CHAPTER

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Approach Towards a Patient with Vertigo

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INTRODUCTION

The term vertigo is derived from the Latin word 'vertere' meaning 'to turn'.

Vertigo is an illusion of self or environmental motion. It may be subjective (patient is moving) and objective (environment is moving) or both. Vertigo is a symptom and never a disease and it is very common complaint in clinical practice. It is due to asymmetry of vestibular system due to damage or dysfunction of the labyrinth and vestibular nerve, or central vestibular structures in the brainstem. Vertigo may be physiologic eg. occurring during or after a sustained head rotation.

VERTIGO V/S DIZZINESS

Dizziness is an imprecise term that means consciousness of disordered orientation of the body in space. Dizziness is a variety of symptoms; it could mean vertigo, dysequilibrium, lightheadedness / presyncope, rocking or swaying as if on a ship, motion sickness, nausea & vomiting, oscillopsia, floating, swimming, spinning inside of head, etc.

ANATOMY AND PHYSIOLOGY

Balance calls upon contributions from vision, vestibular sense, proprioception, musculoskeletal coordination, and even cognitive skills. All these streams of information combine and integrate in central vestibular apparatus in the brain to assess the stability and orientation.

Asymmetrical or defective input into the vestibular apparatus or asymmetrical central processing leads to vertigo whereas abnormal bilateral vestibular activation results in truncal ataxia.

Vestibular system contributes to our balance system, maintains spatial orientation, stabilizes vision and provides information related to movement and head position.

It constitutes:-

Semicircular Canals

The three canals are set at right angle to each other which allow them to perceive rotational or angular acceleration and deceleration in all three planes of three dimensional space. Semicircular canals provide afferent input for the vestibulooccular reflex to keep the eye steady as the head turns. It contains sensory hair cells that are activated by movement of endolymph. As the head moves, hair cells in the semicircular canals send nerve impulses to the brain by way of the vestibular part of IIIV nerve.

Otolith organs

The two otolith organs viz. utricle and saccule are oriented roughly perpendicular to each other, utricle in horizontal plane and saccule in vertical plane. Both organs are sensitive to linear rather than rotatory acceleration. The utricle is sensitive to change in horizontal movement. The saccule is sensitive to the change in vertical acceleration (such as going up in an elevator).

CLASSIFICATION OF VERTIGO

Vertigo can either be due to peripheral causes (cervical, auditary, ocular) or central causes (brainstem, cortical, psychogenic).

Peripheral causes are related to damage of the inner ear receptors or to the vestibulocochlear nerve. Central causes include damage to centers that process vestibular signals in the central nervous system. These may be tabulated as follows:

HISTORY

A careful history remains the cornerstone of diagnosis. A patient complaining of dizziness necessitates a thorough history taking precisely because of the many different meanings the term can have (Table 1).

Following protocol is helpful-

1. Symptoms - when did the symptoms start?

a. Describe the symptoms (avoid leading questions),

b. Time course (resolving, fluctuating),

c. Persistence (constant, resolving or episodic),

d. Length and frequency of episodes,

e. Are episodes spontaneous or provoked (eg. by head movement)?

f. Are there any associated symptoms (nausea, anxiety, neurological symptoms)?

g. Do the symptoms go away completely between episodes?

2. History of any ear symptoms,

3. Any Ophthalmological symptoms,

4. Any Neurological symptoms,

5. Any Other risk factors (head injury, ototoxic drugs, spondylosis, whiplash injury).

Table 1: History Taking and Mechanism of Dizziness

Types of Dizziness	Patient Experiences	Mechanism
Vertigo	Illusion of movement of patient or surroundings.	Disturbance of peripheral or CNS pathways of vestibular system.
Dysequilibrium	Imbalance or unsteadiness while standing or walking.	Vestibulospinal, propioceptive, visual or motor dysfunction, joint pain or instability, psychological factors.
Syncope or Presyncope	Impending loss of Consciousness.	Momentary reduction in blood flow to brain eg. cardiac problems.
Mal de débarquement	Sense of rocking or swaying as if on a ship.	Vestibular adaptive process to the continuous, passive motion os lost and is unable to re-adapt once environment is stable.
Motion sickness	Episodic dizziness, tiredness, pallor, diaphoresis, salivation, nausea & vomiting.	Visual-vestibular mismatch eg. riding in a car or viewing action sequence in large screen theater.
Nausea & Vomiting		Stimulation of vagus centers in medulla.
Oscillopsia	Subjective illusion of visual motion	Spontaneous : acquired nystagmus Head induced : severe, bilateral loss of the VOR
Floating, swimming, spinning inside of head		Frequently psychological symptoms of anxiety, somatoform disorders, and depression.

EXAMINATION (TABLE 2)

Unfortunately there are only few reliable clinical signs to detect or rule out vestibular disease, however, the key points are

Table 2: Differences between Peripheral and Central Vertigo

Symptoms	Peripheral	Central
Onset	Paroxysmal	Slow or acute
Duration	Seconds to minutes	Weeks to months
Intensity	Severe	Moderates
Frequency	Episodic, recurrent	Constant, progressive
Nystagmus	Unidirectional - horizontal or rotatory	Bidirectional - horizontal or vertical
Triggered by changing head positions	Yes	No
Unsteadiness	Mild/moderate	Severe
Nausea/ vomiting	Severe	Varying
Auditory symptoms	Common	Rare
Neurological symptoms	Rare	Common
Compensation/ resolution	Rapid	Slow

- Begin assessing as soon as you meet a patient even in waiting room.
- Observe the patient while walking along several adjoining hallways, making abrupt turns both to right and left, and traversing an uphill and downhill incline.
- A patient with a vestibular deficit will often stare at the floor to keep his balance, especially in an unfamiliar setting such as hospital. When patient with vestibular pathology walks, he often veers towards the side of the lesion, and uses a wide based gait. You should be wary of patients who try to "show you how bad my balance is" by using a narrowed base of gait or swaying excessively when standing still.
- The following list of examination techniques is by no means prescriptive, but is useful when examining a patient with vertigo –

- Ears- Otoscopy, tuning fork tests for hearing, audiometry.
- Eyes-Examine eye movement for saccades, smooth pursuit, and nystagmus (spontaneous and gaze evoked).
- Central nervous system- Examine cranial nerves and look for cerebellar signs.
- Vestibulo-occular reflex testing: By asking patients to read letters on a fixed object while they shake their head from side to side.
- Head-shake test.

- Head-impulse test (rapid dolls).
- Maneuvers which evoke nystagmus, such as the Dix-Hallpike maneuver, are helpful for diagnosing benign paroxysmal positional vertigo (BPPV).

Dix-Hallpike maneuver - This test consists of a series of two maneuvers: with the patient sitting on the examination table, facing forward, eyes open, the physician turns the patient's head 45 degrees to the right (A). The physician supports the patient's head as the patient lies back quickly from a sitting to supine position, ending with the head hanging 20 degrees off the end of the examination table. The patient remains in this position for 30 seconds (B). Then the patient returns to the upright position and is observed for 30 seconds. Next, the maneuver is repeated with the patient's head turned to the left. A positive test is indicated if any of these maneuvers provoke vertigo with or without nystagmus.

NYSTAGMUS

Nystagmus from an acute peripheral lesion is unidirectional, with fast phases beating away from the ear with the lesion. Nystagmus that changes direction with gaze is due to a central lesion.

- Transient mixed vertical-torsional nystagmus occurs in BPPV, but pure vertical or pure torsional nystagmus is a central sign.
- Nystagmus from a peripheral lesion may be inhibited by visual fixation, whereas central nystagmus is not suppressed.
- Absence of a head impulse sign in a patient with acute prolonged vertigo should suggest a central cause.
- Unilateral hearing loss suggests peripheral vertigo. Findings such as diplopia, dysarthria, and limb ataxia suggest a central disorder.

ANCILLARY TESTING

- Audiometry - Unilateral sensorineural hearing loss supports a peripheral disorder (e.g. vestibular schwannoma). Predominantly low-frequency hearing loss is characteristic of Meniere's disease.
- Electronystagmography or videonystagmography.
- Caloric evaluation.
- Neuroimaging - CT Scan, MRI, MRA.

SYMPTOMATIC PHARMACOLOGIC TREATMENT

Medical treatment should be reserved for short term control of active vertigo, such as during the first few days of acute vestibular neuritis or for acute attack of Meniere's disease. One should not prescribe (especially in peripheral causes of vertigo) vestibular suppressant drugs for longer than a few days because they can cause sedation and psychomotor impairment, which hampers central compensation. This central vestibular compensatory mechanism is a faculty of the brain that restores normal balance function after a vestibular assault.

The differential diagnoses of vertigo are discussed in Table 3.

For suppression of vertigo commonly used medications are:-

I. ANTIVERTIGO

A. Vestibular Suppressant

a. Ca antagonist: Flunarizin (5-10 mg daily),

b. Vasodilator: Betahistine (24-48 mg/day),

c. Tranquilizer: Diazepam (2.5 mg 1-3 times daily), Clonazepam (25 mg. 1-3 times daily),

d. Antihistamine: Dimenhydrinate (50 mg. 1-2 times daily), Meclizine (25-50 mg 3 times daily), Promethazine (25 mg. 2-3 times daily, also can be given rectally and IM), Cinnarizine (25 mg. 2-3 times daily),

B. CNS stimulant: Like Ephedrin and Amphetamin - not used much.

II. ANTIEMETIC

A. Anticholinergic: Atropine, Scopolamine (transdermal patch),

B.. Antidopaminergic: Prochlorperazine (5-10 mg. 2-3 times daily), Metoclopramide (10-15 mg. 3-4 times daily).

C. Antihistamine: Dimenhydrinate (50 mg. 1-2 times daily),

III. PSYCHOAFFECTIVE:

A. Clonazepam, diazepam, etizolam, etc. for anxiety and panic attacks.

B. Selective serotonin reuptake inhibitors for psychological vertigo

C. Piracetam a nootropic drug and cyclic derivative of GABA. It is said to have neroprotective and anticovulsant properties, and improves neuroplasticity.

Betahistine is a histamine analog with an agonistic action. It is a H1 receptor agonist and antagonize the h3 receptors. It increases cochlear and cerebral blood flow and regulates firing activity of the vestibular nuclei. Advantage of betahistine is that only non-sedating anti vertigo drug. All other anti-vertigo drugs have some sedative effect, and therefore depress the CNS, which detrimental to the central vestibular compensatory mechanism. Simultaneously it Has a side effect in form of precipitate preexisting bronchial asthma, precipitation of peptic ulcer, etc.

VESTIBULAR REHABILITATION THERAPY

This is a physiotherapy comprising of various eye movements and head exercises which help central adaptation processes that compensate for vestibular loss and also may help habituate motion sensitivity and other

Differential Diagnosis

Disorder	Duration of Episodes	Auditory Symptoms	Clinical Features	Treatment
Peripheral Causes				
BPPV	Seconds	No	Commonest cause of vertigo, brief episodes provoked by change in head position relative to gravity,	Canalith repositioning maneuver (Brandt-Daroff, Epley, Semont),
Labyrinthitis	Days	Yes	Sudden unilateral loss of vestibular function and hearing, nausea & vomiting,	Antibiotics, removal of infected tissue, vestibular rehabilitation,
Vestibular Neuronitis	Days	No	Tendency to fall and vomiting lasting for days,	Brief course of high-dose steroids, vestibular rehabilitation,
Meniere's Disease	Hours	Yes	Episodes of vertigo, tinnitus, feeling of fullness or pressure in the ear,	Low-salt diet, diuretic, surgery, transtympanic gentamicin,
Perilymphatic Fistula	Seconds	Yes	Triggered by sound or pressure changes,	Bed rest, avoidance of straining,
Cerebellopontine Angle Tumours (Acoustic Neuroma, Maningiomas)	Months	Yes	Associated symptoms of 5^{th} and 7^{th} cranial nerve,	Surgery,
Cervicogenic Vertigo	Seconds	No	Vertigo triggered by somatosensory input from head and neck movements,	Conservative, physiotherapy,
Central causes				
Cerebrovascular disease (TIA or stroke)	TIA - seconds to hours, Stroke - days	Usually not	Associated with neurological deficit symptoms,	Control of vascular risk factors, e.g. antiplatelet agents,
Vestibular migraine	Hours	No	Associated with headache, visual aura,	Beta-blockers, calcium channel blockers, tricyclic amines,
Multiple sclerosis	Months	No	Sensory and Motor signs and symptoms disseminated in space and time,	Immunomodulatory drugs,
Cerebellar degeneration	Months	No	Cerebellar sign and symptoms,	Symptomatic,
Cerebellar tumour	Months	No	Cerebellar sign and symptoms,	Surgery,
Other causes				
Anxiety disorder	Variable	Usually not	Personality usually of an obsessive-compulsive, Attack are precipitated by typical situations,	Psycho-educative therapy, desensitization by self exposure, SSRI
Vestibular, Ototoxicity	Months	Yes	History of vestibulo ototoxic drug therapy,	Avoid drugs,

symptoms of psychosomatic dizziness. They should only be practiced after the acute episode is over.

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