Vertigo Management - An overview for Oto-laryngologists

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Subject : ENT [OTO RHINO LARYNGOLOGY]

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Abstract

Aim : To present an overview of vertigo management for otolaryngologists

Patients and Methods: Cross sectional study was done and results were charted over a period of 18 months at the Department of ENT [Oto-laryngology] ,Bhaskar Medical College , Telangana ,India . A sample of 100 Patients presenting with vertigo ,central causes ruled out ,sent to Department of ENT were evaluated using the vertigo examination protocols ,followed by performing diagnostic maneuvers for each semi circular canal and is followed by canal repositioning maneuvers . Other cases like vestibular neuritis ,meniere's disease were diagnosed by clinical features and investigations.

Results : 100 vertigo cases were selected using eligibility criteria.

Among them, 91% were aged 20 to 60 years, with 64% male and 36% female. Tuning fork tests showed that 82% were normal, 3% had conductive hearing loss (CHL), and 15% had sensorineural hearing loss (SNHL). Additionally, 73% had abnormal vestibular function tests (VFT); 31% tested positive for Romberg's test on the

left side while 47% were positive on the right side. Cranial nerves (CN) were normal in all 100 cases, and nystagmus was present in 86% of cases. The Dix-Hallpike Maneuver was positive in 57% of cases and success rate ranging from 77-97%, while Epley and Semont Maneuvers showed improvement in 58% of cases and success rate ranging approximately 72-98%. Sensorineural hearing loss was identified in 2% of cases via pure tone audiometry (PTA). In this study most of the patients improved with maneuvers and did not require review for the same. The most common diagnosis for vertigo was right posterior canal benign paroxysmal positional vertigo (BPPV) at 35%. Other differential diagnoses included labyrinthitis, left lateral canal BPPV, left posterior canal BPPV, Meniere's disease, right anterior canal BPPV, right lateral canal BPPV, vestibular migraine, and vestibular neuronitis.

Conclusion: Our study concludes that the most common age group affected by peripheral vertigo is between 20 and 60 years, more in male. This indicates that the prevalence of vertigo increases in the later stages of life, potentially compromising the quality of life for elderly individuals. The most frequent causes of peripheral vertigo in our study area include benign positional paroxysmal vertigo, vestibular neuritis, vestibular migraine, and Meniere's disease.

As a part of Neurotological practice, management of BPPV forms the most important chunk followed by others. Various manoeuvres based on the involved canal are to be practised for repositioning of the debris into the utricle. symptomatic management of various other differential diagnosis have to be understood to be a successful vestibular practitioner. Our study highlighted the same.

- **Key words**: Vertigo , Benign paroxysmal positional vertigo , Dix hallpike maneuver , Epleys maneuver

INTRODUCTION

Vertigo is defined as an illusion of either oneself or the environment rotating, is a reliable symptom. It is a common clinical symptom and is usually described as a rotational, spinning movement. It indicates involvement of the angular motion sensing system, i.e., the semi-circular canals and their central projections. (1)

It is estimated that about 30% of people will need medical care once in their life due to this symptom. (2) The rise in ageing population worldwide is likely to increase these

percentages since dizziness occurs in almost 50% of people over the age of 75 years. BPPV lifetime prevalence of 2.4%. In studies of both young adults and elderly a prevalence of 9% has been described.(4)

It is a demographic study to know the recent trends and newer diagnostic modalities with latest measures and diagrams.

Vertigo, anxiety and stress tend to be concomitant, and attacks of vertigo seem to produce increased levels of stress related hormones. [2] The vestibular system includes the parts of the inner ear and brain that process the sensory information involved with control of balance and eye movements.

Vertigo results from lesions in diverse locations such as inner ear, the deep stretch receptors of the neck, and the visual or vestibular interaction centres in the brainstem.

[5]

There are two broad categories of vestibular disorders that include the central vestibular disease and the peripheral vestibular disease. The peripheral vestibular Peripheral vertigo is of sudden onset and characterized by episodic attacks. Vestibular symptoms like nausea and vomiting are a common characteristic of peripheral vertigo.

The site of the lesions in vertigo is identified by history and associated symptoms. Vertigo with abnormal caloric response together with hearing loss signifies a peripheral pathology. [5] In addition to vertigo the lesions of VIII cranial nerve produces auditory symptoms like hearing loss, tinnitus, and sensations of fullness in the ear or pain in the ear. [6]

The incidences of peripheral vertigo are very high as compared to central vertigo which occurs only in 10% of the individuals. [6] Because of the high

frequency of illness it has been proposed that the cause of peripheral vertigo is mostly acquired.

Approximately 80 percent of vertigo is peripheral, whereas 20 percent is central. Peripheral vertigo is most commonly due to a benign process, with benign paroxysmal positional vertigo (BPPV) being the most common cause of peripheral vertigo. The exposure of neurotropic viruses [herpes family] was found to be high in population studies. [8]

As the importance of diagnosis and management of vertigo is in an increasing note by the neurotologist, and with the addition of newer diagnostic modalities like VIDEO NYSTAGMOGRAPHY, we tried to study various clinical presentations and differential diagnosis of peripheral vertigo.

Rationale of the study

Various presentations of vertigo, differentiate central vertigo from peripheral vertigo by performing different vestibular and cerebellar function tests,
Various diagnostic and canal repositioning maneuvers were meant to be understood in depth.

VERTIGO- An overview :

Vertigo is a common presenting complaint in primary care offices and emergency departments. It is a symptom of vestibular dysfunction and has been described as a sensation of motion, most commonly rotational motion. It is important to differentiate vertiginous symptoms from other forms of dizziness, such as lightheaded, which is most often associated with pre-syncope. [10]

Dizziness- the feeling that everything is turning around you and that you are not able to balance.

Giddiness- the feeling that everything is moving and that you are going to fall.

Lightheaded- not completely in control of your thoughts or movements; slightly faint.

Presyncope - feeling like one was going to pass out but without actual loss of consciousness

Vertigo affects all ages. In younger patients, middle ear pathology is most often the cause. In the elderly, specific assessment is needed due to the risk of falls and their complications. [12]

- **Etiology:**

Vertigo is most often caused by a dysfunction in the vestibular system from a peripheral or central lesion. [10]

Peripheral etiologies include the more common causes of vertigo, such as benign paroxysmal positional vertigo (BPPV) and Ménière disease. [12] BPPV results from calcium deposits or debris in the posterior semi-circular canal and causes frequent transient episodes of vertigo lasting a few minutes or less. [10]

Unlike BPPV, the patients with Ménière disease often experience tinnitus, hearing loss, and aural fullness in addition to vertigo. Endolymphatic hydrops is a distinct pathologic feature of Ménière disease. [13] Symptoms of Ménière disease result from an increased volume of endolymph in the semi-circular canals. Two additional distinct causes of peripheral vertigo include acute labyrinthitis and vestibular neuritis. Both arise from inflammation, often caused by a viral infection. [10]

Another viral-induced cause of vertigo includes Herpes zoster oticus, also known as Ramsay Hunt syndrome. [14]

In Ramsay Hunt syndrome, vertigo results from reactivation of latent Varicella-zoster virus (VZV) in the geniculate ganglion leading to inflammation of the vestibulocochlear nerve. The facial nerve is often involved as well, resulting in facial paralysis. (10)

Less common peripheral causes include cholesteatoma, otosclerosis, and a Peri lymphatic fistula. Cholesteatomas are cyst-like lesions filled with keratin debris.(15) Cholesteatomas most often involve the middle ear and mastoid. Otosclerosis is characterized by abnormal growth of bone in the middle ear, which leads to conductive hearing loss and may affect the cochlea, also causing tinnitus and vertigo. (16) A peri lymphatic fistula is another less common cause of peripheral vertigo and results from trauma. (10)

Central etiologies of vertigo should always be considered in the differential. Ischemic or haemorrhagic strokes, particularly involving the cerebellum or vertebrobasilar system, are life-threatening and must be ruled out by history, physical and other diagnostic tests if warranted. (10,17) Other more serious central causes include tumors, particularly those arising from the cerebellopontine angle. (18) Examples of such tumors include a brainstem glioma, medulloblastoma, and a vestibular schwannoma, which can lead to sensorineural hearing loss as well as vertiginous symptoms. (10) Tumours can cause vertigo. Schwannoma is the most common lesion in the cerebellopontine angle. (23)

Meningioma is the most common extra-axial tumour in adults. It is the second most common lesion in the cerebellopontine angle. Glomus jugulare and glomus jugulo-tympanicum are tumours of the chemoreceptor system and are the main primary

tumors of the jugular foramen. Metastases should be a consideration in patients with known primary neoplasia or multiple brain lesions. Infectious causes should be considered. Viral labyrinthitis is the most common example. Oto-mastoiditis is an infection of the tympanic and mastoid cavities. It is typically caused by bacterial agents with the most common being Streptococcus pneumoniae and Haemophilus influenzae. Acute cerebellitis is encephalitis that is restricted to the cerebellum. It is most common in children. Varicella-zoster virus is the leading cause. Cholesteatoma can be acquired or congenital, occurring in the pars flaccida or pars tensa. It is the proliferation of keratinized stratified squamous epithelium. (24)

Vestibular migraines are a common central cause of vertigo. They are characterized by unilateral headaches associated with other symptoms, including nausea, vomiting, photophobia, and phonophobia. Finally, multiple sclerosis has been associated with both central and peripheral causes of vertigo. Centrally, multiple sclerosis can cause vertigo with the development of demyelinating plaques in the vestibular pathways. (19) BPPV is a common peripheral cause of vertigo in patients with multiple sclerosis. (10)

Other causes can lead to vertigo. These include medication-induced vertigo and psychologic disorders, including mood, anxiety, and somatization. Medications that have been associated with vertigo include anticonvulsants such as phenytoin and salicylates. (10)

- Acute unilateral deafness
- Acute (occipital) headache
- Any central symptoms or signs
- A negative (normal) head-impulse test

Table 1: Seemungal and Bronstein - Red flags for Brain Imaging in Acute vertigo (54)

- **Epidemiology**

Vertigo affects both men and women but is about two to three times more common in women than men. [10] It has been associated with various comorbid conditions, including depression and cardiovascular disease. Prevalence increases with age and varies depending on the underlying diagnosis. Based on a survey of the general population, the 1-year prevalence of vertigo is about 5% and an annual incidence of 1.4%. Dizziness including vertigo affects about 15% to over 20% of adults yearly. [20] For benign paroxysmal positional vertigo, the one-year prevalence is about 1.6%, and it is less than 1% for vestibular migraine. The impact of vertigo should not be underestimated as nearly 80% of survey respondents reported an interruption in daily activities, including employment and the need for additional medical attention. The prevalence of Menière's disease has been recently reported to be 0.51% which is much higher than previous reports. [20,21] Menière's disease prevalence of 43.2 per 1,00 000 persons and an incidence of 4.3 definite Ménière's disease per 1,00 000 persons per year was reported (1992–1996). Only 1–7% of Ménière's disease cases are seen in the paediatric population.

BPPV estimated lifetime prevalence of 2.4%. In studies of both young adults and the elderly a prevalence of 9% has been described. Benign paroxysmal positional vertigo (BPPV) is probably the most common cause of vestibular vertigo accounting for approximately 20–30% of diagnoses in specialised dizziness clinics

In the adult population, around 40% of all dizziness is caused by a peripheral vestibular pathology (which includes the vestibular end organs of the inner ear and/ or the vestibular nerve).

Vestibular migraine: A high prevalence of VM (~30%) is reported among Indians suffering from dizziness.

- **Patho- physiology**

Asymmetry in the vestibular system accounts for the symptom of vertigo. Asymmetry may result from damage or dysfunction in the peripheral system, such as the vestibular labyrinth or vestibular nerve or a central disturbance in the brainstem or cerebellum. [10] Though there may be a permanent vestibular disturbance, the symptom of vertigo is never permanent as the central nervous system adapts over days to weeks. [22]

In BPPV otoconia from the utricle are thought to collect in the semi-circular canals, making them abnormally gravity-sensitive. The net result is that changes in head position with respect to gravity result in an abnormal displacement of the cupula and stimulation of the corresponding vestibular afferents. This results in the characteristically abnormal eye movements and vertigo.

There are two theories: Cupulo-lithiasis proposes that degenerative otoconia stick to the cupula making it gravity-sensitive. The more recent theory of canalo-lithiasis suggests that degenerative otoconial debris float freely in the endolymph of the semi-circular canal. When exposed to gravity, the otoconia fall to the lowest part of the canal, causing a change in endolymph pressure with subsequent displacement of the cupula. The change in pressure is due to a hydrodynamic ‘plunger effect’ where the otoconial debris act like a piston creating endolymph flow which deforms the cupula. This theory explains the ‘latency’ (delay in onset) of the nystagmus that is related to the time taken for the otoconial debris to fall and the initial adherence to the membranous canal. [4]

Ménière's disease: Based on a large human temporal bone study, Merchant et al. suggested that the endolymphatic hydrops observed in Ménière's disease might be a marker for disordered inner ear homeostasis in which a yet unknown factor produces both the clinical symptoms of Ménière's syndrome and endolymphatic hydrops. Potential etiological or precipitating conditions include genetic variations, infection, vascular risk factors, diet, allergy, autonomic, endocrine and autoimmune factors. (35)

Vestibular neuritis: A selective inflammation of the vestibular nerve is the most favourable aetiology. Inflammation of a nerve may cause demyelination and associated loss of function, which can be irreversible. Temporal bone studies have shown evidence of chronic inflammatory changes of the vestibular nerve with diffuse perivascular lymphocytic infiltrates, consistent with post-infectious inflammatory changes. These changes can be seen months to years after the initial acute episode.

Vestibular migraine: Theories about the pathology of VM derive from theories of pathogenesis of migraine. Migraine is considered a disorder of sensory modulation, involving both neural and vascular tissue. Many theories of migraine pathogenesis centre on the 'trigemino-vascular reflex', with aminergic brainstem nuclei activating trigeminal afferents responsible for the pain of migraine headache that is a central characteristic of the disorder. Migraine is well recognized as a familial disorder and there are documented pedigrees of families with VM.

Feature	Peripheral vertigo	Central vertigo
Nystagmus		
• Type	Combined horizontal and torsional	Purely vertical (most common), horizontal, or torsional
• Direction	One direction	May change direction
• Visual fixation	Inhibits	No change
• Fatigable	Yes	No
• Latency	Present	Absent
Imbalance (in acute conditions)	Mild to moderate but able to walk	Severe and unable to stand or walk
Nausea and vomiting	Usually present and severe	Varies
Hearing loss, tinnitus	Common	Rare
Neurologic symptoms (motor and sensory deficiencies, ataxia, Horner's syndrome)	Absent	Common
Recovery	Begins within days	Slow
Head thrust sign	Present	Absent
Common causes	Benign paroxysmal positional vertigo, vestibular neuritis, Ménière's disease, trauma to labyrinth, infection and drugs	Migraine, vertebrobasilar insufficiency, cerebrovascular accidents, multiple sclerosis, brain tumors and cerebellar disorders

Table 2: Distinguishing characteristics of peripheral and central vertigo [55]

PERIPHERIAL VERTIGO

Approximately 80 percent of vertigo is peripheral, whereas approximately 20 percent is central. Peripheral vertigo is most commonly due to a benign process; benign paroxysmal positional vertigo (BPPV) is by far the most common cause of peripheral vertigo. In contrast, central vertigo often indicates more serious pathology. Peripheral vertigo typically presents with acute, severe episodes. Peripheral vertigo usually is made worse with head movements and is generally associated with horizontal/rotary nystagmus, which is fatigable and unidirectional.

A disturbance of the vestibular system, semi-circular canals, or cranial nerve 8 is the underlying issue. This disturbance could be related to damage to one of these organs or simply confused neuronal input. It is important to remember that the central nervous system receives inputs bilaterally from these structures/systems, assembles the input and then, forms a response. The CNS also coordinates these bilateral

inputs with our visual and sensory inputs creating an overall picture of whether we are moving in space/time or if the environment around us is moving. Suffice it to say that conflicting inputs from these various symptoms overwhelm the central nervous system causing dizziness, nausea, and the perception of movement. [25,26]

Vertigo breaks down into two types: peripheral and central. As the main focus of this review is on peripheral vertigo, we shall have a cursory overview of central vertigo to help distinguish between the two. Usually, peripheral vertigo is, although not always, due to a benign process, whereas central vertigo often indicates a more serious pathology.

Etiology of Peripheral Vertigo are following:

- Benign paroxysmal positional vertigo (BPPV)
- Vestibular neuritis
- Meniere disease
- Vestibular migraine
- Labyrinthitis
- Herpes zoster
- Otitis media
- Aminoglycoside toxicity
- Peri-lymphatic fistula
- Viral infections
- Acoustic neuroma
- Cogan syndrome

Benign Paroxysmal Positional Vertigo:

It is classically described as a sudden onset of spinning brought on by a rapid head movement or a quick turn in bed before getting up. There is no associated ear pain, tinnitus, or hearing loss. The pathophysiology behind this is usually the displacement of otolith or calcium debris located in the posterior semicircle canal. This type of vertigo classically can be made worse with the Dix Hallpike maneuver and subsequently fixed with the Epley maneuver by relocating these otoliths. Other less commonly used maneuver include Semont, Lempert.

Vestibular neuritis: It is usually a post-viral inflammatory syndrome. Patients typically develop rapid, severe nausea, vomiting, vertigo, and gait instability. Despite gait instability, patients are still able to ambulate. They display the typical peripheral vertigo physical findings discussed below. If there is an associated unilateral hearing loss, it is then called labyrinthitis. Often, due to the severity of the symptoms, this can be confused with a central process. Consequently, magnetic resonance imaging is performed if clinician suspicion is high to aid in the diagnosis.

Meniere's disease: Excess endolymphatic fluid causes Meniere's disease. The excess pressure causes inner ear dysfunction. Patients present with episodic unilateral tinnitus, hearing loss, nausea, vomiting, gate instability, and vertigo. Audiometry testing demonstrating a low sensorineural hearing loss can aid in diagnosis.

Cogan syndrome is an autoimmune process that presents with symptoms similar to those of Meniere disease, so it seems relevant to mention (even though not one of the more comm on causes). Caloric testing usually demonstrates absent vestibular

function.

Clinical features of peripheral vertigo

Peripheral vertigo is described as dizziness or a spinning sensation. Other symptoms associated with peripheral vertigo include:

- Loss of hearing in one ear
- Ringing in one or both ears
- Difficulty focusing vision
- Loss of balance

The diagnosis and workup consist of taking a very accurate and detailed history along with symptomatic/physical findings. Peripheral vertigo is typically episodic and acute/severe. Alternatively, central vertigo typically is over a longer duration of time, and “most” of the time, less severe symptoms occur. Peripheral vertigo usually can be made worse with head movements and typically has been associated with horizontal/rotary nystagmus, which is fatigable and unidirectional.

Central vertigo can have nystagmus in any direction is not fatigable and typically multidirectional.

Caloric test: The principle of the caloric test is that changes in temperature in the external auditory canal influence the level of activity of the vestibular labyrinth.

In the conventional procedure, two temperatures are used, one above and one below body temperature. Water irrigation at 30 °C and 44 °C (37 ± 7 °C) is the standard technique. In the original description by Fitzgerald and Hallpike, the procedure follows the order left cold, right cold, left warm, right warm, for standardization purposes, with each irrigation lasting 40 sec onds,49 but this order is not essential. Temperature in the temporal bone has to be minimally stable before the next irrigation, so at least 5 minutes since the end of the previous irrigation has to be allowed for.

cold irrigation induces horizontal nystagmus beating in the opposite direction of irrigation, and ipsilaterally during warm irrigation (cold-opposite-warm-same (COWS)). In this way, left cold and right warm irrigation induce right-beating nystagmus, and vice versa. (33)

The Dix Hallpike test : can help aid in the diagnosis of posterior semi- circular canal BPPV, typically making symptoms worse and nystagmus more obvious.

Figure 4: Dix-Hallpike test [32]

Repositioning manoeuvre for PSCC:

Epley's manoeuvre: Epley developed the canalith repositioning procedure (CRP) in 1992 based on the theory of 'canalo-lithiasis' in order to move the particles from the posterior canal into the utricle via the common crus.

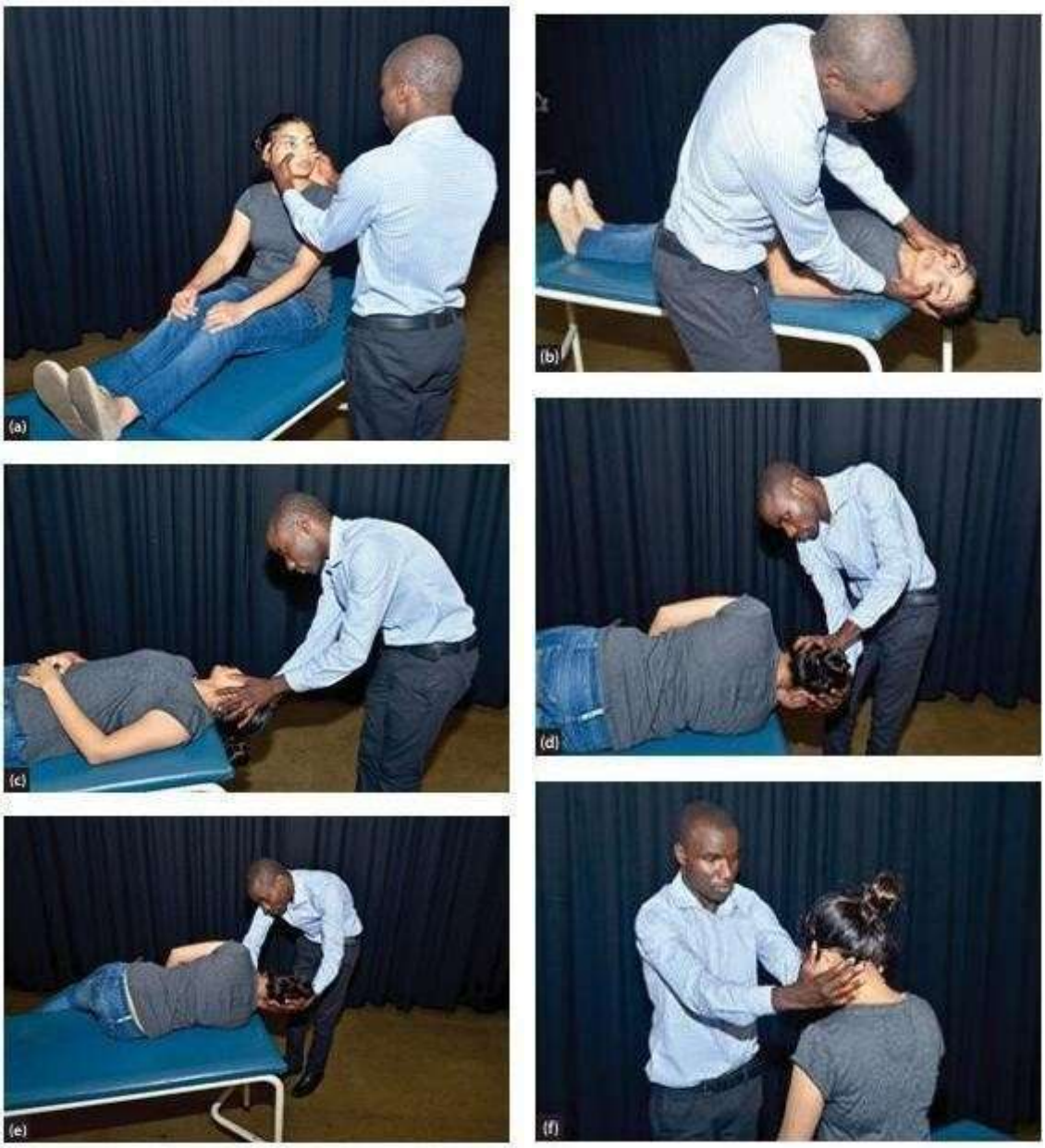


Figure 5: Epley's Canal Repositioning manoeuvre [31]

Semont manoeuvre: SM is performed with the patient sitting in the upright position with the head turned by 45 degrees toward the unaffected ear and quickly leaned to the affected side. After a waiting time, the patient is then rapidly tilted to the unaffected side still with head turned by 45 degrees toward the unaffected ear

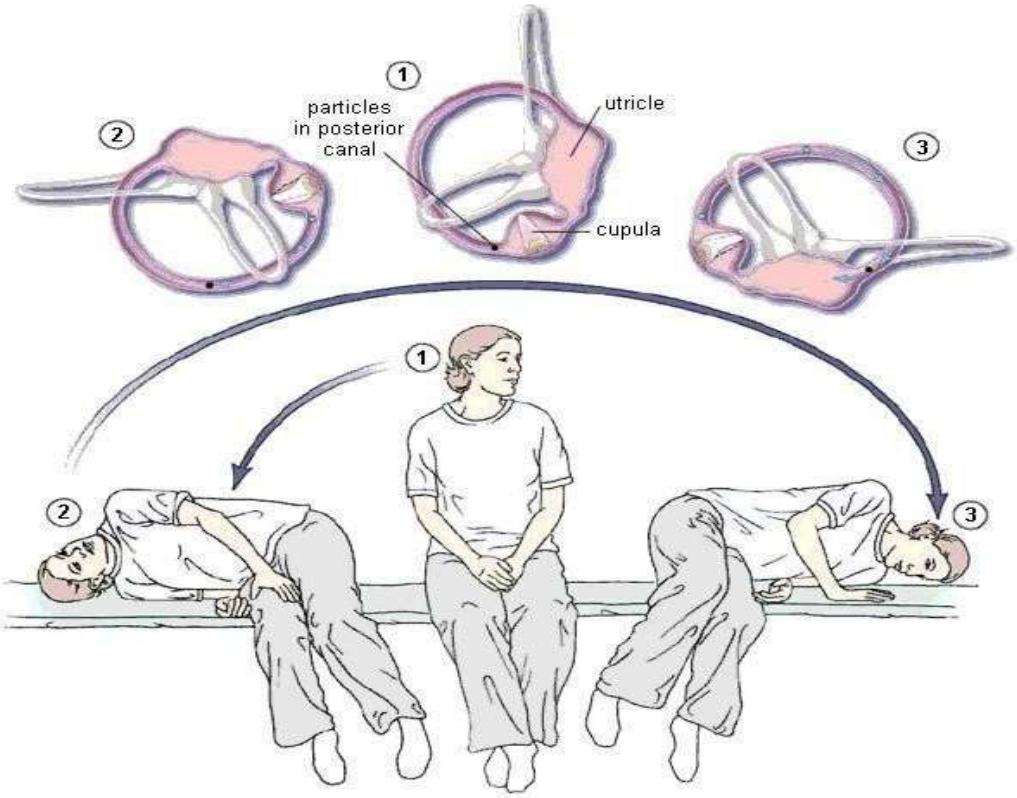


Figure 6: Semont maneuver

Brandt Daroff exercise: Patients were made to lie on their side rapidly, sit up, lie on the opposite side, and then sit up again. Each position was maintained for at least 30sec, and repeated serially 10 times. The patients were instructed to perform this exercise themselves at home three times daily for a week.

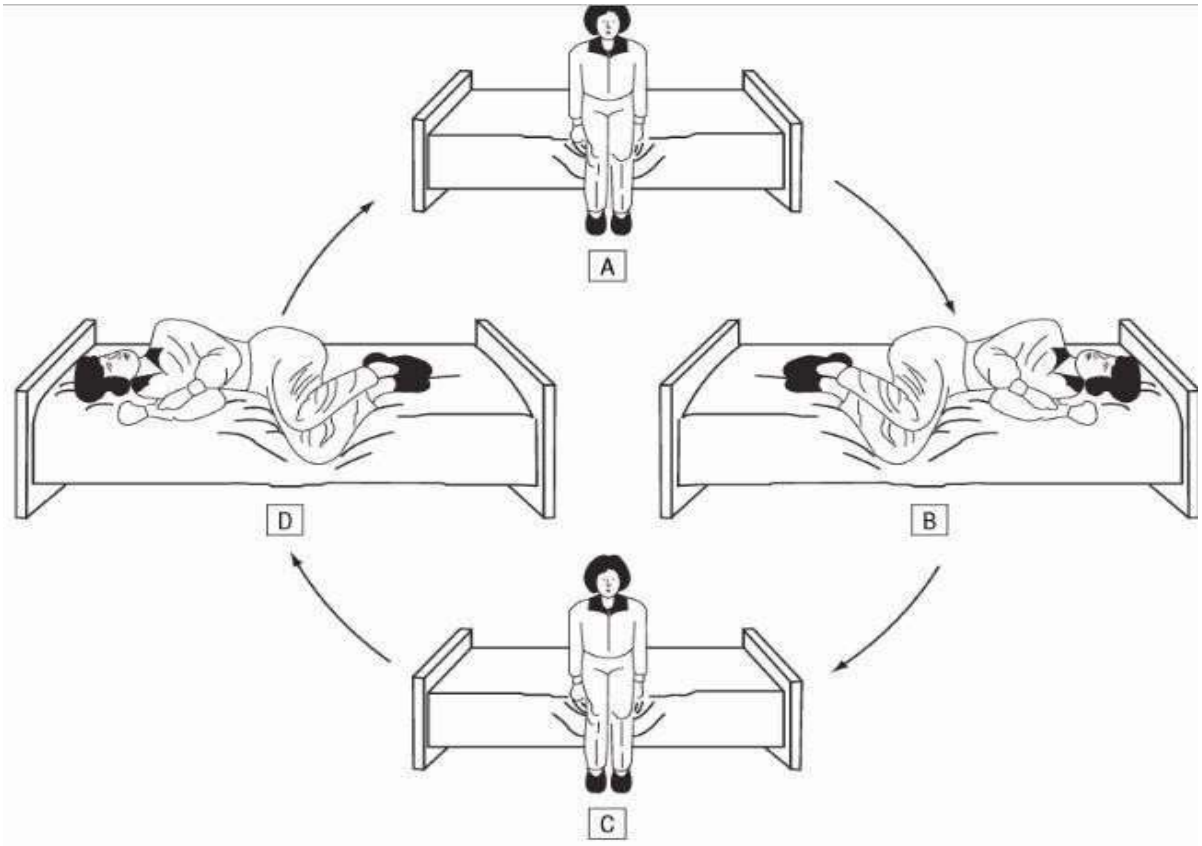


Figure 7: Brandt Daroff exercise LSCC

Therapeutic tests for Lscc:

Gufoni manoeuvre: In geotropic HC-BPPV, the patient quickly lies down on the side of the unaffected ear and remains in this position for 1-2 min, until the evoked nystagmus subsides. The head is then quickly rotated 45° toward the floor and kept in this position for another 2 min, after which the patient resumes an upright position.

In apogeotropic HC-BPPV, Gufoni Manoeuvre entails rapid positioning of the patient starting from the sitting position onto the lesion side and remains in this position for 1-2 min, until the evoked nystagmus subsides, then quickly turning the patient's head 45° upwards and kept in this position for another 2 min, after which the patient resumes an upright position

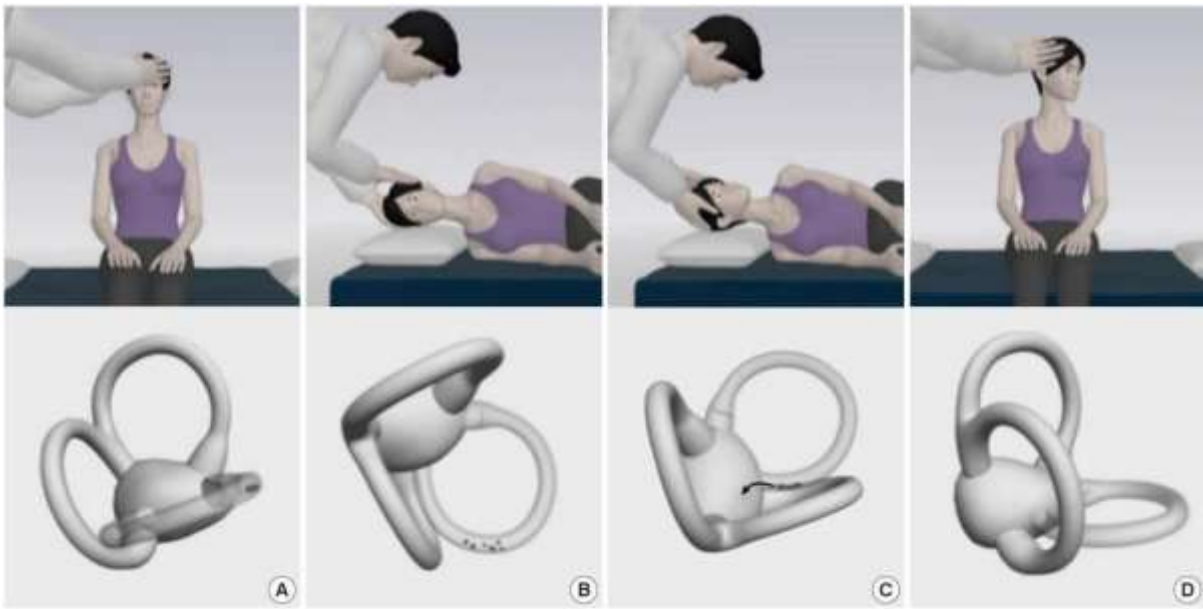


Figure 8: Gufoni manoeuvre

Lempert / Barbeque maneuver: turning the patient's head initially and then the body from the supine position in three 90-degree-step rotations (total 270 degrees) towards the unaffected ear. The body will eventually assume the prone position with the

affected ear facing down, following which the patient will sit up. The rotation is

performed within half a second and the head positions are maintained for 30- 60 seconds.

Figure 9: Lempert / Barbeque manoeuvre

Superior SCC diagnostic tests:

Supine straight head extension test: The simulation shows that in the deep head-hanging position, there is ampullo-fugal movement of the debris, which leads to an excitation of the anterior canal. This causes a downbeat nystagmus with torsion toward the side of involvement when the individual looks straight ahead. This implies that the supine head-hanging test is useful for the diagnosis of both anterior canals.

In the second step, when the subject comes back to the sitting position, the debris moves further toward the utricle (continuing the ampullo-fugal movement) and not back toward the ampulla. This explains why there is no inversion of nystagmus when the subject returns to sitting position and the natural remission.

Diagnostic tests:

Yacovino manoeuvre: As the anterior canal lies in the vertical plane, the head should remain straight on starting the manoeuvre. In the next step, the head of the subject is taken down to 30° below the horizontal plane. This inverts the anterior canal such that the ampullary arm lies at the most superior position, whereas the non-ampullary arm is placed inferiorly. The otolith debris move ampullo-fugally to reach the most dependent position in the canal. Next, the subject is taken to the chin- to-chest position. This takes the debris further ahead in the canal. However, the simulation shows that at this point, there is a risk that the debris enters the posterior canal, leading to a canal switch. In the final step, the subject sits up and bends the head forward, leading to the debris being repositioned to the utricle.

Figure1

0: Yacovino manoeuvre

Other specialized tests include:

- Electro/ Video-Nystagmo-Graphy (ENG)

Management

Treatment usually involves giving the body time to heal and treatment of the underlying process. There is some data to suggest, antihistamines, benzodiazepines, corticosteroids, antiemetics, and anticholinergic's may be of use depending on the etiology of peripheral vertigo. Vestibular rehabilitation therapy (VRT) may also offer relief to some patients. Vestibular rehabilitation therapy is a form of physical therapy that takes advantage of the plasticity of the brain using specialized exercises and head movements to help gaze and gait stabilization. [27,28]

The prognosis for peripheral vertigo is typically quite favorable. It may lead to some morbidity; however, following correct identification of the etiology is correctly identified, symptoms can usually be quite tolerable if not completely resolved.

AIM AND OBJECTIVES

Aim

To study various clinical presentations and differential diagnosis of peripheral vertigo.

Objectives

1) To examine patients with various presentations of vertigo
2) To elicit the common physical examination findings- differentiating central vertigo from peripheral vertigo
3) To perform various diagnostic maneuvers
4) To analyze the various Audiological investigations in vertigo patients
5) To arrive at various differential diagnosis of vertigo and manage using canal repositioning maneuvers

PATIENTS AND METHODS

Study place

The study was conducted among patients attending Department of ENT

(Otorhinolaryngology), Bhaskar medical college and Hospital, Moinabad, Telangana.

Study Design : Cross sectional study

Study period : March 2023 to September 2024
Sample size : 100 patients
Ethical approval- Institutional ethical committee approval was obtained prior to the initiation of the study

Study population

All patients who present with symptoms of vertigo to the department of

Otorhinolaryngology at Bhaskar Medical College & Hospital, Moinabad.

Inclusion criteria:

Patients of age group between 5 to 50 years presenting with complaints like:

1. Giddiness
2. Ringing sensation
3. Spinning sensation
4. Nausea
5. Vomiting
6. Abnormal eye movements
7. Hard of hearing
8. Difficulty balancing
9. Inability to concentrate

10.Fever

Exclusion criteria:

1. Less than 5 years and more than 50 years
2. Trauma
3. Tumours
4. Neck injury patients
5. Bleeding
6. Stroke
7. Post operative cases.

Observations and results:

Table 3: distribution according to age

Age category	Count	Percentage
<20	3	3.0%
21-40	**51**	**51.0%**
41-60	**40**	**40.0%**
61-70	6	6.0%
Total	100	100.0%

A total of 100 cases of vertigo were enrolled based on eligibility criteria. 91% belonged to 20-60 years of age.

Figure 11: Column chart showing age

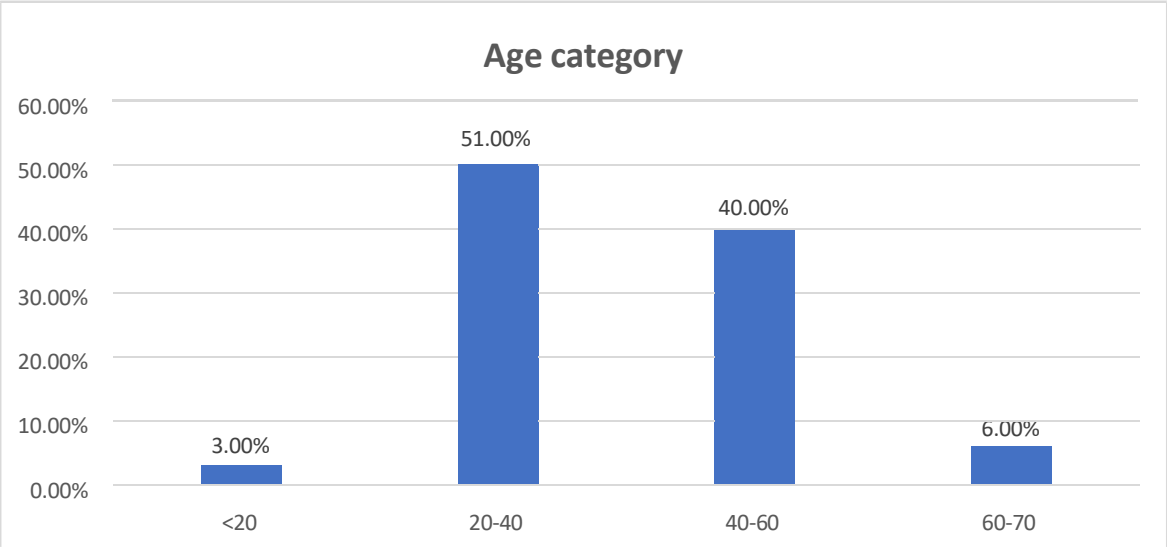


Table 3 shows that the majority of individuals (91%) fall within the 21-60 age range, with the 21-40 group being the largest (51%), while the <20 and 61-70 groups are significantly smaller, representing only 3% and 6% respectively.

Table 4: Gender distribution

Gender	Count	Percentage
Female	36	36.0%
Male	64	64.0%
Total	**100**	**100.0%**

Table 4 shows distribution of 100 cases among those- 64% were male and 36% were female.

Figure 12: Pie chart showing gender

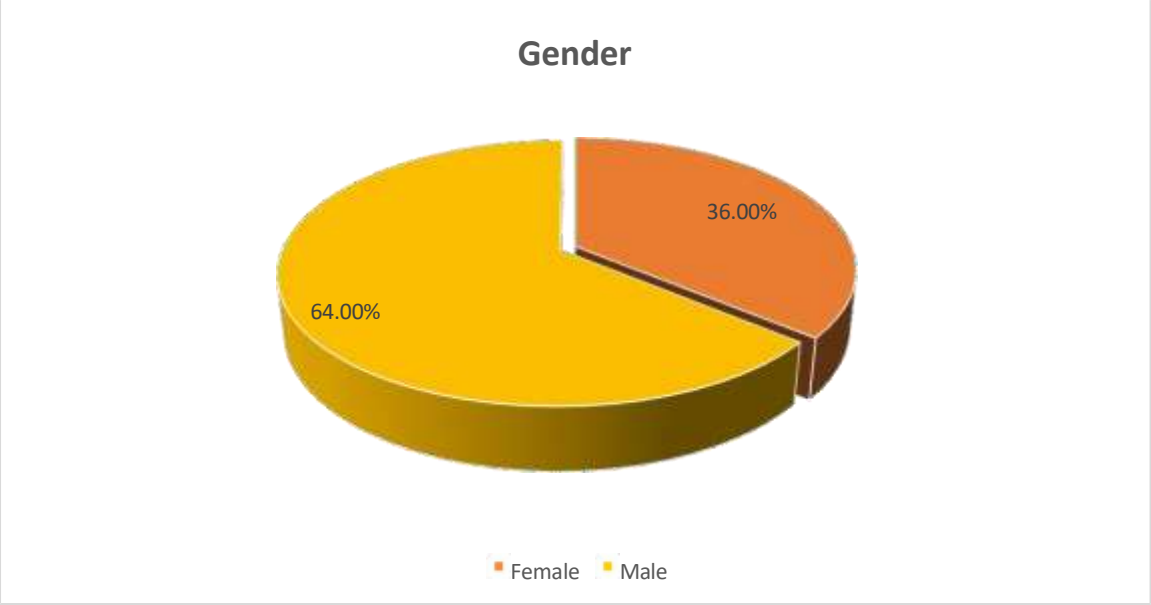


Table 5: TFT distribution

TFT	count	Percentage
CHL	3	3.0%
Normal	82	82.0%
SNHL	15	15.0%

Table 5 shows "TFT" illustrates the distribution of three categories: Normal (82%), SNHL (15%), and CHL (3%), with Normal being the dominant segment, followed by SNHL and CHL.

Figure 13: Bar chart showing TFT distribution

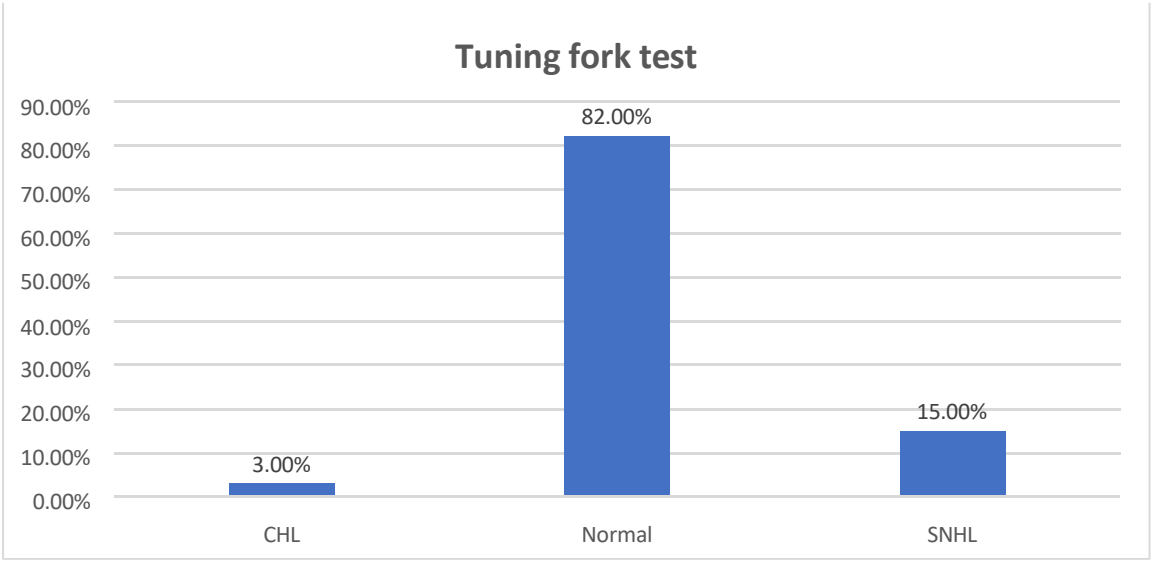


Table 6: VFT & CFT distribution

		Count	Percentage
VFT	Abnormal	77	77.0%
	Normal	23	23.0%
CFT	Normal	22	18.0%
	Romberg's positive L	31	31.0%
	Romberg's positive R	47	47.0%
CN	normal	100	100.0%

73% had abnormal VFT; 31% had romberg's test positive left side and 47% had Romberg's test positive right side. CN were normal in all 100 cases.

Figure 14: Bar chart showing VFT & CFT distribution Count

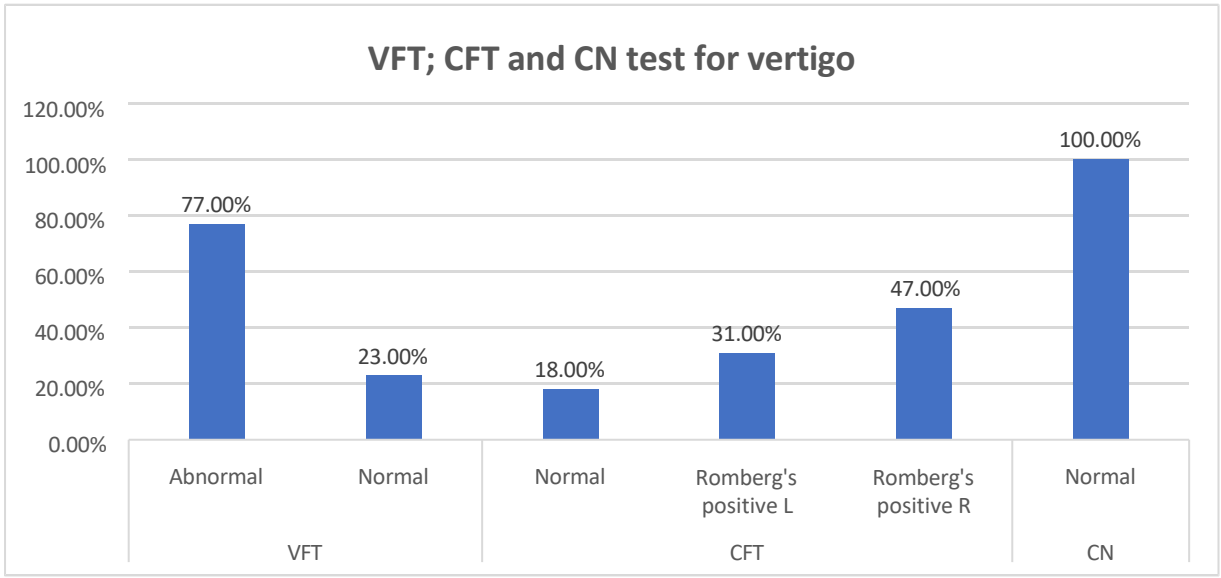


Table 7: occurrence of nystagmus

Nystagmus	count	Percentage
Elicited	86	86.0%
not elicited	14	14.0%

Table 7 shows the occurrence of nystagmus, showing that it was elicited in 86 cases (86.0%) and not elicited in 14 cases (14.0%). This highlights a higher prevalence of elicited nystagmus in the observed instances.

Figure 15: Pie chart showing Nystagmus distribution

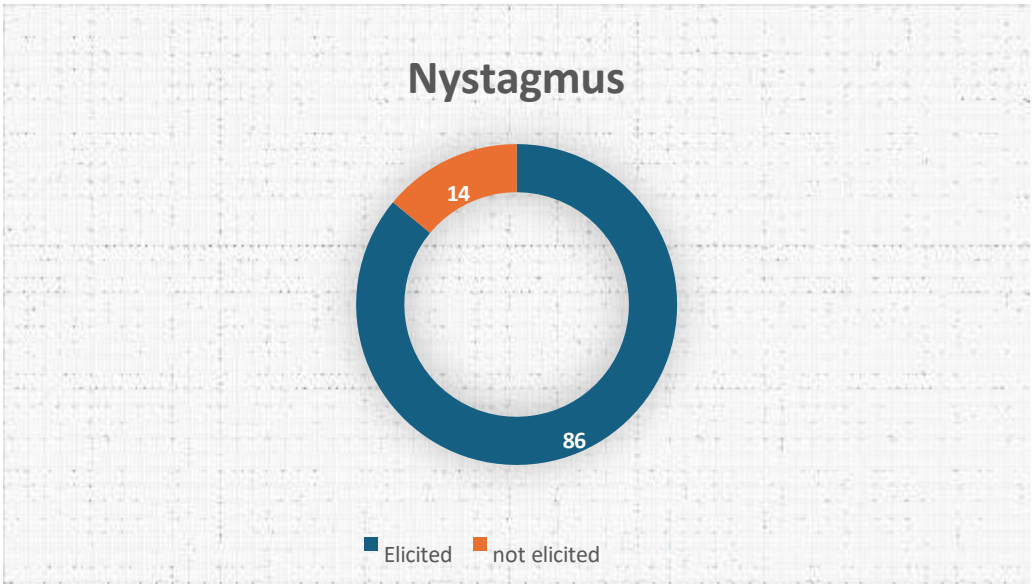
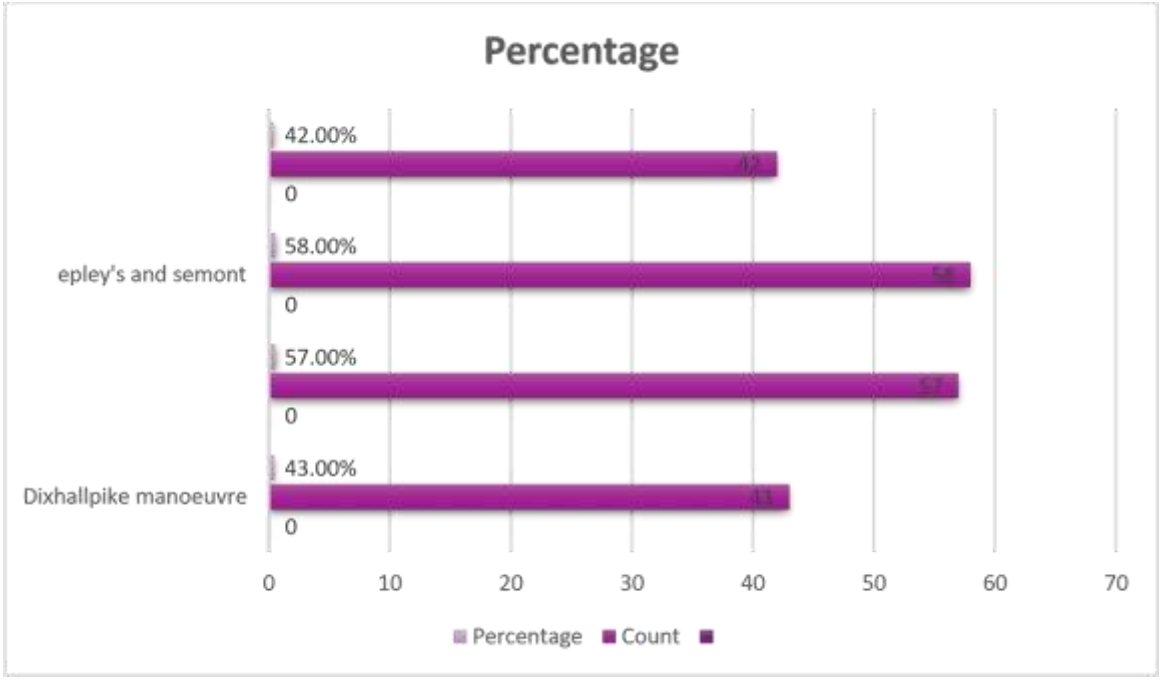


Table 8: dix-Hallpike & Epley's and Semont

		Count	Percentage
Dix-Hallpike manoeuvre	negative	**43**	**43.0%**
	positive	**57**	**57.0%**
Epley's and Semont	Improved	**58**	**58.0%**
	not improved	**42**	**42.0%**

Among 100 case; Dix-Hallpike manoeuvre was positive among 57% cases. Epley's and Semont was improved in 58% cases.

Figure 16: Bar chart showing Dix-Hallpike manoeuvre & Epley's and Semont



The table 8 presents data on two vestibular diagnostic and treatment manoeuvres.

The

Dix-Hallpike manoeuvre, used to diagnose BPPV, showed positive results in 57 cases (57.0%), while 43 cases (43.0%) had negative results. Following treatment with Epley's and Semont manoeuvres, 58 cases (58.0%) showed improvement, whereas 42 cases (42.0%) did not improve. This data highlights the effectiveness of positional manoeuvres in managing vestibular conditions.

Table 9: PTA percentage

PTA	count	Percentage
Normal	**95**	**95.0%**
SNHL	**2**	**2.0%**

Figure 17: pie chart showing PTA percentage

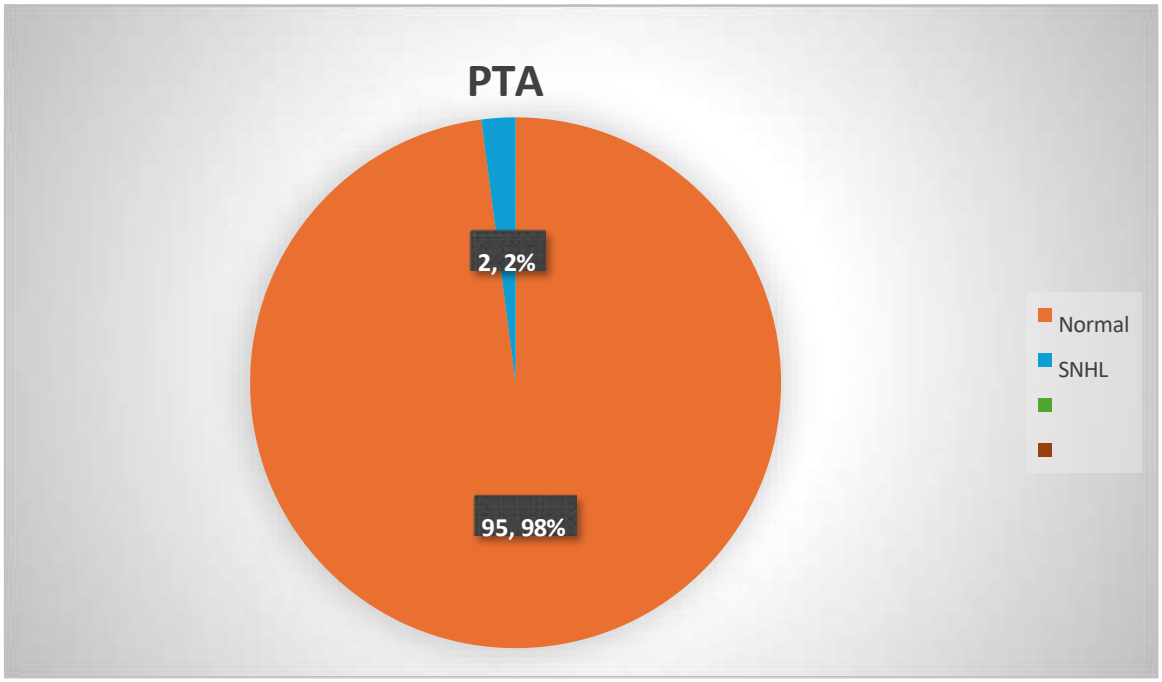


Table 9 gives Out of the total cases, 95 individuals (95.0%) had normal Pure Tone

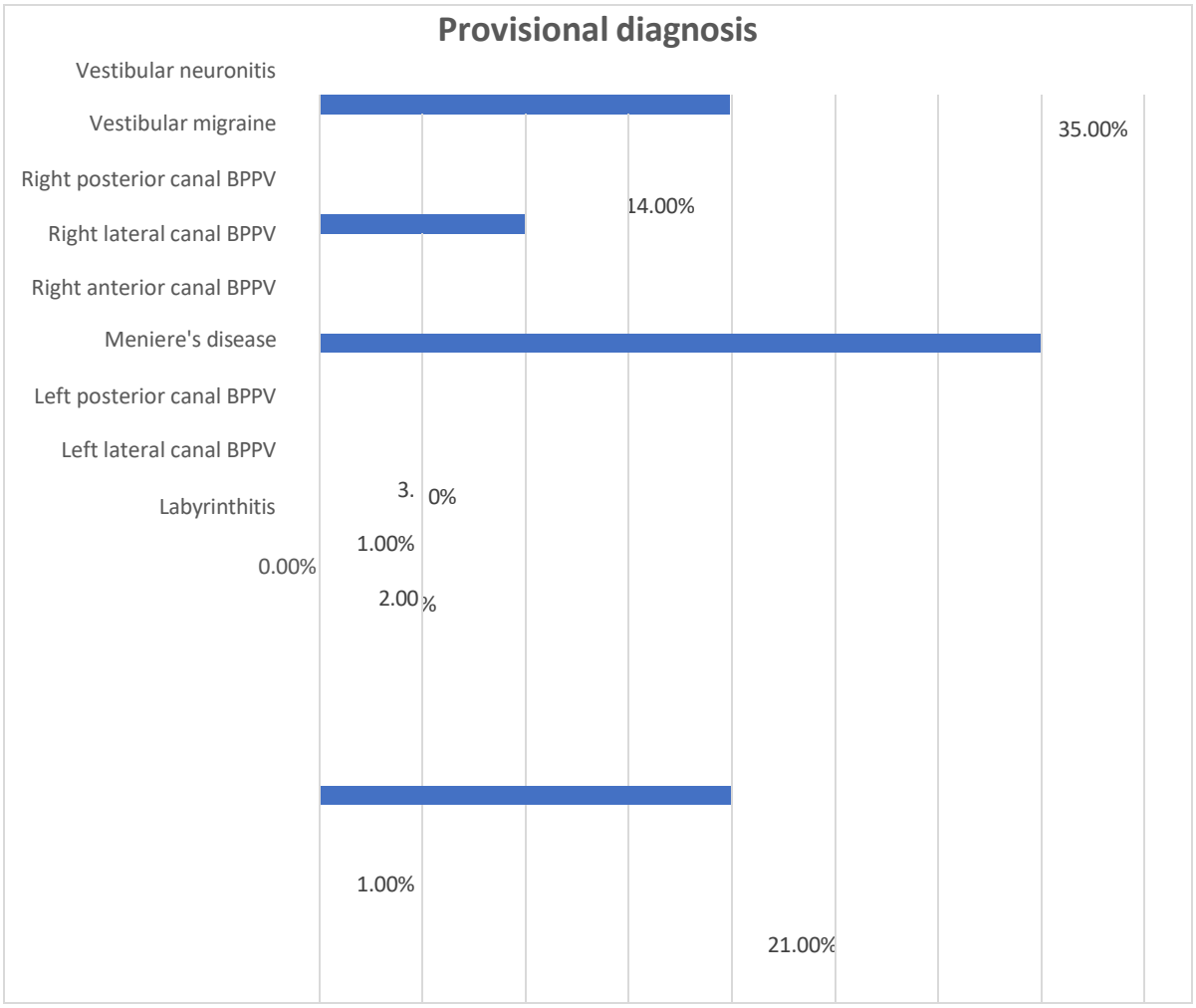
Audiometry (PTA) results, while 2 individuals (2.0%) were diagnosed with Sensorineural Hearing Loss (SNHL).

Table 10: provisional diagnosis

Provisional diagnosis	Count	Percentage
Labyrinthitis	**3**	**3.0%**
Left lateral canal BPPV	**1**	**1.0%**
Left posterior canal BPPV	**21**	**21.0%**
Meniere's disease	**2**	**2.0%**
Right anterior canal BPPV	**1**	**1.0%**
Right lateral canal BPPV	**3**	**3.0%**
Right posterior canal BPPV	**35**	**35.0%**
Vestibular migraine	**14**	**14.0%**
Vestibular neuronitis	**20**	**20.0%**

Most common diagnosis for vertigo was Right posterior canal BPPV (35%). Other differential diagnosis were Labyrinthitis; Left lateral canal BPPV; Left posterior canal BPPV; Meniere's disease; Right anterior canal BPPV; Right lateral canal BPPV; Vestibular migraine and Vestibular neuronitis. Overall, BPPV was the most common aetiology for vertigo, followed by Vestibular migraine, Vestibular neuronitis, and Labyrinthitis.

Figure 18: histogram chart for Provisional diagnosis



DISCUSSION

Vertigo refers to the perception of self-motion even when there is no actual movement or the perception of distorted self-motion during a regular movement of the head. [35] The symptoms of peripheral vertigo can occur spontaneously or be provoked by a variety of circumstances, depending on the underlying cause of the illness. About 6.5% of people will experience peripheral vestibular problems at some point in their lives.[36] In the emergency department (ED), peripheral vestibular diseases are typically misdiagnosed or mistreated, with rates reported to be between 74% and 81%.[37,38] People frequently misdiagnose diseases like vestibular neuritis and benign paroxysmal positional vertigo (BPPV) as more serious central causes like stroke, and they can also confuse them with one another.[37,38] The management is not optimal and not grounded in evidence.[38,39] Proper clinical history-taking and bedside examinations can well differentiate between peripheral vertigo and vertigo of central origin. In acute vestibular syndrome, a normal horizontal head impulse test, abnormal skew deviation (vertical ocular misalignment), or direction-changing nystagmus in eccentric gaze were jointly determined to be 100% sensitive and 96% specific for stroke in comparison to early MRI. [40]

There are several reasons why proper diagnosis of peripheral vestibular problems should be taken seriously. A proper clinical evaluation and oculographic examination can save the time, money, and life of the patients rather than waiting for sophisticated radiological and other imaging studies to rule out the central causes of vertigo.[40] The Dix-Hallpike test and the other effective canalith repositioning procedure are recommended by systematic studies and clinical guidelines for BPPV, which is the most common peripheral vestibular disorder.[41] Comprehensive history-taking and

clinical examination generally distinguish peripheral vertigo, including conditions like

Meniere's disease, vestibular neuritis, and labyrinthitis. In some instances, further investigations may be necessary to aid in the diagnosis. The primary factors contributing to vertigo are vestibular migraine, vertebrobasilar insufficiency, posterior fossa stroke, cerebellopontine angle tumours, multiple sclerosis, and episodic ataxia. These factors are less prevalent compared to peripheral causes. Bedside procedures have the potential to serve as a quick and inexpensive alternative to current treatment methods in a time when both efficiency and cost reduction are of utmost importance.[40]

In this study, 100 vertigo cases were enrolled according to eligibility criteria. Among them, 91% were aged 20 to 60 years, with 64% male and 36% female. TFT examinations showed that 82% were normal, 3% had conductive hearing loss (CHL), and 15% had sensorineural hearing loss (SNHL). Additionally, 73% had abnormal vestibular function tests (VFT); 31% tested positive for Romberg's test on the left side while 47% were positive on the right side. Cranial nerves (CN) were normal in all 100 cases, and nystagmus was present in 86% of cases. The Dix-Hallpike manoeuvre was positive in 57% of cases and success rate ranging from 77-97%, while Epley and Semont Manoeuvres showed improvement in 58% of cases and success rate ranging approximately 72-98%. Sensorineural hearing loss was identified in 2% of cases via pure tone audiometry (PTA). In this study most of the patients improved with manoeuvres and did not require review for the same. The most common diagnosis for vertigo was right posterior canal benign paroxysmal positional vertigo (BPPV) at 35%. Other differential diagnoses included labyrinthitis, left lateral canal BPPV, left posterior canal BPPV, Meniere's disease, right anterior canal BPPV, right lateral canal BPPV, vestibular migraine, and vestibular neuronitis.

Management includes medical treatment for other peripheral diseases like labyrinthitis, vestibular neuritis, vestibular migraine, Meniere's disease- antihistamines, benzodiazepines, corticosteroids, antiemetics, anticholinergics and Tryptans .

In India, life expectancy has increased from 56 to 70 years over the last 40 years, resulting in a larger elderly population. As the incidence of dizziness increases with age, our study in this era will help to identify any new trends in the distribution of patients
with peripheral vertigo. The most common age group in Sarkar et.al study was 51-60 years [42] but other studies by Bhatia et al and Gopal et al, where the most common age group was 30-40 years. [43,44]

Staibano et al found that 66.4% of the patients were male. [45] Yin et al conducted clinical epidemiologic research on vertigo and discovered that 59.28% of the 2,169 patients were male. [46] Bittar et al conducted a study in Sao Paulo to examine the occurrence of dizziness. They found that out of 1,960 patients, 53% were male. [47] The findings of all these studies coincide with our study, in which we found a male predominance of 60.5% of the total sample but Katsarkas found a female predominance with 62.81% of prevalence in females. [48]

In Sarkar et.al. study, the distribution of patients according to aetiology was as follows: 60.5% BPPV, 13.2% acute vestibular neuritis, 7.9% Meniere's disease, and 18.4% others. [42] Our study was comparable with the studies done by Raman et al, Hulse et al, and Yin et al. [36,46,49] In the research done by Yin et al [46], it was shown that BPPV is the predominant peripheral cause, followed by vestibular neuritis and Meniere's illness as the second and third most prevalent causes, respectively.

Meniere's disease is the primary cause of peripheral vertigo, making up 23.0% of cases, as found in investigations done by Muelleman et al and Bhatia et al. [44,50] Muelleman et al found that vestibular migraine and BPPV occur at a rate of 19.1% each. [50] Neupane et al conducted research that revealed that vestibular migraine is the primary cause of vertigo, with a prevalence rate of 36%. [51] BPPV follows closely at 35%, while Meniere's disease is at 8%. PPPD, vestibular neuritis, and seizures account for 5%, 2%, and 2%, respectively. 4% of cases. exhibited a central cause, whereas 8% remained unidentified.

In Sarkar et.al. study [52], the occurrence rate of posterior canal BPPV is 56.5%, horizontal canal BPPV is 30.4%, and anterior canal BPPV is 17.1%. These rates align with the majority of existing literature on the subject. Our study's findings regarding the canal's involvement pattern are consistent with those of Swain et al's study. [52] However, Seok et al reported that the horizontal canal BPPV was the most prevalent, accounting for 57.1% of cases in a sample size of 49.23 The posterior canal and anterior canal were involved in 38.8% and 2.0% of cases, respectively. [53]

CONCLUSION

Our study concludes that the most common age group affected by peripheral vertigo is between 20 and 60 years, more in male. This indicates that the prevalence of vertigo increases in the later stages of life, potentially compromising the quality of life for elderly individuals. The most frequent causes of peripheral vertigo in our study area include benign positional paroxysmal vertigo, vestibular neuritis, vestibular migraine, and Meniere's disease.

As a part of Neurotological practice, management of BPPV forms the most important chunk followed by others. Various maneuvers based on the involved canal are to be done for repositioning of the debris into the utricle. symptomatic management of various other differential diagnosis have to be understood to be a successful vestibular practitioner. Our study highlighted the same.

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