EFFECT OF OCCUPATIONAL NOISE EXPOSURE ON EFFERENT AUDITORY SYSTEM

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CERTIFICATE

This is to certify that this dissertation entitled "**Effect of occupational noise exposure on efferent auditory system**" is a bonafide work submitted as a part for the fulfilment for the degree of Master of Science (Audiology) of the student Registration Number: 16AUD031. This has been carried out under the guidance of the faculty of this institute and has not been submitted earlier to any other University for the award of any other Diploma or Degree.

Mysore

April, 2018

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CERTIFICATE

This is to certify that this dissertation entitled "**Effect of occupational noise exposure on efferent auditory system"** has been prepared under my supervision and guidance. It is also being certified that this dissertation has not been submitted earlier to any other University for the award of any other Diploma or Degree.

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DECLARATION

This is to certify that this dissertation entitled **"Effect of occupational noise exposure on efferent auditory system"** is the result of my own study under the guidance of Dr. Ganapathy M.K., Lecturer in Audiology, Department of Audiology, All India Institute of Speech and Hearing, Mysore and has not been submitted earlier to any other University for the award of any other Diploma or Degree.

Mysore **Registration No: 16AUD031**

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Dedicated to Pappa, Amma,

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Abstract

Exposure to occupational noise affects efferent auditory system. This makes speech understanding difficult in the presence of noise. Efferent system can be assessed by measuring contralateral suppression of otoacoustic emissions and measuring speech identification scores in the presence of noise.

Objective: To study the effect of occupational noise exposure on efferent auditory system by measuring contralateral suppression of otoacoustic emissions (CSOAE) and measuring speech identification scores (SIS) in the presence of contralateral noise between groups of individuals who are not exposed to occupational noise (Group1) and individuals who are exposed occupational noise (Group2) and also to check for the correlation between CSOAE and shift in SIS in the presence of contralateral noise.

Method: Group1 included 35 participants and Group2 included 30 participants who had average pure tone threshold < 25 in all frequencies. TEOEs were measure with and without contralateral BBN at 30dB SL and subtracted to get the amount of suppression and SIS was measured with and without contralateral BBN 30dB SL both at ipsilateral +10 dB SNR and +15 dB SNR.

Results: Results indicated no significant difference in SIS with and without contralateral BBN between groups. There was significant difference in SIS with and without contralateral BBN in group2. There was significant difference in the CSOAE between group, Group1 showed greater suppression compared to Group2. No significant ear effect was seen for both the measures. There was no significant correlation between CSOAE and shift in SIS with contralateral BBN.

Conclusion: From this study it can be concluded that efferent auditory system is getting affected by occupational noise exposure and it leads to difficulty in understanding speech in the presence of noise.

TABLE OF CONTENTS

List of Tables

List of Figures

Abstract

Noise exposure can lead to permanent hearing loss or even changes of the cellular properties within the central auditory pathway (Groschel, Ryll, Gotze, Ernst & Basta, 2014). Kumar, Ameenudin, and Sangamanatha, (2012) reported that individuals exposed to occupational noise had poorer temporal processing and speech recognition. De souza Alcaras et al., 2013 reported that there was poor suppression effect in individuals exposed to occupational noise. Suggesting that chronic exposure to noise may have impaired the functioning of the medial olivocochlear efferent auditory system. Medial olivocochlear fibers are myelinated and terminate at outer hair cells (Ciuman, 2010). The medial olivocochlear bundle plays an inhibitory role on the activity of outer hair cells (Kemp, 1978). Its stimulation reduces auditory nerve response, basilar membrane motility and OAEs amplitude (Kemp, 1978). Thus, exposure to occupational noise may affect efferent auditory system. This may lead to reduced contralateral suppression and difficulty to understand speech perception in the presence of noise in individuals who are exposed to occupational noise.

Objective: To study the effect of occupational noise exposure on efferent auditory system by measuring contralateral suppression of otoacoustic emissions (CSOAE) and measuring speech identification scores (SIS) in the presence of contralateral noise between groups of individuals who are not exposed to occupational noise (Group1) and individuals who are exposed occupational noise (Group2). Further to check for the correlation between CSOAE and shift in SIS in the presence of contralateral noise.

Method: Group1 included 35 participants without history of occupational noise exposure and Group 2 included 30 participants with history of occupational noise exposure. Both the groups had average pure tone threshold ≤ 25 at octave frequencies from 500 Hz to 4000 Hz. For both the groups TEOEs were measure with and without contralateral BBN at 30dB SL.

And behaviourally SIS was measured with and without contralateral BBN 30dB SL both at ipsilateral +10 dB SNR and +15 dB SNR.

Results: Results indicated significant difference in SIS with and without contralateral BBN between groups. There was no significant difference in SIS with and without contralateral BBN both at ipsilateral +10 dB SNR and +15 dB SNR in Group1 but there was significant difference in Group 2. Also there was significant difference in the CSOAE between groups; Group1 showed greater suppression compared to Group 2. Further no significant ear effect was seen for both the measures. In the current study there was also no significant correlation between CSOAE and shift in SIS with contralateral BBN.

Conclusion: From this study it can be concluded that efferent auditory system is getting affected by occupational noise exposure and thus may lead to difficulty in understanding speech in the presence of noise. Further, CSOAE results indicate that it can be a reliable tool to assess the efferent system in individuals with occupational noise exposure, and thus aid in early detection of problems listening in background noise.

CHAPTER 1

Introduction

The olivocochlear system was first reported by Grant Rasmussen (1946), and since then numerous studies have been carried out to understand how it exactly works. The higher organs control the peripheral receptor – the cochlea by means of efferent feedback pathway (Huffman & Henson, 1990). The main segments of efferent pathway are cortex, rostral brainstem and olivocochlear bundle. The olivocochlear bundles are well studied and there are lateral and medial efferent fibers (Guinan, 1979; Guinan, Warr & Norris, 1983). The lateral olivocochlear bundles terminate on the afferent nerve fibers of inner hair cells. Whereas, the medial olivocochlear fibers are myelinated and terminate directly to outer hair cells. As the medial olivocochlear fibers are myelinated and thus the medial olivocochlear (MOC) fibers efferent physiology is well understood. This efferent system supports adaptation and frequency selectivity by modification of the micromechanical properties of outer hair cells (Ciuman, 2010). Consequently, the medial efferent system forms the basis for localization of a sound stimulus and thus enabling to function in a three-dimensional auditory world. Further, the efferent system's distinctive functions include – protection from noise, mediation of selective attention and improvement in signal to noise ratio. Various neurotransmitters are involved in the subtle mechanisms of fine regulation of the efferent system ensuring above mentioned functions (Ciuman, 2010).

The medial efferent system innervates the inner ear contralateral and ipsilateral, whereas the lateral efferent system projects mainly ipsilateral. The fibers of the lateral efferent system mainly correspond to the inner spiral bundle and innervate the dendrites of radial afferent fibers under inner hair cells, whereas the fibers of the medial efferent system continue to run in the tunnel spiral bundle, and to a less extent at the floor of the tunnel of

Corti as outer spiral fibers together with type2 spiral ganglion cell peripheral processes. The medial efferent fibers directly innervate the outer hair cells to a lesser extent, they also form synapses on afferent and efferent fibers. Lateral system of efferent olivocochlear bundles are uncrossed and innervate inner hair cells whereas Medial system are crossed and innervates Outer hair cells (Warr and Guinan, 1979).

The electrical stimulation of the crossed olivocochlear bundle leads to the suppression of auditory nerve response (Action potential) (Galambos, 1956; Warr & Guinan, 1979). Later Kemp (1978) discovered Otoacoustic emission and through this, the efferent cochlear system became accessible for investigation in humans. The medial olivocochlear bundle plays an inhibitory role on the activity of outer hair cells. Its stimulation reduces auditory nerve response, basilar membrane motility and OAEs amplitude. Due to presence of the crossed olivocochlear bundle, an ipsilateral stimulation of efferent fibers results in both ipsilateral and contralateral response. Collet and Kemp, Veuillet, Duclaux, Moulin & Morgon (1990) observed that otoacoustic emissions (OAE) in humans can be suppressed by contralateral white noise and OAEs suppression after contralateral auditory stimulation seems to be the only objective and none invasive method for evaluation of the functional integrity of the medial efferent system and of the structures lying on its course.

The MOC bundle attenuates the response of the cochlea to sound by reducing the gain of the outer hair cell mechanical response to stimulation. The MOC system probably functions in a protective role by acting to reduce receptor damage during intense acoustic exposure. In natural environments the system could function as a mechanism for "unmasking" biologically significant acoustic stimuli by reducing the response of the cochlea to simultaneous low-level noise (Kirk & Smith, 2003). The contralateral suppression of

OAEs are absent or reduced in cases with auditory dyschrony (Starr, Picton, Sininger & Hood & Berlin, 1996), retrochchlear pathology (Prasher, Rayn & Luxon, 1994) and also in auditory processing disorder cases (Muchink et.al., 2004). Also improvement in speech identification scores in noisy environment was reported when efferent system was activated by contralateral noise (Kumar & Vanaja, 2004). Further these results had positive correlation with contralateral suppression of OAEs. These studies show that behaviourally and by using OAEs the contralateral mechanism can be studied non-invasively in humans.

Occupational noise exposure can be classified as traumatic noise i.e., > 105 dBA which causes permanent threshold shift (Eggermont, 2012), threatening noise i.e., > 80 dBA which causes temporary threshold shift with damage to inner hair cells ribbon synapse (Kujawa & Liberman, 2009) and safe noise level of < 80 dBA. Eggermont (2017) reported even exposure to moderate level sounds of < 80 dBA for longer duration causes changes in central gain, increased spontaneous firing rate, reduced neural synchrony and reorganization of cortical tonotopic map. Schaette and McAlpine (2011) reported in animal studies that exposure of 8 hours of 85 dBA noise led to loss at high frequencies, low spontaneous firing rates and auditory nerve firing rates did not saturate by background noise. With these findings they hypothesised that these changes may affect speech perception in noise.

Several animal experiments (Kujawa & Liberman, 2009; Liu et.al., 2012; Shi et.al., 2013) reported that even for months of continuous noise exposure, there is no apparent effect on behavioural or ABR thresholds, cochlear potentials or hair cell morphology. Norena et.al., (2006) reported that when a cat was exposed for 76 dB(A) noise for almost 4 months for 24 hrs per day, the results showed no changes in ABR thresholds but the central auditory responses were affected. However, it is not clear what effect long exposure times can result on the central auditory system in individuals with normal audiometric thresholds.

The behavioural and objective tests of efferent system have been reliably recorded and are non-invasive. Further, the noise exposure shows central auditory changes even with normal audiometric thresholds. Thus, warrants studying the effect of different duration of occupational noise exposure on the efferent auditory system of humans.

1.1 Need for the study

Noise exposure can lead to permanent hearing loss or even changes of the cellular properties within the central auditory pathway (Groschel, Ryll, Gotze, Ernst & Basta, 2014). It is discussed if the observed effects are related to changes of peripheral input or due to changes in central auditory system.

Kumar, Ameenudin, and Sangamanatha, (2012) studied temporal and speech processing skills in normal hearing individuals exposed to occupational noise. Gap detection test, modulation detection test, duration pattern tests were carried out to evaluate temporal processing and speech recognition in presence of multitalker babble at 5dB SNR. The results showed individuals exposed to occupational noise had poorer temporal processing and speech recognition. They suggested that noise can cause significant distortions in the processing of suprathreshold temporal cue which may add to difficulties in hearing in adverse listening condition.

Kowalska and Kotylo (2002) reported decreased suppression of OAEs in individuals with occupational exposure to noise. They used white noise as contralateral stimuli at 40 and 70dBSPL and they found that TEOAE and DPOAE had poorer suppression in individual exposed to noise at higher frequencies.

The contralateral suppression of OAEs are absent or reduced in cases with auditory dyssynchrony (Starr, Picton, Sininger & Hood & Berlin, 1996), retrochchlear pathology

(Prasher, Rayn & Luxon, 1994) and also in auditory processing disorder cases (Muchink et.al., 2004). Also improvements in speech identification scores in noisy environment were reported when efferent system was activated by contralateral noise (Kumar & Vanaja, 2004). Further these results had positive correlation with contralateral suppression of OAEs. These indicate that behavioural and objective tests for studying efferent auditory system are reliable and non-invasive.

Also as several studies indicate that with months of continuous noise exposure there was no changes in behavioural and objective thresholds, whereas they reported central auditory changes (Kujawa & Liberman, 2009; Liu et.al., 2012; Shi et.al., 2013; Norena et.al., 2006). Kujala et.al., in 2004 suggested long term exposure to noise in clinically normal hearing subjects had persistent effect on central auditory processing and led to behavioural deficits. Thus using behavioural and objective tests of efferent system in individuals with years of exposure to occupational noise may provide insight into the neurophysiological alteration due to exposure to noise. Thus, helping to better understand the central mechanism in noise-induced hearing problems. Also by controlling the degree of peripheral hearing level, the changes in the central auditory system could be attributed to the effect of noise.

1.2 Aim:

To study the effect of occupational noise exposure on efferent auditory system.

1.3 Objectives of the study:

1. Effect of occupational noise exposure on speech identification scores with and without contralateral stimulus.
2. Effect of occupational noise exposure on objective test of efferent auditory system (contralateral suppression of OAE).

3. Comparison of speech identification scores with contralateral stimulus and suppression of TOAE in individuals exposed to occupational noise.

CHAPTER 2

Review of literature

This chapter reviews literature on role of efferent auditory system and how noise exposure affects efferent auditory system which in turn affects contralateral suppression of OAEs and the Identification of speech in noise under the following headings:

2.1. Anatomy of efferent auditory system

2.2. Investigation of Medial olivary cochlear bundles (MOCB) functions

2.3. Functional role of MOCB

2.4. Role of MOCB in the identification of speech in noise

2.5. Effect of noise exposure on Auditory system

2.6. Effect of noise exposure on MOCB

2.1. Anatomy of efferent auditory system

Rasmussen (cited in Maison et al., 1999) gave the first description of group of nerve fibers coming superior olivary complex (SOC), crossing the midline at the level of fourth ventricle and making synapse into the cochlea. In 1960 he supplemented his description by reporting another group of nerve fibers which are reaching cochlea without crossing the midline. War and co- worker (cited in Maison et al., 1999) who distinguished two types of olivocochlear fibers according to cell body location, proposed a new classification. First type corresponds to lateral efferent fibers, the cell bodies of which are situated in the, lateral superior olivary nucleus. These unmylinated fibers make synapse with radial afferent fiber dendrites (Liberman, 1980, cited in Maison et al., 1999), mainly on the ipsilateral side. The second type consists of medial olivocochlear fibers (MOC), the cell bodies of which are located around the preolivary nuclei of the SOC. The projections of these myelinated fibers

are mainly contralateral and make direct synaptic contact with basolateral membrane of the cochlear outer hair cells (Liberman and Brown, 1986, cited in Maison et al., 1999). These projections are tonotopically organized, with density of innervation decreasing from baseto apex (Brown, 1989, cited in Maison et al., 1999)

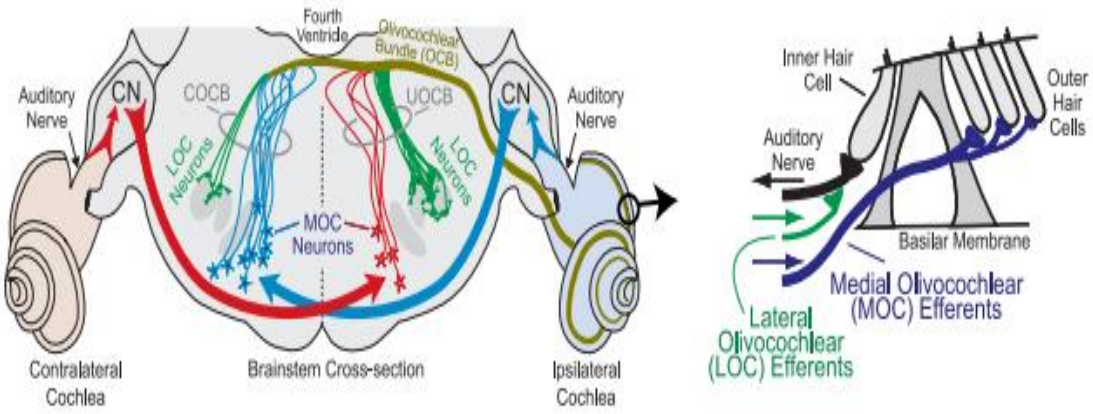

Figure 2.1: Schematized anatomic view of the olivocochlear reflex to the right cochlea.

LEFT: An outline of a transverse section of a cat's brainstem showing the location of lateral olivocochlear (LOC, green) and medial olivocochlear (MOC, blue or red) neurons. The pathways for the ipsilateral and contralateral MOC reflex to the right ear are shown in blue and red, respectively.

RIGHT: Schematic of the organ of Corti showing the main terminations of MOC on the outer hair cells and of LOC neurons on the dendrites of auditory nerve fibers.

This neurons within the medial superior olivary complex or trapezoid body, sends axons either to the contralateral (70%) or the ipsilateral (30%) cochlea and synapse with the basal pole of outer hair cells (Pujol, 1994). MOC fibers can either be seen as the feedback branch of cochlea- cochlear loop or as the component of an inter- cochlear link (Maison et al., 1999). This can be explained with the fact that there is an existence of direct projections from the cochlear nucleus (CN) into the SOC (Robertson and Winter, 1988. cited in Maison et al.,

1999), because these projections are crossed, MOC fibers having their cell bodies on one side are excited by acoustic stimulation presented to the other side. Also, a majority of MOC fibers project to the contralateral cochlea. As a result, MOC fibers mainly project onto the cochlea from which they indirectly drive their inputs (Liberman, Dodds and Pierce, 1990 cited in Maison et al., 1999) or otherwise stated MOC fibers forms a clean “feedback” loop (Maison et al., 1999).

In conclusion, results from various studies have now established that efferent olivocochlear system is divided anatomically, into lateral and medial efferent fibers. Also, the MOC fibers form a feedback branch of a cochlea-cochlear loop.

2.2. Investigation of MOCB functions

Since OHCs receive rich medial efferent innervations and OAEs are a normal by product of cochlear amplifier activity and reflect OHC integrity, they provide appropriate index of changes in cochlear function as MOC fibers are activated (Abdala, Ma and Sinniger, 1999). The OAEs are recorded in one ear in the presence and in the absence of a contralateral acoustic stimulation. Three types of OAEs have been used viz., spontaneous OAEs (Mott, Norton, Neely and Warr, 1989, cited in Collet et al., 1994) transient evoked OAEs with linear clicks (Collet, 1993), non-linear clicks (Collet et al., 1994), tone pips (Berlin et al., 1993, cited in collet et al., 1994) and acoustic distortion product OAEs (Abdala et al., 1999). The contralateral auditory stimulation can be a pure tone (Berlin et al., 1993, cited in collet et al., 1994), click (Veuillet, collet and Duclaux, 1991, cited in Collet, 1994); narrow band noise (Veuillet et al., 1991, cited in Collet et al., 1994) or broad band noise (Veuillet et al., 1991, cited in Collet et al., 1994).

Studies have also shown that for TEOAEs and DPOAEs the suppression effect is greatest when the level of the ipsilateral stimulation is lowest (Veuillet et al., 1991, cited in

Collet et al., 1994). Hood et al., (1996), cited in Parthasrathy, (2001) also showed that when the ipsilateral click stimulus level was kept at or below the suppressor noise level; the suppression effect was significantly greater. Veuillet et al (1991), cited in Collet et al., (1994) also showed that TEPAEs have greater suppression when the ipsilateral stimulus level is low, suggesting that MOCB function best at low ipsilateral stimulation level. Several other investigations (Veuillet et al., 1991, cited in Collet et al., 1994) have shown that this suppression effect is not related to artifacts caused by middle ear muscle contraction or crossover from the contralateral stimulus ear. This is supported by the fact that suppression is present in subjects without middle ear acoustic reflexes but is absent in subjects who have undergone a vestibular neurectomy (Williams, Brookes and Prasher, 1993)

It has been reported in literature that broad band noise is the most effective stimulus for the contralateral suppression (Collet et al., 1990, cited in Maison et al., 1999). Norman nd Thornton (1993) investigated the influence of stimulus bandwidth on contralateral EOAE attenuation. Their results revealed that the contralateral EOAE suppression affect increased with the contralateral stimulus bandwidth. A study by Maison, Micheyl and Collet (1999) Suggested a greater effectiveness of increase in bandwidth on the upper than on the lower side of center frequency of the noise. Maison, Micheyl and Collet (1999) explained this observation of increased MOCB activation with increased stimulation bandwidth by the spatial integration properties of certain neurons in the cochlear nucleus (CN). Onset units have large tuning curves, with occasionall inhibitory lateral bands in their response maps, these units are able to carry our spatial integration of several auditory nerve fibers responses of different best frequencies (Maison et al., 1999), thus, simple models of MOCB activation mechanisms including peripheral band pass filtering, within-channel compression and across channel summation by the afferent paths may account for the fact that MOCB activation increaes with stimulus bandwidth, whether or not the overall energy is kept constant (Maison

et al., 1999). Veuillet, Collet and Margon (1992), cited in Collet (1993) have shown white noise contralateral stimuli to be less effective at EOAE frequencies around 4 kHz suggesting a more fragile cochlear area. At higher and lower frequencies, contralateral auditory stimulation reduces the other components.

Subjects age is another important variable in the measurement of OAEs and interpretation of efferent mediated suppression effect. Morlrt, Collet, Salle and Morgan, (1993) found that BBN presented contralaterally had no effect on TEOAE amplitude for a group of premature nenates ranging in conceptional age from 33 to 39 weeks. However, other investigators have observed contralateral suppression of TEOAEs in term born neonates and even in some premature subjects. (godforth, Hood, and Berlin, 1997, cited in Abdala et al., 1999), Abdala et al., (1999) reported that significant suppressive effect on DPOAE amplitude can be seen when broad band noise (BBN) is presented contralaterally. The magnitude and pattern of contralateral suppression in term-born neonates is comparable to that of adults suggesting that medial efferent effect on cochlear function is matured by 40 weeks gestation. However the data obtained on premature babies in the study by Abdala et al., (1999) suggest that earlier a baby was born, the more likely it is they will show non-adult like expressions of efferent function (i.e. contralateral enhancement of DPOAE amplitude instead of contralateral suppression).

2.3. Functional role of MOCB

The functional role of the auditory efferent is still a matter of debate. Since studies by Buno (1978) and Murata et al., (1980), cited in Collet, (1993), it has been agreed that acoustic stimulation of one cochlea can alter afferents nerve-fibers responses in the contralateral cochlea in both animals and humans, eventhough the functional role is not so clear, continued attempts to understand the functions of the efferent olivocochlear system by researches in

animal and humans have clearly identified interesting properties of medial efferent fibbers, many of which have clinical relevance. These interesting functions are

2.3.1. Protective function against acoustic stimulation

2.3.2. Modulation of auditory sensitivity

2.3.3. Frequency and intensity discrimination

2.3.4. Modulation of signal detection in noise

2.3.1. Protective function against acoustic stimulation

Previous studies have provided strong evidence that the efferent pathways to the mammalian cochlea can protect that cochlea from damage caused by loud sounds (Cody and Johnston 1989, cited in Sahley et al., 1997), this hypothesis is based on the experiment work of animals showing a diminution of the PTS in case of acoustical or electrical stimulation of the olivocochlear bundle during noise exposure and an increase of the PTS after section of OCB. However, Liberman, (1990), cited in Sahley et al., (1997) was not able to replicate the results. These investigations suggest that activation of the medial efferent serves a protective function the mammalian auditory periphery (Patuzzi and Thompson, 1991, cited in Sahley et al., 1997) demonstrated that the whole nerve action potential in guinea pigs following monaural acoustic overstimulation was significantly reduced, from 12.7dB to 5dB, when a frequency matched acoustic stimulus at a lower stimulus intensity is delivered to the contralateral ear. This frequency specific temporary threshold shift (TTS) suggested that the activation of contralateral medial efferent system reduce the susceptibility of the cochlea to the effects of acoustic trauma. However, other researches have pointed out that there are certain ambiguities to the mechanism underlying such effects (Liberman, 1992, cited in Sahley et al., 1997).

2.3.2. Modulation of auditory sensitivity

The activation of medial efferent neurons by the delivery of contralateral stimulus (BBN) has been shown, to result in discharge suppression within primary auditory neurons in animals (Wiederhold and King, 1970). Clinical investigations in human subjects have also demonstrated suppression of the auditory nerve compound action potential following the delivery of a contralateral auditory stimulus (Folson & Owsely, 1987, cited in Sahley et al., 1997). In view of the preferential innervation of OHCs by descending medial efferent fibers (Liberman et al., 1990, cited in Sahley et al., 1997), the prevailing view has been that stimulation of medial efferent alters IHC sensitivity indirectly by altering the mechanical properties of organ of corti. Subsequently auditory sensitivity is also changed (Brownell, 1990, cited in Sahley et al., 1997). Based on this evidence, it was proposed that medial efferent system regulates the length, tension and stiffness of OHCs along their longitudinal axis, providing a gain control for the active, non-linear biometrics of the cochlear partition (Kim, 984, Cited in Sahley et al, 1997) for low intensity auditory (i.e., 45dB to 55dBSPL or 30 to 40 dB above threshold)

2.3.3. Frequency and intensity discrimination

There is some evidence to suggest that medial effect fibers transaction may impair the frequency resolving capacity of the auditory system (Capps and Ades, 1968, cited in Sahley et al., 1997). Focussed ultrasonic lesions of the medial efferent fibers in monkeys resulted in an increase in the frequency difference (threshold) needed to maintain a 75% level of correct discrimination performance. These results suggested that efferent transection produces marked deficits in frequency discrimination performance.

Igarshi and associates (1979) reported that transaction of the midline efferent olivocochlear bundle in the cats fails to produce changes in the suprathreshold (75dBSPL)

intensity discrimination limen. The interpretation of these remains equivocal because the animals had bilaterally intact cochlea and were tested in a sound field.

2.3.4. Modulation of signal detection in noise

It has been reported that OCB is involved in the detection of signal (tone or speech) in noise in animals and humans (Igarashi, Alford, Nakai and Gordon, 1972., Micheyl and Collet., 1996, Girand et al., 1997, Zeng, Lehmann, Soni and Linthicum, 1994). These findings indicate that inhibitory function of efferent system could lead to an improvement in coding of signals embedded in noise (Libermann, 1988, cited in Sahley et al., 1997). This also suggests that efferent system aid in masking.

Micheyl and Collet (1996) found that greater the contralateral EOAE attenuation effect, the better the detection performance of signal in presence of noise, such an observation raises the question as to how a system that inhibits the auditory periphery (reduction in compound action potential of the auditory nerve and auditory afferent fiber discharge, (Wiederhold, and king 1970)) can finally enhance detection performance. Neurophysiological studies on the influence of OCB stimulation on auditory-nerve (AN) fibers have suggested a positive involvement of the OCB in perception in noise compatible with its inhibitory function on AN fibers. The OCB induced change in AN activity that could explain enhanced detection in noise with OCB stimulation is the antimasking effect which has been demonstrated for both shock- evoked and sound evoked OCB activity (Kawase, Delgutte and Libermann, 1993, cited in Micheyl and Collet., 1996).

2.4. Role of MOCB in the identification of speech in noise

Libermann and Guinan (1998) have described how anti-masking effects of the middle ear muscles (MEM) and olivocochlear efferent neurons affects feedback control of the

auditory periphery. According to them the anti- masking properties of the middle ear muscle and medial olivary complex systems are based on different mechanisms and complement each other in the sense that middle ear muscle system helps to control masking from low frequency noise while the MOC system helps with medium and high frequency noise. Addition of noise can raise the thresholds ANFs in two fundamentally different ways. These two mechanisms have been called “excitatory masking” and “Suppressive masking” (Libermann and Guinan, 1998). Neurophysiological studies have shown that the MEM reflex can decrease the masking of high frequency signals by low frequency noise (i.e., the upward spread of masking) also known as suppressive masking. The MOC reflex is believed to minimize masking if high frequency transient signals by high frequency conditions noise, also known as excitatory masking.

Excitatory masking (Figure 2.2) is illustrated by the effect of the high frequency noise “masker” on the response of the high center frequecy fiber to a “signal”. In the absence of noise, the signal is within the fiber’s response area (dashed tuning curve). Thus in the absence of noise, the fibers responds to the signal by increasing its discharge rate, as schematized by the train of action potentials in the “noise off” column. However, the noise bands also contains energy at frequencies and levels to which fiber responds, as illustrated by overlap between the noise spectrum and the dashed tuning curves. Thus, while noise is on, the fiber responds vigorously for the duration as shown by the long spike train in the “noise on” column. This noise driven excitation raises the fiber’s threshold to tones so that signals no longer elicits a response when the noise is on. This “excitatory masking” occurs for two reasons. First, the excitation of the fiber by the steady noise is like increasing is background discharge rate. Thus, for a tone signal to cause a “response” it must elicit an additional increase in rate, and its level must be higher than normal. This has been called the “line busy” effect. The second reason for excitatory masking is that ANFs become fatigued by continuous

stimulation by noise and when fatigued, they are less responsive to an additional signal such as the tone burst.

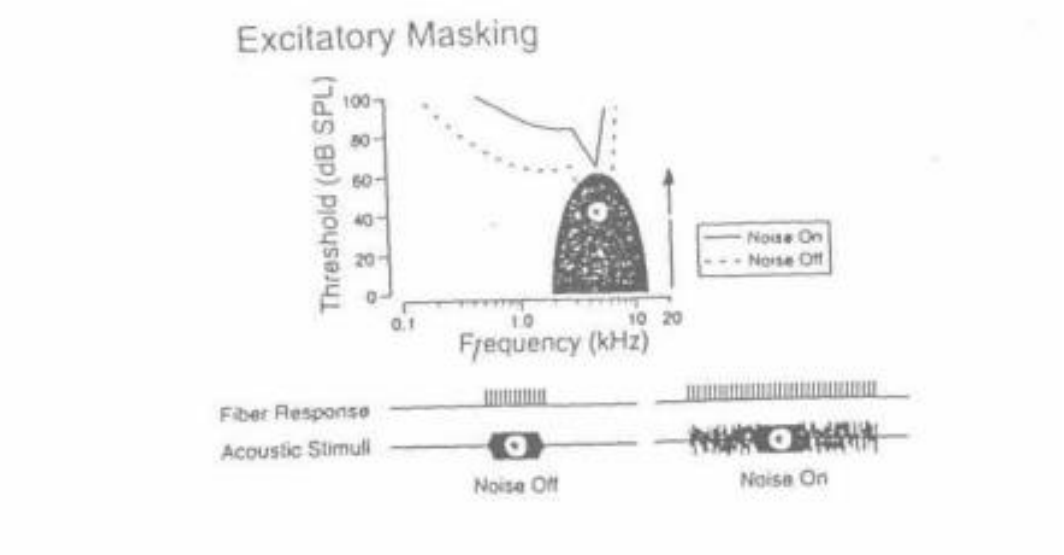

Figure 2.2: shows the tuning curve of the fiber in quiet and excitatory masking, (adapted from Libermann and Guinan, 1998)

The mechanism the response to transient stimuli such as m underlying this fatigue or adaptation as it is also called probably involves depletion of chemical neurotransmitter from the synapse between IHC and ANF. This transmitter that can only be synthesized and packaged at a limited rate, on continuous stimuli such as masking noise decreases the response to transient stimuli such as tone bursts.in this situation, even though the signal is within the masked response area the increment in rate, which it elicits, is very small. Such as small rate change will be difficult for the central nervous system to detect, and small differences in the sound level of the signal will also be difficult to detect.

Stimulation of MOCs decreases the steady response to the noise, thereby increasing the response to the signal transient because the degree of adaptation is reduced. This type of antimasking is illustrated in Figure 2.3. The resulting increment in response to the signal will

be easier to detect and the ability to discriminate suprathreshold stimuli in noise will be improved. However, masked thresholds may not be improved.

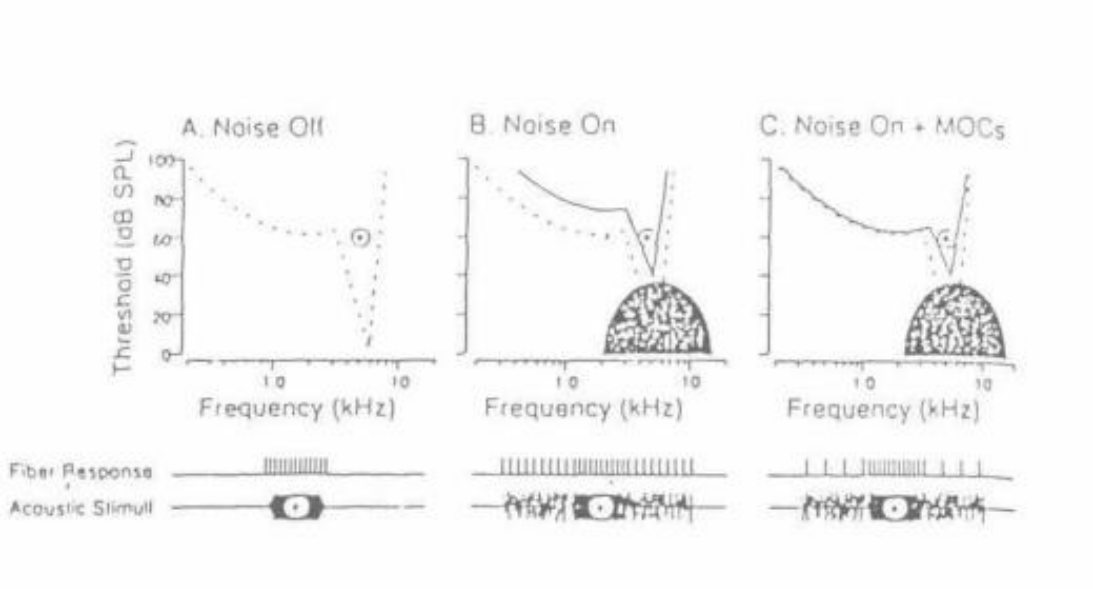

Figure 2.3: Shows the anti-masking effects of the MOC reflex, (adapted from Libermann and Guinan, 1998)

This is important to note that the MOC system does not suppress the noise more effectively than the signal because the noise is broad band whereas the signal is narrow band. Rather, the important difference is that noise is continuous and speech is transient.

Micheyl and Collet (996) investigated the involvement of auditory efferent in hearing in noise in humans. Olivocochlear bundle function was assessed in terms of contralateral attenuation is evoked OAE i.e., the reduction in EOAE amplitude elicited by 30dBSL contralateral BBN. The detection thresholds for 1 and 2 kHz tone pips embedded in 50dbSPL BBN were measured. EOAEs were measured in the same ear with and without contralateral BBN of 30dbSPl. The results indicated that the contralateral attenuation of EOAEs correlated significantly with the detection threshold for 2 kHz tone pips embedded in noise. It also correlated with the shift in threshold at 1 kHz and 2 kHz induced by contralateral acoustic

stimulation. This suggest that the OCB is involved in the detection of tones in noise only when noise is present in the contralateral ear or when background noise is binaural.

Zeng et al (1994) studied the effect of vestibular neurectomy on pure tone intensity discrimination and speech perception in noise in six subjects, by comparing performance in the surgery ear and the non-surgery ear when available between the pre and post-operative conditions. It is assumed that MOCB are severed during vestibular neurectomy. Five of the six subjects had normal or near normal pure tone average thresholds (<30dBHL). BBN was used for intensity discrimination and speech spectra shaped noise was used in speech reception threshold measurement. Both types of noise were presented binaurally at several different levels, whereas tone or speech was varied adaptively based on patient's response. Preliminary results showed that loudness dynamic range is not affected by surgery and also intensity and speech perception in noise was significantly worsened after the surgery in some subjects but not others. Thus, Zeng et al., (1994) concluded that in cases where MOCB was severed the perception of speech in noise became poor.

Girand et al., (1997) investigated speech perception in noise in vestibular neurectomized patients and in normal. In normal, contralateral noise improved speech intelligibility in noise and this was correlated with magnitude of contralateral suppression of OAE. This improvement was absent in de-efferented ears of vestibular neurectomized patients. Conclusion given was olivocochlear efferents play an anti-masking role in speech perception in noisy environment.

The contralateral suppression of OAEs are absent or reduced in cases with auditory dyssynchrony (Starr, Picton, Sininger & Hood & Berlin, 1996), retrochchlear pathology (Prasher, Rayn & Luxon, 1994) and also in auditory processing disorder cases (Muchink et al., 2004). Also improvement in speech identification scores in noisy environment was

reported when efferent system was activated by contralateral noise (Kumar & Vanaja, 2004). Further these results had positive correlation with contralateral suppression of OAEs. These indicate that behavioural and objective tests for studying efferent auditory system are reliable and non-invasive.

A study by Kumar (2001) reinforces this hypothesis that the efferent system augments speech perception in noise. Results showed that contralateral noise significantly improved the speech identification scores (SIS) at +10dB and +15dB signal to noise ratio, but not in children with learning disorder. More shift in SIS scores was seen at +10dB and +15dB in normal children, and this shift showed positive correlation with the physiological measures of OCB (CSOAE). Subjects with learning disorder showed absent CSOAEs and here was no improvement in the SIS scores in the presence of contralateral noise. An investigation by Veuillet et al (1999) also reported significant reduction in MOC functioning in learning disorder children.

2.5. Effect of noise exposure on auditory system

2.5.1. Cellular changes in auditory system by noise exposure

Exposure to intense sound or noise can result in purely temporary shift (TTS), or leave a residual permanent threshold shift (PTS) along with alterations in growth functions of auditory nerve output (Kurabi et al., 2016). The principle cause of NIHL is damage to cochlear hair cells and associated synaptopathy (Kurabi et al., 2016).

- Mechanism of TTS

Temporary loss of hearing sensitivity is often viewed as a less severe form of the same changes that lead to permanent cochlear damage. However, recent evidence suggests that TTS may be mediated by distinct mechanisms. Housley et al., (2013) found that low

level TTS is mediated by ion channels that are activated by extracellular ATP, since mice deficient in a specific channel (P2RX2) do not experience TTS after noise exposure that normally causes about 15dB of temporary sensitivity loss. However more extensive, TTS (up to 50dB) can also recover to normal threshold level over time (Ryan and Bone, 1978), these higher levels of TTS are due to additional mechanisms Nordmann et al., (2000) noted that uncoupling of the outer hair cells (OHC) stereocilia from the tectorial membrane was the primary morphological feature associated with 43dB of TTS in animals. Other investigations have noted swelling of the afferent endings underneath the inner hair cells (IHC) after noise exposure, suggestive of excitotoxicity due to the release of excessive glutamate from overstimulated HCs (Puel et al., 1998). Supporting this mechanism, Puel et al. (1998) found that pre-treatment with the glutamate antagonist kynurenate not only prevented this swelling, but also reduced the amount of TTS. This findings suggests that reversible excitotoxicity to cochlear afferent neurons can also contribute to TTS.

- Mechanism of PTS

When sufficient noise exposure is present the ability of the cochlea to recover is overwhelmed, and hearing loss becomes irreversible. Such permanent changes in auditory thresholds have primarily been linked to cochlea HC damage and loss, although damage to neurons and the lateral wall can also mediate long-term loss of hearing (Schuknecht, 1993). Sufficiently intense overstimulation of the cochlea, as can occur with blast exposure, will produce mechanical damage to the cochlea. This damage includes direct mechanical disruption of HC stereociliary arrays (Liberman and Beil, 1979; Slepecky, 1986; Patuzzi et al., 1989), which can reduce or even eliminate function. The majority of NIHL reflects HC damage mediated by biochemical processes that occur within the cells themselves (Kurabi et al., 2016).

2.5.2. Responses of auditory neurons after noise exposure

Early noise exposure may produce alterations of neuronal responses, in particular, in frequency tuning and tonotopy (Sanes and Constantine-Paton, 1983, 1985). Different types of exposure may lead to very different results, depending on numerous characteristics of noise. Exposure to tonal pulses may lead to broadening of frequency tuning in the auditory cortex (Zhang et al., 2001), but not in the inferior collicullus (Sanes and Constantine-Paton, 1985).Noise exposure of rat pups may have a permanent effect on the neuronal tuning curves in the rat inferior collicullus (Grecova et al., 2009), whereas in the auditory cortex,the tuning curves properties may recover after the animal is placed into normal housing conditions (Chang et al., 2005).

After early noise exposure, the bandwidth of the frequency tuning curves (FTC) usually increases, indicating worsened frequency selectivity. After exposure to tones, distorted tuning bandwidths were reported in the dorsal cochlear nucleus of rats (Zhang and Kaltenbach, 1998). In the IC, broadened tuning curves were reported after the exposure of mice to repetitive clicks (Sanes and Constantine-Paton, 983, 1985) the changes were more prominent between 0 and 5 kHz.

2.5.3. Effect of noise exposure on auditory thresholds

Jin et al. (2013) did study on marching band individuals to see the effect of noise exposure during band on auditory thresholds and reported that they showed high frequency notch (there was no notch at 3 kHz and 4 kHz but at 6 kHz there was notch) compared to control group (Collage students) which could be an early indicator of NIHL.

Loch et al. (1943) reported evidence of tonal dips of at least 15 dB at 4,096 cycles per second in an initial sample of 1,365 school children age 8 to 14 years. In this early study, 15% of boys and 5% of girls evidenced a high frequency notched audiometric pattern.

Helfer et al. (2011) conducted study on army soldiers to see the effect of noise on hearing thresholds over time from 2003 to 2009 since they are exposed high noise during explosion and reported that soldiers showed significant threshold shift (STS) as the year of noise exposure increased from 2003 to 2009.

- Temporary threshold shift (TTS)

Furman et al. (2013) used 4 and 8 kHz noise, at 106 dBSPL for 2 h and found that after 2 weeks the ABR thresholds recovered to normal as did frequency tuning, dynamic range, first spike latency in guinea pigs but suprathreshold ABR amplitudes were reduced. Liu et al. (2012) and Shi et al. (2013) used broad band noise presented at 105 – 110 dB SPL for 2 h in Guinan pigs. They found that the ABR thresholds and the initial loss of synaptic ribbons largely recovered within 14 days to a month after the noise exposure.

2.5.5. Effect of noise exposure on OAE

In case of permanent damage to hair cells or damage to the mechano-sensory function, the more widely used diagnostic tests include pure-tone audiometry and OAEs which would reveal an increment in the thresholds and a decrement in the amplitude of OAEs in those damaged frequency regions. However, OAEs when compared to pure-tone audiometry has a better sensitivity in identifying the damage to the auditory structures (Attias, Horovitz, El-Hatib, & Nageris, 2001). It is identified that the Otoacoustic emissions are evoked by the OHCs within the cochlea, and this is the first site to be affected by noise exposure (Furst et al., 1992). In case of DPOAEs, wherein two pure-tone stimuli are presented, a notch at 3000

Hz is seen, resembling the configuration of hearing loss which is usually present as a notch at 4000-6000 Hz range (Attias et al., 1996). In a study by Attias et al., there was a clear relationship between the OAEs and the thresholds that were obtained behaviourally. There was narrowing of the emission range and also a decrease in the amplitude of OAEs as the severity of the damage increased due to noise exposure. However, in few of the subjects with noise exposure, the OAEs were still present along with normal thresholds behaviourally (Attias et al., 1996).

Later, morphological studies on animals revealed that there is a swelling of auditory nerve fiber (ANF) terminals at the site of connection with the hair cells i.e. the synaptic junction after acoustic overexposure (Liberman, 1982; Robertson, 1983; Spoendlin, 1971). And, this swelling was seen only at the synaptic connection area of the inner hair cells (IHCs), and not at the OHC area (Pujol et al., 1999). This is also supported by many other studies revealing normal OHC functioning despite acoustic exposure. In one of the studies did on human subjects, they assessed the changes seen due to acoustic exposure in different tests including inter-aural time difference (ITD), click-evoked otoacoustic emissions (CEOAEs), and ABR (analysis of wave I and wave V in the presence of masker noise). In normal hearing individuals without noise exposure the shift in wave V latency with increase in masker level was more compared to individuals with noise exposure and normal hearing. And also the performance in sound localization task which required discrimination of ITDs in envelops of sound was better in without noise exposure group than with noise exposure group. Hence, it was seen that there could be damage at the synaptic level and not at the OHC region which was supported by the results obtained indicating a significant difference for the ITD and ABR measures and not for the CEOAEs (Mehraei et al., 2015).

2.5.6. Effect of noise exposure on ABR

Recent work on animals shows that overexposure to acoustic stimulation causing only transient threshold elevation, without any hair cell damage, nevertheless can cause irreversible loss of the synapses between inner hair cells and cochlear nerve fibers (Kujawa and Liberman 2009). Furman and his colleagues carried out an experiment on guinea pigs exposed to noise in 4 kHz-8 kHz octave bands at 106 dB SPL for 2 hours, wherein they recorded potentials from single auditory nerve fibers. They found that 2 weeks post-exposure the ABR thresholds as well as the amplitude of DPOAEs recovered to normal, suggesting that there was recovery in the hair cell functioning. However, the suprathreshold ABR amplitudes had reduced and a loss of 30% of synapses between the ANFs and inner hair cells were confirmed by Immunostaining pre and post synaptic markers of sensory epithelium. They concluded saying, this condition (cochlear synaptopathy) is selective for the subset of auditory fibers with high thresholds and low spontaneous rates (Furman et al. 2013). Evidence also shows that the difficulty in hearing in everyday setting and in understanding speech in noise with normal hearing could be due to the differences in the fidelity with which supra-threshold sound is coded in the auditory pathway (Bharadwaj et al., 2015). Cochlear synaptopathy due to noise-exposure has been studied extensively in animals wherein, there is a reduction in the amplitude of ABR wave I at the supra-threshold levels and not significant at the threshold level (Kujawa & Liberman, 2009; Lin et al., 2011; Hickox et al., 2015). However, there are very few studies to see whether the same results holds good for humans as well. Prendergast and his colleagues did a study on young human adults with a wide range of noise-exposure and normal hearing when tested through audiometry. ABR was done for high-pass filtered clicks (> 1.5 kHz) at 80 and 100 dB peSPL. The bandwidth chosen was 3-6 kHz for the ABR stimuli and the carrier frequency of transposed tones as this frequency region is commonly associated with damage due to noise-exposure in humans. They found

that there was no relation between the noise-exposure and the amplitude of ABR waves, especially wave I, which was seen in animals when exposed to noise.

2.5.7. Effect of noise exposure on central auditory system

Kujala et al. (2004) had earlier reported that long term exposure to noise in clinically normal hearing subjects had a persistent effect on central auditory processing and lead to concurrent behavioural deficits. They studied a group of 10 noise exposed participants including 8 shipyard workers. The exposure to noise in the noise exposed participants had to be at least 2 years in duration. In shipyards, the noise level during working days was around 95- 100 dBA, which brings it solidly in the TTs range. The 10 control participants had been working in silent or moderate noise level conditions. Both groups were age and sex matched. The participant's audiograms were measured before the experiment for sound frequencies from 12 Hz to 8 kHz. These audiograms indicated no significant group effects. However Kujala et al. (2004) found that phoneme discrimination was impaired in noise exposed individuals as indicated by behavioural responses and the mismatch negativity (MMN) brain response. Thus long term exposure to noise has lasting detrimental effects on central temporal auditory processing.

Brattico et al. (2005) replicated and extended the findings of Kujala et al. (2004), and found that the central auditory discrimination impairment in the noise exposed participants was specific to speech processing, and involves not only discrimination of fine acoustic differences present in stop consonants, but also larger physical speech sound differences present in vowels. Brattico et al. (2005) also found that small contrasts between speech and non-speech sound elicited longer latency MMNs in the noise exposed than control group.

Kumar et al. (2012) also explored temporal processing and speech perception skills in individuals who were exposed to occupational noise of more than 80 dB(A) and still had

clinically normal hearing (< 25 dB HL from 250 Hz to 8 kHz). They found a trend of reduced temporal modulation and gap detection in individuals with noise exposure. Speech recognition scores in the presence of noise were also significantly poor in the noise exposed individuals.

2.6. Effect of noise exposure on MOCB

Noise exposure can lead to permanent hearing loss or even changes of the cellular properties within the central auditory pathway (Groschel, Ryll, Gotze, Ernst & Basta, 2014). It is discussed if the observed effects are related to changes of peripheral input or due to changes in central auditory system.

Otoacoustic emissions are evoked by the OHCs within the cochlea, and this is the first site to be affected by noise exposure (Furst et al., 1992). In case of DPOAEs, wherein two pure-tone stimuli are presented, a notch at 3000 Hz is seen, resembling the configuration of hearing loss which is usually present as a notch at 4000-6000 Hz range (Attias et al., 1996). In a study by Attias et al., there was a clear relationship between the OAEs and the thresholds that were obtained behaviourally. There was narrowing of the emission range and also a decrease in the amplitude of OAEs as the severity of the damage increased due to noise exposure.

Kumar, Ameenudin, and Sangamanatha, (2012) studied temporal and speech processing skills in normal hearing individuals exposed to occupational noise. Gap detection test, modulation detection test, duration pattern tests were carried out to evaluate temporal processing and speech recognition in presence of multitalker babble at 5dB SNR. The results showed individuals exposed to occupational noise had poorer temporal processing and speech recognition. They suggested that noise can cause significant distortions in the processing of

suprathreshold temporal cue which may add to difficulties in hearing in adverse listening condition.

Kotylo, (2002) reported decreased suppression of OAEs in individuals with occupational exposure to noise. They used white noise as contralateral stimuli at 40 and 70dBSPL and they found that TEOAE had poorer suppression in individual exposed to noise. Similar results were obtained by Patica Arruda in 2013.

Prasher at al., 1998 did study to see the effect of noise exposure on efferent auditory system. They stimulated subjects with octave band noise for as long as 1hour (Auditory fatigue) and then measured contralateral suppression of TEOAEs. Results showed significant reduction in contralateral suppression of TEOAEs. They suggested that contralateral sound activated efferent suppression may provide an early indication of auditory damage after exposure to noise.

Kotylo et al., 2002 did study on occupational exposure to noise decreases otoacoustic emission efferent suppression. They used white noise as contralateral stimulation at 40 and 70 dBSPL (15 to 45dB higher than threshold). OAE efferent suppression in normal hearing subjects, occupationally exposed to noise, was compared with respective effects in healthy, non-exposed subjects. Results showed poor contralateral suppression of TEOAEs in individuals exposed to noise.

De souza Alcaras et al., 2013 did study on evoked otoacoustic emission and suppression effect on workers exposed to noise. There were two groups in the study one experimental (exposed to occupational noise) and control group (not exposed to occupational noise). Suppression effect in TEOAE and DPOAE were checked and result showed poorer suppression effect in individuals exposed to occupational noise. Suggesting that chronic

exposure to noise may have impaired the functioning of the medial olivocochlear efferent auditory system.

Muller et al., 2008 did study to investigate whether distortion product otoacoustic emissions (DPOAEs) are a suitable means for detecting small changes in cochlear amplifier functionality due to occupational noise exposure. They measured contralateral suppression of DPOAEs by using BBN as contralateral stimuli which was presented at 60 dBSPL. Results showed 1.6dB suppression on an average in individuals exposed to occupational noise and 1.9 in individuals not exposed to noise. Though there was less suppression in individuals exposed to noise since the difference was not significant they concluded that DPOAEs are not a suitable means for detecting small changes in cochlear amplifier functionality due to occupational noise exposure.

CHAPTER 3

Method

The present study was carried out to understand the effect of occupational noise exposure on the efferent auditory system. To evaluate these, behavioural and objective tests were carried out systematically across individuals with noise exposure.

3.1. Participants:

Group I: Thirty five normal hearing individuals age range of 25 to 45yrs who are working in silent or moderate noise level conditions were included.

Group II: Thirty normal hearing individuals age range of 25 to 45yrs who are exposed to occupational noise >5 to <7yrs were included.

Prior to subject selection it was ensured that the noise level at their work place was not > 105 dBA using an SLM. Individuals working in noise levels <105 dBA were not included and a detailed case history was obtained.

3.2. Subject selection criteria:

Individuals with no history of otological and neurological problems were included in the study. Also it was ensured that the individuals with recreational noise exposure, cardiovascular risk factors, smoking, and diabetes were not included in the study. Pure-tone thresholds were obtained at octave frequencies between 125 Hz to 8000 Hz for air-conduction and between 250 Hz to 4000 Hz for bone-conduction. The participants who had audiometric thresholds ≤ 25 dB HL for octave frequencies 125 to 4000 Hz were included in the study. Normal middle ear functioning was ensured based on tympanometry (middle ear pressure between +50 to -50 daPa; middle ear compliance between +0.3 to +1.6 ml (Jerger,

1970), with a probe tone frequency of 226 Hz, and acoustic reflex threshold obtained at 1000 Hz.

For Group I individuals to avoid possible temporary threshold shift effect after work, audiological tests was carried out at least after 10 hours between last shift at work and the tests.

3.3. Test environment:

All tests were carried out for each individual in an air-conditioned acoustically treated double- room setting. The noise levels in these rooms were within permissible limits (ANSI S 3.1 1991).

3.4. Instrumentation:

3.4.1. Instrumentation for Behavioural test

Calibrated dual channel clinical audiometer (MADSEN Astera) with TDH 39 ear phones was used for pure tone audiometry and speech audiometry. Broad band noise was fed through the insert receiver of the same audiometer, and was used as contralateral stimuli (CAS) to activate efferent system.

3.4.2. Instrumentation for Objective/Physiologic test

Calibrated Immittance meter (GSI Tympstar) was used for evaluating middle ear status. ILO V6 a clinical otoacoustic emission analysing software was used to measure otoacoustic emissions. . Broad band noise was fed through the insert receiver of the calibrated audiometer (MADSEN Astera), and was used as contralateral stimuli (CAS) to activate efferent system.

3.5. Material:

Speech identification test material developed by Yathiraj and Vijayalakshmi (2005) was used in the study. Speech material consists of 100 phonetically balanced words which were divided into 5 lists. Speech material was mixed with broad band noise (BBN) at +10dB and +15dB using MATLAB software.

3.6. Procedure:

The behavioural speech identification test with contralateral noise and objective test of contralateral suppression of OAE was carried out.

3.6.1. Behavioural test:

The tests were carried out in a two-room situation, which was acoustically treated and had adequate illumination. Speech material was presented through a CD connected to a two channel clinical audiometer. A calibration tone recorded at the beginning of each list was used to adjust the deflection of the VU meter to 0 while presenting the material. Verbal responses were obtained from the subjects.

Speech identification scores were obtained at 50 dB HL in the following conditions:

i) In quiet (no noise was given in the ipsilateral ear and the contralateral ear)

ii) In the presence of ipsilateral BBN with signal to noise ratio of +10, +15 dB and no noise in the contralateral ear. (Speech was kept constant at 50 dB HL, and noise was varied to achieve different signal to noise ratios)

iii) No noise in the ipsilateral ear and 30 dB SL BBN in the contralateral ear (i.e., threshold of noise)

iv) In the presence of ipsilateral BBN (signal to noise ratio of +10, +15 dB) and contralateral BBN at 30 dB SL (i.e., threshold of noise).

The order of the presentation of experimental conditions was randomized to eliminate the order effect. Every correct response was scored as 1 and the corresponding percentage was calculated. Based on the obtained results effect of contralateral stimuli on speech identification scores were analysed and difference between Right and Left ear was calculated by subtracting mean shift without contralateral acoustic stimuli and mean shift with contralateral acoustic stimuli.

3.6.2. Objective test:

Participants were instructed to be seated on a chair and to be quiet during testing procedure. Transient evoked otoacoustic emissions (TEOAEs) were recorded using a ILO V6, Otodynamics OAE equipment. The TEOAE were recorded for click stimulus at 70dBSPL. A probe with a foam tip was positioned in the external ear canal and adjusted to give a flat stimulus spectrum across the frequency range. Amplitudes of TEOAE were measured. This procedure was repeated in the presence of contralateral BBN at 30 dB SL (i.e., threshold noise) which is presented through the insert ear phone. Care was taken to ensure the position of the probe to be the same during both the recordings. Amount of suppression induced by contralateral acoustic stimuli was calculated by taking the difference of TEOAE amplitude with and without contralateral acoustic stimuli.

3.7. Statistical analysis

Shapiro-Wilks test for normality was administered to check whether the data follows normal distribution for both TEOAE and speech identification scores. It was found that both TEOAE and speech identification scores data did not follow normal distribution and hence, non- parametric tests were administered. The statistical tests administered are as follows.

1. Descriptive statistics was performed to examine the central tendency and variation of amplitude in TEOAE with and without contralateral BBN and speech identification scores with and without contralateral BBN.
2. Mann-Whitney U test was administered to compare the amount of suppression and variation in speech identification scores with and without contralateral stimulus between two groups.
3. Wilcoxon sign ranked test was performed to compare amplitude of TEOAEs with and without contralateral BBN and speech identification scores with and without contralateral BBN.
4. Spearman's rank correlation test was performed to check for the correlation between contralateral suppression of OAE and shift in speech identification scores in the presence of contralateral BBN.

CHAPTERS - 4

RESULTS

The aim of the study was to investigate effect of noise exposure on efferent auditory system using contralateral suppression of TEOAE and speech identification in the presence of contralateral noise. The measures used for analysis of TEOAE are amplitude of OAE at different frequencies 1 kHz, 1.5 kHz, 2 kHz, 3 kHz, 4 kHz with and without contralateral noise. Also speech identification scores were measured at different conditions by keeping speech level at 50dBHL 1- In quite, 2- in the presence of ipsi BBN at 10 dB SNR [SIS (I10)], 3- in the presence of ipsi BBN at 15 dB SNR [SIS (I15)], 4- in the presence of only contralateral BBN at 30dBSL, 5- in the presence of ipsi BBN at 10 dB SNR with contralateral BBN at 30dBSL[SIS (I10C)], 6- in the presence of ipsi BBN at 15 dB SNR with contralateral BBN at 30dBSL [SIS (I10C)]. The responses from these conditions were compared between two groups namely individuals without occupational noise exposure (Group 1) and individuals with occupational noise exposure (Group 2).

4.1. Comparison of speech identification scores (SIS) with and without contralateral noise.

Comparison of SIS with and without contralateral noise was done between groups, within group and between ears.

4.1.1. Comparison of SIS with and without contralateral noise between groups.

Descriptive statistics were carried out to find the mean, median, and standard deviation of SIS with and without contralateral noise with ipsi 10 dB SNR and 15 dB SNR between groups.

Table 4.1:

Mean, median and standard deviation (SD) of speech identification scores at different ipsilateral SNR with and without contralateral stimulus between groups.

	Group1			Group2		
	Mean	SD	Median	Mean	SD	Median
SIS(I10)	99.74	1.07	100	97.13	3.38	100
SIS(I10C)	99.60	1.34	100	90.87	6.42	92
SIS(I15)	99.80	0.97	100	97.73	2.79	100
SIS(I15C)	99.71	1.03	100	94.07	5.53	100

The descriptive statistics results of SIS indicate that there is decrease in SIS in Group2 in the presence of contralateral noise compare to only ipsilateral noise but there is no much change in SIS when compared between with and without contralateral noise in Group1. The same is depicted in Table 4.1.

Mann-Whitney U test was administered to compare SIS with and without contralateral stimulus between groups and the results are as shown in Table 4.2.

Table 4.2:

Test statistics (|Z|) and significance values for comparison of SIS with and without contralateral noise between groups.

	|Z|
SIS(I10)	5.59 *
SIS(I10C)	9.10 *
SIS(I15)	5.57 *
SIS(I15C)	7.81*

* p value < 0.05

The results showed significant difference between the groups as shown in Table 4.2

4.1.2. Comparison of SIS with and without contralateral noise within group.

For group 1 descriptive statistics were carried out to find the mean, median, and standard deviation of SIS with and without contralateral noise with ipsi 10 dB SNR and 15 dB SNR for group1. The same is depicted in Table 4.3.

Table 4.3:

Mean, median and standard deviation (SD) of speech identification scores at different ipsilateral SNR with and without contralateral stimulus within Group1.

Group1	SIS(I10)	SIS(I10C)	SIS(I15)	SIS(I15C)
Mean	99.74	99.60	99.80	99.71
SD	1.07	1.34	0.972	1.03
Median	100	100	100	100

Descriptive statistics results of SIS indicates that there is no much difference in the mean values of with and without contralateral noise conditions at both +10 dB SNR and +15 dB SNR.

Wilcoxon sign ranked test was administered to check the difference in SIS with and without contralateral noise at ipsi +10 dB SNR and +15 dB SNR within group1.

Table 4.4:

Test statistics (|Z|) and significance values for comparison SIS with and without contralateral BBN within Group1.

	|Z|
SIS(I10) and SIS(I10C)	1.06
SIS(I15) and SIS(I15C)	1.08

p value >0.05

Results indicate that there was no significant difference in SIS with and without contralateral BBN both at +10 dB SNR and +15 dB SNR.

For group 2.descriptive statistics were carried out to find the mean, median, and standard deviation of SIS with and without contralateral noise with ipsilateral 10 dB SNR and 15 dB SNR for group2. The same is depicted in Table 4.5.

Table 4.5:

Mean, median and standard deviation (SD) of speech identification scores at different ipsilateral SNR with and without contralateral stimulus within Group 2.

Group2	SIS(I10)	SIS(I10C)	SIS(I15)	SIS(I15C)
Mean	97.13	90.80	97.73	94.07
SD	3.38	6.42	2.27	5.32
Median	100	100	100	100

Descriptive statistics result indicates that there is decrease in the mean value of SIS with contralateral noise conditions at both +10 dB SNR and +15 dB SNR.

Wilcoxon sign ranked test was administered to check the difference in SIS with and without contralateral noise at ipsi +10 dB SNR and +15dB SNR.

Table 4.6:

Test statistics (|Z|) and significance values for comparison SIS with and without contralateral BBN within Group2.

	|Z|
SIS(I10) and SIS(I10C)	6.39**
SIS(I15) and SIS(I15C)	5.39**

**P value < 0.01

Results indicate that there was significant difference in SIS with and without contralateral BBN both at +10 dB SNR and +15 dB SNR.

4.1.3. Comparison of SIS with and without contralateral noise between ears.

Descriptive statistics were carried out to find the mean, median, and standard deviation of SIS with and without contralateral noise with ipsi 10 dB SNR and 15 dB SNR between ears.

Table 4.7:

Mean, median and standard deviation (SD) of speech identification scores at different ipsilateral SNR with and without contralateral stimulus within group across ears.

Group1	Right ear			Left ear		
	Mean	SD	Median	Mean	SD	Median
SIS(I10)	99.60	1.35	100	99.43	1.65	100
SIS(I10C)	99.77	0.94	100	99.43	1.65	100
SIS(I15)	99.89	0.67	100	99.71	1.20	100
SIS(I15C)	99.77	0.94	100	99.66	1.13	100
Group2						
SIS(I10)	96.80	3.69	98	97.47	3.06	100
SIS(I10C)	90.80	6.90	92	90.93	6.02	92
SIS(I15)	97.47	3.23	100	98	2.28	100
SIS(I15C)	94.27	5.21	96	93.87	5.53	96

Descriptive statistics result indicates that there is no much variation in mean values of SIS with and without contralateral BBN between ears in both the groups. The same is depicted in Table 4.7.

Mann-Whitney U test was administered to compare SIS with and without contralateral noise between ears.

Table 4.8:

Test statistics (|Z|) and significance values for comparison SIS with and without contralateral BBN between ears.

Group1	|Z|
SIS(I10)	0.41
SIS(I10C)	0.89
SIS(I15)	0.60
SIS(I15C)	0.46
Group2	|Z|
SIS(I10)	0.58
SIS(I10C)	0.22
SIS(I15)	0.35
SIS(I15C)	0.22

p value > 0.05

The results for both group1 and group 2 indicated that there is no significant difference between right ear and left ear.

4.2. Comparison of contralateral suppression of TEOAE.

Comparison of contralateral suppression of TEOAEs was done between groups and between ears.

4.2.1. Comparison of amount of contralateral suppression of TEOAE between groups.

Descriptive statistics were carried out to find the mean median and standard deviation of contralateral suppression of TEOAE.

Table 4.9:

Mean, median and standard deviation (SD) of contralateral suppression of TEOAEs between groups.

CSOAE*	Group1			Group2	
	Mean	SD	Median	Mean	SD	Median
1 kHz	1.01	0.89	0.85	0.00	0.74	0.10
1.5 kHz	1.02	0.85	0.90	0.54	1.29	0.40
2 kHz	0.99	0.75	1.00	0.29	0.64	0.25
3 kHz	0.84	0.62	0.70	0.12	0.68	0.30
4 kHz	0.50	0.65	0.50	0.16	0.66	0.10

*CSOAE= contralateral suppression of otoacoustic emission.

Results showed decreased mean values of contralateral suppression of TEOAE across all frequencies in group2 than compared to group1. The same is depicted in Table 4.9.

Mann-Whitney U test was administered to compare difference in contralateral suppression of TEOAE between groups.

Table 4.10:

Test statistics (|Z|) and significance values for comparison CEOAE with and without contralateral BBN between Groups.

CSOAE	|Z|
1 kHz	6.35**
2 kHz	5.20**
3 kHz	5.55**
4 kHz	2.67**

** p value < 0.01

Results indicate that there is significant difference between groups at all frequencies.

4.2.2. Comparison of amount of contralateral suppression of TEOAE between ears.

For group 1 descriptive statistics were carried out to find the mean median and standard deviation of contralateral suppression of TEOAEs between ears.

Table 4.11:

Mean, median and standard deviation (SD) of contralateral suppression of TEOAEs in right ear and left ear of Group1.

Group1	Right ear	Left ear
Mean	1.04	0.86
SD	0.58	0.50
Median	0.90	0.80

Results showed that there was no much difference in the mean values of contralateral suppression of TEOAE between ears in Group1. The same is depicted in Table 4.11.

Mann-Whitney U test was administered to compare amount of contralateral suppression of TEOAE between ears.

Table 4.12:

Test statistics (|Z|) and significance value (p) for comparison CEOAE with and without contralateral BBN between ears for Group1.

Group1	|Z|
Right ear and Left ear	1.15

P value > 0.05

Results indicate that there is no significant difference in contralateral suppression of TEOAE between ears.

For group 2 descriptive statistics were carried out to find the mean median and standard deviation of contralateral suppression of TEOAE between ears.

Table 4.13:

Mean, median and standard deviation (SD) of contralateral suppression of TEOAEs in right ear and left ear of Group2.

Group2	Right ear	Left ear
Mean	0.22	0.19
SD	0.37	0.29
Median	0.20	0.20

Results showed that there was no much difference in the mean values of contralateral suppression of TEOAE between ears in Group2. The same is depicted in Table 4.13.

Mann-Whitney U test was administered to compare amount of contralateral suppression of TEOAE between ears.

Table 4.14:

Test statistics (|Z|) and significance value (p) for comparison CEOAE with and without contralateral BBN between ears for Group2.

Group2	|Z|
Right ear and Left ear	0.769

P value > 0.05

Results indicate that there is no significant difference in contralateral suppression of TEOAEs between ears in group 2.

4.3. Correlation between contralateral suppression of TEOAE and shift in SIS in the presence of contralateral BBN.

Spearman's rank correlation test was administered to check whether there is any correlation between contralateral suppression of TEOAE and shift in SIS in the presence of contralateral BBN.

Table 4.15:

Correlation coefficient and significance value (p) for CSOAE and shift in SIS with contralateral noise.

Group1	Correlation coefficient
CSOAE and SIS(I10C)	0.12
CSOAE and SIS(I15C)	0.19

p value > 0.05

For group 1 Results indicate that there is no significant correlation. The same is depicted in Table 4.15.

Table 4.16:

Correlation coefficient and significance value (p) for CSOAE and shift in SIS with contralateral noise.

Group2	Correlation coefficient
CSOAE and SIS(I10C)	0.09
CSOAE and SIS(I15C)	0.11

p value > 0.05

For group 2 Results indicate that there is no significant correlation. The same is depicted in Table 4.16.

CHAPTER 5

DISCUSSION

In this study, the aim was to study the effect of occupational noise exposure on efferent auditory system. The study had 35 participants who were not exposed to occupational noise and 30 participants who were exposed to occupational noise.

Speech identification scores in the presence of contralateral stimulus were checked for both the groups at +10 and +15 ipsilateral SNR. SIS in the presence of contralateral stimulus has been reported to improve scores when ipsilateral SNRs are +10 and +15 (Kumar and Vanaja, 2004). Contralateral stimulus activates efferent auditory system which suppresses the response to steady state which in turn decreases the adaptation of auditory nerve indirectly increasing the SIS (Kumar and Vanaja, 2004). So individuals who are not exposed to occupational noise are expected to get improved SIS in presence of contralateral stimulus along with ipsilateral noise. In current study individuals not exposed to noise had mean scores of 99.74 at +10 ipsilateral SNR and 99.60 at +15 ipsilateral SNR. This could be because of the cealing effect.

But individuals who are exposed to noise got decreased scores in the presence of contralateral stimulus. Mean scores were 90.80 at ipsilateral +10 dB SNR and 94.07 at +15 dB SNR. This can be attributed to damaged efferent auditory system which failed to suppress the response to steady state and failed to decrease the adaptation of auditory nerve which would have increased the SIS in the presence of contralateral acoustic stimulus. Libermann and Guinan, 1998 reported that medial olivo cochlear bundle helps in releasing from masking. Zeng et al, 1994 reported reduced speech identification scores in the presence of noise in individuals who underwent vestibular neurectomy and it was assumed that MOCB were severed during surgery. They concluded that in cases where MOCB was severed the

perception of speech in noise become poor. There was no significant difference for SIS in the presence of contralateral noise between groups. Further, there was no significant ear effect seen for speech identification scores. Similarly no ear effect was reported by Kumar and Vanaja in 2004.

The current study evaluated contralateral suppression of TEOAE's in individuals exposed to occupational noise and individuals who are not exposed to occupational noise. All individuals had normal hearing thresholds (not more than 25 dB HL) at standard audiometric frequencies. So cochlear damage was diagnosed in none of the subjects. However, elevated mean thresholds at >4 kHz in some of noise exposed individuals (5 participants) were present when compared with the control non exposed individuals who showed minor changes to cochlea due to occupational noise. This was further confirmed by poorer OAEs in exposed individuals, which are considered to be an early sign of industrial noise induced hearing loss. In the case of NIHL, two types of damage can be found: a pattern of hair cell degeneration in the first row of the outer hair cells (OHCs), then in the inner hair cells (IHCs), subsequently in the second and third row of OHCs; and a massive destruction of dendrites of the primary auditory neurones below the IHCs. Thus reduction in incidence of OAEs in the noise exposed group may be associated with sensory-cell damage to localised cochlear regions (Desai et al., 1999. cited in Kotylo, 2002).

The inhibitory effect of contralateral stimulation depends on the normal function of crossed efferent fibers and the good condition of outer hair cells, which are the main effector cells in the auditory system (Kotylo, 2002). Occupational noise exposure can be classified as traumatic noise i.e., > 105 dBA which causes permanent threshold shift (Eggermont, 2012), threatening noise i.e., > 80 dBA which causes temporary threshold shift with damage to inner hair cells ribbon synapse (Kujawa & Liberman, 2009) and safe noise level of < 80 dBA. In this study contralateral suppression of TEOAEs is more in non exposed individuals compared

to exposed individuals and this difference is significant. Amplitudes of TEOAEs are also reduced in exposed individuals compared to non-exposed individuals. This can be attributed to damaged efferent auditory system due to occupational noise exposer which failed to suppress TEOAEs and damaged outer hair cells due to noise exposer which may lead to reduced amplitudes in noise exposed individuals. Exposure to high occupational noise damages efferent auditory system of individuals who are exposed to occupational noise showed reduced amplitude and lesser contralateral suppression of TEOAEs. Kotylo, 2002 reported decreased suppression of OAEs in individuals with occupational exposure to noise. Similar results were obtained by Patica Arruda in 2013. Prasher et al., 1998 reported significant reduction in contralateral suppression of TEOAEs. They suggested that contralateral sound activated efferent suppression may provide an early indication of auditory damage after exposure to noise. The current findings also suggest that the physiological test is a relable measure for ealy detection of central dysfucntion due to noise exposure.

Correlation between physiological and behavioral measures were analyzed. There was no significant correlation between contralateral suppression of TEOAEs and SIS in the presence of contralateral acoustic stimulus. Since there was ceiling effect in SIS scores the shift due contralateral acoustic stimulation was very less due to which there is no significant correlation found. However Govil and Vanaja in 2002 reported significant correlation between contralateral suppression of OAEs and shift in speech identification scores in the presence of contralateral stimulus. Similarly Kumar and Vanaja in 2004 reported significant correlation. Current study result of correlation was contradicting the results found by above authors.

CHAPTER 6

SUMMARY AND CONCLUSION

Noise exposure can lead to permanent hearing loss or even changes of the cellular properties within the central auditory pathway (Groschel, Ryll, Gotze, Ernst & Basta, 2014). De souza Alcaras et al., (2013) reported poorer suppression effect in individuals exposed to occupational noise. Suggesting that exposure to noise may have impaired the functioning of the medial olivocochlear efferent auditory system. However several animal experiments (Kujawa & Liberman, 2009; Liu et.al., 2012; Shi et.al., 2013) reported that even for months of continuous noise exposure, there is no apparent effect on behavioural or ABR thresholds, cochlear potentials or hair cell morphology. Norena et.al., (2006) reported that when a cat was exposed for 76 dB(A) noise for almost 4 months for 24 hrs per day, the results showed no changes in ABR thresholds but the central auditory responses were affected. However, it is not clear what effect long exposure times can result on the central auditory system in individuals with normal audiometric thresholds.

Occupational noise exposure can be classified as traumatic noise i.e., > 105 dBA which causes permanent threshold shift (Eggermont, 2012), threatening noise i.e., > 80 dBA which causes temporary threshold shift with damage to inner hair cells ribbon synapse (Kujawa & Liberman, 2009) and safe noise level of < 80 dBA. De souza Alcaras et al., 2013 reported reduced suppression of OAEs in individuals who are exposed to noise. Similar results were obtained by Kotylo in 2002 and Patica Arruda in 2013. Kumar et al., 2012 reported reduced speech recognition scores in individuals who had occupational noise exposure. The behavioural and objective tests of efferent system have been reliably recorded and are non-invasive. Thus, the aim of the study was to see the effect of occupational noise exposure on the efferent auditory system of humans.

Two groups of participants in the age range of 25 to 45yrs were included in the study. Control group (Group1) included 35 individuals who are not exposed to occupational noise and Experimental group (Group2) included 30 individuals who are exposed to occupational noise. Amplitudes of TEOAEs were measured with and without contralateral BBN at 30dBSL and amount of suppression were calculated by subtracting TEOAEs amplitude with and without contralateral stimulus. Speech identification scores were obtained at ipsilateral +10 dB SNR and +15 dB SNR with and without contralateral stimulus conditions.

The data obtained was analysed using statistical package of social science (SPSS) software version 20.0. Shapiro Wilks test of normality was administered to check whether the data is normally distributed or not and was found to be not normally distributed ($p<0.05$). Hence, non-parametric inferential statistics were done. Mann- Whitney U test was administered for between group comparisons, Wilcoxon sign ranked test was performed to compare amplitude of TEOAEs with and without contralateral BBN and speech identification scores with and without contralateral BBN. Spearman's rank correlation test was performed to check for the correlation between contralateral suppression of OAE and shift in speech identification scores in the presence of contralateral BBN.

The results revealed that there is no significant difference in SIS with and without contralateral stimulus both at ipsilateral +10 dB SNR and +15 dB SNR in individuals not exposed to occupational noise but in individuals exposed to occupational noise there was significant reduction in SIS when contralateral stimulus was presented both at ipsilateral +10 dB SNR and +15 dB SNR. There was significantly less contralateral suppression of TEOAEs in individuals exposed to occupational than compared to who are not exposed to occupational noise. There was no significant correlation found between amount of contralateral suppression and shift in SIS in the presence of contralateral stimulus both at ipsilateral +10 dB SNR and +15 dB SNR. It can be concluded that efferent system will be affected in

individuals who are exposed to occupational noise exposure and they are at risk for noise induced hearing loss.

5.1 Implications of the study

1. The study helped to understand efferent auditory system in individuals exposed to occupational noise as the results showed recued suppression and reduced SIS scores in them.
2. This would help in counselling the patient about affected speech identification in presence of noise and about being risk for central damage which leads to difficulty in speech identification in the presence of noise

5.2 Future directions

1. To carry out the study with different duration of occupational noise exposure.
2. To carry out the study with different levels of noise exposure for different durations.

6.3 Limitations of the study

The sample size is less, since the variation in amplitude of TEOAEs are more larger sample size would have accounted for it.

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