Habituation therapy for chronic vestibular dysfunction: Preliminary results

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Chronic vestibular dysfunction is often a frustrating problem for both patient and physician. A program of customized vestibular habituation therapy is introduced and its efficacy in a group of 65 patients is evaluated. Preliminary findings suggest that 59% of patients will have a dramatic improvement, after which their vestibular symptoms no longer cause any restriction in their lifestyles. An additional 23% of patients note considerable improvement, but have persistent symptoms that continue to restrict their activities. Vestibular habituation therapy is a rational, multidisciplinary approach to the treatment of chronic vestibular dysfunction that is a significant alternative to traditional pharmacologic management. Failure of vestibular compensation after involvement in a disciplined program of habituation therapy constitutes a much stronger indication for vestibular surgery in patients with unilateral peripheral lesions. (OTOLARYNGOL HEAD NECK SURG 1990;103:89.)

Among the special senses of the human nervous system, vestibular function is arguably the most complex. It requires complementary bilateral input from the vestibular end organs, which then must be integrated with visual and somatosensory signals in the central nervous system. Many disease processes involving the vestibular labyrinth are self-limited and recover spontaneously because of the remarkable ability of the central nervous system to compensate for labyrinthine injury. However, the otologist is frequently faced with patients who manifest persistent vestibular symptoms, either as a result of continued dysfunction in the vestibular end organ, or failure of vestibular compensation. In many cases, these symptoms result in significant disability.

Vestibular compensation can be defined as the process of recovery following a vestibular lesion by which an ideal functional state is re-established. This process requires a wide reorganization of central nervous system activity at the brainstem and cerebellar level.[1-4] When this process successfully achieves compensation, functional deficits are eliminated and the individual has appropriate equilibrium during daily activities. It should be noted, however, that compensation is a dynamic process that can be influenced by multiple factors, including medications,[5,6] level of arousal,[7] and input from the environment.[8,9]

In the treatment of patients with continued disability from vestibular lesions, the otologist has traditionally relied upon two modalities: medications to control symptoms, or surgery to stabilize or ablate inappropriate input from the vestibular end organ. It is not unusual to encounter patients in whom both approaches are either untenable or unsuccessful. For this reason, a program of vestibular habituation therapy for patients with chronic vestibular dysfunction was developed. For the purposes of this article, vestibular habituation refers to the reduction in a pathologic vestibular response brought about by repeated exposure to a provocative stimulus. This is a longlasting, albeit dynamic, process to be distinguished from adaptation, a transient neurophysiologic phenomenon characterized by a slow decline of afferent neuronal responses to a constant stimulus, which is not believed to contribute to long-term compensation.[10]

METHODS

Sixty-five patients treated after January 1988 completed vestibular habituation therapy (VHT) before January 1989. All patients underwent neurotologic evaluation by a physician, as well as vestibular function

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Table 1. Evaluation of disability

	Disability scale
0	No disability—negligible symptoms
1	No disability—bothersome symptoms
2	Mild disability—performing usual duties
3	Moderate disability—disrupts usual duties
4	Recent severe disability—medical leave
5	Established severe disability

Table 2. Assignment of response groups on the basis of level of persistent vestibular symptoms

	Response group
A	Complete resolution of symptoms
B	Marked improvement/mild symptoms
C	Mild improvement/persistent symptoms
D	No improvement
E	Worse

testing consisting of electronystagmography, rotational chair testing, and dynamic posturography. Involvement in VHT was suggested whenever one or more of the following findings was evident: (1) a history of positionally provoked vertigo; (2) significant abnormalities of equilibrium or actual falls on the sensory organization portion of posturography; (3) dramatic postural control abnormalities on posturography; or (4) other evidence of uncompensated vestibular lesion, such as positional nystagmus or rotational chair asymmetry. The managing neurotologist decided whether the patient should be referred for therapy, and discussed the rationale for such treatment with each patient. These patients represent our early experience with this modality and, because our goal was to gain familiarity with VHT, no attempt was made to provide matched controls. However, to be eligible for treatment, all patients had to have experienced 2 months of continuous symptoms prior to therapy. In reality, almost all had been symptomatic longer than 6 months, suggesting that spontaneous recovery within 12 to 16 weeks was unlikely (mean duration of symptoms, 3.1 years). No effort was made to select patients relative to their clinical diagnosis, and treatment was offered to all who met these criteria—even when central disease or mixed central/peripheral pathology was evident.

The program consisted of an evaluation by a physical therapist requiring approximately 1 hour. This included a general evaluation of muscle strength, joint limitations, postural and gait abnormalities, as well as an evaluation of response to 19 specific rapid positional changes.[11] These findings, along with the posturography results, were used to design a series of customized exercises unique to each patient. Because this program has a customized design, no rigid protocol can be provided in this publication for widespread use. However, for almost all patients the program included these three elements: (1) vestibular habituation exercises designed to facilitate habituation of specific pathologic responses; (2) postural control exercises designed to address underlying maladaptive patterns of postural control that may have contributed to continuing disability; and (3) a generalized conditioning program that was gradually advanced on the basis of the patient's age and functional capacity. The majority of patients performed these exercises twice daily at home, with program adjustments made by periodic telephone followup. Occasionally patients with special problems or restrictions returned for frequent outpatient evaluation to assist their progress. When habituation of abnormal responses was accomplished or no further benefit from therapy was evident, the patient was changed to a maintenance program, usually consisting of general conditioning exercises. Occasional patients required continued habituation exercises to maintain the level of compensation initially achieved.

Response to therapy was evaluated by a functional disability scale (Table 1). The score for each patient before and after therapy was determined by interviewing the patient and/or family members. In addition, each patient was assigned to a response group (Table 2) on the basis of the level of persistent vestibular symptoms.

RESULTS

Patient Population

Sixty-five patients completed therapy and were converted to a maintenance program during the study period. There were 27 men and 38 women in this study. They ranged in age from 20 to 89 years, with a mean age of 52 years (SD ± 16). The clinical diagnoses are listed in Table 3. There was a wide range of disability level before therapy. Thirty-five patients had disability scores of 1 through 3, which means that they were functioning in society at a relatively productive level, with various degrees of disruption resulting from vestibular symptoms. Twenty-six patients considered themselves disabled by their symptoms; sixteen of these had previously established permanent disability defined by earlier determinations of inability to pursue gainful employment, usually associated with compensation payments.

Table 3. Diagnoses of patients treated (N = 65)

Clinical diagnoses	
Unilateral peripheral disease (31)	11
Vestibular neuritis	6
S/P vestibular surgery	
Positional vertigo	
Classical BPPV	3
Nonclassical	1
Perilymph fistula	3
Labyrinthitis	2
Autoimmune	2
Delayed onset vertigo	2
Meniere's disease	1
Bilateral peripheral disease (4)	
Ototoxic drugs	2
Diabetic neuropathy	1
Idiopathic	1
Central disease (6)	
S/P CVA	3
S/P craniotomy	1
Vertebrobasilar insufficiency	1
Probable multiple sclerosis	1
Mixed central/peripheral (21)	
Vestibulopathy/anxiety	9
Head injury	7
Multifactorial/aging	4
Temporal bone fracture	1
Undetermined	3
TOTAL	65

S/P, Status post; *BPPV*, benign paroxysmal positional vertigo; *CVA*, cerebrovascular accident.

Response to Therapy

Figure 1 demonstrates that 82% of patients treated were assigned to a response group which indicated improvement in their symptoms (A, B, or C). Fifty-nine percent of the total group were placed in category A or B, which would indicate a dramatic improvement, and 23% of patients were assigned to group C, which indicates mild improvement. Most of these patients believed the therapy program had been worthwhile and had offered them a technique for management of active symptoms. Eight patients (12%) were assigned to group E, which indicates that they were made worse by therapy. As a group, these patients were more severely affected before therapy, with disability levels of 3 through 5 in each case. Their average objective findings on preoperative testing—including an objective measurement of posturography performance and their response to 19 positional maneuvers—demonstrated more significant abnormalities in balance function than those seen in patients who completed VHT in response groups A through C. Five of the group E patients had

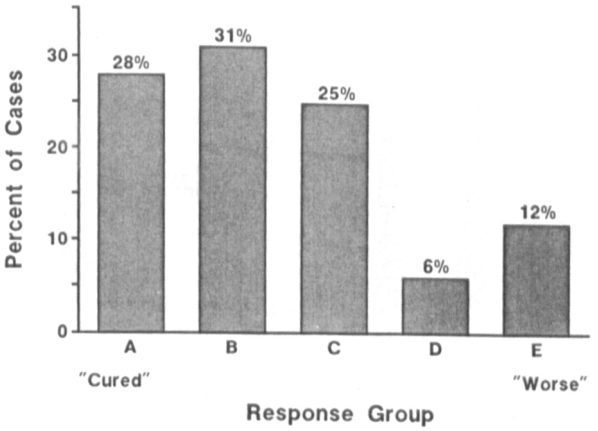

Fig. 1. Percentage of patients in each response group following vestibular habituation therapy.

clear peripheral lesions and were considered surgical candidates. Three have had repairs of perilymph fistulae, one has had a vestibular nerve section, and another had a labyrinthectomy, all with satisfactory outcome. The other three patients in group E all had earlier permanent disability—two had central nervous system findings on their objective testing, and the third was a patient who had failed to compensate after a vestibular nerve section several years earlier.

Improvement of Functional Capabilities (Disability Scores)

Figure 2 illustrates the pre- and post-therapy disability scores. Figs. 3 through 6 illustrate the results of therapy, separating patients by level of pre-therapy disability. Generally, patients with lesser disability levels improved considerably after therapy. A comparison of Figs. 5 and 6 would indicate that recent disability suggests a better prognosis for success in therapy. Seven of the ten patients who had not yet been placed on permanent disability (score = 4) had a dramatic response in their disability level. On the other hand, no patients who had established severe disability (score = 5) achieved dramatic recovery. Two patients in this latter group did achieve a disability score of 2 after therapy. Both of these patients had clear peripheral etiology for their vestibular dysfunction—one patient had labyrinthitis secondary to chronic otitis media, and the other secondary to an earlier viral endolymphatic labyrinthitis. An additional five patients improved to a better functional level (score = 3), but still had considerable difficulty with vestibular symptoms.

Of all disabled patients (score 4 or 5) who failed to improve in therapy (11 patients), three improved after

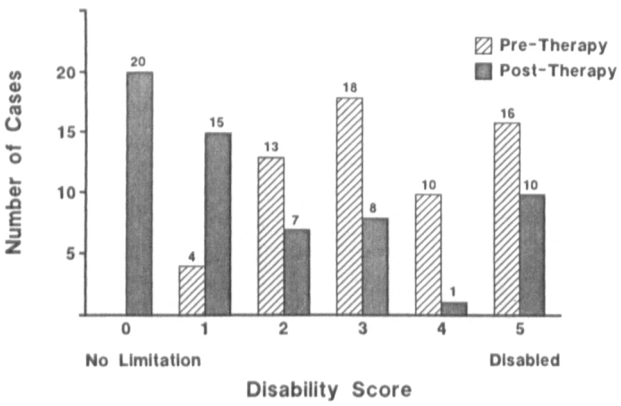

Fig. 2. Histogram demonstrates disability score before and after VHT.

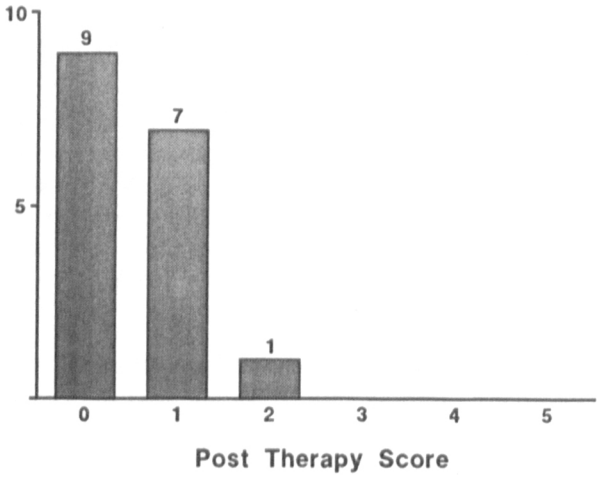

Fig. 3. Disability scores following VHT in patients with mild disability before therapy (score 1 and 2) (N = 17).

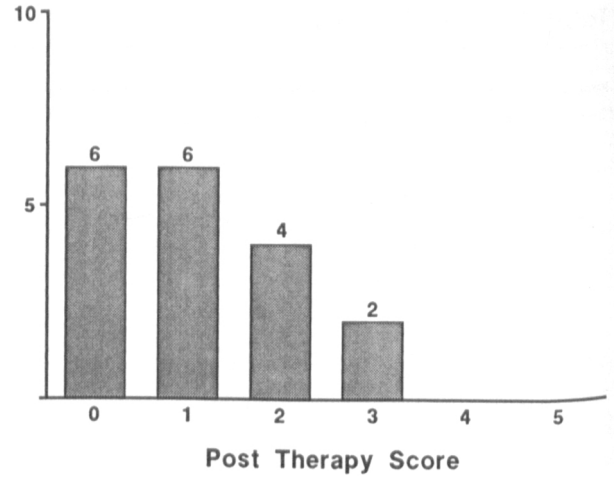

Fig. 4. Disability scores following therapy for patients with moderate disability before therapy (score 3) (N = 18).

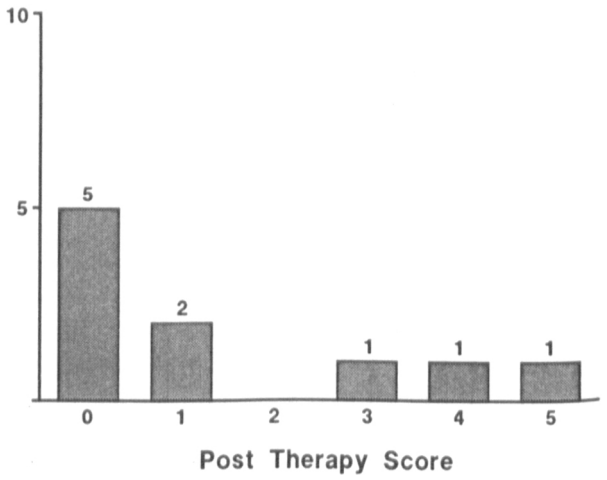

Fig. 5. Disability scores following therapy for patients with recent severe disability (score 4) (N = 10).

surgical treatment of peripheral vestibular lesions. The other eight patients had problems that were not amenable to surgical treatment—specifically, head injury with mixed central and peripheral abnormalities (3), bilateral vestibular paresis (2), complicating psychiatric disease (2), and probable multiple sclerosis (1 case).

Figure 7 suggests a linear relationship between pre- and post-therapy disability scores for groups 1 through 4, with all groups having a mean disability score of less than 2.0. The results in group 5 were much poorer, as previously discussed.

Predictors of Success in Therapy

Statistical analysis of independent variables, consisting of multiple linear regression analysis and single-pair correlation coefficients for the pre-therapy history and objective findings, was performed. No single independent variable was strongly reliable in predicting a positive outcome from VHT. Analysis did suggest that objective findings—especially posturography—were consistent with the subjective disability score assigned to the patients.

It is noteworthy that patients' age and duration of symptoms did not adversely influence the outcome of therapy. Specifically, duration of symptoms greater than 1 year had no negative impact upon response group or post-therapy disability score.

When the findings on the pre-therapy evaluation (Table 4) suggested a lesion that was objectively uncompensated (persistent spontaneous nystagmus, rota-

Table 4. Lesion status on pretherapy testing

Group	Objective results	
	Compensated (29)	Uncompensated (35)
A	41%	14%
B	30%	31%
C	11%	34%
D	7%	6%
E	11%	14%

tional chair asymmetry, or abnormal posturography with falls), the patient was less likely to achieve an excellent outcome (response group A or B) when compared to patients with evidence for satisfactory compensation. It should be noted, however, that if group C results are included, the overall improvement rate is equal.

Table 5 displays the pre-therapy test results, separated according to site of lesion. Several interesting findings emerged. If the testing suggested an isolated unilateral peripheral lesion, 80% of patients improved, almost invariably achieving a post-therapy disability score of 0 or 1. It should be noted, however, that in every case VHT caused either an improvement or a worsening of symptoms in this group. The five patients (20%) in this group who became worse were considered surgical candidates. Patients with bilateral peripheral disease demonstrated minimal response to therapy. Surprisingly, patients with pure central nervous system involvement showed excellent results, but this group is too small to reliably evaluate. Twenty-eight patients had mixed peripheral and central findings, with many of these being patients with head injury. Patients in this category responded more poorly than the overall group.

Implications of Diagnosis

There were 12 patients with vestibular complaints after head injury treated in the therapy program. Only two of these showed dramatic improvement, with an additional five showing minor improvement. Seven of twelve patients remained permanently disabled following VHT. Only one patient with head injury achieved a disability score of 0 after therapy.

Patients with vestibular neuritis or labyrinthitis, despite poor initial compensation requiring therapy, responded extremely well. Ninety percent of patients achieved disability scores of 0 through 2, and only two patients failed to show improvement. No patients in this group were made worse by therapy.

Similar satisfactory results were obtained in patients treated for serious failure of compensation or delayed

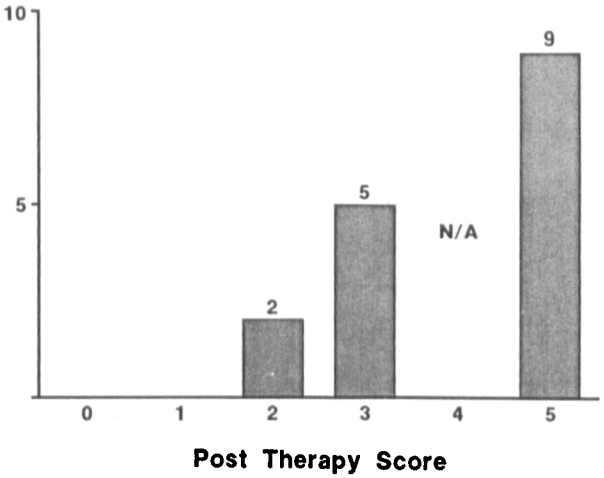

Fig. 6. Disability scores after therapy for patients with previous established severe disability (score 5) (N = 16).

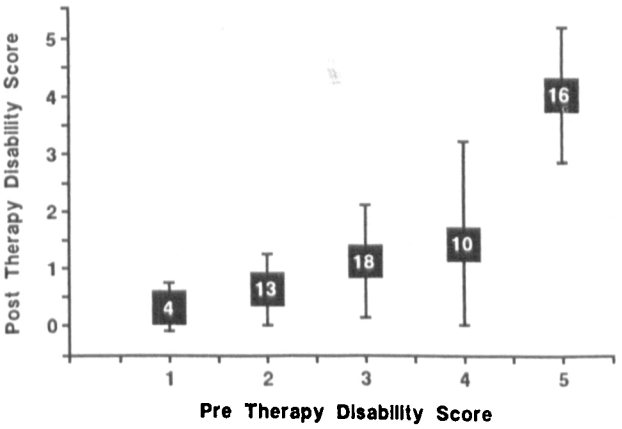

Fig. 7. Mean and standard deviation post-therapy disability scores plotted against pre-existing disability level. A linear relationship exists in groups 1 through 4, with dramatically poorer results in group 5. Number in boxes = number of patients in each group.

decompensation after surgery that ablated unilateral peripheral vestibular function. These included patients after labyrinthectomy, retrolabyrinthine vestibular nerve section, or acoustic neuroma resection. Most patients achieved a post-therapy disability score of 0 or 1.

All patients with benign paroxysmal positional vertigo uncomplicated by head injury or central nervous system findings improved to a disability score of 0 or 1. In one remarkable case, the patient continued to have dramatic nystagmus (classical for BPPV) when the right Hallpike maneuver was performed, but was completely free from vertigo.

Table 5. Results of therapy, separated according to site of lesion

Response group	Unilateral peripheral (N = 25)	Bilateral peripheral (N = 4)	Pure central (N = 3)	Mixed (N = 28)	Total (N = 65)
A	36%	—	67%	18%	28%
B	32%	—	33%	36%	31%
C	12%	75%	—	29%	23%
D	—	25%	—	7%	6%
E	20%	—	—	10%	12%

DISCUSSION

Although vertigo can be a very troubling symptom for our patients, it is almost always caused by benign self-limited disease. While it is generally agreed that surgical treatment is reserved for patients who have not responded to conservative treatment, a rational non-surgical approach to patients who fail to compensate for vestibular disease has been slow to emerge. Treatment with medications and immobilization of patients during and after an acute vestibular crisis is probably counterproductive.[12]

Since the concept of head exercises as a treatment for vertigo was introduced by Cawthorne,[13] this modality has received rare attention in the literature. McCabe[14] reviewed the physiologic basis for such treatment and discussed his personal approach to counseling patients about such treatment. He found labyrinthine exercises to be most useful in patients with stable peripheral vestibular lesions, and believed they were "our most useful single tool in the alleviation of protracted recurrent vertigo." Norré[15] has brought about renewed interest in vestibular habituation therapy in recent years, as well as offering a systematic approach to the assessment of positionally evoked symptoms. He has applied this modality primarily for treatment of patients with benign paroxysmal positional vertigo or unilateral peripheral vestibular hypofunction.

Recent improvements in the understanding of human postural control[16] and the commercial availability of equipment for clinical assessment have allowed a more critical analysis of patients who report dysequilibrium. Most noticeably, it has improved our ability to recognize defects in the interactions between the vestibular system and other sensorimotor inputs to the central nervous system. It also allows the detection of patients who have adopted maladaptive strategies to maintain stance following a vestibular insult.[17] In harmony with these advancements, Horak and Shumway-Cook[18-19] have taken a leadership role in developing assessment and treatment tools for physical therapists who are interested in becoming involved in a multidisciplinary approach to the treatment of patients with disorders of equilibrium.

The present study represents preliminary results of a 2-year program designed to assess the efficacy of VHT in the treatment of patients with a wide variety of vestibular disorders and symptoms. The most prominent findings to date are as follows:

1. Advanced age or lengthy duration of symptoms prior to therapy did not adversely influence the outcome of VHT.
2. As expected, patients with stable unilateral vestibular lesions had an extremely favorable response, including patients with initially poor compensation or decompensation after vestibular surgery.
3. There seems to be a group of patients with peripheral vestibular lesions who become worse during therapy. This suggests unstable peripheral vestibular function, offering a more definite indication for vestibular surgery.
4. Patients with mixed central and peripheral findings after head injury responded more poorly than the average patient.
5. Patients with objective test results suggesting poor compensation for peripheral vestibular lesions were less likely to obtain an outstanding recovery; however, their overall improvement rate was similar to patients with evidence for satisfactory compensation.
6. Patients who manifest established severe disability only rarely showed improvement. Because this cannot be attributed to the duration of symptoms, it may suggest that these patients have more severe underlying disease, or that psychologic or financial disincentives or both to recovery are preexisting.

Overall, despite the unrestricted nature of the patient selection process, 82% of our patients showed some improvement, with 59% improving dramatically. While some of this improvement may have occurred spontaneously, it is our impression that VHT contributed to

the satisfactory outcome in most of these cases. A prospectively randomized controlled study is underway to compare customized VHT with traditional therapy to better address this question.

Patients with chronic vestibular dysfunction, by virtue of their deep desire for relief from their symptoms, are vulnerable to risks from unproven medical and surgical treatment modalities. It is our impression that VHT represents a rational and conservative approach that is useful in the treatment of these patients. Large clinical studies detailing the indications and expected outcome of such therapy are lacking, and prospective studies are needed to establish the role of VHT in the future treatment of this challenging group of patients.

REFERENCES

1. Igarashi M. Vestibular compensation: an overview. Acta Otolaryngol (Stockh) 1984;406:78-82.
2. Llinas R, Walton K. Vestibular compensation: a distributed property of the central nervous system. In: Asanyma H, Wilson VT, eds. Integration in the nervous system. Tokyo: Igaku-Shoin, 1979:145-66.
3. McCabe BF, Ryu JH, Sekitani T. Further experiments on vestibular compensation. Laryngoscope 1972;82:381-96.
4. Schaeffer KP, Meyer DL. Compensation of vestibular lesions. In: Kornhuber, ed. Handbook of sensory physiology. Vol VI. Vestibular system. Berlin: Springer-Verlag, 1974:463-90.
5. Bienhold H, Abeln W, Flohr H. Drug effects on vestibular compensation. In: Flohr H, Precht W, eds. Lesion-induced neuronal plasticity in sensorimotor systems. New York: Springer-Verlag, 1981:265-73.
6. Zee DS. Perspectives on the pharmacology of vertigo. Arch Otolaryngol Head Neck Surg 1985;111:609-12.
7. Collins WE. Arousal and vestibular habituation. In: Kornhuber, ed. Handbook of sensory physiology. Vol. VI. Vestibular system. Berlin: Springer-Verlag, 1974:361-8.
8. Collins WE. Habituation of vestibular responses with and without visual stimulation. In: Kornhuber, ed. Handbook of sensory physiology. Vol. VI. Vestibular system. Berlin: Springer-Verlag, 1974:369-88.
9. Jones GM. Vestibular plasticity. OTOLARYNGOL HEAD NECK SURG 1983;91:72-5.
10. Baloh RW, Honrubia V. Clinical neurophysiology of the vestibular system. Philadelphia: FA Davis Co., 1979:64-6.
11. Norré ME. Treatment of unilateral vestibular hypofunction. In: Oosterveld WJ, ed. Otoneurology. New York: John Wiley & Sons Ltd., 1984:23-39.
12. Katsarkas A, Segal BN. Unilateral loss of peripheral vestibular function in patients: degree of compensation and factors causing decompensation. OTOLARYNGOL HEAD NECK SURG 1988;98: 45-7.
13. Cawthorne T. The physiological basis for head exercises. J Chart Soc Physiother 1944:106-7.
14. McCabe BF. Labyrinthine exercises in the treatment of disease characterized by vertigo: their physiologic basis and methodology. Laryngoscope 1970;80:1429-33.
15. Norré ME. Rationale of rehabilitation treatment for vertigo. Am J Otolaryngol 1987;8:31-5.
16. Nashner LM. Strategies for organization of human posture. In: Igarashi M, Black FO, eds. Vestibular and visual control in posture and locomotion equilibrium. Basel, Switzerland: Karger, 1985:1-8.
17. Nashner LM, Black FO, Wall C III. Adaptation to altered support and visual conditions during stance: patients with vestibular deficits. J Neurosci 1982;2:536-44.
18. Shumway-Cook A, Horak F. Assessing the influence of sensory interaction on balance. Phys Ther 1986;66:1548-50.
19. Horak F. Clinical measurement of postural control in adults. Phys Ther 1987;67:1881-5.