A Dissertation On

"A STUDY OF PREVALENCE OF NOISE INDUCED HEARING LOSS IN AUTO DRIVERS."

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For the Award of the degree

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CERTIFICATE

This is to certify that the dissertation titled **"A STUDY OF PREVALENCE OF NOISE INDUCED HEARING LOSS IN AUTO DRIVERS"** submitted by **Dr. SABARISH.S.,** appearing for M.S. (Otorhinolaryngology) Branch IV degree examination in April 2020, is a bonafide record of work done by him under my guidance and supervision in partial fulfillment of requirements of The Tamilnadu Dr. M.G.R Medical University, Chennai. I forward this to The Tamilnadu Dr. M.G.R Medical University, Chennai.

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DECLARATION

I, **Dr. SABARISH. S,** certainly declare that this dissertation titled, **"A STUDY OF PREVALENCE OF NOISE INDUCED HEARING LOSS IN AUTO DRIVERS"**, represent a genuine work of mine done at the Department of Otorhinolaryngology, Stanley Medical College, under the supervision of the **Prof.V.RAJARAJAN, M.S., DNB., Professor, Department of Otorhinolaryngology, Stanley Medical College, Chennai – 600 001.**

I, also affirm that this bonafide work or part of this work was not submitted by me or any others for any award, degree or diploma to any other university board, neither in India or abroad. This is submitted to The Tamil Nadu Dr.MGR Medical University, Chennai in partial fulfilment of the rules and regulation for the award of Master of Otorhinolaryngology Branch IV .

Dr.SABARISH. S

Date :

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CONTENTS

INTRODUCTION

Noise is one of the occupational health disorders affecting workers in various professions. Traffic noise is a one of the source of environmental pollution in developed and developing nations. Recent reports suggest that, outside Europe and North America, up to 25% of adult males of working age have evidence of occupational noise induced hearing loss. This seems to be a particular problem in those countries moving from agriculture to a stronger manufacturing base for their developing economies .noise induced hearing loss refers to exposure of loud sounds beyond 85db over a period of time. Sounds of 130 dB (A) or greater will cause hearing damage after even short time periods in almost all exposed individuals. noise induced hearing loss is preventable through early intervention. There is evidence that the use of hearing protection reduces the risk of noise induced hearing loss from noise in both recreational and occupational contexts. As regards hearing protection, the choice is between earplugs, earmuffs earplugs can be assumed to give approximately 10–15 dB of sound

attenuation and earmuffs at least 15 dB and active noise reduction early detection of noise induced hearing loss can be made by use of pure tone audiometry

WHO GRADING OF HEARING IMPAIRMENT

Grades of impairment	**Audiometric ISO values(average 500,1000,2000,4000Hz)**	**Impairment description**
0(no impairment)	UP TO 25dBHL	No or very slight hearing problems. Able to hear whispers
1(slight impairment)	26-40dBHL	Able to hear and repeat words spoken in normal voice at 1 metre
2(moderate impairment)	41-60dBHL	Able to hear and repeat words using raised voice at 1 metre
3(severe impairment)	61-80dBHL	Able to hear some words when shouted into better ear
4(profound impairment)	81 dBHL and above	Unable to hear and understand even shouted voice

Audiometric ISO(average of 500,1000,2000Hz)	Grade of Impairment
0 to 25 dB	Normal hearing level
26 to 40dB	Mild deafness
41 to 55dB	Moderate deafness
56 to 70dB	Severe deafness
71 to 90 dB	Very severe deafness
Above 90dB	Profound deafness

AIM AND OBJECTIVES

The main objective of the study is to assess the prevalence pattern of NIHL and its relation with duration of exposure to noise in auto drivers of various parts of Chennai city

REVIEW OF LITERATURE

COCHLEA:

The **cochlea** is formed of three parallel canals , coiled in a spiral around a central 'stalk', the **modiolus** . The axons of the central projections of the auditory nerves that innervate the sensory epithelia, and the vessels of the cochlear blood supply, the cochlear artery and cochlear vein, run through the length of the modiolus. There are 2.5 turns in the human cochlea.

The number of turns varies between different mammalian species but at present this is not known to have any specific functional significance. The central canal, the **scala media**, is lined by epithelia (part of the membranous labyrinth) and is filled with endolymph. In cross sections of the cochlea, the scala media is bounded by three 'walls' and appears approximately triangular in shape The low frequency fibers occupy the centre and high frequency fibers occupy the periphery of the nerve.

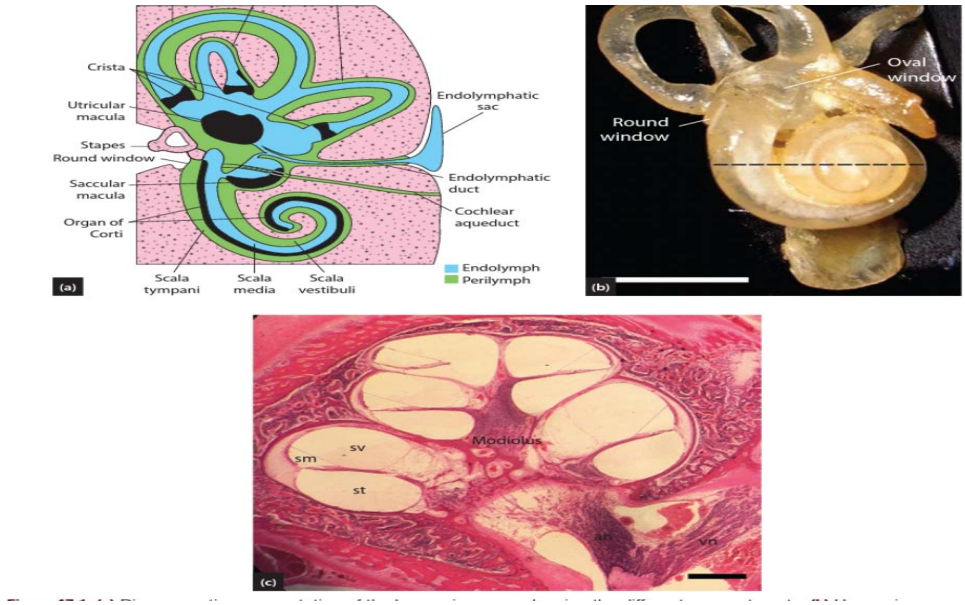
The sensory epithelium, the **organ of Corti,** running along the **basilar membrane**, forms the 'floor' of the triangle. The primary ion-transporting epithelium, the **stria vascularis**, runs along the lateral side and **Reissner's membrane** forms the 'roof' of the scala media . Above

Reissner's membrane is the **scala vestibuli**, and underneath the basilar membrane is the **scala tympani**.

These two scalae are filled with perilymph. Reissner's membrane acts as the barrier between endolymph and perilymph in the scala vestibuli. Perilymph from both the scala vestibuli and scala tympani is freely permeable into the intercellular spaces of the spiral ligament that underlies the stria vascularis but, unlike the vestibular system, there is a barrier to direct diffusion of ions from the spiral ligament into the ion-transporting epithelium.

The height and width of all the three scalae decrease systematically from base to apex of the spiral At the basal end, the scala tympani terminates at the **round window** , a flexible membrane formed of two epithelial sheets sandwiching connective tissue, containing collagen and blood vessels. The apical surface of the outer epithelium is exposed to air in the middle ear; that of the inner epithelium is bathed in perilymph. The scala vestibuli at its basal end is continuous with the vestibule and the perilymphatic compartment of the vestibular system. . The **oval window**, opening over the vestibule, is covered by a membrane and is filled with the footplate of the stapes. The gap between the border of the stapes footplate and the edge of the oval window is sealed with a ligament. At the apical end of the cochlea, the scala media is closed by epithelial tissue,

arising partly by extension of Reissner's membrane, leaving a small opening, the helicotrema, through which the scala vestibuli and scala tympani are connected.



Sound-induced movements of the tympanic membrane drive piston-like 'in–out' movements of the stapes footplate displacing incompressible perilymph along the scala vestibuli, through the **helicotrema** and down the scala tympani leading to 'out–in' movements of the round window.

As fluid is displaced, the pressure difference across the scala media between the scala vestibule and scala tympani, produces vibrational movement of the basilar membrane, described by Von Bekesy. This

‘travelling wave’ stimulates the sensory cells housed in the organ of Corti that sits on the vibrating basilar membrane

ORGAN OF CORTI:

The organ of Corti is a ridge of cells resting on the basilar membrane and overlain by the tectorial membrane . The length of the coiled basilar membrane and attendant organ of Corti varies with species; in humans it is about 35 mm long (range, 28–40 mm), ~12 mm in mice, ~20 mm in guinea pigs and ~40 mm in whales. The widths of the basilar membrane and the organ of Corti increase systematically from the base to the apex of the cochlea. The thickness of the basilar membraneand the height (and mass) of the organ of Corti also both increase systematically from base to apex. The consequent changes in the inherent mechanical properties of the basilar membrane, combined with changes in the mass on the membrane, result in sounds of different frequencies producing maximum vibrations at different locations along the cochlea; high frequencies are detected at the basal end and low frequencies at the apex. This frequency-place, or ‘**tonotopic**’ relationship is preserved along the neural pathways in the brain: nerves that innervate the hair cells at the high-frequency, basal end of the cochlea project to a specific place in the cochlear nucleus, and those that innervate hair cells in the apical low-frequency region project to a different but specific place in the cochlear nucleus, i.e. there is a tonotopic map projected onto the cochlear nucleus

and this tonotopicity is carried on up the auditory pathway. There are two hair cell types in the organ of Corti, the **inner** and **outer hair cells** (IHCs and OHCs) . In most mammals these are regularly arranged into a single row of IHCs on the medial or inner side of the spiral and three, sometimes four, rows of OHCs on the lateral or outer side. The human organ of Corti, however, appears less well ordered than the cochleae of lower mammals, with regions containing two ranks of IHC and areas where the rows of OHCs are less clearly definable, less evenly spaced and with occasional discontinuities With in the body of the organ of Corti are large extracellular spaces . the **spaces of Nuel** around the OHCs, and the **tunnel of Corti** between the OHC region. Both of these spaces are created during late stages of developmental maturation of the organ of Corti as a result of morphological specializations of the supporting cells, during which the phalangeal processes between the cell body region and expanded head at the apical (lumenal) end become reduced in width. The spaces are filled with perilymph as the basilar membrane is not a permeability barrier.

The actual border and permeability barrier between the perilymph of the scala tympani and endolymph in the scala media is created by theintercellularjunctions at the lumenal side of the organ of Corti and lies at the level of the reticular lamina. Mutations in the proteins that comprise the tight (occluding) junctions in the organ of Corti, result in hearing

impairment, emphasizing the essential nature of the junctions and the barrier that they form.

BASILAR MEMBRANE:

The basilar membrane (BM) is a sheet formed predominantly of extracellular matrix It is composed of filaments within a ground substance, with a discontinuous layer of thin, elongated tympanic border cells on the underside facing the perilymph of the scala tympani.The fibrils of the BM run predominantly radially, and are composed of collagen, mostly collagen type IV α1–α5 chains (COL4A1–COL4A5).78 In addition, fibronectin79 and laminin type 11,80 adhesive-type molecules common to extracellular matrices, are localized to the BM and presumably compose the ground substance in which the collagen fibrils reside. The composition of the BM does not appear to be unique in comparison with basement membranes elsewhere in the body, except for a novel extracellular matrix protein (named 'usherin') that has been identified through the genetic mutation which is associated with Usher syndrome type 2A, in which there is high-frequency hearing loss. Mutations in the genes for the proteins composing the BM might be expected to affect the mechanics of the organ of Corti in response to sound and thereby cause hearing impairment. X-linked Alport syndrome has been attributed to mutations in the *COL4A5* gene. It has been suggested that it is the loss of this

protein from the BM that results in the high frequency hearing loss associated with this condition

TECTORIAL MEMBRANE :

The tectorial membrane is the structured sheet of extracellular matrix material that overlies the organ of Corti. At its inner edge it is attached to the inter dental cells of the spiral limbus, a bony prominence to the inside of the organ of Corti .The longest stereocilia of each OHC are embedded in the underside of the TM .The TM is not merely a fibrous mass but it is quite highly structured, with a defined shape .Over the top surface densely packed fibres are arranged in a network, the 'covernet' .The outermost tip is also distinguished by a higher density of fibre packing . On the underside – facing the apical surface of the organ of Corti – there is a thickened ridge known as Hensen's stripe located just lateral to the position of the IHC stereocilia which contribute to the fluid-coupled deflection of the IHC stereocilia.

The body of the TM is formed of fibre bundles running approximately radially, embedded within a matrix composed of striated sheets formed of fine cross-linked fibrils. The fibre bundles are formed of collagen types II, V and IX different types to those found in the BM. Associated with the collagen bundles are the glycoproteins otogelin and α- and β-tectorin and ceacam . Otogelin, α- tectorin and β-tectorin are

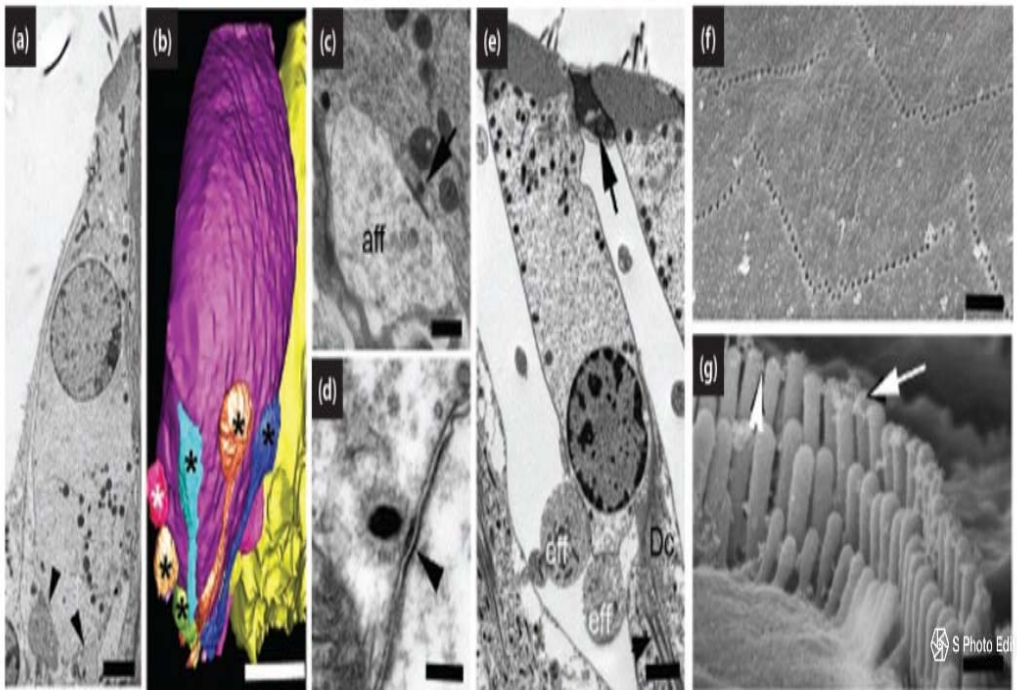
unique and essential to the inner ear, since mutations in the genes that encode for them are associated with non-syndromic hearing loss.

Expression of mRNA for otogelin and the tectorins is detected only during development of the cochlea. The cessation of mRNA production indicates that there is no turnover of these key proteins, suggesting that the TM is a lifelong structure produced only during cochlear development. The implication is that damage to the TM would not be repaired and would result in permanent hearing loss.

INNER HAIR CELLS :

The IHCs reside above a thin and inflexible bony extension from the bone that surrounds the modiolus .They are roughly flask-shaped and the shape of their hair bundles approximates to a straight line or a shallow 'U'-shape so they appear to form an almost continuous 'fence' on inner

aspect of the organ of Corti . IHCs are innervated by afferent nerve fibres and 95% of all the afferent fibres from the cochlea to the brain arise



from IHCs making them the principal receptor cells that send auditory information to the brain. Efferent nerve fibres that terminate in the IHC region arise from the ipsilateral lateral superior olive in the mid-brain. The efferent fibres contact the afferent fibres that terminate on the IHCs. These lateral olivocochlear efferent nerve fibres constitute approximately 20% of the efferent innervation to the organ of Corti. Each IHC forms synapses with several different afferent nerve endings that

surround its basolateral membrane but a single auditory nerve fibre-ANF innervates only a single IHC. The synapses between an IHC and its innervating afferent nerve endings are specialized 'ribbon' synapses .

On the pre-synaptic, IHC side of the synapse adjacent to the membrane there is an elongate or rounded structure, the ribbon, surrounded by tethered secretory vesicles containing the neurotransmitter (glutamate), which release the glutamate into the synaptic gap or cleft when the hair cell is stimulated. The neurotransmitter binds to receptors on the post-synaptic membrane to initiate action potentials in the synapsing ANF. The 'ribbon' specialization provides for a constant pool of neurotransmitter at the site of neurotransmission to allow rapid and sustained release of glutamate and so rapid and sustained firing of the ANFs necessary for audition.

At least two subpopulations have been identified: afferent fibres with low spontaneous firing rates(i.e. the numbers of action potentials fired per second in quiet conditions) and high thresholds (i.e. requiring a relatively high sound intensity input to elicit a change in that firing rate); and fibres with high spontaneous rate and low thresholds. This diversity in the firing characteristics of the nerves innervating a single IHC provides the dynamic range of a single IHC to accurately encode a wide range of sound intensities

OUTER HAIR CELLS :

OHCs are located across the most flexible part of the BM . They are cylindrical cells with a basally positioned nucleus . Their hair bundles form a characteristic 'W'-shape and contact the underside of the overlying TM in which impressions of the longest OHC stereocilia can be seen . A fibrous protein, stereocilia, appears to link the tips of the longest stereocilia to the insertion in to the TM.

Mutations in the gene for stereocilina are associated with hearing impairment.

Coupling between the longest stereocilia and the TM provides the mechanism by which OHCs are stimulated. The up-and down movements of the travelling wave that is generated along the BM in response to the sound-induced fluid displacements along the scala vestibuli and scala tympani translate into radial (medial to lateral) movements at the apical surface of the organ of Corti. The TM mass remains static, so there is a relative shearing motion between the reticular lamina (the apical surface of the organ of Corti) and the TM.

Since the longest stereocilia of OHCs are embedded in the underside of the TM, this differential movement results in the deflection of the hair bundle along the line of functional polarity resulting in opening and

closing of the Mechano electrical transduction channels. Deflection of OHC stereocilia generates a change in membrane potential in the cell, a receptor potential. OHCs increase in length systematically from the base of the cochlear spiral to the apex. The longest stereocilia on OHCs also increase in height systematically along the base-to-apex length of the cochlea, in humans increasing from around 2.5 μm in the basal coil to around 7.0 μm at the apex. OHCs, in contrast to IHCs, are directly innervated at their basal ends, by several large bouton like efferent endings

About 80% of the efferent cochlear innervation terminates on OHCs. These medial olivo cochlear efferent nerves that synapse with the OHCs arise primarily from the medial portion of the contralateral superior olive. The afferent innervation pattern suggests that OHCs may signal more

global, large displacements of the BM, i.e. at high sound pressures.The extensive efferent innervation, on the other hand, strongly indicates that OHCs have a modulatory role in the cochlea.

Thus OHCs are critical for the exquisite frequency discrimination of which the cochlea is normally capable. The OHCs are the cellular basis of the 'cochlear amplifier'. A unique form of motility is exhibited by OHC. Isolated OHCs maintained in short-term culture undergo fast reversible axial length changes at up to auditory frequencies (20 kHz) when stimulated electrically. The changes in OHC-membrane potential deriving from the normal MET mechanism drive the length changes. Thus motion of the BM induces these motile responses, which are thought to feedback into the BM motion, thereby removing local damping and enhancing the movements.

The end result of this cochlear amplification process is to fine-tune and amplify the signal before it reaches the IHCs. The excess energy released by this active process generates sound waves that are emitted from the ear as an otoacoustic emission (OAE).These OAEs can be recorded from probe microphones fitted in the external ear canal and they form the basis of the now routine audiological screening test for hearing impairment that can be performed in newborns or adults, and that provides an objective measure of the 'health' of the OHCs. The fast motile responses of the

OHCs, and the concomitant OAEs, are driven by a motor protein called prestin, which is unique to OHCs and which is densely packed all down the lateral plasma membrane of the cell. Mutation or functional absence of the gene encoding prestin causes hearing impairment due to loss of cochlear amplification

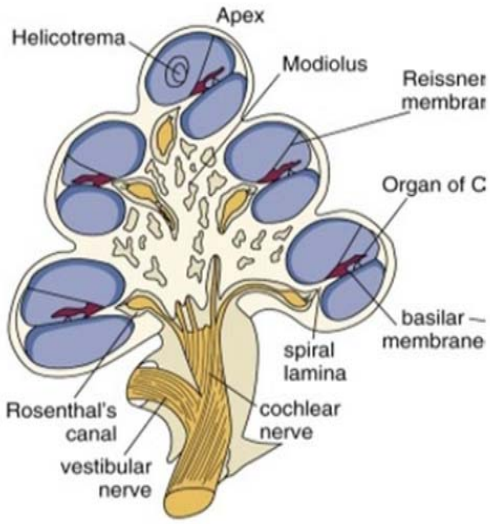
The activity of OHCs upon stimulation is thought to generate a radial flow of endolymph across the surface of the organ of Corti which deflects IHC stereocilia and stimulates IHC responses. In essence, at lower sound pressure levels (below about 60 dB) OHC activity 'drives' IHC responses, but at higher sound pressure levels the larger movements of the organ of Corti produce fluid flow sufficient to deflect IHC stereocilia, and stimulation of the cell, directly

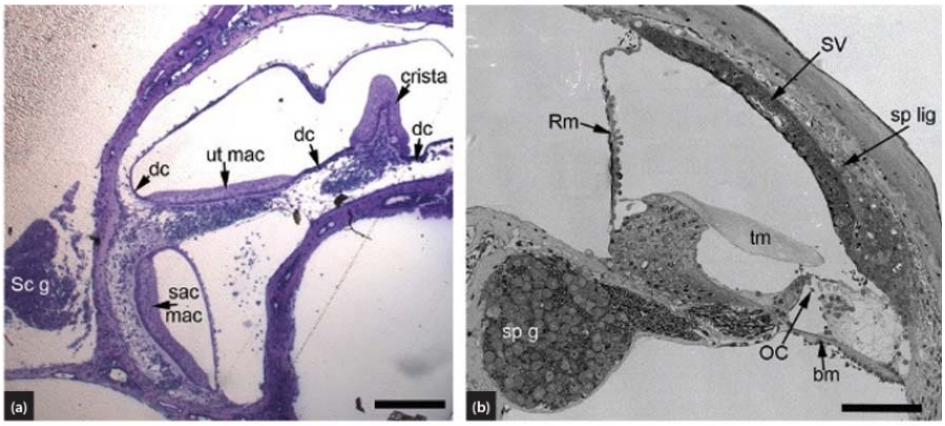
INNERVATION:

The afferent ANFs that innervate the inner and outer hair cells arise from two distinct neuronal populations.The majority (90–95%) are myelinated type 1 neurons that innervate IHCs. The remaining 5–10% are unmyelinated ,thin type 2 neurons that innervate OHCs. The cell bodies of both these types of neurons are collected together in the spiral ganglion (hence the designation 'spiral ganglion neurones' that is sometimes used) which is enclosed in Rosenthal's canal and located within the bony lip of the modiolar wall just medial and below the organ

of Corti itself . The central axonal projections of these bipolar neurons collect together in the modiolus .

The number of axons and the width of the nerve (and the modiolus itself) systematically increasing from apex down to the base of the cochlea as the centrally projecting axons from the cell bodies of neurons at successive locations are incorporated. The large bundle of neurons exits the inner ear via the internal auditory meatus as the VIIIth cranial nerve. The peripheral neurites from the cell bodies access the sensory epithelium through a series of holes, the habenulaperforata, through the thin bone that separates the organ of Corti from the ganglion and over which the IHCs are located .





The type 1 neurons lose their myelination as they reach the habenula, and so are unmyelinated in the organ of Corti as they project directly to the base of the IHCs. The peripheral neurites of the type 2 cells, after crossing through the habenula, turn to run between the bodies of the inner pillar across the floor of the tunnel of Corti, then between the bodies of the outer pillar cells into the region of OHCs. Here they branch, each one sending projections between the Deiters cell bodies to an average of nine OHCs, their terminals mostly synapsing at the very base of the cell.

The innervating efferent neurons have their cell bodies in the mid brain regions from which they project. The peripheral axons of these neurons also enter the organ of Corti through the habenulaperforata. Those projecting from the contralateral medial superior olive in the brain that innervate the OHCs contribute about 80% of the total efferent innervation of the organ of Corti. They pass between the phalangeal processes of

adjacent inner pillar cells, cross the tunnel of Corti at a level about the middle of its height, and then pass between phalangeal processes of the outer pillar cells into the OHC region where each individual nerve branches, sending projections to OHCs in each of the three rows. Axons of the efferent nerves arising from the ipsilateral medial superior olive, afte

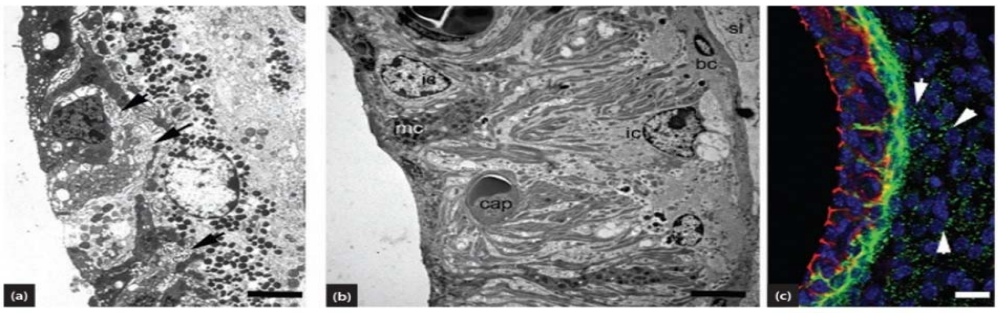
passing through the habenulaperforata, projects directly to the region of

the IHCs, branch and form terminals that synapse with the terminals of the type 1 afferent neurons beneath the IHCs.

The striavascularis (SV) is a strip of tissue 150–300 μm wide lining the lateral wall of the scala media and running along its entire length . It is responsible for the production and maintenance of both the high endolymphatic K+ concentration and the EP. The SV encloses a complex capillary network and is composed of three cell types :

- Marginal cells that line the endolymphatic compartment
- Intermediate cells in a discontinuous layer enclosed entirely with in the body of the epithelium
- Basal cells that separate the SV from the underlying spiral ligament.

The SV is said to have the highest rate of oxidative metabolism in the entire body, most likely due to the huge energy demand resulting from the mass of active ion transport that takes place in this tissue. The EP provides a source of energy or ‘battery’ to drive the cochlear amplifier.



MARGINAL CELL:

The marginal cells of the SV are primarily involved with the transport of K+ and are essentially the same as the dark cells of the vestibular system. Their basolateral membranes are extensively infolded, enclosing numerous large mitochondria and they contain high levels of Na+/ K+-ATPase, both α- and β-isoforms and the Na+/ K+/Cl–- cotransporter NKCC1 (SLC12A2). NKCC1 is the therapeutic target of action for loop diuretics in the kidney and loop diuretics have rapid, acute ototoxic side effects through an action on the cotransporter in the strial marginal cells inhibiting ion transport, which results in accumulation of ions in the extracellular spaces of the stria and a consequent oedema

The apical membranes of the marginal cells (like the dark cells) contain a K+ channel which is formed of two subunits, the KCNE1 regulatory

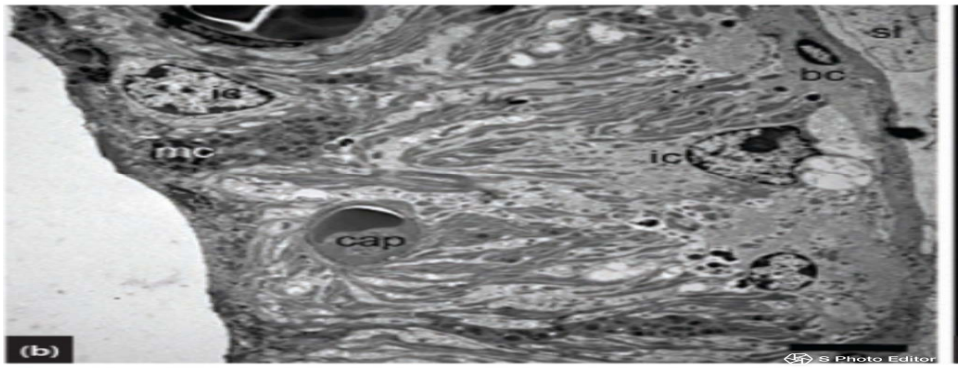
protein and the KCNQ1 channel proteins that provide the pathway through which K+ is secreted into endolymph.Mutations in the KCNE1 gene disrupt endolymph production, leading to collapse of Reissner's membrane and deafness. In the vestibular system they cause collapse of the epithelia of the roof of the utricle, saccule and ampullae indicating dysfunction of the vestibular sensory organs.

During development, high levels of K+ are found in cochlear endolymph, and strial marginal cells show high Na+/K+ ATPase activity, indicate that strial marginal cells, like dark cells, are primarily concerned with active transport to maintain the endolymphatic K+ concentration, but the generation of EP is a separate phenomenon. The absence of any other cell type from the ion-transporting tissue of the vestibular system suggests that the strial intermediate and basal cells play a role inEP generation. During cochlear development the onset and rise in EP coincide with the incorporation of intermediate cells and blood vessels into the body of the stria and the formation of the limiting basal cell layer.

INTERMEDIATE CELLS:

The intermediate cells are a type of melanocyte – melanin pigment-containing cells – that arise during development from cells that migrate from the neural crest. They are entirely enclosed within the corpus of the stria, inter digitating with the other two cell types . They contain a variety

of enzymes that enable energy production from alternative substrates such as lipids as well as enzymes that detoxify oxidative wastes. Melanin can act as a free radical scavenger. These cellular properties suggest that intermediate cells is to protect the stria under conditions of stress and to provide alternative energy sources to maintain activity during periods of reduced blood supply. Intermediate cells also sends thin processes to contact the capillaries and express a number of proteins that are found in macrophages, further indicating the role of these cells in protecting the stria from damage. These cells have been termed perivascular macrophage-type melanocytes.



The crucial role of intermediate cells,is in the generation and maintenance of EP. The intermediate cells are characterized by very high levels of the K+ transporter protein Kir4.1 in their plasma membranes facing the intercellular spaces and the basolateral infoldings of the marginal cells.Thus EP is considered to be generated by rapid and extensive passage of K+ via Kir4.1 across the intermediate cell membrane into

the intercellular space, followed by immediate uptake of K+ by marginal cells such that a high positive potential is generated in that space. The basal cell layer provides an insulating seal that prevents dissipation of the potential into the perilymph in the intercellular spaces of the spiral ligament.

BASAL CELLS:

The basal cells are flattened and elongated, forming between one and three layers delimiting the basal aspect of SV . They arise during development from the mesenchymal cells that also form the spiral ligament. Basal cells closely appose each other and there is extensive sealing of the intercellular spaces between them by tight junctions. This creates an impermeable barrier between the perilymph in the underlying spiral ligament and the body of SV. During development, the initial formation of these tight junctions and the increase in their complexity coincides with onset of EP, suggesting that these junctions are necessary to provide the electrical insulation required for the potential difference to be generated and maintained within the body of SV. Mutations in the gene for the protein claudin 11, which is present in the basal cell tight junctions, cause the loss of EP and so hearing impairment.

Large numbers of gap junctions are also associated with basal cells .They are present between adjacent basal cells, between basal and intermediate

cells and between basal cells and fibrocytesin the spiral ligament. Thus, basal cells appear to be the central element in a functionally coupled unit or syncytium, connected together by intercellular gap junctions, consisting of basal cells, intermediate cells and spiral ligament fibrocytes. Marginal cells are excluded from this syncytium; they do not form gap junctions either with each other or with either basal or intermediate cells and are thus separated, functionally, from each other and from the basal cell/intermediate cell/ligament fibrocyte syncytium.

The gap junction-mediated intercellular communication between basal cells and ligament fibrocytes can provide a pathway for access of ions into SV cells from the ligament that bypasses the tight junctional sealing.Fibrocytes of the spiral ligament possess Na+/K+-ATPase activity and thus probably function to take up K+ from the perilymph in the intercellular spaces of the ligament. The gap junctions between fibrocytes provide an intracellular route for K+ to those fibrocytes beneath the SV, which are coupled to strial basal cells and which in turn are coupled by gap junctions to the intermediate cells. This gap junctional system, therefore, provides a route for recycling K+ from endolymph, through hair cells to perilymph in the spaces in the organ of Corti and into supporting cells; out to the intercellular spaces of the spiral ligament and

into fibrocytes; then into strial basal cells; into intermediate cells out to the intrastrial spaces and back to endolymph via the marginal cells.

The intercellular communication provided by gap junctions may, therefore, be important for the maintenance of EP. During development, the onset and subsequent rise in EP corresponds with the formation of the permeability barriers that isolate the SV from the surrounding tissues but also with the initial formation and subsequent increase in size and number of gap junctions associated with basal cells.

CAPILLARIES:

The functional isolation of the SV from the surrounding tissues created by the permeability barriers at the level of the basal cells means there is little or no access of oxygen and nutrients required for the active processes necessary for EP maintenance. These are supplied by the network of intrastrial capillaries that run predominantly longitudinally along the SV with short interconnecting branches running across the strial thickness. The capillaries are often closely surrounded by extended processes from marginal cells.

While allowing passage of oxygen and essential nutrients, the walls of the capillaries form a ‘blood labyrinth barrier’ that controls exchange between the intrastrial spaces and the blood supply. The capillaries are supplied

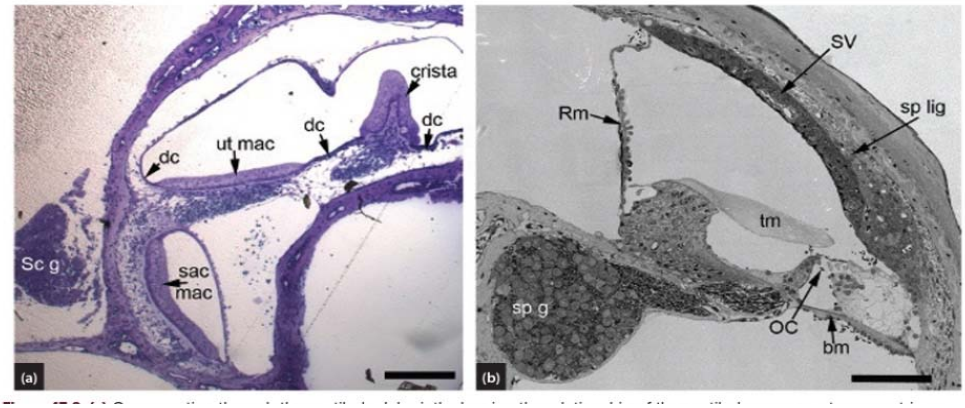
from branches of the cochlear artery that run from the modiolus through the connective tissue over the roof of the scalavestibuli and further branch into the spiral ligament and SV where the network of capillaries forms. They drain through capillaries that run inthe connective tissue beneath the scala tympani to the cochlear vein in the modiolus.

The strial capillaries are unfenestrated and the junctions space.This tight sealing of the capillary lumen from the body of the stria in addition to the tight junctions between marginal cells and the extensive tight junctional sealing between basal cells are crucial to the functional isolation of stria from the surrounding tissues that prevents dissipation of EP as it is generated.

The endothelial cells are surrounded by a basement membrane. Pericytes discontinuously cover the basement membrane extending long, thin processes along the capillary wall. Where these cells are present the basement membrane thickens to cover them. It has been suggested that macrophage-like melanocytes (the intermediate cell subpopulation), pericytes, BM and endocytes act as a complex to monitor and regulate exchange between the intrastrial spaces and the vessel lumen and maintain the blood-labyrinth barrier.

REISSNER'S MEMBRANE:

Reissner's membrane is a thin epithelial sheet formed of two cell layers .A continuous layer of epithelial cells lines the scala media side and, since their apical surfaces face endolymph, they have tight junctions sealing the spaces between adjacent cells. The basal side of the epithelium, in the scala vestibuli, is covered with mesenchymal cells that are bathed in perilymph. The primary role of Reissner's membrane, to maintain the electrical and chemical separation of endolymph and perilymph, is achieved at the level of the tight junctions between the epithelial cells.



Reissner's membrane appears to have some elasticity as it can accommodate by swelling outwards into the scala vestibuli quite large increases in endolymph volume endolymphatic hydrops such as occurs

with ion imbalance that results in water influx into the scala media, before it ruptures

PHYSIOLOGY OF HEARING

THE COCHLEA:

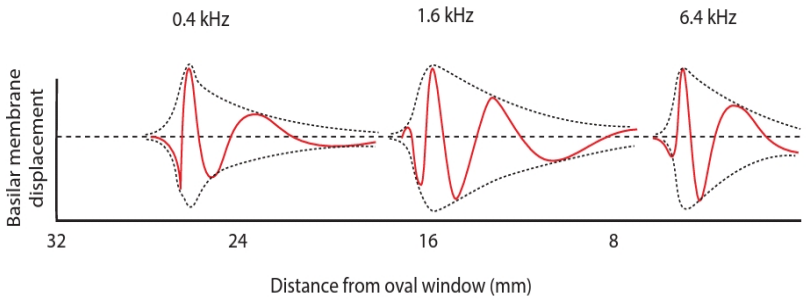
The stapes–oval window interface delivers the mechanical and kinetic energy of the incident acoustic signal to the cochlea. The cochlea is a 2.5-turn coil. Sound introduced to the cochlea undergoes two major processes: a biological and active compression/amplification by the outer hair cells (OHCs) and an electrochemical transduction by the inner hair cells (IHCs), both contained in the scala media of the cochlea. The resulting neural output is a coded and gated signal conveyed by the auditory nerve via the spiral ganglion cells to the brainstem, which preserves frequency information, the amplitude and the phase of the sound by cochlear tonotopicity (frequency specificity), auditory nerve firing rate, phase locking and synchrony.

The cochlea is characterized by amplification to make hearing more sensitive, tuning to make it sharply frequency selective and compressive non-linearity so that relatively large stimuli are translated to proportionally lesser systematically encoded mechanical functions to maintain integrity. Cochlear mechanisms for the above actions are therefore responsible for

the essential functions of the cochlea that include frequency analysis, loudness discrimination, temporal resolution, and the spectral analysis of the incoming signal.

Cochlear OHC function is measured by otoacoustic emissions that include transient emissions in response to a broadband click and distortion product emissions in response to frequency-specific pure tones. It can also be measured by electrocochleography and cochlear microphonics, the latter being incorporated in the software for auditory brainstem response (ABR) testing

COCHLEAR TRAVELLING WAVE:



cochlear travelling wave.

The mechanical displacement of the stapes–oval window junction is conducted along the cochlea, impinging on the base first and then travelling all the way up to the apex. In its wake, it causes mechanical movement of the BM which divides the cochlea into two compartments:

the scalavestibuli above and the scala tympani below with a small connection between the compartments at the helicotrema in the apex. The scalamedia between the scala vestibuli and the scala tympani separated from the scala vestibuli by the Reissner's membrane and the scala tympani by the basement membrane. Three different travelling waves generated: the wave as a result of the pressure difference of the two compartments, the wave as a result of the mechanical displacement of the BM, and the acoustic energy wave which displaces the cochlear fluids. The displacement wave is by far the most important wave for cochlear function.

The travelling wave propagates aided by the gradual diminution of the thickness and stiffness of the basement membrane from base to apex. As it propagates, it is acted upon by numerous critical oscillators, the characteristic frequency of which is specific to a particular region of the BM. These oscillators move the BM along with the travelling wave by expending active energy and are coupled with OHCs in the organ of Corti.The oscillators become active when they compress or modify this signal and passive when they allow the signal to pass. There is a critical point at which these may cancel each other out called the Hopf bifurcation. Hence the oscillators must possess an auto regulation process or a self-tuning property which generates a cochlear tuning curve.

The critical oscillation function and the compressive function of the OHC are responsible for tuning the BM in response to an acoustic signal, which is variable along the length of the BM and is spatially represented, it can be inferred that the acoustic output, the end result of the BM function, is a non-linear output.

In pure tones with frequencies well below the characteristic frequency the function is linear and passive. In disease processes, the non-linearity may become linear and can be measured in the growth function of distortion-product otoacoustic emissions. Pathologies such as ototoxicity and possibly age-induced hearing loss will interfere with the non-linear function of the cochlea.

COCHLEAR TUNING CURVE :

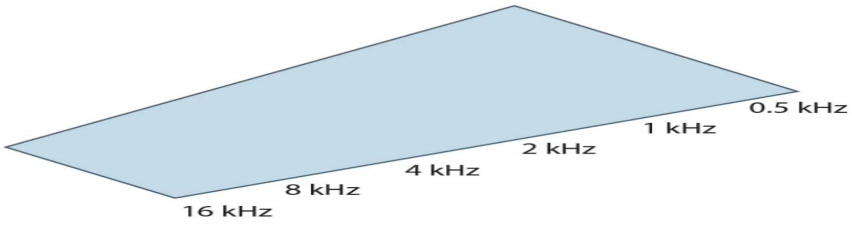


Figure 48.16 Tonotopic representation of the cochlear BM.

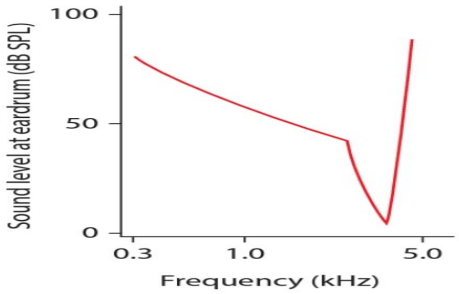


Figure 48.17 The cochlear tuning curve.

The cochlear travelling wave in response to an acoustic stimulus reaches a maximum displacement along the BM which it starts to dissipate. High frequency sounds produce peak displacement towards the base of the cochlea whereas the peak moves progressively towards the apex as sound frequency decreases. The frequency at which the maximum displacement occurs is also called the characteristic frequency at a specific place in the BM making it highly frequency-specific or tonotopic(Figure 48.16). A cochlear tuning curve is the response of the cochlear BM to changing intensities to achieve a maximum amplitude response and is plotted as a function of intensity with frequency. The human ear has a dynamic range up to 120 dB which inherently dictates

that signals of very high intensity must undergo modification at the cochlear level without damaging the cochlea and need to be compressed. The cochlea does this via the OHCs and their non-linear function, and depends on the frequency specificity or the tonotopicity of the BM.

Compression is achieved by the OHCs to generate the tuning curve. This compression is essential for maintaining the integrity of the BM and increasing its stability in the presence of high-intensity stimuli. For lower-intensity signals, the OHCs amplify the BM response by mechanical elongation/compression of their cell bodies which sharpen the tuning curve; this is variable across the response, preserving non-linearity. Another property of the cochlea is its filtering action. This is indirectly dependent on the tonotopicity. The characteristic frequency is other neighbouring frequencies are filtered so as not to interfere with the frequency selectivity i.e., the characteristic frequency is best resolved at a given point in the BM in preference to other frequencies .The filtering explains the attribute of perceptual streaming. This is essentially a central auditory function contributed by the ability of the cochlea to discern a rapid sequence of sounds coming from a single or a multiple source . When the sounds are close together in frequency, fusion

may occur and the perception will be of a single sound as the filtering action becomes less in neighbouring frequencies.

The dynamic range is significantly lost and reduced in cochlear pathologies. The tuning curve loses its sharpness and frequency selectivity is compromised along with perceptual streaming. Different pathologies may affect the shape of the tuning curve in different ways: for example, some genetic losses involve the lower-frequency sensitivity while ototoxicity affects the high-frequency sensitivity. Cochlear hearing loss therefore may show some characteristic features, including loss of frequency selectivity leading to difficulties to understand speech and appreciate music, loudness recruitment due to reduced dynamic range and a compromise of perceptual streaming which manifests itself as an inability to perceive or segregate multiple sounds at a given time.

COCHLEAR FLUID:

The cochlea contains perilymph in the scala vestibuli and scala tympani. This is an ultrafiltrate of blood plasma and the CSF, rich in sodium and low in potassium and calcium. The scala media or the cochlear partition consists of the endolymph which is actively pumped by the striavascularis in the scala media. Endolymphis rich in potassium, low in sodium and has negligible calcium. The stereocilia of the hair cells are bathed in the endolymph while the hair cell bodies are bathed in perilymph. There is

another compartment called the intrastrial space which is the space between the extracellular matrix of the striavascularis. The cochlea is supplied by the spiral modiolar artery, which is a branch from the vestibulocochlear artery (VCA) from the anterior inferior cerebellar artery. The VCA is an end artery.

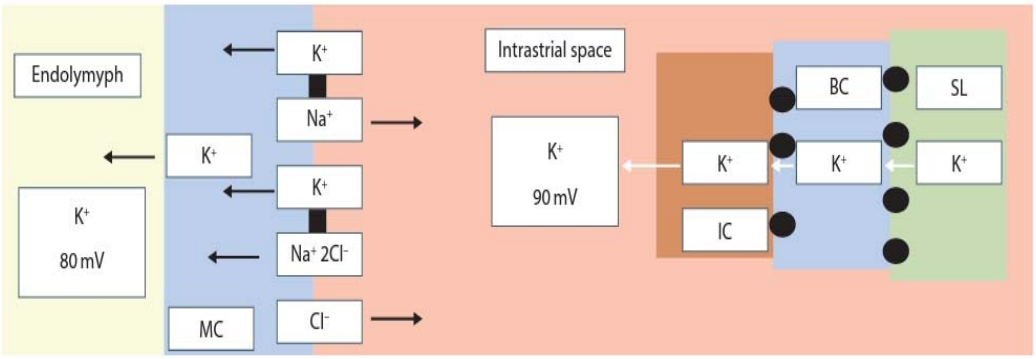
The capillary network is arranged in a parallel fashion in the lateral wall of the cochlea; there is an abundance of pericytes and fibrocytes in the network, and the cochlear blood flow is autoregulated by multiple factors include local metabolites such as lactate, nitrous oxide and potassium, the pericytes and the fibrocytes, and the blood vessel smooth muscle cells. Cochlear microcirculation ensures the integrity of the cochlear fluids and there exists a blood–labyrinth barrier which is responsible for ultrafiltration and abundantly supplied with enzymes and protein subunits, for example the Na+ - K+ ATP subunit so important for ionic transport.

The objective is to deliver energy and provide processes for ionic transport for the cochlear internal milieu. Cochlear microvasculature is affected in a number of disease processes with secondary involvement of cochlear fluid hydrodynamics which in turn lead to impaired cochlear functions. These include noise-induced hearing loss, endolymphatic hydrops and possibly age-induced hearing loss. In addition, the artery is an

end artery, which implies that a blockade or obstruction to it and if it persists beyond a certain period of time (about 15–30 minutes) will lead to irreversible damage to the organ it supplies, which is the cochlea vestibular apparatus.

The highly intricate cochlear microvasculature ensures nutrition in the cochlear compartment and also provides the internal environment for cochlear function. The main driving ion for cochlear function as furnished by different cochlear processes is potassium. The way it is recycled is called a potassium cycle, responsible for the endocochlear potential. This potential is the resting potential kept at a constant level in the scala media. Potassium utilizes a concentration gradient. However, the role of Na+ for maintaining the endolymphatic potential cannot be ignored as the Na+Cl-ATP co transporter in the marginal cells and in the fibrocytes of the spiral ligament ensures further potassium efflux and influx by coupling with the potassium. The endocochlear potential results in a dynamic flux of ion transport in the cochlear endolymphatic space in the scala media. The stria vascularis has K+ four cell types: the marginal cells related to the medial scala media, responsible for maintaining a low potassium composition in the intrastrial space by continuous active uptake of the ion from the endolymphatic space; the intermediate cells, which have the marginal cells medially and the basal cells, laterally, are connected to the basal cells by

gap junctions regulated by the connexin family genes and where the endocochlear potential is generated; and the basal cells, with the intermediate cells in their medial end and laterally connected to the spiral ligament in the lateral wall of the cochlea by gap junctions as well.



Potassium from the blood is actively taken up by the fibrocytes of the spiral ligament and pumped to the basal, cells which in turn deliver the ion to the interstitial cells. These cells present the ion to the intrastrial space from where they are taken up by the marginal cells. The scala media receives its ions from the marginal cells. All the active processes are regulated by various enzymatically driven potassium channels and Na–K ATP systems; the end result is maintenance of a high potassium ionic composition in the scala media which is vital for hair cell function by virtue of the endocochlear potential.

Cochlear hair cell integrity depends on enzymatically driven functions to maintain structural stability. Thus mutations in the protein- encoding genes are likely to cause a sensory hearing loss. Hyperacoustic stimulation depresses the potassium cycle for protecting the cochlea and actually leads to a drop in the endolymphatic potential and stimulation of the P2X by the ATP pathway, which inhibits OHC motility, while beta adrenergic stimulation as a part of sympathetic stimulation swings the cycle to the opposite side. Genetic mutations in the connexin family or the potassium transport family interfere with maintaining endocochlear potential and therefore sensory epithelia function. Examples include connexin 26/30 hearing loss, the commonest genetic autosomal non-syndromicprelingual genetic hearing loss, and Jervall–Lange–Nielsen syndrome with long QT interval where the KCNQ1 ionic transport gene in the striavascularis and cardiac conductive system is deficient.

COCHLEAR SENSORY EPITHELIA AND SUPPORTING CELLS:

The mammalian cochlea is characterized by the presence of two distinct subtypes of sensory epithelia which differ in orientation, morphology and function. The OHCs number about 12 000 in humans; are arranged in three rows; participate in electromotility leading to cochlear tuning, amplification, compression and frequency selectivity and are innervated

by type 2 spiral ganglion afferents and cochlear efferents from the medial olivocochlear bundle. IHCs are arranged in one row; number about 3500 in humans; participate in cochlear transduction of the incoming acoustic signal; largely generate the cochlear afferents to the spiral ganglion type 1 cells from where the auditory nerve commences and also receive efferents from the lateral olivocochlear bundle. Both these cells form the sensory epithelium in the organ of Corti with their apical or stereocilia side bathed in the endolymph of the scala media and their bases bathed in the perilymph of the scala tympani.

Undulations or mechanical vibrations from the BM lead to a shearing force in the tectorial membrane which in turn moves the stereocilia of the hair cells in the endolymph.

THE OUTER HAIR CELLS :

The OHCs are cylindrical with bundles of stereocilia composed of actin filaments which project at their apical ends in the scala media. BM-led tectorial membrane displacements in response to acoustic stimuli result in movement of these stereocilia in a plane parallel to their plane of orientation: depolarization when the movement is towards the tallest stereocilia and hyperpolarization/ repolarization when the movement is towards the smallest stereocilia. Displacement opens up the cation-selective ionic gates responsible for transduction and generation of an

action potential. A displacement of just 0.3 nm can displace the stereociliary bundle. An action potential thus generated happens within 10 ms, which is a graded action potential (AP) mediated through the tip links between adjacent stereocilia which regulate ionic flow in this case potassium from the potassium-rich endolymph. The OHC itself is more negative than the endolymph, which facilitates movement of potassium without any energy expenditure.

Potassium entry through the transduction channels then generates voltage gated calcium channels and results in the influx of Ca2+ ions in the cells which participate in synaptic transmission and 10% of the depolarizing voltage. The entry of potassium constitutes the depolarization stage. Repolarization occurs in two distinct processes: by the efflux of cell-rich potassium to the potassium-poor perilymph across the concentration gradient through the cation-selective ionic channels in the basolateral portion of the OHC, and by the influx of the Ca2+ . The action potential is transmitted to the type 2 spiral ganglion cells, which are essentially non-myelinated and smaller than their IHC counterparts and not as developed. They show reciprocal synapses with their OHC counterpart, i.e. the type 2 cells feed back to the OHC providing a pathway for a closed loop neuronal circuit for bidirectional signalling and reverse transduction.

The action potential in the OHCs does not carry auditory information to the auditory nerve. Its job is to provide a motor for altering OHC physical dimensions for further displacement of the BM. The force contributed by this motor is sufficient to drive the acoustic signal through the entire length of the cochlea as an additional travelling wave as well as providing enhanced modulation at a local, site-specific area of the BM 70. It also leads to movements of the tectorial membrane itself to open up further ionic gates and augment the AP and amplification process.

The OHC exhibits the special feature of electromotility, which is a highly sensitive attribute as cochlear motility translates frequency specificity and amplification and is responsible for fine-tuning of the acoustic signal. In response to acoustic stimulation of the BM, this motility is driven by two forces: a voltage-dependent mechano transduction that moves the hair bundle with an active movement and a somatic non-linear capacitance prestin-mediated motility which modulates the stiffness of the stereocilia and makes them alter their sizes. Prestin is abundantly located in the lateral membrane of the OHC, which belongs to the SLC26A family and participates in selective anion transport and binder, in this case chloride and carbonate. The action of prestin is voltage dependent and results in either contraction or elongation of the OHC necessary for augmenting the acoustic signal incident on the BM. Cochlear electro

motility is sensitive to frequencies and intensities of the signal. The OHC in addition receives efferents from the medial olivocochlear bundle in the brainstem which synapse with the spiral ganglion type 2 nerves, offering a regulation of the reverse transduction process.

THE INNER HAIR CELLS

The IHCs are the true sensory end organs for hearing and are responsible for generating the action potential which then is conducted to the type 1 spiral ganglion cells to be delivered to the auditory nerve. The spiral ganglion type 1 nerve endings are myelinated and synapses are well developed with ribbons and glutamate activity, unlike the OHC synapses. The IHCs are non-actively motile and receive the output from the OHCs through the modified movement of the BM at a given region of the BM. The IHCs are depolarized as a result of BM movements causing deflection of the stereocilia. The depolarization is mediated by cations potassium and Ca2+, the latter being crucial for synaptic action through chemical neurotransmitters especially glutamate.The IHC synapses do not exhibit any reciprocal arrangement, unlike the OHC. Ca2+ release is mediated by voltage-dependent depolarization mediated by potassium of the IHC apical membrane, i.e. the stereociliary deflections. Since it delivers the eventual output of cochlear function, the IHC needs to contain all the information amassed so far in the form of coding to the cochlear nerve.

There are two types of coding, namely frequency coding and intensity coding. The IHCs phase-lock with the stimulation frequency under 5 kHz while, above this frequency, the frequency-specific depolarization/repolarization is too fast for the IHCs to phase-lock. Intensity coding is by way of an increasing number/rate of action potentials proportional to stimulus intensity.

THE COCHLEAR SUPPORTING CELLS

They (i.e. Hansen cells, Deiters cells, pillar cells, inter phalangeal cells and border cells) provide the scaffold for anchoring the hair cells, these cells also participate in cochlear function and stability. The supporting cells contribute to the development, differentiations, patterning and synaptogenesis of the hair cell sensory epithelia. Another important function of the supporting cells is that they contribute to the planar cell polarity (PCP) of the hair cells, which essentially is the stable plane of orientation of the basal and apical ends of the hair cells which expose them to the right environment for mechano transduction.

In the mature cochlea, the supporting cells provide the anchor and the platform for tight adherence of the sensory epithelia to the BM at the basal surface and form the reticular lamina at the apical surface where it separates the endolymph from the perilymph and helps maintain the endocochlear potential. They provide proteins for maintaining the

extracellular matrix of the BM and thus maintain rigidity and stability of the hair cell population.

- they take up Na^+ from the endolymph, ensuring a low concentration of the ion in the scala media.

- They assist in the potassium cycle, especially in the basolateral part of the BM.

- They influence glutamate activity at the hair cell synaptic regions.

By virtue of their gap junctions (which are regulated by connexin26), they are involved in the passage of ions and compounds either across the concentration gradient or actively by the ATP mechanism. This ATP-mediated energy-expending process also leads to Ca^{2+} coupling in the hair cells;

Deiters cells have an internal Ca^{2+} store. Supporting cells may play a role in the eventual repair and regeneration of the hair cells in response to damage. There is nerve innervation of the supporting cells which implies that these cells can generate an action potential. These nerve endings do not synapse with the cochlear efferents or contribute to the formation of the cochlear nerve; they are considered to be a part of the internal neural circuitry which characterizes OHC afferents and participates in OHC non-linear fine-tuning of the acoustic signal through BM activity.

Mutations in genes encoding for supporting cell proteins will lead to cochlear sensory loss. connexin 26 is also expressed in the supporting cells and a mutation will lead to the loss of structure, stability and integrity of the hair cells through the supporting cell mechanism. DFNB29, a cause of autosomal recessive hearing loss, is characterized by a mutation in the gene containing claudin, which is a structural protein of the reticular lamina responsible for tight junctions. A loss of this tightness leads to increased potassium permeability from the scala media to the hair cells, resulting in a drop of the endocochlear potential and loss of function.

PATHOPHYSIOLOGY OF COCHLEAR HEARING LOSS:

Loss of cochlear function for any reason will result in a drop in auditory sensitivity; a drop in complex sound appreciation and analysis due to the loss of frequency selectivity; loss of fine-tuning of the acoustic signal with narrowing of the dynamic range of hearing due the loss of non-linearity, amplification and compressive properties; loss of perceptual streaming due to the loss of frequency selectivity; and loss of temporal pattern of sounds due to the loss of spatial discrimination and the ability of the cochlea to distinguish between closely following frequencies and intensities.

• Structural abnormality: A cochlear dysplasia of any kind leads to ill-formed or absent cochlear components. This can follow a congenital mal development, trauma or a space-occupying lesion and will lead to a cochlear hearing loss.

• Abnormal metabolic activity: Since ionic transport dictates cochlear function, it follows that a metabolic abnormality (systemic or otherwise) that interferes with cochlear ionic transport as a result of either a genetic syndrome or an acquired condition (e.g. problems with glucose metabolism or thyroid metabolism) will lead to changes in the endo lymphatic potential and affect cochlear function.

• Vascular changes: Cochlear vascular afflictions (either systemic or local) will lead to a compromise in stria vascularis function; a diminution in cochlear nutrition and hypoxia; sluggish vascular flow or complete obstruction of the cochlear arterial blood flow. Examples include pressure effects due to any cause, noise trauma and ototoxicity, transient ischaemic attacks (TIAs) and hyperviscosity Endolymphatic hydrops, i.e. an expansion of the endolymphatic space for any reason including a lack of integrity of the striavascularis (e.g. Ménière's disease or endolymphatic fluid disturbances such as third window syndromes) will lead to a compromise in function of the striavascularis.

• Overloading the BM: A crowded or loaded BM due to local or systemic causes (e.g. a metabolic syndrome such as diabetes, hyper lipidemias, iron overload, inflammation or autoimmune conditions) will prevent OHC motility and IHC loss of transduction.

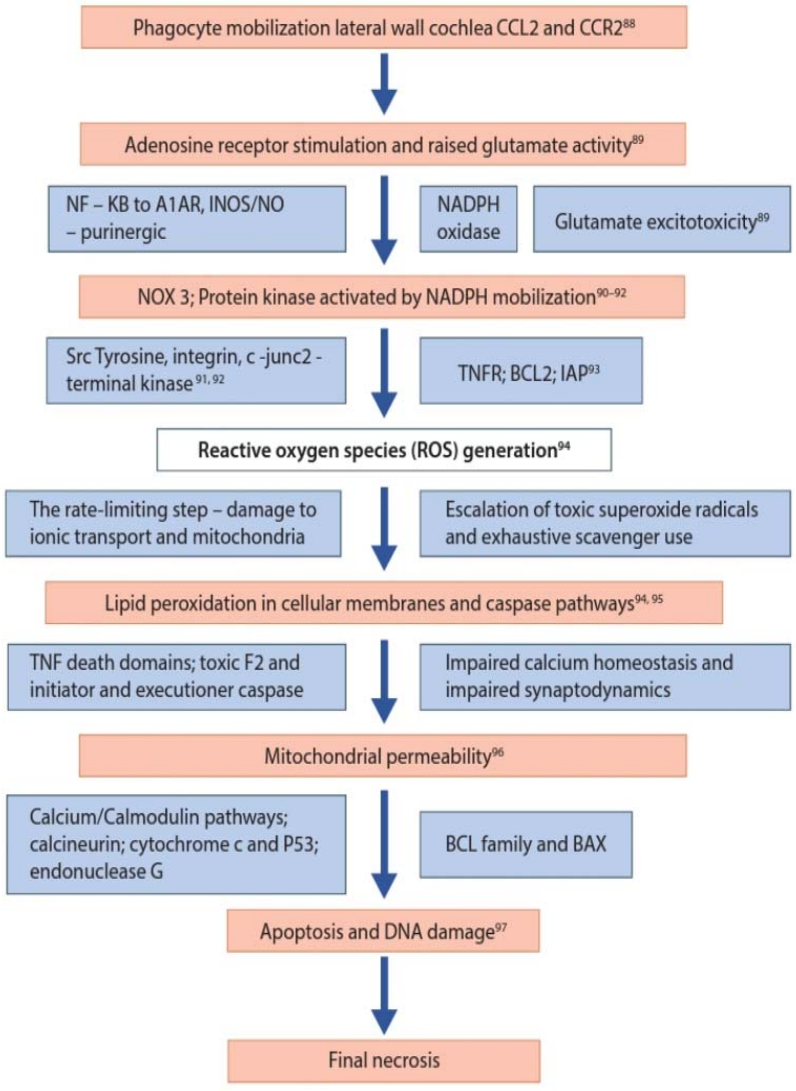
• Infection and inflammation: An inflammatory involvement in the cochlea leads to the disruption of the biochemical pathway of cochlear function (see below) and the accumulation of toxic lipid-derived substrates which hamper cochlear function.

•Genetic mutations: genetic mutation in the genes encoding for numerous proteins involved in cochlear function, from ionic transport to structural proteins, will lead to a cochlear hearing loss.

•Biochemical pathway abnormalities: In response to noise trauma, ototoxicity and due to cumulative wear and tear action in presbycusis, a biochemical cascade for cochlear hair cell integrity loses its balance leading to apoptosis and subsequent cell death.

Otoprotective endogenous compounds play an important role in preserving the delicate cochlear structures and influence the biochemical cascade. These include heat shock factors (HSFs) and heat shock proteins (HSPs), both abundantly expressed in the cochlea, which can modify the apoptosis response down regulation of some growth factors; neurotrophin,

mainly glial cell-derived neurotrophic factor(GDNF) regulation, to modify the end result of apoptosis for recovery of function and the coenzymes Q9 and Q10



THE AUDITORY NERVE AND COCHLEAR EFFEREENTS:

The auditory or cochlear nerve, cranial nerve VIII, originates from the joining of the spiral ganglion cells in the cochlea where their cell bodies lie. There are two types of nerve fibres originating from the spiral ganglion cells: type 1, which have large diameters, innervate the IHCs constituting 95% of the nerve fibre population and are myelinated, and type 2 fibres, which are smaller diameter, innervate the OHCs and are unmyelinated. The nerve traverses the internal auditory canal and joins with the vestibular nerve forming the vestibulo cochlear nerve. One type 1 fibre innervates one IHC but one IHC may synapse with several nerve fibres. The cochlear nerve fibres terminate at the cochlear nuclear complex located in the medulla of the brainstem. The cochlear nucleus synapses with the cochlear nerve and is represented tonotopically, which implies that frequency encoding by the cochlea is carried up to the central nervous system to be further analyzed.

The action potential of the cochlear nerve in response to acoustic stimulation entails a release of bundle synaptic receptors proportional to the stimulus, which results in spike potentials which are short-lived and attain their peaks quickly. The rates of the spikes vary depending on the intensity and the frequency of the incident sound. Each fibre fires most at a given frequency (called the characteristic frequency) like the spatially

represented characteristic frequency in the cochlear BM which facilitates transfer of the tonotopicity to the nerve from the cochlea. For example, fibres from the base of the cochlea will lead to increased firing of the fibres with a high characteristic frequency.

The discharges of the fibres to low-frequency sounds occur at particular times, in other words, there is a phase locking mechanism which occurs up to 5 kHz. Phase locking is important to convey temporal information of the incoming signal.A two-tone suppression model is also present in auditory nerve function which dictates that a second stimulus close to the characteristic frequency of the first one will suppress the first, facilitating the representation of complex and multiple stimuli in the nerve which is a non-linear function. Intensity coding also occurs in the nerve fibres. Nerve fibres possess spontaneous firing activity without sound stimulation. Fibres with high spontaneous firing rates have a low threshold for intensity while fibres with intermediate and low spontaneous firing rates have a high threshold. Stimulus intensity thus influences the firing rate of the nerve fibre: the higher the stimulus intensity, the higher the firing rate, with a saturation point where it plateaus off which is important for adaptation.

The auditory nerve is the beginning of central processing of the acoustic stimulus delivered from the periphery. This is in the form of auditory

nerve adaptation which entails a spike frequency adaptation where the spike in response to a stimulus quickly adapts and reaches a plateau that is more pronounced in high-frequency fibres than the lower ones. Adaptation, which is found in neurons, is crucial in the auditory context as it provides vital cues to the brain regarding timing of the signal. Adaptation gets progressively complex and sophisticated in the brainstem and the cortex. The auditory nerve can be affected by a variety of disease processes which include pressure effects by space- occupying lesions such as a VS; the demyelination processes such as in multiple sclerosis; granulomatous lesions; head trauma; viral illnesses; congenital dysplasias and autoimmune disorders.

Essentially, the tuning curve and the phase-locking properties are affected and lead to a failure in encoding time, frequency and intensity information. This is a classical neural deafness. ABRs are a robust way of measuring auditory nerve function and may be used for prognosticating a demyelinating lesion. The condition auditory neuropathy spectrum disorder (ANSD) is defined as a disorder with abnormal auditory brainstem morphology but preserved cochlear OHC function. Structurally, the nerve is unremarkable, especially on imaging, and most likely the damage is at the molecular level precipitated by a few known risk factors such as hypoxia, hyperbilirubinemia and prematurity.

COCHLEAR EFFERENTS:

The cochlear efferent system consists of projections from both the lateral olive and the medial olivary complex which synapse mostly with type 1 spiral ganglion cells and type 2 spiral ganglion cells respectively and thus connect to both the IHCs and the OHCs. The efferent fibres are carried by the inferior vestibular nerve and meet at the anastomosis of Oort through the saccular branch of the nerve to join up with the cochlear nerve. The ratio of efferent to afferent fibres in the OHC is 1 : 2 whereas those in the IHCs is 1 : 7, suggesting that the biological amplifiers are the main substrates of efferent function which in turn inherently implies that the signal modulation is primarily a function of the OHC.

The medial system innervates both ears while the lateral system supplies only the ipsilateral cochlea. Both project to the different parts of the ventral cochlear nucleus. The cochlear efferents serve an important function by virtue of their modulation of inhibitory and excitatory neurotransmitter release in the cochlear processes thereby offering cochlear protection from loud noise. The activation of the efferent system modifies frequency specific gain at the tonotopic BM by acting on the voltage-dependent OHC motility and attempts to linerarize the signal with a damping effect. Other functions include fine perception of the acoustic signal for localization, improving the signal-to-noise ratio and supporting

adaptation and frequency selectivity. Their function may be compromised in acoustic trauma, ototoxicity, tinnitus and ANSD.The medial olivocochlear bundle function can be measured by contralateral suppression of otoacoustic emissions.

ASCENDING AUDITORY PATHWAY:

The very low frequency fibers branch to form two end bulbs. These end bulbs contain large number of neurotransmitter vesicles and helps in rapid transmission of signal. AVCN responsible for the original frequency selectivity and sensitivity or cochlear response. The cells in these nucleus analyse the pattern of sound and determine the intensity. PVCN receive input of wide range of frequency and is responsible for the precise time of arrival of sound. The signal is sent to the motor nucleus in the brain stem and midbrain and involved in acoustic startle response. Complex response in the DCN determines what sounds are.

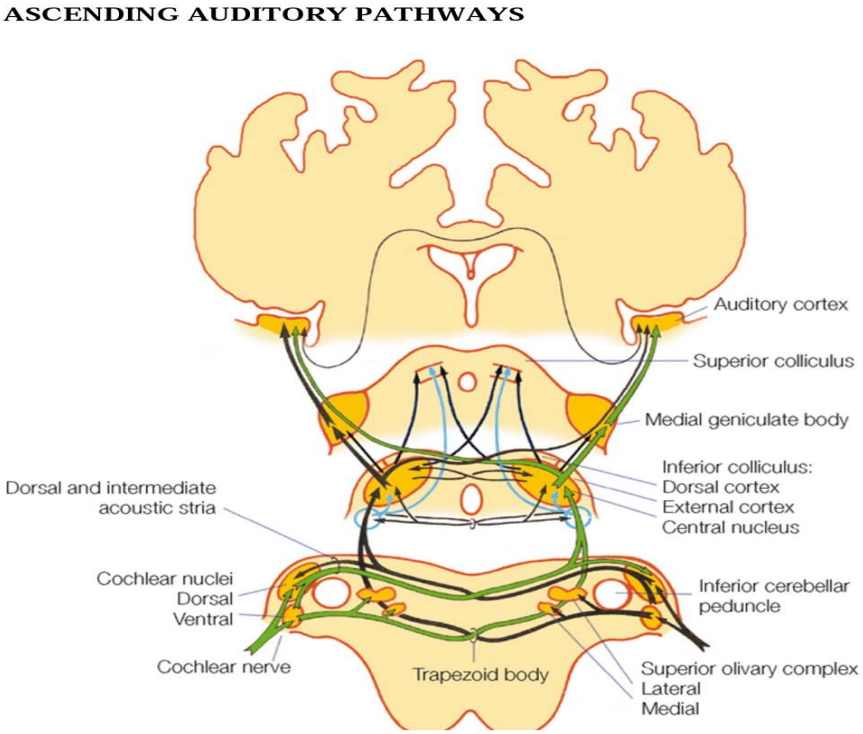
All the auditory pathway leaves the cochlear nuclear complex and divides in to dorsal and ventral pathways. The dorsal pathway project directly to inferior colliculus and ventral pathway projects to ipsilateral and contra lateral superior olivary complex. This makes binaural comparison of sound possible. SOC helps in sound localization. Superior olivary complex contains 'S' shaped lateral olivary nucleus, medial olivary nucleus, medial nucleus of the trapezoid body together with

smaller periolivary nucleus. Medial olivary nucleus helps in detecting inter aural time differences. The 'S' shaped nuclei receives an excitatory input from the ipsilateral cochlear nuclei and inhibitory from the contra lateral cochlear nuclei. This helps in detection of difference of sound intensity. Through the lateral lemniscus the input from the brain stem auditory nuclei is projected to inferior colliculi.

The two pathways emerge from the cochlear nuclear complex and join in the inferior colliculus and further analysis is made. The inferior colliculi contain a central nucleus and outer region composed of dorsal cortex and external lateral cortex. The external portion receives information from cerebral cortex. A tonotopic map is made in the inferior colliculi, by arranging high frequency bands towards the midline of the brain and low frequency bands towards outside. This map is the basis for recognizing patterns in sound and sound localizations. Inferior colliculus also involved in motor response like controlling middle ear muscle, turning head or moving eye in response to sound. The thalamus receives information from the inferior colliculi. Thalamus has medial geniculate body, posterior nucleus and reticular nucleus, which are involved in auditory function.

The ventral division is organized tonotopically into low frequency layers and receives input from the central nucleus of inferior colliculus.

The dorsal division is not tonotopically organized and medial division receives multimodal inputs. From the ventral division of the medial geniculate nucleus, fibers project to Brodmann area 41 within the lateral fissure of the temporal lobe and dorsal division project to non primary areas around A1. Auditory area also organized into ISO frequency layers, arranged tonotopically, the low frequency sound in the rostral end and high frequency in the caudal end. Most cells in A1 respond to binaural stimulation. The main function of primary auditory cortex is sound localization. Areas around A1 have complex response and it detects specific delays and simultaneous occurrence of harmonically related frequencies.



DESCENDING PATHWAYS:

The descending pathway may participate in attention level and anticipation of signals. The major one is olivocochlear feedback loop which originate in SOC and projects back to cochlea. It projects to outer hair cells and is called medial efferent system. It helps in suppression of outer hair cell mobility to make less sensitive and provides protection from loud sounds. The lateral efferent system from lateral superior olivary complex supply inner hair cells which helps in sound localization and binaural comparison.

PHYSIOLOGY OF SOUND TRANSMISSION :

Sound is transmitted to the inner ear through the middle ear ossicles, When the sound waves strike the tympanic membrane, it increases tympanic membrane pressure in a frequency sensitive way. An efficient middle ear impedance transformer will change the low pressure high displacement vibration of the sound waves into low displacement and high pressure vibrations. A compression wave is developed in the inner ear fluid due to the vibration of the stapes footplate, which travels across the scala vestibuli, around the helicotrema, and out across the scala tympani toward the round window.

An inward motion of the stapes causes an outward motion of the round window. This compression wave travels across the scala vestibule. The pressure in the scala vestibuli is higher than the pressure in the scala tympani. This set up a pressure gradient, which causes the cochlear partition to vibrate .A travelling wave is set up in the basilar membrane. This movement is from base to apex .A shearing motion is developed between reticular lamina and tectorial membrane. This shearing force causes a deflection of the hair cell stereocilia. This reaches maximum at a particular place of the basilar membrane and decays. Molecular structure at that location of the basilar membrane determines the characteristic frequency. The cochlea is tuned for higher frequency upto 20kHz. This tonotopic gradient is manifested in hair cell height also.

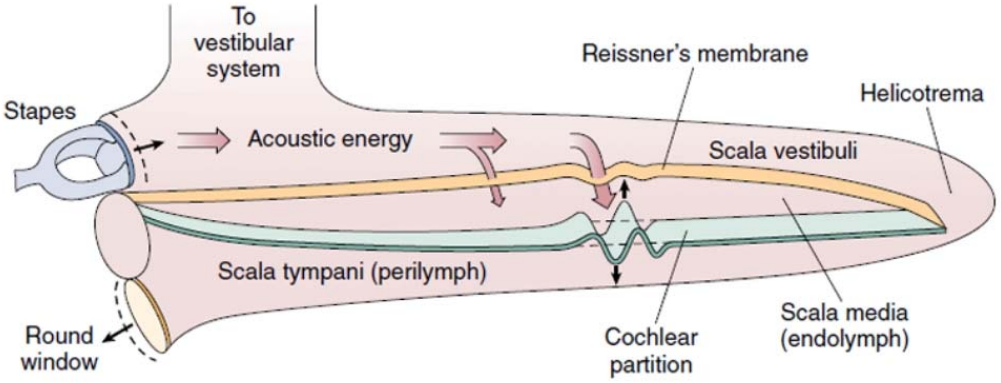


Fig 4.4, schematic presentation of sound conduction in inner ear.

TRANSDUCTION BY HAIR CELLS:

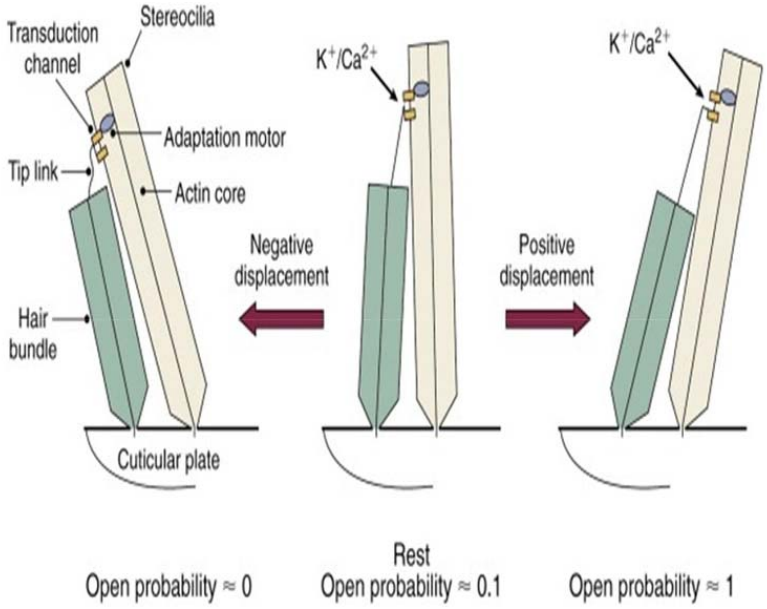
The stereocilia on the hair cells are rigid and braced together with cross links, so they move as a stiff bundle. When stereocilia is deflected in the direction of tallest stereocilia, the tip links are stretched and result in the opening of ion channels. Ca2+ ions play an important role in the opening of ion channels. The relative motion between tectorial membrane and recticular lamina produce a stimulus which is coupled to stereocilia. This result in opening of ionic channels of stereocilia. Na+, K+ and Ca2+ will enter the cell. The apical surface which face the endolymph has high positive potential of +80 milli volt and high K+ ion concentrations. Inside the cell negative intracellular potential of - 45 milli volt for inner hair cell and - 70mv for outer hair cells is maintained.

These potentials combine to give a total of 125mv for inner hair cells and 150 mv for outer hair cells of potential drop across the channels. The K+ ions from the endolymph enters the cell and makes the cell more positive inside and when channels shut cells become more negative during opposite phase of sound wave. K+ is the main ion involved in transducer mechanism. The main energy comes from the striavascularis by ion pumping. All these mechanism produce a receptor

potential. And neuro transmitters are released from the basolateral membrane of inner hair cell

INNER HAIR CELLS AND ELECTRICAL TRANSDUCTION:

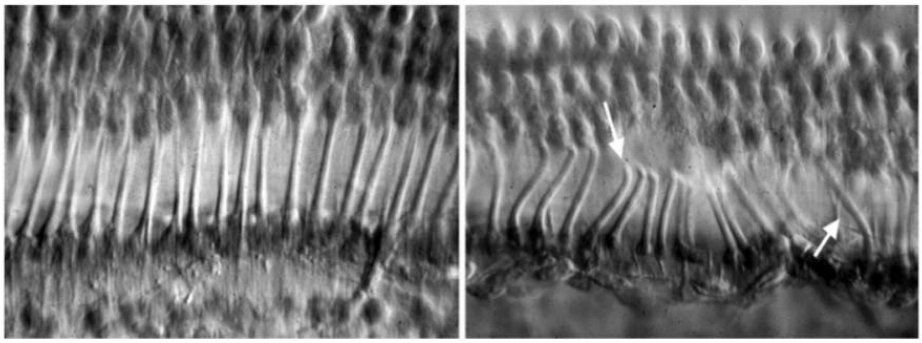
The inner hair cells convert mechanical stimulation into electric signals which is transported to brain. This transduction occurs near the tips of steriocilia. Because of shearing motion between adjacent stereocilia, it is transmitted to all hair cells. This leads to opening of the channel protein upon stimulation and leads to depolarization of the cell. This is followed by hyper polarization of the cell where stereocilia are deflected to shorter stereocilia. In these two processes, rapid channel closure and slow adaptation occurs. IHC detects movement of basilar membrane and responds to velocity changes. The basolateral wall of IHC act as a capacitor.



ELECTRICAL RESPONSE OF OUTER HAIR CELL:

Outer hair cells are mainly for amplification and sharp tuning of basilar membrane. It is for amplification of sounds at a low pressure level. Outer hair cells act by changing it's length. It will contract upon stimulation which leads to depolarization and pull the basilar membrane. It elongates its length which leads to hyperpolarisation. Prestin is the motor protein in the outer hair cells, responsible for the actions of OHC. On depolarization, the anions dissociate and the surface area decreases leading to contraction. Similarly on hyperpolarisation surface area increases

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 deiters cells

ELECTRICAL RESPONSE OF THE COCHLEA :

In response to acoustic stimuli, electric potential can be recorded from the cochlea. Cochlear microphonic potentials represent the mass effects of the transducer currents flowing through outer hair cells. It is an AC response.

SUMMATING POTENTIAL :

It may be either positive or negative, depends on the stimulus. It is the distortion component of the outer hair cell response and small contribution from inner hair cells also. It reaches maximum amplitude after the onset of stimulus.

NEURAL POTENTIAL:

It is produced at the onset of stimulus from the massed action potential of auditory nerve.

PHYSIOLOGY OF NOISE :

The term noise is defined as an undesirable sound. It is excessive sound that has potential to harm hearing. It is defined as superfluous, unwanted random sound energy unrelated to sounds being measured, amplified or otherwise studied. Noise is usually a periodic sequence of vibration. Physically, physiologically, psychologically the meaning of noise varies. Physically it is a complex sound having little periodicity that can be measured on its characteristics analyzed. And the physical attributes of noise include frequency, sound pressure, particle velocity, sound intensity, sound energy density etc. Physiologically it is defined as a signal that has no information and intensity varies. Psychologically it is any sound which is unpleasurable and unwanted. The Psychological attribute includes pitch, loudness, timber intelligibility, annoyance. The physiological measurable attributes include potential to damage hearing.

The frequency of noise is measured in Hertz (Hz), intensity in sound pressure level (SPL) and expressed in decibel (dB). Since it is expressed in decibels it reduces the wide ranging of values to a manageable numbers.

Noise may be continuous, fluctuating, intermittent or impulsive. Continuous noise may be relatively constant. Fluctuating noise increases in level over time. Intermittent noise is interrupted for varying time. Impulsive or input noise may be caused by explosions, more common in military environment.

It is characterized by short lasting rapidly changing wave fronts and followed by small reverberations and echoes. The amount of noise, sound pressure level is measured by sound level meter in decibel (dB) using a frequency weighting formula called A-Scale. A-Weighted measurements are preferred in calculating noise exposure. It reduces the sensitivity of the sound level meter in both extreme ranges of audible spectrum. A standard sound level meter has electronic network and measures noise magnitude automatically in dB.There are four types of specification for sound level meter type 0, 1, 2, 3. Type 0 sound level meter is used as a laboratory reference standard, type 1 mainly for laboratory and also field where acoustical environment in closely controlled. Type 2 is for general field application and type 3 is for field noise survey application. The frequency range responds for all types from 10Hz to 20000Hz. Sound level meter measures noise according to equal energy principle.

NOISE INDUCED HEARING LOSS:

Noise induced hearing loss is the one of the common causes of permanent hearing impairment. Millions of individuals worldwide have noise-induced hearing loss (NIHL), resulting in a reduced quality of life because of social isolation, and impaired communication with family members, co-workers, and friends. The costs in terms of compensation and early retirement payments for work-related NIHL are immense.

The aim of the study is to illustrate the effects of occupational noise exposure on hearing and to improve evaluation helping in early detection of hearing loss in high frequency tones before affecting speech frequency using a high frequency pure tone audiometer. Noise has obviously a serious impact on hearing and may cause hearing impairment in terms of hearing loss and tinnitus. The working environment is a major factor for noise-induced hearing loss and noise is the source of most prevalent occupational diseases in many countries.

NATURE OF HEARING LOSS :

Noise is an undesirable sound. Noise may be continuous, fluctuating, intermittent or impulsive. Continuous means a constant steady noise. Fluctuating means noise varies over time, while intermittent noise are interrupted over time periods Depending on the time period of

exposure, hearing loss may be temporary or permanent. Temporary hearing loss is a reversible loss i.e. temporary threshold shift. It may be accompanied by tinnitus, dysplacusis. Recovery may range from minute to hours. Magnitude of TTS depends on the intensity, frequency and temporal pattern of noise. High frequency sounds are more dangerous than low frequency sounds. If the ear is re-exposed to loud noise before recovery, Permanent Threshold Shift(PTS) will occur. PTS is due to the structural damage of cellular system of cochlea. Repeated episode of TTS leads to PTS. In PTS focal loss of hair cells and degeneration of nerve

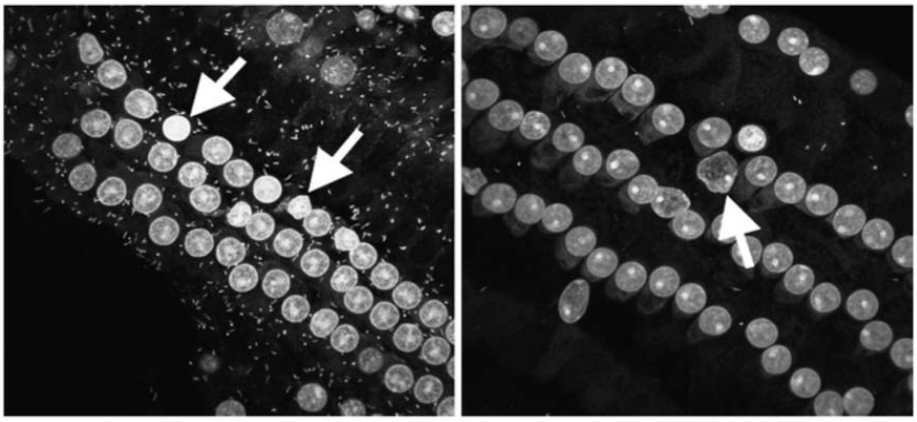
NIHL occurs due to continuous chronic exposure of sound. Occupational noise is always sensorineural and affects mainly OHC. Typically the threshold shift occurs bilaterally. It produces characteristic notching at 4 kHz with high frequency hearing loss. NIHL produce maximum 75dB HL at high frequencies and 40dB at low frequencies. Hearing loss is initially at higher frequency. Once noise exposure is discontinued hearing loss does not progress. The development of hearing loss mainly starts in the high frequency level and gradually progress to middle and low frequencies. Regular exposure to low intense noise also increases the hearing threshold. Asymmetric pattern of hearing loss is also noticed in some persons involved in sports like shooting etc.

PATTERN AND MECHANISM OF NOISE INDUCED COCHLEAR PATHOLOGY:

Cochlea is a highly energy consumptive biological system. Each cellular system of cochlea is vulnerable to noise exposure. Basilar membrane is 200 times stiffer at the base. It results in an impedance gradient from apex to base. So high frequency creates a maximum vibration at base and low frequency at the apex. The damage to pillar cells leads to abrupt change in the basilar membrane impedance gradient. It may lead to cell death. Outer hair cells are most vulnerable to noise exposure. Noise exposure distort the stereocilia of OHCs. Brownell showed that OHC movement is due to the synchronized depolarization of stereocilia .With complete loss of OHC 40 to 50 db HL can occur. The K+ ions are circulated out of hair cells through the supporting cells namely Claudius cells and Henson cells. Eventually the fibrocytes in spiral ligament transfer the potassium ions into striavascularis.

Type II fibrocytes are the most vulnerable to noise exposure and its loss closely corresponds to spectrum of noise exposure. The stereocilia may be shortened or break on noise exposure.IHC are more resistant to noise exposure. When complete loss of OHC in a region leads to loss of IHC and VIII nerve fibers. Temporary effect of noise can be seen in IHC and VIII nerve 6 Because of high rate of synaptic

activity VIIIth nerve fibers swells up due to intense noise exposure. Failure to recycle glutamate accumulated in the dendrite terminal cause excitotoxic effect leads to swelling of postsynaptic cell bodies and dendrites 7 Phase-contrast views of outer pillar cells. Left panel shows normal anatomy.Right panel is after exposure to 50–155 dB SPL impulses. Notice (arrows) the detachment at the level of the cuticular plate



Examples of apoptotic cell death (left) and necrotic cell death (right).

NOISE AS A STRESSOR TO THE COCHLEA:

Cochlea function at a high metabolic rate and the main energy source is stria vascularis. It contains large number of mitochondria which maintain ionicity and polarity of endolymph by extruding k+ constantly. The reactive oxygen species (ROS) in the cochlea are produced by mitochondria. Mitochondria consumes 98% of Oxygen and converts ADP to ATP. 1-2% O2 is converted to superoxide. Because of high

level noise exposure high level of ROS is generated. These are two factors for high level of ROS generation. First cochlear metabolism increase at a faster rate. Secondarily cochlear blood flow decreases, ischemia develops leading to shortage of O2 for mitochondrial functions with resultant superoxide production. When reperfusion occurs blood flow increases providing a rich O2 supply leading to a burst of superoxide generation. Finally cells are destroyed with extrusion of cellular contents into extracellular matrix. Reactivity continues for several days due to which hair cells continue to die after exposure to loud noise.

PATHWAYS OF SENORY CELL DEATH:

Noise exposure produces both apoptosis and necrotic cell death. Following noise exposure the level of phosphates, calcineurin and Bclxl/Bcl-2 associated death promoter level increases. There is a short latency before apoptosis starts following noise exposure and once started apoptosis continues. It may be converted to necrosis because of lack of energy to finish apoptosis. The apoptosis starts from thecentre to basal end of cochlea and is driven by lipid peroxidation19. Impulse noise produce damage to OHC by ROS production. OHC are shortened and their nuclei migrate from basal pole to middle of the cell and finally it shrinks.

COCHLEAR RESPONSE TO STRESS FROM NOISE:

Cochlea has several defense mechanisms to protect it from high level noise. It produces heat shock proteins. It also increases the activities of antioxidant system namely glutathione reductase, catalase, gamma glutamyl cysteine synthatase. In short, noise damage cochlea and causes hearing loss up to 50dB. OHC are more vulnerable to noise exposure. Noise damage cellular system of cochlea by production of ROS which in turn initiates death by apoptosis and necrosis and this continues for few days after noise exposure.

CLINICAL FEATURES:

The middle aged male people with complaints of tinnitus with or without hyperaccusis In the early stage patients present with a history of hearing difficulty in the presence of background noise. They usually describe lack of clarity to speech. Tinnitus is an early symptom, especially post exposure tinnitus. Hyperaccusis may be found in 40% of noise exposed individuals. With further progression of disease, patients may complain of obvious hearing loss. Noise induced hearing loss is more common in men and it leads to social isolation. There may be embarrassment, loss of confidence, anxiety, and frank depression.

MATERIALS AND METHODS

STUDY DESIGN : A Cross Sectional Study Of Hearing Loss In Auto Drivers

SETTINGS : Department of ENT and Head and Neck Surgery, Govt Stanley Medical College and Hospital, Chennai

METHODOLOGY :Otoscopy, tuning fork tests and Pure Tone Audiometry (PTA) were performed in 100 apparently healthy auto drivers aged from 21 to 55

STUDY PERIOD: april2019 to july2019

INCLUSION CRITERIA :

Age: above 21yrs

Sex: Male

Apparently healthy Auto drivers working in different parts of Chennai city for a continous period of three years or more

EXCLUSION CRITERIA:

Age: below 21 or above 60 yrs

Sex :female

Subjects suffering from pre existing ear disease such as chronic suppurative otitis media, otitis media with effusion, otosclerosis, throat infection, those on ototoxic drugs, suffering from any systemic disease such as hypertension or diabetes mellitus were excluded.

METHODOLOGY:

The study was conducted in the department of Otorhinolaryngology. Government Stanley medical college and Hospital Chennai 600001. A total of 100 subjects were examined within the age group 21 to 55 years. All of them working for more than 3 Years. clinical history noted.

All persons were first examined by Otoscopy and ruled out of any external ear and middle ear pathology. All cases subjected to pure tone audiometry.

From 125 to 8 kHz's the response were based on subject activation of Hand held response buttons. Steps of 5dB were used in obtaining results. The results were expressed in dB HL. Also bone conduction from 250 to 4 kHz.

RESULTS AND ANALYSIS

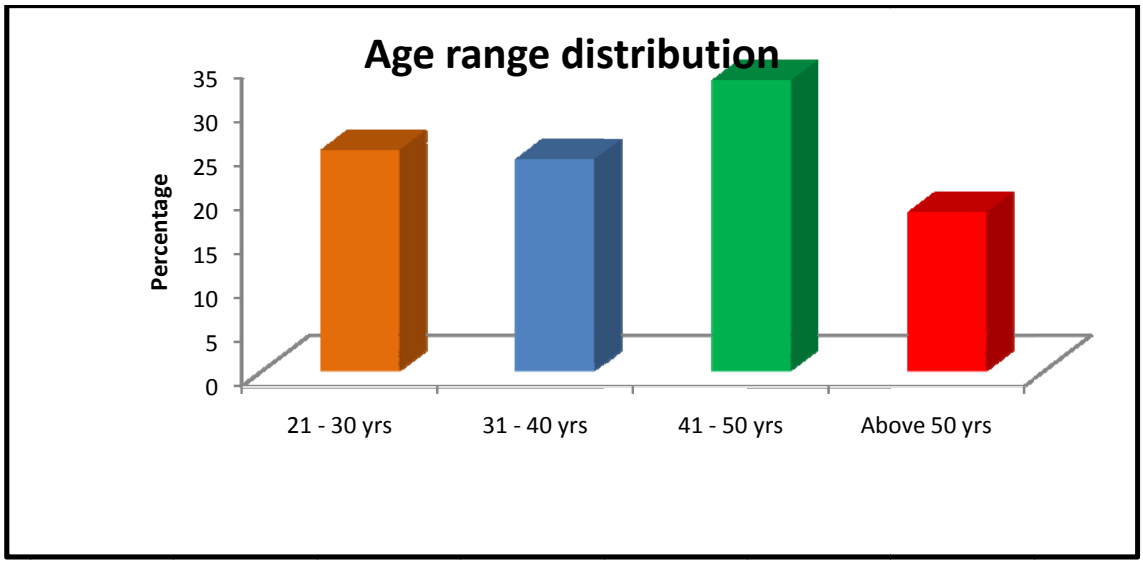
The data obtained from study conducted in auto drivers in dept of otorhinolaryngology, govt.stanley medical college were analysed

The following parameters were measured-

- Age distribution
- years of work
- daily work duration
- Normal PTA with high frequency hearing loss
- Unilateral or bilateral noise induced hearing loss

AGE DISTRIBUTION:

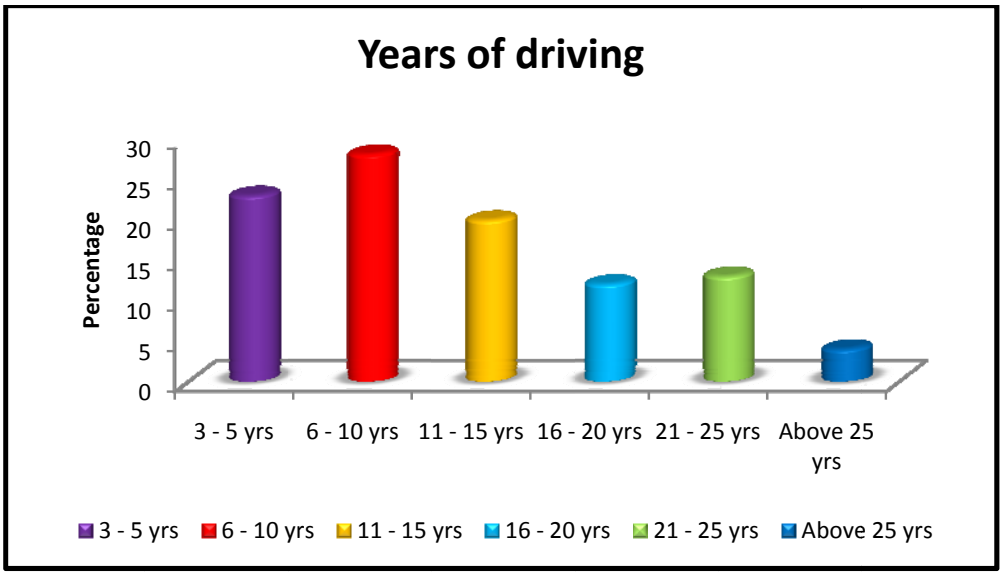
Age	Frequency	Percent
21 - 30 yrs	25	25.0
31 - 40 yrs	24	24.0
41 - 50 yrs	33	33.0
Above 50 yrs	18	18.0
Total	100	100.0



In this study 33% of subjects belongs to age group of 41-50 yrs, 18% were above 50 yrs

YEARS OF DRIVING:

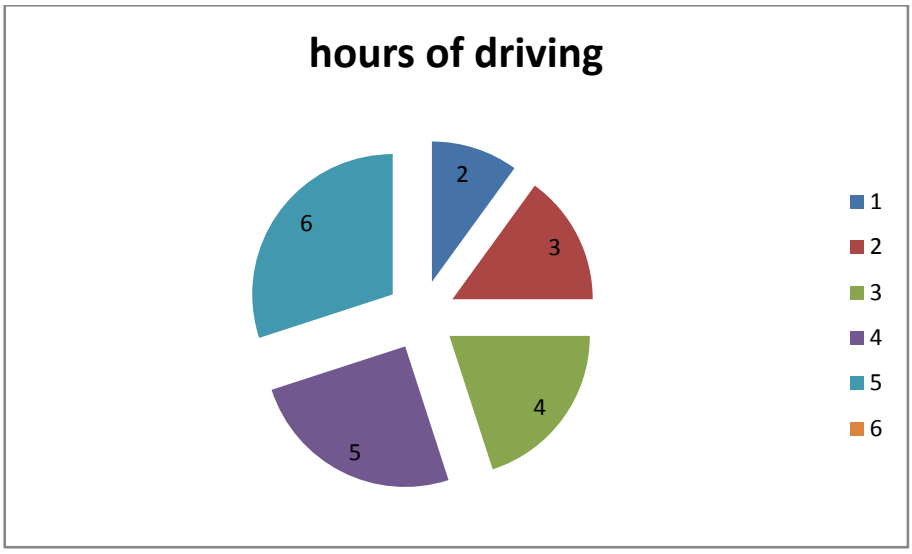
years of driving	Frequency	Percent
3 - 5 yrs	23	23.0
6 - 10 yrs	28	28.0
11 - 15 yrs	20	20.0
16 - 20 yrs	12	12.0
21 - 25 yrs	13	13.0
Above 25 yrs	4	4.0
Total	100	100.0



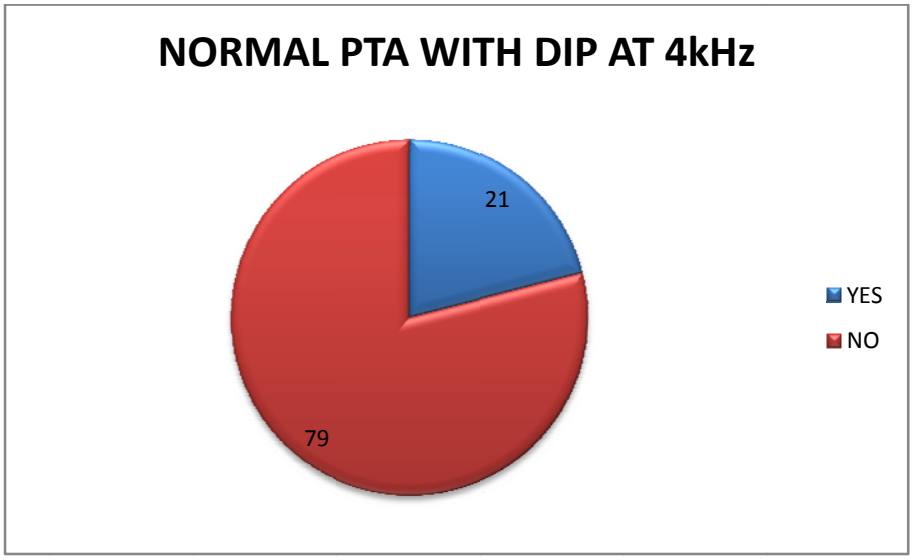
As for as years of driving is concerned 28% of subjects fall in to 6-10 yrs of driving, 23% falls in to 3-5 yrs, only 4% falls in to above 25 yrs of driving

DAILY HOURS OF DRIVING:

HOURS OF DRIVING	FREQUENCY
2	7
3	26
4	30
5	26
6	11



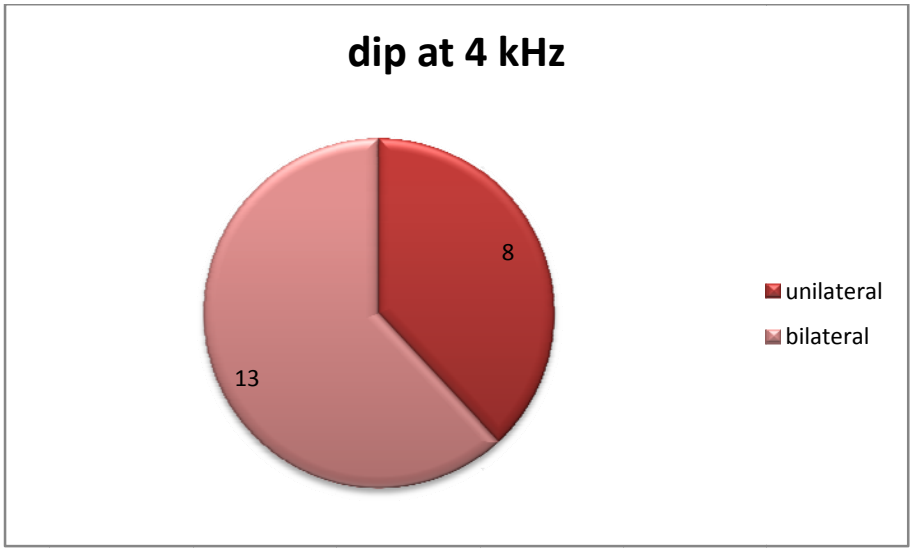
NORMAL PTA WITH DIP AT 4 kHz.:



Out of 100 subjects screened by PTA 21 were found to have dip at 4 kHz

UNILATERAL OR BILATERAL NOISE INDUCED HEARING LOSS:

Out of 21subjects who had dip at 4 kHz , unilateral dip was found in 8 subjects ,bilateral dip was found in 13 subjects



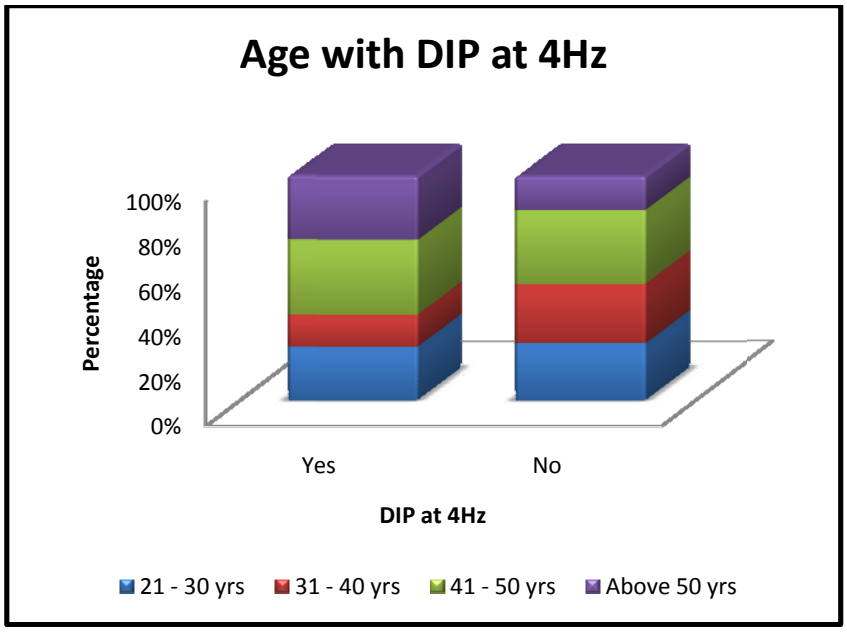
AGE DISTRIBUTION:

Out of 21 subjects who had NIHL, 5(23.8%) were found in age group of 21-30 yrs

Age * DIP at 4hz							
			DIP at 4hz		Total	x 2 - value	P-value
			Yes	No			
Age	21 - 30 yrs	Count	5	20	25	2.712	0.438
		%	23.8%	25.3%	25.0%		
	31 - 40 yrs	Count	3	21	24		
		%	14.3%	26.6%	24.0%		
	41 - 50 yrs	Count	7	26	33		
		%	33.3%	32.9%	33.0%		
	Above 50 yrs	Count	6	12	18		
		%	28.6%	15.2%	18.0%		
Total		Count	21	79	100		
		%	100.0 %	100.0 %	100.0 %		

No Statistical Significance at P>0.05 level

3(14.3%) were found in age group of 31-40 yrs,7(33.3%) falls in to age group of 41-50 yrs.6(8%) were above 50 yrs.

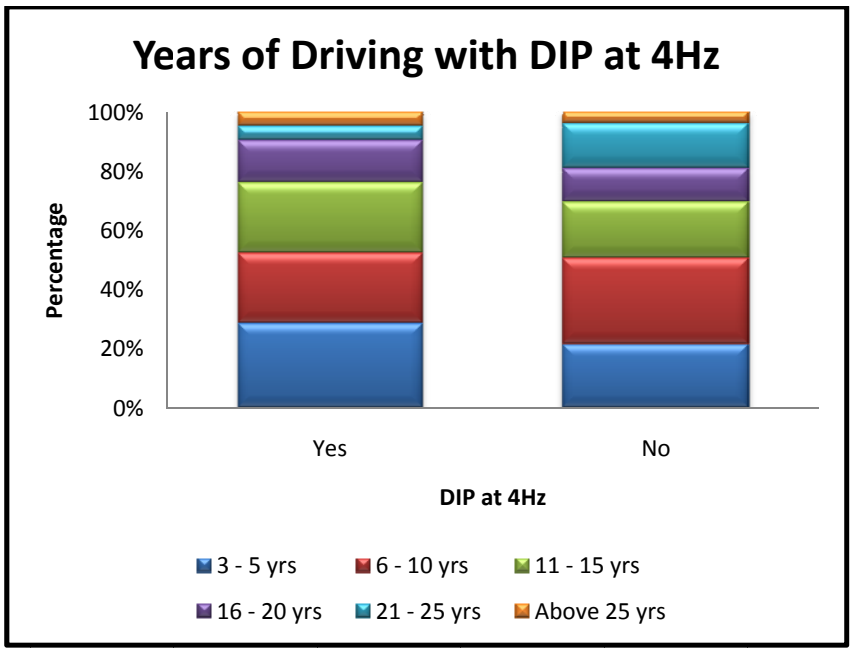


DURATION OF EXPOSURE:

years of driving * DIP at 4hz							
			DIP at 4hz		Total	χ^2 – value	P-value
			Yes	No			
years of driving	3 - 5 yrs	Count	4	17	23	2.26	0.032#
		%	19.04%	21.5%	23.0%		
	6 - 10 yrs	Count	5	23	28		
		%	23.8%	29.1%	28.0%		
	11 - 15 yrs	Count	7	15	20		
		%	33.33%	19.0%	20.0%		
	16 - 20 yrs	Count	3	9	12		
		%	14.3%	11.4%	12.0%		
	21 - 25 yrs	Count	1	12	13		
		%	4.8%	15.2%	13.0%		
	Above 25 yrs	Count	1	3	4		
		%	4.8%	3.8%	4.0%		
Total		Count	21	79	100		
		%	100.0%	100.0%	100.0%		

Statistically significant if # P <0.05

As with duration of exposure maximum of 7(33%) were found 11-15 yrs of exposure, 5 (23.8%) were found in exposure group of 6-10yrs

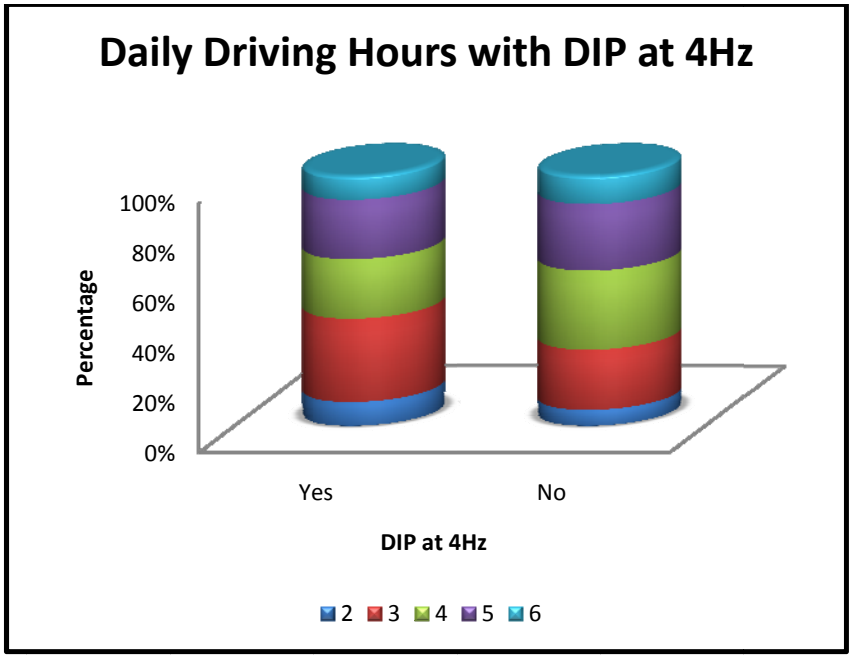


DAILY HOURS OF DRVING:

daily driving hours * DIP at 4hz							
			DIP at 4hz		Total	χ^2 - value	P-value
			Yes	No			
daily driving hours	2	Count	2	5	7	1.233	0.873
		%	9.5%	6.3%	7.0%		
	3	Count	7	19	26		
		%	33.3%	24.1%	26.0%		
	4	Count	5	25	30		
		%	23.8%	31.6%	30.0%		
	5	Count	5	21	26		
		%	23.8%	26.6%	26.0%		
	6	Count	2	9	11		
		%	9.5%	11.4%	11.0%		
Total		Count	21	79	100		
		%	100.0%	100.0%	100.0%		

No Statistical Significance at P>0.05 level

While compiling daily hours of driving ,maximum subjects of 7 with NIHL were found in group of 3 hrs,followed by 5 in group of 4 hrs



DISCUSSION

A cross sectional study to detect noise induced hearing loss in auto drivers working in different parts of Chennai was conducted in department of oto rhinolaryngology govt.stanley medical college and hospital. No one in study population have any complaints of hard of hearing. All members of study were subjected to routine PTA

PTA.

Total No of Cases : 100

Normal- PTA : 79

PTA with dip at 4KHz: 21

Among 100 subjects 79 were having normal pure tone audiometry ,21 have PTA dip at 4KHz which signifies noise induced hearing loss

AGE DISTRIBUTION:

Out of 21 subjects who had NIHL, 5(23.8%) were found in age group of 21-30 yrs. 3(14.3%) were found in age group of 31-40 yrs,7(33.3%) falls in to age group of 41-50 yrs.6(28%) were above 50 yrs.

DURATION OF EXPOSURE:

we divide the Subjects in to following age group

3-5 yrs

6-10 yrs

11-15 yrs

16-20 yrs

21-25 yrs

out of which subjects who have worked for 6 and more years have more considerable NIHL. With a P value of 0.032 which is statistically significant

HOURS OF DRIVING EXPOSURE PER DAY: in this category the group falls in to

- 2 hrs
- 3 hrs
- 4 hrs
- 5 hrs
- 6 hrs

Subjects who have worked for 3 hrs or more had considerable NIHL

UNILATERAL/ BILATERAL HEARING LOSS:

Out of 21 subjects who had NIHL 13 were found have bilateral disease

INTERPRETATION OF RESULTS:

A cross sectional study to detect noise induced hearing loss in auto drivers working in different parts of Chennai was conducted in department of oto rhinolaryngology govt.stanley medical college and hospital

PREVALENCE OF HEARING LOSS:

The prevalence of hearing loss in auto drivers in this study was 21%

This study was comparable to following studies:

A study conducted in bus drivers in brazil year 2012 by andrea cintra lopes and collegues showed a prevalence of 22% which was similar to my study

In other study conducted in bus and truck drivers of Mazandaran province in north Iran in year 2016 a preavelence of 32% was found

Prevalence of Noise Induced Hearing Loss in Indian Air Force Personnel by nair et al 2009 showed a prevalence of 22.6%

Effect of age and duration of driving on hearing status of Indian agricultural tractor drivers by abhijit khadatkar et al showsThe mean hearing threshold values at different audiometric frequencies (0.125–8 kHz) for the left and right ears are higher for higher age group tractor drivers having more driving experience. This shows that with the increase

in age group and increase of driving experience, which was similar to this study

Prevalence of noise induced hearing loss among vehicle drivers at Bandar Abbas freight terminal, south of Iran by Leila Rezaei, Vali Alipour Audiometry test was done Approximately 52% of drivers studied had a degree of hearing loss.

Survey of Noise-Induced Hearing Loss and Health in Professional Drivers by Nazanin Izadi , Mahdi Sadeghi , Maryam Saraie et al showed similar association with this study

Noise-Induced Hearing Loss (NIHL): literature review with a focus on occupational medicine Mirella Melo Metidieri,[1] Hugo Fernandes Santos Rodrigue.had similar results as of this study

A study on "Temporary threshold shift in military pilots measured using conventional and extended high-frequency audiometry after one flight by Kuronen P, Sorri MJ et al Finnish Air Force Headquarters, Finland International Journal of Audiology 2003 conducted study on 51 Finnish Air Force military personnel as subjects using HFA and conventional pure tone audiometry. A statistically significant temporary threshold shifts (TTS) at several frequencies and with all aircraft types involved was noted"

CONCLUSION

From this study the following conclusions were made

The prevalence of hearing loss was assessed based on audiogram findings by pure tone average in both Right and Left ears and early hearing loss was assessed using a dip in 4khz in PTA.

The prevalence of hearing loss in this study among the auto drivers was nearly 21% age ,years of exposure , daily workers hours , were taken in to account of which years of exposure(duration of work) was found to have relation with NIHL which is statistically significant

SUGGESTIONS FROM THIS STUDY:

Noise induced hearing loss is one the leading cause of morbidity in india with relation to occupational health .auto drivers who are constantly exposed to environmental noise, are at more risk of developing NIHL by using ear muffs during driving autodrivers can be able to attenuate excessive noise pollution during driving yearly routine screening is necessary in this sub set of population for earlier prevention of NIHL

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ANNEXURES

ABBREVIATIONS

NIHL-NOISE INDUCED HEARING LOSS

PTA-PURE TONE AUDIOMETRY

IHC-INNER HAIR CELL

OHC-OUTER HAIR CELL

CN-COCHLEAR NUCLEI

AVCN-ANTEROVENTRAL COCHLEAR NUCLEI

PVCN-POSTERO VENTRAL COCHLEAR NUCLEI

SOC-SUPERIOR OLIVARY COMPLEX

OAE-OTOACOUSTIC EMISSION

HOH-HARD OF HEARING

WHO-WORLD HEALTH ORGANISATION

ROS-REACTIVE OXYGEN SPECIES

CASE SHEET PROFORMA

Name:

Age:

Sex:

Occupation:

Address:

Hours of driving per day:

Chief complaints & Duration:

History of present illness:

History of past illness:

Personal History:

Family History:

Treatment History:

General Examinations:

Systemic Examinations:

EXAMINATION OF EAR

RIGHT EAR	LEFT EAR
Pre auricular region	Pre auricular region
Pinna	Pinna
Post auricular region	Post auricular region
External auditory canal	External auditory canal
Tympanic membrane	Tympanic membrane
Tragal sign	Tragal sign
Three finger test Middle finger Index finger Thumb	Three finger test Middle finger Index finger Thumb
Fistula test	Fistula test
Tuning fork test Rinne Weber Absolute Bone Conduction test	Tuning fork test Rinne Weber Absolute Bone Conduction test
Facial nerve function tests	Facial nerve function tests
Vestibular function tests	Vestibular function tests

EXAMINATION OF NOSE:

External contour of the nose:

Root

Dorsum

Supratip

Tip

Ala

Nasolabial groove

Naso alveolar groove

Naso maxillary groove

Vestibule of the nose

Anterior rhinoscopy

Medial wall

Nasal septum

Lateral wall

Inferior turbinate

Inferior meatus

Middle turbinate

Middle meatus

Floor

Nasal cavity

Colour of the Nasal mucosa

Posterior rhinoscopy

Air-way patency test:

Cold spatula test

Cotton wool test

Cottles test

Examination of Paranasal Sinuses

EXAMINATION OF THROAT

Examination of oral cavity:

Upper and lower lips

Angle of the Mouth

Gingivo labial sulcus

Gingivobuccalsulcus

Gums

Teeth

Hard palate

Anterior two third of tongue

Floor of the mouth

Vestibule of mouth

Cheek mucosa

Retromolar trigone

Examination of oropharynx

Bilateral Anterior pillar

Bilateral Tonsils

Bilateral Posterior pillar

Posterior pharyngeal wall

Soft palate

Uvula

INDIRECT LARYNGEAL MIRROR EXAMINATION:

EXAMINATION OF NECK:

PURE TONE AUDIOMETRY:

APPROVAL LETTER

GOVERNMENT STANLEY MEDICAL COLLEGE & HOSPITAL,CHENNAI -01

INSTITUTIONAL ETHICS COMMITTEE

TITLE OF THE WORK : A STUDY OF PREVALENCE OF NOISE INDUCED HEARING LOSS IN AUTO DRIVERS.

PRINCIPAL INVESTIGATOR : DR. S. SABARISH,
DESIGNATION : PG IN MS ENT,
DEPARTMENT : DEPARTMENT OF OTORHINOLARYNGOLOGY, GOVT. STANLEY MEDICAL COLLEGE.

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 03.04.2019 at the Council Hall, Stanley Medical College, Chennai-1 at 10am.

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

MEMBER SECRETARY,
IEC, SMC, CHENNAI

CONSENT FORM

STUDY TITLE : - "A Study Of Prevalence Of Noise Induced Hearing Loss In Auto Drivers"

I …… hereby give consent to participate in the study conducted by DR.S.SABARISH Post Graduate in dept of otorhinolaryngology Govt. Stanley Medical College, 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 consent for further investigations.

Signature / Thumb impression Place: date:

Of patient:

Patient Name and Address:

Signature of the Investigator:

Signature of the Guide:

MASTER CHART

serial no	age	sex	years of driving	daily driving hours	OTOSCOPY	Tuning fork test	PTA	DIP at 4hz
1	23	M	3	4	B/L TM INTACT	WNL	WNL	
2	47	M	7	3	B/L TM INTACT	WNL	WNL	+
3	45	M	14	5	B/L TM INTACT	WNL	WNL	
4	42	M	12	3	B/L TM INTACT	WNL	WNL	+
5	54	M	17	4	B/L TM INTACT	WNL	WNL	
6	43	M	20	3	B/L TM INTACT	WNL	WNL	+
7	46	M	12	5	B/L TM INTACT	WNL	WNL	
8	34	M	6	4	B/L TM INTACT	WNL	WNL	
9	25	M	4	3	B/L TM INTACT	WNL	WNL	
10	38	M	20	3	B/L TM INTACT	WNL	WNL	+
11	37	M	10	4	B/L TM INTACT	WNL	WNL	
12	42	M	11	4	B/L TM INTACT	WNL	WNL	+
13	41	M	14	3	B/L TM INTACT	WNL	WNL	
14	40	M	10	5	B/L TM INTACT	WNL	WNL	+
15	51	M	20	3	B/L TM INTACT	WNL	WNL	
16	32	M	5	4	B/L TM INTACT	WNL	WNL	+
17	53	M	12	4	B/L TM INTACT	WNL	WNL	
18	48	M	12	4	B/L TM INTACT	WNL	WNL	+
19	55	M	8	5	B/L TM INTACT	WNL	WNL	
20	22	M	3	3	B/L TM INTACT	WNL	WNL	
21	26	M	4	2	B/L TM INTACT	WNL	WNL	
22	30	M	5	3	B/L TM INTACT	WNL	WNL	+
23	45	M	7	3	B/L TM INTACT	WNL	WNL	
24	40	M	6	5	B/L TM INTACT	WNL	WNL	
25	28	M	7	4	B/L TM INTACT	WNL	WNL	
26	46	M	20	3	B/L TM INTACT	WNL	WNL	+
27	43	M	9	6	B/L TM INTACT	WNL	WNL	
28	35	M	7	3	B/L TM INTACT	WNL	WNL	
29	51	M	16	2	B/L TM INTACT	WNL	WNL	
30	52	M	17	5	B/L TM INTACT	WNL	B/L CHL	
31	49	M	15	3	B/L TM INTACT	WNL	WNL	
32	35	M	7	5	B/L TM INTACT	WNL	WNL	+
33	56	M	25	4	B/L TM INTACT	WNL	WNL	
34	36	M	8	4	B/L TM INTACT	WNL	WNL	+
35	37	M	13	5	B/L TM INTACT	WNL	WNL	
36	29	M	8	4	B/L TM INTACT	WNL	WNL	
37	28	M	6	5	B/L TM INTACT	WNL	WNL	
38	50	M	20	4	B/L TM INTACT	WNL	B/L MIXED HEARING LOS	
39	45	M	8	3	B/L TM INTACT	WNL	WNL	
40	30	M	8	5	B/L TM INTACT	WNL	WNL	+
41	27	M	4	4	B/L TM INTACT	WNL	WNL	
42	48	M	5	3	B/L TM INTACT	WNL	WNL	
43	38	M	12	6	B/L TM INTACT	WNL	WNL	
44	36	M	15	5	B/L TM INTACT	WNL	WNL	+
45	25	M	3	3	B/L TM INTACT	WNL	WNL	
46	54	M	20	5	B/L TM INTACT	WNL	WNL	+
47	49	M	5	3	B/L TM INTACT	WNL	WNL	
48	48	M	11	4	B/L TM INTACT	WNL	WNL	
49	48	M	25	6	B/L TM INTACT	WNL	WNL	
50	46	M	25	4	B/L TM INTACT	WNL	WNL	+
51	29	M	3	6	B/L TM INTACT	WNL	WNL	
52	38	M	3	3	B/L TM INTACT	WNL	WNL	
53	51	M	20	2	B/L TM INTACT	WNL	WNL	
54	47	M	15	3	B/L TM INTACT	WNL	WNL	+

55	49	M	20	5 B/L TM INTACT WNL	WNL	
56	39	M	18	3 B/L TM INTACT WNL	WNL	
57	28	M	3	5 B/L TM INTACT WNL	WNL	
58	35	M	8	4 B/L TM INTACT WNL	WNL	
59	35	M	8	2 B/L TM INTACT WNL	WNL	
60	53	M	15	6 B/L TM INTACT WNL	WNL	+
61	25	M	4	2 B/L TM INTACT WNL	WNL	
62	38	M	14	3 B/L TM INTACT WNL	WNL	+
63	31	M	6	3 B/L TM INTACT WNL	WNL	
64	45	M	15	4 B/L TM INTACT WNL	WNL	+
65	55	M	28	2 B/L TM INTACT WNL	WNL	
66	45	M	26	4 B/L TM INTACT WNL	WNL	+
67	25	M	4	3 B/L TM INTACT WNL	WNL	
68	40	M	10	5 B/L TM INTACT WNL	WNL	
69	34	M	9	4 B/L TM INTACT WNL	WNL	
70	54	M	22	5 B/L TM INTACT WNL	WNL	
71	50	M	15	3 B/L TM INTACT WNL	WNL	
72	24	M	4	6 B/L TM INTACT WNL	WNL	
73	53	M	10	4 B/L TM INTACT WNL	WNL	
74	35	M	9	2 B/L TM INTACT WNL	WNL	
75	45	M	22	4 B/L TM INTACT WNL	WNL	
76	55	M	31	5 B/L TM INTACT WNL	WNL	
77	53	M	25	4 B/L TM INTACT WNL	WNL	
78	23	M	3	5 B/L TM INTACT WNL	WNL	
79	46	M	21	4 B/L TM INTACT WNL	WNL	
80	51	M	29	5 B/L TM INTACT WNL	WNL	
81	43	M	13	6 B/L TM INTACT WNL	WNL	
82	22	M	3	5 B/L TM INTACT WNL	WNL	
83	24	M	3	5 B/L TM INTACT WNL	WNL	
84	47	M	21	4 B/L TM INTACT WNL	WNL	
85	50	M	25	4 B/L TM INTACT WNL	WNL	
86	48	M	21	5 B/L TM INTACT WNL	WNL	
87	37	M	5	4 B/L TM INTACT WNL	WNL	
88	30	M	3	5 B/L TM INTACT WNL	WNL	
89	52	M	21	6 B/L TM INTACT WNL	WNL	
90	21	M	3	6 B/L TM INTACT WNL	WNL	
91	40	M	8	4 B/L TM INTACT WNL	WNL	
92	46	M	22	5 B/L TM INTACT WNL	WNL	
93	28	M	7	6 B/L TM INTACT WNL	WNL	
94	35	M	10	3 B/L TM INTACT WNL	WNL	
95	23	M	4	3 B/L TM INTACT WNL	WNL	
96	27	M	6	5 B/L TM INTACT WNL	WNL	
97	50	M	12	6 B/L TM INTACT WNL	WNL	
98	51	M	21	4 B/L TM INTACT WNL	WNL	
99	30	M	9	5 B/L TM INTACT WNL	WNL	
100	42	M	15	4 B/L TM INTACT WNL	WNL	