though there was no significant difference in their self-reported Dizziness Handicap Inventory scores. At the end of 16 weeks, both groups showed improvement in their functional testing and self-reported symptom scores. The sample size for the study, however, was too small to permit the generalisation of results.

In a recent larger scale experimental study by Krebs et al,[7] the authors sought to determine whether VRT improves gait stability (i.e., increased speed of walking and decreased base of support). One hundred and twenty-four subjects with both UVL and BVL were recruited for the study. Eighty-six subjects completed the 12-week intervention.

Subjects were randomly assigned to 2 groups. The experimental group received 6 weeks of outpatient VRT (one session a week), followed by 6 weeks of home VRT. The control group received 6 weeks of placebo treatment (i.e., isometric strengthening exercise), followed by 6 weeks of outpatient VRT (one session a week). The VRT programme consisted of both adaptation and substitution exercises for gaze stability training and balance training, according to the individual's rate of progression.

Gait-dependent variables were assessed at preferred speed using the 10-metre walk test and at a paced gait of 120 steps/min. The experimental group demonstrated greater improvement in gait velocity and stability at 6 weeks post-VRT treatment. At 12 weeks (after both groups had had VRT), both groups showed improvement in gait, with the experimental group demonstrating slightly more significant improvement.

Most of the above studies included training for gaze stability but did not include outcome measures to assess this variable. A recent study by Herdman et al[19] evaluated the effect of a VRT intervention on visual acuity during head movement in patients with UVL. This study is to date the only research that has used a dynamic visual acuity (DVA) test to assess improvement in gaze stability post-intervention. In this study, 21 subjects were randomly allocated into the experimental group and control group. The experimental group received a progressive home exercise programme that included adaptation, eye-head coordination and substitution exercises to improve gaze stability, and also performed balance and gait training exercises. The control group did placebo home exercises that consisted of saccadic eye movement with the head stationary, and balance and gait exercises without head movement. Both the experimental and control groups underwent 4 to 5 weeks of the home exercise programme at a frequency of 4 to 5 times daily (approximately 40 minutes of exercises daily, including 20 minutes of balance and gait exercises).

DVA during predictable and unpredictable head movements were used to evaluate changes in the subject's DVA. The subject's symptom of oscillopsia was measured on a visual analogue scale of 0 to 10. The authors demonstrated that there was significant improvement of DVA during predictable head movements in the experimental group after less than 5 weeks of treatment. Both the experimental and control groups reported a significant decrease in self-perceived oscillopsia. There was no correlation between improvement in oscillopsia and improvement in DVA during predictable or unpredictable head movements. In addition, age and time from onset did not contribute to the recovery of DVA, suggesting that the elderly or individuals with chronic vestibular hypofunction may benefit from VRT. The results suggested that the use of vestibular exercises fostered the recovery of DVA during predictable head movements.

Conclusion

VRT has been shown to improve vestibular function in patients with both UVL and BVL. VRT facilitates the reduction of self-perceived dizziness provoked by head movement or movement in a busy visual environment, improves mobility and balance function, and improves gaze stability associated with head movement. VRT needs to be customised to the individual's impairments and the exercises should be tailored according to each patient's rate of progression.

REFERENCES

1. Horak FB, Nasher LM, Diener HC. Postural strategies associated with somatosensory and vestibular loss. Exp Brain Res 1990;82:167-77.
2. Smith PF, Darlington CL. Neuromechemical mechanisms of recovery from peripheral vestibular lesions. Brain Res Brain Res Rev 1991;16:117-33. Erratum in: Brain Res Brain Res Rev 1992;17:183.
3. Horak FB, Jones-Rycewicz C, Black FO, Shumway-Cook A. Effects of vestibular rehabilitation on dizziness and imbalance. Otolaryngol Head Neck Surg 1992;106:175-80.
4. 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;109:735-41.
5. Shepard NT, Telian SA, Smith-Wheelock M, Raj A. Vestibular and balance rehabilitation therapy. Ann Otol Rhinol Laryngol 1993;102: 198-205.
6. Black FO, Angel CR, Pesznecker SC, Gianna C. Outcome analysis of individualized vestibular rehabilitation protocols. Am J Otol 2000;21: 543-51.
7. Krebs DE, Gill-Body KM, Parker SW, Ramirez JV, Wernick-Robinson M. Vestibular rehabilitation: useful but not universally so. Otolaryngol Head Neck Surg 2003;128:240-50.
8. Cohen HS, Kimball KT. Increased independence and decreased vertigo after vestibular rehabilitation. Otolaryngol Head Neck Surg 2003;128: 60-70.
9. Clendaniel RA, Helminaski JO. Rehabilitation strategies for patients with vestibular deficits. In: Arenberg LK, editor. Dizziness and Balance Disorders. New York: Kugler Publications, 1993:663-75.
10. Herdman, SJ, Whitney SL. Treatment of vestibular hypofunction. In: Herdman SJ, editor. Vestibular Rehabilitation (Contemporary Perspectives in Rehabilitation). 2nd ed. USA: F A Davis, 2000:387-423.
11. Herdman SJ. Assessment and treatment of balance disorders in vestibular deficient patient. In: Duncan P, editor. Balance Proceedings of the APTA Forum. Nashville, TN; 1990:87-94.
12. Herdman SJ. Role of vestibular adaptation in vestibular rehabilitation. Otolaryngol Head Neck Surg 1998;119:49-54.
13. Shumway-Cook A, Woollacott MH. Motor Control. Theory and Practical Applications. 2nd ed. USA: Lippincott Williams & Wilkins, 2001: 91-109.
14. Shepard NT, Telian SA. Programmatic vestibular rehabilitation. Otolaryngol Head Neck Surg 1995;112:173-82.
15. Bhansali SA, Stockwell CW, Borjrab DI. Oscillopsia in patients with loss of vestibular function. Otolaryngol Head Neck Surg 1993;109:120-5.
16. Herdman SJ, Blatt P, Schubert MC, Tusa RJ. Falls in patients with vestibular deficits. Am J Otol 2000:847-51.
17. Herdman SJ, Schubert MC, Tusa RJ. Role of central preprogramming in dynamic visual acuity with vestibular loss. Arch Otolaryngol Head Neck Surg 2001;127:1205-10.
18. Herdman SJ, Clendaniel RA, Mattox DE, Holiday MJ, Niparko JK. Vestibular adaptation exercises and recovery: acute stage after acoustic neuroma resection. Otolaryngol Head Neck Surg 1995;113:77-87.
19. Herdman SJ, Schubert MC, Das VE, Tusa RJ. Recovery of dynamic visual acuity in unilateral vestibular hypofunction. Arch Otolaryngol Head Neck Surgery 2003;129:819-24.