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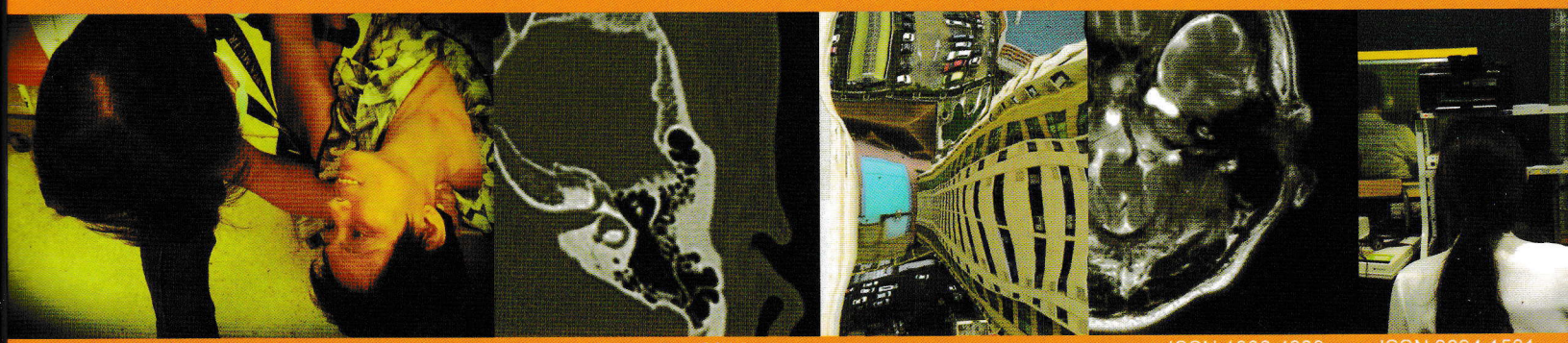

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Clinical Practice Guidelines Vertigo in Adults

Second Edition

PHILIPPINE SOCIETY OF OTOLARYNGOLOGY - HEAD AND NECK SURGERY, INC.
UNIVERSITY OF THE PHILIPPINES - PHILIPPINE GENERAL HOSPITAL
DEPARTMENT OF OTORHINOLARYNGOLOGY
SECTION OF OTOLOGY, NEUROTOLOGY, LATERAL SKULL BASE SURGERY AND AUDIOLOGY

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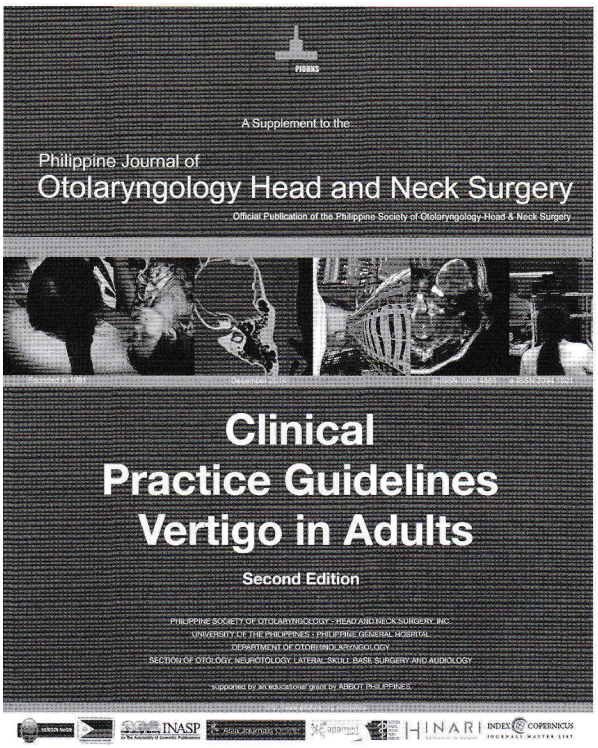

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Clinical Practice Guidelines Vertigo in Adults – 2nd Edition

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CLINICAL PRACTICE GUIDELINES VERTIGO IN ADULTS – 2nd Edition

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INTRODUCTION

The clinical practice guidelines (CPG) on vertigo in adults was first published in 2003. It was made by the Task Force on Clinical Practice Guidelines-Vertigo of the Philippine Society of Otolaryngology-Head & Neck Surgery, Inc. which is composed of general otolaryngologists, otologists and neurotologists from the different accredited ENT training institutions and provincial ENT practitioners. This material is an update of the previous CPG incorporating new studies and researches from 2003 to present.

This report will need to be reviewed, modified and updated periodically according to the availability of new knowledge.

SCOPE OF THE PRACTICE GUIDELINE

These clinical practice guidelines are for the use by general otorhinolaryngologists. It covers the diagnosis and management of vertigo in adults (19 years old and above) in an ambulatory care setting.

OBJECTIVES

The objectives of the guideline are (1) to assist general ENT practitioners to determine true vertigo in adults; (2) to evaluate current diagnostic techniques; and (3) to describe treatment options.

LITERATURE SEARCH

The National Library of Medicine's PubMed database was searched in the first edition, using the keyword vertigo. The search was limited to articles involving humans and those published in English in the last fifteen years, WHO reports, and the PGH 2002 Annual Report. In this update, the National Library of Medicine's Pubmed database was again searched for literature using the 2003 CPG on vertigo guide. The search was limited to articles involving humans from 2003 and present. After review of the search, 80 abstracts were deemed relevant to the clinical questions posed by the guidelines developers. Full text articles were obtained when possible.

All materials were assessed for relevance and further classified according to levels of evidence and grades of recommendations based on guidelines from the Oxford Centre for Evidence-based Medicine Levels of Evidence May 2001 [1].

THESE CLINICAL PRACTICE GUIDELINES ARE FOR THE GENERAL OTORHINOLARYNGOLOGIST IN THE APPROACH OF ADULT PATIENTS WITH VERTIGO. IT IS GEARED TO DIAGNOSE THE COMMON CAUSES OF VERTIGO WITH THE USE OF CLINICAL HISTORY, PHYSICAL EXAMINATION AND MINIMAL DIAGNOSTIC TESTING. THE EMPHASIS IS THE DIAGNOSIS AND TREATMENT OF COMMON CAUSES OF VERTIGO WHILE COGNIZANT OF DISEASES REQUIRING REFERRAL TO A SPECIALIST FOR SPECIFIC INTERPRETATION OF WORK-UP AND TREATMENT.

DEFINITION

Vertigo is defined as an illusion of movement self or of the environment. [2]

RECOMMENDATIONS ON THE DIAGNOSIS OF VERTIGO

1. **If a patient presents with dizziness, the first step should be to ascertain if it is a true vertigo or not.**

   Grade D Recommendation, Level 5 Evidence

   When collecting a patient's history, the physician must first determine whether the patient truly has vertigo versus another type of dizziness. There are four types of dizziness: *vertigo, lightheadedness, presyncope, and dysequilibrium*. The main causes of vertigo are benign paroxysmal positional vertigo, Meniere's disease, vestibular neuritis, and labyrinthitis. Many medications can cause presyncope and these should be elicited in history. Parkinson's disease and diabetic neuropathy should be considered with the diagnosis of disequilibrium. Psychiatric illness can cause vague lightheadedness. [2,3,4]

2. **A carefully obtained medical history is the most important part in the evaluation of a patient with vertigo. The history may be very suggestive of a diagnosis. It guides the examination and work-up.[5,6]**

   Grade D Recommendation, Level 5 Evidence

   The history should include the following:

   2.1 chief complaint
   2.2 history of present illness (recent history of viral infections, colds, co-morbid symptoms)
   2.3 past medical history to include previous head trauma, medications, medical and surgical illnesses
   2.4 pertinent family, personal and social history
   2.5 brief review of symptoms

3. **The chief complaint of vertigo should be evaluated and described thoroughly.[7]**

   Grade D Recommendation, Level 5 Evidence

   The vertigo episode must be described as to the following:

   3.1 what the patient actually felt in his own words
   3.2 mode of onset
   3.3 frequency, severity, intensity and duration of individual and succeeding episodes (diminishing or increasing) and compare it with initial episode
   3.4 triggering and alleviating factors
   3.5 associated auditory symptoms (hearing loss, tinnitus or ear fullness)
   3.6 effects of medication

4. Appropriate physical examination should be done on a patient complaining vertigo.

Grade D Recommendation, Level 5 Evidence

The physical examination should include the following [2]:

4.1 Vital signs – blood pressure (lying, sitting and standing position to rule out orthostatic hypotension e.g. BP change of greater than 10-20 mm Hg), heart rate and respiratory rate

4.2 ORL examination (otoscopy, fistula test and tuning fork tests)

4.3 Evaluation of the vestibular system (e.g. at the least, observation for geotropic rotator spontaneous fatigable nystagmus should be included and the Dix-Hallpike maneuver should be performed)

4.4 Neurological testing (with emphasis on the cranial nerves and vestibulospinal tests e.g. Romberg's test and cerebellar tests)

5. Vertigo of sudden onset (onset within the past few minutes to hours) associated with headache, neurological signs and symptoms warrant urgent referral and investigation to avoid life-threatening or severe neurologic sequelae

Grade D Recommendation, Level 5 Evidence

Stroke should be a serious consideration in the patient who presents with acute vertigo. Fifty percent of stroke patients may present with true vertigo, imbalance problems, vague dizziness sensation or a combination of these. [8]

The **head impulse** test is a useful tool in patients complaining of symptoms of acute vertigo. This is particularly important in differentiating acute unilateral peripheral vestibulopathy (vestibular neuritis), cerebellar stroke or migraine. In the acute setting, head impulse test, when applicable, is negative in stroke patients. [9,10]

6. It is important to differentiate if vertigo is central or peripheral in origin.

Grade D Recommendation, Level 5 Evidence
Table 1 describes peripheral versus central causes of vertigo

7. If vertigo is suggestive of a peripheral cause, pure tone audiometry and speech testing must be performed.

Grade D Recommendation, Level 5 Evidence

Table 1. Peripheral vs. Central Vertigo[3]

Feature	Peripheral Vertigo	Central Vertigo
Nystagmus	Combined horizontal and torsional; inhibited by fixation of eyes onto object; fades after a few days; does not change direction with gaze to either side	Purely vertical, horizontal or torsional; not inhibited by fixation of eyes onto object; may last weeks to months; may change direction with gaze towards fast phase of nystagmus
Imbalance	Mild to moderate; able to walk	Severe; unable to stand still or walk
Nausea and vomiting	May be severe	Varies
Hearing loss, tinnitus	Common	Rare
Nonauditory neurologic symptoms	Rare	Common
Latency following provocative diagnostic maneuver	Longer (up to 20 sec)	Shorter (up to 5 sec)

In patients with central cause of vertigo, further investigation and referral is recommended.

Conventional pure tone and speech audiometry remains to be the most useful and cost effective screening tool in defining patients who should undergo further testing such as an imaging study.
The panel is cognizant of the fact that speech testing may be difficult in some situations e.g. language barriers, and in these cases a pure tone audiometry may suffice.

8. Abnormal PTA-ST results and significant asymmetric hearing loss that would warrant further diagnostic testing (e.g. MRI) are 1) 20 dB or more difference in threshold average (1,2,4, and 8 kHz); 2) 15% SDS difference.

Grade B Recommendation, Level 2 Evidence

In a study by Mangham et al (1991), further evaluation with magnetic resonance imaging (MRI) was recommended when there is an average threshold difference at 1 to 8kHz of 20 dB. [11]

9. For PTA-ST results that are highly suggestive of a retrocochlear pathology such as an acoustic neuroma, MRI is the gold standard imaging study. [12]

Grade B Recommendation, Level 2 Evidence

10. The use of ABR in the diagnosis of a retrocochlear pathology is not recommended.

Grade B Recommendation, Level 2 Evidence

In a study by Cueva in 2004, ABR has been demonstrated to have low sensitivity (71%) and specificity (74%) in finding causative lesions in patients with asymmetric sensorineural hearing loss. The false positive rate was found to be 76.84% while false negative rate was 29%. [12]

11. The history, physical examination and diagnostic tests should be correlated to arrive at a logical diagnosis.

Grade D recommendation, Level 5 Evidence

The *table 2* provides a simple tabulation of the history, PE and diagnostic test results of the most common peripheral causes of vertigo that can help the clinician have a quick working impression. The patient should fit all the criteria in the diagnosis of each specific condition. In cases of partially met criteria and doubtful diagnosis, the patient should be referred.

BENIGN PAROXYSMAL POSITIONAL VERTIGO (BPPV)

DEFINITION [15,16,17,18]

Benign paroxysmanl positional (BPPV) is a brief, episodic, and transient vertigo induced by a rapid change in head position.

The nystagmus is linear or rotatory, geotropic, and lasts less than 1 minute; it has a latency period between the maneuver that evokes it and its appearance; diminished or abolished when the maneuver is repeated (fatigability); and changes direction as soon as the patient returns to a sitting position at the end of the maneuver (reversibility); the rate of nystagmus typically begins gently, increases in intensity, and then declines in intensity as it resolves (crescendo-decrescendo nystagmus)

There is a 1- to 40-second latency before the onset of vertigo and nystagmus, and the vertigo and nystagmus has a duration of less than 60 seconds.

BPPV temporarily becomes less intense and disappears with repeated positioning.

Table2. Common causes of vertigo [6,13,14]

Disease Entities	History	Physical Exam	Diagnostics
Benign Paroxysmal Positional Vertigo (BPPV)	• brief, episodic, and transient vertigo induced by a rapid change in head position • (1-)to 40-second latency before the onset of vertigo and nystagmus • vertigo and nystagmus with a duration of less than 60 seconds • BPPV temporarily becomes less intense and disappears with repeated positioning	• (+) Dix-Hallpike with the following characteristics (preferably done without fixation) o Latency o Geotropic o Brief (30 sec) o Symptomatic o Fatigable o Reverses on sitting position	• PTA-ST: normal • Calorics: normal
Meniere's Disease	an inner ear disorder characterized by: • recurrent episodes of rotational vertigo • fluctuating progressive hearing loss (typically on low frequencies in early stages) • aural fullness • tinnitus	• May be normal	• PTA-ST: an inherent upward-sloping configuration of the mean audiometric curve at all time points during the disease • >10 dBHL change in the hearing levels at all tested audiometric frequencies before, during and after the attacks of vertigo • No significant changes in hearing thresholds were observed during vertigo attacks associated with Meniere's disease
Vestibular neuronitis	• Sudden vertigo with unsteadiness, nausea or vomiting • Persistent vertigo (days-weeks) • (-) auditory deficits • (-) other neurologic	• spontaneous nystagmus to the contralateral ear for the 1st 3 days	• PTA-ST: normal • Calorics: reduced or absent caloric response in one ear

Disease Entities	History	Physical Exam	Diagnostics
Acoustic neuroma	• Non-specific • Patient may complain more of unsteadiness rather than vertigo • May have unilateral tinnitus or hearing loss	• May have cranial nerve deficits (e.g. CN V- decreased corneal reflex)	• PTA ST: o 20 dB or more difference in threshold average (1,2,4, and 8 kHz) o 15% SDS difference. • MRI with gadolinium o (+) intracanalicular mass
Cervicogenic vertigo	• One of these symptoms appear when head/neck positions assumes a certain position/ change of position: o Headache o Vertigo o Syncope o Tinnitus o hearing loss o Nausea and vomiting o Visual symptoms e.g. flashing symptoms	• On PE, one of these symptoms may be elicited on cervical ROMs o Headache o Vertigo o Syncope o Tinnitus o hearing loss o Nausea and vomiting o Visual symptoms eg flashing lights o Supra-clavicular bruit	• PTA-ST: mostly normal • Calorics: mostly normal • Neck APL: may show cervical spondylosis or degenerative changes

THEORIES FOR THE MECHANISM OF BENIGN PAROXYSMAL POSITIONAL VERTIGO [15,19]

Schucknecht in 1969 was the first to provide a pathophysiological concept of BPPV and proposed the theory of "cupulolithiasis". The cupula, which became heavy due to attached otolithic debris, could be deflected by position changes. Reorientation of the canal relative to gravity deflects the cupula, exciting or inhibiting the ampullary organ, thus evoking nystagmus. [20]

Hall proposed in 1979, the concept of "canalolithiasis", which states that otolithic debris from the utricular macule migrates into the semicircular canal via the non-ampullary portion, causing vertigo and nystagmus by moving freely inside the semicircular canal and inducing endolymph flow during positional changes. This concept of canalolithiasis became the theoretical basis of the canalith repositioning maneuvers (CRMs) to treat BPPV. [21]

RECOMMENDATIONS ON THE DIAGNOSIS OF BENIGN PAROXYSMAL POSITIONAL VERTIGO (BPPV)

1. The Dix-Hallpike maneuver is the gold standard test for the diagnosis of posterior canal BPPV. [22,23,24]

Grade B Recommendation, Level 2A Evidence

2. Puretone Audiometry (PTA) is not required in diagnosing BPPV. However, it is a screening tool for patients suffering from vertigo in general (see Recommendations on the diagnosis of vertigo, #7).

Grade B Recommendation, Level 2B evidence

The benefits of obtaining audiometry in the evaluation of BPPV include the ability to establish baseline stability or, alternatively, to help distinguish BPPV from other associated conditions such as Ménière's disease and vestibular schwannoma.

Kentala et al in 1999 found in a study of 564 cases, that the presence of a normal audiogram was corroborating for a diagnosis of BPPV.[25]

In the vast majority of cases of BPPV with stable hearing by history, the audiogram is most likely to be normal or demonstrate an age-appropriate sensorineural hearing loss and, therefore, likely will not influence the diagnosis of BPPV.

RECOMMENDATIONS ON THE TREATMENT BENIGN PAROXYSMAL POSITIONAL VERTIGO (BPPV)

1. Particle repositioning maneuvers are the primary modality of treatment for BPPV.

1.1 Epley maneuver provides effective control and symptom resolution in patients with posterior canal BPPV after 1 maneuver. However, there is no study on how frequent Epley maneuver can be done for persistent disease.

Grade A Recommendation, Level 1A Evidence

The canalith repositioning procedure (CRP), developed by Epley, was designed to use gravity to treat canalithiasis of the posterior canal. The clinician moves the patient through a series of four positions. With each position, the otoconia settle to the lowest part of the canal, move around the arc of the posterior canal, and finally deposit in the vestibule. This procedure requires a 180-degree turn of the head and a

return to a sitting position from lying on the uninvolved side. To enable the otoconia to settle, each position is maintained for at least 30 seconds.[26]

In another prospective cohort study of 86 patients with BPPV who were treated with the Epley maneuver and evaluated within the two weeks following treatment, 70% had complete resolution within two days after first maneuver. Additional 9% had complete resolution after one week after first maneuver.[27]

In a cross-sectional retrospective study, most of the patients analyzed (76%) had a complete symptom resolution and Dix-Hallpike test resolution with only one modified Epley Maneuver.[28]

In a randomized controlled trial, a total of 106 BPPV patients were randomly assigned to three treatment groups: Brandt and Daroff habituation exercises, Semont maneuver and Epley maneuver. Their results indicate that: 1) the Epley and Semont maneuvers are more effective than Brandt and Daroff habituation exercises; 2) the initial response to the Epley maneuver was similar to the Semont maneuver; and 3) after three months of treatment, better results were obtained with Epley maneuver than with the Semont maneuver.[29]
In another randomized controlled trial, 87 subjects diagnosed with posterior canal BPPV were randomly assigned to three treatment groups: modified Epley maneuver, modified Epley maneuver with augmented head rotations and modified Semont maneuver. Their data suggested that augmented head rotations are unnecessary and that the modified Epley and modified Semont maneuvers are equally effective. [30]

In a study done by Radtke et al (2004), they found that self-treatment with Modified Epley Procedure (95% of patients) is more effective compared with self-treatment with Modified Semont Maneuver (58%) in abolishing posterior canal BPPV within one week.[31]

1.2 Semont maneuver provides symptom resolution for BPPV and a maximum of four maneuvers is recommended.

Grade B Recommendation, Level 2A Evidence

The liberatory maneuver, developed by Semont et al (1989), was designed to use inertia and gravity to treat cupulolithiasis of the posterior canal. To evacuate the particles, the patient is rapidly swung from lying on the involved side to lying on the uninvolved side through a 180-degree cartwheel motion with a duration of less than 1.3 seconds[32,33]

In the retrospective chart review, 127 cases of objective BPPV and 35 cases of subjective BPPV underwent the Semont liberatory maneuver and was assessed after three weeks for complete, partial and failure of resolution. There was 90% improvement after an average of 1.5 maneuvers, about 91% with objective BPPV after 1.6 maneuvers had improvements with 29% recurrence rate after first maneuver, while 96% of the remaining responded to the succeeding maneuvers. More than 90% of patients were cured after a maximum of 4 Semont's maneuvers and 83.5% were cured after 2 maneuvers. The efficacy decreased each time it was repeated. [34]

Repeating Semont's maneuver several times has been shown to progressively increase the percentage of cured patients, with symptoms abolished after the 1st maneuver in 61.6% of cases. Further maneuvers, increased the percentage of cured patients to 82.5% after the 2nd, 90.7% after 3rd and 94.1% after the 4th. [35]

Levrat et al in 2003 demonstrated that the Semont maneuver is effective, with a 90.3% recovery rate after 4 maneuvers. Results showed that most patients (83.5%) are cured after only 2 Semont maneuvers and that ensuing maneuvers only slightly improve (6.8%) the global recovery rate. Nevertheless, this supplementary recovery rate, apparently weak, represents 41.3% (19/46) of patients not cured after the first 2 maneuvers. It is, therefore, well worth the effort to repeat the maneuver several times. Similar results have been reported by Serafini et al, who obtained a 94% success rate after 4 maneuvers. [16]

In a randomized, controlled trial, 40 patients affected by BPPV were treated with Semont's maneuver. Cure rates with Semont's maneuver were significantly higher than those obtained with no-therapy (92.5% versus 37.5%). Within a six month follow-up, relapse rates were lower among patients treated with Semont's maneuver than among the no-treated ones (5% versus 60%). [36]

1.3 Postural restriction after reposition maneuvers among patients with BPPV is not recommended.

Grade B Recommendation, Level 2B Evidence

In a prospective double-blind study, 391 consecutive patients diagnosed of posterior canal BPPV with a positive Dix-

Hallpike test were included to establish if postural restrictions are useful after repositioning maneuvers in posterior canal benign paroxysmal positional vertigo (BPPV). There were no statistically significant difference between patients who restricted their movements (80.2% of success with 1 maneuver) and those who did not (72.3%). [37]

In a study done by Moon SJ et al (2005), about 70 patients diagnosed with BPPV of the posterior semicircular canal were studied and treated utilizing the modified Epley maneuver. Postural restriction therapy after the modified Epley repositioning maneuver, did not have a significant effect on the final outcomes of BPPV. [38]

In a prospective randomized controlled study among 50 patients presenting with BPPV of the posterior semicircular canal and treated with Epley Maneuver done by Simoceli et al in 2005, it was shown that there is no significant difference in the efficacy of Epley Maneuver with and without post-maneuver restrictions. [39]

1.4 Brandt-Daroff exercises do not follow the pathophysiology of BPPV, hence we do not recommend it as primary treatment. [22,40]

Grade D Recommendation, Level 5 Evidence

In 1980, Brandt and Daroff described home repositioning exercises that involve a sequence of rapid lateral head/trunk tilts repeated serially to promote loosening and ultimately dispersion of debris toward the utricular cavity. In these exercises, the patient starts in a sitting position and moves quickly to the right-side lying position, with the head rotated 45 degrees and facing upward. This position is maintained for 30 seconds after the vertigo stops. The patient then moves rapidly to a left-side lying position, with the head rotated 45 degrees and facing upward. [41]

In a prospective, randomized controlled trial in 124 patients with posterior semicircular canal BPPV done by Cohen and Kimball in 2005, it was shown that vertigo decreased significantly after liberatory maneuver, canalith repositioning procedure, and Brandt-Daroff exercise. Patients from those three groups also did not differ significantly. [30]

2. Repeated repositioning maneuvers may be attempted on recurrent attacks of BPPV. However, recurrent attacks may warrant further investigation. A patient suspected of having BPPV should be referred if: (1) the Dix-Hallpike test is negative despite repeated testing in the recurrently symptomatic patient; (2) the nystagmus seen in Dix-Hallpike test is atypical (not torsional, ageotropic); (3) remains symptomatic despite treatment and (4) with other otologic or neurologic symptoms.

Grade D Recommendation, Level 5 Evidence

3. Vestibular suppressants (e.g. benzodiazepines) and anti-vertigo drugs (e.g. H3 receptor antagonists) may be given for symptomatic relief of patients with BPPV.

Grade B Recommendation, Level 2B Evidence

In one double- blind controlled trial comparing diazepam, lorazepam, and placebo, all groups showed a gradual decline in symptoms with no additional relief in the drug treatment arms. [42]

In a prospective study among 103 patients diagnosed with BPPV, it was shown that liberatory maneuver and Brandt-Daroff exercises associated with betahistine produced faster recovery rates than the mentioned procedures alone. [43]

In a double-blind, randomized, controlled clinical trial in 72 patients with BPPV, it was shown that Epley maneuver, alone or combined with betahistine or placebo, was found to be effective (86.2%) with regard to improvement of symptoms in certain patients. Thus there is some evidence that betahistine may help in BPPV but stronger evidence is needed. [44]

MENIERE'S DISEASE

DEFINITION

Menière's Disease (MD) is an inner ear disorder characterized by recurrent episodes of rotational vertigo, most typically associated with fluctuating progressive hearing loss, typically on low frequencies in early stages, fullness and tinnitus. [45,46]

RECOMMENDATIONS ON THE DIAGNOSIS OF MENIERE'S DISEASE

1. The characteristic clinical presentation is the gold standard in diagnosing Meniere's disese.

Grade D Recommendation, Level 5 Evidence

Criteria used to diagnosed MD are the presence of at least two episodes of vertigo of at least 20 minutes' duration, audiometrically confirmed sensorineural hearing loss on at least one occasion, and

tinnitus or aural fullness during episodes. [45,46]

2. A low frequency sensorineural hearing loss is suggestive of Meniere's.

Grade B Recommendation, Level 2B Evidence

In the early stage of Meniere's Disease, when the first vertiginous episodes occur, it is the presence of a low-tone hearing loss that indicates the onset of the disease and the beginning of the disabling state.

In a prospective cohort study by Mateijsen et. al (2001), the affected ears significantly show low frequency hearing losses. The hearing loss, however, does not correlate with the duration of the disease. [47]

As Meniere's Disease progresses through the cochlea, low frequency fluctuation is followed by fluctuation in the mid frequencies, leading to fluctuation across all frequencies. Use of a self-hearing test may facilitate diagnosis and hearing aid fitting for this population, as clinical audiograms may not provide accurate information of hearing fluctuation. [48]

Audiometry results corrected for patient age show an inherent upward-sloping configuration of the mean audiometric curve at all time points during the disease (>10 dBHL change in the hearing levels at all tested audiometric frequencies before, during and after the attacks of vertigo). No significant changes in hearing thresholds were observed during vertigo attacks associated with Meniere's Disease. [49,50]

RECOMMENDATIONS ON THE TREATMENT OF MENIERE'S DISEASE

1. For acute attacks of vertigo associated with Meniere's disease, vestibular suppressants and/or anti-vertigo drugs may be given.

Grade B Recommendation, Level 2b Evidence

Medications that may be used for relief of acute attacks of vertigo are:

1.1 **Droperidol –** given through intramuscular route. The panel, however, is cognizant that Droperidol is not available locally. [51]

1.2 **Diazepam** - widely used as a vestibular suppressant because of its additional tranquilizing effect. This should not be given for more than three days. [52]

1.3 **Dimenhydrinate** [52]

1.4 **Diphenhydramine** [52]

1.5 **Meclizine** [52]

1.6 **Anti-vertiginous medications, anti-emetics, sedatives, antidepressants, and psychiatric management** have been reported to be beneficial in reducing the severity of vertigo and vegetative symptoms and in improving tolerance of Meniere's symptoms. [2]

1.7 **Calcium antagonists, e.g. nimodipine and cinnarizine** [53]
In a comparative double blind study involving 181 subjects, two calcium antagonists, namely nimodipine and cinnarizine, were found to be equally effective in the symptomatic treatment of vestibular vertigo. The dosages were at 30 mg nimodipine tablet taken TID and 150 mg cinnarizine tablet taken OD for 12 weeks. Both had similar safety profiles. [53]

2. Because of the episodic nature of Meniere's disease, a trial of treatment to prevent the attacks should be instituted for 2-3 months.

2.1 The use of diuretics (with serum potassium monitoring) in Meniere's disease is recommended.

Grade A Recommendation, Level IB Evidence

In a cross-over placebo-controlled study of 33 patients with Meniere's disease, dyazide (50mg triamterene and 25mg hydrochlorothiazide) was found to decrease significantly the vestibular complaints, but had no effect on hearing and tinnitus. [54]
Hydrochlorthiazide single preparation is currently available in the Philippines and may be given as Hydrochlorthiazide 50mg/tab once a day with potassium supplementation and monitoring of serum K level.

2.2 Betahistine dihydrochloride, at 24 mg BID or 16 mg tablet TID for 2-3 months, is recommended for patients diagnosed with Meniere's disease.

Grade A Recommendation, Level IB Evidence

Betahistine dihydrochloride significantly reduced the

number of vertigo attacks, their intensity score and duration in Meniere's disease. 16 mg TID and 24 mg BID provide similar efficacy and tolerabililty in the treatment of vertigo in patient with Meniere's disease. [55,56]

Dosage can be given as 16 mg twice per day for three months [55,57]to as little as 8 mg/tab BID[58] . There is evidence that high dose, 48 mg TID, was more effective in long term control of vertigo. [59]

In the intercritical phase of Menière's Disease, betahistine therapy offers excellent results when prolonged (at least three months) and administered at an adequate dosage (at least 48 mg/day).The positive results obtained with betahistine, as compared with those of other drugs, are statistically more strongly correlated. Betahistine's efficacy is based on facilitating compensation after vestibular deafferentation. [60]

In the management of tinnitus, betahistine (36 mg/day for 12 weeks) showed greater efficacy versus cinnarizine and dimenhydrinate, without causing significant side effects. [60,61]

In a randomized controlled trial comparing betahistine dihydrochloride (16mg tab TID) and acetazolamide (125mg tablet OD) on 95 patients with Meniere's Disease, betahistine was significantly more effective than acetazolamide in reducing the severity and frequency of vertigo spells. Treatment duration was 6 months. [62]

3. **Salt restriction is recommended as a non-pharmacologic treatment option for Meniere's Disease.**

Grade C Recommendation, Level 3 Evidence

Salt restriction and diuresis are believed by many to be the best medical therapy for those with Meniere's disease. The goal is to reduce endolymph volume by fluid removal or reduced production. [63] Duration of disease is not correlated with degree of response to diuretics and salt restriction. [54]

4. **The panel is cognizant of other treatments reported for Meniere's disease including hyperbaric oxygen therapy and pressure therapy (Meniette device). It is recommended that these be considered only within the limits of a well-controlled clinical trial subject to further evaluation for evidence of effectivity.**

Grade D Recommendation, Level 5 Evidence

5. **Alternative treatments for intractable Meniere's Disease that are invasive such as intratympanic injections (Gentamicin and Dexamethasone), laser therapy, picrotoxin and surgery cannot be recommended in this guideline because such therapies will require a more thorough evaluation in order to exclude other diseases that are not part of this guideline.**

Grade D Recommendation, Level 5 Evidence

VESTIBULAR NEURONITIS

DEFINITION

Vestibular neuronitis is an acute onset of severe rotatory vertigo that is aggravated by head movement, with disequilibrium, nausea, and vomiting, and with no other auditory or neurologic signs or symptoms. The etiology of vestibular neuronitis is thought to be viral, particularly reactivation of human herpes simplex virus 1. [64,65,66,67,68]

RECOMMENDATIONS ON THE DIAGNOSIS OF VESTIBULAR NEURONITIS

1. **Careful history and assessment of the character of vertigo should be done in patients suspected for vestibular neuronitis. The character of symptoms should be properly evaluated.**

Grade B Recommendation, Level 2C Evidence

1.1 Acute Phase [65,66]

The acute phase is characterized by continuous vertigo that persists for more than 24 hours. The usual course is that the patient is bedridden for a few days, and then there is gradual recovery.

In the acute phase, there is postural imbalance with falls toward the affected ear.

In the acute phase, there is horizontal spontaneous nystagmus (with a rotational component) toward the unaffected ear, usually lasting for three days, but may last up to eight days in some patients. [69]

This spontaneous nystagmus is typically reduced in amplitude during fixation because visual fixation suppresses the vestibuloocular reflex. The intensity of spontaneous nystagmus is enhanced by eye closure, Frenzel's glasses, and during convergence.

If there are no significant differences on the intensity of nystagmus with or without fixation, this indicates a central origin and lesion and excludes vestibular neuritis.

Amplitude and slow-phase velocity are increased with gaze shifts in the direction of the fast phase, and decreased with gaze shifts in the direction of the slow phase of the nystagmus.

1.2 Rehabilitation Phase

In the rehabilitation phase, which can last for weeks to months, the patient will experience increased discomfort (dizziness, nausea and fatigue) of activity that involves motion. [64,66]

2. Careful anamnesis for vascular risk factors and a general neurologic examination are required to rule out the common finding of a vertebrobasilar infarction as an acute peripheral vestibular deficit.

Grade B Recommendation, Level 2C Evidence

Kerber (2009) stated that a small stroke within the posterior fossa can present as acute severe dizziness that may mimic vestibular neuronitis. However, on further history and physical exam, only in extremely rare cases will a stroke mimic all features of vestibular neuronitis. [8]

3. Bedside vestibular exams may be done to confirm if the findings are consistent with the diagnosis of vestibular neuronitis.

Grade B recommendation, Level 2B Evidence

Bedside **head impulse/ head thrust** test should be done in the acute phase to rule out central pathology.

The head-impulse test was performed by rapidly rotating the patient's head (abrupt, high-acceleration rotations of about 20° amplitude) to the right and to the left. The examiner stood in front of the patient, who was instructed to fix on the examiner's nose. Results of the head-impulse test were considered abnormal if there was an obvious corrective saccade supplementing an inadequate slow phase with acceleration toward one (affected) side. [64]

The disadvantage of this test is that you do not get any documentation of the disability. In addition, the impulse test is less sensitive in the limited damage of the balance nerve. [65,66]

RECOMMENDATIONS ON THE TREATMENT OF VESTIBULAR NEURONITIS

1. In the acute attacks of vertigo associated with vestibular neuronitis, vestibular suppressants may be given. However, prolonged administration of vestibular suppressants may delay central compensation.

Grade D Recommendation, Level 5 Evidence

Although there are no clinical trials found investigating the effect of vestibular suppressants on acute vestibular neuronitis, these drugs have been used in trials as part of their protocol, for symptomatic treatment for acute attacks of vertigo caused by vestibular neuronitis (Shupak et al 2008). [70] Vestibular suppressants are also advised in reviews and practice recommendations, and are advised to be withdrawn as soon as symptoms lessen or become tolerable, usually after three days. [65,71,72]

The following vestibular suppressants may be used:

1.1 Dimenhydrinate

Strupp and Brandt (2009) suggests that during the first 1 to 3 days, when nausea is pronounced, vestibular sedatives such as dimenhydrinate 50 to 100 mg every 6 hours. [65]

1.2 Benzodiazepines

A retrospective case review was done in 2005 to describe the clinical features of patients with failed vestibular nerve section that have a response to anticonvulsant medication. They reported three patients who were afflicted with frequent brief spinning spells, or "quick spins," after vestibular neurectomy for Meniere's disease. Results showed that all cases had an excellent therapeutic response to carbamazepine or oxcarbazepine. (Moon and Hain 2005) [73]

If the vertigo is severe, the use of diazepam, 5 to 10 mg IV, may be beneficial. [2]

1.3 Promethazine

Promethazine is another potent drug that can be administered IV, per rectum, and by mouth. Doses from 25 to 50 mg are often required every 4 to 6 hours. [2]

2. The benefits of the administration of other anti-vertigo drugs for vestibular neuronitis still need further investigation.

Grade D Recommedation, Level 5 Evidence

There are no strong evidences or recommendations from the above sources on the use of other anti-nausea or anti-vertigo drugs in the symptomatic treatment of vestibular neuronitis.

2.1 Flunarizine

In a case series reported by Corvera (2002) involving 23 subjects with vestibular neuritis, oral flunarizine at 5mg tablet daily in a single dose was taken together with physical exercises. Flunarizine appears to be useful in the treatment of vertigo caused by vestibular neuritis. However, it could not be determined whether the portion of the change was obtained by flunarizine and exercises and what was due to spontaneous evolution. [74]

2.2 Meclizine

There were no clinical studies found on the use of Meclizine specifically on treatment of vestibular neuronitis.

2.3 Cinnarizine

There were no clinical studies found on the use of Cinnarizine specifically on treatment of vestibular neuronitis.

2.4 Betahistine

There were no clinical studies found on the use of betahistine specifically on treatment of acute vestibular neuronitis.

Studies on Betahistine simply investigated their effect on vestibular compensation on the recovery phase of the disease.

In a review by Lacour (2006), betahistine was demonstrated to strongly facilitate vestibular compensation in the cat. The cat model showed dose- and duration-dependent effects of betahistine; low doses of betahistine, close to those given to humans (2 or 5 mg/kg/day), had significant effects only when treatment was given for a sufficient period (2-3 months). It was concluded that functional recovery can be facilitated pharmacologically using betahistine treatment in the long-term to accelerate the rebalancing of the vestibular loss-induced asymmetries. [71]

Redon et al (2011) provided evidence on betahistine's role of promoting earlier recovery of static functions in humans. [55]

3. Vestibular rehabilitation initiated as early as possible to improve balance function is recommended.

Grade A Recommendation, Level 1A Evidence

In the randomized controlled trial by Strupp et al, 39 patients (20 in the control group, 19 in the physiotherapy group) diagnosed with vestibular neuronitis were analyzed. Vestibular exercises were found to improve vestibulospinal compensation in these patients, thereby improving balance function. It seems best to start as early as possible with the exercises after symptom onset.[75]

In a COCHRANE database systematic review, 21 RCTs on unilateral vestibular pathology were reviewed in a meta-analysis. It was concluded that there is moderate to strong evidence that vestibular rehabilitation is a safe, effective management for unilateral peripheral vestibular dysfunction, based on a number of high quality randomised controlled trials. There is moderate evidence that vestibular rehabilitation provides a resolution of symptoms in the medium term. [76]

Vestibular exercise involves a gradual program of physical exercise under the supervision of a physiotherapist. Static stabilization is concentrated on, then dynamic exercises are done for balance control and gaze stabilization during eye–head-body movements. It is important that the degree of difficulty of exercises for equilibrium and balance be successively increased above normal levels, both with and without visual stabilization. [65]

4. Steroids may be useful for improving peripheral vestibular function in vestibular neuronitis.

Grade B Recommendation, Level 2B Evidence

In a randomized controlled trial of 114 patients (randomized into (a) thirty patients in the placebo group, (b) 29 in the methylprednisolone group, (c) 27 in the valacyclovir group, and (d) 28 in the methylprednisolone-plus valacyclovir group). Methylprednisolone (or a matching placebo) was administered for 3 weeks as follows: Days 1 through 3 - single morning dose of 100 mg; days 4 through 6 – 80 mg; days 7 through 9 – 60 mg; days 10 through 12 – 40 mg; days 13 through 15 – 20 mg; days 16 through 18 – 10 mg; days 20 and 22 – 10 mg; Valacyclovir, (or matching placebo), was given as two 500-mg capsules three times daily for seven days.

The mean (±SD) improvement in peripheral vestibular function

(based on caloric test) at the 12-month follow-up was: 9.6±28.1 percentage points in the placebo group; 62.4±16.9 percentage points in the methylprednisolone group; 36.0±26.7 percentage points in the valacyclovir group; 59.2±24.1 percentage points in the methylprednisolone-plus-valacyclovir group. Analysis of variance showed a significant effect of methylprednisolone (P<0.001) but not of valacyclovir (P=0.43). The combination of methylprednisolone and valacyclovir was not superior to corticosteroid monotherapy. [77]

A retrospective cohort study was done in 28 patients with vestibular neuronitis. Outcome measures were caloric tests and questionnaires two years after onset. Result showed that canal improvement was 50% in the nonsteroid-treated group and 75% in the steroid-treated one. In cases with severe canal paresis (CP > or = 60%), canal improvement was 33% in the nonsteroid-treated group and 67% in the steroid-treated one. Steroid therapy at the acute stage of this disease significantly reduced the duration of spontaneous nystagmus and handicap in daily life due to dizziness induced by head and body movement, decreasing mood disturbance. (Kitahara T et al 2001) [78]

A COCHRANE meta-analysis looked at four trials, involving a total of 149 participants concluded that there is insufficient evidence from the trials to support the administration of corticosteroids to patients with vestibular neuronitis. All the trials were small and of low methodological quality.

However, it was still found in this review that there is an overall significant effect of corticosteroids compared with placebo medication on complete caloric recovery at one month; but they stated that no significant effect was seen on complete caloric recovery at 12 months; or on the extent of caloric recovery at either one month or at 12 months. In addition, there was no significant difference between corticosteroids and placebo medication in the symptomatic recovery of vestibular function following idiopathic acute vestibular dysfunction with respect to vertigo at 24 hours and use of the Dizziness Handicap Inventory score at one, three, six and 12 months. [79]

In a prospective controlled trial of 30 vestibular neuronitis patients, 15 in the study and 15 in the control group, the study group was treated by 1 mg/kg prednisone for five days, followed by gradually reduced doses of prednisone for an additional 15 days, and vestibular sedatives for symptomatic relief during the first five days after presentation. The control group received a placebo and similar vestibular sedatives. The patients had a baseline evaluation and follow-up examinations after 1, 3, 6, and 12 months. The groups were compared for the presence of symptoms and signs, caloric lateralization on the electronystagmography (ENG), the presence of other pathologic findings in the ENG, and Dizziness Handicap Inventory scores. Results showed no differences between the groups in the occurrence of symptoms and signs, degree of caloric lateralization, presence of other ENG pathologic findings, and Dizziness Handicap Inventory scores at the end of the study. Complete resolution was observed in 64% of the study and in 80% of the control group. The study group showed earlier recovery of ENG lateralization at the 1- and 3-month follow-up evaluations and higher rates of complete resolution at the 3- and 6-month follow-up points. The study concluded that prednisone therapy might enhance earlier recovery but does not improve the long-term prognosis of VN. The clinical and laboratory parameters in VN are not correlated, and both are required for complete patient evaluation. [70]

The proportion of patients with caloric complete recovery was significantly different between the corticosteroids and placebo groups both at 1 (OR, 12.64; 95% CI, 2.6-61.52; p = 0.002; heterogeneity, p = 0.53; fixed effects model) and 12 months (OR, 3.35; 95% CI, 1.45-7.76; p = 0.005; heterogeneity, p = 0.03; random effects model) after the initiation of therapy.

The caloric extent of canal paresis at 12 months after the initiation of therapy seemed to differ significantly between patients who received corticosteroids and those who received placebo.

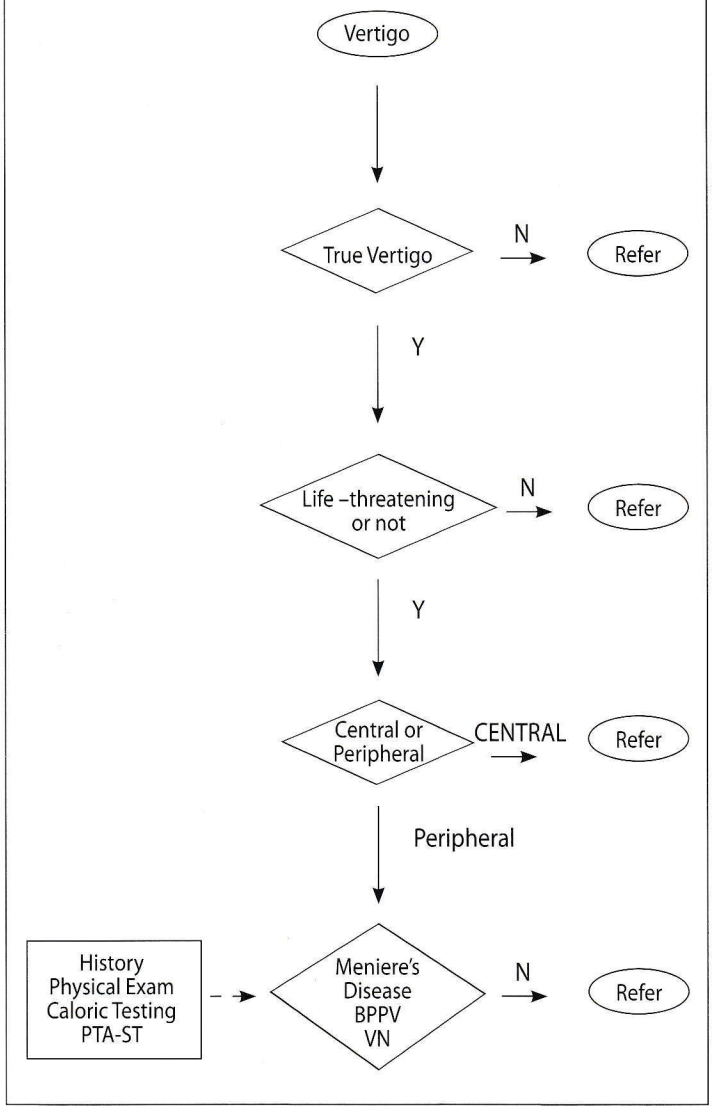
The study of Goudakos et al 2010 where three studies were meta-analyzed, and the conclusion was that corticosteroids improve the caloric extent and recovery of canal paresis of patients with vestibular neuritis. However, they stated that clinical recovery does not seem be better in patients receiving corticosteroids. [80]

5. Antiviral medication should not be included in the treatment of vestibular neuronitis.

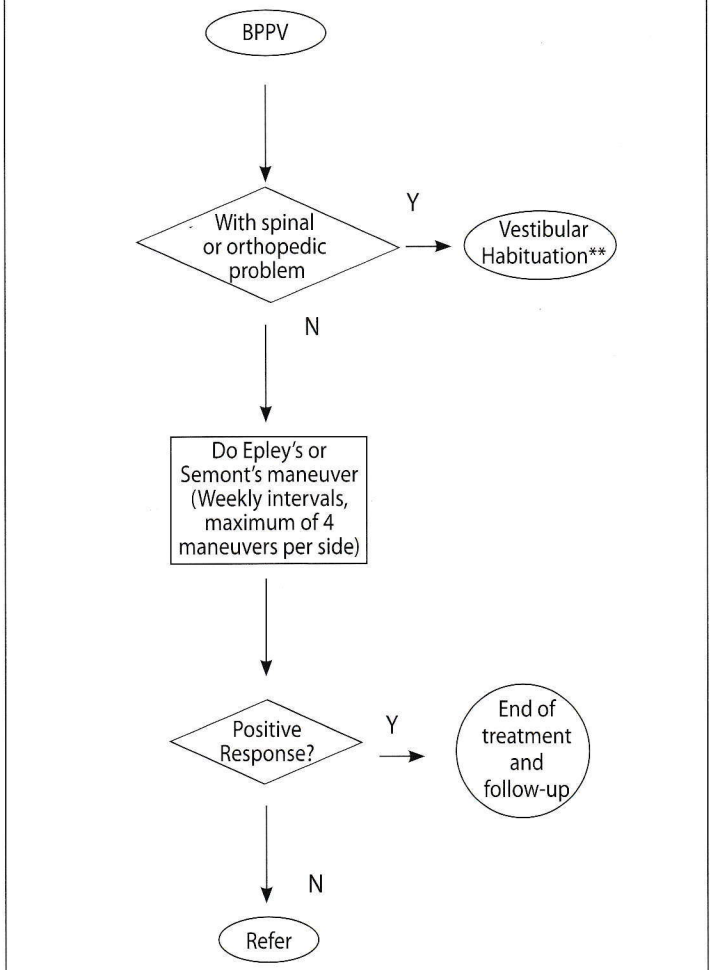
Grade B Recommendation, Level 2B Evidence

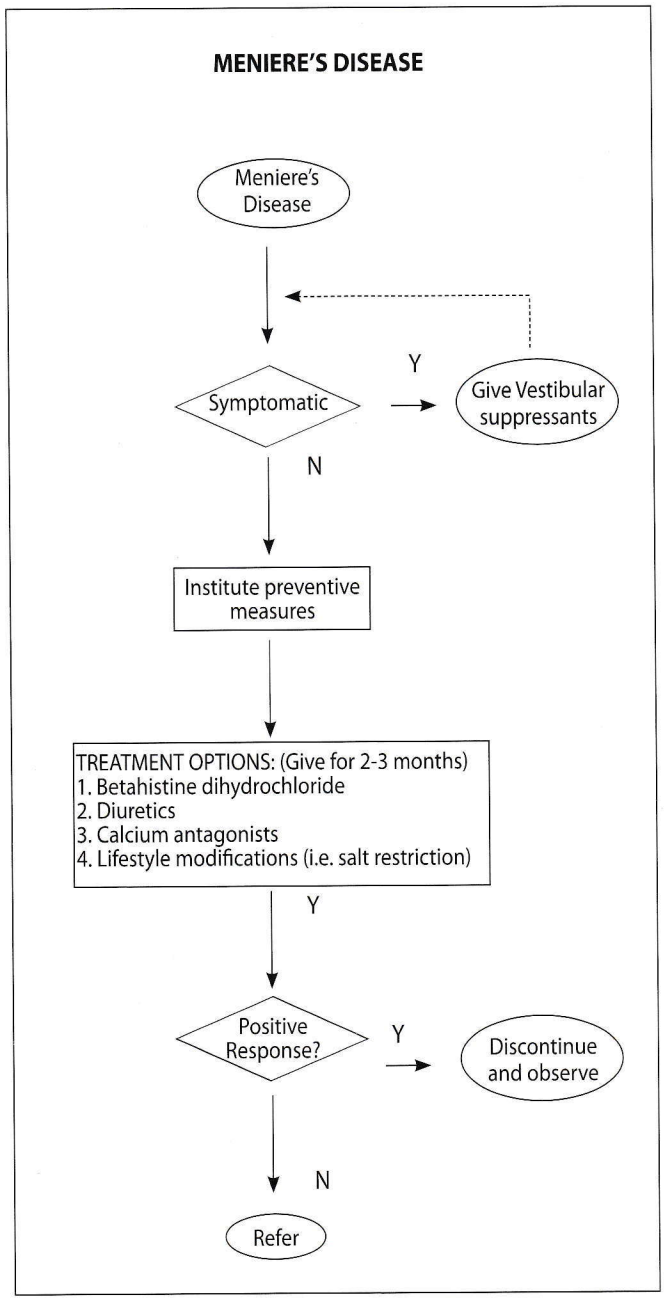
The study by Strupp et al (2004) provided no additional benefit with antivirals compared to steroids alone. [77]

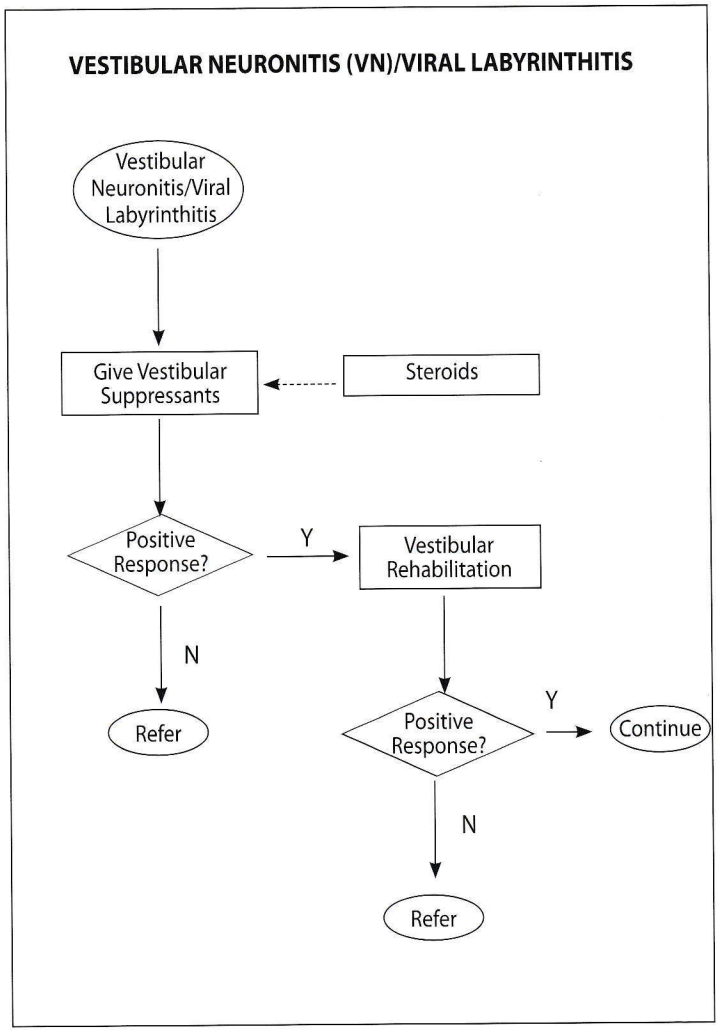
APPROACH TO A PATIENT WITH VERTIGO



BENIGN PAROXYSMAL POSITIONAL VERTIGO (BPPV)




MENIERE'S DISEASE
Meniere's Disease
Symptomatic
Y
Give Vestibular suppressants
N
Institute preventive measures
TREATMENT OPTIONS: (Give for 2-3 months)
1. Betahistine dihydrochloride
2. Diuretics
3. Calcium antagonists
4. Lifestyle modifications (i.e. salt restriction)
Y
Positive Response?
Y
Discontinue and observe
N
Refer


VESTIBULAR NEURONITIS (VN)/VIRAL LABYRINTHITIS
Vestibular Neuronitis/Viral Labyrinthitis
Give Vestibular Suppressants
Steroids
Positive Response?
Y
Vestibular Rehabilitation
N
Refer
Positive Response?
Y
Continue
N
Refer

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Betahistine dihydrochloride

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PRESCRIBING INFORMATION

Formulation: Each tablet contains Betahistine dihydrochloride 24mg. **Indications:** Meniere's Disease, Meniere-like syndrome (with symptoms of vertigo, tinnitus, and sensorineural deafness) and vertigo of peripheral origin. **Dosage:** 1 tablet BID. **Contraindications:** Hypersensitivity to any of the components of the product. **Warnings and Special Precautions for Use:** Patients suffering from pheochromocytoma and patients with bronchial asthma need to be carefully monitored during therapy. Caution should be taken in the treatment of patients with history of peptic ulcer. **Adverse Effects:** Mild gastric complaints have been reported which can be dealt with by taking the dose during meals or by lowering the dose. **Storage:** Store at temperatures not exceeding 30°C.

Full prescribing information is available upon request.

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