“AUDIOLOGICAL EVALUATION OF EARLY NOISE-INDUCED HEARING LOSS IN TRAFFIC POLICE PERSONNEL IN BELAGAVI”– A COMMUNITY-BASED ONE YEAR CROSS SECTIONAL STUDY

BY

REG. NO:BE0118003

Dissertation

Submitted to the

KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH,

Belagavi, Karnataka

In partial fulfilment

of the requirements for the degree of

MASTER OF SURGERY

IN

OTORHINOLARYNGOLOGY AND HEAD AND NECK SURGERY

DEPARTMENT OF OTORHINOLARYNGOLOGY AND HEAD AND NECK SURGERY, JAWAHARLAL NEHRU MEDICAL COLLEGE, BELAGAVI, KARNATAKA

APRIL – 2021

KLE ACADEMY OF HIGHER EDUCATION AND RESEARH,
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This is to certify that the dissertation entitled **"Audiological Evaluation of Early Noise-Induced Hearing Loss in Traffic Police Personnel In Belagavi"**– A Community-Based One Year Cross Sectional Study, is a bona fide research work done by **REG. NO: BE0118003**

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LIST OF ABBREVIATIONS USED

dB	:	Decibel, unit of sound intensity
Hz	:	Hertz
et al	:	et alii *(Latin; 'and others')*
i.e.	:	id est *(Latin; 'that is')*
SNHL	:	Sensory neural hearing loss
NIHL	:	Noise induced hearing loss
PTA	:	Pure tone audiometry
SRT	:	Speech recognition threshold
SIS	:	Speech identification score
IHC	:	Inner hair cells
OHC	:	Outer hair cells
SPL	:	Sound pressure level
OAE	:	Otoacoustic emissions
ABR	:	Auditory Brainstem Response
DPOAE:		Distortion-product Otoacoustic emissions
TEOAE:		Transient evoked otoacoustic emissions

ABSTRACT

Background:

Potentially hazardous sources of excessive exposure to noise include occupational noise and sociocusis. The negative influence of excessive noise on cochlear hearing is known. Noise Induced Hearing Loss is termed as a gradual deterioration of sensory neural hearing as an effect to continuous excessive exposure of the auditory system to noisy surrounding. There have been studies assessing NIHL among traffic police personnel with audiometry, however using DPAOE in this study population for early detection of cochlear pathology is rarely studied.

Aims:

1. To measure prevalence of noise-induced hearing loss amongst traffic police personnel in a tier-2 city like Belgaum.
2. To identify early noise-induced hearing loss in traffic police personnel.

Materials and methods:

This is a community based cross-sectional study conducted in the department of Otorhinolaryngology of Dr. Prabhakar Kore Hospital, Belagavi, from January 2019 to December 2019. 80 road traffic police personnel were included in the study, Their symptoms noted, detailed history and examination taken including Smith hearing loss severity questionnaire. Later, they were subjected to pure tone audiometry and Distortion-product OtoAcoustic emissions, its findings were assessed and compared.

Results:

All road traffic police personnel worked for a minimum duration of 8 hours per day on road. Out of 80 traffic police examined, 56.25% (45) had hearing loss detected by PTA. The prevalence of early noise induced hearing loss assessed with DPOAE is 62.5% (50). DPOAE gives higher sensitivity. Unilateral cases as detected by audiometry were 6.25% (5: right-1 and left-4) and bilateral cases were 50% (40). And 11.25% (9: right-3 and left-6) of unilateral NIHL and 55% (44) of bilateral NIHL was detected by DPOAE. Though majority of NIHL are symmetrical losses, in early phase of disease, unilateral pathology is found. 16% of the subjects experienced tinnitus.

Conclusion:

Mean age at which NIHL was prevalent in noise exposed traffic policemen were 36.84 years. Prevalence of noise induced hearing loss is high in Belgaum traffic police personnel with most of them having mild to moderate degree of hearing loss. Study has proven that increasing age and more duration of service are significant risk factors causing noise induced hearing loss. Lack for proper knowledge and reduction in protective aids and interventions are factors prone for NIHL. DPOAEs represent a sensitive test for monitoring the effects of noise in preclinical conditions. With further reductions in acquisition time, the measurement procedure will be applicable in clinical routine in the near future.

Keywords: Noise induced hearing loss, Early detection, Traffic police, Pure tone audiometry, Distortion-product Oto-Acoustic emissions, tier two city.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF GRAPHS

LIST OF FIGURES

INTRODUCTION

The normal physiological mechanisms of human body face new challenges everyday due to modernization and industrialization. This cause adverse impacts on our health including hearing.

The 2017 estimates of WHO reports 360 million individuals suffering from severe hearing loss and 1.1 billion among them are aged 12-35yrs[1]. It's the 2nd most prevalent aetiology for reduced hearing after Presbycusis[2]. WHO has stated, among worldwide occupational hazards, Noise induced hearing loss[NIHL] is the commonest[3].It is an irreversible occupational hazard, with highest burden in developing countries[4]. And ironically, it is a preventable pathology.

Potentially hazardous sources of excessive exposure to noise include occupational noise and sociocusis. NIHL is termed as a gradual deterioration of sensory neural hearing as an effect to continuous excessive exposure of the auditory system to noisy surrounding[2]. Auditory fatigue is the initial clinical feature of NIHL, and hearing can gradationally improve if subject cease the exposure[7]. A single exposure of intense noise resulting in sudden hearing loss is defined Acoustic trauma[5].

The normal human hearing frequency is 20-20,000 Hz[6]. NIHL affects high frequency in initial stages. The loss is left unnoticed unless the lower speech frequencies are involved. Initial brunt of attack in chronic exposure to loud noise is to hair cells associated with high-frequency sounds. Around 16,000 hair cells are present at birth, and up to 30-50% hair cells may be exterminated until hearing loss can be ascertained.

As time progresses, a continuous burden of an inordinate amount of noise prime to inebriated transmission of high and low frequency sounds to brain.

Unfortunately, there is limited ability to ascertain the preliminary phase of NIHL. And a permanent damage to large group of hair cells are done by the time this noticeable. This loss is not reversible and cannot typically be corrected by hearing aids[7].

Cochlear damage in NIHL presents with mild-moderate symmetrical loss with/without tinnitus. Per contra, an indicative presence of asymmetrical thresholds are reported and, based on the exposure duration, loss can be severe to profound.[8].

Mills explains the reduction of auditory acuity post exposure to continuous and impulse noise. Characteristics of the hearing loss engendered by acoustic impulses is different in attribute from that effected by continuous exposure to steady state noise at moderate levels. An immoderate displacement of basilar membrane by an acute profound acoustic impulse bring about mechanical tearing and tattering, whereas injuries effected by lower intensity impulses may be produced by effects, including the exhaustion of energy stores, change in shape of the tectorial membrane due to mechanical trauma, and cochlear vasoconstriction[9].

NIHL has a significant effect on affected person. It considerably impairs one's social relationships, professional capabilities, quality of life and in turn economy. Moore et al. postulated that Noise-induced tinnitus occur before measurable NIHL, warning more strict exposure limits for protection against noise-induced tinnitus[10]. And it is also proven that acoustic damage result in vestibular dysfunction like vertigo[8].

Traffic noise has been increasing since years as a harmful byproduct to growing transportation and industrialization especially in developing countries like India. As of 'The Traffic Index 2019', India ranks the highest in world for traffic congestion globally and holds 4 out of top 10 most traffic congested cities in the

world which are Bengaluru, Mumbai, Pune and New Delhi at 1st, 4th, 5th and 8th position respectively.

Traffic police personnel are bearing a major brunt of this attack due to intensification of vehicular traffic. Depending on heavy & light traffic, road traffic noise differs from place to place. Level of sound calibrated was 40-70 dB in light traffic zones and 70-102 dB in heavy traffic areas.Cities are classified into different tiers based on their population. The traffic congestion, noise pollution created, and its occupational hazards are highest in tier 1 cities and gradually decrease with population density.

Unguarded exposure of noise pollution is defined by Occupational Safety and Health Administration [OSHA] as 85 dB for a period of 8-hours or more[11]. For every 5-dB increase in intensity, time is halved (i.e., 4-hour limit at 95 dBA, 2-hour at 100dBA, etc.). Normal conversation falls in 50–65 dB[12], 80–85 is average disciplined city traffic noise[2], 95–110 is motorbike[13]. Motor vehicle horns produce sound of 109–112 dB. NIHL is a high frequency SNHL. Traffic policemen, amid this noise pollution are in constant exposure to this hazard[14].

Certain adjunctive risk factors are associated with NIHL, which can invoke its progression[2]. Increasing age, ethnicity, genetic and, male gender are nonmodifiable risk factors. While, modifiable factors are voluntary exposure to intense sound, lack of protection, smoking, lack of exercise, poor diet, diabetes and cardiovascular disease[15]. Overlapping of these elements with sound exposure, accelerate NIHL occurrence. Early detection and further prevention are the need of the hour.

Pure tone audiometry (PTA) is a diagnostic tool to detect NIHL in exposed individuals. As speech frequency lies lower range, significant damage occur before a

person begin to appreciate hearing loss[16].NIHL is more widespread than revealed by pure tone audiometry.

Otoacoustic emissions (OAE) are frequency-specific non-invasive aid for calibrating cochlear damage with high sensitivity[17]. Two types of OAEs are Distortion product otoacoustic emissions (DPOAEs) and Transient-evoked otoacoustic emissions (TEOAEs)[18]. It assess the Outer hair cell (OHC) function with high precision, which are fundamental targets for NIHL[19].

Markers of OAE signal de-escalate at frequencies representing the damaged region. Signal processing and OAE generation is hindered in injury to basal turn of cochlea. In workers working in noisy environment, the OAE amplitude decrease in those frequencies specific of acoustic injury. OAE changes happen before PTA threshold shift, supporting the higher sensitivity of OAEs in diagnosing NIHL in earlier stage. Hence, OAEs can function as a quantitative tool for hearing loss evaluation and monitoring in occupational NIHL[17].

Identification of early NIHL allows us to focus hearing conservation efforts on those individuals who are most in need of those services. We can halt the process of NIHL by certain measures like usage of hearing protective measures, community awareness, enforcement of noise regulations, periodic hearing assessment, reducing on-road duty hours, changing them to desk-jobs, etc. Prevalence mapping, treatment, and prevention of NIHL is of prime significance. Currently, prevention and protection from NIHL are the first lines of defense, as treatment is indefinite.[8].

There are only few studies performed estimating early auditory effects of noise pollution in traffic police personnel, particularly in India. Hence, this study has a pivotal role.

OBJECTIVES

1. To measure prevalence of noise-induced hearing loss amongst traffic police personnel in a tier-2 city like Belgaum.
2. To identify early noise-induced hearing loss in traffic police personnel.

REVIEW OF LITERATURE

Research works in the field of NIHL is present since early 1900s. Shahid et al in 2019 conducted a cross-sectional, descriptive study in 329 traffic policemen of Lahore to determine the frequency of Noise induced hearing loss Among Traffic Wardens of Lahore city, by an interviewer-administered questionnaire followed by PTA[4].

A study conducted by Chauhan et al in 88 traffic police man in 2018 included a questionnaire followed by Smart phone based pure-tone audiometry in Vadodara, Gujarat. [20].

Ghimire et al in 2018 conducted a cross-sectional study among 36 traffic police personal about screening of hearing ability and hearing threshold in Biratnagar, Nepal[21].

An observational comparative study was performed in 2017 by Indora et al in 35 male traffic policemen with field posting of more than 3 years to assess the hearing pathway in traffic policemen by means of brainstem evoked response audiometry (BERA), mid latency response (MLR), and slow vertex response (SVR)[22]. They found that chronic exposure of traffic policemen to noise resulted in delayed conduction in peripheral part of the auditory pathway, *ie*, auditory nerve up to the level of superior olivary nucleus; no impairment was observed at the level of sub-cortical, cortical, or the association areas[23].

In 2016, Sanju et al determined self-assessment of noise-induced hearing impairment in traffic police and bus drivers by a questionnaire-based study in Mysore, Karnataka. The study was done on 60 non-smokers, male traffic police, and 80 long route bus drivers in the age range of 30–50 years. There were 15 questions related to

self-assessment of hearing quality, annoyance evaluation, noise-related attitude and knowledge[24].

A prospective observational study was conducted by Suri et al in 2015 among 150 adults in the age group of 21 to 65 years, attending outpatient department with non-otological pathology. It was aimed to determine whether deterioration in cochlear function, as evaluated by distortion product otoacoustic emissions (DPOAE), exists before the elevation of audiometric threshold[25].

A study in 2014, was conducted among 60 traffic policemen of Aurangabad, Maharashtra by Khan ST et al with an objective to assess the hearing loss in traffic police and to determine hazardous effects of traffic noise [144].

Gupta et al in 2014 carried out a study on 90 non-smoking male traffic policemen aged 20-50 years during a general health check-up camp organized at Government Dispensary, Police Lines, Patiala, Punjab. The study assessed Self-assessment of hearing quality and noise-related attitudes among traffic policemen by a questionnaire[26].

A cross-sectional questionnaire study in 60 traffic policemen of Bangalore city, Karnataka was conducted by Venkatappa et al in 2012 to assess the knowledge of traffic policeman regarding the assessment of knowledge, attitude and practices and relation of auditory effects of noise and noise exposure showed positive correlation between the duration of service and NIHL prevalence.[27].

Shrestha et al in 2011 conduced a cross sectional, descriptive study 110 responding traffic police personnel aged 21-50 years, to measure permanent threshold shift in traffic police personnel due to noise exposure and to assess its associated with duration of noise exposure, years of work and risk factors. The study showed

significant positive relationship between NIHL and duration of work, tobacco smoking, alcohol consumption and family history[11].

Thomas et al in 2007 among 30 traffic point duty personnel by audiometric tests revealed significant positivity for noise-induced hearing loss. A questionnaire survey revealed a lack of knowledge on occupational safety and personal protective equipment[28].

Conditions of work, sound pressure levels on the city roads, direct and indirect hearing loss risk factors and hearing were studied in 200 traffic policemen working on the roads and 50 policemen working in offices by Sokolova et al in 2006. It was found that working conditions correlate with hearing loss in traffic policemen, so hearing loss in the policemen working on the roads is occupational and requiring adequate prophylactic and therapeutic management.

In 2006, Tiwari et al conducted questionnaire-based study was among 86 traffic policemen in Ahmedabad, Gujarat who were randomly selected for an awareness workshop for prevention of noise pollution. The questionnaire included questions regarding the self-assessment of the policemen about their hearing ability, past and present exposure to loud sound and the use of personal protective devices such as earplugs and earmuffs[29]

According to study carried out by Sharif et al among randomly selected 100 traffic police in 2004, to determine NIHL prevalence. It was concluded that significant respondents developed sensorineural hearing loss due to noise exposure and it was related to the duration of exposure [30].

Xiong et al in 2019 performed a study to investigate if tinnitus patients with normal audiograms have hearing loss missed by standard PTA testing, referred as

"missed hearing loss" in the paper. 106 tinnitus patients were tested using fine frequency resolution (1/24 octave step) audiometry. DPOAEs revealed significantly reduced OAE amplitude in the tinnitus patients, suggesting OHC dysfunction.

Suri et al performed a study in 2018 to detect early hearing impairment and deterioration in cochlear function by DPOAE in individuals with normal pure tone audiometry[31].

In 2017 Zelle et al governed a study in Germany with an aim is to improve the accuracy of inner-ear diagnosis by DPOAE generation mechanisms[18].

Nadon et al in 2017 performed a pilot study for Field Monitoring of Otoacoustic Emissions During Noise Exposure.This study demonstrates that the monitoring of an individual's OAEs could be useful in monitoring temporary and permanent changes in hearing status induced by exposure to ambient noise and could be considered as a new tool for effective hearing conservation programs in the workplace.

A study was conducted by Guida et al in 2012 to investigate the correlation between the findings of audiometry results and distortion product otoacoustic emissions (DPOAE) in 200 military police officers of Sao paulo[**32**].

Kujawa et al in 2009 postulated that hearing loss may be caused by damages of hair cells or cochlear synaptopathy which was referred as synaptopathy or “hidden hearing loss”.

A study conducted in 2007 by Renata et al with an aim to investigate the capability of otoacoustic emission (OAE) in the detection of low levels of noise-induced hearing loss, audiometric and otoacoustic data of young workers (age: 18-35) exposed to different levels of industrial noise showed that correlation between

DPOAE levels and audiometric hearing threshold is sufficient to design OAE-based diagnostic tests with good sensitivity and specificity also in a very mild hearing loss range, between 10 and 20 dB.

A study conducted by Paul et al in 2005 assessed relation between Distortion-product otoacoustic emission spectra and high-resolution audiometry in noise-induced hearing loss. DPOAEs performed better than audiogram patterns.

Mariola et al in 2001 assessed Otoacoustic emissions in industrial hearing loss. OAE changes may occur prior to the audiometric threshold shift, which supports the superiority of OAEs in early detection of noise-induced damage. Therefore, OAEs may be applied as a quantitative test for individual assessment and monitoring of industrial hearing loss.

ANATOMY AND PHYSIOLOGY

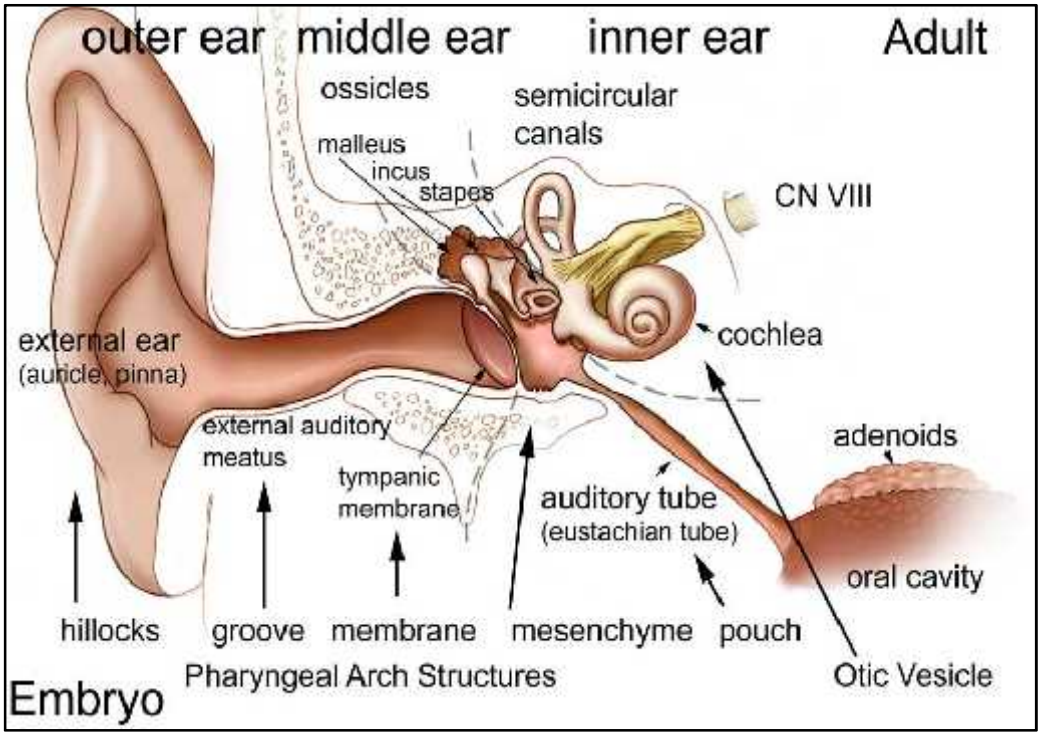


Fig 1: Anatomy of Ear

Development of Inner ear (Labyrinth)

The internal ear initially develops independently and become attached to the stapes footplate thereby giving continuity to the auditory pathway. There is initial development of generalized structure of the membranous labyrinth, followed by encasement by the bony labyrinth and then production of a further series of spaces within this bony shell that in turn become the perilymphatic spaces of the complete structure[33].

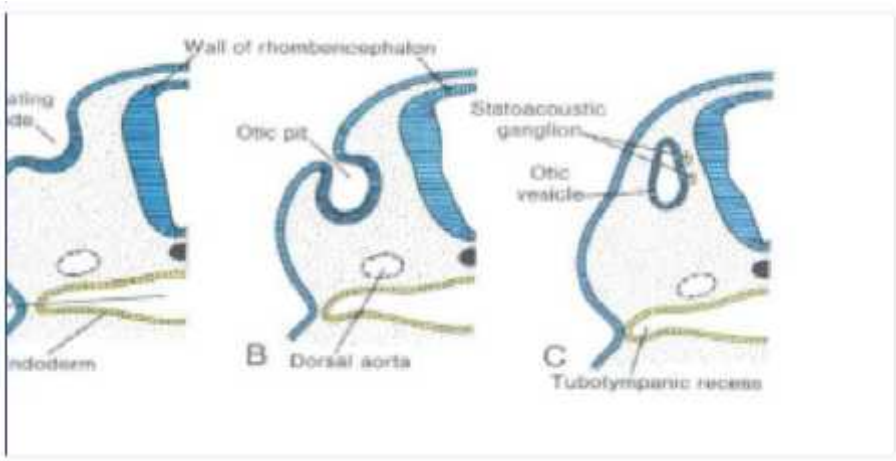


Fig 2: Development of Otocyst

On day 22-23 of intrauterine life, otocyst develops as a thickening called the oticplacode on the surface of the embryo just cranial to the first somite. This thickening sinks into the mesoderm and eventually the otic pit closes off to leave the isolated otocyst/otic vesicle, which subsequently becomes the membranous labyrinth by 25 weeks. An assemblage of neural crest cells lie surrounding the otocyst which develop to become the geniculate (facial), spiral (auditory) and Scarpa's (vestibular) ganglion.

Then cochlea begins to develop. And saccule get separated from utricle and it exhibits a solitary outpouching that deepens and then starts coiling at base and reach apex to form its full 2.5 coils in a period of 25 weeks.

In membranous labyrinth, sensory cells of 3 cristae, 2 maculae and the organ of Corti develop from ectoderm. This is due to ingrowth of cochlea-vestibular ganglion nerve endings or vice versa.

Development of organ of Corti starts begins at 11 weeks from a single chunk of ectodermal cells. The inner and outer hair cells and specialized supporting cells develop from this block[34]. Clusters of stereocilia and a single kinocilium develop on each hair cell. The cochlear kinocilium regresses leaving the adult configuration of stereocilia and the spaces between the outer hair cells open as the supporting cells (Deiter's cells) change shape.

Differentiation progresses from base to apex so that at any single time various stages of development can be seen. Epithelium in proximity to sensory areas grow into specialized cell groups, which balances the endolymphatic electrical and ionic stability. It forms striavascularis of the cochlear duct and the 'dark cell' regions belonging to vestibular sensory epithelium[34].

Introduction to sound

Sound is defined as vibrations of molecules that traverse via air or another medium and can be heard when they reach ear. Sound is characterized by pitch and intensity Pitch is termed as frequency and intensity as loudness of sound[35].

The Auditory system

It consists of the peripheral and central auditory system, which if normal gives perfect auditory acuity. Ear is an efficient transducer, converting sound pressure in the air into a neural-electrical signal that is translated by the brain as speech, music, noise, etc.[36].

ULTRASTRUCTURE OF COCHLEA

Adult cochlea is "snail-shell" in look. The adult human cochlea have 2.5 turns, but this can vary up to 2.75 or even 3 turns[37].

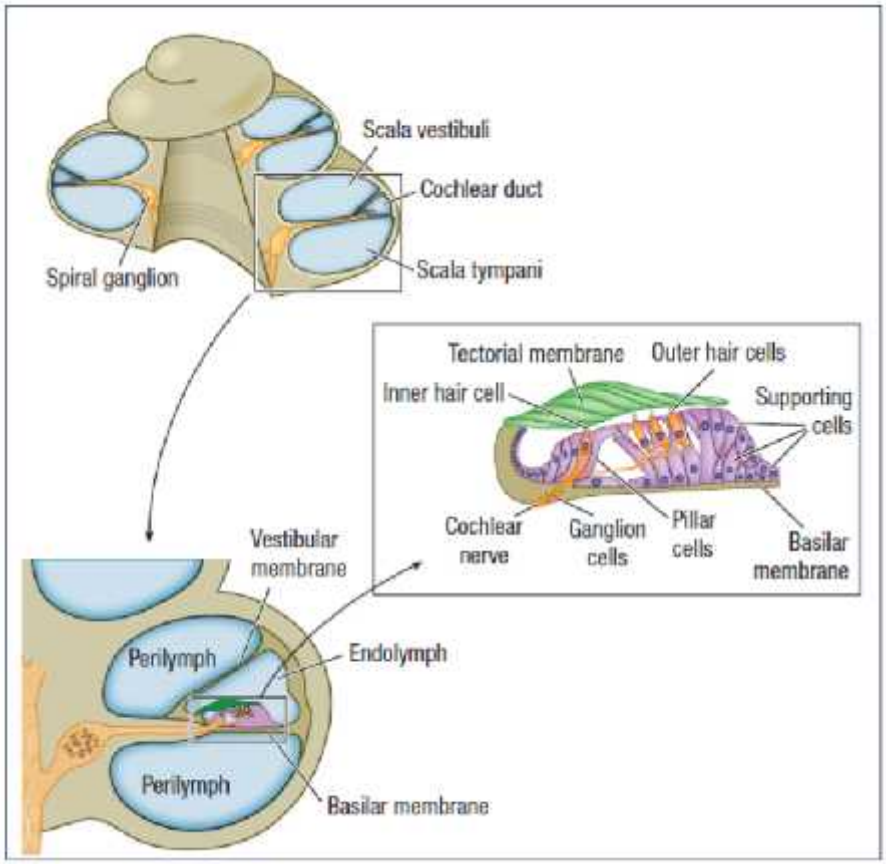


Fig 3: Anatomy of Cochlea

Cochlear duct

Its divided into three separate fluid compartments; two contain perilymph (scala tympani and scalavestibuli), like the body's extracellular fluid. And the other,

scala media, contains endolymph, which is like intracellular fluids. Central bony axis of spiral, termed modiolus, consist the spiral ganglion enclosing bipolar neurons that innervate hair cells in periphery and form cochlear nerve centrally.

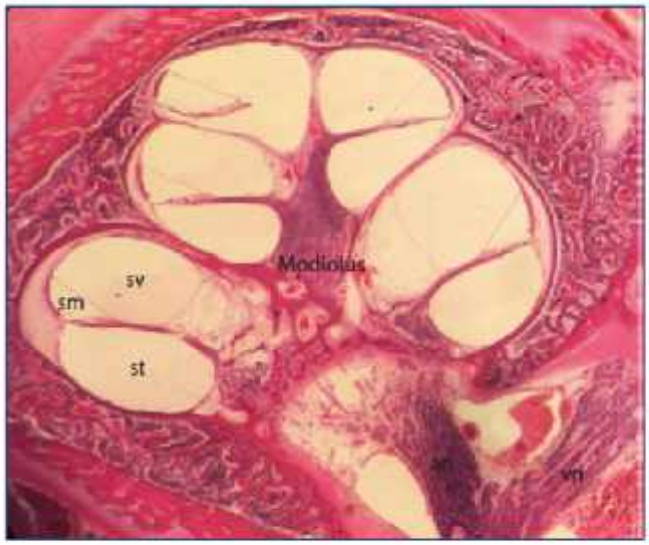


Fig 4: Section through human temporal bone at approximately the mid-modiolar level of the cochlea. sm – scala media; st – scala media; sv – scala vestibuli; an – auditory nerve; vn – vestibular nerve[34]

Endolymph

Fig 5: **Scanning electron micrograph of a dissected guinea pig organ of** Corti (oC) showing its location spiralling around the modiolus (mod). As indicated, high frequency sounds are detected near the base of the spiral and low frequencies near its apex. Scale bar = 1 mm.

This fluid has high K+ and low Na+ with an elevated positive electrical potential (180mV) called endolymphatic potential (EP). The organ of Corti cells

facing scala media are joined by tight junctions. Thus there is a chemical and electrical isolation, the only communication being through ion channels in the sensory cells[34].

Maintenance of the EP is crucial to hearing. It drives currents through transduction channels that are fundamental to hair-cell function and is thus a vital component required for producing the high sensitivity to the cochlea[38].

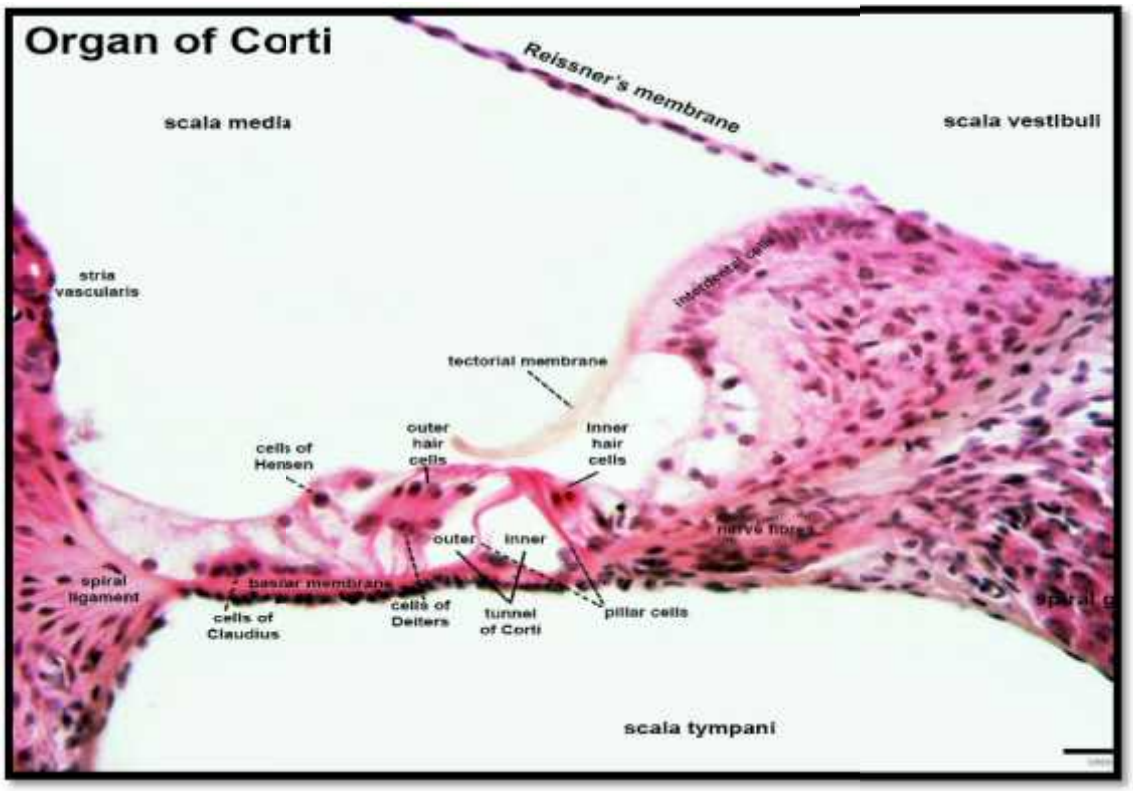


Fig 6: **Cellular architecture of Organ of Corti**

Organ of Corti

The organ of corti was first identified in 1851 by Alfonso GiacomoGaspareCorti, an Italian anatomist. The organ of Corti extends for 35mm in humans. The hair cells in sensory zone have an apical hairs termed stereocilia. Apical end of supporting cells and hair cells reach the upper surface, while the basal ends lie on basement membrane only in supporting cells[39].

Inner hair cells

Apical surface of IHCs are flat to lightly incurved. The cell body is flask-shaped with a wide centre, tapering towards base and apex. The IHC stereociliarybundle usually form a single linear row, their axes running along the hair-cell row. Additional IHC rows may be present.

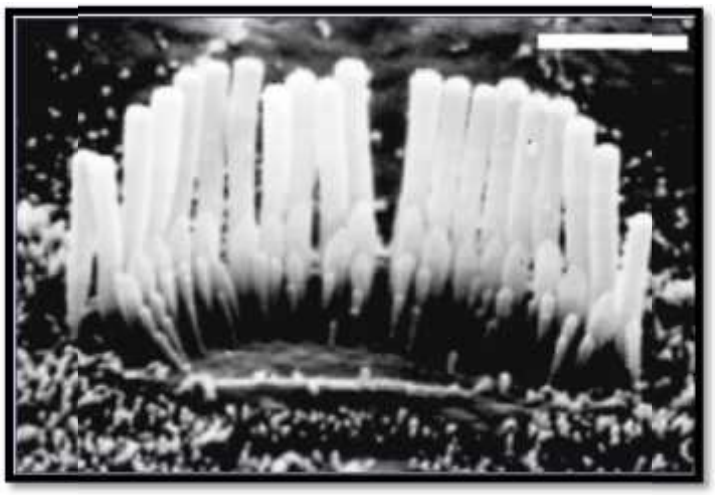


Fig 7a: Linear pattern of IHC stereocilia

Outer hair cells

OHCs are 3 rows and lie exterior to epithelium. At the base of cochlea, rows may increase.

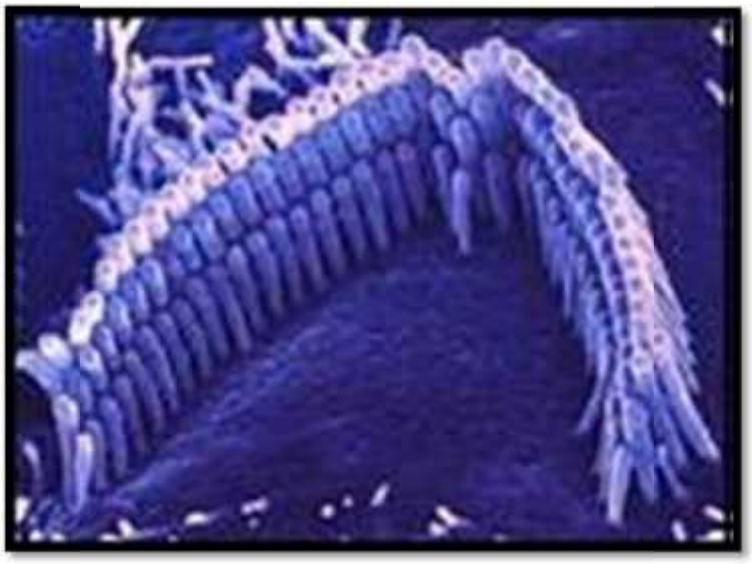


Fig 7b: W/V pattern of outer hair cell stereocilia

Towards the base, OHC and supporting cell's height decreases, longer in furthermost row and shorter at inner side. Towards basal region, there is graded length reduction and numerical increment of the stereocilia on both IHCs and OHCs[40].

Major contribution to neural signaling, representing sensory transduction and cochlear processing is by passive sensory role of IHCs. But, the cochlear active amplifier which enhance frequency detection and separation is by OHCs. The vibrations generated by sound from basilar membrane are mechanically boosted to produce a sharp tuned and high sensitive displacement pattern, which will be reported by IHCs[41].

MECHANISM OF HEARING

Airborne sound is an alternate phase of condensation and rarefaction. The auricle collects sound waves which are at greater amplitude and lesser force. It is transmitted to the EAC and then reach tympanic membrane[42]. On arriving the tympanic membrane, the catenary lever action, ossicular lever action and Hydraulic Lever action plays a major role. And the vibrations are converted to greater force and lesser amplitude, which are transmitted to stapes foot plate.

Footplate rocks at the oval window, setting inner ear fluids in motion[43]. It then leads to displacement of the basilar membrane, leading to shearing movements between the hair cells and the tectorial membrane of the Organ of corti. Vibrations are then transmitted to the round window. The nerve impulses sally along the auditory nerve fibers to the auditory nuclei and finally receives at the auditory cortex which is perceived as sound[41].

Theories of hearing

1. **Helmholtz place theory** (1883)→ Basilar membrane act as a series of tuned resonators, each causing a resonant vibration, particularly to its own place. High frequency waves excite the basal region and low frequency excite the apical region.
2. **Rutherford's frequency or telephone theory** (1886)→ Basilar membrane and hair cells, at their entire length, are activated by all frequencies. Firing rate of auditory nerve fibers represent the signal frequency.

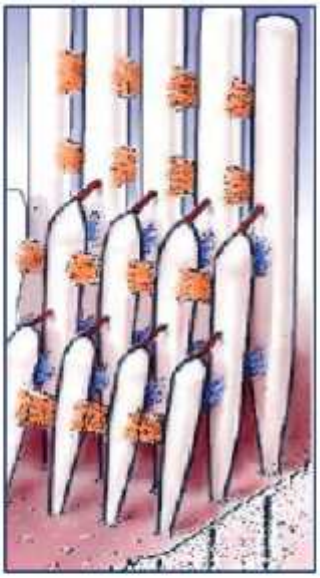
3. **Wever's volley resonance theory** (1949)→ Basal turn perceives high frequency. Low frequencies stimulate nerve action potential equal to frequency stimulation. Intermediate frequencies are represented by asynchronous discharges which combine actively to represent the stimulation frequency.

4. **Von bekesy's travelling wave theory** (1960)→ Wave move from base towards the apex. High pitched sounds cause short travelling wave not beyond the basal turn and low stimuli cause maximum displacement near the apex. Mid frequency changes occur in middle".

Hair cell Mechanics

The stereocilia are connected by extracellular filaments. A single filament, the tip link runs from the tip of each stereocilium of the shorter rows to the side of the adjacent stereocilium immediately behind.

At the upper attachment of the tip link, there is a distinctive electron dense plaque lying between the membrane and the actin core. At the lower attachment there is dense material over the actin core, separated by a gap from the membrane of the tip.



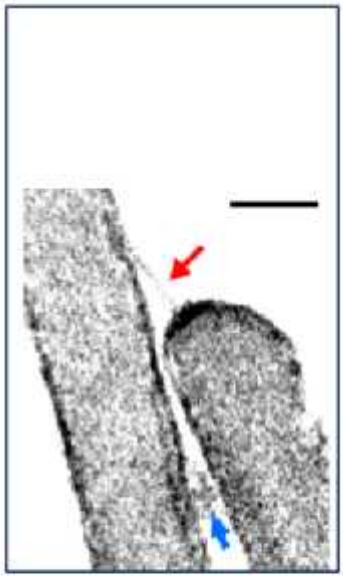


Fig 8a: Stereocila arranged in three rows of graded lengths. In addition to thin tip links (shown here in red) which are involved in the mechano-transduction process, stereocilia are attached by transverse links (shown in blue), both in the same row and from row to row.

Fig 8b: With transmission electron microscopy (TEM), the tip link (red arrow) and a lateral link (blue arrow) between medium and tall stereocilia. At both ends of the tip link, a membrane condensation is seen.

Below the lower attachment is a zone called the 'contact region' where the membranes of the two converging stereocilia approach very closely. Lateral links connect the shafts of adjacent stereocilia, both within and between rows. Where the lateral links connect to the stereocilia, the membrane and the adjacent actin filaments inside show an increased density[44].

Transduction function of the cochlea

Transduction is the conversion of mechanical energy of sound to electrical energy. When stapes is pressed onto the oval window, pressure is exerted to the perilymph in the scalavestibuli which is transferred to the scalar media. This causes

downward movement of the basilar membrane exerting pressure in the scala tympani. This is transmitted in turn to the round window which bulges into the middle ear[45].

When the stapes and oval window move out, there is an upward movement of the basilar membrane. The elastic tension built up in the basilar fibers initiates a wave which travels towards the helicotrema. Auditory nerve endings are not only stimulated electrically but also by chemical transmitters[46].

Major steps involved in transduction by hair cells are:

1. With sound stimulus, basilar membrane and organ of corti move up and down, producing a shearing action between the tectorial membrane and reticular lamina causing stereocilia to bend sideways.

2. This bending of hair bundles opens channels, which allow potassium entry into hair cell mediating in depolarization.

3. Depolarization spreads to the lower part of the cell leading to opening of calcium channels.

4. Calcium mediates fusion of the vesicles to the basal part of the cell membrane, releasing transmitter substance- amino acid glutamate.

5. Glutamate, diffuses across the synaptic cleft to initiate action potential in the auditory nerve[41].

Conduction of electrical impulses to the brain

From peripheral auditory system, the electrical impulses are carried by the cochlear nerve. It reaches the ventral and dorsal cochlear nucleus and then goes to

superior olivary nucleus. Then to the lateral lemniscus, the inferior colliculus and reach the auditory cortex via the medial geniculate body.

PATHOPHYSIOLOGY OF NIHL

Fundamental equal-energy principle

NIHL results from the interaction of genetic and environmental factors but is generally still postulated by the extent of biological damage caused by noise. The energy level is a function of the sound pressure of noise (in decibels) and of the duration of exposure over time. The equal-energy principle effectively states equal energy will cause equal damage, such that similar cochlear damage may result after exposure to a higher level of noise over a short period of time as would occur after exposure to a lower level of noise over a longer period of time[47].

Environmental factors

Exposure to impulse noise is more deleterious than exposure to steady state noise[48]. Noise trauma can result in two types of injury to the inner ear, depending on the intensity and duration of the exposure: either transient attenuation of hearing acuity a.k.a. temporary threshold shift (TTS), or a permanent threshold shift (PTS)[49]. Hearing generally recovers within 24–48 h after a TTS. The recovery of TTS is a result of reversible uncoupling of OHC stereocilia from the tectorial membrane [14] and/or reversible central gain increase and associated hyperacusis and tinnitus.

However, even when there is recovery of auditory pure tone thresholds, there can be considerable damage to the ribbon synapses, a rapid degeneration termed synaptopathy. This pathology results in loss of connections between the inner hair cells and their afferent neurons in the acute phase of noise-induced cochlear trauma and is most likely a result of glutamate excitotoxicity causing damage to the post-

synaptic terminals. This is referred to as NIHL, as it is not accompanied by a pure-tone threshold shift.

The characteristic pathological feature of NIHL with PTS is the loss of hair cells, particularly the prominent loss of outer hair cells at the basal turn, while loss of inner hair cells was limited. Degeneration of the auditory nerve followed the loss of outer hair cells in both temporal bone histopathology and in a mouse model. A crucial characteristic of hair cell loss due to any cause (noise, ototoxic medications, age) is the inability of mammalian sensory cells to regenerate.

With sufficient intensity and duration of noise, not only the hair cells but the entire organ of corti may be disrupted. Destruction of the organ of corti can be the result of two mechanisms: mechanical destruction by short exposure to extreme noise intensities or metabolic decompensation after noise exposure over a longer period of time. Mechanical destruction is acquired by exposure to noise intensities above 130 dB sound pressure level (SPL) leading to disassociation of the organ of Corti from the basilar membrane, disruption of cell junctions, and mixing of endolymph and perilymph. The pathology observed as a result of metabolic decompensation includes stereocilia disruption, swollen nuclei, swollen mitochondria, cytoplasmic vesiculation, and vacuolization.

Current theories of metabolic damage center on the formation of free radicals or reactive oxygen species (ROS) and glutamate excitotoxicity evoked by excessive noise stimulation, followed by activation of signalling pathways leading to cell death. ROS emerge immediately after noise exposure and persist for 7–10 days thereafter, spreading apically from the basal end of the organ of Corti, thus widening the area of necrosis and apoptosis.

Glutamate is the excitatory neurotransmitter that acts at the synapses of the inner hair cells with the eighth cranial nerve. High levels of glutamate can over-stimulate postsynaptic cells and cause swelling of cell bodies and dendrites, a process referred to as glutamate excitotoxicity. Another consequence of noise exposure is an increase of free calcium (Ca2+) in outer hair cells immediately after acoustic overstimulation contributed to by both entry through ion channels and liberation from intracellular stores. Ca2+ overload can also trigger apoptotic and necrotic cell death pathways independent of ROS formation [30]. Aside from direct effects on the auditory system, noise also can cause psychological and physiological stress.

The hypothalamus-pituitary-adrenal (HPA) axis can modulate the sensitivity of the auditory system and be activated by acoustic stress. Mice lacking corticotropin-releasing factor receptor (a critical factor in HPA function) in the cochlea exhibited loss of homeostasis and protection against noise-induced hearing loss, leading to an increased susceptibility to noise trauma.

Genetic factors

The genetic susceptibility to NIHL has been clearly demonstrated in animals. Mouse strains (C57BL/6 J) exhibiting age-related hearing loss were shown to be more susceptible to noise than other strains. Also, several heterozygous and homozygous knockout mice including Cdh23, Pmca2, Sod1, Gpx1, Trpv4, Vasp, and Hsf1were shown to be more sensitive to noise than their wild-type littermates.

These studies indicate that there are some genetic deficits that disrupt specific pathways and structures within the cochlea and predispose the inner ear to NIHL[8]. Screening of Single Nucleotide Polymorphisms (SNPs) of different genes known to play a functional and morphological role in the inner ear has been adopted. SNPs are common point mutations in the genome (occurring every 100 – 300 base pairs), and

their genotyping is believed to be a successful tool in analysing the genetic background of complex diseases, such as NIHL. In such studies, a disease susceptibility allele is expected to occur more often among susceptible groups than resistant ones.

The most promising results were obtained for the inner ear potassium (K+) ion recycling and heat shock protein (HSP) genes. K+ recycling genes are indispensable for the process of hearing, as evidenced by the fact that multiple mutations in these genes (GJB2, GJB3, GJB6, KCNE1, KCNQ1 and KCNQ4) lead to both syndromic and non-syndromic forms of hearing loss. HSPs form a group of conserved proteins assisting in synthesis, folding, assembly and intracellular transport of many other proteins. HSPs are ubiquitously expressed in cells under physiological and pathological conditions, and their expression increases under stressful conditions, including noise exposure having protective effects[8]. Recently, the significance of genetic variation in NIHL development has also been shown for otocadherin and myosin genes.

Age factor

Aging is associated with a down-regulation in metabolism, which may underlie an increased sensitivity to stress agents and a decreased repair of tissues following stress. In the auditory system this could lead to increased sensitivity to noise induced hearing loss (NIHL) with age.

Miller et al in 1998 demonstrated that ABR threshold shifts and hair cell losses which followed noise exposure increased with increasing age[50].

Indora et al in 2017 conducted a study in Delhi among 35 traffic policemen working at heavy traffic junctions who are continuously exposed to high level of noise. The objective was to assess the hearing pathway by means of brainstem evoked

response audiometry (BERA), mid-latency response (MLR), and slow vertex response (SVR). The study showed increase in the latencies of waves I and III of BERA, and IPL I-III. The MLR and SVR waves showed no significant changes in studied policemen. It was inferred that chronic exposure noise resulted in delayed conduction in peripheral part of the auditory pathway, ie, auditory nerve up to the level of superior olivary nucleus; no impairment was observed at the level of sub-cortical, cortical, or the association areas.[21]

Hidden hearing loss and noise-induced hearing loss

Some people have no obvious hearing loss but find it difficult to hear clearly in noisy environments. The hearing acuity and speech discrimination rate of those people is abnormal. This phenomenon is known as 'hidden hearing loss'[51]. It is difficult for regular hearing tests (PTA) to detect hidden hearing loss in time to take steps to prevent further damage. At the early stage of noise induced hearing loss, noise can cause transient changes in ribbon synapses which are located between hair cells and spiral ganglion neurons.

The quantity and quality of ribbon synapses significantly decreases after noise exposure. Although hearing can recover several days later, the number of ribbon synapses does not totally recover[52]. Various perceptual abnormalities then begin to emerge, including tinnitus and hyperacusis[53]. Long-term noise exposure can lead to continuous apoptosis of hair cells and degeneration of spiral ganglion neurons, which results in the decrease of speech discrimination rate and an increase of the hearing threshold, eventually causing a permanent hearing impairment[54]

Tinnitus in Sensory neural hearing loss

Research proves that many subjects with tinnitus doesn't no signs of hearing loss up to 8 kHz in the audiometric test, which is routinely performed using pure tone

sound stimuli. It is thought that one of the possible causes of tinnitus in these patients may be related to the cochlear disorder in the basal region[55].

Therefore, it was thought that it would be useful to extend this range to 16 kHz in audiometric tests when evaluating patients with tinnitus[56]. Another factor in the development of tinnitus may be the cochlear synaptopathy, which is described as the loss of inner hair cell synapses without any evidence of increased hearing thresholds[57]. It can be detected by ABR and electrocochleography.

Measure of Noise pollution

Daily noise exposure level [$L_{EX,8h}$] is the average noise energy a person is exposed to during a working day. L_{EX} forms the basis of risk assessment. The formula is $L_{EX,8h} = L_{Aeq} + 10\ Log(T/8)$, where L_{Aeq} stands for equivalent continuous level and T is the time in hours of noise exposure per day. NIHL can be caused by sound levels above 75dB. Most cases of NIHL arise over years of exposure.

Peak sound pressure [P_{peak}] is the maximum value of frequency weighted instantaneous sound pressure level. It is measured by a sound level meter. An excessive P_{peak} in excess of 150dB can cause instantaneous hearing loss.

Noise assessment is pivotal is occupation involving noisy environment. In some cases, it provides prima facie evidence. It is an ordain for risk stratification, pathology identification, compensatory action like eliminate risk/establish controls, provide hearing protection & conservation, provide information to workers, health surveillance and review them. Occupational anamnesis is thus an essential tool for risk identification. It is important to detail the exposure to allow correlation between the exposure and the signs and symptoms of NIHL.

Background Noise Measurement:

Smartphones are an effective tool for noise measurement. The principal factor is microphone, with phones incorporating one or more internal microphones[58].

Filter type(i.e., Max RF, Enhanced RF) and its impedance are factors determining the quality of the readings[59]. The microphone responds to vibrations in the air, converting them into electrical current fluctuations. Then, these electrical signals can be evaluated as a signal in the time domain or in the frequency domain[60]. The sound signal can be processed and its loudness computed over long time intervals[61].

Audiometric configuration of NIHL

Pure tones are presented to each ear in order of increasing frequency and the lowest perceived intensity for each tested frequency is recorded, which is called threshold for that frequency. The average increase in the threshold off speech frequencies gives the magnitude of hearing loss in decibels.

Air conduction is tested by microphones in the external auditory canal and bone conduction is tested by placing a specialized microphone over the mastoid process in the postauricular region. The rationale is that impairment in hearing will reduce the perception of low intensity sounds[6].

Audiometric inferences are used to: (1)

Determine the severity of hearing loss (2)

Diagnose the type (i.e., conductive, sensorineural, or mixed) by comparing air- and bone-conduction thresholds.

(3) Determine the need for rehabilitative measures.

The audiometric configuration resulting from noise exposure may be similar for either traumatic loss or NIHL. A typical progression of hearing loss as a function of exposure to industrial noise.

The maximum hearing loss occurred in the 4000-Hz region which forms the 'notch' and it's because the hair cells in this region were more susceptible to damage.

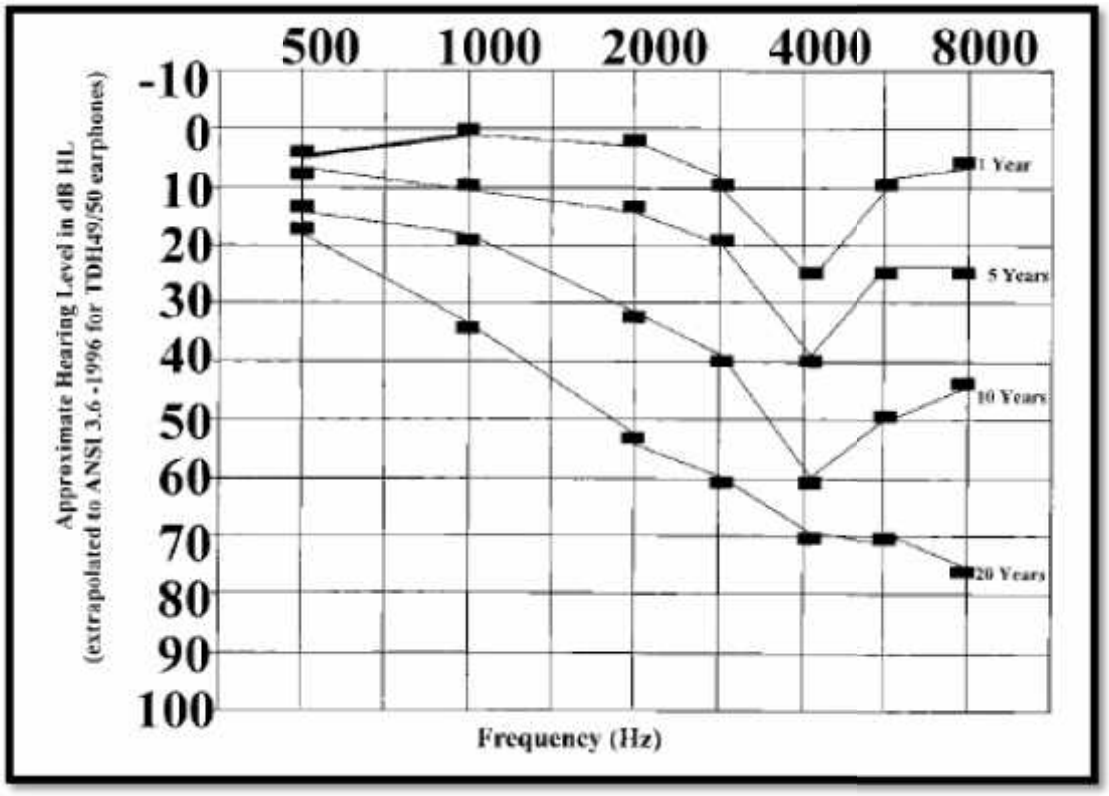


Fig 9: Typical progression of noise-induced hearing loss (NIHL) as a function of years of exposure. (Adapted from Newby HA. *Audiology*, 4th ed. Englewood Cliffs)

As the exposure continues, the "notch" typically involving 3000 to 6000 Hz tends to broaden to the frequency region below 3000 Hz, and the magnitude of hearing loss at 3000 to 6000 Hz increases.

The audiometric configurations of NIHL cover a wide range. The three representative audiometric configurations of NIHL are:

Type I → Audiometric configuration typical of only several years of exposure to noise, is characterized by near normal hearing through 2000 Hz.

Type II → Representative of many years of excessive exposure to noise, is typically illustrated by audiograms revealing hearing loss extending into the lower frequencies (i.e., below 2000 Hz).

Type III → Less common. More extreme case. Hearing is near normal for the low frequencies only, and the audiometric configuration has a precipitous slope into the high frequencies[52].

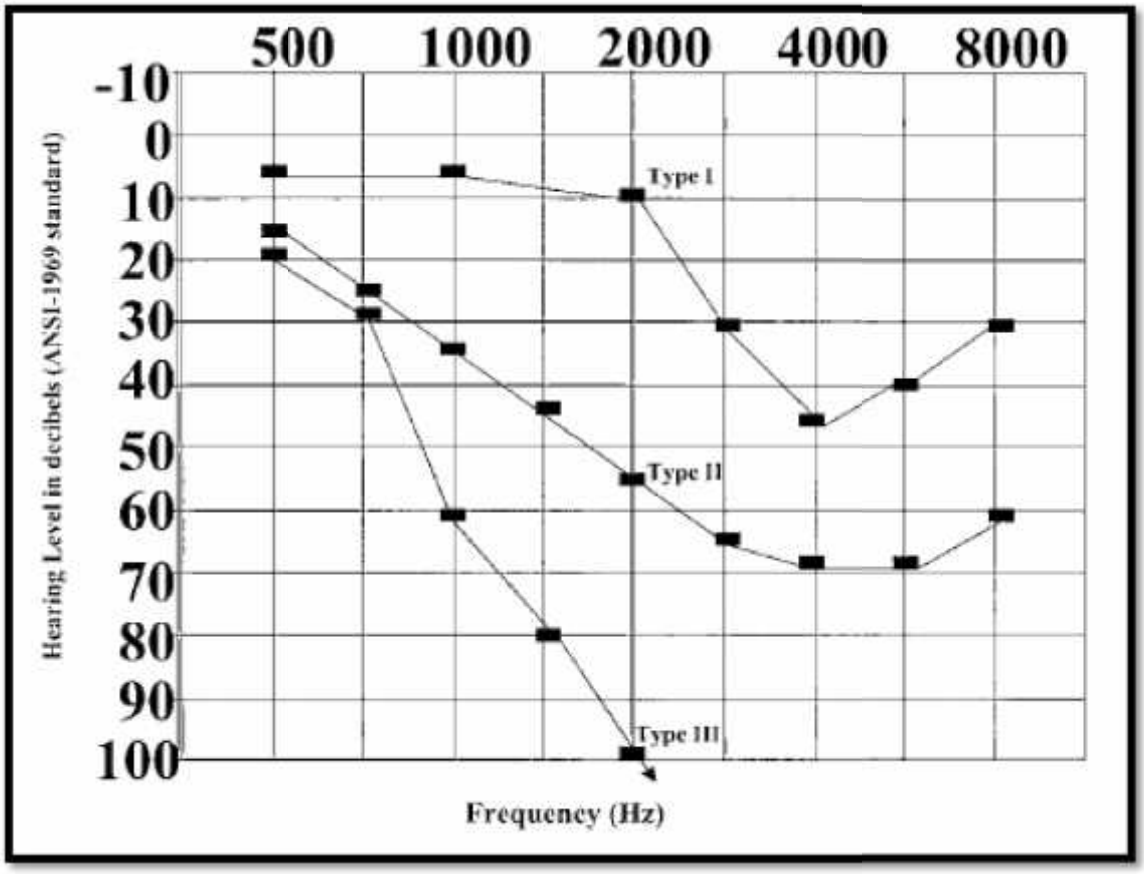


Fig 10: Audiometric configurations representative of three types of NIHL. (Adapted from Newby HA. *Audiology*, 4th ed. Englewood Cliffs)

PTA does not distinguish between IHC and OHC dysfunction. Noise exposure can cause substantial cochlear neuropathy without affecting sensitivity to weak

sounds. This hidden hearing loss, may be the physiological basis for many of the cases of hearing disability with a normal audiogram. This is supported by evidence from human studies that noise exposure may cause perceptual difficulties without affecting the audiogram[53].

Noise-Induced Temporary Threshold Shifts

Davis et al. explored tolerance of human ear sustained sound. Subjects were exposed to sound in turns exposing one ear at a time and measuring the recovery of sensitivity from their temporary threshold shift (TTS). If recovery was complete, defined as within 5 dB in 24 hours, the exposure was considered 'Safe'.

When ears were exposed to a bullhorn pure tone, the TTS was always greatest at a frequency about half an octave above that of the exposure tone. Sometimes the threshold at the exposure tone frequency was shifted no more than 10 dB, while half an octave higher the shift 2 min after exposure might be 50 dB. Prolonged exposure to an intense 500 Hz tone or to noise of wide frequency spectrum caused severe speech discrimination loss at a low (40 dB SPL) presentation level but only moderate loss at a high (100 dB SPL) level[57].

Kujawa et al showed that acoustic overexposure for 2 h with an 8-16 kHz band of noise at 100 dB SPL in the mouse caused a moderate, but completely reversible, threshold elevation as measured by ABR. The absence of permanent changes in the OAEs indicated that the exposure left outer hair cells (OHCs) intact. Using confocal imaging of the inner ear, they found that despite the normal appearance of the cochlea and normal hearing thresholds there was an acute loss of the ribbon synapses located on the medial side of the inner hair cell (IHC) connected to high-threshold, low-SFR ANFs followed by a delayed progressive diffuse degeneration of the cochlear nerve[63].

Speech Recognition Threshold

SRT is the lowest hearing level at which the patient correctly recognizes speech stimuli 50% of the time for each ear independently. The most commonly used and recommended speech stimuli for obtaining SRTs are spondaic words.A spondee is a bisyllabic word with equal stress on both syllables (e.g., baseball, hotdog, airplane).

Because PTA reflects hearing thresholds in the speech frequency region and the SRT is measured using speech stimuli, close agreement between the latter two measures is anticipated. Functional hearing loss (i.e., pseudo-hypoacusis) should be considered whenever there is an SRT-PTA discrepancy of more than 12 dB[6].

In addition to functional hearing loss, a variety of other reasons must be considered when an SRT-PTA discrepancy exists, including (1) audiometer malfunction; (2) eighth cranial nerve disorders; (3) significant hearing loss with an island of normal hearing; and (4) cognitive or language disorders.

Although the literature is not extensive, there is some evidence that listeners with a history of noise exposure, but with near-normal threshold sensitivity, show deficits in complex discrimination tasks.

Word Recognition Measures in NIHL

There are four primary reasons listeners with hearing loss have decreased word recognition skills:

(1) Reduced audibility of the speech cues that are important for correct recognition of words.

(2) Cochlear distortions that are manifested as reductions in frequency selectivity, temporal resolution, gap detection, and frequency and/or intensity discrimination.

(3) Central auditory nervous system deficiencies.

(4) Deficits in cognitive processing.

Regardless of the cause of diminished word recognition ability, word recognition measures, particularly in quiet, are poor prognostic indicators of success with amplification for patients with NIHL. Word recognition scores are excellent for some when presented in quiet and become difficult in noisy environments.

Oto-Acoustic Emission Testing

Thomas Gold introduced OAEs in the year 1948 and David Kemp demonstrated its use in 1978. The taxonomy arises from nonlinear, electromechanical distortion within the human cochlea, which creates a source of energy that is measured in the outer ear as emission.

Indications of OAE include:

• Infants over 90 days old and children up to 4 years of age

• Children and adults who are unable to cooperate with other methods of hearing testing (e.g., individuals with autism or stroke)

• Children with developmental or delayed speech or language disorders

• Individuals with tinnitus, acoustic trauma, noise induced hearing loss, or sudden hearing loss

• Individuals with Auditory Neuropathy or auditory processing disorder (APD), also known as central auditory processing disorder (CAPD)

• Individuals with Sensorineural Hearing Loss confirmed by audiometry

• Individuals with abnormal auditory function studies or failed hearing exam

• Individuals who may be feigning a hearing loss

• Monitoring of ototoxicity in individuals before, during, and after administration of agents known to be ototoxic (e.g., aminoglycosides, chemotherapy agents)"[64]

OAEs can be measured with a sensitive microphone in the ear canal and provide a non-invasive measure of cochlear amplification.

There are two types of OAEs in clinical use.

(1) TEOAEs are evoked using a click stimulus. The evoked response from a click covers the frequency range up to around 4 kHz[63].

(2) DPOAEs – When two tones are presented simultaneously to a healthy cochlea, the response measured in the ear canal will contain several tones that are not present in the eliciting stimuli[64]. These additional tones are called distortion products (DPs) which are attributed to the nonlinear processes of the normally functioning cochlea. DPOAEs are effective in identifying subjects with known sensorineural hearing loss[65].

The most robust one is at the frequency equal to 2f1-f2 and is the most commonly measured, where f1 indicates the lower frequency tone and f2 indicates the higher frequency tone of the pair. An f2/ f1 ratio of 1.22 produces the largest 2f1-f2 DPs, on the average, in adults. Optimal stimulus intensities range from 50 to 70 dB SPL, with the higher frequency tone (L2) presented 10 to 15 dB less intense than the lower frequency tone (L1). Under these stimulus conditions, the interaction of primary tones on the basilar membrane occurs near the region of f2. It is the cochlear integrity being assessed using these stimulus parameters[65].

METHODOLOGY

STUDY DESIGN: Cross sectiontional Observational study

STUDY PERIOD: January 2019 to December 2019 [1 year]

SAMPLE SIZE: 80 traffic policemen

ETHICAL CLEARANCE: Obtained from the institutional ethical committee. **Ref: MDC/DOME/61**

INCLUSION CRITERIA: Traffic police personnel from the two divisions of Belagavi city.

- Age group of 18-50 years.
- Candidates who have noise exposure more than 85dB for 8 hours a day for a period of six months or more.
- Excluded certain traffic police personnel as mentioned under exclusion criteria by history, clinical examination, and relevant investigations.

EXCLUSION CRITERIA:

- Candidates with acute or chronic otitis media – squamosal and mucosal.
- Congenital causes and those with family history of hearing loss.
- Traumatic causes of hearing loss including head trauma and ear surgery.
- Sudden sensory neural hearing loss.
- Candidates with history of ototoxic drugs and with any systemic illness and metabolic disorders causing SNHL

MATERIALS AND MEHODS:

- Written informed consent from all participants. Prior to start, the purpose and procedures were explained.

- **Interview and Data Collection**:

Data collection by Interviewer-administered structured questionnaire regarding awareness, detailed medical history, history of trauma, ear surgery, occupational history, duration of exposure, familial history of hearing loss. Personal habits including alcohol intake and smoking were included. Questions were read aloud to subjects and subject response recorded.Questionnaire includedSmithquestionnaire, which subjectively assessed the severity of hearing loss.Questions were related to self-assessment of hearing quality, attitude and knowledge about NIHL, annoyance, correlation between other associated factors. Presence of NIHL were statisticallyanalysed.

These questionnaires were translatedinto Kannada, Marathi and Hindi, and reverse translation was carried out make sure that the meaning of the content remains the same. These translatedquestions were proofread by a native speaker of these languages as well as having knowledge of English too.

- All traffic policemen underwent general and systemic examination.
- An elaborate ENT assessment including tuning fork tests, otoscopic and examination under microscopy were carried out.
- **Background Noise Measurement:**

Road traffic noise measurements were taken in smartphone using Decibel X Pro application. Decibel X Pro has highly reliable, pre-calibrated measurements and supports dBA, dBC. It turns your iOS device into a professional sound level meter, precisely measures the sound pressure level (SPL) all around you[1]. It calibrates measurements ranging from 30 dB up to about 130 dB. Its precision is as good as real

SPL devices. Noise was measured from 10 different light and heavy traffic zones in Belgaum city and it ranged from 71.2 to 95 dB.

- **Hearing assessment by PTA:**

Assessment of hearing was done separately for each ear in a sound-proof room, on an out-patient basis, with MAICO MA53 audiometer. Active noise cancelling headsets with the option to toggle the active noise cancellation feature were checked, to make sure the active noise cancellation feature was turned on.

Fig 11: **Pure tone audiometry in progress**

The hearing thresholds were assessed for air conduction at frequencies of 125-8000 Hz and bone conduction at 250-4000 Hz, using 10db increments. Type, degree of severity and frequency pattern of pure-tone thresholds studied.

The following audiometric patterns of noise-induced threshold shift if present, in one or both ears is taken as NIHL.

1. Pure-tone thresholds at 0.5 and 1 kHz are 15 dB HL better than higher frequencies[66].

2. Ahigh-frequency notch - When thresholds at 3, 4, or 6 kHz exceeded the average threshold at 0.5, 1 and 2 kHz by ≥15dB HL→4KHz notch and then worsening hearing loss curve in high frequencies as disease advances.

3. Recovery at 8kHz →8 kHz threshold should be >10 dB lower (better) than the maximum (poorest) threshold value for 3, 4, or 6 kHz[66].

Table 1: Interpretation of Audiogram: Degree of hearing loss[4].

DEGREE OF HEARING LOSS	HEARING THRESHOLD
Minimal	16-25dB
Mild	26-40dB
Moderate	41-55dB
Moderately Severe	56-70dB
Severe	71-90dB
Profound	>90dB

- The threshold for hearing at different hertz was taken. Each ear was evaluated separately.
- The speech recognition threshold (SRT) is noted. It is defined as the lowest level at which a person can identify a sound from a closed set list of disyllabic words. It was compared to Noise induced hearing loss. Both ears tested.
- Speech Identification score (SIS) was also recorded and its association with NIHL assessed. Both ears tested.

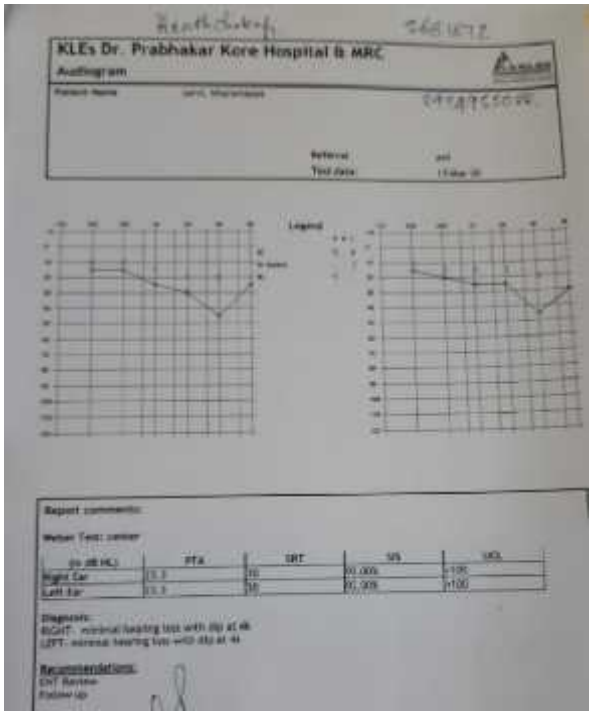


Fig 12: **PTA report of Bilateral minimal Noise induced hearing loss**

- Statistical correlation of the SRT and SIS values with presence of Noise induced hearing loss was assessed.

- **<u>OAE Assessment</u>**

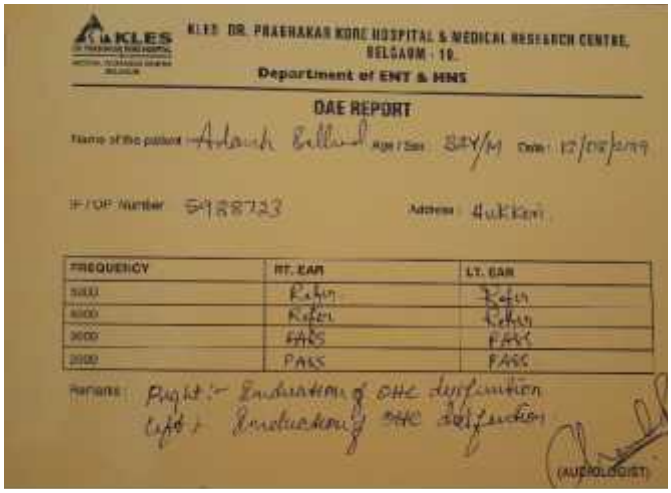
A normal external ear and tympanic membrane with a clean external auditory canal is a pre-requisite.

DPOAEwith Biologic AUDUX-Pro machine in a sound-proof room was measured. For DPOAEacquisition a special OAE probe was used. This probehas built-in miniature speakers and a microphone, whichwas inserted into the external auditory canal of the patient and sealed with a flexible rubber mold.

The DPOAE was obtained by simultaneously presenting two pure tones, f1 and f2. These pure tones, called primary frequencies, have a ratio of 1.22. In this study, we used the equivalent response to 2f1-f2, and the intensity ratio was L1 = 65 dB and L2

= 55 dB SPL (sound pressure level). Thef2 frequency was swept from low to high for eachmeasurement and considered for analysis frequencies (f2) of 2, 3, 4 and 6 kHz [5].

Fig 13: **Otoacoustic Emission testing in progress**



KLES DR. PRABHAKAR KORE HOSPITAL & MEDICAL RESEARCH CENTRE, BELGAUM - 10.

Department of ENT & HNS

OAE REPORT

Name of the patient: Adarsh Bellad Age / Sex: 22Y/M Date: 12/02/2019

IP / OP Number: 5988723 Address: Gokak

FREQUENCY	RT. EAR	LT. EAR
5000	Refer	Refer
4000	Refer	Refer
3000	FAIL	FAIL
2000	PASS	PASS

Remarks: Right :- Evaluation of OHC dysfunction
Left :- Evaluation of OHC dysfunction

(AUDIOLOGIST)

Instrument indicates ‘‘Pass’’ or ‘‘Refer’’ for each ear. ‘‘Refer’’ is equivalent to an absent DPOAE.

Fig 14**: OAEreported as Bilateral Outer hair cell dysfunction**

DPOAE was not administered when otoscopic examination could not be conducted or in cases of bilateral occlusions, presence of blood, a foreign object/substance in both ears, impacted wax in both ears, the participant was unwilling.

- Correlation between OAE and normal hearing threshold PTA were analysed. Special note was taken on subjects with Normal audiometric threshold.
- Also, association between subjective assessment of hearing loss by Smith scale and OAE proven NIHL documented.

SATISTICAL METHODS

- Data was entered in excel spread sheet by using numerical codes. Data analysis was done using Microsoft excel software or SPSS. Chi-square test and multiple logistic regression were used for the analysis.
- The level of significance was 5% ($p < 0.05$) and the confidence interval was constructed with 95% statistical confidence.
- Demographic characteristics and study variables wereanalysed using descriptive statistics.
- Means, standard deviations and ranges were reported for continuous variables and percentages were reported for categorical variables.
- Research for possible asymmetries in the DPOAE measurement between the right and left ears (n = 80) through the Wilcoxon test of signed stations.
- The Spearman correlation analysis for determining the correlation between the results of pure-tone audiometry and DPOAE (n = 80)

RESULTS

A total of 80 traffic police personnel of tier two city Belagavi where 160 ears were assessed for NIHL by undergoing history taking & examination, Interviewer-administered questionnaire, Pure tone audiometry and Distortion-product otoacosutic emissions. All participants were male traffic police personnel serving fir a duration of more than 2 years.

Out of 80 traffic police examined, 56.25% (45) had hearing loss detected by PTA. The prevalence of early noise induced hearing loss assessed with DPOAE is 62.5% (50).

Out of both right and left 80 ears, unilateral cases as detected by audiometry were 6.25% (5: right-1 and left-4) and bilateral cases were 50% (40). And 11.25% (9: right-3 and left-6) of unilateral NIHL and 55% (44) of bilateral NIHL was detected by DPOAE.

All road traffic police personnel worked for a minimum duration of 8 hours per day on road.

Age:

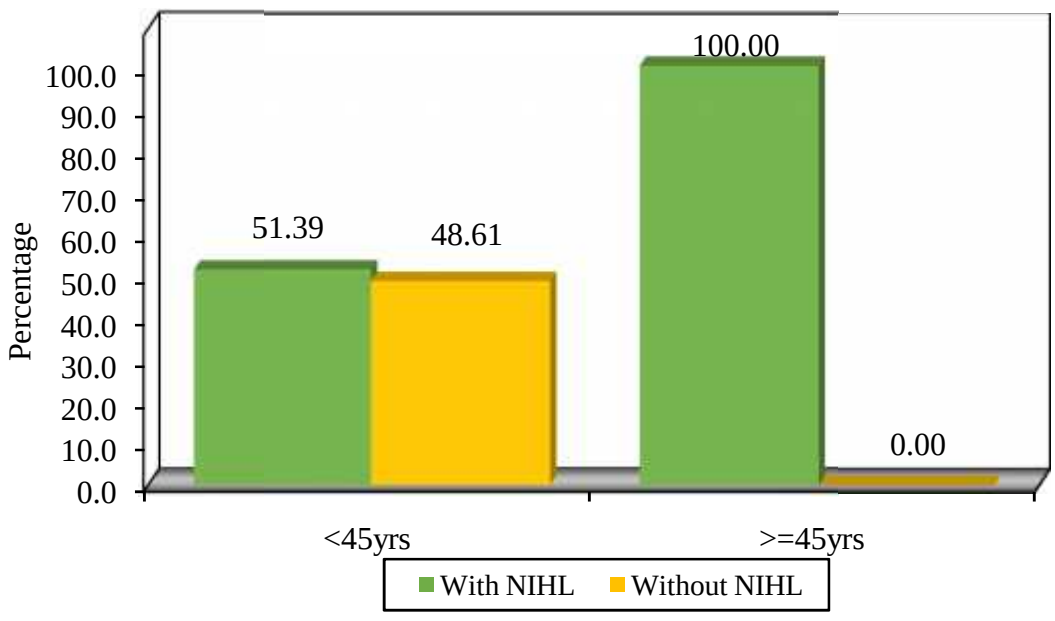
The mean age group is 36.84±2.6 years. 43.75% (35) of the traffic police belonged to the age group 21-30 years, 33.75% (27) in the age 31-40 years and 22.5% (18) in the age 41-50 years. Prevalence increases as age advances in our study, with 100% (8) after 45 years.

Table 2: Association between NIHL by age groups:

Age groups	With NIHL	%	Without NIHL	%	Total	%
<45yrs	37	51.39	35	48.61	72	90.00
>=45yrs	8	100.00	0	0.00	8	10.00
Total	45	56.25	35	43.75	80	100.00
Chi-square with Yates's correction = 5.0792 P = 0.024 0*						

*p<0.05

Graph 1: Association between total NIHL by age groups



Duration of exposure:

Direct correlation with duration of exposure and occurrence of NIHL is seen in our study. 83.78% (31) of the participants working for a duration of more than 5 years have developed NIHL as per study. There is a statistically significant positive association between increasing age and duration of exposure to prevalence of NIHL.

Table 3: Association between NIHL and duration of exposure

Duration	With NIHL	%	Without NIHL	%	Total	%
<5yrs	14	32.56	29	67.44	43	53.75
≥5yrs	31	83.78	6	16.22	37	46.25
Total	45	56.25	35	43.75	80	100.00
Chi-square= 19.1750 P = 0.0001*						

*p<0.05

Graph 2: Association between total NIHL by duration

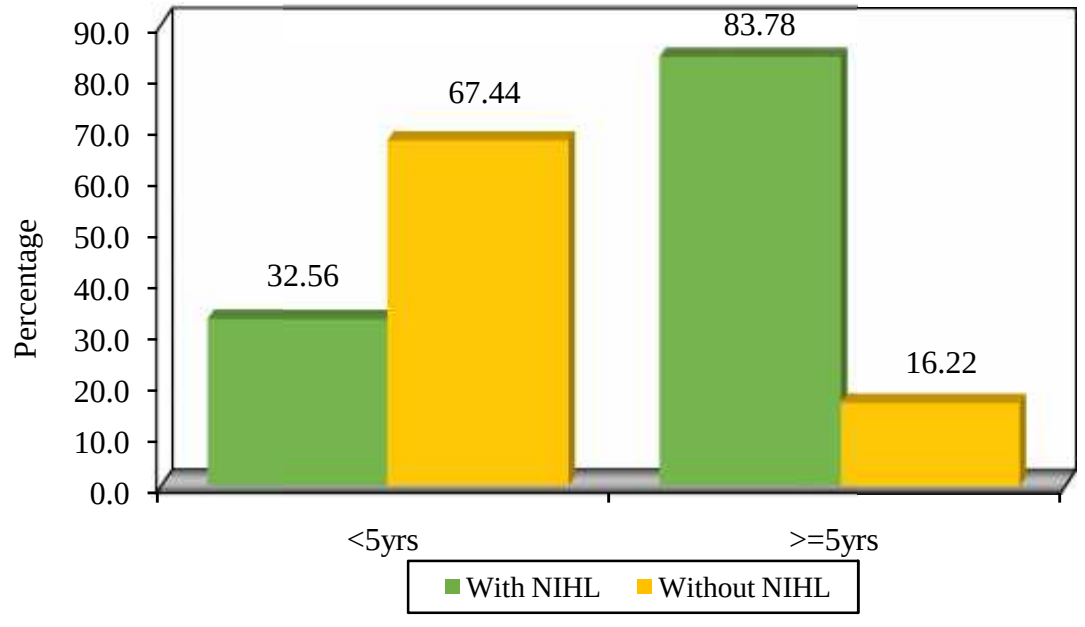


Table 4: Comparison of with and without total NIHL by mean age, years of exposure and per week exposure by t test

Variable	Total NIHL	Mean	t-value	P-value
Age in years	With NIHL	36.84	5.9221	0.0001*
	Without NIHL	27.66		
Years of exposure	With NIHL	6.16	5.9107	0.0001*
	Without NIHL	2.89		
Per week exposure	With NIHL	58.78	0.6626	0.5095
	Without NIHL	58.31		

*p<0.05

Graph 3: Comparison of with and without total NIHL by mean age, years of exposure and per week exposure

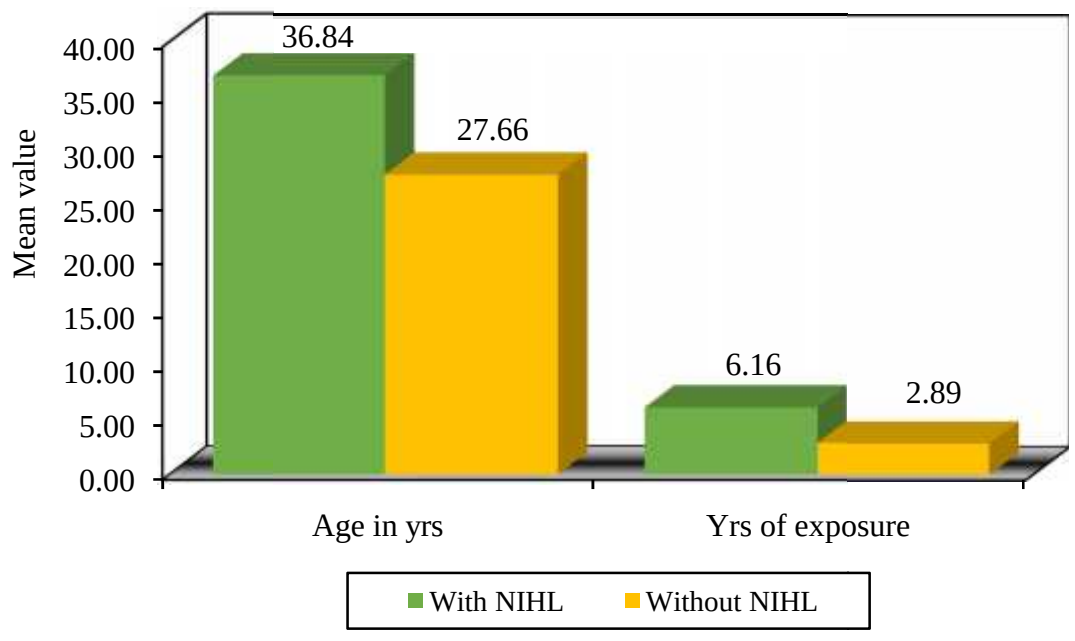


Table 5: Smith questions wise responses of respondents

Smith questions	Never	%	Occasional	%	Often	%	Always	%
S1: Noisy environment work	0	0.00	0	0.00	0	0.00	80	100.00
S2: Trouble following conversation when 2 or more people talk?	34	42.50	17	21.25	23	28.75	6	7.50
S3: Turn TV volume high?	34	42.50	19	23.75	21	26.25	6	7.50
S4: Misunderstand words or ask to repeat?	37	46.25	18	22.50	22	27.50	3	3.75
S5: Do people get annoyed?	47	58.75	18	22.50	13	16.25	2	2.50
S6: Make inappropriate response due to misunderstanding?	45	56.25	22	27.50	10	12.50	3	3.75
S7: Difficulty hearing whisper?	47	58.75	17	21.25	13	16.25	3	3.75
S8: Cause family arguments	52	65.00	20	25.00	7	8.75	1	1.25
S9: Problem listening TV/radio?	59	73.75	12	15.00	9	11.25	0	0.00

Graph 4: Smith questions wise responses of respondents

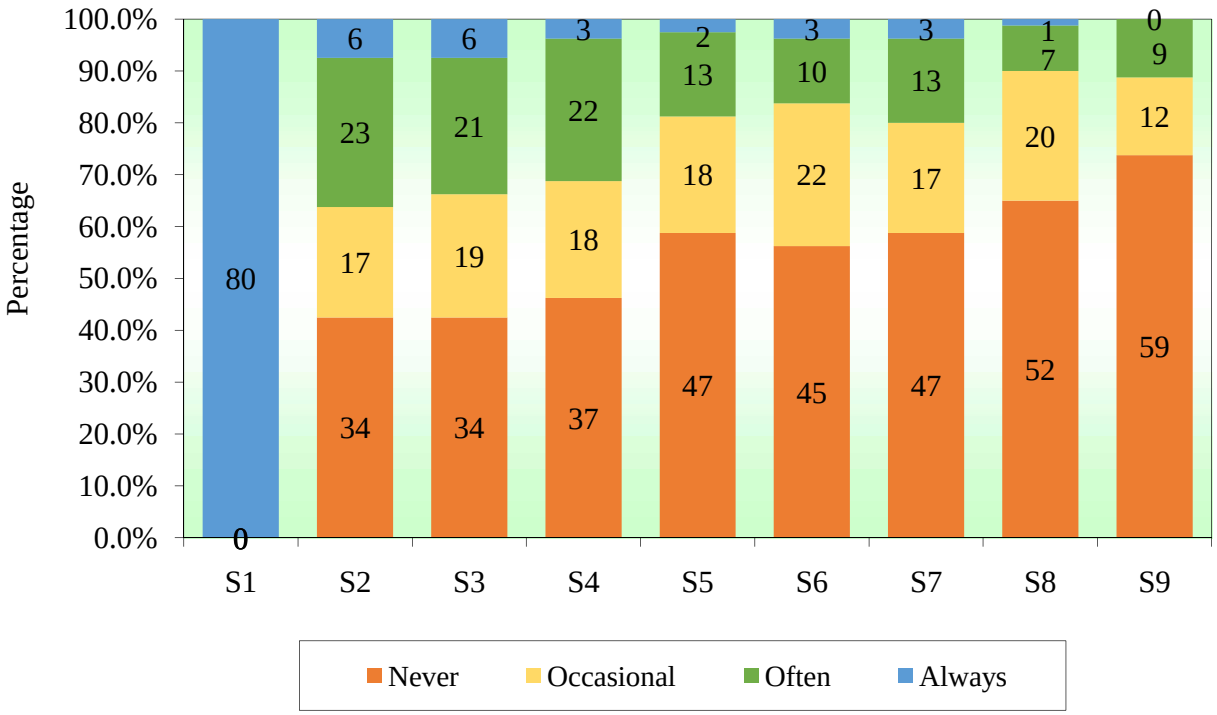


Table 6: Comparison of each question of Smith scale to presence and absence of NIHL by Mann-Whitney U test

Smith Questions	Mean With NIHL	Mean Without NIHL	p-value
S1	3.00	3.00	1.0000
S2	1.78	0.03	0.0001*
S3	1.73	0.03	0.0001*
S4	1.56	0.03	0.0001*
S5	1.11	0.00	0.0001*
S6	1.11	0.03	0.0001*
S7	1.13	0.03	0.0001*
S8	0.82	0.00	0.0001*
S9	0.67	0.00	0.0004*
Total	12.91	3.14	0.0001*

Graph 5: Comparison of each question of Smith scale to presence and absence of NIHL

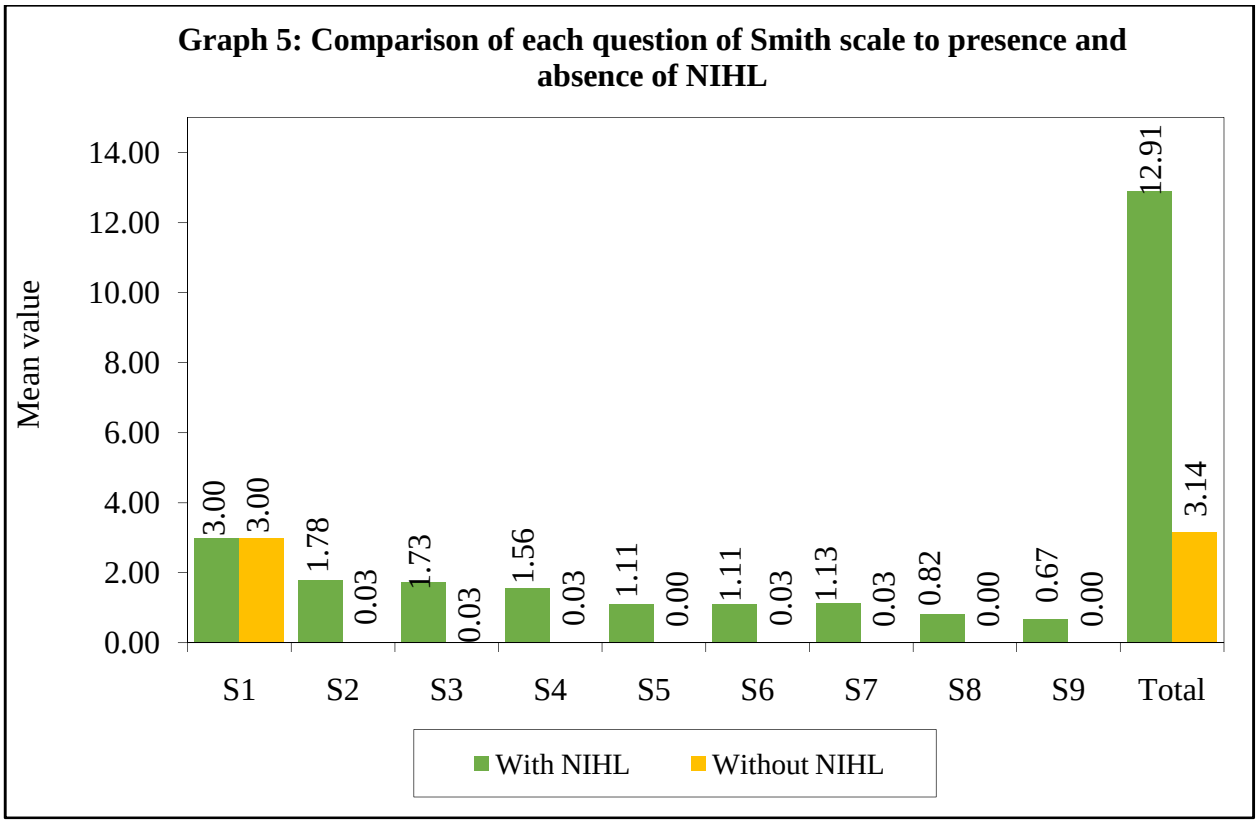


Table 7: Prediction of NIHL with Smith Questionnaire

Smith grades	With NIHL	%	Without NIHL	%	Total	%
Normal	0	0.00	35	100.00	35	43.75
Mild hearing loss	16	35.56	0	0.00	16	20.00
Moderate hearing loss	24	53.33	0	0.00	24	30.00
Severe hearing loss	5	11.11	0	0.00	5	6.25
Total	45	100.00	35	100.00	80	100.00

Graph 6: Association between Smith grades with presence of NIHL

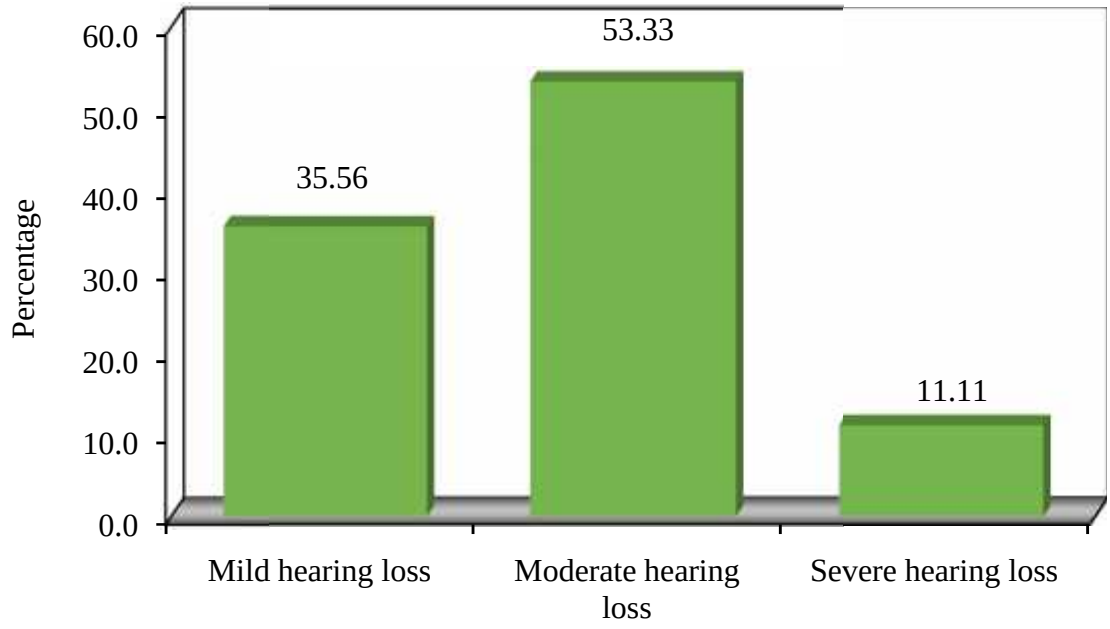
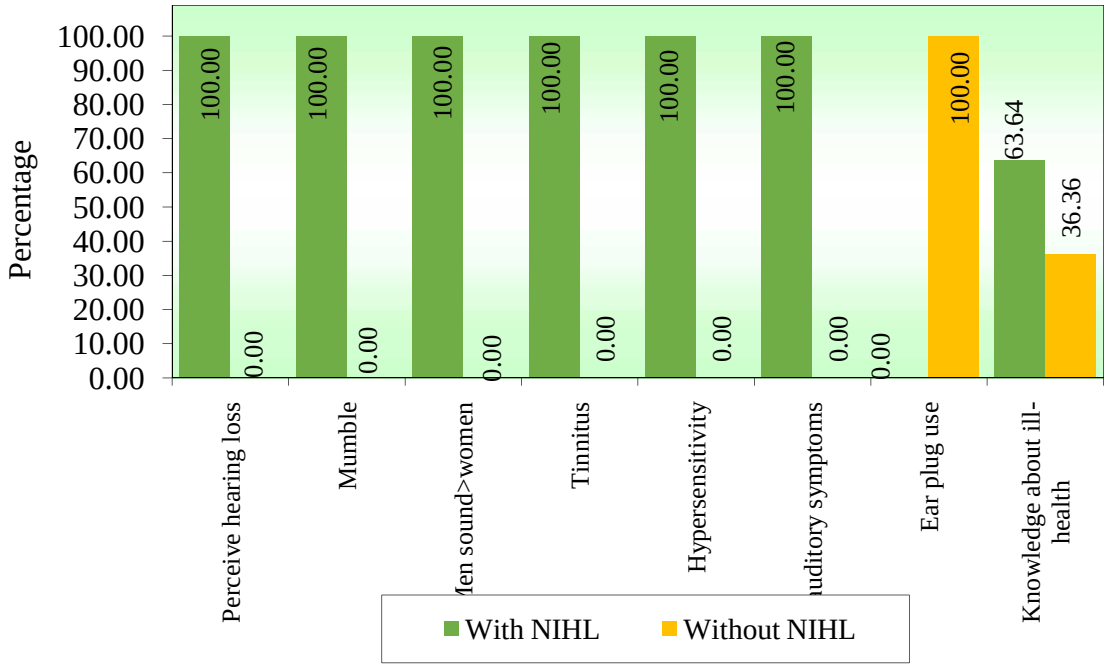


Table 8: Association between NIHL with presence of associated factors

Associated factors	With NIHL	%	Without NIHL	%	Total	%	Chi-square	p-value
Perceive hearing loss								
No	37	51.39	35	48.61	72	90.00	6.9140	0.0090*
Yes	8	100.00	0	0.00	8	10.00		
Mumble								
No	17	32.69	35	67.31	52	65.00	33.5040	0.0001*
Yes	28	100.00	0	0.00	28	35.00		
Men sound>women								
No	26	42.62	35	57.38	61	76.25	19.3810	0.0001*
Yes	19	100.00	0	0.00	19	23.75		
Tinnitus								
No	29	45.31	35	54.69	64	80.00	15.5560	0.0001*
Yes	16	100.00	0	0.00	16	20.00		
Hypersensitivity								
No	38	52.05	35	47.95	73	91.25	5.9670	0.0150*
Yes	7	100.00	0	0.00	7	8.75		
Non-auditory symptoms								
No	34	49.28	35	50.72	69	86.25	9.9190	0.0020*
Yes	11	100.00	0	0.00	11	13.75		
Ear plug use								
No	45	63.38	26	36.62	71	88.75	13.0380	0.0001*
Yes	0	0.00	9	100.00	9	11.25		
Knowledge about ill-health								
No	38	55.07	31	44.93	69	86.25	0.2830	0.5950
Yes	7	63.64	4	36.36	11	13.75		
Total	45	56.25	35	43.75	80	100.00		

*p<0.05

Graph 7: Association between NIHL with presence of associated factors



It was observed that a majority were oblivious to the harmful effects of noise. The workplace environment was perceived to be extremely noisy by most of them. It was observed that only 9 subjects used ear plugs and none of the police personnel with NIHL had ever used an ear plug or ear muff, for which the main reason cited was its unavailability. A majority of them did not use any method to protect themselves against noise.

Table 9: Comparison of NIHL with different variables by independent t test

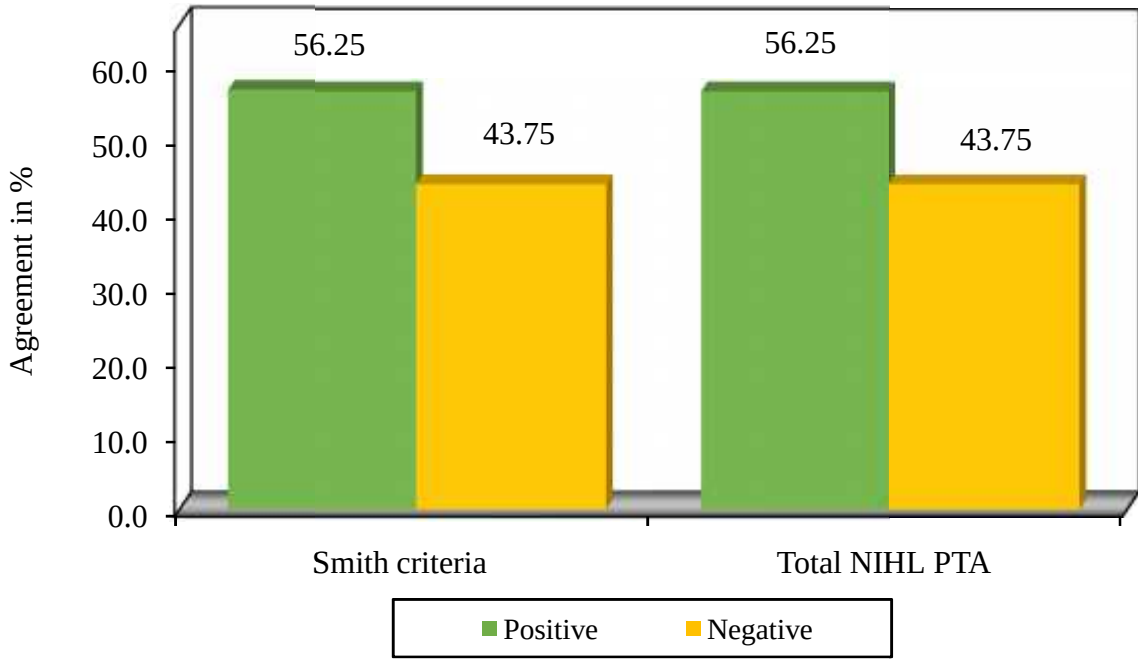
Variables	With NIHL		Without NIHL		t-value	p-value
	Mean	SD	Mean	SD		
R 250Hz	18.56	9.92	8.71	2.53	5.7184	0.0001*
R 500Hz	24.44	13.11	14.57	1.42	4.4293	0.0001*
R 1KHz	27.11	13.63	14.71	1.18	5.3577	0.0001*
R 2KHz	32.11	19.79	14.86	0.85	5.1482	0.0001*
R 4KHz	55.67	21.68	15.86	7.81	10.3418	0.0001*
R 8KHz	41.22	19.40	16.86	5.01	7.2358	0.0001*
R PTA	28.12	14.93	14.75	1.07	5.2811	0.0001*
R SRT	37.44	19.44	19.57	1.42	5.4198	0.0001*
R SIS %	85.24	7.56	92.51	2.39	-5.4734	0.0001*
L 250Hz	18.67	7.42	8.86	2.99	7.3649	0.0001*
L 500Hz	24.11	12.03	14.57	1.42	4.6609	0.0001*
L 1KHz	26.89	12.58	14.86	0.85	5.6399	0.0001*
L 2KHz	31.11	17.87	14.71	1.69	5.4032	0.0001*
L 4KHz	56.56	17.64	19.00	8.56	11.5694	0.0001*
L 8KHz	40.44	16.71	18.57	6.25	7.3446	0.0001*
L PTA	27.79	13.79	14.66	1.14	5.6096	0.0001*
L SRT	37.33	18.27	19.57	1.42	5.7314	0.0001*
L SIS %	85.47	7.30	92.51	2.39	-5.4777	0.0001*

*$p<0.05$

Table 10: Agreement between Smith criteria and NIHL detected by PTA

Smith criteria	NIHL detected by PTA			
	Positive	**Negative**	**Total**	**%**
Positive (>=6)	45	0	45	56.25
Negative (<=5)	0	35	35	43.75
Total	45	35	80	100.00
%	56.25	43.75	100.00	

Graph 8: Agreement between Smith criteria and total NIHL detected by PTA

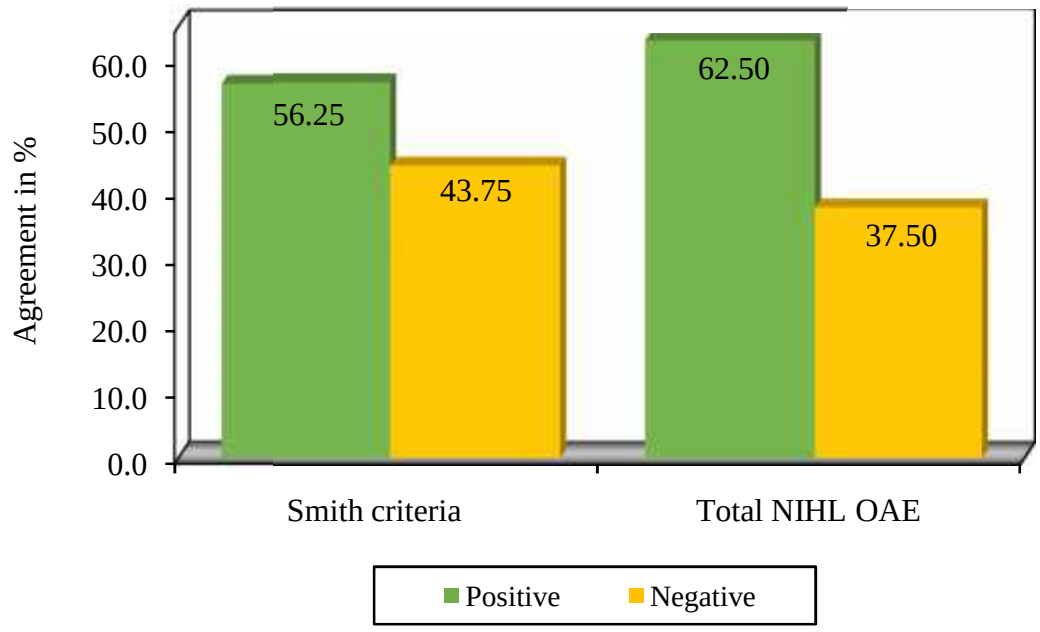


All subjects with NIHL detected by Pure tone audiometry had subjective auditory impairment as assessed by Smith Questionnaire.

Table 11: Agreement between Smith criteria and NIHL detected by OAE

Smith criteria	NIHL detected by OAE			
	Positive	**Negative**	**Total**	**%**
Positive (>=6)	45	0	45	56.25
Negative (<=5)	5	30	35	43.75
Total	50	30	80	100.00
%	62.50	37.50	100.00	

Graph 9: Agreement between Smith criteria and NIHL detected by OAE

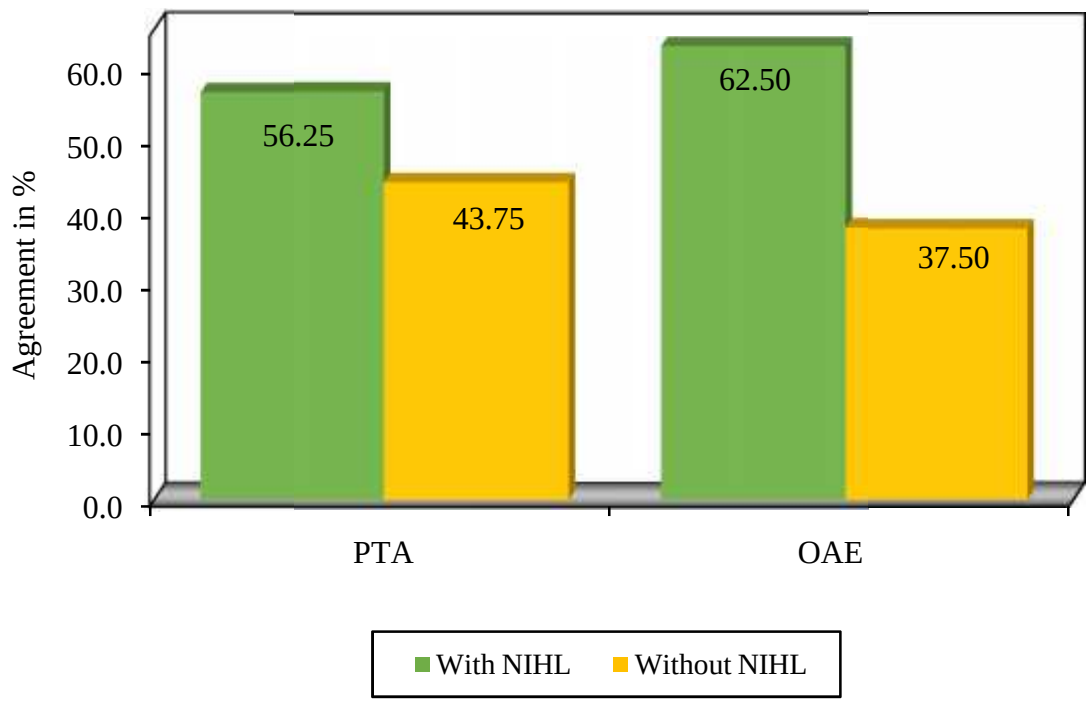


Distortion-product otoacoustic emissions detected NIHL even in subjects who did not experience subjective hearing abnormality. Hence proving higher sensitivity even in pre-clinical phase of NIHL.

Table 12: Agreement between NIHL PTA and NIHL OAE

OAE	NIHL PTA			
	With NIHL	**Without NIHL**	**Total**	**%**
With NIHL	45	5	50	62.50
Without NIHL	0	30	30	37.50
Total	45	35	80	100.00
%	56.25	43.75	100.00	

Graph 10: Agreement between NIHL PTA and NIHL OAE



Prevalence of noise induced hearing loss as detected by PTA is 56.25% and that by DPOAE is 62.5% proving the higher sensitivity of this method.

Table 13: Sensitivity and specificity (NIHL PTA and NIHL OAE)

Summary	Value	95% CI
Sensitivity	100.00%	92.13% to 100.00%
Specificity	85.71%	69.74% to 95.19%
Positive predictive value	90.00%	79.99% to 95.30%
Negative predictive value	100.00%	-
Disease prevalence	56.25%	44.70% to 67.32%

Table 14: Agreement between NIHL PTA and NIHL OAE on right side

Right OAE	Right PTA			
	With NIHL	Without NIHL	Total	%
With NIHL	40	6	46	57.50
Without NIHL	1	33	34	42.50
Total	41	39	80	100.00
%	51.25	48.75	100.00	

Graph 11: Agreement between NIHL PTA and NIHL OAE on right side

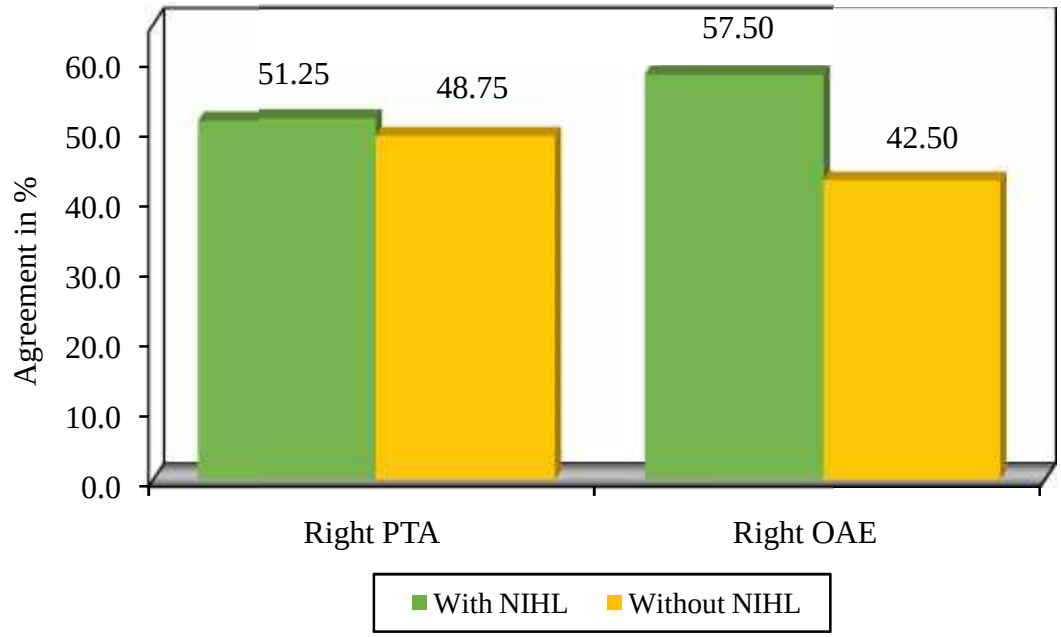


Table 15: Agreement between NIHL PTA and NIHL OAE on left side

Left OAE	Left NIHL PTA			
	With NIHL	Without NIHL	Total	%
With NIHL	44	3	47	58.75
Without NIHL	0	33	33	41.25
Total	44	36	80	100.00
%	55.00	45.00	100.00	

Graph 12: Agreement between NIHL PTA and NIHL OAE on left side

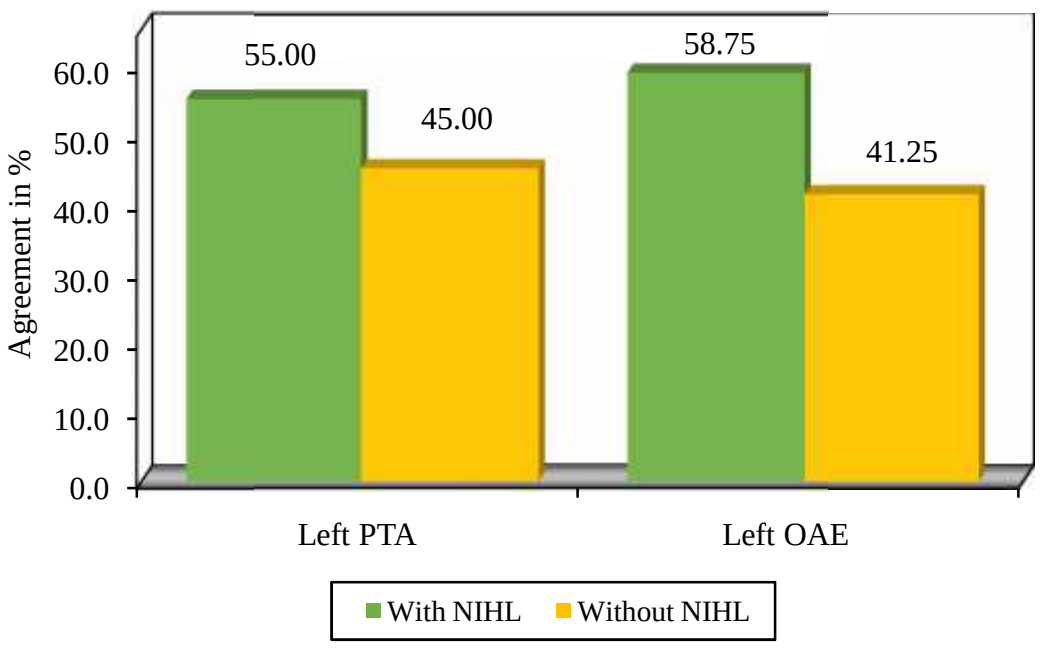


Table 16: Sensitivity and specificity (right NIHL PTA and right NIHL OAE)

Summary	Value	95% CI
Sensitivity	97.56%	87.14% to 99.94%
Specificity	84.62%	69.47% to 94.14%
Positive predictive value	86.96%	76.12% to 93.31%
Negative predictive value	97.06%	82.58% to 99.57%
Disease prevalence	51.25%	39.81% to 62.59%

Table 17: Sensitivity and specificity (left NIHL PTA and left NIHL OAE)

Summary	Value	95% CI
Sensitivity	100.00%	91.96% to 100.00%
Specificity	91.67%	77.53% to 98.25%
Positive predictive value	93.62%	83.23% to 97.74%
Negative predictive value	100.00%	-
Disease prevalence	55.00%	43.47% to 66.15%

The sensitivity and specificity of DPOAE to detect NIHL is higher than PTA. And though majority of NIHL are bilateral, in our study, symmetrical NIHL in either left or right ear is also observed proving its asymmetrical occurrence in initial stages of disease.

DISCUSSION

The results of the present study is based on the study conducted in 80 traffic police personnel – 160 ears.

Mean age of traffic police personnel

Studies have shown that a sound intensity of more than 85dB for a period for more than 2 years are apt for the detection of permanent damage caused by noise pollution.

Table 18: Comparison of tier of city, sample size and mean age

Studies	Tier of city	Sample size (number)	Mean age (years)
Shahid et al	Two tier	329	35.35 +/- 1.21
Chauhan et al	Two tier	88	36.43+/-1.87
Present study	Two tier	80	39.61+/-2.6

Prevalence of NIHL among traffic police personnel by PTA

Ghimire et al, described traffic policemen who could not hear 25dB sound were taken as short of hearing[21].

Khan et al accounted hearing loss greater than 10 dB for NIHL and hearing threshold ≤10 dB was ignored. Out of 60 traffic polices 39 (66%) showed NIHL from PTA assessment[13].

In present study, traffic police personnel with hearing loss greater than 15dB were taken as reduced hearing.

Following are the prevalence of NIHL among traffic police personnel in similar studies. We have compared our city Belagavi which is a tier two city based on population density, with our tier two cities in India. Our study showed high prevalence.

Table 19: Comparison of tier of city, sample size and prevalence of NIHL

Studies	Tier of city	Sample size	Prevalence
Shahid et al	Two tier	329	52.9% (174)
Chauhan et al	Two tier	88	90.9% (80)
Ghimire et al	Two tier	36	47.2% (17)
Khan et al	Two tier	60	66% (39)
Shrestha et al	Two tier	76	51.6%
Present study	Two tier	80	56.25% (45)

Degree of hearing loss

In the present study, 56.25% NIHL was detected after PTA assessment, out of which 33% had minimal, 7% had mild, 20% had moderate and 9% had moderately severe hearing loss.

Age and noise-induced hearing loss

Age has a cumulative effect on hearing loss. In study by Tiwari et al, majority of the policemen with NIHL belonged to 35-45 years of age[29]. According to Scott Brown 8th edition, the prevalence of age-associated hearing loss is tabulate.

Table 20: Age distribution and occurrence of noise induced hearing loss

Age group(years)	Prevalence (%)
18-30	2
31-40	5
41-50	10
51-60	17
61-70	30
71-80	53

Yulia et al in a study conducted in 3500 participants in USA demonstrated that the presence of an audiometric notch increased with age ($p<0.01$), ranging from 19.2% among persons aged 20–29 years, 27.3% in 31-40 years and 36% among persons aged 41-50 years[68].

Almaayeh et al found Age >35 years (OR = 2.7; 95% CI: 1.2– 6.1 significantly associated with increased odds of NIHL occurrence[69].

In the present study, a positive correlation exists between age and hearing loss.

- Prevalence of NIHL was % in the age groups 20-30, 31-40 and 41-50 years respectively. Maximum prevalence found in the age group of 41-50 years.
- The mean age of traffic police personnel with NIHL is 36.91 years and in those without NIHL is 27.83 years.
- In our study population, 90% (72) of the traffic policemen were aged less than 45 years while 10% (8) were aged more than 45 years. We found that all 8 traffic policemen aged more than 45 years and 51.39% (37) of those less than 45 years had NIHL.

Table 21: Prevalence of NIHL in age group >45years and <45years

Studies	**Age <45yrs**		**Age>45yrs**	
	Number of subjects	**Prevalence of NIHL**	**Number of subjects**	**Prevalence of NIHL**
Tiwari et al	66	43%	40	76%
Present study	71	50%	9	100%

Duration of exposure and Noise-induced hearing loss

In all the comparable studies, traffic policemen had a daily exposure of road traffic noise for a minimum duration of 8 hours per day. Duration of service gives an additive effect to noise in causing NIHL.

In a study Tiwari et al, most of them had recently joined the police service, with the mean years of exposure being 2.1 ± 1.8 years[29].

In a study by Khan et al, most of the traffic policemen were exposed to noise for 3-5 years (i.e. 41% of the policemen) some for 2-3 years (i.e. 25%) and others to below 2 years (33.3%). This showed hearing loss occured more with increased duration of exposure[13].

In the present study, all policemen had a duration of working hours as 8-10 hrs per day. The mean duration of exposure in NIHL group was 6.14+/- 3.10 years.

In our study, 83.78% (31) of traffic police-personnel who served for more than 5 years on road and 32.56% (14) who served less than 5 years on road developed NIHL. The positive correlation between duration of exposure and prevalence of NIHL was in agreement with other studies.

Table 22: NIHL prevalence in those with noise exposure for >5 years and <5 years

Studies	Sample size	Prevalence of NIHL (%)	
		Noise exposure <5 years	Noise exposure ≥ 5 years
Tiwari et al	66	86% (74)	14% (12)
Ghimire et al[21]	36	94.45%(34)	5.55% (2)
Present study	80	43(54%)	37(46%)

Symmetry in NIHL

Rabinowitz et al suggested that, though noise induced hearing loss (NIHL) is typically bilateral it can also be unilateral[70].

In study by Ghimire et al, 8 (22.22%) had PTA threshold involvement in 4 KHz where the percentage of unilateral or bilateral hearing loss was same, however in 3Hz frequency loss, unilateral loss was more. Out of both right and left ears, when PTA was done in 4000 kHz, 15 (41.7%) had hearing loss in left ear and 11 (30.6%) had in right ear. In 3000 kHz, 6 (16.7%) had hearing loss in left ear and 4 (11.1%) had loss in right ear. In 2000 kHz, 2(5.6%) had hearing loss in left ear where no hearing loss was seen in right ear. The threshold of hearing was more in left ears[21].

Table 23: NIHL in Bilateral, Unilateral, Right and Left ears

Studies	Bilateral NIHL	Unilateral NIHL	Right NIHL	Left NIHL
Shrestha et al	61.6% (45)	38.4% (28)	14% (4)	86% (24)
Present study	50% (40)	6.25%(5)	51.25%(41)	54%(44)

Shrestha et al also found asymmetrical NIHL with left side affected more than right. Mean threshold of hearing at 4 kHz on right ear is 26.31dB with standard deviation 9.42(range 15-70dB). Mean threshold on left ear is 28.95dB with SD 10.09 (range 10- 70dB)[11].

Suri et al studied DPOAE-Prevalance of NIHL. In the age group of 21-35 years, 28%(14) subjects were refer ears and all 100% of them were unilateral- 5 right and 9 left. In the age group of 36-50 years, 22%(15) were refer of which 64%

unilateral and 36% bilateral refer ears. In the older age group of 51-65 years, out of 66% refer result, 61% were unilateral and 31% were bilateral. This study concludes the detection of asymmetric pathology[25]

The two ears are linked with a neural pathway such that stimulation of one ear has a modulating effect on the contralateral cochlea[71]. Most studies report better responses for otoacoustic emissions in right ear. Some explanations to these findings are related to differences in ears associated with the slight advantage of right aural awareness and the dominance of the left hemisphere in the perception of speech and language function, besides the effect of higher suppression of otoacoustic emissions in the right ear, which proves that there is an asymmetric activity between ears and favours the acoustic signal detection and morphological asymmetry between the right and left craniofacial regions[72].

However, some studies have not shown significant differences in responses to otoacoustic emissions as to ear side.

In the present study, out of 80 policemen (160 ears), on comparison of hearing between left and right ears, right sided NIHL was seen in 41(51.25%) and left side in 44(54%) with a mean PTA threshold of 28.12dB in right and 27.79dB in left ear. In present study, left side was affected more compared to right ear. Its also in agreement with the above studies and the studies done in Biratnagar and Kathmandu city.

Self-assessment about hearing quality, tinnitus and noise-related attitudes

Shahid et al found only 12 (3.6%) had tinnitus, 140 (42.6%) used any hearing protection during duty hours and 42 (12.8%) said they had difficulty in hearing and frequently asked people to repeat themselves[4].

Ghimire et al found 22.2% (8) of the traffic policemen showed ear fullness. 22.2% (8) had tinnitus and only 5.6% (2)of them used protective measures[21].

Table 24: Self-assessment about NIHL in traffic policemem

Studies	Perceived ear fullness/ Problem in hearing	Ask to repeat/ Difficulty in crowd/ Telephone	Tinnitus	Use of Hearing protection aids
Shahid et al	12.8%	12.8%	3.6%	42.6%
Ghimire et al	22.2%	NA	22.2%	5.6%
Sanju et al	43.33%	33.2%	NA	0%
Gupta et al	40%	16.7%	19.96%	0%
Tiwari et al	41.9%	8.3%	11.6%	4.6%
Shrestha et al	35.5%	25%	16.6.6%	0%
Majumear et al	11.8%	NA	NA	7.3%
Venkatappa et al	3.33%	25%	16.6%	0%

Sanju et al in his study on 60 traffic policemen came to the conclusion that 43.33% of traffic police reported to have difficulty in hearing over phone and 33.2% of the traffic have difficulty of hearing in crowd[24].

Gupta *et al*, reported a larger percentage of subjects having trouble during normal (40%), and telephonic conversation (16.7%) respectively. None of the policemen (100%) had ever used ear muffs/ear plugs. Also reported 19.96% to have tinnitus. It was found that even though a majority of subjects rated their hearing as excellent, the supplementary questions that probed their hearing ability, revealed a different picture. A relatively more number of subjects reported having difficulties in conversation with others over the phone or in crowded places, due to which they had to ask the other person to talk a bit loudly, or they were themselves pointed out as

talking loudly by others. It was observed that a majority of our subjects were oblivious to the harmful effects of noise and did not consider it as an occupational hazard. The workplace environment was perceived to be extremely noisy by most of them. It was observed that only 9 subjects used ear plugs and none of the police personnel with NIHL had ever used an ear plug or ear muff, for which the main reason cited was its unavailability.[26].

In study by Tiwari et al, 6 subjects reported that they usually missed a lot of words in phone conversation, while 2.3% reported similar condition while talking to someone in a crowd. 11.6% reported that while watching television they usually kept the sound louder to hear properly. 32.6% mentioned that others often indicated to them that they were talking louder, while 41.9% felt that people usually talked louder with them so as to enable them to hear. 11.6% complained of regular tinnitus, while 62.8% had work-related tinnitus and experienced it during working hours only. Only 4.7% used earplugs and that too, very seldom[29].Among the participants of study by Shrestha et al; 26(23.6%) had tinnitus and 39(35.5%) say that they feel some blocked sensation in ear and have difficulty hearing in noisy environment.

Shrestha et al, in his study found 26(23.6%) subjects with tinnitus and 39(35.5%) subjects who said that fell some blocked sensation in ear and have difficulty hearing in noisy environment.13% subjects reported that they usually missed some conversation over phone while 25% reported similar condition while talking to someone in crowd. 16.66% had work related tinnitus (> once a day) and experienced it more during working hours. None of them used ear plugs/ear muffs and the reason for non-usage was non-availability (100%)[27].

Results obtained by Majumder et al.,[19] from traffic constables in Kathmandu, Nepal in 2010; 11.8% of their subjects gave a below average rating to

their hearing ability. Also, a substantial number of the subjects (92.7%) admitted not having used any kind of hearing protection device.

In the early stages of NIHL, the patients have a very few symptoms and hence they are usually unaware of the deleterious effects of sound. Any level of NIHL may muffle high-frequency sounds such as whistles or buzzers and may result in difficulty discriminating speech consonant sounds such as those in the words fish and fist, particularly in noisy environments with background noise, many voices, or room reverberation [11].

Talking loud in crowd could be because they become habituated talking loudly and do not realize the same. The auditory system too gets adapted to the louder input due to constant stimulation to loud noise[15].

A common finding between these studies was that although the self-assessed prevalence of NIHL showed a lower percentage of subjects, a closer scrutiny of the supplementary questions to assess the hearing status showed that the number of participants with hearing impairment is in large proportion, though hearing impairment may not be significant to disturb day-to-day conversation.

In the present study, out of the 36 policemen with normal PTA, 3 policemen reported difficulty in comprehending speech when 2 or more people talk simultaneously or in a noisy surrounding.

Only (2.3%) of the policemen felt that the need of using ear plugs/muffss hearing ability was below average. This could be due to ignorance about the hazards caused by continuous exposure to noise.

The study also revealed that the traffic police, in general, do not use any personal protective equipments and the non-availability of these PPEs is the common

reason for it. Thus it is suggested that not only should these PPEs be made available, but also periodic workshops should be carried out to motivate the subjects for their correct and regular usage. The effectiveness of the PPEs over other methods to reduce noise exposure should also be demonstrated noise level, exposure of more than 10 years, and not using hearing protective devices were significantly associated with increased risk of NIHL[69].

Non-audiological side effects

In the study by Himanshu et al, 16.66% of the traffic police had headache, 36.66% reported annoyance from occupational noise, 23.33% showed irritation even after work, 33.33% reported poor quality of sleep after work, because of exposure to occupational noise during work[24].

In the present study, 11% of the road traffic policemen reported non-auditory side effects and it was found statistically significant with the prevalence of NIHL(0.0020).

Effect of smoking and alcohol

Studies have shown a positive correlation with smoking and alcoholism with the incidence of NIHL. However, our study did not have a statistically significant difference between traffic police personnel with NIHL whether or not were habituated. This maybe because, exact packs years were not calculated and compared. Since there is no positive correlation, smoking and alcoholism will not become a confounding factor in our study[71].

NIHL and Frequencies affected, SRT, SIS

Studies have shown that 4KHz is mainly affected however neighbouring frequencies are also affected[8]. The speech recognition threshold [SRT] is known to be reduced in cases of NIHL. Speech identification score [SIS] is lowered in auditory neuropathy.

Ghimire et al showed, out of 17 traffic policemen in his study, 8 of them had hearing loss in 4 kHz, 4 in 3kHz and remaining 5 in mixed 2 kHz, 3kHzand 4 kHz frequencies.

In the early stages of NIHL, the speech frequencies are less affected and frequency area 4-6 kHz is usually affected first with maximum at 4 kHz[73].

In present study, higher frequencies were affected more. On right, the mean of individual frequencies in NIHL subjects for 1kHz, 2kHz, 4kHz and 8kHz are 27.11dB, 32.11dB, 55.67dB and 41.22dB, respectively. On left, the frequency means are 26.89dB, 31.11dB, 56.56dB and 40.44dB respectively for the same frequencies. There was a statistically significant difference between the NIHL and Non-NIHL subjects in higher frequencies.

Narne et al compared the SIS score of 10 normal controls and 10 subjects with auditory neuropathy. Results revealed that speech identification scores were significantly poorer in auditory neuropathy than that of individuals with normal hearing[74]. Alvord reported a significant deficit of 10% in word identification in background noise for 10 audiometrically normal males with a history of occupational noise exposure[75] .

In our study, both SRT and SIS had a statistically significant reduction in subjects with NIHL and those without NIHL. The mean SRT was 37 in NIHL

policemen and 19.5 in subjects without NIHL. The mean SIS(%) was 85 in NIHL policemen and 92 in subjects without NIHL.

Correlation between PTA and DPOAE

There is a lack in the number of studies done in traffic policemen to detect early noise-induced hearing loss by Otoacoustic emissions (OAE).

Table 25: DPOAE abnormality found in ears with normal PTA threshold

Study	No. of ears with Normal threshold (n)	DPOAE		
		Right	Left	Bilateral
Suri et al	150	12.7% (19)	14.7% (22)	11.3% (17)
Present study	75	8% (6)	4% (3)	4% (3)

Suri et al found that in subjects with normal audiometric thresholds, DPOAE can be refer. and asymmetrical hearing loss can occur. Our study also showed, abnormal DPOAE I certain subjects with PTA normal hearing threshold. This stands true for the hidden hearing loss in PTA detected by OAE.

Gorga et al reported DPOAE test performance, it was best for mid and high frequencies and poorest for lower frequencies and for the highest frequency tested (8000 Hz)[76].

Rosati et al in 2017 conducted a study among urban outdoor workers aimed to compare the DPOAEs with the PTA and to analyze the changes in participants exposed to noise. The results show a prevalence of participants with impaired DPOAEs higher than the prevalence of participants with impaired audiometries[20].

Neely et al conducted a study in 2018 among 32 normal hearing subjects and 124 sensorineural hearing loss subjects found that compared with Cochlear reserve measurements, DPOAE measurements have the advantages in a screening paradigm of better test performance and shorter test time to identify early noise induced hearing loss[77].

In our study, among the 80 traffic police personnel, in right ear 41 (51.25%) showed PTA abnormality suggestive of NIHL and 46(57.5%) policemen reported "refer" in OAE. It had a sensitivity of 97.56%, specificity of 84.62%, PPV of 86% and NPV of 97.06% at 95% CI. On left side, 44 reported NIHL on PTA and 47 had "refer" on OAE. It had a sensitivity of 100%, specificity of 91.67%, PPV of 93.62% and NPV of 100% at 95% CI. The findings in this study are in agreement with the above studies. The study observed a statistically significant difference in DPOAE amplitudes among age groups at four test frequencies .Older ears had reduced DPOAE amplitude compared with young adult ears.

Studies have shown subjective abnormalities even in subjects with normal hearing threshold. In a study by Ghimire, out of the 36 policemen with normal PTA, 3 policemen reported difficulty in comprehending speech when 2 or more people talk simultaneously or in a noisy surrounding.

Some listeners with normal audiometric thresholds report difficulties in understanding speech in noisy environments[78]. For example, in a large UK survey, 26% of adults reported great difficulty hearing speech in noise, while only 16% had abnormal sensitivity (audiometric thresholds 525 dB hearing level [HL] averaged between 0.5 and 4 kHz).

These results suggest that as age advances and as duration of noise exposure increases, the prevalence of NIHL will also increase. NIHL is generally affects both sides equally, but can affect one side alone. Questionnaire, PTA and OAE detect NIHL in increasing order of sensitivity and specificity. Early NIHL can be detected by an abnormal OAE conducted in PTA-normal subjects.

CONCLUSION

People employed in noisy environment should be aware of noise pollution and NIHL, its possible effects, and its preventive measures. This will allow a careful attitude and conscious effort for prevention, without hindrance in their job. Considering this, an early detection of this irreversible but preventable occupational hazard is of prime importance.

The auditory and non-auditory effects of noise pollution as an occupational hazard have been extensively studied both clinically and experimentally. There are less studies vouching for otoacoustic emission as the main screening tool for NIHL.

Till date Indian studies have been using Pure tone audiometry for the detection of NIHL, which lacks a clear picture of this occupational hazard. There have been near to nil Indian studies one amongst road traffic police personnel on NIHL using Otoacoustic emission. OAE gives a better clarity of this occupational hazard.

Distortion-product otoacoustic emission is found to be highly frequency specific and is recordable over a broad frequency range. Thus, DPOAE might be the revolutionary new objective, non-invasive, rapid and accurate test of hearing with its application as screening test for not only infants but also as an early indicator of hearing impairment in healthy adults before the elevation of audiometric threshold.

But our study is one of a kind and is one of the initial studies done among road traffic police personnel using OAE, in this geographical region.

Utilization of health-related applications in smartphones has gained increased focus. These are useful, less time consuming, and has increased efficiency by

allowing early diagnosis of hearing loss. Also it will reduce the shortage of audiology service in developing countries like ours.

With the rapid increase in noise pollution as a byproduct to industrialization and technical modernization, along with loopholes in jurisdiction related to occupational hazards, screening with higher accuracy tool is pivotal. This study assess awareness and detect early NIHL amongst traffic police personnel of Belagavi.

It is also expected that our work will inspire many other researchers to take up similar studies and then bring about a framework for early detection of NIHL.

In this study, prevalence of NIHL and various factors associated with NIHL were studied.

1. Mean age at which NIHL was prevalent in noise exposed traffic policemen were 36.84 years.
2. Prevalence of noise induced hearing loss is high in Belgaum traffic police personnel with most of them having mild to moderate degree of hearing loss.
3. Study has proven that increasing age and more duration of service are significant risk factors causing noise induced hearing loss.
4. Lack of proper knowledge and reduced usage and availability of protective aids and interventions are factors prone for NIHL.
5. DPOAEs represent a sensitive test for monitoring the effects of noise in preclinical conditions. With further reductions in acquisition time, the measurement procedure will be applicable in clinical routine in the near future.

We therefore strongly vouch for certain strategies for protection of traffic policemen. Regular hearing assessment of traffic policemen is necessary to identify

the problems of noise pollution since it is an insidious process and takes long duration to be overt. Early detection and timely prevention by providing ear protective devices & regular rotation of working place will provide cheap and long-term benefits. Introduction of stringent legislations regarding usage of horns and organization of intensive public awareness campaigns regarding ill effects of noise, via print, and electronic media will also prove beneficial.

World Hearing Day is observed yearly on 3rd March with the primary objective to raise awareness about different aspects of hearing loss. The rationale for selecting this date was that the number 3.3 represents the shape of two ears. Taking cognisance of these facts, we should take adequate measures and work towards it.

SUMMARY

This study was conducted in KLES Dr Prabhakar Kore hospital, Belagavi during a period of one year amongst the traffic policemen in Belagavi district. We performed this community-based study with an objective to identify the prevalence of NIHL in traffic policemen of a tier two city, and to detect Early NIHL.

In our study, 80 traffic police personnel, aged from 21 to 50 years, employed for a duration of at least 6 months participated. In all policemen, thorough history taking and examination was done. For evaluating NIHL, these traffic policemen were subjected to an Interview-based questionnaire including the Modified Smith hearing severity questionnaire, Pure tone audiometry and Distortion-product Otoacoustic emission.

After the analysis, the following results were obtained:

1. Mean age of traffic policemen with NIHL was 36.91+/-1.12 years.
2. Prevalence of NIHL among road traffic police personnel are high and most reported mild to moderate loss. Prevalence by PTA is 56.25% and that of early-NIHL by OAE is 62.50%.
3. Prevalence of NIHL increased with increasing age.
4. Mean duration of noise exposure in NIHL was 6.14+/- 3.10 years; Prevalence of NIHL increase with duration of exposure.
5. There was significant agreement between subjective detection of hearing loss by the Modified smith hearing severity questionnaire and the prevalence of NIHL.

6. NIHL is associated with other auditory effects like tinnitus and hypersensitivity to loud sound, and non-auditory effects like headache, irritability.
7. Speech recognition score and Speech identification score can be abnormal in NIHL.
8. NIHL produced a high frequency sensory neural hearing loss with 4k notching at initial stage of disease process.
9. DPOAE is a sensitive tool than PTA in detection of NIHL in early stage even in an asymptomatic PTA-normal subject.

This study confirms the findings of similar studies performed in noise exposed occupations. Subjects in our study comprised the economically productive age groups. Permanent damage of hearing disability this early, would leave them in lifelong disability. Hence prevention and early detection play pivotal role.

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ANNEXURE - I

ETHICAL CLEARANCE CERTIFICATE

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed – to- be- University)
Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (GoI)
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Ref: MDC/DOME/ 61 **Date: 24/11/2018**

To,
REG. NO:BE0118003
PG student in Otorhinolaryngology and Head & Neck Surgery,
J.N.Medical College
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"AUDIOLOGICAL EVALUATION OF EARLY NOISE –INDUCED HEARING LOSS IN TRAFFIC POLICE PERSONNEL IN BELGAUM-A COMMUNITY –BASED ONE YEAR CROSS SECTIONAL STUDY",** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Arathi Darshan)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Roopa M Bellad)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

ANNEXURE – II

INFRORMED CONSENT

WRITTEN AND INFORMED CONSENT

TITLE: "AUDIOLOGICAL EVALUATION OF NOISE-INDUCED HEARING LOSS IN TRAFFIC POLICE PERSONNEL IN BELGAUM, INDIA"– A ONE YEAR COMMUNITY-BASED CROSS-SECTIONAL STUDY.

STUDY INVESTIGATOR:REG. NO: BE0118003
Post graduate resident
Department of Otorhinolaryngologyand
Head and Neck Surgery,
JNMC, Belagavi

CO-INVESTIGATOR:Dr. _____
Professor,
Department of Otorhinolaryngology and
Head and Neck Surgery,
JNMC, Belagavi

INTRODUCTION AND PURPOSE:

A study**:** "AUDIOLOGICAL EVALUATION OF NOISE-INDUCED HEARING LOSS AMONGST TRAFFIC POLICE PERSONNEL INBELGAUM, INDIA"– A ONE YEAR COMMUNITY-BASED CROSS SECTIONAL STUDY" at KLE's Dr. Prabhakar Kore Charitable Hospital and Medical Research Centre, Belagaviis beingconducted by **REG. NO: BE0118003,** Postgraduate resident in the Department of Otorhinolaryngology and Head & Neck surgery at J. N. Medical College Belagavi, Karnataka, under the guidance of Dr._____ ,Professor,

Department of Otorhinolaryngology and Head & Neck surgery, J. N. Medical College, KLE University, Belagavi.

We request you to participate in this study as you are eligible to be included. During the study you will be asked questions regarding your present & past medical history and awareness about noise induced hearing loss and you will be required to answer to the best of your knowledge.

Your participation in this study is voluntary. Your decision whether or not to participate in the study will not affect your relationship with J.N.Medical College. Even if you decide to participate, you are free to withdraw from the study at any point of time.

OBJECTIVES:

1. To measure prevalence of noise-induced hearing loss in traffic police personnel, in a second tier city like Belgaum.
2. To identify early noise-induced hearing loss among them.

EXPLANATION OF PROCEDURE

Each patient will be required to fill out a questionnaire addressing the presence of noise induced hearing loss symptoms and signs. All of them will be required to undergo otoscopic and otoendoscopic examination, followed by pure-toneaudiometry and Distortion-product otoacoustic emission, if required.

BENEFITS: Patient will not be eligible for any kind of monetary benefits or free services by virtue of your participation in the study.

RISKS: Methods applied to do the study are safe. No adverse effects are expected.

COST OF PARTICIPATION: The cost of the investigation will be borne by the study subject. The other indirect expenses will be borne by the investigator.

PRIVACY AND CONFIDENTIALITY: The results of the study may be published in journals for scientific purposes. However your identity will not be revealed. All information collected will be coded so that no one other than the investigator will know your identity.

WITHDRAWAL FROM THE STUDY: You can withdraw from the study at any time if you wish to do so.

AUTHORIZATION TO PUBLISH THE RESULTS: The researcher may use the information gathered from this study for presentation in scientific meetings. However your identity will not be revealed.

QUERIES AND CONTACT: If you have any queries regarding the study, you can contact **REG. NO: BE0118003**without any hesitation on Mobile no: _____ and the guide Dr. _____on Mobile no: _____. If you have any questions about rights as a research participant you can contact Dr. Rupa M. Bellad Professor, Department of Paediatrics and Chairman, Jawaharlal Nehru Medical College Institutional Ethics Committee on human subjectsresearch.

CONSENT SUMMARY: I have been explained all the contents of this consent form in my local language and having understood and clarified all my queries about the study to the best of my knowledge, I hereby give my voluntary consent for participation in the study. I do sign the informed consent form in front of an eyewitness whom I recognize.

Name and Signature/ left thumb impression of the participant:

Name and Signature of the interviewer:

Name and Signature/ left thumb impression of the eyewitness (Relative):

Signature of the guide:

Date:

ANNEXURE - III

PROFORMA

"AUDIOLOGICAL EVALUATION OF EARLY NOISE-INDUCED HEARING LOSS IN TRAFFIC POLICE PERSONNEL IN BELGAVI"– A COMMUNITY BASED ONE YEAR CROSS SECTIONAL STUDY

Date:O.P. No:

Name: Age

Sex: Occupation:

Address: Phone No:

QUESTIONNAIRE

1. Number of years of service in traffic branch:
2. Average duty hours per day:
3. Average number of duty hours in a week:
4. Place of duty:

CLINICAL ASSESSMENT

1. Do you feel you have a hearing loss?
2. Do you currently have any medical problems with your hearing or ears?
3. Does one of your ears hear better than the other?
4. Do you or have you had ringing in the ears or tinnitus?
5. Do you have trouble following conversations when two or more people talking at the same time?
6. Do people complain you have the TV or radio volume too high?
7. Do you find you need to ask people to repeat themselves as you misunderstand some words?

8. Can you hear and understand men's voices better than women or children?
9. Do voices sound blurry, like people mumbling?
10. Do you sometimes miss common sounds (heard by others) i.e. doorbells or the telephone?
11. Do people get annoyed if u misunderstand what they say?
12. Does a hearing problem cause you to have arguments with family members?
13. Do you have difficulty in hearing over whispers or at increased distances, i.e. calling from other room?
14. Does your hearing ever seam out of balance i.e. louder on one side than the other?
15. Have you had infections or discharges from your ears previously?
16. Any history of sudden high intensity /acute noise exposure?
17. Have you ever had operations on your ears for any conditions, including (but not limited to) Mastoids, Drum repair, perforation of the ear drum?
18. Have you taken medications for meningitis, tuberculosis [Ototoxic drugs] ?
19. Do you have history of previous occupational noise exposure?
20. Do you wear any hearing protection during work?

CLINICAL PROFILE:

HISTORY

Chief Complaint:

History of Present Illness:

Past History:

Personal History:

Family History:

GENERAL PHYSICAL EXAMINATION

Pulse: Blood Pressure:Temperature:

Respiratory Rate:Pallor: Icterus:

ClubbingCyanosis: Lymphadenopathy:

Oedema:

ENT EXAMINATION

1.EAR EXAMINATION Right Left

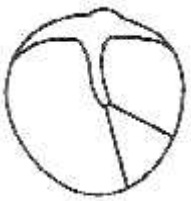
Pinna

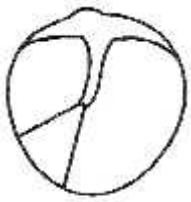
Pre auricular area

Post auricular area

External auditory canal

Tympanic membrane


Right


Left

TUNING FORK TESTS:

Rinne's test 256 Hz

512 Hz

1024 Hz

Weber's test:

Absolute Bone Conduction test:

FACIAL NERVE EXAMINATION:

2.NOSE EXAMINATION

External appearance

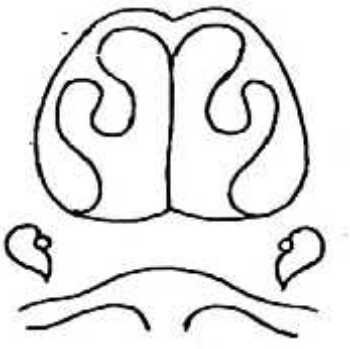
- Root
- Bridge
- Dorsum
- Tip
- Ala
- Columella

Cold spatula test:

Tip elevation:

Anterior Rhinoscopy

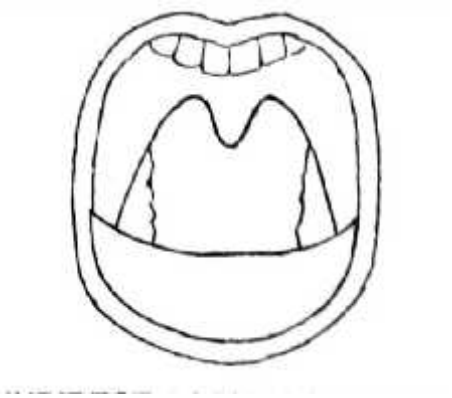
Posterior Rhinoscopy:



Paranasal Sinus Examination:

3. ORAL CAVITY AND OROPHARYNX, INDIRECT LARYNGOSCOPY

Oral cavity and Oropharynx:



Indirect laryngoscopy

4. NECK EXAMINATION

DIAGNOSIS:

INVESTIGATIONS

Routine Blood Investigations**CBC:** **GRBS:**

LFT: **MR:**

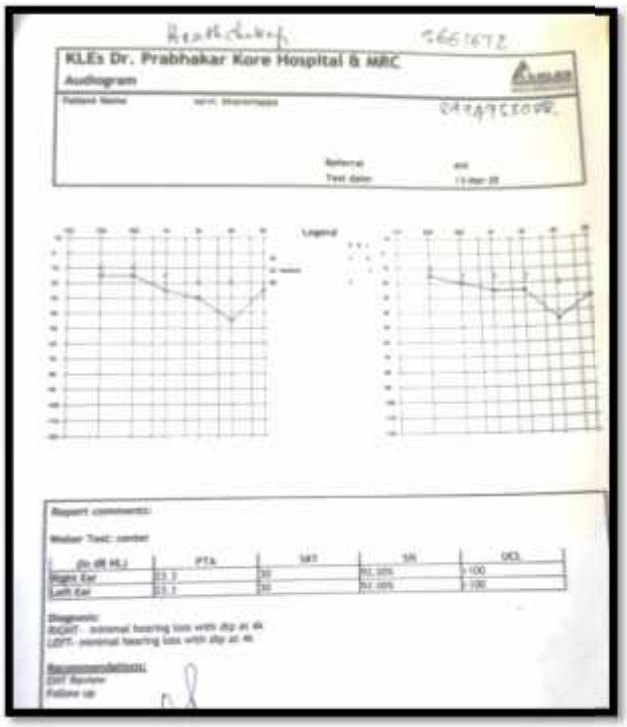
PTA:

DPOAE:

ANNEXURE - IV

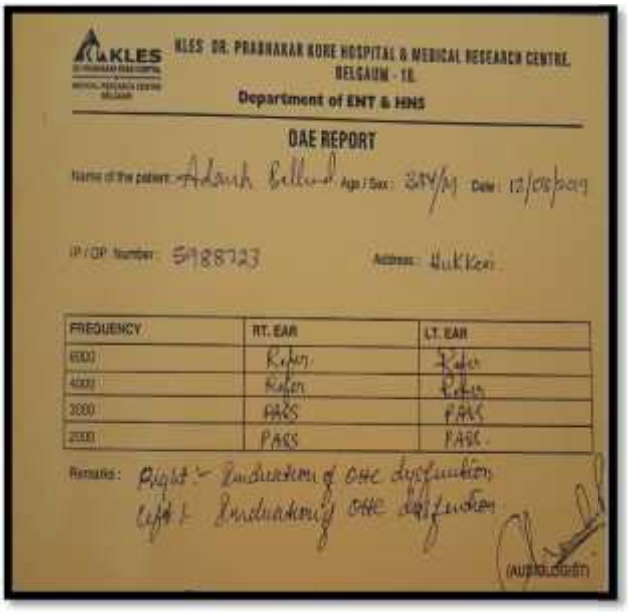
PHOTOGRAPHS

1. PTA in progress



2. PTA report showing bilateral 4k notch

3. OAE in progress



KLES DR. PRABHAKAR KORE HOSPITAL & MEDICAL RESEARCH CENTRE, BELGAUM - 10

Department of ENT & HNS

OAE REPORT

Name of the patient: Adarsh Bellad Age / Sex: 24Y/M Date: 12/05/2019

IP / OP Number: 5188723 Address: Mukteen

FREQUENCY	RT. EAR	LT. EAR
6000	Refer	Refer
4000	Refer	Refer
2000	PASS	PASS
2000	PASS	PASS

Remarks: Right - Indication of OHC dysfunction
Left - Indicating OHC dysfunction

(Audiologist)

4. OAE report – “Refer” at bilateral 4k and 6k

ANNEXURE V – KEY TO MASTER CHART

Db	:	Decibel
Hz	:	Hertz
PTA	:	Pure tone audiometry
u/l	:	Unilateral
B/L	:	Bilateral
L	:	Left
R	:	Right
NIHL	:	Noie induced hearing loss
SRT	:	Speech recognition threshold
SIS	:	Speech identification score
OAE	:	Otoacoustic emissions

ANNEXURE VI – MASTER CHART

Sl. No.	AGE	YEARS OF EXPOSURE	EXPOSURE PER WEEK	SMOKING	ALCHOHOL	S1: Work in Noisy environment?	S2:Trouble following conversation when 2 or more people talk?	S3:Turn TV/Radio volume high?	S4: Misunderstand words or ask to repeat?	S5: Do people get annoyed?	S6: Make inappropriate response due to misunderstanding?	S7: Difficulty hearing whisper?	S8: Cause family arguments?	S9: Problem listening TV/radio?	S TOTAL	SMITH QUESTIONNAIRE INFERENCE
1	38	3	56	X	Y	3	1	1	1	0	0	0	0	0	NIHL	NIHL
2	27	5	56	X	X	3	0	0	0	0	0	0	0	0	Normal	NIHL
3	23	3	60	X	X	3	0	0	0	0	0	0	0	0	Normal	Normal
4	46	7	63	X	X	3	3	3	3	3	3	3	3	2	NIHL	NIHL
5	42	7	56	Y	X	3	2	2	1	1	1	1	0	0	NIHL	NIHL
6	45	5	56	X	Y	3	2	1	2	1	1	1	0	0	NIHL	NIHL
7	38	6	56	X	X	3	1	2	1	1	1	1	0	0	NIHL	NIHL
8	28	5	56	Y	Y	3	0	0	0	0	0	0	0	0	Normal	Normal
9	40	2	63	X	X	3	0	0	0	0	0	0	0	0	NIHL	Normal
10	34	6	56	X	X	3	1	1	1	0	0	0	0	0	NIHL	NIHL
11	43	10	63	Y	X	3	3	2	1	2	1	1	1	1	NIHL	NIHL
12	50	15	63	X	Y	3	3	3	3	3	3	3	2	2	NIHL	NIHL
13	23	3	56	X	X	3	0	0	0	0	0	0	0	0	Normal	NIHL

14	39	7	56	Y	X	3	1	2	0	1	1	1	1	0	NIHL	NIHL
15	33	2	60	X	X	3	0	0	0	0	0	0	0	0	Normal	Normal
16	33	2.5	63	Y	Y	3	0	0	0	0	0	0	0	0	Normal	Normal
17	26	2.5	63	X	X	3	0	0	0	0	0	0	0	0	Normal	Normal
18	36	2	56	X	Y	3	0	0	0	0	0	0	0	0	Normal	Normal
19	23	2	56	X	Y	3	0	0	0	0	0	0	0	0	Normal	Normal
20	38	9	56	Y	X	3	2	1	1	2	1	1	1	0	NIHL	NIHL
21	22	2.5	56	X	X	3	0	0	0	0	0	0	0	0	Normal	Normal
22	32	4	63	X	X	3	2	2	1	1	2	0	0	0	NIHL	NIHL
23	26	5	56	X	X	3	0	0	0	0	0	0	0	0	Normal	Normal
24	25	2	56	X	X	3	2	2	1	0	0	0	0	0	NIHL	NIHL
25	23	2	60	X	Y	3	0	0	0	0	0	0	0	0	Normal	Normal
26	33	6	63	X	Y	3	1	1	1	0	0	0	0	0	NIHL	NIHL
27	23	1.5	63	X	X	3	0	0	0	0	0	0	0	0	Normal	Normal
28	26	5	56	X	X	3	0	0	0	0	0	0	0	0	Normal	Normal
29	22	2	56	X	X	3	0	0	0	0	0	0	0	0	Normal	Normal
30	48	3.5	56	Y	X	3	1	1	2	2	1	1	2	1	NIHL	NIHL
31	40	9	63	X	Y	3	1	1	0	1	1	1	1	0	NIHL	NIHL
32	28	6	63	X	X	3	1	1	0	0	0	0	0	0	NIHL	NIHL
33	26	2	56	X	X	3	0	0	0	0	0	0	0	0	Normal	Normal
34	28	2	56	X	X	3	0	0	0	0	0	0	0	0	Normal	NIHL
35	24	3	60	X	Y	3	0	0	0	0	0	0	0	0	Normal	Normal
36	27	2	63	Y	Y	3	1	1	1	0	0	0	0	0	NIHL	NIHL
37	25	2	63	X	X	3	0	0	0	0	0	0	0	0	Normal	Normal
38	27	5	56	X	X	3	2	2	2	0	2	2	0	0	NIHL	NIHL
39	43	12	56	X	X	3	3	3	2	2	3	3	2	1	NIHL	NIHL
40	26	4	56	Y	X	3	0	0	0	0	0	0	0	0	Normal	Normal
41	36	7	56	X	Y	3	2	2	2	1	1	1	1	1	NIHL	NIHL

42	23	2	63	X	X	3	1	1	1	0	0	0	1	0	NIHL	NIHL
43	38	8	56	X	Y	3	2	2	2	1	1	1	1	1	NIHL	NIHL
44	27	2	56	X	X	3	0	0	0	0	0	0	0	0	Normal	Normal
45	24	2	60	X	X	3	0	0	0	0	0	0	0	0	Normal	Normal
46	28	4	60	Y	X	3	2	2	2	0	0	0	0	0	NIHL	NIHL
47	25	3	56	X	X	3	0	0	0	0	0	0	0	0	Normal	Normal
48	33	2.5	56	X	X	3	0	0	0	0	0	0	0	0	Normal	Normal
49	23	3	60	X	Y	3	0	0	0	0	0	0	0	0	Normal	Normal
50	45	11	63	X	X	3	3	3	3	2	2	2	2	1	NIHL	NIHL
51	42	2	56	X	Y	3	1	1	2	1	1	1	1	0	NIHL	NIHL
52	38	3	56	Y	Y	3	1	1	1	0	0	0	0	0	NIHL	NIHL
53	32	7	56	Y	X	3	2	2	2	1	2	1	1	1	NIHL	NIHL
54	33	8	56	X	X	3	2	2	2	2	1	2	1	1	NIHL	NIHL
55	45	7	63	X	X	3	2	1	2	1	1	1	1	1	NIHL	NIHL
56	44	5	56	X	X	3	0	0	0	0	0	0	0	0	Normal	Normal
57	47	6	56	X	X	3	2	2	2	2	2	2	2	2	NIHL	NIHL
58	38	2	60	X	X	3	1	1	1	0	1	1	0	0	Normal	Normal
59	41	5	60	X	Y	3	2	2	2	2	1	2	1	2	NIHL	NIHL
60	34	7	63	X	X	3	1	1	1	0	0	0	0	0	Normal	Normal
61	42	10	56	X	X	3	2	2	2	1	2	2	1	1	NIHL	NIHL
62	38	11	56	X	X	3	2	2	2	1	1	2	1	2	NIHL	NIHL
63	43	3	60	X	Y	3	0	0	0	0	0	0	0	0	Normal	Normal
64	26	7	63	X	X	3	2	2	2	2	1	1	1	2	NIHL	NIHL
65	39	2.5	56	X	X	3	2	2	2	1	1	1	1	2	NIHL	NIHL
66	23	2.5	60	X	Y	3	1	1	1	2	0	0	0	0	NIHL	NIHL
67	23	2.5	56	Y	X	3	0	0	0	0	0	0	0	0	Normal	Normal
68	38	2	56	Y	X	3	1	1	1	0	1	0	0	0	NIHL	NIHL

69	37	8	56	X	Y	3	2	2	2	2	2	2	2	1	NIHL	NIHL
70	32	7	63	X	Y	3	0	0	0	0	0	0	0	0	NIHL	Normal
71	26	2	56	X	X	3	2	2	2	1	1	2	1	0	NIHL	NIHL
72	25	2	56	X	X	3	0	0	0	0	0	0	0	0	Normal	Normal
73	33	6	60	Y	X	3	2	3	2	2	2	2	1	2	NIHL	NIHL
74	23	2	56	X	X	3	0	0	0	0	0	0	0	0	Normal	Normal
75	23	2	56	X	X	3	0	0	0	0	0	0	0	0	Normal	Normal
76	26	3.5	60	X	X	3	2	1	1	1	1	2	0	0	NIHL	NIHL
77	44	10	63	X	Y	3	3	3	2	2	2	2	2	2	NIHL	NIHL
78	40	6	56	X	Y	3	1	1	1	1	1	1	1	0	NIHL	NIHL
79	48	5	63	X	X	3	2	2	2	1	2	2	1	1	NIHL	NIHL
80	22	3	63	X	X	3	0	0	0	0	0	0	0	0	Normal	Normal

PERCEIVE HEARING LOSS?	MUMBLING	MEN voice heard betteer than WOMEN	TINNITUS	HYPERSENTIVITY	Non-auditory symptoms	EAR PLUG use	KNOWLEDGE about ill-health	R 250Hz	R 500Hz	R 1KHz	R 2KHz	R 4KHz	R 8KHz	R PTA	R SRT	R SIS %
N	N	N	N	N	N	N	N	10	15	15	15	45	30	15	20	92
N	N	N	N	N	N	Y	N	10	15	15	15	15	15	15	20	92
N	N	N	N	N	N	N	Y	15	15	15	15	20	25	15	20	92
Y	Y	Y	y	Y	Y	N	N	55	55	60	100	110	95	68.3	75	72
N	Y	Y	N	N	N	N	N	15	20	25	25	45	30	23.3	30	92
N	Y	N	N	N	N	N	y	15	20	25	30	45	25	23.3	30	92
N	N	Y	N	N	N	N	N	15	15	20	25	50	40	20	30	92
N	N	N	N	N	N	Y	N	10	15	15	15	20	15	16.3	20	92
N	N	N	N	N	N	Y	N	10	15	15	15	10	15	15	20	92
N	N	N	N	N	N	N	N	5	10	10	15	60	50	11.6	20	92
N	Y	Y	Y	Y	Y	N	N	30	45	50	65	90	70	53.3	70	72
Y	Y	Y	Y	Y	Y	N	N	35	50	55	65	90	70	56.6	70	72

N	N	N	N	N	N	N	N	10	15	15	15	15	15	15	20	92
N	N	y	N	N	N	N	N	15	15	25	30	45	25	23.3	30	92
N	N	N	N	N	N	N	N	10	15	15	15	10	15	15	20	92
N	N	N	N	N	N	N	N	10	15	15	15	15	20	15	20	92
N	N	N	N	N	N	N	N	5	10	15	15	20	15	13.3	15	100
N	N	N	N	N	N	N	N	10	15	15	15	30	35	15	20	92
N	N	N	N	N	N	N	Y	10	15	15	15	10	15	15	20	92
N	Y	N	Y	N	N	N	N	15	15	25	30	45	25	23.3	30	92
N	N	N	N	N	N	N	N	10	15	15	15	10	15	15	20	92
N	Y	Y	N	N	N	N	N	10	15	15	15	10	15	15	20	92
N	N	N	N	N	N	Y	N	10	15	15	15	25	15	15	20	92
N	Y	y	N	N	N	N	N	15	15	15	15	25	15	15	20	92
N	N	N	N	N	N	N	N	10	15	15	15	10	15	15	20	92
N	N	N	N	N	N	N	N	10	15	15	15	10	15	15	20	92
N	N	N	N	N	N	N	N	10	15	15	15	45	30	15	20	92
N	N	N	N	N	N	N	N	5	10	10	10	15	10	10	15	100
N	N	N	N	N	N	Y	N	10	15	15	15	25	15	15	20	92
N	N	N	N	N	N	N	N	10	15	15	15	20	15	15	20	92
N	N	N	N	N	N	N	N	20	20	20	20	45	30	20	30	82
N	N	N	N	N	N	N	N	15	15	20	25	80	70	20	20	82
N	N	N	N	N	N	N	N	10	15	15	15	40	30	15	20	92
N	N	N	N	N	N	N	N	5	15	15	15	10	15	15	20	92
N	N	N	N	N	N	Y	N	10	15	15	15	10	15	15	20	92
N	N	N	N	N	N	N	N	5	15	15	15	20	20	15	20	92
N	N	N	N	N	N	N	N	10	15	15	15	45	30	15	20	92
N	N	N	N	N	N	N	N	10	15	15	15	10	15	15	20	92

N	Y	Y	N	N	N	N	Y	10	15	15	15	10	10	15	20	90
Y	Y	Y	Y	Y	Y	N	N	30	45	50	65	90	70	53.3	70	72
N	N	N	N	N	N	Y	N	10	15	15	15	10	15	15	20	92
N	Y	Y	Y	N	N	N	N	15	25	25	30	55	40	28.3	40	82
N	N	N	N	N	N	N	N	10	15	15	15	45	30	15	20	92
N	Y	Y	N	N	N	N	N	15	25	25	30	55	40	28.3	40	82
N	N	N	N	N	N	N	N	10	15	15	15	10	10	15	20	90
N	N	N	N	N	N	N	N	5	15	15	15	10	15	15	20	90
N	Y	N	N	N	N	N	N	10	15	15	15	20	20	15	20	92
N	N	N	N	N	N	N	N	5	15	15	15	10	15	15	20	92
N	N	N	N	N	N	N	N	10	15	15	15	25	15	15	20	90
N	N	N	N	N	N	Y	N	5	15	15	15	10	15	15	20	92
Y	Y	N	Y	Y	Y	N	Y	30	45	50	65	90	70	53.3	70	72
N	N	N	N	N	N	N	Y	15	15	20	25	80	70	20	20	82
N	N	N	N	N	N	N	N	10	15	15	15	45	30	15	20	92
N	Y	N	N	N	N	N	N	15	15	25	30	45	25	23.3	30	92
N	Y	Y	Y	N	N	N	N	15	25	25	30	55	40	28.3	40	82
N	Y	N	N	N	N	N	N	15	25	25	30	55	40	28.3	40	82
N	N	N	N	N	N	N	N	10	15	15	15	15	20	15	20	92
N	Y	Y	y	N	Y	N	N	30	40	40	45	60	45	41.6	60	80
N	N	N	N	N	N	N	N	10	15	15	15	45	30	15	20	92
N	Y	Y	y	N	N	N	N	30	40	40	45	60	45	41.6	60	80
N	N	N	N	N	N	N	Y	10	15	15	15	40	30	15	20	92
Y	Y	Y	Y	Y	Y	N	N	30	45	50	65	90	70	53.3	70	72
N	Y	N	Y	N	Y	N	N	30	40	40	45	60	45	41.6	60	80

N	N	N	N	N	N	N	N	10	15	15	15	25	15	15	20	92
N	Y	N	N	N	N	N	N	15	15	25	30	45	25	23.3	30	92
N	N	Y	N	N	N	N	Y	15	15	20	15	60	45	18.3	30	82
N	N	N	N	N	N	N	N	10	15	15	15	45	30	15	20	92
N	N	N	N	N	N	N	N	5	15	15	15	10	15	15	20	92
N	N	N	N	N	N	N	N	10	15	15	15	45	30	15	20	92
Y	Y	Y	Y	N	Y	N	N	30	40	40	45	60	45	41.6	60	80
N	N	N	N	N	N	N	N	10	15	15	15	20	25	15	20	92
N	Y	N	N	N	N	N	N	15	15	25	30	45	25	23.3	30	92
N	N	N	N	N	N	N	Y	5	15	15	15	10	15	15	20	92
N	Y	Y	Y	N	N	N	N	30	40	40	45	60	45	41.6	60	80
N	N	N	N	N	N	N	N	5	10	10	15	15	20	11.6	15	100
N	N	N	N	N	N	Y	N	10	15	15	15	10	15	15	20	92
N	Y	N	N	N	N	N	N	15	15	15	15	45	25	23.3	30	92
Y	Y	N	Y	Y	Y	N	N	30	45	50	65	90	70	53.3	70	72
N	N	N	N	N	N	N	N	15	15	20	25	80	70	20	20	82
Y	Y	N	Y	N	Y	N	Y	30	40	40	45	60	45	41.6	60	80
N	N	N	N	N	N	N	Y	10	15	15	15	10	15	15	20	92

L 250Hz	L 500Hz	L 1KHz	L 2KHz	L 4KHz	L 8KHz	L PTA	L SRT	L SIS %	R GRADE PTA	L GRADE PTA	SIDE NIHL PTA	R OAE	L OAE	SIDE OAE
15	15	15	15	45	30	15	20	92	NIHL	NIHL	B/L	NIHL	NIHL	B/L
10	15	15	15	20	30	15	20	92	Normal	Normal	X	Normal	NIHL	U/L
15	15	15	15	20	25	15	20	92	Normal	Normal	X	Normal	Normal	X
20	30	35	60	90	70	41.6	40	82	NIHL	NIHL	B/L	NIHL	NIHL	B/L
15	20	25	25	45	30	23.3	30	92	NIHL	NIHL	B/L	NIHL	NIHL	B/L
15	20	25	25	45	30	23.3	30	92	NIHL	NIHL	B/L	NIHL	NIHL	B/L
10	15	15	15	45	30	15	30	92	NIHL	NIHL	B/L	NIHL	NIHL	B/L
10	15	15	15	25	20	15	20	92	Normal	Normal	X	Normal	Normal	X
10	15	15	15	15	20	15	20	92	Normal	Normal	X	NIHL	Normal	U/L
5	10	10	10	45	30	10	20	92	NIHL	NIHL	B/L	NIHL	NIHL	B/L
30	45	50	65	90	70	53.3	70	72	NIHL	NIHL	B/L	NIHL	NIHL	B/L
30	45	50	70	90	70	55	70	72	NIHL	NIHL	B/L	NIHL	NIHL	B/L
10	15	15	15	20	35	15	20	92	Normal	Normal	X	Normal	NIHL	U/L
15	15	25	30	45	25	23.3	30	92	NIHL	NIHL	B/L	NIHL	NIHL	B/L
10	15	15	15	15	20	15	20	92	Normal	Normal	X	Normal	Normal	X

10	15	15	15	20	15	15	20	92	Normal	Normal	X	Normal	Normal	X
5	10	15	10	25	30	11.6	15	100	Normal	Normal	X	Normal	Normal	X
10	15	15		35	35	15	20	92	Normal	Normal	X	Normal	Normal	X
10	15	15	15	20	15	15	20	92	Normal	Normal	X	Normal	Normal	X
15	15	25	30	45	25	23.3	30	92	NIHL	NIHL	B/L	NIHL	NIHL	B/L
10	15	15	15	20	15	15	20	92	Normal	Normal	X	Normal	Normal	X
15	20	25	25	45	20	23.3	30	92	Normal	NIHL	U/L	NIHL	NIHL	B/L
10	15	15	15	25	15	15	20	92	Normal	Normal	X	Normal	Normal	X
15	15	15	15	45	30	15	20	92	Normal	NIHL	U/L	NIHL	NIHL	B/L
10	15	15	15	10	10	15	20	92	Normal	Normal	X	Normal	Normal	X
15	15	15	15	45	30	15	20	92	NIHL	NIHL	B/L	NIHL	NIHL	B/L
5	10	15	10	15	15	11.6	15	100	Normal	Normal	X	Normal	Normal	X
10	15	15	15	25	15	15	20	92	Normal	Normal	X	Normal	Normal	X
10	15	15	15	20	15	15	20	92	Normal	Normal	X	Normal	Normal	X
25	30	30	30	50	40	30	40	82	NIHL	NIHL	B/L	NIHL	NIHL	B/L
15	15	20	15	60	45	18.3	30	82	NIHL	NIHL	B/L	NIHL	NIHL	B/L
10	15	15	15	40	30	15	20	92	NIHL	NIHL	B/L	NIHL	NIHL	B/L
5	15	15	15	10	15	15	20	92	Normal	Normal	X	Normal	Normal	X
10	15	15	15	20	15	15	20	92	Normal	Normal	X	Normal	NIHL	U/L
5	15	15	15	20	20	15	20	92	Normal	Normal	X	Normal	Normal	X
15	15	15	15	45	30	15	20	92	15 NIHL	NIHL	B/L	NIHL	NIHL	B/L
10	15	15	15	10	15	15	20	92	Normal	Normal	X	Normal	Normal	X
10	15	15	15	40	25	15	20	90	Normal	NIHL	U/L	NIHL	NIHL	B/L
30	45	50	65	90	70	53.3	70	72	NIHL	NIHL	B/L	NIHL	NIHL	B/L
10	15	15	15	10	15	15	20	92	Normal	Normal	X	Normal	Normal	X

15	25	25	30	55	40	28.3	40	82	NIHL	NIHL	B/L	NIHL	NIHL	B/L
15	15	15	15	45	30	15	20	92	NIHL	NIHL	B/L	NIHL	NIHL	B/L
15	25	25	30	55	40	28.3	40	82	NIHL	NIHL	B/L	NIHL	NIHL	B/L
5	15	15	15	20	15	15	20	90	Normal	Normal	X	Normal	Normal	X
5	15	15	15	10	15	15	20	90	Normal	Normal	X	Normal	Normal	X
15	15	15	15	45	30	15	20	92	Normal	NIHL	U/L	NIHL	NIHL	B/L
5	15	15	15	10	15	15	20	92	Normal	Normal	X	Normal	Normal	X
10	15	15	15	25	15	15	20	90	Normal	Normal	X	Normal	Normal	X
5	15	15	15	10	15	15	20	92	Normal	Normal	X	Normal	Normal	X
30	45	50	65	90	70	53.3	70	72	NIHL	NIHL	B/L	NIHL	NIHL	B/L
15	15	20	15	60	45	18.3	30	82	NIHL	NIHL	B/L	NIHL	NIHL	B/L
15	15	15	15	45	30	15	20	92	NIHL	NIHL	B/L	NIHL	NIHL	B/L
15	15	25	30	45	25	23.3	30	92	NIHL	NIHL	B/L	NIHL	NIHL	B/L
15	25	25	30	55	40	28.3	40	82	NIHL	NIHL	B/L	NIHL	NIHL	B/L
15	25	25	30	55	40	28.3	40	82	NIHL	NIHL	B/L	NIHL	NIHL	B/L
10	15	15	15	15	20	15	20	92	Normal	Normal	X	Normal	Normal	X
30	40	40	45	60	45	41.6	60	80	NIHL	NIHL	B/L	NIHL	NIHL	B/L
15	15	15	15	45	30	15	20	92	Normal	Normal	X	Normal	Normal	X
30	40	40	45	60	45	41.6	60	80	NIHL	NIHL	B/L	NIHL	NIHL	B/L
10	15	15	15	20	15	15	20	92	NIHL	Normal	u/l	Normal	Normal	X
30	45	50	65	90	70	53.3	70	72	NIHL	NIHL	B/L	NIHL	NIHL	B/L
30	40	40	45	60	45	41.6	60	80	NIHL	NIHL	B/L	NIHL	NIHL	B/L
10	15	15	15	25	15	15	20	92	Normal	Normal	X	Normal	Normal	X
15	15	25	30	45	25	23.3	30	92	NIHL	NIHL	B/L	NIHL	NIHL	B/L

15	15	20	25	80	70	20	20	82	NIHL	NIHL	B/L	NIHL	NIHL	B/L
15	15	15	15	45	30	15	20	92	NIHL	NIHL	B/L	NIHL	NIHL	B/L
5	15	15	15	10	15	15	20	92	Normal	Normal	X	Normal	Normal	X
15	15	15	15	45	30	15	20	92	NIHL	NIHL	B/L	NIHL	NIHL	B/L
30	40	40	45	60	45	41.6	60	80	NIHL	NIHL	B/L	NIHL	NIHL	B/L
10	15	15	15	20	15	15	20	92	Normal	Normal	X	NIHL	Normal	U/L
15	15	25	30	45	25	23.3	30	92	NIHL	NIHL	B/L	NIHL	NIHL	B/L
5	15	15	15	10	15	15	20	92	Normal	Normal	X	Normal	Normal	X
30	40	45	45	65	45	45	60	80	NIHL	NIHL	B/L	NIHL	NIHL	B/L
5	10	10	10	15	20	10	15	100	Normal	Normal	X	Normal	Normal	X
10	15	15	15	10	15	15	20	92	Normal	Normal	X	Normal	Normal	X
15	15	15	15	45	25	23.3	30	92	NIHL	NIHL	B/L	NIHL	NIHL	B/L
30	45	50	65	90	70	53.3	70	72	NIHL	NIHL	B/L	NIHL	NIHL	B/L
15	15	20	25	80	70	20	20	82	NIHL	NIHL	B/L	NIHL	NIHL	B/L
30	40	40	45	60	45	41.6	60	80	NIHL	NIHL	B/L	NIHL	NIHL	B/L
15	15	15	20	40	20	15	20	92	Normal	Normal	X	Normal	Normal	X