

# Vertigo-An Overview

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## ABSTRACT

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Human beings maintain equilibrium utilizing a sophisticated system. Vertigo is a very disquieting symptom. Analysis of the symptomatology and clinical picture is needed to ascertain the cause of vertigo in any given case.

**Keywords:** Vertigo, Types and evaluation

\*See End Note for complete author details

Man has developed a very sophisticated system by which perfect equilibrium is maintained. Sensory information from the eyes and vestibular apparatus together with proprioceptive information from the neck and limbs passes to the central nervous system where, at the level of the vestibular nuclei, it is integrated and modulated by activity arising in the cerebellum, extra pyramidal system and cortex. Pathways arising from the nuclei connect with five main systems; the cerebral cortex, oculomotor nuclei, the motor part of spinal cord, the cerebellum and the autonomic nervous system resulting in static and dynamic spatial orientation and control of locomotion and posture. Pathology affecting the central nervous system, cardiovascular system, the eyes, the ears, the locomotor system, blood and endocrine gland may all alter this balance of neural information and result in disequilibrium.

## Dizziness I VERTIGO

Dizziness is a term that comprises a number of symptoms of disequilibrium including light headedness, faintness, giddiness, sensations of floating, imbalance ataxia, mental confusion or loss of consciousness. In other words it is a feeling of “as if about to fall”.

Vertigo is a specific symptom related directly to dysfunction of the vestibular system. By definition, vertigo is a “hallucination of movement (Cawthorne 1952) or “disagreeable sensation of instability or disorder of orientation in space.

It may not be always possible for the patient to differentiate between the two symptoms and they may often complain only of a feeling of instability.

## Physiology Vestibular System

Vestibular labyrinth is composed of two parts:

1. Semicircular Canals that respond to angular acceleration.
2. The Otolith Apparatus that respond to linear acceleration.

The two halves of the vestibular system should be maintained in perfect balance for equilibrium.

During head movements vestibular input alters along with visual signals and cervical and proprioceptive input. From birth, this information is stored in the reticular formation of the brain stem (data centre). Afferent information is always compared with this data bank and normally there is a perfect match and equilibrium is maintained. If the function of any of these is impaired and mismatch occur between the information generated by one sensory modality and that of the other, symptoms of disequilibrium arise.

Table 1. Causes of Dizziness/Vertigo

General Medical	Otological	Neurological	Miscellaneous
a. Haematological: anaemia, polycythaemia	1. Trauma, 2. Infection	1. Disorders of VIII <sup>th</sup> Nerve 2. Brainstem disease	1. Cervical vertigo 2. Ocular vertigo
b. Cardiovascular: c. hypotension, cardiac failure, dysarrhythmia	3. Vascular 4. Menieres disease	3. Cerebellar disease 4. Cerebrovascular disease	3. Laterogenic
d. Metabolic: Diabetes mellitus, hypoglycemia, chronic renal failure, alcohol	5. Auto immune disorders 6. Ototoxicity 7. Metabolic bone disease 8. Structural abnormalities of the vestibular labyrinthine	5. Multiple sclerosis 6. Trauma 7. Infection 8. Epilepsy	

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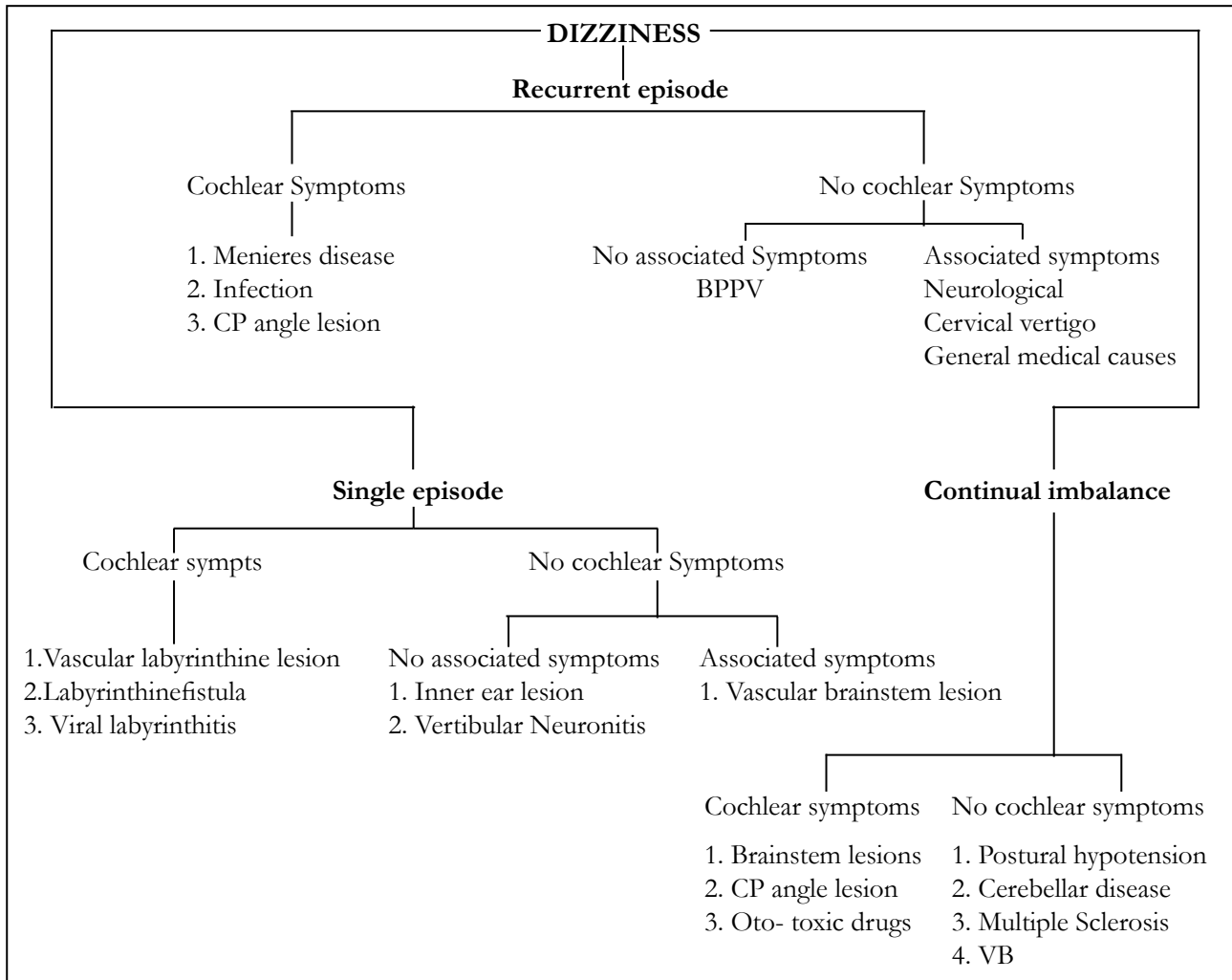


Figure 1. Diagnostic approach to Dizziness / Vertigo

**Diagnosis:** is based on

1. A proper history
2. Full medical examination with reference to the ears, eyes, neurological assessment
3. Specific special investigations.

By considering the character of complaint, duration of illness and presence or absence of associated symptoms-cochlear, neurological or cardiovascular we often get a clue to the diagnosis.

**Some Generalizations:**

1. Vertigo is commonly associated with a vestibular disorder
2. Dizziness is commonly related to general medical disorder
3. Sudden, unexpected, short lived episodes of vertigo- peripheral vestibular disorders
4. Gradual, insidious onset of continual imbalance - central disorder (exception include temporal lobe

epilepsy and vertebrobasillar ischaemia)

5. Duration  
BPPV- duration of individual attacks 30-40 seconds  
Menieres disease lasts upto 24 hours.  
Labyrinthine failure vertigo lasts for many days
6. Associated symptoms: Audiology symptoms like hearing loss, tinnitus, sensation of fullness in the ear and painful lesions of labyrinth or VIIIth Nerve.

**Benign Paroxysmal Positional Vertigo ( BPPV)**

Most common clinical syndrome following minor head injury. Symptoms develops after a symptom free interval of days or weeks. It can also be seen after viral infections of upper respiratory tract.

**Symptoms**

Brief severe episode of rotatory vertigo lasting less than a minute upon sudden changes of head position especially on lying down and turning towards the affected ear.

**Findings:**

*Dix Hall pike Maneuvers*

- Latent period 2-20 sec followed by vertigo/nystagmus with or without nausea nystagmus - linear rotatory with fast phase towards affected ear lasts <1 minute.
- Absence of symptoms and signs on repeated testing (fatigability)

**Pathophysiology:**

Thought to arise due to pathology in the posterior semi circular canal

1. Theory of “Cupulolithiasis” proposed by S-chuknecht in 1969.
2. Theory of “Canalolithiasis’ proposed by Brandt and Stedden in 1993.

According to this theory, Otoconialdebris forms a free-floating clot in the posterior semicircular canal. Rapid changes of head position with respect to gravity causes the clot to move and induce endolymph flow and cupular deflection

**Pharmacotherapy**

1. Role not clearly established
2. Best avoided where possible to allow central nervous system compensatory mechanism
3. Drugs employed symptomatically are
  - Phenothiazines
  - Ca++ channel antagonists
  - Antihistamines
4. Betahistine (histamine receptor agonist) has a prophylactic role and is widely used.

**Liberatory Manoeuvre - Canalith Repositioning Procedure**

- Proposed by Epley
- To use head position and vibration to cause free canaliths to migrate out of PSCC to the inert region of the utricle.

This maneuver has produced successful results in most patients and if the symptoms recurs, the maneuver can be repeated.

**Migrainous Vertigo**

Migrainous vertigo, although not recognized in the International Headache Society Schema, is a commonly diagnosed entity among neuro-otologists. The diagnosis requires clinical suspicion and is one of

Table 2. Characteristics of Peripheral Vs Central Vertigo

Symptoms or sign	Peripheral	Central
1. Latency (time of onset of vertigo/nystagmus)	0-40 seconds	No latency, begins immediately
2. Duration	1 minute Yes	Symptoms may persist
3. Fatigability (lessening of signs and symptoms with 1 repetition of provocative manoeuvre)	Yes	No
4. Nystagmus direction	Fixed, torsional, up, upper pole of eyes towards ground	Direction changes variable
5. Intensity of signs and Symptoms	Severe vertigo marked nystagmus, nausea	Usually mild vertigo, less intense nystagmus, rare nausea
6. Reproducibility	Inconsistent	More consistent

exclusion, Neuhauser et al have suggested diagnostic criteria

**Definitive criteria**

1. Episode of vestibular symptoms of at least moderate severity vertigo, positional dizziness and head motion intolerance
2. Migraine according to International Headache Society Criteria
3. One or more of the following features during at least two vertigo attacks
  - Migrainous headache
  - Headache
  - Photophobia
  - Phonophobia
  - Migrainous aura
4. Other diagnoses excluded by appropriate test

**Probable:**

Criterion 1 and 4 as above plus s at least ONE of the following

- migrainous headache
- migraine symptoms during vertigo
- migraine specific triggers of vertigo response of anti migraine drugs.

Migrainous vertigo is a diagnosis of exclusion and because some patients may have symptoms and signs (including nystagmus) suggestive of central dysfunction, neuroimaging may be required at first presentation. AI though there have been no adequate randomized trials of treatment of migrainous vertigo in the clinic setting, most neurologist use standard anti-

migraine prophylactic drugs (propranolol, amitryptilline etc) with reasonable success.

## INVESTIGATIONS

The laboratory investigation, like the physical examination, should be directed particularly by the patients history. If there is a history of presyncope or syncope, the patient must have a cardiac evaluation, and an electrocardiogram. All patients with undiagnosed vertigo should have metabolic screening tests, including blood cell count, electrolytes, blood glucose, ESR and thyroid function test. The presence of auditory symptoms requires audiometric tests. Multiple or recurrent cranial neuropathy requires screening test for collagen vascular disease or basal skull lesions or meningitic process. Vertigo with cerebellar signs definitely requires CT scan or MRI of brain.

## END NOTE

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**Conflict of Interest:** None declared

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