# Primary Care Management Pathway
# Dizziness

## Background

This primary care management pathway has been developed by a group of specialist and primary care physicians to help support the management of common non-urgent conditions with long wait-times for specialist care. These pathways are intended to help identify patients with concerning features and facilitate early referrals as needed.

Many different types of disorders can cause a patient to present with a complaint of "dizziness". Most of these patients will not have an inner ear (i.e. Vestibular) cause. Most dizzy patients can be managed in primary care or by an allied health professional (such as physiotherapy). "Vestibular disorder" is an umbrella term used to encompass many different conditions that affect the inner ear and those parts of the central nervous system involved in maintaining balance. This pathway was developed to aid family physicians in the diagnosis, management, and outline when/to whom to refer of patients with dizziness.

## Defining Dizziness

Dizziness is a vague term that patients use to describe a range of sensations, such as:

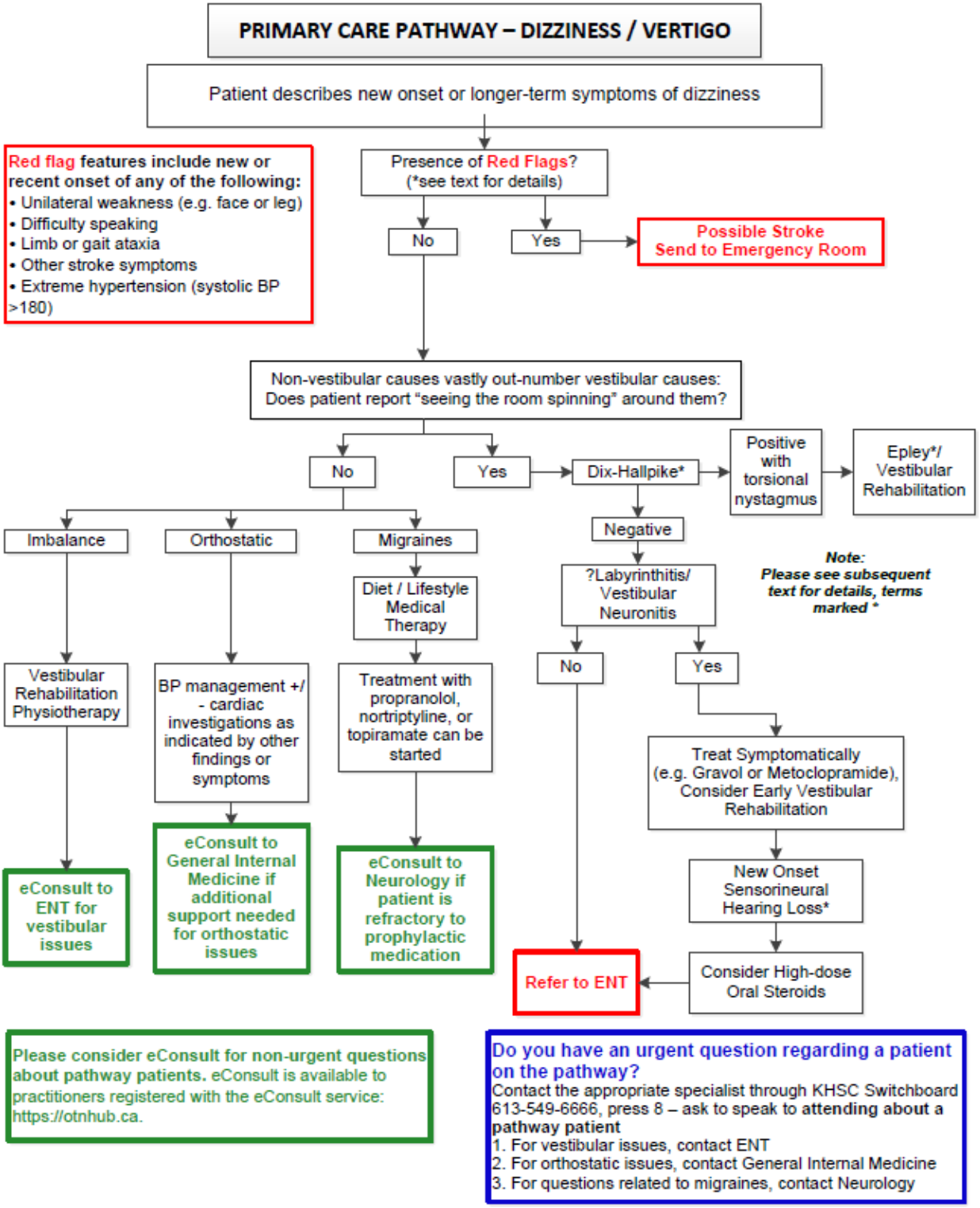
- lightheadedness
- giddiness
- confusion
- presyncope/syncope
- spinning in the head
- brain fog
- imbalance
- an illusion of motion (vertigo);

Dizziness, and related sensations, are sometimes caused by a vestibular disorder. Prior to consideration of a vestibular disorder, consider much more common entities, such as orthostatic dizziness, imbalance, and migraine dizziness.

## Patient information

It is possible that your patient and/or their family member may express a desire for additional information about the primary care management pathway and their role or experience throughout the process of being on a pathway. Additional information for patient education has been provided in "Appendix B – Patient Information".

# Primary Care Management Pathway – Clinical Flow Diagram
## Dizziness



# Appendix A – Expanded Detail

## Diagnosing Dizziness

Many patients with dizziness, imbalance, or vertigo have trouble obtaining a diagnosis, with many different types of vestibular disorders causing dizziness.

In the general population, the most common causes of patients presenting with a complaint of dizziness are orthostatic dizziness, imbalance, or migraine dizziness.

Less commonly, patients may present with a vestibular disorder such as benign paroxysmal positional vertigo (BPPV), labyrinthitis or vestibular neuritis, Ménière's disease, and secondary endolymphatic hydrops. This primary care management pathway offers non vestibular as well as vestibular causes of dizziness, indicating when urgent referral to the Emergency Room or referral to Otolaryngology (i.e. ENT) should be considered.

It is important to first rule out presence of red flags, which may indicate signs of stroke, and would warrant sending the patient to the Emergency Room. On the dizziness pathway, red flag features include new/recent onset of any of the following:

- Unilateral weakness (e.g. face or limb(s))
- Difficulty speaking
- Limb or gait ataxia
- Other stroke symptoms
- Extreme hypertension (systolic BP >180)

The Canadian Stroke Best Practice Guidelines recommend use of the FAST (Face, Arms, Speech, Time) acronym to facilitate memory and recognition of possible stroke signs.

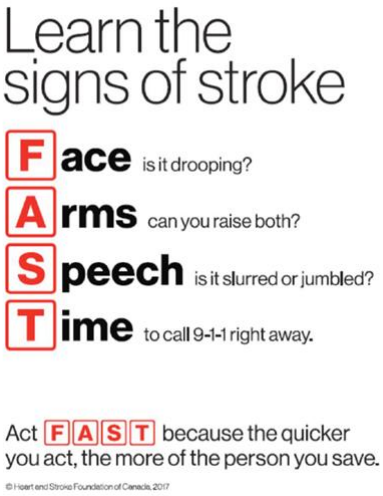



Image 1: Signs of Stroke – FAST[2]

### Benign Paroxysmal Positional Vertigo (BPPV)

Diagnosis: Torsional geotropic nystagmus on Dix-Hallpike testing. Dix-Hallpike testing can be used to elicit nystagmus for diagnosis. For instruction on how to complete the Dix-Hallpike maneuver, please visit: Dix-Hallpike Maneuver.

### Viral Labyrinthitis/Vestibular Neuronitis

Diagnosis: These are two closely related disorders. They present typically with 5 or more days of essentially constant vertigo. Viral labyrinthitis has associated unilateral hearing loss, vestibular neuronitis does not have hearing changes. Diagnosis is clinical, with audiometric testing as soon as available.

## Treatment of Dizziness

### Vestibular Causes and Treatment of Dizziness

### BPPV

Epley* maneuver, or referral to physiotherapy for Epley maneuver. For more information on the treatment of BPPV, please refer to the following link, which provides instruction on completing the Epley maneuver – also known as the canalith repositioning procedure (CRP): Canalith Repositioning Procedure.

### Viral Labyrinthitis/Vestibular Neuronitis

Supportive management is provided. Early vestibular rehabilitation physiotherapy expedites recovery. Vestibular suppressants (such as benzodiazepines) should be avoided as these delay recovery. If unilateral sensorineural hearing loss is suspected, refer urgently to Otolaryngology, and consider starting on high dose oral steroids (1mg/kg for 7 days, then tapered over an additional 7 days. Consider max dose of 60mg, or even lower based on patient age and comorbidities.).

### Non-Vestibular Causes and Treatment of Dizziness

### Vestibular Rehabilitation[3]

Vestibular Rehabilitation is an exercise-based therapy that specifically targets the symptoms of vestibular disorders such as vertigo, dizziness, motion sensitivity, visual disturbance, and imbalance.

A critical component of vestibular rehabilitation is the initial assessment. This involves a thorough evaluation to understand not only the individual's symptoms but also how these symptoms are affecting them in their daily life. As even the same vestibular condition can

cause different symptoms, a careful history and thorough physical assessment is critical to understanding an individual's specific impairments and is crucial to implementing a customized and effective treatment plan.

Components of the examination will include testing of gaze stability (the eye's ability to remain stable when the head is moving), motion sensitivity, and static and dynamic balance. There are many standardized tests that may be used in a vestibular examination such the Modified Clinical Test of Sensory Interaction in Balance (m-CTSIB), the Functional Gait Assessment (FGA) or the Dynamic Visual Acuity Test. These and other tests help not only to determine the degree of vestibular impairment but also guide treatment and provide accurate measures to ensure the expected progress towards short-term and long-term goals is being made.

While the deficits of a vestibular disorder may be permanent, a decrease in symptoms and improvements in function are achievable. A vestibular rehabilitation program stimulates both vestibular and other sensory inputs and challenges the body's ability to accurately detect motion, balance, and stabilize gaze thus promoting central compensation and habituation.

The exercises of a vestibular rehabilitation program are typically completed at home and regular daily practice is critical to success. Depending on the condition a typical exercise program will include gaze stability exercises such as maintaining visual focus on a target while the head or body is moving, a variety of balance exercises, and exercises to target any motion sensitivity. Regular in-clinic re-assessment allows for the progression of the exercises to ensure they remain challenging and are ensuring progress towards patient goals.

Originally developed in the 1940s to treat dizziness after a head injury, vestibular rehabilitation has evolved from a simple uniform movement protocol to an individualized, patient- and goal-centered approach. Vestibular rehabilitation is supported by an extensive and growing body of scientific evidence and is the preferred treatment approach for many vestibular conditions. It has brought symptom relief and improved function to thousands of people and continues to evolve and become more effective all the time.

### Orthostatic Hypotension[4]

Orthostatic Hypotension is defined as a sustained and persistent drop in systolic BP (SBP) ≥20 mm Hg or diastolic BP ≥10 mm Hg within 3 minutes of achieving a standing position or head-up tilt to at least 60 degrees on tilt table. There are many potential causes, as outlined in the table below:

| | |
|---|---|
| Medication | Antidepressants (most commonly implicated: TCA, MAOIs), Anticholinergics (benztropine, oxybutynin), Antihypertensives, |

| | |
|---|---|
| | Dopamine agonists (levodopa, rotigotine, bromocriptine, ropinirole, pramipexole),<br>Ethanol,<br>Insulin (may exacerbate OH in the setting of diabetic neuropathy),<br>Vasodilators (hydralazine, nitroglycerin),<br>Phosphodiesterase inhibitors (sildenafil, tadalafil),<br>Opioids/sedatives (morphine, benzodiazepines, barbiturates, promethazine),<br>Antipsychotics (thioridazine, iloperidone),<br>Neurotoxic drugs (vincristine, cisplatin) |
| Neurogenic | Idiopathic OH (1/3 of cases of OH),<br>Central autonomic nervous system disease (40% of patients with Parkinson's Disease have OH),<br>Peripheral autonomic nervous system diseases (e.g. diabetic autonomic neuropathy – very common) |
| Non-Neurogenic | Cardiac disorders (heart failure, arrythmias, pericardial disease, severe aortic stenosis, idiopathic hypertrophic subaortic stenosis, hypertrophic obstructive cardiomyopathy, acute aortic regurgitation, acute myocardial infarction),<br>Deconditioning<br>Intravascular volume depletion (bleeding, diarrhea, diabetes insipidus, diuretics, poor oral intake, vomiting)<br>Metabolic (adrenal insufficiency, hypoaldosteronism, pheochromocytoma, carcinoid syndrome, hypokalemia (severe), hypothyroidism, porphyria),<br>Sepsis<br>Systemic mastocytosis<br>Venous pooling (heart of vigorous exercise, postprandial splanchnic dilation, prolonged recumbency or standing) |

Risk factors for OH include old age (particularly patients in long-term care facilities), multiple comorbidities (including HTN, diabetes, neurodegenerative disorders, neuropathy), as well as polypharmacy.

Initial approach to diagnosing OH includes: detailed history and physical examination with a focus on neurodegenerative disorders and neuropathy, thorough medication review, screening for reversible causes: 12-lead ECG, CBC, BMP (hypokalemia, alkalosis, and renal insufficiency suggesting volume depletion), TSH, 24-hour ambulatory BP monitor, B12/SPEP/UPEP if neuropathy on exam. ECG/Holter if arrhythmias suspected.

In completing the history, consider:

- Postural symptoms: dizziness, light-headedness, palpitations, visual blurring, syncope, or presyncope. Elderly patients may have vague complaints even before frank syncope: generalized weakness, fatigue, nausea, difficulty with concentration or cognition, leg buckling, pure vertigo, visual blurring, headache or "coat-hanger" pattern neck–shoulder pain, orthostatic dyspnea, or angina.
- Aggravating factors: warm environments, exertion, prolonged standing, ingestions of large/carbohydrate-rich meals, alcohol intake (vasodilation)
- Volume depletion: vomiting, diarrhea, poor oral intake, polyuria
- Cardiac pump failure: orthopnea, edema, paroxysmal nocturnal dyspnea, angina
- Peripheral neuropathy: numbness, pain, paresthesia, imbalance, or falls
- Associated diseases: diabetes, PD, dementia
- Autonomic symptoms: altered sweating (hyper- or hypohidrosis), GI dysfunction (bloating, nausea, vomiting, constipation), impotence, bladder dysfunction, sicca symptoms
- May be asymptomatic

In completing the physical exam, consider:

- Measure BP while supine and standing: Patient should be supine for 5 minutes and then after standing, check the BP at 3 minutes. Use sitting measurements only if the patient is too dizzy or weak to stand. Use fall precautions: Do not check for OH in patients with supine SBP <90 mm Hg (shock) because it adds no useful information. Tachycardic response to standing may be a sign of hypovolemia or cardiac pump failure, whereas minimal or no change in heart rate may suggest a neurogenic cause. Orthostatic tachycardia without a significant drop in BP does not meet criteria for OH and may suggest postural orthostatic tachycardia syndrome (POTS).
- Cardiac exam: jugular venous distention, pulse irregularity, edema, murmurs, S3
- Neurologic exam: hypomimia, gait, tremor, cogwheel rigidity, motor strength, fine touch, pain sensation, proprioception, Romberg maneuver, cerebellar signs, and myoclonus

Differential diagnoses include: Neurally mediated (reflex) syncope [vasovagal syncope, situational syncope (cough, micturition, defecation, swallowing)], carotid sinus hypersensitivity, falls related to a neurologic disorder, postprandial hypotension, POTS, shock (must have a normal lying BP before testing).

<u>Treatment</u>

- Abdominal binder or compression stockings.
- Moving gradually when switching to standing from a sitting position. Also, physical counter maneuvers (to increase vascular resistance and preload): isometric contraction of leg muscles for 30 seconds at a time, repeated feet dorsiflexion, leg crossing and contraction, squatting, bending at the waist, leg elevation, and respiratory maneuvers, such as inspiration through pursed lips and inspiratory sniffing, moderate exercise, elevate head of bed 20 degrees

- Increase water and sodium intake: 1.25 to 2.50 L of fluid a day and up to 6 to 9 g/day of sodium). Encourage drinking water with meals and before exercise. Small and frequent rather than large meals.
- Avoid prolonged recumbency; increased intrathoracic pressure (straining, coughing); large meals, especially if high in carbohydrates; and alcohol.
- Medication: Discontinue all potentially aggravating medications.

**Migraines**

Prophylactic Treatment

- Suggest treatment for frequent (e.g. more than 6-8 headache days per month) and/or disabling migraines
- Choice of prophylactic treatment depends primarily on comorbidities and potential side effects.
- Initial options include:
    - Propranolol, e.g. starting at 10mg BID and increasing in 2 weeks to 20mg BID, then in a further 2 weeks to 40mg BID.
        - Potential side effects primarily include hypotension and bradycardia
        - Can be beneficial for patients with hypertension
        - As it is a beta-blocker, propranolol is contraindicated in patients with asthma, or with pre-existing bradycardia.
    - Topiramate, e.g. starting at 25mg qhs and increasing in 25mg increments every 2 weeks, to a target of 75mg BID
        - Potential side effects include weight loss, cognitive slowing, paresthesias in the extremities, and rarely kidney stones or glaucoma.
        - Can be beneficial for patients with obesity, as it may cause weight loss
        - Contraindicated in patients with low body mass index or history of kidney stones
    - Amitriptyline or nortriptyline, e.g. starting at 10mg qhs, increasing in 2 weeks to 25mg qhs. Option to increase further to 50mg qhs if well tolerated and only partially effective.
        - Nortriptyline can be better tolerated but is more expensive, so is a reasonable choice for patients with drug coverage
        - Potential side effects include weight gain, sedation or morning grogginess, and anti-cholinergic effects such as dry mouth or dry eyes. Can also impact cognition and should be used with caution in elderly patients.
        - Can be beneficial for patients with low mood or poor sleep.

If patient is refractory to medication, please consider seeking specialist advice from Neurology through eConsult.

## When should I refer my patient to a specialist?

Most individuals with dizziness can be managed by their primary care provider. eConsults are encouraged for questions or if additional support is needed. Additionally, there are certain instances when referral to the appropriate specialist is indicated.

Referral to Otolaryngology (ENT) should be considered once more common causes of dizziness have been ruled out, and a primary vestibular disorder is being considered. Furthermore, if new-onset unilateral sensorineural hearing loss is suspected, patients should be urgently referred to Otolaryngology.

For orthostatic issues, consider a referral to General Internal Medicine.

For migraines, consider a referral to Neurology.

# Appendix B – Patient Information

*Please note: This information is intended to be given to the patient, either as a handout or in the form of a conversation with their primary care provider.*

## What is Dizziness?

Dizziness is a term used to describe a range of feelings like a false sense of motion (also known as vertigo), feeling lightheaded of faint, or imbalance. There could be various reasons for feeling dizzy and in some cases it can be caused by different types of vestibular disorders.

## How will my doctor follow my condition?

Your family doctor will follow you with a clinical assessment and investigations, which could include administering certain tests or prescribing certain medications. Your family doctor will refer you to a specialist only if it is felt required.

## You are enrolled on a "Dizziness" clinical pathway. What does this mean?

The dizziness primary care management pathway was developed by family doctors, otolaryngologists, and neurologists at Kingston Health Sciences Centre to help with the screening and treatment of people with dizziness.

Clinical pathways are an evidence-based tool for common conditions seen frequently by family doctors. The pathways ensure that patients receive standardized care for their conditions. Clinical pathways help identify patients with high-risk features and facilitate early referral to specialists as needed. They also identify patients with low-risk symptoms who can be monitored by their family doctors.

For more information or if you are interested in learning more, visit: https://vestibular.org/

# Appendix D – Endnotes

## References

1. Vestibular Disorders Association. (2022). Types of Vestibular Disorders. https://vestibular.org/article/diagnosis-treatment/

2. Heart and Stroke Foundation Canada. (2021). Signs of Stroke – FAST [Infographic]. https://www.heartandstroke.ca/stroke/signs-of-stroke

3. Dougal Bale Phty. Hons. (Registered Physiotherapist, Vestibular Rehabilitation Kingston Ear Institute)

4. Wolters Kluwer Health Inc. (2023). 5MinuteConsult – Hypotension, Orthostatic. https://5minuteconsult.com/collectioncontent/1-151758/diseases-and-conditions/hypotension-orthostatic