Management of Peripheral **Vestibular Disorders** in Primary Care

Updated 2025

CONTENTS

Expert Committee 3

Foreword 4

Introduction 5

Diagnosis 7

- How to Address a Dizzy Patient 10

Management 12

- Pharmacotherapy 12
- Surgical Management 14
- Particle Repositioning Manoeuvres 14
- Manoeuvres in Special Conditions 26
- Vestibular Rehabilitation Therapy 28

Paediatric Vertigo 32

Dizziness in the Elderly 34

Recurrent Dizziness 36

Drugs 37

- Symptomatic 37
- Disease Specific 40

When to Refer to a Specialist 44

Appendix 45

- Appendix 1 – ICVD Secondary Vestibular Symptom Definitions 45
  - » 1.A – Spontaneous and Triggered Vertigo 45
  - » 1.B – Vestibulovisual Symptoms 46
  - » 1.C – Postural Symptoms 46
- Appendix 2 – Bárány Society Diagnostic Criteria 47
- Appendix 3 – MALPIP Screening Tool for Older Adults 51

References 53

Acknowledgements 56

EXPERT COMMITTEE

The development of this guide was made possible by the active participation and contribution of all members of the Expert Committee.

Committee Chair

Dato' Dr Philip Rajan Devesahayam
Consultant Otorhinolaryngologist
Hospital Raja Permaisuri Bainun, Perak

Committee Members

Dato' Professor Dr Prepageran Narayanan
Consultant Otorhinolaryngologist
Faculty of Medicine,
Universiti Malaya, Kuala Lumpur

Dr Carren Teh
Consultant Otorhinolaryngologist
Hospital Sungai Buloh, Selangor

Dr Fadzilah Ismail
Consultant Otorhinolaryngologist
Hospital Sungai Buloh, Selangor

Associate Professor Dr Noor Dina Hashim
Consultant Otorhinolaryngologist
Faculty of Medicine,
Universiti Kebangsaan Malaysia,
Kuala Lumpur

Dr Rosdan Salim
Consultant Otorhinolaryngologist
Universiti Sains Malaysia, Kota Bharu,
Kelantan

Professor Dr Mazlina Mazlan
Consultant Rehabilitation Physician
Faculty of Medicine,
Universiti Malaya, Kuala Lumpur

Professor Dr Tang Ing Ping
Consultant Otorhinolaryngologist
Universiti Malaysia Sarawak,
Sarawak General Hospital, Sarawak

Dr Sakina Mohd Saad
Consultant Otorhinolaryngologist
Hospital Sultan Abdul Halim, Kedah

Dr Jeyasakthy Saniasiaya
Consultant Paediatric Otorhinolaryngologist
Faculty of Medicine,
Universiti Malaya, Kuala Lumpur

Dr David Chang Chee Tao
Research Pharmacist
Clinical Research Centre Perak,
Hospital Raja Permaisuri Bainun, Perak

FOREWORD

The update of this guide is timely as progress in scientific research and new medical knowledge continue to give us a better understanding of dizziness, vestibular disorders and their management. A panel of experts comprising otorhinolaryngologists, rehabilitation physicians and researchers have assembled to provide relevant and updated information to assist primary care doctors in the diagnosis and management of patients with dizziness, particularly peripheral vestibular disorders which form a significant proportion of dizzy patients. I would like to take this opportunity to congratulate Dato' Dr Philip Rajan and all members of the expert panel for their tireless contribution in updating this guide.

Professor Emeritus Dato' Dr Lokman Saim
President (2024–2025)
College of Otorhinolaryngologists,
Head and Neck Surgeons,
Academy of Medicine of Malaysia (CORLAMM)

INTRODUCTION

Dizziness is a common condition encountered by physicians in a variety of specialties. It is estimated to account for 5 percent of primary care clinic visits**.** The burden of this problem is expected to increase in Malaysia as we head towards an ageing society, as many common causes of dizziness such as benign paroxysmal positional vertigo, orthostatic hypotension, Parkinson's disease and diabetic neuropathy affect older patients. **[1]**

This guide has been prepared to assist the primary care physician in assessing the dizzy patient; from relevant history, clinical examination and appropriate tests to help diagnose common causes of dizziness. Common pharmacologic agents used in the management of vestibular disorders are described. Potential side-effects and drug interactions are also highlighted. In addition, common particle repositioning manoeuvres for benign paroxysmal positional vertigo (BPPV) are described.

According to a Dutch study, the one-year prevalence of dizziness in the elderly patients aged 65 years or older was 8.3 percent. **[2]** Symptomatic dizziness has been associated with an increased risk of mortality and morbidity, as well as a significant impact on quality of life (i.e., problems with dizziness, balance and falls), and this is where correct and adequate treatment is important. **[3]**

Dizziness is a generic symptom which includes vertigo, disequilibrium, presyncope and light-headedness. A detailed history is required to specifically elicit the nature of the patients presenting with a complaint of dizziness. The main categories of dizziness are summarized in **Table 1**. **[4]**

Malaysian studies report that:

- 46.7 percent of Malaysians aged 60 years and above suffer from vestibular impairment. **[5]**
- There is a higher prevalence of vestibular disorders in women than men in Malaysia. Previous studies have linked vestibular disorders to female health-related conditions such as migraine and pre-menstrual syndrome. **[6]**
- The older population in Malaysia do not seek assistance for their vestibular-related problems due to low awareness of available vestibular clinics in Malaysia. **[6]**
- In patients who presented with dizziness, approximately 66 percent were caused by peripheral vestibular causes, the majority of which 31 percent was BPPV. **[7,8]**
- A study in a tertiary centre in Malaysia showed that BPPV was the most prevalent diagnosis, followed by Ménière's Disease. **[6]**

Category	Description	Percentage of patients with dizziness
Vertigo	False sense of motion, possible spinning sensation	45 to 54
Disequilibrium	Off-balance and wobbly	Up to 16
Presyncope	Feeling of losing consciousness or blacking out	Up to 14
Light-headedness	Vague symptoms, possibly feeling disconnected with the environment	Approximately 10

Table 1. Main Categories of Dizziness
Adapted from Post RE and Dickerson LM. Am Fam Physician 2010;82(4):361-8.

Vestibular disorders (Table 2) are a leading cause of dizziness and consists of peripheral (inner ear and vestibular nerve) and central (brainstem and central nervous system) disorders. **[9]** The International Classification of Vestibular Disorders (ICVD) definitions of secondary vestibular symptoms can be viewed in **Appendix 1. [10]**

Vestibular Syndromes	Diseases and disorders
Acute Vestibular Syndrome	Vestibular neuritis, Acute stroke
Episodic Vestibular Syndrome	Ménière's disease, Vestibular migraine, Transient Ischemic Attack (TIA)
Chronic Vestibular Syndrome	Bilateral vestibular failure, Cerebellar degeneration

Table 2. Vestibular Syndromes Based on Duration
Adapted from Bisdorff AR, et al. Neurol Clin 2015;33:541-50.

DIAGNOSIS

Acute dizziness is frequently reported in primary care and emergency departments. **[11,12]** The primary focus of care should be prompt identification of any potential cerebrovascular events to initiate emergency management. Once such events have been ruled out, it is then essential to establish a precise diagnosis to enable targeted management strategies. **[13]**

The following chart **(Figure 1)** is a quick guide to help primary care practitioners better understand the different possible causes of dizziness.

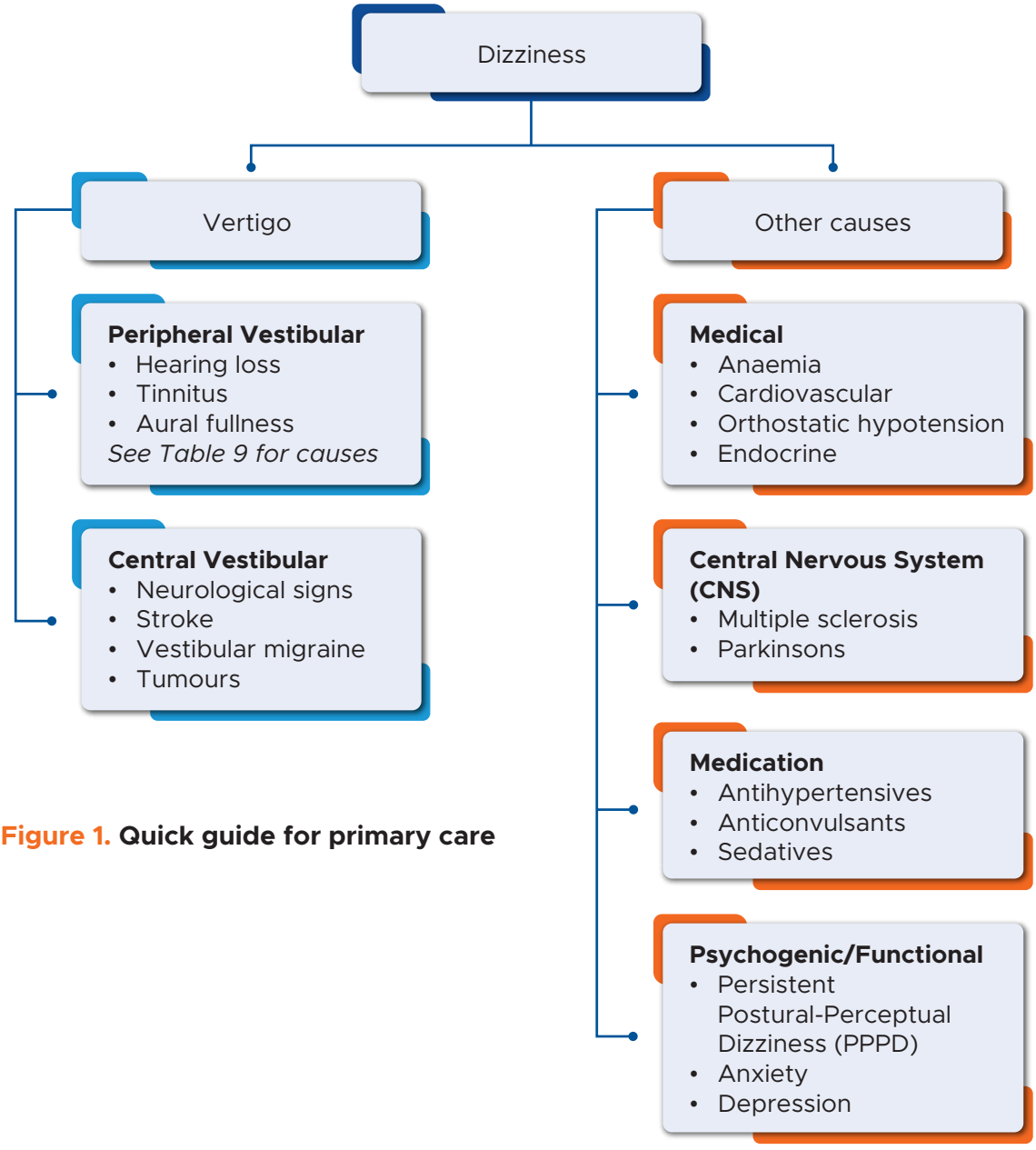


Figure 1. Quick guide for primary care

Figure 2 is a **simple algorithm** that can aid in deriving a diagnosis efficiently through careful history-taking which begins with identifying if the symptoms are vertiginous in nature or not. If the complaint was that of acute vertigo, central symptoms such as numbness, ataxia, speech difficulties, central nystagmus, skew deviation, double vision, new headache or inability to walk should be investigated. **BEFAST** which stands for **B**alance (loss of balance), **E**yes (sudden loss of vision or double vision), **F**ace (facial asymmetry), **A**rms (numbness or weakness), **S**peech difficulties, **T**ime to call for help; is often used to recognize the signs of stroke. **[14,15]** A targeted examination should be performed to rule out any cerebrovascular event including **HINTS**. **[13]**

A central **HINTS** sign was defined as present if one or more of the following clinical signs were detected: normal head impulse test, direction-changing nystagmus, and/or skew deviation in presence of a spontaneous nystagmus. **HINTS (Table 3)** is an acronym for the three tests included **[16]**:

Examination	How to perform this test	Peripheral Vertigo	Central Vertigo
Head **I**mpulse Test (**HI**-)	Fast, low amplitude (10–15°) head rotations to the left or right while the patient is looking at a fixed target (e.g., the doctor's nose)	Abnormal, corrective saccade to midline with quick rotation of head	Normal, no corrective saccade
Nystagmus (-**N**-)	Fixation on an object (e.g., tip of a pen) during lateral gaze (~20 to 30°) for at least 5 seconds	Unidirectional horizontal	Horizontal & direction changing, vertical, torsional
Test of **S**kew (-**TS**)	Rapid covering then uncovering of one eye after the other while the patient is looking at a fixed target in space (e.g., the doctor's nose). The examiner should focus on only one eye	None	Present

Table 3. Head Impulse, Nystagmus, Test of Skew (HINTS) Examination

Infarction of the territory of Anterior Inferior Cerebellar Artery (AICA) may result in a labyrinthine infarction and the HINTS can misdirect the diagnosis toward a peripheral vestibular disorder. By assessing for hearing loss, **HINTS Plus examination** increases the sensitivity of diagnosing a cerebrovascular event. **[17]**

HINTS Plus hearing loss was introduced when it was found that a patient with cochlear or brainstem infarction may present with HINTS that indicated peripheral vertigo. **HINTS Plus hearing loss** could increase the diagnostic accuracy of acute vestibular syndrome. **[17]**

If this is negative, further history and a targeted examination should be attained to derive a peripheral vestibular cause as shown in the algorithm below (**Figure 2**). The standardised diagnostic criteria of many vestibular diagnoses are listed in **Appendix 2**.

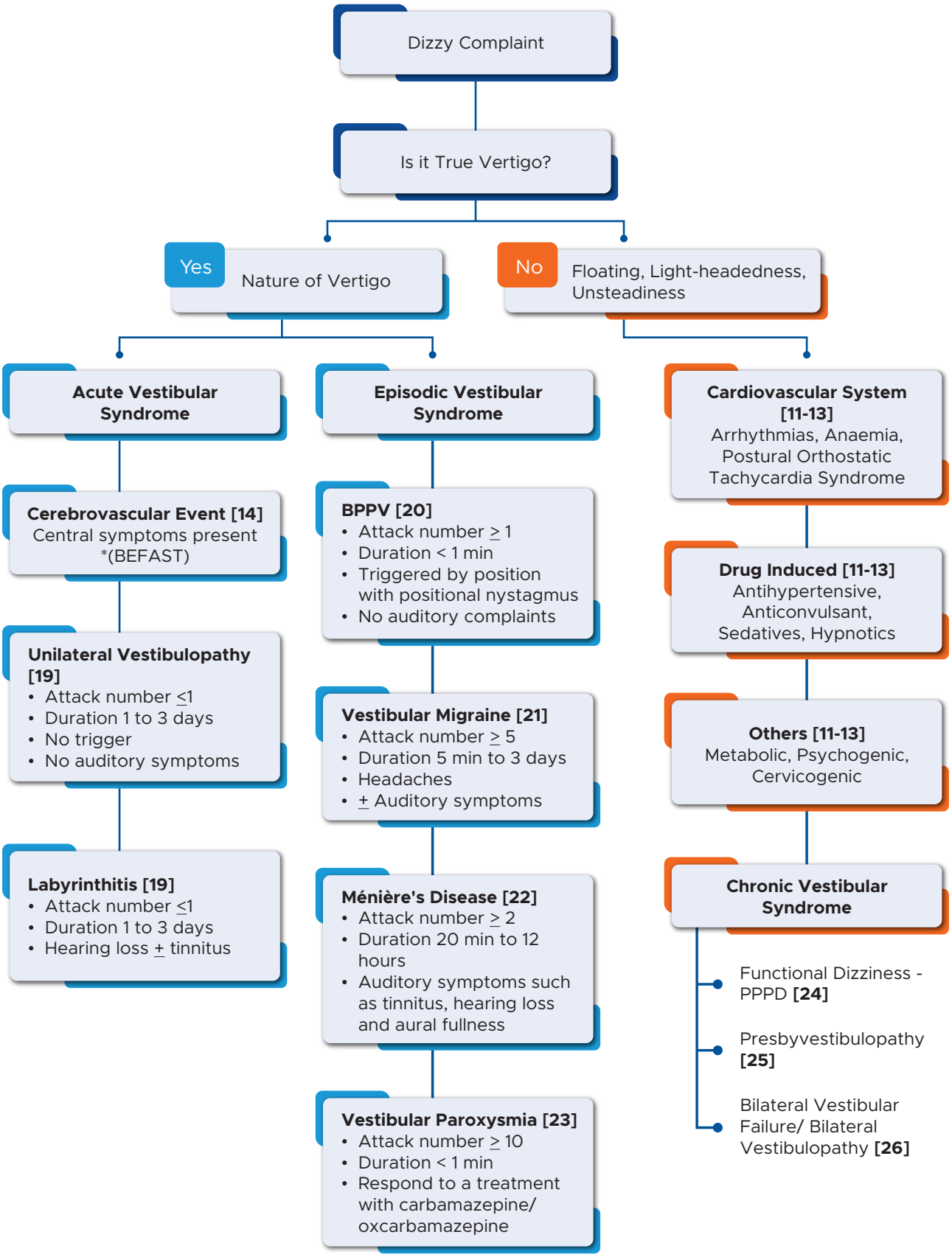
The common causes of peripheral vertigo are summarised in **Table 4**, and the less common causes are shown in **Figure 3**. **[18]**

	Acute	Episodic			
	Vestibular Neuritis	**Benign Paroxysmal Positional Vertigo**	**Ménière's Disease**	**Recurrent Vestibulopathy**	**Vestibular Paroxysmia**
Duration	Days-to-weeks	Seconds	Minutes to hours	Minutes to hours	Minutes
Hearing	No hearing loss	No hearing loss	Hearing loss, tinnitus and aural fullness	No hearing loss	Usually normal
Trigger	Preceding upper respiratory tract infection	Change in head position	Spontaneous	Spontaneous	Spontaneous
Aetiology	Viral infection	Otolith in semicircular canals	Endolymphatic hydrops	Vestibular nerve dysfunction	Vestibuloco-chlear nerve compression or irritation
Nystagmus	Spontaneous horizontal nystagmus	Brief rotatory nystagmus on positional manoeuvre	Spontaneous horizontal nystagmus	Spontaneous horizontal nystagmus	Absent or Spontaneous horizontal / rotatory

Table 4. Common Causes of Peripheral Vertigo

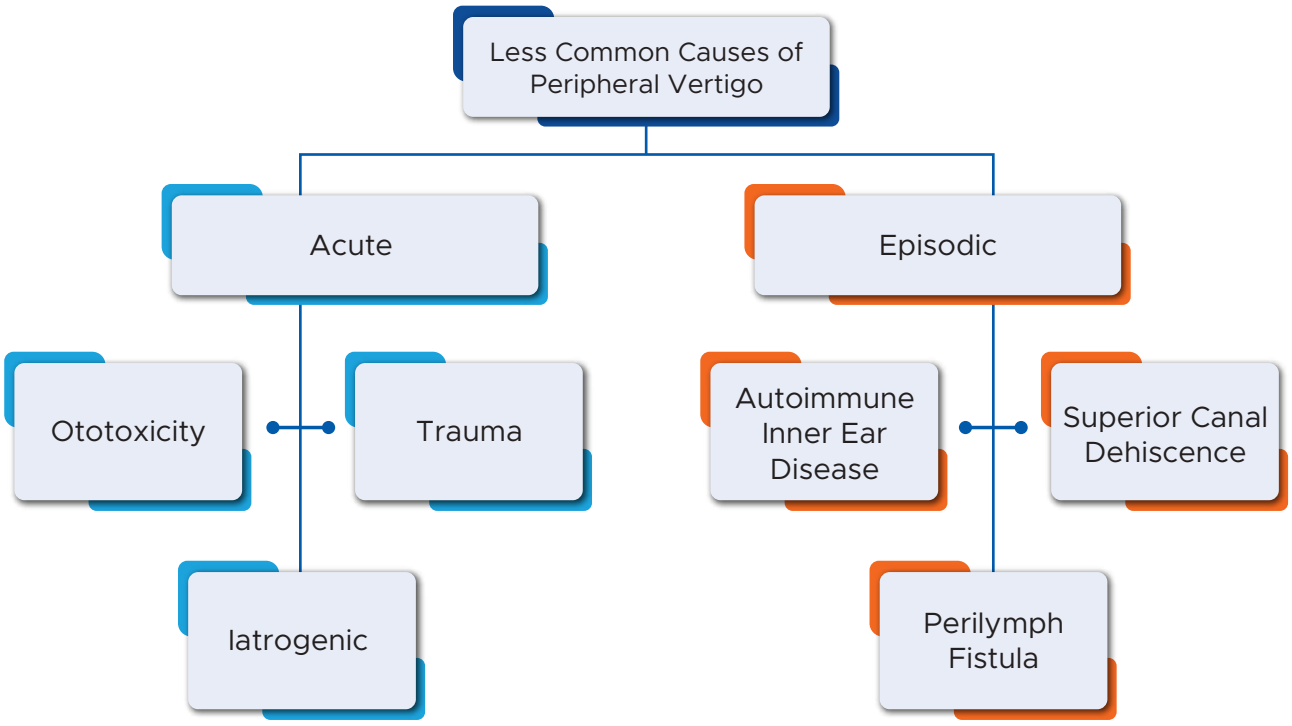
How To Address A Dizzy Patient?

Figure 2. Algorithm to Guide Diagnosis of a Dizzy Patient



*Balance (loss of balance), Eyes (sudden loss of vision or double vision), Face (facial asymmetry), Arms (numbness or weakness), Speech difficulties, Time to call for help

Figure 3. **Less Common Causes of Peripheral Vertigo**



MANAGEMENT

Pharmacotherapy

The options of pharmacotherapy **[27-30]** in peripheral vestibular disorders include symptomatic treatment and disease-specific approaches. The goal of symptomatic treatment is control of acute symptoms and autonomic complaints, such as vertigo and vomiting whereas a disease-specific approach involves treating the possible underlying cause of the vertigo.

For guidance on when to refer patients to specialists, please refer to the '**When to Refer to A Specialist**' chapter of this booklet on **page 44**.

Symptomatic Control

1. Vestibular Suppressants

This group of pharmacotherapies include benzodiazepines, antihistamines and anticholinergics. These are drugs that reduce the intensity of attacks of vertigo and nystagmus evoked by vestibular disturbances, as well as associated motion sickness. They are best prescribed and restricted for acute episodes. Prolonged use is discouraged since it may slow down the process of central vestibular compensation. **[9]**

2. Antiemetics

The vestibular and vomiting center in the brain is strongly connected. Stimulation of the vestibular system either by movement or vertigo activates the vomiting center leading to nausea and vomiting which can be more debilitating than the vertigo itself. Other autonomic symptoms that can be present include salivation, pallor, abdominal cramps and diarrhoea. Vestibular suppressants and antiemetics works hand in hand to either reduce or eliminate these symptoms.

Disease-Specific Treatment

1. Vestibular Neuritis

Corticosteroid treatment may reduce inflammation and improve the recovery of the affected peripheral vestibular nerve. **[31,32]** Antivirals provide no benefit in treating this condition even though viral infection is believed to cause this. Symptomatic treatment should be prescribed in the first few days to alleviate acute symptoms and discontinued once controlled. **[33]**

2. Ménière's Disease

The aim of pharmacological treatment is to address the acute episodes of vertiginous attacks, prevent new attacks and treat audio-vestibular dysfunction. Patients are advised to follow strict dietary salt restriction, adequate hydration, avoid caffeine, control blood sugar and stop smoking. Betahistine has been shown to improve blood flow to the inner ear through vasodilation and increased vascular permeability, therefore helping to relieve inner ear pressure. Betahistine has also been shown to have a significant effect on the frequency of the attacks. As betahistine does not suppress CNS function, vestibular compensation is thus not jeopardized. **[34-38]** A mild diuretic such as hydrochlorothiazide-triamterene helps to reduce the frequency of attacks.

3. Vestibular Migraine

A holistic, multidisciplinary treatment strategy that includes medications, lifestyle modifications, vestibular rehabilitation, trigger avoidance as well as addressing comorbidities is the most effective method for treating vestibular migraine. For reduction of occurrence, the prophylactic treatment follows the migraine headache protocols which include antidepressants, beta blockers, calcium-channel blockers and anticonvulsants **(Figure 4)**. Vestibular suppressants, antiemetics, non-steroidal anti-inflammatory drugs (NSAIDs) can be deployed in acute attacks as monotherapy or in combination. Regular exercise, stress reduction, intake of regular meals and good sleep hygiene should be practiced. Limiting one's consumption of caffeine, alcohol, monosodium glutamate (MSG), and highly processed foods is one way to avoid triggers. Emerging research suggests that vestibular migraine may also benefit from neuromodulation, botulinum toxin, and calcitonin gene-related peptide antagonists. **[36]**

4. Vestibular Paroxysmia

Vestibular paroxysmia is believed to be caused by the neurovascular compression of the cochleovestibular nerve. Vestibular suppressants are not effective in this condition. Clinically, the irregular, unpredictable attacks have been shown to respond well to anticonvulsants (such as oxcarbamazepine or carbamazepine) **[23]** which is helpful to confirm the diagnosis.

5. Recurrent Vestibulopathy

Recurrent vestibulopathy is a clinical syndrome that is similar to Ménière's disease which consists of episodic vertigo lasting minutes to hours, without the auditory symptoms. The exact cause is unknown, but is believed to be due to the periodic reduction of afferent discharge activity of the vestibular nerve secondary to viral infection. Follow-up is required as a number of cases are eventually diagnosed as Ménière's disease or vestibular migraine. The majority of cases resolve with symptomatic management. **[39,40]**

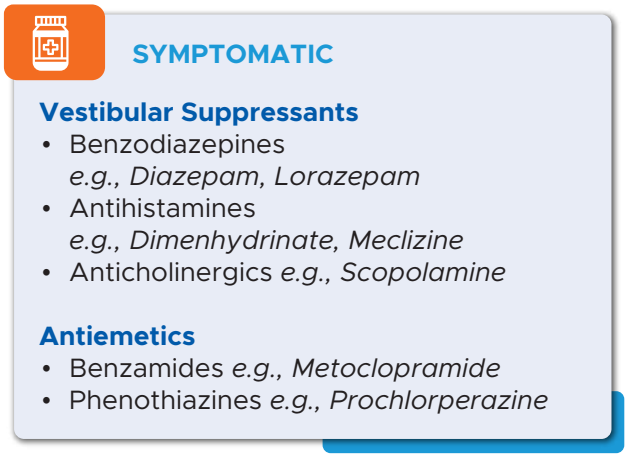


Figure 4. Pharmacotherapy in Peripheral Vestibular Disorders

Pharmacotherapy should not be overused, as any medication that inhibits the CNS will impair vestibular compensation. Except for betahistine, most drugs that are used to eliminate vertigo and its associated acute symptoms can result in CNS depression, which can slow recovery particularly for permanent vestibular lesions like acoustic neuromas, and may even lead to addiction to some medications e.g., benzodiazepines. Therefore, these medications ought to be used exclusively and solely for the purpose of symptom relief for a maximum of three to five days.

Nevertheless, numerous unconventional substances have been marketed to produce noticeable curative qualities, many of which have not been proven to be particularly effective. These substances accelerate vestibular compensation, more apparently in Ménière's disease.

Some alternative treatments such as Gingko Biloba extract (EGb 761) have also been shown to be effective in the management of vertigo. Gingko Biloba contains a mild antiplatelet compound which has been shown to reduce vascular resistance and improve peripheral blood circulation in animal models. Current evidence supports the use of Gingko Biloba extract at higher dosages for medical acceleration of vestibular compensation in surgical or disease-related acute vestibular loss. **[41]**

Surgical Management

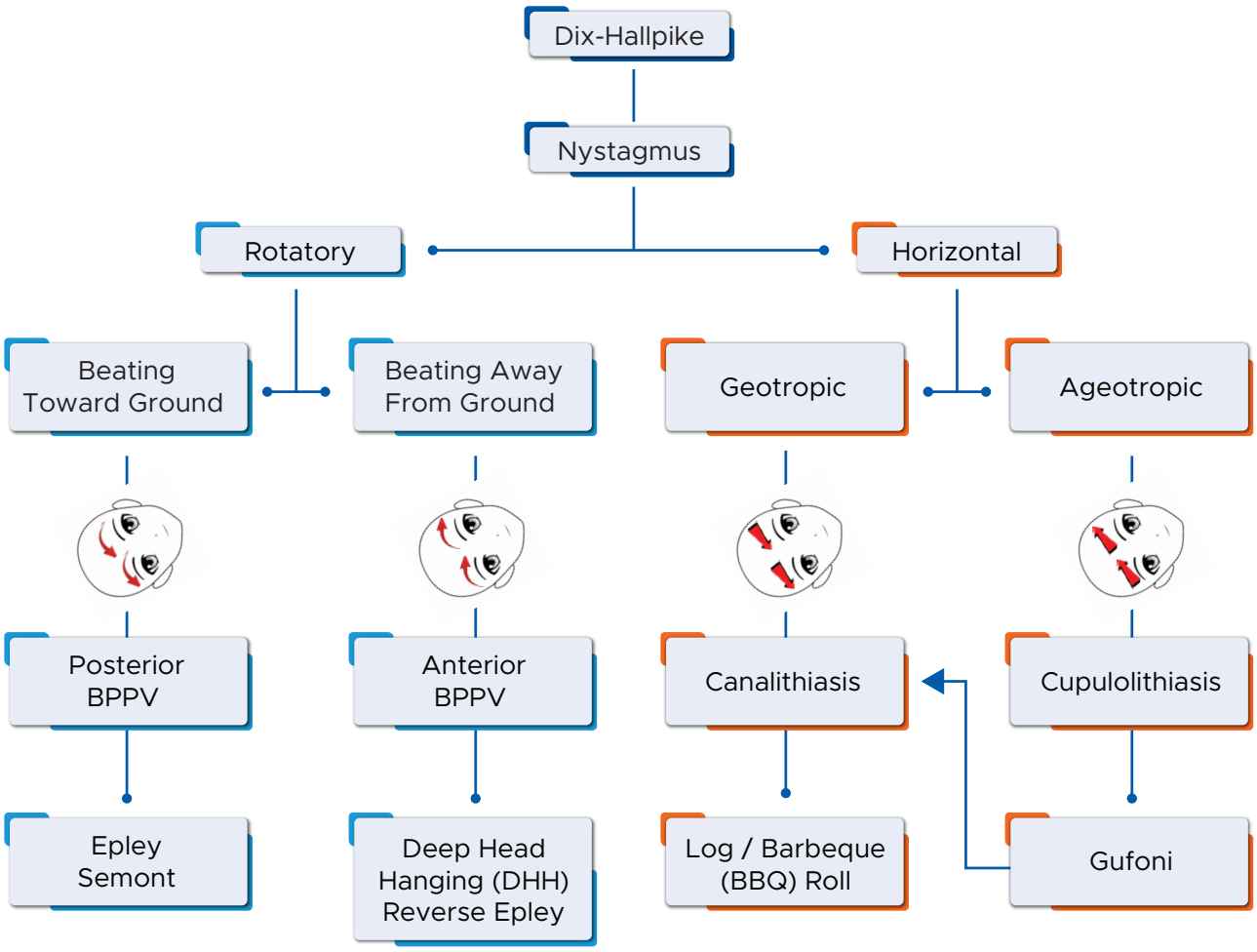
The majority of patients with vertigo have a good prognosis and are treated conservatively. Surgical therapy is seldom required and usually only reserved for patients who have not responded to the common pharmacotherapies. A lengthy counselling on the possible risks, especially hearing loss following destructive vestibular procedures must be performed prior to committing to one.

Following a diagnosis of Ménière's disease, preoperative residual hearing status is one of the pre-determining factors when deciding on a suitable type of surgical procedure.

- Intratympanic gentamicin is an ablative, minimally invasive procedure.
- Endolymphatic sac decompression, neurectomy of the vestibular nerve and labyrinthectomy are other types of destructive procedures reserved for refractory conditions.
- If it is not possible to preserve hearing, a simultaneous cochlear implantation is an option to achieve a positive cumulative outcome of good vertigo control and hearing replacement. Vestibular implants are the current frontier in development for the restoration of the vestibular reflexes in particular the vestibulo-ocular reflexes, that may demonstrate a close-to-reality function in vertigo sufferers. **[42-44]**

Particle Repositioning Manoeuvres

Particle Repositioning Manoeuvres (PRM) are the main treatment for BPPV. They relocate the free-floating otolith from the semicircular canals into the utricle in the vestibule. Depending on the nature and the sites of the dislodged otolith, specific PRMs are used. **Figure 5** shows four most common presentations or types of BPPV and their preferred PRMs. **[45-52]**



*Although Dix-Hallpike may elicit horizontal nystagmus, the Supine Roll Test is a standard diagnostic test for horizontal canal (HC)-BPPV.

Figure 5. Benign Paroxysmal Positional Vertigo (BPPV) Algorithm

Table 5 summarises the types of BPPV, related diagnostic tests and manoeuvres that may be performed to reposition the otolith.

Types of BPPV	Diagnostic test	Repositioning Manoeuvre
Posterior Canal (PC-BPPV)	Dix-Hallpike	Epley Manoeuvre Semont Manoeuvre
Anterior Canal (AC-BPPV)	Dix-Hallpike	Deep Head Hanging (Yacovino) Manoeuvre **[53]**
Horizontal Canal (HC-BPPV)	Supine Roll Test	**Geotropic Horizontal Nystagmus** BBQ Roll / Log Roll / Lempert's Manoeuvre Appiani-Gufoni Manoeuvre Kurtzer's Hybrid Manoeuvre **Apogeotropic Horizontal Nystagmus** Casani Manoeuvre Kurtzer Hybrid Manoeuvre

Table 5. Types of BPPV, Diagnostic Tests and PRMs

1. Posterior Canal BPPV (PC-BPPV)

The PRMs for PC-BPPV are the **Epley Manoeuvre** and **Semont Manoeuvre**.

A. Epley Manoeuvre

A

Begin with patient sitting on the examination couch. This is the same position as for Dix-Hallpike testing.

B

Turn the patient's head toward affected side. Pause in this position for 30 seconds.

Then, lie the patient flat, as for Dix-Hallpike. This and all parts of Epley are undertaken slowly and smoothly. Support the head throughout.

C

The patient's head remains turned to affected side and is lowered about 30 degrees from the horizontal plane. Pause in this position for 30 seconds.

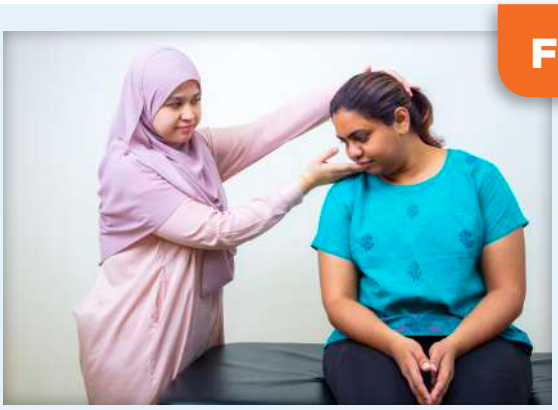
D

Turn the patient's head toward the good ear. This and all parts of Epley are undertaken slowly and smoothly. Pause in this position for 30 seconds.

E

While keeping the head in the same direction, ask the patient to gently move so that they are now lying on the hip and shoulder of the good side. Then, turn head toward the good ear – the patient should now be looking at floor, with chin close to shoulder. All parts of Epley are undertaken slowly and smoothly. Pause in this position for 30 seconds.

F



Gently bring patient to a sitting position. Ensure that head position does not change relative to trunk (chin still on shoulder of good side). This and all parts of Epley are undertaken slowly and smoothly. Pause in this position for 30 seconds.

G

Finally, turn head to centre and flex neck to put chin on chest in one movement. Pause in this position for 30 seconds.

Post-manoeuvre instructions:

- Patient should not drive home after Epley manoeuvre.
- Patient should avoid lying flat for the next two nights.
- For the following 5 nights, the patient should avoid lying on the affected side.
- Sleep on the unaffected side with a pillow behind the back to prevent rolling over.

B. Semont Manoeuvre

A

Begin with patient sitting at the edge of the examination couch.

B

Turn patient's head 45 degrees to the left.

C

The patient is then quickly brought into a right side-lying position, pause in this position for 1 minute.

D

The patient is then guided quickly from the right to the left side-lying positions within 1.5 seconds, without stopping in the centre. The head should be maintained in the initial 45 degrees of leftward rotation, so that at the end of the manoeuvre the patient is facing the table/ground. Pause in this position for 1 minute.

E

The patient is then slowly guided into a seated position, with the head maintained in 45 degrees of leftward rotation before finally...

F

...slowly moved back to the neutral (front-facing) position.

2. Anterior Canal BPPV (AC-BPPV)

The PRM for AC-BPPV is the **Deep Head Hanging Manoeuvre**.

A. Deep Head Hanging Manoeuvre

A

Begin with patient in the long-sitting position.

B

Lower the patient until their head is at least 30 degrees below the horizontal position with the head facing straight up. Hold in this position until the nystagmus induced by this step is over.

C

While the patient is still supine, bring the head up quickly to touch the chest. Pause in this position for 30 seconds.

D

While maintaining head flexion, bring the patient back up into a sitting position.

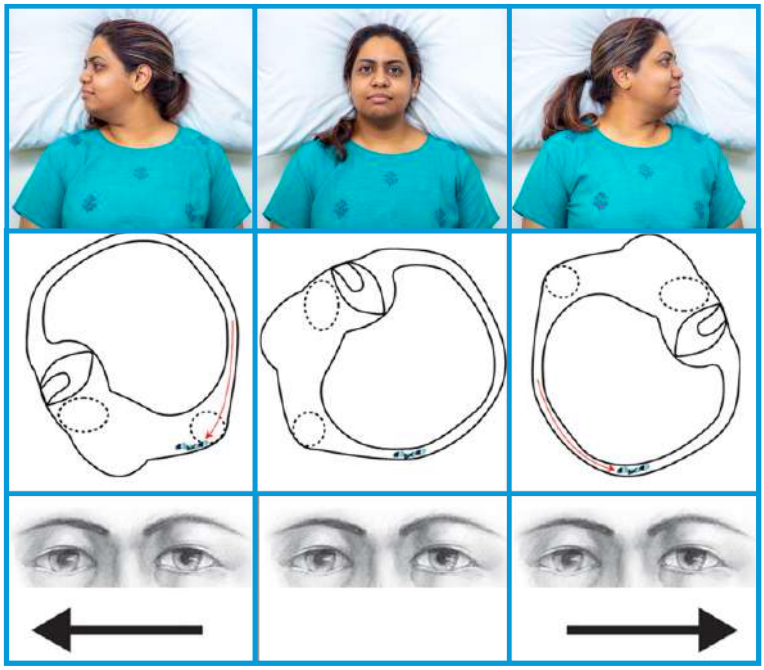
E

Bring the patient's head to a neutral position.

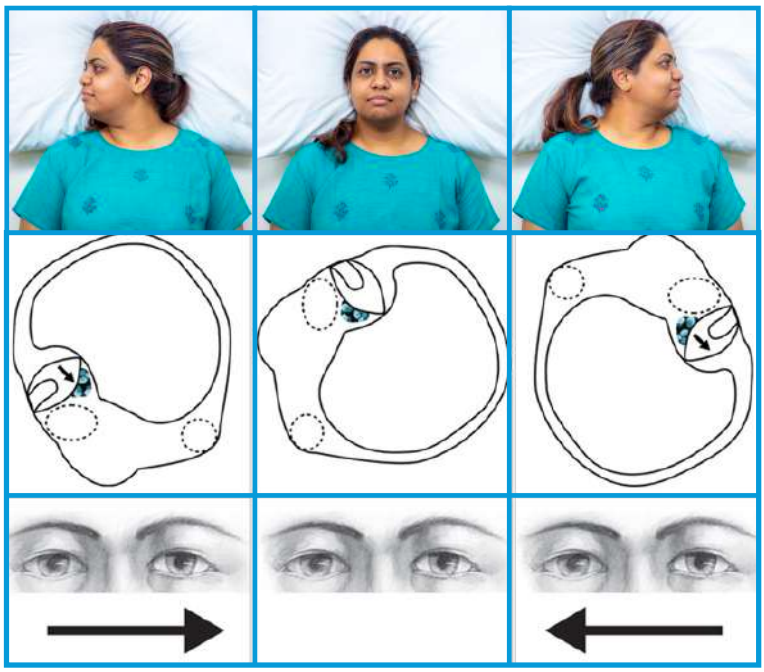
3. Horizontal Canal BPPV (HC-BPPV)

Supine Roll Test

- The Supine Roll Test (Pagnini-McClure manoeuvre) is used for the diagnosis of right HC-BPPV.
- While in a supine position, the head is turned about 90 degrees to each side.
  A. Canalolithiasis
  B. Cupulolithiasis
- The corresponding illustrations demonstrate the location of the otolithic debris in the horizontal canal during each manoeuvre and the direction of the induced nystagmus (arrows). **[50]**



A. Canalolithiasis



B. Cupulolithiasis

There are two known distinct subtypes of HC-BPPV based on the direction of horizontal nystagmus during supine head turns: geotropic and apogeotropic.

In geotropic HC-BPPV (fast phase of nystagmus beats toward the ground), nystagmus and symptoms will be worse on the affected side. Log Roll / Barbeque Roll toward the opposite side is the PRM of choice.

A. Log Roll Manoeuvre

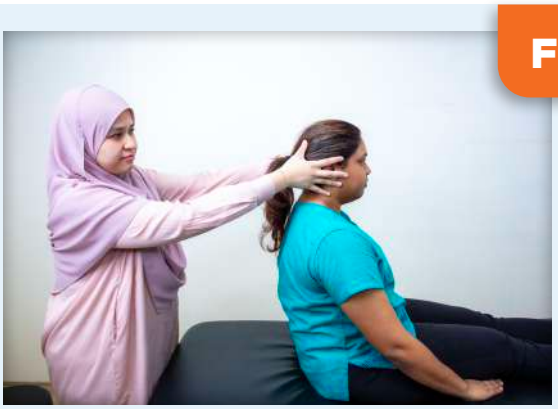
This procedure can be carried out on the examination couch or on the floor.

Begin by positioning the patient so that they are lying on their back with their head turned toward the affected ear (affected ear down).

Then, quickly turn the patient's head 90 degrees toward the unaffected side (face up).

A series of 90 degrees turns toward the unaffected side is then undertaken sequentially until the patient has turned 360 degrees...

...and is back to the affected ear-down position [E].

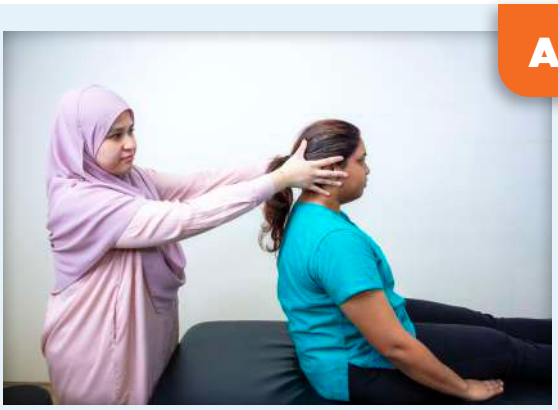


From here, the patient is turned to the face-up position and brought up to a sitting position.

The successive head turns can be done in 15- to 20-second intervals even while the nystagmus continues. Waiting longer does no harm, but may lead to the patient developing nausea, and the shorter interval does not appear to detract from the effectiveness of the treatment.

B. Barbeque (BBQ) Roll Manoeuvre

A



Begin with the patient in the long-sitting position.

B

Move the patient into a supine position with the head elevated 30 degrees.

C

Next, the patient's head (or whole body) should be rotated 90 degrees to the left side and maintained for 30 seconds or until the nystagmus and vertigo cease.

D

From (C) position, rotate the head back to the neutral position...

E

...then 90 degrees to the right side...

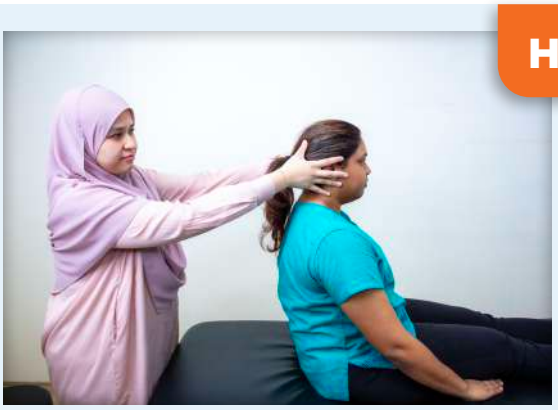
F

...into the prone position...

G

...and 90 degrees to the left side.

H



Finally return the patient to the long-sitting position.

Maintain each of these positions (D-H) for 30 seconds or until the nystagmus and vertigo cease.

In ageotropic/apogeotrophic HC-BPPV, nystagmus and symptoms will be worse on the opposite side. This is much more challenging to treat. One must convert ageotropic HC-BPPV to geotropic HC-BPPV to successfully treat this variant.

Converting ageotropic to geotropic BPPV can be attempted by performing a series of quick head turns from the affected side to the unaffected side. This is performed in an attempt to free or move the otoconia away from the cupula to the long arm of the canal.

C. Gufoni Manoeuvre

A

Begin with patient sitting at the edge of the examination couch.

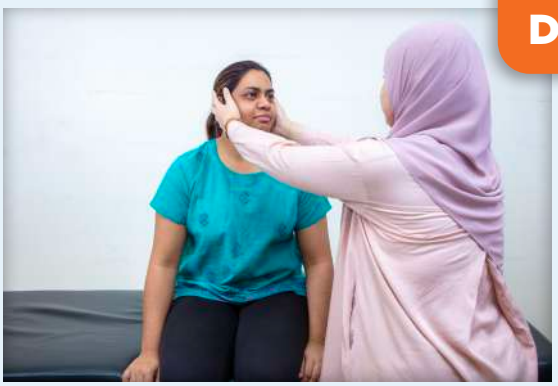
B

Bring the patient down rapidly into the side-lying position on the affected side. Remain in this position for 1 to 2 minutes until the nystagmus has stopped or markedly reduced.

C

Rapidly rotate the patient's head 45 degrees upward/toward the ceiling. Maintain this position for 2 to 3 minutes.

D

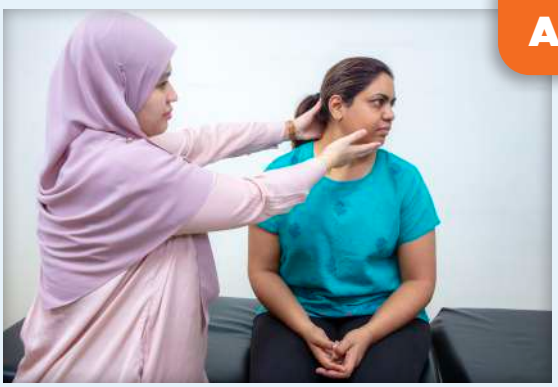


Slowly bring the patient back to the sitting position.

D. Kurtzer Hybrid Manoeuvre

The advantages of Kurtzer Hybrid Manoeuvre are that it can be performed in both geotrophic and ageotrophic HC-BPPV, without needing to know which side is the affected ear. **[51]**

A



Begin with the patient in a similar position to a Side-lying Hallpike test.

B

Then, with the head rotated to the left (30 degrees of neck flexion), ask the patient to lie on their right side. Stay in this position for 1 minute until any nystagmus subsides.

C

Rotate the head nose-down with 30 degrees of flexion and hold for 1 minute until any nystagmus subsides.

D

Then, rotate the patient's head (30 degrees of neck flexion) so that their nose is now facing up. Rotate their body to the opposite side and prepare the patient to repeat steps (B) and (C).

E

Once you have repeated the procedure on this side, rotate the patient's head nose-down with 30 degrees of flexion. Hold this position for 1 minute until any nystagmus subsides.

Manoeuvres in Special Conditions

These manoeuvres are intended for patients with difficulty lying flat on their backs, such as pregnant women, patients with low back pain, and those with obesity or arthritis.

A. Side-Lying Dix-Hallpike (Semont Manoeuvre) [54]

A

Begin with the patient seated in the middle of the examination couch, facing the clinician, with their legs hanging off the side of the examination couch.

B

Then the patient's head is turned 45 degrees away from the test ear.

C

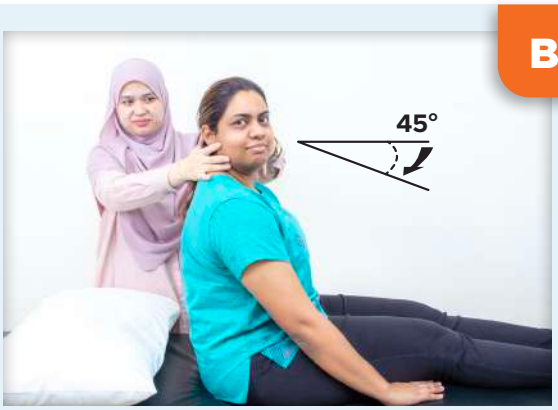
- Inform the patient to lie down so that the test ear is closest to the examination couch.
- While the patient is lying down on their side, instruct them to swing their legs up on the table so that the patient ends up lying completely horizontal on their side.
- Repeat the above process for the other ear.

B. Supported Dix-Hallpike

- This manoeuvre is appropriate for patients who are unable to tolerate the traditional and side-lying procedures.
- The procedure is similar to the traditional Dix-Hallpike; however, the patient's head is fully supported with pillow on the examination table and lowered about 30 degrees from the shoulder. **[48]**

A

B



C. Modified Epley Manoeuvre

This is a modification of the Epley Manoeuvre with a pillow under the shoulders. **[49]**

A

Begin with the patient sitting upright on an examination couch, then rotate the patient's head at a 45 degrees angle toward the affected side.

B

Quickly push the patient backward: the patient's upper body and head are reclined backward with the head extended 20-30 degrees below the pillow, resulting in a natural neck extension with the occiput resting on the surface of the table. Hold in this position for 30 seconds.

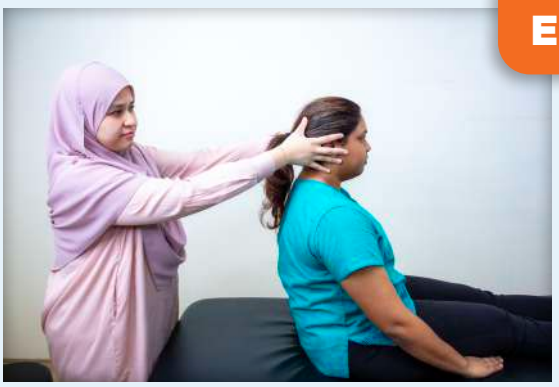
C

Rotate the patient's head 90 degrees in the opposite direction, while supporting and stabilising it with the surface of the table. Guide the direction and angle of the patient's position and hold for 30 seconds, until the dizziness stops.

D

Rotate the patient in the same direction another 90 degrees, with the unaffected side facing down. Hold the patient in this position for 30 seconds, until their dizziness stops.

E



Finally, bring the patient back up to a sitting position.

Vestibular Rehabilitation Therapy

Introduction

Vestibular Rehabilitation Therapy (VRT) for peripheral vestibular disorders is a specific form of physical therapy program aimed at reducing both primary and secondary symptoms of vestibular disorders. Common symptoms are vertigo, dizziness and imbalance. These symptoms can vary in intensity and may fluctuate or be persistent, depending on the underlying cause of the vestibular disorder. The primary goal of VRT is to promote vestibular adaptation through a series of exercises designed to reduce symptoms, optimizes the process of vestibular compensation and improve function.

Overview of VRT

VRT was developed due to the ability of the vestibular system to adjust and recover following injury or dysfunction. VRT uses special exercises that encourage the brain to use alternative ways to maintain balance and spatial orientation (ability to identify the body's position in space).

The primary goals of VRT

1. Reduce vertigo and dizziness
2. Improve balance and postural stability
3. Improve gaze stability
4. Prevent complications such as falls, fear and anxiety, and cognitive fatigue
5. Increase activity levels and functional independence

Core components of VRT

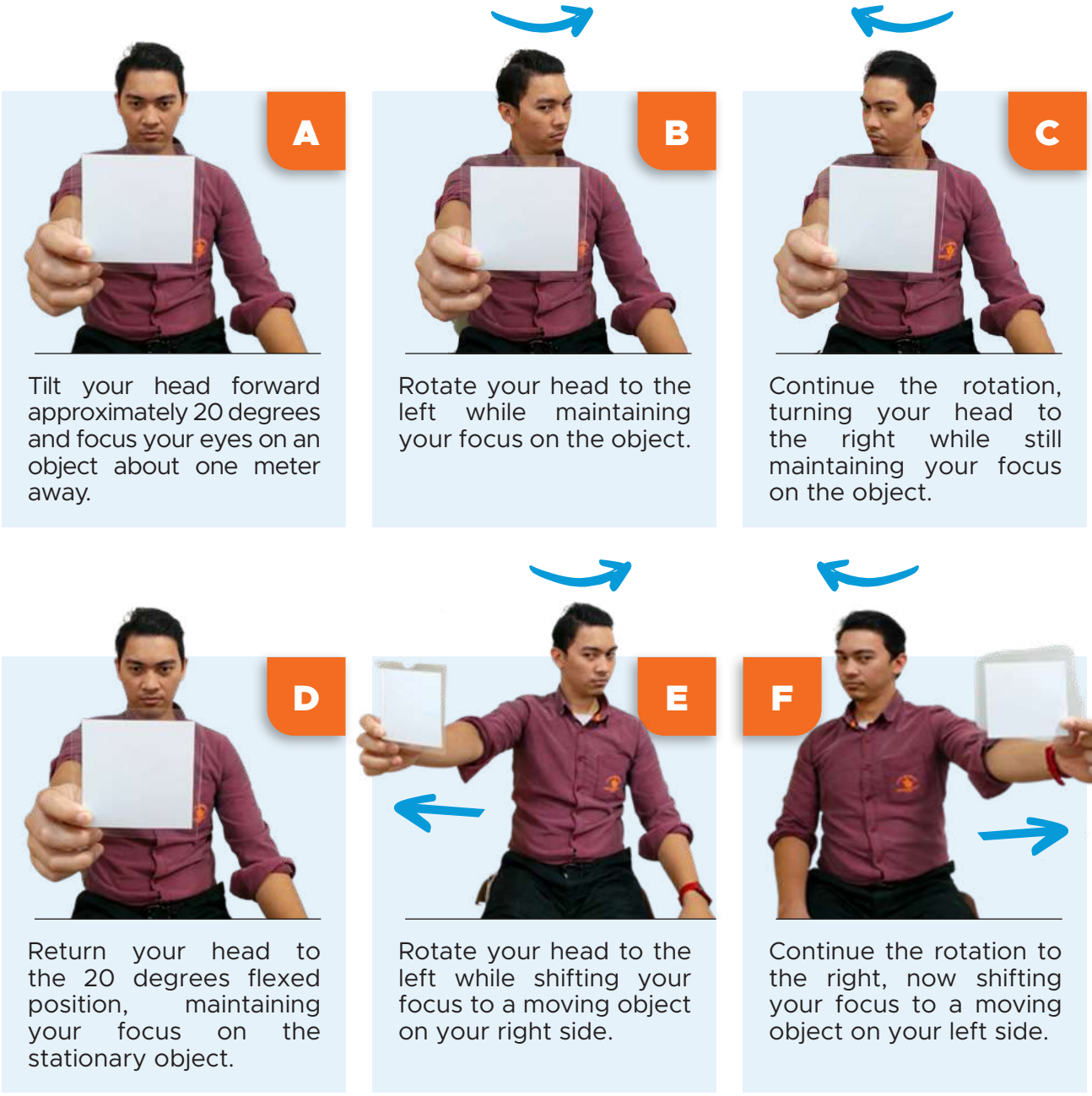
The core components of VRT include habituation exercises, gaze stabilisation exercise and balance/posture training.

1. **Habituation Exercises:** These are designed to reduce the unwarranted responses to head movement and visual stimuli. Patients are gradually and slowly exposed to movements and environments that trigger dizziness, allowing the brain to adjust and diminish symptoms over time.

2. **Gaze Stabilisation Exercises (Figure 6)**: These exercises aim to improve control of eye movements during head movements, thereby improving the ability to focus on a target while the head is in motion. They are useful for patients experiencing oscillopsia, where stationary objects appear to move. Picture A shows the examples of exercises that can be conducted at home.

3. **Balance and Posture Training (Figure 7)**: These exercises improve stability by challenging the balance system in progressively more difficult postures. They often include exercises during sitting, standing, walking on uneven surfaces, with eyes opened and eyes closed, or while performing dual tasks. Picture B shows the examples of standing balance training that can be conducted at home.

Figure 6. **Gaze Stabilisation Exercise**



With permission from the Physiotherapy Division, Department of Rehabilitation Medicine, Universiti Malaya Medical Centre, 2023.

Figure 7. Standing Balance Training

With permission from the Physiotherapy Division, Department of Rehabilitation Medicine, Universiti Malaya Medical Centre, 2023.

Additional Q&A

1. **Who is suitable for vestibular rehabilitation therapy?**
   Vestibular rehabilitation therapy is suitable for patients with acute, subacute and recurrent unilateral or bilateral vestibular hypofunction.

2. **Is it safe for a patient to perform the vestibular rehabilitation therapy at home?**
   Vestibular rehabilitation therapy is a safe and effective method to be performed at home, in the clinic or in combination. However, patients are advised to consult a doctor or a trained physiotherapist before starting the therapy at home. A supervised therapy may be initially required.

3. **How often should a patient repeat each gaze stabilisation exercise?**
   In general, for each exercise that involves head rotation, a patient should perform 10 head rotations continuously. This is considered one cycle. The patient can repeat for 3 to 5 cycles (30 to 50 head rotations per exercise). It is advisable to consult a trained physiotherapist before performing these exercises.

4. **When should one stop vestibular rehabilitation therapy?**
   Clinicians may use achievement of primary goals, resolution of symptoms, or plateau in progress as reasons for stopping the rehabilitation.

5. **Can virtual reality be incorporated into vestibular rehabilitation therapy?**
   Yes, recent studies have shown the effectiveness of incorporating virtual reality to achieve habituation and adaptation through a more motivated vestibular rehabilitation therapy.

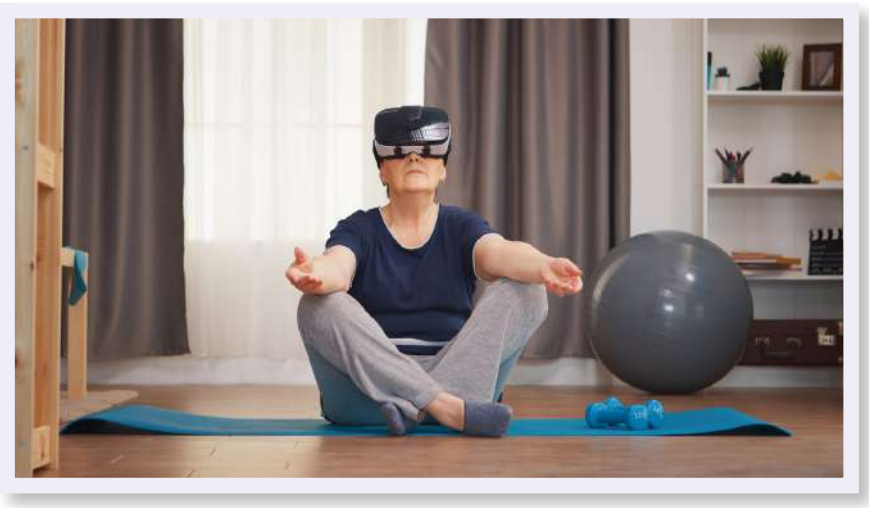


Figure 8.
A patient wearing virtual reality goggles during VRT exercises

Conclusion

VRT is an important intervention for people with balance and vestibular disorders. It uses specific exercises to assist the brain adapt and improve balance, reducing dizziness and helping people return to their daily lives. VRT is customizeable for different types of vestibular dysfunctions, making it very effective for recovery and functional improvement. As research continues to evolve, VRT remains a valuable area within rehabilitation medicine, offering hope and real benefits to those affected by vestibular disorders. **[55, 56]**

PAEDIATRIC VERTIGO

Introduction

The prevalence of vestibular disorders in children has been reported to be between 5% and 15% **[57]**, although the exact prevalence remains unknown due to the varying presentations. Unlike the adult population, identifying vestibular conditions in children is challenging due to several factors, including **[58]**:

1. Difficulty in verbalising symptoms
2. Short-lived nature of the condition and often rapid compensation
3. Delayed presentation due to better tolerance of vestibular symptoms
4. Lack of awareness among parents/caretakers
5. Lack of expertise in vestibular medicine among paediatric providers
6. Limited number of centres with the ability to perform vestibular testing in children

A healthy vestibular system is essential for a developing child to ensure optimal gross motor and postural control development, gaze stability during head movement, perception of head position and motion **[59]**, as well as learning **[60]**, and cognitive development **[61]**.

Aetiology

Dizziness in children can result from a number of vestibular conditions, especially vestibular migraine and sensorineural hearing loss. Primary health care physicians should be aware of non-vestibular conditions that can contribute to dizziness in the paediatric population, including binocular vision disorders (e.g., convergence insufficiency, accommodative dysfunction), autonomic dysfunction, neurological disorders, musculoskeletal disease, haemodynamic intolerance, panic attacks, and medications.

Assessment of Vestibular Function in Children

History taking is crucial in diagnosing vestibular disorders in children, as thorough history can direct appropriate assessments and diagnosis. History is best taken from both the child and the parents or caretaker to obtain the necessary information to make the right diagnosis. **Table 6** summarises the important history to be included.

Key Aspects of Patient History for Comprehensive Evaluation	
Antenatal and perinatal history	✓
Developmental milestone	✓
Vestibular and balance history	✓
Sensory processing/integration disorder	✓
Otological history	✓
School performance	✓
Ophthalmological history	✓
Cardiological history	✓
Neurological/Musculoskeletal history	✓
Psychological history	✓

Table 6: Essential Patient History for Comprehensive Evaluation

It is imperative to note that vestibular assessment in children should be tailored to the child's age and clinical suspicion based on history.

1. Ear examination
2. Complete neurological examinations
3. Head impulse test
4. Single-leg standing test

Warning signs of vestibular disorders in children include:

1. Delayed gross motor milestone
2. Frequent falls, clumsiness
3. Hearing loss
4. Exaggerated motion sickness

Management

Management of paediatric vertigo ranges from simple reassurance, non-pharmacological intervention, including vestibular rehabilitation therapy, and pharmacological treatment to surgical intervention.

Due to the complexity of paediatric vertigo, children with dizziness need to be referred to the tertiary centre.

DIZZINESS IN THE ELDERLY

Dizziness is a common problem in the elderly and can lead to problems with balance, which in turn results in falls with head injuries or long bone fractures. Various terms have been used to describe dizziness among the elderly including multisensory dizziness, disequilibrium of aging or presbyvestibulopathy.

Evaluation of dizziness in the elderly only yields a specific diagnosis in less than a third of the patients. The cause is usually multifactorial and addressing individual issues can help with the patient symptoms. The algorithm to guide diagnosis of a dizzy patient **(Figure 2)** may help in providing a structured approach when evaluating the multiple contributing factors of dizziness.

One of the issues is addressing Potentially Inappropriate Medication (PIM). The elderly often have multiple comorbidities and polypharmacy is common in this population of patients. Reducing medication can help reduce unnecessary prescriptions, drug interactions and side effects such as dizziness. Some common drug classes that can cause dizziness are listed in **Table 7** below. **[62-64]**

No.	Indication	Drug classes	Rationale
1.	Gastrointestinal related	Atropine, belladona alkaloids, clidinium-chlordiazepoxide, dicyclomine, homatropine	Anticholinergic effect
2.	Muscle relaxant	Chlorzoxazone, orphenadrine	Anticholinergic effect
3.	Hypertension	Clonidine, hydrochlorothiazide, methyldopa, moxonidine, reserpine, loop diuretics (e.g., frusemide, bumetanide)	Increased risk of hypotension
4.	Thromboembolic agent	Dipyridamole	Increased risk of hypotension
5.	Antipsychotics	Haloperidol, atypical antipsychotics (e.g., clozapine, olanzapine, risperidone, quetiapine, aripiprazole), phenothiazines (first-generation antipsychotics, e.g., chlorpromazine, perphenazine, trifluoperazine)	Increased risk of orthostatic hypotension
6.	Analgesics	Opioids	Sedation effect
7.	Sedative hypnotics	Zolpidem	Sedation effect
8.	Anxiolytics (anxiety)	Benzodiazepines (e.g., alprazolam, clonazepam, lorazepam)	Increased risk of orthostatic hypotension

No.	Indication	Drug classes	Rationale
9.	Allergy	First-generation antihistamines (e.g., chlorpheniramine, dimenhydrinate, hydroxyzine)	Sedation effect
10.	Benign prostatic hyperplasia	Selective alpha blockers (e.g., tamsulosin, alfuzosin, terazosin, prazosin, doxazosin)	Increased risk of orthostatic hypotension
11.	Depression	Selective serotonin re-uptake inhibitors (e.g., citalopram, escitalopram, fluoxetine, paroxetine, sertraline, fluvoxamine), tricyclic antidepressants (e.g., amitriptyline, doxepin, imipramine)	Increased risk of orthostatic hypotension
12.	Diabetes	SGLT-2 inhibitors (e.g., dapagliflozin, empagliflozin)	Increased risk of orthostatic hypotension

Table 7. Drug Classes that May Cause Dizziness

MALPIP and Its Role in Managing Dizziness in Primary Care [65]

The Malaysian Potentially Inappropriate Prescribing (MALPIP) tool is an innovative screening instrument designed to enhance medication safety and optimize pharmacotherapy in older adults. Developed through a rigorous Delphi study involving a panel of experts, MALPIP aims to identify potentially inappropriate prescriptions that may pose risks to the elderly population, thereby improving overall healthcare outcomes.

Dizziness is a prevalent and concerning issue in older adults, often resulting from various age-related biological factors as well as medication side effects.

MALPIP is particularly useful in primary care settings where clinicians frequently encounter older patients with multiple comorbidities and extensive medication regimens. By incorporating MALPIP into routine practice, primary care providers can systematically screen for medications that may cause dizziness or other adverse effects. The tool provides a comprehensive list of medications deemed potentially inappropriate for older adults, allowing for a thorough review and modification of prescriptions.

The practical aspects of using MALPIP in primary care involve several steps. First, primary care providers can use the tool during regular medication reviews, particularly for older patients presenting with dizziness or at risk of falls. By identifying and discontinuing or substituting potentially inappropriate medications, providers can potentially reduce the incidence of dizziness and falls.

Furthermore, MALPIP can be used to educate and raise awareness among healthcare teams about the importance of appropriate prescribing practices for the elderly.

Apart from supplementing dizziness management, integrating MALPIP in primary care settings is expected to facilitate a proactive approach to medication safety, reduce the risk of adverse drug reactions, and enhance the overall quality of elderly care.

See **Appendix 3** for more information about the MALPIP tool.

RECURRENT DIZZINESS

Diseases and disorders that produce persistent dizziness, **usually lasting three months or more**, fall under the category of recurrent dizziness. A detailed history and examination is required as the aetiology of recurrent dizziness can be diverse, ranging from psychiatric, medical, neurological, vestibular and multifactorial. The common causes of recurrent dizziness are listed in **Table 8**. **[10, 66]**

No.	Aetiology	Common Causes
1.	Recurrent Vestibular Syndrome	• Bilateral vestibular failure • Cerebellar degeneration • Persistent postural perceptual dizziness • Persistent Mal de Débarquement Syndrome (MdDS) • Presbyvestibulopathy
2.	Psychiatric	• Generalised anxiety disorder • Panic and phobic disorders
3.	Neurological	• Traumatic brain injury • Vestibular migraine • Parkinson's disease • Brain tumours
4.	Cardiac	• Dysrhythmias
5.	Medical	• Anaemia • Hypoglycaemia • Chronic fatigue syndrome
6.	Drugs	• Adverse drug reactions • Polypharmacy

Table 8. Common Causes of Recurrent Dizziness

It is also important to consider adverse drug reactions or polypharmacy as potential aetiology.

Symptomatic

Vestibular Sedatives				
Drugs	**Use in Dizziness**	**Dosage**	**Adverse/ Undesirable Effects**	**Special Precautions for Use**
Diazepam	Long-acting benzodiazepine with anxiolytic and sedative properties • During acute attack of vertigo as vestibular suppressants controlling vegetative symptoms. • Psychogenic vertigo occurs in association with disorders such as panic disorder, anxiety disorder and agoraphobia.	**Adult:** 10mg IM/ IV repeated if necessary after 8 hours **Oral** **Adult:** 2mg used 8 hourly. Max: 30mg daily. **Elderly:** Dose reduction may be required. **Renal Impairment:** Dosage adjustments may be needed.	Psychological and physical dependence with withdrawal syndrome. **Potentially fatal: Respiratory and CNS depression, coma.**	Impaired renal and hepatic function, respiratory disease, organic cerebral changes, elderly, psychotic patients, epileptics, history of alcohol or drug addiction, impaired gag reflex, obese patients. May cause CNS depression.
Lorazepam	Short acting benzodiazepine • During acute attack of vertigo as vestibular suppressants controlling the vegetative symptoms. • Psychogenic vertigo occurs in association with disorders such as panic disorder, moderate to severe anxiety disorder and agoraphobia.	**Adult:** 2-3mg/day (PO); Acute or severe anxiety 25-30mcg/kg 6 hourly if needed (IV).	Drowsiness, headache, dizziness, confusion, blurred vision, nausea, weakness, unsteadiness. **Potentially fatal: Respiratory depression.**	Hepatic and renal dysfunction, pulmonary insufficiency, myasthenia gravis, may impair ability to drive or operate machinery, elderly or debilitated patients.
Meclozine	Motion sickness, vestibular disorders.	**Oral:** Vestibular disorders. **Adult:** Up to 100mg daily in divided doses.	Drowsiness, thickening of bronchial secretion, dry mouth, fatigue, blurred vision.	Angle-closure glaucoma, prostatic hyperplasia, pyloric or duodenal obstruction, asthma, elderly. May affect the ability to drive or operate machinery.

Vestibular Sedatives				
Drugs	**Use in Dizziness**	**Dosage**	**Adverse/ Undesirable Effects**	**Special Precautions for Use**
Dimenhydrinate	Treatment and prophylaxis of motion sickness, nausea and vertigo caused by Ménière's disease.	**Oral:** Nausea and vertigo caused by Ménière's disease –**Adult:** 50-100mg 3-4 times daily. For prevention of motion sickness: First dose to be given at least 30 minutes before travelling. **Parenteral:** Nausea and vertigo caused by Ménière's disease – **Adult**: 50mg in a 5% solution given IM or 0.5% slow IV inj. given over 2 minutes.	Sedation, dry mouth, thickened respiratory tract secretions, tightness of chest, bradycardia followed by tachycardia and arrhythmias, blurred vision, urinary retention, constipation, GI disturbance, blood dyscrasias. **Paradoxical CNS stimulation may occur in children and occasionally in adults.**	Angle-closure glaucoma, urinary retention, prostatic hyperplasia, pyloric or duodenal obstruction, asthma, epilepsy, elderly. May affect the ability to drive or operate machinery. Tasks requiring mental alertness.
Scopolamine	Prophylaxis of motion sickness, preoperative sedation.	**Oral:** Prophylaxis of motion sickness – **Adult:** As hydrobromide: 300mcg 30 minutes before a journey, then 300mcg every 6 hours if required. Max dosage: 3 doses in 24 hours. **Transdermal:** Prophylaxis of motion sickness – **Adult:** As patch delivering 1mg over 3 days: Apply 1 patch at least 4 hours before exposure to motion. To be applied behind the ear. **Subcutaneous:** Prophylaxis of nausea and vomiting – **Adult:** As hydrobromide: 0.3-0.6mg. **Paediatric dosage:** Prophylaxis motion sickness in children >10 years old: 1 patch behind the ears 4-6 hours before journey.	Postural hypotension, tachycardia, fibrillation. Rarely psychotic reactions. Decreased diaphoresis, heat intolerance. Somnolence, dermatitis, oedema, exudate, follicular conjunctivitis, increased intraocular pressure, local irritation, photophobia, vascular and respiratory congestion. **Potentially fatal: CNS depression, coma, circulatory and respiratory failure.**	Hepatic/renal disease, pyloric stenosis, urinary retention, prostatic hyperplasia, psychosis, seizure disorders, ulcerative colitis, coronary artery disease, tachyarrhythmias, heart failure, hypertension.

Antiemetics				
Drugs	**Use in Dizziness**	**Dosage**	**Adverse/ Undesirable Effects**	**Special Precautions for Use**
Metoclopramide	Vomiting associated with acute attacks of vertigo.	**Oral** 10mg 4 times/day (max dose 30mg daily or 0.5mg/kg daily). **IM** 10mg stat followed by oral if needed.	Extrapyramidal symptoms, restlessness, drowsiness, anxiety, diarrhoea, hypotension, hypertension, headache, depression, blood disorders (e.g., agranulocytosis, methemoglobinaemia), hypersensitivity reactions (e.g., bronchospasm, rash), galactorrhoea or related disorders, transient increase in plasma aldosterone levels. **Potentially fatal: Neuroleptic malignant syndrome, cardiac conduction disorders may occur with IV dosage form.**	Gastrointestinal haemorrhage, mechanical obstruction and perforation, pheochromocytoma, history of seizures. Children, elderly. Renal or hepatic impairment, porphyria, epilepsy, Parkinson's disease, history of depression. Ability to drive or operate machineries may be impaired. Pregnancy and lactation. Monitor patients on prolonged therapy. Increased risk of tardive dyskinesia in patients on prolonged or high-dose treatment.
Prochlorperazine	Vomiting associated with acute attacks of vertigo.	**Oral** 15-30mg/day in divided doses and reduce gradually to 5-10mg/day. **IM** 12.5mg stat followed by oral if needed.	Cholestatic jaundice, cardiac arrhythmias, orthostatic hypotension, leucopaenia, thrombocytopaenia, dry mouth, blurring of vision, glaucoma, urinary retention, constipation, galactorrhoea, gynecomastia, amenorrhoea and impotence. Buccal: Transient numbness of gum and tongue. **Potentially fatal: Bone-marrow suppression. Cardiac arrhythmias or aspiration.**	Extrapyramidal syndrome, hypotension, epilepsy, impaired hepatic, renal, cardiovascular, cerebrovascular or respiratory function, glaucoma. May impair ability to drive or perform tasks requiring mental alertness or physical coordination. CNS depression. Children <2 years. Pregnancy and lactation.

Disease Specific

Steroids				
Drugs	**Use in Dizziness**	**Dosage**	**Adverse/ Undesirable Effects**	**Special Precautions for Use**
Prednisolone	Vestibular neuritis.	**Oral:** Prednisolone 1mg/kg daily for five days, followed by reducing regimen for the next 15 days.	Please refer to product-specific Prescribing Information for more information on adverse events and precautions.	Please refer to product-specific Prescribing Information for more information on adverse events and precautions.

Diuretics				
Drugs	**Use in Dizziness**	**Dosage**	**Adverse/ Undesirable Effects**	**Special Precautions for Use**
Hydrochlorothi-azides	Ménière's disease.	**Oral:** **Adult:** Initially, 12.5mg daily Usual dose: 25-50mg daily.	Volume depletion and electrolyte imbalance, dry mouth, thirst, lethargy, drowsiness, muscle pain and cramps, hypotension, hypersensitivity reactions e.g., rashes, photosensitivity, thrombocytopaenia, jaundice, pancreatitis, fatigue, weakness; may precipitate an attack of gout, impotence, hyperglycaemia, anorexia, gastric irritation, nausea, vomiting, constipation, diarrhoea, sialadenitis, dizziness, raised calcium concentration. **Potentially fatal: Hypersensitivity reactions.**	Existing electrolyte disturbances, hepatic cirrhosis, severe heart failure, oedema, elderly, renal impairment. Monitor for signs of fluid and electrolyte disturbance. Hepatic impairment, diabetes mellitus, gout, hyperlipidaemia, hypercalcemia, hyperuricemia, electrocardiogram, left ventricular hypertrophy and/or ventricular ectopics (extrasystoles). May exacerbate or activate systemic lupus erythematosus in susceptible patients.
Acetazolamide	Ménière's disease.	**Oral:** **Adult:** 250-375mg once daily.	Drowsiness, paraesthesia, ataxia, dizziness, thirst, anorexia, headache, confusion, malaise, depression, gastrointestinal distress, metabolic acidosis, polyuria, hyperuricemia, renal calculi, nephrotoxicity, hepatic dysfunction. **Potentially fatal: Rarely, skin reactions or blood dyscrasias.**	Potassium supplements may be required. Impaired hepatic or renal function, diabetes. Monitor plasma electrolytes and blood count regularly.

Betahistine				
Drugs	**Use in Dizziness**	**Dosage**	**Adverse/ Undesirable Effects**	**Special Precautions for Use**
Betahistine Dihydrochloride	A strong histamine H3 receptor antagonist and a weak H1 agonist with three sites of action for the treatment of Ménière's disease and the symptomatic treatment of vestibular vertigo.	The dosage for adults is 48mg divided over the day. 24mg tablets: 1 tablet 2 times daily. Dosage should be individually adapted according to response. As betahistine mesilate: 6-12mg 3 times daily.	In some cases, mild gastric complaints have been observed. These can normally be dealt with by taking the dose during meals or by lowering the dose. Gastrointestinal: Nausea or vomiting may occur in rare cases. Hypersensitivity reactions e.g., rash, may occur in rare cases.	Patients suffering from pheochromocytoma and patients with bronchial asthma need to be carefully monitored during the therapy. Caution should be taken in the treatment of patients with a history of peptic ulcer. Careful dosage is necessary if the patient has a history of digestive ulcer or an active digestive ulcer, bronchial asthma, pheochromocytoma.

Calcium Channel Blockers				
Drugs	**Use in Dizziness**	**Dosage**	**Adverse/ Undesirable Effects**	**Special Precautions for Use**
Flunarizine	Vertigo caused by Ménière's disease.	Shown to be beneficial in the treatment of Ménière's disease when administered in doses of 10mg orally twice daily for the first 2 weeks, followed by 10mg once daily thereafter.	Depression (history), extrapyramidal symptoms (preexisting).	Nausea, gastric pain and dry mouth have occurred in less than 1% of patients. Vomiting was reported by approximately 30%. Weight gain has been reported during flunarizine therapy. The extrapyramidal disorders include Parkinsonism, akathisia, orofacial dyskinesia, acute torticollis and facial tremor.
Cinnarizine	Vertigo and vestibular disorders.	Oral vestibular disorders: The usual initial dose for adult patients is 25mg or 30mg 3 times daily (as tab), and 75mg once or twice daily (as cap), followed by a maintenance dose of 75mg once daily.	Central nervous system: Transient fatigue and drowsiness, particularly at the start of therapy, are the most frequently reported CNS complaints. Dizziness, asthenia and headache are also potential adverse reactions to cinnarizine. Extrapyramidal symptoms such as tremor, rigor and hypokinesia depression is infrequently reported. Lupus erythematosus may occur, especially in elderly patients and patients receiving greater than 150mg/day of cinnarizine.	Parkinson's disease

Calcium Channel Blockers				
Drugs	**Use in Dizziness**	**Dosage**	**Adverse/ Undesirable Effects**	**Special Precautions for Use**
Verapamil	Vestibular migraine.	For **adult**: Initially, 240mg daily in 2-3 divided doses. Max: 40mg daily.	AV block, bradycardia, worsening heart failure, transient asystole, hypotension, flushing, fatigue, headache, dyspnoea, peripheral oedema, constipation, nausea, abnormal liver function, skin reactions, gingival hyperplasia, extrapyramidal symptoms. Rarely, gynaecomastia. **Potentially fatal: hepatotoxicity.**	Patient with bradycardia or 1st degree AV block, attenuated neuromuscular transmission, hypertrophic cardiomyopathy. **Avoid abrupt withdrawal. Renal and hepatic impairment.**

Nootropics				
Drugs	**Use in Dizziness**	**Dosage**	**Adverse/ Undesirable Effects**	**Special Precautions for Use**
Piracetam	Treatment of the elderly with some degree of cerebral functional impairment e.g. loss of memory, lack of concentration or alertness and vertigo.	**Dose range:** 2.4-4.8g daily in 2-3 divided doses. **Adult:** As monotherapy or in combination with other anti-myoclonic drugs: Initially, 7.2g daily in 2 or 3 divided doses, then increased by 4.8g every 3-4 days. Max: 24g daily in 2-3 divided doses.	Nervousness, agitation, irritability, anxiety and sleep disturbances. Gastrointestinal problems e.g., nausea, vomiting, diarrhoea and stomach-ache. Other symptoms e.g., vertigo, headaches, trembling and sexual stimulation.	As the principle route of elimination for piracetam is via the kidney, special care must be taken when treating patients known to suffer from renal insufficiency. Monitoring of renal function is recommended in such cases. The increase in half-life is directly related to the decrease in renal function and creatinine clearance. This is also true for the older patient in whom creatinine clearance is dependent on age. The daily dose must be individualized according to renal function. Due to effect of piracetam on platelet aggregation, caution is recommended in patients with underlying disorders of haemostasis, major surgery or severe haemorrhage.

Beta-blockers				
Drugs	**Use in Dizziness**	**Dosage**	**Adverse/ Undesirable Effects**	**Special Precautions for Use**
Propranolol	Vestibular migraine.	**Adult: Oral:** Initially, 40mg 2-3 times daily, maybe increased at weekly intervals according to response. Usual range: 80-160mg daily. Max: 240mg daily. As extended - release capsule: Initially, 80mg once daily, may be increased gradually to achieve optimum migraine prophylaxis. Usual range: 160-240mg once daily. **Child:** Oral: ≤12 years 10-20mg 2-3 times daily; >12 years same as adult dose.	Bradycardia, cardiac failure, bronchospasm, hypoglycaemia, exacerbation of angina and acute myocardial infarct following abrupt withdrawal, thrombocytopaenia, visual disturbances, dry eye, nausea, vomiting, diarrhoea, hypersensitivity reactions, sleep disturbances, alopecia, purpura, psoriasiform skin reaction, rash, Raynaud's phenomenon.	Patient with 1st degree heart block, compensated heart failure, diabetes mellitus, myasthenia gravis, history of anaphylactic reaction to allergens, and Wolff-Parkinson-White syndrome may mask the signs and symptoms of hypoglycaemia and hyperthyroidism, renal and hepatic impairment.

Antidepressants				
Drugs	**Use in Dizziness**	**Dosage**	**Adverse/ Undesirable Effects**	**Special Precautions for Use**
Amitripty-line	Vestibular migraine.	**Adult:** Initially, 10-25mg daily, preferably in the evening. May increase gradually in increments of 10-25mg every 3-7 days as tolerated. **Recommended doses:** 25-75mg daily, preferably in the evening. Use the lowest effective dose for the shortest duration needed to treat symptoms.	Suicidal thoughts and behaviour, may precipitate mania or hypomania, may aggravate psychotic symptoms, cardiac arrhythmias, mydriasis and eye accommodation disorder, dysgeusia, paraesthesia, ataxia, disturbance in attention, micturition disorders, decreased libido, erectile dysfunction, hyperhidrosis.	Patient with cardiovascular disease, electrolyte disturbances, history of suicidal related events or exhibiting a significant degree of suicidal ideation, diabetes mellitus, myasthenia gravis, angle-closure glaucoma, increased IOP, urinary retention, prostatic hypertrophy, pyloric stenosis, paralytic ileus, hyperthyroidism, pheochromocytoma.

Anticonvulsants				
Drugs	**Use in Dizziness**	**Dosage**	**Adverse/Undesirable Effects**	**Special Precautions for Use**
Topiramate	Vestibular migraine.	**Adult: Oral:** Initially, 25mg at night for 1 week, then increased in increments of 25mg daily at weekly intervals until an effective dose is reached. **Usual dose:** 100mg daily, maybe increased to 200mg daily if necessary.	Increased seizure frequency or onset of new types of seizures, oligohydrosis, hyperthermia, cognitive related dysfunction, mood disturbances, serious skin reactions, nephrolithiasis, acute myopia associated with secondary angle closure glaucoma, hyperchloraemic metabolic acidosis, hyperammonaemia.	Patient with predisposition to nephrolithiasis, history of eye disorders, predisposition to acidosis. **Avoid abrupt withdrawal.**
Sodium Valproate	Vestibular migraine.	**Adult: Oral:** As extended release tab: Initially, 500mg once daily for a week, then increased to 1000mg once daily. **As delayed release tab:** Initially, 250mg twice daily. Doses up to 1000mg may be given in certain patients.	Suicidal ideation and behaviour, CNS effects, thrombocytopaenia, acquired von Willebrand disease type 1, haemorrhage stroke, hyperammonaemia, hypothermia, delayed hypersensitivity reactions, porphyria, increased liver enzymes, rhabdomyolysis, major congenital malformation.	Patient with systemic lupus erythematosus, carnitine palmitoyltransferase (CPT) type II deficiency, risk factors for hepatotoxicity. Avoid abrupt withdrawal. **Must not be used in pregnant women or those planning to become pregnant.**
Carbamaze-pine	Vestibular migraine.	**Adult: Oral:** Initially, 100-200mg twice daily. Doses may be gradually increased in increments of up to 200mg daily as needed to achieve freedom from pain. **Maintenance:** 400-800mg daily in divided doses. Max 1200mg daily.	Leucopaenia, thrombocytopaenia, CNS depression, ataxia, somnolence, hypotension, increased intraocular pressure, cardiac conduction abnormalities, elevated hepatic enzymes, hepatic failure, hyponatraemia, suicidal thoughts or behaviour, renal toxicity, isolated macular or maculopapular exanthema, exacerbated atypical absence or myoclonic seizures, toxic epidermal necrolysis, Steven Johnson syndrome.	Patient with mixed seizure disorder, increased intraocular pressure, urinary retention, pre-existing cardiac damage, history of cardiac conduction disturbance. **Avoid abrupt withdrawal. Renal and hepatic impairment.**

Important note: The above information on drug usage, dosage, undesirable effects and special precautions were taken from MIMS.com. For more information on specific drugs and their adverse events, please refer to product-specific Prescribing Information.

WHEN TO REFER TO A SPECIALIST

Table 9 summarises the key features of five common peripheral vestibular disorders (otologic causes only).

Duration	Diagnosis	Related to Position	Hearing Loss / Tinnitus	Aural Fullness
Seconds to minutes (Episodic)	BPPV	✓	-	-
Minutes to hours (Episodic)	Ménière's disease	-	✓	✓
	Recurrent vestibulopathy	-	-	-
	Vestibular paroxysmia	-	-	-
Days to weeks (Persistent)	Vestibular neuritis	-	-	-
	Labyrinthitis	-	✓	-

Table 9. Common Peripheral Vestibular Disorders

Peripheral vestibular causes should be considered only when more serious medical or central causes of dizziness are ruled out. If in doubt consult a physician or neurologist first.

When to refer to an ENT

- Positional vertigo
- History of dizziness is that of a rotatory or spinning sensation i.e., vertigo, no light-headedness, unsteadiness or fainting-like spells
- Episodic in nature
- Associated with aural (ear) symptoms such as tinnitus, hearing loss, aural fullness or ear discharge
- ENT examination shows unilateral hearing loss or otoscopy suggestive of ear infection or cholesteatoma
- Failure to manage BPPV

When to refer to a paediatrician

- Dizziness in children

When to refer to a physician

- Cardiac symptoms (chest pain, shortness of breath, excessive sweating, palpitations)
- Abnormal cardiac evaluation (postural hypotension, arrhythmias)
- Abnormal investigations (electrocardiogram [ECG], full blood count [FBC], blood urea and serum electrolytes [BUSE] and fasting blood sugar [FBS])

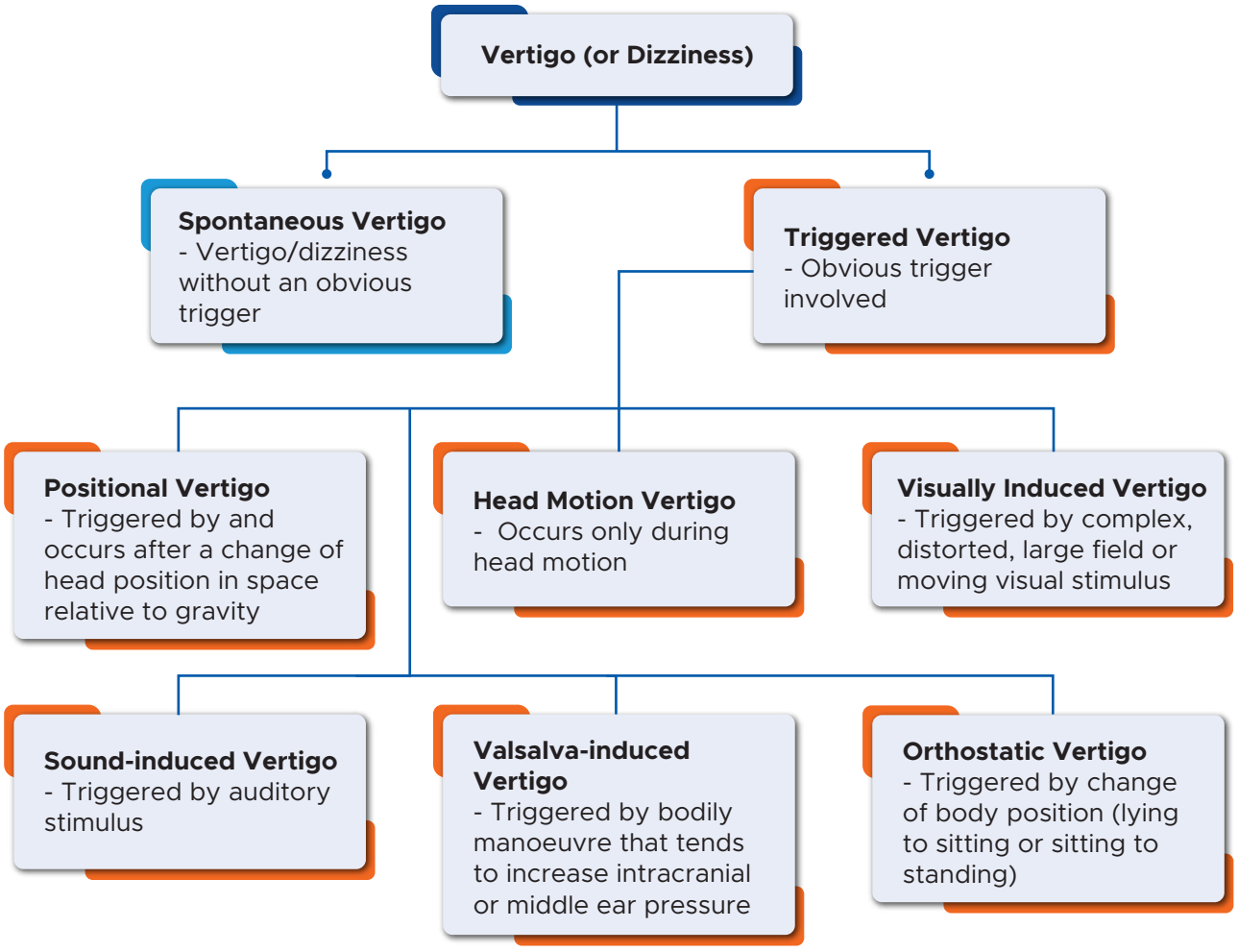
When to refer to a neurologist/physician

- Neurological symptoms (headaches, visual disturbances, hoarseness, dysphagia, numbness, weakness)
- Abnormal neurological evaluation (cranial nerve palsies, paraesthesia, weakness, unsteadiness, cerebellar signs)
- Acute vestibular syndrome

APPENDIX

Appendix 1 – ICVD Secondary Vestibular Symptom Definitions

1.A – Vertigo (or Dizziness)



Bisdorff AR, et al. Overview of the International Classification of Vestibular Disorders. Neurol Clin 2015;33:541-50.

1.B – Vestibulovisual Symptoms

External Vertigo
- False sensation of spinning or flowing surroundings

Oscillopsia
- Oscillating surroundings

Visual Lag
- False sensation that visual surround follows behind a head movement with a delay

Vestibulovisual Symptoms

Visual Tilt
- Visual surround oriented off the true vertical

Movement-induced Blur
- Reduced visual acuity during or shortly after head movement

Bisdorff AR, et al. Overview of the International Classification of Vestibular Disorders. Neurol Clin 2015;33:541-50.

1.C – Postural Symptoms

Unsteadiness
- Feeling of instability while seated, or walking without a directional preferences

Directional Pulsion
- Feeling of instabiliy with tendency to veer or fall in a particular direction (latero-, retro- or anteropulsion) while seated, standing or walking. For lateropulsion, the direction (right or left) should be specified.

Postural Symptoms

A Balance-related Near Fall
- Sensation of imminent fall without a completed fall. Related to strong unsteadiness, directional pulsion, or other vestibular symptom

A Balance-related Fall
- Completed fall related to strong unsteadiness, directional pulsion, or other vestibular symptom

Bisdorff AR, et al. Overview of the International Classification of Vestibular Disorders. Neurol Clin 2015;33:541-50.

Appendix 2 – Bárány Society Diagnostic Criteria

1. Benign Paroxysmal Positional Vertigo (BPPV)

A proper diagnosis must indicate the affected canal and the pathophysiology which is either canalolithiasis or cupulolithiasis. This requires diagnostic positional manoeuvres with observation of canal-specific positional nystagmus. Characteristics of the nystagmus that should be noted include latency, direction and duration.

A. The types of BPPV attacks that lasts under 1 minute

i. Recurrent attacks of positional vertigo or positional dizziness provoked by lying down or turning over in the supine position

ii. Duration of attacks **<1 minute**
AND

iii. Perceived nystagmus based on the positional tests will indicate the type of BPPV

a. Canalolithiasis of the posterior canal (PC-BPPV)
Positional nystagmus elicited after a latency of one or few seconds by the Dix-Hallpike Manoeuvre or Side-Lying Manoeuvre (Semont manoeuvre). The nystagmus is a combination of torsional nystagmus with the upper pole of the eyes beating toward the lower ear combined with vertical nystagmus beating upward (toward the forehead).

b. Canalolithiasis of the horizontal canal (HC-BPPV)
Positional nystagmus elicited after a brief latency or no latency by the Supine Roll Test, beating horizontally toward the undermost ear with the head turned to either side (geotropic direction changing nystagmus)

c. Canalolithiasis of the anterior canal (AC-BPPV)
Positional nystagmus elicited immediately or after a latency of one or few seconds by the Dix-Hallpike Manoeuvre (on one or both sides) or in the supine straight head-hanging position, beating predominantly vertically downward.

d. Lithiasis of multiple canals (MC-BPPV)
Positional nystagmus compatible with canalolithiasis of more than one canal during the Dix-Hallpike Manoeuvre and the Supine Roll Test

v. Not attributable to another disorder

B. The types of BPPV attacks that lasts more than 1 minute

i. Recurrent attacks of positional vertigo or positional dizziness provoked by lying down or turning over in the supine position.

ii. Perceived nystagmus based on the positional tests will indicate the type of BPPV

a. Cupulolithiasis of the horizontal canal (HC-BPPV-cu)
Positional nystagmus elicited after a brief latency or no latency by the supine roll test, beating horizontally toward the uppermost ear with the head turned to either side (apogeotropic direction changing nystagmus), and lasting **>1 minute**

b. Cupulolithiasis of the posterior canal (PC-BPPV-cu)
Positional nystagmus elicited after a brief or no latency by a "Half Dix-Hallpike Manoeuvre", beating torsionally with the upper pole of the eye to the lower ear and vertically upward (to the forehead) and lasting **>1 minute**

iii. Not attributable to another disorder

C. No Observable Nystagmus - Probable BPPV, spontaneously resolved

Probable BPPV, spontaneously resolved

i. Recurrent attacks of positional vertigo or positional dizziness provoked by lying down or turning over in the supine position
ii. Duration of attacks < 1 minute
iii. No observable nystagmus and no vertigo with any positional manoeuvre
iv. Not attributable to another disorder

Notes:

a. Positional vertigo or dizziness must be distinguished from orthostatic symptoms present only on arising but not with other positional triggers
b. Nystagmus may not be as prominent in the elderly with BPPV

2. Vestibular Migraine

A. Vestibular Migraine

I. At least 5 episodes with vestibular symptoms of moderate or severe intensity, lasting 5 minutes to 72 hours
II. Current or previous history of migraine with or without aura according to the International Classification of Headache Disorders (ICHD)
III. One or more migraine features with at least 50% of the vestibular episodes:
    a. Headache with at least two of the following characteristics: one sided location, pulsating quality, moderate or severe pain intensity, aggravation by routine physical activity
    b. Photophobia and phonophobia
    c. Visual aura
IV. Not better accounted for by another vestibular or ICHD diagnosis

B. Probable Vestibular Migraine

I. At least 5 episodes with vestibular symptoms of moderate or severe intensity, lasting 5 minutes to 72 hours
II. Only one of the criteria II and III for vestibular migraine is fulfilled (migraine history or migraine features during the episode)
III. Not better accounted for by another vestibular or ICHD diagnosis

3. Ménière's Disease

A. Definite Meniere's Disease

I. Two or more spontaneous episodes of vertigo, each lasting 20 minutes to 12 hours
II. Audiometrically documented low to medium frequency sensorineural hearing loss in one ear, defining the affected ear on at least one occasion before, during or after one of the episodes of vertigo
III. Fluctuating aural symptoms (hearing, tinnitus or fullness) in the affected ear
IV. Not better accounted for by another vestibular diagnosis

B. Probable Meniere's Disease

I. Two or more episodes of vertigo or dizziness, each lasting 20 minutes to 24 hours
II. Fluctuating aural symptoms (hearing, tinnitus or fullness) in the affected ear
III. Not better accounted for by another vestibular diagnosis

4. Acute Unilateral Vestibulopathy/Vestibular Neuritis

Each of the following criteria have to be fulfilled:

A. Acute or subacute onset of sustained spinning or non-spinning vertigo (i.e., an acute vestibular syndrome) of moderate to severe intensity with symptoms lasting for at least 24 hours
B. Spontaneous peripheral vestibular nystagmus i.e., a nystagmus with a trajectory appropriate to the semicircular canal afferents involved, generally horizontal-torsional, direction fixed, and enhanced by removal of visual fixation
C. Unambiguous evidence of reduced vestibulo-ocular reflex (VOR) function on the side opposite the direction of the fast phase of the spontaneous nystagmus
D. No evidence for acute central neurological symptoms or acute audiological symptoms such as hearing loss or tinnitus or other otologic symptoms such as otalgia
E. No acute central neurological signs, namely no central ocular motor or central vestibular signs, in particular, no skew deviation, no gaze-evoked nystagmus, and no acute audiological signs
F. Not better accounted for by another disease or disorder

5. Vestibular Paroxysmia

A. Vestibular Paroxysmia

Each point needs to be fulfilled:

I. At least ten attacks of spontaneous spinning or non-spinning vertigo
II. Duration < 1 minute
III. Stereotyped phenomenology in a particular patient
IV. Responds to a treatment with carbamazepine/oxcarbazepine
V. Not better accounted for by another diagnosis

B. Probable Vestibular Paroxysmia

Each point needs to be fulfilled:

I. At least five attacks of spinning or non-spinning vertigo
II. Duration <5 minutes
III. Spontaneous occurrence or provoked by certain head-movements
IV. Stereotyped phenomenology in a particular patient
V. Not better accounted for by another diagnosis

6. Persistent Postural-Perceptual Dizziness (PPPD)

PPPD is a chronic vestibular disorder defined by criteria A-E below. All five criteria must be fulfilled to make the diagnosis.

A. One or more symptoms of dizziness, unsteadiness, or non-spinning vertigo are present on most days for 3 months or more
   I. Symptoms last for prolonged (hours-long) periods of time but may wax and wane in severity
   II. Symptoms need not be present continuously throughout the entire day

B. Persistent symptoms occur without specific provocation, but are exacerbated by three factors:
   I. Upright posture
   II. Active or passive motion without regard to direction or position
   III. Exposure to moving visual stimuli or complex visual patterns

C. The disorder is precipitated by conditions that cause vertigo, unsteadiness, dizziness, or problems with balance including acute, episodic, or recurrent vestibular syndromes, other neurologic or medical illnesses, or psychological distress
   I. When the precipitant is an acute or episodic condition, symptoms settle into the pattern of criterion A as the precipitant resolves, but they may occur intermittently at first, and then consolidate into a persistent course
   II. When the precipitant is a chronic syndrome, symptoms may develop slowly at first and worsen gradually

D. Symptoms cause significant distress or functional impairment

E. Symptoms are not better accounted for by another disease or disorder

7. Presbyvestibulopathy

Each of the criteria A through D have to be fulfilled:

A. Recurrent vestibular syndrome (at least 3 months duration) with at least 2 of the following symptoms:
   I. Postural imbalance or unsteadiness
   II. Gait disturbance
   III. Chronic dizziness
   IV. Recurrent falls

B. Mild bilateral peripheral vestibular hypofunction documented by at least 1 of the following:
   I. Vestibulo-ocular reflex (VOR) gain measured by video-head impulse test (HIT) between 0.6 and 0.83 bilaterally
   II. VOR gain between 0.1 and 0.3 upon sinusoidal stimulation on a rotatory chair (0.1 Hz, Vmax = 50-60°/second)
   III. Reduced caloric response (sum of bithermal maximum peak SPV on each side between 6 and 25°/second)

C. Age ≥60 years

D. Not better accounted for by another disease or disorder

8. Bilateral Vestibulopathy

A. Bilateral Vestibulopathy

i. Chronic vestibular syndrome with the following symptoms
   a. Unsteadiness when walking or standing, plus one or both of the following:
   b. Movement-induced blurred vision or oscillopsia during walking or quick head/body movements, **and/or**
   c. Worsening of unsteadiness in darkness and/or on uneven ground

ii. No symptoms while sitting or lying down under static conditions

iii. Bilaterally reduced or absent angular VOR function documented by:
   a. Bilaterally pathological horizontal angular VOR gain <0.6, measured by the video-HIT or scleral-coil technique, **and/or**
   b. Reduced caloric response (sum of bithermal max. peak SPV on each side <6°/second), **and/or**
   c. Reduced horizontal angular VOR gain <0.1 upon sinusoidal stimulation on a rotatory chair (0.1Hz, Vmax=50°/second) and a phase lead >68 degrees (time constant <5 second)

iv. Not better accounted for by another disease

B. Probable Bilateral Vestibulopathy

i. Chronic vestibular syndrome with the following symptoms
   a. Unsteadiness when walking or standing, plus one or both of the following:
   b. Movement-induced blurred vision or oscillopsia during walking or quick head/body movements, **and/or**
   c. Worsening of unsteadiness in darkness and/or on uneven ground

ii. No symptoms while sitting or lying down under static conditions

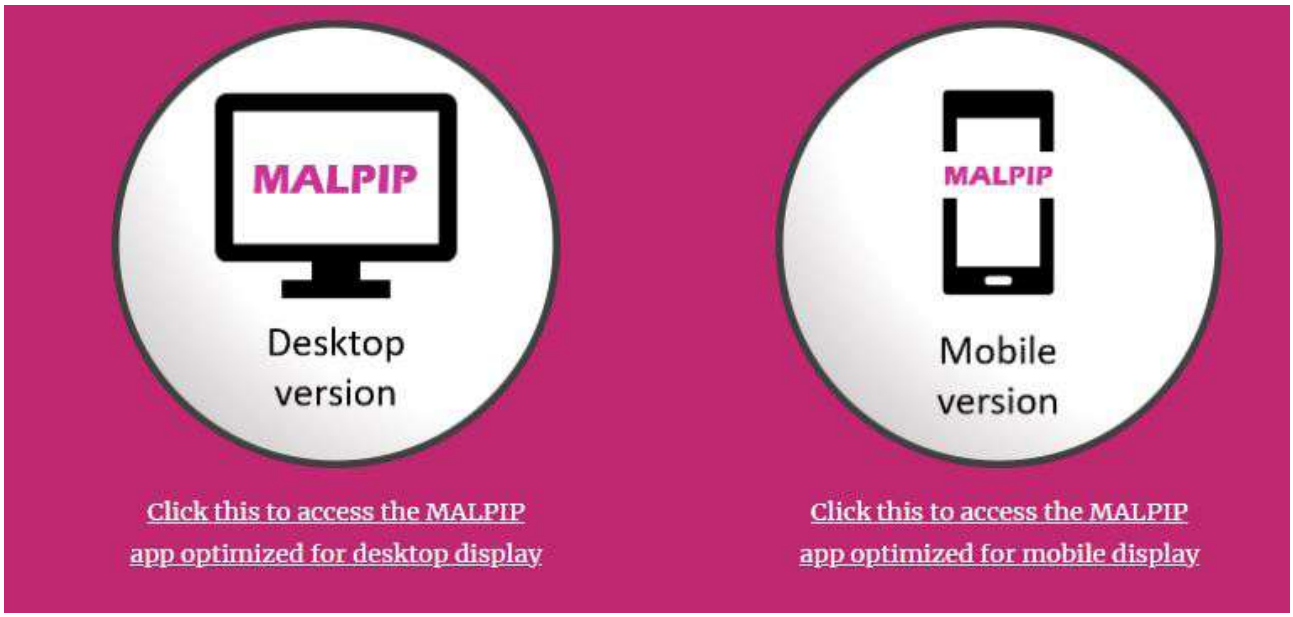
iii. Bilaterally pathological horizontal bedside head impulse test

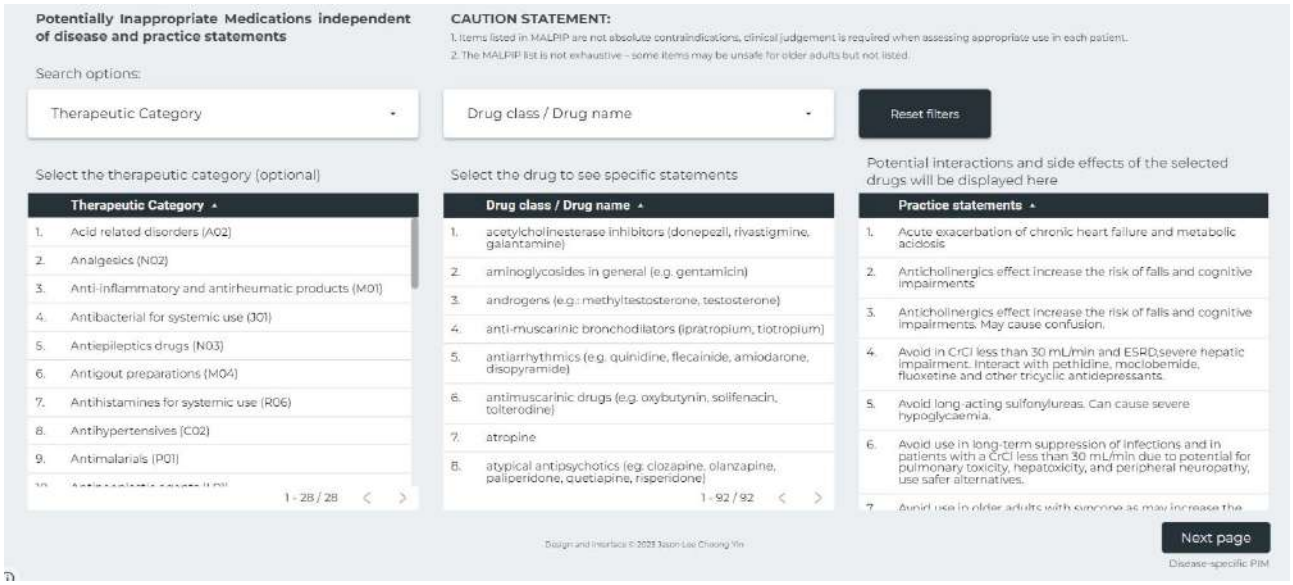
iv. Not better accounted for by another disease

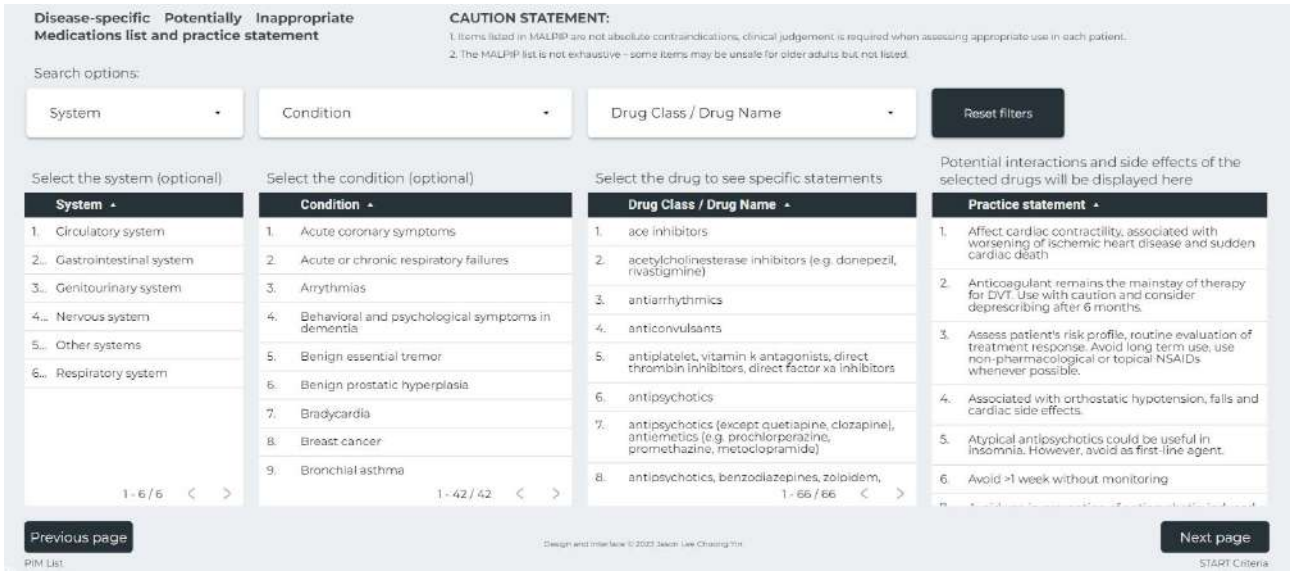
Appendix 3 – MALPIP Screening Tool for Older Adults

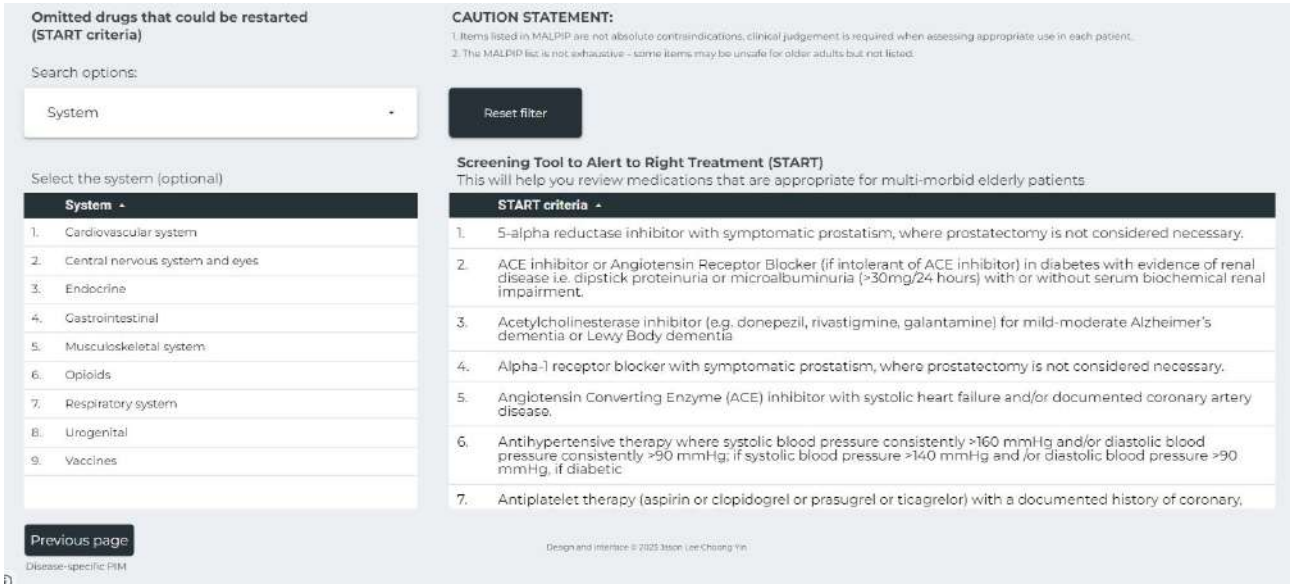
The **MAL**aysian **P**otentially **I**nappropriate **P**rescribing Screening Tool (MALPIP) can be accessed via https://sites.google.com/moh.gov.my/malpip/home. There are two version available for access, a Desktop version and a Mobile version.

The following are screenshots taken from the Desktop version, demonstrating the utility of the MALPIP tool:






Disease-specific Potentially Inappropriate Medications list and practice statement
CAUTION STATEMENT:
1. Items listed in MALPIP are not absolute contraindications, clinical judgement is required when assessing appropriate use in each patient.
2. The MALPIP list is not exhaustive - some items may be unsafe for older adults but not listed.
Search options:
System
Condition
Drug Class / Drug Name
Reset filters
Select the system (optional)
Select the condition (optional)
Select the drug to see specific statements
Potential interactions and side effects of the selected drugs will be displayed here
System
1. Circulatory system
2. Gastrointestinal system
3. Genitourinary system
4. Nervous system
5. Other systems
6. Respiratory system
1 - 6 / 6
Condition
1. Acute coronary symptoms
2. Acute or chronic respiratory failures
3. Arrythmias
4. Behavioral and psychological symptoms in dementia
5. Benign essential tremor
6. Benign prostatic hyperplasia
7. Bradycardia
8. Breast cancer
9. Bronchial asthma
1 - 42 / 42
Drug Class / Drug Name
1. ace inhibitors
2. acetylcholinesterase inhibitors (e.g. donepezil, rivastigmine)
3. antiarrhythmics
4. anticonvulsants
5. antiplatelet, vitamin k antagonists, direct thrombin inhibitors, direct factor xa inhibitors
6. antipsychotics
7. antipsychotics (except quetiapine, clozapine), antiemetics (e.g. prochlorperazine, promethazine, metoclopramide)
8. antipsychotics, benzodiazepines, zolpidem,
1 - 66 / 66
Practice statement
1. Affect cardiac contractility, associated with worsening of ischemic heart disease and sudden cardiac death
2. Anticoagulant remains the mainstay of therapy for DVT. Use with caution and consider deprescribing after 6 months.
3. Assess patient's risk profile, routine evaluation of treatment response. Avoid long term use, use non-pharmacological or topical NSAIDs whenever possible.
4. Associated with orthostatic hypotension, falls and cardiac side effects.
5. Atypical antipsychotics could be useful in insomnia. However, avoid as first-line agent.
6. Avoid >1 week without monitoring
Previous page
PIM List
Next page
START Criteria


Omitted drugs that could be restarted (START criteria)
CAUTION STATEMENT:
1. Items listed in MALPIP are not absolute contraindications, clinical judgement is required when assessing appropriate use in each patient.
2. The MALPIP list is not exhaustive - some items may be unsafe for older adults but not listed.
Search options:
System
Reset filter
Select the system (optional)
Screening Tool to Alert to Right Treatment (START)
This will help you review medications that are appropriate for multi-morbid elderly patients
System
1. Cardiovascular system
2. Central nervous system and eyes
3. Endocrine
4. Gastrointestinal
5. Musculoskeletal system
6. Opioids
7. Respiratory system
8. Urogenital
9. Vaccines
START criteria
1. 5-alpha reductase inhibitor with symptomatic prostatism, where prostatectomy is not considered necessary.
2. ACE inhibitor or Angiotensin Receptor Blocker (if intolerant of ACE inhibitor) in diabetes with evidence of renal disease i.e. dipstick proteinuria or microalbuminuria (>30mg/24 hours) with or without serum biochemical renal impairment.
3. Acetylcholinesterase inhibitor (e.g. donepezil, rivastigmine, galantamine) for mild-moderate Alzheimer's dementia or Lewy Body dementia
4. Alpha-1 receptor blocker with symptomatic prostatism, where prostatectomy is not considered necessary.
5. Angiotensin Converting Enzyme (ACE) inhibitor with systolic heart failure and/or documented coronary artery disease.
6. Antihypertensive therapy where systolic blood pressure consistently >160 mmHg and/or diastolic blood pressure consistently >90 mmHg; if systolic blood pressure >140 mmHg and /or diastolic blood pressure >90 mmHg, if diabetic
7. Antiplatelet therapy (aspirin or clopidogrel or prasugrel or ticagrelor) with a documented history of coronary.
Previous page
Disease-specific PIM

REFERENCES

1. Karatas M. Central vertigo and dizziness: epidemiology, differential diagnosis, and common causes. Neurologist 2008;14(6):355-64.
2. Maarsingh OR, et al. Dizziness reported by elderly patients in family practice: prevalence, incidence and clinical characteristics. BMC Fam Pract 2010;11:2.
3. Lin ME, et al. Association of symptomatic dizziness with all-cause and cause-specific mortality. JAMA Otolaryngol Head Neck Surg 2024;150(3):257-64.
4. Post RE, Dickerson LM. Dizziness: a diagnostic approach. Am Fam Physician 2010;82(4):361-8.
5. Onn MA, Daud K, Salim R. Prevalence of vestibular dysfunction with presbycusis among elderly in Malaysia. PJMHS 2021;15(12):3478-80.
6. Wahat NHA, et al. Prevalence of vestibular disorders among otology patients in a tertiary hospital in Malaysia. Int Med J 2013;20(3):316-8.
7. Richard WKY, et al. Perceived changes in symptoms and quality-of-life amongst patients with dizziness: A single-centre experience in Malaysia. Med J Malaysia 2022;77(6):730-5.
8. R Philip, N Prepageran. Dizziness, a review of walk-in patients at a specialized neurotology clinic. Med J Malaysia 2009;64(1):56-8.
9. Strupp M, et al. Vestibular disorders. Diagnosis, new classification and treatment. Dtsch Arztbl Int 2020;117(17):300-10.
10. Bisdorff AR, et al. Overview of the International Classification of Vestibular Disorders. Neurol Clin 2015;33:541-50.
11. Edlow JA, et al. Guidelines for reasonable and appropriate care in the emergency department 3 (GRACE-3): Acute dizziness and vertigo in the emergency department. Acad Emerg Med 2023;30:442-86.
12. Bosner S, et al. Prevalence, aetiologies and prognosis of the symptom dizziness in primary care – a systematic review. BMC Fam Pract 2018;19:33.
13. Filippopulos FM, et al. Validation of a comprehensive diagnostic algorithm for patients with acute vertigo and dizziness. Eur J Neurol 2022;29:3092-101.
14. El Ammar F, et al. BE-FAST: a sensitive screening tool to identify in-hospital acute ischemic stroke. J Stroke Cerebrovasc Dis 2020;29:104821.
15. Chen X, et al. A systematic review and meta-analysis vomparing FAST and BEFAST in acute stroke patients. Front Neurol 2022;12:765069.
16. Tarnutzer AA, Edlow JA. Bedside testing in acute vestibular syndrome – Evaluating HINTS Plus and beyond – A critical review. Audiol Res 2023;13(5):670-85.
17. Orinx C, et al. Moving from H.I.N.T.S. to H.I.N.T.S. PLUS in the management of acute vestibular syndrome. Ear, Nose Throat J 2022;0(0).
18. Dlugaiczyk J, et al. Recurrent vestibular symptoms not otherwise specified: Clinical characteristics compared with vestibular migraine and Ménière's disease. Front Neurol 2021;12:674092.
19. Strupp M, et al. Acute unilateral vestibulopathy/vestibular neuritis: Diagnostic criteria. J Vestib Res 2022;32(5):389-406.
20. von Brevern M, et al. Benign paroxysmal positional vertigo: Diagnostic criteria. J Vestib Res 2015;25(3-4):105-17.
21. Lempert T, et al. Vestibular migraine: Diagnostic criteria1. J Vestib Res 2022;32(1):1-6.

22. Lopez-Escamez JA, et al. Diagnostic criteria for Menière's disease. J Vestib Res 2015;25(1):1-7.
23. Strupp M, et al. Vestibular paroxysmia: Diagnostic criteria. J Vestib Res 2016;26(5-6):409-15.
24. Staab JP, et al. Diagnostic criteria for persistent postural-perceptual dizziness (PPPD): Consensus document of the committee for the Classification of Vestibular Disorders of the Bárány Society. J Vestib Res 2017;27(4):191-208.
25. Agrawal Y, et al. Presbyvestibulopathy: Diagnostic criteria Consensus document of the classification committee of the Bárány Society. J Vestib Res 2019;29(4):161-70.
26. Strupp M, et al. Bilateral vestibulopathy: Diagnostic criteria Consensus document of the Classification Committee of the Bárány Society. J Vestib Res 2017;27(4):177-89.
27. Zwergal A, Strupp M, Brandt T. Advances in pharmacotherapy of vestibular and ocular motor disorders. Expert Opin Pharmacother 2019;20(10):1267-76.
28. Kalla R, et al. Update on the pharmacotherapy of cerebellar and central vestibular disorders. J Neurol 2016;263:24-9.
29. Muncie HL, et al. Dizziness: Approach to evaluation and management. Am Fam Physician 2017;95(3):154-62.
30. Dyhrfjeld-Johnsen J, Attali P. Management of peripheral vertigo with antihistamines: new options on the horizon. Br J Clin Pharmacol 2019;85(10):2255-63.
31. Bogdanova A, et al. Corticosteroids in patients with vestibular neuritis: An updated meta-analysis. Acta Neurol Scand 2022;146:429-39.
32. Strupp M, et al. Pharmacotherapy of vestibular and ocular motor disorders, including nystagmus. J Neurol 2011;258:1207-22.
33. de Sousa FA, et al. Pharmacological treatment of acute unilateral vestibulopathy: a review. J Audiol Otol 2024;28(1):18-28
34. Sanchez-Vanegas G, Castro-Moreno C, Buitrago D. Betahistine in the treatment of peripheral vestibular vertigo: results of a real-life study in primary care. Ear Nose Throat J 2020;99(6):356-60.
35. Della Pepa C, Guidetti G, Eandi M. Betahistine in the treatment of vertiginous syndromes: a meta-analysis. Acta Otorhinolaryngol Ital 2006;26(4):208-15.
36. Smyth D, et al. Vestibular migraine treatment: a comprehensive practical review. Brain 2022;145(11):3741-54.
37. Nauta JJP. Meta-analysis of clinical studies with betahistine in Ménière's disease and vestibular vertigo. Eur Arch Otorhinolaryngol 2014;271:887-97.
38. Adrion C, et al. Efficacy and safety of betahistine treatment in patients with Ménière's disease: primary results of a long term, multicentre, double blind, randomised, placebo controlled, dose defining trial (BEMED trial). Brit Med J 2016;352:h6816.
39. Rutka JA, Barber HO. Recurrent vestibulopathy: third review. J Otolaryngol 1986;15(2):105-7.
40. van Leeuwen RB, Bruintjes TD. Recurrent vestibulopathy: natural course and prognostic factors. J Laryngol Otol 2010;124(1):19-22.
41. Lindner M, et al. Ginkgo biloba extract EGb 761 improves vestibular compensation and modulates cerebral vestibular networks in the rat. Front Neurol 2019;10:147.
42. Volkenstein S, Dazert S. Recent surgical options for vestibular vertigo. GMS Curr Top Otorhinolaryngol Head Neck Surg 2017;16:Doc01.
43. de Azevedo YJ, et al. Vestibular implant: does it really work? A systematic review. Braz J Otorhinolaryngol 2019;85(6):788-98.
44. Guyot JP, Perez Fornos A. Milestones in the development of a vestibular implant. Curr Opin Neurol 2019;32(1):145-53.

45. Wang YH, Chan CY, Liu QH. Benign paroxsymal positional vertigo – recommendations for treatment in primary care. Ther Clin Risk Manag 2019;15:719–25.
46. Gold DR, et al. Repositioning maneuvers for benign paroxysmal positional vertigo. Curr Treat Options Neurol 2014;16:307.
47. Kurtzer DA, Gans RE, McLeod H. New horizontal canal benign paroxysmal positional vertigo treatment: Kurtzer Hybrid maneuver. Glob J Oto 2017;6(3):555686.
48. Jeon EJ, et al. The efficacy of a modified Dix-Hallpike test with a pillow under shoulders. J Vestib Res 2019;29(4):197-203.
49. Lee HJ, Jeon EJ, Lee DH, Seo JH. Therapeutic efficacy of the modified Epley maneuver with a pillow under the shoulders. Clin Exp Otorhinolaryngol 2020;13(4):376-80.
50. Lee SH, Kim JS. Benign paroxysmal positional vertigo. J Clin Neurol 2010;6(2):51-63.
51. Bhattacharyya N, et al. Clinical Practice Guideline: Benign Paroxysmal Positional Vertigo (Update). J Otolaryngol Head Neck Surg 2017;156(S3):S1-47.
52. van den Broek EM, van der Zaag-Loonen HJ, Bruintjes TD. Systematic review: efficacy of Gufoni maneuver for treatment of lateral canal benign paroxysmal positional vertigo with geotropic nystagmus. J Otolaryngol Head Neck Surg 2014;150(6):933-8.
53. Bhandari A, et al. Diagnostic and therapeutic maneuvers for anterior canal BPPV canalithiasis: three-dimensional simulations. Front Neurol 2021;12:740599.
54. Cohen HS. Side-lying as an alternative to the Dix-Hallpike test of the posterior canal. Otol Neurotol 2004; 25(2):130-4.
55. Hall CD, et al. Vestibular rehabilitation for peripheral vestibular hypofunction: an evidence-based clinical practice guideline: from The American Physical Therapy Association Neurology Section. J Neurol Phys Ther 2016;40(2):124-55.
56. Heffernan A, Abdelmalek M, Nunez DA. Virtual and augmented reality in the vestibular rehabilitation of peripheral vestibular disorders: systematic review and meta-analysis. Sci Rep 2021;11:17843.
57. Brodsky JR, Lipson S, Bhattacharyya N. Prevalence of pediatric dizziness and imbalance in the United States. Otolaryngol Head Neck Surgy 2020;162:241-7.
58. Saniasiaya J, Islam MA, Salim R. The global prevalence of vestibular dysfunction in children and adolescents: a systematic review and meta-analysis. Eur Arch Otorhinolaryngol 2023;280:2663-74.
59. Almutairi A, Cochrane GD, Christy JB. Vestibular and oculomotor function in children with CP: Descriptive study. Int J Pediatr Otorhinolaryngol 2019;119:15-21.
60. Rine RM, et al. Evidence of progressive delay of motor development in children with sensorineural hearing loss and concurrent vestibular dysfunction. Percept Mot Skills 2000;90(3 Pt 2):1101-2.
61. Wiener-Vacher SR, Hamilton DA, Wiener SI. Vestibular activity and cognitive development in children: perspectives. Front Integr Neurosci 2013;7:92.
62. Lindell E, et al. Dizziness and health-related quality of life among older adults in an urban population: a cross-sectional study. Health Qual Life Outcomes 2021;19:231.
63. Anand P, et al. Characterization of potentially inappropriate medications use in Indian elderly population and their impact on quality of life using Beers criteria. Aging Med (Milton) 2022;5(1):45-52.
64. Thorell K, et al. Use of potentially inappropriate medication and polypharmacy in older adults: a repeated cross-sectional study. BMC Geriatr 2020;20:73.
65. Chang CT, et al. Development of a Malaysian potentially inappropriate prescribing screening tool in older adults (MALPIP): a Delphi study. J Pharm Policy Pract 2023;16:122.
66. Stabb JP, Ruckenstein MJ. Expanding the differential diagnosis of chronic dizziness. Arch Otolaryngol Head Neck Surg 2007;133(2):170-6.

ACKNOWLEDGEMENTS

The committee for this guide would like to express their gratitude and appreciation to the following for their contributions:

- **Dr Amirah binti Sufian**
  Department of Otorhinolaryngology, Hospital Sungai Buloh, Selangor
- **Dr Shamini Shanmugalingam**
  Department of Otorhinolaryngology, Hospital Sungai Buloh, Selangor
- **Omar Mohd Nawi**
  Physiotherapy Division, Department of Rehabilitation Medicine,
  University Malaya Medical Centre, Kuala Lumpur
- **Ahmad Akmal Noor Rashidi**
  Physiotherapy Division, Department of Rehabilitation Medicine,
  University Malaya Medical Centre, Kuala Lumpur
- All external reviewers:
  - **Dr Puteri Shanaz Jahn Kassim**
    Family Medicine Specialist, Universiti Putra Malaysia, Kuala Lumpur
  - **Dr Mohd Hazmi bin Mohamed**
    Consultant Otorhinolaryngologist, Faculty of Medicine and Health Sciences,
    Universiti Putra Malaysia, Kuala Lumpur
  - **Leena Lee Poh Chen**
    Rehabilitation Manager and Senior Physiotherapist,
    Department of Rehabilitation Medicine, Universiti Malaysia Medical Centre,
    Kuala Lumpur

Funding acknowledgement

The development of the **"Management of Peripheral Vestibular Disorders in Primary Care – Updated 2024"** guide was made possible by an education grant from Abbott Laboratories Sdn Bhd (Malaysia).

COLLEGE OF OTORHINOLARYNGOLOGISTS
HEAD & NECK SURGEONS,
ACADEMY OF MEDICINE OF MALAYSIA

Supported By

Abbott