DIZZINESS AND VERTIGO

VERTIGO IS DEFINED AS A SENSATION OF DISORINETATION IN SPACE COMBINED WITH A SENSATION OF MOTION

THE DIAGNOSTIC PROCESS IS CONSISTENTLY BASED ON TWO BASIC CONCEPTS → whether the patient has true vertigo and then whether its cause is central or peripheral

PATHOPHYSIOLOGY:

- The maintenance of equilibrium and awareness of the body in relationship to its surroundings depends on the interaction of THREE INPUTS:
    - VISUAL
    - PROPRIOCEPTIVE
    - VESTIBULAR
        - THESE SENSE ORGANS ARE CONNECTED WITH THE CEREBELLUM BY WAY OF THE VESTIBULAR NUCLEI
        - Any disease that interrupts the integration of these three systems may give rise to vertigo and disequilibrium
- Nystagmus occurs when the synchronised vestibular information becomes unbalanced
    - This unopposed activity causes a slow movement of the eyes toward the side of the stimulus, and the cerebral cortex then corrects for these eye movements and rapidly brings the eyes back to the midline
    - The direction of nystagmus is denoted by the direction of the fast, cortical movement
    - If caused by vestibular disease, the nystagmus tends to be unidirectional and horizontal
        - If VERTICAL → a central lesion is usually the cause
- Connections between the vestibular nuclei and the autonomic system account for the associated symptoms of nausea, diaphoresis and vomiting that often accompany attacks of vertigo

DIAGNOSTIC APPROACH:

DESCRIBE THE SENSATION *WITHOUT* USING THE WORD *DIZZY!*

- The sensation of light-headedness is more consistent with presyncope → think arrhythmia, AMI, sepsis, hypovolaemia, drug effects, PE
- Once vertigo established → is it central or is it peripheral

PIVOTAL FINDINGS:

Table 12-1 Characteristics of Peripheral and Central Vertigo

CHARACTERISTIC	PERIPHERAL	CENTRAL
Onset	Sudden	Gradual or sudden
Intensity	Severe	Mild
Duration	Usually seconds or minutes; occasionally hours, days (intermittent)	Usually weeks, months (continuous) but can be seconds or minutes with vascular causes
Direction of nystagmus	One direction (usually horizontorotary), never vertical	Horizontal, rotary, or vertical (different directions in different positions)
Effect of head position	Worsened by position, often single critical position	Little change, associated with more than one position
Associated neurologic findings	None	Usually present
Associated auditory findings	May be present, including tinnitus	None

- Time of onset and duration of vertigo are important clues to the cause
    - If severe, lasts hours and has symptom-free periods suggests peripheral labyrinth disorder
- Presence of auditory symptoms → suggests peripheral cause (i.e. middle or inner ear)
    - Progressive hearing loss over months, think ACOUSTIC NEUROMA
    - Hearing loss, vertigo and tinnitus → TRIAD OF MENIERES
- ASSOCIATED NEUROLOGICAL SYMPTOMS?
    - Ataxia of recent and relatively sudden onset suggests cerebellar haemorrhage, or infarction of PICA/SCA territory
- Prior trauma:
    - Vertigo is common after head injury → labyrinth concussion
    - Neck injury → strain of muscle proprioceptors or vertebral artery injury
- It has been clearly shown that isolated vertigo can be the only initial symptom of cerebellar or other posterior circulation TIA or CVA → risk stratify with age, male sex, HT, CAD, DM, AF → high risk

PHYSICAL EXAMINATION:

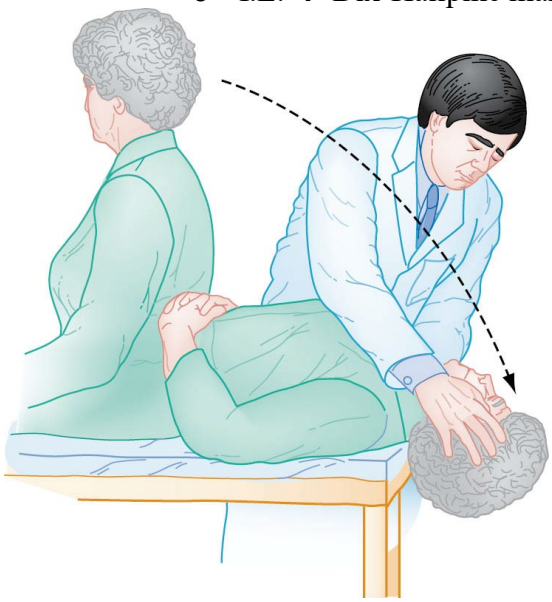
- HEAD AND NECK:
    - Bruits of carotid/vertebral arteries
    - ENT:
        - Perforated eardrum/scarring suggests PERILYMPH FISTULA
        - Impacted wax

- EYES:
  - Sixth cranial nerve palsy may result from early brainstem compression in ICH
  - INO → almost pathognomic of MS, involvement of MLF

Table 12-2 Distinguishing Characteristics of Nystagmus with Central and Peripheral Vertigo

CHARACTERISTIC	CENTRAL	PERIPHERAL
Direction	Any direction	Horizontal or horizontorotary
Laterality	Unilateral or bilateral	Bilateral
Position testing effects:		
Latency	Short	Long
Duration	Sustained	Transient
Intensity	Mild	Mild to severe
Fatigability	Nonfatigable	Fatigable
Effect of visual fixation	Not suppressed, may be enhanced	Suppressed

- POSITIONAL TESTING:
  - I.E. → Dix-Hallpike manoeuvre



- Patient is moved quickly from upright seated position to a supine position and the head is turned to one side and extended to 30 degree beyond horizontal plane
- Use with care in suspected vertebro-basilar insufficiency
- Positive elicitation of symptoms and signs generally indicates vestibular pathology → although sensitivity and specificity NOT GREAT

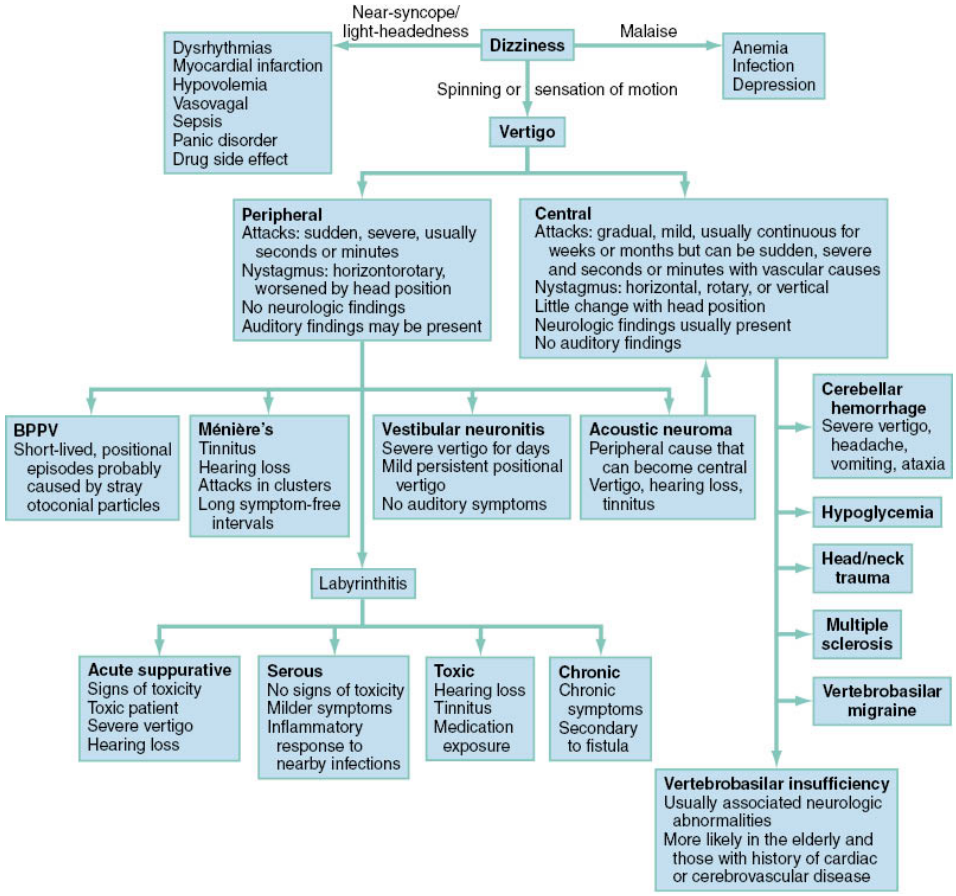
- NEUROLOGICAL EXAMINATION:
  - Presence of cranial nerve deficits suggests a space-occupying lesion in the brainstem or cerebellum
  - EVALUATE FOR CEREBELLAR DYSFUNCTION:
    - DYSMETRIA → inability to arrest a muscular movement at the desired point → past-pointing (finger-nose)
    - DYSDIADOCHOKINESIS → inability to perform coordinated muscular movement smoothly

- GAIT → broad-based, truncal tremor, irregularity of steps, lurching from side-to-side

ANCILLARY TESTING:

- BSL in all cases
- IMAGING:
  - If cerebellar involvement suspected → CT or MRI.
    - MRI particularly useful for diagnosis of acoustic neuroma, demyelinating white matter lesions

DIAGNOSTIC ALGORITHM:



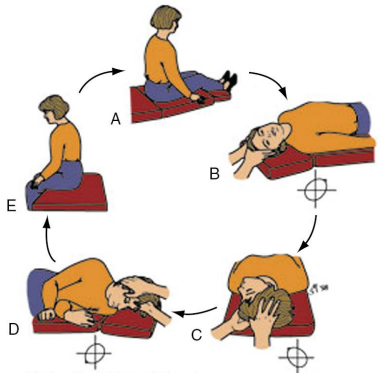
DIFFERENTIAL DIAGNOSIS OF PERIPHERAL VERTIGO:

Table 12-3 Differential Diagnosis of Patients with True Vertigo

CAUSE	HISTORY	ASSOCIATED SYMPTOMS	PHYSICAL
Peripheral			
1. Benign paroxysmal positional vertigo	Short-lived, positional, fatigable episodes	Nausea, vomiting	Single position can precipitate vertigo. Horizontorotary nystagmus often can be induced at bedside.
2. Labyrinthitis			
A. Serous	Mild to severe positional symptoms. Usually coexisting or antecedent infection of ear, nose, throat, or meninges	Mild to severe hearing loss can occur	Usually nontoxic patient with minimal fever elevation
B. Acute suppurative	Coexisting acute exudative infection of the inner ear. Severe symptoms	Usually severe hearing loss, nausea, vomiting	Febrile patient showing signs of toxicity. Acute otitis media
C. Toxic	Gradually progressive symptoms: Patients on medication causing toxicity	Hearing loss that may become rapid and severe, nausea and vomiting	Hearing loss. Ataxia common feature in chronic phase
3. Ménière's disease	Recurrent episodes of severe rotational vertigo usually lasting hours. Onset usually abrupt. Attacks may occur in clusters. Long symptom-free remissions	Nausea, vomiting, tinnitus, hearing loss	Positional nystagmus not present
4. Vestibular neuronitis	Sudden onset of severe vertigo, increasing in intensity for hours, then gradually subsiding over several days. Mild positional vertigo often lasts weeks to months. Sometimes history of infection or toxic exposure that precedes initial attack. Highest incidence is found in third and fifth decades	Nausea, vomiting. Auditory symptoms do not occur	Spontaneous nystagmus toward the involved ear may be present.
5. Acoustic neuroma	Gradual onset and increase in symptoms. Neurologic signs in later stages. Most occur in women between 30 and 60	Hearing loss, tinnitus. True ataxia and neurologic signs as tumor enlarges	Unilateral decreased hearing. True truncal ataxia and other neurologic signs when tumor enlarges. May have diminution or absence of corneal reflex. Eighth cranial nerve deficit may be present.

OTHER THAN ACUTE BACTERIAL LABYRINTHITIS (which needs admission for IV antibiotics and occasionally surgery), treatment of acute attacks of vertifo caused by peripheral disorders is symptomatic

- IV diazepam
- Anticholinergic drugs/antihistamines with anticholinergic activity (diphendhyramine, promethazine)
- Prochlorperazine
- EPLEY MANEOUVRE



- Sequential movements, staying in each position for ~30 seconds
- Very effective in BPPV

CENTRAL CAUSES OF VERTIGO:

Central			
1. Vascular disorders			
A. Vertebrobasilar insufficiency	Should be considered in any patient of advanced age with isolated new-onset vertigo without an obvious cause. More likely with history of atherosclerosis. Initial episode usually seconds to minutes	Often headache. Usually neurologic symptoms including dysarthria, ataxia, weakness, numbness, double vision. Tinnitus and deafness uncommon	Neurologic deficits usually present, but initially neurologic examination can be normal.
B. Cerebellar hemorrhage	Sudden onset of severe symptoms	Headache, vomiting, ataxia	Signs of toxicity. Dysmetria, true ataxia. Ipsilateral sixth cranial nerve palsy may be present.
C. Occlusion of posterior inferior cerebellar artery (Wallenberg's syndrome)	Vertigo associated with significant neurologic complaints	Nausea, vomiting, loss of pain and temperature sensation, ataxia, hoarseness	Loss of pain and temperature sensation on the side of the face ipsilateral to the lesion and on the opposite side of the body, paralysis of the palate, pharynx, and larynx. Horner's syndrome (ipsilateral ptosis, miosis, and decreased facial sweating)
D. Subclavian steal syndrome	Classic picture is syncopal attacks during exercise, but most cases present with more subtle symptoms.	Arm fatigue, cramps, mild light-headedness may be only other symptoms than vertigo	Diminished or absent radial pulses in affected side or systolic blood pressure differentials between the two areas occur in most patients.

- Also consider head trauma, neck trauma, vertebrobasilar migraine, MS, temporal lobe epilepsy, hypoglycaemia