Vertigo

A neurobiological review

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ABSTRACT

Dizziness is one of the most common presenting symptoms in clinical practice. Yet, the meaning of this symptom is patient-dependent and can span from true vertigo due to vestibular dysfunction to syncope or vertebro-basilar stroke. This review addresses the neurobiological background of vertigo and the most common syndrome of benign paroxysmal positional vertigo, with an outline of the approach towards localization and management of the acute vertiginous patient.

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Vertigo is an illusion of movement indicating an imbalance within the vestibular system.[1] Dizziness is a term commonly used by patients visiting physicians at all levels of health care services including primary care, ear, nose and throat, internal medicine, neurology, neurosurgery and psychiatry clinics. It ranks as the ninth most common chief complaint that causes patients to seek medical help.[2] The exact meaning of dizziness for a given patient spans a wide spectrum of possibilities, including true vertigo, unsteadiness, ataxia, nausea, hypersomnolence, altered conscious state, seizure, transient weakness, migraine headache, transient ischemic attacks (TIAs), stroke or combinations of some, or all of these. Acute vertigo may be associated with vestibular neuritis, labyrinthitis, labyrinthine infarct, perilymph fistula or brain stem and cerebellar infarction.[3] Thus, there is a remarkable diversity of the underlying neurological localizations with different pathologies that dictate different management approaches. Hence, the clinician's dilemma is with respect to where, why, what is the problem and how to work it out, and offer the suffering patient the required optimal therapy and appropriate management.

Neurobiological basis. The vestibular system is the oldest phylogenetic balance system in the animal kingdom.[4,5] In man, it is part of the inner ear and made up of the membranous labyrinth (**Figure 1**),[6] that occupies the bony labyrinth space in the petrous portion of the temporal bone of the skull. Each membranous labyrinth is a system of small cavities (utricle and saccule) and 3 semicircular canals containing a dense fluid known as the endolymph and surrounded from outside with a thinner fluid the perilymph. The basic receptor cells in the system are hair cells that are made up into complex receptor structures, named the macula or the otolithic organ in the utricle and saccule and the cupola in the dilated portion of each semicircular canal known as the ampulla. Each of the 3 semicircular canals (lateral, anterior and posterior) is disposed at right angles to each other. The main function of the vestibular system is the control of the position of the head in space, that in turn will be the "spatial reference" for controlling the body posture and balance in space.[7] Functionally the utricle and saccule are considered as the static system that monitors linear acceleratory displacements of the

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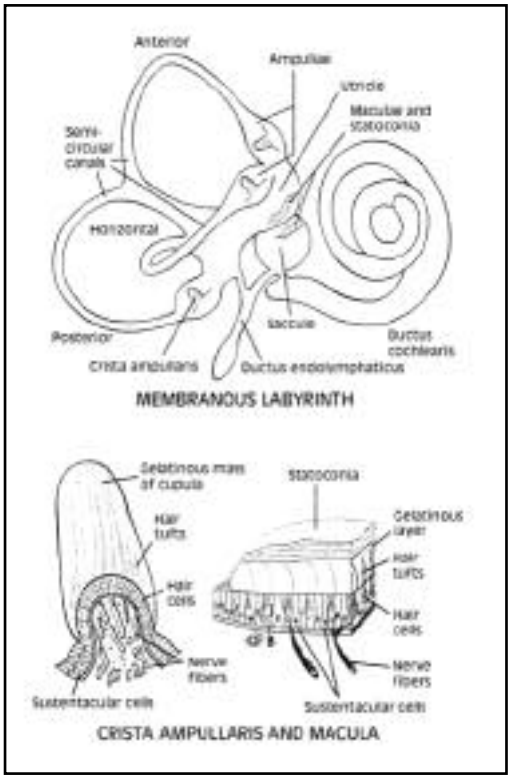

Figure 1 - Membranous labyrinth and organization of the crista ampullaris and the macula (modified from Goss CM: Gray's Anatomy of the human body. Lea & Febiger, Philadelphia).[6]

Table 1 - Useful clinical characteristics for localization in vertigo.

Characteristics	Peripheral (BPPV)	Central
Symptoms		
Precipitating factors	Head movement	Spontaneous
Latency-to-onset	Present	Absent
Duration	Short	Long
Frequency	Paroxysmal	Continuous
Nausea and vomiting	Severe	Variable
Imbalance	Mild to moderate	Severe
Severity	Moderate to severe	Mild
Hearing loss	Common	Rare
Tinnitus	May be present	May be present
Signs		
Provocation procedures	Yes	Spontaneous
Nystagmus	Unidirectional inhibited by fixation	Changing directions Not inhibited by fixation
Compensation	Rapid	Slow
Focal neurological signs	Rare	Cranial nerves, ataxia motor, sensory deficits
Pathology	Otolithiasis	Structural brainstem lesion
Treatment	Symptomatic	Symptomatic + cause if feasible

BPPV - Benign paroxysmal positional vertigo

Table 2 - Useful drugs for symptomatic treatment of vertigo.

Drug group	Generic name	Trade name	Route	Dose	Clinical effect Anti-vertiginous	Anti-emetic
Antihistamines	Dimenhydrinate	Dramamine	Oral	50mg q4-6hr	Moderate	Mild
	Cyclizine	Marezine	Oral/IM/IV	50mg q8hr	Moderate	Mild
	Meclizine	Antivert	Oral	25-50mg q8hr	Moderate	Mild
	Cinnarizine	Stugeron	Oral	25mg q8hr	Moderate	Mild
	Promethazine	Phenergan	Oral/IM suppositories	25mg q6hr 50mg q12hr	Moderate	Moderate
Benzodiazepines	Diazepam	Valium	Oral IV	2-10mg q12hr 5-10mg q4hr	Major	Mild
	Lorazepam	Ativan	Oral IV	1-2mg q8hr 2mg	Major	Mild
Anticholinergic	Scopolamine	Transderm	Patch	1 patch q3d	Moderate	Mild
Phenothiazines	Prochlorperazine	Stemetil Compazine	Oral IM/IV Suppositories	5-10mg q6hr 25mg q12hr	Mild	Major
	Chlorpromazine	Largactil Thorazine	Oral IM Suppositories	10-25mg q6hr 50-100mg q6-8hr	Mild	Major
Benzamides	Metoclopramide	Maxolon	Oral/IM/IV	5-10mg q4-6hr	Mild	Major

IM - intramuscular, IV - intravenous, q - every, hr - hour

head; whereas the 3 pairs of semicircular canals form the dynamic system, that monitors the angular or rotational acceleratory displacements of the head. In microgravity, gravitational acceleration to the otolith receptors is reduced by approximately 1000-folds.[8] The afferent synchronous output from the vestibular components resolves the displacements in the position of the head into the 3 spatial planes of antero-posterior, medio-lateral and superior-inferior planes. Substance P has been recently implicated as a neurotransmitter in the peripheral vestibular system.[9] The vestibular function is a major contributor to the reflex mechanisms controlling body posture that include, in addition, the visual and proprioceptive inputs. However, there is mounting evidence that the vestibular input is required for the resolution of the ambiguous visual and somatosensory reference and it plays the dominant role in the sensorimotor control of posture on earth.[7] The 3 afferent inputs, therefore, work synergistically for the overall maintenance of body posture but functional impairment of any of these 3 inputs will compromise the control of body posture in certain situations. Thus, impairment of the vestibular function will be most hazardous for the patient when the other proprioceptive and visual mechanisms are no more available, for example by disease or during submerged underwater diving. The main presenting symptom of disturbed vestibular function is vertigo and the main objective of the practicing physician is to identify true vertigo that indicates peripheral or central vestibular dysfunction.

Manifestations of vestibular dysfunction. Peripheral vestibular dysfunction exemplified by benign paroxysmal positional vertigo (BPPV), is one of the most common causes of episodic dizziness with an incidence of 64/100,000 patients per year,[10,11] which increases with the age of the patient.[2,12,13] The pathogenesis of BPPV is still not very well defined and its cause is most likely a disorder of otoconic metabolism leading to utriculolithiasis and subsequently canalithiasis.[14] The patient typically presents with a complaint of vertigo that lasts less than one minute and occurs after head movements with respect to gravity, for example sitting up from a lying position or looking up or bending down.[2] The attacks are usually worse in the morning due to the aggregation of the otolithic debris with little head movement during sleep. The condition may be precipitated by mild head trauma or upper respiratory tract infection but it is self-limited to 2 weeks and can be recurrent in approximately 20% of patients. **Table 1** gives some useful characteristic features for peripheral and central vertigo. The other recognized diseases that cause prolonged vertigo include vestibular neuritis, labyrinthitis, labyrinthine infection, perilymph fistula, basilar migraine, Meniere's disease, drug ototoxicity and vertebro-basilar insufficiency. The relevant investigations that may be useful for making definitive diagnoses include audiometry, electronystagmography, brainstem auditory evoked response, cranial MRI and screening for risk factors of cerebrovascular disease.

Management of acute vestibular dysfunction. **Table 2** shows the various drugs that are commonly used for symptomatic treatment of vertigo. Although BPPV is a self-limiting condition, it tends to be less responsive to medications. Thus, the use of Epley's procedure[15,16] for canalith repositioning has demonstrated good efficacy in providing rapid and long-lasting relief of symptoms.[2,17] However, the overall recurrence rate in a recent study was 37% at 60 months with higher rates (50%) for patients with horizontal canal BPPV than those with posterior canal (26%) disorder.[17]

Dizziness remains one of the most common presenting symptoms in clinical practice and the onus on the clinician is to identify first, whether it is vertiginous in origin and then whether it is of peripheral or central localization in order to be really effective in the management of the patient.

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