Vestibular Rehabilitation: Critical Decision Analysis

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Vestibular Diagnosis and Rehabilitation: Science and Clinical Applications

Richard E. Gans, Ph.D., FAAA

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INTRODUCTION

Vestibular Diagnosis and Rehabilitation: Science and Clinical Applications

Vestibular science, clinical evaluation, and treatment have advanced significantly in a short span of 50 years. Sir Isaac Newton, with regard to his own scientific contributions, once said, "If we can see far, it is because of those who have come before us; we are merely dwarfs riding on the shoulders of giants." This is certainly germane for those of us who continue to build on and advance the work of those who have contributed so much to our knowledge of this field.

This issue of *Seminars in Hearing* includes clinically oriented aspects of vestibular science. While there is an abundance of literature from the disciplines of audiology, otology, neurology, aviation, space medicine, physical therapy, and occupation therapy, the articles that have been included here have the most applicability for the rank and file clinician. Virtually all audiologists, physicians, and therapists have the ability to participate in some aspect of the continuum of care for the vestibular patient. Clinicians—even those that do not specialize in this field—should possess the ability to identify and triage the vestibular patient.

We begin this issue with an article by T. Oma Hester and Herbert Silverstein who examine the importance of good history taking from the otologist's point of view. Dr. Silverstein has been in the vanguard in the development of otologic surgical innovations, including the endo-lymphatic shunt, vestibular nerve section, laser assisted stapedectomy, and most recently, the Microwick intratympanic gentamicin treatment for intractable Meniere's disease.

Our next contributor, Anita Pikus, has had a 25-year career with the National Institutes of Health. Dr. Pikus' article on heritable vestibular conditions is particularly important for those clinicians working with a pediatric population. We often forget that hearing-impaired children also may have vestibular anomalies that occur within the impaired auditory vestibular system.

Michael Lombardo, a renowned biochemist whose career has focused on research in prostate and breast cancer, takes an interesting look at the biochemistry of benign paroxysmal positional vertigo. Dr. Lombardo presents a hypothesis that may explain the prevalence of the disorder in some individuals, while others benefit from spontaneous resolution.

A clinical article by Richard and Patricia Gans on Benign Paroxysmal Positional Vertigo (BPPV) examines the longitudinal treatment histories of BPPV patients over a seven-year period. The doctors Gans review treatment efficacy with modified Canalith Repositioning / Liberatory Maneuvers. Results from 376 patients suggest that both methods are highly effective in successfully treating this condition.

The importance of the vestibular ocular reflex, and its role and importance in human equilibrium is reviewed by Dennis O'Leary. Dr. O'Leary, a professor of otolaryngology at the University of Southern California, has spent much of his career in the development of tests of the Vestibular Ocular Reflex (VOR) during active head movement. It is the development and clinical implication of many of these simple and easily accessible technologies

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that often reveal otherwise undetected vestibular dysfunction in patients.

A review of Vestibular Rehabilitation Therapy, along with a critical decision analysis of the triage of vestibular patients in their continuum of care, is presented by Richard Gans. Dr. Gans' contribution will assist clinicians in identifying the appropriate management of patients with nonmedical or nonsurgically manageable chronic vestibular dysfunction.

The final contribution comes from Gary Jacobson. His prolific writing has provided us with a wealth of articles and edited textbooks that have become classic references for those pursuing this specialty. Dr. Jacobson's article, "Development of a Clinic for the Assessment of Risk of Falls in Elderly Patients," is timely. Falling among the elderly has been identified as a potentially major crisis for the healthcare system. Healthcare providers and insurers have, over the past decade, begun to seriously consider the long-term benefit of prophylactic intervention.

It is hoped that this issue of *Seminars* will provide its readers with a combination of clinically applicable tools, new ideas and theories to contemplate, as well as information that will assist those who serve the needs of the vestibular patient.

I wish to thank all of our contributing authors who have taken time from their hectic schedules to write for this *Seminars'* issue. I also wish to thank my support and clinical staff at The American Institute of Balance for their assistance and support in the preparation of my article. I would like to recognize the late David Cyr, Ph.D., who spent much of his career at Boys Town Neurological Institute researching and writing on pediatric vestibular issues.

Richard E. Gans, Ph.D., FAAA
Guest Editor[1]

Treatment Efficacy of Benign Paroxysmal Positional Vertigo (BPPV) with Canalith Repositioning Maneuver and Semont Liberatory Maneuver in 376 Patients

Richard E. Gans, Ph.D., FAAA,[1] and Patricia A. Harrington-Gans, Au.D., FAAA[1]

ABSTRACT

Canalith Repositioning and Semont Liberatory Maneuvers have been shown to be highly efficacious in the successful treatment of Benign Paroxysmal Positional Vertigo (BPPV). The differentiation of canalalithiasis, cupulolithiasis, and correct identification of canal involvement, particularly through the use of Video-oculography, have enhanced treatment decisions and outcomes.

Since 1994, approximately 700 BPPV patients have been treated at the authors' clinical facility. An anecdotal study of 376 of these patients followed over a 7-year period is presented. The patients in the historical study all presented with BPPV- PC and were treated with modified Canalith Repositioning Maneuver and Semont Liberatory Maneuver treatment procedures. The review indicated no significant differences in treatment outcomes between the two procedures. Seventy-nine percent of the patients required only one treatment, while 17% required two treatments, 3.5% required three treatments, and 0.05% required four treatments. The average number of treatments was 1.3. The SLM did show a reduced recurrence rate compared to the CRP method.

KEYWORDS: Benign Paroxysmal Positional Vertigo (BPPV), canalalithiasis, cupulolithiasis, otolith

Learning Outcomes: As a result of this activity, the reader will: (1) have a historical perspective of the development of the diagnosis and treatment of BPPV; (2) have a review of the literature of BPPV treatment methodologies; and (3) be able to determine and perform appropriate BPPV treatment methods.

Vestibular Diagnosis and Rehabilitation: Science and Clinical Applications; Editor in Chief, Catherine V. Palmer, Ph.D.; Guest Editor, Richard E. Gans, Ph.D., FAAA. *Seminars in Hearing*, volume 23, number 2, 2002. Address for correspondence and reprint requests: Richard E. Gans, Ph.D., The American Institute of Balance, 11290 Park Boulevard, Seminole, FL 33772. E-mail: rgans@dizzy.com. [1]The American Institute of Balance, Seminole, Florida.
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Benign paroxysmal positional vertigo (BPPV) is the most common form of vertigo[1] and is among the most easily treated without the need for surgery or medication.[2] The past 14 years has provided clinicians a wealth of research, retrospective studies, identification of BPPV variants and new treatment techniques to consider and practice. This article will present a review of, as well as the author's experience with, the treatment efficacy for posterior canal BPPV with both the Semont Liberatory and Canalith Repositioning Maneuvers in treating 376 patients over an 8-year period. A historical perspective will precede a comprehensive discussion of clinical protocols, and a review and discussion of the anecdotal findings.

DESCRIPTION AND PATHOPHYSIOLOGY

It has been 80 years since Barany[3] first described the abnormal condition eliciting rotatory-torsional nystagmus and vertigo with change in head position, a condition we now know as BPPV. It would be 30 years before Dix and Hallpike would name it Benign Paroxysmal Positional Vertigo (BPPV) in 1952.[4] Their work contributed to our understanding of the positioning maneuver to elicit the response and the cardinal manifestations. It did not explain its underlying pathophysiology. There was much controversy as to the cause of the condition until Schuknecht's classic cupulolithiasis explanation was published in 1969.[5] Prior to that time, many thought that the rotatory-torsional nystagmus and transient intense vertigo was too complicated to be attributable to anything but a central nervous system disorder.[6]

Schuknecht's histological and temporal bone studies demonstrated that the otoconia from the utricle migrated into the posterior semicircular canal (PSCC) and embedded onto the cupula. The weighted cupula, when moved from the vertical to the horizontal plane, which occurs when the head is tilted back, would cause the posterior canal to inadvertently become a gravity sensor. Although Schuknecht's work advanced the theory of the biomechanical aspects of BPPV, it did not completely explain all aspects of the condition. Ten years later, the term canalithiasis was coined by Parnes and McClure.[7] While attempting to blockade the ampullae of the PSCC, they observed the presence of free-floating otoconia within the long process of the PSCC. The biomechanics of this form of BPPV readily explains the fatigability aspect of the response, as had previously been postulated by Hall et al.[8] It is now accepted that while BPPV may manifest itself in a variety of variants and/or combinations, canalithiasis is the most prevalent form of BPPV.[9]

THERAPEUTIC TREATMENT METHODS

A physical therapeutic maneuver to specifically ameliorate BPPV was first proposed by Brandt and Daroff in 1980.[10] Their repetitive side-lying maneuver, although less than completely efficacious, did have some degree of success in reducing or eliminating the BPPV response in some patients. The self-induced vertigo, and uncomfortable and unnatural positions of the patient, limits its efficacy. Various techniques and successes in the treatment of BPPV have been put forth, beginning with the Liberatory maneuver by Semont, Freyss and Vitte[11] in 1988, and followed by the Canalith Repositioning by Epley.[12] The reported success in 1993 of Herdman et al[13] using the single treatment technique began a litany of reports of successful treatment options by more than a score of researchers. Table 1[11–24,27,29,39,44] provides a review of these studies.

Most clinicians who perform Repositioning and Liberatory Maneuvers have modified many of the protocols into a hybrid that has produced successful outcomes for them in their own clinics. It appears that regardless of what methodology is used, if the differential diagnosis of canalithiasis from cupulolithiasis is made, and appropriate canal involvement has been obtained, there is high treatment efficacy.[14–24]

Treatment of BPPV by either Liberatory or Canalith Repositioning Maneuver is widely recognized as the most efficacious, noninvasive and cost effective way to manage the condi-

Table 1 Studies of Posterior Canal Benign Paroxysmal Positional Vertigo Treatment Methods and Efficacy

Study	N	Year	Procedure	Results
Norre and Beckers[45]	51	1988	Other	Vestibular habituation training produces good results, when Benign Paroxysmal Positional Vertigo is diagnosed correctly.
Semont et al[11]	711	1988	Semont Liberatory	Semont Liberatory Maneuver is an effective form of treatment showing a low recurrence rate.
Epley[12]	30	1992	Canalith Repositioning Maneuver	Canalith Repositioning Maneuver, as described by Epley, is the most effective form of treatment.
Troost & Patton[46]	N/A	1992	Other	There is an excellent chance that exercise therapy will be curative with patients having Benign Paroxysmal Positional Vertigo.
Herdman et al[13]	60	1993	Semont Liberatory and Canalith Repositioning Maneuvers	Both the Semont and Epley maneuvers are effective treatments for Benign Paroxysmal Positional Vertigo.
Steenerson and Cronin[14]	40	1996	Canalith Repositioning	Canalith Repositioning Maneuver is as effective as vestibular habituation training and requires less patient time for treatment.
Coppo et al[15]	165	1996	Semont Liberatory Maneuver	Over 80% of patients were successfully treated after 1 to 3 sessions.
Herdman & Tusa[29]	85	1996	Canalith Repositioning Maneuver	Canalith Repositioning Maneuver can cause debris to move into the anterior or horizontal canal.
Lempert et al[16]	15	1997	Other	360° rotation of the posterior/semicircular canal proved to be an effective treatment.
Cohen and Jerabek[17]	87	1999	Both	Found that augmented head rotations are unnecessary and the modified Epley and Semont maneuvers are equally effective in the treatment of benign paroxysmal positional vertigo.
Gall et al[18]	16	1999	Semont Liberatory Maneuver	The findings showed a statistically significant change in subjective visual vertical post Hallpike and Semont maneuvers.

Table 1 (Continued)

Study	N	Year	Procedure	Results
Wolf et al[19]	107	1999	Canalith Repositioning Maneuver	The modified Epley maneuver is an excellent treatment for benign paroxysmal positional vertigo.
Blatt et al[47]	33	2000	Canalith Repositioning Maneuver	Treatment with Canalith Repositioning Maneuver appears to improve balance, but not in all patients.
Dornhoffer and Colvin[20]	52	2000	Canalith Repositioning Maneuver	Canalith Repositioning Maneuver was effective in 99% of patients. Any recurrence is thought to correlate to etiology.
Gross et al[48]	9	2000	Canalith Repositioning Maneuver	Meniere's disease may predispose patients to intractable benign paroxysmal positional vertigo.
Macias et al[21]	259	2000	Canalith Repositioning Maneuver	Patients with benign paroxysmal positional vertigo not located in a single posterior semicircular canal are more likely to need multiple Canalith Repositioning procedures.
Nuti et al[33]	56	2000	Semont Liberatory Maneuver	Restrictions following Semont Liberatory Maneuver are not necessary when treating benign paroxysmal positional vertigo.
Nunez et al[22]	168	2000	Canalith Repositioning Maneuver	Canalith Repositioning Maneuver is an effective treatment for benign paroxysmal positional vertigo with only 1 or 2 sessions; however, there is a 15% recurrence of benign paroxysmal positional vertigo symptoms per year after treatment.
O'Reilly et al[23]	72	2000	Canalith Repositioning Maneuver	Benign paroxysmal positioning vertigo is effective in relieving the vertigo associated with benign paroxysmal positional vertigo.
Tirelli et al[24]	118	2000	Canalith Repositioning Maneuver	Success rates were significantly higher with the Modified Repositioning Maneuver.
Sargent et al[30]	168	2001	Canalith Repositioning Maneuver and other	Mastoid oscillation does not significantly improve the efficacy of the Canalith Repositioning Maneuver.

tion.[2] The high incidence of BPPV among the elderly persons has been documented. An incidence rate as great as 50% in persons over the age of 70, and the associated morbidity with undiagnosed and untreated BPPV has been established.[25] Gans and Crandell[26] have reported significant improvement in the subjective report (SF-36)[27] of BPPV patients post-treatment in quality of life, general health, mental health, and vitality.

TECHNICAL ADVANCES

Recent advances in technology, specifically infrared video-oculography, have provided clinicians with the capability to record and review eye movements associated with BPPV. This has allowed better differentiation of the involved canal(s). Approximately 90 to 95% of BPPV will affect the PSCC and, to a lesser degree, the HSCC.[28] The predominant occurrence of HC-BPPV is usually secondary to the treatment of PC-BPPV with migration due to the repositioning itself. The occurrence of AC-BPPV rarely is seen or reported. Its existence may be questionable in view of the anatomical position of the anterior canal. When it does occur, it may appear as a cupulolithiasis variant.

The use of this new technology gives the clinician the opportunity of repeated viewing of the associated eye movement. The otherwise brief and transitory nature of the clinician's observation of the nystagmus in real time may be insufficient or inaccurate for a differential diagnosis. Figure 1 is a screen display of a computerized videonystagmography (VNG) system. A differential diagnostic clinical pathway is presented in Figure 2 for canalalithiasis vs. cupulolithiasis.

TREATMENT OF CANALITHIASIS

Canalithiasis may be treated using either the Canalith Repositioning Maneuver (Appendix A), or the Semont Liberatory Maneuver (Appendix B). The decision of which method to follow becomes a matter of comfort or choice for the clinician. The patient's physical characteristics or capabilities need to be taken into consideration. The Liberatory Maneuver requires the movement of the patient en masse and may be inappropriate for those patients who have undergone a hip replacement within 90 days of the treatment. The Canalith Repositioning Maneuver tends to be more comfortable for patients, as it only requires them to move their head and roll onto their side. Physical limitations, whether they are orthopedic, neuromuscular, or due to obesity, may make one technique easier and more comfortable for both patient and clinician than the other. The success rate of either procedure has been reported to be greater than 90% by most investigators.

TREATMENT OF CUPULOLITHIASIS

According to Schuknechts'[10] theory of cupulolithiasis, the debris adhers to the cupula rather than free floating in the long process of the posterior canal. This theory, according to the Semont's Liberatory Maneuver, recognizes that the debris cannot merely be repositioned by rotation of the head but some basic principals of physics must be used to dislodge the debris from the cupula so that it can then be released and allowed to return to the utricle and dissolve. The differentiating factor and the diagnosis are based on whether the symptoms will abate with repetition of a provoking maneuver. For patients with acute symptoms, including nausea and emesis, repetition of three and four modified Hallpike Maneuvers as part of the differential diagnostic process may be difficult.

USE OF VIBRATION

The original work of Epley,[12] as well as Li,[29] recommends the use of vibration to maximize treatment outcome. However, a review of the literature suggests that most researchers and clinicians have not found the vibrator to be a critical component in the treatment of BPPV.[13–24] There are other options that range from bone conduction oscillators, tuning forks,

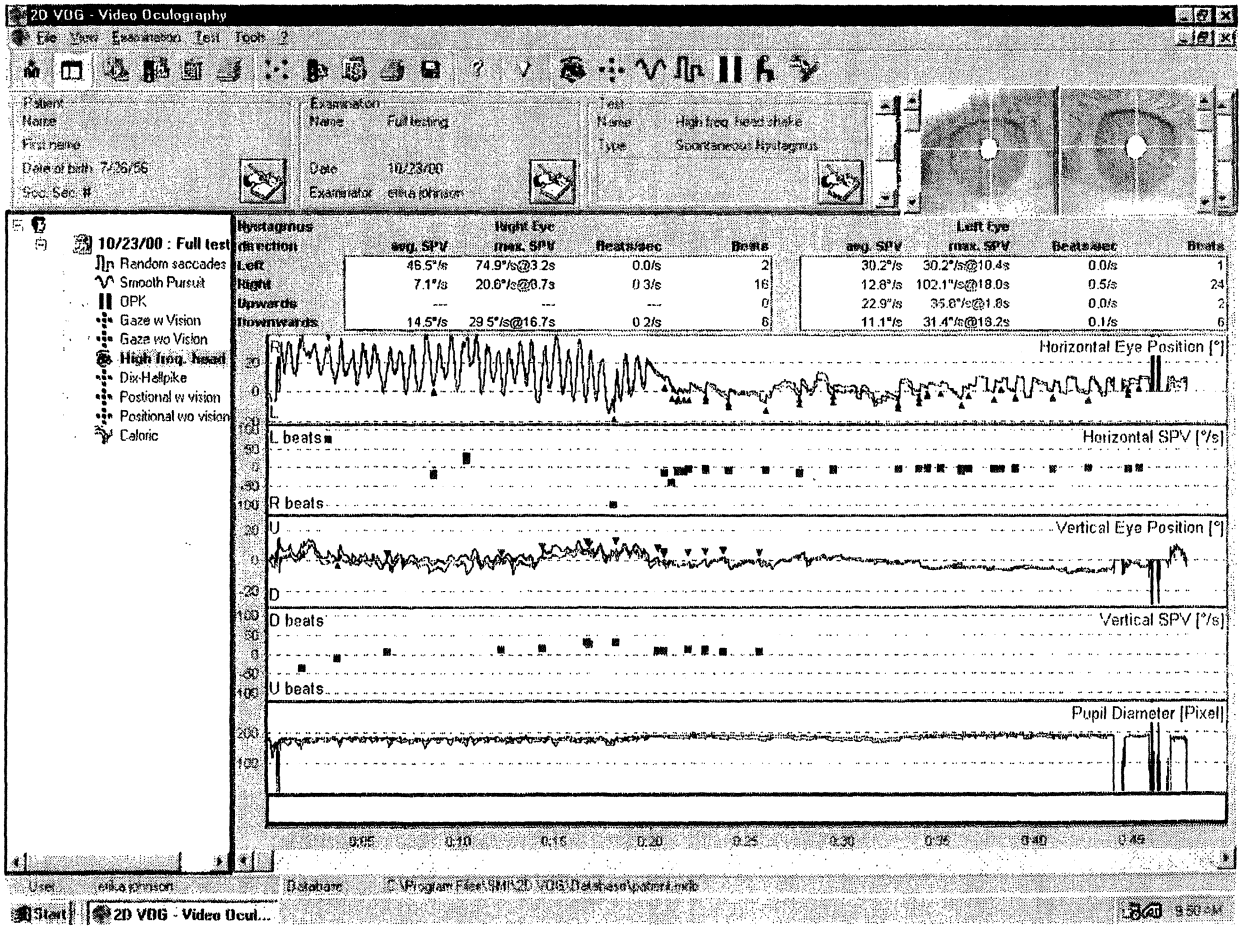

Figure 1 Videoculography technology allows visualization and video recording of eye movement in addition to the computer graphics.

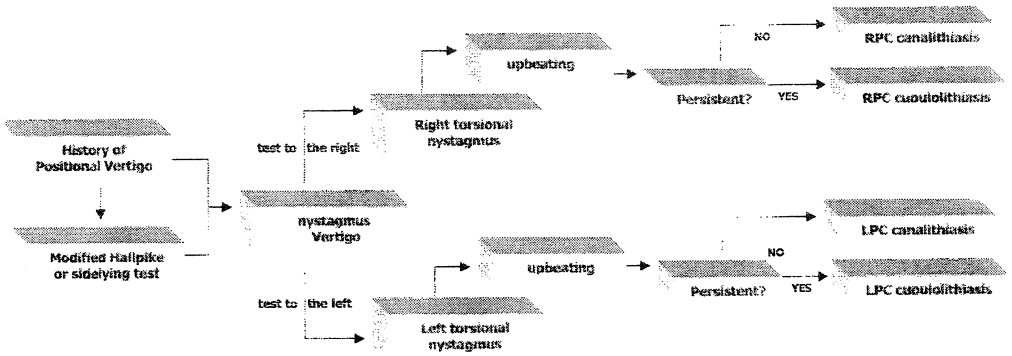

Figure 2 A clinical pathway for the differential diagnosis of ear(s) involved and the differentiation of canalithiasis and cupulolithiasis for PC-BPPV.

or tapping of the mastoid process during the treatment procedure to facilitate movement of the debris. Our experience indicates that in patients requiring more than the average number of treatments, tapping the mastoid process has been somewhat successful in clearing the debris. A horizontal canal migration during, or subsequent to a Canalith Repositioning Maneuver, however, has occurred in greater numbers in those patients who have received the tapping.[31]

POSTTREATMENT RECOMMENDATIONS

There are a wide variety of reported activity limitations for the patient from 24 to 48 hours following treatment. Many reports have asked that the patient not lie supine or tip their head for 48 hours following treatment. This is often assisted by wearing a soft cervical collar. Then, for several nights or up to a week following treatment, the patient is asked to refrain from lying on the treated or affected side while sleeping.

A modified prohibition of having the patient avoid tipping the head or lying supine for over 24 hours has proven to be sufficient in our clinic (Appendix C). This is supported by the work of Zucca,[32] who reported that otolith debris in the vestibular system of frogs would dissolve within the calcium-poor endolymph well within the 24-hour period. Recent reports have suggested that it may not be necessary for any prohibition of head movement or activity.[33]

COMPLICATIONS OF TREATMENT

Clinicians participating in the treatment and management of BPPV patients should recognize that several complications, although not common, could occur and when they do, they must be dealt with quickly and efficiently.

HORIZONTAL CANAL MIGRATION

Migration of the otolith debris into the horizontal canal has been reported in the literature.[34–41] For audiologists performing ENG without the benefit of video-oculographic recordings, this phenomenon may be the explanation for a direction changing nystagmus seen in positional ENG sub tests.

The debris may migrate into the horizontal canal following Canalith Repositioning or during movements through the positioning. The patient will present with a burst of linear horizontal nystagmus with its fast phase beating geotropically towards the undermost involved horizontal canal. If the patient's head is turned onto the opposite or unaffected ear, there is a linear horizontal ageotropic nystagmus beating away from the normal undermost ear towards the affected ear. Patients may then

be treated with either the Appiani Liberatory, or a 360° barbeque roll maneuver. Appiani et al[34] described a simplified method of treatment for the horizontal canal BPPV migration. The treatment requires a side lying liberatory-lateral type movement followed by a downward head turn while the patient is in the lateral body position. This method is more comfortable and easily performed by heavier or older patients.

The barbeque roll technique requires the patient to roll in 90° quadrants until the debris is cleared. Both methods of treating the horizontal canal begin with the initial treatment position away from the affected ear. This differs from Canalith Repositioning and Semont Liberatory Maneuvers that initiate towards the affected ear.

CANALITH JAM

A rare but often frightening occurrence is a canalith jam.[44] This occurs when the otolith debris is unable to clear the common crus as the debris falls downward from the posterior canal into the utricle when the patient returns to a seated position following the final stage of the Canalith Repositioning Maneuver or Semont Liberatory Maneuver. The patient will become symptomatic and the response will not be transient and it will not fatigue the patient. The sensation of falling or rapidly tumbling in a Tumarkin's-like crisis may be severe. The method used to clear the jam is to reverse the Repositioning protocol in the order in which it was performed.

METHODS AND PATIENTS

A retrospective case review of 376 patients treated for PC-BPPV at the American Institute of Balance from May of 1994 to July of 2001 was conducted. To date, nearly 700 patients have been treated. The selection of the patients for this study was based on completeness of records and follow-up contact. All patients were treated by a single clinician. All patients had been medically referred for evaluation and treatment of vertigo. Comprehensive cochleovestibular testing included ENG/VNG with air calorics. The diagnosis of PC-BPPV was made with the classic criteria of: (1) transient rotatory–torsional nystagmus toward the undermost ear, (2) subjective vertigo that parallels the nystagmus, (3) latency of onset of nystagmus, and (4) a possible reversal of nystagmus upon return to sitting position. Patients that were evaluated from 1994 to 1997 were confirmed by direct visual observation. Patients that were evaluated between 1998 and 2001 were video recorded with video-oculography (Micro Medical-monocular, SMI monocular video-goggles or SMI binocular 2D VOG). In instances where monocular recording was performed, the infrared camera was placed to record the ipsilateral eye of the undermost test ear. Figure 2 presents a clinical pathway for differential diagnosis of the ear(s) involved, and differentiation of canalithiasis vs. cupulolithiasis. Preceding all positioning maneuvers, a vertebral artery-screening test, as previously recommended by this author,[30] was performed. Positive indicators on a Vertebral Artery Screening test include: (1) diplopia, (2) dysarthria, (3) dizziness or near syncope, (4) nausea, and (5) in rare cases, nystagmus. A positive indicator on the Vertebral Artery Screening Test is reported to the referring physician, and the patient is not positioned with the neck hyperextended.

Modified Dix-Hallpike maneuvers in three variations were used to elicit the PC-BPPV response. The American Institute of Balance protocol excludes the traditional-classic Dix-Hallpike method due to the potentially negative biomechanical impact on both clinician and patient. The positioning techniques are: (1) modified Dix-Hallpike with hyperextension (head hanging with neck fully supported), (2) supine (without head hanging, fully supported) minimal hyperextension and head rotation, and (3) side-lying with minimal hyperextension and rotation. The positioning-test maneuver selection was based on patient comfort and on each patient's individual physiognomy or physical condition. A patient with a history of a fused vertebra, for example, is a poor candidate for any type of head hanging maneuver. Likewise, a patient with a recent hip

replacement (less than 90 days) is a poor side-lying candidate.

Patients identified as having HC-BPPV or AC-BPPV upon initial diagnosis or secondary to migration post initial treatment were excluded from the anecdotal review. All patients who were deemed candidates for treatment had medical clearance and/or a subsequent referral for treatment. Prior to treatment, patients received written materials describing their condition, reprints and articles detailing the treatment procedure, post treatment restrictions, possible complications, and the Institute's treatment outcomes. Treatment was never performed on the same day as the diagnostic evaluation.

TREATMENT METHODS

Patients seen from 1994 to 1998 (N=272) were treated only with the Semont Liberatory Maneuver (SLM). Patients from 1999 to 2001 (N=195) were treated with the Canalith Repositioning Maneuver (CRM) as the first treatment of choice. The CRM patients who did not clear/fatigue on the initial treatment maneuver or those who were still positive for PC-BPPV on their first follow-up visit were treated with SLM. Descriptions of both treatment methods are presented in Appendices A and B for modified Canalith Repositioning Maneuver and Semont Liberatory Maneuver, respectively.

POST TREATMENT RESTRICTIONS AND FOLLOW-UP

Patients were provided with written instructions, as presented in Appendix C, restricting activity. Patients also were advised to return in approximately one week for a post treatment follow-up visit. Patients educated in advance of the importance of the follow-up visit(s) as part of their treatment were highly compliant. Their follow-up appointment was made at the time of their initial treatment and all visits were confirmed by telephone prior to their appointment day. A subjective report of treatment benefit by telephone inquiry has not shown to be a reliable predictor of treatment efficacy. Patients often are reluctant to aggressively test themselves. While reporting that they are feeling better and have resumed normal activities, they may consciously or unconsciously avoid the most provocative head positions. Concomitant vestibular symptomtology (i.e., noncompensated high frequency vestibulopathy) or unrelated symptoms (i.e., lightheadedness) secondary to other medical conditions, may contaminate the patient's subjective report of treatment benefit.

Confirmation of extinction of the rotatory-torsional nystagmus and vertigo was obtained through the repetition of the diagnostic positioning maneuvers at the time of the patients' first follow-up visit. The patients were placed in at least two provocative positions. The first position was an appropriately modified Dix-Hallpike position, and the second was the side-lying position. Patients also were roll tested to ensure there had been no migration into the horizontal canal. A patient was considered cleared if no recordable nystagmus was noted through direct observation or videooculography recording, and there was no subjective report of vertigo by the patient. If there was observable or recordable nystagmus and a subjective report of vertigo, even though significantly ameliorated, the patient was considered to remain positive for PC-BPPV, and retreated.

RESULTS

The 376 patients (Table 2) in the anecdotal review were comprised of 110 males (mean age 71 years), and 266 females (mean age 69 years). The patients received a total of 480 treatments. Of these, 195 received CRM, 272

Table 2 Summary of 376 Patients by Gender, Age, and Involved Ear(s)

	Age Range	Mean Age	Affected Ear		
			Right	Left	Both
Males	32–91	71	63	42	5
Females	28–91	69	148	106	12

Table 3 Summary of 376 Patients' Outcomes by Treatment Method and Gender

	Treatment			% Improved		
	Canalith Repositioning	Semont Liberatory	Both	Canalith Repositioning	Semont Liberatory	Both
Males	56	84	2	97.9	92.8	75
Females	139	188	11	98.9	96.8	95.9

received SLM, and 13 patients received both treatments (Table 3). The right ear was affected in 211 patients and the left ear in 148 patients. Both ears were affected in only 17 patients. There was a 56% predominance of a right ear involvement. The right ear predominance was not gender specific, with right ear involvement occurring in 57% of males, and in 56% of females.

As can be seen in Table 3, the treatment efficacy was comparable for males and females receiving both CRM and SLM treatments. The CRM treatment efficacy was approximately 98% in males, and 99% in females. SLM treatment efficacy was approximately 93% for males, and approximately 97% in females. For patients receiving both methods of treatment, there was a difference in the treatment efficacy, with females showing a positive outcome of approximately 96% versus males with 75%.

A consideration in the selection of treatment methods is the success rate with a single treatment. As indicated in Figure 6, 79% of the patients had a complete resolution following only one treatment (Table 4). Both males and females had a success rate of 98% with a single treatment using Canalith Repositioning maneuver. The Semont Liberatory Maneuver produced success in a single treatment in 91% of the males, and in approximately 97% of the females. Seventeen percent of the patients required two treatments, while 3.7% required three treatments. Only one-half of one percent (0.5%) of the 376 patients required four treatments.

Another consideration of treatment method selection and follow-up is the recurrence rate. The data presented in Table 5 illustrate a recurrence rate for the Canalith Repositioning Maneuver of approximately 7% for both males and females. The Semont Liberatory Maneuver presented with a recurrence rate of only 3.6% and 1.5% for males and females, respectively. A recurrence was considered to have occurred 30 days following the initial treatment period and post follow-up period.

DISCUSSION

A review of the literature over the past 14 years has shown the treatment efficacy of both Canalith Repositioning and Semont Liberatory Maneuvers. The patients in our anecdotal review with PC-BPPV demonstrated successful outcomes using both treatment methods. Seventy-nine percent of the patients were successfully cleared of symptoms with a single treatment and 17% required two treatments. These data compare favorably with Epley's finding that 80% of his 30 patients were successfully cleared with one treatment. Epley's data further suggested that 98% were symptom-free by the end of 3 months. Our data indicated that 96%

Table 4 Comparison of Single Treatment Efficacy by Method and Gender

	Canalith Repositioning	Semont Liberatory
Males	98.06	91.47
Females	98.41	96.78

Table 5 Comparison of Recurrence Rate by Treatment Method and Gender

	Recurrence Rate (%)		
	Canalith Repositioning	Semont Liberatory	Both
Males	7.2	3.6	0.9
Females	6.8	1.5	0

of the patients were successfully cleared following just two treatments. Semont et al reported similar findings with 84% of his patients requiring one treatment and 93% successfully cleared with two treatments.

Semont's recurrence rate over an 8-year period was approximately 4%. Our recurrence rates ranged between 2 to 4% using a Modified Semont Liberatory Maneuver, and 7% for the Canalith Repositioning Maneuver. To our knowledge, there has not been a longitudinal study that has reported a recurrence rate over an 8-year period with Canalith Repositioning Maneuver. Generally, there was no correlation between gender and treatment efficacy with any of the methods. The patient's mobility and compliance to the post-treatment restrictions are probably the two most important predictors of the patient's success. We did not differentiate the underlying pathophysiology or etiology of the BPPV. For example, would a patient with BPPV secondary to vestibular neuritis be less likely clear from a single treatment versus an idiopathic occurrence of BPPV? The high (79%) success rate of a single treatment, and a 96% success rate with just two treatments, suggests that it is unlikely that underlying etiology would provide information to affect treatment of choice.

CONCLUSION

1. CRM and SLM are equally efficacious methods of treating PC-BPPV.
2. Ninety-six percent of patients will be successfully treated with two treatments. Seventy-nine percent require only a single treatment.
4. The recurrence rate of BPPV is relatively small (approximately 7%) using CRP, and as low as 2 to 4% using SLM.

Both CRM and SLM are highly efficacious treatment methods for PC-BPPV. Undiagnosed and untreated BPPV, particularly in the older population, is a significant and often catastrophic health hazard. The cost in dollars and human suffering in allowing patients to go untreated have been well documented. With 96% of patients requiring two or less treatments, CRM and SLM are highly cost-effective methods in the treatment of this often-debilitating condition. Third-party payers, primary care physicians, and patients themselves should be aware of this highly efficacious and cost-effective treatment for the leading cause of dizziness and vertigo.

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ABBREVIATIONS

AC-BPPV	anterior canal benign paroxysmal positional vertigo
ASCC	anterior semicircular canal
BPPV	benign paroxysmal positional vertigo
CRM	Canalith Repositioning Maneuver
HC-BPPV	horizontal canal benign paroxysmal positional vertigo
HSCC	horizontal semicircular canal
PC-BPPV	posterior canal benign paroxysmal positional vertigo
PSCC	posterior semicircular canal
SLM	Semont Liberatory Maneuver
VNG	videonystagmography

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APPENDIX A. DESCRIPTION OF MODIFIED CANALITH REPOSITIONING MANEUVER (CRM)

Step 1. The patient is seated on the long axis of the examination table (we recommend placing a pillow under the legs to avoid cramping the hamstrings). The clinician stands at the head of the table to support the patient's neck and back. The patient turns the head *toward* the affected ear as he or she lies backwards. The patient is then placed in the hyperextended head hanging position while the clinician, sitting on a properly height-adjusted stool, provides full support for the patient's neck. Following the anticipated latency, there should be an onset of nystagmus and subjective vertigo. There must be a provocation in this initial position. The patient remains in this position for 3 minutes.

Step 2. The patient is then asked and cued to rotate the head to the opposite side while maintaining a hyper-extension with the clinician supporting the neck at all times. Regardless of whether there is a provocation, the patient maintains this position for 3 minutes.

Step 3. The patient now must roll onto his or her side (nonaffected ear side). This will place the patient face down. As in the hyperextended, somewhat head-hanging position in Step 1, the head should be sufficiently off the table so the chin is extended beyond the edge of the table. The physics of this position is such that the head at this point no longer needs to be supported by the clinician. The position may cause a strong provocation, so it is imperative to provide sufficient kinesthetic feedback to the patient. The patient maintains this position for 3 minutes.

Step 4. The patient now swings the legs off the table and transfers to a fully seated, erect position by using elbow and hands to push themselves up. The head remains turned and is brought frontward only when the patient reachs the seated position. The head is then cued for a slight downward tilt and then returned to the normal or neutral position. The clinician must be prepared for the possibility that the patient may experience a Tumarkin's crisis. This is an intense sensation of falling or being pulled to the ground. The patient is placed in a soft cervical collar as a reminder to not move or pitch the head in the vertical plane.

APPENDIX B. DESCRIPTION OF MODIFIED SEMONT LIBERATORY MANEUVER TREATMENT

Step 1. The patient is seated on the side of the examination table. The head is turned *away* from the affected ear and the side to be laid on.

Step 2. The patient is briskly laid on the side as they swing the legs up onto the table (similar movement as one does getting into bed). As the head has maintained the same position it is now resting on the table with the nose pointing upward at approximately a 45-degree angle. The patient maintains this position for five minutes following the cessation of nystagmus.

Note: The patient must be provoked (onset of nystagmus and vertigo) in the initial position. The nystagmus should appear following a brief latency. A few rare patients will show latencies as long as 50 seconds. The nystagmus and accompanying vertigo will last for 5 to 20 seconds.

Step 3. The patient is then moved to the opposite side through a transfer method whereby the legs hang from the table. The patient pushes up using elbow and hands, and is laid in the reverse direction on the opposite side. Because the patient has not moved the position of the head, the nose now faces down into the table. After waiting 10 seconds or so for a spontaneous burst of nystagmus and vertigo. If it does not occur, the patient is cued to move their head side to side. The head movement is initiated, whether there is nystagmus or not. The patient maintains this position for 5 minutes.

Step 4. The patient now swings the legs off the table and uses the elbows and hands to transfer to the seated-erect beginning position. At this point, it is critical to maintain a physical hold of the patient for at least 20 seconds as, on occasion, patients may have a Tumarkins-like crisis. There is no way to predict in advance which patients may experience this intense sense of falling, dropping, or being pulled to the earth. The patient is then placed in a soft cervical collar for the sole purpose of serving as a friendly reminder to not move or pitch the head in the vertical plane.

APPENDIX C. POST TREATMENT PATIENT INSTRUCTIONS

Reminder on Day of Treatment

1. Keep your head upright. Do not pitch your head up or down. Try to keep your head vertical, as if trying to balance a book on it.
2. Side-to-side turns are okay.

Sleeping for Tonight Only, and Next Day

1. Do not lay flat. Try to sleep propped-up, about 30 degrees.
2. You may remove the cervical collar 24 hours post treatment.

Sleeping for the Next Three Days

1. Avoid sleeping on the side of the treated ear.
2. Please call should you have any questions or concerns.
3. It is not unusual for you to feel a sensation of floaty-headedness, and you may be slightly off-balance for several days following treatment.

Patients are provided with a soft cervical collar.

Vestibular Rehabilitation: Critical Decision Analysis

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ABSTRACT

Vestibular Rehabilitation Therapy (VRT) is used by audiologists, physical and occupational therapists, and physicians as an efficacious treatment and management strategy for patients with noncompensated vestibular dysfunction secondary to a vestibular disease or disorder. Although vestibular rehabilitation techniques were first reported over a half-century ago, it has been in the past decade that these techniques have become a widely accepted nonmedical or nonsurgical treatment strategy for patients with chronic and often debilitating symptoms. The use of diagnosis-based strategies or individualized and programmatic therapy protocols and programs has been documented as providing the best treatment outcomes. Critical decision analysis and clinical pathways provide a straightforward method for clinicians in the identification and treatment of this population.

KEYWORDS: Vestibular, vestibulopathy, noncompensated, stabilized, unilateral vestibular dysfunction, vestibular rehabilitation

Learning Outcomes: Upon completion of this article, the reader will be able to (1) state the primary causes and disease processes of vestibular dysfunction, (2) describe the physiological characteristics of noncompensated vestibulopathy, and (3) list the physiological theories for vestibular rehabilitation and its appropriate application.

According to the National Institutes of Health (NIH),[1] patients' complaints of dizziness and vertigo will account for nearly 7 million doctor visits. Vestibular disorders will account for 85% of these symptoms.[2] The vestibular system is subject to insult, trauma, and disease (Table 1). The disorder or disease process may cause the vestibular system to suffer a reduction or loss of function in one or both systems. The damage or change may occur either as a loss of sensory receptors within the end organ, or within the nerve itself. The sudden loss or reduction of function unilaterally will typically produce debilitating vertigo with associated autonomic and parasympathetic nervous system responses of nausea, emesis,

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Table 1 Common Causes of Unilateral Vestibular Dysfunction

Autoimmune disorders
Labyrinthitis
Labyrinthine concussion
Labyrinthine ischemia
Meniere's disease
Ramsey Hunt syndrome
Vestibular migraine
Vestibular neuritis

Table 2 Common Causes of Bilateral Vestibular Dysfunction

Aminoglysides
Arteriosclerosis
Diabetes
Microvascular disease

and diaphoresis. This is similar to what occurs in acute motion sickness. Fortunately, the acute phase of most conditions will pass within hours. Other conditions, however, may linger for days, weeks, or—as in the case of Meniere's—be problematic for years. During the acute phase of the condition, the patient will require medical and pharmacological management. Patients usually wish to stay quiet and immobilized until they recover their homeostasis and manage to move about.

STATUS OF THE VESTIBULAR DYSFUNCTION

Patients with vestibular disorders may have had insult or damage to either the peripheral (labyrinth part of the inner ear) or central (brainstem or cerebellum) portions of the vestibular mechanism. Common inner ear disorders that cause vestibular dysfunction include labyrinthitis, vestibular neuritis, herpes zoster oticus (shingles), vestibular migraine, labyrinthine ischemia, and Meniere's disease. Many of these patients may have had only a relatively short phase of acute vertigo, or have post-surgery treatment of intractable inner ear disease such as Meniere's disease. Once out of the acute phase, they may be left with chronic symptoms affecting their sense of spatial orientation, gaze stabilization, or balance.

Vestibular disorders affect two output modalities—the vestibulo-ocular reflex (VOR) that controls eye movement and gaze stabilization during active head movement, and the vestibulo-spinal reflex (VSR) that influences postural stability, translated through the musculoskeletal system and antigravity muscles. Patients with vestibular disorders may present with defects in one or both symptoms of gaze stabilization problems or unsteadiness, usually when challenged by uneven surfaces, quick turns or with reduced vision.

For those with slow, insidious vestibular changes that culminate over years, patients will not experience vertigo, but rather a loss of equilibrium and increased unsteadiness with ambulation. This often occurs in the older adult or those with a variety of nonvestibular or nonotologic related disease processes (Table 2). Individuals who have had bilateral vestibular losses secondary to aminoglyside toxicity usually present with an associated complaint of oscillopsia during head movement.

VRT is a highly effective management strategy for those patients who have chronic symptoms related to a labyrinthine event.[3] In order to provide the most efficacious treatment possible, the clinician will benefit by following a critical decision analysis. The first step is to understand the status of the disorder that has caused the patient's condition.

Prior to the referral for, and undertaking of VRT, it will be necessary to properly define and categorize the status of the vestibular involvement. The ideal candidate for VRT will be a patient with stabilized but noncompensated unilateral vestibular dysfunction (UVD).

STABILIZED VS. NONSTABILIZED

A stabilized condition can best be described as one that is no longer producing attacks or episodes of debilitating vertigo and other otologic or parasympathetic responses, such as acute nausea, emesis, etc. This has been described as the labyrinthine storm. Most conditions, such as vestibular neuritis or labyrinthitis, become

stabilized once the acute phase has passed. The anatomical and physiological damage to the system is typically caused either by a viral or bacterial infection of the labyrinthine fluids, or a viral/bacterial inflammation of the vestibular portion of CN VIII. The patient may have symptoms related to the chronic VOR dysfunction, but no longer is subject to attacks.

Meniere's disease is perhaps the best example of a chronic nonstabilized vestibular disease that may persist in an active or agitated state for years. Another example of a nonstabilized disease would be a nonoperable acoustic neuroma, or vestibular schwannoma. Because BPPV is caused by a biomechanical phenomenon rather than a neurophysiological state, it does not fit into the categorization of a stabilized vs. nonstabilized type lesion.

COMPENSATED VS. NONCOMPENSATED

The central nervous system (CNS) will, within days, weeks, or months, see the asymmetrical labyrinthine function and, without any external help, will reset or retune the VOR function. Central compensation is believed to occur through the plasticity of the CNS within the brainstem and cerebellum. It has been described as a neurophysiological motor re-learning phenomenon and has been documented in the literature with animal and human models.[4,5]

Patients who have a spontaneous recovery of the VOR function subsequent to an acute vestibulopathy are considered to have a compensated lesion. Although these patients will continue to present with abnormal findings (i.e., reduced labyrinthine reactivity or caloric weakness on a VNG/ENG caloric), they will have no subjective report or hallucination, or exaggerated sense of motion, oscillopsia, or visually provoked symptoms. On tests of VOR function such as Vestibular Autorotation Testing, patients will present with normal gain and phase, just as if they were otherwise normal. Tests of dynamic visual acuity will appear normal, despite the caloric weakness. The caveat to clinicians is that just because there is a reduced labyrinthine reactivity, it does not mean that it is the origin of the patient's complaints. Likewise, patients may have normal caloric responses either because the horizontal canal was not involved, or the problem is in the higher frequency sensitivity of the system. The caloric test evaluates only the ultra low frequency sensitivity of the horizontal semicircular canal. (See the article in this issue by O'Leary for more information on the limitations of caloric testing.)

IDENTIFYING VRT CANDIDATES

Patients who find their everyday function is adversely affected or limited by this stabilized, but noncompensated asymmetry in vestibular function, may benefit from VRT. The vestibulopathy may affect the full range of acceleration, or frequency, or just regions of acceleration, similar to a frequency-specific hearing loss within the cochlea. Likewise, the direction of the acceleration also may be involved. Typically, the patient's symptoms will be more provoked with acceleration toward the involved or impaired labyrinth. The manifestation of a vestibulopathy will often result in a VOR deficiency. The VOR is responsible for stabilizing eye/head position at frequencies starting at about 1.5 Hz. The errors that occur in the VOR function may affect the gain or accuracy and the phase or timing of the reflex. Correct VOR function is dependent on the brain's ability to correctly signal the extraocular eye muscles to correspond their response with the initiating head movement. The hydromechanical movement of the fluid within the semicircular canals initiates this signal.

The inability of the eyes to be correctly positioned with active head movement causes a retinal slippage that results in an oscillopsia. The image to be viewed appears to jump or jiggle. It may be restricted to the plane of the involvement. The extraocular eye muscles are correlated with the specific plane of the balance canals.

VRT consists of systematic repetitive exercises and protocols that extinguish, or ameliorate patients' motion-provoked symptoms, as well as enhance postural stability and equilibrium. VRT is not new; it has reached its half-century mark. In the 1940s, Cawthorne[6]

and Cooksey[7] discussed the benefit of active eye and head movement exercise for patients who experienced labyrinthine problems. Since then, research and clinical experience has greatly advanced the scientific application of this treatment methodology.[8–12]

CLINICAL CORRELATES

Patient evaluation may include a variety of assessment tools that are specific to revealing VOR abnormalities. This may include tests of gaze stabilization and visual acuity with active head movement. Tests of VSR function, such as Computerized Dynamic Posturography (CDP),[13] or simple modified Clinical Test of Sensory Integration of Balance (CTSIB)[14] may reveal UVD patterns. Traditionally performed tests, such as VNG/ENG calorics, or less commonly performed rotary chair, may not reveal a high frequency UVD. Patients who experience VOR problems at a higher frequency can often be identified with tests that disrupt visual acuity during active head movement, or that quantify the VOR gain and phase.[15,16]

Recent investigation and reports of gaze stabilization tests[17] hold significant promise in simple and straightforward analysis of VOR function beyond what is currently available. All too often, patients with undetected UVDs whose history and symptoms strongly correlate with a stabilized, but noncompensated UVDs are dismissed as being a nonvestibular patient as a result of an unremarkable caloric or VNG/ENG study.

SUBJECTIVE HANDICAP SCALES AND PATIENT REPORT

Patient rating scales have been used to identify and quantify the functional disability created by physical ailment or illness. Disease or activity-specific and global health status patient handicap scales provide a valuable resource for establishing baseline, as well as serial or outcome measures. Table 3[17–21] provides several scales used and clinically documented in the literature.[17–23] These may provide excellent insight for clinicians in determining candidates for VRT or for those who may require further testing.

Table 3 Subjective Handicap Instruments

Dizziness Handicap Inventory[17]
Health Survey Questionnaire—SF-36[18]
Meniere's Disease—Patient Oriented Subjective Improvement[19]
Vestibular Disorder Activities of Daily Living Scale[20]
Activities-Specific Balance Confidence Scale[21]

PHYSIOLOGICAL BASIS OF VRT

The underlying physiological basis for VRT is the plasticity of the central nervous system. VRT does not actually involve a regeneration or treatment of the damaged vestibular end organ itself. Instead, it works by allowing the central nervous system and the brain to acclimate or adapt to asymmetrical/conflicting input from the two vestibular systems. Possible mechanisms include the spontaneous rebalancing of the tonic activity within the vestibular nuclei, recovery of the VOR through adaptation, and the habituation effect that is a lessening of response to the same stimuli over time. Theoretically, central compensation should occur within 90 days following dysfunction or loss of one of the vestibular systems. Many lesions, particularly those that occur with rapid onset, do not benefit from this compensation phenomenon.

Complicating or delaying the phenomenon of central compensation is the reluctance of the patient to perform any of those activities involving active head motion that produce symptoms of dizziness. This may be a primary factor as to why central compensation does not occur in many individuals. Other complicating factors include commonly prescribed drugs such as meclizine, Antivert, Valium, and other pharmaceuticals that suppress either peripheral vestibular or CNS function. These drugs will delay or prevent the central nervous system from relearning or adapting to asymmetrical sensory input. Unfortunately, the dizzy patient,

in his/her heightened state of anxiety about becoming dizzy (especially while at work or driving), becomes reliant on those pharmaceuticals that assist in suppressing their symptoms.

VRT works best when it is used with individuals who are outside of the acute phase of a condition. The patient who is in the midst of a labyrinthine storm secondary to labyrinthitis, vestibular neuritis, or active Meniere's disease will receive little or no benefit from VRT. Most patients, however, will be in a stabilized condition.

These patients present symptoms that are provoked with active head movement, often at a particular frequency of motion and in a particular direction. For example, there may be an inability to ride down a particular street where there appears to be numerous telephone poles when the patient looks out of the side window. It is common for patients to express a sensation of motion sickness while they are looking at certain patterns of floor tiles or wall coverings. One of the most common patient complaints is difficulty walking down an aisle of a grocery store while turning their head from side-to-side and up-and-down while shopping.

DIAGNOSIS-BASED STRATEGIES

Gans,[23] Shepard,[24] Black,[25] and Cohen,[26] as well as others have supported and promoted the use of specific rehabilitation strategies correlating with specific underlying categorization of functional disability. Many well-meaning practitioners continue to use the 50-year-old Cawthorne and 30-year-old Brandt exercises for dizzy patients, regardless of the patient's diagnosis or condition. Clinical experience and the literature strongly indicate that the success of vestibular rehabilitation is related to applying the correct treatment methodology to the appropriate corresponding dysfunction. Table 4 identifies those appropriate treatment methodologies with their corresponding functional components.

Diagnosis Based Strategies as an individualized or customized therapeutic approach have been shown to produce successful outcomes.[27] These strategies link the underlying physiological changes that occurred due to the disease or insult with the patient's functional symptoms. There are three approaches to therapy: adaptation, substitution, and canalith

Table 4 Diagnosis-Based Strategies

Diagnostic Category	Symptoms	Strategy	Objective
Unilateral vestibular dysfunction, high frequency	Disrupted gaze stabilization with active head movement. May also be visually provoked.	Adaptation	Return normal VOR function. Extinguish symptoms.
Unilateral vestibular dysfunction, no balance component	Motion or positionally provoked sense of dizziness or exaggeration of motion.	Adaptation	Extinguish or ameliorate motion or visually provoked symptoms. Enhance VOR function.
Unilateral vestibular dysfunction with balance component	Same as above, with addition of imbalance and unsteadiness when walking, particularly on dynamic surfaces.	Adaptation and substitution	Same objectives as above with the addition of improved stability and equilibrium. Reduce visual and surface dependence.
Bilateral vestibular dysfunction	Surface and visual dependence. May present with oscillopsia, if post aminoglycoside.	Substitution, conditioning, fall prevention strategies.	Enhance balance function. Strengthen remaining sensory systems. Increase independent ambulation, reduce oscillopsia.

repositioning-liberatory maneuvers. They may be used independently or in conjunction with one another, depending on the patient's needs.

Adaptation will reset or retune the VOR by repetitive activities. These activities will include those situations or movements that provoke the very symptoms the patient has been trying to avoid. Activities will incorporate mostly coordinated head and eye movement, along with head and eye movement with full ambulation. Presenting the patient with multiple simultaneous acceleration stimuli in a variety of planes will provide an excellent activity with a minimum of space, cost, and equipment. A good example of this would be to have the patient sit on a balance ball, with a slight bounce while they turn their head from side-to-side and read two separate word lists, such as a grocery list. Activities that disrupt the predictability of gaze stabilization or somatosensory input will be useful. Gaze stabilization exercises may progress from easy to more difficult as a progression from the patient performing side-to-side head turns while seated on a stationary chair to those while seated on a ball.

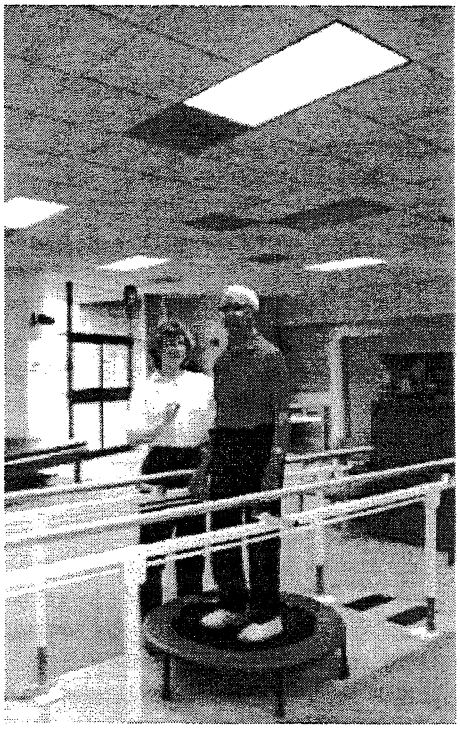

Figure 1 A VRT Patient's other sensory modalities are challenged to strengthen vestibular function.

Baseline, serial, or final performance can be evaluated with any technique that evaluates the VOR function. This may be as simple as testing dynamic visual acuity with a Snellen eye chart, or as complicated as using the technologically advanced Vestibular Autorotation Testing that provides a computerized analysis of the eye and head velocity.

Substitution protocols will strengthen the weakened systems by reducing the dependence on the remaining ones. Or, in the case where a sensory modality is missing, will work to strengthen or make the remaining systems more trustworthy. Figure 1 is a good example of a substitution protocol application. A patient with a weakened vestibular system is forced to make it more dominant by reducing or challenging the somatosensory input by standing on a trampoline. The visual sense could be further disrupted or diminished by having the patient close his eyes or watch a moving visual stimulus while maintaining his balance.

Evaluation of a patient's performance may include tests of postural stability with dynamic surface and absent vision. A simple version of this test is Horak and Schumway-Cook's classic Clinical Test of Sensory Integration of Balance (CTSIB). A more complex test would consist of the Equitest Computerized Dynamic Posturography, used by NASA to evaluate balance function of returning shuttle astronauts. Figure 2 shows a patient being trained on a Balance Master unit providing him with visual feedback about his limits of stability and balance function during therapy.

Canalith Repositioning and Liberatory Maneuvers are used to treat Benign Paroxysmal Positional Vertigo (BPPV), the number one cause of vertigo in older adults. The diagnosis and treatment of these patients are dependent on the therapist's skill in understanding the patients eye movement associated with the condition that is termed nystagmus. Because of the transitory nature of the nystagmus that lasts less than 20 sec, the ability to capture the nystagmus on videotape is invaluable. This allows the therapist to review the tape and to

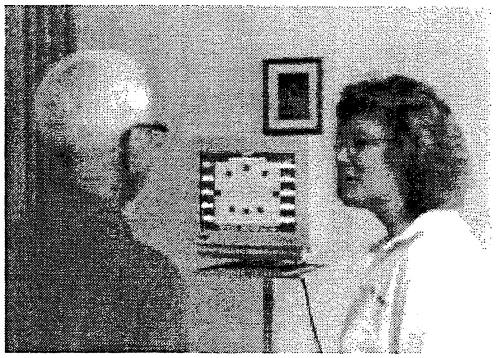

Figure 2 Balance Master provides visual feedback for the patient's postural control training.

document the efficacy of treatment. It is also an excellent teaching tool for patients, their families, and referring physicians. This technology is simple and requires only a TV/VCR to plug into for review and taping, as seen in Figure 3. It is important that clinicians make a differential diagnosis in BPPV, identifying the involved canal as well as the canalithiasis vs. cupulolithiasis variant.

Figure 4 provides a simplified clinical pathway for diagnosis and treatment of BPPV.

THERAPY MODELS

Self-Directed Exercises This is a home-based therapy that patients can do on their own. Each program is individually designed for patients (based on their test results) to include situations that bring on their symptoms. This approach is most commonly done with patients who do not require supervision while they exercise. Best results occur when the patient spends 20 to 30 min per session, 2 to 3 times daily. Most patients report a significant reduction in symptoms within a 2 to 4 week span. This has been a particularly valuable method for patients who have busy social or professional lives, and who cannot find the time (and are not required) to come into the clinic (see Table 5).

Clinician-Directed Exercises This program is designed for patients whose symptoms may be rather severe and require supervision during the exercises. Therapy sessions usually include the use of a variety of special vestibular therapy equipment that most people thoroughly enjoy using. Fall prevention also is emphasized for

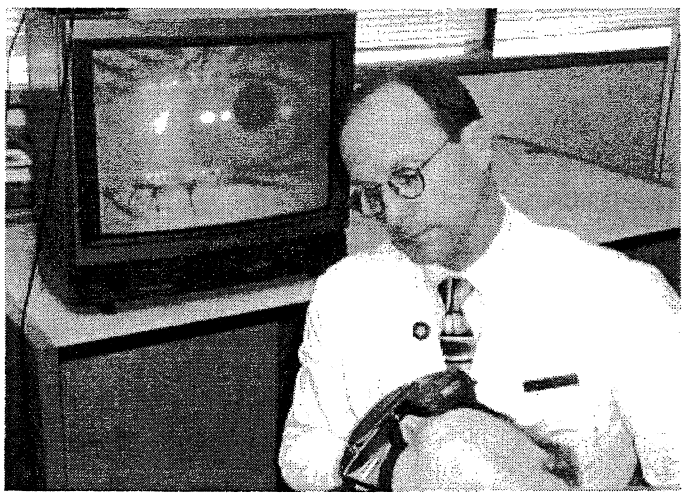

Figure 3 Video-oculography provides clinicians with the ability to review and record eye movement.

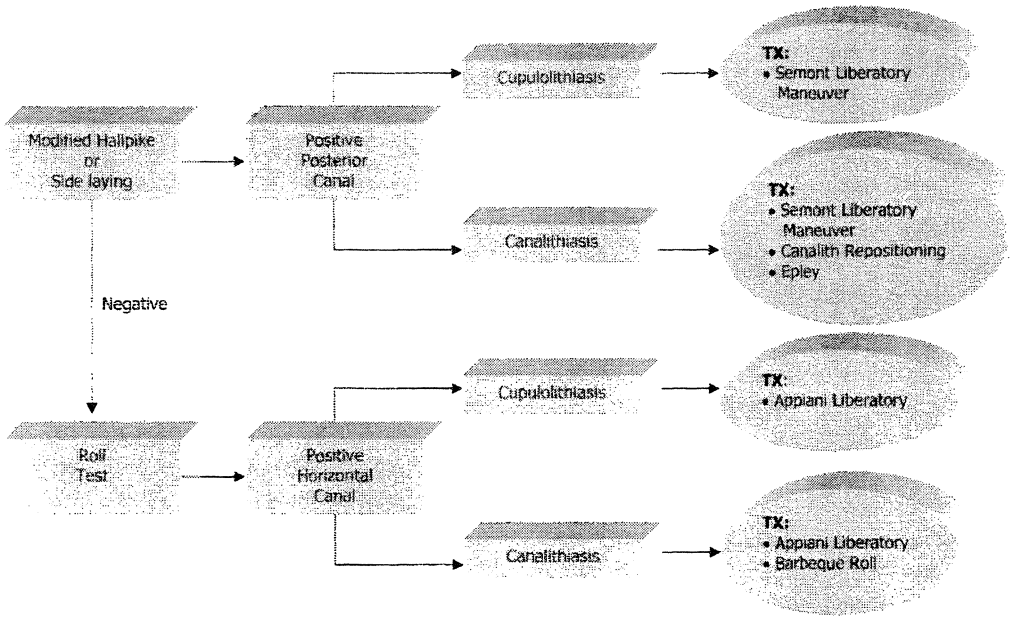

Figure 4 Diagnostic and treatment clinical pathways for BPPV of the posterior and horizontal canal.

older patients. Typically, the patient participates in one or two 60-min sessions per week, with an average of 7 to 12 sessions. As the patient progresses, he/she is given some self-directed exercises for use at home to accelerate improvement (see Table 6).

CASE STUDY

The following case study and clinical pathway is provided as an example of a typical vestibular patient.

History and Symptoms

A 49-year-old female was referred to the clinic by her primary care physician with a chief complaint of acute positional vertigo. The physician obtained a magnetic resonance image (MRI) and blood work profile, all of which were reported as normal. Her history included episodes of vertigo, the first of which began approximately four months prior. The initial episode lasted for approximately 7 to 8 days, with severe nausea and emesis throughout the episodic period. She enjoyed a spontaneous recovery of the

Table 5 Self-Directed Vestibular Rehabilitation Therapy

Ideal for high frequency UVDs without acute symptoms or imbalance.
Minimizes disruption of work or home lifestyle.
Cost effective, no need for third-party involvement.
Requires motivated patient willing to perform protocols 2–3 times per day for 30 to 90 days.
Treatment efficacy as successful as clinician directed.

Table 6 Clinician-Directed Vestibular Rehabilitation Therapy

Practical for patients with acute motion or visually provoked symptoms.
Those who are unable to proceed without a coach present.
Patients with UVDs with imbalance or unsteadiness.
Therapy sessions 1–2 times per week for 6 weeks or less.
Patients supplement therapy with home protocols.

vertigo and was symptom-free until approximately two months later. The second attack lasted about 7 to 8 days. The only other condition or symptom that occurred during these acute episodes was a significant outbreak of cold sores. Following the last attack (within 4 to 5 days), the patient reported an acute episode of vertigo when she would lie flat or turn her head while lying down. This vertigo would last only seconds. Her complaints also included a sensation that the world was "jiggling" when she looks from side-to-side or when walking. She felt as though her eyes "didn't have any shock absorbers." Although the sensation of her world bouncing is not as frightening as the acute positional vertigo, it is annoying and has limited her activity level. No hearing loss was associated with the attacks or subsequent to them, nor was there a history of migraine or migraine equivalent.

Clinical Findings

Video-Oculography revealed a left posterior canalithiasis during modified Hallpike positioning. A 43% left unilateral weakness was revealed on the caloric portion of VNG testing. Vestibular Autorotation Testing indicated abnormal (hypofunction) gain in the horizontal and vertical plane. Computerized Dynamic Visual Acuity Test (CDVAT) produced a 25% decrease in visual acuity with active head movement in the vertical plane. All other audiologic studies were unremarkable. The patient had normal hearing acuity for all test frequencies. Normal emission studies and distortion product otoacoustic emissions also were obtained.

Recommendations

The treatment strategy with this patient was twofold. First, the recurring attacks and nature of symptoms (including outbreak of cold sores) was suggestive of a viral vestibular neuritis. An otology consult was conducted. Following positive lab results for the herpes simplex type 1, the patient was placed on an antiviral medication prescription and a daily regimen of a Lysine supplement by the otologist to control or inhibit further outbreaks. The patient was referred back to the clinic for treatment of the left posterior canal BPPV and noncompensated left UVD.

Treatment and Outcomes

As can be seen in the clinical pathways of Figure 5, the patient's VRT pathway included several visits. During visit one, the left posterior canal BPPV was treated with a modified Canalith Repositioning Maneuver. The patient was placed in a soft cervical collar to reduce head movement over the next 24 hours, and provided with written and verbal instructions. She was scheduled to return to the clinic for a second visit, at which time Video-Oculographic recording would be used to ensure that the BPPV had been cleared. If the patient's oscillopsia-type symptoms persisted, she would then be provided with a Self-Directed Vestibular Rehabilitation Program. On the second visit, the patient was deemed to have been successfully cleared of the BPPV based on the absence of rotatory-torsional nystagmus and vertigo with modified Hallpike positioning. There was no migration into any of the adjoining semicircular canals. She did report her oscillopsia-type complaint and the visual provoked symptoms (as when walking through the grocery store or through a mall) persisted. She was provided with a Self-Directed Vestibular Rehabilitation Program.

On the third visit, which was 60 days post initiation of the program, she reported a 100% reduction in symptoms. Retesting of Vestibular Autorotation and Computerized Dynamic Visual Acuity Testing indicated a recovery and return to normal function on both tests.

The patient was pleased with these results and was discharged from clinical care. She was encouraged to follow-up with both her primary care physician and the otologist should she have a recurrence of the acute-phase vestibular neuritis. She was encouraged to recom-

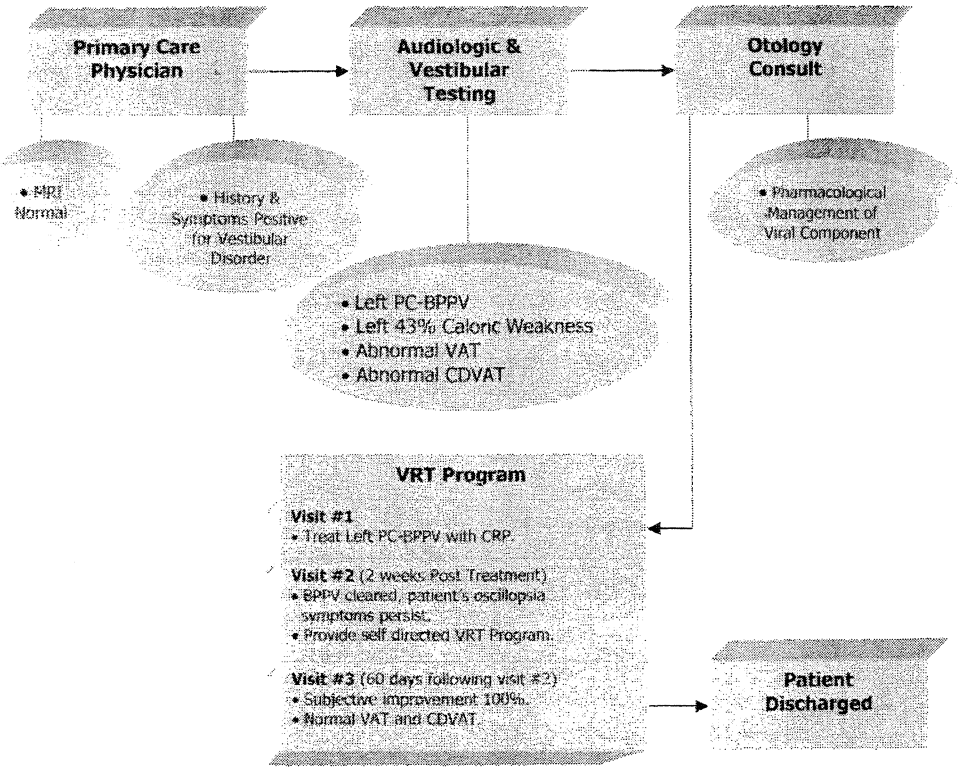

Figure 5 Diagnosis and treatment clinical pathway for case study of 49-year-old female with left posterior canal Benign Paroxysmal Positional Vertigo, and noncompensated left unilateral vestibular dysfunction, postvestibular neuritis.

mence with her Self-Directed Vestibular Rehabilitation Protocols if at any time she felt a return of any of the oscillopsic symptoms with active head movement.

SUMMARY

Fifty years of literature, clinical experience, and a growing patient acceptance has solidified VRT as an important and efficacious nonmedical treatment for the symptoms of noncompensated vestibular disorders. Our understanding of the importance of intact VOR function in human equilibrium, and new and simplified tests of this system will allow us to continue to quickly identify and treat this population. New and exciting research in cochleovestibular hair cell regeneration may someday make VRT unnecessary. In the meantime, VRT presents as one of the most simple and successful treatment options available.

ABBREVIATIONS

BPPV	Benign Paroxysmal Positioning Vertigo
CDP	Computerized Dynamic Posturography
CDVAT	Computerized Dynamic Visual Acuity Test
CNS	central nervous system
CTSIB	Clinical Test of Sensory Integration of Balance
MRI	magnetic resonance image
NIH	National Institutes of Health
VOR	vestibulo-ocular reflex
VRT	Vestibular Rehabilitation Therapy
VSR	vestibulo-spinal reflex

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