COCHLEAR FUNCTIONING IN INDIVIDUALS WITH SENSORINEURAL HEARING LOSS WITH AND WITHOUT TINNITUS

Sreelakshmi, H. D.

Register No.: 15AUD032

A Dissertation Submitted in Part Fulfilment of Degree of

Master of Science [Audiology]

University Of Mysore

ALL INDIA INSTITUTE OF SPEECH AND HEARING

MANASAGANGOTHRI, MYSURU- 570 006

May, 2017

CERTIFICATE

This is to certify that this dissertation entitled '**Cochlear functioning in individuals with sensorineural hearing loss with and without tinnitus'** is a bonafide work submitted in part fulfilment for degree of Master of Science (Audiology) of the student Registration Number: 15AUD032. This has been carried out under the guidance of a faculty of this institute and has not been submitted earlier to any other University for the award of any other Diploma or Degree.

Mysuru
May, 2017

Dr. S. R. Savithri,
Director
All India Institute of Speech and Hearing,
Manasagangothri, Mysuru- 570 006

CERTIFICATE

This is to certify that this dissertation entitled '**Cochlear functioning in individuals with sensorineural hearing loss with and without tinnitus'** has been prepared under my supervision and guidance. It is also been certified that this dissertation has not been submitted earlier to any other University for the award of any other Diploma or Degree.

Mysuru
May, 2017

Mr. Sreeraj K.
Guide
Lecturer in Audiology
All India Institute of Speech and Hearing,
Manasagangothri, Mysuru- 570 006

DECLARATION

This is to certify that this dissertation entitled **'Cochlear functioning in individuals with sensorineural hearing loss with and without tinnitus'** is the result of my own study under the guidance a faculty at All India Institute of Speech and Hearing, Mysuru, and has not been submitted earlier to any other University for the award of any other Diploma or Degree.

Mysuru,
May, 2017

Registration No. 15AUD032

ACKNOWLEDGEMENTS

First of all I wish to express my sincere gratitude to my guide **Mr. Sreeraj**, who has been supporting and inspiring me throughout the dissertation, without him I would have not completed my dissertation.

My sincere gratitude to all lecturers of speech and hearing, **AIISH**, who have inspired me to become a better researcher, a better clinician and, ultimately a better audiologist from the time I began my journey into this field.

I would like to thank **Dr. Santhosha C. D.** for helping in statistics and always ready to clear my doubts. I would like to thank **Prashanth sir** for helping me in analysing the data. I would also like to thank **Vikas sir, Srikar sir**, and all the clinical staffs, who have been very cooperative during data collection and for opening department whenever we had asked for.

I would like to thank **MR (Sanjeev), Sujan, Jeena, Shanthu, Sneha, Shama, Raji and Preethi** who helped me throughout my dissertation work. A special thanks to **Meenamma (menu)** for taking permission to open the Department.

My dissertation will be incomplete if I forget to thank my bestiees **Pavana, Devu, Usha, Amulya** and all the people from my beloved group **NAVAS.** Thank you for encouraging me throughout and motivating me to keep moving from all ups and downs in the journey.

I am very much thankful to my friends, classmates, seniors and juniors for their support and love.

Thanks a ton to my **Amma and Suni** who constantly supported me in every single stage of my life. Thank you mom for all the struggle and sacrifices you have made for me, I would consider myself very lucky to be your daughter.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

Abstract

The aim of the study was to evaluate the cochlear functioning in sensorineural hearing impaired individuals with and without tinnitus. The study consists of two group of hearing impaired participants in the age range of 18 to 45 years. Group 1 consist of 15 participants of hearing impaired with tinnitus and Group 2 consists of 15 participants of hearing impaired without tinnitus. Each group were further divided into 3 sub-groups depending on degree of hearing loss in to minimal, mild and moderate (each subgroup consists of 5 participants). The cochlear function was assessed through SWPTC, TEN test and ECochG. Results revealed, reduced Q10 of PTC and reduced CM amplitude in individuals with tinnitus compare to individuals without tinnitus; whereas no significant difference was found between groups for tip frequency of PTC and TEN test. It was also found that the there was no significant difference within individuals with tinnitus across all parameters. However, PTCs obtained from individuals with tinnitus reduced from minimal to moderate hearing loss. Thus, it can be concluded from the study that, damage to OHC are more common in individuals with tinnitus than in those without tinnitus also that OHC are the probable site of generation for tinnitus. Also, it can be concluded that, as degree of hearing loss increases the frequency resolution of cochlea tend to become poorer.

Chapter 1

Introduction

Tinnitus is the perception of sound in the absence of any external sound. The word tinnitus is derived from Latin word 'tinnire', which means 'to ring'. As defined by McFadden (1982), 'Tinnitus is the conscious expression of a sound that originates in an involuntary manner in the head of its owner, or may appear to him to do so'.

There is an increased risk of tinnitus associated with hearing loss. Studies have shown a clear relation between tinnitus and hearing loss (Axelsson & Barrenas, 1992; Davis & Refaie, 2000), and most of individuals with tinnitus have certain degree of hearing loss (Axelsson & Ringdahl, 1989; Davis & Refaie, 2000; Henry & Wilson, 2001; Vernon, 1998). A prevalence study done by Davis (1995) reported tinnitus prevalence of 15.1% in the age range of 41 to 70 year. A similar result was obtained by Dawes et al. (2014), wherein, he reported that out of 10.7% of adult population who had significant hearing impairment, 16.9% reported to have tinnitus. The other risk factors include head and neck injuries, noise exposure, ear diseases, cardiovascular diseases, medication, mental status, and lifestyle factors (Ahman & Seidman, 2004; Hoffman & Red, 2004). Tinnitus can also occur in the absence of hearing loss with no specious change in audiometric threshold (Schaette & McAlpine, 2011; Weisz, Hartmann, Dohrmann, Schlee, & Norena, 2006).

The exact pathophysiology underlying tinnitus is yet to be understood. No single theory, hypothesis or the model can explain pathophysiology of tinnitus, but it is the multiple mechanism which results in perception of tinnitus. The hypothesis of discordant damage has postulated that, the tinnitus is produced on the portion of the

basilar membrane which has preserved IHC but temporarily dysfunctional or damaged OHC (Bohne & Clark, 1982; Bohne, Yohman, & Gruner, 1987; Liberman, 1987; Liberman & Dodds, 1987; Liberman and Mulroy, 1982; Liberman & Kiang, 1978).

Animal studies have reported a strong link between the presence of tinnitus and damage to the auditory peripheral system (Bauer, Turner, Caspary, Myers, & Brozoski, 2008; Brozoski, Bauer, & Caspary, 2002; Heffner & Harrington, 2002; Kaltenbach, Zacharek, Zhang, & Frederick, 2004). However, the tinnitus perception was still reported even after the ablation of auditory nerve (Sasaki, Babitz & kauer, 1981). This indicates that tinnitus is majorly a central phenomenon, such as cortical reorganization (Eggermont & Komiya 2000; Rajan & Irvine, 1998) or hyperactivity present in the central auditory pathway (Sasaki, Kaner, & Babitz, 1980; Kaltenbach, Rachel, Mathog, Zhang, Falzarano, & Lewandowski, 2002; Eggermont, 2007; Bauer et al., 2008). Therefore, damage in the inner ear is likely necessary, but not adequate, for tinnitus to occur (Cacace, 2003; Lanting, De Kleine, & Van Dijk, 2009).

Sensorineural hearing impairment consists of outer hair cells, inner hair cells damage or both, with outer hair cell being more susceptible to damage (Hawkins, 1973; Jastreboff, 1990), but studies have also shown that inner hair cell damage with subsequent neural degeneration can co-occur with outer hair cells being functionally normal (Kujawa & Liberman, 2009).

It is unclear that which of this cochlear damage might cause the central changes and results in tinnitus perception. A strong relationship between tinnitus and the lack of afferent fibres to central auditory structures was found by Bauer et al. (2008). This suggests that OHC dysfunction might not be vital to tinnitus perception.

Thus, it is important to study peripheral dysfunction which might have predominant effect on tinnitus perception in individual with hearing loss.

Hearing loss is mainly assessed through the audiometry; still auditory threshold assessed through audiometry gives limited information about the status of the cochlea at the signal frequency. Studies have reported only moderate correlation between degree of hearing loss and OHC dysfunction (Davis, Qiu, & Hamernik, 2004). Also, damage to IHC can result in less responsive region in cochlea which results in off- frequency listening (Moore, Huss, Vickers, Glasberg, & Alcantara. 2000; Moore, 2004). Hence, detailed assessment of inner ear is necessary, which should include test other than audiometry which can assess inner hair cell and outer hair cell damage independently and help to draw a conclusion about tinnitus and hearing loss.

PTC (Psychophysical Tuning Curves) measures the frequency resolution of cochlea. For individuals with normal hearing, the tip of the PTC lies close to the signal frequency (Moore, 1978; Moore et al., 2000; Moore & Alcantara, 2001). Studies have reported that variations which present in psychophysical and physiologic tuning curves which shows a reduced sharpness of tuning which is measured from damaged OHCs (Ryan, Dallas, & McGee, 1979; Robertson, Cody, Bredberg, & Johnstone, 1980; Harrison, Aran, & Erre, 1981; Liberman & Dodds, 1984; Smith, Moody, Stebbins, & Norat, 1987).

Moore (2004) employed PTC to explore dead region in cochlea (which is basically complete loss of IHC in certain place of cochlea), his results suggested that tip of the PTC's will shifted towards the edge frequency where effective masking takes place. The cochlear dead region can be also found using TEN (Threshold-

Equalizing Noise) test, given by Moore et al. (2000). The TEN test is employed in detection of pure tone signal in the presence of masker. The masker is broadband noise which has fixed intensity. If the dead region is present in the individual, the TEN noise should cause the masker threshold to be elevated to threshold measured in quite in the frequency region with dead region (Moore et al., 2000; Moore, 2004).

Other test to assess the functionality of cochlea includes Electrocochleography (ECochG). ECochG is a method for recording the electrical potentials of the cochlea. The component of ECochG includes Cochlear microphonic (CM), Summating potential (SP) and Compound action potential (CAP). The CM is believed to directly reflect the functional status of the OHC predominantly (Yoshie & Yamaura, 1969; Elberling & Salomon, 1973; Eggermont, 1976). About 25% of the OHC loss along the cochlear partition will result in reduction of CM potential by 25% or less than 3dB. OHC loss may evident approximately in the order of 15 to 25 dB loss of auditory sensitivity in response to tone from 500 to 4000 Hz (Davis, Ahroon, & Hamernic, 1989).

In order to evaluate the involvement of cochlear pathology, that is extent of OHC and IHC damage which leads to tinnitus perception, the test battery should include tests which assess OHC and IHC functions independently. Thus, test battery of PTC, TEN and EcochG test will be helpful assessing functionality of OHC and IHC independently.

Need of the study.

Tinnitus consists of many ontological symptoms, which requires audiological assessment in detail. Many models suggest that tinnitus is central in origin (Bauer et al. 2008; Brozoski et al. 2002; Kaltenbach et al. 2004). At the same time, many

studies report that it's the peripheral pathology which leads tinnitus of central origin. The discordant damage hypothesis suggests that tinnitus is generated with damaged or temporarily dysfunction of OHC but preserved IHC. (Bohne & Clark, 1982; Bohne et al., 1987; Liberman, 1987; Liberman & Dodds, 1987; Liberman & Mulroy, 1982; Liberman & Kiang, 1978).

A study done by Tan, Lecluyse, McFerran and Meddis (2013) assessed cochlear function in hearing impaired individual with tinnitus and without tinnitus using psychophysical measures and their result suggested better OHC functioning in individual with tinnitus. Another complimenting study done by Kiani,Yoganantha,Tan, Meddis and Schaette (2013) reported presences of dead region in hearing impaired individual with tinnitus which is proportional to hearing impaired individual without tinnitus using PTC. Both studies report better OHC functioning and IHC dysfunction.

In contrast, study done by Mitchell and Creedon (1995) employed Psychophysical tuning curves and studied difference in PTC in individual with tinnitus and without tinnitus and their result showed significantly different between individual with and without tinnitus, and subjects often had some elevated tips and hypersensitive tails. The shapes of tuning curves were consistent with cochlear lesions which involve the damage to outer hair cells.

It is unclear that which part of the cochlea is involved in generation of tinnitus. Few studies reported that it's the OHC damage/ hyper functioning in the cochlea which causes the tinnitus, in contrast few studies reported that presence of the cochlear dead region which involves in generation of tinnitus.

There are many studies in literature which reports that the peripheral pathology which results in tinnitus of central origin (Bauer et al. 2008; Brozoski et al. 2002; Heffner & Harrington, 2002; Kaltenbach et al. 2004). But, it is imprecise as in whether peripheral pathology includes OHC or IHC dysfunction. Thus the present study is taken up with purpose of identifying the specific role of OHC and IHC in tinnitus perception.

Aim of the study.

The present study aims to assess the OHC and IHC functioning in individual with and without tinnitus having various degrees of hearing loss.

Objectives of the study.

1. To compare findings of PTC, ECochG and TEN test in individual with (Group 2) and without tinnitus (Group 1) across different degree of hearing loss.
2. To compare findings of PTC, ECochG and TEN test in individual with tinnitus (Group 2) across different degrees of hearing loss.

Chapter 2

Review of literature

2.1. Tinnitus definition and its representation.

Tinnitus is defined as a phantom auditory perception. As defined by Jastreboff (1990) it is a perception of sound without corresponding acoustic or mechanical correlates in the cochlea. The episodes of tinnitus can be very short or it may be continuous. Consensual criteria that differentiate such normal ear noises from pathologic tinnitus have not been developed. Some authors have specified that tinnitus must exceed 5-min duration (Coles, 1984; Davis, 1995; Hazell, 1995) to consider it as a relevant symptom. Dauman and Tyler (1992) proposed that pathologic tinnitus is head noise lasting at least 5 min that occurs more than once per week. Both of these definitions would constitute low-fence criteria to define an internal sound that is present most or all of the time for the typical tinnitus patient (Meikle, Creedon, & Griest, 2004). Tinnitus can be subjective or objective. Objective tinnitus is caused by the internal stimuli such as blood flow pulsation, whereas in subjective tinnitus there is no actual physical sound outside or inside the body that could account for tinnitus (Eggermont, 1976).

Tinnitus is a frequent phenomenon occurring in an estimated 10 to15% of the population (Hoffman & Reed, 2004; Henry et al., 2005). Most of the studied link tinnitus to the presence of hearing loss. A study done by Axelsson and Barrenas (1992) reported that tinnitus was more common in individuals with hearing loss than in individuals without hearing loss (Davis & Refaie, 2000) although tinnitus can also occur in the absence of hearing loss (Weisz et al., 2006). Vernon and Meikle (2000) reported that 70% to 80% of tinnitus patients have ‘significant hearing difficulties’.

A prevalence study reported by Axelsson and Ringdahl (1989) where in a three thousand six hundred subjects were randomly selected and questionnaire was administered, the results Showed 14.2% of individuals suffered from tinnitus 'often' or 'always' and it was more common in males than in females; and it was seen that tinnitus was more common in left ear than in right ear. They also reported that tinnitus is more common in individuals with hearing loss than with normal hearing. A prevalence study done by Davis (1995) reported tinnitus prevalence of 15.1% in the age range of 41 to 70 year.

Nondahl et al. (2002) had conducted a longitudinal study on incidence of tinnitus which was a population based study in hearing impaired adults with age range of 48 to 92 years. The self-reported data on tinnitus were obtained initially during baseline examination and after 5 years and they reported an incidence of 5.7% and also tinnitus is more commonly seen in older adults. Another incidence study reported by Sanchez, Boyd & Davis (1999) estimated an incidence of 7% in elderly individuals.

2.2. Causes of tinnitus.

Hypotheses concerning mechanisms of generation of tinnitus are plentiful. There exists heterogeneity in causes of tinnitus observed in the tinnitus population (Moller, 1997). It may be noted that no single theory, model or hypothesis can explain the presence of tinnitus in all individuals with tinnitus. Rather, a multiple mechanisms may be involved in generation of tinnitus (Baguley, 2002). Tinnitus can also occur as a symptom of certain pathology like otosclerosis, vestibular schwannoma and Meniere's disease which can be correct through medical or surgical intervention.

2.2.1. Cochlear models. *Spontaneous oto-acoustic emissions:* Individuals with normally functioning cochlea may generate low level tonal or narrow band sound without external acoustic stimulation (Gold, 1948). Kemp (1978) reported a small amount of acoustical signal are generated in cochlea as a by-product of electromotile activity of the OHCs and propagated into the external auditory canal and he called this as Spontaneous otoacoustic emissions (SOAEs). These SOAEs generated by cochlea can be perceived as tinnitus.

Discordant damage of IHC and OHC: According to Jastreboff in 1990, one of the mechanisms which explain of the source of tinnitus is 'Discordant damage of IHC and OHC'. According to this theory, differential damage or dysfunction of OHC (being more prone to the damage than IHC) which results in the disinhibition of neurons in the dorsal cochlear nuclei (DCNs). The increase in Spontaneous activity is seen only when neurons in the DCN receive excitation from IHCs but not from the damaged OHCs, and this is perceived as tinnitus (Jastreboff & Hazell, 1993).

2.2.2. Non-cochlear mechanisms of tinnitus generation. Neurophysiological model of tinnitus perception by Jastreboff (1990) considered the role of 'signal recognition and classification circuits' for perception of tinnitus. He believed that cochlear dysfunction might generate weak perception of tinnitus but when the 'negative emotional reinforcement' was attached, by activation of limbic system and autonomic activation, which cause tinnitus activity to be enhanced and persistent.

a) Synchronisation of spontaneous neural activity. According to Moller (1984) certain forms of tinnitus can be related to abnormal neural phase locking phenomenon. The reason he gave for tinnitus perception was the artificial synapses, which occur as a result of damaged cranial nerve and leads to ephaptic transmission

between nerve fibres. Such cross talk between nerves results in phase locking of spontaneous activity which results in tinnitus.

Eggermont (1990) hypothesised that excess influx of K+ or Ca2+ ions into the hair cell results in transient hair cell depolarizations causing synchronous transmitter release at all hair cell synapses. The model produces the excess of short inter-spike intervals found in auditory nerve fibre recordings in animal models of tinnitus as well as the theoretically required correlation in the activity of neighbouring neurons.

Kaltenbach et al. (2004) made an hypothesis, where they have categorized the various forms of plasticity that characterize tinnitus and searched for their neural underpinnings in the dorsal cochlear nucleus (DCN). They believed DCN as a possible site for the generation of tinnitus-producing signals owing to its tendency to become hyperactive following exposure to tinnitus inducing agents such as intense sound and cisplatin.

b) Somatic modulation. Lockwood et al. (1998) used PET to map brain regions responding to changes in tinnitus loudness in four individuals who could alter tinnitus loudness by performing voluntary oral facial movements (OFMs) and six individuals without tinnitus who represented controls. The cerebral blood flow was measured in three conditions, at rest, during the OFM, and during stimulation with pure tones. OFM-induced loudness changes affected the auditory cortex contralateral to the ear in which tinnitus was perceived, whereas unilateral cochlear stimulation caused bilateral effects, suggesting a retrocochlear origin for their tinnitus. Individuals with tinnitus, compared with controls, showed evidence for more widespread activation by the tones and abnormal links between the limbic and auditory systems.

These abnormal patterns provide evidence for cortical plasticity that may account for tinnitus and associated symptoms.

In 1999, Levine investigated this phenomenon by asking all patients attending the tinnitus clinic to perform a series of head & neck contractions. Over two-thirds (68%) reported a change in their tinnitus: loudness, pitch & laterality could all be affected. Decrease in tinnitus was more likely to occur, if the tinnitus was unilateral. The findings were used to suggest that somatic inputs could disinhibit the Ipsilateral dorsal cochlear nucleus (DCN), acting via the medullary somatosensory nucleus. This disinhibition could affect spontaneous activity in the DCN, altering tinnitus perception. However, the anatomical evidence in humans is less clear.

c) Analogies with pain. Moller (1997) found considerable evidence that both chronic pain and some forms of tinnitus are caused by changes in the central nervous system and they study showed no correlation between ear of tinnitus and anatomical location of pain. Such changes in the central nervous system may have been induced by peripheral processes such as tissue damage, but the changes can persist a long time after complete healing of a peripheral lesion.

d) Cortical re-organisation. The precise tonotopicity that has been demonstrated in the central auditory pathways indicates de-afferentation of a specific portion of the cochlea, in the short-term, leading to reduced activity in the cortical area with corresponding characteristic frequency (CF). If similar measurements are made some months later, that area is again responsive to sound, but many neurones now have CFs adjacent to that of the lesioned region (Salvi, Lockwood, & Burkard, 2000). One consequence of this re-organisation is that a disproportionately large number of neurones will be sensitive to frequencies at the upper and lower borders of

the hearing loss. Evidence for re-organisation of the auditory cortex being a mechanism of tinnitus generation in humans was reported by Mulnickel, Elbert, Taub and Flor (1998) and Dietrich, Nieschalk, Stoll, Rajan and Pandey (2001).

2.3. Tinnitus and Hearing loss.

There exists a strong link between presences of tinnitus and hearing loss, but not all individual who have hearing loss will be having tinnitus.

A study done by Axelsson and Sandh (1985) analysed perception of tinnitus in 94 individuals with noise induced hearing loss. The result revealed, tinnitus was more common at high frequency and mean levels of tinnitus was corresponding to audiometric thresholds. Whereas, subjective tinnitus rating showed reduced correlation between audiometric threshold and sensation level.

Most of the studies have reported a relation between the presence of tinnitus and damage to the auditory peripheral system (cochlea). A study done by Bauer et al. (2008) who selectively damaged the cochlea hair cell in chinchillas. In first condition only cells responsible for low frequencies were selectively damaged, in second condition prominent OHC loss with some IHC loss was given and in third condition prominent IHC loss and some OHC loss was given and last was controls which had normal hearing. It was revealed from the results that first and second condition showed increases in spontaneous activity at the level of contralateral inferior colliculus. They concluded cochlear dysfunction likely necessary which causes multiple changes in central system and generate tinnitus.

Mulders and Robertson (2009) studied the effect of peripheral pathology on central changes. The experiment was done on guinea pigs, where they have induced acoustic trauma by giving continuous 10000 Hz tone at 124 dBSPL for one hour. Results showed a small and permanent hearing loss in restricted frequency region of

cochlea and hyperactivity was seen in inferior colliculus which corresponds to the frequency of the cochlear damage. Thus, they conclude that the dependency of hyperactivity in the central system is mediated by the integrity of the peripheral receptor.

Schaette and McAlpine (2011) conducted a study, wherein they have considered tinnitus with individuals with normal hearing and studied cochlear functioning in them through electrophysiological test. 30 individuals with normal hearing were included in the study; in which 15 were with tinnitus and 18 were without tinnitus. Auditory brain stem responses were obtained in these individuals. Results reveal a significant reduction in wave I amplitude in subjects with tinnitus and showed normal amplitude of wave V which is more centrally generated wave. They concluded that, there is a hidden hearing loss which manifests as decrease in neural cochlear output and results in renormalization of the auditory brainstem.

2.4. Cochlear functioning in individuals with sensorineural hearing loss with tinnitus.

Sensorineural hearing impairment consists of outer hair cells, inner hair cells damage or both; with outer hair cell being more susceptible to damage (Hawkins, 1973; Jastreboff, 1990), but studies have also shown that inner hair cell damage with subsequent neural degeneration can co-occur with outer hair cells being functionally normal (Kujawa & Liberman, 2009). Damage to the outer hair cell on certain part of the basilar membrane with intact inner hair cell might involve in generation of tinnitus where functional damage to the OHC may not be indicated in the audiometric testing.

2.4.1. Cochlear functioning and PTC. One of the method to study frequency resolution of the auditory system is through psychophysical tuning curve (PTC) given

by Chistovich, 1957). It is obtained by presenting masking noise or a tone just required to mask the probe tone, which is usually presented at 10 dB above the threshold as a function of masker frequencies. The probe tone is a sinusoidal tone which is fixed in frequency and intensity and narrow band noise will be used as masker since it avoids perception of beads which might influence the result. In individuals with normal hearing the tip of the PTCs will lie near the probe frequency (Moore, 1978). In individual with cochlear hearing loss the PTCs will be usually broader and lacks the sharp tip (Hoekstra & Ritsma, 1977; Zwicker & Schorn, 1978; Kluk & Moore, 2005).

The important function of outer hair cell is frequency selectivity, which is bought about by active mechanism of OHCs (Dallos 1992; Nobili et al. 1998., Griffiths, Blakemore, Elliott, Moore & Chinnery, 2001) and when there is a lesion in OHCs the PTCs will be broaden (Ryan, Dallas, & McGee, 1979; Robertson et al., 1980; Harrison, Aran, & Erre, 1981; Liberman & Dodds, 1984; Smith et al., 1987).

Bonding (1979) reports that frequency selectivity and speech discrimination in individuals with sensorineural hearing loss is affected. Psychoacoustic tuning curve (PTC) was obtained at 1000 Hz and was compared to speech discrimination capacity in patients with cochlear disorders having relatively flat audiometric pattern. The result revealed that there was a change in PTC with increasing hearing loss which showed rapidly deterioration beyond normal limit values when the hearing loss exceeded 30 to 40 dBHL; and the same was observed in speech discrimination score obtained in presence of noise. There was a deterioration of speech discrimination score when the hearing loss was greater than 30 to 40 dBHL. Thus, he concluded that, PTC is most effective measure to evaluate frequency selectivity and impaired speech discrimination is caused by impaired frequency selectivity in individuals with cochlear disorders.

Florentine, Buus, Scharf and Zwicker (1980) studied frequency selectivity in individuals with and without hearing loss using psychoacoustic tuning curves. A total of five groups participated in the study. Group 1 without hearing loss and other groups consist of individuals having conductive pathology, otosclerotic, noise-induced, or hereditary degenerative disorder. The frequency selectivity was reduced for individuals with cochlear hearing loss compared to normal hearing individuals and reduction in the frequency selectivity was greatly correlated with the extent of cochlear damage.

The animal study done by Smith, Moody, Stebbins, and Norat (1987) wherein they have selectively damaged the OHCs and recorded PTCs for the frequency of 500 Hz, 2000 Hz, 4000 HZ and 8000 Hz at 10 dBSL. Their result showed elevation in the tip region of PTCs which was associated with the increase in threshold. With increases in threshold of up to 30-40 dB, there was a selective elevation and broadening of the tip region in the PTC response. Once the threshold is 50 dB or greater, the tip response were completely absent. This suggests that the final transition of the PTC to low-pass filter function concomitant with shifts in threshold of greater than 40-50 dB is a result of complete removal of OHC influence.

Nelson (1991) studied PTC using forward masking procedure. The study consists of 26 normal hearing individual and 24 hearing impaired individuals and PTCs were obtained at 1000 Hz probe tone at different levels. The result indicated that the low-frequency slopes of PTCs from hearing-impaired listeners were not different from those of normal-hearing listeners. Hearing-impaired listeners did not demonstrate abnormal upward spread of masking when equivalent masker levels were compared. An abnormally broder PTCs were obtained in ten individuals with coclear

hearing loss, indicating cochlear hearing losses greater than 40 dBHL influence the sharp tuning capabilities usually associated with outer hair cell function.

PTCs may be useful as a general diagnostic tool (Zwicker & Schorn, 1978) and specifically for the diagnosis of dead regions in the cochlea and estimation of their edge frequencies (Moore et al, 2000; Moore & Alcántara, 2001; Kluk & Moore, 2005, 2006). Dead region refers to complete loss of inner hair cell in some part of basilar membrane and it can also lead to deafferentation of the auditory nerve (Moore, Glasberg & Stone, 1997) as a result, off – frequency listening can occur. A signal may be detected by an adjacent cochlear region that responds at a lower sound level even though it is not tuned to the signal frequency (Moore et al., 2000; Moore, 2004).

When a hearing-impaired person has a dead region in the cochlea at the signal frequency (a region where the inner hair cells and/or neurons are functioning very poorly), the tip of the PTC may be shifted away from the signal frequency i.e. the vibration can be spread to basal or apical of basilar membrane where there is surviving neurons (Thornton & Abbas, 1980; Turner et al., 1983; Florentine & Houtsma, 1983; Moore et al., 2000; Moore & Alcántara, 2001; Kluk & Moore, 2005, 2006).

Moore and Alcantara (2001) evaluated cochlear dead region using PTCs. The subjects consist of five individuals with hearing loss having various audiometric configurations. PTCs were obtained in these individuals. The result showed mid-frequency dead region in individuals with mid-frequency loss and high-frequency hearing dead region in individuals with high-frequency loss. Thus, they concluded that PTC can be used to detect and define cochlear dead region and tip of the PTC is used to define approximately one boundary of the dead region. Moore (2004)

employed PTC to explore dead region in cochlea, his results suggested that tip of the PTC's will be shifted towards the edge frequency where effective masking takes place.

2.4.2. Cochlear dead region and TEN. Cochlear dead region are characterised by presence of non-functional IHCs and is not necessarily shown in the audiogram (Moore, 2000). For example in the presence of low frequency dead region, the neurons with characteristic frequency (CF) about dead region can respond instead for stimulation, thus the audiometric threshold may be better. (Thornton & Abbs, 1980; Florentine & Houtsma, 1983).

TEN (Threshold-Equalizing Noise) test is the most time efficient tool to diagnose hearing impaired individuals with dead region, given by Moore et al. (2000). The TEN Test employs detection of pure tone signal in the presence of masker. The masker is broadband noise which has fixed intensity. If the dead region is present in the individual, the TEN noise should cause the masker threshold to be elevated to threshold measured in quite in the frequency region with dead region (Moore et al, 2000; Moore, 2004). In individual with moderate to severe cochlear hearing loss without dead region, the masked threshold at signal frequency is usually 2-3 dB higher than normal hearing individual (Glasberg & Moore 1990; Moore, Glasberg & Stone, 1997). In individual with hearing loss the masked threshold of 10 dB or higher above the TEN level will considered as presence of dead region (Moore et al., 2000).

Summers et al. (2003) used PTC and tone detection thresholds in threshold-equalizing noise (TEN) in identifying dead regions in listeners with high frequency hearing loss. Seventeen individuals (18 ears) having moderate to severe hearing loss with steeply sloping at high frequencies were included in the study. The author found that only 56% agreement between results of PTC and TEN (10 individuals out of 18)

and they could also find a conflicting results between PTC and TEN test at more than one frequencies. Finally, author concluded that excessive amount of masking might cause error in TEN test result and they considered PTC as a reliable tool in diagnosing dead region in cochlear rather TEN .

Warnaar and Dreschler (2012) studied the agreement between the PTC and TEN in identifying the dead region in cochlea. Twenty four individuals with audiometric loss of greater than 60 dBHL were included in the study. PTC and TEN test were administered to find dead region. Depending on the criteria used the agreement between the PTC and TEN test varied. The result showed highest agreement when PTC shift was 20% and 8 dB in probe elevation above TEN masked threshold was used for diagnosing dead region.

2.4.3. Cochlear functioning and Electrocochleography. Other test to assess the functionality of cochlea includes Electrocochleography (ECochG). ECochG is a method for recording the electrical potentials of the cochlea. The component of ECochG includes Cochlear microphonic (CM), Summating Potential (SP) and Compound Action Potential (CAP). The Cochlear Microphonic (CM) believed to directly reflects the functional status of the OHC predominantly (Yoshie & Yamaura, 1969; Elberling & Salomon, 1973; Eggermont, 1976).

An experiment done by Spoendlin and Baumgartner (1997), wherein they have selectively damaged the IHCs alone and OHC alone and recorded ECochG reports that damage to the auditory nerve resulted in normal CM, but reduction in CAP and damage to the organ of corti resulted in abolition of CM wherein CAP was barely affected even with few OHC loss and small number of functional IHCs. There for they concluded presence of CM is indication functioning of OHC.

2.5. Cochlear functioning in individuals with sensorineural hearing loss with tinnitus.

A study done by Zhou, Henin, Long and Parra et al. (2011) assessed cochlear functioning in individuals with tinnitus with without tinnitus in normal hearing as well as hearing impaired individuals. To assess functioning of cochlea, the author carried out perceptual thresholds testing and measurement of distortion product otoacoustic emissions (DPOAEs). The individuals with tinnitus also underwent 'tinnitus likeness spectrum' where the subjects rated their tinnitus in agreement to the stimulus presented which varied across frequency and bandwidth. Result showed that subjects with tinnitus had elevated thresholds, reduced DPOAE, and increased slope of the DPOAE input-output function in high frequency region ranging from 4000 Hz to 10000 Hz. Also, elevation in the perceptual threshold correlated with the tinnitus rating and this was also indicated by reduced amplitude in DPOAE in those frequency regions, which suggest impaired cochlear functioning in individual with tinnitus.

Dauman, and Cazals (1989) has studied frequency selectivity in individual with tinnitus using Psychoacoustic tuning curves with simultaneous pure tone masking method. They could clearly identify broadening of frequency selectivity in individual with tinnitus having bilateral hearing loss and also they could find broadening was more in the ear with the tinnitus than the ear without tinnitus which strongly suggests tinnitus originates in the cochlea and outer hair cell are site of generation for tinnitus.

A recent study done by Tan, Lecluyse, McFerran and Meddis (2013) assessed cochlear function in hearing impaired individual with tinnitus and without tinnitus using psychophysical measures. The study included 27 individuals having SNHL with tinnitus and 15 individuals having only SNHL without tinnitus. Author found that

hearing impaired individual with tinnitus had better compression and frequency selectivity than those without tinnitus indicating better OHC functioning in individual with tinnitus. This suggested that subjective tinnitus is not strongly liked to dysfunction of OHC also dysfunction of IHC which subsequently causes reduction in auditory nerve might be possible generator for tinnitus.

Another complimenting study done by Kiani, Yoganantha, Tan, Meddis and Schaette (2013) aimed to see presence of off- frequency listening in individuals with chronic tinnitus. The author used psychophysical tuning curves using a forward-masking paradigm in 20 individuals with tinnitus having varying degree of hearing threshold. 16 out of 20 individuals showed the presence of dead region indicating better OHC functioning and IHC dysfunction.

In contrast, study done by Mitchell and Creedon (1995) employed PCTs to study cochlear functioning in individual with tinnitus and without tinnitus. A total of 18 individual participated in the study; seven individuals with tinnitus and 11 individuals without tinnitus having normal audiometric threshold till 8000 Hz. PTC were obtained in the tinnitus matched frequency in tinnitus population whereas for normal individual PTC were obtained at the same frequency to that of tinnitus population. Result showed significantly different between individual with and without tinnitus, and subjects often had some elevated tips and hypersensitive tails. The shapes of tuning curves were consistent with cochlear lesions which reflect damage to the outer hair cells.

A study done by Thabet (2009) evaluated the functioning of cochlea in individuals with tinnitus having normal hearing through TEOAEs and TEN test. Twenty individuals having unilateral tinnitus with normal hearing sensitivity

participated in this study. Their other ear acted as control ears. Result showed 85% of tinnitus ears showed abnormality in TEOAEs when compared to only 20% in control ears. The abnormal TEOAEs frequency bands in the tinnitus ears were statistically significant above 2000 Hz when compared to the control ears and were more common for the 4000 and 5000 Hz. This suggests that OHCs dysfunction may be important in the generation of tinnitus. TEN test demonstrated dead regions in the cochlea in 15% of the tinnitus ears only. This might be attributed to increased resistance of IHCs to damage compared to OHCs vulnerability.

A study done by Konadath and Puttabasappa (2016) has studied cochlear functioning in normal hearing individuals with tinnitus and without tinnitus. A total of 40 individuals participated in the study in which 20 individuals had tinnitus with normal hearing sensitivity and 20 individuals with normal hearing. SWPTC and extended high frequency audiometry were carried out on these individuals. The results of SWPTC showed a change in tip frequency at 4000 and 6000 Hz. The result of extended high frequency audiometry showed significant elevation in threshold in individuals with tinnitus compare to individuals without tinnitus. The overall study indicated that, the presence of hidden hearing loss in individuals with tinnitus which originates at the level of basilar membrane itself; more likely to be damage associated with IHC at high frequency region.

Chapter 3

Method

The present study tested the null hypothesis which states that 'there is no significant difference in the result of PTC, TEN test and ECochG test in individuals with sensorineural hearing loss, with tinnitus and without tinnitus'. To test the hypothesis SWPTC, TEN test and ECochG were used in individuals in the same target group. The results of these tests were further analysed to assess the functioning of IHCs and OHCs. The following method was used in the study to test the hypothesis.

3.1. Selection of participants.

The study involved two groups of individuals with hearing impairment in the age range of 18 to 45 years. Group 1 consist of 15 individuals having sensorineural hearing loss without tinnitus and Group 2 consists of 15 individuals having sensorineural hearing loss with tinnitus. Each group were further divided into 3 sub-groups depending on degree of hearing loss in to minimal, mild and moderate (5 participants in each sub-group). All participants gave signed consent form before testing, which specifies their willingness to take part in the study. The following inclusion and exclusion criteria were used for participant selection in the study.

3.1.1. Inclusion criteria. The participants who fulfilled the following criterion was included in the study.

Group 1

- Sensorineural hearing loss of either minimal, mild or moderate degree and having flat audiometric configuration and
- Individual with SIS of 70% and above.

Group 2

- Apart from the criteria governing intake for participants selected in Group 1, all individuals in Group 2 were required to have a score of moderate and above in Tinnitus Handicap Inventory (THI), a questionnaire to assess the individual's reaction to tinnitus (Newman, Jacobson, & Spitzer 1996).

3.1.2. Exclusion criteria. Participants who are presented with one or more of the following characteristics were excluded from the study:

Group 1

- Any history or presence of middle ear disorders and
- Presence of retrocochlear pathology.

Group 2

- Any somatosensory or other conditions those are typically associated with tinnitus (vestibular schwannoma or Meniere's Diseases) and
- Any history or presence of psychological problems.

Table 3.1 shows the pure- tone average obtained by averaging 500, 1000, 2000 and 4000 Hz for individuals having SNHL without tinnitus (Group 1) and with tinnitus (Group 2). Based on the pure-tone average, the group were further sub-categorised as minimal, mild and moderate.

Table 3.1.
The pure-tone average in dBHL which is averaged across 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz of participants in Group 1 and Group 2.

Minimal SNHL		Mild SNHL		Moderate SNHL	
Group 1	Group 2	Group 1	Group 2	Group 1	Group 2
20.00	23.75	36.25	27.50	52.50	47.50
22.50	18.75	33.75	29.68	55.00	53.75
21.25	22.50	31.25	26.25	53.75	41.25
18.75	20.00	27.50	28.50	52.50	52.40
23.75	18.75	32.50	27.50	45.00	42.50

3.2. Instrumentation.

A calibrated dual channel audiometer along with TDH-39 headphone mounted in a supra-aural MX-41/AR cushion was used to obtain air conduction threshold; whereas Radio ear B-71 was used to obtain bone conduction threshold. Grason-Stadler GSI Tympstar middle ear analyser was used for evaluation of middle ear status and to obtain acoustic reflex threshold at 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz. PTC is administered using SWPTC software (version 1.4.50.1) installed in personal computer and TEN test was administered through TEN(HL) CD (Moore, 2014). To record ECochG, Biologic navigator pro AEP (version 7.2.1) was used.

3.3. Test environment.

All tests were carried out in acoustical treated audiometric room where the ambient noise level were within the permissible limits as specified by ANSI S3.1 (1999).

3.4. Procedure.

3.4.1. Routine evaluation. Pure tone threshold was obtained using calibrated dual channel audiometer using modified Hughson and Westlake procedure (Carhart & Jerger, 1959). This was carried out across frequencies ranging from 250 Hz to 8000 Hz for air conduction thresholds and 250 Hz to 4000 Hz for bone conduction thresholds. Based on four frequency pure tone average the individuals were categorized into three sub-groups having minimal, mild and moderate hearing loss.

- The hearing threshold of >15 dB, but <25 dB is considered as minimal,
- The hearing threshold of >26 dB, but <40 dB is considered as mild and
- The hearing threshold of >41 dB but <55 dB is considered as moderate (Clark, 1981).

Also, only those individuals who are having the difference between air conduction threshold and bone conduction threshold less than 10 dB were included in the study. Speech recognition threshold were obtained by using Kannada paired words (Rajashekar, 1976). Speech Identification Scores (SIS) was obtained using the PB word lists in Kannada language developed by Yathiraj and Vijayalakshmi (2005). Tympanometry and Acoustic reflex using 226 Hz probe tone at 500 Hz, 1000 Hz, 2000 Hz & 4000 Hz were assessed. Based on the results of the above tests, those participants who satisfy the selection criteria were included for the study.

All the individuals with continuous tinnitus were given a Tinnitus Handicap Inventory (THI), a questionnaire which consist of 25 questions were sub classified under 3 divisions, namely functional, emotional and catastrophic reactions to tinnitus. Basically, questions assess difficulties experienced by the individuals with tinnitus. Each question was scored in 3 levels: yes, sometimes and no. Individuals with a score of moderate and above were selected for the study.

3.4.2. Software Psychophysical tuning curves (SWPTC). The software PTC (SWPTC, version 1.4.50.1) was installed in personal computer fitted with soundcard and output was delivered through TDH 39 head phone. Before starting testing, the software was calibrated to ensure correct amount of sound level being delivered by the system.

The probe signal used was a pulsed and fixed in frequency. The same was presented at an intensity of 10 dB above the absolute threshold at 500 to 4000 Hz in mid octave step. The signal duration at each frequency was maintained at 0.2 second; with an interval of 0.2 second between the pulses. The noise used for masking was swept in forward sweeping manner with a rate of change of 2dB/s. The initial noise level for the test was set at 50 dB SPL and this level was kept constant across all the test frequencies. The participants were instructed to press and hold the space bar in keyboard as long as the tone is heard and to leave the key once the tone becomes inaudible. The participants were also instructed to ignore the noise and only concentrate on tone and then respond to only tone.

3.4.3. TEN Test. For the administration of TEN test the unmasked pure-tone thresholds were obtained through routine audiological examination and TEN masked threshold were obtained through TEN CD which contains special masking noise called TEN noise (Threshold Equalizing Noise). For conducting TEN test, right and left output from the computer was connected to the right and left input socket of audiometer respectively. The Track 1 contained calibrated tone which was used to calibrate output from audiometer. Later, the tracks from both the channel was mixed and presented to the same ear such that both TEN noise and warble tone are delivered to same ear.

The test frequencies consist of 500, 750, 1000, 1500, 2000, 3000 and 4000 Hz. The TEN levels were specified as the level of a one ERBn (equivalent rectangular bandwidth) wideband centred at 1000 Hz (Glasberg & Moore, 1990; Moor, 2004). The level of the signal and the TEN was controlled by using attenuator in the audiometer. The TEN masking noise was always kept constant at 70 dBHL (Vinay & Moore, 2007). The signal level was varied in 2 dB steps to determine the threshold (Moore, Glasberg, & stone, 2004). A 'no response' was indicated if subject did not respond for maximum output level of the audiometer.

3.4.4. Electrocochleography. ECochG was done using a single channel recording. Initially skin was prepared for electrode placement by using skin preparation gel and subject was made to relax on an inclining chair. Tip- trode electrode was used to record ECochG. The impedance of the each electrode was within 5 kΩ and between electrodes was 2 kΩ. The protocol used to record ECochG is given in table 3.2

Table 3.2.

Stimulus and acquisition parameters for recording ECochG

Stimulus parameters	
Transducer type	ER-3A Insert headphone
Type of stimulus	Click
Intensity	80dBnHL
Stimulus polarity	Rarefaction
Stimulus rate	7.1/s
Acquisition parameters	
Analysis time	10ms
gain	10000
Filter setting	10-3000 Hz
No of sweep	1500
Electrode montage	Inverting (-) = Non test ear Mastoid (M_1/M_2) Non inverting (+) = Ear canal (A_1/A_2) Ground = Forehead (Fz)

3.5. Analysis of responses.

3.5.1 Analysis of SWPTC. The Q10 values were analysed; i.e. the ratio of central frequency to the band width measured 10 dB above the lowest point on the tuning cure. Tip frequency was measured to assess the function of IHC. The software itself offers different method to estimate tip frequency and sharpness of PTC. One of such method, two point moving average was used to estimate tip frequency and sharpness of PTC which is defined as Q10 value. The moving average was obtained by smoothing the data by two- point and the frequency corresponding to the minimum of the moving average was taken as the tip frequency.

3.5.2. Analysis of TEN test. For identifying cochlear dead region through TEN test, the following two criteria were considered and the individuals who met both the criteria were considered as having cochlear dead region.

- Firstly, the masked threshold in the TEN should be 10 dB or more above the TEN level/ERBN,
- Secondly, the masked threshold in the TEN should be 10 dB or above the absolute threshold or unmasked threshold**.**

3.5.3. Analysis of waveform of EcochG. The latency and the amplitude of cochlear microphonics were measured by using rarefaction stimuli. The waveforms were analysed subjectively. The waveforms recording were given to the two qualified audiologists for the analysis of parameters. If there was agreement between both the audiologists, then only the waveform were taken for further analysis.

3.6. Statistical analyses.

The data obtained was tabulated using Software packages for Statistical Analysis (SPSS, Version 21.0). Appropriate statistical analysis was carried out in SPSS software.

Chapter 4

Results

The current study aimed to compare the cochlear functioning in individual having sensorineural hearing loss with and without tinnitus. This was achieved through following objectives.

- To compare findings of PTC, ECochG and TEN test in individual having sensorineural hearing loss without (Group 1) and with tinnitus (Group 2) across different degree of hearing loss.
- To compare findings of PTC, ECochG and TEN test in individual having sensorineural hearing loss with tinnitus (Group 2) across different degrees of hearing loss.

To analyse the result, following statistics were carried out:

- Shapiro Wilk test was performed to check whether all the data points are following normal distribution. It was found that the data points were not following normal distribution ($p>0.05$) for any parameter. Thus non-parametric tests were performed to analyse the data.
- Descriptive statistics were carried out to find the median and range values for all parameters.
- Mann Whitney U test was carried out between Group 1 and Group 2 across different degree of hearing loss to look for any difference in test results obtained.
- Kruskal Wallis test was performed to see, if there are any differences in all parameter of Group 1 across different degrees of hearing loss. Also, the same

was performed to check for the difference in results across various degree of hearing loss in Group 2.

4.1. PTC, ECochG and TEN test results in Group 1 and Group 2.

Under this section, which address the first objective of the study, includes results of PTC (Q10 and tip frequencies), TEN masked thresholds and cochlear microphonic (latencies and amplitude). Results were compared between individuals having sensorineural hearing loss without tinnitus (Group 1) and individuals with sensorineural hearing loss with tinnitus (Group 2) across minimal, mild and moderate degrees of hearing loss. The results are subcategorised based on degree of hearing loss.

4.1.1. Minimal hearing loss. The results of PTCs, TEN test and ECochG between Group 1 and Group 2 are discussed under the following headings.

4.1.1.1. Psychophysical tuning curves.

a) Q10 values. Descriptive statistics was carried out to find the median and range of Q10 values in individuals having minimal SNHL without tinnitus (Group 1) and with tinnitus (Group 2). It was found that most of the individuals in Group 1 had higher Q10 values compare to Group 2. Higher value indicates better frequency resolution capability of outer hair cells. Median and range values are given in Table 4.1. It can be seen from the Figure 4.1, that the median for Q10 of PTCs are higher (in most of the cases) for Group 1 than Group 2.

Table 4.1.

Median and range for Q10 values of PTC for Group 1 and Group 2

	Median (dB)		Minimum (dB)		Maximum (dB)	
	Group 1	Group 2	Group 1	Group 2	Group 1	Group 2
500 Hz	4.00 (5)	3.00 (5)	3.00	2.00	6.00	4.00
1000 Hz	4.00 (5)	3.00 (5)	3.00	3.00	6.00	4.00
1500 Hz	4.00 (4)	4.00 (5)	4.00	3.00	4.00	4.00
2000 Hz	4.00 (5)	3.00 (5)	3.00	3.00	5.00	4.00
3000 Hz	3.00 (5)	4.00 (3)	3.00	2.00	6.00	5.00
4000 Hz	3.00 (3)	2.00 (3)	3.00	2.00	5.00	2.00

Note: N given in parenthesis

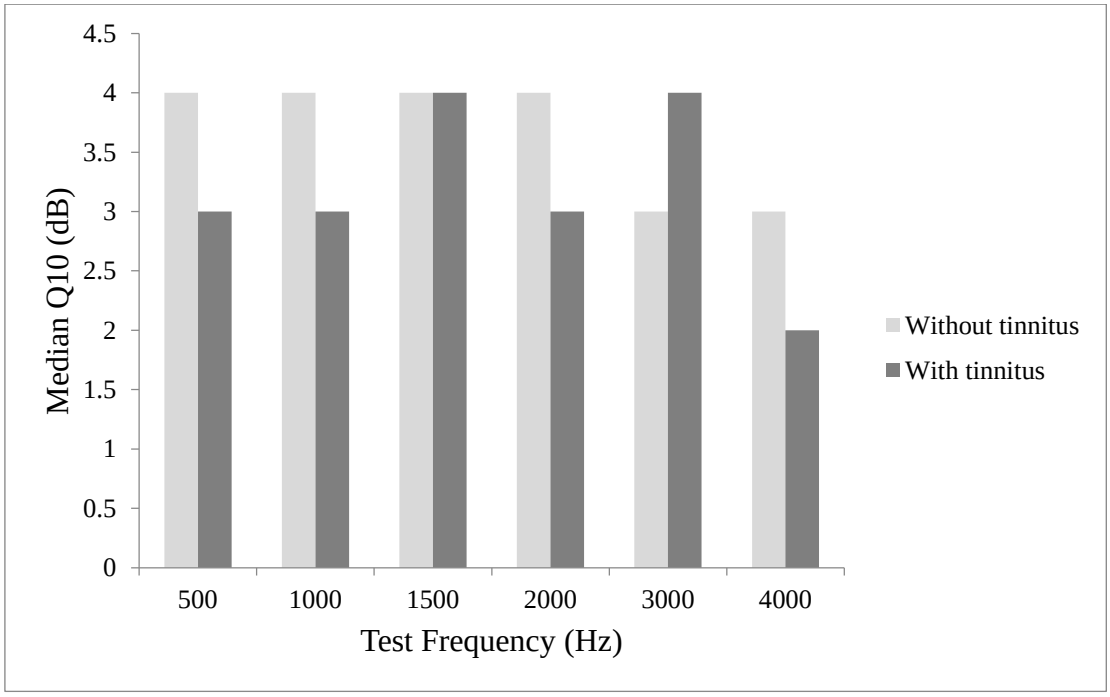

Figure 4.1. Median for Q10 values of PTC for Group 1 and Group 2.

To compare results of Q10 values of PTC in minimal SNHL with and without tinnitus Mann Whitney U test was carried out. Results showed no significant difference in Q10 value between two groups ($p>0.05$), except at 4000 Hz ($p<0.05$). The p and Z value obtained in the Mann Whitney u test is given in Table 4.2.

Table 4.2.

/Z/ value and level of significance obtained on Mann Whitney U test for Q10 comparison

Q10 (Hz)	500	1000	1500	2000	3000	4000
/Z/	1.643	1.417	1.352	1.315	0.461	2.121
Level of significance	p>0.05	p>0.05	p>0.05	p>0.05	p>0.05	p<0.05

b) Tip frequencies. Descriptive statistics was carried out to find the median and range for tip frequencies. It was found that median was similar between both groups across all frequencies. The median and range for tip frequencies of PTC for Group 1 and group 2 are shown in the table 4.3.

Table 4.3.

Median and range for tip frequencies of PTC for Group 1 and Group 2

Frequencies (Hz)	Median (Hz)		Minimum (Hz)		Maximum (Hz)	
	Group 1	Group 2	Group 1	Group 2	Group 1	Group 2
500	513 (5)	549 (5)	498	496	556	598
1000	1094 (5)	1062 (5)	963	1015	1260	1164
1500	1513 (4)	1642 (5)	1487	1064	1605	1756
2000	2075 (5)	2088 (5)	1905	1942	2643	2134
3000	3172 (5)	3033 (3)	3091	3011	3528	3784
4000	3960 (3)	4147 (3)	3704	3981	4178	5358

Note: N given in parenthesis

To compare results of tip frequencies in individuals with minimal SNHL with and without tinnitus, Mann Whitney U test was carried out. Results showed no

significant difference in tip frequency between two groups (p>0.05) Table 4.4. shows the /Z/ value and level of significance obtained on Mann Whitney U test for comparison of tip frequency.

Table 4.4.

/Z/ value and level of significance obtained on Mann Whitney U test for comparison of tip frequency

Tip frequency (Hz)	500	1000	1500	2000	3000	4000
/Z/	0.313	0.104	1.225	0.104	0.745	1.091
Level of significance	p>0.05	p>0.05	p>0.05	p>0.05	p>0.05	p>0.05

4.1.1.2. TEN test. Descriptive statistics was carried out to find the median and range for TEN masked threshold in individuals having minimal SNHL without tinnitus (Group 1) and with tinnitus (Group 2). It was found that median was similar between both groups across all frequencies. The median and range for TEN masked threshold for Group 1 and group 2 are shown in the table 4.5.

Table 4.5.

The median and range for TEN masked thresholds for Group 1 and group 2

Frequencies (Hz)	Median (dB)		Minimum (dB)		Maximum (dB)	
	Group 1	Group 2	Group 1	Group 2	Group 1	Group 2
500	4.00 (5)	4.00 (5)	2.00	4.00	4.00	12.00
750	4.00 (5)	4.00 (5)	4.00	4.00	6.00	6.00
1000	6.00 (5)	6.00 (5)	4.00	4.00	6.00	8.00
1500	4.00 (5)	4.00 (5)	2.00	4.00	4.00	6.00
2000	4.00 (5)	6.00 (5)	2.00	4.00	6.00	8.00
3000	4.00 (5)	4.00 (5)	2.00	2.00	6.00	6.00
4000	4.00 (5)	8.00 (5)	4.00	6.00	8.00	10.00

Note: N given in parenthesis

To compare results of TEN test in individuals with minimal SNHL Mann Whitney U test was carried out. The TEN masked thresholds were subjected to Mann Whitney U test to see the difference between Group 1 and Group 2. Results showed no significant difference in TEN masked threshold between two groups ($p>0.05$). The /Z/ and p values obtained on Mann Whitney U test for comparison TEN masked threshold is given in Table 4.6.

Table 4.6.

/Z/ value and level of significance obtained on Mann Whitney U test for comparison of TEN masked threshold

Frequencies (Hz)	500	750	1000	1500	2000	3000	4000
/Z/	1.342	0.655	0.949	1.678	1.643	0.454	1.844
Level of significances	p>0.05	p>0.05	p>0.05	p>0.05	p>0.05	p>0.05	p>0.05

4.1.1.3. EcochG. Descriptive statistics was carried out to find the median and range for latency and amplitude of cochlear microphonics in individuals having minimal SNHL without tinnitus (Group 1) and with tinnitus (Group 2). It was found that median was similar between both groups for latency of cochlear microphonics and amplitude was higher for Group 2 (SNHL without tinnitus). The median and range for latency and amplitude of cochlear microphonics for Group 1 and Group 2 are shown in the Table 4.7. The median for CM latency and amplitude are depicted in the Figure 4.2. and 4.3 respectively. It can be observed from the figure the median of

CM amplitude is higher for the individuals without tinnitus (Group 1) than with tinnitus (Group 2).

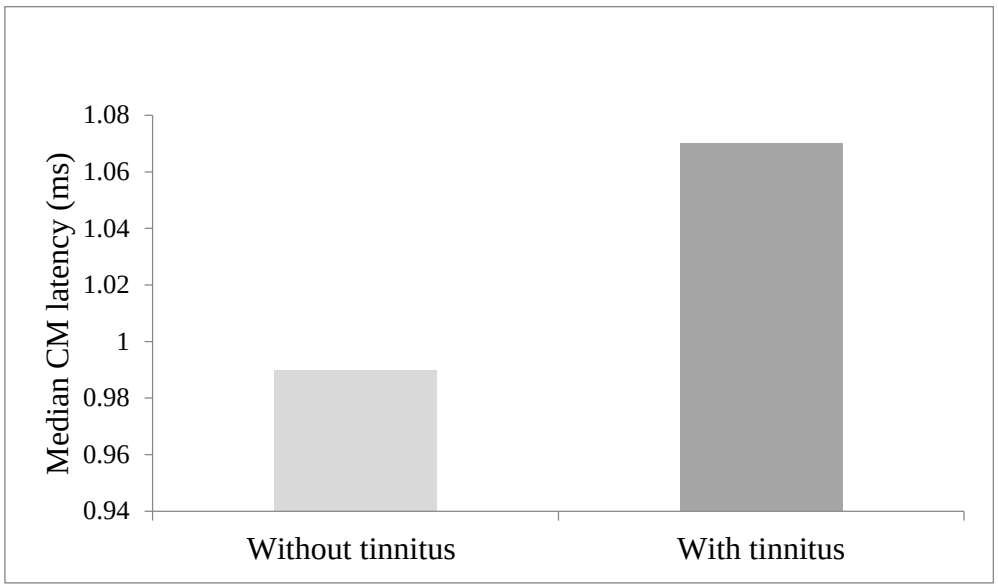

Figure 4.2. Median for latency of cochlear microphonics for Group 1 and Group 2.

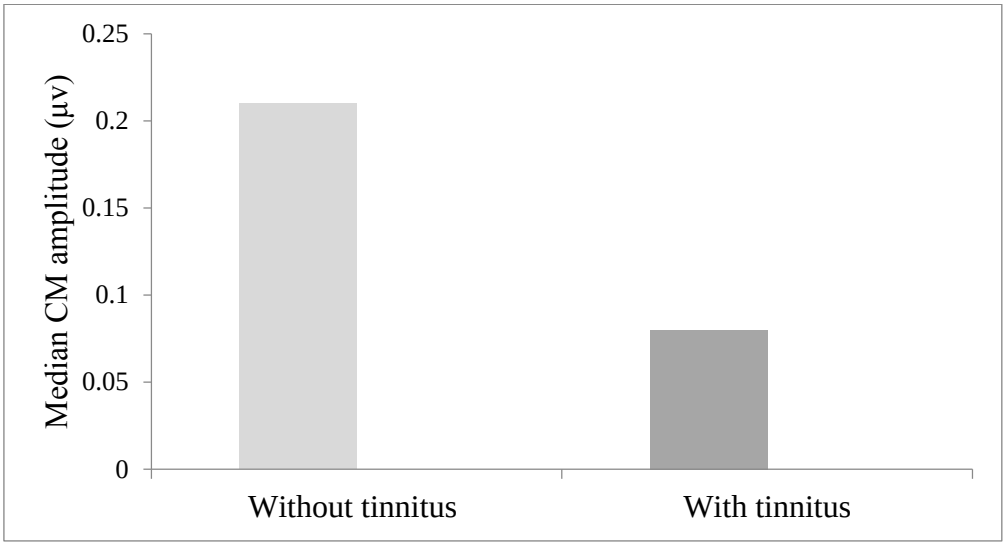

Figure 4.3. Median for amplitude of cochlear microphonics for Group 1 and Group 2.

Table 4.7.

The median and range for latency and amplitude of cochlear microphonics for Group 1 and group 2

	Median		Minimum		Maximum	
	Group 1	Group 2	Group 1	Group 2	Group 1	Group 2
CM latency (ms)	0.99 (3)	1.07 (5)	0.89	0.99	1.07	1.28
CM amplitude (μv)	0.21(3)	0.08 (5)	0.19	0.01	0.41	0.18

Note: N given in parenthesis

Following descriptive statistics, the cochlear microphonics latency and amplitude was analysed using Mann Whitney U test to study for any difference between Group 1 and Group 2. Results showed no significant difference in cochlear microphonics latency between two group ($p>0.05$), whereas significant difference was found in CM amplitude between two group ($p<0.05$). The /Z/ and p value obtained on Mann Whitney U test for comparison of CM latency and amplitude is given in Table 4.8.

Table 4.8.
/Z/ value and level of significance obtained on Mann Whitney U test for comparison of CM latency and amplitude

	Latency	Amplitude
/Z/	1.439	2.236
Level of significances	P>0.05	P<0.05

4.1.2. Mild hearing loss

The results of PTCs, TEN test and ECochG between Group 1 and Group 2 are discussed under the following headings.

4.1.2.1. Psychophysical tuning curves:

a) Q10 values: Descriptive statistics was carried out to find the median and range of all parameter in individuals having mild SNHL without tinnitus (Group 1) and with tinnitus (Group 2). Median and range values are given in Table 4.9. It can be seen from the figure, the median for the Q10 at 3000 Hz was higher in Group 1 than Group 2.

Table: 4.9.

Median and range for Q10 values of PTC for Group 1 and Group 2

Frequencies	Median (dB)		Minimum (dB)		Maximum (dB)	
(Hz)	Group 1	Group 2	Group 1	Group 2	Group 1	Group 2
500	4.00 (5)	1.50 (4)	2.00	1.00	6.00	4.00
1000	3.00 (3)	3.00 (4)	3.00	2.00	4.00	4.00
1500	3.00 (4)	3.00 (3)	2.00	2.00	4.00	3.00
2000	5.00 (3)	4.00 (3)	4.00	2.00	5.00	4.00
3000	4.00 (3)	2.00 (3)	4.00	2.00	5.00	3.00
4000	2.00 (3)	4.00 (1)	1.00	4.00	2.00	4.00

Note: N given in parenthesis

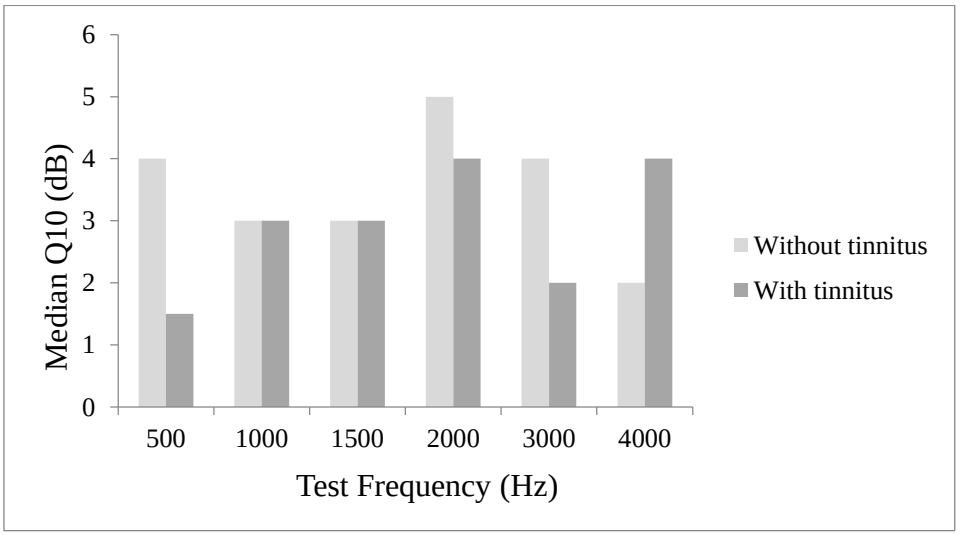

Figure 4.4. Median for Q10 values of PTC for Group 1 and Group 2

To compare results of Q10 values of PTC in mild SNHL with and without tinnitus a Mann Whitney U test was carried out. Results showed no significant difference in Q10 value between two groups ($p>0.05$) except at Q10 value of 3000 Hz ($p<0.05$). Table 4.10. shows the /Z/ value and level of significance obtained on Mann Whitney U test for comparison of Q10 value.

Table 4.10.

/Z/ value and level of significance obtained on Mann Whitney U test for Q10 comparison

Q10 (Hz)	500	1000	1500	2000	3000	4000
/Z/	1.634	0.592	0.592	1.650	2.023	1.414
Level of significances	p>0.05	p>0.05	p>0.05	p>0.05	p<0.05	p>0.05

b) Tip frequency: Descriptive statistics was carried out to find the median and range for tip frequencies. It was found that median was similar between both groups across all frequencies. The median and range for tip frequencies of PTC for Group 1 and group 2 are shown in the Table 4.11.

Table 4.11.

Median and range for tip frequencies of PTC for Group 1 and Group 2

Frequencies(Hz)	Median (dB)		Minimum (dB)		Maximum (dB)	
	Group 1	Group 2	Group 1	Group 2	Group 1	Group 2
500	563 (5)	474 (4)	473	406	592	520
1000	1040 (3)	1109 (4)	1033	1041	1100	1118
1500	1518 (4)	1637 (3)	1484	1553	1578	1740
2000	2143 (3)	2168 (3)	2117	2117	2143	2189

3000	3194 (3)	2886 (3)	3046	2763	3316	3398
4000	3755 (3)	3188 (1)	3729	3188	4517	3188

Note: N given in parenthesis

Mann Whitney U test was carried out to compare results of tip frequencies of PTC in mild SNHL with and without tinnitus. Results showed no significant difference in tip frequency between two groups ($p>0.05$). Table 4.12. shows the /Z/ value and level of significance obtained on Mann Whitney U test for comparison of tip frequency.

Table 4.12.

/Z/ value and level of significance obtained on Mann Whitney U test for comparison of tip frequency.

Tip frequency (Hz)	500	1000	1500	2000	3000	4000
/Z/	1.476	1.768	1.768	0.899	0.655	1.342
Level of significance	p>0.05	p>0.05	p>0.05	p>0.05	p>0.05	p>0.05

4.1.2.2. TEN test: Descriptive statistics was carried out to find the median and range for TEN masked threshold in individuals having mild SNHL without tinnitus (Group 1) and with tinnitus (Group 2). It was found that median was similar between both groups across all frequencies. The median and range for TEN masked threshold for Group 1 and group 2 are shown in the Table 4.13.

Table 4.13.

The median and range for TEN masked thresholds for Group 1 and group 2

frequencies	Median (dB)		Minimum (dB)		Maximum (dB)	
(Hz)	Group 1	Group 2	Group 1	Group 2	Group 1	Group 2
500	6.00 (5)	4.00 (5)	4.00	4.00	8.00	10.00

750	4.00 (5)	4.00 (5)	4.00	2.00	6.00	8.00
1000	6.00 (5)	6.00 (5)	4.00	2.00	8.00	8.00
1500	4.00 (5)	6.00 (5)	4.00	2.00	6.00	8.00
2000	4.00 (5)	4.00 (5)	4.00	2.00	4.00	4.00
3000	6.00 (5)	4.00 (5)	4.00	4.00	8.00	8.00
4000	8.00 (5)	8.00 (5)	4.00	6.00	8.00	10.00

Note: N given in parenthesis

To compare the results of TEN test in individual with mild SNHL a Mann Whitney U test was carried. Results showed no significant difference in TEN masked threshold between two groups ($p>0.05$). The /Z/ and p values obtained on Mann Whitney U test for comparison TEN masked threshold is given in Table 4.14.

Table 4.14.

/Z/ value and level of significance obtained on Mann Whitney U test for comparison of TEN masked threshold

Frequencies (Hz)	500	750	1000	1500	2000	3000	4000
/Z/	-0.339	-0.112	-0.108	-1.337	-1.000	-1.474	-1.459
Level of significances	p>0.05	p>0.05	p>0.05	p>0.05	p>0.05	p>0.05	p>0.05

4.1.2.3. ECochG: Descriptive statistics was carried out to find the median and range for latency and amplitude of cochlear microphonics in individuals having mild SNHL. It was found that median was similar between both groups for latency and amplitude of cochlear microphonics. The median and range for latency and amplitude of cochlear microphonics for Group 1 and Group 2 are shown in the Table 4.15.

Table 4.15.

The median and range for latency and amplitude of cochlear microphonics for Group 1 and group 2

CM	Median		Minimum		Maximum	
	Group 1	Group 2	Group 1	Group 2	Group 1	Group 2
Latency (ms)	1.03 (5)	1.07 (5)	0.86	0.66	1.16	1.16
Amplitude (μv)	0.21 (5)	0.22 (5)	0.12	0.16	0.38	0.27

Note: N given in parenthesis

To compare results of ECochG results in mild with and without tinnitus a Mann Whitney U test was carried out. The cochlear microphonics latency and amplitude were analysed using Mann Whitney U to look for any difference between Group 1 and Group 2. Results showed no significant difference in cochlear microphonics latency and amplitude between two groups ($p>0.05$). The /Z/ and p value obtained on Mann Whitney U test for comparison of CM latency and amplitude is given in Table 4.16.

Table 4.16.

/Z/ value and level of significance obtained on Mann Whitney U test for comparison of CM latency and amplitude

	Latency	Amplitude
/Z/	0.316	0.104
Level of significances	P>0.05	P>0.05

4.1.3. Moderate hearing loss.

The results of PTCs, TEN test and ECochG between Group 1 and Group 2 are discussed under the following headings.

4.1.3.1. Psychophysical tuning curves.

a) Q10 values. Descriptive statistics was carried out to find the median and range of Q10 values in individuals having moderate SNHL without tinnitus (Group 1) and with tinnitus (Group 2). The Q10 values could not obtained in most of the frequencies in individuals having SNHL without tinnitus (Group 1). Median and range values are given in Table 4.17.

Table 4.17.

Median and range for Q10 values of PTC for Group 1 and Group 2

Frequencies	Median (dB)		Minimum (dB)		Maximum (dB)	
(Hz)	Group 1	Group 2	Group 1	Group 2	Group 1	Group 2
500	- (0)	4.00 (1)	-	4.00	-	4.00
1000	- (0)	3.50 (2)	-	3.00	-	4.00
1500	- (0)	3.00 (3)	-	3.00	-	3.00
2000	- (0)	3.50 (2)	-	3.00	-	4.00
3000	2.00 (1)	3.50 (4)	2.00	2.00	2.00	6.00
4000	1.00 (1)	2.00 (3)	1.00	2.00	1.00	3.00

Note: N given in parenthesis

To compare results Q10 values of PTC in moderate SNHL with and without tinnitus, a Mann Whitney U test was carried out. In many of the individuals with moderate hearing, Q10 value could not be obtained for the frequency 500 Hz, 1000 Hz, 1500 Hz and 2000 Hz, therefore Mann Whitney U test could not be performed for these parameters. The Q10 value for the frequency 3000 Hz and 4000 Hz were analysed to see the difference between Group 1 and Group 2. Results showed no significant difference in Q10 value between two groups ($p>0.05$). The p and /Z/ value obtained from the Mann Whitney u test is given in Table 4.18.

Table 4.18.

/Z/ value and level of significance obtained on Mann Whitney U test for comparison of Q10 values.

Q10 (Hz)	3000 Hz	4000 Hz
/Z/	1.088	1.414
Level of significance	p>0.05	p>0.05

b) Tip frequencies. Descriptive statistics was carried out to find the median and range for tip frequencies. The tip frequency could not obtained in most of the frequencies in individuals having SNHL without tinnitus (Group 1). Median and range values are given in table 4.19.

Table 4.19.

Median and range for tip frequencies of PTC for Group 1 and Group 2

Frequencies	Median (dB)		Minimum (dB)		Maximum (dB)	
(Hz)	Group 1	Group 2	Group 1	Group 2	Group 1	Group 2
500	- (0)	499 (1)	-	499	-	499
1000	- (0)	1006 (2)	-	953	-	1059
1500	- (0)	1516 (3)	-	1428	-	1903
2000	- (0)	1964 (2)	-	1886	-	2041
3000	3066 (1)	3142 (4)	3066	2936	3066	3592
4000	3364 (1)	3563 (3)	3364	3306	3364	4720

Note: N given in parenthesis

To compare results of tip frequencies of PTC in moderate SNHL with and without tinnitus, a Mann Whitney U test was carried out. In many of the individuals with moderate hearing, tip frequency could not be obtained for the frequency 500 Hz, 1000 Hz, 1500 Hz and 2000 Hz, therefore Mann Whitney U test could not be

performed for few parameters. In those frequencies where comparisons were possible, results showed no significant difference in tip frequencies between two groups ($p>0.05$). Table 4.20. shows the /Z/ value and level of significance obtained on Mann Whitney U test for comparison of tip frequency.

Table 4.20.

/Z/ value and level of significance obtained on Mann Whitney U test for comparison of tip frequency

Tip frequency (Hz)	3000	4000
/Z/	0.707	0.447
Level of significance	p>0.05	p>0.05

4.1.3.2. TEN test. Descriptive statistics was carried out to find the median and range for TEN masked threshold in individuals having moderate SNHL without tinnitus (Group 1) and with tinnitus (Group 2). It was found that median was similar between both groups across all frequencies. The median and range for TEN masked threshold for Group 1 and group 2 are shown in the table 4.21. It can be seen from the figure 4.5, median of TEN masked threshold was greater Group 1 compare to Group 2.

Table 4.21.

The median and range for TEN masked thresholds for Group 1 and group 2

Frequencies (Hz)	Median (dB)		Minimum (dB)		Maximum (dB)	
	Group 1	Group 2	Group 1	Group 2	Group 1	Group 2
500	4.00 (5)	8.00 (5)	4.00	6.00	10.00	10.00
750	6.00 (5)	6.00 (5)	4.00	4.00	6.00	8.00
1000	6.00 (5)	6.00 (5)	2.00	4.00	8.00	6.00
1500	8.00 (5)	4.00 (5)	6.00	2.00	8.00	10.00

2000	8.00 (5)	4.00 (5)	6.00	4.00	10.00	6.00
3000	8.00 (5)	6.00 (5)	6.00	4.00	12.00	10.00
4000	12.00 (5)	8.00 (5)	8.00	6.00	12.00	12.00

Note: N given in parenthesis

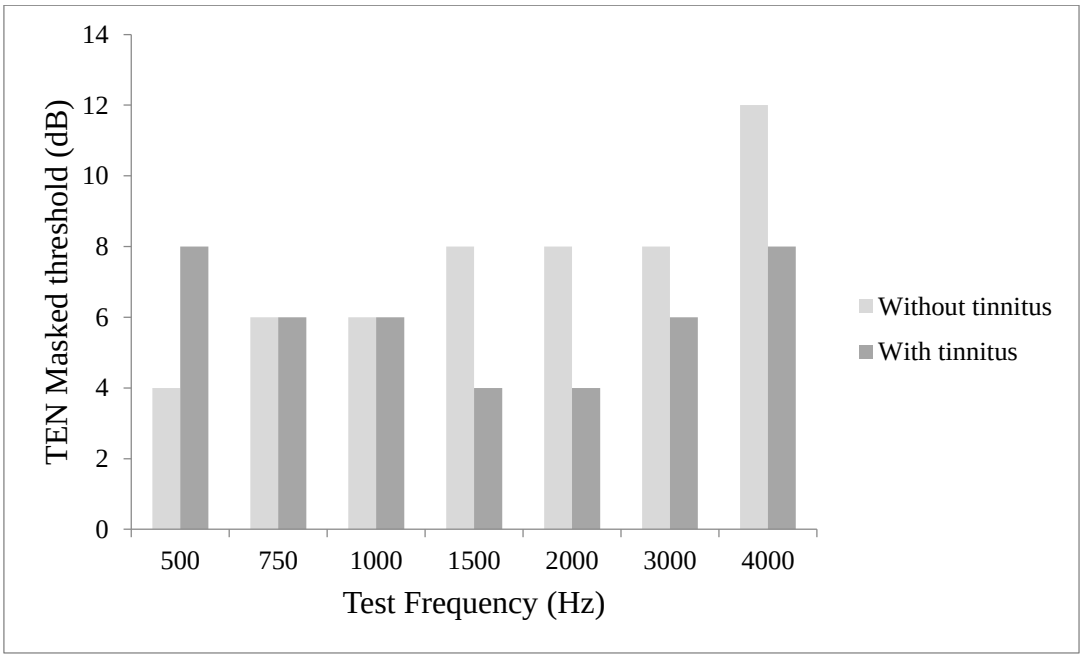

Figure 4.5. The median for TEN masked thresholds for Group 1 and group 2.

To compare the results of TEN test in individuals with moderate SNHL, Mann Whitney U test was carried out. Results showed no significant difference in TEN masked threshold between two groups ($p>0.05$), except for Ten 2000 Hz ($p<0.05$). The /Z/ and p values obtained on Mann Whitney U test for comparison TEN masked threshold is given in Table 4.22.

Table 4.22.

/Z/ value and level of significance obtained on Mann Whitney U test for comparison of TEN masked threshold.

Frequencies (Hz)	500	750	1000	1500	2000	3000	4000
/Z/	1.078	0.346	0.438	1.611	2.479	1.708	1.017
Level of significances	p>0.05	p>0.05	p>0.05	p>0.05	p<0.05	p>0.05	p>0.05

4.1.3.3. EcochG. Descriptive statistics was carried out to find the median and range for latency and amplitude of cochlear microphonics in individuals having moderate SNHL without tinnitus (Group 1) and with tinnitus (Group 2). It was found that median was similar between both groups for latency and amplitude of cochlear microphonics. The median and range for latency and amplitude of cochlear microphonics for Group 1 and Group 2 are shown in the Table 4.23.

Table 4.23.

The median and range for latency and amplitude of cochlear microphonics for Group 1 and group 2

	Median		Minimum		Maximum	
	Group 1	Group 2	Group 1	Group 2	Group 1	Group 2
CM latency(ms)	1.11 (1)	0.99 (4)	1.11	0.81	1.11	1.16
CM amplitude(μv)	0.29 (1)	0.15 (4)	0.29	0.06	0.29	0.31

Note: N given in parenthesis

To compare results of ECochG results in moderate SNHL with and without tinnitus, Mann Whitney U test was carried out. The latency and amplitude of cochlear microphonics were analysed by using Mann Whitney U to study the difference between Group 1 and Group 2. Results showed no significant difference in cochlear

microphonics latency and amplitude (p>0.05). The /Z/ and p value obtained on Mann Whitney U test for comparison of CM latency and amplitude is given in Table 4.24.

Table 4.24.

/Z/ value and level of significance obtained on Mann Whitney U test for comparison of CM latency and amplitude

	Latency	Amplitude
/Z/	0.725	0.707
Level of significances	P>0.05	P>0.05

4.2. PTC, ECochG and TEN test results in individual with tinnitus.

To study the difference in PTC, ECochG and TEN test results across different degrees of hearing loss (objective 2 of the study), Kruskal Wallis test was performed and result revealed no significant difference found across different degree of hearing loss in SNHL individuals with tinnitus. Table 4.25, 4.26, 4.27 and 4.28 indicates the Chi-Square values and level of significance obtained on Kruskal Wallis test for comparison of Q10 values of PTCs, tip frequency of PTC, TEN masked threshold and CM latency and amplitude respectively.

Table 4.25.

Chi-Square values and level of significance obtained on Kruskal Wallis *test for comparison of Q10 values*

Test Frequencies (Hz)	χ^2	p
500	2.613	p>0.05
1000	0.844	p>0.05
1500	4.917	p>0.05
2000	0.490	p>0.05

3000	2.017	p>0.05
4000	1.391	p>0.05

Table 4.26.

Chi-Square values and level of significance obtained on Kruskal Wallis *for comparison of tip frequencies*

Test frequencies (Hz)	χ^2	p
500	2.455	p>0.05
1000	2.223	p>0.05
1500	0.242	p>0.05
2000	3.545	p>0.05
3000	1.573	p>0.05
4000	3.143	p>0.05

Table 4.27.

Chi-Square values and level of significance obtained on Kruskal Wallis *test for comparison of TEN masked threshold*

Test frequencies (Hz)	χ^2	p
500	3.615	p>0.05
750	0.598	p>0.05
1000	1.248	p>0.05
1500	1.372	p>0.05
2000	4.827	p>0.05
3000	2.646	p>0.05
4000	1.191	p>0.05

Table 4.28.

Chi-Square values and level of significance obtained on Kruskal Wallis *test for comparison of CM latency and amplitude*

	χ^2	p
CM Latency	1.053	p>0.05
CM Amplitude	4.787	p>0.05

Since there was no difference found in Group 2 (SNHL with tinnitus) the Kruskal Wallis test was also performed to look for any difference in PTC, TEN test and ECochG in Group 1 (SNHL without tinnitus). It was found that there was no significant difference found in PTC and EcochG results obtained. Table 4.29, 4.30, 4.31 and 4.32 indicates the Chi-Square values and level of significance obtained on Kruskal Wallis test for comparison of Q10 values of PTCs, tip frequency of PTC, TEN masked threshold and CM latency and amplitude respectively.

Table 4.29.

Chi-Square values and level of significance obtained on Kruskal Wallis *test for comparison of Q10 values*

Test frequencies (Hz)	χ^2	p
500	.048	p>0.05
1000	.921	p>0.05
1500	4.000	p>0.05
2000	2.016	p>0.05
3000	2.695	p>0.05
4000	5.057	p>0.05

Table 4.30.

Chi-Square values and level of significance obtained on Kruskal Wallis *test for comparison of tip frequencies*

Test frequencies (Hz)	χ^2	p
500	0.538	p>0.05
1000	0.556	p>0.05
1500	0.190	p>0.05
2000	0.202	p>0.05
3000	1.440	p>0.05
4000	2.286	p>0.05

Table 4.31.

Chi-Square values and level of significance obtained on Kruskal Wallis *test for comparison of TEN masked threshold*

Test frequencies (Hz)	χ^2	p
500	4.341	p>0.05
750	2.240	p>0.05
1000	0.040	p>0.05
1500	10.937	P<0.05
2000	9.918	P<0.05
3000	7.687	P<0.05
4000	9.051	P<0.05

Table 4.32.

Chi-Square values and level of significance obtained on Kruskal Wallis *test for comparison of CM latency and amplitude*

	χ^2	p
CM Latency	1.383	p>0.05
CM Amplitude	0.284	p>0.05

Since majority of parameter did not showed significant difference between the subgroup, the data were combined except for TEN results. Further, Mann Whitney U test was performed to check for the significant difference between Group 1 and Group 2 after combining data. The Z and p value obtained from the Mann Whitney U test is given in table 4.33, 4.34 and 4.35 for comparison of Q10, tip frequency and ECochG respectively.

Table 4.33.

Z value and level of significance obtained on Mann Whitney U test for comparison of Q10

Test frequency	/Z/	Level of significance
500	1.995	P<0.05
1000	1.394	p>0.05
1500	1.185	p>0.05
2000	1.989	P<0.05
3000	1.130	p>0.05
4000	0.736	p>0.05

Table 4.34.

Z value and level of significance obtained on Mann Whitney U test for comparison of tip frequency

Test frequency	/Z/	Level of significance
500	1.059	p>0.05
1000	0.495	p>0.05
1500	1.612	p>0.05
2000	0.444	p>0.05
3000	0.898	p>0.05
4000	0.640	p>0.05

Table 4.35.

Z value and level of significance obtained on Mann Whitney U test for comparison of CM latency and amplitude

	Z	Level of significance
CM latency	0.510	p>0.05
CM amplitude	2.050	P<0.05

It was wound that there was a significant difference in Q10 value of 500 Hz, 2000 and CM amplitude between the groups indicating more pathophysiological changes at the level of OHCs compared to that of IHCs ($p<0.05$).

Summary of results: The results of the first objective revealed significant difference in Q10 values of PTC at 4000Hz and 3000Hz in minimal and mid hearing loss respectively and Q10 values did not show any difference in moderate hearing loss between individuals with and without tinnitus. Results of tip frequency of PTC showed no significant difference across degrees of hearing loss between individuals with and without tinnitus. TEN test also showed no difference in TEN masked

threshold in individuals with and without tinnitus across degrees of hearing loss except at 2000 Hz in moderate hearing loss.

The results of ECochG showed a significant difference in CM amplitude only in minimal hearing loss and CM latency showed no difference across degrees of hearing loss in individuals with and without tinnitus.

The results of second objective revealed no significant difference in PTC, TEN test and ECochG across degrees of hearing loss in individuals with tinnitus. Whereas, when the data were combined across degrees of hearing loss and comparison was made between the group, results showed significance difference in Q10 value at 500 Hz and 2000 Hz and CM amplitude irrespective of degrees of hearing loss.

Chapter 5

Discussion

The purpose of the present study is to compare the cochlear function in individual without and with tinnitus. The outcomes of the experiment were discussed in following headings.

5.1. PTC, ECochG and TEN test in individual having sensorineural hearing loss without and with tinnitus across different degree of hearing loss.

5.1.1. PTC result across different degrees of hearing loss.

a) Q10. On comparison of Q10 values in individuals with and without tinnitus across degrees of hearing loss, it was found that there was no significant difference between the group except at 4000 Hz in minimal and at 3000 Hz in individuals having mild degree of SNHL. But, it was seen that Q10 was lower for individuals with tinnitus compare to individual without tinnitus, which indicates poorer frequency resolution in OHC of individuals with tinnitus compared to those without tinnitus.

Literature shows, more of OHC dysfunction in individuals with tinnitus and IHC being intact. A study done by Shiomi, Tsuji, Naito, Fujiki and Yamamoto (1997) found significant decreases in DPOAE amplitude over a limited frequency range in DP- gram in individuals with tinnitus compared to individuals without tinnitus with normal hearing, also moderate correlation were found between DPOAE amplitudes and hearing levels. Reduction in DPOAE amplitude directly indicated OHC dysfunction in these individuals. Mitchell and Creedon (1995) also found irregularity in PTC curve in individuals with tinnitus and these irregularities included hypersensitive tail and elevated tips in individuals with tinnitus compare to

individuals without tinnitus indicating OHC dysfunction in individuals with tinnitus without IHC or nerve damage.

A study done by Zhou et al. (2011) reported that, subjects with tinnitus had elevated thresholds, reduced DPOAE, and increased slope of the DPOAE input-output function in high frequency region ranging from 4000 Hz to 10000 Hz. Also, elevation in the perceptual threshold correlated with the tinnitus rating and this was indicated reduced amplitude in DPOAE in those frequency regions, which suggest impaired cochlear functioning in individual with tinnitus. In the present study, it has been seen that individuals with tinnitus had lower Q10 values, which directly reflects the broadening of the auditory filter. To draw support for these findings; Dauman and Cazals (1989) indicated that frequency selectivity in individuals were abnormally affected. They could clearly identify broadening of frequency selectivity in individual with tinnitus having bilateral hearing loss and also they reported broadening was more in the ear with the tinnitus than the ear without tinnitus, which strongly suggests tinnitus originates in the cochlea and outer hair cell are site of generation for tinnitus.

There are contradicting studies which reports IHCs being affected in individuals with tinnitus rather than OHCs. A recent study done by Tan et al. (2013) reported the presence of off frequency listening (phenomenon which results when there is intact OHCs and non-function IHC) and better frequency selectivity in individuals with tinnitus compare to individuals without tinnitus. They also report changes observed between these individuals were relatively minor and the involvement of OHCs dysfunction cannot rule out completely.

In the present study, most of the individuals with moderate hearing loss, PTCs obtained were relatively flat and lacked in tip. Therefore Q10 and tip frequency could

not be obtained in these individuals. In the present study, Q10 values for only 3000 Hz and 4000 Hz could be compared between the group (due to small N) and no significant result was found between the groups. But Q10 was present in most of the individual with tinnitus (15) when compare to individuals without tinnitus (2) in moderate hearing loss. The reason can attribute to the absolute threshold of individuals. Most of the individuals with tinnitus had threshold within 50 dB (mean threshold of 47.48 dB) where as individuals without tinnitus had threshold more than 50 dB (mean threshold of 51.74 dB).

Many other studies in literature also reports of difficulty measuring Q10 value in moderate degree of hearing loss. A study done by Tan et al. (2013) reported that Q10 value were difficult to obtain since PTCs obtained were flat or inverted in some instance, as the threshold increases. Further, Smith et al (1987) reported that, with increases in threshold of up to 30-40 dBHL, there was a selective elevation and broadening of the tip region in the PTC response. Once the threshold is 50 dBHL or greater the tip response was completely absent. This suggests that, threshold of greater than 40-50 dBHL is a results in of complete removal of OHC functioning. Nelson (1991) also found abnormally broader PTCs in individuals with coclear hearing loss, indicating cochlear hearing loss of greater than 40 dBHL influence the sharp tuning capabilities usually associated with outer hair cell function.

b) Tip frequency. The result of tip frequency between individual with and without tinnitus showed no significant difference across all degrees of hearing loss. Since shift in the tip frequency indicates the presence of dead region; in the present study there no such shift in the test frequencies found, which indicates the presence of the intact IHC in these individual. Also, the results of tip frequencies of PTC showed

no change between the groups; again might be indicating IHCs are least susceptible to damage compare to OHCs (Hawkins, 1973; Jastreboff, 1990; Thabet, 2009).

The overall finding of the PTC indicated presence of OHC damage and intact IHCs in both the group, but the extent of damage was more in individuals with tinnitus compared to without tinnitus. Individuals with tinnitus showed less shaper tuning curve when compared to individuals without tinnitus (who showed sharper tuning curve) which was estimated through Q10. Also, it was found that there was no shift in the tip frequency which indicates presences of functional IHCs in both individuals.

5.1.2. TEN test.

The results of TEN obtained from the present study indicated no change in TEN masked threshold in individuals with tinnitus and without tinnitus in minimal and mild hearing loss. Similar result were obtained in a study done by Thabet (2009), wherein he reported that in individuals with tinnitus had abnormal TEOAEs; and only 15% of the individuals with tinnitus had dead region which was estimated through TEN test. This might be attributed to increased resistance of IHCs to damage compared to OHCs vulnerability.

The results of the TEN test indicated increased masked threshold in individuals without tinnitus compare to with tinnitus in moderate hearing loss. It was found that result was significant at only 2000 Hz, but TEN masked thresholds were within 10 dB of the TEN level at 2000 Hz again indicating presence of no dead region. Most of the studies in literature have shown presence of dead region when the absolute threshold was greater than 70 dBHL. (Aazh & Moore, 2007; Vinay &

Moore, 2007b). Since in the present study has included only individuals with absolute threshold less than 55 dBHL, presence of dead region was not seen.

To summarise the results of TEN test, there was no dead region found in both the groups across degrees of hearing loss which could be attributed to the fact that the peripheral hearing sensitivity was not beyond 55 dBHL to have a definite IHC damage.

5.1.3. EChcoG.

The result of the EChcoG showed the higher amplitude for cochlear microphonics in individuals without tinnitus than with tinnitus in minimal hearing loss, whereas no significant results were found in mild and moderate hearing loss. The latency of CM showed no significant difference between both the groups across degrees of hearing loss. The presence of cochlear microphonic is a good indicator of OHC functioning (Yoshie & Yamaura, 1969; Elberling & Salomon, 1973; Eggermont, 1976). In the present study it was seen that cochlear microphonic amplitudes are lesser in individuals with tinnitus, again indicating poorer functioning of OHC in individuals with tinnitus.

In the present study, most of the individuals (4) with tinnitus had CM present than individuals without tinnitus (1) again, could be attributed to absolute threshold of individuals. Most of the individuals with tinnitus had threshold less than 50 dBHL compare to individuals without tinnitus whose thresholds were greater than 50 dBHL. There are few studies done on effect of hearing loss on cochlear microphonics. One of such study done by Davis et al. (1989) reported about 25% of the OHC loss along the cochlear partition will result in reduction of CM potential by 25%.

All the findings in the present study indicate poorer functioning of OHCs in individuals with tinnitus than without tinnitus. The finding of all results can be summarised in terms of functioning of OHCs. In the present study it was found that lower Q10 values and lower amplitude of CM in individuals with tinnitus which directly indicate poorer functioning of OHC in individuals with tinnitus and results of tip frequency and TEN test showed normal functioning of IHCs. Thus, we can infer from the overall findings that OHC dysfunction is profound in individuals with tinnitus than damage seen at IHCs; this suggests that OHCs may be the site for generation for tinnitus.

4.2. PTC, ECochG and TEN test results in individual with tinnitus.

The result of PTC revealed no significant difference in all parameter across degree of hearing loss in individuals with tinnitus. Although it showed no difference across degrees of hearing loss, it was found that the number individuals in which PTCs obtained varied across different degrees of hearing loss. The total number of PTCs obtained in minimal, mild and moderate hearing loss was 26, 18 and 15 respectively (including all the test frequencies/parameters). As the loss increased from minimal to moderate, the sharpness of tip of PTCs were reduced and PTC was more flat. Tip frequency and TEN results did not show any difference across degrees of hearing loss within individuals with tinnitus, indicating absence of dead region irrespective of degrees of hearing loss. The results of EcochG showed no difference in CM amplitude and latency across degrees of hearing loss. However, it was shown that in individuals with minimal hearing loss the CM amplitude was relatively less, which indicants poorer functioning of OHCs at the initials stages of hearing loss. It was also seen that CM was present only 4 individuals with moderate hearing loss, which can be attributed to greater extent of OHC dysfunction in individuals with moderate loss.

To summarise the results of second objective; the function of OHCs reduces as the loss progresses from minimal to moderate hearing loss.

When the data was combined for all degrees of hearing loss, results only showed significance difference in Q10 value at 500 Hz and 2000 Hz and in CM amplitude; where Q10 was lower and CM amplitude was reduced for individuals with tinnitus irrespective of degrees of hearing loss, again indicating poorer functioning of OHCs. Tip frequencies did not show any significant difference between groups, indicating intact IHC functioning.

Thus, it can be concluded from the present study that OHCs are more affected in individuals with tinnitus than in individuals without tinnitus. Also, normal functioning of IHCs in individuals with tinnitus were seen, which suggests that OHCs are the probable site of generator for tinnitus.

Chapter 6

Summary and Conclusion

Tinnitus is the perception of sound in the absence of any external sound. The exact pathophysiology underlying tinnitus is yet to be understood as no single theory, hypothesis or the model can explain pathophysiology of tinnitus; but it is the multiple mechanism which results in perception of tinnitus. Studies have reported perception of tinnitus is majorly central phenomenon such as cortical reorganization (Eggermont & Komiya 2000; Rajan & Irvine, 1998) or hyperactivity present in the central auditory pathway (Sasaki et al., 1980; Kaltenbach et al., 2002). But, most of the studies report presence of peripheral pathology likely necessary for these changes to occur in central system (Bauer et al., 2008; Brozoski et al., 2002; Heffner & Harrington, 2002; Kaltenbach et al., 2004). It is unclear that which of this cochlear damage might cause the central changes and results in tinnitus perception.

Hence, this study was taken to compare the cochlear functioning in hearing impaired individuals with and without tinnitus; also to compare the functional difference across degrees of hearing loss in individuals with tinnitus. The study consists of two group of hearing impaired participants in the age range of 18 to 45 years. Group 1 consist of 15 participants of hearing impaired with tinnitus and Group 2 consists of 15 participants of hearing impaired without tinnitus. And each group were further divided into 3 subgroups depending on degree of hearing loss in to minimal, mild and moderate and each subgroup consists of 5 participants. The cochlear function was assessed through SWPTC, TEN test and ECochG.

The result of the first objective showed higher Q10 values in mild SNHL individuals without tinnitus and higher amplitude of cochlear microphonics in

minimal SNHL individuals without tinnitus. But, Q10 was present in most of the individual with tinnitus (15) when compare to individuals without tinnitus (2) in moderate hearing loss. The reason can attribute to the absolute threshold of individuals. Most of the individuals with tinnitus had threshold within 50 dBHL (mean threshold of 47.48 dBHL) where as individuals without tinnitus had threshold more than 50 dBHL (mean threshold of 51.74 dBHL). Since threshold itself was lower in individuals with tinnitus, PTCs seems to be present. There was no difference in Tip frequency found across degrees of hearing loss and TEN test also showed no difference in TEN masked threshold except for moderate hearing loss (2000Hz).

The result of second objective showed no significant difference in PTC, TEN test and ECochG in individuals with tinnitus across degrees of hearing loss. However, it was found that the number of individuals in whom PTCs were obtained varied across different degrees of hearing loss. The total number of PTCs obtained in minimal, mild and moderate hearing loss was 26, 18 and 15 respectively. The reduction in the response across degress of hearing loss directly reflects the effect of the absolute threshold.

Since data did not show significant difference across degrees of hearing loss in individuals with tinnitus and without tinnitus across PTC and ECochG. The data were combined and comparison was made between the groups for these parameter, results showed significance difference in Q10 value at 500 Hz and 2000 Hz and CM amplitude, where Q10 was lower and CM amplitude was reduced for individuals with tinnitus irrespective of degrees of hearing loss, again indicating poorer functioning of OHCs. Tip frequency did not show any significant difference between groups, indicating intact IHC functioning.

From the result of Q10 and ECochG it can be concluded that there will be better frequency resolution in hearing impaired individuals without tinnitus than with tinnitus. Further, tip frequency of PTC showed presence of no dead region and TEN test also showed presence of no dead region except at 2000 Hz in moderate hearing loss. Thus, it can be concluded from the study that damage to OHC are more common in individuals with tinnitus than without tinnitus; also, OHCs are the probable site of generation for tinnitus. Further, it can be concluded that, as degree of hearing loss increases the frequency resolution tend to become poorer.

Implication of the study.

1. The present study helps in better understanding of pathophysiology underling generation of tinnitus. It was found that OHCs are the site for generation of tinnitus and IHCs are found to be intact in these individuals. Also, understanding of pathophysiology is important while counselling individuals with tinnitus with SNHL.

2. In literature there are reports stating that the perception of tinnitus reduces after the patients are fitted with amplification devices. The understating to this could be drawn from the findings obtained in this study. Providing amplification will increase the output from the cochlear amplifier (OHCs); which might reduce perception of tinnitus as it results in providing better output to the higher centres.

3. From the findings of the present study, it is found that PTCs are better measures to evaluate cochlear functioning in individuals with sensorineural hearing loss, than TEN test and ECochG. Hence, PTCs could be used clinically to study the changes in OHCs and IHCs specifically.

Future directions.

1**.** The number of individuals considered in each sub-group was limited (5 individuals in each sub-group). Hence, the present study can be replicated using a large number of individual across various degrees of hearing loss, so it helps in better generalization of results.

2. The present study can also be extended to include individuals in moderately sever and sever hearing loss categories as most of the existing literature addresses tinnitus in individuals up to moderate loss.

3. As Fine structure OAEs are considered the most proficient tool to assess the OHC functioning, including this test would help to understand the pathophysiology at the level of OHCs much efficiently. This can be applicable to hearing loss up to mild degree.

References

Aazh, H., & Moore, B. C. (2007). Dead regions in the cochlea at 4 kHz in elderly adults: relation to absolute threshold, steepness of audiogram, and pure-tone average. *Journal of the American Academy of Audiology*, *18*(2), 97-106.

Ahmad, N., & Seidman, M. (2004). Tinnitus in the older adult: epidemiology, pathophysiology and treatme`nt options. *Drugs Aging*,21:297–305.

Axelsson A, Barrenas ML. (1992) . Tinnitus in noiseinduced hearing loss. In: Dancer AL, Henderson D, Salvi RJ, Hamernik RP, eds. Noise-Induced Hearing Loss. Boston: Mosby Year Book, 269-276 .

Axelsson, A., & Ringdahl, A. (1989). Tinnitus : a study of its prevalence and characteristics. *British Journal of Audiology*, 23: 53-62.

Axelsson, A., & Sandh, A. (1985). Tinnitus in noise-induced hearing loss. *British journal of audiology*, *19*(4), 271-276.

Baguley, D. M. (2002). Mechanisms of tinnitus. *British medical bulletin*, *63*(1), 195-212.

Bauer, C. A., Turner, J. G., Caspary, D. M., Myers, K. S., & Brozoski, T. J. (2008). Tinnitus and inferior colliculus activity in chinchillas related to three distinct patterns of cochlear trauma. *Journal of Neuroscience Research*, 86: 2564-2578.

Bohne, B. A., & Clark, W. W. (1982). Growth of hearing loss and cochlear lesion with increasing duration of noise exposure. In *New perspectives on noise-induced hearing loss* (pp. 283-302). Raven Press New York.

Bohne, B. A., Yohman, L., & Gruner, M. M.(1987). Cochlear damage following interrupted exposure to highfrequency highfrequency noise. *Hearing Research*, 29: 251-64.

Bonding, P. (1979). Frequency selectivity and speech discrimination in sensorineural hearing loss. *Scandinavian audiology*, *8*(4), 205-215.

Brozoski, T.J., Bauer, C.A., & Caspary, D.M., (2002). Elevated fusiform cell activity in the dorsal cochlear nucleus of chinchillas with psychophysical evidence of tinnitus. *Journal of Neuroscience*, 22: 2383-2390.

Cacace, A. T. (2003). Expanding the biological basis of tinnitus: crossmodal origins and the role of neuroplasticity. *Hearing research*, *175*(1), 112-132.

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

Chistovich, L. A. (1957). Frequency characteristics of masking effect. *Biofizika*, *2*(6), 714-725.

Clark, J. G. (1981). Uses and abuses of hearing loss classification. *Asha*, *23*(7), 493-500.

Coles, R. R. A. (1984). Epidemiology of tinnitus:(1) prevalence. *The Journal of Laryngology & Otology*, *98*(S9), 7-15.

Dallos, P. (1992). The active cochlea. *Journal of Neuroscience*, *12*, 4575-4575.

Dauman, R., & Cazals, Y. (1989). Auditory frequency selectivity and tinnitus. *Archives of oto-rhino-laryngology*, *246*(5), 252-255.

Dauman, R., and Tyler, R. S. (1992). Some considerations on the classification of tinnitus. In J. M. Aran., and R. Dauman., eds. *Proceedings of the fourth international tinnitus seminar* Bordeaux, France, pp 225-229

Davis, A., & Rafaie, E. A. (2000). Epidemiology of tinnitus. *Tinnitus handbook*, 1-23.

Davis, B., Qiu, W., & Hamernik, R. P. (2004). The use of distortion product otoacoustic emissions in the estimation of hearing and sensory cell loss in noise-damaged cochleas. *Hearing Research*, 187:12-24.

Davis, P. B. (1995). *Living with tinnitus*. Gore & Osment.

Davis, R. I., Ahroon, W. A., & Hamernik, R. P. (1989). The relation among hearing loss, sensory cell loss and tuning characteristics in the chinchilla. *Hearing Research*, *41*: 1–14.

Dawes, P., Fortnum, H., Moore, D. R., Emsley, R., Norman, P., Cruickshanks, K., **&** Davis, A. et all. (2014). Hearing in middle age: a population snapshot of 40- to 69-year olds in the United Kingdom. *Ear and Hearing*, 35: 44-51.

Dietrich, V., Nieschalk, M., Stoll, W., Rajan, R., & Pantev, C. (2001). Cortical reorganization in patients with high frequency cochlear hearing loss. *Hearing research*, *158*(1), 95-101.

Eggermont, J. J. (1976). Electrocochleography. In *Auditory system* (pp. 625-705). Springer Berlin Heidelberg.

Eggermont, J. J. (1990). On the pathophysiology of tinnitus; a review and a peripheral model. *Hearing research*, *48*(1), 111-123.

Eggermont, J. J.(2007). Correlated neural activity as the driving force for functional changes in auditory cortex. *Hearing Research*,229: 69–80.

Eggermont, J. J., & Komiya, H. (2000). Moderate noise trauma in juvenile cats results in profound cortical topographic map changes in adulthood. *Hearing Research*, 142: 89–101

Elberling, C., & Salomon, G. (1973). Cochlear microphonics recorded from the ear canal in man. *Acta oto-laryngologica*, *75*(2-6), 489-495.

Florentine, M., & Houtsma, A. J. (1983). Tuning curves and pitch matches in a listener with a unilateral, low-frequency hearing loss. *The Journal of the Acoustical Society of America*, *73*(3), 961-965.

Florentine, M., Buus, S., Scharf, B., & Zwicker, E. (1980). Frequency selectivity in normally-hearing and hearing-impaired observers. *Journal of Speech, Language, and Hearing Research*, *23*(3), 646-669.

Glasberg, B. R., & Moore, B. C. (1986). Auditory filter shapes in subjects with unilateral and bilateral cochlear impairments. *The Journal of the Acoustical Society of America*, *79*(4), 1020-1033.

Glasberg, B. R., & Moore, B. C. (1990). Derivation of auditory filter shapes from notched-noise data. *Hearing research*, *47*(1-2), 103-138.

Gold, T. (1948). Hearing. II. The physical basis of the action of the cochlea. *Proceedings of the Royal Society of London B: Biological Sciences*, *135*(881), 492-498.

Griffiths, T. D., Blakemore, S., Elliott, C., Moore, B. C. J., & Chinnery, P. F. (2001). Psychophysical evaluation of cochlear hair cell damage due to the A3243G mitochondrial DNA mutation. *JARO-Journal of the Association for Research in Otolaryngology*, *2*(2), 172-179.

Harrison, R.V., Aran. J.M., & Erre, J.P. (1981). AP tuning curves from normal and pathological human and guinea pig cochleas. *Journal of the Acoustical Society of America*, 69: 1374-1385.

Hawkins, J. E. (1973). Comparative otopathology: aging, noise, and ototoxic drugs. In *Otophysiology* (pp. 125-141). Karger Publishers.

Hazell, J. W. (1995). Models of tinnitus: Generation, perception, clinical implications. *Mechanisms of tinnitus*, 73-93.

Heffner, H. E., & Harrington, I. A. (2002). Tinnitus in hamsters following exposure to intense sound. *Hearing Research*, 170:83-95.

Henry, J. L., & Wilson, P. H. (2001). The psychological management of chronic tinnitus. Needham Heights, MA: Allyn & Bacon

Hoekstra, A., & Ritsma, R. J. (1977). Perceptive hearing loss and frequency selectivity. *Psychophysics and physiology of hearing*, 263-271.

Hoffman, H.J., & Red, G.W.(2004). Epidemiology of tinnitus:Tinnitus Theory and Management. London: BC Decker Inc;2004. p. 16–41.

induced hearing loss .St. Louis, MO: MosbyYear Book.

Jastreboff, P. J.(1990). Phantom auditory perception (tinnitus): mechanism of generation and perception. *Neuroscience Research* , 8: 221–54

Jastreboff, P. J., & Hazell, J. W. (1993). A neurophysiological approach to tinnitus: clinical implications. *British journal of audiology*, *27*(1), 7-17.

Kaltenbach, J. A., Rachel, J. D., Mathog, T. A., Zhang, J., Falzarano, P. R. & Lewandowski, M. (2002). Cisplatin-induced hyperactivity in the dorsal

cochlear nucleus and its relation to outer hair cell loss: relevance to tinnitus. *Journal of Neurophysiology*, 88: 699-714.

Kaltenbach, J.A., Zacharek, M.A., Zhang, J., & Frederick, S. (2004). Activity in the dorsal cochlear nucleus of hamsters previously tested for tinnitus following intense tone exposure. *Neuroscience Letters*, 355: 121-125.

Kemp, D. T. (1978). Stimulated acoustic emissions from within the human auditory system. *The Journal of the Acoustical Society of America*, *64*(5), 1386-1391.

Kiani, F., Yoganantha, U., Tan, C. M., Meddis, R., & Schaette, R. (2013). Off-frequency listening in subjects with chronic tinnitus. *Hearing research*,*306*, 1-10.

Kluk, K., & Moore, B. C. J. (2005). Factors affecting psychophysical tuning curves for hearing-impaired subjects with high-frequency dead regions. *Hearing Research*, 200: 115-131.

Konadath, S., & Puttabasappa, M. (2016). Psychophysical Tuning Curves and Extended High Frequency Audiometry Findings in Individuals with Normal Hearing Having Tinnitus. *International Journal of Health Sciences and Research (IJHSR)*, *6*(9), 371-378.

Kujawa, S. G., & Liberman, M. C. (2009). Adding insult to injury: cochlear nerve degeneration after "temporary" noise-induced hearing loss. *Journal of Neuroscience*, 29: 14077-14085.

Lanting, C. P., De Kleine, E., & Van Dijk, P. (2009). Neural activity underlying tinnitus generation: results from PET and fMRI. *Hearing research*, *255*(1), 1-13.

Levine, R. A. (1999, September). Somatic modulation appears to be a fundamental attribute of tinnitus. In *Proceedings of the Sixth International Tinnitus Seminar* (pp. 193-197). The Tinnitus and Hyperacusis Center.

Liberman, M. C. (1987). Chronic ultrastructural changes in acoustic trauma: Serial-section reconstruction of stereocilia and cuticular plates. *Hearing Research*, 26: 65- 88.

Liberman, M. C., & Dodds, L. W. (1987).Acute ultrastructural changes in acoustic trauma: Serial-section reconstruction of stereocilia and cuticular plates. *Hearing Research,* 26: 45-64.

Liberman, M. C., & Kiang, N. Y. (1978). Acoustic trauma in cats: cochlear pathology and auditory-nerve activity. *Acta oto-laryngologica*, 358: 1-63.

Liberman, M. C., & Mulroy, M. J. (1982). *Acute and chronic effects of acoustic trauma: Cochlear pathology and auditory nerve pathophysiology*. Raven Press.

Lockwood, A. H., Salvi, R. J., Coad, M. L., Towsley, M. L., Wack, D. S., & Murphy, B. W. (1998). The functional neuroanatomy of tinnitus Evidence for limbic system links and neural plasticity. *Neurology*, *50*(1), 114-120.

McFadden, D. (Ed.). (1982). *Tinnitus: Facts, theories, and treatments*. National Academies.

Meikle, M. B., Creedon, T. A., & Griest, S. E. (2004). Tinnitus archive 2d ed. Retrieved April 29, 2004.

Mitchell, C. R., & Creedon, T. A. (1995) Psychophysical tuning curves in subjects with tinnitus suggest outer hair cell lesions. *Otolaryngology Head Neck Surgery*, 113:223–233.

Møller, A. R. (1997). Similarities Between Chronic Pain and Tinnitus. *Am J (Orol*, *18*, 577-585.

Moore, B. C. J. (1978). Psychophysical tuning curves measured in simultaneous and forward masking. *Journal of the Acoustic al Society of America*, *63*: 524–532.

Moore, B. C. J. (2004). Dead regions in the cochlea: conceptual foundations, diagnosis, and clinical applications. *Ear and Hearing*, 25: 98-116.

Moore, B. C. J., & Alcantara, J. I. (2001). The use of psychophysical tuning curves to explore dead regions in the cochlea. *Ear and Hearing*, 22: 268-278.

Moore, B. C. J., Huss, M., Vickers, D. A., Glasberg, B. R., & Alcántara, J. I. (2000). A test for the diagnosis of dead regions in the cochlea. *British Journal of Audiology*, *34*: 205–224.

Moore, B. C., Glasberg, B. R., & Stone, M. A. (2004). New version of the TEN test with calibrations in dB HL. *Ear and hearing*, *25*(5), 478-487.

Møsller, A. R. (1984). Pathophysiology of tinnitus. *Annals of Otology, Rhinology & Laryngology*, *93*(1), 39-44.

Mühlnickel, W., Elbert, T., Taub, E., & Flor, H. (1998). Reorganization of auditory cortex in tinnitus. *Proceedings of the National Academy of Sciences*, *95*(17), 10340-10343.

Mulders, W. H. A. M., & Robertson, D. (2009). Hyperactivity in the auditory midbrain after acoustic trauma: dependence on cochlear activity. *Neuroscience*, *164*(2), 733-746.

Nelson, D. A. (1991). High-Level Psychophysical Tuning CurvesForward Masking in Normal-Hearing and Hearing-Impaired Listeners. *Journal of Speech, Language, and Hearing Research*, *34*(6), 1233-1249.

Newman, C. W., Jacobson, G. P., & Spitzer, J. B. (1996). Development of the tinnitus handicap inventory. *Archives of Otolaryngology–Head & Neck Surgery*, *122*(2), 143-148.

Nobili, R., Mammano, F., & Ashmore, J. (1998). How well do we understand the cochlea?. *Trends in neurosciences*, *21*(4), 159-167.

Nondahl, D. M., Cruickshanks, K. J., Wiley, T. L., Klein, R., Klein, B. E., & Tweed, T. S. (2002). Prevalence and 5-year incidence of tinnitus among older adults: the epidemiology of hearing loss study. *Journal of the American Academy of Audiology*, *13*(6), 323-331.

Rajan, R., & Irvine, D. R. F. (1998). Absence of plasticity of the frequency map in dorsal cochlear nucleus of adult cats after unilateral partial cochlear lesions. *Journal of Comparative Neurology*, *399*(1), 35-46.

Rajasekhar, B. (1976). Development and standardization of a picture SRT test for adult and children in Kannada. *Unpublished Master's Dissertation. University of Mysore*.

Robertson, D., Cody, A. R., Bredberg, G. & Johnstone, B. M. (1980). Response properties of spiral ganglion neurons in cochleas damaged by direct

mechanical trauma. *Journal of the Acoustical Society of America*, 67: 1295-1303.

Ryan, A., Dallas, P., & McGee, T. (1979). Psychophysical tuning curves and auditory thresholds after hair cell damage in the chinchilla. *Journal of the Acoustical Society of America*, 66: 370-378.

Sahey, T. L., & Nodar, R. H. (2001). A biochemical model of peripheral tinnitus. *Hearing Research,*152: 43–54

Salvi, R. J., Lockwood, A. H., & Burkard, R. (2000). Neural plasticity and tinnitus. *Tinnitus handbook*, *1*, 123-148.

Sanchez, L., Boyd, C., & Davis, A. (1999). Prevalence and problems of tinnitus in the elderly. In *Proceedings of the Sixth International Seminar* (pp. 05-09).

Sasaki, C. T., Kaner, J.S., & Babitz, L. (1980). Differential 2-deoxyglucose uptake after deafferentation of the mammalian auditory pathway - a model for examining tinnitus. Brain Research,194: 511-6.

Sassaki, C.T., Babitz, L., & Kauer, J. S. (1981). Tinnitus: development of a neurophysiologic correlate. *Laryngoscope,* 91:2018–2024

Schaette, R., & McAlpine, D. (2011). Tinnitus with a normal audiogram: physiological evidence for hidden hearing loss and computational model. *Journal of Neuroscience*, 31: 13452-13457.

Sek, A., Skrodzka, E., & Moore, B. C. (2003). Development of a fast method for determining psychophysical tuning curves. *The Journal of the Acoustical Society of America*, *113*(4), 2287-2288.

Shiomi, Y., Tsuji, J., Naito, Y., Fujiki, N., & Yamamoto, N. (1997). Characteristics of DPOAE audiogram in tinnitus patients. *Hearing research*, *108*(1), 83-88.

Smith, D.W., Moody. D.B., Stebbins, W.C. & Norat, M.A.(1987). Effects of outer hair cell loss on the frequency selectivity of the patas monkey auditory system. *Hearing Research*, 29: 125-138.

Spoendlin, H., & Baumgartner, H. (1977). Electrocochleography and cochlear pathology. *Acta oto-laryngologica*, *83*(1-6), 130-135.

Summers, V., Molis, M. R., Musch, H., Walden, B. E., Surr, R. K., et al. (2003). Identifying dead regions in the cochlea: Psychophysical tuning curves and tone detection in threshold-equalizing noise. *Ear and Hearing*, 24: 133-142.

Tan, C. M., Lecluyse, W., McFerran, D., & Meddis, R. (2013). Tinnitus and patterns of hearing loss. *Journal of the Association for Research in Otolaryngology*, *14*(2), 275-282.

Thabet, E. M. (2009). Evaluation of tinnitus patients with normal hearing sensitivity using TEOAEs and TEN test. *Auris Nasus Larynx*, *36*(6), 633-636.

Thornton, A. R., & Abbas, P. J. (1980). Low-frequency hearing loss: Perception of filtered speech, psychophysical tuning curves, and masking. *The Journal of the Acoustical Society of America*, *67*(2), 638-643.

Turner, C., Burns, E. M., & Nelson, D. A. (1983). Pure tone pitch perception and low-frequency hearing loss. *The Journal of the Acoustical Society of America*, *73*(3), 966-975.

Vernon, J. A. (1998). *Tinnitus: treatment and relief*. Allyn & Bacon.

Vernon, J. A., & Meikle, M. B. (2000). Tinnitus masking. *Tinnitus handbook*, 313-356.

Vinay, & Moore, B. C. (2007). Speech recognition as a function of high-pass filter cutoff frequency for people with and without low-frequency cochlear dead regions. *The Journal of the Acoustical Society of America*, *122*(1), 542-553.

Warnaar, B., & Dreschler, W. A. (2012). Agreement between psychophysical tuning curves and the threshold equalizing noise test in dead region identification. *International journal of audiology*, *51*(6), 456-464.

Weisz, N., Hartmann, T., Dohrmann, K., Schlee, W., & Norena, A. (2006). High-frequency tinnitus without hearing loss does not mean absence of deafferentation. *Hearing research*, *222*(1), 108-114.

Yathiraj, A., & Vijayalakshmi, C. S. (2005). Phonemically balanced word list in Kannada. *Developed in Department of audiology, AIISH, Mysore*.

Yoshie, N., & Yamaura, K. (1969). Cochlear microphonic responses to pure tones in man recorded by a non-surgical method. *Acta oto-laryngologica*,*67*(sup252), 37-69.

Zhou, X., Henin, S., Long, G. R., & Parra, L. C. (2011). Impaired cochlear function correlates with the presence of tinnitus and its estimated spectral profile. *Hearing research*, *277*(1), 107-116.

Zwicker, E., & Schorn, K. (1978). Psychoacoustical tuning curves in audiology. *Audiology*, *17*(2), 120-140.