"ROLE OF VITAMIN D SUPPLEMENTATION IN ADDITION TO BETAHISTINE AS COMPARED TO BETAHISTINE ALONE IN PATIENTS OF BENIGN PAROXSYMAL POSITIONAL VERTIGO: A ONE YEAR RANDOMIZED CONTROLLED STUDY IN KLES Dr PRABHAKAR KORE HOSPITAL "

BY

REG. NO: BE0120004

Dissertation

Submitted to the KLE Academy of Higher Education and Research, Belagavi, Karnataka

In Partial Fulfilment
of the Requirements for the Degree of

M.S

IN

OTORHINOLARYNGOLOGY

AND HEAD AND NECK SURGERY

DEPARTMENT OF OTORHINOLARYNGOLOGY AND HEAD AND NECK SURGERY
JAWAHARLAL NEHRU MEDICAL COLLEGE,
BELAGAVI, KARNATAKA

JUNE / JULY - 2023

KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH

BELAGAVI, KARNATAKA

ENDORSEMENT BY THE HOD, PRINCIPAL/HEAD OF THE INSTITUTION

This is to certify that the dissertation entitled **"ROLE OF VITAMIN D SUPPLEMENTATION IN ADDITION TO BETAHISTINE AS COMPARED TO BETAHISTINE ALONE IN PATIENTS OF BENIGN PAROXSYMAL POSITIONAL VERTIGO: A ONE YEAR RANDOMIZED CONTROLLED STUDY IN KLES Dr PRABHAKAR KORE HOSPITAL "** is a bonafide and genuine research work carried out by **REG. NO: BE0120004.**

Dr. B.P. Belaldavar MS, Ph.D
Professor and Head,
Department of Otorhinolaryngology
and Head and Neck Surgery,
J. N. Medical College,
Nehru Nagar, Belagavi

Professor & Head
Department of E.N.T.
J. N. Medical College
Belagavi

Date : 22/12/22
Place : Belagavi

KAHER * BELAGAVI * J.N. MEDICAL COLLEGE

Dr (Mrs) N. S. Mahantashetti MD (Paeds)
Principal,
J.N. Medical College,
Nehru Nagar, Belagavi

PRINCIPAL
J.N. Medical College,
BELAGAVI- 590 010

Date : 02/01/2023
Place : Belagavi

UNDERTAKING

I **(REG. NO: BE0120004),** hereby declare that the information and the data mentioned in my dissertation entitled **"ROLE OF VITAMIN D SUPPLEMENTATION IN ADDITION TO BETAHISTINE AS COMPARED TO BETAHISTINE ALONE IN PATIENTS OF BENIGN PAROXSYMAL POSITIONAL VERTIGO: A ONE YEAR RANDOMIZED CONTROLLED STUDY IN KLES Dr PRABHAKAR KORE HOSPITAL "**belongs to me and is original.

I am aware of the definition of plagiarism as detailed below:

- An act or instance of using or closely imitating the language and thoughts of another author without authorization and the representation of that author's work as one's own, as by not crediting the original author.
- A piece of writing or other work reflecting such unauthorized use or imitation.
- The deliberate or reckless representation of another's words, thoughts or ideas as one's own without attribution in connection with submission of academic work, whether graded or otherwise.

I herby declare that the dissertation prepared by me is original-one and does not involve plagiarism anywhere. In case at a later stage, it is found that I have indulged in plagiarism, then I am solely responsible for the same and the institution is liberty to take any disciplinary action against me including cancellation of dissertation or any other penalties imposed by the University.

Date: 02/01/2023

Place: Belagavi

REG. NO: BE0120004

PLAGIARISM ACCEPTED LETTER

KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH

JAWAHARLAL NEHRU MEDICAL COLLEGE

(Recognized by Medical Council of India, New Delhi)

Accredited 'A+' Grade by NAAC (3rd Cycle) **Placed in Category 'A' by MHRD (GoI)**

Nehru Nagar, Belagavi- 590 010, Karnataka, INDIA

0831 - 2471350 0831 - 2470759 www.jnmc.edu principal@jnmc.edu

Ref No: MDC/PG/ Date: 30-11-2022.

ACCEPTANCE LETTER

The softcopy of thesis entitled: **"ROLE OF VITAMIN D SUPPLEMENTATION IN ADDITION TO BETAHISTINE AS COMPARED TO BETAHISTINE ALONE IN PATIENTS OF BENIGN PAROXYSMAL POSITIONAL VERTIGO"** has been submitted for Anti-Plagiarism check through Turnitin software. The scan has been carried out and the scanned output reveals a match percentage of **06%** which is within the acceptable limits of **10%** as per the guidelines given by UGC.

Guide.

KAHER BELAGAVI J. N. MEDICAL COLLEGE

Dr. (Mrs.) N.S. Mahantashetti.
Chairperson-Antiplagiarism Committee &
Principal,
J. N. Medical College, Belagavi.

To,
Reg. No. BE0120004.
Postgraduate Student,
2020-21 Batch,
Department of ENT,
J. N. Medical College, Belagavi.

ETHICAL CLEARANCE

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed – to- be- University)
Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (GoI)
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
Website: http//www.jnmc.edu
E-Mail : dome@jnmc.edu
Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/ 59 **Date: 25/01/2021**

To,

REG. NO: BE0120004

PG student in Otorhinolaryngology and Head & Neck Surgery,
J.N.Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"ROLE OF VITAMIN D SUPPLEMENTATION IN ADDITION TO BETAHISTINE AS COMPARED TO BETAHISTINE ALONE IN PATIENTS OF BENIGN PAROXYSMAL POSITIONAL VERTIGO –A ONE YEAR RANDOMIZED CONTROLLED STUDY IN KLES DR. PRABHAKAR KORE HOSPITAL, BELAGAVI"**, is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Smita Sonoli)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Harsha Hegde)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

LIST OF ABBREVIATIONS

GLOSSARY	ABBREVIATIONS
BPPV	Benign Paroxysmal Positional Vertigo
CNS	Central Nervous System
PSC	Posterior Semicircular Canal
HSC	Horizontal Semicircular Canal
CSF	Cerebrospinal Fluid
EP	Endocochlear Potential
VOR	Vestibulo-Ocular Reflex
VCR	Vestibulo Collic Reflex
VSR	Vestibulo Spinal Reflex
SCN	Suprachiasmatic Nucleus
SCC	Semicircular canal
RA	Right Anterior
LA	Left Anterior
aVOR	Angular Vestibulo Ocular Reflex
LARP	Left Anterior Right Posterior
RALP	Right Anterior Left Posterior
HTN	Hypertension
Sr.	Serum
Vit.	Vitamin

ABSTRACT

TITLE :ROLE OF VITAMIN D SUPPLEMENTATION IN ADDITION TO BETAHISTINE AS COMPARED TO BETAHISTINE ALONE IN PATIENTS OF BENIGN PAROXSYMAL POSITIONAL VERTIGO: A ONE YEAR RANDOMIZED CONTROLLED STUDY IN KLES Dr PRABHAKAR KORE HOSPITAL

BACKGROUND: BPPV is defined as a unilaterally predominating vestibular impairment of peripheral origin. It is marked by episodes of vertigo. In clinical practice, it is the most frequently occurring peripheral vestibular disease and ranks the top most among otolaryngology variety of vertigo [1].

Otoconia are in a dynamic state whose mineralization and turnover depend on calcium. Vitamin D is essential for calcium metabolism and may have an impact on the density and matrix of CaCO3 crystals . The generation of otoconial particles in the vestibular system and the method by which Ca2+ & P are deposited in bone and teeth are all directly impacted by vitamin D[11]. On the Ca2+ channel transport system of the labyrinth, vitamin D receptors can be seen, and they regulate the proper calcium balance. Deficiency of vitamin D leads to cochlear demineralization and cochlear deafness[12]Severe BPPV is prevalent in vitamin D deficient patients, as this deficiency is linked to developmental progression or severity of several skeletal or non-skeletal conditions.[17]

OBJECTIVE : To study the effect of Vitamin D supplementation along with Betahistine as compared to Betahistine alone in treatment of patients of BPPV.

MATERIALS AND METHODS : This is a Randomised Controlled Trial conducted in the Department of Otorhinolaryngology and Head & Neck Surgery at Jawaharlal Nehru Medical College from January 2021 to December 2021 to examine the effectiveness of vitamin D supplementation in treating BPPV patients. 50 patients , more than 18 years of age with complaints of dizziness were evaluated using a Dizziness questionnaire and Dix Hallpike Test . Once the diagnosis of BPPV was confirmed , patients underwent Sr. Vit D level testing . 25 patients received Vitamin D 60,000 IU weekly for three months along with Betahistine while the other 25 received Betahistine alone and the results of the treatment were compared.

RESULT: Among the 50 patients ,group A received Vitamin D along with Betahistine while Group B received Betahistine alone ,the mean age was 41.96 ± 15.01 years, and group A consisted of 16 females and 9 males (range: 21–60 years). There were 13 females and 12 males found in group-B, and their mean age ranged from min to max at 41.88 ± 12.33 year.Patients with Left BPPV were determined to be 9 (36%) and 16 (64%) in groups A and B, while those with Right BPPV were 16 (64%) and 9 (36%).In group A, 11 (44%) patients had diabetes mellitus, 6 (24%) had HTN, and 5 (20%) had no co-morbid conditions. There were conditions in group-B individuals with diabetes mellitus 5(20%), HTN 5(20%), migraine 12 (48%), and 3(12%) patients had no comorbidities.Before starting therapy, patients in Group A had an average of 10 episodes per day (41.67%), while only 7 episodes per day (29.17%).After one month of treatment in group A, a maximum of 14 patients (58.33%) had six episodes per day. After 2 and 3 months, there were 11 (45.83%) patients who had 3 episodes, 6 (25%) patients who had only one episode, and 16 (66.67%) patients who had no any episodes at all .The maximum number of patients in group-B who had four episodes per day after a month of treatment was 16(64%), 19

patients (76%) had only three episode, while 14 (56%) used 2 episodes after two and three months and 3(12%) patients who had no any episodes at all after 3 months.In the present study, vitamin D levels were compared between Group-A and Group-B. Before treatment, group-A had a vitamin D level of 16.68, while group-B had a vitamin D level of 17.48. Whereas, Group-A showed 19.68 and Group-B showed 18.42 after treatment

CONCLUSION : The occurrence and recurrence of BPPV are both correlated with low vitamin D levels. A lower probability of BPPV relapse was associated with lessening vitamin D insufficiency, and this association was statistically significant. Following our research, we came to the conclusion that comorbidities would exacerbate BPPV and increase the frequency of otolith detachment. So even after a successful repositioning effort, it will increase the likelihood of recurrence. Therefore, in addition to repositioning techniques, individuals who present with BPPV should also be assessed and treated for any comorbidities.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF GRAPHS

LIST OF FIGURE

INTRODUCTION

BPPV is defined as a unilaterally predominating vestibular impairment of peripheral origin. It is marked by episodes of vertigo. In clinical practice, it is the most frequently occurring peripheral vestibular disease and ranks the top most among otolaryngology variety of vertigo [1].BPPV is the frequently occurring disease in vertigo patients affecting 2.4% of the general population.[2] Prevalence of BPPV exceeds with age from 0.5% at age 18 to 39 years, increasing to 3.4% after 60 years of age. [3] In our randomized controlled study, the patients age parameter ranged from 18 year to 60 years . The life time cumulative incidence of the disease hikes upto 10% at age of 80 years.[4] It is more commonly seen in females and most cases are idiopathic in nature. The predilection of BPPV among females is two folds higher than males[5]. This dissimilarity is often because of sex hormones. The cause of BPPV is mostly unknown, while some occurrences may be linked to extended recumbent positions, such as those in dental chairs, salons, or while on enforced bed rest, head trauma, different inner ear disorders (including Meniere's disease), migraines, or vitamin D insufficiency (10ng/ml) [6] . However ,in our study the patients analyzed were absolutely normal without any past trauma history or medical history .This randomized control study was carried out in general population. Benign paroxysmal positional vertigo is delineated by transient fierce rotational vertigo and specific nystagmus,which results from otoconia displacement from urticle and flow in to semicircular canals[7]. This condition arises by head position due gravitational direction.

The most prevalent type of BPPV is posterior canal BPPV, which makes about 90% of cases. BPPV in the horizontal canal occurs at a rate of 5–15%. BPPV is caused by the displacement of microscopic calcium crystals termed otoconia from their

natural position on the utricle, an inner ear sensory organ[8]. Otoconia are biocrystals in the utricle and saccule that link mechanical stresses to sensory hair cells. This is necessary for someone to detect linear acceleration and gravity in order to keep their body in balance[9].In BPPV, calcium crystals detach and flow freely in fluid-filled inner ear regions, including the semicircular canal, which causes head rotation or vertigo. [10]. Thus, aberrant eye movements and vertigo are a result of the changes in head position w.r.t gravity. Otoconia are in a dynamic state,whose mineralization and turnover depend on calcium. Vitamin D is essential for calcium metabolism and may have an impact on the density and matrix of CaCO3 crystals . The generation of otoconial particles in the vestibular system and the method by which Ca2+ & P are deposited in bone and teeth are all directly impacted by vitamin D[11]. On the Ca2+ channel transport system of the labyrinth, vitamin D receptors can be seen, and they regulate the proper calcium balance. Deficiency of vitamin D leads to cochlear demineralization and cochlear deafness[12]Vitamin D deficiency affects calcium metabolism because calcium ions are crucial for maintaining membrane permeability.[13] The nerve needs ionised calcium to function effectively, and a lack of it may prevent the cochlea from producing action potentials. Otic capsule demineralisation, spiral ligament, stria vascularis, and cochlear hair cells degeneration are all caused by low vit.D and Ca2+ levels.[14] Aging causes otoconia to degrade and fragment as a result of increasing demineralization, which causes balance problems. Three lines of evidence are described in the literature as being related to the development of BPPV and defective calcium metabolism: Calcium carbonate and vitamin D supplementation may help those with BPPV and subnormal Sr.Vit D levels avoid further BPPV attacks. BPPV development has been associated to lower Sr.Vit D levels. (1) Patients with BPPV had lower BMD more commonly than healthy controls. (2) Oestrogen is essential for

sustaining otoconia, and oestrogen shortage seems to hasten otoconia degeneration and BPPV development. 3) Low Sr. Vit D levels are seen with the onset of BPPV, & calcium carbonate and vit.D supplements might help those who currently have the condition avoid recurrent BPPV attacks. Otoconia crystals are made up of a central core and an outer zone. With a lower amount of Ca2+, the centre is primarily organic (which is primarily glycoprotein), while the periphery is primarily inorganic (polymorph of CaCO3), having greater level of Ca2+.The crystals' inside, exterior surface, and interconnecting fibrous material are all organised and have varying sizes. Otoconia crystals are produced by the vestibular organ's active calcium metabolism. Otoconia crystals are joined to hair cells by a protein fibre that is partially encased in a fibrous matrix. Vitamin D increases calcium absorption from the kidney and stomach. The hormone parathyroid controls the upkeep of blood calcium. There are similarities between the biomineralization of bone and otoconia. Since the two tissues' protein composition and matrix architecture are comparable. In order to achieve biomineralization in otoconia, an organic matrix must be carefully controlled to form at only certain locations, and mineral crystallites must deposit in an organised pattern like to that of bone and teeth. The disruption in calcium and vitamin D metabolism found in osteoporosis is a major factor in the aetiology of BPPV. The severity of BPPV and its recurrence were both connected with vitamin D insufficiency. In fact, vitamin D administration may reduce the frequency of BPPV attacks. Osteoporosis risk was 1.28 times higher when BPPV was present. Additionally, osteoporosis was linked to 1.34 times the likelihood of BPPV. Adequate level of vitamin D is maintained through its cutaneous photosynthesis and oral ingestion. [16] Vitamin D deficiency or insufficiency is prevalent in nearly one billion people worldwide. Severe BPPV is prevalent in vitamin D deficient patients, as this deficiency is linked to

developmental progression or severity of several skeletal or non-skeletal conditions.[17] Persistent recurrent attacks of vertigo particularly in elderly leads to impaired daily activities, decrease muscle strength, postural imbalance and fall due to dizziness. This may affect quality of life. Vitamin D reduces the risk of fracture in elderly due to fall because of vertigo and dizziness; the Due to photosynthesis and vitamin D bioavailability, there is a possibility of reduced vit.D status, and many other components can affect this risk. [18] Changes in exposure to sun due to variation in latitude, time of the day, exposure to solar radiation, season, weather, air pollution, clothing, use of sunscreen, and skin colour, as well as variations due to age, obesity, and the incidence of certain chronic conditions, are among these factors. According to a study, maintaining the right level of vitamin D helped older people with BPPV experience a 78% decrease in falls. Betahistine is the most common anti–vertigo drug prescribed since 40years. It is a structural analogue of histamine .[19] A modest agonist of H1 receptor and an antagonist of H3 receptor are the two ways that betahistine works. Betahistine increases microcirculation in the inner ear by creating vasodilation.[20] It is likely the only non-sedative anti-vertigo medication without any anticholinergic and anti-dopaminergic effects, relieving symptoms via sedating the vestibular labyrinth. The vestibular suppressant is the one that prevents CNS depression. N-Methyl-2-(2-pyridyl) ethylaminohydrochloride is betahistine.

It directly affects the vestibular complex's neurons. Betahistine mostly undergoes hepatic transformation into pyridylacetic acid, which is eliminated in the urine, amino ethylpyridine, and hydroxyethylpyridine. This drug is mainly used in surgery resistant cases.Use of 48mg\day of betahistine for 3 months is the most effective for peripheral vertigo. Despite the recovery of BPPV with betahistine leads to remissionin days and weeks, recurrence may develop in 50% of patients. Therefore, in

these situations, treating the underlying vitamin D deficit can avoid recurrence. Due to pharmacokinetic interactions, vitamin D can increase the efficacy and side effect rate of betahistine. Sunshine vitamin has a wider spectrum of drug-related effects.As vitamin D maintains the drug concentration. Although the outcomes of vitamin D supplementation with betahistine and effect of betahistine alone has not be published or studied till date, so this is our first study of interrelationship of vitamin D supplementation along with betahistine drug in general population. Hence, in this study we will be assessing the effect of Vitamin D along with betahistine and betahistine alone in patients of BPPV.

AIMS AND OBJECTIVES

To study the effect of Vitamin D supplementation along with Betahistine as compared to Betahistine alone in treatment of patients of BPPV.

REVIEW OF LITERATURE

BPPV is defined as spontaneous occurrence of vertigo arising from vestibular dysfunction of peripheral origin and unilateral predominance.[21] Clinically, BPPV, which tops list in otolaryngology-related peripheral vestibular diseases, is the most prevalent..[22]

If actual motion is not present , it is described as an illusory impression of movement in oneself or one's surroundings.

BPPV is a type of giddiness brought on by shifts in the head's posture in relation to gravity. A condition known as BPPV is an inner ear illness characterized by recurrent bouts of positional vertigo.

The labels "benign" and "paroxysmal" have traditionally been used to describe the various types of positional vertigo.

BPPV is a balance disorder related to crystals of CaCO3 and is the disease of otolith organs. It is induced due to the degeneration of otoconia which sprains into the semicircular canal, which sensitize them to gravity.[23]

About 50% of patients express their dissatisfaction with the subjective imbalance between BPPV's classic episodes. The prevalence of BPPV is 2.4% over a lifetime and 1.6% over a single year. Prevalence of BPPV increases with advancing age. It is commonly observed in geriatric patients above 60 years of age. It is prevalent more in female in comparison to male i.e. 2 :1 ratio. [24]

85% of cases with BPPV are idiopathic in origin, and the precise reason is unknown. More likely causes of BPPV include injury to head (6%–18%), conditions of the inner ear such as infection, inflammation, or disturbances in circulation of otolithic organs (3%–9%), Meniere's disease, vestibular neuritis, ear surgeries, migraine , and prolonged bed rest.[25]

There are two topographical variations of BPPV disease.

1. The Post. SCC BPPV (PSC -BPPV).
2. The horizontal SCC BPPV (HSC-BPPV).

PSC-BPPV is more prevalent than HSC-BPPV and made up 85 - 95 percent of BPPV.[26]

Thalmann et al reported that deterioration in the elasticity and adhesivity of the subunits in the macula matrix, where otoconia are located are responsible for the BPPV.[27] BPPV has high frequency of intermittence. Migraine, autoimmune disorders, hyperlipidemia, vascular disorders hypertension and diabetes mellitus are been considered as facilitating factors for recurrent episodes of vertigo. The risk factors for recurrent BPPV are increased antithyroid autoantibodies and decreased vitamin D .[28]

About 90% of Indians are considered to be in the "INSUFFICIENCY AND DEFICIENCY" level range. Vitamin D is important in management of the low BMD in osteoporosis which is prime concern in geriatric patients due to imbalance in position and falls due to disequilibrium, because of this reason there is strong association of vitamin D in BPPV.[29]. The main goal of our study is to increase the therapeutic value of the drug betahistine and improve the standard of care and

outcomes for BPPV by developing more precise and effective treatment modalities, correcting vitamin D deficiency, reducing irrational use of vest. Suppressants.

HISTORICAL BACKGROUND

BPPV has a long history and it was first described by Adler in 1879 and was further elaborated by Barany (1921). The term BPPV was formulated by 2 British otologists Margaret Dix and Charles Hallpike in 1952 .

Positionally generated vertigo was initially described in the medical literature by Adler, and later by Barany, who clung to the theory that it was caused by an issue with the otolith organs. Barany noted the presence of "appearance of a strong rotatory nystagmus to the right with a vertical component upwards, which when looking to the right was purely rotatory, and when looking to the left was purely vertical" in a 27-year-old woman who felt dizzy after turning her head side to side while lying down.[30]

In 1952 at Queen Square Hospital, Margaret Dix (1911–1981) and Charles Hallpike (1900–1979) used 100 patients to create a diagnostic and provoking positioning examination for "positional nystagmus of the benign sort." "The complaints of patient are characteristically of dizziness on lying down or turning over in bed, or lying down below car or in throwing the head backward to paint a ceiling," the letter that they provided as supporting documentation stated. As part of the diagnostic procedure, the subject is made to sit on a sofa ,their head tilted to one side , eyes fixed on examiner's forehead.Examiner pushes the patient back in critical position [30 degree below couch level and between his hands, holding the patient's forehead firmly in place].[31] The resultant reaction necessitates a thorough explanation. The superior pole of eye was pounding directed towards ground when Barany first noticed

a torsional nystagmus, and it "fatigued" during subsequent testing. A reaction delay of around 5 seconds, a nystagmus crescendo and drop, and a nystagmus reversal as the patient sits up were also noted. They evaluated individuals on a device that avoided neck rotation to rule out the idea that the response could be caused by vascular blockage. The identical reaction took place. In Britain, Hallpike was a leader in the field of temporal bone histology. The otolithic membrane was absent from the utricle's macula. In their conclusion, they stated that the overall picture was one of persistent tissue changes brought on by either trauma or infection. The author concluded that the lesion coincides with the side of the nystagmus. Hallpike strengthened the case about this theory by removing symptoms in two individuals with a chemical labyrinthectomy of an acoustically dead ear and in one patient with an eighth nerve slice. According to Barany, Dix, and Hallpike, the utricular macula condition was discovered to be the primary source of benign positional nystagmus.

Although the inner ear's bony and some membranous structures were physically well documented, their functions remained unknown till 19th century. Semicircular canals were thought to be responsible for bone conduction of sound transmission & to perceive sound directions, while the cochlea was thought to mediate the type & pitch of sound, saccule& utricle for loudness perception, and cochlea itself to perceive loudness.[32]

At Harvard University in Boston, Harold Schuknecht (1917-1996) made the hypothesis - BPPV "could be caused by detached utricular otoconia, operating upon the cupula of the posterior semicircular canal." This hypothesis was made in 1962. Since there were no conclusive human pathological investigations at the time, the idea appeared plausible from a strictly theoretical standpoint.[33]In patients with BPPV

symptoms in 1969, Schuknecht verified discovering basophilic staining masses linked to the posterior canal cupula. He diagnosed this condition as cupulolithiasis (heavy cupula) and surmised that the masses were detachable utricular otoliths that had been prepared by decalcification. Gacek's study on five patients, in whom the selective excision of the posterior ampullary nerve eliminated BPPV symptoms, provided support for this.[34,35,36]For almost thirty years, cupulothiasis predominated despite the fact that it was unable to account for the nystagmus' varied and frequently extended latency and fatiguability.[37]

BPPV (85% posterior canal) - is most common cause of vertigo in adults. Estimates show that 2.4% of people experience at least one episode throughout their lifetime .[38]BBPV was discovered in 9% of senior inhabitants of a facility. [39]. The fifth to seventh decades are when it typically manifests. It is the most frequent reason for dizziness following a head injury. [40, 41]. A vestibular neuritis episode and a period of bed rest are frequent causes. [42,43].

ANATOMY:

THE INNER EAR -The 3 parts of the ear are outer,middle and inner ear.Inner ear-part of ear-close to the body, contains the vestibuloccochlear organs. It accomplishes two main goals:

This can send data to the auditory system in the brain by converting mechanical signals from the middle ear into electrical signals.

ANATOMICAL POSITION AND STRUCTURE

Inner ear is a part of petrous part of temporal bone. It is situated between the IAM, which is medially located, and the middle ear, which is laterally located. The membranous labyrinth & bony labyrinth are 2 main parts of inner ear.

Bony labyrinth: There are numerous bony cavities in petrous temporal bone . Cochlea, vestibule, and 3 SCCs make up its structure. Perilymph is a fluid that is present inside all of these structures, which are internally lined with periosteum.

Membranous labyrinth – The bone labyrinth is enclosed by the membrane-filled labyrinth. The cochlear duct, semicircular ducts, utricle, and saccule make up this structure. Endolymph is a liquid that fills the membranous labyrinth.

There are two membrane-covered channels leading from the inner ear to middle ear. While middle part is situated between round window and scala tympani, vestibule & oval window are where the middle ear is situated (part of the cochlear duct).

Vestibule

The centrepiece of the bony labyrinth is the entryway. It communicates anteriorly to cochlea and posteriorly to SCCs ,separated from middle part by oval window.

Vestibule contains saccule& utricle, 2 parts of membranous part.

Cochlea

Cochlear duct, which is a component of the auditory portion of the membranous labyrinth, is housed within the cochlea. It forms a cone-shaped structure that points anterolaterally as it spirals around the modiolus, the middle section of the bone. Cochlear part of vestibulocochlear nerve gives it's branches at base of modiolus .

A bone ledge called the spiral lamina, which extends from the modiolus and connects to the cochlear duct, holds it in place. Two perilymph-filled chambers are created above and below the cochlear duct:

Scala vestibuli: The location of it is above cochlear duct. It continues into vestibule.

Semi-circular Canals

There are anterior, lateral, and posterior semi-circular canals. They house the semi-circular ducts, which are in charge of maintaining equilibrium (along with the utricle and saccule).

In a superoposterior position to the vestibule, the canals are positioned at an angle to one another. They have an ampulla, or swelling, at one end.

Cochlear Duct

Within cochlea's bony framework is where the cochlear duct is situated. The spiral lamina keeps it in place. The duct's presence results in making of the scala vestibuli and scala tympani, two canals that run above and below the duct. It can be described as triangular in shape:

Lateral wall – called spiral ligament-thickened periosteum

Roof –.formed by Reissner's membrane- separates the cochlear duct from the scala vestibuli.

Floor – formed by basilar membrane-separates cochlear duct & scala tympani.Organ of Corti present here .

Saccule and Utricle

The vestibule contains two membrane sacs called the saccule and utricle. They are balancing organs that are able to recognise when the head moves or accelerates in the vertical and horizontal planes, respectively.

The three semicircular ducts enter the utricle, which is the bigger of the two. The cochlear duct enters the globular-shaped saccule.

The utricle and saccule release endolymph into the endolymphatic channel. To reach the back of the petrous portion of temporal bone, the duct goes via the vestibular aqueduct. Here,duct widens into a sac that allows for the secretion and absorption of endolymph.

Semi-circular Ducts

The semicircular canals contain the semicircular ducts, which are oriented similarly. The endolymph flow within the ducts alters speed and/or direction in response to head movement. Sensory receptors in the semicircular canals' ampullae notice this change and communicate with the brain in order to process balance.

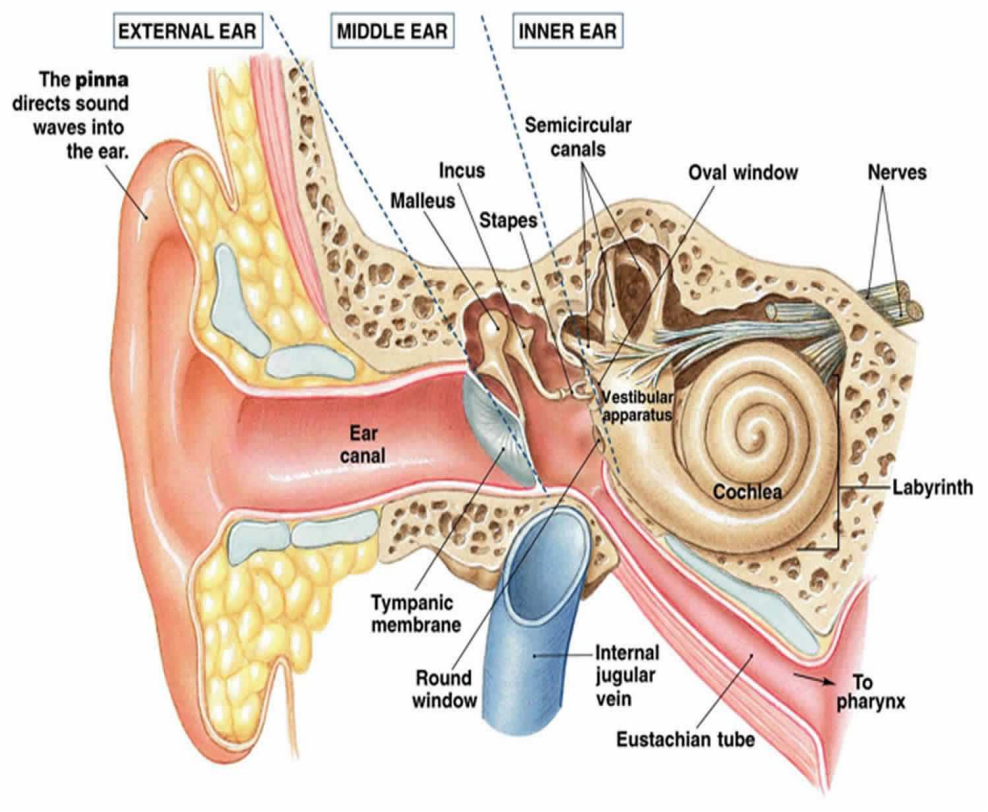


Figure1- Semicircular canal

Function: The inner ear gathers, organises, and transmits sensory data pertaining to hearing through cochlea & balance through the vestibular system. It is in charge of mechanoelectrical transduction, which is the translation of movements—started by sound waves in the cochlea or by changes in the location of the head in space—into electrical signals that can then be transmitted to the brain via the auditory or vestibular nerves. It is formed by the series of bony channels that enclose interconnected fluid-filled tubes (the membranous labyrinth), the inner walls of which are lined by epithelial tissues. The bony channels are filled with perilymph, which surrounds the membranous canals. Perilymph is essentially a typical extracellular fluid which is similar, but not identical, to cerebrospinal fluid (CSF) or serum; it has a higher Na+ and lower K+ concentration. The perilymphatic compartment joins the arachnoid

space of the brain via the cochlear aqueduct so there is potential continuity between cochlear perilymph and CSF, although the exact compositions of the two fluids are different from each other and also from serum, indicating that perilymph is produced or at least significantly modified locally in the inner ear and is not derived directly from CSF (or serum). The fluid in the membranous canals - endolymph . It is higher in K+ (~140mM) and lower in Na+ (~1mM). Electric potential of endolymph is +80mV, the endocochlear potential (EP), but although the compartments are interconnected a similar electrical potential is not observed in the vestibular system. The connections between the epithelial cells that enclose the endolymphatic regions mark the boundary between perilymph and endolymph. The preservation of this permeability barrier, which is formed by these junctions, is crucial for the inner ear's proper operation.

THE VESTIBULAR SYSTEM:

In general, the vestibular system is comprised of two parts: utricle and 3 SCCs that arise and end in utricle and run in orthogonal planes - horizontal (lateral), posterior, and superior - are anatomically distinct chambers from the saccule.The BPPV of the posterior semicircular canal constitute approximately 85 % -95% of BPPV.[44]

OTOCONIAL MEMBRANES AND CUPULA:

The utricular and saccular maculae are each overlaid by an ‘otoconial membrane’ which consists of a large number of otoconia (Greek: ‘ear dust’), crystalline particles composed of calcium carbonate surrounding a proteinaceous core that sit on a honeycomb-like perforated sheet of non- collagenous fibrillar extracellular matrix. This matrix is composed of proteins only seen in inner ear including otogelin,

α & β tectorins and ceacam 16 .[45] In fish, rather than numerous small particles, there is a single, large calcium carbonate particle, the otolith (Greek: 'ear stone'), hence the term 'otolithic organs' is sometimes used (incorrectly) to describe the saccule and utricle of mammals. Hair bundles of the utricular and saccular hair cells appear to align with the perforations in the sheet of extracellular matrix with the longest stereocilia, possibly in contact with the edge of the hole. Maculae detect translational motion of the head in the horizontal and vertical planes. With a linear motion,because of its mass, movement of otoconia lags in relation to movement of the epithelium itself, which follows the head movement without any inertia. As a result, the stereocilia are deflected and hair cells stimulated. With a head tilt, the force of gravity acts on the otoconial membrane to produce a relative displacement between the membrane and the tilting surface of the epithelium that deflects the stereocilia. In the semicircular canals, the extracellular structure overlying a crista, known as the cupula, is a dense fibrous mass with no otoconia that extends to the roof of the ampulla . The principal proteinaceous component is otogelin. The longest stereocilia of hair cells of the cristae appear to contact the underside of the cupula. The cristae detect rotational acceleration of the head. When the head rotates, movement of endolymph through the semicircular canals lags behind that of the crista epithelium because of the fluid's inertia.

The consequent differential movement displaces the cupula, leading to deflection of the stereocilia.

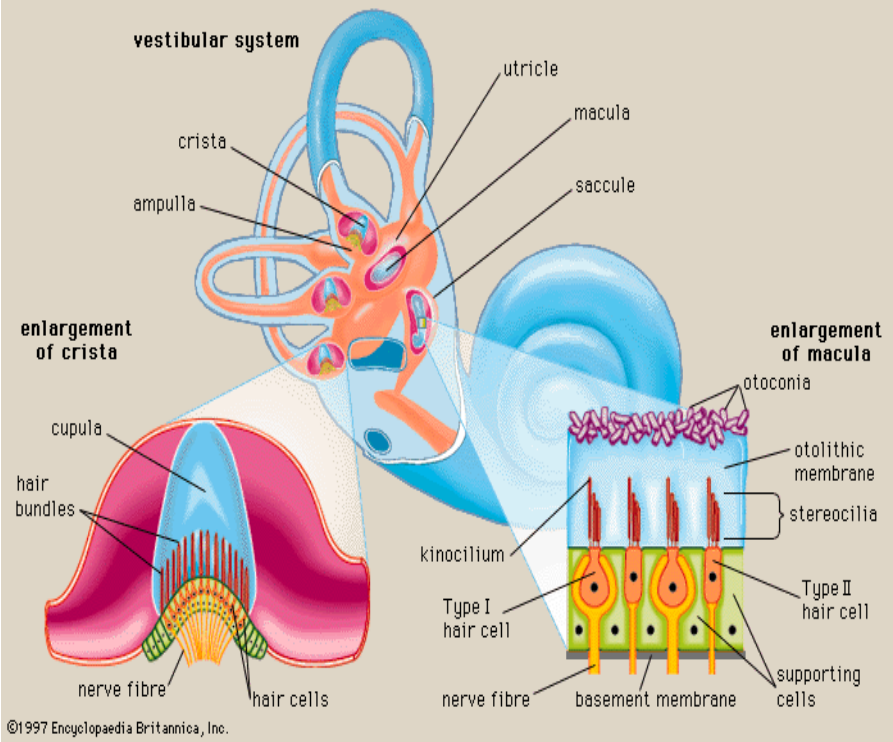


Figure2- Otoconial membranes and cupula

THE COCHLEA

Three parallel canals, or scalae (Latin for "ladders"), make up the cochlea. These canals wrap in a spiral around modiolus. Cochlear artery and cochlear vein, as well as the axons of auditory nerves' central projections that feed the sensory epithelia with blood, run along length of modiolus. Cochlea in humans has 2.5 turns. The number of turns varies throughout the various mammalian species, although as of yet, it is unknown whether this has any particular functional importance. The membranous labyrinth's central canal, known as the scala media, is lined with epithelia and is filled with endolymph.The scala media is about triangular in form and is surrounded by three "walls" in cross sections of the cochlea. Floor - Basilar memb & OAC. The stria vascularis, the principal ion-transporting epithelium, runs along the lateral side, and Reissner's membrane creates the scala media's "roof."

Scala vestibuli is located above Reissner's membrane and scala tympani are located below the basilar membrane. The perilymph fills these two scalae. In the scala vestibuli, Reissner's is partition b/w endo & perilymph .The intercellular gaps (spiral ligament) are freely permeable to perilymph from both scalae.

From spiral's base to its peak, the three scalae's height and width all gradually decline. The scala tympani finishes at the round window at the basal end, a flexible membrane made of 2 epithelial sheets with connective tissue in middle - contains arteries &veins and collagen. In middle ear, the superior portion of the inner epithelium is covered in perilymph while the outer one is open to air. The vestibular system's vestibule and perilymphatic compartment are continuous with the scala vestibuli at its basal end. Oval window that opens over the vestibule is covered in a membrane and contains the stapes footplate. A ligament fills the space between the stapes footplate's border and the oval window's edge. There is a small gap called the helicotrema through which the S.vestibuli & S.tympani are joined at the apical part of cochlea. The epithelial tissue that closes the scala media was partially formed by the extension of Reissner's membrane.

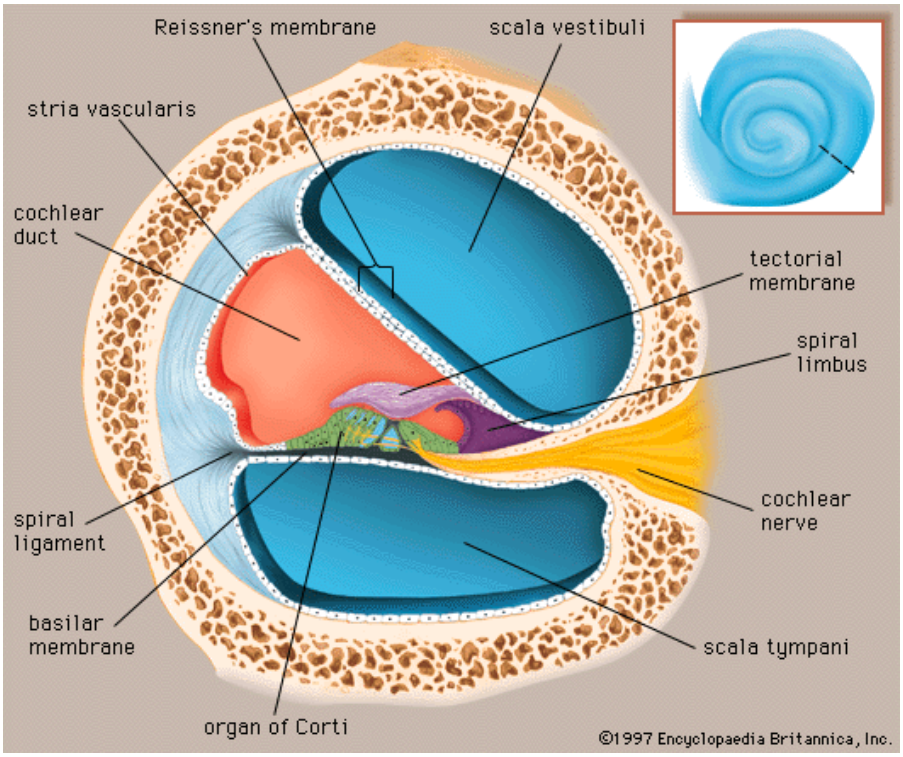


Figure3- The Cochlea

PHYSIOLOGY:

THE VESTIBULAR SYSTEM'S ROLE: The vestibular system's primary job: create info for the CNS, serving 4 purposes:

1. To guarantee eye stabilization
2. To allow for balanced movement and posture.
3. To give the body an overall direction with respect to gravity.
4. To refocus the body after autonomic functions have been adjusted.

The optical, vestibular, and proprioceptive systems, as well as other signals like hearing, even if they are of lesser importance, all provide information to the brain.

The unique outcomes that result from the brain's core processing of all of this information are as follows:

- The vestibulo-ocular reflex (VOR) for gaze stability.
- The vestibulospinal reflex (VSR) and vestibulocollic reflex (VCR) maintain the body in an upright position and stabilize the trunk and head in space.
- Adjustments to autonomic function follow changes in body orientation.

Since the Vestibular system is proven that it affects circadian rhythm and related to cognitive performance, this list of outcomes is by no means limited to these four tasks.

It is evident that balance cannot rely just on eyesight when walking in the dark and must instead rely on somatosensory & vestibular senses. All systems are required for daily operations to run smoothly, and there is limited system redundancy.

Motion decomposition & orientation in head:

Each movement in space is examined using 3 translational & 3 rotational degrees of freedom (yaw, pitch, and roll) (left–right, up– down, fore–aft). Anatomy of motion sensors in the peripheral vestibular system of the inner ear reflects these 6 degrees of freedom. Maculae detect primarily translation, whereas SCCs assess predominantly rotations. At first , one can assume the left & right canal as parallel systems, i.e. right anterior (RA) is parallel to left posterior (LP) and both lie in RALP plane.

The LARP plane is made up of LA and RP canal. Additionally both horizontal canals are parallel in lateral plane. The upright head's horizontal axis and horizontal SCC are around 30° angle to each other w.r.t sagittal plane of skull, vertical canal angle is roughly 45°.

Movement detection:

The vestibular system uses fundamental physical principles to sense movement or orientation. According to Newton's first law, "moving objects will continue to move until acted upon by a force." The direction or speed of the object may alter as a result of this force. According to the second law, an F(force) = m(mass) * a(acceleration).

Head and body are constantly moving throughout daily life (the head and body are even somewhat shaken during each pulse), & these movements are influenced by force or acceleration. The vestibular organ detects these movements thanks to a strict connection of the sensory epithelium to the bone structure. As a result, a system filled with fluid is fastened to the skull, and inertial forces cause the fluid to move. Any movement of the head causes the fluid to travel more slowly & this displacement serves as signal for movement detection.Canal system is constructed so that the deflection of the hair cells is proportionate to the head velocity in order to reduce the movement of these cells caused by the accelerations that propel the head movements.

OTOLITH ORGANS:

Starting a motion in a straight line causes the otolithic organs to sense linear acceleration and gravity.Sense of motion and orientation to gravity are worse in humans and animals whose otolith organs are injured or lacking. A group of hair cells and a pile of stones are connected. In relation to the hairs, the stones accelerate and

apply a shearing force to the hairs. The vestibular nerve branches carry this force to the brain after being picked up by the hair cells. Both the saccule& the utricle get input from nerve's superior and inferior divisions, respectively.Depending on their orientation, the otolithic organs detect motion. The utricle in the head is mostly horizontal and primarily detects accelerations in head's axial/horizontal plane. Saccule in the head is primarily vertical, or parasagittal, it senses accelerations in the sagittal plane. However, the saccule function has a downward-facing "blind spot" (Dimiccoli, M., et al., 2013). Numerous reflexes rely on the motion perception provided by the otoliths respectively. The structure of the utricle and saccule is significantly more complex, with several types of hair cells.

Otolith-Ocular reflexes

l-nystagmus (linear nystagmus)

Off-vertical axis nystagmus and ocular counter-roll

- Reflexes in the otolith body

  Reflex of Righting

- The Otolith-canal interactions

  "Dumping" the VOR

o Tilt-dependent modulation of the canal time constant (e.g. Hain and Buettner, 1990).

It may be challenging to stabilise the eyes and the body if the otoliths or their core connections are damaged (Lempert et al, 1997). Additionally, it appears that intra-vestibular conflict, which is indicated by otolithic sensors that indicate the head is rotating more slowly than the canals, could be the cause of certain motion

intolerance.The otoconia deteriorate with age, becoming hollowed down, fragmented, and rough. Demineralization is primary pathway for deterioration. Walther and Westhoven stated saccule volume ratio as 100:21 while the utricular otoconia had a ratio of 100:42. This explains the increasing decline in VEMP amplitude with age and is a result of natural ageing.

BPPV is assumed to be due to debris, likely otoconial, that has accumulated inside a portion of inner ear. BPPV is seen in higher rate in older age possibly as a result of otoconia degeneration.

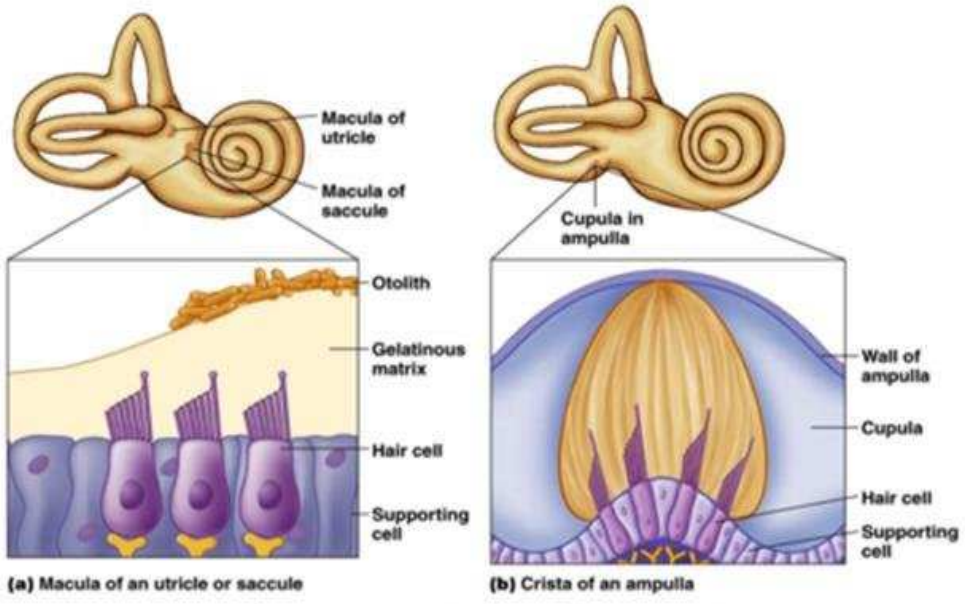


Figure4 - Otolith organs

SEMICIRCULAR CANALS:

The primary signal for the head's movement sensing is the moving endolymph in the SCCs.Transient rotations predominate in natural movements, such as head

movements back and forth (such as nodding) or walking and sprinting. These movements are catered for by the SCC system.

The sensory epithelium is activated by head accelerations, but the signal that is sent to the brain is proportionate to head velocity. While head velocity is restricted to several hundred degrees per second, head accelerations can reach up to several thousand degrees per second square. Sustained velocity is no longer detected nor perceived after approximately 30 seconds.3 forces act on endolymph & cupula in canal as it is rotated about an axis:

- Inertial force, proportional to the mass of the endolymph and cupula.
- Elastic restoring force of the cupula that positions the cupula back to the centre position after stimulation
- Fluid's resistance to sliding past the tube's internal wall due to viscous forces. This viscous force is influenced by how quickly the endolymph moves relative to the wall.

A variety of responses are triggered by any stimulus (head angular acceleration), which determines how the SCC behaves and is known as the pendulum model.

BENIGN PAROXYSMAL POSITIONAL VERTIGO:

It is a vestibular labyrinth condition characterized by one or more episodes of a transient, persistent, and intense spinning feeling brought on by adjustments to posture and head position.[48] Most frequent cause of peripheral vertigo and frequently impacts roughly one-third of persons with balance issues.[49] Compared to adults, children have

a decreased prevalence of BPPV. [50,51]Youngsters experience BPPV at a lower rate than adults do because otoconia separation is caused by less common conditions in children (hypertension, metabolic disorders, atherosclerosis).[52]

The otoconia debris that enters SCCs (canalolithiasis) / sticks to cupula (cupulolithiasis), altering the SCC' sensitivity to gravity, is one of the pathophysiological reasons of BPPV that is well understood. Old age, head injury, vestibular neuritis, Meniere's illness, migraines, ear surgery, and extended bed rest are a few of the established risk factors for BPPV.

PATHOPHYSIOLOGY:

The two dominant ideas surrounding the pathogenesis of BPPV are canalithiasis and cupulolithiasis.Otoconia sticks to cupula itself in cupulolithiasis . System becomes sensitive to gravitational force as a result of this cupular loading, and changes in cupular deflection that follow result in abnormal perceptions of motion. According to certain views, this mechanism may represent the more chronic type of BPPV.[53]

In their in vivo study, Parnes and McClure were the first to show that the canalithiasis model mentions free-floating particles within the canal lumen as the primary component. [54]One can see the free-floating particles. Gravity forces the particles into the endolymph canal, producing a plunger-like action that causes ipsidirectional cupular displacement, which results in an abnormal signal. The most frequent subtype, according to a clinicopathological investigation, is canalithiasis.[55.] It has been widely speculated that the canaliths responsible for the development of illness may be caused by displaced portions of the otolithic membrane.

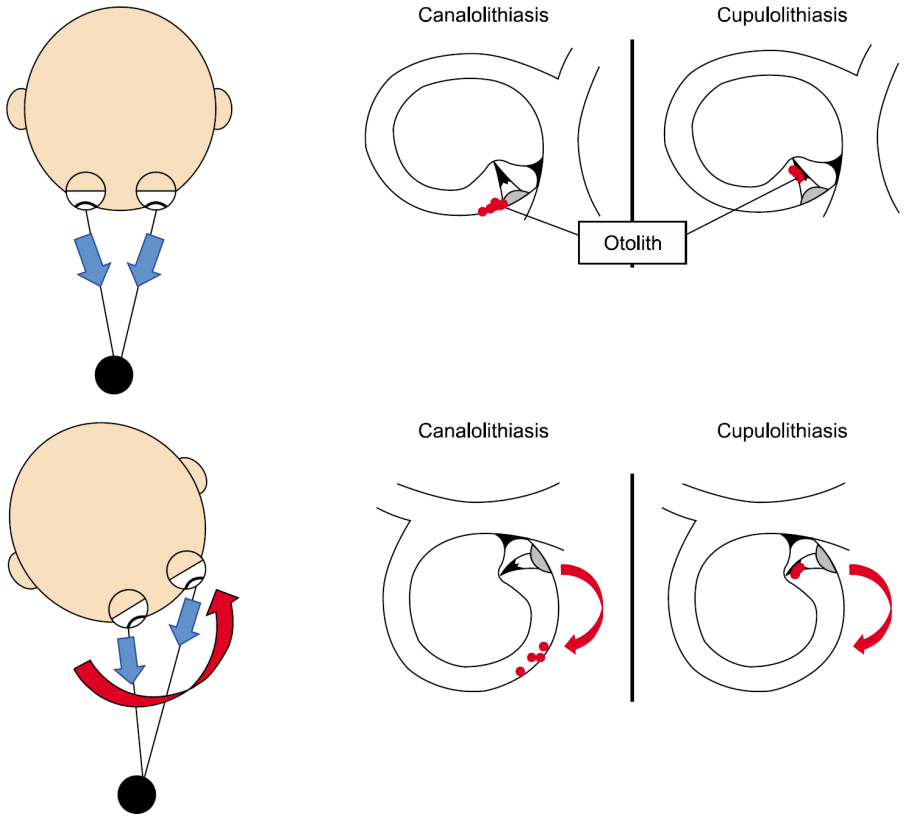


Figure5- Pathophysiology of BPPV

Canalolithiasis may be converted to cupulolithiasis when otoconial debris floating in the canal attach to the cupula.

SYMPTOMS AND NATURAL COURSE OF THE DISEASE:

Post.SCC is the most prevalent site of BPPV onset (p-SCC). Anterior SCC (a-SCC) is very seldom damaged while the horizontal SCC(h-SCC) is involved much less commonly.Size of the common crus of post. & sup. SCC, its placement underneath the utricle when supine, and its dependent posture while both erect and supine are thought to be the morphological correlates of the tendency for particle accumulation in the posterior canal.

Particles can become trapped in the posterior canal; any debris that may have entered the superior canal is more likely to fall back into the utricle. In the upright position, the horizontal canal's opening into the utricle is undermost, thus facilitating particle migration out of the canal, while the posterior canal's opening is uppermost. Therefore, the horizontal canal can be easily cleared by natural head movements but the posterior canal is a trap for any particles that have entered it. Similarly, particles may leave the horizontal canal just by the action of the person rolling over in bed whereas particles in the posterior canal will only be shifted backwards and forwards. This may explain the infrequent presentation of h-BPPV. During manoeuvres , p-BPPV can be converted to either a-BPPV or h- BPPV to p-BPPV and a-BPPV to p-BPPV. Similarly, bilateral apogeotropic h-BPPV may spontaneously convert to bilateral geotropic and is attributed to debris located in anterior part of SCC sticking to cupula dislodging & shifting to the post. part of canal. Plugging of the horizontal canal presenting with persistent spontaneous nystagmus, vertigo and oscillopsia has also been described .Vertigo lasting a few seconds, with or without nausea, and imbalance when rolling over in bed, sitting up from a laying position, or lying down, as well as when extending or contracting the neck, are the distinguishing features of p-BPPV. These symptoms can present in clusters with several attacks per day. In between attacks or shortly after successful treatment, patients are either symptom-free or experience a sensation of imbalance. This sensation of imbalance can be described as 'walking on pillows' and may be attributed to the underlying damage of the otolith organs. However, some patients may report atypical symptoms, and it is worthwhile conducting the positional tests in all patients presenting with episodic vertigo.

DIAGNOSIS:

Nystagmus and nausea, which are typically brought on by certain positional tests, are used to make the diagnosis of BPPV.

Understanding the characteristic eye movements during these tests will help in making the diagnosis. Ewald has described the following vestibular eye movements :

- The direction of eye movement is in the plane of the canal or canals that are stimulated.

- In the horizontal canal, endolymph flow towards the ampulla (ampullopetal) results in an excitatory and stronger response than flow away from the ampulla (ampullofugal), which is inhibitory. The opp. holds true for vertical SCC. When doing Dix-Hallpike test, for instance, the posterior canal and anterior canal pairs are activated. In order to line the vertical canals with the sagittal plane, the patient is seated with their feet up and their head tilted 45 degrees in the direction of the side being checked. The head is immediately positioned 45 degrees to the side being evaluated while being quickly lowered down over the end of the couch to rest there.

LARP and RALP canals are stimulated during right Dix-Hallpike and the left Dix-Hallpike. The right posterior canal experiences ampullofugal (excitatory) flow during the right Dix-Hallpike, while the left anterior has ampullopetal (inhibitory) flow. Normally this balances out and there is no nystagmus. However, if there were particles in the posterior canal, the ampullofugal forces would be greater, resulting in a net excitatory effect and eye movement consistent with the plane of the canal. The slow-phase eye movements when excitatory would be towards the right ear or

downwards and inwards via superior oblique muscle of the same side & inferior rectus muscle of opp side.Fast phase would be upwards and outwards or upbeating geotropic-torsional nystagmus. This is best seen by closely watching the scleral vessels. The nystagmus typically reverses when sitting up. A cupulolithiasis type BPPV can also rarely occur and is characterized by nystagmus that occurs without latency and does not fatigue.

An option that has been demonstrated to have outcomes comparable to those of the Dix-Hallpike test is the side-lying test. The vertical canals are in the frontal plane, as opposed to the Dix-Hallpike, where the head is rotated 45 degrees away from the side being tested. The patient is quickly moved to a supine-lying position with the neck hyperextended 20 degrees. The typical torsional nystagmus is seen beating towards the lowermost ear.

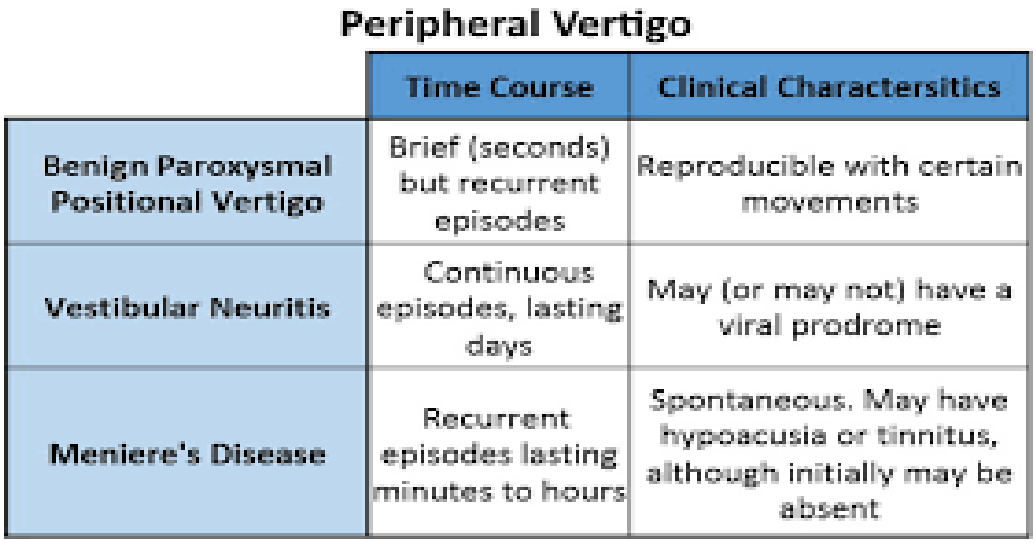
Similarly, the anterior canal during a right Dix-Hallpike or side-lying tests will be inhibited, i.e. flow will be ampullopetal. If particles are present, they will cause an inhibitory effect which will predominate causing the opposite nystagmus to the posterior canal – beating downwards and intorsional in the ipsilateral eye.The straight head-hanging method, in which the head is extended backwards to a head-hanging posture of the end of the bed from seated to supine, has also been proven to be beneficial for diagnosing a-BPPV.. Horizontal canal BPPV is assessed using the roll test. The head is flexed 30 degrees, bringing the horizontal canal into the axial plane, and is then briskly rolled to one side. The same is repeated to the opposite side. In the majority of cases the nystagmus will be horizontal and geotropic and towards the ear being tested. When turned to the opposite side, the nystagmus will reverse and beat towards the undermost ear again. When a canal is stimulated with an ampullopetal

force, the nystagmic response is greater (Ewald's second law), therefore the ear containing the loose particles would generally present with the brisker nystagmus hence helping to make the diagnosis. An apogeotropic nystagmus is also described and is thought to be related to cupulolithiasis. In this situation, when the head is turned to the affected side, the force is ampullofugal hence the nystagmus will beat away (apogeotropic) from the affected ear. Frenzel glasses can be used to better observe the nystagmus or it can be recorded with electro- or video-oculography. As BPPV can sometimes coexist with other vestibular disorders and the history can sometimes be atypical, many would perform a Dix– Hallpike routinely as part of a vestibular assessment.

DIFFERENTIAL DIAGNOSIS:

There are several potential differential diagnoses for benign paroxysmal positional vertigo, including:

- Ménière disease[56]
- Vascular loop dysfunction; labyrinthitis or vestibular neuronitis with alcohol consumption; concussion of the inner ear and orthostatic hypotension.
- Nodular lesion caused by diseases such ischemia, intoxication, multiple sclerosis, Arnold-Chiari malformation, stroke, and multiple sclerosis[58].
- Acoustic neuroma and meningioma
- Vertebral artery blockage



Peripheral Vertigo

	Time Course	Clinical Characterisitics
Benign Paroxysmal Positional Vertigo	Brief (seconds) but recurrent episodes	Reproducible with certain movements
Vestibular Neuritis	Continuous episodes, lasting days	May (or may not) have a viral prodrome
Meniere's Disease	Recurrent episodes lasting minutes to hours	Spontaneous. May have hypoacusia or tinnitus, although initially may be absent

Figure 6 - Differential diagnosis

Cause	Type of dizziness	Clinical characteristics	Treatment
BPPV	Vertigo, lasting seconds	Episodic vertigo on lying down or looking up, without hearing loss Positive Dix-Hallpike test	Canalith repositioning manoeuvre
Meniere's disease	Vertigo, minutes to hours	Episodic vertigo associated with hearing loss, tinnitus, or aural fullness	Vestibular sedative, low-salt diet, diuretics, vasodilator, intratympanic gentamicin, surgery
Vestibular neuritis	Vertigo, hours to days	Vertigo without hearing loss	Vestibular sedative in acute phase, vestibular rehabilitation
Hyperventilation	Lightheadedness	Associated with perioral or limb numbness; reproduce on hyperventilation	Rebreathing into paper bag, treat underlying psychiatric problem
Orthostatic hypotension	Pre-syncope	SBP decrease >20 mm Hg, DBP decrease >10 mm Hg, or pulse increase of 30 bpm	Review medication; fludrocortisone, midodrine in severe cases
Cardiovascular	Pre-syncope	Exertional chest pain, palpitation	Need cardiovascular assessment
Vestibular migraine	Vertigo	Episodic vertigo with migrainous aura or headache	Migraine prophylaxis
Peripheral neuropathy	Disequilibrium	Decreased extremities sensation	Treatment of underlying cause
Labyrinthitis	Vertigo	With hearing loss and ear discharge	Treat infection, vestibular sedative
Perilymph fistula	Vertigo	Episodic vertigo with hearing loss, fistula sign positive	Surgical repair, bed rest
Cerebrovascular disorder	Vertigo	Persistent vertigo with other neurological signs	Need neurological assessment
Disequilibrium of ageing	Disequilibrium	Multi-sensory deficits	Walking aid, vestibular rehabilitation

Fugure 7 – Causes, types, characteristics and treatment

MANAGEMENT:

Aims of management:

- Increase or modify the blood flow to the inner ear.
- Improve circulation of endolymph
- Treat underlying cause if any

Drugs used :

- Vasodilators − Eg: Betahistine HCl
- Labyrinthine suppressants− Eg: Cinnerizine
- Diuretics − Eg: Acetazolamide
- Anti-anxiety agents − Eg: Diazepam
- Anti-depressants − Eg: Amitriptiline
- Corticosteroids − Eg: Methyl- prednisolone
- Anti-bacterial drugs − Eg: Amoxicilline-clavulinic acid

Betahistine :

Mechanism of action :

Acts specifically on the precappilary sphincter- increasing blood flow through inner ear by 50% Increases blood flow through the vascular system of inner ear - generates of collateral blood circulation.

Decreases lymphatic or inner ear pressure , accompanying secretion or absorption of the endolymph.

Removes endolymphatic hydrops of inner ear - improves the permeability of the vascular walls of small blood vessels

Vitamin D in BPPV:

The migration of calcium carbonate crystals (otoconia) from the utricle into the semicircular canals is one of the established ideas for the aetiology of BPPV (most commonly the posterior canal). Vitamin D is essential for calcium metabolism and may have an impact on the density and matrix of calcium carbonate crystals (otoconia). Otoconia crystals mostly consist of a central core and an outer zone. With a lower amount of Ca2+, the centre is primarily organic (which is primarily glycoprotein), while the periphery zone is primarily inorganic (which is primarily a polymorph of calcium carbonate, or CaCO3), with a greater level of Ca2+. The crystals' inside, exterior surface, and interconnecting fibrous material are all organised and have varying sizes. Otoconia crystals are created by the vestibular organ's active calcium metabolism. Otoconia crystals are joined to hair cells by protein fibre and are partially encased in a fibrous matrix. Vitamin D affects and increases the kidney's and intestine's absorption of calcium. Parathyroid hormone regulates the blood calcium's ability to stay in balance. Vitamin D directly affects the mechanisms of calcium and phosphate deposition in bone, teeth, and the creation of otoconial particles in the vestibular system. There are similarities in the biomineralization of bone and otoconia. Since the two tissues' protein composition and matrix architecture are comparable.In order to achieve biomineralization in otoconia, an organic matrix must be carefully controlled to form at only certain locations, and mineral crystallites must deposit in an organised pattern like to that of bone and teeth. Similar to how vitamin D levels and calcium crystal deposition affect bone structures, so do the otoconia matrix and density. The severity of BPPV and its recurrence were both associated with vitamin D deficiency. In fact, vitamin D administration may reduce BPPV recurrent attacks. It's interesting to note that several instances have seen a full recovery following vitamin D

treatment studies. The calcium channel transport systems of the labyrinth are the basis of the vitamin D receptors, which control the right calcium balance. This process might make it easier to understand how vitamin D supports healthy auditory function.Cochlear demineralization and cochlear deafness have been linked to vitamin D deficiency. Since calcium ions are crucial for membrane permeability, a vitamin D deficiency may manifest as impaired calcium metabolism. For the nerve to operate normally, ionised calcium is required, and a lack of it may impair the cochlea's ability to generate action potentials. Low vitamin D and calcium levels can cause the otic capsule to demineralize as well as spiral ligament, stria vascularis, and cochlear hair cell degenerative alterations. The degree of hearing was reported to have improved by Brooks et al. after serum vitamin D levels were restored.

PROGNOSIS:

At three weeks, one-third of patients experience recurrence. Most patients experience remission after six months. [59] Only 1% of BPPV patients will ever require surgery, but since BPPV is a common disorder, there are a lot of surgical possibilities.[60]

COMPLICATION:

The complications are ongoing nausea and vomiting. A collision may happen as a result of benign paroxysmal positional vertigo brought on by abrupt head movements when riding a bike or driving. Serious mishaps involving work or recreational pursuits could happen after an episode of BPPV.

METHODOLOGY:

From January 2021 to December 2021, the Department of Otorhinolaryngology and Head & Neck Surgery at Jawaharlal Nehru Medical College conducted the current study to examine the effectiveness of betahistine and vitamin D supplementation in treating BPPV patients. A total of 50 patients were selected at random for the investigation.

STUDY PERIOD : 1 year

STUDY DESIGN: Randomised Controlled Trial .

STUDY POPULATION:

Study was conducted in patients who are confirmed cases of BPPV above 18 years of age that came to department of Otolaryngology who filled inclusion criteria .

INCLUSION CRITERIONS:

All patients who are confirmed cases of BPPV above 18 years of age that came to department of Otolaryngology.

EXCLUSION CRITERIONS:

All patients with other forms of peripheral and central vertigo, individuals with traumatic head injury , who have had middle ear surgeries , pregnant women, patients receiving chemotherapy or ototoxic drugs will be excluded.

SAMPLE SIZE: The minimal sample size was 50 and they were divided into two groups with minimum of 25 patients in each groups.

METHOD OF RANDOMIZATION - Computer generated Randomisation.

STATISTICAL ANALYSIS: The comparison of the two groups is the study's principal goal. We shall compute the mean and standard deviation for the continuous quantitative data. The unpaired student's t test and other appropriate statistical methods will be used to compare the continuous variables between the groups. The student will compare two quantitative variables within a group using the paired t test.

Rates, ratios, and percentages will be used to express the categorical data. Using the Chi-square test or Fisher's exact test, the relationship between the result, clinical, and demographic factors will be examined. The median will serve as a representation for discrete variables. Discrete variables will be compared using non-parametric testing.

The appropriate graphs will be used to display the comparison. For all tests, the value of p less than 5% (0.05) will be taken into account.

STUDY SETTING:The patients coming to the Otolaryngology clinic with complaints of frequent attacks of dizziness were evaluated for Vertigo using a Dizziness History Questionnaire.

The subjects were evaluated for signs of peripheral vertigo using Dix Hallpike Manoeuvre. Once the diagnosis of BPPV was confirmed , the serum Vitamin D levels were measured .Informed consent was taken before performing the procedure.The subjects were allocated into two groups randomly:

Group A received 60,000 IU Cholecalciferol weekly along with Betahistine for three months .

Group B received only Betahistine .

The results of treatment was compared between the two groups .

The compliance was recorded by maintaining a log book.

SPECIAL INVESTIGATION: The study required special investigation of Serum Vitamin D level and supplementation of vitamin D.

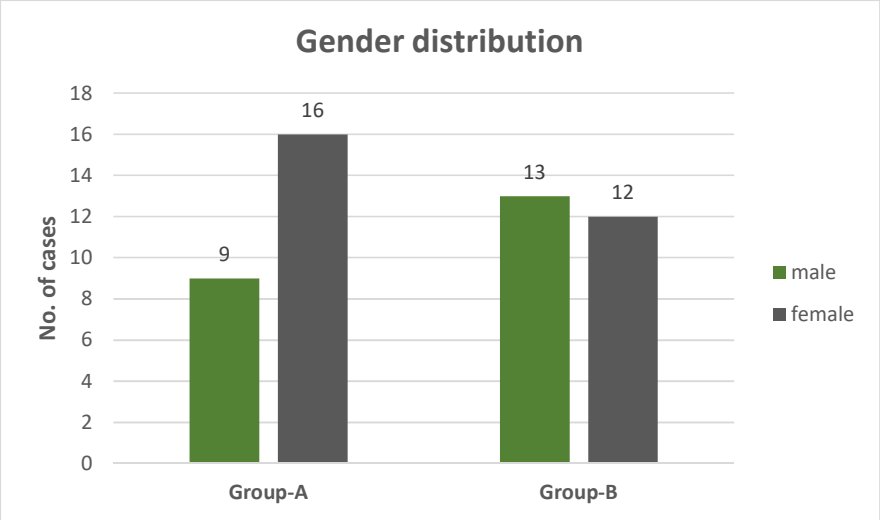
RESULTS-

Group A received 60,000 IU Cholecalciferol weekly along with Betahistine for three months.

Group B received only Betahistine.

Table-1: Gender wise distribution of patients in both groups.

Demographic data	Group-A	Group-B
Gender	**No. of cases (%)**	
Male	9 (36%)	13(52%)
Female	16 (64%)	12(46%)

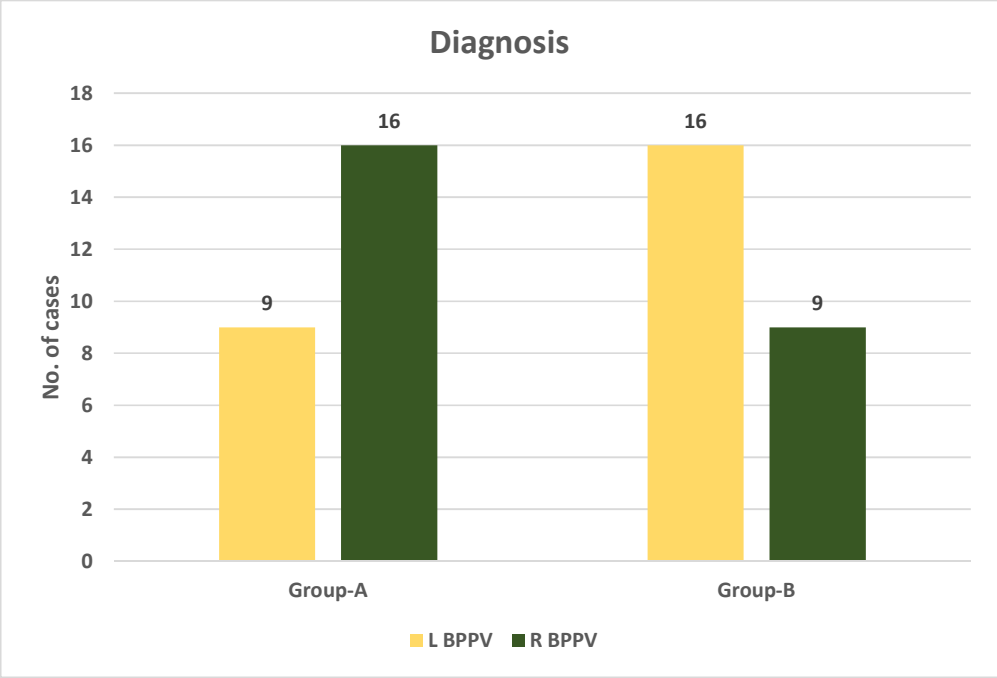


Graph -1: Gender wise distribution of patients in both groups.

Their mean age was 41.96 ± 15.01 years, and group A consisted of 16 girls and 9 males (range: 21–60 years). There were 13 girls and 12 males found in group-B, and their mean age ranged from min to max at 41.88 ± 12.33 years (Table 1, figure-1)

Table-2: Diagnosis types in group-A and group-B.

Diagnosis	Group-A	Group-B
L BPPV	9 (36%)	16(64%)
R BPPV	16 (64%)	9 (36%)



Graph -2: Diagnosis types in group-A and group-B.

Table 2 shows that patients with L BPPV were determined to be 9 (36%) and 16 (64%) in groups A and B, while those with R BPPV were 16 (64%) and 9 (36%). (Figure-2).

Table-3: Associated symptoms in groups

ASSOCIATED SYMPTOMS	Group-A	Group-B
	No. (%)	
DECREASED HEARING	6	11
NIL	5	4
TINNITUS	14	10

Both groups observed the patients as having symptoms such as decreased hearing and symptoms of tinnitus six and eleven. 14, 10 patients were observed in groups A and B, and 5, 4 patients in groups A and B had no symptoms. (Table-3).

Table-4: Comorbidities in both groups.

COMORBIDITIES	Group-A	Group-B
DM	3(12%)	5 (20%)
HTN	6 (24%)	5 (20%)
MIGRAINE	11 (44%)	12 (48%)
NIL	5 (20%)	3(12%)

Table 4 shows that in group A, 11 (44%) patients had diabetes mellitus, 6 (24%) had HTN, and 5 (20%) had no co-morbid conditions. There were conditions in group-B individuals with diabetes mellitus 5(20%), HTN 5(20%), migraine 12 (48%), and 3(12%) patients had no comorbidities.

Tabel-5: Previously Treated For Dizziness.

PREVIOUSLY TREATED FOR DIZZINESS	Group-A	Group-B
Yes	8 (32%)	11 (44%)
No	17 (68%)	14 (56%)

From table-5 patients with dizziness in group-A 8(32%), 11(44%) from group-B were treated for dizziness.

Table-6: Before treatment use of episodes on daily.

Before Treatment (Group-A)		
No. of episode	**No. of cases**	**Percentage**
6	2	**8.33%**
7	4	**16.67%**
8	10	**41.67%**
9	7	**29.17%**
10	2	**8.33%**

According to Table 6, before starting therapy,10 patients in Group A had an average of 8 episodes per day (41.67%), while only 2 patients had a maximum of 10 episodes per day (8.33%)

Table-7: Distribution of episodes in following months in group-A

After Treatment (Group-A)		
No. of episode (1 month)	**No. of cases**	**Percentage**
3	1	4.17%
4	5	20.83%
5	1	4.17%
6	14	58.33%
7	4	16.67%
No. of episode (2 month)	**No. of cases**	**Percentage**
1	1	4.17%
2	3	12.50%
3	11	45.83%
4	10	41.67%
No. of episode (3 month)	**No. of cases**	**Percentage**
0	16	66.67%
1	6	25.00%
2	3	12.50%

After one month of treatment in group A, a maximum of 14 patients (58.33%) had six episodes per day. After 2 and 3 months, there were 11 (45.83%) patients who had 3 episodes, 6 (25%) patients had only one episode, and 16 (66.67%) patients who did not have any episodes at all (Table-7).

Table-8: Before treatment use of episodes on daily.

Before Treatment in group-B		
No. of episodes	**No. of cases**	**Percentage**
4	3	12.00%
5	6	24.00%
6	13	52.00%
7	2	8.00%
8	1	4.00%

Table-9: Distribution of episodes in following months in group-B

After Treatment in group-B		
No. of episode after 1month	**No. of cases**	**Percentage**
2	2	8.00%
3	3	12.00%
4	16	64.00%
5	3	12.00%
6	1	4.00%
No. of episode after 2 months	**No. of cases**	**Percentage**
2	6	24.00%
3	19	76.00%
No. of episode after 3 months	**No. of cases**	**Percentage**
0	3	12.00%
1	8	32.00%
2	14	56.00%

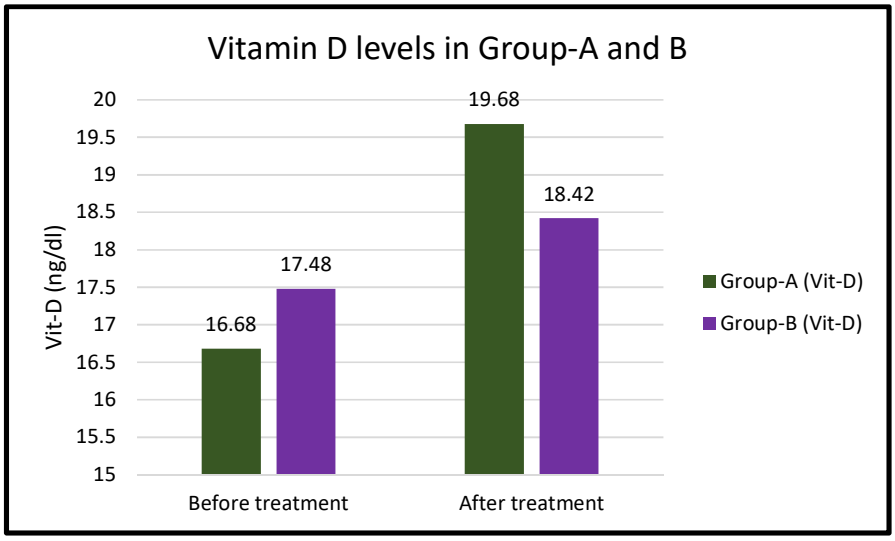
The maximum number of patients in group-B who had four episodes per day after a month of treatment was 16(64%), 19 patients (76%) had only three episode, while 14 (56%) had 2 episodes after two and three months and 3(12%) patients who did not have any episodes at all after 3 months.

Table-10: Vitamin D level in group-A.

Vitamin D in Group-A		p-value
Before treatment	**After treatment**	
16.68 (4.89)	19.68 (5.08)	0.001 [S]

Table-11: Vitamin D level in group-B.

Vitamin D in Group-B		p-value
Before treatment	**After treatment**	
17.48 (3.66)	18.42(3.57)	0.001 [S]



Graph -4: Graphical representation of vitamin D level between both groups.

In the present study, vitamin D levels were compared between Group-A and Group-B. Before treatment, group-A had a vitamin D level of 16.68, while group-B had a vitamin D level of 17.48. Whereas, Group-A showed 19.68 and Group-B showed 18.42 after treatment as illustrated in (Table-10, 11 and Figure-4).

DISCUSSION:

BPPV is one of the most prominent peripheral vestibular conditions. Numerous comorbidities, including hypertension, diabetes mellitus, thyroid disease, hearing loss, hyperlipidemia, and vitamin D deficiency, are interrelated with BPPV.

Recurrence of vertigo attacks is usual in such circumstances. In order to limit chronicity and decrease frequency of recurrence, these illnesses may benefit from the gold standard therapy technique.

This study's objectives were to measure vitamin D serum levels in BPPV patients and look into the connection between vitamin D insufficiency and repeated BPPV attacks. Additionally, it evaluated whether vitamin D intake in addition to betahistine reduced the number of recurrent BPPV attacks more than betahistine alone.

From January 2021 to December 2021, the study was carried out at the department of Otorhinolaryngology and Head & Neck Surgery at Jawaharlal Nehru Medical College ,Belagavi.

50 patients were considered in the study and the patients were confirmed cases of BPPV above 18 years of age.

In our study, subjects were allocated into 2 groups. Group A was receiving 60,000 IU Cholecalciferol weekly along with Betahistine for 3 months. Group B was receiving only betahistine.

In our study, the two groups had mean age of 41.96 ± 15.01 years, and group A consisted of 16 females and 9 males (range: 21–60 years). There were 13 females and 12 males found in group-B, and their mean age ranged from min to max at 41.88 ± 12.33 years. Out of total 50 cases which showed no statistical gender preponderance between both the groups.

Similar findings were made in a study by Rhim, G. I., who came to the hypothesis that serum vitamin D concentrations influence BPPV as a factor for recurrences regardless of age, gender, follow-up length, and kind of BPPV.[61] So the two semicircular canals most frequently impacted by BPPV are the posterior and lateral semicircular canals. Patients in groups A and B of our study with L BPPV were found to be 9 (36%) and 16 (64%) whereas those with R BPPV were found to be 16 (64%) and 9 (36%) respectively.

Repositioning techniques are used to treat BPPV with the goal of bringing the particles back to the utricle. Some individuals experienced vestibular-related tinnitus, which later went away right away after repositioning techniques. Tinnitus can be a symptom of numerous medical disorders, but its exact cause is still unknown. In our study, both groups of patients were observed having symptoms such as decreased hearing and symptoms of tinnitus.

While 14% in group A and 10% in group B experienced tinnitus symptoms, 6% in group A and 11% in group B experienced hearing loss symptoms. Patients in groups A and B, respectively, were 5% and 4% symptom-free. According to Ece Kocabaş, Ahmet Kutluhan, Banu Müjdec, et al study 's tinnitus in BPPV patients was found to be confined in the ear that was afflicted by the condition.[62] 23 tinnitus sufferers experienced tinnitus eradication following treatment procedures.

Pillai NG, Gopinath I. A in their study in 2019 studied the relationship between BPPV and vitamin D deficiency.[63] In this one year prospective study they found that, the definite co-morbidity for the development and recurrence of BPPV is vitamin D. Hence, patient presenting with BPPV should be evaluated for serum Vitamin D along with otoconial re-positioning maneuvers since vitamin D plays a vital role in mechanism of formation and maintenance of otoconial particles in vestibular system .

In our study, group A, 11 (44%) patients had diabetes mellitus, 6 (24%) had HTN, and 5 (20%) had no co-morbid conditions. There were conditions in group-B individuals with having diabetes Mellitus in 5(20%), HTN in 5(20%), migraine in 12 (48%) and 3(12%) patients had no comorbidities.

Such comorbidities were discovered in the study of Ioanna Sfakianaki, Paris Binos, and George Psillas et al., which looked at how individuals with hyperlipidemia and hypertension had the highest recurrence rates of BPPV, at 67.80% and 55.89%, respectively.[64] Additionally, more than half (53.48%) of patients with diabetes mellitus experienced a BPPV recurrence. Furthermore, considering that BPPV recurs in 41.71% of cases, it is likely that migraines also contribute to the condition's recurrence.

One of the most common medical symptoms, vertigo, often known as dizziness, affects 15-35% of the general population at some point in their lives (Kroenke and Price, 1993; Yardley et al., 1998; Hannaford et al., 2005; Gopinath et al., 2009; Wiltink et al., 2009; Mendel et al., 2010).[65]

Due to their frequent recurrence, dizziness and vertigo have a substantially higher yearly prevalence than incidence. In unselected adults, the annual incidence of dizziness, including vertigo, was estimated to be 3%. (Neuhauser et al., 2008). In our study , patients with dizziness in group-A 8(32%) and 11(44%) from group-B had treated for dizziness.[66]

Reduced BMD and the development or recurrence of BPPV are associated, according to Talaat HS, Abuhadied G, et al. Additionally, low vitamin D levels were linked to BPPV recurrence while low levels were responsible for BPPV formation, before starting therapy in our study , patients in Group A had an average of 10 episodes per day (41.67%), while only 7 episodes per day (29.17%) patients had. After one month of treatment in group A, a maximum of 14 patients (58.33%) had six episodes per day. After 2 and 3 months, there were 11 (45.83%) patients who had 3 episodes, 6 (25%) patients who had only one episode, and 16 (66.67%) patients who had no any episodes at all.[67] Similarly, The maximum number of patients in group-B who had four episodes per day after a month of treatment was 16(64%), 19 patients (76%) had only three episode, while 14 (56%) had 2 episodes after two and three months and 3(12%) patients who had no any episodes at all after 3 months.

In the present study, vitamin D levels were compared between Group-A and Group-B. Before treatment, group-A had a vitamin D level of 16.68, while group-B had a vitamin D level of 17.48. Whereas, Group-A showed 19.68 and Group-B showed 18.42 after treatment.

Recurring BPPV episodes were noticeably less frequent in group A than in group B. Low serum Vitamin D levels and BPPV recurrence are related.

Jeong SH, Kim JS, et al in the study conducted in 2013 suggest a interrelationship between decreased serum vitamin D and an link of osteoporosis with BPPV.[68] Since most of the studies correlating the role of vitamin D and BPPV have been conducted abroad and there are a very few studies conducted in India we need more studies to draw conclusion regarding the same. Therefore, in this research, we have examined how vitamin D and betahistine are used to treat BPPV. Recurring BPPV episodes were noticeably less frequent in group A than in group B.

Despite the recovery of BPPV with betahistine leads to remission in days and weeks, recurrence may develop in 50% of patients. So in such cases the prevention of recurrence can be obtained by treating the predisposing vitamin D deficiency level.

Hence, in this study we have assessed the effect between serum Vitamin D levels along with betahistine and betahistine alone in patients of BPPV.

Thus,Vitamin D had a significant role in treatment of BPPV along with Betahistine and routine testing of Sr.Vit D levels should be advocated . Correcting the vitamin D levels leads to lower recurrence of BPPV and the associated symptoms .Further studies with larger sample size can help to determine if Vitamin D supplementation can be a routine practise. The limitations of the study were the high cost of Vitamin D estimation and the follow up.

CONCLUSION:

The occurrence and recurrence of BPPV are both correlated with low vitamin D levels. A lower probability of BPPV relapse was associated with lessening vitamin D insufficiency, and this association was statistically significant. Following our research, we came to the conclusion that comorbidities would exacerbate BPPV and increase the frequency of otolith detachment. So even after a successful repositioning effort, it will increase the likelihood of recurrence. Therefore, in addition to repositioning techniques, individuals who present with BPPV should also be assessed and treated for any comorbidities. Our study's objective was to compare two groups' performance, and results showed that group A performed better than group B.

SUMMARY-

The department of otorhinolaryngology and head and neck surgery at Jawaharlal Nehru Medical College in Belagavi conducted a randomised control trial study from January 2021 to December 2021 comparing the effects of vitamin D supplementation with betahistine versus betahistine alone in the treatment of patients with BPPV. Fifty samples were used in the study.

After complete pre-operative evaluation, the patients were randomly divided into even and odd groups.

GROUP A- Received 60,000 IU cholecalciferol weekly along with betahistine for 3 months.

GROUP B- Received only betahistine.

Treatment outcomes for the two groups were contrasted. The unpaired student's t test and other appropriate statistical methods were used to compare the intergroup continuous variables. Using a paired t test, two quantitative variables within a group were compared.

Rates, ratios, and percentages were used to express the categorical data. Using the Chi-square test or Fisher's exact test, the relationship between the result, clinical, and demographic factors will be examined.

In the present study, vitamin D levels were compared between Group-A and Group-B. Before treatment, group-A had a vitamin D level of 16.68, while group-B had a vitamin D level of 17.48. Whereas, Group-A showed 19.68 and Group-B showed 18.42 after treatment. Though it was found in our study that group A patient showed better outcomes in contrast to group B patients.

BIBLIOGRAPHY

1. Thompson TL, Amedee R. Vertigo: a review of common peripheral and central vestibular disorders. Ochsner J. 2009 Spring;9(1):20-6. PMID: 21603405; PMCID: PMC3096243.

2. von Brevern M, Radtke A, Lezius F, Feldmann M, Ziese T, Lempert T, Neuhauser H. Epidemiology of benign paroxysmal positional vertigo: a population based study. J Neurol Neurosurg Psychiatry. 2007 Jul;78(7):710-5. doi: 10.1136/jnnp.2006.100420. Epub 2006 Nov 29. PMID: 17135456; PMCID: PMC2117684.

3. Liu DH, Kuo CH, Wang CT, Chiu CC, Chen TJ, Hwang DK, Kao CL. Age-Related Increases in Benign Paroxysmal Positional Vertigo Are Reversed in Women Taking Estrogen Replacement Therapy: A Population-Based Study in Taiwan. Front Aging Neurosci. 2017 Dec 12;9:404. doi: 10.3389/fnagi.2017.00404. PMID: 29311896; PMCID: PMC5732995.

4. Agrawal Y, Ward BK, Minor LB. Vestibular dysfunction: prevalence, impact and need for targeted treatment. J Vestib Res. 2013;23(3):113-7. doi: 10.3233/VES-130498. PMID: 24177344; PMCID: PMC4069154.

5. Chen, ZJ., Chang, CH., Hu, LY. et al. Increased risk of benign paroxysmal positional vertigo in patients with anxiety disorders: a nationwide population-based retrospective cohort study. BMC Psychiatry 16, 238 (2016).

6. Gyo K. Benign paroxysmal positional vertigo as a complication of postoperative bedrest. Laryngoscope. 1988 Mar;98(3):332-3. doi: 10.1288/00005537-198803000-00019. PMID: 3343883.

7. Parnes LS, Agrawal SK, Atlas J. Diagnosis and management of benign paroxysmal positional vertigo (BPPV). CMAJ. 2003 Sep 30;169(7):681-93. PMID: 14517129; PMCID: PMC202288.

8. Hornibrook J. Benign Paroxysmal Positional Vertigo (BPPV): History, Pathophysiology, Office Treatment and Future Directions. Int J Otolaryngol. 2011;2011:835671. doi: 10.1155/2011/835671. Epub 2011 Jul 25. PMID: 21808648; PMCID: PMC3144715.

9. Lundberg YW, Xu Y, Thiessen KD, Kramer KL. Mechanisms of otoconia and otolith development. Dev Dyn. 2015 Mar;244(3):239-53. doi: 10.1002/dvdy.24195. Epub 2014 Oct 18. PMID: 25255879; PMCID: PMC4482761.

10. Argaet EC, Bradshaw AP, Welgampola MS. Benign positional vertigo, its diagnosis, treatment and mimics. Clin Neurophysiol Pract. 2019 Apr 6;4:97-111. doi: 10.1016/j.cnp.2019.03.001. PMID: 31193795; PMCID: PMC6542326.

11. Abdelmaksoud AA, Fahim DFM, Bazeed SES, Alemam MF, Aref ZF. Relation between vitamin D deficiency and benign paroxysmal positional vertigo. Sci Rep. 2021 Aug 19;11(1):16855. doi: 10.1038/s41598-021-96445-x. PMID: 34413436; PMCID: PMC8377140.

12. Brookes GB. Vitamin D deficiency--a new cause of cochlear deafness. J Laryngol Otol. 1983 May;97(5):405-20. doi: 10.1017/s0022215100094330. PMID: 6602194.

13. Gröber U, Kisters K. Influence of drugs on vitamin D and calcium metabolism. Dermatoendocrinol. 2012 Apr 1;4(2):158-66. doi: 10.4161/derm.20731. PMID: 22928072; PMCID: PMC3427195.

14. Taneja MK, Taneja V. Role of vitamin D in prevention of deafness. Indian J Otol [serial online] 2012 [cited 2022 Oct 3];18:55-7.

15. Jeong SH, Kim JS. Impaired Calcium Metabolism in Benign Paroxysmal Positional Vertigo: A Topical Review. J Neurol Phys Ther. 2019 Apr;43 Suppl 2:S37-S41. doi: 10.1097/NPT.0000000000000273. PMID: 30883492.

16. 16.Büki B, Ecker M, Jünger H, Lundberg YW. Vitamin D deficiency and benign paroxysmal positioning vertigo. Med Hypotheses. 2013 Feb;80(2):201-4. doi: 10.1016/j.mehy.2012.11.029. Epub 2012 Dec 14. PMID: 23245911; PMCID: PMC4196321.

17. 17.Sheikhzadeh M, Lotfi Y, Mousavi A, Heidari B, Monadi M, Bakhshi E. Influence of supplemental vitamin D on intensity of benign paroxysmal positional vertigo: A longitudinal clinical study. Caspian J Intern Med. 2016 Spring;7(2):93-8. PMID: 27386060; PMCID: PMC4913711.

18. Iwasaki S, Yamasoba T. Dizziness and Imbalance in the Elderly: Age-related Decline in the Vestibular System. Aging Dis. 2014 Feb 9;6(1):38-47. doi: 10.14336/AD.2014.0128. PMID: 25657851; PMCID: PMC4306472.

19. 19.Casani AP, Gufoni M, Capobianco S. Current Insights into Treating Vertigo in Older Adults. Drugs Aging. 2021 Aug;38(8):655-670. doi: 10.1007/s40266-021-00877-z. Epub 2021 Jun 23. PMID: 34159566; PMCID: PMC8342368.

20. Lacour M, Sterkers O. Histamine and betahistine in the treatment of vertigo: elucidation of mechanisms of action. CNS Drugs. 2001;15(11):853-70. doi: 10.2165/00023210-200115110-00004. PMID: 11700150.

21. Elmoursy MM, Abbas AS. The role of low levels of vitamin D as a co-factor in the relapse of benign paroxysmal positional vertigo (BPPV). Am J Otolaryngol. 2021 Nov-Dec;42(6):103134. doi: 10.1016/j.amjoto.2021.103134. Epub 2021 Jun 19. PMID: 34166965.

22. Palmeri R, Kumar A. Benign Paroxysmal Positional Vertigo. [Updated 2022 Jan 3]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK470308/

23. Benign Paroxysmal Positional Vertigo (BPPV): History, Pathophysiology, Office Treatment and Future Directions Jeremy Hornibrook Department of Otolaryngology, Head and Neck Surgery, Christchurch Hospital, 2 Riccarton Avenue, Christchurch 8011, New Zealand Correspondence should be addressed to Jeremy Hornibrook, jeremy@jhornibrook.com Received 12 March 2011; Accepted 18 May 2011

24. von Brevern M, Radtke A, Lezius F, Feldmann M, Ziese T, Lempert T, Neuhauser H. Epidemiology of benign paroxysmal positional vertigo: a population based study. J Neurol Neurosurg Psychiatry. 2007 Jul;78(7):710-5.

doi: 10.1136/jnnp.2006.100420. Epub 2006 Nov 29. PMID: 17135456; PMCID: PMC2117684.

25. You P, Instrum R, Parnes L. Benign paroxysmal positional vertigo. Laryngoscope Investig Otolaryngol. 2018 Dec 14;4(1):116-123. doi: 10.1002/lio2.230. PMID: 30828628; PMCID: PMC6383320.

26. Bressi F, Vella P, Casale M, Moffa A, Sabatino L, Lopez MA, Carinci F, Papalia R, Salvinelli F, Sterzi S. Vestibular rehabilitation in benign paroxysmal positional vertigo: Reality or fiction? Int J Immunopathol Pharmacol. 2017 Jun;30(2):113-122. doi: 10.1177/0394632017709917. Epub 2017 May 9. PMID: 28485653; PMCID: PMC5806799.

27. İnan HC, Kıraç M. An Evaluation of the Effects of Betahistine and Dimenhydrinate on Posterior Canal Benign Paroxysmal Positional Vertigo. Turk Arch Otorhinolaryngol. 2019 Dec;57(4):191-196. doi: 10.5152/tao.2019.4185. Epub 2019 Dec 1. PMID: 32128517; PMCID: PMC7032557.

28. Teggi R, Guidetti R, Gatti O, Guidetti G. Recurrence of benign paroxysmal positional vertigo: experience in 3042 patients. Acta Otorhinolaryngol Ital. 2021 Oct;41(5):461-466. doi: 10.14639/0392-100X-N1233. PMID: 34734582; PMCID: PMC8569667.

29. Laird E, Ward M, McSorley E, Strain JJ, Wallace J. Vitamin D and bone health: potential mechanisms. Nutrients. 2010 Jul;2(7):693-724. doi: 10.3390/nu2070693. Epub 2010 Jul 5. PMID: 22254049; PMCID: PMC3257679.

30. Hornibrook J. Benign Paroxysmal Positional Vertigo (BPPV): History, Pathophysiology, Office Treatment and Future Directions. Int J Otolaryngol. 2011;2011:835671. doi: 10.1155/2011/835671. Epub 2011 Jul 25. PMID: 21808648; PMCID: PMC3144715.

31. Dix MR, Hallpike CS. The pathology, sympomatology and diagnosis of certain common disorders of the vestibular system. Annals of Otology, Rhinology and Laryngology. 1952;61:987–1016. [PubMed] [Google Scholar]

32. Ruben RJ. The development and acceptance of the association of diseases of the ear and disorders of balance. Meniere's disease. In: Nadol JB, editor. Proceedings Second International Symposium on Meniere's Disease. Kugler; 1989. pp. 3–11. [Google Scholar]

33. Schuknecht HF. Positional vertigo. Clinical and experimental observations. Transactions of the American Academy of Ophthalmology and Otolaryngol. 1962;66:319–331. [PubMed] [Google Scholar]

34. Schuknecht HF. Cupulolthiasis. Archives of Otolaryngology-Head & Neck Surgery. 1969;70:765–778. [PubMed] [Google Scholar]

35. Schuknecht HF, Ruby RRF. Cupulolithiasis. Advanced Oto-Rhino-Laryng. 1962;20:434–443. [PubMed] [Google Scholar]

36. Gacek RR. Transection of the posterior ampullary nerve for the relief of benign paroxysmal vertigo. Annals of Otology, Rhinology and Laryngology. 1974;83:596–605. [PubMed] [Google Scholar]

37. Cawthorne T. The physiological basis for head exercises. The Chartered Society of Physiotherapy. 1944;30:106–107. [Google Scholar]

38. von Brevern M, Radtke A, Lezias F, et al. Epidemiology of bengin paroxysmal positional vertigo: a population based study. Journal of Neurology Neurosurgery and Psychiatry. 2007;78:710–715. [PMC free article] [PubMed] [Google Scholar]

39. Oghalai JS, Mandolidis S, Bath JL, Stewart MG, Jenkins HA. Unrecognised benign positional vertigo in elderly patients. Otolaryngology-Head & Neck Surgery. 2000;122:630–634. [PubMed] [Google Scholar]

40. Davies RA, Luxon LM. Dizziness following head injury: a neuro-otological study. Journal of Neurology. 1995;242(4):222–230. [PubMed] [Google Scholar]

41. Hornibrook J. Immediate onset of positional vertigo following head injury. The New Zealand Medical Journal. 1998;111(1073):p. 349. [PubMed] [Google Scholar]

42. Lindsay JR, Hemmenway WG. Postural vertigo due to unilateral sudden partial loss of vestibular function. Annals of Otology, Rhinology and Laryngology. 1956;65:695–708. [PubMed] [Google Scholar]

43. Gyo K. Benign paroxymsal positional vertigo as a complication of postoperative bedrest. Laryngoscope. 1988;98(3):332–333. [PubMed] [Google Scholar]

44. Balzanelli C, Spataro D, Redaelli de Zinis LO. Benign Positional Paroxysmal Vertigo in Children. Audiol Res. 2021 Feb 1;11(1):47-54. doi: 10.3390/audiolres11010006. PMID: 33535402; PMCID: PMC7930952.

45. Lundberg YW, Xu Y, Thiessen KD, Kramer KL. Mechanisms of otoconia and otolith development. Dev Dyn. 2015 Mar;244(3):239-53. doi: 10.1002/dvdy.24195. Epub 2014 Oct 18.

46. 46..PMID: 25255879; PMCID: PMC4482761. Fetter M. Vestibulo-ocular reflex. Dev Ophthalmol. 2007;40:35-51. doi: 10.1159/000100348. PMID: 17314478.

47. Wallace B, Lifshitz J. Traumatic brain injury and vestibulo-ocular function: current challenges and future prospects. Eye Brain. 2016 Sep 6;8:153-164. doi: 10.2147/EB.S82670. PMID: 28539811; PMCID: PMC5398755.

48. Von Brevern, M.; Bertholon, P.; Brandt, T.; Fife, T.; Imai, T.; Nuti, D.; Newman-Toker, D. Benign paroxysmal positional vertigo: Diagnostic criteria. J. Vestib. Res. 2015, 25, 105–117. [CrossRef] [PubMed]

49. . Marcelli, V.; Piazza, F.; Pisani, F.; Marciano, E. Neuro-otological features of benign paroxysmal vertigo and benign paroxysmal positioning vertigo in children: A follow-up study. Brain Dev. 2006, 28, 80–84. [CrossRef] [PubMed]

50. Davitt, M.; Delvecchio, M.T.; Stephen, C. The differential diagnosis of vertigo in children: A systematic review of 2726 cases. Pediat. Emerg. Care 2020, 36, 368–371. [CrossRef]

51. Balzanelli C, Spataro D, Redaelli de Zinis LO. Benign Positional Paroxysmal Vertigo in Children. Audiol Res. 2021 Feb 1;11(1):47-54. doi: 10.3390/audiolres11010006. PMID: 33535402; PMCID: PMC7930952.

52. Fife TD, Iverson DJ, Lempert T, Furman JM, Baloh RW, Tusa RJ, et al. Practice parameter: therapies for benign paroxysmal positional vertigo (an evidence-based review): report of the Quality Standards Subcommittee of the American Academy of Neurology. Neurology. (2008) 70:2067–74. 10.1212/01.wnl.0000313378.77444.ac [PubMed] [CrossRef] [Google Scholar]

53. Parnes LS, Agrawal SK, Atlas J. Diagnosis and management of benign paroxysmal positional vertigo (BPPV). CMAJ 2003;169(7):681–693.

54. . Parnes LS, McClure JA. Free-floating endolymph particles: a new operative finding during posterior semicircular canal occlusion. 1992. Laryngoscope 2015;125(5):1033.

55. Lee S-H, Kim JS. Benign paroxysmal positional vertigo. J Clin Neurol 2010; 6(2):51–63.

56. Swartz R, Longwell P. Treatment of vertigo. Am Fam Physician. 2005 Mar 15;71(6):1115-22. [PubMed]

57. Parnes LS, Agrawal SK, Atlas J. Diagnosis and management of benign paroxysmal positional vertigo (BPPV). CMAJ. 2003 Sep 30;169(7):681-93. [PMC free article] [PubMed]

58. Swartz R, Longwell P. Treatment of vertigo. Am Fam Physician. 2005 Mar 15;71(6):1115-22. [PubMed]

59. White J, Savvides P, Cherian N, Oas J. Canalith repositioning for benign paroxysmal positional vertigo. Otol Neurotol. 2005 Jul;26(4):704-10. [PubMed]

60. Shaia WT, Zappia JJ, Bojrab DI, LaRouere ML, Sargent EW, Diaz RC. Success of posterior semicircular canal occlusion and application of the dizziness handicap inventory. Otolaryngol Head Neck Surg. 2006 Mar;134(3):424-30. [PubMed].

61. Rhim GI. Serum vitamin D and recurrent benign paroxysmal positional vertigo. Laryngoscope Investig Otolaryngol. 2016 Oct 20;1(6):150-153. doi: 10.1002/lio2.35. PMID: 28894811; PMCID: PMC5510269.

62. Kocabaş E, Kutluhan A, Müjdeci B. The evaluation of tinnitus and auditory brainstem response in benign paroxysmal positional vertigo accompanied by tinnitus. Eur Arch Otorhinolaryngol. 2021 Sep;278(9):3275-3280. doi: 10.1007/s00405-020-06413-w. Epub 2020 Oct 10. Erratum in: Eur Arch Otorhinolaryngol. 2020 Nov 20;: PMID: 33037892.

63. Dimple Sahni, Amandeep Kaur, Sanjeev Bhagat, Parvinder Singh, Rajwant Kaur, Deeksha Bharadwaj Department of Otorhinolaryngology, Government Medical College, Patiala, Punjab, India.

64. 64.Sfakianaki I, Binos P, Karkos P, Dimas GG, Psillas G. Risk Factors for Recurrence of Benign Paroxysmal Positional Vertigo. A Clinical Review. J Clin Med. 2021 Sep 24;10(19):4372. doi: 10.3390/jcm10194372. PMID: 34640391; PMCID: PMC8509726.

65. Yardley L, Owen N, Nazareth I, Luxon L. Prevalence and presentation of dizziness in a general practice community sample of working age people. Br J Gen Pract. 1998 Apr;48(429):1131-5. PMID: 9667086; PMCID: PMC1410052.

66. Neuhauser HK. The epidemiology of dizziness and vertigo. Handb Clin Neurol. 2016;137:67-82. doi: 10.1016/B978-0-444-63437-5.00005-4. PMID: 27638063.

67. Talaat HS, Kabel AM, Khaliel LH, Abuhadied G, El-Naga HA, Talaat AS. Reduction of recurrence rate of benign paroxysmal positional vertigo by treatment of severe vitamin D deficiency. Auris Nasus Larynx. 2016 Jun;43(3):237-41. doi: 10.1016/j.anl.2015.08.009. Epub 2015 Sep 16. PMID: 26386496.

68. Büki B, Ecker M, Jünger H, Lundberg YW. Vitamin D deficiency and benign paroxysmal positioning vertigo. Med Hypotheses. 2013 Feb;80(2):201-4. doi: 10.1016/j.mehy.2012.11.029. Epub 2012 Dec 14. PMID: 23245911; PMCID: PMC4196321.

ANNEXURES

ANNEXURE I – CONSENT FORM

ROLE OF VITAMIN D SUPPLEMENTATION IN ADDITION TO BETAHISTINE AS COMPARED TO BETAHISTINE ALONE IN PATIENTS OF BENIGN PAROXSYMAL POSITIONAL VERTIGO: A ONE YEAR RANDOMIZED CONTROLLED STUDY IN KLES Dr PRABHAKAR KORE HOSPITAL

PRINCIPAL INVESTIGATOR : _____

CO-INVESTIGATOR : _____

INTRODUCTION AND PURPOSE:

The present study is conducted among patients who are confirmed cases of BPPV in ENT & HNS department in KLE's Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi.

PROCEDURE:

If you agree to participate in this study, the relevant data will be collected as per the proforma and the final diagnosis will be confirmed.

After getting enrolled in the study, you will be evaluated for Serum Vitamin D level.

BENEFITS:

This study may help to benefit in improving the line of treatment of the patients coming with complaints of dizziness.

RISKS:

Methods applied and the drugs used in the study are safe.

COST OF PARTICIPATION:

The cost of the Investigation will be borne by the researcher.

PRIVACY AND CONFIDENTIALITY:

The results of the study may be published in journals for scientific purposes. However your identity will not be revealed. All information collected will be coded so that no one other than the investigator will know your identity.

WITHDRAWAL FROM THE STUDY:

You ca n withdraw from the study at any time if you wish to do so.

AUTHORIZATION TO PUBLISH THE RESULTS:

The researcher may use the information gathered from this study for presentation in scientific meetings. However your identity will not be revealed.

QUERIES AND CONTACT:

If you have any query about rights as a research participant you can contact Dr. Harsha Hegde,Chairperson, J.N.Medical College, IEC & Scientist D, ICMR, National Institute of Traditional Medicine,Belagavi on Mobile no: 9480422500 on human subjects research.

CONSENT SUMMARY:

I have been explained all the contents of this consent form in my local language and having understood and clarified all my queries about the study to the best of my knowledge, I hereby give my voluntary consent for participation in the study. I do sign the informed consent form in front of an eyewitness whom I recognize.

Name and Signature/ left thumb impression of the participant

Name and Signature of the interviewer:

Name and Signature/ left thumb impression of the eyewitness (Relative):

Signature of the guide:

Date:

Date:

ANNEXURE II – PROFORMA

ROLE OF VITAMIN D SUPPLEMENTATION IN ADDITION TO BETAHISTINE AS COMPARED TO BETAHISTINE ALONE IN PATIENTS OF BENIGN PAROXSYMAL POSITIONAL VERTIGO: A ONE YEAR RANDOMIZED CONTROLLED STUDY IN KLES Dr PRABHAKAR KORE HOSPITAL

Date:

O.P. No: IP No:

Name: Age:

Sex: Occupation:

Address: Phone No:

D.O.A D.O.D:

CLINICAL PROFILE:

Chief Complaint:

History of Present Illness

Past History:

Personal History:

Family History

Physical Examination:

I) General Physical Examination -

Vital signs: Pulse: Blood Pressure : Respiratory rate :

Pallor Icterus clubbing cyanosis Lymphadenopathy

Oedema

II) ENT Examination

1. EAR EXAMINATION-	**Right ear**	**Left ear**
Pinna		
Preauricular area		
Post auricular area		
External auditory canal		
Tympanic membrane		

Tuning Fork Test:	**Right ear**	**Left ear**
Rinne's test:		
256hz		
512hz		
1024hz		
Weber's test:		
Absolute Bone Conduction test:		
Dix Hallpike Test		

2.THROAT EXAMINATION -

2.1ORAL CAVITY and OROPHARYNX:

2.2 INDIRECT LARYNGOSCOPY-

3.NECK EXAMINATION

4.NOSE EXAMINATION

1.External appearance

Root Bridge Dorsum Alae Tip columell

Cold spatula test

Cottle's test

Anterior Rhinoscopy

Posterior Rhinoscopy

Paranasal Sinus Examination

SERUM VITAMIN D LEVEL:

Before **After**

DIAGNOSIS

Dizziness History Questionnaire[10]

Name:_____

DOB:_____ **Date:**_____

- **Duration of symptoms:**
- **Currently, my dizziness…**

1. is constant.
2. is always there, but changes in intensity.
3. Comes and goes

.

- **If comes and goes:**

  1. How long does it typically last? _____seconds / minutes / hours (Circle ONE)
  2. How often does it typically occur? _____times per: hour / day / week / month / year

- **My dizziness mostly consists of…** (Tick **ALL** that apply)

1. Spells of spinning with nausea.
2. Off-balance sensation without dizziness.
3. A light-headed or near faint sensation.
4. Other. Please explain:_____

- **Between episodes I feel…** (Tick **ONE**)

  1. Dizzy or off balance all the time
  2. Normal
  3. Other. Please explain:_____

- **My episodes occur…** (Tick **ALL** that apply)

1.spontaneously. Nothing I do seems to bring them on or turn them off._____
2.only when standing or walking.
3.in relation to any head motion.
4.in relation to only certain head positions. Please describe:

- **When I roll over in bed…** (Tick **ONE**)

1.Nothing unusual happens.
2.The room seems to spin sometimes.
3.The room spins every time.

- **Is there anything that you can do to make the dizziness go away?** (sit, lay down, close eyes…)

  Please explain:_____

- **Circle all that apply:**

  I have hearing difficulty……..Right…..Left…..Both I have ringing or other sounds…….Right…..Left…..Both I have fullness……..Right…..Left…..Both I have had ear surgery……..Right…..Left…..Both

- Circle **YES** or **NO**

Did you have cold, flu, or virus type symptoms shortly before the onset of your dizziness?	YES	NO
Did you have cough, lift, sneeze, fly in a plane, swim under water, or have a head trauma shortly before the onset of your dizziness?	YES	NO
If you had head trauma prior to your dizziness, did you lose consciousness completely?	YES	NO
Were you exposed to any irritating fumes, paints, etc. at the onset of your dizziness?	YES	NO
Do you get dizzy when you have not eaten for a long time?	YES	NO
Is your dizziness connected with your menstrual period?	YES	NO
Did you get new glasses recently?	YES	NO
I consider myself to be an anxious or tense type of person…	YES	NO
I am under a great deal of stress…	YES	NO

- **In the past year I have had… (Tick ALL** that apply)

1. loss of consciousness
2. Seizures or convulsions
3. Slurring of speech
4. Difficulty swallowing
5. Weakness in one hand, arm, or leg
6. Double vision
7. Spots before eyes
8. Occasional loss of vision
9. Severe pounding headache or migraine
10. Palpitation of the heartbeat
11. Tingling around mouth
12. Tendency to fall
13. Loss of balance when walking

I have or have had… (Check ALL that apply)

1. Diabetes
2. high Blood Pressure
3. Arthritis
4. Irregular Heartbeat
5. Stroke
6. Migraine Headaches
7. A neck and/or back injury
8. Allergies

Have you ever been previously evaluated for dizziness?_____

ANNEXURE III – MASTERCHART

Sr. No.	AGE (Years)	GENDER	DIAGNOSIS	VITAMIN D LEVELS BEFORE (ng/ml)	VITAMIN D LEVELS AFTER (ng/ml)	TREATMENT GIVEN	NUMBER OF EPISODES BEFORE TREATMENT (PER DAY)	NUMBER OF EPISODES AFTER TREATMENT(PER DAY)			ASSOCIATED SYMPTOMS	COMORBIDITIES	PREVIOUSLY TREATED FOR DIZZINESS
1	21	M	R BPPV	14	18	BETAHISTINE ,VIT D	7-8 PER DAY	5-6 PER DAY	2-3 PER DAY	0 PER DAY	TINNITUS	MIGRAINE	YES
2	29	F	R BPPV	15	17	BETAHISTINE ,VIT D	8-10 PER DAY	6-7 PER DAY	3-4 PER DAY	0 PER DAY	TINNITUS	MIGRAINE	YES
3	46	M	L BPPV	20	20	BETAHISTINE	5-6 PER DAY	5-6 PER DAY	2-3 PER DAY	1-2 PER DAY	DECREASED HEARING	DM	NO
4	22	F	L BPPV	12	15	BETAHISTINE ,VIT D	6-7 PER DAY	5-6 PER DAY	2-4 PER DAY	0 PER DAY	TINNITUS	MIGRAINE	NO
5	58	F	R BPPV	15	17	BETAHISTINE ,VIT D	9-10 PER DAY	6-7 PER DAY	3-4 PER DAY	1-2 PER DAY	DECREASED HEARING	HTN	NO
6	45	F	L BPPV	18	18	BETAHISTINE	5-6 PER DAY	4-5 PER DAY	2-3 PER DAY	0 PER DAY	DECREASED HEARING	DM	NO
7	41	M	L BPPV	16	16	BETAHISTINE	4-5 PER DAY	3-4 PER DAY	2-3 PER DAY	1-2 PER DAY	DECREASED HEARING	MIGRAINE	YES
8	56	F	R BPPV	20	22	BETAHISTINE ,VIT D	7-8 PER DAY	5-6 PER DAY	3-4 PER DAY	0 PER DAY	DECREASED HEARING	HTN	NO
9	34	F	R BPPV	19	21	BETAHISTINE ,VIT D	8-10 PER DAY	5-6 PER DAY	2-3 PER DAY	0 PER DAY	TINNITUS	MIGRAINE	YES
10	22	F	R BPPV	15	17	BETAHISTINE ,VIT D	5-6 PER DAY	3-4 PER DAY	0-2 PER DAY	0 PER DAY	TINNITUS	NIL	NO
11	26	F	L BPPV	20	20	BETAHISTINE	7-8 PER DAY	4-5 PER DAY	2-3 PER DAY	0-1 PER DAY	DECREASED HEARING	MIGRAINE	NO
12	53	M	R BPPV	17	17	BETAHISTINE	6-7 PER DAY	3-4 PER DAY	2-3 PER DAY	1-2 PER DAY	NIL	NIL	NO
13	58	F	L BPPV	14	16	BETAHISTINE ,VIT D	7-8 PER DAY	5-6 PER DAY	3-4 PER DAY	0 PER DAY	DECREASED HEARING	HTN	YES
14	36	F	L BPPV	13	13	BETAHISTINE	5-6 PER DAY	3-4 PER DAY	2-3 PER DAY	0-1 PER DAY	TINNITUS	DM	YES
15	60	F	R BPPV	17	21	BETAHISTINE ,VIT D	7-8 PER DAY	3-4 PER DAY	1-2 PER DAY	0 PER DAY	DECREASED HEARING	MIGRAINE	NO
16	59	F	L BPPV	24	34	BETAHISTINE ,VIT D	8-9 PER DAY	5-6 PER DAY	3-4 PER DAY	0-1 PER DAY	TINNITUS	HTN	NO
17	40	F	R BPPV	3	10	BETAHISTINE ,VIT D	6-7 PER DAY	2-3 PER DAY	1-2 PER DAY	0 PER DAY	TINNITUS	MIGRAINE	NO
18	55	F	R BPPV	17	17	BETAHISTINE	6-7 PER DAY	4-5 PER DAY	2-3 PER DAY	1-2 PER DAY	DECREASED HEARING	DM	YES
19	32	F	L BPPV	19	19	BETAHISTINE	3-4 PER DAY	3-4 PER DAY	1-2 PER DAY	0-1 PER DAY	DECREASED HEARING	MIGRAINE	NO
20	60	F	R BPPV	12	15	BETAHISTINE ,VIT D	8-10 PER DAY	5-6 PER DAY	3-4 PER DAY	0-1 PER DAY	NIL	DM	NO
21	55	F	L BPPV	19	22	BETAHISTINE ,VIT D	7-8 PER DAY	3-4 PER DAY	2-3 PER DAY	0 PER DAY	TINNITUS	HTN	YES
22	44	F	L BPPV	17	17	BETAHISTINE	3-4 PER DAY	1-2 PER DAY	1-2 PER DAY	0 PER DAY	TINNITUS	MIGRAINE	NO
23	60	F	R BPPV	15	15	BETAHISTINE	5-6 PER DAY	3-4 PER DAY	2-3 PER DAY	1-2 PER DAY	DECREASED HEARING	HTN	YES
24	40	F	R BPPV	18	18	BETAHISTINE	5-6 PER DAY	3-4 PER DAY	2-3 PER DAY	1-2 PER DAY	TINNITUS	MIGRAINE	NO
25	28	F	L BPPV	22	22	BETAHISTINE	4-5 PER DAY	2-3 PER DAY	1-2 PER DAY	0-1 PER DAY	TINNITUS	MIGRAINE	YES

26	40	M	L BPPV	24	24	BETAHISTINE	5-6 PER DAY	3-4 PER DAY	2-3 PER DAY	1-2 PER DAY	NIL	MIGRAINE	NO
27	60	M	L BPPV	17	17	BETAHISTINE	3-4 PER DAY	1-2 PER DAY	1-2 PER DAY	0-1 PER DAY	DECREASED HEARING	HTN	NO
28	57	M	L BPPV	18	18	BETAHISTINE	4-5 PER DAY	2-3 PER DAY	1-2 PER DAY	0-1 PER DAY	TINNITUS	DM	NO
29	34	M	R BPPV	10	12	BETAHISTINE ,VIT D	7-8 PER DAY	5-6 PER DAY	3-4 PER DAY	1-2 PER DAY	NIL	MIGRAINE	YES
30	60	M	L BPPV	13	13	BETAHISTINE	5-6 PER DAY	3-4 PER DAY	2-3 PER DAY	1-2 PER DAY	DECREASED HEARING	HTN	YES
31	59	M	R BPPV	26	29	BETAHISTINE ,VIT D	8-10 PER DAY	6-7 PER DAY	2-3 PER DAY	0 PER DAY	TINNITUS	DM	NO
32	30	M	L BPPV	16	16	BETAHISTINE	5-6 PER DAY	3-4 PER DAY	2-3 PER DAY	1-2 PER DAY	TINNITUS	MIGRAINE	NO
33	22	M	L BPPV	11	11	BETAHISTINE	5-6 PER DAY	3-4 PER DAY	2-3 PER DAY	0-1 PER DAY	NIL	NIL	YES
34	59	M	R BPPV	19	21	BETAHISTINE ,VIT D	6-7 PER DAY	4-5 PER DAY	2-3 PER DAY	0 PER DAY	DECREASED HEARING	NIL	NO
35	39	M	R BPPV	23	23	BETAHISTINE	4-5 PER DAY	2-3 PER DAY	1-2 PER DAY	0-1 PER DAY	TINNITUS	MIGRAINE	NO
36	50	M	L BPPV	24	24	BETAHISTINE	4-5 PER DAY	3-4 PER DAY	2-3 PER DAY	1-2 PER DAY	TINNITUS	HTN	YES
37	32	F	R BPPV	24	24	BETAHISTINE	5-6 PER DAY	3-4 PER DAY	2-3 PER DAY	1-2 PER DAY	TINNITUS	MIGRAINE	YES
38	29	M	R BPPV	16	19	BETAHISTINE ,VIT D	7-8 PER DAY	5-6 PER DAY	2-3 PER DAY	0 PER DAY	TINNITUS	MIGRAINE	NO
39	39	M	R BPPV	18	20	BETAHISTINE ,VIT D	8-9 PER DAY	5-6 PER DAY	3-4 PER DAY	0-1 PER DAY	TINNITUS	NIL	NO
40	30	M	L BPPV	15	19	BETAHISTINE ,VIT D	9-10 PER DAY	5-6 PER DAY	2-3 PER DAY	0 PER DAY	TINNITUS	NIL	NO
41	28	F	R BPPV	14	18	BETAHISTINE ,VIT D	7-8 PER DAY	5-6 PER DAY	2-3 PER DAY	0-1 PER DAY	NIL	MIGRAINE	NO
42	60	M	L BPPV	17	17	BETAHISTINE	5-6 PER DAY	3-4 PER DAY	2-3 PER DAY	1-2 PER DAY	DECREASED HEARING	HTN	YES
43	24	F	L BPPV	18	21	BETAHISTINE ,VIT D	8-10 PER DAY	6-7 PER DAY	3-4 PER DAY	0-1 PER DAY	NIL	MIGRAINE	NO
44	32	M	R BPPV	22	22	BETAHISTINE	5-6 PER DAY	3-4 PER DAY	2-3 PER DAY	1-2 PER DAY	DECREASED HEARING	NIL	NO
45	59	M	L BPPV	18	19	BETAHISTINE ,VIT D	7-8 PER DAY	5-6 PER DAY	3-4 PER DAY	1-2 PER DAY	TINNITUS	DM	YES
46	24	F	L BPPV	17	18	BETAHISTINE ,VIT D	6-7 PER DAY	3-4 PER DAY	2-3 PER DAY	0 PER DAY	NIL	MIGRAINE	NO
47	36	F	R BPPV	13	13	BETAHISTINE	4-5 PER DAY	3-4 PER DAY	2-3 PER DAY	1-2 PER DAY	TINNITUS	MIGRAINE	NO
48	23	F	R BPPV	12	12	BETAHISTINE	4-5 PER DAY	3-4 PER DAY	2-3 PER DAY	1-2 PER DAY	NIL	MIGRAINE	YES
49	46	M	L BPPV	24	24	BETAHISTINE	5-6 PER DAY	3-4 PER DAY	2-3 PER DAY	1-2 PER DAY	TINNITUS	HTN	YES
50	44	F	R BPPV	23	26	BETAHISTINE ,VIT D	7-8 PER DAY	5-6 PER DAY	2-3 PER DAY	0 PER DAY	DECREASED HEARING	NIL	NO