DISSERTATION ON

"INCIDENCE OF NOISE INDUCED HEARING LOSS AMONG METROPOLITAN CITY TRAFFIC POLICE PERSONNEL"

Dissertation submitted

in partial fulfilment of the regulations for the award of the degree of

M.S.DEGREE BRANCH-IV OTORHINOLARYNGOLOGY

Of

THE TAMIL NADU Dr. M.G.R. MEDICAL UNIVERSITY

UPGRADED INSTITUTE OF OTORHINOLARYNGOLOGY

MADRAS MEDICAL COLLEGE

CHENNAI

APRIL 2011.

CERTIFICATE

This to certify that this dissertation **"INCIDENCE OF NOISE INDUCED HEARING LOSS AMONG METROPOLITAN CITY TRAFFIC POLICE PERSONNEL"** submitted by **KARUPPASAMY.C,** appearing for M.S ENT Branch IV Degree examination in April 2011 is a bonafide record of work done by him under my direct guidance and supervision in partial fulfilment of the Tamilnadu Dr.M.G.R Medical University, Chennai. I forward this to the Tamilnadu Dr.M.G.R Medical University, Chennai, Tamilnadu, India.

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In keeping with the maxim, "All is well that ends well", I was able to carry out my study to my fullest satisfaction. I thank the guidance, encouragement, motivation and constant supervision extended to me by my respected Teacher **Prof. Jacinth C.Cornelius M.S., D.L.O., The Director & Professor,** Upgraded Institute of Otorhinolaryngology.

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I am extremely thankful to my family members for their continuous support. Above all I thank God Almighty for his immense blessings.

CONTENTS

INTRODUCTION

The term noise induced hearing loss is related to reduction in auditory acuity associated with noise exposure. This situation may be temporary and is described as temporary threshold shifts (TTS). The hearing loss may be from hours to days and recovered within 16 hours to 48 hours. The hearing loss may be permanent and described as permanent threshold shift (PTS). PTS may occur following single episode of noise exposure. PTS shifts divided into Acoustic trauma and Noise included hearing loss. Acoustic trauma results from single exposure to an intense sound and leads to an immediate hearing loss. NIHL results from prolonged noise exposure that is more than 85dB of sound level. It depends on the intensity, frequency and duration of the noise exposure. The pure tone audiometry shows characteristic notch with maximum reduction in sensitivity to stimulation in the range from 3 to 6 kHz and recovery at 8 kHz. NIHL results from loss of structural hair cell function. NIHL associated with other causes of hearing loss may not be present with the characteristic 4kHz notch. Absence of the notch does not excluded noise induced hearing loss. Pure tone audiometry is the corner stone of investigation to find out the noise induced hearing loss. And to find out the earlier pathology of hair cell dysfunction before the NIHL present clinically, Otoacoustic emission is the best investigation.

AIMS OF THE STUDY

1. To evaluate the incidence of noise induced hearing loss among Metropolitan city traffic police personnel.

2. To evaluate the degree of hearing loss with respect to the amount of noise exposure.

3. To evaluate the duration of noise exposure with respect to the development of hearing loss.

REVIEW OF LITERATURE

1) Ramazzini 1713 – reported that workers who hammered copper for a living became hard of hearing.

2) Nils skragger over 200 years ago wrote a thesis on occupational deafness in Coppersmiths and blacksmiths.

3) Kylin-1960 and forstroke – 1831 – Accurate description of NIHL in blacksmiths coining the expression as blacksmith deafness.

4) 1880 – Rock and Holt in America, Bezold in Germany and Bear in Great Britain – recognized the importance of industrial noise as a cause of hearing loss.

5) Holt in 1882- and Glasgow 1886 first describe the Boiler maker's deafness Haberman – 1890- site and nature of the lesion in the ear produced by noise – partial disappearance of organ of corti with destruction of the hair cells.

6) Fowler 1979 – observed dips at 4 kHz in Pure Tone Audiometry.

7) Bunch 1939- Published the first audiometric data demonstrating the typical high frequency hearing loss.

8) Howkins 1976 – Excellent Historical services of NIHL.

9) George Van Bekesy 1961 – Travelling wave theory laid the foundation to a modern theory of how the Cochlea functions.

10) 1950 and 1960 – Katsuli, Erans and kiang study the individual fibres of acoustic nerve.

11) 1970 – Heinrich spoendlin- To map out the innervation of the organ of corti.

12) Waver's volley resonance theory – 1949.

13) Helmholt's place theory – 1883.

14) Rutherford's frequency/ Telephone Theory – 1886.

15) Alphonsa Corti – 1st described the organ of corti – 1851.

MATERIALS AND METHODS

AIMS AND OBJECTIVE: To find out the incidence of noise induced hearing loss among metropolitan city traffic police personnel

STUDY PLACE: Government General Hospital, Chennai 600003.

COLLABORATING DEPARTMENT: Upgraded Institute of Otorhinolaryngology,

Department of Neurotology

Department of Audiology

STUDY DESIGN: Prospective

STUDY PERIOD: FEBRUARY 2010 TO APRIL 2010

INCLUSION CRITERIA: Police personnel working in Chennai city as traffic Regulators

.

EXCLUSION CRITERIA:

People with previous ear disease like CSOM, congenital malformations,

People with previous ear surgeries

People on ototoxic drugs, head injury, any other major exanthematous illness

People with positive family history of HOH

INVESTIGATION: Pure Tone Audiometry [PTA]

METHODOLOGY

This study was conducted at the Upgraded Institute of Otorhinolaryngology, Government General Hospital, Madras Medical College, Chennai-3.From the Period of February 2010 to December 2010.1367 subjects were analyzed all are males and within the age group of 30 to 57 years. Among these 660 subjects-Metropolitan city traffic police personnel working as traffic regulators in the main traffic signal areas for more than three months duration, history and ENT examination wise normal were considered as **cases.**707 subjects selected as **controls,** from the general population, other than traffic regulators, without known history of noise exposure. The cases and controls were investigated by Pure Tone Audiometry and screened for hearing loss. According to the PTA finding the cases were divided in to Normal, Noise Induced Hearing Loss, Sensory Neural Hearing Loss, Mixed Hearing Loss and Conductive Hearing Loss.NIHL cases again evaluated for severity, age distribution, unilateral or bilateral and duration of work. Intensity of noise exposure in the main traffic signal areas was measured during the peak period of traffic with sound pressure meter in sound pressure level [SPL] dB- SPL A mode. Chennai Metropolitan city divided into three zones by two roads Poonemalle high road &Annasalai. Cases with normal hearing with 4 kHz dip, High frequency hearing loss-3kHz to 6 kHz with some

recovery at 8 kHz and Sensory neural hearing loss with typical 4 kHz dip. Cases with Sensory neural hearing loss underwent special audiological tests- Speech Audiometry and Tone Decay Test.Conductive Hearing Loss further evaluated by Tympanometry.

DESCRIPTIVE ANATOMY OF THE LABYRINTH

Inner ear lies in the petrous part of the temporal bone. Bony labyrinth consists of cochlea, vestibule and semicircular canals. Membranous labyrinth lies within the bony labyrinth, it consists of cochlear duct, utricle and saccule and three semicircular ducts. The space between the inner periosteum of the bony labyrinth and membranous labyrinth is filled with perilymph which is an extracellular like fluid that is high in sodium and calcium and low in potassium. The membranous labyrinth contains endolymph which is an intracellular like fluid rich in potassium and low in sodium and calcium. The ionic composition and potentials are essential to the primary function of the inner ear because they provide the driving force for mechanotransduction.

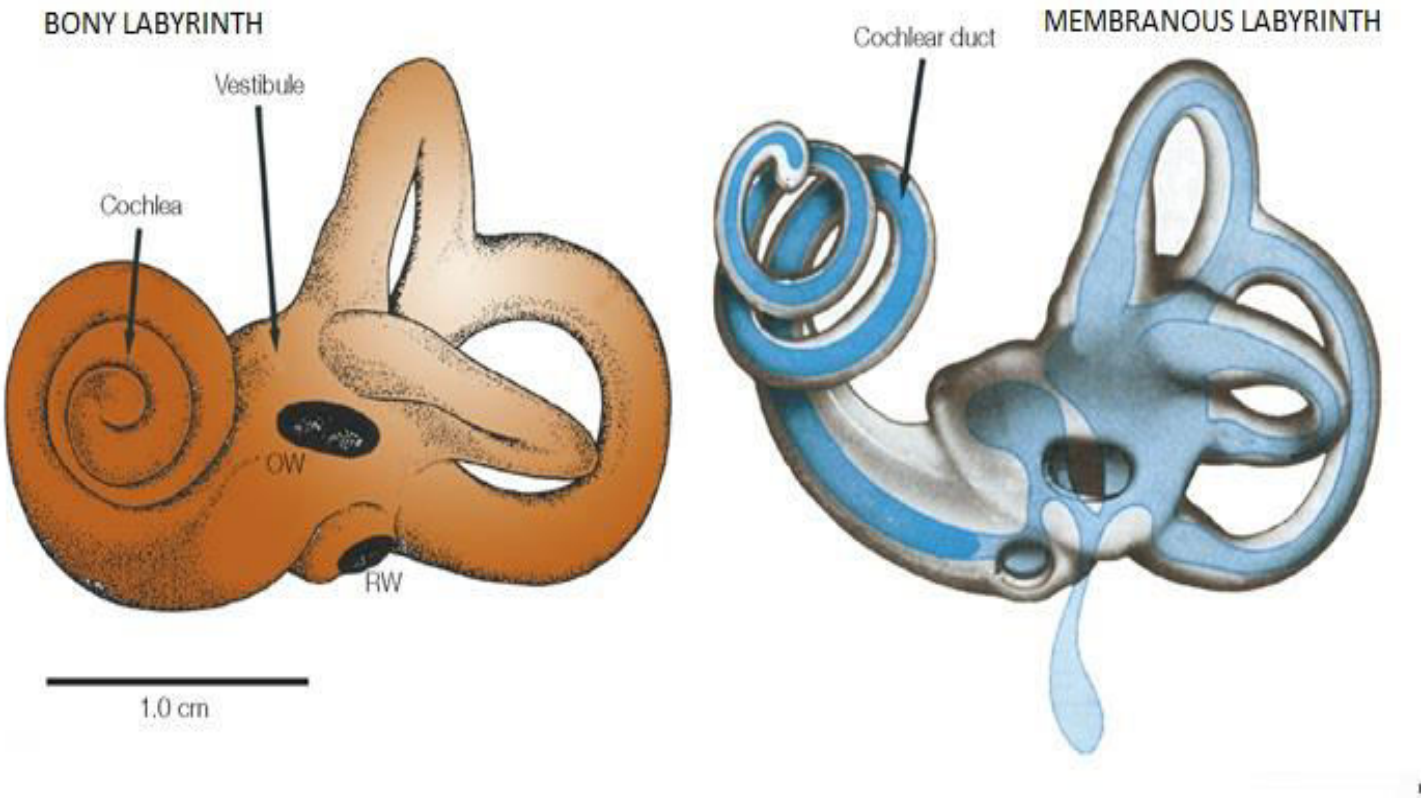
COCHLEA:-

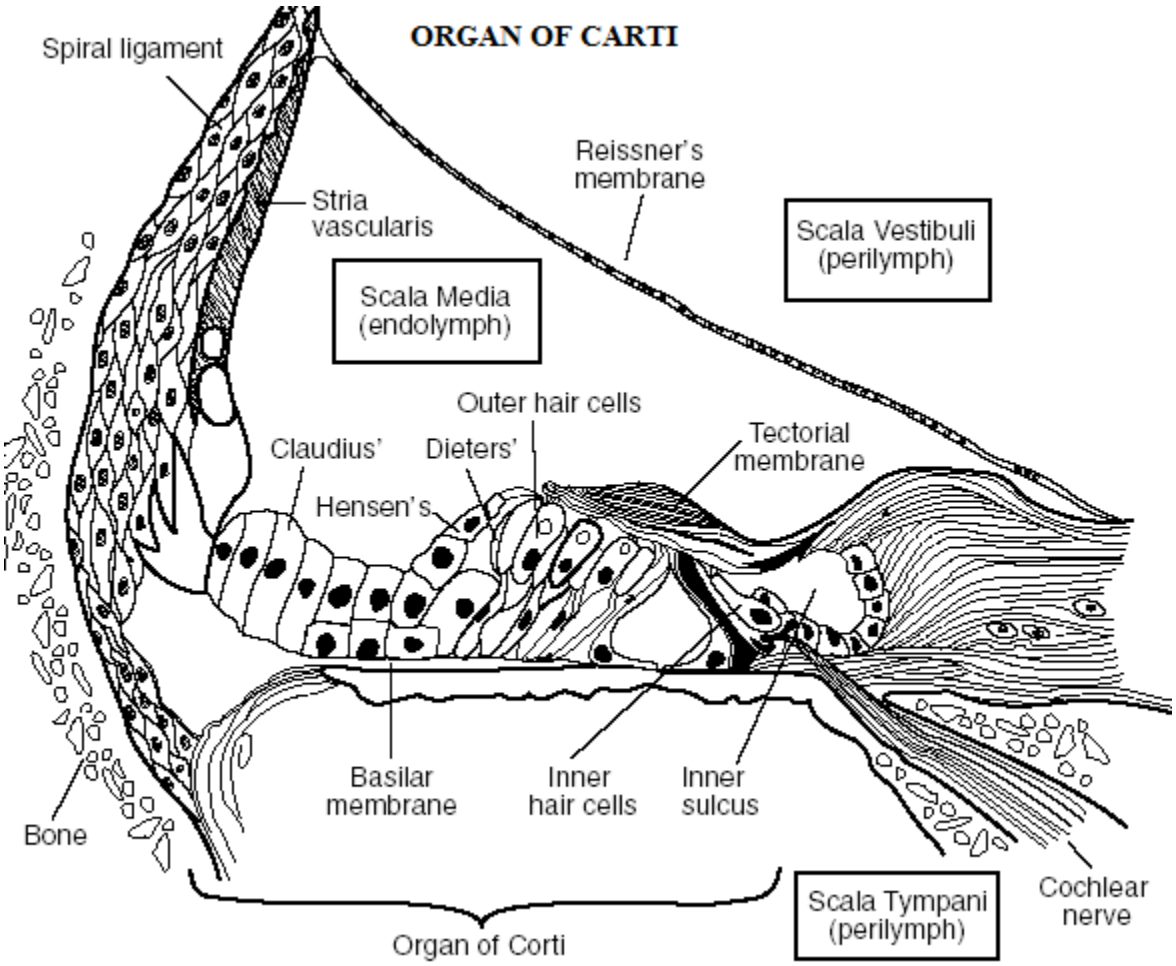
Organ of hearing, the name derived from the Greek word cochlea means snail. It is the most anterior part of labyrinth lying in front of the vestibule. The shell has 2 ¾ turns and its height is about 5 mm, greater distance across the base in 9m, and 35mm long. The coil of the cochlea turns about a central core or modiolus. Its apex or cupula points towards the anterosuperior part of the medial wall of the tympanic cavity. The base faces the bottom of the internal acoustic meatus, basal coil forms the bulge in the medial of the middle ear called the promontory. Bony

spiral lamia arising from the edge of the modiolous, membranous spiral lamina or basilar membrane extends from the edge of the bony spiral lamina to the outer wall of the cochlea, there by dividing each coil into upper part scala vestibule, which commences from the fenestra vestibule or oval window, sealed by annular ligament and foot plate of stapes and lower part, scala tympani commences from the fenestra cochlea or round window covered by secondary tympanic membrane. At the apex these two perilymphatic spaces communicate with each other via minute channel called the helicotrema. The middle part-scala media or cochlear duct. At the base of the cochlea in the floor of the scale tympani another opening is present from which the perilymphatic fluids communicates with the subarachnoid space via the cochlear aqueduct in the base of the osseous spiral lamina, Rosenthal canal is present which accommodate the bipolar ganglion cells of the spiral (cochlea] ganglion. From the Rosenthal canal many tiny canals habnerula perforta radiate through the osseous spiral lamina to its rim, they carry fascicles of the cochlear nerves to the organ of corti.

COCHLEAR DUCT OR SCALA MEDIA:-

It is a spirally arranged tube lying on the upper surface of the spiral lamina. The average length is around 34mm (29 to 40mm). It is triangular in cross section.


BONY LABYRINTH
MEMBRANOUS LABYRINTH
Vestibule
Cochlea
Cochlear duct
OW
RW
1.0 cm


ORGAN OF CARTI
Spiral ligament
Reissner's membrane
Stria vascularis
Scala Vestibuli (perilymph)
Scala Media (endolymph)
Outer hair cells
Claudius'
Dieters'
Tectorial membrane
Hensen's
Basilar membrane
Inner hair cells
Inner sulcus
Bone
Scala Tympani (perilymph)
Cochlear nerve
Organ of Corti

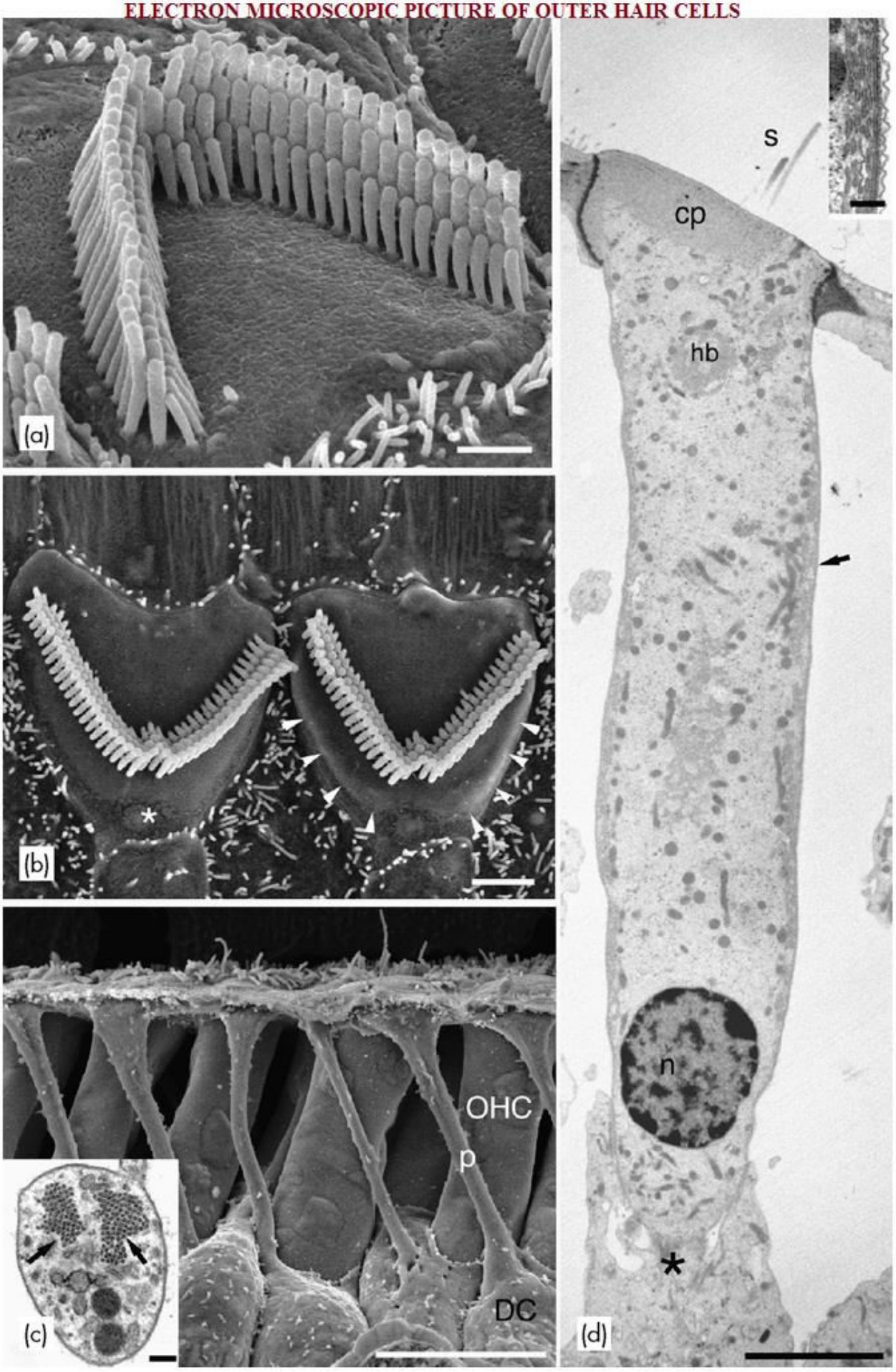
FLOOR OF THE COCHLEAR DUCT:-

Floor is formed by bony spiral lamina which separates into two ridges, upper ridge in the spiral limbus, from which the tectorial membrane originates, lower ridge gives rise to the membranous spiral lamina (Basilar membrane) through which the acoustic nerve fibers reach the organ of corti, the outer thicker zone pectinata starts beneath the base of the outer pillar cells to basilar crest, Basilar membrane length 35mm width increases from 0.21mm basally to 0.36mm at tis apex. Its separates the scala media from the scala tympani. Basilar membrane is separated from the spiral limbus by the inner sulcus, it is an open channel bounded by the lateral edge of the spiral limbus, medial edge of the organ of corti and apically by the tectorial membrane. Separated from the lateral wall by the internal sulcus, it is an open channel between the spiral prominence and the Claudius' cells. The organ of corti consists of epithelial structures that are lies on the zona arcuata of the basilar membrane. It consists of supporting cells and the auditory sensory cells. Arrangements of cells from lateral to medial are Hensen's cells, Outer tunnel of corti, 3 to 5 rows outer hair cells, Dieters' cell with its phalangeal process, spaces of Nuel, outer pillar cells, inner tunnel of corti, inner pillar cells, inner hair cell, inner phalangeal cell and inner border cell. Inner pillar cells are 60000 in number, form 60 degree angle with basilar membrane. These two pillar cells are contact in the apex and form tunnel of corti, Deiters' cells support the outer hair cells. Space

of the Nuel is a fluid filled space between the outer hair cell bodies and the phalangeal process of the Deiters' cells. Inner border cell and phalangeal cell separate the inner sulcus cells from the medial surface of the inner hair cell. The sensory cells are inner and outer hair cell. Each hair cell consists of body which lies within the organ of corti, thickened upper surface called the cuticular plate, from which cluster of stereocilia or hair cells projects.

Inner hair cells form single row, 3500 in number small apex bears 50 to 60 stereocilia and large flask shaped body, nerve fibres and nerve endings are located around the lower half of the body. The outer hair cells form 3 to 5 rows, 12000 in numbers, long cylindrical shape body, apex bears several rows of stereocilia but the configuration varies from 'w' shape at the base 'v' shape in the middle and linear array at the apex. The no of stereocilia also decrease from the base to apex, longest row of stereocilia in outermost and shortest row in innermost. There is a side link. Tip links were present between the adjacent stereocilia. The tectorial membrane arises from the spiral limbus and extends over the organ of corti and attach close to the Hensen cell region. The membrane is an acellular gel like matrix. The tips of the longest stereocilia of the outer hair cells are attached to or embedded in the undersurface of the tectorial membrane.

ELECTRON MICROSCOPIC PICTURE OF OUTER HAIR CELLS



LATERAL WALL OF THE COCHLEAR DUCT:-

It consists of three distinct zones, stria vascularis above, spiral prominence below and the transitional zone between the two and spiral ligament. Stria vascularis form the bulk of the lateral wall and consists of three cell layers, marginal, intermediate and basal cell layer. Marginal cells are seems to be main functional unit. It is thought to play an active role in the maintenance of the ionic composition and electrical potential of the endolymph.

ROOF OF THE COCHLEAR DUCT:-

Reissner's membrane → It stretches between the bony spiral lamina to the upper part of the lateral wall of the cochlear duct. It consists of typical squamous epithelial cell with microvilli on their surface, perilymphatic surface contain melanocytes.

INNERVATION OF THE COCHLEA:-

Cochlea is innervated by three types of fibres, Autonomic, afferent and efferent. Autonomic fibres travel along the blood vessels supplying the cochlea[6].

Afferent nerve fibres carrying sensory information to the brainstem, their cell bodies are located in the spiral ganglion. Cochlear nerve contains 30000 myelinated nerve fibres, virtually all afferent. These fibres pass through the modiolus to the spiral canal, 95% spiral ganglion cells are type I cells both

myelinated and unmyelinated.These are bipolar and innervate the inner hair cells. Each inner hair cell receives 20 peripheral processes of the dendrites, 5% are type II cells, unmyelinated, and their cell bodies are unipolar and supply the outer hair cells. Central process reaches the cochlear nuclei. It is sub divided into Dorsal and Ventral cochlear nuclei. Ventral cochlear nuclei again subdivided into Anteroventral (AVCN) and posteroventral (PVCN) nuclei.

The auditory nerve afferent in the AVCN terminate on the principal projection neurons of the cochlear nuclear complex and expands into a very large terminal called the end bulb of Held. Fibers for low frequency <1 kHz may branch to form two end bulbs. AVCN is responsible for the original frequency selectivity and sensitivity of the cochlear response. The DCN or PVCN responsible for the precise time arrival of sounds, It may be involved in acoustic startle response. DCN are important in determining what sounds are. The dorsal pathway from cochlear nuclei reaches the inferior colliculus. The ventral pathway divides further and projects to both the ipsilateral and contralateral superior olivary complex. It is the first part of the ascending pathway where major binaural comparison can be made. SOC is to function in sound localization, it contains 'S' shaped lateral nucleus, it receives an excitatory input from ipsilateral CN and an inhibitory input from the contralateral CN, LSON detect the differences in the sound intensity. Disc shaped medial olivary nucleus (MON) together with the trapezoid body form periolivary

nuclei. MON localize the sound and detect specific inter aural sound differences. The inferior colliculi receive direct input from the brainstem auditory nuclei via a tract called the lateral lemniscus, located in the Midbrain.The central nucleus of inferior colliculi is layered into isofrequency bands, the higher frequency bands are found towards the midline and low frequency bands towards the outside producing a tonotopic map.It is basis for recognizing pattern in sound and sound localization. The thalamus contain the medial geniculate body - three major division each receiving a separate parallel pathway from the inferior colliculi, the ventral division organized tonotopically into isofrequency layers, Dorsal division not tonotopically organized the medial division receives multimodels input. The primary auditory cortex (A1) corresponding to Broadman's area 41 within the lateral fissure of the temporal lobes it receives main projection from ventral division of the MGB. Low frequency is the rostral end and high frequency in the dorsal end organized into isofrequency layers. Complex reponses can be found in neurones in the area surrounding A1 it detects specific delays and simultaneous occurrence of harmonically related frequencies.

EFFERENT OR DESCENDING PATHWAYS:-

Olivocochlear bundle in the major descending projection, largely supply the outer hair cells minimal amount to the inner hair cells. Their cell bodies are located in the brainstem SOC

VASCULAR SUPPLY:-

Labyrinthine Artery → common cochlear artery → spiral modiolar artery → radial arteries.Cochlear branch of the vestibulocochlear artery supply the spiral ganglion, osseous spiral lamina, limbus, spiral ligament

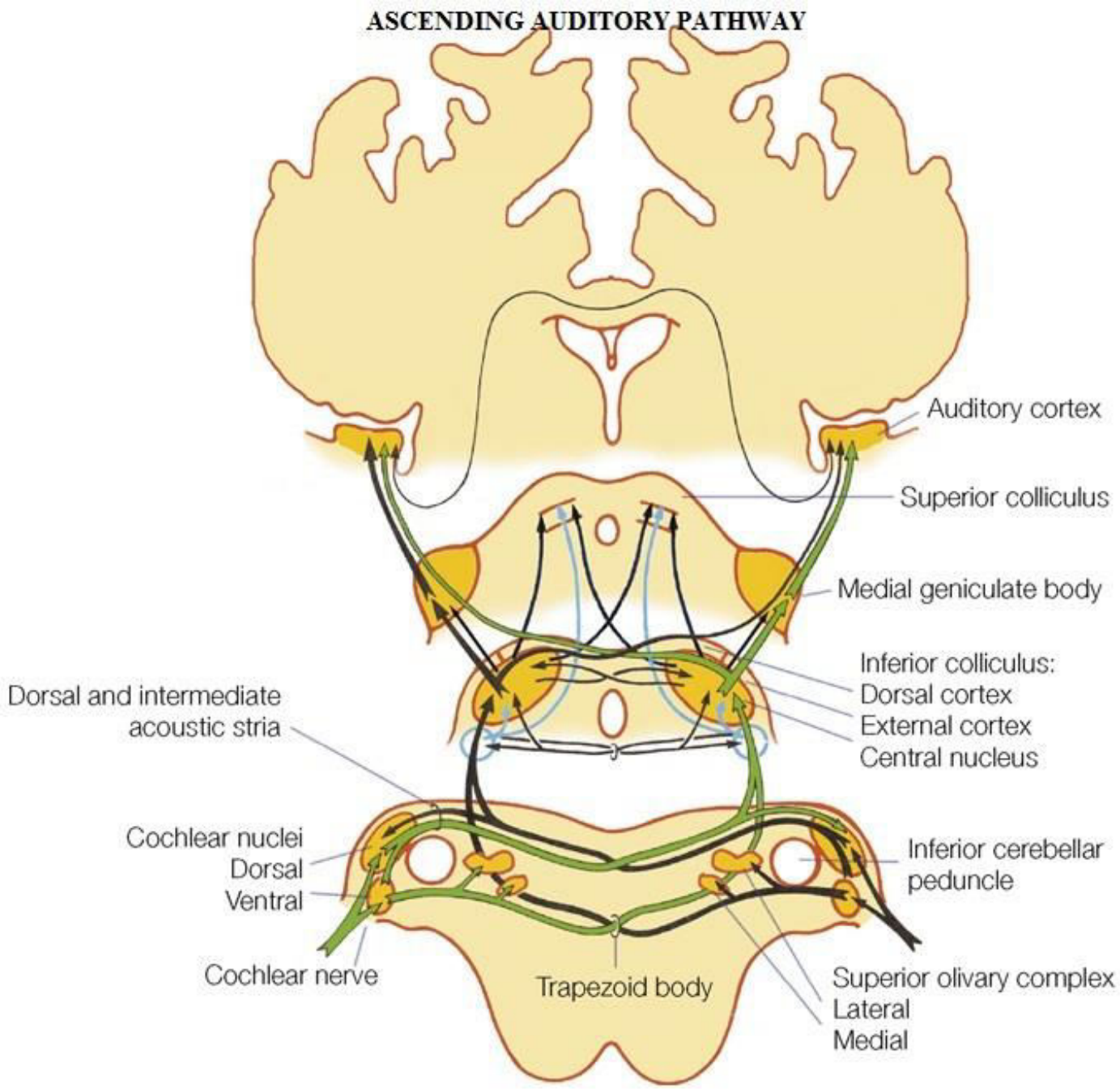
VENOUS DRAINAGE:-

Apical region – Anterior spiral vein

\Basal region – posterior spiral vein

These two joins with the anterior and posterior division of the vestibular vein in the region of the basal turn of cochlea, to form the vein of the cochlea which empties into the jugular bulb.

ASCENDING AUDITORY PATHWAY



PHYSIOLOGY OF THE COCHLEA:-

The sound wave collected in the external ear canal produce vibration of the tympanic membrane, it increases the tympanic membrane pressure in a frequency sensitive way, which in turn vibrates middle ear ossicles. An efficient middle ear impedance transformer will change the low pressure, high displacement vibration of the air into high pressure, low displacement vibration suitable for driving the cochlear fluids. The vibratory motion of the stapedial foot plate that transmit the mechanical energy of the ossicular chain directly through the oval window of the cochlea, effectively delivering sound pressure waves to the scala vestibule and translating mechanical motion into pressure waves that propagate through the virtually compressible cochlear fuids at a velocityof approximately 1.5 Km/Sec. Higher pressure scala vestibule relative to scala tympani produce a pressure differential across the cochlear partition that creates intra cochlear forces that set the passion in motion. The basilar membrane width decreases from base to apex. As well as thickness and compositions of radial filaments decreases basoapically. The basilar membrane and organ of corti complex is stiffen less massive at the base than at the apex. The high frequency acoustic events are preferentially transduced in the base because it is stiff and less massive. The inverse is true for low frequency acoustic events.

The outer hair cells play a fundamental role in the active cochlear mechanics, which uses the biological energy to boost the mechanical vibration of the basilar membrane. The basilar membrane much more sharply tuned for the frequency filtering. The high frequency is detected at the base and low frequency at apex and mid frequency in between this two- Von Bekesy-1960. The displacement of the basilar membrane results in a radial shearing motion between the reticular lamina and tectorial membrane, a motion that serves as the mechanical trigger of transudation currents. The shearing motion between the reticular lamina and tectorial membrane causes stereocilia to bend in the direction of the modiolus or spiral limbus depending on whether the basilar membrane is displaced towards the scala tympani or scala vesibuli respectively. The relative mechanical stimuli to the IHC stereocilia seems to be the flow of endolymph within the subcortical space.it is the narrow channel between the reticular lamina and the tectorial membrane, when the OHCs contract, the reticular lamina and the tectorial membrane are pulled together, enhancing overall basilar movement displacement during active mechanics.The magnitude and phase of the displacement vary in radial dimension. The tectorial membrane required for OHCs to amplify displacement of the basilar membrane at low frequency levels[2].

TRANSDUCTION BY HAIR CELLS:-

The individual stereocilia on the apical surface of the hair cells are mechanically rigid due to the presence of the actin filaments. Apical surface are braced together with cross links so that they move as a stiff bundle, when the stereocilia are deflected in the direction of the taller stereocilia the tip links are stretched, opening the ion channels in the cell membrane. When the stereocilia deflected in the opposite direction, the tension is taken of the links and the channels are closed. The tip links are responsible for coupling the stimulus movements to the transducer channel. The apical surface of the hair cells is faced by the endolymph with a high positive potential 85 mV. Inside the cell there is a negative intracellular potential -45 mV for inner hair cells and -70 mV for outer hair cells. The potential combines to give 125 mV for inner hair cells or 150 mV for outer hair cells (OHCs). If potential drop across the channel, when the channel open, K^+ ions from endolymph tend to be driven into the cell by their gradient and also by Ca^+ ions and thus making the cell become more positive inside. Most of the transducer current may be carried by the K^+ ions and also by Ca^+ ions. The energy for the whole process comes from the stria vascularis. This inward transducer current flow across the basolaterel membrane of the inner hair cells, producing the receptor potential. The voltage gated Ca channels are activated resembling in an influence

of Ca ions and release of the neurotransmitter from the basolateral membrane of the inner hair cells.

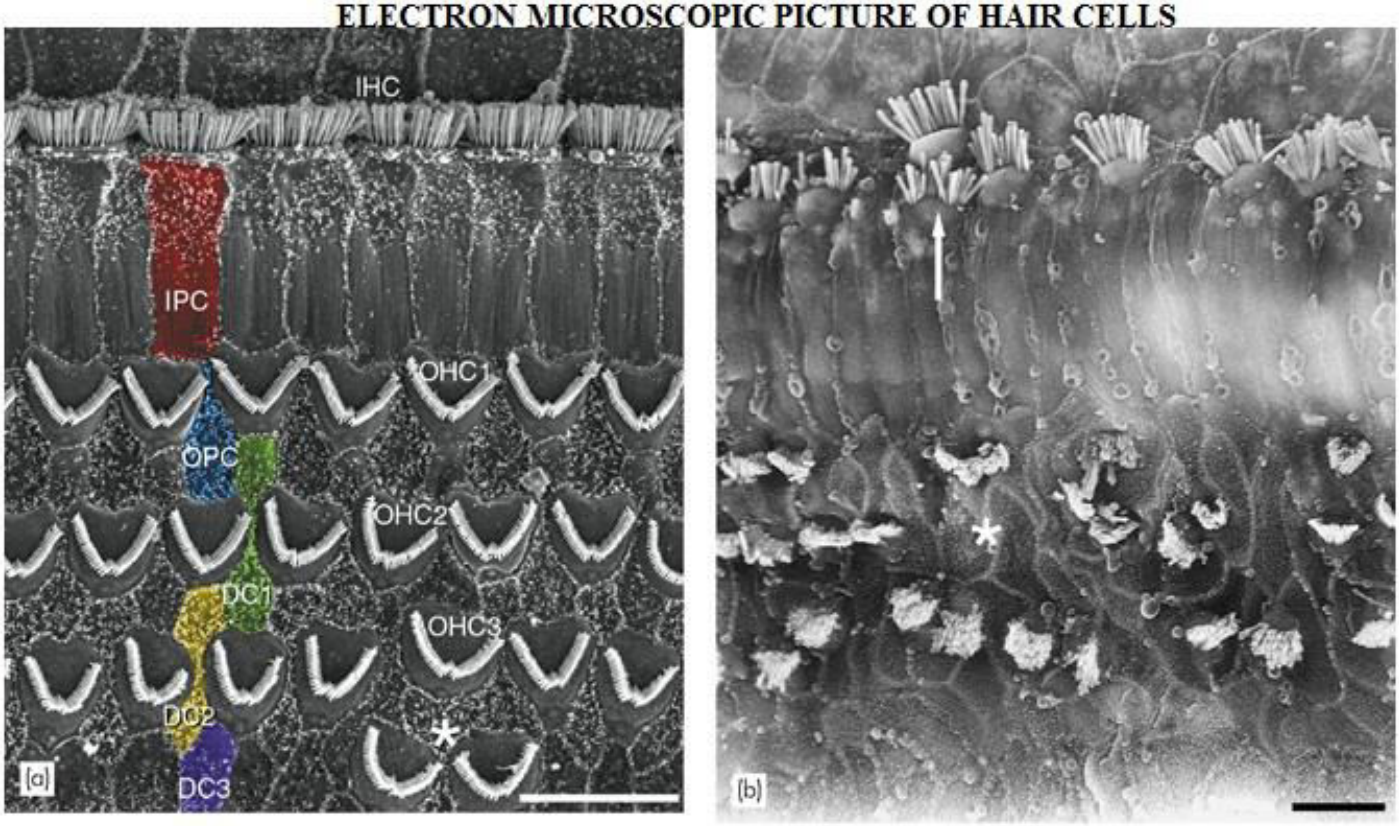
THE ELECTRCAL RESPONSE OF THE COCHLEAR CELLS:-

Inner hair cells → the role of the inner hair cell is to detect the movement of the basilar membrane and transmit it to the auditory nerve. Inner hair cells respond to the velocity rather than just displacement of the basilar membrane. Whenever the tip links ion channels are opened, the cell become positive charged and depolarized. The basolateral wall of the IHCs act as a capacitor. And as the frequency is raised more and more transducer current will flow out through the capacitance of the basolateral wall of the IHCs to the auditory nerve.

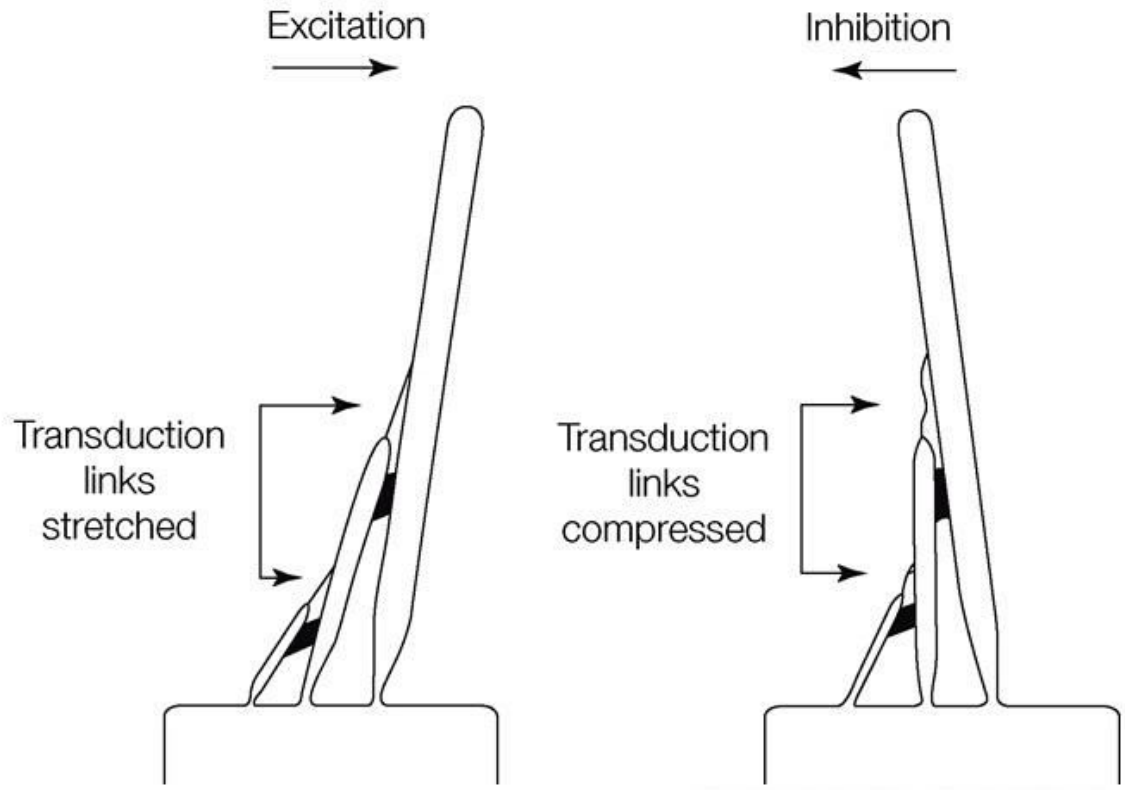
OUTER HAIR CELLS:-

Outer hair cells are potentially involved in generating the active mechanical amplification of the basilar membrane[3]. Vibration which gives rise to large amplitude and sharply tuned mechanical travelling waves. They also create cochlear micro phonics and this may be the essential step in the mechanical amplification. The motile response of the OHCs amplifies the travelling wave to increase the cochlear sensitivity and frequency selectivity.

ELECTRON MICROSCOPIC PICTURE OF HAIR CELLS



Electron microscopic picture of the upper surface of the organ of corti shows single row of inner hair cells and four rows of outer hair cells, inner and outer pillar cells and deiter's cell.



The role of tip links between stereocila in transduction. Three rows of stereocilia are shown in cross section.

THE CROSS ELECTRICAL PESPONSE OF THE COCHLEA:-

1. Neural potential arise from the massed action potential in the auditory nerve fibres as the onset of the stimulus.

2. Cochlear micro phonics – AC current produced by outer hair cells

3. Summating potential – can appear as either a positive or negative shift. It is most likely generated as the distortion components of the OHCs response , and small contribution from inner hair cells

RESPONSE OF THE AUDITORY NERVE FIBRES:-

Neurotransmitter released in the synapses at the base of the IHCs and this gives rise to action potential in the auditory nerve fibres. As the intensity of the stimulus is increased, the amplitude of the basilar membrane vibration grows. Similarly the activation of the inner hair cells grows and also does the firing rate of the auditory nerve fibres.

CENTRIFUGAL INNERVATION OF THE COCHLEA:-

Olivo cochlear bundles arrive from the medial border of the superior olivary complex and projects mainly contralarerally.And innervates the OHCs directly. Acetylcholine is the major neurotransmitter, affects the gains of the active motile response in the isolated outer hair cells.

The steady receptor potential is related in size to the amplitude of the acoustic stimulus. At the same time afferent neurons are stimulated by the synaptic action at the bases of the inner hair cells. They fire more rapidly as the vibration of the basilar membrane increases in amplitude up to a threshold that depends on the sensitivity of the specific nerve fibre involved. This is carried away through the central auditory pathway. The transformation of the physical characteristics of the sound into auditory object is thought to occur in the transition from primary to secondary auditory cortex.

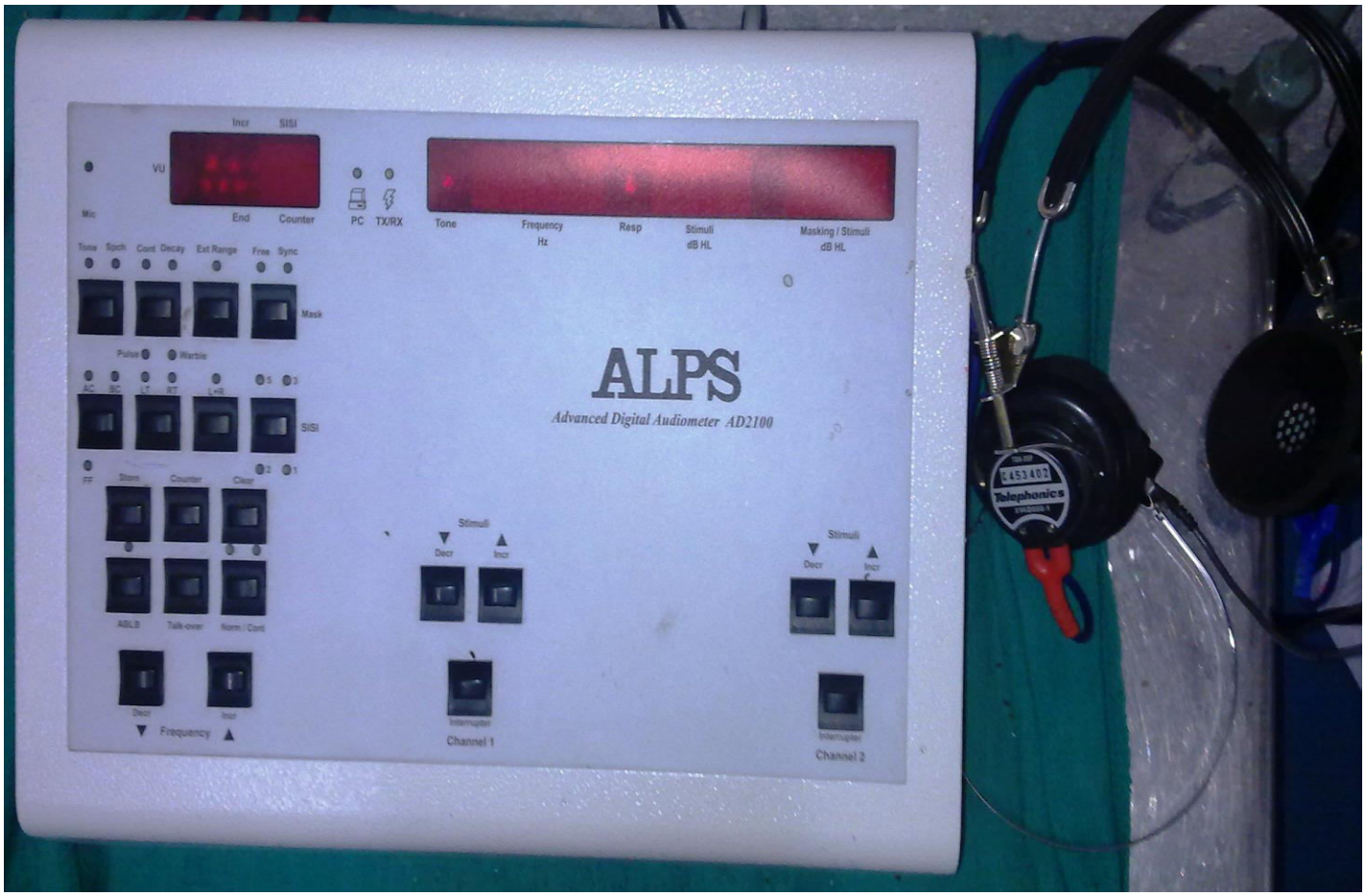
PHYSILOGY OF NOISE

The term noise is commonly used to designate an undesirable sound. In the hearing field, the term has come to mean any excessively loud sound that has the potential to harm hearing[8]. Noise has physical, physiological and psychological connotations all of which differ. Physically - It is a complex sound having little or no periodicity which can be measured on its characteristics analyzed. Physiologically – Noise is defined as a signal that bears no information and whose intensity varies randomly in time. Psychologically – Noise is any sound, irrespective of its wave form which unpleasured or unwanted. Noise like any sound in defined in terms of its duration, frequency spectrum, measured in Hz and intensity is measured in sound pressure level (SPL) and expressed in decibel (dB). It may be continuous, intermittent impulsive or explosive. Impact noise, it is dangerous and frequent in military. The

characteristics of the impact noise vary enormously. Rise and decay times range from abrupt to gently sloping and reverberation varies. The temporal patterns of environmental noise are typically described as continuous, fluctuating, intermittent, or impulsive. Continuous or steady-state noise remains relatively constant, whereas fluctuating noise rises and falls in level over time, and intermittent sounds are interrupted for varying time periods. Impulsive or impact noises caused by explosive or metal-on-metal mechanical events have rapidly changing pressure characteristics consisting of intense, short-lasting (i.e., in terms of milliseconds) wave fronts, followed by much smaller reverberations and echoes that occur over many seconds. The amount of noise, usually referred to as the sound pressure level (SPL), is conventionally measured by a sound-level meter (SLM) in decibel (dB) units using a weighting formula called the A-scale. The dBA-scale measure of sound level essentially mimics the threshold-sensitivity curve for the human ear, so that the low- and high-frequency components are given less emphasis as auditory hazards. Standard SLMs have electronic networks designed to measure noise magnitude automatically in dBA, whereas to measure impulse or impact noise, a special peak-reading SLM is needed that are capable of accurately measuring sounds with essentially instantaneous onset times[3]. The personal noise dosimeter is typically used to measure noise exposure in the workplace. This instrument provides readout of the noise dose or the percent exposure experienced by a

worker, typically over a single shift. The logging dosimeter integrates a function of sound pressure over time and calculates the daily (8-hours) dose with respect to the current permissible noise level for a continuous noise of 85dBA or less SPL lasting 8 hours.

SOUND LEVEL METER



ADVANCED DIGITAL AUDIOMETER AD2100

NOISE-INDUCED HEARING

INTRODUCTION

One of the most common causes of hearing impairment is exposure to excessive sounds. Millions of people are suffer from noise-induced hearing loss (NIHL), resulting in a reduced quality of life because of social isolation and possible inexorable tinnitus, as well as impaired communication with family members, coworkers, and friends. Moreover, the costs in terms of compensation and early retirement payments are immense. The purpose of this study is to present and discuss the most recent perspectives about the effects of noise on hearing that address the scientific and practical aspects of the affliction. Although NIHL has been studied experimentally for over 100 years, it has only been over the last decade or so that some major breakthroughs have occurred in our basic understanding of the ear's reaction to damaging sounds. These increments in our knowledge base about NIHL promise to significantly improve the detection and treatment of this disorder over the coming years

NATURE OF THE LOSS

Depending on the level of the sound exposure, either reversible or permanent damage can occur to the peripheral auditory end organ. The reversible loss,

typically referred to as a *temporary threshold shift (TTS)*, results from exposures to moderately intense sounds that might be encountered, for example, at a philharmonic orchestra concert. Hearing problems associated with TTS include elevated thresholds, particularly for the higher mid-frequency region that includes the 3- to 6-kHz frequencies. The TTS condition is often accompanied by a number of other common symptoms of hearing impairment including tinnitus, loudness recruitment, muffled sounds, and diplacusis. Depending on the duration of the exposure, recovery from TTS can occur over periods ranging from minutes to hours and days. After exposure, if TTS does not recover before the ear is reexposed to excessive sound, a permanent change in hearing occurs that is referred to as a Permanent Threshold shift (PTS).In PTS, the elevation in hearing thresholds is irreversible because, permanent structural damage occurs to the critical elements of the cochlea. The precise relationship between the TTS and PTS stages of hearing loss due to noise exposure is unknown[15]. Thus, TTS was correlated with a buckling of the supporting pillar bodies in the frequency region of the maximal exposure effect. In contrast, the morphologic abnormality that was consistently correlated with PTS was a focal loss of hair cells and a complete degeneration of the corresponding nerve-fiber endings. Because PTS eventually develops from repeated exposures to stimuli that initially produce only TTS, it is likely that the latter condition is also associated with subtle changes to the sensitive

outer hair cell (OHC) system that go undetected by light microscopy. Traditionally, PTS caused by acoustic overstimulation has been separated into two distinct classes. One type, called acoustic trauma, is caused by a single, short-lasting exposure to a very intense sound (e.g., an explosive blast) and results in a sudden, usually painful, loss of hearing. The other type of hearing loss is commonly referred to as NIHL and results from chronic exposure to less intense levels of sound[5]. Consequently, it is well established that a single exposure to a severe sound can result in direct mechanical damage to the delicate tissues of the peripheral auditory apparatus including components of the middle ear (tympanic membrane, ossicles) and inner ear (organ of Corti). In contrast, regular exposure to less intense but still noisy sounds involves the insidious destruction of cochlear components that eventually and unavoidably leads to an elevation in hearing levels, along with other common symptoms of hearing impairment. Irreversible NIHL is a specific pathologic state exhibiting a recognized set of symptoms and objective findings. (1) A permanent sensorineural hearing loss with damage principally to cochlear hair cells, and primarily to OHCs. (2) a history of a long-term exposure to dangerous noise levels (i.e., >85 dB for 8 hours/day) sufficient to cause the hearing loss. (3) a gradual loss of hearing over the first 5 to10 years of exposure; (4) a hearing loss involving initially the higher frequencies from 3 to 6 kHz before including low frequencies; (5) Speech-Recognition Scores that are

consistent with the audiometric loss; and (6) a hearing loss that stabilizes once the noise exposure is terminated. A patient with NIHL commonly consults a physician because of difficulties in hearing and understanding ordinary speech, especially in the presence of background noise. The beginning region of impairment involves the sensitive mid-frequency range, primarily between 3 and 6 kHz, and the corresponding impairment is classically described as the 4-kHz notch. This particular pattern of maximal hearing loss, with little or no loss below 2 kHz. .Noise Induced Hearing Loss is usually symmetrical for both ears, particularly for individuals who have been working in noisy environment. Other forms of noxious sound, is the gunfire associated with sports shooting, which causes an asymmetric pattern of hearing loss. In this case, the ear pointed towards the source of noise (gun barrel), for example, the right ear (open circles) of this left-handed shooter, would have worse hearing than the ear directed away from the source the left or protected ear by 15to 30 dB or more and particularly at higher frequencies due to the absence of the protective head-shadow effect. Beginning stages of noise-induced hearing loss produce symmetrical pattern of "4-kHz notch" in the audiogram.

PATHOLOGY OF NIHL

The primary site of anatomical damage is at the level of mechanosensory receptors of the auditory system’s end organ. That is the loud sound damages the outer hair

cells of the organ of corti initially.Prlonged exposure or very intense acoustic stimulation can damage the inner hair cells, supporting cell elements and nerve fibres also. Noise can cause damage to hair cells ranging from total destruction to effects evident only in ultrastructure of the specialized subcellular regions.NIHL depends on the intensity, frequency and duration of noise exposure and individual susceptibility.Prlonged and continuous exposure of the noise over years can lead to total destruction of the sensory and neural elements in the basal turn of cochlea, resulting in an abrupt loss of mid and high frequency hearing. Almost symmetric pattern of degeneration observed in both ears. Recovery from TTS is thought to imply a role for metabolic mechanism, the persistence of PTS that for structural change mechanism[16].

METABOLIC MECHANISMS

Acoustic over stimulation could potentially lead to the excessive release of excitatory neurotransmitter glutamate, which is associated with the transduction function of the cochlea leads to metabolic exhaustion of the activated cell. Change in the cochlear blood flow associated with acoustic stimulation, sound of moderate intensity increases cochlear blood flow, high intensity decreases the cochlear blood flow, this being the potential mechanism for cochlear dysfunction. NIHL and cochlear hypoxia due to vascular narrowing precede changes in cochlear blood flow. Mechanical injury caused by severe motion of basilar membrane. Ionic

poisoning from the interruption of the normal chemical gradiants of the cochlea. Other possible mechanisms are OHC plasma membrane fluidity, the role of glucocorticoid receptors, oxidative stress and free radicals. NIHL pathology is a multifactorial and complex situation

STRUCTURAL MECHANISMS

Changes to micromechanical structures within the cochlea have been reported as possible mechanisms of NIHL.Depolymerization of the actin filaments in stereocilia may be a substrate of TTS.Changes to neurosensory elements of cochlea such as swelling of the stria vascularis, afferent nerve endings and supporting cells have been noted. Alteration in the stereocilia in the form of shortened or brokened rootlets are involved in the initial process that leads to TTS and PTS[10]. The progression of OHCs death due to apoptotic changes like nuclear condensation and cell shrinkage have been detected five minutes after exposure to impulse noise, necrotic changes like nuclear swelling appeared 30mins following exposure. A family of cysteine dependent aspartate specific proteases involved in cochlear hair cell apoptosis

The metabolic and structural changes in the organ of corti following noise exposure are indicative of necrotic mechanism being involved[13]. High frequency hair cells die rapidly after noise injury, but low frequency hair cells may survive without auditory function. Well accepted origin of the 4 kHz notch in the

NIHL is related to the resonator function of the external auditory canal rather than to indeterminable innate properties of the cochlea. The hair bundles are capable of rebuilding their ultrastructure from top to bottom over a 48 hours period, however if the damage is so severe that it overwhelm this self-repair mechanisms, as exposure continues a discrete but direct mechanical disruption probably results in a toxic mixing of endolymph and perilymph through micro breaks in the structural framework of the cochlear duct which leads to secondary effects, including loss of hair cells and their corresponding nerve fibres

PREDISPOSING FACTORS OF NIHL

A potential genetic basis for susceptibility to NIHL has been continued, ahl gene[18]

1) Age- the effect of ageing upon threshold altered NIHL is showed but for adjacent frequencies is accelerated.

2) Smoking[14]

3) Diabetes

4) Cardiovascular disease

5) Recreational drug users

6) Exposure of ototoxic agent and noise are synergistic

7) Individual variability in human susceptibility to NIHL.

CLINICAL FEATURES

Usually males, in early middle age. Younger patients complaint most about tinnitus with or without hyperacusis than deafness. Acoustic shock is seen more frequently in women.

In the early stage patient gives H/o. hearing difficulties in the presence of backround noise, the description involves a lack of clarity rather than a volume.

Tinnitus is a common accompanying symptom of NIHL. Occur early in the course of the condition.

Post Exposure tinnitus is a useful symptom when making the diagnosis.

- Hyperacusis
- Obvious hearing loss – Frequently having to ask others to repeat themselves.
- H/o social withdrawal – Often increasing reliance on the spouse for social and family interaction – lead to marital stress.
- Embarrassment, loss of confidence
- Anxiety and
- Frank depression.

COMPLICATIONS

1. Vestibular dysfunction-symmetrically, centrally compensated decrease in the Vestibular End Organ function associated with NIHL[16,21]..

a. Anatomical proximity to acoustic noise exposure

b. Greater similarity of the vestibular and cochlear hair cells

c. Common arterial supply

2. General health impairment -due to activation of the autonomic nervous system and pituitary adrenal axis, because prolonged noise exposure act as a biological stressor, leads to

a.Hypertension

b.Peptic ulcer

c.Emotional instability.

INVESTIGATIONS

The cornerstone of investigation is Pure Tone Audiometry.

Classical audiometric pattern is of a high tone hearing loss 3 and 6 kHz with a notched appearance central on 4 or 6 kHz with some recovery at 8kZ.

Tympanometry – is helpful to confirm normal middle ear function.

BERA – to provide a more objective measure of hearing thresholds, loudness discomfort level- useful measure of the presence of hyperacusis.

OAE – decreased, contralateral OAE with early noise damage.

MRI – to exclude a vestibular schwannoma.

Tinnitograms- Pitch and intensity matching are often performed.

DIAGNOSIS

Unfortunately there is no specific test available to make the diagnosis of NIHL

Individuals with a clear and prolonged history of unprotected exposure to excessive noise with no evidence of any other otological pathology and an audiogram showing good preservation of mid and low frequencies, but a significance high frequency hearing loss with classical notching at 4 to 6 kHz.

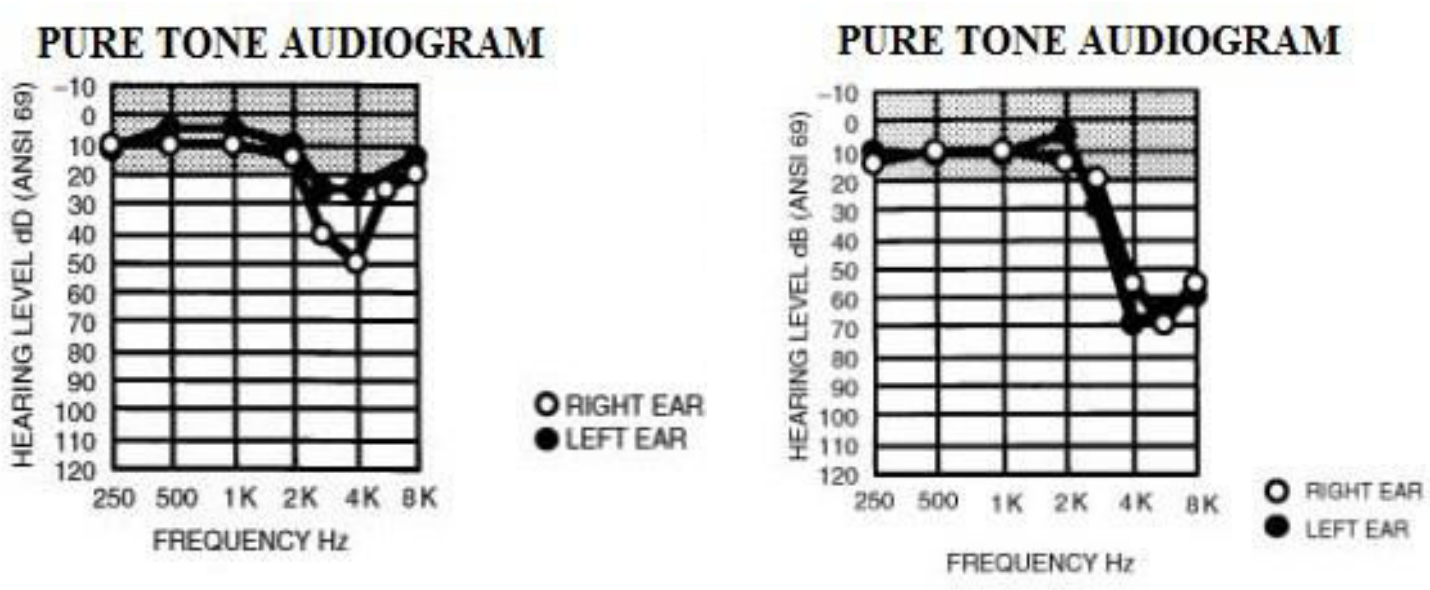
These are additional problems that must be considered when diagnosing the NIHL.

1) Separating the effect of ageing from the effect of noise. Removal of an average value for Age Related Hearing Loss has left an assumed NIHL, according to NIHL tables ISO 7029 and ISO 1999.

2) Remove all other otological pathology such as ear disease, head injuries and positive family history of hearing loss.

3) An individual will have a hearing loss composed of three parts; an age-related component, noise induced component and idiopathic degenerative component, must be considered when diagnosing a NIHL.

1 **2**



1. Pure tone audiogram shows typical 4 kHz dip of noise induced hearing loss.

2. 2nd audiogram shows high frequency noise induced hearing loss.

HISTORY TO R/O

Head injury

Meningitis

Serious systemic illness

Previous aminoglycoside treatment

Strong family H/o. Early hearing loss

There must be a H/o. Noise exposure.

EXMINATION

- Otological examination will be normal.
- Evidence of either active or quiescent otitis media does not preclude the diagnosis.

MANAGEMENT

NIHL is not medically or surgically treatable condition, it is almost entirely a preventable condition

PREVENTIVE MEASURES

Health and safety Act 1974, to minimize risks to employees, including those from excessive noise exposure. The 1989 Noise Act at Work Regulation described two

action level at 85dB (A) and a second action at 90dB (A), peak action level of 140 dB. These have been recently been replaced by the Act of noise at work regulation April 6, 2006, where each action level is 5dB lower respectively.

Employee education and provides appropriate hearing protection, the use of this hearing protection is at the discretion of the employee until the second or peak action level is reached when it becomes compulsory.

Regular hearing test should be offered when a potential risk is recognized.

Reduction of noise at exposure, use of 4 strokes motorcycles instead of 2 strokes.

1. Reduction of noise level at the source of production

2. Specific protection of the individuals who were at risk

a. Ear plugs[1]

Can be assumed to give approximately 10 to 15dB of sound protection.

b. Ear muffs.

Ear muffs at least 15 to 30dB sound protection.

3. Health education-about NIHL and preventive aspects

4. Early detection-perodic audiological checkup. It is also useful to prevent further worsening of the hearing loss.

PHARMACOLOGICAL TREATEMENT

1. Acetyl -L-carnitine - an endogenous mitochondrial membrane compound protect the hair cells from oxidative stress

2. Carbamathione – Glutamate antagonist

3. D-methionine – glutathione repletion drug[12, 20].

4. Adeno viral Math – 1 gene therapy – new hair cell generator – future trend[4'9'22]

REHABLITATION

Patient with moderate to severe hearing loss were benefited by hearing aids, particularly digital hearing aids with amplification of particular frequency is useful.

RESULTS AND ANALYSIS

The following data were obtained from 660 patients and 707 controls, total 1367 at upgraded institute of otorhinolaryngology in Government General Hospital attached to Madras Medical College, Chennai during the period of February 2010 to December 2010.

1) Type of Hearing Loss

2) Bilateral/ Unilateral NIHL

3) Severity of HL

4) Duration of Work

5) Age Distribution

6) Zone Distribution.

7) Sound level exposure.

NIHL

ZONE DISTRIBUTION

1) Type of Hearing Loss

2) Unilateral / Bilateral

3) Severity of Hearing Loss

4) Duration of work

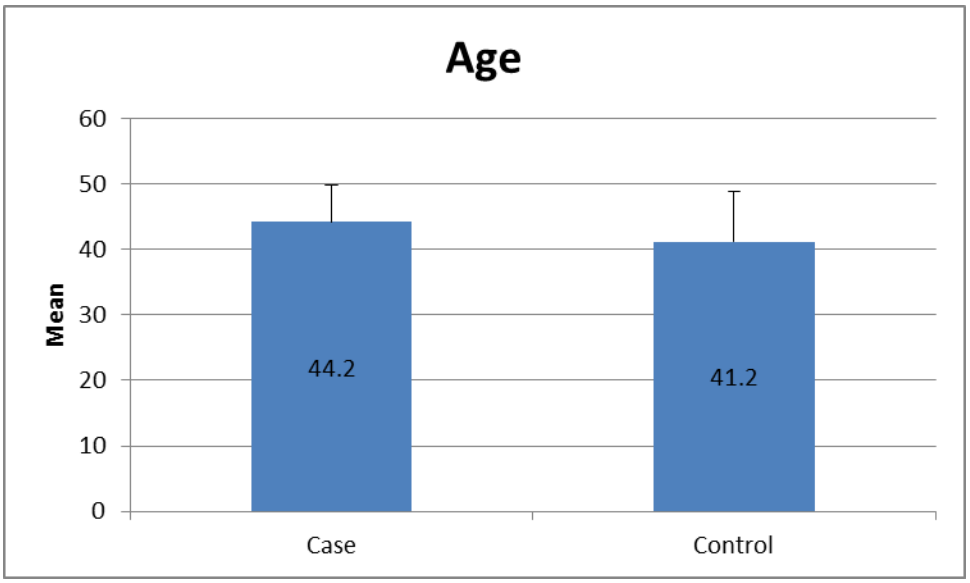
5) Age Distribution.

AGE DISTRIBUTION IN CASE AND CONTROL

	Case			Control			t	df	p value
	N	Mean	S D	N	Mean	S D			
Age	660	44.2	5.65	707	41.2	7.72	8.03	1365	0

	Case	Control
Mean	44.2	41.2
SD	5.65	7.72

This table shows mean age for the case - 44.2, control 41.2, the cases occur in somewhat higher age group, compared to control group, which is significant.

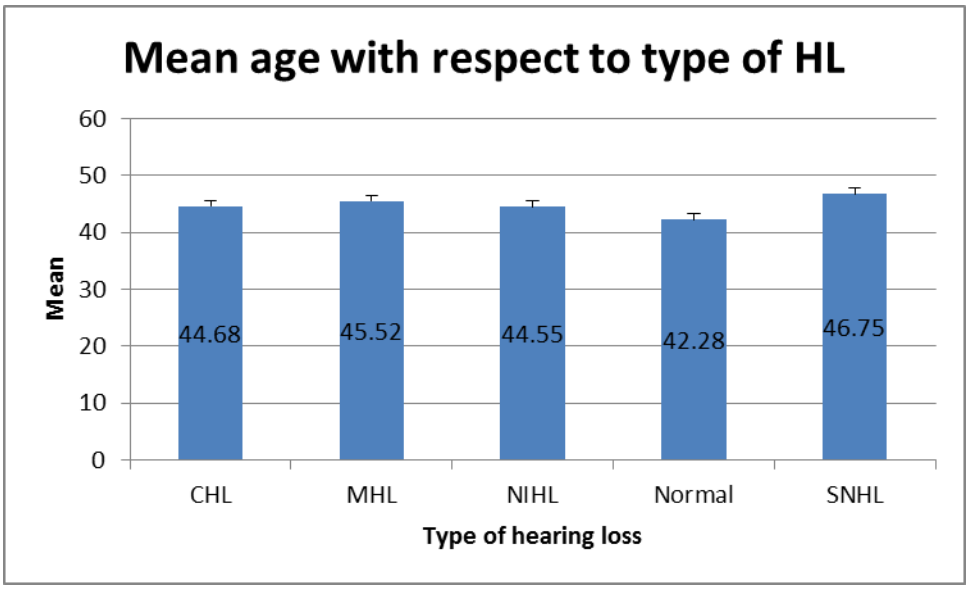


TYPE OF HEARING LOSS

Age	N	Mean	Std. Deviation	Minimum	Maximum
CHL	47	44.68	5.831	30	57
MHL	21	45.52	4.760	36	54
NIHL	364	44.55	5.611	31	57
Normal	172	42.28	5.493	32	53
SNHL	56	46.75	4.889	34	55
Total	660	44.19	5.646	30	57

($p < 0.001$, significant) One way ANOVA

According to the age of NIHL distribution, mean age is 44.55 which is significant, compared to control



BILATERAL / UNILATERAL

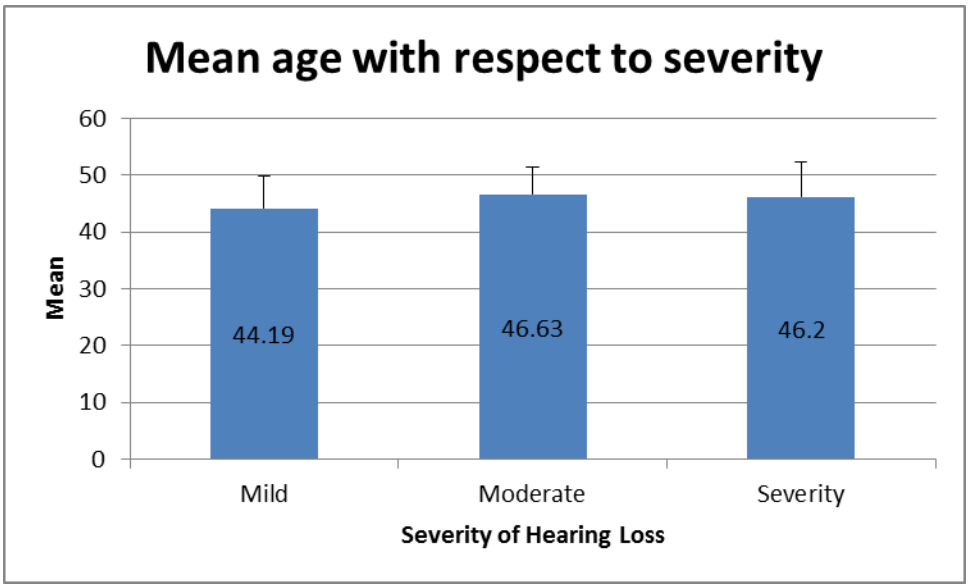
BILATERAL/ UNILATERAL	Control		Case		Total
	Count	%	Count	%	
	707	100	296	44.8	1003
Bilateral	0	0	278	42.1	278
Left Ear	0	0	48	7.3	48
Right Ear	0	0	38	5.8	38
Total	707	100	660	100.0	1367

This table shows bilateral and unilateral cases of NIHL, Bilateral 42.1% and Unilateral 13.1%

SEVERITY OF HEARING LOSS

SEVERITY OF HL	Control		Case		Total
	Count	%	Count	%	
	707	100	296	44.8	1003
Mild	0	0	308	46.7	308
Moderate	0	0	46	7.0	46
Severe	0	0	10	1.5	10
Total	707	100	660	100.0	1367

This table shows severity hearing loss, mild cases 46.7%, moderate cases 7% and severe 1.5 %.



DURATION OF WORK

DURATION OF WORK	Control		Case		Total
	Count	%	Count	%	
	707	100	0	0.0	707
< 1 year	0	0	154	23.3	154
1 - 5 years	0	0	145	22.0	145
5 - 10 years	0	0	148	22.4	148
> 10 years	0	0	213	32.3	213
Total	707	100	660	100.0	1367

This table shows duration of work in relation to the cases, less than 1 year 23.3%, 1-5 years 22%, 5-10 years 22.4% and more than 10 years 32.3%. More than 5 years 54.7% which is significant when compared to less than 5 years.

AGE DISTRIBUTION

Age

	N	Mean	Std. Deviation	Minimum	Maximum
Mild	308	44.19	5.646	31	57
Moderate	46	46.63	4.809	37	57
Severity	10	46.20	6.106	38	55
Total	364	44.55	5.611	31	57

(p = 0.014, significant) One way ANOVA

This table shows zonal wise age relation with the severity of the hearing loss. Mild cases mean age 44.19, moderate 46.63 and severe cases 46.2 which is significant.

ZONE DISTRIBUTION

ZONE	Control		Case		Total
	Count	%	Count	%	
	707	100	0	0.0	707
Central	0	0	311	47.1	311
North	0	0	244	37.0	244
South	0	0	105	15.9	105
Total	707	100	660	100.0	1367

This table shows zonal wise severity of hearing loss. Central zone 47.1%, North zone 37% and South zone 15.9%.

SOUND LEVEL EXPOSURE

SOUND LEVEL dB

	N	Mean	Std. Deviation	Minimum	Maximum
Mild	308	94.96	14.110	70	122
Moderate	46	95.22	13.943	74	122
Severity	10	95.90	11.995	75	110
Total	364	95.02	14.003	70	122

(p = 0.974, not significant) One way ANOVA

This table shows sound level exposure in relation severity of the cases. Mild cases minimum level of noise exposure- 70dB, maximum–122dB. Moderate cases minimum level of noise exposure-74dB, maximum-122dB. Severe cases minimum level of noise exposure-75dB, maximum-110dB.Mean sound level exposure to all NIHL cases-95.02dB.

ZONE WISE AGE AND SOUND LEVEL EXPOSURE

Case Summaries

ZONE		Age	SOUND LEVEL dB
Central	N	171	171
	Mean	44.87	104.11
	Std. Deviation	5.462	10.906
	Minimum	31	74
	Maximum	57	122
North	N	141	141
	Mean	44.90	85.84
	Std. Deviation	5.466	12.428
	Minimum	31	70
	Maximum	57	122
South	N	52	52
	Mean	42.58	90.02
	Std. Deviation	6.162	6.304
	Minimum	31	74
	Maximum	56	100
Total	N	364	364
	Mean	44.55	95.02
	Std. Deviation	5.611	14.003
	Minimum	31	70
	Maximum	57	122

This table shows more number of NIHL cases were distributed central zone. Mean age and sound level of exposure was high in the central zone

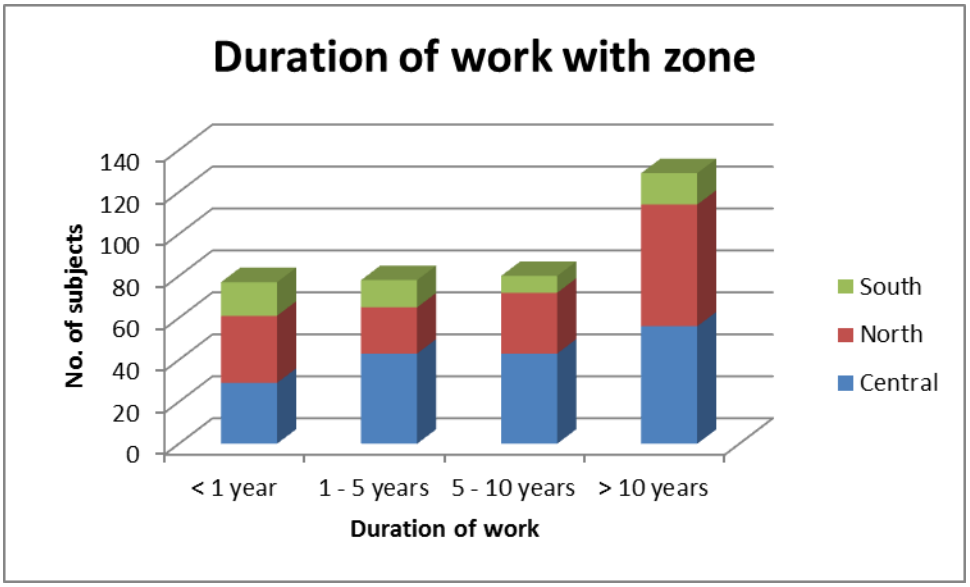
DURATION OF WORK

ZONE	< 1 year	1 - 5 years	5 - 10 years	> 10 years
Central	29	43	43	56
North	32	22	29	58
South	16	13	8	15
Total	77	78	80	129

p = 0.069 (chi square test)

This table shows zonal wise duration of work and cases. Less than 5 years 155 cases.

More than 5 years of noise exposure cause more number of cases 209, which is significant.

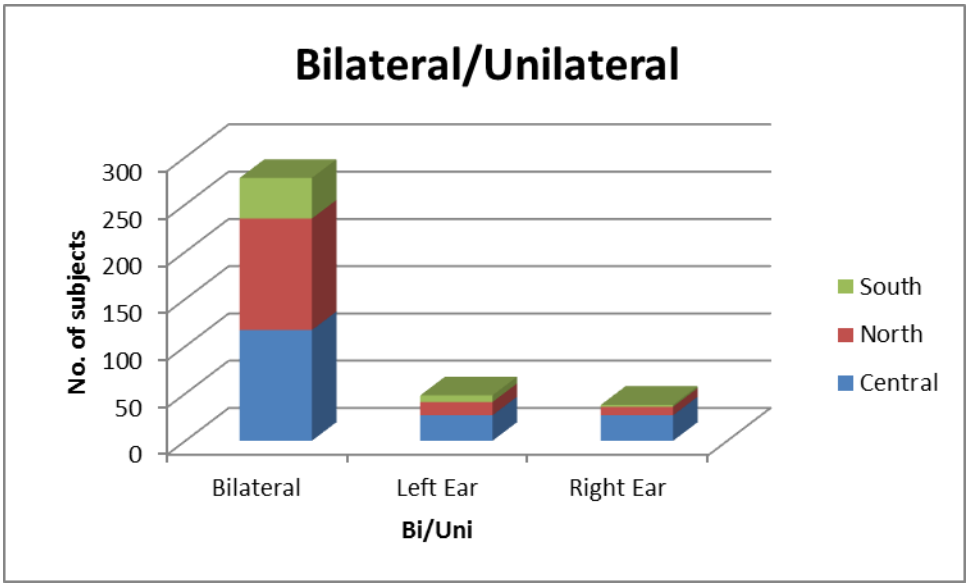


ZONE * BILATERAL/UNILATERAL Cross tabulation

		BILATERAL/UNILATERAL		
	ZONE	Bilateral	Left Ear	Right Ear
	Central	117	27	27
	North	118	14	9
	South	43	7	2
Total		278	48	38

$p < 0.01$ (chi square test)

North and central zone bilateral NIHL cases were equal but unilateral cases are more common in central zone.

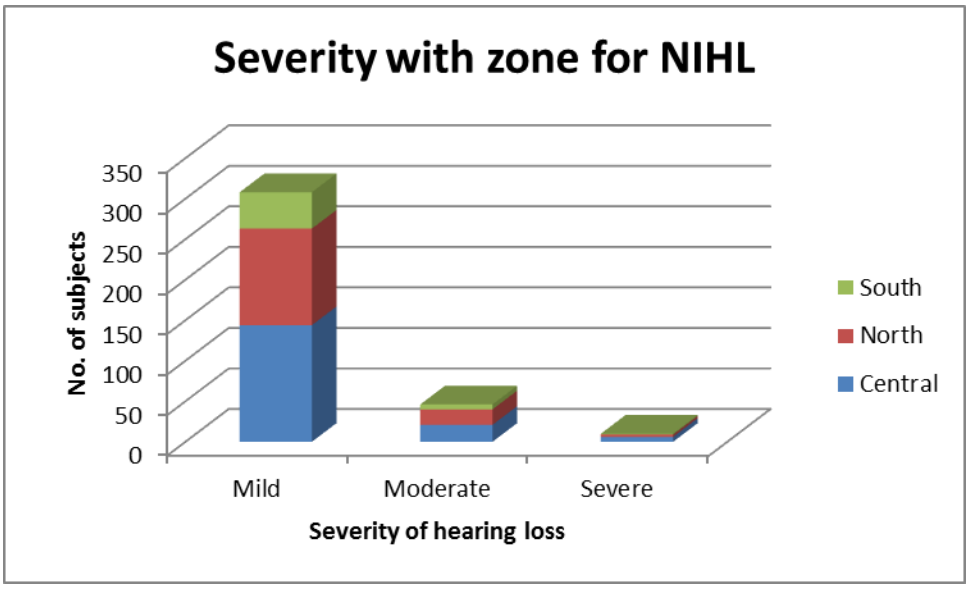


ZONE * SEVERITY OF HL Cross tabulation

		SEVERITY OF HL		
	ZONE	Mild	Moderate	Severe
	Central	144	21	6
	North	119	19	3
	South	45	6	1
Total		308	46	10

p 0.930 (chi square test)

This table shows zonal wise severity hearing loss. Central zone- mild cases-144, moderate-21 and severe-6. North zone –mild cases-119, moderate-19 and severe-3. South zone mild cases-45, moderate-6 and severe-1.



DISCUSSION

1. Michael Hoffer-Australia-2006 ,conducted a study about NIHL in industrial Workers, the incidence rate were 56%.
2. Hwang et al-USA-New York 2007 studied about occupational NIHL among crops spraying farmers, incidence -62%.
3. McBride et al-New Zealand studied about occupational NIHL in farmers (tractor driver) incidence -62%.
4. Marvel et al-crop specialist & extension professor studied about occupational NIHL in Dairy farmers, incidence -65%
5. March 2007, WHO & Washington DC, Centre for disease control and prevention(CDC) after series of studies had concluded NIHL is the most common occupational illness in North America
6. RA Klick, J Royse C & Rain bold studied the incidence of NIHL among music teacher. Study was conducted among 104 subjects, incidence-19%
7. Sri Harmadji & Heri Kabullah, NIHL among steel factory workers , 50 subjects from Sidarjo East Java during Jan to June 2004, incidence -84%
8. De – Beers 2002 studied the incidence of NIHL among mine workers, 223 subjects, and Incidence-40%.

9. Susan L Phillips, Vincent C Henrich studied the prevalence of NJHL in student musicians of 15 to 25 years. Sample size-329, the incidence rate was 45%.

10. Sharif A, Taous A, Siddique BH and Dutta PG-Prevalence of NIHL in Dhaka Metropolitan city traffic police, study period Jan 2003 to Dec 2004.Study place-Department of Otorhinolaryngology and Head &Neck surgery, Mymensingh Medical College,Bangaladesh.Incidence rate was 24%, mild to moderate SNHL in relation to the duration of exposure.

11. Another prospective study was conducted in Abbasi Shaheed Hospital, Karachi; about 200 selected subjects were studied, among 74 traffic constables as cases, 126 shopkeepers as controls. Age group 15 to 80 years. Incidence of NIHL was 33.81.

In our prospective study our aim is to find out the incidence of Noise Induced Hearing Loss by pure tone audiometry among traffic police personnel working as traffic regulators. The study was conducted in our Upgraded Institute of Otorhinolaryngology, Government General Hospital, Madras Medical College from the period of February 2010 to December 2010. Two groups of subjects were studied. Traffic police personnel were case, about 660 in number,

Peoples other than traffic police personnel considered as control with normal pure tone audiometry finding, about 707 in number.

Cases – 660

Control – 707

Total – 1367

TYPE OF HEARING LOSS

Among 660 cases

Normal hearing level	172
Noise induced hearing loss	364
Sensory neural hearing loss	56
Mixed hearing loss	21
Conductive hearing loss	47

SEVERITY OF HEARING LOSS

Mild - 26 to 40dB loss

Moderate – 41 to 55 dB

Severe – 56 dB and above

In bilateral hearing loss with in equal affection of both ears, the severity was calculated by using the formula.

Rt ear – (hearing loss- 25) x 1.5%

Lt ear – (hearing loss – 25) x 1.5%

Percentage of Hearing loss.

$$\frac{(\text{Better ear \% x 5}) + \text{Worse ear\%}}{6}$$

Among the 364 NIHL

Mild NIHL – 308

Moderate NIHL – 46

Severe NIHL-10

BILATERAL / UNILATERAL

NIHL commonly produce bilateral symmetrical hearing loss, the exposure of the noise to both ears were varying according to the traffic, to the side of ear exposed. Here we find outs, cases of unilateral as well varying degree of hearing loss among both ears

Bilateral – 278

Unilateral – 86

Rt Ear – 38

Lt Ear – 48

Bilateral hearing loss was common. Among the unilateral cases the left ear was affected somewhat more when compared to the Rt ear.

DURATION OF WORK

Here we divide the working period into 4 categories

- Less than 1 year
- 1 to 5 years
- 5 to 10 years and
- More than 10 years

According to the duration of noise exposure more number of cases were found, when the duration of the noise exposure was more than 10 years. It was significantly more where the duration of work more than 5 years when exposed to less than 5 years.

AGE DISTRIBUTION

The age of cases were distributed between 30 to 57 years. The mean age group affected by NIHL 44.55.

ZONE DISTRIBUTION

Three zones were divided, North zone, Central zone and South zone by two roads Poonemalle High Road and Annasalai.

North zone – 141

Central Zone – 171

South Zone – 56

More number of cases was distributed in the Central zone. Because the Sound level of exposure was high 104.11dB when compared to other zones. The next zone affected was North zone the last one affected was South zone.

SOUND LEVEL EXPOSURE (dB)

The severity& no. of cases of NIHL depends on the intensity of the sound, higher the intensity leads to more hearing loss. The minimum level of sound exposure is 70dB; maximum level 122 dB Sound level was measured by the sound level meter. It was measured in SPL (A)

Minimum Level – 70 dB

Maximum – 122 dB

Mean – 95.02 dB.

BENEFITS TO THE COMMUNITY:

1. Early diagnosis.
2. Better patient care.
3. Awareness to traffic police personnel about noise exposure and hearing loss.

Highlights the importance of wearing the protective devices during the working hours to prevent Noise Induced Hearing Loss.

CONCLUSION

All the patients were underwent Pure Tone Audiogram. The following observations were made.

1) The incidence of Noise Induced Hearing Loss was 55.2%.

2) The most common age group affected was more than 40 years, mean age group was 44.55 years.

3) The most of the NIHL cases were mild type- 46.7%,moderate-7%&severe-1.5%

4) Bilateral NIHL was common when compared to the unilateral hearing loss. Bilateral-42.1% and unilateral-13.1%.

5) There was considerable relationship between the duration of noise exposure and hearing loss, more than 5 years of work cause significant hearing loss-54.7%.

6) When intensity of noise exposure was high, the hearing loss is early and more. Mean level of noise exposure was 95.02dB.

CONSENT FORM

I _____ hereby give consent to participate in the study conducted by **Dr. C.KARUPPASAMY,** post graduate in Upgraded institute of otorhinolaryngology, Madras Medical College & Government General Hospital, Chennai and to use my personal clinical data and result of investigation for the purpose of analysis and to study the nature of disease.

I also give the consent for further investigation.

Signature of participant

PROFORMA

INCIDENCE OF NOISE INDUCED HEARING LOSS IN METROPOLITAN TRAFFIC POLICE

Name:

Age:

Sex:

OP No:

Occupation:

Working Place:

Duration of Work:

Hours of work per day:

Any hearing protection device used during work:

Average sound pressure level in that area:

H/o. Ear discharge:

H/o. Ear Pain:

H/o. Ear Block:

H/o. Tinnitus/ Vertigo:

H/o. Trauma:

Family H/o. Hearing Loss:

ENT EXAMINATION

Ear:

Nose:

Throat:

TUNING FOR TEST

	Right	Left
Rinne:		
Weber:		
ABC:		

PURE TONE AUDIOMETRY:-

At the time of recruitment:

Present Level:

Pure Tone Audiometry

Normal	Abnormal
↓	↓
CHL	SNHL
↓	↓
Impedance Audiometry	Special Audiological Test Speech Audiometry

BIBLIOGRAPHY

1. Abel SM, Rockley T, Goldfarb D: Outer ear canal shape and its relation to the effectiveness of sound attenuating earplugs, *J Otolaryngol* 19:91–95, 1990.

2. Ahmad M, Bohne BA, Harding GW: An in vivo tracer study of noise-induced damage to the reticular lamina, *Hear Res* 175:82–100, 2003.

3. Bermingham-McDonogh, Rubel EW: Hair cell regeneration: winging our way towards a sound future, *Curr Opin Neurobiol* 13:119–126, 200.

5. Bies DA, Hansen CH: An alternative mathematical description of the relationship between noise exposure and hearing loss, *J Acoust Soc Am* 88:2743–2754, 1990.

6. Bredberg G: Cellular pattern and nerve supply of the human organ of Corti, *Acta Otolaryngol Suppl* 236:1–135, 1968.

7. Brownell WE: Outer hair cell electromotility and otoacoustic emissions, *Ear Hear* 11:82–92, 1990.

8. Cantrell RW: Physiologic effects of noise, *Otolaryngol Clin North Am* 12:537–549, 1979.

9. Kawamoto K and others: Math1 gene transfer generates new cochlear hair cells in mature guinea pigs in vivo, *J Neurosci* 23:4395–4340, 2003.

10. Lieberman MC: Chronic ultrastructural changes in acoustic trauma: serial-section reconstruction of stereocilia and cuticular plates, *Hear Res* 26:65–88, 1987.

11. Lusk SL: Preventing noise-induced hearing loss, *Nurs Clin North Am* 37:257–262, 2002.

12. McFadden SL and others: The influence of superoxide dismutase and glutathione peroxidase deficiencies on noise-induced hearing loss in mice, *Noise Health* 3:49–64, 2001.

13. McGill TJI, Schuknecht HF: Human cochlear changes in noise-induced hearing loss, *Laryngoscope* 86:1293–1302, 1976.

14. Mizoue T, Miyamoto T, and Shimizu T: Combined effect of smoking and occupational exposure to noise on hearing loss in steel factory workers, *Occup Environ Med* 60:56–69, 2003.

15. Nordmann AS, Bohne BA, Harding GW: Histopathological differences between temporary and permanent threshold shift, *Hear Res* 139:13–30, 2000.

16. Schuknecht HF: *Pathology of the ear,* Cambridge, Massachusetts, 1974, Harvard University Press.

17. Shupak A and others: Vestibular findings associated with chronic noise induced hearing impairment, *Acta Otolaryngol* 114:579–585, 1994.

18. Toppila E and others: Individual risk factors in the development of noise-induced hearing loss, *Noise Health* 2:59–70, 2000.

19. Vavrina J, Muller W: Therapeutic effect of hyperbaric oxygenation in acute acoustic trauma, *Rev Laryngol Otol Rhinol (Bord)* 116:377–380, 1995.

20. Yamasoba T and others: Role of glutathione in protection against noise-induced hearing loss, *Brain Res* 784:82–90, 1998.

21. Ylikoski J and others: Subclinical vestibular pathology in patients with noise-induced hearing loss from intense impulse noise, *Acta Otolaryngol* 105:558–563, 1988.

22. Zheng JL, Gao WQ: Overexpression of Math 1 induces robust production of extra hair cells in postnatal rat inner ears, *Nat Neurosci* 3:480–586, 2000.

23. Scott-Brown's Otorhinolaryngology, Head and Neck Surgery-7th edition volume-3, Part 19, Chapters-226,229&238b.

24. Scott-Brown's Otorhinolaryngology, Head and Neck Surgery-6th edition Volume-3/4/3&3/4/28, Volume-2/11/1-28.

25.Mawson's Diseases of the Ear-5th edition,Chapter-2 Page 45,Chapter 3 Page74& Part 5, Chapter 24 Page-593.

26. Mawson's Diseases of the Ear-6th edition, Part II Chapter-8, Page 34-41, Part VI Chapter 35 Page-483 to 94.

27. Glasscock-Shambaugh SURGERY 0f the EAR, Fifth Edition, Part II Chapter 3, Page 66& Part II Chapter 7, Page 153.

28. OTOLARYNCOLOGY, HEAD&NECK SURGERY-CUMMINGS Volume 4, Chapter 128, Page 2908 to2923.

29. OTOLARYNCOLOGY, HEAD&NECK SURGERY-CUMMINGS Volume 4, Chapter 149&150, Page 3373 to3452

30. Logan Turner's Diseases of the NOSE THROAT AND EAR-Chapter 3.14, Page 350 to 352.

ABBREVIATION

NIHL	-NOISE INDUCED HEARING LOSS
TTS	-TEMPORARY THRESHOLD SHIFT
PTS	-PERMANENT THRESHOLD SHIFT
CN	-COCHLEAR NUCLEI
AVCN	-ANTERO VENTRAL COCHLEAR NUCLEI
PVCN	-POSTERO VENTRAL COCHLEAR NUCLEI
SOC	-SUPERIOR OLIVARY COMPLEX
PTA	-PURE TONE AUDOIMETRY
SPL	-SOUND PRESSURE LEVEL
CHL	-CONDUCTIVE HEARING LOSS
MHL	-MIXED HEARING LOSS
SNHL	-SENSORINEURAL HEARING LOSS
IHC	-INNER HAIR CELL
OHC	-OUTER HAIR CELL

INSTITUTIONAL ETHICAL COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI -3
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CERTIFICATE OF APPROVAL

To
Dr. C. Karuppasamy
PG in MS ENT
Madras Medical College, Chennai -3

Dear Dr. C. Karuppasamy

The Institutional Ethical Committee of Madras Medical College reviewed and discussed your application for approval of the project / proposal / clinical trail entitled **"Incidence of noise induced hearing loss among metropolitan city traffic police personnel"** No 18072010

The following members of Ethical committee were present in the meeting held on 21.07.2010 conducted at Madras Medical College,

1.	Prof. S.K. Rajan, MD	-- Chairperson
2.	Prof. J. Mohanasundaram, MD,Ph.D,DNB Dean, Madras Medical College, Chennai -3	-- Deputy Chairman
3.	Prof. A. Sundaram, MD Vice Principal , MMC, Chennai -3	-- Member Secretary
4.	Prof. R. Sathianathan, MD Director, Institute of Psychiatry	-- Member
5.	Prof R. Nandhini, MD Director, Institute of Pharmacology, MMC, Ch-3	-- Member
6.	Prof. Pregna B. Dolia , MD Director, Institute of Biochemistry, MMC, Ch-3	-- Member
7.	Prof. C. Rajendran , MD Director, Institute of Internal Medicine, MMC, Ch-3	-- Member -- Member
8.	Prof. Geetha Subramanian, MD,DM Professor & Head , Dept. Of Cardiology	-- Member
9.	Prof. V. Shruti Kamal, MS Professor of Surgery, MMC, Ch-3	-- Member
10.	Prof. Md. Ali, MD, DM Professor & Head ,,Dept. of MGE, MMC, Ch-3	-- Member -- Member

We approve the trail to be conducted in its presented form.

Sd/. Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, any SAE occurring in the course of the study, any changes in the protocol and patient information / informed consent and asks to be provided a copy of the final report

Member Secretary, Ethics Committee.