To establish normative data in an Indian population for static and dynamic Subjective Visual Vertical and Horizontal (SVV & SVH) examination and to determine the role of SVV & SVH in evaluation of patients with migrainous vertigo

A DESSERTATION SUBMITTED IN PARTIAL FULFILLMENT OF M.S.BRANCH-IV (OTORHINOLARYNGOLOGY) EXAMINATION OF THE TAMIL NADU DR.M.G.R.MEDICAL UNIVERSITY TO BE HELD IN APRIL 2015

DEPARTMENT OF OTORHINOLARYNGOLOGY

CHRISTIAN MEDICAL COLLEGE

VELLORE

CERTIFICATE

This is to certify that the dissertation entitled,' **To establish normative data in an Indian population for static and dynamic Subjective Visual Vertical and Horizontal (SVV & SVH) examination and to determine the role of SVV & SVH in evaluation of patients with migrainous vertigo**' is a bonafide original work of Dr Gaurav Ashish carried out under my guidance, in partial fulfilment of the rules and regulations for the MS Branch IV,Oto-rhino-laryngology examination of The Tamil Nadu Dr M.G.R Medical University to be held in April,2015.

Principal
Christian Medical College
Vellore 632004

Dr John Mathew,
Professor and HOD,
Department of ENT,
Christian Medical College,
Vellore.

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Dr Achamma Balraj,

Professor and Head (unit 4),

Department of ENT,

Christian Medical College,

Vellore.

CERTIFICATE

I declare that this dissertation entitled "**To establish normative data in an Indian population for static and dynamic Subjective Visual Vertical and Horizontal (SVV & SVH) examination and to determine the role of SVV & SVH in evaluation of patients with migrainous vertigo** 'submitted towards fulfilment of the requirements of the Tamil Nadu Dr. M.G.R. Medical University for the MS Branch IV, Otorhinolaryngology examination to be conducted in April 2015, is the bonafide work of Dr.Gaurav Ashish, postgraduate student in the Department of Otorhinolaryngology, Christian Medical College, Vellore.

Dr.Gaurav Ashish

Postgraduate Student (M S Otorhinolaryngology)

Register Number: 221314351

Department of Otorhinolaryngology

Christian Medical College

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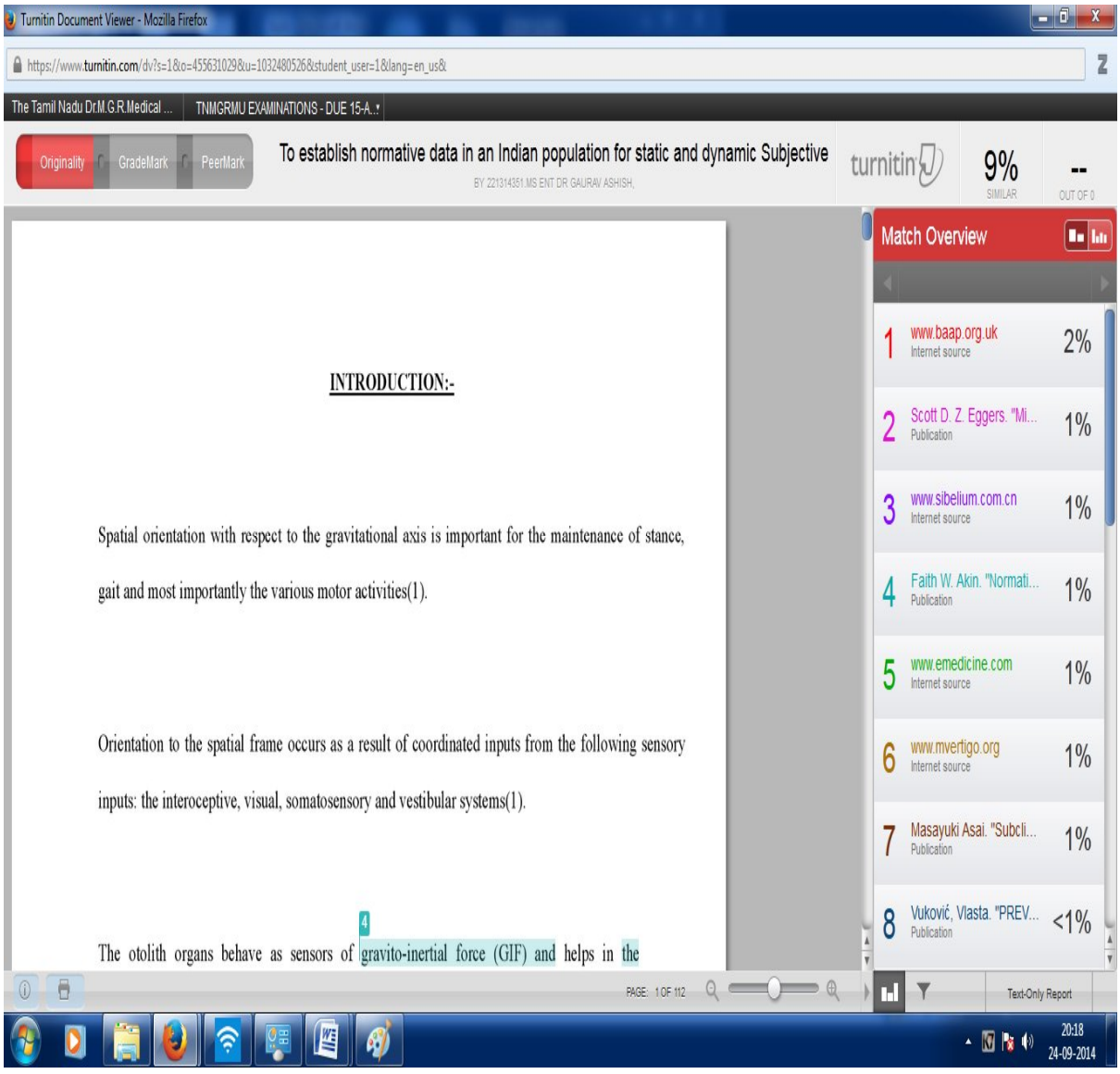
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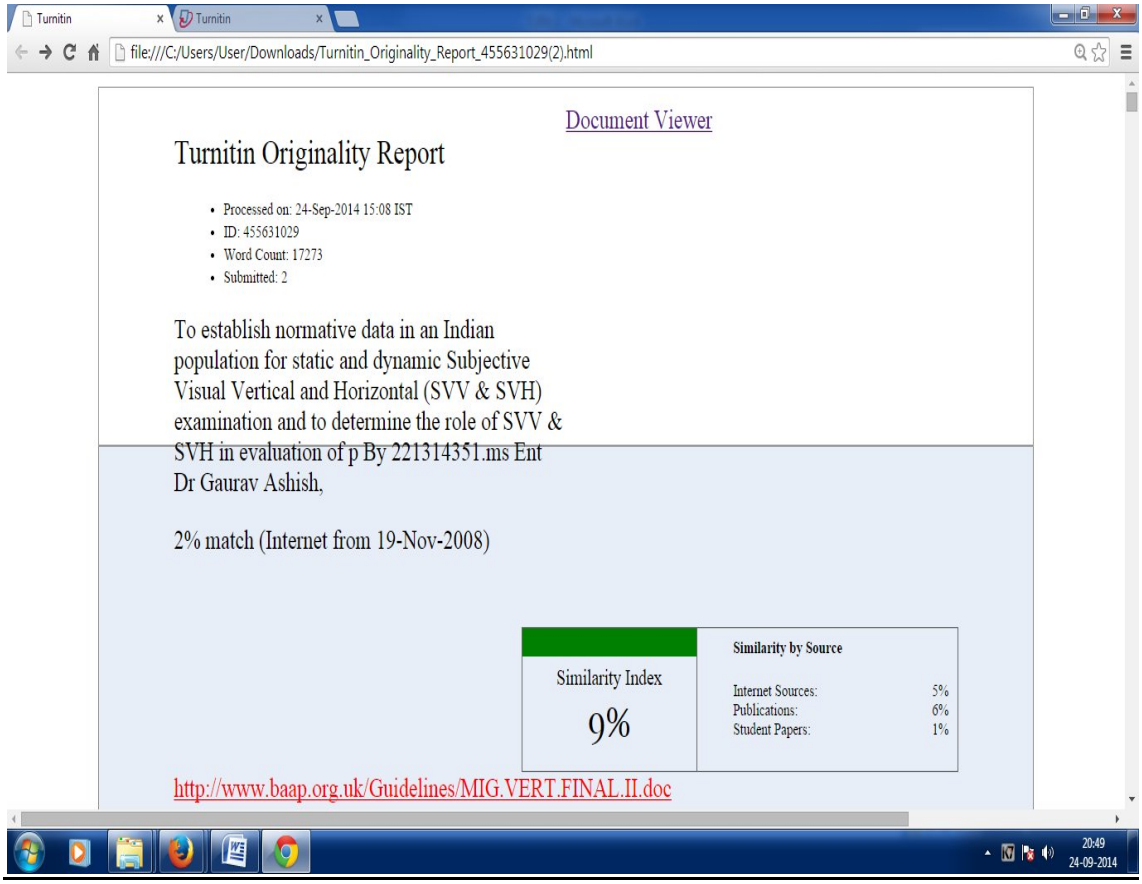
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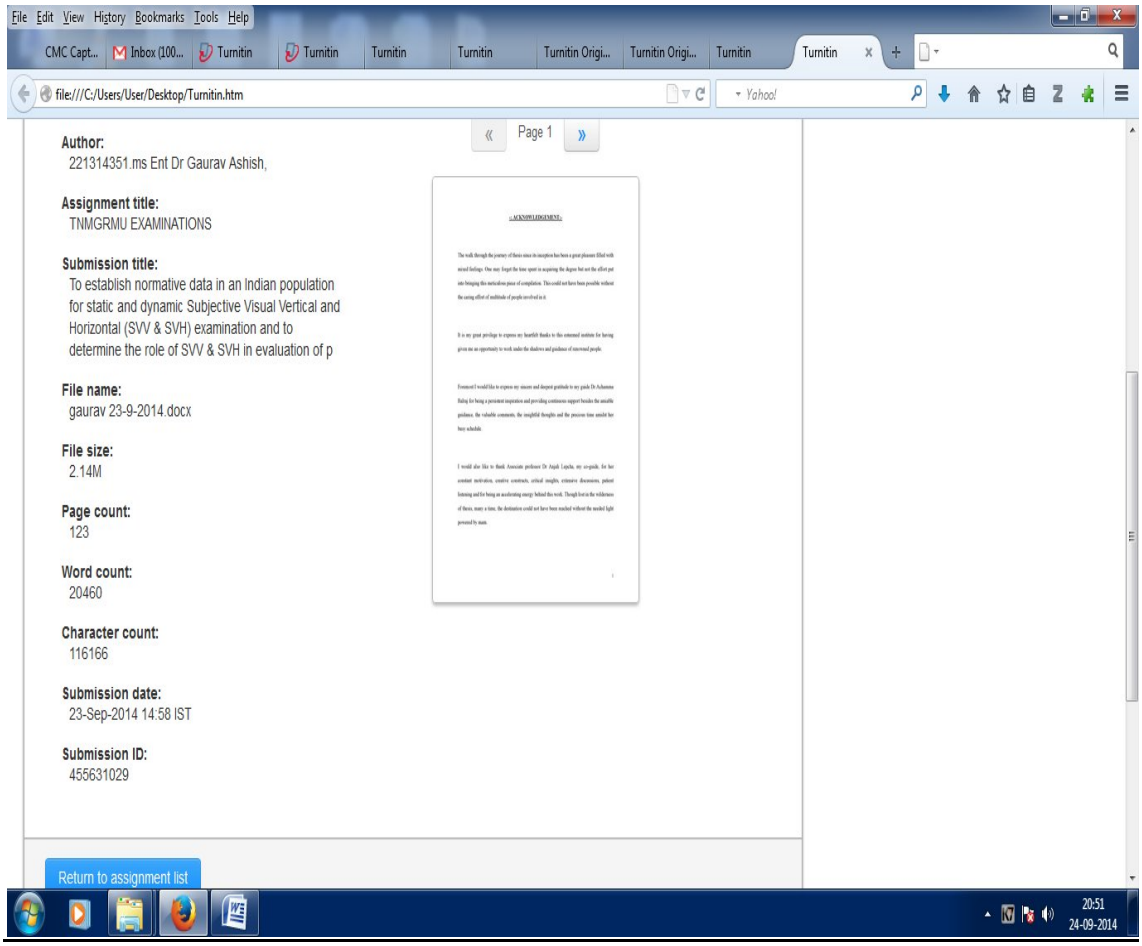
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CONTENTS

INTRODUCTION:-

Spatial orientation with respect to the gravitational axis is important for the maintenance of stance, gait and most importantly the various motor activities(1).

Orientation to the spatial frame occurs as a result of coordinated inputs from the following sensory inputs: the interoceptive, visual, somatosensory and vestibular systems(1).

The otolith organs behave as sensors of gravito-inertial force (GIF) and helps in the perception of spatial orientation. They comprise saccule and utricle and interpret inputs pertaining to linear acceleration and position with respect to the head, with reference to gravity (head tilt), and further adds to postural stability. The saccules are oriented vertically and perceive linear acceleration in the vertical axis. On the contrary the utricles are oriented horizontally and perceive linear acceleration in the horizontal axis (2).

The interpretation of gravitational vertical, also known as the true vertical, can be analysed by asking the subject either to orient his/her body to the vertical (postural vertical or to adjust a computer simulated light bar vertically also called subjective visual vertical (SVV) . Similarly, the interpretation of gravitational horizontal (a plane at 90 degrees to true gravitational vertical), also known as true horizontal, can be assessed by asking the subject to orient a computer simulated light bar to the horizontal also called subjective visual horizontal (SVH) is the angle measured in degrees between perceptual vertical and true vertical.

Studies have suggested that SVV and SVH in normal volunteers in an vertical static position are within +/- 2.5 from true vertical or horizontal(3). The tilt of SVV and SVH is a very accurate indicator of vestibular tonus imbalance when analysed in roll plane (4).

Static SVV and SVH are sensitive to acute vestibular loss. Static SVV and SVH get compensated very fast as compared to dynamic Subjective visual vertical and Subjective visual horizontal values. Thus dynamic Subjective visual vertical and Subjective visual horizontal values can be analysed much later and can hint at any insult that must have occurred earlier to any area involving the utricular pathway(5).

It is important to note that it is difficult to distinguish subjects suffering with prolonged unilateral vestibular loss from normal subjects based on their SVV and SVH values. This can be only done if complicated non-physiological stimulation techniques are used to stimulate the utricle and saccule.

Patients with migraine often report of vertigo. However, the pathological basis of the above has not been established. Migraine is believed to cause benign recurrent vertigo apart from being associated with numerous vestibular and cochlear syndromes.

Some reports showed that that subjects with migraine have abnormalities in the vestibular spinal reflex system, due to involvement of otolith macula and these remain subclinical until demonstrated by sophisticated tests(6).

Propioceptive cues for postural control may be influenced by false or inappropriate inputs, leading to unsteadiness. All conventional otoneurological tests in these patients are usually found to be normal. An area which is often not covered in these tests is the utricular pathway and a test which detects pathology in this pathway may give valuable information.

Justification for this study:

Available methods for assessing utricular pathways abnormalities are the chair rotation test, the bucket test, the gondola equipment which are either impractical or space occupying in a busy clinical practice. More recently computer software based methods are becoming available but have not been yet widely utilised. This study was designed to determine the normative values for SVV and SVH for a subset Indian population using the software using the (MUS_VS-V1.3.2.Rev B) Synapsis Company-France)

The target of this study was to analyse the role of SVV/SVH in subjects diagnosed as migrainous vertigo to evaluate their SVV and SVH, which may be associated to the subjective unsteadiness.

This project also encompasses analysis and detail assessment of various otoneurological tests done for patients of migranous vertigo and their respective outcomes. The clinical profile of these patients was also studied and it was tried to observe whether any correlation exists between these factors and the SVV/SVH values.

AIMS AND OBJECTIVE:-

AIMS:-

To determine normative data for static and dynamic Subjective Visual Vertical and Horizontal (SVV & SVH) in a group of normal Indian volunteers and to compare their reading against that of a group of patients with migrainous vertigo.

OBJECTIVES:-

1. To establish normative data for age specific groups in Indian population for static and dynamic Subjective Visual Vertical and Horizontal (SVV & SVH) examination and to determine whether SVV and SVH could identify specific abnormalities in patients suffering with migranous vertigo since the test identifies utricular pathway involvement..

2. To compare SVV and SVH values in migranous vertigo when compared to normative values calculated among the normal volunteers.

3. To assess the otoneurological profile of patients diagnosed as migranous vertigo in terms of the clinical presentation, imaging characteristics and results of audiovestibular tests conducted.

-: PRESENT KNOWLEDGE AND REVIEW OF LITERATURE:-

BASIC ANATOMY:-

Vestibular system integrates the balance in human beings. It comprises of membranous and bony labyrinth which is situated in petrous bone. It contains the following specific sense organs namely the three semicircular canals {superior, lateral, posterior}and the otolith organs namely utricle and saccule(2).

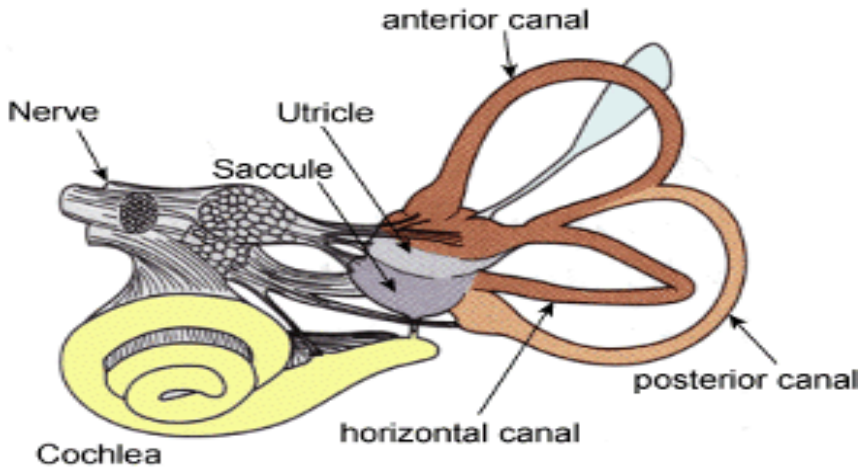

Figure 1:-showing anatomy of vestibular apparatus(7).

The inner ear lodges the sensory organs that detect angular movements by the semicircular canals (SCC), and the linear movements by the otoliths-namely utricle and saccule. The saccule is placed in a vertical axis and helps to detect linear vertical movements Utricle is aligned in the horizontal plane and detects linear horizontal movements(2).

Therefore otoliths serve as gravito-inertial force (GIF) sensors and help us to orient in the 3 dimensional spatial orientations. Spatial interpretation with respect to gravitational axis is important to maintain gait, stance and also most of the motor activity. (8).

EMBRYOLOGY OF INNER EAR

During the development of embryo in the third week, the otic placode arises out of neuroectoderm and ectoderm. In the 4th week otocyst or otic vesicle and the formation of endolymphatic duct happens(9).

Utricular chamber gets transformed into utricle and semicircular canals whereas the saccular chamber gets transformed to saccule and the cochlea. Later the separation of saccule and cochlea occurs along with the formation of the ductus reunions.

In the 3 rd week another major development occurs where the sensory epithelium develops from the ectoderm leading to the formation of 3 cristae and 2 maculae respectively. Embryologically the Vestibulocochlear ganglion starts as a single entity and then later separates as the inferior and superior branches respectively (9) .

The superior branch innervates the superior and lateral semicircular canals and the utricle whereas on the other hand the inferior division innervates saccules and the posterior semi circular canal via the singular nerve.

APPLIED ANATOMY:-

The three semicircular canals are oriented orthogonal with respect to one another. The lateral canal is angulated at 30 degrees to the horizontal while the superior and postererior canals are oriented at approximately 45 degrees off the sagittal plane. Utricle is aligned in the horizontal plane whereas saccule is oriented in vertical axis. It is a well known fact that there are five openings into area of utricle .The saccule is situated in spherical recess and the utricle in the elliptical recess(8).

Membranous labyrinth has perilymph all around it, which is rich in sodium whereas the endolymph lies inside the vestibular end organs as well as the cochlea.

The sensory end organ structures are the ampulla of the semicircular canals. These are the dilated ends of the semi circular canals. These contain sensory neuroepithelium, the, the cupula and the various cells that act as supporting entities.

The cupula is a gelly like material which is oriented at right angle. Although it lies across in its full extent yet it is not gravity responsive.

The Crista ampullaris consists of sensory hair cells along with supporting cells(8).

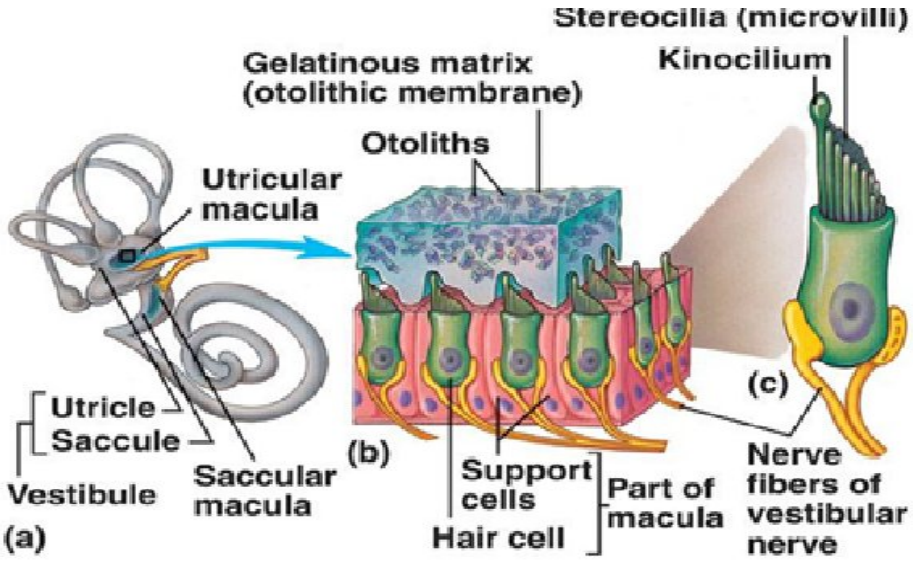

Figure 2:-showing microanatomy of vestibule(10).

The sensory cells can be type I or type II cells. Type I cells is usually flask shaped with a chalice shaped ending. Another peculiarity is that a single chalice could synapse with as many as two to four other Type I cells. On the other hand the type II cells are cylindrical in shape and have multiple efferent and afferent fibres(8).

Cell attains polarity as a result of kinocillium being situated at either of the end of the cells. The kinocillium have a peculiar 9+2 arrangement of microtubule doublets. Absence of inner dynein arms, and the central portion of microtubules are also the hallmark features .If it is so then it may mean that they are either immobile or partially mobile(9).

Every afferent neuron has a basic rate of firing. It is well established fact that deviation of stereocilia toward kinocillium leads to afferent neuron being fired at an increased rate while any deviation away causes retardation in the firing rate.

The kinocilia are situated closest to utricle in the lateral canals and are on canalicular side in the other canals. Thus any flow that is towards the ampulla -Ampullopetal flow-is excitatory in nature in lateral canals and inhibitory in nature for the superior and posterior canals.Simillarly any flow that is away from the ampulla -Ampullofugal flow has an opposite impact(2).

Semicircular canals are oriented in paired fashion where the horizontal canals are oriented complimentary to each other whereas the right posterior and left superior are aligned with respect to each other. Similarly are the left posterior and right superior semi circular canals oriented. These allow redundant reception of movement and this fact also explains compensation after unilateral vestibular loss has an opposite effect(8).

Otolithic organs namely the utricle and saccule contain cilia arising out of hair cells which lie in a gelly like layer. The otoliths, more specifically the otoconia are situated on the upper surface. The otoconia are made of calcium carbonate materials. The size range from 0.5-30um with a specific gravity of approximately 2.71-2.94.Striola is situated in the centre of otolithic membrane. (8).

Hair cells in the saccule are situated away from the striola whereas in utricle the hair cells are aligned towards the striola .Moreover it is known that striola being curved help to perceive and interpret linear motion in any directions.

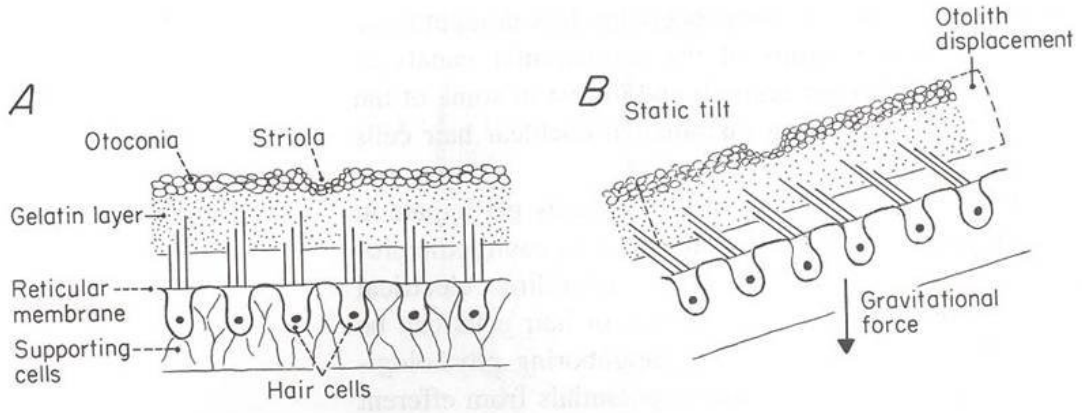

Figure3:-Showing tilting of striola and physiology involved(11).

BLOOD SUPPLY:-

There are variations in the blood supply of the inner ear. In approximately 45%the main blood supply is from AICA (anterior inferior cerebellar artery).On the other hand 24%of the blood supply is from superior cerebellar artery and in about 16% from basilar artery respectively. The blood supply mainly occurs from the two major subdivisions namely the anterior vestibular and common cochlear artery(12).

CENTRAL CONNECTIONS:-

The Scarpa's ganglion lies within internal acoustic meatus and it consists of bipolar ganglion cells belonging to the category of first order neurons. The superior and inferior branches fuse together to form a common entity which penetrates the brainstem. It is noteworthy to remember that none of the primary vestibular afferents travel midline to actually cross them (13).

The afferent fibers end in the vestibular nucleus which is situated in the floor of fourth ventricle. There are four vestibular nuclei namely

– Superior vestibular nuclei

– Lateral vestibular nuclei

– Medial vestibular nuclei

– Descending vestibular nuclei

They are complexely projected into the Cerebellum, Extra ocular nuclei, Spinal cord and the vestibular nuclei of the opposite side as well as other groups of central vestibular nuclei like Interstital Nucleus of Cajal and Brachium Conjuntivum(13). The medial longitudinal fasciculus (MLS) forms the main pathway by which these various centres are connected.

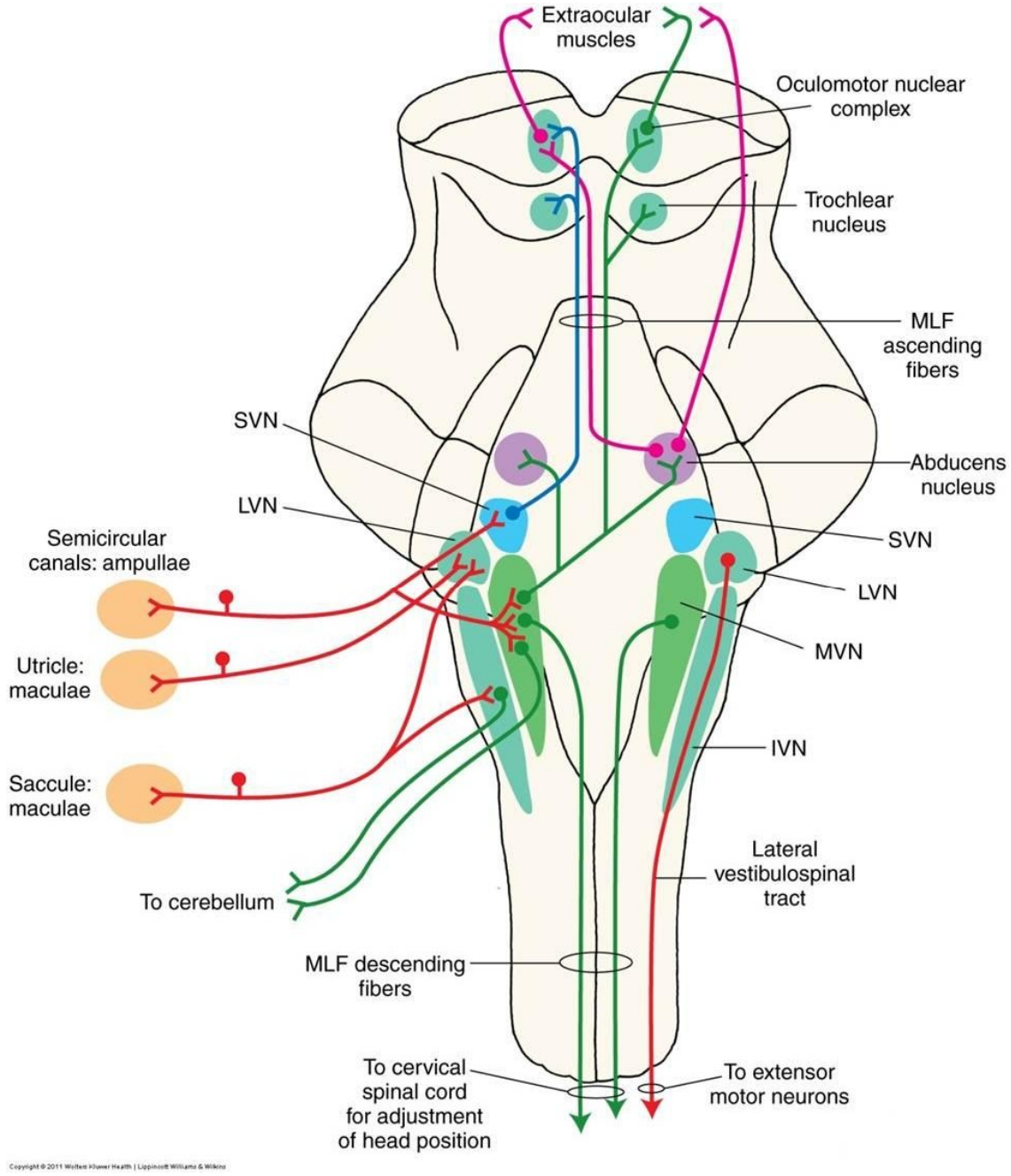

Figure 4:-Showing central connections of vestibular apparatus(14).

The central vestibular connections main role is in integrating vestibular sensation with other sensory information from proprioceptive, visual and autonomic organs so as to provide sense of normal orientation, and maintenance of balance at rest and during motion (13).

VESTIBULOCULAR REFLEX:

The vestibuloocular reflex is a short latency reflex (8 millisecond) required for stabilising images on the retina when head is in motion. By this reflex the eye moves to the same distance but in opposite direction of the head and at the same speed as the head so as to avoid a retinal slip of the image during head motion (6). In the absence of VOR the visual acuity declines by more than 50% at a point that is even 2° away from the centre of the fovea. (Jacobs and Carpenter). The fovea has the maximum concentration of the photoreceptor on the retina and thus the visual acuity is highest at this particular point(15) .

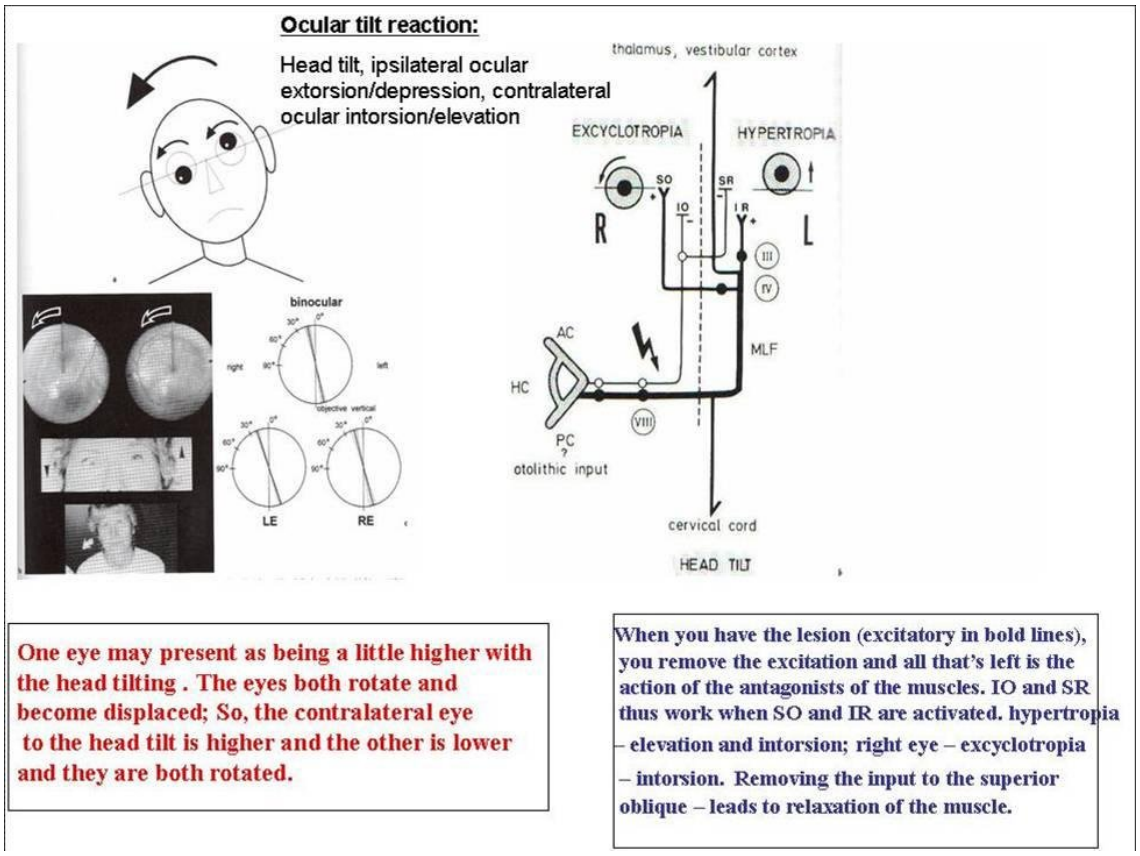

Figure 5:-Showing ocular tilt reaction in relation to VOR**(16)**.

Vestibuloocular reflex (VOR) involves a complex intercorrelation of 3-neuron involving the vestibular ganglion, vestibular nuclei, and oculomotor nuclei. The main components of VOR are described as follows:-

Rotational vestibuloocular reflex

While the head is in rotation, the motion of endolymph within the semicircular canals causes deflection inside cupula. The endolymph movement towards ampulla causes excitation of horizontal semicircular canals, whereas the endolymphatic movement away from ampulla is excitatory in nature for the posterior and superior semicircular canals.(17).

All the four nuclei of vestibule namely inferior or descending vestibular nucleus, medial vestibular nucleus, superior nucleus, and lateral vestibular nucleus receive both excitatory and inhibitory signals from the afferent nerves from the ampulla. The oculomotor nuclei contain representation from cranial nerves III, IV, and VI and the various areas from these vestibular nuclei have varied representation upon the oculomotor nuclei. Efferent output from these nuclei cause contraction and relaxation of the corresponding ocular muscles (18).

An up beating rotational eye movement occurs due to stimulation of the superior canal results. The stimulation of superior canal causes contraction of the superior rectus of the same

side and also the contraction of inferior oblique muscle of the same side. However there is relaxing effect on the inferior rectus of the same side and l superior oblique muscles of the opposite side.

A downward rotational eye movement is caused due to excitation of the posterior canal. This can be understood by the fact that stimulation in the posterior semi circular canal leads to a contracted state of the superior oblique of the same side and inferior rectus muscles of opposite side along with relaxation of the inferior oblique on the same side and superior rectus muscles on the opposite side.

Similarly a horizontal eye movement toward the opposite ear is caused due to the excitation of the lateral canal resulting in contraction of the ipsilateral medial rectus and contralateral lateral rectus muscles and relaxation of the contralateral medial rectus and ipsilateral lateral rectus muscles (18)(19).

The oculomotor connections and the various vestibular nucleus are complexely related to the nucleus prepositus hypoglossi, and the neuronal connections in the paramedian tracts and these centres are closely connected to flocculus of the cerebellum(19) . The vestibulocerebellum plays a pivotal role in the complex arc it compares the inputs from visual and vestibular receptors separately. This causes alternation in the vestibuloocular reflex following any assault to vestibular apparatus or any change in visual functions.

Reverse projections arising and leading to the cerebellum help in the fine movement of the eyes. The rotational vestibuloocular reflex (r-VOR) has a latency period of approximately fifteen milliseconds. Practically this is the time taken for the eyes to react in an equivocal but opposite magnitude to that of motion of the head. The latency of r-VOR is very rapid when as compared to the delay in performing visually mediated eye movements, which are approximately seventy five milliseconds.

Cerebral function may also influence the vestibuloocular reflex and has been shown to have the potential for suppression of the vestibuloocular reflex. Literature has supported the fact that any assault to the occular gyrus and more importantly the parietal vestibular cortex has demonstrated interference of visual suppression of the vestibuloocular reflex(18)(19).

Translational vestibuloocular reflex:-

The otoliths have major role in stimulating the translational vestibuloocular reflex (t-VOR) pathways. The utricle reacts to any stimulus leading to translational movement, whereas the saccule reacts to any vertical movement. The complex pathways involving translational vestibuloocular reflex have been studied very minimally when compared to that of the pathways for the rotational vestibuloocular reflex (20).

Projections from the ocular motor nuclei influence the translational vestibuloocular reflex pathways due to connections arising out of vestibular nucleus. If the macula within utricle is stimulated it causes contraction of the superior rectus, medial rectus and superior oblique rectus muscles and on the contrary causes relaxation of the contra lateral rectus, inferior oblique and inferior rectus(18)(20).

3D ORIENTATION OF PLANES OF ACTION OF VOR

For physiological and pathological purposes it is best to describe the neuronal network of VOR in relation to three main axes are:-

1. Horizontal action which occurs around a vertical axis- **"Yaw"plane**

2. Head flexion and extension which occurs about horizontal Y axis, known **as "Pitch"plane**

3. Tilting of head occurring about horizontal x axis, called as **"Roll" plane**(21).

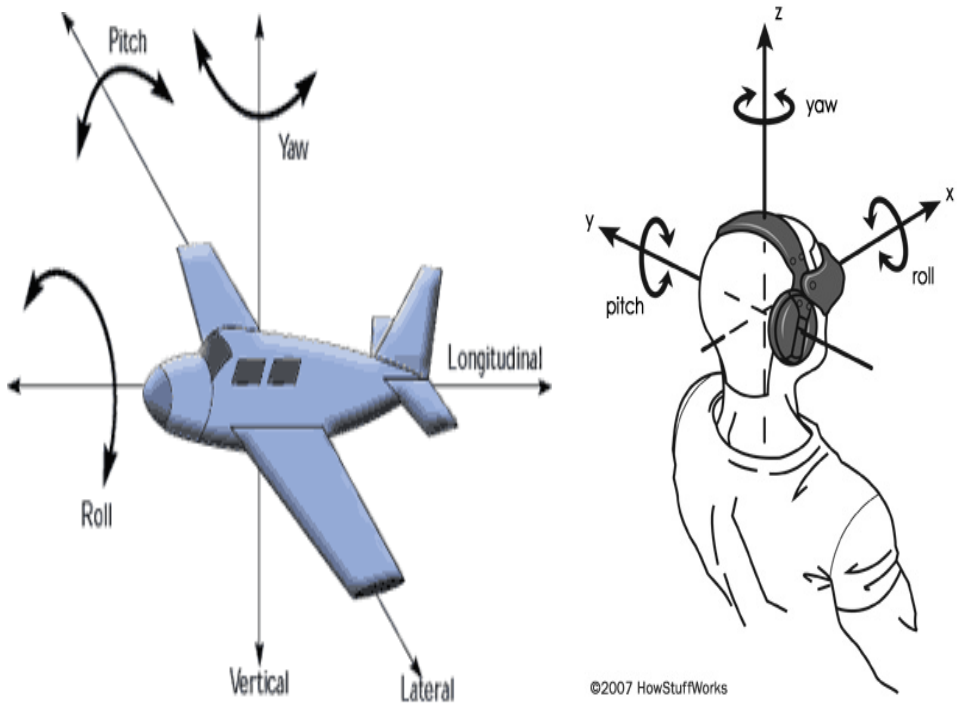

Figure 6:-3D orientations of VOR (22).

Disorder of Yaw plane is seen as horizontal nystagmus usually seen in pseudo vestibular neuritis as in cases of AICA and PICA infarcts. Similarly disorders of pitch plane manifests as down beating or up beating nystagmus associated with vertigo. and the disorders of Roll plane is manifested as occular tilt reaction or "lateropulsion"(21)(23).

These above planes of VOR have been applied on to the SVV and this has been implicated on SVV as follows(23).

Types of per-rotary SVV are namely

• **On-Axis**: where the rotation happens around a central vertical axis

• **Off-Vertical Axis (OVAR)**: where the rotation occurs around a tilted yaw axis

• **and lastly Off-Axis** (also termed as unilateral centrifugation): shifted outward placing one otolith directly over axis of rotation and the other significantly outward from this axis of rotation.(3)

Since the utricle is a major contributor within the vestibular organ to the spatial orientation, we can evaluate it separately if the influence of other systems can be eliminated and compel the subject to rely mainly on otolithic input. Therefore when we are measuring Off axis left ear then the left utricle is activated and Subjective visual vertical is tilted to the right and

similarly when evaluating Off-axis right ear, the right utricle is activated and the Subjective visual vertical tilted to the left respectively(24).

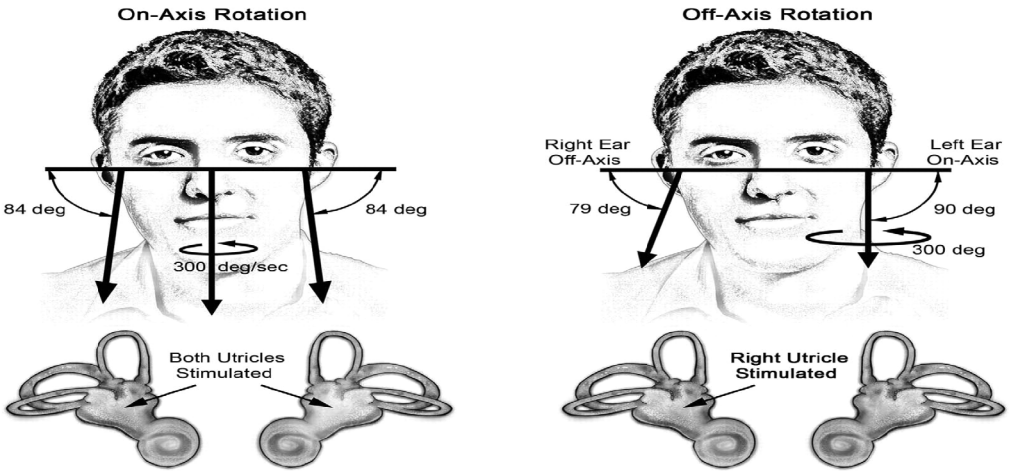

Figure: 7- showing on axis and off axis rotation(25).

VESTIBULOCULAR REFLEX DYSFUNCTION

The importance of VOR is only understood when the vestibuloocular reflex (VOR) malfunctions. The anatomical location of the pathology forms the basis of deciding the manifestation of acute vestibuloocular reflex dysfunction may have varied manifestations and may be a result of various disorders involving the complex labyrinthine pathways or the central vestibular pathways.

A peripheral vestibular lesion affecting only one side may demonstrate unequivocal responses to rotation. Whereas unilateral lesion which has compensated shows a specific

pattern seen at low frequency stimulus which demonstrates decrease in gain and increase in phase.

Peripheral vestibular disease involving both sides have specific pattern seen on sinusoidal testing showing a low gain and phase lag.. These subjects frequently report sense of vertical or horizontal motion of the surrounding, or continuous unsteadiness, which is aggravated in dark. These patients can be assessed by the rotational chair testing .This is because both labyrinths get stimulated at a single time and high end frequencies are tested which is different to caloric testing (26).

Central vestibular pathologies also influence the vestibuloocular reflex. Thurston et al showed in patients with cerebellar deficits have increased demonstrated gain where as Cerebellar atrophy, on the other hand, demonstrated a asynchronous nystagmus with variations in the amplitude with respect to every beat.

Post blast exposure has demonstrated influence on the high-velocity angular vestibuloocular reflex function as has been earlier studied(27).

It is also noteworthy that VOR is influenced by systemic illness for example migraine, depressive illness, and anxious spectrum disorders. In cases of vestibulopathy of migranous origin, elevated gain is demonstrated with visually enhanced VOR (VVOR).VVOR is a modification of VOR where the VOR is performed in a well lighted (i.e., visually enhanced) environment as compared to a dark booth(28).

Anxiety disorders subjects demonstrate a higher vestibular sensitivity demonstrating a high VOR gains and comparatively smaller time constants. Lastly candidates diagnosed with major depression demonstrate hypoactive vestibular nuclei, leading to decrease in the slow phase of the nystagmus(29).

OTHER VESTIBULAR REFLEX:-

There are 3 important vestibular reflexes. Apart from the VOR the vestibulospinal and vestibulocollic reflexes influence the postural and spatial orientation. Vestibulospinal reflex can be clinically assessed by using computerized dynamic posturography.

The vestibulocollic reflex has been the least understood of the various types of vestibular reflexes. The vestibulocollic reflex is an indirect reflection of the integrity of saccules. This can be studied in the following ways namely direct (Intraoperative) stimulation of the inferior vestibular nerve stimulation intraoperatively, acoustic stimuli, forehead stimulation by mechanical means, and galvanic voltage mediated stimulation (30).

The result is analysed in terms of a muscle evoked potential in the sternocleidomastoid muscle and the trapezius muscles of the same side. Therefore vestibular evoked myogenic potential (VEMP) test is being further studied in detail with respect to the reflex.

Vestibulospinal Reflex

This is responsible for analysing the head movement and also movement with respect to gravity. This influences the muscles acting against gravity by means of three major pathways .These are the reticulospinal tract, Medial vestibulospinal tract and the lateral vestibulospinal tract (31).

UTRICULAR AND VESTIBULAR FUNCTION TESTS:

Over the years the most of clinical testing of peripheral balance function has been limited to evaluating the functions of the lateral semicircular canals and their respective connections with the CNS. Many times we have patients who complain about tilting sensation, vertigo or leaning sensation in the presence of normal ENG/VNG exam, VEMP, Rotary Chair and / or imaging studies.

This has provoked the clinicians and researchers to explore ways to clinically evaluate the function of other components within the peripheral vestibular apparatus which may be the root cause for such complaints.

Vestibular evoked myogenic potentials (VEMPs) provide information regarding the function of the saccules but there were no tests which measure the integrity of utricle or rather utricular pathways. The answer to the above has been recently the subjective visual vertical and horizontal.

Tests of saccular function are Vestibular Evoked Myogenic Potential (VEMP), Cervical VEMP (cVEMP) and the Ocular VEMP (oVEMP)

Tests of Utricular function like Parallel swing and linear sled testing have found very limited role in clinical practice due to equipment cost, increased space requirement and poor sensitivity(32).

Other tests of utricular function like Static Subjective Visual Vertical (SVV), SVV coupled to Dynamic unilateral centrifugation and Off-Vertical Axis Rotation popularly known as(OVAR).But it is very difficult to evaluate the utricles due to the limited availability of very expensive and space occupying equipment(5,33).

Another more recent test that is becoming popular is subjective visual vertical and horizontal

BACKGROUND:-

The true vertical is actually the perception of gravitational vertical, can be analysed by asking the subject either to adjust his/her body position to the vertical (postural vertical or to align a light bar to the vertical which is computer stimulated (subjective visual vertical).

Similarly, the perception of gravitational horizontal (a plane at 90 degrees to true gravitational vertical), termed as true horizontal, can be evaluated by asking an subject to align a computer simulated light bar to the horizontal (Subjective Visual Horizontal SVV is the angulation between the adjusted light bar better known as perceptual vertical and true vertical.

SVV is a valid otoneurological test. The SVV tilts can be used to identify vestibular dysfunction especially in the otoliths. The test measures the deviations of the perceived vertical from the true vertical which is measured in degrees and this capacity depends on the integrity of visual and vestibular pathway in the brainstem (34).

Visual cortex has the cells very systematically organized, and is responsible for responding to a specific alignment arranged at 90 degrees to the surface of brain cortex in columns(35,36).Vestibular inputs integrates the static gravitational alignment and cephalic linear motions. This helps in better maintenance of posture and balance. The otolithic organs

contribute to the spatial orientation by providing inputs from subconscious postural reflexes(37).

A modification of the conventional SVV and SVH is known as the dynamic SVV and SVH test .This is similar to static SVV (adjusting the virtual line in the vertical position occurs irrespective to any reference suggestive of the real vertical), but the modification is that the background continuously rotates. This is based on the concept that if the peripheral field is made to rotate and subject is asked to follow the motion, it is perceived that the individual is himself rotating(38). This highlights the fact that the dynamic SVV reflects replacement of vestibular signals with visual signals (39).

Literature supports the fact that the SVV and SVH in healthy subjects in an upright static position do not demonstrate deviation greater than +/- 2.5 from true vertical or horizontal. The tilt of SVV and SVH is a very important indicator of vestibular tonus imbalance demonstrable in the roll plane (4).

Static SVV and SVH are sensitive to acute vestibular loss. Static SVV and SVH get compensated very fast as compared to dynamic Subjective visual vertical and Subjective visual horizontal values. Thus the dynamic SVV and SVH values can be analysed much later and can hint at any insult that must have occurred earlier to any area involving the utricular pathway(40) .

However, subjects with long standing vestibular loss cannot be differentiated by comparing their SVV or SVH from normal subjects till the sophisticated non-physiological techniques are used to stimulate the otolith organs separately.

Other available methods for assessing are the conventional chair rotation test, the bucket test, the gondola equipment, computer software based methods and various others.

Rotational chairs are most commonly available test which are usually used to cause stimulation of otoliths (dynamic SVV test) by simulating using a computer and rotating the background in clockwise and anticlockwise pattern to stimulate otoliths. The mean values obtained from other studies for SVV are: static(-0.372+/-1.21), dynamic clockwise 1.53+/-1.80 and dynamic counter clockwise -1.11+/-2.46}(41)(1)(34)(42).There have been also some studies done with a computer based and remote controlled potentiometer operated equipments. These have also demonstrated similar values. There have been no studies done on an Indian population and there is no normative data for the above for India on a healthy population.

METHODS TO MEASURE SVV AND SVH:-

There are various methods listed in literature about the SVV and SVH, however the important ones are listed below as follows:

Hemispheric dome method:

In this method the patient sits on a chair with the chin resting on a fixed pad and the patient is asked to look straight inside a dome which is hemispherical in shape. The diameter of the dome is around 0.6 metres and it should be sufficient enough to completely fill one's visual field. The dome has a characteristic pattern where it is randomly covered with colored dots. This sort of arrangement is done to avoid any cues to gravitational orientation(43).

A linear target is placed at a distance of thirty centimetres in front of the subject .The centre of the presumed target is fixed on the shaft of the computer base remote controlled potentiometer . While performing the test, the target is rotated in the subject's frontal plane as per the examiner specifications (44).

The target is rotated in a random fashion from vertical and the subject is asked to orient the target according to the subject's perceived vertical utilising joystick {remote controlled potentiometer}. Consequently the difference between the subject's adjusted alignment and

true vertical were analysed by the investigator using the system computer. This is done after taking the average of such ten calculations. SVV is determined for both the eyes(44).

Bucket method:-

This is a method which has gained popularity in many centres worldwide. In this method the patient is instructed to sit vertically straight and look into a semitransparent bucket made of plastic. The rim of the bucket helps to completely cover the visual field. Towards the bottom of the bucket, a straight line running along the diameter is displayed. (45).

On the contrary on the outside of the bottom a perpendicular line arising from the centre of a quadrant is demarcated which is further subdivided into degrees by the zero line which is adjusted to the dark line corresponding inside.

Now the bucket is rotated in no specific pattern to either clockwise or anti clockwise by the examiner, this is done to remove the haptic clues to a minimum as possible, to numerable terminal positions and then it is rotated and brought slowly back to zero degree neutral position.

Patients are instructed to indicate to the tester as soon as the patient feels that the internal line at the bottom is truly vertical by saying “stop” or by raising one hand. Readings are taken from the outside calibrated scale by the principal investigator. Measurements essentially should be made with both eyes open (binocular) and also with either eyes covered separately (monocular left/right) as well(44)(45).

Light bar technique:-

This method is being used most acceptably and has been incorporated into a computer software programme. Here the patients are instructed to be seated upright with the head restrained using a head band, Approximately 150 cms in front of a stationary wall. Patients are instructed to fix their visual field at a poorly lit light bar which is wall mounted and measures approximately 30 x 1 cm.

This whole test should be performed ideally in absolutely darkened exam room in Oder to avoid any visual cues as far as possible. Before testing the investigator randomly places the bars at preset non vertical and non horizontal angulations. While testing, the light bar is rotated around its centre axis by the examiner as per the subjects indication till the point where the bar reaches the patient's orientation of verticality(3).

Thus the readings are made by the examiner as to between the true and perceived vertical. When this is incorporated into a computer base software then the line is displayed on the screen and a remote control potentiometer is used to align the displayed light bar.

Horizontal mechanical device:-

It consists of a spherical background consisting of circles and light bar. The subject has to orient this in the vertical direction as perceived. This methodology helps in calculation of static Subjective visual vertical and dynamic Subjective visual vertical by using a static and rotating disk respectively. The drawback of the above method is that it utilises additional sensory information in terms somatosensory information, as a result of which, the result generated does not accurately assess the values as it was not possible to isolate the involved sensory system(46).

Gondola Test:-During this gondola exercise subjects are exposed to controlled rotational head movements which results in a gravitoinertial force of vector 2.5 G, with an inclination of 66 degrees on gondola. In this the subjects are subjected to monitored movements of head in rotational plane with an angular speed 27 degrees/s which is around 40 degrees. This rotation occurs around the yaw (body z-) axis which occurs as a result of mechanical motor operated helmet(47).

MEASUREMENT PARAMETERS:-

Conditions of measurement of subjective visual vertical or horizontal are mainly static (stationary) or per-rotary (dynamic) where during active rotation the subject is seated in a seated in micro-centrifuge chair. This can be also simulated on computer based software where the background of the screen on which the light bar is projected can be kept stationary or can be allowed to randomly rotate.

The SVV is analysed by subjecting the candidate to align a luminous bar in the vertical axis as perceived. This is accomplished in absence of any clues of the real vertical, in the complete darkness without any visual cues(48).

In a similar study involving a software based assessment of SVV and SVH vales was done. This consisted aligning a virtual line by a mouse. The movement was usually possible in both clockwise (CW) direction and the counter clockwise (CCW) direction. Such software's provide capability of control by the either the investigator or the candidate(48).The display is displayed in a full screen mode and either binocular pinhole spectacles or a tube connected to the display. This screen is left at affixed distance ranging from 30 cms to 1 metre and the stimulus ids projected at a preset angle. The subject was seated in upright position .Head can be also strapped to avoid any motion related artefacts(48).Both dynamic and static SVV and SVH values were analysed and recorded. The test is usually repeated many times to minimise learning effect. While assessing the dynamic values the background is rotated at a constant velocity

It is instructed to the subjects to inform the examiner when subjects feels that the or she has aligned the displayed line absolutely horizontal or vertical to the best of his ability(48).

Subjective spatial perceptions of verticality can be also evaluated with respect to the following parameters namely the subjective straight ahead, subjective visual vertical , subjective haptic vertical and subjective postural vertical (SVV) (1).

SUBJECTIVE HAPTIC VERTICAL is estimated utilising a wooden /metal bar which can be manipulated with respect to-vertical position of earth when the candidate's eyes are closed. The subjective haptic vertical shows the integrity haptic orientation which arises from the inputs of various mechanoreceptors located in skin, muscle bulk, various tendons and multiple joints which play a vital role while manually exploring of the metal bar(1)(49).

The subjective postural vertical is analysed where the subject is seated upon a manoeuvrable chair which can rotate in any particular direction or planes..This chairs mobility is restricted by providing stability in lateral motion which prevents response to any changes in posture of the individuals. The subjects signal when he feels that their body orientation is in the vertical position. This is noteworthy that subjective postural vertical is based on the information arising from graviceptors located in the trunk and also from inputs situated in the region of head and neck (1).

SUBJECTIVE STRAIGHT AHEAD (SVA). This is assessed by subjecting the candidate to align the stimulus given to an orientation which he/she feels as to straight ahead with respect to egocentric framework(50).

NORMAL DEVIATION RANGE:-

Normal subjects usually adjust the SVV or SVH {perceived vertical) within a few degrees on either side from the actual gravitational vertical. A value of normative values ranging from 2 to 3 degrees has been proposed. The various studies are enlisted below with the approximate values of the deviations calculated.

+/- 2.0° (This was substantiated by various studies which include Akin & Murnane in 2009; Bohmer in 1999; Friedmann in 1970; Murray, et al.in 2007; Tabak, et al. in 1997; and Vibert& Häusler in 2000)

+/- 2.5° (This was substantiated by various studies which include Tribukait, et al. in 1996; Tribukait, & Bergenius in 1998; Tribukait, & Eiken in 2005; and Tribuikait, et al. in 2004)

+/- 3.0° (this figure is supported by studies done by Hafstrom, et al. in 2004 and Karlberg, et al. in 2002).

The literature reports normal variation of around 0.5 to 1 degrees from the above listed figures.

Literature has well supported the fact that among the normal people the vertical perception is near accurate and is well reproducible. As seen above in few of the prior conducted studies, normal volunteers have suggested angles of SVV tilts within 2 to 3 degrees(24)(3)(48). In our present study, values of SVV deviations in normal volunteers lie well within the normal data for other countries as per the literature available. Thus it is very indicative that presence of abnormal values of SVV test values indicated lesions in the otolith organs or the involved graviceptive pathways.

AGE RELATED CHANGES ON SVV AND SVH

Additionally Kobayashi et al has clearly stated 1n 2002 as per his study that there exists no significant change in SVV with aging however on the contrary it is an established fact that the vestibular system becomes weak over the years as a person becomes old.

This can be substantiated by the fact that as a person becomes old the propioceptive inputs become lesser and lesser. And also due to the age related changes which have demonstrated anatomically in the vestibular systems .Moreover the sense of verticality also depends on a greater extent to the visual input which also becomes weak as the age progresses due to various refractory errors(51)(52).

IMPLICATION AND APPLICATION OF SVV AND SVH:-

Can be affected in lesions involving the peripheral vestibular apparatus and complex pathways which are graviceptive any extend from anywhere between medulla and mesencephalon, thus helping us indirectly to lead us to the probable diagnosis can help us to prognostigate the clinical situation

Although the static SVV and SVH values may return to normal after compensation yet the dynamic take long time to compensate or rather do not get compensated, therefore these values can indicate whether there was any insult in the past or not which involved the utricular pathway.

Abnormal tilt in SVV have been reported in cases affecting brain stem lesions , vestibular diseases or peripheral vestibulopathy; Vestibular neuritis (sudden idiopathic unilateral peripheral vestibular loss) and Viral labyrinthitis (sudden vestibulo-cochlear incident) this overall suggests of an otolithic dysfunction which may be undiagnosed and if not treated then the patient would show refractory therapeutic response (33,46,53).

In patients affected with vestibular affliction limited to one side, the SVV tilts occurs on the ipsilateral side of the vestibular lesion(54).Thus the above test can help us to detect as to which side is the affected side.

SVV deviations following stroke has been attributed to involvement of central vestibular pathways (brainstem, thalamus, cortex), sensory pathways (thalamus, sensory cortex), and pathology affecting visuospatial analysis as in cases of parietal lesions (55).

Patients with Parkinson's disease demonstrate abnormalities in SVV task which could be associated with putamen atrophy usually seen in cases of Parkinson's disease patients(56).

Multiple sclerosis patients also demonstrate abnormal SVV probably as a result of brainstem and cerebellar involvements (57).

More importantly it has been observed that there are demonstrable subclinical deviations of SVV and SVH .It was observed that the values of subjective visual vertical (SVV) deviations in cases of tension-type headache (1.3+/-1.1 degrees) and in those with migraine (1.5+/-1.2 degrees) were comparatively higher with those of patients without headache (0.6+/-0.4)(58).

It was also seen that the result of various stabillometric tests done on tension type headache and those suffering with migrane headache were abnormal .This also suggested underlying dysfunction of vestibulospinal system(59).

In cases of acute Vestibulocochlear incident, abnormal SVV deviations are seen on the same side of vestibular involvement. This indirectly is a manifestation of It the ocular tilt reaction in response to the vestibular disorder (60).

Similarly in various central lesions such as tegmental pontomedullary brainstem lesions demonstrate SVV tilt to the same side however in case of tegmental pontomesencephalic lesions, opposite SVV tilts are seen(61).

It is well known fact that people with vestibular lesions may have the SVV deviations upto 10 degrees(62).Usually the SVV deviations come back to the normal in cases of labyrinthectomy by one year. Although in cases of transection, full compensation may not happen and a minimal deviation may still be evident after neurectomy even after 4 years. In cases of diagnosed Menieres disease that underwent labyrinthectomy usually a large deviation is seen on the side operated soon after postoperatively, this usually resolves within weeks.

MIGRANE:-

DEFINITION OF MIGRAINE

Migraine has been sub classified under the broad category of *"Vascular headache of migraine" by the* Ad Hoc Committee on Classification of Headache as well as the Headache Classification Committee of the International Headache Society(63).

Ad Hoc Committee has defined migraine briefly as: *"Recurrent attacks of headache widely varied in intensity, frequency and duration. These attacks are usually unilateral at onset; and are usually associated with anorexia, and sometimes, with nausea and vomiting; in some are preceded); or associated with, conspicuous sensory, motor and mood disturbances (aura) and are often familial"* .

However in some migraine episodes, visual and/or other sensory or motor disturbances occur in absence of headache and these conditions have been classified as migraine without aura by the IHS.

A migraine is a common type of headache that is seen in patients presenting to the clinic. Most often it is associated with symptoms such as sense of vomiting, dyspepsia, vomiting, or photophobia or to loud sound. It is more often than not unilateral in presentation.

Usually migraine headache sufferers have associated alerting symptoms. These are called an aura and thee usually present before the typical headache sets in(64). An aura is a constellation of symptoms which may manifest as visual disturbances, heaviness of head, nausea and even parosmia.It. It is believed that an aura is a alerting sign for a bad headache is coming.

Migraine headaches usually runs in families and majority of its sufferers are between the ages of 10 and 45years of age. However it has been reported in much younger and elder people. It is more commonly seen women than men (64)(65).

TRIGGERS OF MIGRANE:-

Well known triggerers of migrane headache are:-(64)(66)

- Caffeine withdrawal {usually found in tea ,coffee and other aerated beverages}
- Alternation in hormonal status during a woman's menstrual cycle {menarche, menopause, contraceptive pills etc}
- Alternation in sleep patterns or lack of sleep
- Alcohol consumption or sudden withdrawal
- Heavy physical exercise or any form of physical stress
- Abnormally bright light and very loud sounds

- Irregular meal timings and quantity
- Various smell perversions
- Excessive smoking and sudden abstinence from smoking
- Any form of stress and anxiety

Certain food indegreints are well known to cause migrane attacks of which most common ones are as following:-(64)(66)

- Baked or roasted items
- Chocolate or Chocó products
- Specific milk products such as cheese, paneer
- Foods with tasteners such as monosodium glutamate (MSG)
- Foods items rich in tyramine, such as red wine, aged cheese, smoked fish-usually consumed in china, chicken livers and figs.
- Fruits like banana and some of the citrus fruits
- Meats items
- Onions
- Nuts and seeds in specific peanuts
- Processed, fermented, pickled, or foods items with preservatives such as sauces.

As described earlier that aura is a group of symptoms usually seen from minutes to hours before the typical attack. The aura is seen in both the eyes and may manifest as following

- momentary blackouts
- or unclear vision
- Pain in and around the eyes
- Seeing stars or zigzag lines or flashes of lights
- Narrowing or tunnelling of vision
- Other occasional associated symptoms include yawning, difficulty to concentrate, felling of vomiting, and rarely inappropriate speech

Usually aura sets in about ten to fifteen minutes before the attack of migrane headache, but can occur just a few minutes to even a day before. It is also noteworthy to remember that not always is a migrane headache associated with aura (67).

EPISODE OF MIGRANE:-

The classical episode of migrane headache(64)(66) –

- Is a dull aching unilateral headache which lasts for minutes or hours
- Headache can throbbing, pounding, or pulsating in nature
- It can be associated with pain behind the eye and pain associated in lower neckache
- It can last minutes to as long as 48 hours

Occasional associated symptoms may be

- Loss of feeling of hunger
- Nausea , vomiting, dyspepsia
- Loss of sensation or abnormal sensation with or without weakness
- phonophobia or photophobia
- Sweating ,excessive urination, depression, irritability

There may be some symptoms which last for many hours after the rue migrane attack has gone off .A few of these may be as follows

- Sense of being dull, lethargic and mental unclearness
- Excessive sleepiness
- Neck or back pain

MIGRANE AND VERTIGO

Patients with migraine headache also have dizziness as one of their complaints. However while evaluating these patients the clinical evaluation of vertigo is underestimated. The tenderness of muscle around the cranium and cervical muscle in patients affected by migraine headache is known to be significantly higher than in normal subjects. This has been found to suggest that, patients with chronic cervical pain and concomitant dizziness have balance disturbance, as compared with healthy subjects. The altered tenderness of cervical muscle can result in altered propioceptive inputs (68).

It is possible that due to abnormal inputs about propioceptive cues for postural control,it can result in unsteadiness. However, till date the exact pathophysiological mechanism explaining the dizziness in patients diagnosed as migrainous vertigo remains unclear.

The objective of this study was to investigate the role of SVV/SVH in patients affected by migrainous vertigo and analyse their subjective vertical and horizontal, which may associated with the subjective imbalance.

This study also encompasses analysis and detail assessment of various otoneurological tests done for patients of migranous vertigo and their respective outcomes. The clinical profile of these patients was also studied and it was tried to observe whether any correlation exists between these factors and the SVV/SVH values.

BACKGROUND FOR MIGRANE ASSOCIATED VERTIGO &MIGRANOUS VERTIGO:-

Patients with migraine often complain of vertigo. However, till date no single hypothesis has explained its pathological basis. Migraine is a well recognized cause of attacks of benign recurrent vertigo and has been linked with several peripheral vestibular and central vestibular abnormalities..

Migraine is a common clinical condition characterized by attacks of unilateral headache and found along with various other neurologic symptoms.

Migraine may be associated with dizziness and vertigo. Clinicians tackling patients of dizziness have stressed over time that migrane and vertigo are more often associated with each other but is often underreported of under diagnosed. Association between migraine and vertigo has been established over a century ago however the exact pathophysiology involved has not been established and infact many theories have been proposed(69).

Migraine-related vertigo (MV) has not been understood clearly as any study aimed to understand is hindered by several factors some of which is listed below. Firstly the way a MV patient presents is very vague especially with regards to the duration of episodes, nature of vertigo, and the unusual temporal relationship between vertigo and headaches.

These patients may present in a very similar pattern that overlaps with other situations such as Ménière's disease or benign paroxysmal positioning vertigo (BPPV), thereby making the diagnosis still more confusing. However most neurotologists accept that migraine is to be ruled out when evaluating and treating patients with recurrent vertigo (70).

MIGRANOUS VERTIGO:-

The Headache Classification Committee of *The International Headache Society (IHS)* has set criteria to classify migraine and has also set criteria's for the various subtypes of migrane. Thus to reach a diagnosis of "migraine related vertigo" or rather migranous vertigo, we need to strictly follow the standards set in by the IHS.

IHS in 1988 classified a term as *basilar migraine* (71)in which vertigo is a symptoms included i n the aura of such migrane. Later *Benign paroxysmal vertigo of childhood* (72) was also sub classified in the in the IHC classification however the *"episodic torticollis"*(73) was excluded in the classification. Similarly *"*Benign *recurrent vertigo"* of adults was not included in the IHC classification as these were the conditions which were considered very similar to episodes of migrane without headache(74)(75).

Although the controversy still exists and some authors still believe it to be equivalent to migrane. Therefore of late the consensus is that if benign recurrent vertigo is associated with migranous features during few episodes along with a history of typical migranous attacks

outside the classical episodes of vertigo, it could be probably classified as *"Definite ,probable or possible" migrainous vertigo depending on the nature and duration of the migrane and vertigo episodes.*

As per the literature available broadly the following guidelines have been layed for helping for categorising

DEFINITE MIGRAINOUS VERTIGO

(Modified from H. K. Neuhauser, et al, 2001)(76);

- Episodes of vestibular symptoms which are of at least moderate severity (characterised by rotational vertigo, other illusory self or object motion, positional vertigo, head motion intolerance).

- Presence of at least two of the following listed below migranous symptoms during at least two vertiginous attacks:

  These are (a) migranous headache;

  (b) Photophobia,

  (c) Phonophobia;

(d) Visual or other aura.

- Attacks of migraine (outside episodes of vertigo) as per the listed IHS criteria.

- Some central and/or peripheral vestibular abnormalities may be present in vertigo-free periods.

All Other causes should be ruled out by appropriate history, physical examination and other appropriate investigations.

PROBABLE MIGRAINOUS VERTIGO

(Modified from H. K. Neuhauser, et al, 2001(76);

- Episodes of vestibular symptoms which are of at least moderate severity (characterized by rotational vertigo, other illusory self or object motion, positional vertigo, head motion intolerance)
- Presence of at least one of the following listed below, in relation to at least one vertiginous attack:

  (a) Migrainous headache,

  (b) Photophobia,

(c) Phonophobia,

(d) migraine-specific triggers e.g. specific foods,
Sleep irregularities, hormone changes

- Positive response to migraine prophylactic drugs.

- Migraine (outside vertiginous attacks) according to the criteria of the IHS

- Some central and/or peripheral vestibular abnormalities may be found in vertigo-free periods.

All Other causes should be ruled out by appropriate history, physical examination and other appropriate investigations.

POSSIBLE MIGRAINOUS VERTIGO(76)

- Episodic vertigo, associated occasionally with tinnitus but usually without hearing loss

- May be accompanied by nausea/vomiting and ataxia

- Nystagmus may be demonstrated during the episode

- Duration of the attack may last from minutes to hours, usually less than one hour , or hours to days

- Presence of episodes of migranous headaches outside vertiginous episodes, and/or positive family history of migraine.

- Normal audiometric findings, or no asymmetry if there is an incidental hearing loss

HISTORY OF MIGRANOUS VERTIGO:

The foundation of migranous vertigo was laid in 1979 when Robert Slater conceptualised the existence of benign recurrent vertigo .Later in 1992Robert Baloh was the first to bring to light something what later came to be known as migraine-associated dizziness .In 1994 the term familial benign recurrent vertigo was established.

Joseph Furman in 1997 suggested that episodes of migrane can have an impact on the vestibular system and suggested a diagnosis of migraine-related vestibulopathy for such situations. It was in 1999 when Thomas Brandt suggested the concept of vestibular migraine which later in 2001 was studied further and termed as migranous vertigo by H. Neuhauser & T. Lampert(77).

NOMENCLATURE

Till date a uniform nomenclature to describe the association between migraine and vertigo has not found acceptance. However various terminologies such as migrainous vertigo, vestibular migraine, migraine-associated dizziness, or migraine-related vestibulopathy have been suggested and used. Some of these terms are used merely to suggest a coincidental association between migraine and vertigo, whereas others are merely descriptive.

SYNONYMS:-

Various synonyms that are used interchangeably to explain the above condition are the following-

Migraine-associated vertigo (MAV)

migranous vertigo

Vestibular migraine

Migraine-associated dizziness

Migraine-related vertigo

Migraine-related vestibulopathy

Benign recurrent vertigo

As mentioned above that many different terms have been developed to describe this concept, of which the more popular being vestibular migraine, migranous vertigo, and migraine-associated vertigo. Till date the most commonly cited diagnostic criteria are according to Neuhauser which has been recently included in the International Classification of Headache Disorders(78).

DEMOGRAPHIC PROFILE:-

Worldwide prevalence of migraine ranges from 6% to 18% in women and 3-6% in men approximately. Large sized population based studies have shown that the occurrence of vertigo in normal population is above 20% with a overall lifelong prevalence of vertigo of around 7%(76).

It has also been suggested that 3.2% of the people has both vestibular vertigo and migraine; of which around 1% of the population suffering from migrainous vertigo(79).

Migrainous vertigo may be under diagnosed in majority of cases due to the fact that some patients have exaggerated headache while having giddiness at the same time and these patients report headache as their chief complaint.

In such cases diagnosis of migranous vertigo should be made by taking into consideration the entire spectrum of the clinical presentation. This should include complaints such as phonophobia, photophobia which may be related to vertiginous attacks of migrane.

Detailed history in patients with migraine and vertiginous symptoms helps to establish a connection if at all it exists. In most cases, a complete chronological sequale of the events and daily recording of events may help could help to analyse as to whether migrane and vertigo were entirely separate entities or had a overlap in presentation. Literature has suggested that maintenance of Migraine diaries have been found limited role in establishing a correct diagnosis(80).

Migraine should be easy to diagnose because strict diagnostic IHS criteria have been proposed but on the other hand diagnosing a vertiginous disorder is not always as easy if in some patients not all criteria could be fulfilled, a diagnosis of probable migrainous vertigo could be established in order to help guide the therapeutic course(81).

PATHOPHYSIOLOGIC MECHANISMS INVOLVED:-

The mechanisms of migraine pathophysiology are still a mystery unsolved. Till date the migrainous vertigo patients share common pathophysiological pathways when compared to migrane attacks.

Vertebral artery is found to be hypoplastic more frequently in migraine patients, especially in migraine patients who have aura also present, which indirectly suggests that it may play a role in migraine pathophysiology(82).

Some reports demonstrated that patients with migraine have subclinical dysfunctions in the vestibular spinal reflex system, which may be partially due to the subclinical damage to otolith macula

Migrane being a heterogeneous disorder has been linked with the mutations in the gene for the a1 subunit component of a voltage-gated calcium channel such as familial hemiplegic migrane .Also it is worth nothing that in some patients with cerebellar dysfunction, a neuronal calcium channelopathy can be detected, thus suggesting a role of calcium channelopathy in the pathophysiology(83).

A genetic study which was performed on14 genetically unrelated patients with migrainous vertigo had suggested that genes causing familial hemiplegic migraine and episodic ataxia type2 represent major susceptibility loci for MV ,however this has not been conclusive till date as a thorough genetic workup is needed, to try to establish existence of a genetic background if such exists(84).

Some studies have also demonstrated a significant association between migrainous vertigo and coronary heart disease and even with diabetes has been established and it has been already established in patient with migrane that while in deranged cardiovascular profile has been linked to adult migraners however, a causal relationship remained unestablished (85).

It has also been suggested that people with low blood pressure are at higher risk of having migraine attacks or that both manifestations could share a common risk factor, since systolic blood pressure values were found in patients having more frequent attacks of migrane(86).

The pathophysiology of MV is vague, due to absence of clear cut systematic investigations and diagnostic criteria. Diagnosis of migranous vertigo based on proper methodology and patient identification are just beginning. Interestingly spreading depression, which has been proposed as a mechanism of the migraine aura, may play a role in patients having brief episodes of migranous vertigo.

Spreading depression is a cortical mechanism which mainly involves the posterior insula and the temporoparietal junction can produce vestibular symptoms when these multisensory cortical areas become involved as these sites are well known to process the vestibular signals. The drawback of the above proposed theory is that during the acute stage of MV, including canal paresis and complex positional nystagmus, cannot be explained by cortical dysfunction mechanism(87).

Various neurotransmitters that are involved in the pathogenesis of migraine namely calcitonin–gene-related peptide, serotonin, noradrenaline, and dopamine are also known to modulate the central and peripheral vestibular pathways and therefore may be related to the etiopathogenesis of MV(88,89).

Therefore release of these substances which occur with episodes of unilateral headache can also cause static vestibular imbalance leading to rotatory vertigo. Release of the above substances have known to influence the dorsal raphe nucleus, nucleus raphe magnus, locus ceruleus, and lateral tegmental region which could probably result in a functional vestibular tone imbalance due to asymmetric activation or deactivation of vestibular neuronal activity(90).

The above mechanism gets reinforced with the support of findings showing brief and recurrent brainstem dysfunction being demonstrated with brainstem auditory evoked potentials {BERA and CERA} and positron emission tomography{PET} in patients of migranous vertigo (89).

As peripheral auditory and vestibular deficits coexist, some authors have suggested that peripheral part of the vestibular system has a role in pathogenesis of migranous vertigo. Another probable theory is that migraine-induced vasospasm causes decrease in regional blood flow to the inner ear (via the internal auditory artery from the anterior inferior cerebellar artery) causing lack of blood supply an ultimately hypoxia to the labyrinth there by resulting in transient or permanent hearing or vestibular loss (91).

Presence of ocular motor (central) and cerebellar dysfunction in migraners between the episodes of migranous vertigo propose subclinical uninterrupted neuronal abnormalities in brainstem and cerebellar centres hinting towards the pathophysiology further(68).

A recent synopsis focussing on the probable etiopathogenesis association among migrainous and vestibular pathways has been suggested. These overall suggest complex correlating interactions involving the nuclei of vestibular pathways, the trigeminal complex, and thalamocortical centres(29).

Its well documented that genetic abnormalities in ion channels have been linked to numerous neurological pathological conditions. On one hand familial hemiplegic migraine (FHM) and episodic ataxia type 2 (EA-2) have common diagnosed abnormality in voltage gated calcium channels and on the other have similar symptoms consisting of vertigo and migrane headaches. This suggests that the gene responsible for MV susceptibility to lie in the same region(92).

Literature has suggested that association of migraine and vertigo is more than just coincidental .In a more clear understanding it would be appropriate to stat that migrane is more commonly associated in patients with chronic vertigo and simultaneously vertigo is more commonly a feature among migraine sufferers. Kayan and Hood's discovered that symptoms such as vertigo which could be both vestibular and nonvestibular in nature are reported to occur as high as fifty five % in migrane patients when compared to thirty percent in tension headache patients (93).

The following additional points suggest that migrane affects the vestibular apparatus or rather both of these have a common pathophysiology which favours the various theories laying foundation for migranous vertigo patients

IT was observed that the patients with migraine who presented to the outpatient department for the treatment of headache had a history of episodic vertigo in as much as 20 to 33 percent of individuals(94).However when the lifetime prevalence of vertigo calculated in general individuals it was found to around 8 percent (95).

Approximately 50-75 percent of migraine suffers also manifested with motion sickness which is around two to five times greater than the prevalence in general population(96).

Numerous studies have suggested that approximately 25 percent of migrane headache patients had abnormal results on the battery of vestibular function tests. This clearly indicates about the coexistence of vestibular anomalies in migrane patients. It is also noteworthy that these patients also had vertigo as one of the symptoms (59)(97).

Prior studies included subjects who underwent the battery of tests in the symptom free periods but as per some studies done during the acute attack, it revealed findings which were suggestive of both central vestibular dysfunction and peripheral dysfunction (98).

Vestibular migraine may be actually the counterparts of recurrent cases of benign paroxysmal vertigo of childhood. If followed up for a long time, a significant fraction of these patients actually turned into cases of Vestibular migrane. Further it should be noted that this entity is accepted by the International Headache Society (IHS) as a migraine equivalent (99).

It is also very interesting to know that the linkage of vertigo and migraine is specific, and this does not extend to other headache syndromes. Studies have suggested that when vertigo was seen to coexist in groups of people with migrane headache and tension headache, it was

found that vertigo was more common among the migrane headache patients. Similarly in another study it was established that the stabillometric findings of vestibular dysfunction was much higher in migrane patients when compared to with those of tension type of headache(59).

Audiological manifestations in migrane patients has been well established earlier .Some of these include hypersensitivity to sound-better known as phonophobia, ringing sound in ear, decreased hearing which maybe sudden or progressive, or even blocked sensation of ear(100).

Another land mark work done by Neuhauser et al. who studied a group of 200 patients recruited from dizziness clinic and was compared to 200 patients who served as controls and had similar demographic profile. These were recruited from orthopedic clinic .These were then studied on detail to calculate the prevalence of migraine in these people as per the IHS criteria. Upon analysis migraine was found to 1.6 times commoner in the dizziness patients when compared to those from the orthopedic clinic (101).

Lee et al evaluated72 subjects prospectively presenting as unexplained isolated vertigo and following detailed otoneurological workup showed that migrane had a prevalence of around 61% when compared to 10% in the control group (102).Lempert and Neuhauser also found that the overall prevalence of MV was around 1% in the uncategorised population. These all data indirectly hints that the above occurrence is not only a coincidence, and suggested MV to a more commoner under diagnosed condition.

CLINICAL PROFILE:-

Migraine-related vertigo or rather migranous vertigo can manifest irrespective of the age with the first attack occurring between as young as seven years and as elderly as 72years but most commonly found in the late thirties and fifties. Women make up the bulk of the load of such patients {60% to 85%}.

Classically, migraine headaches may occur many years before the actual vertigo manifests .These patients usually don't have attacks of headache for years before vertigo develops .Usually females have these headaches precipitated with vertigo around menopause. On the other hand vertigo can be the first presentation of migraine before or in absence of headaches (103).

Patients of migranous vertigo usually present with episodes of unprovoked vertigo or the felling of self motion or that of the surrounding. The character is usually spinning vertigo but may be a back and forward sensation or even head spinning sensation. Other patients may also report other vague complaints such as light-headedness, vertigo, giddiness, a sense of buoyancy or submersion , feeling similar to motion sickness(103).

Some patients may complain of some postural imbalance which usually settles down and they are able to locomote without help. specific posture may also trigger the episodes .Migraine-related positional vertigo has been well documented and can be classically differentiated from BPPV as a result of underlying canalithiasis since the former may have association to some of the migrainous features, attacks which lasts variably from minutes to hours as compared

from weeks to months, it is found early in the life, it occurs repeatedly, and the nystagmus which is positional in nature which is atypical for BPPV (104).

It is also true that Visual vertigo precipitated by observing rapidly mobile objects or fast moving scenes, car-chase ,fast moving objects such as train) is another common symptom of MV that may manifest as one of the vestibular complaints(105).

Patients with migrainous vertigo usually manifest with migrainous headache in about 24-45%.It is also noteworthy to note that reported literature shows in about as high as 48% patients that vertigo occurred with or without headache(106).

A higher proportion of patients usually present with migrainous vertigo associated with visual auras as compared to group of dizziness free migraners (107).

In cases of migranous vertigo the most clinching part is the history as it provides maximum input towards diagnosis when compared to any other vestibular tests. This is true due to absence of any specific abnormalities to MV. Thus practically in patients who have a strong suggestive history, no further audio vestibular tests should be recruited.

The additional audiovestibular testing helps to reassure patients and can be used to rule out other undiagnosed problems mimicking migranous vertigo. Even significant findings on ENG like complete canal paresis and hyperactive responses can be picked up which can suggest alternate diagnosis. Sometimes, minor findings on vestibular testing may also help to detect co existent diagnosis such as BPPV co existing with migranous vertigo If tests are conducted

within the period of attack or soon after the attack, it can suggest us the amount of compensation that has practically occurred with the treatment given(108).

There are various precipitating factors for the migranous vertigo attacks and the nature of these precipitants provides vital diagnostic information. Migranous vertigo attacks are usually influenced any hormonal turbulence, thus it is more likely to be correlated as a diagnosis in vertiginous women with menstrual disturbances, when compared to other vestibular syndromes.

Some of the well known triggers of migraine include lack of sleep or irregularity in sleeping hours; unhabitual stress; peculiar food items, to name a few of them will be refined milk products, wine, and glutamate rich foods; and other provocators of various sense organs such as flashing lights or zig zag lights, particular odours, and loud sound. The triggers are highly subjective and each one affects to a very small group of people suffering from migraine.

To diagnose MV it involves a high degree of critical analysis\ and the art of eliciting the history for any features within the spectrum of migranous disorders. Patient could also be evaluated in detail about present or previous headaches, short lasting somatosensory or ophthalmic aura symptoms, and motion sickness in childhood or periodic vomiting. It is also noteworthy to evaluate the person about any history of childhood sleepwalking, which may be as high as six times in patients with migraine when compared to those nonmigrainous headaches (109).

CLINICAL FEATURES OF MIGRANOUS VERTIGO:-

Symptoms can range from

- Fluctuating dizziness and recurrent episodes of vertigo which can be either surrounding rotatory or head rotatory vertigo.

- Patient can also complain of motion sensitivity (which can be felt in all directions).There can be only nausea in motion and the patient may not even report of any vertigo

-Bilateral tinnitus without progressive hearing loss is also reported in some of these patients .These patients can also present as phonophobia.

- These can also present as eye soreness/heaviness or photophobia and even can present as transient blurred vision/ Visual vertigo

-He /She can also present as neck/shoulder pain and quite a few of these are misdiagnosed as cervical vertigo.

-Nausea or occasionally vomiting can be a part of the manifestations of the above condition.

Many patients who are diagnosed as Migranous vertigo may not present as headache, or rather have chronic non-specific headache which does fit into the migraine classification proposed by the International Headache Society(108).

The exact basis for the above condition is unknown but is being investigated through analysis of clinical experience and via means of genetic research. Once this condition was thought to be extremely rare but is now turning to be common etiology of long lasting recurrent vertigo which were earlier unexplained.

Migranous vertigo was often misdiagnosed as Ménière's Disease, Vestibular Neuritis or was thought to be a part of psychiatric disorder. As per the recent consensus a condition previously known as "atypical Ménière's" is no longer valid and is rather believed to be a part of migrainous vertigo syndrome(110).

Basilar migraine is otherwise also addressed as Bickerstaff syndrome .It is subcategorised under the broad diagnosis of migraine with aura.

Bickerstaff Syndrome usually presents as a combination of the following symptoms namely dizziness, ringing sensation in the ear, hard of hearing, gait disturbances, difficulty in articulating speech, visual disturbances, double vision, abnormal sensation or weakness of a part of body and even altered levels of consciousness which is usually accompanied by an attack of throbbing headache(111).

The vertigo episodes in migraine-associated vertigo have various durations and an approximate duration on an average that was studied is as follows

- Episode lasting for seconds (7%)

- Episode lasting for 2 hours (31%)

-Episode lasting for two to six hours (5%)

-Episode lasting for six hours to one day (8%)

-Episode lasting for more than a day (49%)(111).

Till date there is no gold standard diagnostic test that exists for diagnosing a case of Migranous vertigo. Judgemental combination of inputs from clinical history and the results of various otoneurological tests lead us to the diagnosis

A definite diagnosis of migraine-associated vertigo/Migranous vertigo is usually thought in cases where patients have migraine with aura and is accompanied by concurrent episodes of vertigo.

It may be also considered in cases where the patients have migraine without accompanied aura and such episodes are repeatedly associated with vertigo either soon before or during the episodes of headache(111).

PRECIPITATING FACTORS FOR MIGRANOUS VERTIGO:-

-motion induced vertigo

-exposure to loud sound, excessive bright light, certain particular odours or even high wind flow

-Hunger-inducing the attacks is a well known fact which may be misdiagnosed as hypoglycaemia

- Insomnia, lack of proper sleep and improper dreams are one of the very under evaluated causes.

- Excessive Anxiety and stress are also potential provokers for the attacks which may be misdiagnosed as psychogenic dizziness

-Menstrual irregularities may precipitate the attacks but many a times the co existent anaemia may be thought as the cause for dizziness when actually it is not the cause.

- Postmenopausal dizziness

- Postmenopausal syndrome

Other contributory history that may be present in these patients are following

- History of motion sickness may be present

- These patients may also have history of recurrent giddiness/vertigo during childhood which may be a part of the benign paroxysmal vertigo of childhood.

- Familial migraine or recurrent Migraine-associated vertigo if more often than not present in these patients(112).

DIFFRENTIAL DIAGNOSIS:-

It is important to exclude vestibular causes which can be both central and peripheral before reaching to a diagnosis of migranous vertigo.

Peripheral disorders mimicking Migranous vertigo are Ménière's disease, endolymph fistula, benign paroxysmal positional vertigo, episodes of vestibular neuritis, and cases uncompensated vestibular function (113).

On the other hand central causes include multiple sclerosis, , compromise in the supply from vertebrobasilar artery, compressive pressure effect in cervicomedullary region due to the anomalies/pathologies related to the craniovertebral junction(113).

However the main differential diagnosis is with Ménière's disease. This is due to the fact that the symptoms of Ménière's disease and Migranous vertigo overlap .The common symptoms include episodic vertigo, sensorineural hearing loss, and ringing sensation of sound

occasionally. The differentiation of migranous vertigo from that of Menieres disease very difficult(114).

This can be made easy with the help of detailed history which offers clues that helps make the correct diagnosis. Photophobia, no progressive sensorineural hearing loss, prolonged vertigo, chronic complains of motion intolerance, and episodes of vertigo associated with menstrual periods are few of the symptoms which suggest the clinical diagnosis of migranous vertigo over Menieres disease. It is also very important to know that a single individual can be affected by both Migraine and vestibular disease. Therefore even if a patient gives history of migrane headache, yet if all the criteria are met to reach a diagnosis of Ménière's disease then the subject should be treated as Ménière's disease (111)(5)(113).

This study was done as in the western literature it is suggested that migrane patients have vestibular dysfunction which may be central or peripheral(12). Moreover it is also suggested that patients suffering from migrane and migranous vertigo share similar profile a pathophysiological mechanisms(108) .It has been studied that results of various stabillometric tests and vestibular tests are abnormal in cases of migrane(59). The otoneurological profile of patients with migranous vertigo has also been studied(115) .Importantly none of the studies in western literature have studied the correlation of SVV and SVH values and the profile of migranous vertigo patients .Our study has tried to study the above correlation if any and also aspires to calculate the normative values of SVV/SVH in Indian population as there are no values for the reference in Indian scenario.

MATERIALS AND METHOD

DESIGN:-

This was a hospital based prospective cross sectional study of a group of normal adults and patients with migranous vertigo carried out over a period of 12 months in 2013-14.

SETTING:-

CMC is a tertiary care multispecialty teaching hospital of repute under Tamilnadu Dr.MGR Medical University in the state of Tamilnadu in India. It has approximately 2200 beds and it caters to referred patients with medical and surgical problems from all over India .The department of ENT has 5 units which sub specializes in Head & Neck Surgery, Neuro otology, Rhinology / Endoscopic Sinus Surgery and Paediatric ENT and Audio vestibular Medicine.

It has, in addition, a well-established Audiology and Speech therapy unit, audio-vestibular and temporal bone dissection laboratories. There are more than 2 lakh OPD patients and 6000 inpatients every year. It has outpatient clinics 6 days a week and elective operations 5 days a week with three operating theatres on each weekday. In addition, there are speciality clinics in the afternoon sessions of every day.

Among the facilities for investigation that are available are the full range of diagnostic otoneurological tests like pure tone audiometry, impedance testing, auditory brainstem response audiometry, otoacoustic emission, and ECoch G for investigation of vestibular conditions and Electronystagmography.

RECRUITMENT OF PATIENTS:-

Locations: Outpatient department, SVV room in audiology section of CMC.

The study involved two separate groups, normative group and migranous vertigo group. Normative subjects were volunteers with no history of vertigo or any otological complaints with normal otoneurological physical examination and were recruited from relatives of patients attending the audio vestibular clinic OPD in CMC.

Patients and volunteers were subjected to SVV and SVH testing and the values are recorded after obtaining a written consent. All the recruitment was done by the study physician while examination was done by non-study physicians who were aware that a study was on going, but not involved in the selection or testing of the volunteers or migranous patients. One technician who conducted the test was blinded to the status of the person being tested.

DURATION OF THE STUDY:-

The study was conducted from July 2013 to August 2014. The target was to analyse a total of 72 normal volunteers and 72 cases of migranous vertigo which would be recruited as per the statistical requirement and the inclusion/ exclusion criteria.

INCLUSION CRITERIA FOR NORMATIVE GROUP:-

These were relatives of patients, (males and females) who were between the ages of 20 and 60 years who never in the past or present had a history of one or more of the following:

Giddiness or vertigo or imbalance

Otological complaints (hearing loss/tinnitus/ear discharge/earache/ear trauma)

Persistent headache,

Any history of head-trauma,

Ototoxicity due to drugs,

Or any prior ENT surgeries,

No history of Diabetes mellitus or hypertension or hypothyroidism

They had to be normal after a basic otoneurological examination and were taken as "**clinically normal**" (48). These candidates were provided a patient information sheet which mentioned about the details of test proposed to be conducted upon him/her. Following this the volunteer was asked to sign consent, prior to the test. All this was done by the study physician. A copy of the patient information sheet is attached as annexure 1.

INCLUSION CRITERIA FOR MIGRANOUS VERTIGO PATIENTS:-

Patients for the above study between the ages of 20 to 60 years (consecutive cases) were recruited for the test. These patients were those who were attending the OPD and were clinically diagnosed to have migrainous vertigo by one or more non-study physicians but verified as migrainous vertigo by the study physician as per Neuhausser's classification. Signed consent was obtained prior to the test. This inclusion process was over a 12 month period.

Just as with the normal volunteers, all of the patients were also provided with an information sheet containing details of the tests and following this, a consent form was provided for their signature before proceeding to the test. A copy of the consent form is in annexure 2.

PROCEDURE OF THE TEST-

Static and dynamic tests were conducted on each individual, both on the normal volunteers and patients with migranous vertigo. Standardization of the test had been done earlier on staff volunteers and the person performing the test was a trained technician whose primary role was to carry out these tests. The study physician ordered the tests through the online ordering system. This enabled blinding of the technician about the status of the subject.

A very important aspect was that the room in which the test was conducted was fully dark so that visual cues to the person undergoing the test were reduced to a minimum. It was equally important to calibrate the verticality of SVV and SVH test by using a plumb line which served the reference line for the gravitational vertical.

The Subject was made to sit in a darkened room (to avoid any visual cues, as mentioned earlier).The height of the chair was such that it was at the same level as to that of the screen. The Head and neck were stabilised in erect neural position using a headband fixed to the patient's high back chair.

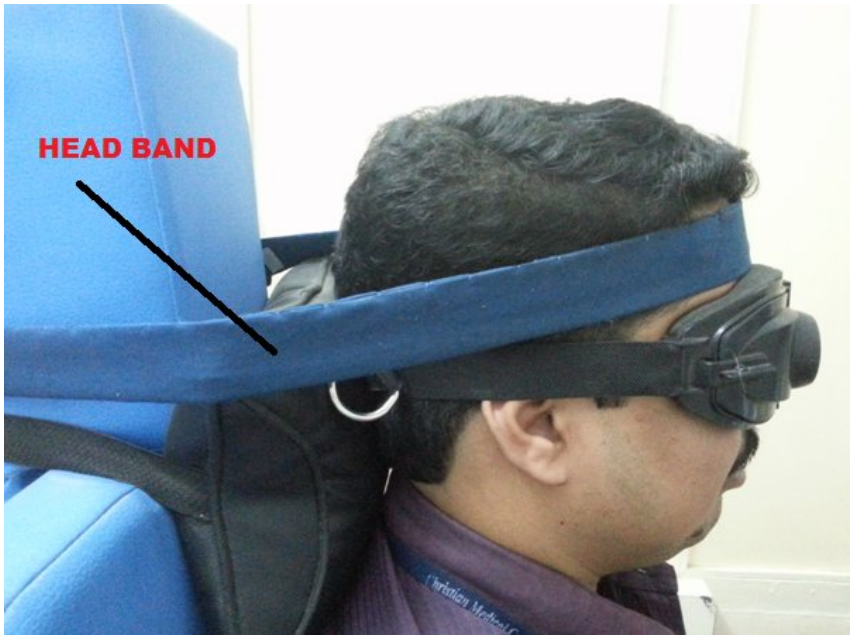

Figure 8: - Showing head being stabilised via a head band

Patient was fitted with a contour mask with binocular vision fitted with a set of 3 obturators so as to reduce the chance of any visual cues. It was confirmed that the vision was binocular and not uniocular after fitting of the contour binocular spectacles.

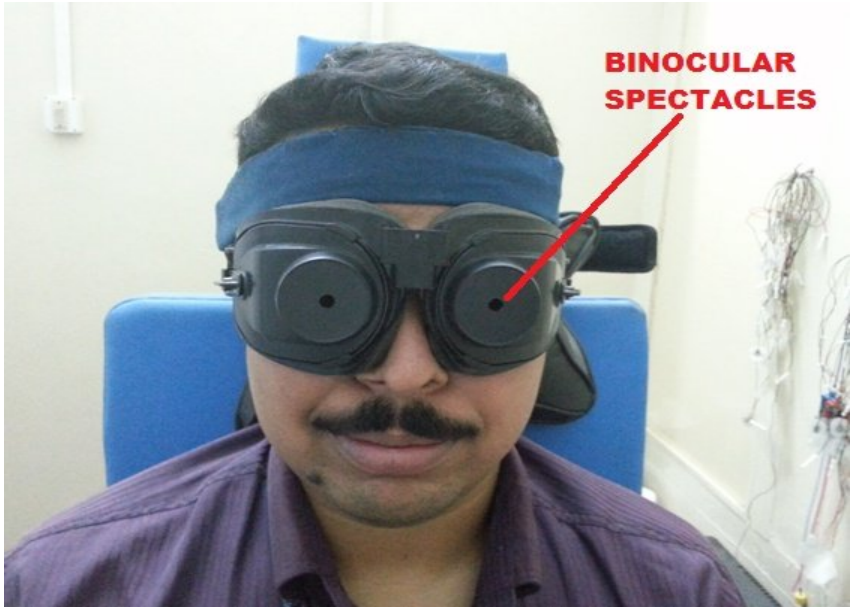

Figure: 9 - showing subject fitted with binocular spectacles and head band

The stimulus is projected on a large screen monitor mounted in front of the patient. The stimulus is a projected vertical illuminated 'line' on the screen provided by the software from the SVV equipment (MUS_VS-V1.3.2.Rev B) Synapsis Company-France)(34,116).

Figure 10: - Showing self illuminated bar on the screen

The 'line' is presented at a preset angle (between 5 degrees and 20 degrees) Patient is required to adjust the 'line' to vertical as perceived by the subject using a joy stick (remote controlled potentiometer). For the dynamic assessment the background is made to rotate clockwise or anti-clockwise.

Figure: 11- showing remote controlled potentiometer

The test is repeated 6 times each for static and dynamic settings and values are recorded. The test is similarly repeated with a stimulus with a projected horizontal illuminated ‘line’ to test the SVH. These at the end of the performance of the test, there are 24 values, 6 for each group. These values may be either depicted as positive or negative according to the direction of deviation. The average is calculated as arithmetic mean irrespective of whether the value is positive or negative.

Test (Examination date)	Test	Angle	Patterns	Frame	Head	Vision
Examination dated: 8-26-2014	Vert.	-2.0	Normal	None	|	oo
Examination dated: 8-26-2014	Vert.	-3.0	Normal	None	|	oo
Examination dated: 8-26-2014	Vert.	-3.0	Normal	None	|	oo
Examination dated: 8-26-2014	Vert.	-1.0	Normal	None	|	oo
Examination dated: 8-26-2014	Vert.	0.0	Normal	None	|	oo
Examination dated: 8-26-2014	Vert.	-1.0	Normal	None	|	oo
Examination dated: 8-26-2014	Vert.	2.0	Normal	Opto +20°/s	|	oo
Examination dated: 8-26-2014	Vert.	3.0	Normal	Opto +20°/s	|	oo
Examination dated: 8-26-2014	Vert.	2.0	Normal	Opto +20°/s	|	oo
Examination dated: 8-26-2014	Vert.	-5.0	Normal	Opto -20°/s	|	oo
Examination dated: 8-26-2014	Vert.	-4.0	Normal	Opto -20°/s	|	oo
Examination dated: 8-26-2014	Vert.	-4.0	Normal	Opto -20°/s	|	oo
Examination dated: 8-26-2014	Horiz.	-3.0	Normal	None	|	oo
Examination dated: 8-26-2014	Horiz.	-1.0	Normal	None	|	oo
Examination dated: 8-26-2014	Horiz.	1.0	Normal	None	|	oo
Examination dated: 8-26-2014	Horiz.	0.0	Normal	None	|	oo
Examination dated: 8-26-2014	Horiz.	1.0	Normal	None	|	oo
Examination dated: 8-26-2014	Horiz.	1.0	Normal	None	|	oo
Examination dated: 8-26-2014	Horiz.	3.0	Normal	Opto +20°/s	|	oo
Examination dated: 8-26-2014	Horiz.	3.0	Normal	Opto +20°/s	|	oo
Examination dated: 8-26-2014	Horiz.	3.0	Normal	Opto +20°/s	|	oo
Examination dated: 8-26-2014	Horiz.	-3.0	Normal	Opto -20°/s	|	oo
Examination dated: 8-26-2014	Horiz.	-3.0	Normal	Opto -20°/s	|	oo
Examination dated: 8-26-2014	Horiz.	-2.0	Normal	Opto -20°/s	|	oo

Figure12:-Result of SVV and SVH measurements for a subject shown above

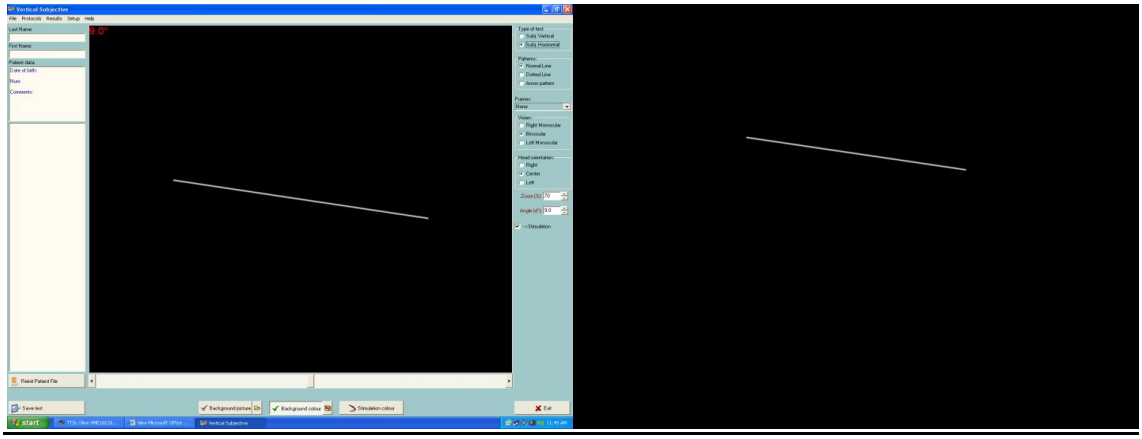

Figure 13: - showing software screen and display screen simultaneously

SELECTION OF SUBJECTS AND TESTING METHODS

Biases were minimized by the following:

1. The identity of the subject as to whether he or she was a normal volunteer or a case of migranous vertigo was not disclosed to the technician conducting the test.

2. The diagnosis of migranous vertigo was made by non-study physicians in the clinic and the study physician had to ensure they fitted the Neuhausser's classification criteria prior to inclusion for the study.

3. The principal investigator was not allowed to conduct the test; instead a trained technician performed the test.

4. Selection of patients and volunteers was consecutive

5. The room in which the test is conducted was kept completely dark to avoid providing any visual cues to the patient

6. The subject had to wear binocular spectacles with a pinhole aperture to see, this was done to further reduce any visual cues.

7. The head of the subject was fixed using a head strap so as to remove any inclination artefacts which could potentially alter the result.

SAMPLE SIZE:-

For a target sensitivity of 90%, an alpha of 0.05 and a precision of ± 10%, a sample size of 36 were required in each study population in each age group.

72 volunteers (36 each in two broad age groups) were targeted to be studied for eliciting normative values. This sample size was expected to provide a near normal distribution. Since the technician had adequate training and standardization in the use of the equipment prior to the start of the study and only one technician carried out the tests, the person to person variation was expected to be low.

Seventy two consecutive patients, (36 each in two broad age groups) with clinically confirmed migrainous vertigo were targeted to be recruited to be studied.

AUDIOVESTIBULAR PROFILE OF PATIENTS DIAGNOSED AS MIGRANOUS VERTIGO

Those diagnosed as migranous vertigo patients were also counselled and explained about the need of various tests and if willing these audiovestibular tests were carried out for them. Consent was taken for them undergoing the SVV and SVH tests. Various other aspects were studies for these patients were analysed such as the:-

The various test outcomes include the following:

Pure tone audiometric findings-average values were calculated

Impedance audiometry findings-type of curve and whether reflexes were present or absent

Outcomes measured using the history and findings:

Duration of vertigo and type of vertigo-vertigo duration was categorised based on the duration and it was also observed as to whether the vertigo was surrounding rotatory, head rotatory or a combination of both.

Duration of headache,-it was analysed as to for how many years was the history of headache present.

Association with tinnitus and hearing loss: it was analysed whether the subjects had ringing sensation in the ears or complained of decreased hearing from either ears. It was also assessed whether the hearing loss was sudden in onset, fluctuating or progressive in nature.

Imaging characteristics-to analyse whether imaging was done and whether these had any incidental /important findings

Photophobia and phonophobia -to analyse any discomfort encountered while exposure to loud sounds or bright light. Some patients could also complains of halos or zigzag light sensation.

ENG profile of these patients-in terms of whether ENG was performed and as to what were the response such as normal study/l canal paresis, left or right canal paresis or whether any hyperactive response was present.

Family history contributing to the illness among the patients.

Neurological complaints among the patients-like complains of tingling sensation in fingers and extremities, loss of consciousness, numbness or paresthesia in any part of body, blackouts, seizures, double vision etc.

Co morbidities:-

Such as diabetes mellitus, hypertension, hypothyroidism, etc.

The data collection questionaire is attached as **annexure 3**.

ETHICS COMITEE AND INSTITUTIONAL REVIEW BOARD:-

The study proposal was accepted by the institutional review board and ethics committee. The research funding was obtained from the fluid research grant of the institution.

A copy of the IRB and ethics committee approval is attached as **annexure 4**.

-: RESULTS AND ANALYSIS:-

Two groups were recruited among patients and normal volunteers belonging to age groups of 20 to 40 years and 41 to 60 years.

Target was to collect 36 for each group, therefore 72 for normal volunteers and 72 for migranous vertigo patients.

At the end of 12 months of recruitment, a total of 82 normal volunteers (43 in 20 to 40 age group and 39 in 41 to 60 years age group) were recruited.

A total of 66 cases of migranous vertigo (31 in 20 to 40 years age group and 35 in 41 to 60 years age group) were recruited, against the target of 72.

NORMAL VOLUNTEERS

GENDER DISTRIBUTION:-

There were 30 females (36.5%) and 52 males (63.5%), among females, 16(53.3%) were within age ranging between 20 to 40 years and 14(46.6%) among those belonging to the age limit of 41 to 60 years. Among males, 29(58 %) were 20 to 40 years and 23(42%) were 41 to 60 years.

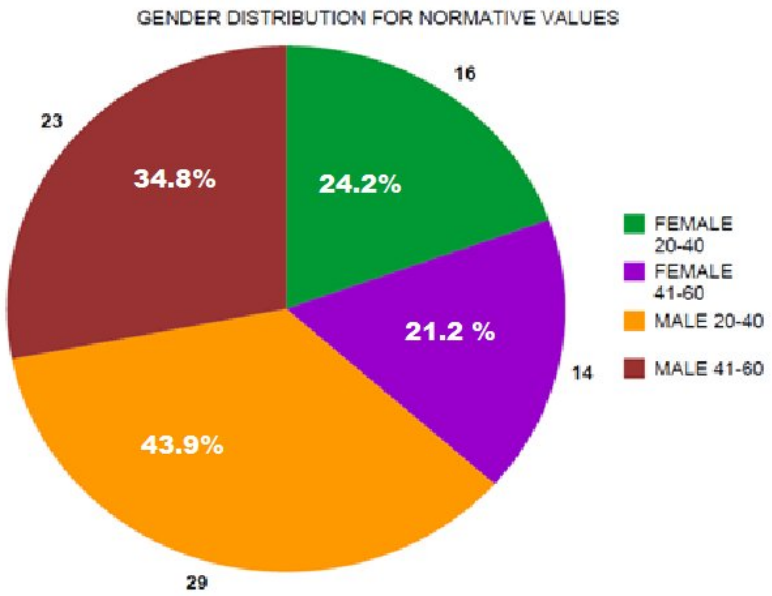

Figure 14:-Pie chart showing age wise groups and corresponding gender distribution

AGE DISTRIBUTION OF THE VOLUNTEERS:-

Average age of group 20-40 years was 28.17 years with the minimum age being 21 years and the maximum age being 38 years in the group.

Average age of group 41-60 years was 48.24 years with the minimum age being 41 years and the maximum age being 60 years.

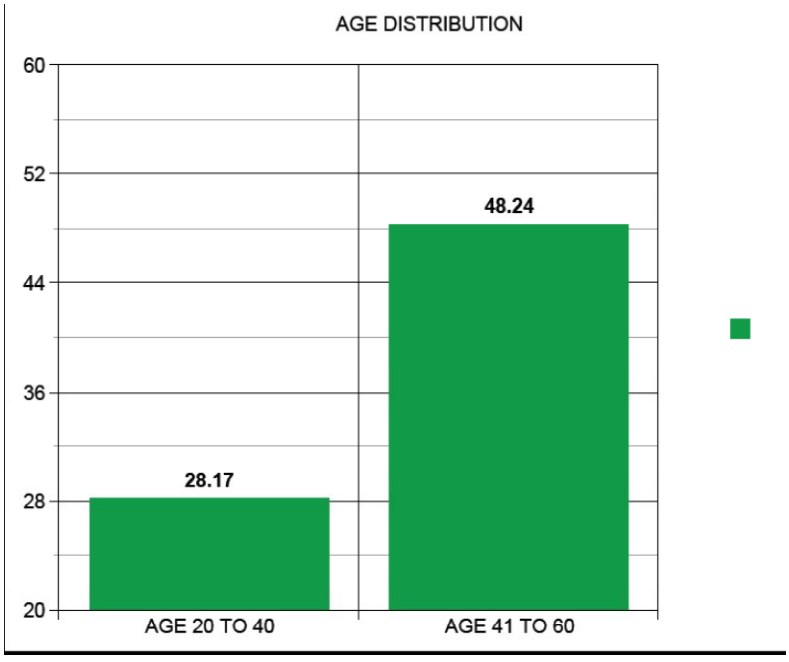

Figure 15: -Bar diagrams showing mean age for normal volunteers recruited.

NORMATIVE VALUES -SVV AND SVH (both static and dynamic)

The normative values of SVV and SVH were calculated after taking the average of 6 readings irrespective of the arithmetic sign as to whether the value was positive or negative. The SVV and SVH values were categorised as per the age and gender groups. The mean values, standard deviation and standard error of SVV and SVH both in dynamic and static tests are depicted below in the table:

	SEX GROUP	N	Mean Value In degrees	Std. Deviation	Std. Error Mean
STATIC-VERTICAL	FEMALE	30	1.4160	.67591	.12340
	MALE	52	1.5756	.70719	.09807
DYNAMIC-VERTICAL	FEMALE	30	1.8150	.63945	.11675
	MALE	52	2.0434	.65027	.09018
STATIC-HORIZONTAL	FEMALE	30	1.6275	.76425	.13953
	MALE	52	1.6507	.83814	.11623
DYNAMIC-HORIZONTAL	FEMALE	30	1.8037	.59989	.10953
	MALE	52	2.0958	.85861	.11907

Table 1:- Showing normative values of SVV and SVH (dynamic and static)

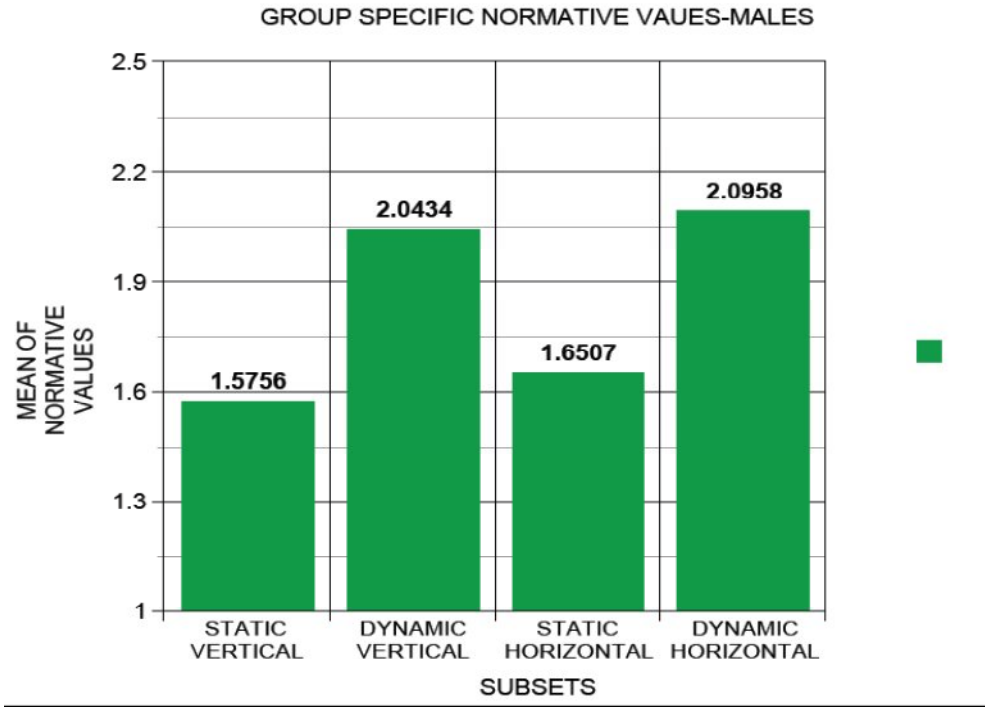

Figure 16:-Bar diagram representing the normative values in males.

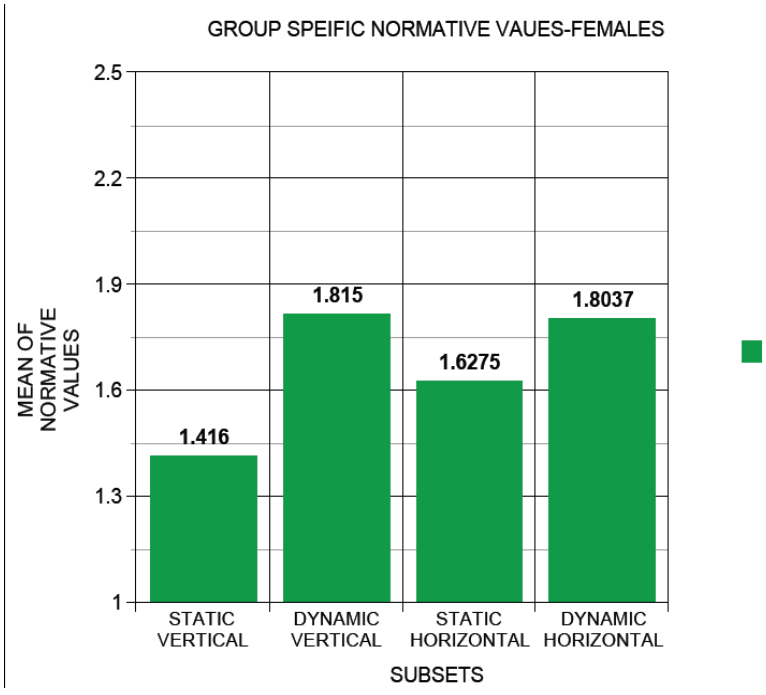

Figure 17:-Bar diagram representing the normative values in females.

Overall normative values for SVV and SVH for both dynamic and static (both age and gender groups). The values are depicted below in the table:-

ENTITY	MEAN VALUES
STATIC-VERTICAL	1.4958
DYNAMIC-VERTICAL	1.9292
STSTIC-HORIZONTAL	1.6391
DYNAMIC –HORIZONTAL	1.9497

Table 2 –showing overall normative values for dynamic and static SVV/SVH

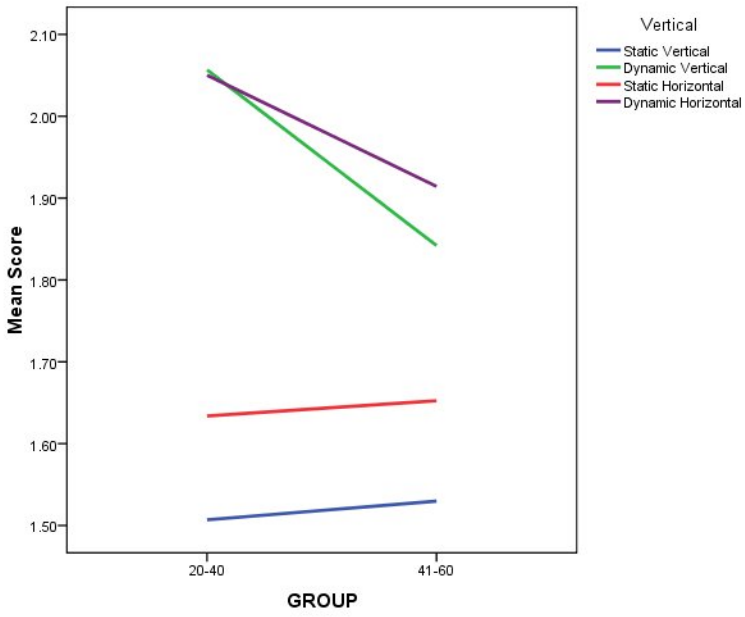

Figure 18: - graph showing the normative values in the two age groups, for males and females shown together.

MIGRANOUS VERTIGO

GENDER DISTRIBUTION OF MIGRANOUS VERTIGO PATIENTS

Out of the 66 patients diagnosed with Migranous vertigo, 26 patients were male (39.9%) and 40 patients were females (60.1%). Out of the 26 male patients, 16 (61.5%) belonged to age ranging within 20 to 40 years and 10 (38.5%) belong to 41 to 60 years of age.Simillarly out of the 40 female patients, 21 (52.5%) were in the age group of 20 to 40 years and 19 (47.5%) belong to 41 to

60 years of age.

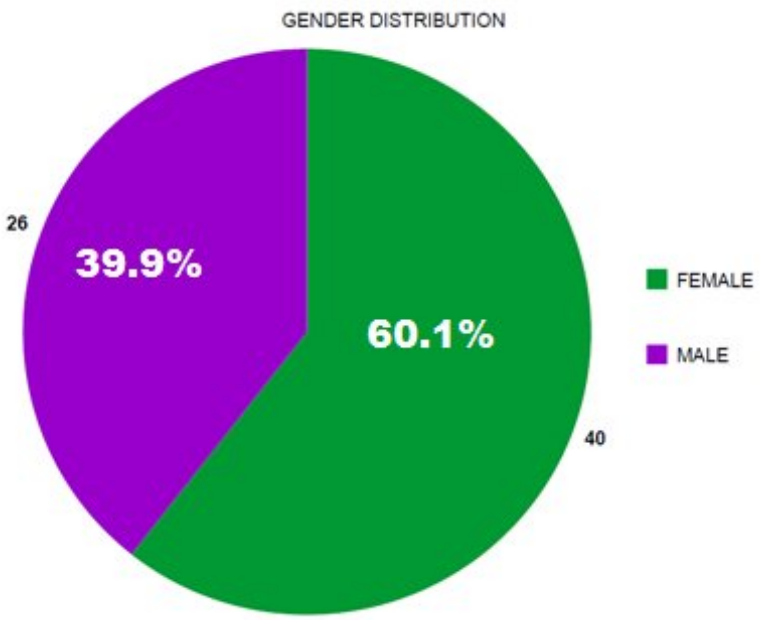

Figure 19:-Pie chart showing gender distribution profile

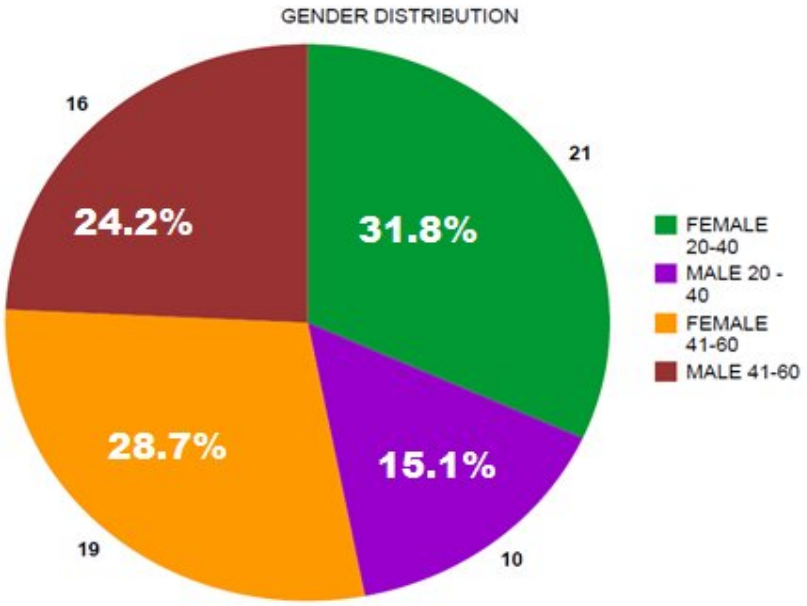

Figure20:-Pie chart shows profile of gender distribution according to age wise groups.

AGE DISTRIBUTION IN MIGRANOUS VERTIGO PATIENTS:-

The average age of the group of 20-40 year old was 29.71years, with the age ranging from 20to 38 years. Similarly the

The average age of group 41-60 years was 48.66 years with the age ranging from 41 years to 60 years.

The overall average age of presentation was 39.76 years.

FAMILY HISTORY PROFILE IN MIGRANOUS VERTIGO PATIENTS:-

46 patients out of 66 (69.6%) had a positive history of migrane headache in their family. The remaining 20 had no contributing family history.

DURATION OF HEADACHE:-

The duration of headache was assessed for these patients diagnosed with Migranous vertigo and was stratified by age groups.

The mean duration of headache was approximately 4.3years.Among those belonging to the age limit of 20 to 40 years, the minimum duration of headache was 1 month and maximum of 10 years. The mean duration of headache in this group was 3 years.

Similarly in patients diagnosed with migranous vertigo belonging to the age limit of 41 to 60 years, the minimum duration of headache was since 4 months and maximum of 30 years. The mean duration of headache in this group was 5.5 years.

GROUP	Mean	Median	N	Minimum	Maximum	Std. Deviation
20-40	3.06290	2.00000	31	.100	10.000	2.811493
41-60	5.53571	3.00000	35	.250	30.000	7.624010
Total	4.37424	3.00000	66	.100	30.000	5.966478

Table 3:-Table depicting the profile of duration of headache in years.

DURATION OF VERTIGO EPISODES IN MIGRANOUS VERTIGO PATIENTS

The patients diagnosed with Migranous vertigo had the episodes of vertigo lasting from momentary to a few hours. For statistical analysis these were categorized into five groups based on the duration (see Table 5). The majority had vertigo lasting for less than 5 minutes

Minutes	Frequency	Percent
<5	28	42.4
6-10	12	18.2
11-30	13	19.7
31-60	9	13.6
>1 hr	4	6.1

Table 4: - Profile showing duration of vertigo in MV patients

The statistical difference between values of SVV and SVH for different durations of vertigo was found to be insignificant using kruskal Wallis test.

AURA IN PATIENTS WITH MIGRANOUS VERTIGO:-

Among those with aura, 53 (80.3%) out of 66 patients presented with feeling of aura like phonophobia and photophobia. In remaining of 13 patients there was no appreciation of aura and features like phonophobia and photophobia.

HEARING LOSS AND TINNITUS PROFILE IN PATIENTS WITH MIGRANOUS VERTIGO:-

Out of the total of 66 patients 9 (13.6%) and 14 (21.2%) complained of hearing loss and tinnitus respectively.

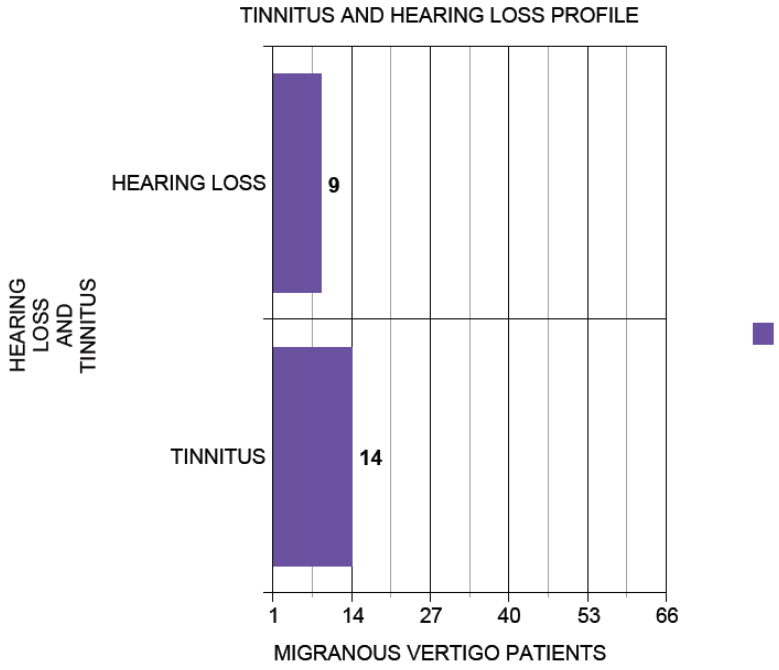

Figure 21:-Showing hearing loss and tinnitus profile in MV patients.

TYPE OF VERTIGO IN MIGRANOUS VERTIGO PATIENTS:-

The patient had varied history of vertigo extending from surrounding rotatory vertigo in a majority to head rotatory vertigo in few, and rarely both types together.54 patients complained of SRV(**81.8%**), 9 patients had HRV(**13.6%**) and 3(**4.5%**) had both SRV and HRV.The SVV and SVH values were similar in both SRV and HRV patients.

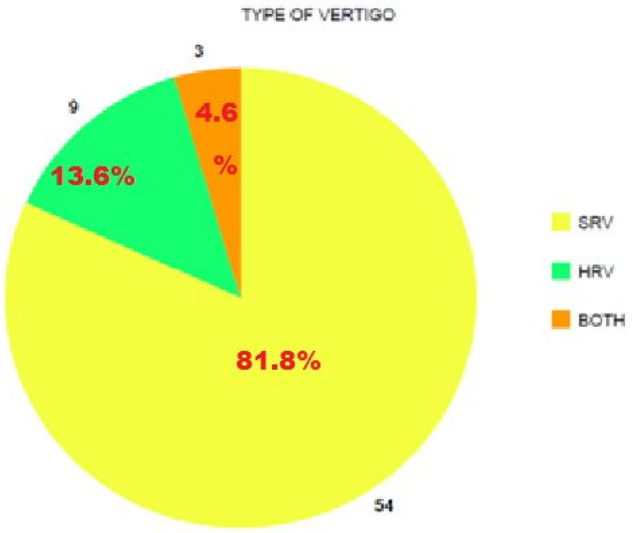

Figure 22:-Pie chart showing distribution of type of vertigo in MV patients

COMORBIDITIES IN ATIENTS WITH MIGRANOUS VERTIGO:-

A significant proportion of the patients had co morbidities present in the Migranous vertigo group.17 out of 66 (**25.7%**) had various co morbidities present.

These included dyslipidemia, diabetes mellitus, hypertension, hypovitaminosis and a few of them had hypothyroidism. A few of these also had a combination of the above said co morbidities.

AUDIOMETRIC PROFILE :-

Pure tone audiometry was performed as a routine test for any patients diagnosed as Migranous vertigo. The average of PTA was calculated and the profile was studied.

The patients whose age was within 20 to 40 years had an average of 18.71 db values for the right ear and 13.31 db for the left years.Simillarly the patients whose age was within 41 to 60 years had an average of 19.86 db values in right ear and 20.41 db in left ear (Table 5).

AGE GROUP		RIGHT EAR	LEFT EAR
20-40	Mean	(db)18.71	(db)13.39
	Median	15.00	15.00
	Number of participants	31	31
41-60	Mean	19.86	20.14
	Median	15.00	15.00
	Number of participants	35	35

Table 5:-showing mean and median values of pure tone audiometry with respect to age and side

ELECTRONYSTAGMOGRAM (ENG) PROFILE OF MIGRANOUS VERTIGO:-

ENG was a part of the tests done for audiovestibular assessment of cases diagnosed with migranous vertigo in majority of patients. It was observed that 47 patients underwent the ENG test and 21 out of these were normal study's whereas 26 patients had abnormal findings on ENG.

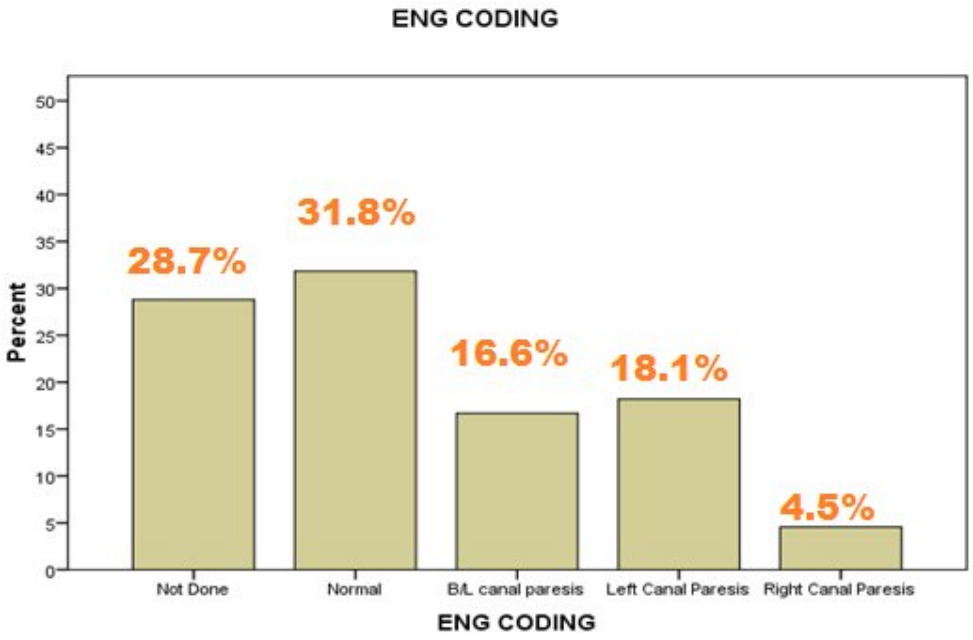

Figure 23:-Bar chart demonstrating distribution of ENG profile

IMAGING PROFILE:-

A significant proportion of the Migranous vertigo patients underwent imaging such as MRI brain to rule out any secondary pathology in the brain that may be responsible for the symptoms. Thirty six patients (54.5 %) out of the 66 underwent imaging and 8 (12%) had important findings on the MRI.

These findings ranged from compression at cervical vertebra, vascular loops, lacunar infarcts, thecal compression, disc degenerative changes and empty spot with bright spot in pituitary.

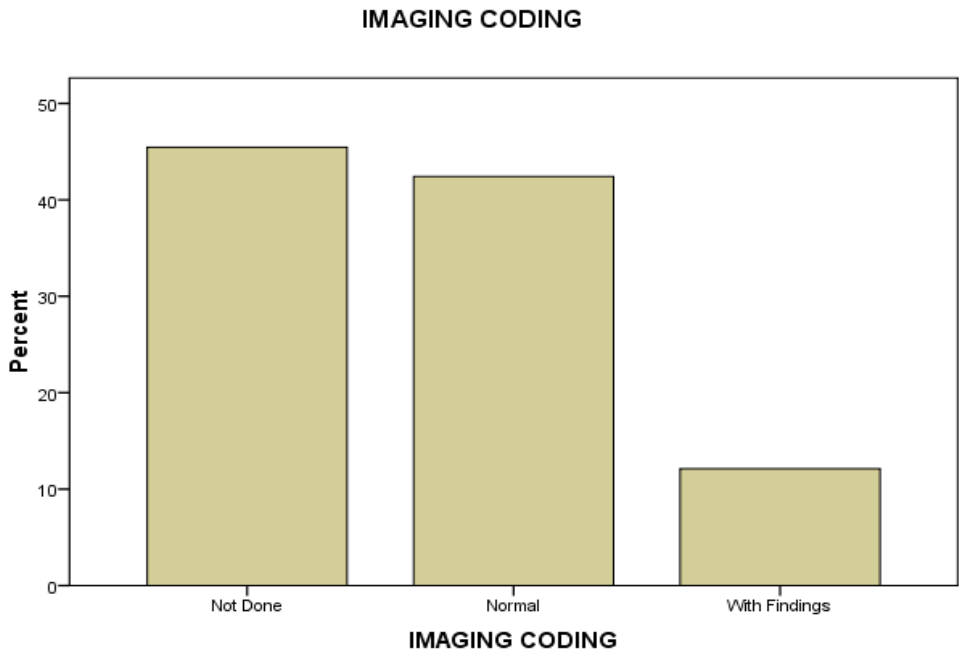

Figure 24:- Imaging and abnormalities identified

SVV and SVH values in diagnosed cases of Migranous vertigo

The values of SVV and SVH were calculated in diagnosed cases of Migranous vertigo after taking the average of 6 readings irrespective of the arithmetic sign as to whether the value was positive or negative. The SVV and SVH values were categorised as per the age and gender groups. The mean values of SVV and SVH both in dynamic and static aspects are depicted below in the table:

	GROUP	N	Mean Value in degrees	Std. Deviation	Std. Error Mean
STATIC-VERTICAL	**20-40**	31	**1.3548**	.80722	.14498
	41-60	35	**1.5580**	.92062	.15561
DYNAMIC-VERTICAL	**20-40**	31	**2.9923**	1.52971	.27474
	41-60	35	**3.0911**	1.93439	.32697
STATIC-HORIZONTAL	**20-40**	31	**1.5600**	1.08636	.19512
	41-60	35	**2.0934**	1.09647	.18534
DYNAMIC-HORIZONTAL	**20-40**	31	**3.0568**	1.57519	.28291
	41-60	35	**3.4637**	1.88851	.31922

Table 6: Showing values calculated for static and dynamic SVH/SVV for migranous vertigo patients.

NPAR test was employed to evaluate the overall mean values of SVV and SVH in patients diagnosed as migranous vertigo. The result of which is depicted in the table 7 below:

ENTITY	N	Mean	Std. Deviation	Minimum	Maximum
STATIC-VERTICAL	66	1.4626	.86862	.25	5.50
DYNAMIC-VERTICAL	66	3.0447	1.74349	.83	10.30
STATIC-HORIZONTAL	66	1.8429	1.11603	.30	5.00
DYNAMIC-HORIZONTAL	66	3.2726	1.74717	.60	10.60

Table 7: - Gross overall split up of mean values of SVV and SVH in Migranous vertigo patients

	Group							
	20 - 40			p- value	41 - 60			p-value
	Mean	SD	N		Mean	SD	N	
Static Vertical:								
NORMATIVE	1.50	0.68	45	0.38	1.53	0.72	37	0.88
MIGRANOUS	1.35	0.81	31		1.56	0.92	35	
Dynamic Vertical:								
NORMATIVE	2.06	0.66	37	0.001	1.84	0.63	37	0.0004
MIGRANOUS	2.99	1.53	31		3.09	1.93	35	
Static Horizontal:								
NORMATTIVE	1.63	0.90	45	0.76	1.65	0.68	37	0.04
MIGRANOUS	1.56	1.08	31		2.09	1.09	35	
Dynamic Horizontal:								
NORMATIVE	2.05	0.78	45	0.0005	1.91	0.79	37	<0.001
MIGRANOUS	3.05	1.57	31		3.46	1.89	35	

TABLE 8:- showing age wise split up and comparison of SVV and SVH

Dynamic values including both dynamic vertical and dynamic horizontal were significantly higher among migranous patients when compared to the normal value. Mann Whitney test was used to calculate the statistical correlation between the dynamic and static values of SVV and SVH. The p values are depicted in the table 9 below:-

TEST	20-40 years p- value	41-60 years p-value
Static Vertical:	**0.38**	**0.88**
Dynamic Vertical:	**0.001**	**0.0004**
Static Horizontal	**0.76**	**0.04**
Dynamic Horizontal:	**0.0005**	**<0.001**

Table 9:-Showing the various 'p'values of the SVV/SVH tests It was also observed that the difference between migranous patients and normal was significantly different in the age group of 41 to 60 years for static horizontal values.

DISCUSSION:-

Many tests have been suggested for the use of the use of SVV in clinical practice. We have used a computer software based SVV equipment (MUS_VS-V1.3.2.Rev B) Synapsis company) which had a remote controlled potentiometer for recording the SVV and SVH values. The specific requirements were met as per requirement for conducting the SVV and SVH tests. The test was easy and enjoyable to use by the patients and was in accordance with other studies which was conducted previously (108)(48).

The values that we calculated was around 1.5 degrees for static SVV, 1.9 degrees for dynamic SVV and 1.6 degrees for static SVH and 1.9 degrees for dynamic SVH. These values were in accordance for the other studies worldwide. Earlier studies have suggested the normal values of static SVV and dynamic SVV to range from +/- 1.5 to 3.0+/- degrees. The various studies are enlisted below with the approximate values of the deviations calculated.

Studies done by Akin & Murnane in 2009; Bohmer in 1999; Friedmann in 1970; Murray, et al.in 2007; Tabak, et al. in 1997; and Vibert& Häusler in 2000 have suggested the values to be +/-2.0 degrees)(3).

Similarly studies done by Tribukait, et al. in 1996; Tribukait, & Bergenius in 1998; Tribukait, & Eiken in 2005; and Tribuikait, et al. in 2004 has proposed the values to be around +/- 2.5 degrees)(117).

Values as high as +/- 3.0° has been reported by Hafstrom, et al. in 2004 and Karlberg, et al. in 2002)(118).

Our values of SVV and SVH were well between the two extremes of values reported. This also reinforced that the methodology and the SVV equipment used in our study were valid.

Pavan et al had studied 30 normal volunteers (23 females and 7 males) for analysing the normative values in these individuals. The values for static SVV was found to be -0.372 +/- 1.21 degrees, clockwise dynamic SVV 1.53+/-1.80 degrees and for counter clockwise Dynamic SVV -1.11+/-2.46 degrees. Computer based software was used for the above study which was very similar to the test used in our study(48).

A similar study done by Griffin et al studies 25 individuals (15 males and 10 females) with an average age of 44.6 year. They used a rotational chair with SVV led light array based test. They concluded that the static values were around 2 degrees and dynamic values were approximately 3 degrees(24). Akin et al also found same values when he evaluated 24 normal volunteers for SVV and SVH values using ultracentrifugation tests.

Literature search showed very few articles on assessment of normative values for SVV and SVH and more importantly there seemed to be none for Indian population. The study group size of the above mentioned studies was also significantly much smaller when compared to our study.

Literature search showed only one study where stratification of SVV and SVH values has been done based on age groups(52). These normative values acquired in our study could serve as a reference values for application of this test for further studies and as a part of application of this tests in other clinical setting. . These values may help us in future evaluation of patients with various vestibular pathologies involving the utricular and frontal pathways.

MV is generally found more commonly in females than males(103,108).As per the reported literature MV can occur at any age(103). It has also been reported that the migranous vertigo patients are usually associated with aura and have a positive family history(83)(103,119,120). Our study also showed a female preponderance (60.1% females). Family history of migrane or chronic headache was found in 69.6 % of our subjects and aura was reported in 80.3 % of the MV patients in our study.

There are studies done which suggest that the values of SVV/SVH increase as the age of individuals increases. This was attributed to decreasing visual acuity and age related changes in the vestibular system(51). However we also observed SVV or SVH values were not significant between various age groups upon stastical analysis. This may be attributed to the differences in the demographical profile, variation in lifestyle and also to the fact that the maximum age limit for our study was upto 60 years only.

Many studies have reported abnormal findings in Ménière's Disease(114), vestibular neuritis(121), Gentamycin toxicity (122)and after Stapedectomy(123).

Migrainous vertigo is the 2nd most common cause of giddiness (124). Patients can present with a spectrum of symptoms from spontaneous room spinning vertigo, positional vertigo or non specific giddiness and symptoms may lasts for seconds to days(108). In our study, a majority of patients diagnosed with migranous vertigo had surrounding rotatory vertigo which were recurrent episodes and lasted less than 1 hour and majority of them lasted for few minutes. This was similar other studies where MV patients presented with spontaneous recurrent episodes of true vertigo lasting for less than 1 hour(115).

We also observed that there existed a female preponderance (60.6 %) among those who were diagnosed with Migranous vertigo. The average age of these patients was 39.7 years. These two finding are comparable with the study done by Augusto P. Casani et all in 2009(115).The attacks of vertigo were of shorter duration in younger patients when compared to the older persons(115).

Casani et al(115) found some central vestibular findings on ENG testing. In our study 26 out of 47 patients (57.7 %) tested had abnormal ENG findings. However in our study the findings were mainly bilateral and unilateral hypofunctioning caloric responses. Similar results were done by Bir et al where the ENG findings were compared in case with and without vertigo. He reported as high as 58 % abnormal ENG results in migranous vertigo patients(125).

We observed a very important difference between the SVV and SVH values in migranous vertigo patients. The dynamic SVV and SVH values were significantly increased in these patients. When compared to the normative values that we calculated ($p < 0.5$). This finding is in keeping with the presently accepted pathoetiology of migrainous vertigo of ion channel abnormality (126) and involvement of the brainstem pathways (127). The static SVV values were not different from the normal which implies that tonic vestibular compensation may have been achieved in these patients during the interictal phase (as these tests are being done during symptom free periods). However the fact that there is a significant difference in the static SVH and dynamic SVV and SVH suggests that the tonic vestibular horizontal and dynamic compensation for the utricular pathway defects have not been achieved in these patients. These abnormalities may also explain some of the symptoms in some of these patients in spite of other normal otoneurological examination and testing.

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Migrainous vertigo is known to be the great ‘mimicker ” of all diagnosis of vertigo(128).When the presentation is typical diagnosis is obvious. However when the presentation is vague SVV and SVH could provide important diagnostic information towards management of this very common but morbid condition.

-: CONCLUSIONS:-

NORMAL VOLUNTEER GROUP

1. The normative values for static and dynamic SVV / SVH are as follows:-

Static vertical:-1.49 degrees

Dynamic vertical:-1.92 degrees

Static horizontal:-1.63 degrees

Dynamic horizontal:-1.94degrees

2. There was no statistically significant difference between the two age groups (20-40 years and 40-60 years) and their corresponding SVV and SVH values.

3. There were no male female differences that were statistically significant between the males and females and SVV and SVH values.

4. The normative values were comparable with those of the western literature.

MIGRANOUS VERTIGO GROUP:-

5. The average age of presentation for migranous vertigo patients was 29.7 and 48.6 in the respective age groups of 20-40 years and 40 to 60 years. The overall mean age was around 39.7 years.

6.The values for the subjective visual vertical and horizontal in both static and dynamic aspects for patients diagnosed with Migranous vertigo were as follows:-

Static vertical:-1.46 degrees

Dynamic vertical:-3.02 degrees

Static horizontal:-1.87 degrees

Dynamic horizontal:-3.24 degrees

7. The above values of dynamic SVV and dynamic SVH were significantly different when compared to the normative values.

8. Static SVH values were statistically significant in the age group of 40-60 years when compared to the corresponding normative values.

9. The values of dynamic SVV and dynamic SVH were not different among the two age groups and males/females upon statistical analysis in patients of migranous vertigo.

10. There was a female preponderance among patients with migranous vertigo.

11. Of 36 patients of migranous vertigo who had imaging, 8 had abnormalities that are considered non-specific by radiologists.

12. Electronystagmogram was done for 47 patients and 21 out of these were normal study whereas 26 patients had abnormal findings on ENG which is a significant finding suggesting involvement of vestibular pathways in \ patients diagnosed as Migranous vertigo.

13. The episodes of vertigo had durations lasting from as less as to a minute to hours. However, a majority of the patients complained of vertigo which was surrounding rotatory (81.8%) and lasting for few minutes (42.4 % patients complained of episodes lasting less than 5 minutes).

14. A significant proportion of the patients diagnosed with Migranous vertigo had associated aura like phonophobia and photophobia (80.3 %).

17. SVV and SVH can be an important test that can be added to the list of audiovestibular tests available for migranous vertigo patients.

LIMITATION OF THE STUDY

1) Due to shortage of time we were unable to recruit the targeted 36 patients for 1 arm in the Migranous vertigo category.

2) The educational status of the various patients was not uniform, on one hand the younger age group found getting oriented to remote controlled potentiometer was very easy and interesting where as the older age group patients generally took more time for getting oriented to the remote controlled potentiometer.

3) Due to financial reasons some of the Migranous vertigo patients did not undergo imaging like MRI brain.

4) As we did not include volunteers whose age was less than 20 years, therefore the normative data value shall be not applicable to people who are less than 20 years of age.

CONTRIBUTION OF THIS STUDY

1. To our knowledge this is the first study on normative values for SV and SVH for Indian population.

2. This study suggested to the fraternity treating Migranous vertigo patients that SVV and SVH can serves as an additional test in the existing battery of audiovestibular tests.

3. Abnormalities in SVV and SVH may explain some of the symptoms in patients whose otoneurological examination is normal but complain of vertigo.

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PATIENT INFORMATION SHEET

STUDY TITLE- Normative data for subjective visual vertical test in Indian population

Subjective visual vertical (SVV) is a recent test for investigating people with giddiness since after routine examination, the cause of giddiness may not be clear and SVV test gives additional information that helps the doctor identify the probable cause of giddiness.

Giddiness is often caused by a problem in the balance organ in the ear and SVV test can assess that. This is a very simple test and takes 10 to 15 minutes to complete.

What do you have to do?

You will be asked to answer a questionnaire and undergo a detail Ear, Nose and Throat examination.

Then you will have to do the SVV This test is a simple and does not involve any injection or instrument that will be pushed in your ear. You will be given a joystick that looks like a computer game that children often play and you will have to use the joystick to make a line displayed on the screen as perfectly straight vertically and horizontally.

This test is routine and will not result in extra cost or harm you in any way.

The benefit of this test is that it will indicate to the doctor if any part of your balance system in the ear is the cause of your giddiness.

Your participation in this study is voluntary and you are free to withdraw at any time, without giving any reason and your withdrawal will not affect any treatment you or your relatives receive in this hospital.

There is no extra risk for you due to your participation in this study since our treatment for you will not be influenced by this test. There is absolutely no additional cost to you as a result of participation in this study.

If you are willing to participate in this study, you will be required to sign in the following consent form.

Contact person

Dr Gaurav Ashish

Dept of ENT, CMC, Vellore

Informed Consent Form to participate in an observational study

Study Title- Normative data for subjective visual vertical test in Indian population

Study number:

Subject's Initial:

Subject's Name:

Date of Birth/Age:

Please put your signature here

(**Subject)**

1. I confirm that I have read and understood the information sheet dated for the above study and have had the opportunity to ask questions.

2. I understand that my participation in the study is voluntary and that i am free to withdraw at anytime, without giving any reason, without my medical care or legal rights being affected.

3. I understand that sponsor of the clinical trial, others working on the sponsor's behalf, the ethics Committee and the regulatory authorities are free to look at my health records both in respect of the current study and any further research that may be conducted in relation to It, even if I withdraw from the trial, for scientific purposes only. However, I understand that my identity will not be revealed in any information released to third parties or published.

4. I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s)

5. I agree to take part in this study.

Signature (or thumb impression) of subject/Legally acceptable representative:

Date:

Signatory's Name: ----------

Signature of the Investigator: ----------

Study investigator's name: ----------

Signature of the witness: ----------

Name of witness: ----------

-: DATA RETREIVAL SHEET:-

Serial Number: Group-A/B

Name:

Age:

Sex: Hospital number:

Occupation: Contact number:

Address: Email id:

Date of interview-

Handedness:

Any refractory errors:

Hearing loss-yes/no

Tinnitus-yes/no

Ear discharge- yes/no

Giddiness/lightheadness/head spinning/imbalance- yes/no

Type of vertigo:-SRV/HRV/BOTH

Duration of vertigo:-

History of ototoxic drugs- yes/no

History of headache- yes/no

Duration of headache:-

Any photophobia/photophobia-Yes/No

Neurological signs if any?

History of head trauma-Yes/no

Diabetes/Hypertension/Hypothyroidism/Dyslipidemia: yes/no

Any medications taking-

Any significant family history-

Any h/o LOC/Seizures-yes/no

Any Palpitations-yes/no

Any syncopal attacks-Yes/no

Any tingling /numbness/weakness of any limb-Yes /no

Any past ENT/Brain surgeries-yes/no

EXAMINATION-

EAR EXAMINATION

Tympanic membrane- (R) ---------- (L) ----------

Tuning fork tests

Rhinnes (R) ---------- (L) ----------

Webers (R) ---------- (L)

Fistula sign: +/-

EYE SIGNS-

Squint: yes/no

Extra ocular movements: normal/restricted

Nystagmus: yes/no

Saccades: yes/no

Smooth pursuit: yes/no

Head thrust test: yes/no

CEREBELLAR SIGNS

Finger nose test

Finger nose finger test

Dysadidokinesia

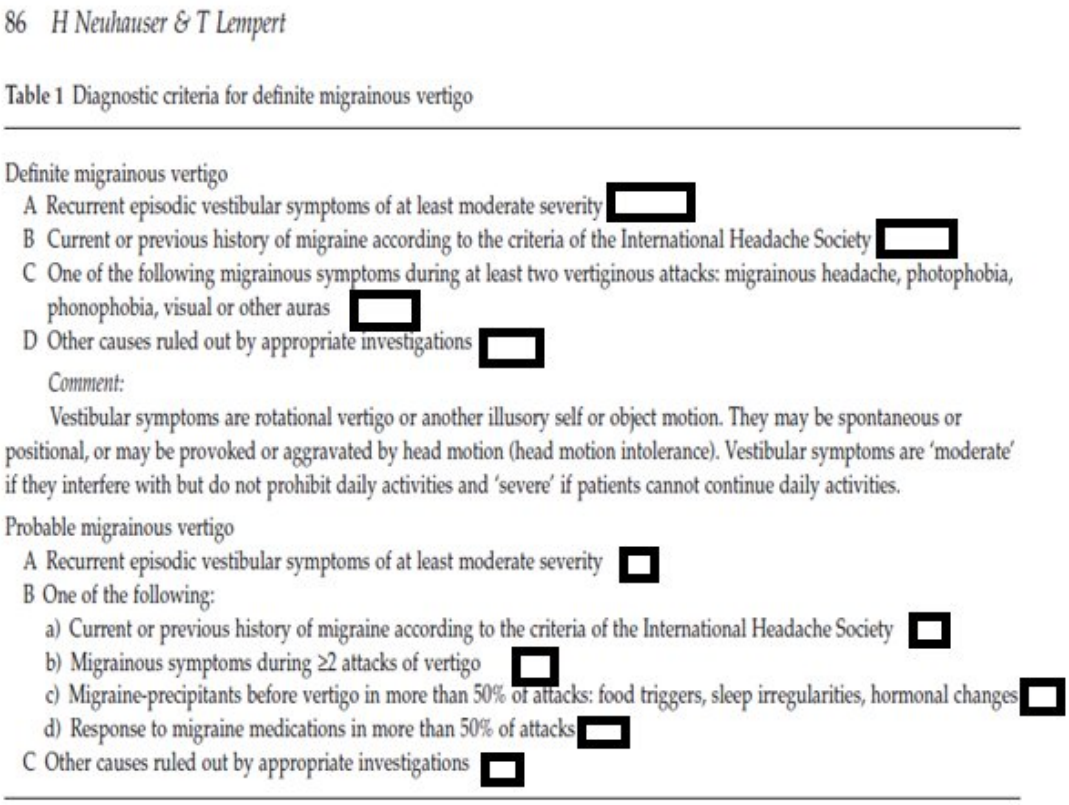
Rhombergs test

GAIT:

DIX HALLPIKES TEST:

CRITERIA FOR MIGRANOUS VERTIGO

Tick against all the criteria met:-



86 *H Neuhauser & T Lempert*

Table 1 Diagnostic criteria for definite migrainous vertigo

Definite migrainous vertigo
- A Recurrent episodic vestibular symptoms of at least moderate severity ☐
- B Current or previous history of migraine according to the criteria of the International Headache Society ☐
- C One of the following migrainous symptoms during at least two vertiginous attacks: migrainous headache, photophobia, phonophobia, visual or other auras ☐
- D Other causes ruled out by appropriate investigations ☐

Comment:

Vestibular symptoms are rotational vertigo or another illusory self or object motion. They may be spontaneous or positional, or may be provoked or aggravated by head motion (head motion intolerance). Vestibular symptoms are 'moderate' if they interfere with but do not prohibit daily activities and 'severe' if patients cannot continue daily activities.

Probable migrainous vertigo
- A Recurrent episodic vestibular symptoms of at least moderate severity ☐
- B One of the following:
  - a) Current or previous history of migraine according to the criteria of the International Headache Society ☐
  - b) Migrainous symptoms during ≥2 attacks of vertigo ☐
  - c) Migraine-precipitants before vertigo in more than 50% of attacks: food triggers, sleep irregularities, hormonal changes ☐
  - d) Response to migraine medications in more than 50% of attacks ☐
- C Other causes ruled out by appropriate investigations ☐

- Whether imaging {CT BRAIN/MRI} done—Yes/no

Any abnormal findings on Imaging—

- ENG Study: - {if done}

- PTA average for right and left ear:-.....db/.....db

- Impedance audiometry:-

OFFICE OF RESEARCH
INSTITUTIONAL REVIEW BOARD
CHRISTIAN MEDICAL COLLEGE,
BAGAYAM, VELLORE 632002, TAMIL NADU, INDIA

Ref: FG/8411/08/2013

September 13, 2013

The Treasurer
Christian Medical College,
Vellore.

Dear Mr. Denzil,

Sub: **Fluid Research grant project:**
To establish normative data in an Indian population for static and dynamic subjective visual vertical and Horizontal (SVV & SVH) examination and to determine the role of SVV & SVH in evaluation of patients with migrainous vertigo.
Dr. Gaurav Ashish, PG Registrar, ENT IV, Dr. Achamma Balraj, Dr. Anjali Lepcha, Dr. Amit Kumar Tyagi, ENT.

Ref: IRB Min. No. 8411 dated 13.08.2013

The Institutional Review Board at its meeting held on August 13, 2013 vide IRB Min. No. **8411** accepted the project *for a total sum of* Rs. 79,900/- (Rupees Seventy Nine Thousand Nine Hundred only) will be granted for 2 years. *If overspent the excess should be debited form the respective departmental or Special funds.* Kindly arrange to transfer the sanctioned amount to a separate account to be operated by Drs. Gaurav Ashish and Achamma Balraj.

Yours sincerely,

Dr. Nihal Thomas
Secretary (Ethics Committee)
Institutional Review Board

Dr Nihal Thomas
MBBS MD MRCPS DNB (Endo) FRACP(Endo) FRCP(Edin)
Secretary (Ethics Committee)
Institutional Review Board

CC: Dr. Gaurav Ashish, ENT IV, CMC
Dr. Achamma Balraj, ENT IV CMC
File

Normative group

NAME	AGE	SEX GROUP	SEX	STATIC-VERTICAL	DYNAMIC-VERTICAL	STATIC-HORIZONTAL	DYNAMIC-HORIZONTAL	GROUP
GLADYS	47	0	FEMALE	0.42	1.38	0.73	0.97	B
HOSSANA	43	0	FEMALE	0.62	1.22	1.03	0.81	B
DILLIBAI	43	0	FEMALE	0.67	2.03	0.72	1.55	B
LALMUKHUNIA	55	1	MALE	0.7	1.52	0.36	1	B
KANCHAN MALA	45	0	FEMALE	0.43	3.19	2.08	1.9	B
SUNAM NOURULA	46	0	FEMALE	2.13	1.4	1.65	2.31	B
SAMSON	45	1	MALE	0.33	2.53	1.65	1.48	B
SOM DUTT	41	1	MALE	1.21	1.98	0.53	1.98	B
REKHA SHARMA	45	0	FEMALE	1.36	2.98	2.93	2.2	B
GHAZI	60	1	MALE	2.58	1.61	2.06	1.81	B
DHANDAPANI	46	1	MALE	0.85	1.4	1.61	1.43	B
ANTARA GIRI	42	0	FEMALE	1.58	2.38	1.5	1.26	B
ALICE DHAMODHARAN	42	0	FEMALE	1.01	0.8	1.51	1.21	B
SEKHARAN	56	1	MALE	1.4	2.71	1.21	2.81	B
KARTUKEYAN	46	1	MALE	0.93	1.6	1.48	2.3	B
MAYEYUNISSA	51	0	FEMALE	2.25	0.66	2.33	2	B
PIYUSH KUMAR	44	1	MALE	2.85	2.83	2	2	B
SOUMEN	55	1	MALE	2.16	2.16	2.33	2.42	B
ABHA DEVI	50	0	FEMALE	2.71	2.44	2.66	1.83	B
KANTHI NATH DUBEY	55	1	MALE	1.66	1.66	3.14	2	B
FRANCIS	55	1	MALE	3.14	1.5	1.88	2.66	B
PRAKASH KUMAR	42	1	MALE	2	1.77	2	2.28	B
HEMANT KUMAR	54	1	MALE	2.16	2	1.14	2.71	B
KEMIN GEORGE	44	0	FEMALE	2.57	1.66	1.5	1.33	B
HASINA BEGUM	59	0	FEMALE	1.42	1.71	1.14	1.66	B
ANSHU JAIN	44	1	MALE	1.57	1.37	1.85	1.33	B
KIRTHI	52	1	MALE	1.83	1.5	1	1	B
RAMAN K	58	1	MALE	1	1.16	1.16	1.83	B
KESHAWANI	50	0	FEMALE	0.83	1	1.42	1.5	B
SALIMULA	46	1	MALE	1.71	1.3	2	1.83	B
JOSHUA	43	1	MALE	1.46	2.81	2.86	2.12	B
RADHIKA	44	1	MALE	1.571	1.375	1.85	1.33	B
GIRIJESH	44	1	MALE	1.48	1.52	2.01	2	B
SUHAZ MONDAL	53	0	FEMALE	2	2	2.13	2	B
DANIEL PAUL	45	1	MALE	1.33	2.81	1.23	5.52	B
GANESH	45	1	MALE	1.15	2.03	0.46	2.46	B
GIRIJESH	50	1	MALE	1.53	2.16	2.01	2	B
DEVAKI	28	0	FEMALE	0.98	1.3	1.23	1.76	A
HANNA JOLLY	21	0	FEMALE	0.66	2.44	1.38	2.33	A
MONIRUL HAQ	27	1	MALE	2.15	1.98	1.76	2.01	A
KALI DAS	32	1	MALE	1.58	1.68	1.75	1.12	A

SAMPATH	38	1	MALE	0.33	0.93	0.68	1.6	A
AYESHA	21	0	FEMALE	1.9	1.43	0.46	1.58	A
INBA CHARLES	31	1	MALE	0.8	2.15	0.46	2.2	A
THILIGAWATHY	28	0	FEMALE	1.48	3.12	0.96	2.43	A
ZALAL	33	0	FEMALE	1.11	2.15	0.81	1.98	A
PRASHANT KUMAR	22	1	MALE	0.75	4.36	2.21	4.2	A
RANJIT	25	1	MALE	2.13	2.16	3.2	1.75	A
BABY RADHIKA	34	0	FEMALE	0.8	1.98	1.43	3.2	A
PRATIK SAMPATH	21	1	MALE	1.03	1.31	1.33	3.02	A
DURAI VELU	30	1	MALE	1.32	2.07	0.33	2.43	A
KARTHIKA	25	0	FEMALE	0.95	1.35	1.35	1.48	A
TANNA SUNNY	38	1	MALE	0.7	1.88	1.06	1.7	A
ABDUL	23	1	MALE	2.28	2.35	0.65	2.03	A
GOURHARI JANA	33	1	MALE	0.95	2.42	0.63	1.2	A
JOSHUA	28	1	MALE	1.47	2.82	2.87	2.12	A
VENKATESH	28	1	MALE	0.75	1.65	0.65	2.37	A
ABDUL KADIR	25	1	MALE	1	1.97	0.35	1.97	A
MAHMOOD ALAM	33	1	MALE	2.02	2.53	1.35	2.28	A
ISHRUF NOORANI	30	0	FEMALE	1.75	1.47	1.47	1.33	A
AJIT	25	0	FEMALE	0.5	1.62	0.51	2.05	A
VISHNU AGARWAL	23	1	MALE	2.13	1.87	1.77	0.82	A
RANA SHOME	25	1	MALE	1.23	1.68	1.45	2.13	A
SWAPAN MAJUMDER	23	1	MALE	2.3	1.98	1.47	1.3	A
R RAJESH	29	1	MALE	1.55	2.06	1.22	3.57	A
ALOK SAHU	36	1	MALE	2.28	1.57	1.83	1.85	A
VENDA R	33	0	FEMALE	2.14	2	2.66	1.25	A
KEERTHANA	33	0	FEMALE	1.85	2.42	2.42	1.85	A
SOM SRIVASTAVA	29	1	MALE	2	1.51	2	3.28	A
HARSHIT MONDAL	22	1	MALE	2.33	2.571	2	2.5	A
HAMAZ ALI MOLLA	23	0	FEMALE	2	2	2.42	2	A
GURU PRASAD	32	1	MALE	2.16	1.71	2.51	1.16	A
KALAI VANI	28	0	FEMALE	0.7	1.5	3.1	3.66	A
KHUSRU	21	0	FEMALE	1.65	1.16	3	1.85	A
M AKBAR BASHA	27	1	MALE	0.42	3.571	1.85	1.85	A
GAURAV KUMAR	22	1	MALE	1.58	2.83	1.74	2.45	A
JOY ROZAR	21	1	MALE	0.56	2.12	4.75	3.54	A
SHANKAR	33	1	MALE	3.33	2.57	1.83	1.83	A
GUHESH	33	1	MALE	1.78	3.23	2.16	1.9	A
ABDUL ROUF	33	1	MALE	2.42	1.42	2.16	0.5	A
BULBUL	30	0	FEMALE	1.85	1.66	1.14	1.33	A
RANJU K	33	0	FEMALE	2.16	2	1.16	1.5	A

MIGRANOUS VERTIGO GROUP

NAME	HOSPITAL NUMBER	SEX	AGE	STATIC-VERTICAL	DYNAMIC-VERTICAL
BAPIETE	728523f	FEMALE	27	0.46	2.98
TECHIAMPI	326287f	FEMALE	36	1.01	7.15
SONALI	678125F	FEMALE	18	1.03	2.7
NITAI BAROI	333192F	MALE	28	1.28	1.9
MANJU DEVI	697846F	FEMALE	28	0.98	5.3
K SARALA	900981C	FEMALE	38	0.91	3.41
PRUYAMALA	697201F	FEMALE	36	1.7	3.78
ANIMESH PATRA	954770D	MALE	34	1.15	0.86
LAV KUMAR	727854F	MALE	34	0.65	1.8
KRISHNA BEET	774196F	FEMALE	36	1.51	2.01
KOYEL ADHIKARI	634529F	FEMALE	22	0.6	3.1
DIPIKA BISWAS	488951F	FEMALE	32	2.3	4.23
SUPRABHAT	748016F	MALE	25	1.13	2.53
SHANTHI HARIZAN	633781F	MALE	25	0.52	1.82
LATARANI SARKAR	718651F	FEMALE	35	1.9	1.85
JANCY RANI	111222D	FEMALE	30	1.43	1.77
TEJA	827674F	MALE	30	1.33	1.6
SARA JOHN		MALE	26	0.6	6.3
SUMAN	757223B	FEMALE	25	0.33	0.83
SOHEL RANA	873537F	MALE	25	0.6	1.3
SOURAV	657646D	MALE	22	1.3	1.6
SENTHIL	869997F	MALE	31	2.3	2.66
PAVITHRA C	895172F	FEMALE	24	3.66	4
RENUGA	895225F	FEMALE	36	1.66	3.5
GOURANGA KARMAKAR	011381G	FEMALE	33	2.5	3.16
SOWBHAGYALASKHMI	881226F	FEMALE	37	0.5	5.3
KRISHNA MAJUMDAR	894477F	FEMALE	20	1.5	3.16
VIMALA	874635F	FEMALE	37	1.5	4.5
VIJI S	853691F	FEMALE	21	0.5	2
LAKSHMI	884565F	FEMALE	35	3.16	2.5
VIMALA E	006399G	FEMALE	35	2	3.16
RAM SURESH RAJAK	977499D	MALE	54	1.13	2.1
CHANDRA	719324F	FEMALE	51	1.45	4.86
DIPA GUHA	480910F	FEMALE	56	1.61	3.23
SHANTHI NATH	715108F	FEMALE	55	1.21	1.4
SANTANA DHARA	668781F	FEMALE	42	0.83	0.96
KRISHNA CHAKRABORTHY	682226F	FEMALE	45	0.25	1.38
KRISHNA DHAR	698811F	FEMALE	60	1.25	2.8
UMESH KUMAR	681361F	MALE	40	0.75	1.2
SHANTHI	461255F	FEMALE	50	1.91	6.8
ADIP ACARYA	663287F	MALE	49	0.76	3.4
MARIYAM HABEEBA	766350F	FEMALE	45	1.2	4.16

THOMAS	762615F	MALE	49	0.7	2.28
ANAWASH	760089F	FEMALE	56	1.13	1.46
MANI	510567F	MALE	60	1.5	5.33
BHASKAR	904869F	MALE	54	2.33	4.5
SUMAN KANT THAKUR	873103F	MALE	46	2.33	2.16
SADDA JAYANTHI	876252F	MALE	42	1.5	2.16
RAO MILAKARJUN		MALE	44	1.61	0.96
JANITY	131433F	FEMALE	50	5.5	2
TAPPA DUTTA	848405C	MALE	47	1.3	1.6
BORRA ARLA	801065F	MALE	41	1.8	10.3
TSHUTU	773242F	MALE	41	1.1	4.3
INBA	267781A	MALE	44	0.8	2.15
ASHA DEVI	873751F	FEMALE	60	0.7	3.3
HEMAVATHI	354829B	FEMALE	45	1	3
BISHWADEEP GHOSH	876313F	MALE	50	1	1.83
AMUDA	007087G	FEMALE	42	1.66	3.3
PUSHPA PRIYA	685174B	FEMALE	42	2.66	3.66
GEETA RANI DAS	879680F	FEMALE	57	2.16	6.5
AMUTHA	024222D	FEMALE	51	1.5	3
MALLIKARJUN	798177F	MALE	50	1.25	0.96
MAMTA SINHA	662042F	FEMALE	47	3.16	3.16
SADAN GHOSH	699291F	MALE	54	2.16	1.83
MITHU MAL	024878G	FEMALE	41	1.5	3.16
THANIKA	001397D	FEMALE	43	1.83	3

STATIC-HORIZONTAL	DYNAMIC-HORIZONTAL	PTA RIGHT	PTA LEFT	ENG CALORIC	ENG SMOOTH PURSUIT
1.36	2.81	10	10	B/L CANAL PARESIS	NORMAL
2.88	2.26	10	10	NORMAL	NORMAL
1.45	1.65	10	10	LEFT CANAL PARESIS	NORMAL
1.68	2.01	15	15	B/L CANAL PARESIS	NORMAL
2.22	4.11	15	15	N/A	N/A
1.85	5.24	15	15	B/L CANAL PARESIS	N/A
1.23	5.16	10	10	N/A	N/A
1.1	0.93	15	15	B/L CANAL PARESIS	NORMAL
0.8	2.8	15	15	LEFT CANAL PARESIS	NORMAL
0.86	2.15	15	15	N/A	N/A
0.5	3.2	10	10	LEFT CANAL PARESIS	NORMAL
0.8	3.71	15	15	LEFT CANAL PARESIS	NORMAL
0.68	1.58	5	5	B/L CANAL	NORMAL

				PARESIS	
				B/L CANAL	
1.22	1.4	15	15	PARESIS	NORMAL
5	1.03	15	15	NORMAL	NORMAL
1.4	2.42	15	15	N/A	N/A
				LEFT CANAL	
2	0.6	20	20	PARESIS	NORMAL
0.8	6	5	5	NORMAL	NORMAL
0.83	1.5	10	10	NORMAL	NORMAL
0.3	1.66	15	15	N/A	N/A
				B/L CANAL	
1.16	2.3	15	15	PARESIS	NORMAL
				LEFT CANAL	
1.5	2.66	85	15	PARESIS	NORMAL
				B/L CANAL	
1.83	4.83	10	10	PARESIS	NORMAL
3.33	3.66	15	15	N/A	N/A
				RIGHT CANAL	
0.83	2.66	90	10	PARESIS	NORMAL
1	3.8	40	25	NORMAL	NORMAL
0.5	3.5	15	15	NORMAL	NORMAL
1	2.6	5	5	NORMAL	NORMAL
1.1	6.6	15	15	NORMAL	NORMAL
				B/L CANAL	
3.15	5.6	20	20	PARESIS	NORMAL
4	4.33	15	15	N/A	N/A
2.1	2.06	20	20	NORMAL	NORMAL
				LEFT CANAL	
4.68	4.98	45	45	PARESIS	NORMAL
				RIGHT CANAL	
2.16	3.2	15	15	PARESIS	NORMAL
				B/L CANAL	
2.6	1.18	15	15	PARESIS	NORMAL
0.83	1.31	15	15	N/A	N/A
				B/L	
1.8	2.83	55	55	HYPERACTIVE	NORMAL
1.18	2.6	25	20	NORMAL	NORMAL
0.55	1.8	15	10	NORMAL	NORMAL
1.41	6.51	40	40	N/A	N/A
0.91	1.5	15	15	N/A	N/A
2	3.16	15	15	N/A	N/A
1.71	3.91	15	15	N/A	N/A
				SUPRESSION	
1.45	1.73	15	15	OF LT NYST	NORMAL
3.83	4.6	25	30	NORMAL	NORMAL
4.5	6	5	5	NORMAL	NORMAL
				B/L CANAL	
2	2.5	10	10	PARESIS	NORMAL
2	2.9	25	25	NORMAL	NORMAL

2.21	2.16	10	10	NORMAL	NORMAL
4.16	4.83	20	25	N/A	N/A
3.3	3.5	15	15	N/A	N/A
				RIGHT CANAL	
2.63	10.6	15	15	PARESIS	NORMAL
2.1	4.4	15	15	N/A	N/A
0.38	2.2	15	15	NORMAL	NORMAL
				LEFT CANAL	
2.1	3.8	30	30	PARESIS	NORMAL
3.83	4.5	15	25	N/A	N/A
0.5	2	40	55	NORMAL	NORMAL
2.5	5.16	15	15	NORMAL	NORMAL
				LEFT CANAL	
1.5	3.66	25	15	PARESIS	NORMAL
				LEFT CANAL	
1.16	5.5	25	15	PARESIS	NORMAL
				LEFT CANAL	
1.33	4.33	20	20	PARESIS	NORMAL
2.21	2	15	20	NORMAL	NORMAL
2.16	1.83	15	15	N/A	N/A
2.33	1.66	10	10	N/A	N/A
1	4	15	15	N/A	N/A
2.16	2.33	10	10	NORMAL	NORMAL

OPTOKINETIC	PENDULAR TESTING	SPONTANEOUS TESTING	IMAGING
NORMAL	NORMAL	NORMAL	NOMAL
NORMAL	NORMAL	R,L PERIPHERAL NYSTAGMUS	NOMAL
NORMAL	NORMAL	RJIGHT PERIPHERAL NYSTAGMUS	NOMAL
NORMAL	NORMAL	NORMAL	NOMAL
N/A	N/A	N/A	N/A
N/A	N/A	N/A	NOMAL
N/A	N/A	N/A	N/A
NORMAL	NORMAL	NORMAL	NOMAL
NORMAL	NORMAL	NORMAL	NORMAL
N/A	N/A	N/A	N/A
NORMAL	NORMAL	R,L PERIPHERAL NYSTAGMUS	NOMAL
NORMAL	NORMAL	NORMAL	N/A
NORMAL	NORMAL	NORMAL	N/A
NORMAL	NORMAL	NORMAL	NOMAL
NORMAL	NORMAL	NORMAL	NOMAL
N/A	N/A	N/A	THECAL COMPRESSION
NORMAL	NORMAL	NORMAL	NOMAL
NORMAL	NORMAL	NORMAL	NOMAL
NORMAL	NORMAL	NORMAL	NOMAL-OUTSIDE FILMS
N/A	N/A	N/A	NOMAL
NORMAL	NORMAL	NORMAL	N/A
NORMAL	NORMAL	NORMAL	N/A

NORMAL	NORMAL	NORMAL	NOMAL
N/A	N/A	N/A	N/A
NORMAL	NORMAL	NORMAL	VASCULAR LOOP AROUND IAC
NORMAL	NORMAL	NORMAL	N/A
NORMAL	NORMAL	NORMAL	NOMAL
NORMAL	NORMAL	NORMAL	N/A
NORMAL	NORMAL	NORMAL	N/A
NORMAL	NORMAL	NORMAL	N/A
N/A	N/A	N/A	N/A
NORMAL	NORMAL	NORMAL	N/A
NORMAL	NORMAL	NORMAL	N/A
NORMAL	NORMAL	NORMAL	NORMAL
NORMAL	NORMAL	NORMAL	N/A
N/A	N/A	N/A	NORMAL
NORMAL	NORMAL	NORMAL	N/A
NORMAL	NORMAL	NORMAL	N/A
NORMAL	NORMAL	NORMAL	NORMAL
N/A	N/A	N/A	EMPTY SELLA,BRIGHT SPOT IN PITUTAI HYPER IN MAX
N/A	N/A	N/A	NORMAL
N/A	N/A	N/A	N/A
N/A	N/A	N/A	ULCERATION IN LEFT CCA
NORMAL	NORMAL	NORMAL	MILD DISC DEGENERATIVE CHANGES
NORMAL	NORMAL	NORMAL	N/A
NORMAL	NORMAL	NORMAL	NORMAL
NORMAL	NORMAL	NORMAL	N/A
NORMAL	NORMAL	NORMAL	NORMAL
NORMAL	NORMAL	NORMAL	NORMAL
N/A	N/A	N/A	N/A
N/A	N/A	N/A	L4-L5 DISC CHANGES
NORMAL	NORMAL	NORMAL	N/A
N/A	N/A	N/A	N/A
NORMAL	NORMAL	NORMAL	NORMAL
NORMAL	NORMAL	NORMAL	NORMAL
N/A	N/A	N/A	N/A
NORMAL	NORMAL	NORMAL	DISC C3-4 L4-L5 DEGERATIVE
NORMAL	NORMAL	NORMAL	N/A
NORMAL	NORMAL	NORMAL	NORMAL
NORMAL	NORMAL	NORMAL	N/A
NORMAL	NORMAL	NORMAL	FEW SCATTERED TR HYPERINTENSE FO
NORMAL	NORMAL	NORMAL	N/A
N/A	N/A	N/A	NORMAL
N/A	N/A	N/A	N/A
N/A	N/A	N/A	NORMAL
NORMAL	NORMAL	NORMAL	N/A

NO OF YEARS OF	NUMBER	DURATION	SRV/HRV/BOTH	AURA

HEADACHE	OF YEARS VERTIGO	OF VERTIGO		
10	10	60 MINUTES	SRV	YES
1	1	20 MINUTES	SRV	NIL
5	5	60 MINUTES	SRV	YES
2.5	2.5	15 MINUTES	SRV	YES
0.1	0.1	10 MINUTES	SRV	NIL
0.5	0.5	10 MINUTES	HRV	YES
5	3	10 MINUTES	SRV	YES
5	5	120 MINUTES	BOTH	YES
1	1	2 MINUTES	SRV	YES
4	3	5 MINUTES	SRV	YES
0.75	0.5	15 MINUTES	SRV	YES
4	4	20 MINUTES	SRV	YES
5	0.5	60 MINUTES	HRV	YES
1.5	1	10 MINUTES	SRV	YES
1	1	60 MINUTES	SRV	YES
0.25	0.25	10 MINUTES	SRV	YES
0.25	0.25	10 MINUTES	SRV	NIL
1	1	5 MINUTES	SRV	NIL
2	2	10 MINUTES	SRV	YES
10	3	2 MINUTES	SRV	YES
2	1	1 MINUTES	SRV	YES
3	0.5	60 MINUTES	SRV	YES
0.1	0.1	30 MINUTES	SRV	YES
4	0.1	1 MINUTES	SRV	YES
4	1	5 MINUTES	BOTH	YES
4	0.5	5 MINUTES	SRV	YES
8	2	30 MINUTES	HRV	YES
0.5	0.5	60 MINUTES	SRV	YES
7	7	30 MINUTES	SRV	YES
0.5	0.5	20 MINUTES	SRV	YES
2	0.25	5 MINUTES	SRV	YES
3	4	60 MINUTES	SRV	YES
1	2	15 MINUTES	SRV	NIL
30	3	1 MINUTES	SRV	NIL
1	1	1 MINUTES	HRV	YES
1	1	10 MINUTES	SRV	YES
3	0.5	20 MINUTES	HRV	YES
25	25	120 MINUTES	SRV	YES
5	5	1 MINUTES	SRV	NIL
5	5	1 MINUTES	SRV	YES
20	10	60 MINUTES	SRV	YES

2	2	5 MINUTES	SRV	YES
3	3	5 MINUTES	SRV	YES
4	3	10 MINUTES	SRV	YES
0.5	0.5	15 MINUTES	SRV	YES
1	1	10 MINUTES	SRV	NIL
1	1	5 MINUTES	SRV	YES
4	3	5 MINUTES	SRV	YES
1	1	10 MINUTES	SRV	NIL
2	2	5 MINUTES	HRV	NIL
7	7	5 MINUTES	HRV	NIL
4	4	60 MINUTES	HRV	YES
1	1	2 MINUTES	SRV	NIL
5	5	15 MINUTES	SRV	YES
8	0.75	1 MINUTES	SRV	YES
0.25	0.25	15 MINUTES	SRV	YES
0.5	3	3 MINUTES	SRV	YES
0.5	0.5	1 MINUTES	SRV	YES
1	0.75	5 MINUTES	SRV	YES
3	3	10 MINUTES	SRV	YES
1	1	5 MINUTES	SRV	YES
3	3	180 MINUTES	SRV	YES
20	10	2 MINUTES	SRV	YES
3	3	5 MINUTES	HRV	YES
20	0.25	2 MINUTES	SRV	NIL
4	4	120 MINUTES	SRV	YES

NEUROLOGICAL SYMPTOMS?	HISTORY OF HEARING LOSS	HISTORY OF TINNITUS	SYMPTOMS OF MENNIERS	FAMILY HISTORY OF MIGRANE/LIKE MIGRANE
NIL	NIL	NIL	NIL	NIL
NIL	NIL	YES	NIL	NIL
DIPLOPIA,LOC	NIL	NIL	NIL	YES
TINGLING	NIL	NIL	NIL	NIL
NIL	NIL	NIL	NIL	YES
NIL	NIL	NIL	NIL	NIL
NIL	NIL	NIL	NIL	NIL
NIL	NIL	NIL	NIL	YES
NIL	NIL	NIL	NIL	NIL
NIL	NIL	NIL	NIL	YES
NIL	NIL	NIL	NIL	NIL
NIL	NIL	NIL	NIL	NIL
NIL	NIL	NIL	NIL	YES
LOC	NIL	NIL	NIL	NIL
TINGLING,NUMBNESS	NIL	NIL	NIL	NIL
LOC	NIL	NIL	NIL	YES

NIL	YES	YES	NIL	NIL
NIL	NIL	NIL	NIL	NIL
NIL	NIL	NIL	NIL	YES
BLACKOUTS	NIL	NIL	NIL	YES
NIL	NIL	NIL	NIL	NIL
NIL	NIL	NIL	NIL	NIL
NIL	NIL	NIL	NIL	NIL
NIL	NIL	NIL	NIL	NIL
NIL	NIL	NIL	NIL	NIL
NIL	YES	YES	NIL	YES
NIL	NIL	YES	NIL	NIL
NIL	NIL	NIL	NIL	NIL
NIL	NIL	NIL	NIL	NIL
NIL	NIL	NIL	NIL	NIL
NIL	NIL	YES	NIL	NIL
NIL	YES	YES	NIL	NIL
NIL	YES	NIL	NIL	YES
NIL	NIL	NIL	NIL	NIL
TINGLING	NIL	NIL	NIL	YES
NIL	NIL	NIL	NIL	NIL
NIL	YES	YES	NIL	NIL
NIL	NIL	NIL	NIL	YES
NIL	NIL	NIL	NIL	YES
NIL	NIL	NIL	NIL	NIL
NIL	NIL	NIL	NIL	NIL
NIL	NIL	NIL	NIL	NIL
NIL	NIL	NIL	NIL	YES
NIL	NIL	NIL	NIL	YES
NIL	NIL	YES	NIL	NIL
NIL	NIL	NIL	NIL	NIL
TINGLING	NIL	NIL	NIL	YES
NIL	NIL	NIL	NIL	YES
NIL	NIL	NIL	NIL	NIL
NIL	YES	YES	NIL	NIL
NIL	NIL	NIL	NIL	YES
NIL	NIL	NIL	NIL	NIL
NIL	YES	YES	NIL	NIL
NIL	NIL	NIL	NIL	NIL
NIL	NIL	NIL	NIL	NIL
NIL	NIL	NIL	NIL	YES
NIL	NIL	YES	NIL	NIL
NIL	NIL	NIL	NIL	NIL
NIL	NIL	NIL	NIL	NIL
TINGLING,DILPOPIA	YES	YES	NIL	YES
NIL	NIL	NIL	NIL	NIL
NIL	NIL	YES	NIL	NIL

		NIL	NIL	NIL	NIL	NIL
		NIL	NIL	NIL	NIL	NIL
		NIL	NIL	NIL	NIL	NIL
	TINGING		YES	YES	NIL	NIL

DRUGS THAT HAVE HELPED	ANY ABNORMAL EXAMN FINDINGS	BLOOD INVESTIGATIONS
N/A	N/A	N/A
N/A	N/A	N/A
N/A	N/A	DYSLIPIDEMIA,SEIZURE
N/A	N/A	DYSLIPIDEMIA
N/A	N/A	N/A
N/A	N/A	DYSLIPIDEMIA
N/A	N/A	N/A
N/A	N/A	N/A
N/A	N/A	DIABETES MELLITUS
N/A	N/A	N/A
N/A	N/A	N/A
N/A	N/A	DYSLIPIDEMIA,DM
N/A	N/A	N/A
N/A	N/A	PLNTAR FASCITIS
N/A	N/A	N/A
N/A	N/A	N/A
N/A	N/A	N/A
N/A	N/A	N/A
N/A	N/A	N/A
N/A	N/A	N/A
N/A	N/A	N/A
N/A	N/A	N/A
N/A	N/A	N/A
N/A	N/A	N/A
N/A	N/A	DYSLIPIDEMIA
N/A	N/A	N/A
N/A	N/A	N/A
N/A	N/A	POLYARTHALGIA
N/A	N/A	N/A
N/A	N/A	N/A
N/A	N/A	N/A
NIL	NIL	NORMAL
NIL	NIL	N/A
NIL	NIL	N/A
NIL	NIL	N/A
NIL	NIL	HYPOTHYROIDISM,ANEMIA
NIL	NIL	N/A
NIL	NIL	DYSLIPIDEMIA,HYPERTENSION

NIL	NIL	DYSLIPIDEMIA
NIL	NIL	DM
NIL	NIL	N/A
NIL	NIL	N/A
NIL	NIL	N/A
NIL	NIL	DM,HYPOVITAMINOSIS D
NIL	NIL	N/A
NIL	NIL	N/A
NIL	NIL	N/A
NIL	NIL	DYSLIPIDEMIA,DM
NIL	NIL	N/A
NIL	NIL	N/A
NIL	NIL	DM,HYPOTHYROIDISM
NIL	NIL	DYSLIPIDEMIA
NIL	NIL	N/A
NIL	NIL	N/A
NIL	NIL	N/A
NIL	NIL	N/A
NIL	NIL	DYSLIPIDEMIA
NIL	NIL	N/A
NIL	NIL	N/A
NIL	NIL	N/A
NIL	NIL	DM,HYPOTHYROIDISM
NIL	NIL	NIL
NIL	NIL	NIL
NIL	NIL	OLD TB
NIL	NIL	NIL
NIL	NIL	NIL