A search to possible pathways for later peaks of VEMP and N3 potential

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Abstract

The VEMP is an inhibitory potential recorded from the sternocleidomastoid (SCM) muscle in response to loud sounds. There are four peaks in VEMP which have been classified according to their latencies, known as p13, n23, n34, p44. Waves p13-n23 which are saccular in origin possibly and have been studied excessively due to the higher response rate in normals whereas the N34- P44 which are believed to be cochlear origin have been scarcely explored thus ignoring their clinical significance. There is another potential (N3 potential) which is thought to be originated from vestibular system is the negative peak at 3 msec in ABR recording. The aim of the study was to know any relation between the later peaks of VEMP and N3 potential and also to explore the possible routes for generation of these potentials. Two groups of subjects participated in the study. First group of subjects (control group) consisted of subjects in the age range of 16 to 45 years, with normal hearing (N=30) and the second group, (experimental group) consisted of 30 subjects with different configuration of sensorineural hearing loss. The experimental group was further divided into three subgroups based upon their hearing loss. Results revealed that all the four groups were significantly different in terms of presence of later peaks of VEMP. One way ANOVA showed significant difference in all the four groups. Results revealed that occurrence of later peaks of VEMP increased with increase in severity of hearing loss. It was observed that in only 6.7% of the subjects when VEMP was absent N3 potential was also absent. In 40% of the subjects when later peaks of VEMP were present N3 potentials were also present suggesting that there might be some similarity in pathway between later peaks of VEMP and N3 potential. Based on the results and literature available it was felt that there could be three possible pathways for later peaks of VEMP, first via Vestibulo-cochlear anastomosis, second through cochlear afferents and third via Olivocochlear bundle. Similarly for N3 potential there could be two sites of origin first could be vestibular nucleus and other one could be cochlear nucleus.

Key words: Later peaks of VEMP, N3 potential, Vestibulo-cochlear anastomosis, cochlear nucleus, olivocochlear bundle

Introduction

The VEMP by definition is a short-latency electromyogram recorded from the tonically contracted SCM in response to high-intensity acoustic stimulation (Bickford, Jacobson & Cody,

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1964; Cody & Bickford, 1969; Colebatch & Halmagyi, 1994). Colebatch et al., (1994) labeled the serial peaks P13, N23, N34 and P44, based on their latencies. The research studies have almost solely investigated wave P13-N23 complex. In contrast, Colebatch, Halmagyi and Skuse (1994) and Robertson and Ireland (1995) reported presence of N34-P44 peaks of VEMP in 60 to 68% of their normal hearing subjects interrupting the investigation of their clinical significance.

There is another potential which is thought of vestibular in origin and can be recorded during the ABR recording. The evoked potential is known as N3 potential which is a large negative deflection with latency of 3 ms (N3) and which has been recorded in patients with peripheral profound deafness (Kato et al., 1998). The later peaks of VEMP are thought to be of cochlear origin and N3 potentials were thought to be of vestibular origin though both of them are elicited by acoustic stimulation. Thus it was essential to know the anatomical and functional relationship between the cochlear and vestibular system which resulted in initial peaks of VEMPs to be of vestibular origin and later peaks to be of cochlear origin.

When n34-p44 peaks recorded from the Sternocleidomastoid muscle are thought to be of cochlear in origin this implies that auditory nerve needs to stimulate the vestibular nerve to elicit responses from the Sternocleidomastoid muscle. Thus it indicates that later potentials are likely to be absent or decreased in cases with hearing loss. However there are a few studies in which n34-p44 potentials are present in profound hearing loss individuals (Ferber-Viart, Dubreuil & Duclaux, 1999) which raised the question whether these potentials are really of cochlear origin. This could be understood if the study is carried out in normal and in subjects with hearing loss. Kato et.al., (1998) studied the N3 potential in profound hearing loss cases and presence of this negative peak confirms vestibular system especially saccule as a site of origin whereas N34-P44 peaks of VEMPs recorded by stimulating the SCM muscle are thought to be cochlear origin.

Hence it suggested that there could be some relation between the two potentials. If there is a relationship between the two then what could be the possible pathway for both later peaks of VEMP and N3 potential? How these potentials can help in understanding Vestibulo-cochlear nerve pathology?

To find out the answers for all these questions the present study was taken up with the following aims:

- To study the relationship between the severity of hearing loss and N34-P44 potentials of VEMP
- To understand the relationship between N3 potentials and VEMP
- To understand the possible route for later peaks of VEMP and the possible route for the N3 potential
- To understand the structural and functional relationship between the Vestibulo-cochlear nerve root

Method

Subjects

The present study comprised of two major subject groups, the Control group and the Clinical group. The Control group comprised of 30 (60 ears) normal hearing individuals in the age range

of 16 to 45 years. The Clinical group consisted of 35 subjects with sensorineural hearing loss in the age range of 16 to 45 years. This clinical group was further sub divided into three groups based on their severity of hearing loss as:

Group A: 10 (20 ears) individuals with mild sensorineural hearing loss
Group B: 10 (20 ears) individuals with severe sensorineural hearing loss
Group C: 15 (30 ears) individuals with profound hearing loss

Subject selection criteria

Control group:

1. All the subjects had normal hearing sensitivity, having puretone thresholds within 15 dBHL at frequencies from 250 to 8000 Hz in octaves, in both ears.
2. All of them showed 'A' type tympanogram with normal reflexes in both ears
3. They did not have any history or presence of any otological problems
4. They reported no complaints of giddiness, vertigo, balancing problem, spondilitis and high blood pressure
5. All the subjects had UCL (uncomfortable level) above 105dB HL

Clinical group:

1. Subjects with pure tone thresholds of varying degrees with sensorineural hearing loss were chosen.
2. Attempts were made to rule out space occupying lesions based on ABR results or neurological assessment as and when it was required.
3. They had 'A' type tympanogram with presence, elevated or absence of acoustic reflexes in both the ears.
4. Subjects did not have any history of middle ear pathology.
5. Subjects had UCL above 105 dBHL, as confirmed by UCL testing.

Instrumentation:

The vestibular evoked myogenic potentials and N3 potentials were recorded using IHS smart EP version 2.39 (Intelligent hearing system, Florida, USA) instrument

Procedure

Phase I: Routine evaluations for the selection of subjects

1. Detailed case history was taken for all the individuals to rule out any history or presence of any otological problems, general weakness, giddiness, vertigo, high blood pressure and spondilitis.
2. The pure tone audiometric thresholds were obtained using modified version of Hugson and Westlake procedure (Carhart & Jerger, 1959) with the help of GSI-61 Audiometer.
3. Tympanometry and acoustic reflexes were tested using GSI Tympstar to rule out the presence of middle ear pathology.

4. ABR was done by cleaning the electrode site with the help of skin preparing gel to rule out the possibility of space occupying lesion.

Subjects who fulfilled the selection criteria either for control group and or for clinical group were considered for the study.

Phase II: Experiment

VEMP: VEMPs were recorded for all the subjects in control and clinical group.
Subjects were instructed:

- To sit straight and turn their head to opposite side of the test ear so as to stretch the Ipsilateral Sternocleidomastoid muscle.
- To close their eyes at the time of recording to avoid interference by occulomotor reflexes.
- To avoid extraneous movements of head, neck and jaw to elude muscle artifacts.

Prior to the VEMP and N3 potential recording the electrode sites were cleaned using skin preparation gel to reduce the impedance. The electrodes were placed on respective sites as given in the table 1 and 2 for VEMP and N3 potential respectively with conducting paste to improve the conduction. It was ensured that impedance was within 5 K ohms at each recording site and inter electrode impedance was within 3 K ohms to obtain good responses.

The protocol proposed by Huang T., Young Y. and Cheng P. (2004) was used in the present study to record VEMP which is given below:

Table 2: Depicts the parameters used for VEMP recording

Electrode Montage	
Inverting electrode	Sternoclavicular joint
Non Inverting electrode	Midpoint of SCM.
Ground electrode	Forehead
Acquisition parameters	
Analysis time	100 msec.
Filter setting	20-2000 Hz.
Amplification	50,000
Stimulus parameters	
Type of stimulus	Clicks
Repetition rate	5/sec.
Polarity	Rarefraction
Intensity	105 dBnHL.
Stimulus duration	500 µsec.

N3 potential: N3 potentials were recorded for individuals with profound hearing loss. Instructions given to the subjects were as follows:

1. Subjects were asked to sit in chair and close their eyes.
2. They were informed not to stretch their neck, instead were asked to sit quietly.

3. They were instructed to avoid extraneous movements of head, neck and jaw to elude muscle artifacts.

Table 3: Depicts the parameters used to record N3 potential

Electrode montage	
Non Inverting electrode	Vertex
Inverting electrode	Ipsilateral mastoid
Ground electrode	Nape of the neck
Acquisition parameters	
Analysis time	10 msec.
Filter setting	100 – 3000 Hz.
Amplification	10,000
Stimulus parameters	
Type of stimulus	Clicks
Repetition rate	10/sec.
Polarity	Rarefraction
Intensity	95 dBnHL
Total no. of stimuli	500 stimuli.
Stimulus duration	100 µsec.

N3 potential was recorded using the protocol proposed by Toshihisa, Iwasakib, Takaib and Takegoshic, (2005) as shown in the table above. For all the subjects VEMPs and N3 potential were recorded for right side first and then for the left side. It was ensured that each recording was repeated to have reproducibility of the responses. For subjects in Group C, N3 potential was recorded after the VEMP recording.

Results

The data was statistically analyzed using One-way ANOVA, Duncan's post-hoc test, Chi-square test and Cramer's V test. All the statistical analysis was carried out using SPSS 10 software.

1. Relationship between the severity of hearing loss and N34 – P44 potentials of VEMP:

Table 4: Depicts the Mean and SD of latencies and peak to peak amplitude of later peaks of VEMP

Entity	**No.**	**Group**	**Mean**	**SD**	**Min.**	**Max.**
N34	13	Mild	32.5538	3.1532	28.80	37.60
	14	Severe	35.1714	2.2228	32.00	38.60
	24	Profound	34.7937	3.0226	29.00	39.80
	31	Normal	33.3161	3.1136	27.80	39.20
P44	13	Mild	42.6154	3.4024	37.60	47.20
	14	Severe	46.0143	2.1260	43.00	49.80
	24	Profound	44.8138	3.2994	38.20	50.00
	31	Normal	43.7574	3.1351	38.40	49.00
PP	13	Mild	4.4885	2.2823	2.18	9.32
	14	Severe	4.1950	1.5442	2.09	6.78
	24	Profound	4.4579	1.8855	1.58	8.97
	31	Normal	3.3110	2.7835	0.90	15.00

Table shows that there is a slight variation in latency and peak to peak amplitude of N34 – P44 peak of VEMP with the varying degree of hearing sensitivity.

The One-way ANOVA was administered to check whether latency and peak to peak amplitude are significantly different or not and if ANOVA was significant then Duncan's post-hoc test was done to see significant differences between the groups of subjects. Results revealed that control group and subgroups of clinical group were significantly different for latency N34 peak and P44 peak and not for peak to peak amplitude for later peaks of VEMP ($p<0.05$).

On Duncan's post-hoc test showed there was significant difference between the data obtained from individuals with mild hearing loss from those with profound and severe hearing loss and rest of the groups were not different for N34 peak of VEMP.

Table 5: Depicts the Duncan's post-hoc test results for N34 peak of VEMP

Groups	**No. of ears**	**Subset for alpha = .05**	
		1	**2**
Mild	13	32.5538	
Normal	31	33.3161	33.3161
Profound	24		34.7937
Severe	14		35.1714
Sig.		0.443	0.079

For P44 peak of VEMP significant difference was observed between individuals with mild hearing loss and with severe and profound hearing loss but not from individuals with normal hearing, whereas individuals with normal hearing differed significantly from individuals with severe hearing loss and not from individuals with mild and profound hearing loss ($p<0.05$).

Table 6: Depicts the Duncan's post-hoc test results for P44 peak of VEMP

Groups	**No. of ears**	**Subset for alpha = .05**		
		1	**2**	**3**
Mild	13	42.6154		
Normal	31	43.7574	43.7574	
Profound	24		44.8137	44.8137
Severe	14			46.0143
Sig.		.270	.308	.247

Further data was analyzed to see if there is any association between the presence or absence of N34- P44 wave and hearing sensitivity based on the data obtained. Chi square test was administered and results revealed that there lies a significant association between the presence of N34 – P44 wave of VEMP and increase in hearing sensitivity, χ^2 (1) = 0.464, ($0.05<p<0.1$).

On Cramer's V test it was seen that association is 23% and the response rate to eliciting wave N34-P44 from group with normal hearing sensitivity, mild, severe group and profound hearing loss group were 51.7%, 65%, 70% and 80% respectively as seen in the following table.

Table 7: No. of ears and percentage of the presence or absence of later peaks of VEMP

Groups	**No. of ears**	**Absent**	**Present**	**Total**
Mild	No. of ears	7	13	20
		35.0%	65.0%	100.0%
Severe	No. of ears	6	14	20
		30.0%	70.0%	100.0%
Profound	No. of ears	6	24	30
		20.0%	80.0%	100.0%
Normal	No. of ears	29	31	60
		48.3%	51.7%	100.0%
Total	No. of ears	48	82	130
		36.9%	63.1%	100.0%

2. Relationship between N3 potentials and N34 – P44 peaks of VEMPs:

The later peaks of VEMP and N3 potential were recorded in individuals with profound hearing loss. The mean and the standard deviation along with minimum and maximum values for N3 potential latency and absolute amplitude are given in the table below:

Table 8: Mean, Standard deviation (SD), minimum and maximum values for N3 potential latency and absolute amplitude in subjects with profound hearing loss

Parameters	**Mean**	**SD**	**Minimum**	**Maximum**
N3	3.3033	0.4782	2.28	4.05
Amplitude	-0.2142	0.3941	0.22	-1.25

It was seen that N3 potential was recorded with a range was of 1.77 msec and for absolute amplitude the range was 1.47 micro volts. The Chi-square test was done to see if there lies any association between the presence and absence of N34-P44 peak of VEMP and presence or absence of N3 potential. Chi-square test showed no significant association between the presence and absence of the two potentials for χ^2 (3) = 7.486, ($p>0.05$). On cross tabulation it was evident that N3 potential was present in 4 ears when N34-P44 peak of VEMP were absent where as 12 ears had absence of N3 potential with presence of later peaks of VEMP.

Form the table it is evident that only 6.7% of the subjects when VEMP was absent, N3 potential was also absent. And for 40% of the subjects when later peaks of VEMP were present, N3 potentials were also present. Thus 46.7% of the population had similar test results and 53.3% did not show the same findings

Table 9: Distribution of data for presence/absence of later peaks of VEMP and N3 potential in subjects with profound hearing loss

Parameter		**N3**		**Total**
		Absent	Present	
N34 –P44	No. of ears	2	4	6
	Absent	6.7%	13.3%	20%
	No. of ears	12	12	24
	present	40%	40%	80%
Total	No. of ears	14	16	30
		46.7%	53.3%	100%

These results are discussed to understand the possible root of VEMP and the physiological relationship between the Vestibulo-cochlear nerve routes.

Discussion

The results of the present study have shown that there is association between presence of later peaks of VEMP and severity of hearing loss. Chi-square test showed that there is association of 23% between presence of N34- P44 wave of VEMP and severity of hearing loss. Duncan's post-hoc test did not show any specific trend in increase or decrease of latencies and amplitude of later peaks of VEMP in individuals with different hearing sensitivity. On the Cramer's V test it was seen that in individuals with normal hearing sensitivity only 51% had presence of N34-P44 peak of VEMP compared to 80% seen in individuals with profound hearing loss. The initial findings reported in the literature by Colebatch et al. (1994) who could record later peaks of VEMP from 60% of individuals with normal hearing, whereas Huang et al. (2004) could record from 80% of the individuals with normal hearing sensitivity.

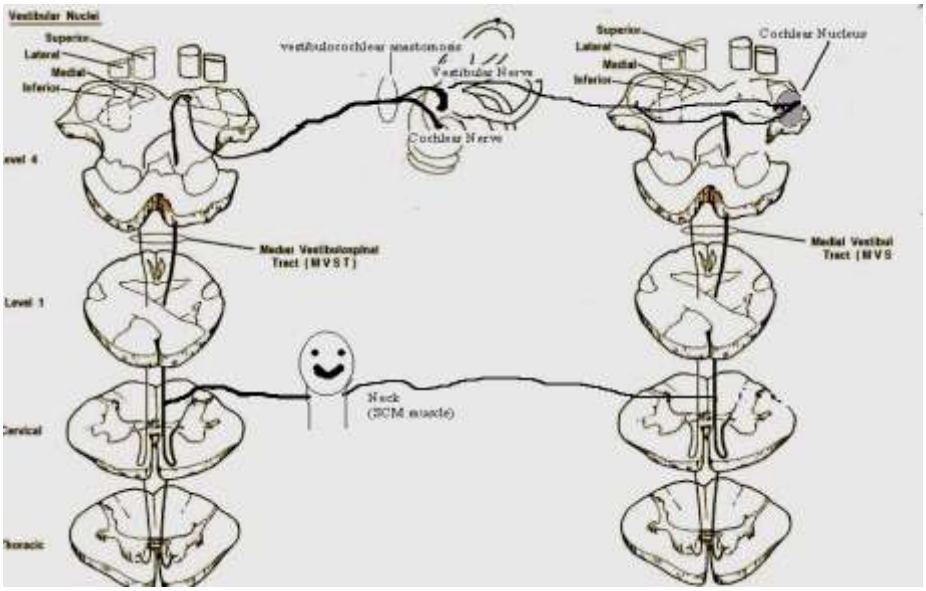
In the present study later peaks of VEMP could be recorded from 80% of the individuals with profound hearing loss which is much higher than what has been reported in the literature by Wu and Young in (2002). They could record later peaks of VEMP only from 45% of the individuals with sudden hearing loss. It is also evident from the current study that as the severity of hearing loss increased the percentage of presence of later peaks of VEMP also increased. However, there are no such studies available in the literature which compared the presence of later peaks of VEMP in population with different degrees of hearing loss.

In the current study the findings suggest that probably later peaks of VEMP may not be cochlear origin as wave complex is present in 80% of individuals with profound hearing loss. However, there are reports in literature which support the fact that later peaks of VEMP are cochlear in origin as Colebatch et al., (1994) found presence of later peaks of VEMP in before and after selective vestibular nerve section.

Taking these results together it might suggests that wave n34–p44 may have both a cochlear and vestibular origin. The possible pathways for generation of N34- P44 peak of VEMP are discussed below:

The first possible pathway:

There are histopathological and morphological studies which have proven that the (saccular nerve) inferior vestibular nerve links to the cochlear nerve in internal acoustic canal and this intimate connection was named as Vestibulocochlear anastomosis (Oort, 1918; Rasmussen, 1940; House, 1961; Kim et al., 1998; Nageris, Kalmanowitz, Segal & Frenkiel, 2000; Labrousse, Ouedraogo, Avisse, Chays & Delattre, 2005). It was also found that cochlear afferent fibers go to the lateral ipsilateral vestibular nucleus (Cazals, Erre & Aurousseau, 1987) may be via vestibulocochlear anastomosis. There are reports which suggest that vestibulospinal nerve fibers from the medial and lateral vestibular nucleus descend down to the SCM muscle via MVST and to leg muscle via LVST, Colebatch (1992 & 1994).



Pathway I **Pathway II**

Figure 1: possible pathways for generation of later peaks of VEMP

It can be explained that from the fig. 1 that there is a possibility of acoustic stimulation to the cochlea stimulates the cochlear afferents which in turn might be stimulating vestibular nucleus in the brainstem via vestibulocochlear anastomosis. From there impulses are sent to the neck muscles via the medial vestibulospinal tract (MVST) and the leg muscles via the lateral vestibulospinal tract (LVST).

Second possible pathway:

On the other hand the current study suggests that the wave n34- p44 could also be obtained in deaf ears, implying that they were probably not of cochlear afferent origin as WU and Young (2002) observed the presence of later peaks of VEMP in 45% of subjects after sudden hearing loss. This implies that these peaks might occur via a polysynaptic pathway also

terminating on the motor neuron of SCM muscles, Wilson et al. (1969), Murofushi, Halmagyi, Yavor & Colebatch (1996) & Kushiro, Zakir, Sato, Ono, Ogawa and Meng (2000).

It can be seen from figure.1 that inferior vestibular nerve originates from saccule and utricle and sends a few projections into the dorsal cochlear nucleus. Hence the possible pathway could be from saccule to the inferior vestibular nerve fibres progress to the vestibular nuclei which are responsible for generation of early peaks of VEMP, whereas few fibers progressed to the cochlear nucleus. These few fibers later via the intimate connection between cochlear and vestibular nucleus terminates in medial and lateral vestibular nuclei. From this vestibular nuclei they innervate the SCM i.e. neck muscle via MVST and to leg muscle via LVST resulting in the generation of later peaks of VEMP.

From the above discussion and information from the literature it may be concluded that the above mentioned route could be the generator of later peaks of VEMP. This could be the possible route as 80% of individuals with profound hearing loss showed presence of later peaks of VEMP. Thus the longer latencies of later peaks could be due to the longer path travelled by the nerve fibers to stimulate the neck muscle.

Third possible pathway:

There could also be a possibility of the third route for generation of later peaks of VEMP via olivocochlear bundle. Brown (1993), Benson and Brown (1996) and Winter, Robertson and Cole (1989) found that the medial and lateral olivocochlear fiber systems give off branches to the inferior vestibular nucleus and the lateral vestibular nucleus respectively, apart from those given to the cochlear nucleus in the mouse and guinea pig. This suggests that these few olivocochlear fibers might progress further to vestibulospinal tract to stimulate SCM muscle via MVST.

Thus from the above discussion it is evident that there could be three possible pathways which help in eliciting later peaks of VEMP in individuals with normal hearing sensitivity as well as in individuals with profound hearing loss. The possible first and third pathway are more applicable for generation of later peaks of VEMP in individuals with normal hearing sensitivity whereas second possible pathway might explain the generation of later peaks of VEMP in individuals with profound hearing loss. So these two mechanisms for generation of later peaks of VEMP for two different groups might have resulted in lesser and more percentage of presence of later peaks of VEMP in individuals with normal hearing sensitivity or individuals with profound hearing loss. Thus, this presence of later peaks of VEMP can give some information of integrity of cochlear nerve which in turn might help in selecting candidates for cochlear implants. The proposed possible second pathway might encourage considering acoustic stimulation of the saccule as an alternative to the cochlear implant.

Pathways for N3 potential:

To understand the possible pathway for N3 potential, later peaks of VEMP and N3 potential were recorded from individuals with profound hearing loss. Results revealed 46.7% of the population had similar results suggesting that there could be similar pathway for later peaks

of VEMP and N3 potential, whereas 53% showed the disagreement between the results suggesting that there could be two different sites for generation of N3 potential. Thus there could also be two different sites of origin for N3 potential, one could be vestibular nucleus and other could be the cochlear nucleus.

The possible site for generation of N3 potential suggested in literature is vestibular nucleus. Mason (1996) reported a short latency negative component during ABR recordings in child candidates for cochlear implant suggesting it to be of vestibular origin hence the relation between sound and the vestibular system is undoubtedly believed. Studies from Elidan and Honrubia (1987) and Cazals et al., (1987) suggested origin of N3 potential could be the vestibular nerve and vestibular nuclei. Nong, Kyuna, Owa and Noda (2002) and Ochi and Ohashi (2001) suggested that N3 potential might be of vestibular origin as is VEMP. Thus, the high level of acoustic stimulation to the cochlea stimulates the saccule. This excitation of saccular cells in the saccule then sends the information to the vestibular nucleus via inferior vestibular nerve and resulted in low amplitude negative potential at around 3 msec. In the process of exploring the possible pathway for later peaks of VEMP it was observed that a few fibers of the vestibulospinal tract progress to the dorsal cochlear nucleus (Bukoswka, 2002). This suggests that negative peak at 3 msec might be of the cochlear nucleus origin also.

The second possible route could be thus explained from the fact that few saccular fibers enter the cochlear nucleus while progressing towards vestibular nucleus (Kevetter & Perachio, 1989). The intense stimulation to the cochlea stimulates the saccule. Stimulation to the sensory cells of saccule in turn sends the information to the dorsal cochlear nucleus which might result in generation of low amplitude negative peak at around 3 msec. The above discussion suggests that there could be three different pathways for the generation of the later peaks of VEMP and two possible pathways for generation of N3 potential.

Conclusion

It can be concluded that later peaks of VEMP can be recorded at higher percentage in profound hearing loss individuals compared to normals. There lies relationship between later peaks of VEMP and N3 potential as 46.7% of the population showed similar results. Thus there might be possibility of multiple pathways for later peaks of VEMP as well as N3 potential. For later peaks of VEMP one pathway might be from cochlea to the Vestibulo-cochlear anastomosis further progressing to the vestibular nuclei ending at SCM muscle and second could be via cochlear nucleus to the vestibular nucleus to the SCM muscle. There could also be a third pathway from cochlea to the medial olivocochlear bundle to the vestibular nucleus descending to the SCM muscle.

For N3 potential there could be two possible sites of origins. First as Vestibular nucleus and second might be the cochlear nucleus by stimulating saccule using acoustic stimulation. Thus this communication between the two systems might have lot of implications in field of Audiology.

Implications of the study:

- The study has implication in knowing the pathophysiology of hearing loss in profound hearing loss.
- Non-invasively the condition of the vestibular system (especially the saccule and inferior vestibular nerve and medial and lateral vestibular nuclei) can be assessed as the later peaks of VEMP and N3 potentials can only be obtained when saccule is intact and can serve as 2 different tests to check saccular system.
- Presence of later peaks of VEMP in profound hearing loss might suggest about the residual function of cochlea/cochlear nerve in turn helping to decide on candidacy for cochlear implant.
- On the basis of all possible pathways it is evident that there might be a possibility for a totally deaf person to process acoustic stimulation via a saccular implant.

Limitations of the study:

- All the possible pathways proposed have been based on the electrophysiological results. It would have been better if the results were correlated with other direct methods like injecting wheat germ agglutinin-horseradish peroxidase (WGA-HRP).
- Multiple pathway for each potential limits to find out exact lesion when they are absent, leading to lack of diagnostic purpose.

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