INVOLVEMENT OF AUDITORY PATHWAY IN MIGRAINOUS VERTIGO AMONG THOSE VISITING THE AUDIO-VESTIBULAR CLINIC IN A TERTIARY CARE HOSPITAL

A dissertation submitted in partial fulfillment of MS Branch IV, ENT examination of the Tamil Nadu Dr.MGR Medical University, to be held in April 2013

DEPARTMENT OF OTORHINOLARYNGOLOGY CHRISTIAN MEDICAL COLLEGE VELLORE

CERTIFICATE

This is to certify that the dissertation entitled **—INVOLVEMENT OF AUDITORY PATHWAY IN MIGRAINOUS VERTIGO AMONG THOSE VISITING THE AUDIOVESTIBULAR CLINIC IN A TERTIARY CARE HOSPITAL** is the bonafide original work of **Dr.JOHN MATHEW** submitted in fulfillment of the rules and regulations for the MS Branch IV, ENT examination of the Tamil Nadu Dr.MGR Medical University, to be held in April 2013.

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AIMS AND OBJECTIVES

1. Review data of all patients with vertigo to assess the frequency of migrainous Vertigo among patients attending the Audio vestibular clinic of CMC over a 6 month period prior to start of a prospective evaluation.

2. Determine the frequency of auditory dysfunction among patients with migrainous vertigo compared to the normal.

3. Evaluate the auditory status of patients with migrainous vertigo

INTRODUCTION

Migrainous vertigo is a vestibular syndrome which is caused by migraine and is characterised by short spells of spontaneous or positional vertigo which can lasts from only a few seconds to weeks. There are no established diagnostic criteria for migrainous vertigo as it is not included presently in the International Headache Society classification of migraine (1). Neuhauser (1) however proposed a diagnostic criterion for the entity and that has been widely accepted.

Migrainous vertigo was a poorly known entity till 1980s. Migrainous vertigo associated with problems in auditory pathway is even less well-known or documented. There are only a few numbers of studies addressing this issue (1, 2, and 3). Migraine and migrainous vertigo are considered to be a result of chemical abnormalities in the Serotonin pathway (7).

Approximately 33% of patients with migraine present with complains of vertigo (1, 2). , Patients with migraine can also present with auditory complaints like tinnitus, aural fullness, decreased hearing and phonophobia (1,2). There are reports of sudden, permanent and fluctuating hearing loss associated with migraine However, the incidence of hearing loss in migraine is low(1,3,4).

Just as migrainous vertigo lacks an established diagnostic criterion, the pathophysiological mechanisms and treatment strategies continue to be elusive. Various pathophysiological mechanisms have been proposed. Migrainous vertigo can present as a migraine aura or a longer lasting vestibular syndrome.

Cortical and brainstem spreading depression occurs as a result of glial and neuronal depolarisation occurring at cortical and brainstem levels and is regarded as the mechanism of migraine aura (5,6).

The longer lasting vestibular symptoms can be attributed to the activity of the trigeminovascular system causing release of various inflammatory neuropeptides into the dural circulation during headache (7)

Migrainous vertigo is essentially a diagnosis of exclusion. Physical examination is likely to be normal.

History however will usually provide the clue to the diagnosis than tests. Various investigations that could be done include imaging, otoacoustic emissions, auditory brain stem evoked response and audiometry in order to exclude other pathological conditions.

Studies have shown that the interpeak and absolute latencies during brainstem evoked response testing could be prolonged in migrainous vertigo.(8) Thus abnormalities of the auditory brainstem response testing may be the earliest indicator of impending auditory involvement in migraine (8).

Migraine has increased incidence in Meneire's disease and benign paroxysmal positional vertigo.

There have been reports of sudden sensory neural hearing loss with acute attacks of migraine (1, 2). Early preventive treatment would result in avoiding the morbidity caused by both these conditions like vertigo, and headaches which could often be incapacitating. It may also help to avoid the effects on hearing if the same chemical pathway is affecting the auditory pathway. Hence this study is preliminary and is intended to determine the frequency of migrainous vertigo and to investigate the involvement of auditory pathway among patients with migrainous vertigo.

REVIEW OF LITERATURE

ANATOMY OF INNER EAR

The inner ear consists of the cochlea, vestibule and the semicircular canal. The cochlea can be compared to a snail as it is a canal which spirals around itself and makes about 2 ½ to 2 ¾ turns. The labyrinth is of two types namely the membranous labyrinth filled with the endolymph and the bony labyrinth which surrounds the membranous labyrinth which is filled with perilymph.

The cochlea is divided into an upper chamber called the scala vestibule, filled with perilymph, and a lower chamber called the scala tympani, also filled with perilymph separated by a middle chamber called scala media or the cochlear duct filled with endolymph. The scala media is separated from the scala vestibule by the Reissner's membrane and the media from the scala tympani by the basilar membrane.

On the floor of the basilar membrane is situated the organ of Corti which is the receptor organ of the inner ear and has a single row of inner hair cells situated medially and three to four rows of outer hair cells laterally on either sides of the rod or tunnel of Corti.

On the medial aspect of the scala media there is a tectorial membrane which is a fibrous structure and it comes in contact with the stereo cilia of the hair cells .The bipolar cells of spiral ganglion situated in the Rosenthal's canal have dendrites which synapse with the base of the hair cells (9).

Auditory pathways

The cochlear division of the auditory nerve is formed by the axons of the bipolar cells and end in the cochlear nuclei, both dorsal and ventral on either side of the medulla. From the cochlear nuclei, crossed and uncrossed fibres travel to the superior olivary nucleus, lateral leminiscus, inferior colliculus and the medial geniculate body and finally reach the auditory cortex of the temporal lobe. The central auditory pathway is from the cochlear nuclei to the auditory cortex in the temporal lobe. Impulses reach the auditory cortex via a four neuron pathway. There are both first order and second order neurons, the first order being the bipolar neurons which connect the hair cells to the cochlear nuclei. The afferent to the cochlear nuclei is always from the ipsilateral cochlea and auditory nerve.

The second order neurons start from the cochlear nuclei and project to the contra lateral inferior colliculus.

The axons of these neurons decussate in the trapezoid body or synapses with neurons in the superior olivary nucleus. Both ipsilateral and contralateral afferent axons from the cochlear nuclei are received by superior olivary complex on both sides.

Neurons from the superior olivary nucleus pass to the lateral leminiscus of the midbrain. The third order neurons connect the inferior colliculus to the medial geniculate body. The fourth order neurons connect the medial geniculate body to the cerebral cortex.

The auditory cortex in the temporal lobe, known as the Brodman's area 41 and 42 is the gyrus of Heschl on the upper part of the superior temporal gyrus in the sylvian fissure. Clinically these areas are important as they control the visual and somaesthetic aspects of language (10).

Physiology of hearing

The ear is the sensory organ which gathers acoustic or sound energy and converts it into neural or electrical stimuli and this neural stimulus is further transmitted to the brain for

processing. The pinna and the external auditory canal capture the sound waves and direct it to the tympanic membrane causing it to vibrate.

These vibrations are transmitted to the ossicles of the middle ear and through them to the perilymph of the inner ear forming a fluid wave. The impedance matching mechanism of the middle ear compensates for about 30 decibels loss of acoustic energy. The middle ear converts sound energy of greater amplitude and lesser force into greater force and lesser amplitude.

The sensory organ of hearing, the organ of Corti has both outer and inner hair cells. The inner hair cells pick up the vibrations of the basilar membrane and convert them into impulses for the auditory nerve. The functions of outer hair cells are,

1. Participate in the micromechanics of the cochlea by amplifying the motion of the basilar membrane.
2. Adds approximately 50 decibels to the sensitivity of the cochlea
3. Increases the frequency selectivity of the cochlea for low intensity sounds, thus improving the ability to detect soft sounds in noisy environments.

However, the outer hair cells do not have any role in the transmission of electrical impulses which is done by the inner hair cells. Each point on the basilar membrane is tuned to a specific frequency. Different groups of hair cells are activated by sounds of varying frequencies within the spectrum of audibility and different inner hair cells are tuned to different frequencies (11).

There are five well studied electro acoustic events in the cochlea which are,

1. Endocochlear potential-positive voltage of 80-100 millivolts seen in the endolymphatic space of cochlea
2. Cochlear microphonics-is an alternating potential and is a true reflection of the electrical activity of the outer and inner hair cells.

3. The Summating potentials-which is a direct current offset and part of cochlear microphonics
4. The compound action potential-which is the sum of synchronous discharges in response to a click or transient stimulus. It is this action potential which forms the wave 1 of the brain stem evoked response auditory.
5. Otoacoustic emissions-are acoustic outputs from the outer hair cells as a result of both mechanical distortion products and direct echoes. (12)

MIGRAINOUS VERTIGO

Migrainous vertigo (MV) is defined as vertigo or dizziness caused by migraine. Approximately 10% of the population has migraine headaches (13) and one third of persons with migraine experience dizziness (14) so the prevalence of MV can be estimated as approximately 3% of the population.

Currently, there is no unifying term or internationally approved criterion for migraine associated vertigo. Without such criteria, determination of the pathophysiology and appropriate treatment regimens for these patients will continue to be elusive.

HISTORICAL BACKGROUND

The association between hearing and balance disorders and migraine has been recognized since ancient Greece, when in 131 B.C. Aretaeus of Cappadocia described precisely and in detail the occurrence of both conditions during a migraine crisis (15). In 1861, Prosper Ménière published a classical paper describing the symptoms of Ménière Syndrome (MS) in migraine patients (16). In 1873, Liveing reported a clear association between vertigo and migraine, which has since been also reported by various authors in various other studies and

these studies defined the concept that vertigo, hearing loss and tinnitus were part of the symptoms that some migraine patients presented(17,18).

The manifestations of migrainous vertigo were studied and published in various case series beginning from Kayan and Hood's publication (18). The disease lacks a generally accepted diagnostic criterion and a proper terminology as it is only an evolving entity and the pathophysiology and treatment are still under research. Various terminologies have been put forth namely migraine associated vertigo, vestibular migraine, migraine associated dizziness, migrainous vertigo, benign recurrent vertigo and migraine associated vestibulopathy.

Vestibular migraine has been increasingly used as the terminology these days as this stresses more on the vestibular symptoms of the disease. The link between vertigo and migraine were promoted by various epidemiological associations.

Many of the dizziness patients have migraine and conversely, many migraine patients report dizziness as a symptom (18, 19). The lifetime prevalence of migraine in the general population was found to be 14% (20) and that of vertigo 7% (3); migraine and vertigo together was found to be 3.2 % (21) in a large population based study.

EPIDEMIOLOGY

In three large population based studies the lifetime prevalence of migraine in the general population was found to be 14% (20) and that of vertigo 7% (3) and that of migraine and vertigo together was found to be 3.2 % (21). In the United States the prevalence rates of migraine ranges from 16-18% in women and 4-6% in men (22). In a study at a dizziness clinic in the United states with 200 patients, the prevalence of migraine was 38% and that of migrainous vertigo was 7% (19).

In a German population survey (3), 1212 out of 4077 patients (24%) who were interviewed telephonically had experienced vertigo in their lifetime. Out of 1003 patients who completed the interviews 243(24%) experienced vertigo due to migraine and out of the 24%, 40%

experienced spontaneous rotational vertigo, 43% positional vertigo and 17% episodic nausea with oscillopsia or imbalance(3). Detailed study of this sample showed 3.2% of these patients with of Migrainous vertigo. In a study by Kayan and Hood of 200 migraine patients in the United Kingdom, 27% had experienced vertigo (18).

DIAGNOSTIC CRITERIA

Neuhauser et al. in 2001 published ‘‘The Interrelations of Migraine, Vertigo, and Migrainous Vertigo’’ in order to provide an acceptable diagnostic criteria for migraine-associated vertigo (19).They made a criterion for definite as well as for probable migrainous vertigo. Probable migrainous vertigo is similar to definite migrainous vertigo though a less specific but more sensitive entity. This entity was created for individuals who could not be strictly diagnosed as migrainous vertigo, but still appeared to be most likely, migrainous vertigo. With the help of this criterion, they were able to delineate a statistically significant association between vertigo and migrainous headaches, with 38% of vertigo patients having migraines compared with only 24% of controls (19).

Radke et al in a recent study in 2011 checked the validity of the diagnostic criterion given by Neuhauser and colleagues. He and has colleagues conducted a long-term follow-up of those diagnosed with definite or probable migrainous vertigo (23).He followed up the patients for a mean period of 8.75 years, the minimum being 5.4 years and maximum being 11 years and found out that 85% of patients as diagnosed with migrainous vertigo, were having the disease, thus the positive predictive value was 85%. 50 % of patients with probable migrainous vertigo were later, on follow up diagnosed to have definite migrainous vertigo and 32% had only probable migrainous vertigo. With the onset of hearing loss in some patients, some of the diagnosed patients of migrainous vertigo were reclassified as Meniere’s disease. Patients with migrainous vertigo had migrainous headache with aura and the

percentage was shown to be 63%.However, only 22% of patients with probable migrainous vertigo had migraine with aura. The criterion given by Radke et al was precise in the long term, had doubts on its diagnostic precision. The criterion given was;

1) Rotational vertigo-at least three attacks which were spontaneous

2) Duration of episode at least a minute

3) No associated one sided decreased hearing.

Radke et al conducted telephone interviews with patients suffering from recurrent vertigo which was benign and who had migraine as well .Among these patients they found that 87% had imbalance ,and 92% had nausea. Headache was present in only 50% of them. They proposed that diagnosis should be based on the pathophysiology rather than clinical features. Another study by Brantberg et al., there was a suggestion that there were different forms of migrainous vertigo which were likely to be present, however research is still going on to probably show the differences between the different types of the disease (24).

In another recent study, Cohen et al. in 2011 did a review of chart of patients diagnosed with migrainous vertigo retrospectively to study the disease further and understand the patho physiology. Their diagnostic criteria were patients with:

1) Migrainous headache according to the International headache society classification and

2) One symptom of migrainous vertigo with at least one vestibular trigger. The most common vestibular symptoms according to their study were light headedness, unsteadiness and balance disorders. Various triggers for the disease were documented like crowds, moving objects and flashing lights. In their study the vestibular symptoms like vertigo were more likely to be associated with migrainous headache with aura than without aura. They also suggested that if the common features of migrainous vertigo can be crafted together, an acceptable diagnostic criteria can be made, although in this study, no definite criteria was made as the data was not sufficient enough.(25).

. In another study by Reploeg and Goebel in 2002, an analysis of patients retrospectively was conducted to investigate the efficacy of treating migraine and thus reducing the dizziness associated with it. In this study the Patients in whom a diagnosis of migrainous vertigo was made, the treatment was given with anti migraine drugs and their response was detected. They did not have a specific diagnostic criterion for migraine. The patients were followed up for a mean period of 54.5 weeks and 72% of the patients had more than 75% reduction in headache and all patients except 4 of them had reduction in their dizzy spells. The clinical response to treatment thus can be used to validate the diagnostic criteria, although the diagnostic criterion was not specified. Thus as finding acceptable treatment options are the final culmination of the controversies research continues to determine the pathophysiology of migrainous. (26).

In any disease, the pathophysiology of the condition will dictate the diagnosis and treatment. But when the pathophysiology of any disease is not fully understood, the diagnostic criteria will have to be substantiated by its treatment outcomes. Studies are still going on to investigate migrainous vertigo, and there no gold standard diagnostic criterion for it. However, Neuhauser et al's criteria (19) presents a broad based starting point in guiding health care workers diagnosing and treating this relatively increasingly recognized entity.

Diagnostic Criteria for Migrainous Vertigo (19)

Definite MV

1) Vestibular symptoms of at least moderate severity which are episodic (rotational vertigo, positional vertigo, head motion intolerance which is a sensation of imbalance or object motion that is provoked by head motion). At least one of the following migrainous symptoms

during at least two attacks of vertigo: migrainous headache, increased sensitivity to lights, increased sensitivity to sounds, visual or other auras.

2) Migrainous headaches according to the International Headache Society criteria (IHS).

3) Other causes of vertigo ruled out by investigations.

Probable MV

1) Vestibular symptoms which are episodic of at least moderate severity (rotational vertigo, other illusory self or object motion, positional vertigo, head motion intolerance).

2) At least one of the following: migrainous headaches according to the criteria of the IHS; migrainous symptoms during vertigo; migraine triggers of vertigo, (e.g. hormonal changes, sleep irregularities, specific foods); or response to anti migraine drugs.

3) Other causes of vertigo ruled out by investigations.

The International Classification of Headache Disorders, 2nd Edition (27)

Migraine without Aura

A. Five attacks of headache fulfilling criteria B–D

B. Headache lasting 4–72 hours which were untreated or unsuccessfully treated.

C. Headache has two of the following characteristics: located unilaterally, of pulsating quality , intensity of pain which is moderate or severe, aggravation by or causing avoidance of routine physical activity.

D. Headache having one of the following: nausea and/or vomiting, photophobia, and phonophobia

E. Cannot be related to another disorder

Typical Aura with Migraine Headache

A. Two attacks of headache fulfilling criteria B–D

B. Aura consisting of at least one of the following, but no motor weakness:

fully reversible visual symptoms including positive and/or negative features, fully reversible sensory symptoms including positive and/or negative features, fully reversible dysphasic speech disturbance.

C. At least two of the following: homonymous visual symptoms and/or unilateral sensory symptoms, at least one aura symptom develops gradually over 5 minutes and/or different aura symptoms occur in succession over 5 minutes, each symptom lasts 5 and 60 minutes.

D. Headache fulfilling criteria for migraine without aura begins during the aura or follows aura within 60 minutes.

E. Cannot be related to another disorder.

PATHOPHYSIOLOGY

The pathophysiology of Migrainous vertigo despite intense study is still poorly understood. Migraine sufferers are more sensitive to numerous unpleasant sensory inputs and these inputs trigger a threshold which causes a cortical event followed by a brainstem event causing more input to be perceived as noxious resulting in headache. Sensory inputs that are not disturbing to non migraineurs like bright light, loud sound, motion and smell can be to people suffering from migraine. Thus the brain of migraine suffers are hyperexitable. Thickening of sensory cortex in migraine sufferers is reported in certain studies. Migraine sufferers are sensitive to sensory inputs even outside the period of headache. According to studies 45 % of children and 50% of adults gave a positive history of motion sickness (18, 28).

Vascular dysregulation is also considered a part of the pathophysiology. Migraine aura may represent vasoconstriction and cortical hypoxia. It is also regarded as a cortical neuronal process (29). Both vascular dysregulation and abnormal electrical activity such as cortical spreading depression of Leao (29) and changes in occipital area seen in visual auras are implicated in aura of migraine (29). The trigeminal brainstem circuit dysfunction is also

implicated as a pathophysiology for Migrainous headache (29). This can be explained by the response to serotonin agonists given during the episode of migraine.

Trigeminal neuron cell bodies contain a vasodilator peptide called calcitonin gene related peptide which is released during a sensory input overload triggered cortical circuitry which in turn increases painful input (29). Triptans block these receptors. Similar mechanisms are proposed for Migrainous vertigo. The cortical spreading depression may play a role in patients who are having short attacks (30). The cortical spreading depression can produce vestibular symptoms when the sensory cortical areas at the posterior insula and temporoparietal junction are involved (7). Canal paresis and complex positional nystagmus however cannot be explained by this phenomenon (31). Calcitonin gene related peptide, serotonin, adrenaline and dopamine involved in the pathogenesis of migraine also modulate the activity of a number of central and peripheral vestibular neurons thus contributing to the pathogenesis of vestibular migraine (7,32 ,33,34). The unilateral release of these substances causes one sided headache and a static vestibular imbalance resulting in rotatory vertigo. Bilateral release of these substances could result in motion sickness type of dizziness. Migraine and vertigo are prominent symptoms in familial hemiplegic migraine and episodic ataxia type 2 and an abnormal voltage gated calcium channel gene were found in them (35). This prompted the search for a susceptibility gene for Migrainous vertigo which so far has not been identified (36, 37).

GENETICS

Episodic vertigo has been associated with certain genetic syndromes, few of them having known genes. Episodic vertigo has been reported in episodic ataxia type 2 and is strongly associated with it. It has also been reported in episodic ataxia TYPES 134 (35,38,39 ,40). EA type 2 is allelic with familial hemiplegic migraine and about 50% of cases report migrainous headaches (35). In his study of 24 probands, Oh found 40% incidence of benign recurrent

vertigo in relatives as compared to only 2% in unrelated spouses and this was correlated with a high incidence (46%) of migraine in them. In 22 of the cases where more than one generation was affected, an autosomal dominant inheritance pattern was found the most likely (7). This study found an association with 22q12. A majority of these cases also had Migrainous headache.

CLINICAL FEATURES

Migrainous vertigo can occur at any age and has a female preponderance; the ratio being 1.5 and 5 to 1 (6,7,19,32,33). It shows an autosomal dominant pattern of inheritance with decreased penetrance in men. Migrainous headache begins earlier than vestibular migraine in most patients (6, 19). Some of them may be free from migraine for a few years and may manifest vestibular migraine later, like women may develop vestibular migraine after menopause that may have had Migrainous headache earlier in their life. Vestibular migraine is common in migraine without aura than in migraine with aura (18).

Vestibular Migraine in Children

One of the early manifestations of vestibular migraine in children is benign recurrent paroxysmal vertigo. This can be characterised by short attacks of vertigo or unsteadiness, and often nystagmus, nausea or vomiting recurring for months or years (41). These children may develop Migrainous headache later in life (42). There is a two fold increase in first degree relatives according to certain studies (42). In a population-based study, the prevalence of recurrent vertigo probably related to migraine was estimated at 2.8% in children between the ages of 6 and 12 years (43).

VESTIBULAR MIGRAINE IN ADULTS

In a recent study of migraine associated vertigo, it was found out that the most common vestibular symptom is rotational vertigo (70%), intolerance to motion (48%) and positional

vertigo (22%) (19). The onset of the disease can be gradual to abrupt. The duration of the disease was also found to be variable. The most common duration according to Neuhauser and his colleagues was 5- 60 minutes (33%), followed by 1-24 hours (21%), few seconds to minutes (18%) and more than 24 hours (2%)(19). It may take several days to weeks for the attack to subside and the attacks can be weeks to months to years apart. The spontaneous vertigo can transform to positional vertigo after several days to weeks (44). Positional vertigo is different from benign paroxysmal positional vertigo in duration of single attack, total duration of symptomatic episodes and the type of nystagmus. The patients need not experience positional vertigo with each attack although 40-70% of patients experience it (18). Head motion intolerance which is similar to motion sickness which is imbalance and nausea or vomiting provoked by head movement is experienced by a certain number of patients (32). Visual vertigo which is dizziness stimulated by a moving object is also a common feature (32,45). Nausea and unsteadiness are also common but are not specific for Migrainous vertigo. The vertiginous attacks may precede the headache, occur along with it or can occur after the episode of headache. Thus some patients experience vertigo with headache constantly (45%) or without headache sometimes (48%) and some never experience headache and vertigo together (6%). A good number of patients have photophobia (70%), phonophobia (64%), osmophobia, visual auras during an attack and auras other than vertigo (36%) (19). Some patients may present with aural complaints like hearing loss (32%) and tinnitus (32%) and some may have fluctuating hearing loss and aural fullness (11%) (8).The hearing loss may be mild and transient which usually is non progressive. Less common symptoms include a sensation of rocking, tilting, and walking on an uneven surface and unsteadiness. Similar to triggers for migraine, numerous triggers for migraine associated vertigo is also identified in a minority of patients. The triggers are numerous but a few seem to occur more frequently than others. The weather related triggers include changes in barometric pressure, and change in

seasons. The sensory stimuli triggers include bright or scintillating lights, intense smells, and noise. Other triggers could include physical exertion, decreased sleep, menses and dehydration while the common dietary triggers include matured cheese, red wine, glutamate, and change in pattern of caffeine intake, alcohol, chocolate and nitrites. Even when the triggers and manifestations are absent a diagnosis of MV can be made after excluding other causes of vertigo. The favourable response to anti migrainous drugs also supports the suspicion of a migraine mechanism. In summary the manifestations of MV is variable and the diagnosis is usually made on clinical suspicion, exclusion of other causes of vertigo, occurrence of migraine triggers and probably response to anti migraine drugs. The various manifestations of vestibular migraine in a study of 33 patients by Neuhauser are given below

Clinical features of definite vestibular migraine in 33 patients(19)

Clinical Features	%
<u>Vestibular symptoms</u>	
Positional vertigo	42%
Head motion intolerance	48%
Rotational vertigo	70%
Other illusory self- or object motion	18%
<u>Duration of vestibular symptoms</u>	
Seconds to 5 minutes	18%
5 to 60 minutes	33%
1 hour to 1 day	21%
>1 day	2%
<u>Migrainous symptoms during vertigo</u>	

Migrainous headache	94%
Always	47%
Sometimes	48%
No headache	6%
Photophobia	70%
Phonophobia	64%
Visual or other auras	36%

PHYSICAL EXAMINATION IN MIGRAINOUS VERTIGO

The clinical examination in these patients, both neurological and otological especially in the symptom free period will be normal (7). At times they can have nystagmus (18, 31) which can be made out by Frenzel goggle examination (46). About 20% of vestibular migraine patients will have a one sided decreased response on caloric testing and 10 % will have directional preponderance of nystagmus responses (6, 7).

These findings are not specific for the disease and can be seen in other vestibular syndromes as well. There are studies which show mild occulomotor deficits in absence of other brain stem or cerebellar signs (6), vestibular migraine patients are four times more nauseous than migraine with other vestibular syndromes during caloric testing (47). In a study of 20 patients of vestibular migraine during their acute phase, all but one had imbalance and swaying during Romberg's and tandem walking. Pathological nystagmus was observed in 14 patients. Unlike benign paroxysmal positional vertigo, the nystagmus is maintained until the position is maintained and it is not in the plane of positioning. Some of the patients had a unilateral deficit of vestibulo-ocular reflex, central vestibular dysfunction and peripheral vestibular dysfunction (31).

In clinical practice as there are no symptoms specific for vestibular migraine, the history will usually provide the clue to diagnosis than vestibular testing. If the history is clear cut, then

the vestibular tests are not necessary. Testing during the period of attack, will reveal certain abnormalities which usually disappear after a few days to weeks and testing when the patients are asymptomatic will show no abnormalities.

TESTING FOR MIGRAINE RELATED VERTIGO

Migrainous vertigo is a diagnosis of exclusion. The most crucial overlap is with other otological diseases and for this reason, an otological and vestibular screening is advisable.

The most useful tests include audiometry (to rule out Meniere's disease and labyrinthitis) and vestibular evoked myogenic potential or video nystagmography, to rule out vestibular disorders. Various other tests done in migraine associated vertigo include otoacoustic emissions (48), brainstem evoked response audiometry (8, 49), posturography (51, 52) vestibular evoked myogenic potential (50), rotatory chair testing (32, 51), and caloric testing (52).

All these are done primarily to exclude other disorders as there is no specific diagnostic test for the disease. Oculomotor testing is also done to exclude other conditions and also according to Von Brevern and colleagues (31), there are minor oculomotor abnormalities in 70% of patients with vestibular migraine. Brain imaging also done to exclude other disorders is normal all patients. However a recent meta–analysis showed that there are white matter abnormalities in 23% of migraine patients and they are at four fold risk of developing white matter abnormalities than people without migraine (53).

The imaging findings are scattered punctate foci of T2 and fluid-attenuated inversion recovery signal hyper intensity in the deep cerebral white matter. There are rare cases of hearing loss in Migrainous vertigo (54, 55) which is usually transient and episodic (56). Cases of permanent hearing loss can be due to Migrainous infarction .However currently there is only limited data to suggest hearing loss in migraine associated vertigo. The present

clinical experiences suggest almost all patients with migraine associated vertigo have normal hearing, except when combined with Meniere's disease.

Pure tone Audiometry (57)

Pure tone audiometry is a behavioural test used to measure the hearing sensitivity and it measures both central and peripheral auditory systems. The tests done are plotted on a graph called audiogram which displays the intensity of sound as a function of frequency. The various degrees of hearing loss are;

Normal hearing loss-0 to 25 d B

Mild hearing loss- 26 to 40 d B

Moderate hearing loss-41 to 55 d B

Moderately severe hearing loss-56 to 70 d B

Severe hearing loss-71 to 90 d B

Profound hearing loss- >90 d B

Types of hearing loss

1. Conductive hearing loss-here the bone conduction thresholds will be normal and but the air conduction thresholds are higher by at least 10 decibels when compared to normal. E g-chronic otitis media.
2. Sensorineural hearing loss-Here the bone and air conduction thresholds are within 10 decibels of each other, but the bone conduction thresholds are higher than 25 decibels. E g- presbyacusis

3. Mixed hearing loss-here there is involvement of both the conductive as well as sensorineural components where the air and bone conduction thresholds are more than 10 decibels apart and the bone conduction thresholds are more than 25 decibels.

Pure tone average is the average of the hearing sensitivity at 500 Hz, 1000 Hz and 2000Hz. The primary purpose of doing an audiogram is to determine the type, degree and configuration of hearing loss. It can not be done in young and unco operative individuals and patient suffering from other medical conditions. No anaesthesia is required for the procedure.

Pure tone audiometry is routinely done in all cases of migrainous vertigo. There are only limited studies which shows migrainous vertigo patients having hearing loss (8). In Dash et al's study 7 out of 38 patients of migrainous vertigo had mild sensorineural hearing loss documented.

Impedance Audiometry.(57)

Tympanometry and acoustic reflexes are routinely done in all cases of migrainous vertigo. A normal (type A) tympanometry confirms a normal middle ear function which is essential for testing for otoacoustic emissions and auditory brain stem potentials.

The acoustic reflex tests the integrity of the acoustic reflex pathway. The reflex pathway consists of an afferent (cochlea and auditory nerve) and efferent pathway (fibres from brainstem to the facial nerve and stapedius muscle

Brain stem evoked response audiometry (BERA).

Auditory brainstem response audiometry testing is an objective test which assesses the auditory pathway from the auditory nerve to the brainstem, electro physiologically. After a

sound stimulus is presented, it occurs in the first 10 milliseconds, hence is considered a short latency potential.

The BERA consists of seven waves and of these waves 1, wave III and wave V are the most seen and robust and are of more clinical significance. The different sites at which these waves are generated are –

Wave 1- most distal portion of the auditory nerve in relation to the brainstem.

Wave II- proximal portion of auditory nerve in relation to the brainstem.

Wave III-cochlear nucleus

Wave IV –superior olivary complex

Wave V-lateral leminiscus

Wave VI-inferior colliculus

Wave VII-medial geniculate body (58,59)

Recordings of this potential may be analysed clinically using various parameters,

1. Morphology of the waves
2. Absolute latency and amplitude of wave I,III and V
3. Interpeak latencies-wave1-III,wave III-V and wave 1-V
4. Wave 1 -V latency and amplitude relation
5. Wave 1 -V latency inter aural difference or wave 5 absolute latency differences.

Of these parameters, the most widely used clinically are the interpeak and absolute latency interval measurements (59).

According to studies, the main clinical aims and uses of BERA are,

1. To establish a minimal auditory response level
2. To delineate the type of hearing loss
3. To assess the maturity of the central nervous system in neonates.
4. To investigate the site of auditory nerve or brain stem injury

5. To monitor surgery of posterior fossa
6. To monitor patients in intensive care units (60)

There are certain factors which can influence the interpeak and absolute latencies of the test. The physiological factors which influence the values according certain studies are subject related features such as age, sex and hormonal status. Certain studies revealed increased wave latencies in BERA testing in subjects aged over 60 years while in some studies there was no statistically significant variation with age (61). Other studies have shown that latency measures (especially wave V) and interpeak intervals (especially in the I-V interval) are higher in male subjects compared with female subjects (62).

Thus, both age and sex are mentioned as variables that may alter BAEP recordings; their true influence, however, remains controversial, requiring additional studies of these issues.

BERA potentials are recorded from the scalp by disc or cup electrodes which are the responses to high intensity clicks which are given at a rate of approximately 10 per second. These potentials are typically present in persons with normal hearing and those with sensorineural hearing loss. There are only few studies on the BERA abnormalities in patients with migrainous vertigo (8). As the degree of sensorineural hearing loss increases, the morphology of the potentials also will also change to the extent that it can be absent in profound sensorineural hearing loss (63).

Otoacoustic emissions

Otoacoustic emissions were first described by Kemp in 1978 as low intensity sounds generated by the outer hair cells of cochlea by their active movements (64,65).

In general, spontaneous OAEs occur in only 40-50% of individuals who have normal hearing. For these adults, the range is about 30-60%; in neonates with normal hearing, the range is approximately 25-80%. These are a marker of cochlear function and the emissions are left intact even after the sectioning of auditory nerve. Transient clicks at 80- 86 db sound pressure

evoke the acoustic echoes and typically responses to 260 stimuli are captured over a time frame of first 20 milliseconds after application of the stimulus.

There are 2 types of emissions; a transient evoked and a distortion product oto acoustic emission. The transient evoked emissions are present if the response amplitude after reduction of background noise is 4 decibels or more and the wave form is reproducible in at least 3 octave bands.

More frequency specific information can be obtained from distortion product emissions than from transient evoked emissions as it allows specific testing of a restricted region of outer hair cells. In distortion product emissions 2 continuous tones of 2 different frequencies (f1 and f2) are given and the emissions are largest at the 2f1-f2 frequency. Otoacoustic emissions represent the acoustic equivalent of electric cochlear microphonics. Both these provide improved tuning of the inner ear and enhance threshold activity.

Though OAES provide valuable information regarding outer hair cell function, there is no accurate way to measure inner hair cell function.

<u>Prerequisites for obtaining otoacoustic emissions</u>

Prerequisites include the following:

- An ear canal which does not have any obstruction.
- The probe should seal the ear canal.

- Correct situation of the probe
- Normal middle ear function: Pressure equalization (PE) tubes alone probably will not interfere with results. However, if emissions are absent, results should be interpreted with caution.
- Normal functioning outer hair cells.
- A quiet patient: Excessive movement or vocalization can give a wrong interpretation.

- A quiet recording environment: a noisy environment may preclude accurate recording.

Non pathologic problems that can cause absence of OAEs

- Poor placement of probe tip or not obtaining proper seal
- Standing waves, however most equipment alerts the person doing the test about these.
- Wax blocking the canal or probe port
- Debris and foreign bodies in the external auditory canal
- Vernix caseosa in neonates: This is common immediately after birth.
- Uncooperative patient

Pathologic problems that can cause absence of OAEs

Outer ear

- Otitis externa
- Stenosis of external auditory canal
- Any lesions in the external canal like a cyst
- Variations in the middle ear pressure
- Perforations of tympanic membrane

Middle ear

- Ossicular discontinuity
- otospongiosis
- chronic otitis media, squamous or mucosal
- Cyst

Inner ear (Cochlea)

- Exposure to ototoxic medication
- noise exposure .
- Any other cochlear pathology

Conditions that do not affect OAEs

- Auditory nerve pathology: If auditory nerve pathology also affects the cochlea, OAEs are affected.
- Central auditory disorder

Conditions that elicit abnormal OAEs and normal behavioural thresholds

- Tinnitus: OAEs may be abnormal in the frequency region of the tinnitus.
- Excessive noise exposure
- Any intake of ototoxic drugs
- Any Vestibular pathology

Conditions that elicit normal OAEs and abnormal behavioural thresholds

- Functional hearing loss
- Attention deficits
- Autism
- Possibly, inner hair cell damage but normal outer hair cells (reported for animals but no human reports yet)
- Auditory neuropathy: This includes central auditory nervous system dysfunction and CN VIII auditory dysfunction.

Uses of Otoacoustic emissions

- Simple, non invasive hearing tests for neonates as well as those not co operating for conventional hearing tests.
- Assists in the differential diagnosis of various cochlear and central auditory pathologies.
- Biometric identification
- As it is an objective test, it is useful in investigating inorganic hearing loss and demonstrating normal cochlear function to the patient. Literature has been silent regarding the abnormalities of OAEs in Migrainous vertigo.

DIFFERENTIAL DIAGNOSIS OF MIGRAINOUS VERTIGO

When headache and dizziness coincide, the most likely diagnosis is vestibular migraine. The difficulty in diagnosis is when these two does not coincide or when there is no headache at all. According to recent studies, migraine may be triggered by dizziness itself the various other disorders we can think of are as follows (66).,

1) Benign paroxysmal positional vertigo(BPPV)-Here the vertigo lasts for seconds to 1 minute and is provoked by changes in head position and there is a positive positional test result with typical geotropic torsional nystagmus.

2) Meniere's Disease- The Vertigo lasts for 20 minutes to 3 hours with Features of fluctuating hearing loss, tinnitus, and aural fullness and progressive hearing loss over years (67).

3) Central positional vertigo-here the features are similar to BPPV but the direction of positional nystagmus, duration and latency are not similar to that of BPPV; as the vertigo is central, neurologic or neurotologic features will also be present.

4) Vertebrobasilar transient ischemic attack-the vertigo lasts for a few minutes, with brain stem symptoms, ataxia, dysarthria, double vision or visual field defects. Elderly patients with vascular risk factors like hypertension, diabetes mellitus and dyslipidemia are prone to have such symptoms.

5) Vascular compression of the eighth nerve presents with short attacks of vertigo lasting a few seconds for several times in a day with or without auditory symptoms and such patients often responds to carbamazepine.

6) Perilymph fistula –the Vertigo occurs after a history of head trauma, barotrauma, or stapedectomy or violent coughing, sneezing, straining, or loud sounds.

7) Autoimmune inner ear disease-there will be attacks of vertigo of variable duration; with rapidly progressing hearing loss and insufficient compensation of unilateral vestibular loss, but bilateral loss is also common. There will be brief and mild spells of vertigo during rapid head movements, oscillopsia when head turns to affected ear and a positive result of head thrust test to the affected side.

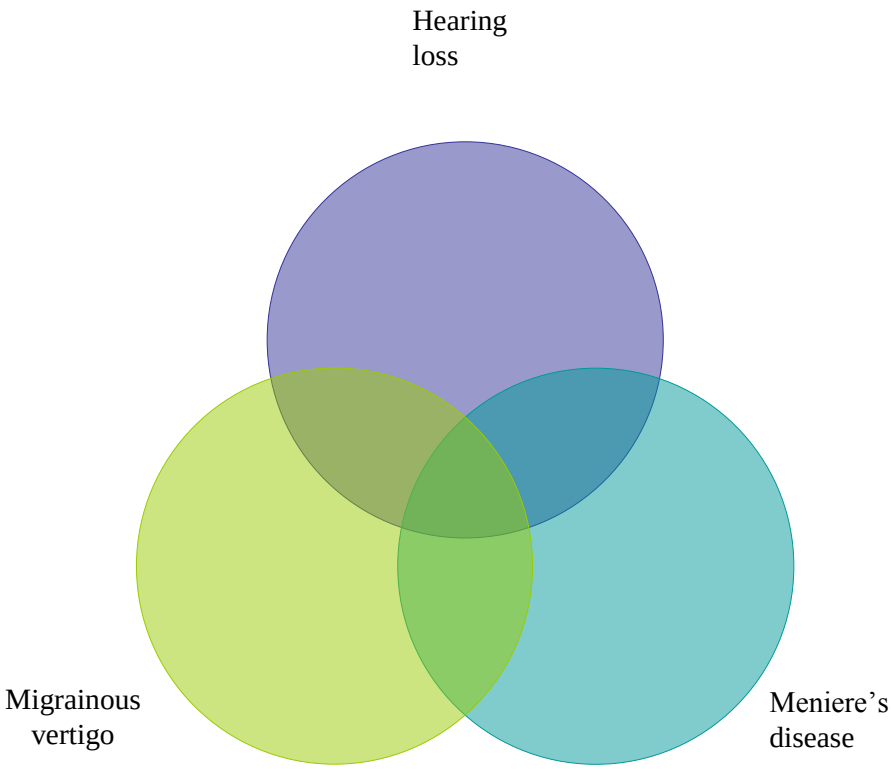
8) Schwannoma of the eighth nerve-in this condition, the patients rarely presents with vertigo as a complaint. They can have mild attacks of vertigo, the most common symptoms are a slowly progressive unilateral hearing loss and tinnitus and abnormal Brain stem evoked response audiometry with poor wave form morphology.

MIGRAINE ASSOCIATED VERTIGO SYNDROMES

Migraine and Meniere's disease

There is an increased prevalence of migraine (22-76%) in patients with Meniere's disease, though the pathophysiologic association is not well understood(68). The prevalence of migraine in Meniere's (American Academy of Otolaryngology 69) is twice as seen in age and sex matched controls. Patient having migraine has an increased susceptibility of developing earlier manifestations of Meniere's disease than without migraine. Vestibular testing also

provide little information to differentiate between the two (70). In vestibular migraine the hearing loss will be mild, transient, and non progressive whereas in Meniere's disease the hearing loss is severe and progressive (33). However migraine can mimic Meniere's disease and can even lead to inner ear damage and delayed endolymphatic hydrops (54). At times it is difficult to differentiate between the two disorders clinically (68). Both these conditions may be different manifestations of the same susceptibility and may have a genetic basis also (70). There are familial forms of Meniere's disease in which there will be increased prevalence of migraine, no hearing loss and episodic vertigo making the migraine-Meniere's syndrome genetically distinct from Meniere's disease alone (71).



Association of migrainous vertigo with Meneire's disease and hearing loss

Migraine and Benign paroxysmal positional vertigo

Although clinically separate entities, certain studies found a link between the two. Migraine was three times more common in those with idiopathic BPPV than with BPPV secondary to

surgery or trauma and two times more common in idiopathic BPPV than in age and sex matched controls (72). A retrospective study by Uneri of 476 patients of BPPV found 54.8% of them having migraine (73). In BPPV, the degenerating otoconia movement produces movement of endolymphatic fluid in the labyrinth resulting in a short spell of dizziness which has a true rotatory vertigo, nausea and imbalance. On positional testing there will be a brief torsional geotropic nystagmus, which is successfully treated by the particle repositioning manoeuvre (74). This has to be distinguished from the positional vertigo in vestibular migraine (75). Thus migraine can be considered as a risk factor for developing BPPV. This association may due to the ischemic changes due to vasospasm to the otoliths (54, 55).

Migraine and motion sickness

Various studies have shown that Motion sickness occurs more frequently in patients who have migraine (30%–70%) than in controls who have tension headache or in headache-free controls (20%–40%) (18,45). The prevalence is increased in children and migraine with aura (76). Optokinetic stimuli induced visual vertigo is also common in migraine (45). Apart from that, photophobia, headache and scalp tenderness can be easily caused by optokinetic stimulation in patients who have migraine than in controls. It may be difficult to differentiate between episodic motion sickness and attacks of VM induced by motion stimuli. The only differentiating points could be the duration and type of symptoms. In vestibular migraine the rotational or positional vertigo will usually persist even after the motion stimuli while the dizziness due to motion sickness will improve after the motion stimuli is removed. Chronic VM may be explained by a constantly lowered threshold to motion stimuli. Interestingly, motion sickness could be prevented using rizatriptan in migraineurs who had VM but not in patients who had migraine alone (77,78).

Benign recurrent vertigo of childhood

Basser in his studies described a recurrent paroxysmal vertigo occurring in children particularly below 8 years which was of very short duration lasting for seconds to minutes and had a benign prognosis (41). Fenichel later in studies described the association of migrainous headaches and benign recurrent vertigo (79). Long term follow up studies suggested that there is a strong association of (both family history and personal history) migraine headaches and benign vertigo in childhood. These children also had a high prevalence of motion sickness (80).

Basilar Migraine

In basilar migraine, vertigo is one of the most common symptoms (2).which has been included in the current international headache society classification of headaches. For a diagnosis of basilar migraine there should be an aura of bilateral visual field abnormalities and at least two symptoms suggestive of brainstem involvement along with migrainous headache (2). The majority of patients with migraine and vertigo do not qualify for a diagnosis of basilar migraine as the vertigo attacks are often separate from the headaches and the duration of the vertigo often does not fall within the restrictions for aura (5-60 min) (1,6,19,24).

Migraine and cerebellar dysfunction

Dizziness can be as a result of Cerebellar dysfunction which causes imbalance. A rare subtype of migraine which is Familial hemiplegic migraine can develop cerebella ataxia which is progressive and can also have nystagmus. In FHM, episodic ataxia type 2 and spinocerebellar ataxia type 6,there are mutations identified in the CACNA1A gene which is coding for the a1A subunit of a neuronal Ca21 channel which is expressed in the cerebellum heavily. EA-2 is characterized by nystagmus which is interictal, cerebellar ataxia and vertigo. Half of the patients who have EA-2 have migrainous headache and FHM and EA-2 are associated with symptoms of basilar migraine. In more common types of migraine, Cerebellar

symptoms are not usually present in the more common type of migrainous headaches, but sub clinical hypermetria and other subtle sub clinical cerebellar signs in patients who have migraine with or without aura have been reported. The possible cause for this could be the gene mutation as mentioned above. This hypothesis relies on findings of involvement of the CACNA1A gene region in some families who have non hemiplegic migraine with and without aura. Another possible link between migrainous vertigo and cerebellar dysfunction was as a result of the mild occulomotor deficits of cerebellar origin observed in patients who have migrainous vertigo. (81,6).

Migraine and non vestibular dizziness

The prevalence of vertigo as well as dizzy spells in migraine patients is more compared to controls. The dizzy spells can be attributed to non vestibular causes. However mild vestibular dysfunction can also present as dizzy spells.

Migraine, orthostatic hypotension and syncope

It has been seen in studies that 5% of 500 migraine patients develop syncope during migraine attacks. In a large based population survey, the prevalence of syncope and orthostatic hypotension was found to be higher in migraine patients than in controls. However orthostatic hypotension can be caused dopamine agonists and can be counteracted by antagonists in migraine patients but not in controls. This suggests dopaminergic hypersensitivity as the underlying cause (83).

Migraine, dizziness and psychiatric disorders

Studies have shown that migraine, dizziness and certain psychiatric disorders are interrelated. It has been shown that migraine is a risk factor for major depression and panic attacks and vice versa and the association of migraine with these disorders have also been documented. Dizziness is the second most common symptom of panic attacks after palpitation as well as depression. Studies have shown there are vestibular tests abnormalities in patients with panic

and anxiety disorders suggesting that those with vestibular abnormalities have an increased risk of developing an anxiety disorder. As compared to other vertigo syndromes vestibular migraine patients show increased rates of concurrent anxiety and panic disorders. Because of this association, a new syndrome, migraine anxiety related dizziness has been proposed (84, 85).

Dizziness and anti migraine drugs

Dizziness is a side effect of many drugs used in the treatment of various conditions as well as for migraine. Thus a detailed drugs history should be asked for during history taking. Beta blockers can cause orthostatic hypotension initially and tricyclic antidepressants used in prophylaxis of migraine can cause orthostatic hypotension, light headedness and blurred vision.

MATERIALS AND METHODS

This study was carried out in the ENT Department's out-patient and in the Audio vestibular Clinic (AVC) of the Christian Medical College, Vellore, a tertiary health care facility.

SELECTION OF CASES

The first part of the study was to review the charts of the patients visiting Audio vestibular clinic in CMC for a period of six months, retrospectively. It is routine practice in the clinic that a preformatted questionnaire is used to record the history of vertigo patients and complete filling up of the questionnaire is strictly implemented. The details required to make a diagnosis of migraine has already been included in this questionnaire. In those records where a diagnosis of Migrainous vertigo had been made, demographic details were collected as well as information of the audiometric tests (pure tone audiometry and impedance audiometry).

The age and sex distribution of Migrainous vertigo was calculated from the data and the type of hearing loss in the patients was also calculated. The average hearing loss in the frequencies 500Hz, 1Hz, 2Hz at different age groups were calculated along with the frequency of Migrainous vertigo.

The second part of the study included thirty patients in whom a diagnosis of Migrainous vertigo was made using the same questionnaire. The inclusion and exclusion criteria for the subjects were as follows:

Inclusion Criteria

a. Age between 18 to 60yrs

b. All patients with Migrainous Vertigo

Exclusion Criteria

Any patients with typical history of BPPV (and a positive Hall pikes test)

Any patients with typical Meniere's disease (AAOC criteria 1995)

Any patient with past history of chronic discharging ears/history of surgery to ear/ history of permanent hearing loss.

Other neurological disorders such as stroke and intracranial tumours, anyone with prolonged noise exposure, history of ototoxic medications, otosclerosis or with a history of head or ear trauma, diabetes mellitus, hypertension or ischemic heart disease.

Relevant blood tests were done to do exclude patients with dyslipidemia, diabetes mellitus and hypothyroidism and anaemia. All those selected for the study patients underwent otoneurological investigations consisting of pure tone audiometry (PTA), impedance audiometry (IA), auditory brainstem response audiometry (BERA) and distortion product otoacoustic emissions(DPOAE). Thirty age matched normal controls were also selected and the additional tests were also done on them to compare the inter-peak and absolute latencies and presence or absence of otoacoustic emissions with that of the subjects.

PROCEDURE

PTA was done using a GSI-Grason Stadler diagnostic audiometer. Hearing thresholds of 15-25 dB across the frequencies 250 Hz -8000 Hz were considered normal. The Glason Stadler GSI Tympstar impedance audiometer was used to obtain immittance measurements. The test was performed using a probe with tone frequency of 226 Hz. An ipsilateral stapedial reflex at 1000 Hz was elicited. The ipsilateral acoustic reflex threshold was taken as normal if the level at which it was elicited fell between 70 dB and 100 dB.

DPOAE were tested using the INTELLIGENT HEARING SYSTEMS. It was recorded using a test protocol where preliminaries were fixed at L1 equalling 65 dB SPL, L2 equalling 55 dB SPL with an f2/f1 ratio of 1.22. The f2 frequencies were carefully selected to correspond closely to audiometric test frequencies of 1000 Hz, 2000 Hz and 4000 Hz. The testing was done at 1000 Hz, 2000 Hz, 3000 Hz, 4000 Hz and

6000 Hz. A response is considered to be present if the SNR is more than 6.13 dB. The DPOAE was recorded with amplitude in dB SPL as a function of stimulus frequency. This is commonly called as a DPOAE gram. The f2 frequencies are presented on the horizontal axis and amplitude of the DPOAE for the different frequencies is plotted on the vertical axis.

Brain stem evoked response audiometry (BERA) was done using INTELLIGENT HEARING SYSTEM launch pad version 2.32 X. Smart EP software under Microsoft windows version XP 2002 and is primarily controlled by the mouse with push buttons and menus.

CASE DEFINITIONS

1. Normal hearing: pure tone average 500 Hz, 1000 Hz and 2000 Hz with bone conduction threshold less than 25 dB and air conduction thresholds at 20 dB.
2. Mild sensory neural hearing loss: pure tone average 500 Hz,1000 Hz and 2000 Hz with bone conduction between 26-40 dB
3. Moderate sensory neural hearing loss: pure tone average 500 Hz,1000 Hz and 2000 Hz with bone conduction between 41-55 dB
4. Moderately severe sensory neural hearing loss: pure tone average 500 Hz,1000 Hz and 2000 Hz with bone conduction between 56-70 dB
5. Severe sensory neural hearing loss: pure tone average 500 Hz,1000 Hz and 2000 Hz with bone conduction between 71-90 dB
6. Profound sensory neural hearing loss: pure tone average 500 Hz,1000 Hz and 2000 Hz with bone conduction more than 90 dB
7. Conductive hearing loss-when air bone gap is more than 25 dB
8. Poor wave form morphology on auditory brainstem response testing: waveforms showing prolongation of inter peak latencies i. e wave I to V delay more than 3.84

milliseconds with standard deviation of 0.16 or abnormal amplitude ratios. The normal wave latencies are shown below.

90 DB nHL	Mean (milliseconds)	Standard deviation
Absolute latency Wave 1	1.54	0.09
Absolute latency Wave III	3.6	0.12
Absolute latency Wave V	5.43	0.14
Interpeak latency wave1-III	2.06	0.14
Interpeak latency wave III-V	1.82	0.14
Interpeak latency of wave I -V	3.85	0.16

(n –reference to the normative group threshold click stimulus)

9. The presence of oto acoustic emissions present was taken as the presence of true validated DPOAE and it means that normal cochlear function is present at the frequency of stimulus

10. DPOAE absent was taken as absence of otoacoustic emissions over a more restricted frequency range, at least third of an octave indicating abnormal outer hair cell function (86).

11. Impedance audiometry consisting of two components Tympanometry and acoustic reflex- A type curve indicates a normal tympanometric graph and indicates normal middle ear function.

Statistical methods:

The retrospective part of the study was descriptive and provided statistics on what had been observed in the patients who were tested in the clinic.

The prospective part of the study involved analysis of the absolute and inter-peak latencies of patients with migraine related vertigo compared to the standard normal.

Sample size: About 400 patients are seen every year with Migrainous Vertigo in the Audio Vestibular Clinic.

Other studies indicate that abnormalities of the auditory pathway may be found from 36 to 56% among patients with Migrainous Vertigo (2,55).

Taking a conservative estimate that 20% of patients in the Audio vestibular clinic with Migrainous Vertigo would have abnormalities of the auditory pathway, a sample size of 88 was taken with 44 patients with Migrainous vertigo and 44 age matched controls to provide an estimate that could range from 15% to 25% with a 95% confidence.

RESULTS AND ANALYSIS

There were two parts to this study.

The first part(retrospective chart review)

The first was a chart review of 400 patients' charts who had visited the audio vestibular unit over a period of 6 months.

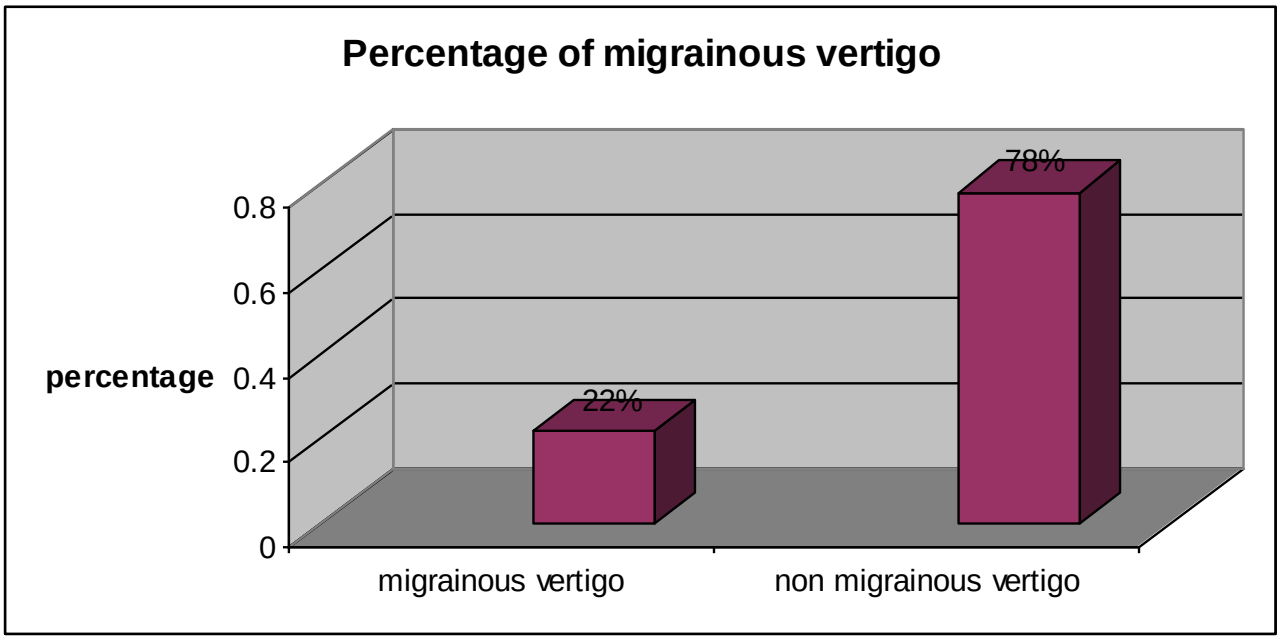
Of the 400 charts reviewed, 363 patients (90.8%) presented with chief complaints of vertigo and 37 (9.3%) presented with complaints other than vertigo.

Among the 363 patients who presented with vertigo, 283 had complaints of vertigo other than migrainous vertigo, 80 (22%) (Table1).

Table 1. Presentation of vertigo among the charts reviewed

Presentation of vertigo	Number of patients	Percent
Migrainous vertigo	80	22%
Non-migrainous vertigo	283	78%
Total	**363**	**100%**

Fig 1.Percentage of migrainous vertigo patients seen in clinic

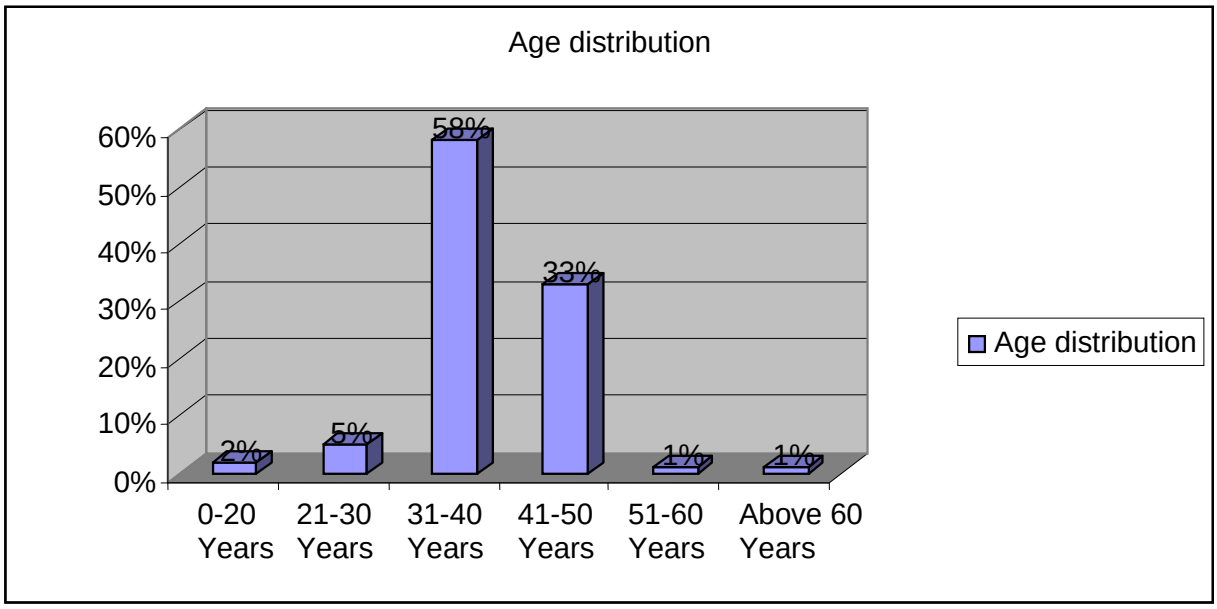


Age breakdown showed that ninety percent were between the ages 31 and 50 (Table 2,fig 1). Most number of patients were found in age group 31-40(58%), followed by age group,41-50(33%).

Table 2-Age distribution of migrainous vertigo patients

Age groups (years)	Number (%)
0-20	2 (3)
21-30	4 (5)
31-40	46 (58)
41-50	26 (33)
51-60	1 (1)
Above 60	1 (1)
Total	**80 (100)**

Fig 2-Age distribution of Migrainous vertigo patients



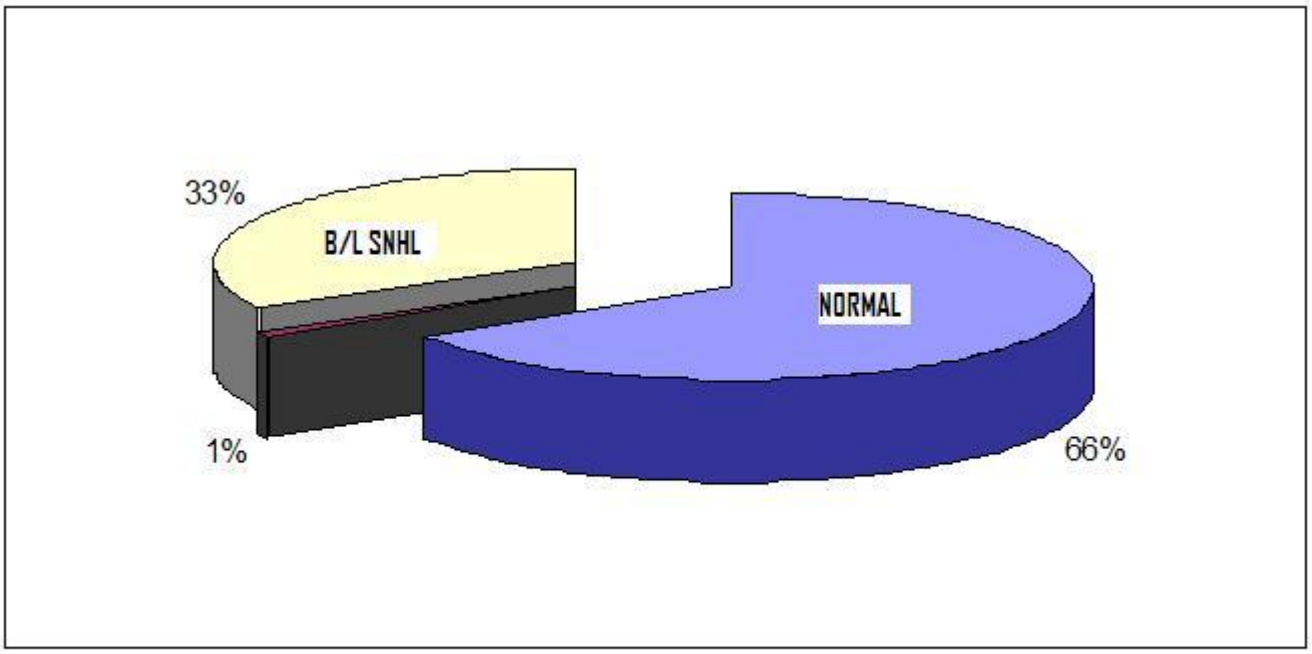
The sex distribution showed 44 (55%) were females and 36 (45%) were males. Most of

the patients with a diagnosis of migrainous vertigo had normal hearing (53 of 80); 26 of 80 had bilateral mild sensorineural hearing loss (SNHL) and one patient had unilateral mild sensorineural hearing loss (Table 3).

Table 3. Type of hearing loss in migrainous vertigo

Hearing loss	**Number (%)**
Normal hearing	53 (66)
Unilateral mild SNHL	1 (1)
Bilateral mild SNHL	26 (33)
Total	**80 (100)**

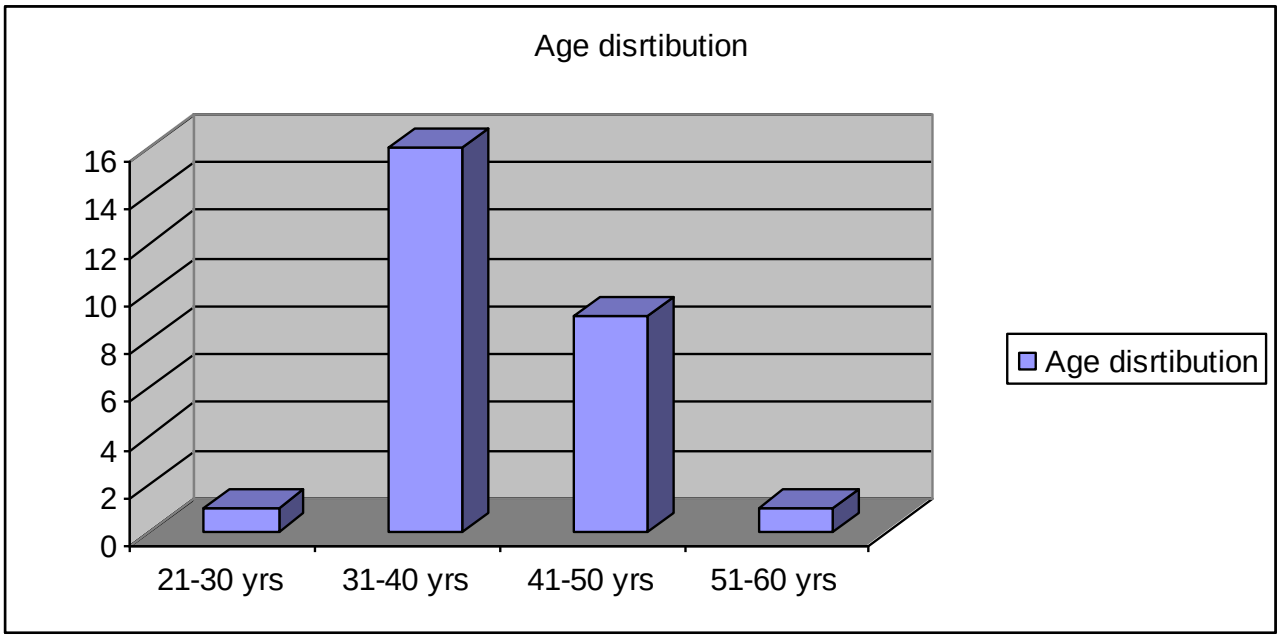
Fig 3.Type of hearing loss in Migrainous vertigo



66%-patients with normal hearing, 33%-patients with mild sensory neural hearing loss,1%-patient with unilateral sensory neural hearing loss.

Of the twenty seven patients who presented with mild sensorineural hearing loss 16 were in the age group of 31-40 years; 9 were in the 41-50 years age group and one each was in the 21-30 and 51-60 years age groups.

Fig 4. Age categories and number of patients with hearing loss



Second part of the study:(prospective case controlled)

Table 4 shows the distribution of age of Migrainous patients and controls. Most of the patients and the controls were in the age groups 20 to 40 years(80% and 93% respectively)

Table 4. Age distribution of cases and controls

Age Years	Cases No. (%)	Controls No. (%)
20-30	8 (27)	18 (60)
31-40	16 (53)	10 (33)
41-50	6 (20)	2 (7)
Total	30	30

There were 11 males and 19 females in the study group while there were 16 males and 14 females in the control group.

The ENT and otoneurological examination in all patients and controls were normal.

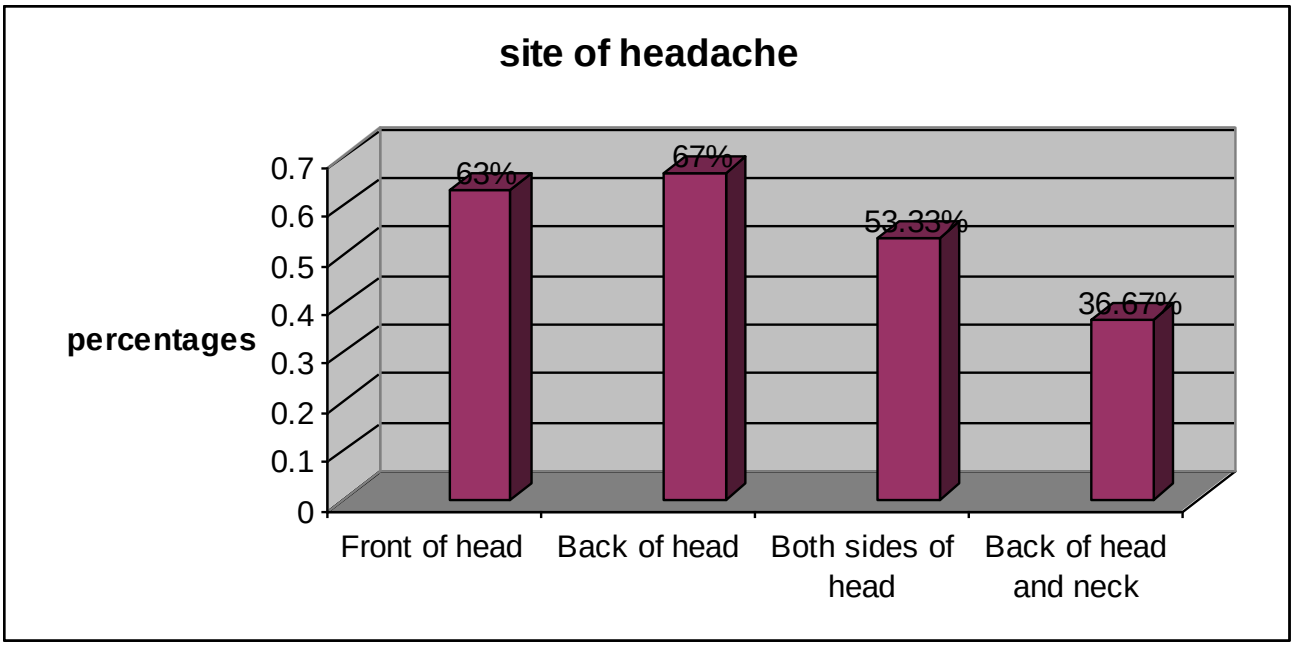
HEADACHE AND ITS DESCRIPTION AMONG THE CASES SELECTED:

All the thirty patients had headache during their lifetime. For 27, the attacks were similar in nature each time (identical). Tables 5 shows the localisation of the headache. Since the choices are not mutually exclusive, each patient could have had more than one response and percentages will add up to more than 100. The most frequent site of headache was at the occipital region.

Table 5. Headache and pain in the neck and its localisation

Site of headache	Number (%)
Front of head	19(63.33%)
Back of head	20(66.67%)
Both sides of head	16(53.33%)
Back of head and neck	11(36.67%)

Fig 5.Headache and its localisation



Twenty one patients had headache more than 11 years; and 9 had headache for 10 years or less. The duration of an average attack of headache lasted for an hour or less in 17 patients, 1 to 2 hours in 12 and in one, for more than 2 hours (Table 6). A majority (83%) had two or more attacks of headache a month (Figure 4).

Table 6. Duration of an average attack of headache

Hours	Number (%)
0-1	17 (57)
1-2	12 (40)
>2	1 (3)

Fig 6.Duration of attacks

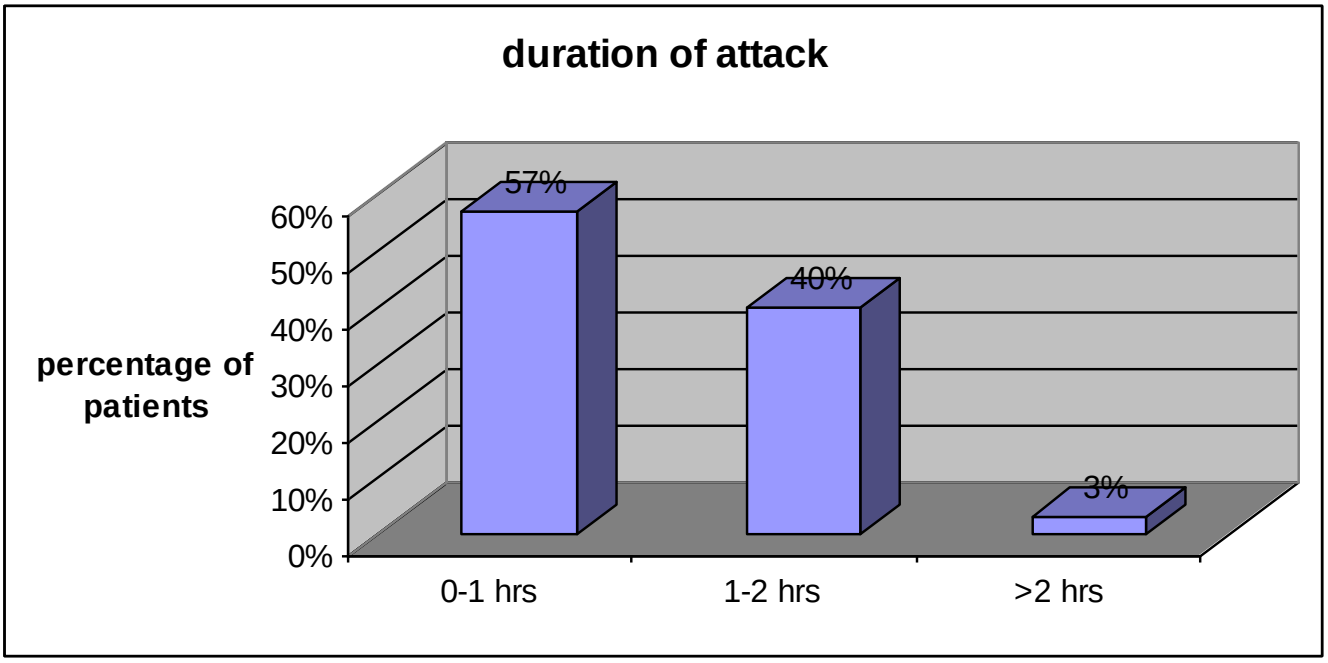
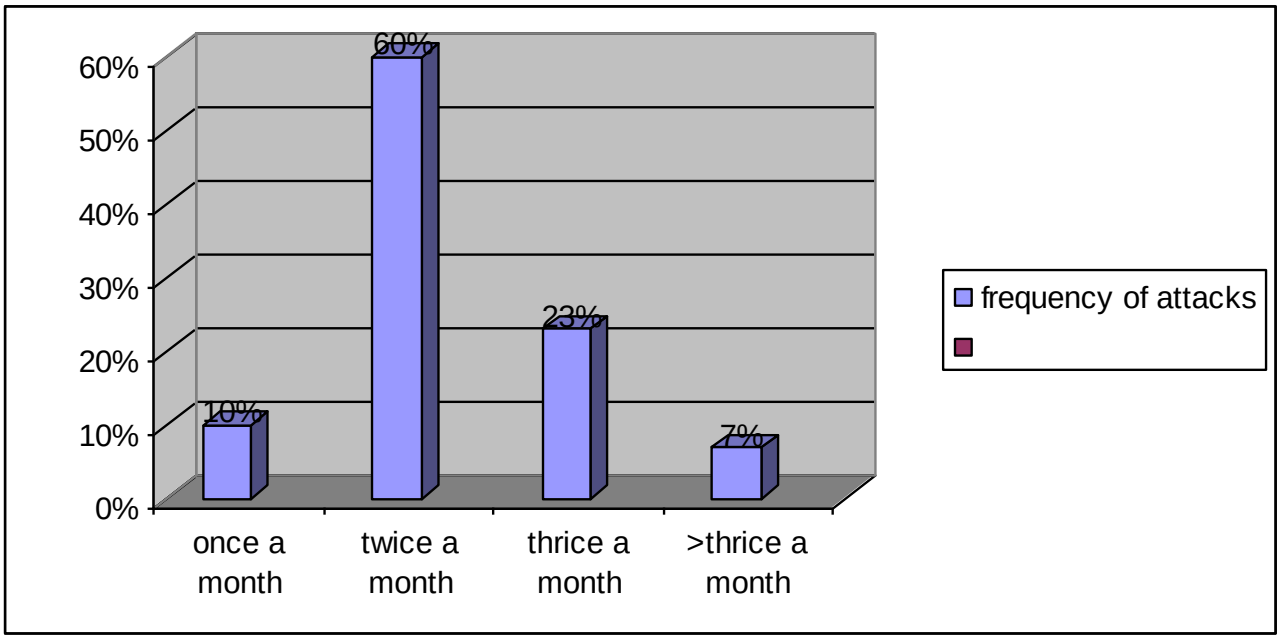


Fig 7-frequency of attacks



All 30 experienced a throb during attacks of migraine while 9 had dizzy spells when they did not have a headache. Nine had positional vertigo and 10 had motion sickness

Fig 8 .percentage of patients having positional vertigo

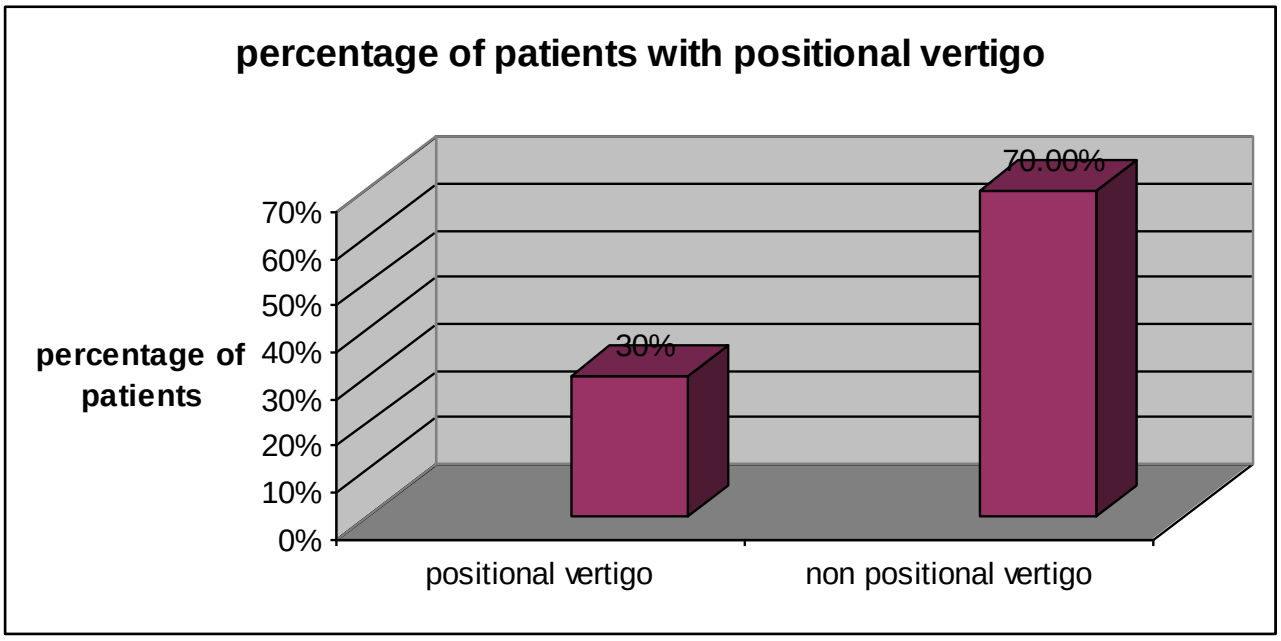
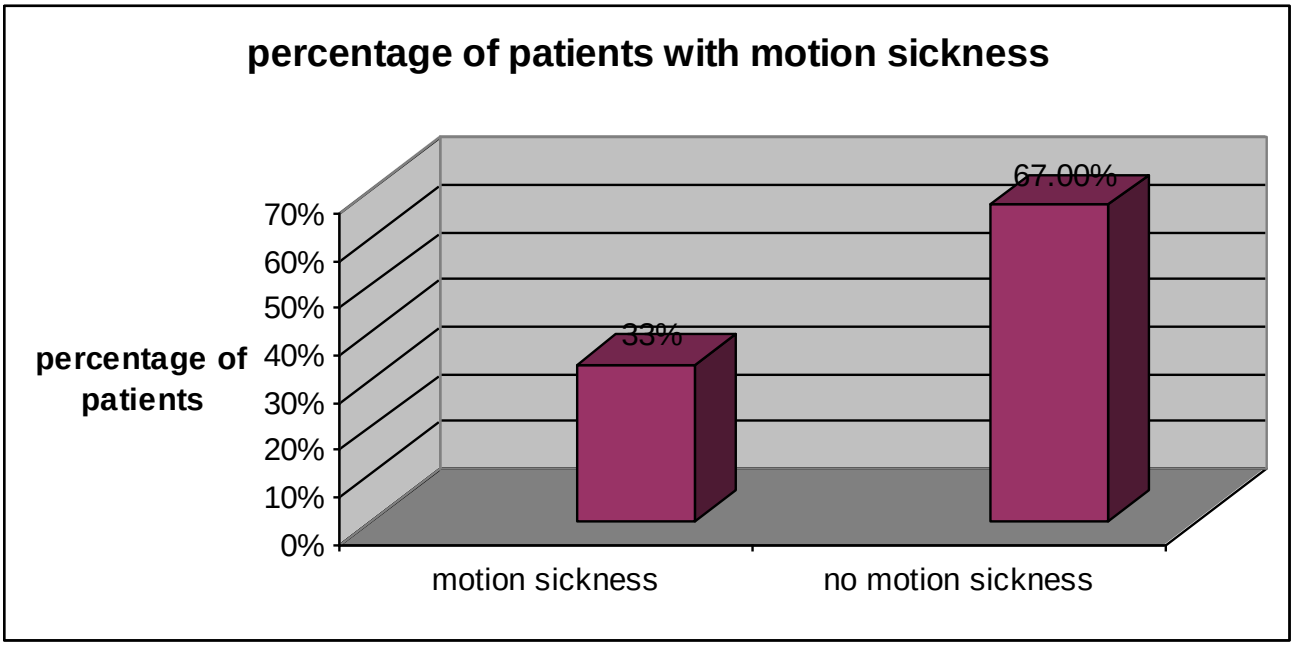
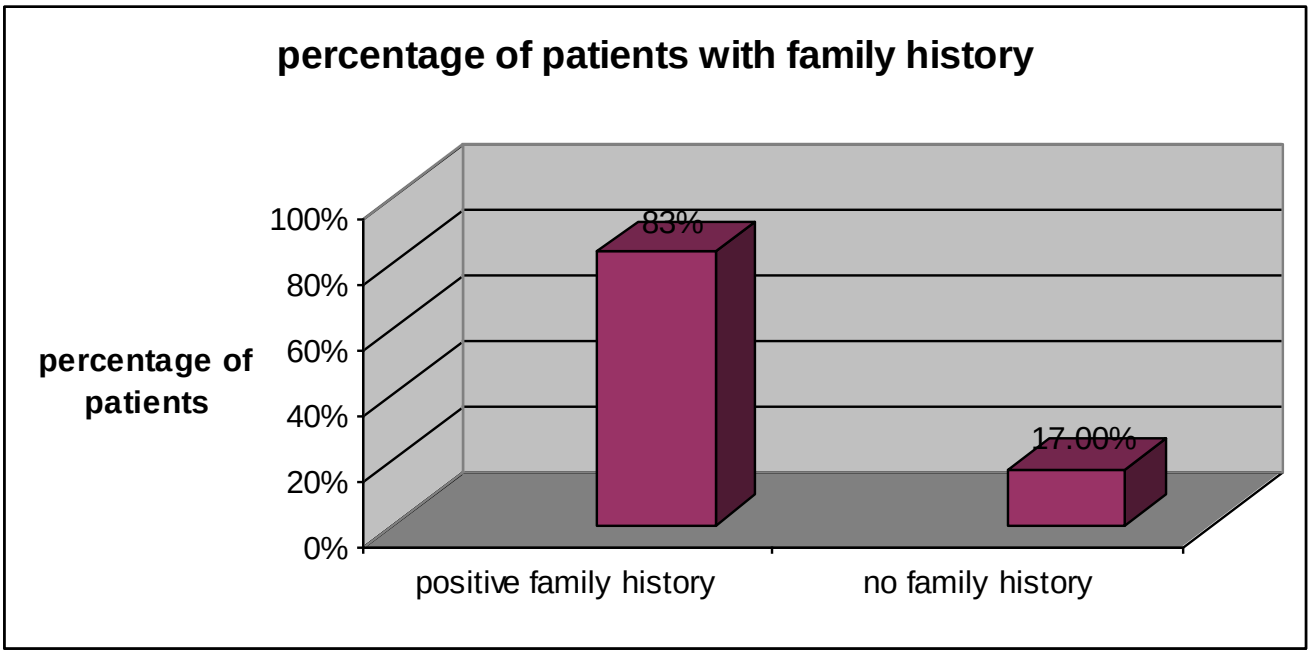


Fig 9.Percentage of patients having motion sickness



Headache in 25 patients was relieved by paracetamol, in 3 it settled on its own and in 2 by other measures.25 out of 30 patients had a positive family history of Migrainous headaches.

Fig 10.Percentage of patients with positive family history



PATIENTS EXPERIENCING MIGRAINE WITH AURA

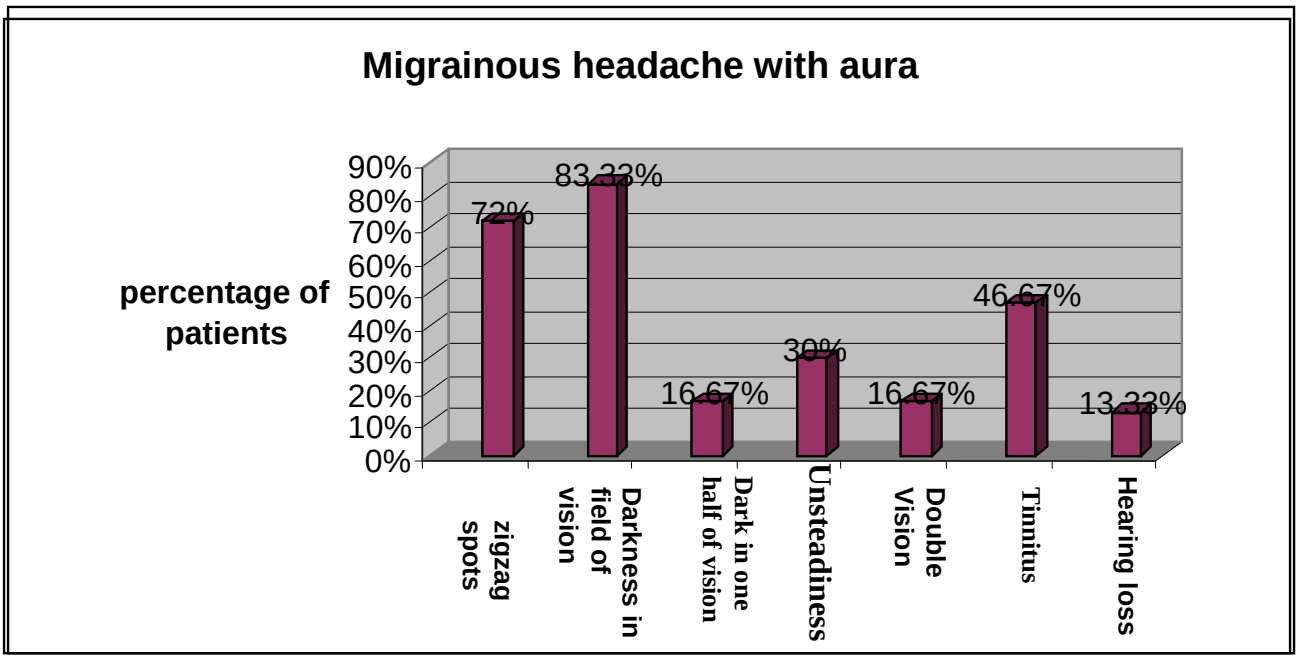
Among the 30 patients, all experienced aura during the attack of headache or an hour before or after the attack of headache. At least one of the brain stem symptoms like unsteadiness, , zigzag lights in vision, darkness in vision and double vision was experienced by all the patients tinnitus and hearing loss .

Eighteen experienced zigzag spots in their field of vision, 25 experienced dark areas in the centre of field of vision, 5 experienced darkness in one half of field of vision.

All patients experienced dizzy spells, unsteadiness was experienced by nine, five experienced double vision as aura during the attack of headache, 14 experienced noises in the ear as an aura, 4 experienced loss of hearing during the attack of migraine as an aura. None of the patients experienced episodes of loss of consciousness or black outs (Figure 5)

.

Fig 11.Symptoms of aura present in patients with Migrainous vertigo



ASSOCIATION OF HEADACHE WITH OTHER SYMPTOMS

All patients experienced nausea with the attack of headache while 28 had both nausea and vomiting, two experienced only nausea. Thirteen of the 30 experienced increased sensitivity to bright lights or photophobia and 13 had increased sensitivity to loud sounds (phonophobia). Five experienced disturbances in smell associated with headache. The dizzy spells of 16 out of 30 patients of Migrainous vertigo were accompanied by auditory symptoms like tinnitus and decreased hearing and phonophobia. Five out of the 30 patients with Migrainous vertigo had olfactory symptoms and 16 had auditory symptoms with vertigo during headache free intervals.

Fig 12. percentage of patients experiencing photophobia

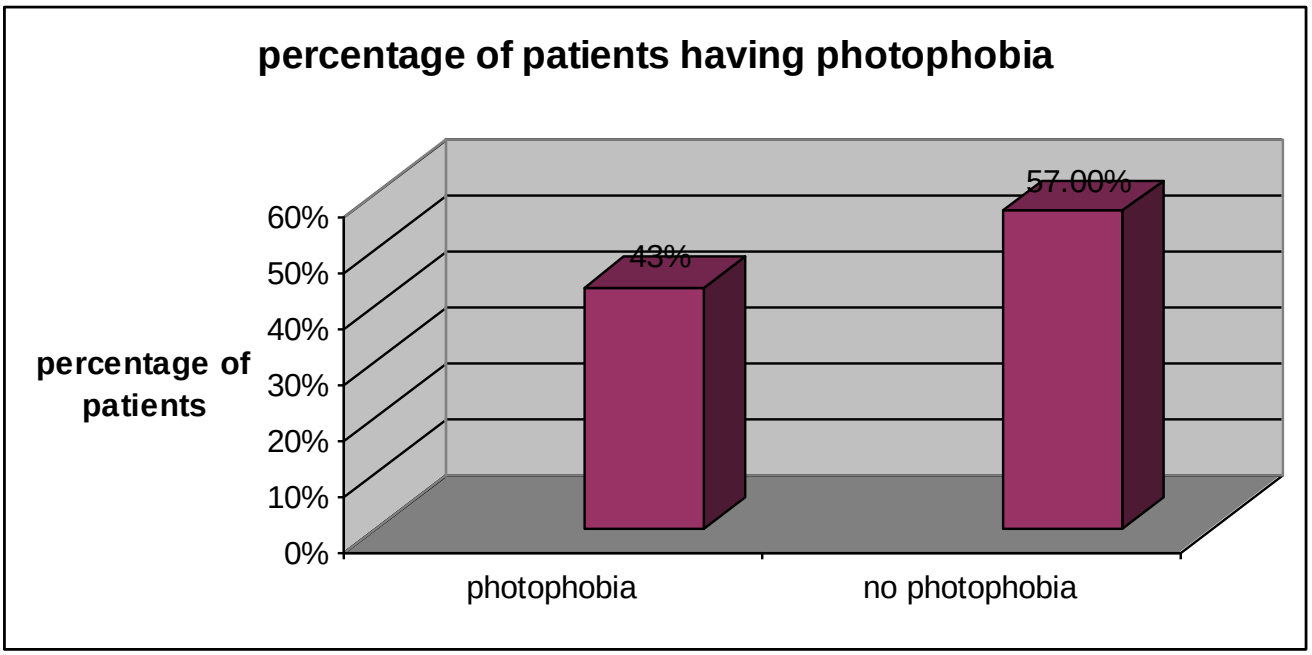


Fig 13.percentage of patients having phonophobia

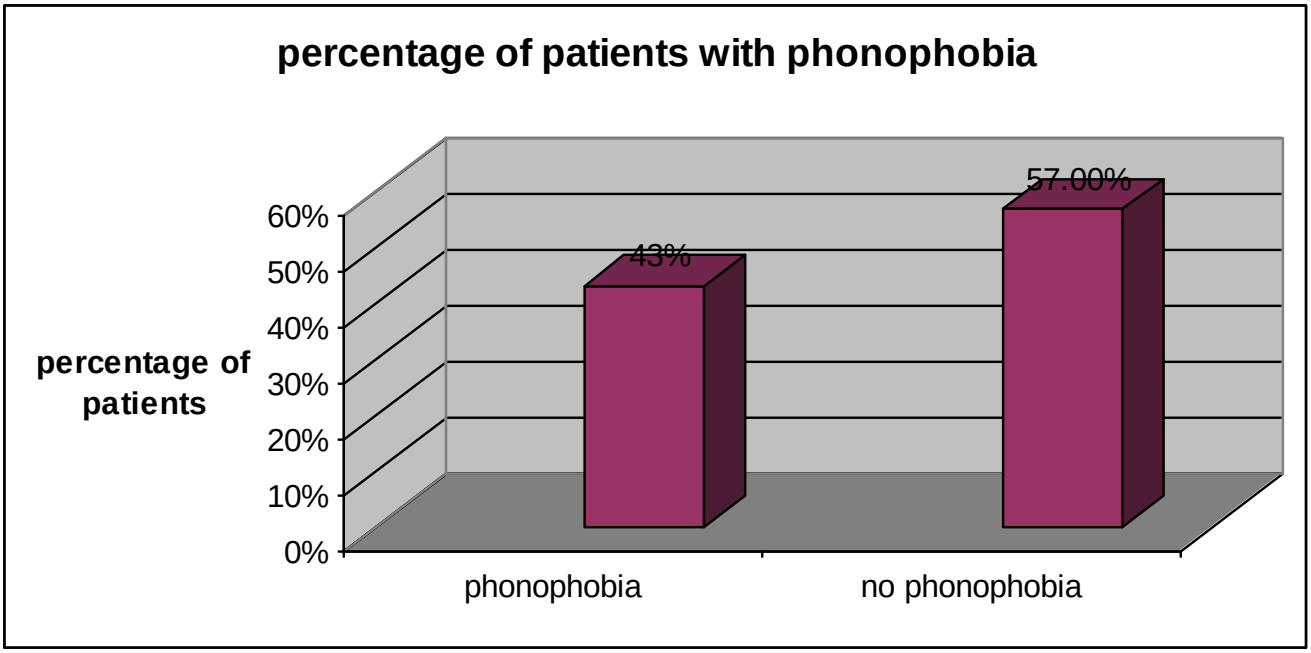

percentage of patients with phonophobia
percentage of patients
60%
50%
40%
30%
20%
10%
0%
43%
57.00%
phonophobia
no phonophobia

Fig 14.percentage of patients experiencing auditory symptoms

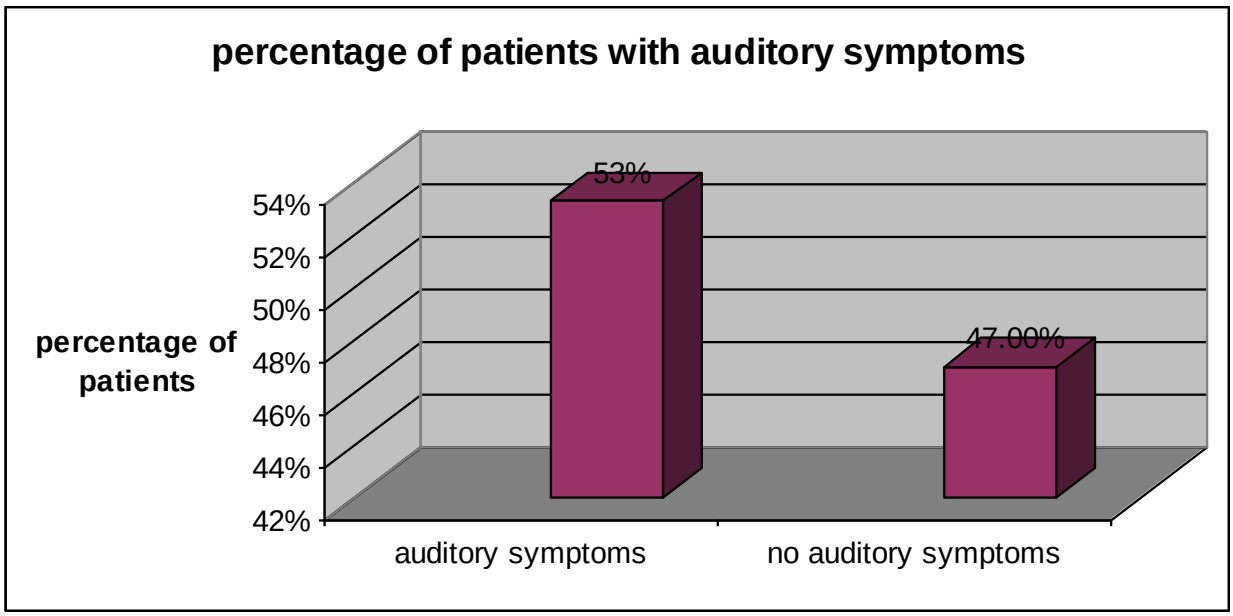
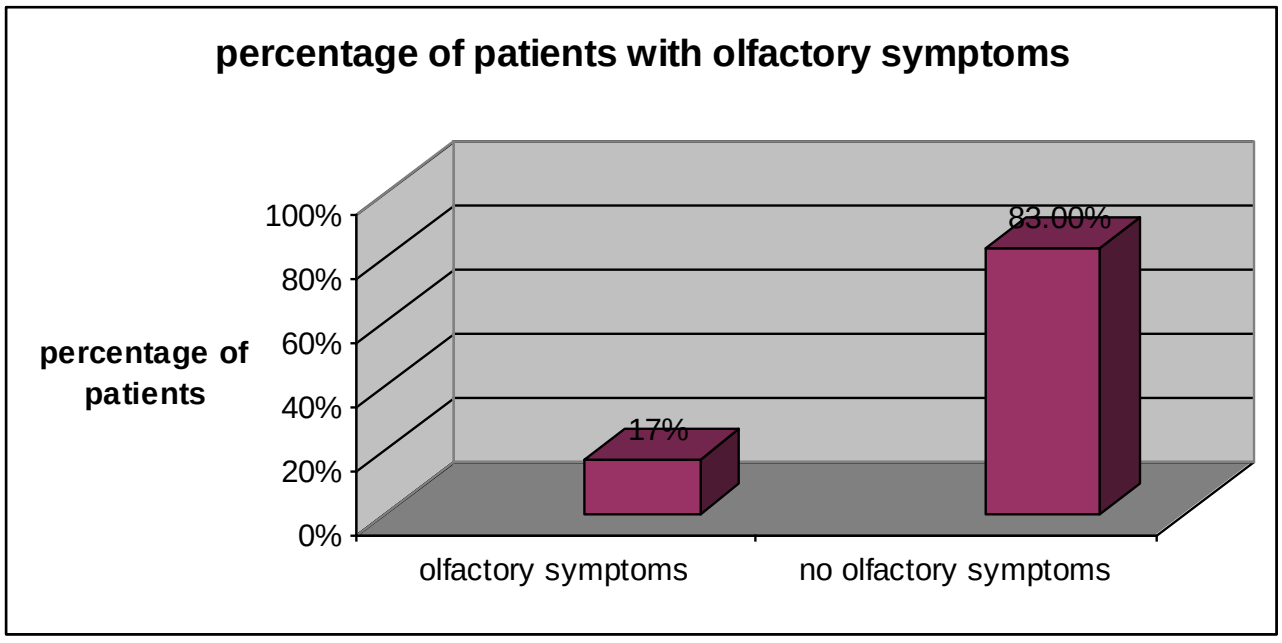


Fig 15. Percentage of patients with olfactory symptoms



PHYSICAL EXAMINATION AND AUDIOLOGICAL INVESTIGATIONS

The ENT and otoneurological examination in all 30 patients were normal.. All thirty patients underwent a pure tone audiometry (PTA), impedance audiometry(IA), distortion product otoacoustic emissions(DPOAE) and brainstem evoked response audiometry(BERA).

Out of the 30 patients 10 had abnormal PTA and 20 had normal PTA

All ten except one patient had bilateral mild sensorineural hearing loss; one patient had unilateral mild sensorineural hearing loss.

Tympanometry done to assess the middle ear function, showed normal A type curve in all patients. The stapedial reflux was present in all the 30 patients except one.

The tables 7 to 9 show the test results in migrainous patients with and without auditory symptoms There were no significant differences between the two groups for these tests.

Table 7: PTA results in migrainous patients with and without auditory symptoms

Auditory symptoms (tinnitus, hearing loss and phonophobia)	PTA abnormal (%)	PTA normal (%)
Present (n=16)	7 (44)	9 (56)
Absent (14)	3 (21)	11 (79)
Total 30	10	20

Chi.Sq=0.8203; df=1; p=0.3651

Table 8. Auditory symptoms and BERA results

Auditory symptoms (tinnitus, hearing loss and phonophobia)	BERA abnormal (%)	BERA normal (%)
Present (n=16)	14 (87.5)	2 (12.5)
Absent (14)	12 (86)	2 (14)
Total 30	26	4

Chi.Sq=0.1558; df=1; p=0.6930

Table 9. Auditory symptoms and OAE results

Auditory symptoms (tinnitus, hearing loss and phonophobia)	BERA abnormal	BERA normal
Present (n=16)	6 (37.5)	10 (62.5)
Absent (14)	5 (35.7)	9 (64.2)
Total 30	11	19

Chi.Sq=0.0775; df=1; p=0.7807

The tables 10 shows DPOAE results in migrainous patients with normal and abnormal PTA.

. There were no significant difference between the two groups for these tests

Table 10 Status of OAE in patients with normal and abnormal pure tone audiometry

Audiogram (PTA)	DPOAE absent	DPOAE present
Normal (20)	7 (35)	13 (65)
Abnormal (10)	4 (40)	6 (60)
Total (30)	11	19

Chi.Sq=0.0179; df=1; p=0.8934

The table 11 shows the BERA test results in migrainous patients with normal and abnormal PTA . There were no significant difference between the two groups for these tests

Table 11. Status of BERA in patients with normal and abnormal pure tone audiometry

Audio	ABR normal	ABR abnormal
Normal (20)	3 (15)	17 (85)
Abnormal (10)	1 (10)	9 (90)
Total (30)	4	26

Chi.Sq=0.0361; df=1; p=0.8494

All thirty patients underwent BERA and DPOAE testing. The values of these tests were compared with that of 30 normal controls.

Table 12 and 19 show the number of patients with normal and abnormal DPOAE in the right ear and left ear respectively in patients and controls. This difference was statistically significant in both ears.

Table 12. OAE Right ear Differences between cases and controls

OAE results		Cases	Controls	Total
Right ear	Normal	22	29	51
	Abnormal	8	1	9
Total		30	30	60

Chi sq 6.4, P=0.011*

Table 13. OAE Left ear Differences between cases and controls

OAE Results		Cases	Controls	Total
Left ear	Normal	20	29	49
	Abnormal	10	1	11
Total		30	30	60

Chi sq 9.01, P=0.003*

Tables 14 to 18 show the number of patients with normal and abnormal BERA absolute latencies of waves I, III, and V in the right ear and left ear in patients and controls. The difference in absolute latency of wave V in the right ear was statistically significant. Difference in all other absolute latencies did not reach significance.

Table 14: Number of patients and controls with normal and abnormal absolute latency of wave I in the right ear and the test of significance

Absolute latency of wave 1,right ear	Number of patients/controls	Mean	Std. Deviation	Std. Error Mean
Case	30	1.5320	.09704	.01772
Control	30	1.5597	.16166	.02952

t= -0.804, p=0.425

Table 15: Number of patients and controls with normal and abnormal absolute latency of wave III in the right ear and the differences

Absolute latency of wave III,RIGHT EAR	N	Mean	Std. Deviation	Std. Error Mean
Case	30	3.5243	.13431	.02452
Control	30	3.6017	.19761	.03608

t= - 1.733, p=0.082

Table 16: Number of patients and controls with normal and abnormal absolute latency of wave V in the right ear and the differences.

Absolute latency of wave V ,right ear	N	Mean	Std. Deviation	Std. Error Mean
Case	30	5.3257	.22337	.04078
Control	30	5.4520	.25600	.04674

t= - 2.03, p= 0.046 *

Table 17: Number of patients and controls with normal and abnormal absolute latency of wave I in the left ear and the diferences

Absolute latency of wave I ,left ear	N	Mean	Std. Deviation	Std. Error Mean
Case	30	1.537	.1056	.0193
Control	30	1.538	.1315	.0240

t= - 0.032, p= 0.974

Table 18: Number of patients and controls with normal and abnormal absolute latency of wave III in the left ear and the differences.

Absolute latency wave III, Left ear	N	Mean	Std. Deviation	Std. Error Mean
Case	30	3.5600	.15779	.02881
Control	30	3.6080	.18376	.03355

t= - 1.085, p= 0.282

Table 19: Number of patients and controls with normal and abnormal absolute latency of wave V in the left ear and the differences.

Absolute latency wave V left ear	N	Mean	Std. Deviation	Std. Error Mean
Case	30	5.3733	.20913	.03818
Control	30	5.4130	.24785	.04525

t= - 0.670, p= 0.506

Tables 20 to 25 show the number of patients with normal and abnormal BERA inter peak latencies of waves I- III, III-V and I-V in the right ear and left ear in patients and controls. The difference in inter-peak latencies of all the waves were not statistically significant

Table 20: Number of patients and controls with normal and abnormal inter peak latencies of waves I- III, right ear and the differences.

Inter peak latency wave I-III, right ear	N	Mean	Std. Deviation	Std. Error Mean
Case	30	1.9980	.11223	.02049
Control	30	2.0503	.13556	.02475

t= - 1.629, p= 0.109

Table 21: Number of patients and controls with normal and abnormal inter peak latencies of waves III-V in right ear and the differences.

Interpeak latency wave III-V, Right ear	N	Mean	Std. Deviation	Std. Error Mean
Case	30	1.803	.1481	.0270
Control	30	1.845	.1238	.0226

t= - 1.173, p= 0.24

Table 22: Number of patients and controls with normal and abnormal inter peak latencies of waves I- V, right ear and the differences.

Interpeak latencies wave I –V, Right ear	N	Mean	Std. Deviation	Std. Error Mean
Case	30	3.8007	.18629	.03401
Control	30	3.8303	.37660	.06876

t= - 0.387, p= 0.700

Table 23: Number of patients and controls with normal and abnormal inter peak latencies of waves I- III, left ear and the differences.

Interpeak latencies wave I To III, Left ear	N	Mean	Std. Deviation	Std. Error Mean
Case	30	2.0257	.11337	.02070
Control	30	2.0713	.10624	.01940

t= - 1.610, p= 0.113

Table 24: Number of patients and controls with normal and abnormal inter peak latencies of waves III-V, left ear and the differences.

Interpeak latencies wave III to V ,Left ear	N	Mean	Std. Deviation	Std. Error Mean
Case	30	1.8087	.11548	.02108
Control	30	1.8063	.12907	.02356

t= 0.074, p= 0.941

Table 25: Number of patients and controls with normal and abnormal inter peak latencies of waves I- V, left ear and the differences.

Interpeak latency wave I-V, left ear	N	Mean	Std. Deviation	Std. Error Mean
Case	30	3.8310	.17639	.03220
Control	30	3.8720	.16890	.03084

t= - 0.920, p= 0.362

DISCUSSION

This study was done in a tertiary care hospital to ascertain the frequency of migrainous vertigo by reviewing the charts of patients who visited the Audio vestibular clinic for various complains retrospectively over a period of 6 months and to evaluate the auditory status of diagnosed cases of Migrainous vertigo prospectively over a 6 month period.

In the first part of the study which was a retrospective chart review, the frequency of migrainous vertigo among the patients visiting the audio vestibular clinic was 20% (80/400), and of the patients presenting with vertigo the frequency of migrainous vertigo was 22.04% (80/363). Both these values are higher than that reported in literature (7%) (19).

The age group at which migrainous vertigo was most common, was found to be between 31 to 40 years(46/80) followed by 41 to 50 years(26/80). In literature migrainous vertigo can occur at any age group (6,7,19). The female to male ratio in our study was 1.22 is to 1 which is similar to that reported in Literature (6,7,19).

A frequency of 27/80 (21%) patients were found to have documented evidence of hearing loss in our chart review and this is far higher than that reported by Dash et al (8) which is 3.5%. He has however also noted that majority of the patients with migrainous vertigo have hearing loss that may be transient and non progressive (8). The type of hearing loss noted by us was mild as mentioned in his study.

In the review of charts the age group at which hearing loss occurred maximum was found to be from 31 to 40 years (16/27). This could be due to the number of patients of migrainous vertigo were mostly in the same age group.

In the second part of the study, which was prospective with controls, of the 30 diagnosed with migrainous vertigo, 19 were females and 11 were males, the ratio being 1.73 which is similar to the ratio in literature (6,7,19).

Of the 30 patients, the maximum numbers of patients were seen in the age group 31-40 years as seen also in the retrospective case review carried out as first part of this study. Patients below 18 years were not included in the study as patients below this age usually attended paediatric ENT. Patients above 60 years were not included in the study to minimise the effect of other co-morbid conditions.

In this study the duration of each attack of headache was most commonly found to be less than one hour (17/30), followed by 1-2 hours (12/30). According to literature the most common duration is also between 5-60minutes (33%) followed by 1-24 hours (21%). Thus the duration is similar to that given in literature (19).

Thirty percent of patients in this study, experienced dizzy spells outside the period of headache, while in literature up to half (48%) can experience dizzy spells outside the period of headache (19).

In this study, 30% of patients experienced positional vertigo, a little less than what we found in literature 42% (19). Motion sickness was found in 33% of patients and again less than what we found in literature 48% (19). Unsteadiness was experienced by 30% of patients and double vision by 17% of patients in this study. Dash et al have reported transient unsteadiness can occur in 16% of the patients with Migrainous vertigo (8).

In migrainous vertigo, the most common vestibular symptom besides vertigo was motion sickness (33%) followed by positional vertigo (30%) which is similar to Neuhauser et al (19).

Auditory symptoms occurring in migrainous vertigo is considered less common than vestibular symptoms (18). In this study, tinnitus was experienced by 47% of patients as an aura during headache, while during the attack of migrainous vertigo, 53% of patients experienced auditory symptoms like hearing loss and tinnitus. Phonophobia was experienced by 43% of patients in our study.

Thirteen percent of patients experienced hearing loss alone as an aura during the attack of headache. The most common auditory symptom in this study was hearing loss with tinnitus, (53%), followed by tinnitus alone (47%), phonophobia (43%) and hearing loss alone (13%). In the study by Dash et al (8), the most common auditory symptom was phonophobia (70%), followed by tinnitus (50%). This was in discordance with our study.

In this study, 43% of patients experienced photophobia while in Neuhauser et al's study (19), 70% of patients experienced photophobia. Visual auras were experienced by all patients in our study while in Neuhauser et al's study, 36% experienced visual auras and auras other than vertigo. Sometimes these may be the only symptoms the patients present with which could help link between vertigo and headache and should be specifically asked for.

In our study 83% of patients had a positive family history of Migrainous headache. In literature it is mentioned that migraine and vertigo were found in familial hemiplegic migraine and episodic ataxia type 2 (35). However a susceptibility gene for the same has not yet been identified (36,37).

In all 30 patients, aura was experienced within one hour of headache. At least one of the brain stem symptoms other than vertigo like unsteadiness, zigzag lights in vision, darkness in vision and double vision, hearing loss and fullness of the ear, tinnitus was experienced by all the patients. This suggests that the commonest involvement in migrainous vertigo may be in the region of brainstem and posterior circulation territory of the brain.

The physical examination was normal in all 30 patients similar to what is known (7).

Of the thirty patients who underwent a pure tone audiometry, 10 (33%) had documented mild sensory neural hearing loss. This value is much higher than that found in Dash's study (8) which was is 14%. 11 patients had absent oto-acoustic emissions in either right or left ear.

We found a larger proportion of patients (26 patients out of 30) had abnormal values in either absolute latency or inter-peak latency in one or both ears compared to the study by Dash et al (8). which had 7 out of 38 patients (18%) had documented evidence of mild sensory neural hearing loss. However in his study all patients had abnormalities in BERA intervals in either right or left ear.

This prospective case controlled study conducted in a tertiary care specialised clinic showed a higher frequency of involvement of auditory pathway than what is reported in literature. From the battery of tests done it is suggestive that the involvement of the central auditory pathway may be earlier than the peripheral pathway as evidenced by abnormalities in BERA in all patients compared to PTA and DPOAE which were abnormal in 10 and 11 patients respectively. There is very little indication of involvement of middle ear function. A search of English literature revealed only one study which highlights this aspect of migrainous vertigo. The use BERA testing in patients with migrainous vertigo would help to pick up early involvement of auditory pathway and these patients if started on prophylactic anti-migraine treatment could prevent progression of auditory involvement in this condition.

Limitations of the study

This study was not powered to bring out differences between cases and controls for the various tests performed. A larger sample size may have provided results that were different. One reason for this smaller sample was the cost involved in recruiting more normal controls. The sample size is smaller than required. This was because of the cost factor involved in recruiting more normal controls.

The matching of patients could have been closer than what was done. Often the recruitment had to depend on the availability of cases that fulfilled selection criteria and time.

CONCLUSIONS

The frequency of migrainous vertigo in a tertiary care specialised audiovestibular clinic was 22%.

The largest age group attending tertiary care for this problem were in age group 31-40 (58%),

The male: female ratio was 1:1.2.

The frequency of hearing loss was 34% among 400 charts screened retrospectively.

Symptoms pointing to posterior circulation territory involvement were present as an aura in all patients.

33% percent of patients had documented bilateral mild sensory hearing loss.

Tympanometry and stapedial reflex were normal in all patients

There was significant difference between cases and control in the presence of distortion product otoacoustic emissions in both ears as well as in the absolute latency of wave V signifying the involvement of auditory pathway in migrainous vertigo.

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1. PROFORMA FOR MIGRAINOUS VERTIGO

Serial no-
Name-
Date-
Hospital Number-
Age-
Sex-

1)Are you or have you been subject to headache? Yes no
2)Do your headaches come in more or less identical attacks? Yes no
3)Are your headaches
a.at the front yes no
b.on both sides of your head yes no
c.at the back of your head yes no
d.in your neck yes no
4)how old were you when the headache started?
5)how long does the headache last on an average?
6)how often do you get headaches?
7)do your headache have a throb any time during the attack.?
8)do you have dizzy spells at other times than with your headache?
9)are your dizzy spells related to position of your head?
10)what are your headaches relieved by?
11)have you ever had motion sickness?
12)during or within an hour before your attack do you have any of the following complaints?
a.spots/shimmering/zigzag lights in your field of vision?
b.dark areas at the centre of your vision?
c.darkness in one half of your vision?
d.numbness or tingling around your mouth?
e.numbness or tingling over one half of your face or body?
f.weakness on one side /both sides of your face or body?
g.Difficulty in your speech?
h.dizziness?
i.unsteadiness?
j.double vision?
k.slurred speech?
l.noises in your ear?
m.hearing loss?
n.decreased level of conciousness/

o.blackouts?

13)do you have any of the following complaints during your attacks

a.nausea

b.nausea and vomiting

c.increased sensitivity to bright lights.

d.increased sensitivity to loud sounds

e.disturbance with smell

14)are your dizzy spells accompanied by noise in your ears or change in your hearing? Yes no

15)is there any family history of similar attacks?

<u>INVESTIGATIONS</u>

1) Pure Tone Audiogram Right ear Left ear

Air Conduction

Bone conduction

2)Impedance Right ear Left ear

Tympanometry

Stapedial reflux

3) ABR Right ear Left ear

Absolute Latency

Wave 1

Wave 3

Wave 5

Inter peak Latency

Wave 1 and 3

Wave 3 and 5

Wave 1 and 5

4)DPOAE Right ear Left ear

2. INFORMED CONSENT DOCUMENT

Study number-
Participant's name-
Date of birth/age-

I son/daughter/wife of give my consent to take part in this study.It has been explained to me that I will be tested for hearing problems that I may have due to my disease.All the tests done (four in number)are accected universally at the moment.Two of the four tests are a protocol for this condition.The two additional tests which will be done are not of any harm/risks to me.I understand that this is a clinical study done to know more about my disease and for its better treatment.

It has been explained to me that I am free to withdraw from the study any time I want and will not in any way compromise the treatment ,the ENT department is giving me.I understand that my identity and participation will not be revealed in any information released to third parties.I am giving this consent on my own free will.I have been explained about the study in a language familiar to me.

Name Signature Physician

Name of relative(guardian/parent)
Signature
Name of witness
Signature

Signature of person taking consent.

slno	idno	name	age	sex	diagnosis	audiogram
1	232098F	YOMKEN BOKO	2	1	2	2
2	232320F	AJIT KUMAR	5	1	2	14
3	23224F	MAMTA PANDEY	2	2	1	2
4	228414F	MAYAKRISHNAN	1	2	2	0
5	974661A	SELVI	2	2	1	0
6	229197F	JAYAPRIYA	1	2	2	9
7	233988F	ROHIT DAS	1	1	3	12
8	226766F	MANIK DAS	4	1	2	2
9	231909F	KUSHBUL BEGUM	2	2	1	2
10	239892F	SELVARAJ	3	1	1	0
11	223593F	SUKDEV JANA	3	1	2	0
12	225280F	DURGA PRASAD	5	1	1	0
13	181915F	ABIMANYU	3	2	1	5
14	225363F	BASANA	3	1	2	1
15	151392F	JAYANTI	2	2	1	0
16	232027F	MATI DEVI	3	2	1	2
17	230345F	ROHIT MONDAL	0	1	2	0
18	231252F	SUNANDA BHUIN	0	2	3	8
19	228304F	SUPRATO MALAKAN	2	1	2	0
20	151487F	AMIT KUMAR CHAN	2	1	3	14
21	229412F	RADHA SHYAM	2	1	2	0
22	215115F	SOMNATH SINGH	2	1	2	0
23	198045F	DEBRUP SARKAR	1	1	3	12
24	225611F	DEEPAK	4	1	2	3
25	232668F	MANIKNATH	5	1	2	0
26	209614F	BRIJ KUMAR	3	1	2	0
27	561221D	MERCY INDRA	4	2	2	2
28	027711B	TAJUNEESA	4	2	2	2
29	12307F	SHADNAM KUMAR	1	1	3	12
30	229306F	VALLIAMMA	5	2	2	2
31	219723F	UGRENGRA KUMAR	5	1	2	0
32	227663F	LELIN.K	4	2	1	2
33	229891F	ARASU	3	1	2	0
34	199124F	MURUGESAN	3	1	2	14
35	199426F	ABDUL RASHHED	4	1	2	0
36	233837F	RAM SWARUP	1	1	2	0
37	236491F	RABEA BEGUM	4	2	2	0
38	221511F	GANGAMMA	5	2	3	13
39	72566A	LLOGANAYAGI	5	2	2	0
40	233970F	DIBANKER MONDAL	3	1	2	0
41	019517F	SRIDHAR	3	1	2	0
42	228304F	SUPRANATH MALAK	3	1	1	0
43	222958F	NARASIMHAN	4	1	2	2
44	189481C	VINAYAN	3	1	2	2
45	962305D	LEELAVATHYSBABY	0	2	3	12
46	871145A	SHYAMALA	5	1	3	2
47	229649F	MUNIYAN	3	1	1	0
48	772094D	BARNALI NANDY	3	2	2	3
49	219347F	CHINNDRA	3	2	1	0
50	905415C	SHAMEEM	3	1	2	0

51	037430C	JAISHANKER	3	1	2	0
52	228713F	MADHABI DAS	2	2	3	2
53	788773C	RAJAN	5	1	2	8
54	477587C	PARUL DEY	3	1	2	1
55	671597D	RAM KALLESHWAR	3	1	2	2
56	283907	RICHARD	5	1	2	6
57	983854C	BIRU KUMAR	1	1	3	12
58	229891F	ARASU SK	3	1	2	0
59	216616F	SUBENDRA DEB	3	1	3	14
60	905415C	SHAMEEM	1	1	2	0
61	037430F	JAISHANKER	2	1	2	0
62	940692D	BIDHAN ROY	4	1	2	7
63	895891D	SUJATHA	2	2	1	2
64	972800D	VIJI	3	2	2	0
65	963340D	PRATIMA PAUL	3	2	2	10
66	817795D	NEHA TAGE	2	2	2	0
67	232906F	SELVI	1	2	2	14
68	239889F	RAJESH PRADHAN	2	1	1	2
69	174616F	DASARADHAN	3	1	2	0
70	247326F	TULSI SUBBA	3	2	2	0
71	730810A	SRINIVASAN	4	1	2	0
72	248376F	ROSIA BIBI	3	2	2	7
73	237080F	RAJA SAHEB	1	1	2	1
74	247264f	BUDD SAHA	4	1	2	3
75	255434F	KACHALA MADHU	1	2	1	0
76	247172F	VICTORIA	4	2	2	9
77	166328F	YUVARAJULU	5	1	2	2
78	198398F	SEEMA BERA	2	2	2	2
79	251788f	TUSHAR KANTI	2	1	2	4
80	862447B	ALAMELU	4	1	2	0
81	364795B	THIRUMAGAL	1	2	1	0
82	856219	ANNAMALAI	5	1	2	0
83	078050C	SHENBAKAVALLI	3	2	1	2
84	841681B	SARALA	2	2	2	0
85	117181F	JANCE G	4	2	2	0
86	224512F	RAMASAMY	5	1	2	4
87	189481C	VINAYAGAM	5	1	2	4
88	287373	JESSIE EDWARD	5	2	2	0
89	233988F	ROHIT DAS	4	1	3	10
90	233858F	DATTARAM	4	1	2	5
91	332692F	PRADIP SARKAR	3	1	2	2
92	232027F	MALTI DEVI	3	2	1	4
93	222958F	NARASIMHAN	3	1	2	4
94	233175F	GOURI DAS	3	2	2	0
95	236295F	LATHA S	2	2	1	0
96	946929D	RABINDRA KUMAR	3	1	2	0
97	956315D	SNEHADRI PACHEL	3	2	2	5
98	237251F	RAJENDER PANDIT	3	1	2	0
99	213234F	KANAHASABHAI	4	1	2	6
100	238887F	PUNANJAY KUMAR	2	1	2	2
101	238443f	ASGAR SABA	4	1	2	2
102	238982F	PRASHANT KUMAR	3	1	2	2
103	166330F	RAM CHANDRA	4	1	2	2
104	237877F	ASHWIN R	2	1	2	0

105	249402F	JAI MUNNA DEV	3	1	2	2
106	174616F	DASARADHAN	3	2	2	0
107	244043F	INDU CHAKRABORT	2	2	1	0
108	130837A	AMRITA DAS	2	2	2	2
109	084727A	SELVAKUMAR	2	1	1	0
110	088991F	SABITA DAS	2	2	2	4
111	730810A	AJ JOSEPH	3	1	2	0
112	964299D	PRAJENDRA CHAND	2	1	1	2
113	778053c	NATARAJAN	4	1	2	0
114	134307F	FAZALUR RAHMAN	2	1	1	14
115	244016F	SRILAKHSMI	3	2	2	0
116	241385F	UTPAL BISWAS	1	1	2	0
117	189604F	SELVI	2	2	1	2
118	166328F	YUVRAJULUU	5	1	2	4
119	101017F	RAJAMANI	3	2	3	8
120	219296F	GEETA SINGH	2	2	2	0
121	242471F	SANTHOSH KUMAR	3	1	2	2
122	244070F	KASHI NATH	3	1	2	2
123	233428F	MINA CHATTARAJ	4	2	2	0
124	242699F	BENU BALA	5	2	2	2
125	122018F	PUNITHA R	1	2	3	10
126	244043F	INDU CHAKRABORT	1	2	1	0
127	231562F	SANKAR DEY	3	1	2	0
128	232906F	SELVI	1	2	2	14
129	239889F	RAJESH PRADHAN	3	1	1	2
130	174616F	DASARADHAN	3	1	2	0
131	247326F	TULSI SUBBA	3	1	2	0
132	230810A	KUMAR	2	1	2	0
133	267737F	SUSHAR BAHAT	2	1	2	0
134	252888F	VISVANADAN	2	1	3	2
135	073886F	GAURAV KUMAR	1	1	3	0
136	261622F	PARVATHI	1	2	2	0
137	121355F	JHUNI PANIGRAHI	2	2	1	0
138	270976F	JOYDEB BISWAS	2	1	2	4
139	273289F	DIPAK SAMLAL	2	1	2	2
140	270379F	RUBIN DAS	3	1	2	0
141	276330F	RANJIT MUKHERJE	3	1	1	0
142	270434F	LAKHI MONDAL	2	2	1	0
143	271665F	INDARDEO	3	1	2	0
144	272428F	NEELAM JAISWAL	2	2	2	0
145	270300f	MINATI MAITI	3	2	3	2
146	273352F	SHUNI RATHI	2	1	3	9
147	272821F	MANOHAR SHIL	3	1	2	4
148	265070C	NIRMALA	3	2	2	0
149	235967F	DHRUBA LAL	4	1	2	0
150	169794F	RAJA	2	1	1	0
151	263994F	KAMLIDASS	3	2	2	7
152	188150F	TUTUL DEBNATH	1	2	3	11
153	114958F	NANDA BHUNIA	1	2	3	11
154	270991F	SAHEDA MAHELI	3	2	2	2
155	266099F	NANDAKUMAR	3	1	2	0
156	248574f	JALAJAKSHI	2	2	2	2
157	272492F	PINTU GUPTA	3	2	2	0

158	275354F	LAKHSMI	4	2	2	4
159	268749F	SHIRU SULTHANA	2	1	1	2
160	256433F	SUSHILA J	2	2	1	2
161	273352F	SHUNI RATHORE	4	2	3	4
162	241732F	NIBEDITA BISMI	1	2	3	12
163	247952F	ARVIND KUMAR	2	1	1	0
164	258460F	KUMKUM DEVI	3	2	1	13
165	364795B	THIRUMAGAL	2	1	1	0
166	261209F	CHANDRA NATH RA	3	1	2	0
167	268955F	SANJEEVI	3	2	1	0
168	270300F	MINATI MAITI	5	2	3	2
169	272021F	MANOHAR SHIL	2	1	2	14
170	270976F	JOYDEB BISWAS	3	1	2	4
171	266307F	MALTI DEVI	3	2	2	0
172	270379F	RUBI DAS	3	2	2	0
173	264590F	BISHNUPRIYA	2	2	2	3
174	271924F	UMAR	1	1	2	0
175	263120F	DIP MONDAL	0	1	3	10
176	263122F	RASHI MONDAL	1	2	3	12
177	239357F	RAJU NT	5	1	2	6
178	245869F	MANI	3	1	2	0
179	268360F	GANESAN	4	1	2	0
180	270434F	LAKHI MONDAL	2	2	1	0
181	272033F	GOURANGA MAITY	3	1	1	1
182	244744F	KOUSHIK NAIYA	4	1	2	6
183	140803F	ARJUN VISVA KAR	3	1	2	4
184	263994F	KAMLI DEVI	3	2	2	7
185	250259F	DRUBHA LAL	3	1	2	2
186	244752F	KULDEEP	3	1	2	0
187	233124F	PARBATI GHOSH	3	2	2	2
188	261188F	MRIND KANTI	3	1	2	3
189	236018F	ANJU SINHA	2	2	1	0
190	219834F	ALAKRAJ DUTTA	4	1	2	14
191	268005F	EVERGREEN SHYLL	1	2	2	2
192	265788F	LALITHA DEVI	2	2	1	2
193	272197F	PRAKASH SINGH	5	1	2	1
194	057615F	TAMALIKA	2	2	2	0
195	263360F	PRABIR BARUAH	3	1	2	0
196	273706F	JAYARAMAN	1	1	3	0
197	246619F	NARESH KUMAR	0	1	1	0
198	263277F	BABU	3	1	2	5
199	266521F	DEVI	2	2	1	0
200	250325F	VIJAYA	2	2	1	2
201	199860F	PRABHA DEVI	3	2	1	0
202	268817F	AMIT KUMAR	2	1	2	9
203	267867F	RAM VISVAKARMA	2	1	3	2
204	265967F	SWAPAN DEY	5	1	2	2
205	268655f	RAKESH KUMAR	4	1	2	8
206	143730F	ARUN MONDAL	1	1	3	12
207	271082F	DEETIPATRA	0	2	1	0
208	204321F	ARUN KUMAR	1	1	3	11
209	260583F	ROSALINE	3	2	2	0
210	264347F	KSHETRI MAYAN	3	1	2	0
211	131978F	TASHI BHUTIA	1	1	3	12

212	260075F	ASIM BAG	3	1	1	2
213	082515F	MAYA DAS	3	2	1	2
214	731306D	PAPPU KUMAR	4	1	2	0
215	262778F	PREMA DEVI MODI	2	2	1	2
216	258028F	MON SAHA	5	1	2	0
217	268187F	KALIPADA SARKAR	2	1	2	14
218	155158F	NIZAMUL HAQUE	2	1	1	0
219	256585F	SARASWATI	4	2	2	5
220	266099F	NANDAKUMAR	4	1	2	8
221	248830F	NANDA	2	2	1	2
222	270508F	SANGITA DEBI	2	2	1	0
223	264590F	BISHNUPRIYA	2	2	2	3
224	267737F	SUBHAS BHAKAT	3	1	2	2
225	261437F	MUBIYA	2	2	2	2
226	253704F	NIBARAM CHANDRA	3	1	2	3
227	127274F	RANGASAMY	4	1	2	0
228	258460F	KUMKUM DEVI	3	2	2	13
229	304628D	VENKATESAN	2	1	2	0
230	249709F	SWETA KARMAKAR	1	2	3	12
231	262420F	CHARANJEDI	3	1	2	4
232	262217F	ZAKIR HUSSAIN	4	1	2	0
233	266521F	DEVI N	2	2	1	2
234	242533F	ASHOK YADAV	4	1	2	9
235	131978F	TASHI DHUNDUP	2	1	3	11
236	253704F	NIBARBAN MONDAL	3	1	2	3
237	258767F	ARUP BATABEL	2	1	2	11
238	260575F	RABANI ADITYA	2	1	2	1
239	260922F	AMIT MAITY	3	1	2	0
240	305892F	HARADHAN SAHA	3	1	2	8
241	188902F	SHYAM SUNDER	2	1	2	3
242	303304F	DILIP SINGH	4	1	2	7
243	703297F	VIJAYA R	2	2	1	2
244	306439F	PINTU DEBNATH	2	2	2	4
245	302706F	BIMAL KUMAR	2	1	1	0
246	301264F	ABHIJIT SADU	2	1	1	0
247	304126f	AKBAR ALAM	2	1	2	7
248	298795F	LATHA	3	2	2	0
249	107230f	HARADHAN PAUL	3	1	2	4
250	111677F	SUBRATA GHOSH	3	1	2	0
251	276784f	RAM PRASAD	2	1	2	5
252	288036F	SOMA DUTTA	3	2	2	0
253	288392F	KRISHNA KANTA	2	1	2	1
254	300007F	SYED NAZIRUDDIN	3	1	2	0
255	307201F	ALOK KUMAR	4	1	2	0
256	950425B	PARTHSARTHY	5	1	2	0
257	185681F	RAJENDRAN	3	1	2	2
258	292116F	SUBHADEEP DEY	2	1	1	0
259	023767B	TARA SAMUEL	4	2	2	4
260	538499B	SWAPNA	3	2	1	0
261	300945F	SUSANG LEPCHA	1	2	3	12
262	279607F	RADHA SHYAM	1	1	3	10
263	212307F	SHADNAM KHAMAR	1	1	2	3
264	303945F	SADHAN KUMAR DE	2	1	3	9
265	303934F	GAYATHRI DEVI	3	2	2	8

266	284129F	JHARNA MONDAL	2	2	2	13
267	258786F	SUNIL KUMAR	4	1	2	7
268	297624F	NEMAI MONDAL	2	1	2	7
269	537511D	JOSEPH	5	1	2	0
270	302226F	PRAITY JAEL	2	1	1	0
271	303910F	KAKALI	2	2	2	11
272	249175F	RAYALAMMAL	4	2	2	0
273	147823F	NANDAKUMAR	2	1	2	10
274	291254F	JOHN KENNEDY	3	1	2	10
275	283791F	PUTUL SHIL	2	1	2	0
276	305666F	APARESH BANERJE	2	1	2	12
277	301444F	PRATIMA MIDYA	3	2	2	0
278	281229F	GURUSAMY	2	1	2	2
279	303752F	SIKDAR	4	1	2	0
280	291771F	RAJAVELI	3	1	2	0
281	251539F	JAISHANKER	5	1	2	0
282	242405F	ANWAR BASHA	3	1	2	0
283	295934F	ROSE MARY	4	2	2	0
284	294507F	TAPAS DUTTA	3	1	2	0
285	071446A	VIMALA RAM	5	2	2	4
286	307762F	KANNIYAMMAL	2	2	1	0
287	368777A	SUSEELA	4	2	2	2
288	111517F	SULTHAN B	3	1	2	2
289	144788F	GOUTAM GHOSH	3	1	2	3
290	300034F	SANJAY KUMAR	4	1	2	8
291	302260F	DULAL DEBNATH	3	1	2	0
292	296442F	PUSHPA SINGH	3	2	2	0
293	305546F	SURENDRA PRASAD	3	1	2	2
294	302748F	ARCHANA SINGHA	2	2	2	0
295	300942F	RABINDRA SARKAR	2	1	2	11
296	290486F	SHAKUNTHALA	2	2	2	0
297	294851F	ATWARI RANA	3	1	2	0
298	301583F	MEBANJOP	2	2	2	0
299	964940F	AINUL HAQUE	4	1	2	0
300	305570F	SATYABAMA	4	2	2	4
301	285208B	MURALIDHARAN	5	1	2	0
302	197218F	BHAKTI PRASAD	2	1	2	12
303	144255F	CHANDRASHEKAR	2	1	2	12
304	258669F	MANISHA RAY	3	2	1	2
305	296442f	PUSHPA SINGH	2	2	2	0
306	083465F	ABIRAMI	0	2	3	10
307	772629D	SWAPAN SEN	3	1	2	5
308	709190C	ZAMRUTH BEE	3	2	2	0
309	311771F	NAYAN DUTTA	2	1	1	2
310	312133F	SUSIT CHAKRABOR	4	1	2	0
311	144788F	GOUTAM GHOSH	1	1	2	0
312	479698	SHANMUGAM	4	1	2	12
313	183656F	RAMAKRISHNAN	4	1	2	2
314	243115C	RAJENDRA REDDY	3	1	1	0
315	588587D	MANIVELU	4	1	2	9
316	302260F	DULAL DEBNATH	4	1	2	0
317	241436F	ARCHANA BASUALI	3	2	2	0
318	299869F	ASHISH KUMAR	3	1	2	3

319	310288F	CHARU BALA DEVI	3	2	2	0
320	185681f	RAJENDRA	3	1	2	1
321	315989F	KIRAN DEVI	3	2	2	11
322	797313B	SHAKTHIVEL	3	1	1	2
323	252985F	SRINIVASAN	2	1	1	0
324	256433F	SUSHILA	4	2	2	5
325	305952F	PRABIR BARUA	2	1	2	0
326	312121F	SWAPAN KUMAR	2	1	2	0
327	879723C	LEELAVATHY	4	2	2	7
328	302271F	ROSALINE	3	2	2	0
329	104061F	SHYAMA MONDAL	3	2	1	0
330	305892F	HARADHAN SAHA	3	1	2	8
331	315937F	DEBJIT GHORUI	2	1	2	0
332	292110D	JAI PRAKASH	3	1	2	0
333	311250F	SWETHA DUTTA	2	2	1	0
334	291220f	BHAKTHI PRASAD	3	1	2	11
335	311122F	KRISH BANI	2	1	2	0
336	312465F	BIRENDRA NATH	4	1	2	0
337	327094F	MINCHI ADHIKARI	3	2	2	0
338	307201F	ALOK MUKERJEE	3	1	2	3
339	313360F	MATIUR RAHMAN	3	1	2	0
340	298810F	RUBI NANDI	2	2	2	0
341	290489F	LAKSMI RANI DUT	3	2	2	8
342	305783F	AJAY SARKAR	2	1	1	2
343	144560F	SUDHAM MAIHAP	3	1	2	4
344	298803F	ANURADH KUMAR	4	1	2	10
345	273741F	BHOLANATH SAHU	5	2	2	9
346	169559D	NEELAM SINGH	2	2	2	2
347	325871B	IRUCHAPPAN	5	1	2	4
348	503658F	VASANTHA	2	2	2	7
349	655824B	MANIVANNAN	3	1	2	2
350	312024F	PAIPAYA ACHARYA	2	1	1	0
351	315963F	RAJENDRAN	3	1	2	11
352	640186B	SUBRAMANI	3	1	2	14
353	981973B	DROPADICHANDELI	2	2	2	0
354	812940B	SRIMATHI R	3	2	2	0
355	330199	GOVINDAMMA	4	2	2	2
356	148651F	PREMA S	3	2	2	0
357	313124F	MAHMUDA KHATUN	2	2	2	12
358	751602D	CHITTIAMMA	3	2	2	0
359	320237F	GOLAM MUSTAFA	3	1	2	0
360	183656F	RAMAKRISHNAN	3	1	2	2
361	299509F	BISHAK BERA	1	1	2	0
362	956575D	LAKSMI M	2	2	2	2
363	302533D	SHAKEEL BASHA	3	1	2	3
364	399595D	RABINDRA SINGH	3	1	2	0
365	315304F	ILA BISWAS	2	2	2	8
366	170737F	AMALENDU PANDEY	3	1	1	0
367	274883B	JANAKI	5	2	2	4
368	312566F	VINCENT JOHN	3	1	1	2
369	312933F	SWAPAN SARKAR	3	1	1	0
370	299000F	KHAGENDRANATH	5	1	2	4
371	303592F	PRASHANT KUMAR	2	1	2	10

372	297072F	RABINDRA DHALI	3	1	1	0
373	310537F	LINDA SUSANNE	2	2	1	0
374	273334F	MOUMITA BANERJI	2	2	2	10
375	503966D	LIMY MATHEW	1	2	1	2
376	851479D	SANJIB GHOSH	4	1	2	9
377	315805F	SHASHI CHANDAN	3	1	2	2
378	252842F	ANWAR BASHA	2	1	2	2
379	409648A	VIJAY KUMAR	2	1	1	0
380	674276C	FAIROON	2	2	2	0
381	279604F	DIPANKER MONDAL	0	1	3	0
382	262446F	ABHISHEK B	2	1	2	0
383	312465F	BIRENDRA NATH	3	1	1	0
384	675810C	LAKSMI KANTA	3	1	2	0
385	286407F	FESTING	4	1	2	2
386	310526F	MANIK SUTRADHAR	4	1	2	0
387	312933F	SWAPNA SARKAR	2	2	2	0
388	301036F	SOMNATH DEY	2	1	1	0
389	247474F	RANJIT BHOUMIK	4	1	2	4
390	310797F	BASANTI DEVI	3	2	2	0
391	312388F	PADMA MOHAN	3	1	2	0
392	310681F	MINA PATEL	2	2	2	14
393	308931F	KHAILAS NATH	4	1	2	7
394	178853F	AJITHA	2	2	1	0
395	239533F	DAMODHAR DAS	4	1	2	14
396	254823F	SURESH BABU	2	1	1	0
397	328754f	LATHA	2	2	1	0
398	326346F	ANIL KUMAR	4		2	1
399	761795D	DEBI PRASAD	4	1	2	0
400	615058D	BRINDA DEVI	5	2	2	4

gr	sl	name	id	age	sex	date	headache	nature	aat	bon	cat	din	when	duration in	duration in hours	frequency	throbbing	dizzy	position	relief	motion	aspotszi	bdark	cdarkness	dtingling	etingling	fweakness	gdifficul	hdizzines	iunsteadi	jdouble	kslurred	lnoises	mhearing	ndecrease	oblack
1	1	SARAWATI ROY	109635F	41	2	11-01-2012	1	2	2	2	1	1	38	3	1	3	1	1	2	1	2	1	1	2	2	2	2	2	1	2	2	2	2	2	2	2
1	2	HUSNE ARABEGUM	118700F	35	2	24-01-2012	1	1	2	2	1	1	15	20	2	4	1	2	1	1	2	1	1	2	2	2	2	2	1	2	2	2	2	2	2	2
1	3	SIVASHANKARI	215239D	36	2	11-02-2012	1	1	1	2	1	2	45	9	2	3	1	2	2	1	1	1	1	2	2	2	2	2	1	1	2	2	2	1	2	2
1	4	SHANMUGHAM	061117A	34	1	13-02-2012	1	1	1	1	2	2	17	17	1	2	1	2	2	1	1	1	1	1	2	2	2	2	1	2	1	2	2	2	2	2
1	5	NIRMALA.R	130528F	38	2	16-02-2012	1	1	1	1	2	2	15	23	2	3	1	2	2	1	1	1	1	2	2	2	2	2	1	2	2	2	2	2	2	2
1	6	MAMTA PRASAD	101670F	41	2	18-02-2012	1	1	1	1	2	2	20	26	1	2	1	2	1	1	1	1	1	2	2	2	2	2	1	2	2	2	2	2	2	2
1	7	HEMAVATHI	992907B	26	2	18-02-2012	1	1	2	1	1	2	16	10	3	3	1	1	2	3	2	1	2	2	2	2	2	2	1	1	2	2	1	1	2	2
1	8	MANOJ CHATRI	140198F	32	1	24-02-2012	1	1	1	1	2	2	10	22	2	2	1	1	2	3	1	1	1	2	2	2	2	2	1	2	2	2	2	2	2	2
1	9	ASHOK KUMAR	135456F	44	1	25-02-2012	1	1	1	2	1	2	20	24	2	2	1	2	2	1	2	1	1	2	2	2	2	2	1	2	2	2	2	2	2	2
1	10	KALADEVI	823816A	38	2	03-03-2012	1	1	1	1	2	2	20	18	1	2	1	1	1	1	1	1	1	2	2	2	2	2	1	2	1	2	1	2	2	2
1	11	OBELASU	158565F	35	1	28-03-2012	1	1	1	1	2	2	15	20	1	2	1	2	2	1	2	1	1	1	2	2	2	2	1	2	2	2	2	2	2	2
1	12	BED MAYA POUDEL	190765F	27	2	07-05-2012	1	1	1	1	2	2	17	10	1	2	1	2	2	1	2	1	1	2	2	2	2	2	1	2	2	2	2	2	2	2
1	13	CHHATRA PAL SINGH	196595F	34	1	16-05-2012	1	1	1	1	2	2	15	19	1	3	1	2	1	1	2	1	1	2	2	2	2	2	1	1	2	2	2	2	2	2
1	14	JAHAN ARA	062962F	26	2	19-05-2012	1	1	1	1	2	2	13	13	2	2	1	2	1	1	2	1	1	2	2	2	2	2	1	2	2	2	2	2	2	2
1	15	VIJAYA	428383C	37	2	21-05-2012	1	2	1	2	1	1	30	7	1	2	1	1	2	1	2	2	2	2	2	2	2	2	1	2	2	2	2	2	2	2
1	16	SARADEVI.K	457685D	27	2	11-06-2012	1	2	1	1	2	2	26	1	2	4	1	1	2	1	2	1	1	2	2	2	2	2	1	1	2	2	2	2	2	2
1	17	INDU CHAKRABORTY	244043F	29	2	16-06-2012	1	1	2	2	1	1	17	12	1	2	1	2	2	1	1	2	1	1	2	2	2	2	1	2	2	2	1	2	2	2
1	18	PADMA	233402F	42	2	25-07-2012	1	1	1	1	1	2	13	29	1	1	1	2	1	2	2	2	1	2	2	2	2	2	1	2	2	2	1	2	2	2
1	19	SELVA KUMAR	084727A	34	1	20-07-2012	1	1	2	2	1	1	17	17	1	2	1	2	2	1	2	1	2	2	2	2	2	2	1	2	2	2	1	2	2	2
1	20	BITTU KUNDU	249060F	21	2	27-07-2012	1	1	2	1	1	2	18	3	2	1	1	2	2	2	1	2	2	2	2	2	2	2	1	1	2	2	1	2	2	2
1	21	REKHA PANDIT	251470F	30	2	30-07-2012	1	1	2	2	1	1	16	14	2	2	1	2	2	2	2	2	1	2	2	2	2	2	1	2	2	2	1	2	2	2
1	22	THIRUMAGAL	364795B	33	1	06-08-2012	1	1	2	1	1	2	17	16	2	2	1	1	2	1	2	2	1	2	2	2	2	2	1	2	1	2	1	2	2	2
1	23	KOTTESWARI	956258A	34	2	20-08-2012	1	1	2	2	1	1	20	14	1	1	1	2	1	1	1	2	1	2	2	2	2	2	1	2	2	2	1	1	2	2
1	24	REBECCA	307886D	25	2	08-09-2012	1	1	1	2	1	1	15	10	1	3	1	2	2	1	2	2	1	2	2	2	2	2	1	1	1	2	1	2	2	2
1	25	PADMA P	290702F	40	2	17-09-2012	1	1	1	2	1	1	18	22	2	2	1	2	2	1	2	1	1	2	2	2	2	2	1	2	2	2	1	2	2	2
1	26	MANORANJAN DAS	552968D	42	1	07-08-2012	1	1	2	1	1	2	20	22	1	2	1	1	2	1	2	2	1	1	2	2	2	2	1	2	2	2	1	2	2	2
1	27	SRINIVASAN	252985F	42	1	05-10-2012	1	1	1	1	1	1	41	1	1	3	1	2	1	1	2	2	1	2	2	2	2	2	1	1	1	2	1	1	2	2
1	28	AJAY SARKAR	305783F	39	1	08-10-2012	1	1	1	2	1	2	20	19	1	2	1	2	2	1	1	1	1	2	2	2	2	2	1	2	2	2	2	2	2	2
1	29	KANNIYAMMAL	307762F	40	2	24-10-2012	1	1	1	2	1	2	15	25	1	2	1	1	2	1	2	2	2	1	2	2	2	2	1	1	2	2	1	2	2	2
1	30	SURESH BABU	254823F	34	1	27-10-2012	1	1	2	2	1	1	15	19	2	2	1	2	1	1	2	2	1	2	2	2	2	2	1	1	2	2	2	2	2	2
2				34	1																															
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2				42	1																															
2				24	2																															
2				32	1																															

anausea	bnausea	cincrease	dincrease	edisturba	hearing	family	right	left	nose	throat	ac	a	bc	b	tymps	tymps1	stapreflu	staperefl	dp	dp1	ab	abl	abslat	ltablwave1	ltabsolute	leftablwav	wave	rtwave	rtwav	wav	wve	wv
1	1	2	2	2	2	1	1	1	1	1	20 db	20 db	20 db	20 db	1	1	1	1	2	1	1.45	3.58	5.38	1.5	3.55	5.28	2.13	1.8	3.93	2.05	1.73	3.78
1	2	2	2	2	2	1	1	1	1	1	20 db	20 db	20 db	20 db	1	1	1	1	1	1	1.43	3.4	5.22	1.45	3.43	5.25	1.98	1.82	3.8	1.98	1.83	3.8
1	2	2	2	2	2	1	1	1	1	1	20 db	35 db	20 db	35 db	1	1	2	1	2	2	1.7	3.75	5.88	1.75	3.95	5.95	2.05	2.13	4.18	2.2	2	4.2
1	1	2	2	2	2	2	1	1	1	1	20 db	20 db	20 db	20 db	1	1	1	1	1	1	1.48	3.65	5.38	1.43	3.65	5.53	2.17	1.73	3.9	2.22	1.88	4.1
1	1	2	2	2	1	1	1	1	1	1	35 db	35 db	35 db	35 db	1	1	1	1	1	1	1.55	3.45	5.28	1.43	3.5	5.22	1.9	1.83	3.73	2.08	1.72	3.8
1	1	2	2	2	1	1	1	1	1	1	35 db	35 db	35 db	35 db	1	1	1	2	1	1	1.45	3.5	5.3	1.55	3.45	5.38	2.05	1.8	3.85	1.9	1.92	3.83
1	1	1	2	1	1	1	1	1	1	1	20 db	20 db	20 db	20 db	1	1	1	1	1	1	1.58	3.4	5.43	1.6	3.58	5.3	1.83	2.03	3.85	1.98	1.72	3.7
1	1	2	2	2	2	1	1	1	1	1	20 db	20 db	20 db	20 db	1	1	1	1	1	1	1.55	3.45	5.25	1.4	3.58	5.3	1.9	1.8	3.7	2.18	1.72	3.9
1	1	2	2	2	2	1	1	1	1	1	20 db	20 db	20 db	20 db	1	1	1	1	1	1	1.65	3.68	5.43	1.65	3.83	5.72	2.03	1.75	3.78	2.18	1.9	4.07
1	1	2	2	2	2	1	1	1	1	1	35 db	35 db	35db	35 db	1	1	1	1	1	1	1.58	3.4	5.2	1.6	3.4	5.2	1.83	1.8	3.63	1.8	1.8	3.6
1	1	2	2	2	2	1	1	1	1	1	20 db	20 db	20 db	20 db	1	1	1	1	2	2	1.7	3.78	5.6	1.6	3.65	5.6	2.08	1.82	3.9	2.05	1.95	4
1	1	1	2	2	2	1	1	1	1	1	20 db	20 db	20 db	20 db	1	1	1	1	1	1	1.43	3.58	5.2	1.5	3.6	5.35	2.15	1.63	3.78	2.1	1.75	3.85
1	1	1	2	2	2	1	1	1	1	1	20 db	20 db	20 db	20 db	1	1	1	1	1	1	1.45	3.55	5.45	1.48	3.6	5.55	2.1	1.9	4	2.13	1.95	4.07
1	1	1	2	1	2	1	1	1	1	1	20 db	20 db	20 db	20 db	1	1	1	1	1	2	1.55	3.45	5.1	1.48	3.43	5.18	1.9	1.65	3.55	1.95	1.75	3.7
1	1	2	2	2	2	1	1	1	1	1	35 db	35 db	35 db	35 db	1	1	1	1	2	2	1.6	3.4	5	1.45	3.3	5.08	1.8	1.6	3.4	1.85	1.6	3.4
1	1	2	2	2	2	1	1	1	1	1	20 db	20 db	20 db	20 db	1	1	1	1	1	1	1.45	3.45	5.45	1.7	3.65	5.58	2	2	4	1.95	1.93	3.88
1	1	1	1	2	1	1	1	1	1	1	20 db	20 db	20 db	20 db	1	1	1	1	1	1	1.48	3.55	5.45	1.48	3.48	5.45	2.07	1.9	3.98	2	1.98	3.98
1	1	2	1	2	1	1	1	1	1	1	20 db	20 db	20 db	20 db	1	1	1	1	2	2	1.48	3.5	5.2	1.48	3.43	5.28	2.03	1.7	3.73	1.95	1.85	3.8
1	1	2	1	1	1	2	1	1	1	1	20 db	20 db	20 db	20 db	1	1	1	1	1	1	1.55	3.35	5.1	1.63	3.45	5.1	1.8	1.75	3.55	1.83	1.65	3.47
1	1	2	1	2	1	1	1	1	1	1	20 db	20 db	20 db	20 db	1	1	1	1	1	1	1.5	3.4	5.03	1.48	3.45	5.2	1.9	1.63	3.53	1.98	1.75	3.73
1	1	1	1	2	1	1	1	1	1	1	20 db	20 db	20 db	20 db	1	1	1	1	2	2	1.5	3.5	5.1	1.43	3.4	5.18	2	1.6	3.6	1.98	1.78	3.75
1	1	1	1	1	1	2	1	1	1	1	20 db	20 db	20 db	20 db	1	1	1	1	1	1	1.43	3.3	5.13	1.43	3.3	5.2	1.87	1.83	3.7	1.87	1.9	3.78
1	1	2	1	2	1	1	1	1	1	1	35 db	35 db	35 db	35 db	1	1	1	1	2	2	1.48	3.55	5.18	1.45	3.48	5.18	2.07	1.63	3.7	2.03	1.7	3.72
1	1	1	1	2	1	2	1	1	1	1	20 db	20 db	20 db	20 db	1	1	1	1	1	1	1.58	3.5	5.3	1.48	3.5	5.3	2.03	1.85	3.88	2.03	1.8	3.83
1	1	2	1	2	1	2	1	1	1	1	35 db	35 db	35 db	35 db	1	1	1	1	1	1	1.45	3.48	5.18	1.48	3.55	5.18	2.03	1.7	3.72	2.07	1.63	3.7
1	1	1	1	1	1	1	1	1	1	1	40 db	40 db	35 db	35 db	1	1	1	2	1	1	1.83	3.9	5.98	1.68	3.8	5.63	2.08	2.07	4.15	2.13	1.83	3.95
1	1	1	1	2	1	1	1	1	1	1	20 db	20 db	20 db	20 db	1	1	1	1	1	2	1.63	3.5	5.47	1.7	3.75	5.65	1.88	1.97	3.85	2.05	1.9	3.95
1	1	1	1	2	1	1	1	1	1	1	35 db	35 db	35 db	35 db	1	1	1	1	1	2	1.45	3.65	5.45	1.43	3.58	5.4	2.2	1.8	4	2.15	1.83	3.98
1	1	1	1	2	1	1	1	1	1	1	35 db	35 db	30 db	30 db	1	1	1	1	1	1	1.55	3.63	5.2	1.68	3.83	5.4	2.08	1.58	3.65	2.15	1.58	3.73
1	1	1	2	2	2	1	1	1	1	1	20 db	20 db	20 db	20 db	1	1	1	1	2	2	1.45	3.45	5.45	1.7	3.65	5.58	2	2	4	1.95	1.93	3.88
																			1	1	1.93	3.95	5.93	1.85	3.93	6	2.03	2.08	4.15	2.07	2.08	4.15
																			1	1	1.63	3.6	5.3	1.5	3.58	5.28	1.98	1.7	3.68	2.08	1.7	3.78
																			1	1	1.5	3.68	5.58	1.6	3.78	5.53	2.17	1.9	4.08	2.17	1.75	3.93
																			1	1	1.4	3.43	5.2	1.38	3.4	5.13	2.03	1.78	3.8	2.03	1.73	3.75
																			1	1	1.48	3.48	5.33	1.4	3.4	5.2	2	1.85	3.85	2	1.8	3.8
																			1	1	1.43	3.43	5.13	1.43	3.45	5.18	2	1.7	3.7	2.03	1.72	3.75
																			1	1	1.33	3.5	5.38	1.43	3.43	5.25	2.17	1.88	4.05	2	1.83	3.83
																			1	1	1.43	3.68	5.68	1.38	3.55	5.4	2.25	1.7	3.95	2.17	1.85	4.03
																			1	1	1.5	3.5	5.2	1.63	3.63	5.25	2	1.7	3.7	2	1.63	3.63
																			1	1	1.6	3.8	5.78	1.6	3.8	5.78	2.2	1.98	4.18	2.2	1.98	4.18
																			1	1	1.65	3.65	5.72	1.6	3.85	5.63	2	2.07	4.07	2.25	1.78	4.03
																			1	1	1.4	3.43	5.13	1.43	3.38	5.03	2.03	1.7	3.73	1.95	1.65	3.6
																			1	1	1.48	3.63	5.53	1.63	3.63	5.3	2.15	1.9	2.05	2	1.67	3.68
																			1	1	1.33	3.35	5.08	1.3	3.48	5.08	2.03	1.73	3.75	2.17	1.6	3.78
																			1	1	1.43	3.6	5.45	1.63	3.75	5.58	2.17	1.85	4.03	2.13	1.83	3.95
																			1	1	1.43	3.45	5.1	1.38	3.35	5.03	2.03	1.65	3.68	1.98	1.68	3.65
																			1	1	1.48	3.3	5.05	1.45	3.3	5.05	1.82	1.75	3.58	1.85	1.75	3.6
																			1	1	1.8	3.95	5.72	1.65	3.8	5.58	2.15	1.77	3.93	2.15	1.78	3.93
																			1	1	1.58	3.8	5.78	1.55	3.73	5.58	2.22	1.98	4.2	2.17	1.85	4.03
																			1	1	1.53	3.35	5.22	1.68	3.7	5.45	1.83	1.87	3.7	2.03	1.75	3.78
																			1	1	1.55	3.4	5.43	1.63	3.43	5.43	1.85	2.03	3.88	1.8	2	3.8
																			1	1	1.75	3.65	5.47	1.6	3.65	5.65	1.9	1.82	3.72	2.05	2	4.05
																			2	2	1.98	3.9	5.9	1.75	3.9	5.7	1.92	2	3.93	2.15	1.8	3.95
																			1	1	1.58	3.4	5.3	1.48	3.43	5.22	1.83	1.9	3.72	1.95	1.8	3.75
																			1	1	1.5	3.55	5.4	1.43	3.5	5.35	2.05	1.85	3.9	2.08	1.85	3.93
																			1	1	1.45	3.55	5.47	1.45	3.48	5.58	2.1	1.92	4.02	2.03	2.1	4.13
																			1	1	1.63	3.9	5.7	1.65	3.85	5.6	2.28	1.8	4.08	2.2	1.75	3.75
																			1	1	1.8	3.98	5.75	1.68	3.8	5.75	2.17	1.78	3.95	2.13	1.95	4.08
																			1	1	1.58	3.48	5.47	1.58	3.73	5.4	1.9	2	3.9	2.15	1.68	3.83
																			1	1	1.63	3.68	5.38	1.38	3.55	5.4	2.25	1.7	3.95	2.17	1.85	4.03

bwave	cwave	dwave	ewave	fwave	gwave	hwave	iwave	jwave	kwave	lwave
3.93	6	2.07	2.08	4.15	1.93	3.95	5.93	2.03	2.08	4.15
3.58	5.28	2.08	1.7	3.78	1.63	3.6	5.3	1.98	1.7	3.68
3.78	5.53	2.17	1.75	3.93	1.5	3.68	5.58	2.17	1.9	4.08
3.4	5.13	2.03	1.73	3.75	1.4	3.43	5.2	2.03	1.78	3.8
3.4	5.2	2	1.8	3.8	1.48	3.48	5.33	2	1.85	3.85
3.45	5.18	2.03	1.72	3.75	1.43	3.43	5.13	2	1.7	3.7
3.43	5.25	2	1.83	3.83	1.33	3.5	5.38	2.17	1.88	4.05
3.55	5.4	2.17	1.85	4.03	1.43	3.68	5.68	2.25	1.7	3.95
3.63	5.25	2	1.63	3.63	1.5	3.5	5.2	2	1.7	3.7
3.8	5.78	2.2	1.98	4.18	1.6	3.8	5.78	2.2	1.98	4.18
3.85	5.63	2.25	1.78	4.03	1.65	3.65	5.72	2	2.07	4.07
3.38	5.03	1.95	1.65	3.6	1.4	3.43	5.13	2.03	1.7	3.73
3.63	5.3	2	1.67	3.68	1.48	3.63	5.53	2.15	1.9	2.05
3.48	5.08	2.17	1.6	3.78	1.33	3.35	5.08	2.03	1.73	3.75
3.75	5.58	2.13	1.83	3.95	1.43	3.6	5.45	2.17	1.85	4.03
3.35	5.03	1.98	1.68	3.65	1.43	3.45	5.1	2.03	1.65	3.68
3.3	5.05	1.85	1.75	3.6	1.48	3.3	5.05	1.82	1.75	3.58
3.8	5.58	2.15	1.78	3.93	1.8	3.95	5.72	2.15	1.77	3.93
3.73	5.58	2.17	1.85	4.03	1.58	3.8	5.78	2.22	1.98	4.2
3.7	5.45	2.03	1.75	3.78	1.53	3.35	5.22	1.83	1.87	3.7
3.43	5.43	1.8	2	3.8	1.55	3.4	5.43	1.85	2.03	3.88
3.65	5.65	2.05	2	4.05	1.75	3.65	5.47	1.9	1.82	3.72
3.9	5.7	2.15	1.8	3.95	1.98	3.9	5.9	1.92	2	3.93
3.43	5.22	1.95	1.8	3.75	1.58	3.4	5.3	1.83	1.9	3.72
3.5	5.35	2.08	1.85	3.93	1.5	3.55	5.4	2.05	1.85	3.9
3.48	5.58	2.03	2.1	4.13	1.45	3.55	5.47	2.1	1.92	4.02
3.85	5.6	2.2	1.75	3.75	1.63	3.9	5.7	2.28	1.8	4.08
3.8	5.75	2.13	1.95	4.08	1.8	3.98	5.75	2.17	1.78	3.95
3.73	5.4	2.15	1.68	3.83	1.58	3.48	5.47	1.9	2	3.9
3.55	5.4	2.17	1.85	4.03	1.43	3.68	5.38	2.25	1.7	3.95