Correlation between vHIT and caloric test in individuals with Auditory Neuropathy Spectrum Disorder

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Abstract

Auditory neuropathy spectrum disorder features the presence of Otoacoustic emissions, poor speech identification scores and absent auditory brainstem response. The present study aimed at correlation between vHIT and caloric test in individual with ANSD. Standard group comparison was performed in the study. Total 30 individuals participated in the study where Group I consisted 15 individuals with auditory neuropathy spectrum disorder and Group II consisted 15 normal hearing individuals. All participants underwent case history, pure tone audiometry, immittance, otoacoustic emissions, auditory evoked response, caloric test and video head impulse test. Based on the Hex plot, the vHIT response was analysed where the VOR gain of each of all six semi-circular canals and the refixation saccades (if any) at the time of head thrust i.e. covert saccade, after the head thrust i.e. overt saccade were represented. Similarly, in case of caloric test, the cumulative frequency was taken in consideration to be denoted on the butterfly chart. The four response waves achieved with bi-thermal stimulation were calculated and the cumulative frequency was achieved. All the tests were performed within noise permissible criteria of ANSI S3.1 (1991) in an acoustically treated room. Shapiro-Wilk test revealed normal distribution of the data ($p>0.05$). Hence a parametric independent sample't' test was done to compare the VOR gain and VOR gain asymmetry between the normal individuals and individuals with auditory neuropathy spectrum disorders. Pearson correlation test was done to check the Correlation between VOR gain and duration of six semi-circular canals in individual with ANSD. Also, Pearson's correlation was performed to understand the relation between the VOR gain and VOR gain asymmetry with the duration of disease in individuals with ANSD. Independent sample 't' test revealed significantly lower vestibulo-ocular reflex gain values in individuals with auditory neuropathy spectrum disorder. Presence of 100% corrective refixation saccades was seen in the same group. Pearson's correlation revealed no significant correlation between vestibulo-ocular reflex gain with duration of hearing loss and puretone thresholds for any of the three orthogonal planes. Huge percentage of individuals with auditory neuropathy spectrum disorderhave been found to have associated vestibular dysfunction as well. Therefore, video head impulse testcan be usedas one of the important tests of vestibular test battery to evaluate all six semi-circular canals in individuals with auditory neuropathy spectrum disorder.

Key words: *Auditory neuropathy spectrum disorder, semi-circular canals, Vestibulo-ocular reflex gain, caloric test, bithermal, refixation saccades, duration, puretone threshold.*

Introduction

Auditory neuropathy spectrum disorder (ANSD) is the term being used across the age group ranging from infants to adults with most of the characteristic criteria fulfilled such as: presence of otoacoustic emission or cochlear microphonics in ECohGs resembling preserved OHC's functioning; absence of auditory evoked brainstem response and acoustic reflex displaying the lack of synchronous firing of 8th cranial nerve; affected speech identification score that doesn't correlate with the pure tone average (PTA) of the individual and the variable degree as well as pattern of hearing loss (Starr et al, 1991; Starr et al, 1996; Sheykholeslami et. al, 2000; Sininger & Starr, 2001; Starr, 2001; Zeng, 2006).

As the auditory and vestibular receptors (first order neuron) of 8th cranial nerve are intrinsically linked with the common pathway, the frequent pathophysiological demyelination at periphery level of 8th cranial nerve are likely to involve both auditory and vestibular divisions leading to neuropathy on both the sides (Buetti & Luxon, 2014). However immense literature on auditory neuropathy (Davis & Hirsh, 1979; Kowalski, Rasheva, & Zakrzewska, 1991; Scaioli et al, 1992; Starr et al, 1996; Berlin, 1999; Sheykholeslami et al, 2000; Tang et al, 2004; Kumar & Jayaram, 2006) and rare description of vestibular neuropathy (Starr et al, 1996; Fujikawa & Starr, 2000; Sheykholeslami et al, 2000; Sheykholeslami et al, 2005; Kumar et al, 2007;) can be due to the slow progressive development of the pathology, likely, not much expressed due to the effective vestibular compensatory mechanism, and more consideration given on proprioception and cerebellar functioning for the imbalance problem in individual with ANSD (Melgaard & Zilstorff, 1979).

Few reports are available in the literature regarding the presence of vestibular dysfunction in individual with ANSD. Presence of horizontal nystagmus on lateral gaze, absence of caloric response, abnormal/absent vestibular evoked myogenic responses (VEMP) response suggest the presence of generalized peripheral pathologic changes at the level of vestibulocochlear nerve (Starr et al, 1991; Kaga et al, 1996; Kumar et al, 2007). Furthermore, abnormal Romberg's, Mann's and

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Fukuda stepping tests response in eyes closed condition correlates with the earlier reports suggesting the involvement of neuropathologic condition at the vestibular branch of 8th cranial nerve (Sheykholeslami et al., 2005). Hence these studies explain the existence of affected peripheral vestibular branch and normal central vestibular connections in individual with ANSD.

Aim of the Study

The present study aimed at correlation between vHIT and caloric test in individual with ANSD.

Objectives of the study

1. To assess VOR gain function in the individual with ANSD.
2. To check for refixation saccades which may be covert or overt in nature in the individual with ANSD.
3. To check for any correlation with degree of hearing loss for Caloric and v-HIT test in individual with ANSD.
4. To find out correlation between the Caloric and v-HIT test results in individual with ANSD.

Method

The study was conducted with an aim of understanding the correlation between vHIT and caloric test in individual with ANSD. To fulfil the aim, following methods were adopted.

Participants

Two groups of subjects with total number of 30 individuals participated in the study.

Group I comprised of 15 individuals, 7 males and 8 females, n= 30 ears with the age range of 17 to 38 years (mean age= 25.2yrs) with ANSD.

Group II comprised of 15 individuals, 10 males and 5 females in the age range of 18 to 24years (mean = 22.1yrs) with normal hearing sensitivity.

Participant selection Criteria:

Individuals with ANSD (Group I)

1. All participants were diagnosed to have bilateral ANSD based on the criteria of poor speech in noise scores, absence of ipsilateral and contralateral acoustic reflex response, presence of otoacoustic emissions/ cochlear microphonics and absence of auditory brainstem response.
2. The participants were identified to have sensorineural hearing loss.
3. No restriction regarding the degree of hearing loss was considered as there could be large variability in individual with ANSD (Kumar & Jayaram, 2006).
4. The participants with "A" type tympanograms and no history or presence of middle ear pathology were taken for the study.
5. Neurological examination excluded the presence of space occupying lesion in all the participants.
6. None of the participants had undergone vestibulotoxic medication and none complained any other illness earlier to the testing.

Normal Hearing Individuals (Group II)

The subjects included in the control group were age matched to the experimental group and fulfilled the following criteria.

1. The participants were examined to have normal hearing sensitivity (? 15 dBHL) at octaves from 250 Hz to 8000 Hz for air conduction and from 250 Hz to 4000 Hz for bone conduction.
2. The participants had 'A' type tympanogram with both ipsilateral and contralateral reflexes in immittance evaluation.
3. No history or presence of any relevant middle ear pathology, vestibular related abnormalities, neurological problems or had undergone vestibulotoxic medication.

Test instrument

All the tests were performed within noise permissible criteria of ANSI S3.1 (1991) in an acoustically treated room.

Instrumentation

1. Calibrated GSI audiometer (GSI VIASYS Healthcare, Wisconsin, USA) was used to perform threshold estimation (pure tone audiometry and speech audiometry) for ruling out any hearing loss components in both the groups to meet within inclusion criteria. Calibrated TDH 39 headphones (Telephonics, 815 Broad Hollow Road, Farmingdale, New York 11735) for AC threshold and calibrated B-71 bone vibrator (Radioear, KIMMETRICS, 22050 Mohawk Drive, Smithsburg, MD 21783) for BC threshold were used.
2. Calibrated GSI Tympstar Immittance (GSI VIASYS Healthcare, Wisconsin, USA) meter was used to measure tympanometry with a probe tone frequency of 226 Hz. The same equipment was used for measuring ipsilateral as well as contralateral reflexometry at 500, 1000, 2000, and 4000 Hz.
3. Calibrated ILO 292 V-6 (Otodynamics Ltd., Hatfield, Herts, UK) system was used to measure otoacoustic emissions.

4. Calibrated IHS (Intelligent Hearing System) Smart EP (3.94 USBez) system (Intelligent Hearing System, Florida, USA) was used to administer click evoked auditory brainstem response with ER-3A insert phones (Etymotic Research, Inc., 61 Martin Lane, Elk Grove Village, IL 60007, USA).
5. GN Otometrics manufactured v-HIT (GN Otometrics, Taastrup, Denmark) instrument along with laptop running Otosuite software and frenzel glasses were used for measuring VOR gain function of six semi-circular canal.
6. Videonystagmography (BioMed Jena GmbH, LutherstraBe 148, 07743 Jena, Germany) was used for performing caloric test in both the groups.

Procedure:

All the participants underwent various audiological and vestibular evaluations which are listed as follows:

Clinical Case History and Questionnaire Administration

A detailed case history was taken for all the participants in the study regarding the nature and onset of the hearing loss and attacks of vertigo (if any). Information on medical history and associated problems (if any such as neurological, visual conditions) were obtained. Also, Maryland Dizziness Questionnaire was administered on each individual of both the groups. Out of 5 sections of the questionnaire, only the 2nd section which was related with the features of dizziness was administered.

Pure Tone Audiometry:

Using the modified Hughson and Westlake procedure (Carhart & Jerger, 1959), air conduction threshold with the TDH 39 headphones and bone conduction thresholds with B-71 bone vibrator were obtained for octave frequencies from 250 to 8000 Hz and 250 to 4000 Hz respectively to investigate the hearing sensitivity of the each participants. Pure tone average (PTA) was calculated by taking mean at 500 Hz, 1 kHz, 2 kHz and 4 kHz.

Impedance audiometry:

Tympanometry and reflexometry were done to rule out any pathology at middle ear or auditory nerve level. Tympanometry was done at 226 Hz probe tone and acoustic reflex threshold was elicited for both ipsilateral and contralateral stimulation at 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz.

Otoacoustic emission:

Transient evoked OAEs were recorded in double walled sound treated room with 260 pairs of clicks stimuli. Probe fit was assessed by examining the stimulus spectrum and was adjusted to produce minimal ringing and flat spectrum. The stimulus was presented at 80 dB peak SPL. The OAEs were considered as present if the OAE SNR was greater than 6 dB for 3 consecutive frequencies. OAEs were recorded in non-linear mode.

Auditory Brainstem Response

Participants were placed in a reclining chair and cleaned at both the mastoids (M1, M2), and forehead (Fz) with skin abrasive followed by the vertical electrode placement. Also intra-electrode impedance and inter-electrode impedance were maintained < 5 kΩ and < 2 kΩ respectively. It was performed with making participants relaxed with reduced extraneous body movements.

Auditory brainstem response was recorded with 1500 sweeps of click stimulus through insert earphones. 90dBnHL click stimulus (rarefaction polarity) at 11.1/s repetition rates and the response was recorded in ipsilateral mode in single channel with minimum of two replications of the waveform in 12msec analysis time. The waveform response are recorded with bandpass filter between 100Hz to 3000Hz.

vHIT:

With laptop running Otosuite vestibular software, Video Head Impulse test was administered in well-lit room. Frenzel glasses with clean attached face cushion were tightened appropriately to avoid slippage such that the camera can track participants' pupil movement. The vHIT instrument had two laser pointers projected onto the wall, placed at the distance of 1 metre from participant seated adjustable for the purpose of calibration. Based on the Hex plot, the vHIT response was analysed where the VOR gain of each of all six semi-circular canals and the refixation saccades (if any) at the time of head thrust i.e. covert saccade, after the head thrust i.e. overt saccade were represented. Also effect of duration of ANSD in the results of vHIT was analysed. Also the age of the individuals and effect of duration of ANSD in the results of vHIT were analysed.

Caloric test:

VNG instrument was used for recording the response of caloric test. With the placement of the inverting electrode at 1.5cm lateral to the outer canthi of the left eye, non- inverting electrode at 1.5 cm lateral to the outer canthi of the right eye and ground at forehead, single channel recording was performed. Prior to the recording of each participants, calibration of the VNG instrument was carried out. This was followed by plotting the cumulative frequencies for the four recordings achieved from the individuals with ANSD on Hail-Stoll butterfly chart. With the help of Hail-Stoll Butterfly chart, the caloric responses were interpreted as normal, hypoactive or hyperactive based on its location i.e. within, below or above the normal limits of culmination frequency respectively. Also with digits as normal = 0; hypoactive = 1 and hyperactive = 2, the responses were labeled.

Statistical Analysis

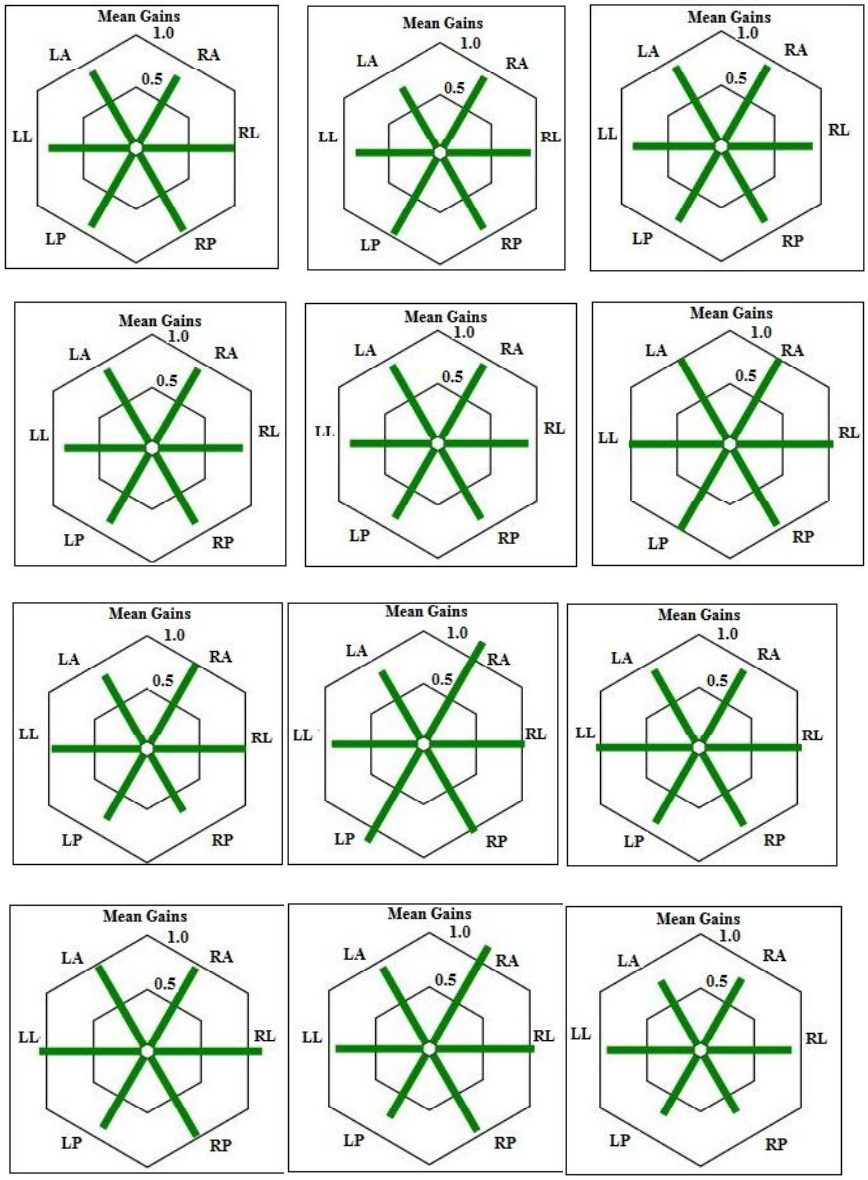
The entire statistics was done with the help of IBM SPSS 20.0 software. Shapiro Wilk test was done to check the normality of the data. Descriptive statistics was done to find out the mean and the standard deviations for VOR gain, VOR gain asymmetry. Independent sample t test was done to compare the VOR gain and VOR gain asymmetry between the normal individuals and individuals with auditory neuropathy spectrum disorders. Pearson correlation test was done to check the correlation between VOR gain and duration of six semi-circular canals in individual with ANSD. Pearson's correlation was performed to understand the relation between the VOR gain and VOR gain asymmetry with the duration of disease in individuals with ANSD. Chi square test was administered to understand the association of VOR gain with the degree of hearing loss in specific ear in individuals with ANSD. Chi square test was performed to understand the association of caloric test result and the degree of hearing loss in specific ear in individuals with ANSD. Chi square test was administered to check the association of caloric test result with the VOR gain of lateral canals in specific ear in individuals with ANSD.

Results

The present study was carried out with an aim to understand the correlation between vHIT and caloric test in individual diagnosed with auditory neuropathy spectrum disorder.

VOR gain

VOR gain was calculated for three planes of semi-circular canals. The hex plot representing VOR gain of vHIT responses recorded from each individuals of both the groups are given in Figures 1 and 2 where the VOR gain lesser than 0.8 was taken as abnormal(Patterson et al, 2015).



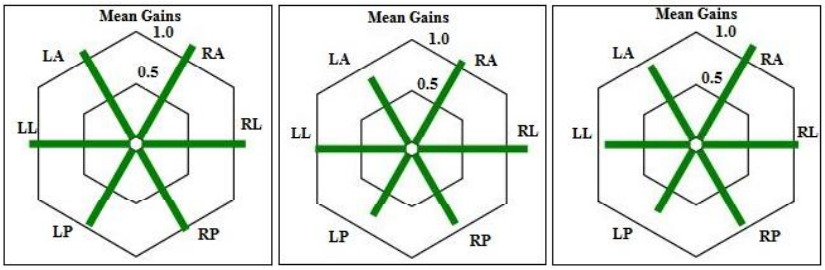


Figure.1: Hex plot of VOR gain measured with video head-impulse test response in three different planes of semi-circular canals in 15 normal hearing individuals.

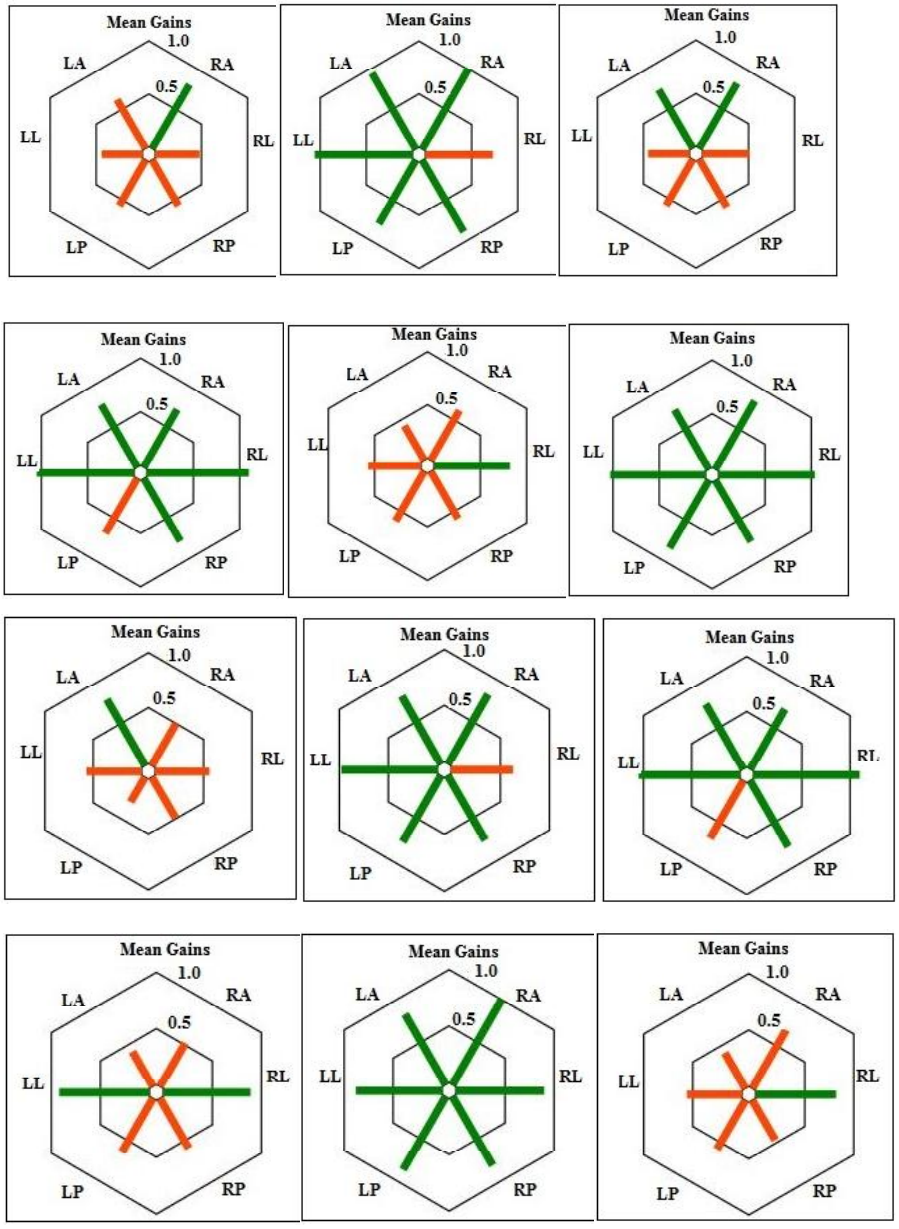


Figure 2: Hex plot of VOR gain measured with video head-impulse test response in three different planes of semi-circular canals in 15 individual with ANSD.

Descriptive statistics was done for both the groups to measure the mean and standard deviation of VOR gain in all three planes as represented in Table 1.

Table 1: Mean and standard deviation of VOR gain for all six semi-circular canals for both the groups.

Planes	ANSD Individuals (N= 15)		Normal Individuals (N=15)	
	x̄	SD.	x̄	SD.
Left Lateral (LL)	0.76	0.06	0.95	0.02
Right Lateral (RL)	0.77	0.05	1.00	0.02
Left Anterior (LA)	0.69	0.05	0.89	0.03
Right Posterior (RP)	0.70	0.04	0.89	0.04
Left Posterior (LP)	0.66	0.04	0.88	0.03
Right Anterior (RA)	0.77	0.04	0.97	0.04

Note: N= number of participants; x? = Mean; SD = Standard Deviation.

To find out if there exists any significant difference of VOR gain for all six canals between the groups, Independent sample't' test was administered as seen in Table 2.

Table 2: Independent sample test results for parameters of VOR gain between the groups.

Planes	Independent sample 't' test between ANSD and normal Individuals	
	t	p
Left Lateral (LL)	2.99	0.01
Right Lateral (RL)	4.61	0.00
Left Anterior (LA)	3.50	0.002
Right Posterior (RP)	3.58	0.001
Left Posterior (LP)	4.58	0.001
Right Anterior (RA)	3.37	0.002

VOR gain asymmetry

Descriptive statistics was administered for both the groups to measure the mean and standard deviation of VOR gain asymmetry in all three planes as represented in Table 3.

Table 3: Mean and standard deviation of VOR gain asymmetry of all three planes in both the groups.

Planes	ANSD Individuals (N= 15)		Normal Individuals (N=15)	
	x̄	SD.	x̄	SD.
Lateral	16.93	12.31	7.87	1.31
RALP	19.67	8.67	8.53	1.20
LARP	17.86	12.00	6.27	1.50

Note: N= number of participants; x? = mean; SD = standard deviation; LARP= Left Anterior Right Posterior; RALP= Right Anterior Left Posterior

It can be seen that all the planes of VOR gain asymmetry was significantly different in individuals with ANSD compared to that of normal individuals.

Refixation saccades

The corrective refixation saccades i.e. covert and overt saccades were studied in both the groups where it was observed to be present in individuals with ANSD. However it was found to be absent in normal hearing individuals. Table 4 represents the presence of the refixation saccades in each of the semi-circular canals in individuals with ANSD. Similarly, figure 3 shows the presence and absence of refixation saccades in different canals measured in one of the individuals with ANSD.

Table 4: Refixation saccades in each of the semi-circular canals present in the individuals with ANSD.

Semi-circular Canals	Covert Saccade	Overt Saccade	Covert + Overt	None
Left Lateral	3	2	7	3
Right Lateral	5	0	9	1
Left Anterior	2	0	1	12
Right Anterior	4	0	0	10
Left Posterior	2	1	2	10
Right posterior	2	0	3	9

From the table 4, it can be observed that the only covert saccade condition was present in all six semi-circular canals in individuals with ANSD. Similarly the both covert and overt saccades condition was found in all six canals except right anterior (RA). However the only overt saccade condition was present in left lateral (LL) and left posterior (LP) canals. Also the presence of saccades were witnessed more in number for lateral semi-circular canals. There were few individuals with ANSD in whom there were no saccades in different canals. None of the normal hearing individuals had saccades.

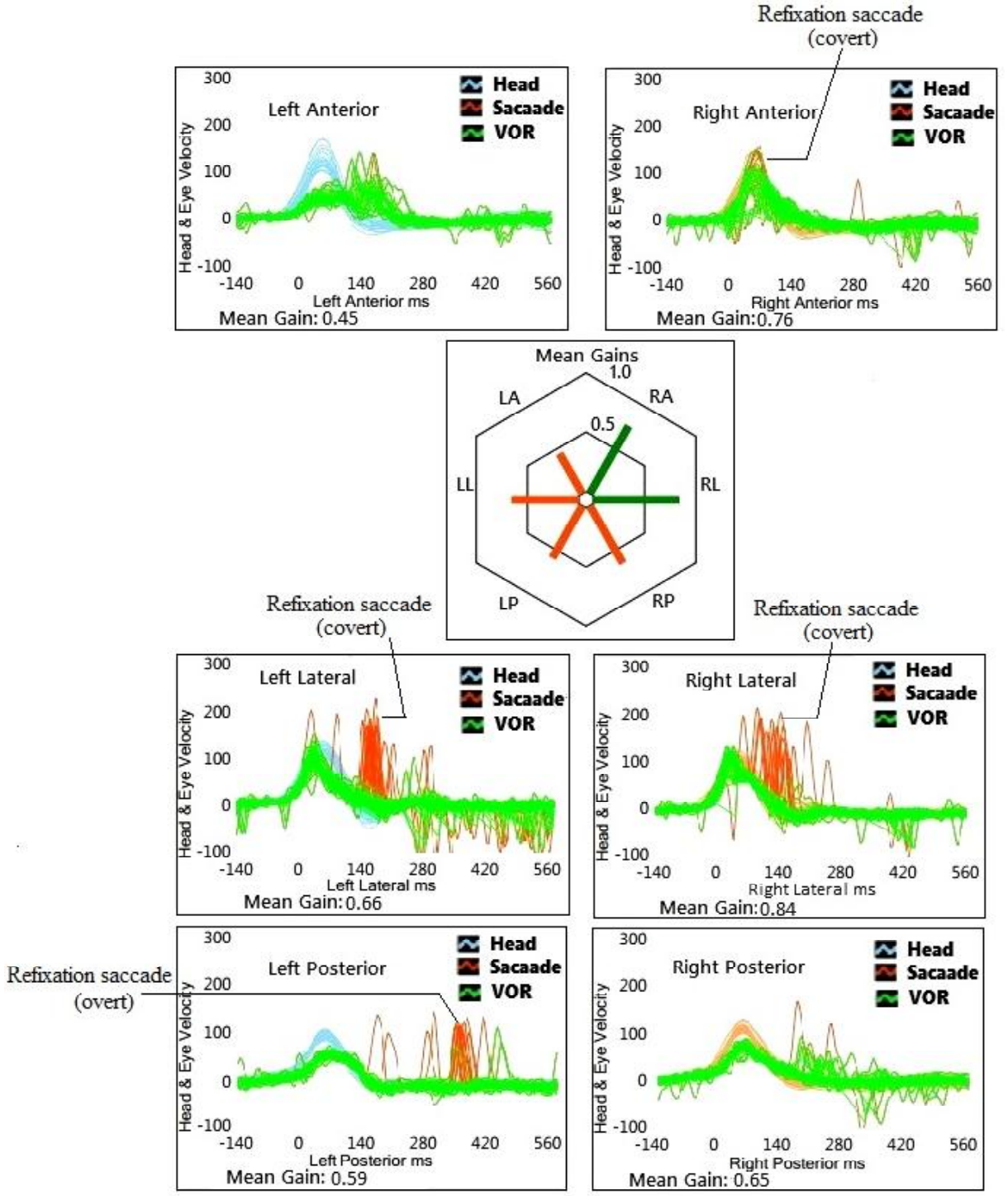


Figure 3: Video head-impulse test results in three different planes of an individual with ANSD. The head and eye velocities throughout different head impulses to the right or left side along with VOR gain and refixation saccades are shown.

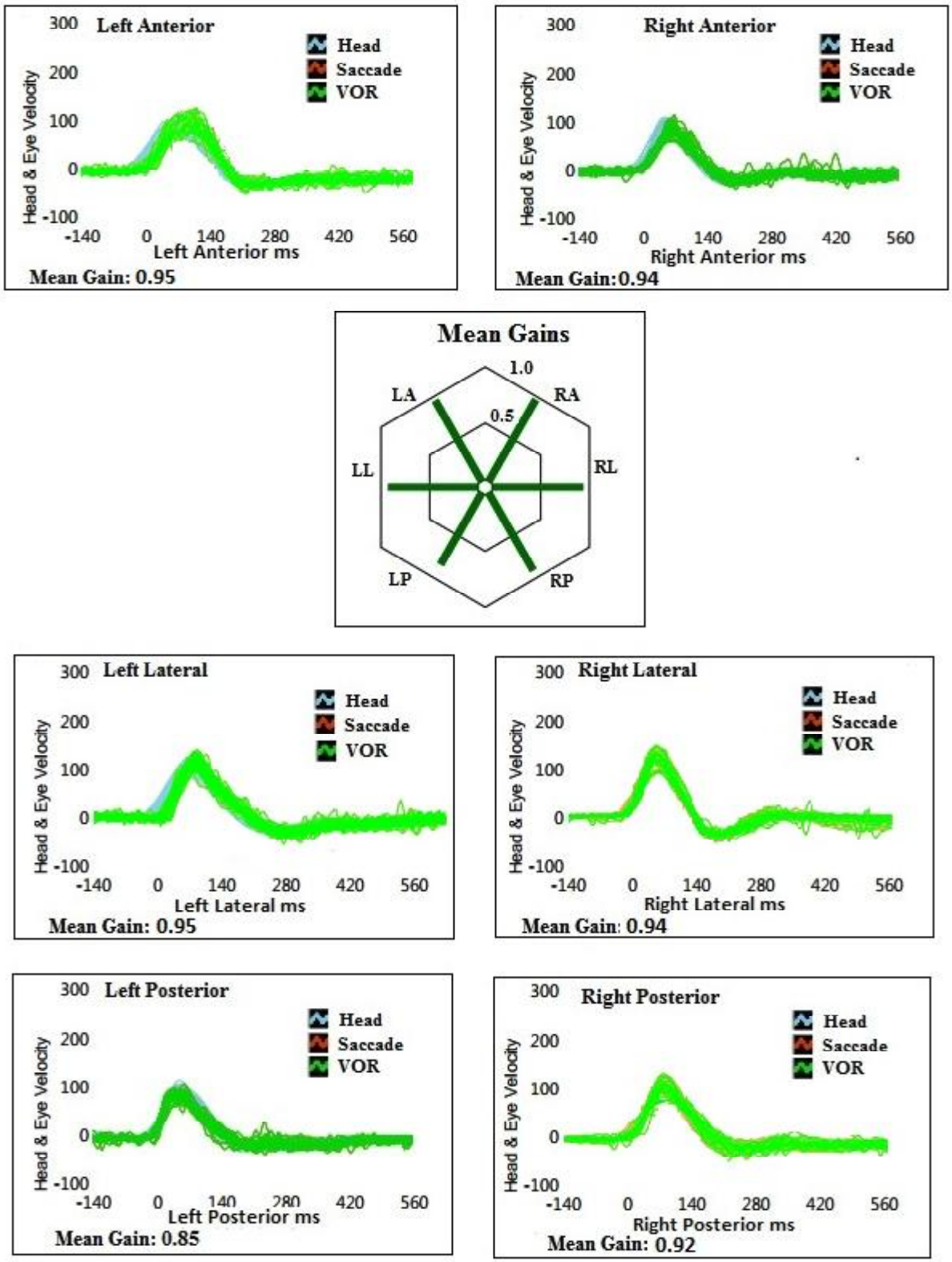


Figure 4: Video head-impulse test results in three different planes of a normal hearing individual with ANSD. The head and eye velocities throughout different head impulses to the right or left side along with VOR gain and absence of refixation saccades are shown.

Caloric test results in individuals with Auditory Neuropathy Spectrum Disorder

Bithermal caloric stimulation in all the normal hearing individuals participated in the study yielded cumulative frequencies for each of the four stimulations i.e. right cold (RC), left cold(LC), right warm(RW) and left warm(LW). It was found to have normal caloric response in all the normal hearing individuals based on the normative range of cumulative frequencies given. Moreover the Figure 4 represents Hail-Stoll butterfly chart with cumulation frequency of one of the normal hearing individual based on the number of nystagmus beats per 30 seconds in each of the 4 different stimulations.

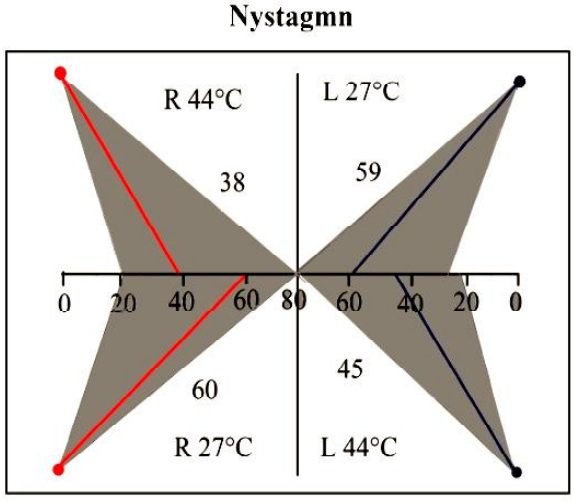


Figure 5: Hail-Stoll butterfly chart of one of the individuals with normal hearing.

The bithermal caloric test could be completed in ten individuals with ANSD with auditory neuropathy spectrum disorders due to various subject related problems. Four out of the five participants developed severe vertigo & vomiting sensation during the entire testing and the test was stopped for them, as these participants did not want to go ahead with the testing. In most of the individuals with ANSD, there was prevalence of hypoactive response followed by few normal responses. It was found to have 66.67% of hypoactive response for RW irrigation, 86.67% for LW irrigation, 86.67% for RC irrigation and 100% for LC irrigation in these individuals. Yet, three of them had normal cumulation frequency only in right warm (RW) caloric irrigation. However, none of the individuals had hyperactive response present.

These obtained results were plotted against the normative in Hail-Stoll butterfly chart. Hail-Stoll butterfly charts obtained of an individual with ANSD is represented in figure 6.

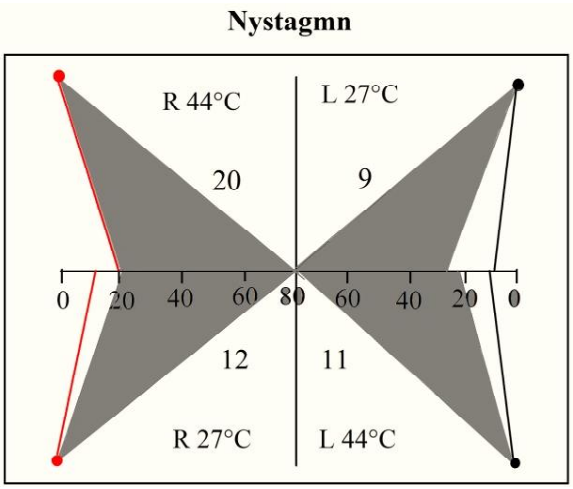


Figure 6: Hail-Stoll butterfly charts of 10 individuals with ANSD.

Correlation of VOR gain and asymmetry with duration

Pearson's correlation was performed to understand the relation between the VOR gain and VOR gain asymmetry with the duration in individuals with ANSD. However, no significant correlation was observed.

Association of VOR gain with degree of hearing loss

Chi square test was administered to understand the association of VOR gain with the degree of hearing loss in specific ear in individuals with ANSD. No significant association was seen between any of the canals and degree of hearing loss in both the ears.

Association of caloric test result with degree of hearing loss

Chi square test was performed to understand the association of caloric test result and the degree of hearing loss in specific ear in individuals with ANSD. No significant association was seen in either of the bithermal stimulation and degree of hearing loss in both the ears.

Association of caloric result with lateral canals VOR gain

Chi square test was administered to check the association of caloric test result with the VOR gain of lateral canals in specific ear in individuals with ANSD. No significant association was seen between any of the bithermal stimulation and lateral canals.

DISCUSSION

Present study revealed significantly lower VOR gain in individuals with ANSD than their normal hearing counterparts. Similar reports of reduced VOR gain values have been reported in various peripheral vestibular disorders (Weber et al, 2008; Macdougall et al, 2013; Blodow, Pannasch & Walther, 2013; Blödow et al, 2014; MacDougall et al, 2016; Taylor et al, 2016; Neupane et al, 2017). Reduced VOR gain in individuals with ANSD

indicates the probability of peripheral vestibular nerve involvement such that the functioning of its innervated end organ is also affected (Sinha et al, 2013). Hence these structural bases of peripheral vestibular nerve in individuals with ANSD results in reduced neural input as well as impaired synchronous firing of the vestibular nerve resulting in the presence of reduced VOR gain in these individuals. Also, in the present study, three individuals with ANSD had normal VOR gain which could be due to the slower development of the neuropathic degeneration in vestibular branch of eighth cranial nerve. Hence the evident manifestation and development of auditory deficits in ANSD would be more than the vestibular manifestation which could provide more chances for compensation in vestibular symptoms resulting normal VOR gain values in these 3 individuals with ANSD (Kumar et al., 2007).

Individuals with ANSD had significantly higher VOR gain asymmetry in all three coplanar axes (Lateral, LARP and RALP) than their normal hearing counterparts. Similar findings have been reported by Neupane et al (2017) in individuals with motion sickness where it was explained to have occurred due to the intra-sensory conflict between the semicircular canals of same planes. This phenomenon in return would have given rise to the discrepancy in neural input from three orthogonal planes to the cortical balance areas. Such a dilemmatic circumstance could generate difficulty in understanding the precise postural alignment of the body even when whole body is stimulated with similar acceleration. None of the other studies have yet been published reporting VOR gain asymmetry in any of the vestibular pathologies. However, before considering VOR gain asymmetry as a vital parameter in detecting vestibular pathology, there is a need of evaluating the sensitivity of VOR gain asymmetry across various different clinical vestibular conditions.

All normal hearing individuals had absent corrective refixation saccade, however, it was present in 100% of individuals with ANSD in the present study. Existence of these refixation saccades have been reported in various vestibular related pathologies (MacDougall et al, 2009; Macdougall et al, 2013; Jiménez & Fernández, 2016; Redondo-Martinez et al, 2016; Neupane et al, 2017) as an outcome for VOR gain reduction in individuals with vestibulopathy. Refixation saccades are suggestive of impaired semicircular canals when the variation is present between the stimulated sides of the coplanar canals to that of the non-stimulated side therefore, making VOR generate compensatory eye movement to maintain gaze stability even during head rotation (Matin, 1974; Bronstein & Gresty, 1991; Weber et al, 2008; MacDougall & Curthoys, 2012).

In the present study, in caloric test, for right warm stimulation 66.67% of the participants with ANSD had hypoactive responses, 86.67% had hypoactive responses for left warm irrigation, 86.67% had hypoactive responses for left warm and 100% of the participants had hypoactive responses for left cold irrigation. Such an response with significant absence of any quantifiable nystagmus beats has been reported as an indicative factor for the presence of predominant peripheral vestibular pathology (Kaga et al, 1996; Abdel-Nasser et al, 2006; Biswas, 2009). Hypoactive responses could be attributed to the structural variations at the level of vestibular nerve such as its beaded distorted appearance, fragmented myelin layer with larger gaps than the diameter of the nerve fiber itself reduction in number of nerve fibers joining receptor organ and scarpa ganglion, resulting in impairment of the action potentials conduction along these fibers.

There was found to be no correlation between VOR gain and asymmetry with duration of disorder in individuals with ANSD. Similar studies were reported in the literature where the group of individuals with ANSD having similar degree of hearing loss and speech identification scores even though the duration of the disorder was variable and vice versa (Spoendlin, 1974; Starr et al, 1996; Zeng, 2000; Jijo & Yathiraj, 2012). Also, there was found to have no association between VOR gain and caloric response with degree of hearing loss in these individuals. Similar result was reported by Sujeet et al. (2014) where they found dissociation between the caloric response and cVEMP response with degree of hearing loss. This highlights the heterogeneity of the disorder and its nature that varies across each individual with auditory neuropathy spectrum disorders. Also, the processing of these functions at the level of auditory nerve or vestibular nerve may go differently, resulting in lack of association between the VOR gain and caloric response with degree of hearing loss.

There was found to be dissociation between VOR gain of the lateral canals and canal paresis as indicated by bithermal caloric test in the individuals with ANSD. The major differences between the vHIT and caloric test are the temporal frequency of the stimulus used in each test (vHIT with high frequency stimulus vs caloric with low frequency stimulus) as well as the stimulus delivery mode (vHIT with head jerks vs caloric with thermal gradient) (McCaslin et al, 2014). Anatomically, afferent fibres from the peripheral zone of crista that synapse mainly with Type II hair cells, have slow conduction velocities retaining regular tonic neural spike timing and have higher gain at low frequencies with small phase shifts. However, afferent fibres from the central zone of crista that synapse mainly with Type I hair cells have irregular firing rates and have higher gain at high frequencies with large phase leads (Goldberg, 1991; Haque, Angelaki, & Dickman, 2004; Eatock & Songer, 2011). Therefore, it can be presumed that a short duration head jerk in vHIT may stimulate the irregular fibres on the central zone of crista and

tonic caloric stimulation may stimulate the regular fibres on the peripheral zone of crista. Thus its presently accepted that there can be selective weakening of different zones of crista that code low and high frequency movements of head or any other body parts (Park et al, 2005).

Conclusions

Present study revealed reduced VOR gain in individuals with ANSD. Refixation saccades in these individuals indicate the presence of compensatory mechanism of VOR. Hypoactive response for caloric test reveals the condition as the probable involvement of superior vestibular nerve or/and its innervated end organ in individuals with ANSD. However, the lack of correlation between VOR gain and asymmetry with the duration of disorder, as well as, VOR gain and caloric response with degree of hearing loss highlights the heterogeneity of the disorder and its nature that may vary across each individual. Moreover, the lack of association between VOR gain of the lateral canals and canal paresis in the individuals with ANSD indicates the variation in stimulus as well as response parameters between the two tests.

References

Abdel-Nasser, A., Elkhayat, N. M., Khalil, S. H., & Mahmoud, L. H. (2006). Audio-vestibular and neurological correlates in patients with auditory and peripheral neuropathy. Egyptian J Neurol Psych Neurosurg, 43, 253-267.

Berlin, C. I. (1999). Auditory neuropathy: using OAEs and ABRs from screening to management. Paper presented at the Seminars in Hearing.

Biswas, A. (2009). Clinical audio-vestibulometry for otologists and neurologists. Electronystagmography, 3rd edn. Bhalani Publishing house, Mumbai, 109-147.

Blödow, A., Heinze, M., Bloching, M. B., von Brevern, M., Radtke, A., & Lempert, T. (2014a). Caloric stimulation and video-head impulse testing in Meniere's disease and vestibular migraine. Acta Otolaryngol, 134(12), 1239-1244.

Blodow, A., Pannasch, S., & Walther, L. E. (2013). Detection of isolated covert saccades with the video head impulse test in peripheral vestibular disorders. Auris Nasus Larynx, 40(4), 348-351. doi: 10.1016/j.anl.2012.11.002

Bronstein, A., & Gresty, M. (1991). Compensatory eye movements in the presence of conflicting canal and otolith signals. Exp Brain Res, 85(3), 697-700.

Buetti, B., & Luxon, L. M. (2014). Vestibular involvement in peripheral neuropathy: A review. Int J Audiol, 53(6), 353-359.

Carhart, R., & Jerger, J. (1959). Preferred method for clinical determination of pure-tone thresholds. Journal of Speech & Hearing Disorders.

Davis, H., & Hirsh, S. (1979). A slow brain stem response for low-frequency audiometry. Audiology, 18(6), 445-461.

Eatock, R. A., & Songer, J. E. (2011). Vestibular hair cells and afferents: two channels for head motion signals. Annual review of neuroscience, 34, 501-534.

Fujikawa, S., & Starr, A. (2000). Vestibular neuropathy accompanying auditory and peripheral neuropathies. Archives of Otolaryngology-Head & Neck Surgery, 126(12), 1453-1456.

Goldberg, J. M. (1991). The vestibular end organs: morphological and physiological diversity of afferents. Current opinion in neurobiology, 1(2), 229-235.

Haque, A., Angelaki, D. E., & Dickman, J. D. (2004). Spatial tuning and dynamics of vestibular semicircular canal afferents in rhesus monkeys. Exp Brain Res, 155(1), 81-90.

Jijo, P., & Yathiraj, A. (2012). Audiological characteristics and duration of the disorder in individuals with auditory neuropathy spectrum disorder (ANSD)-a retrospective study. J Indian Speech Hear Assoc, 26(1), 17-26.

Jiménez, G. G., & Fernández, N. P. (2016). Reduction in posterior semicircular canal gain by age in video head impulse testing. Observational study. Acta Otorrinolaringologica (English Edition), 67(1), 15-22.

Kaga, K., Nakamura, M., Shinogami, M., Tsuzuku, T., Yamada, K., & Shindo, M. (1996). Auditory nerve disease of both ears revealed by auditory brainstem responses, electrocochleography and otoacoustic emissions. Scandinavian audiology, 25(4), 233-238.

Kowalski, J., Rasheva, M., & Zakrzewska, B. (1991). Visual and brainstem auditory evoked potentials in hereditary motor-sensory neuropathy. Electromyography and clinical neurophysiology, 31(3), 167-172.

Kumar, K., Singh, N., Sinha, S., Bharti, A., & Barman, A. (2007). Vestibular evoked myogenic potentials as a tool to assess vestibulocolic pathway dysfunction in individuals with auditory neuropathy. Asia Pac J Speech Lang Hear, 10, 110-118.

Kumar, U. A., & Jayaram, M. (2006). Prevalence and audiological characteristics in individuals with auditory neuropathy/auditory dys-synchrony: Prevalencia y caracteristicas audiológicas de la neuropatía/disincronía auditiva. Int J Audiol, 45(6), 360-366.

MacDougall, H. G., & Curthoys, I. S. (2012). Plasticity during vestibular compensation: the role of saccades. Front Neurol, 3, 21.

MacDougall, H. G., McGarvie, L. A., Halmagyi, G. M., Curthoys, I. S., & Weber, K. P. (2013). The video head impulse test (vHIT) detects vertical semicircular canal dysfunction. PLoS One, 8(4), e61488.

MacDougall, H. G., McGarvie, L. A., Halmagyi, G. M., Rogers, S. J., Manzari, L., Burgess, A. M., . . . Weber, K. P. (2016). A new saccadic indicator of peripheral vestibular function based on the video

head impulse test. Neurology.

MacDougall, H. G., Weber, K. P., McGarvie, L. A., Halmagyi, G. M., & Curthoys, I. S. (2009). The video head impulse test: diagnostic accuracy in peripheral vestibulopathy. Neurology, 73(14), 1134-1141.

Matin, E. (1974). Saccadic suppression: a review and an analysis. Psychological bulletin, 81(12), 899.

McCaslin, D. L., Jacobson, G. P., Bennett, M. L., Gruenwald, J. M., & Green, A. P. (2014). Predictive properties of the video head impulse test: measures of caloric symmetry and self-report dizziness handicap. Ear Hear, 35(5), e185-191.

Melgaard, B., & Zilstorff, K. (1979). Central vestibular involvement in peroneal muscle atrophy: a preliminary report. Annals of neurology, 5(2), 118-120.

Neupane, A., Gururaj, K., & Sinha, S.K. (2017). Higher Asymmetry Ratio and Refixation Saccades in Individuals with Motion Sickness. Journal of American Academy of Audiology, 29(3), 1-12.

Park, H. J., Migliaccio, A. A., Della Santina, C. C., Minor, L. B., & Carey, J. P. (2005). Search-coil head-thrust and caloric tests in Meniere's disease. Acta Otolaryngol, 125(8), 852-857.

Patterson, J. N., Bassett, A. M., Mollak, C. M., & Honaker, J. A. (2015). Effects of Hand Placement Technique on the Video Head Impulse Test (vHIT) in Younger and Older Adults. Otol Neurotol, 36(6), 1061-1068.

Redondo-Martinez, J., Becares-Martinez, C., Orts-Alborch, M., Garcia-Callejo, F. J., Perez-Carbonell, T., & Marco-Algarra, J. (2016). Relationship between video head impulse test (vHIT) and caloric test in patients with vestibular neuritis. Acta Otorrinolaringol Esp, 67(3), 156-161.

Scaioli, V., Pareyson, D., Avanzini, G., & Sghirlanzoni, A. (1992). F response and somatosensory and brainstem auditory evoked potential studies in HMSN type I and II. Journal of Neurology, Neurosurgery & Psychiatry, 55(11), 1027-1031.

Sheykholeslami, K., Kaga, K., Murofushi, T., & Hughes, D. W. (2000). Vestibular function in auditory neuropathy. Acta Otolaryngol, 120(7), 849-854.

Sheykholeslami, K., Schmerber, S., Habiby Kermany, M., & Kaga, K. (2005). Sacculo-collic pathway dysfunction accompanying auditory neuropathy. Acta Otolaryngol, 125(7), 786-791.

Sinha, S., Barman, A., Singh, N., Rajeshwari, G., & Sharanya, R. (2013). Vestibular test findings in individuals with auditory neuropathy: review. The Journal of Laryngology & Otology, 127(05), 448-451.

Sininger, Y., & Oba, S. (2001). Patients with auditory neuropathy: who are they and what can they hear. Auditory neuropathy: A new perspective on hearing disorders, 15-35.

Sininger, Y., & Starr, A. (2001). Auditory neuropathy: a new perspective on hearing disorders: Cengage Learning.

Spoendlin, H. (1974). Optic and cochleovestibular degenerations in hereditary ataxias. Brain, 97(1), 41-48.

Starr, A. (2001). The neurology of auditory neuropathy. Sininger I, Starr A. Auditory neuropathy, a new perspective on hearing disorders. San Diego: Singular Publishing Group, 37-49.

Starr, A., McPherson, D., Patterson, J., Don, M., Luxford, W., Shannon, R., . . . Waring, M. (1991). Absence of both auditory evoked potentials and auditory percepts dependent on timing cues. Brain, 114(3), 1157-1180.

Starr, A., Michalewski, H. J., Zeng, F. G., Fujikawa?Brooks, S., Linthicum, F., Kim, C. S., . . . Keats, B. (2003). Pathology and physiology of auditory neuropathy with a novel mutation in the MPZ gene (Tyr145? Ser). Brain, 126(7), 1604-1619.

Starr, A., Picton, T. W., Sininger, Y., Hood, L. J., & Berlin, C. I. (1996). Auditory neuropathy. Brain, 119(3), 741-753.

Sujeet, K. S., Niraj, K. S., Animesh, B., Rajeshwari, G., & Sharanya, R. (2014). Cervical vestibular evoked myogenic potentials and caloric test results in individuals with auditory neuropathy spectrum disorders. Journal of Vestibular Research, 24(4), 313-323.

Tang, T. P., McPherson, B., Yuen, K. C., Wong, L. L., & Lee, J. S. (2004). Auditory neuropathy/auditory dys-synchrony in school children with hearing loss: frequency of occurrence. Int J Pediatr Otorhinolaryngol, 68(2), 175-183.

Taylor, R. L., McGarvie, L. A., Reid, N., Young, A. S., Halmagyi, G. M., & Welgampola, M. S. (2016). Vestibular neuritis affects both superior and inferior vestibular nerves. Neurology, 87(16), 1704-1712.

Weber, K., Aw, S., Todd, M., McGarvie, L., Curthoys, I., & Halmagyi, G. (2008). Head impulse test in unilateral vestibular loss Vestibulo-ocular reflex and catch-up saccades. Neurology, 70(6), 454-463.

Zeng, F.-G. (2000). Auditory neuropathy: why some hearing-impaired listeners can hear but do not understand and how can DSP technology help them. Paper presented at the IEEE Signal Processing Society, Ninth DSP (DSP 2000) Workshop, Hunt, TX.

Zeng, F.-G. (2006). Speech perception in individuals with auditory neuropathy. Journal of Speech, Language, and Hearing Research, 49(2), 367-380