DISSERTATION ON

A STUDY OF HEARING IMPAIRMENT AMONG PATIENTS WITH ESSENTIAL HYPERTENSION

Dissertation submitted to

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY

In the partial fulfillment of the regulations for the award of the degree of

M.S. IN OTORHINOLARYNGOLOGY

BRANCH-IV

REGISTRATION NUMBER : 220420101007

GOVERNMENT STANLEY MEDICAL COLLEGE

CHENNAI -600 001.

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI

MAY - 2022

CERTIFICATE

I certify that the dissertation titled **"A STUDY OF HEARING IMPAIRMENT AMONG PATIENTS WITH ESSENTIAL HYPERTENSION"** presented by **DR.VINEETH.S** for the degree of master of surgery (Otorhinolaryngology) in partial fulfillment of regulations of The Tamil Nadu Dr.M.G.R. Medical university, Chennai is the result of original research work undertaken by him during the academic year 2020- 2022, in the department of Otorhinolaryngology and Head & Neck Surgery, Government Stanley Medical College and Hospital, Chennai.

PROF.Dr.M.GOWRI SHANKAR
M.S., DNB., D.L.O
Head of the Department,
Department of Otorhinolaryngology,
Govt Stanley Medical College,
Chennai-01

PROF. Dr.P. BALAJI
MS.,FRCS.,Ph.D.,FCLS.,
THE DEAN
Govt Stanley Medical College
Chennai-01

Date :

Place: Chennai.-01

CERTIFICATE BY GUIDE

This is to certify that the Dissertation titled **"A STUDY OF HEARING IMPAIRMENT AMONG PATIENTS WITH ESSENTIAL HYPERTENSION"** presented by **DR.VINEETH.S** is an original work done in the Department of Otorhinolaryngology, Government Stanley Medical College and Hospital, Chennai in partial fulfillment of regulations of the Tamil Nadu Dr.M.G.R. Medical university for the award of degree of M.S. (Otorhinolaryngology) Branch IV, under my guidance, during the academic period of 2020-2022.

DR.ATHIYAMAN.K M.S.,
ASSOCIATE PROFESSOR
DEPARTMENT OF OTORHINOLARYNGOLOGY
GOVERNMENT STANLEY MEDICAL COLLEGE
CHENNAI - 01

Date:

Place: Chennai

DECLARATION

I **DR.VINEETH.S** solemnly declare that the dissertation, titled **"A STUDY OF HEARING IMPAIRMENT AMONG PATIENTS WITH ESSENTIAL HYPERTENSION"** a descriptive cross sectional study submitted by me is a result of the original work carried out by myself under the guidance of**. DR. ATHIYAMAN K, M.S**, ASSOCIATE PROFESSOR, Department of Otorhinolaryngology and Head and Neck surgery, Government Stanley medical college and hospital, Chennai. I further declare that the result of research has not been submitted previously by myself or other persons in any conferences or journals.

This dissertation is submitted to The Tamil Nadu Dr. M.G.R Medical University in the partial fulfilment of the rules and regulations for the M.S. Degree examination in Otorhinolaryngology to be held on May 2022.

Signature of the candidate

DR. VINEETH.S

Date –

Place – Chennai 01

ACKNOWLEDGEMENT

First and foremost I would like to express my sincere gratitude to **PROF.DR.P.BALAJI,MS.,FRCS.,Ph.D.,FCLS.,**The dean, Stanley medical college, for having permitted me to undertake this study.

I am immensely grateful to my ,Head of the department **Prof.Dr.M.GOWRISHANKAR,M.S.D.L.O.,DNB.,** Department of Otorhinolaryngology and Head & Neck surgery, Government Stanley Medical College & Hospital**,** for his guidance, suggestions, encouragement, constant motivation, supervision, valuable support in conducting the study.

I am also immensely grateful to **PROF DR. V. RAJARAJAN, M.S., DNB,** Professor, Department of Otohinolaryngology and Head & Neck surgery and extremely indebted to my guide Associate Professor **DR. K.ATHIYAMAN. M.S**. for his guidance, suggestions, encouragement, constant motivation, supervision, valuable support in conducting the study. I wish to thank my Co-Guide Dr.V.Ashwin, Assistant Professor Of Otorhinolaryngology for his thorough supervision, hard questions and valuable tips. I am grateful to my Assistant Professors Dr.V.Suresh , Dr.P.Chozhan, Dr.Vivek Narayan, Dr.V.Saravana Selvan, and Dr.Benazir for their valuable time and guidance in completing my dissertation work.

I express my sincere thanks to THE SECRETARY AND CHAIRMAN, INSTITUTIONAL ETHICAL COMMITTEE, Government Stanley Medical College and hospital.

I am extremely thankful to Dr. Venkatesan for carrying out the statistical analysis for this study.

I am grateful to all the other post-graduates who most willingly helped me during this study period.

I also thank the staff nurse and theatre personnel, Government Stanley Hospital for their co-operation and assistance in the conduct of this study.

I thank all my colleagues for their constant encouragement and valuable criticism. Last but not the least, I express my gratitude for the generosity shown by all the patients who participated in the study.

I dedicate this work to my beloved parents and brother and without their constant love, support and encouragement it would have been impossible to complete this library dissertation. Their sacrifices have borne fruit in the form of my success.

Above all I thank the Almighty God for showering his blessings and love on me and providing with the inspiration and zest to tread through the path of life.

CONTENTS

INTRODUCTION

According to World health organization (WHO), a person who has hearing thresholds of 25db or more in both ears is said to have hearing loss or impairment. [1]Hearing loss affects the quality of life of people when acquired in adulthood. The estimated prevalence of adult-onset deafness in India was found to be 7.6%. [2]The hearing system affection may cause psychosocial effect, amongst which low self-esteem, isolation, depression and irritability and these problems interfere with quality of life.[3]

Essential hypertension is a form of hypertension that has no identifiable cause. It is a common type of hypertension affecting 95% hypertensive population.[4] In people with hypertension presbycusis can set in at an early decade which can become worse by added factors like noise pollution and vascular pathologies.[5]

Hypertension is a common disease in India and our set of population does not undergo master health check-up and periodic blood pressure check-up. The public health awareness is improving but not to the extent of health consciousness and staying fit. Health consciousness like obesity, hip abdominal ratio and BMR are not prevailing consciousness in our population.

Hearing is affected in hypertensive patients in the following manner.

- The atherosclerotic blood vessels in hypertensive patient cause changes in the

Blood hemodynamics [6, 7]

- The rapidity of blood flow in the cochlea may affect the cochlear dynamics and produce hearing loss at high frequencies at basal turn and apical turn at low frequencies.[6,7,]

- Sodium and potassium perfusion variation can also affect hearing loss.[8]

- In hypertensive patients due to rapid flow of blood there is no proper exchange of oxygen and nutrients, so they depend on the integrity of the heart and blood vessels. The circulatory system pathology can also affect the hearing directly by increase in blood viscosity.[6,7]

- Reduced capillary blood flow leads to reduced oxygen supply resulting in hypoxia which results in hearing loss.[6,7]

- Apart from this the arterial hypertension can cause ionic changes in cell potentials and causes hearing loss.[8]

Studies exist on hearing loss and hypertension but there is minimum data regarding hypertension as a risk factor of hearing loss.[3] Hence there is a need for more studies on hypertension as a risk factor for hearing loss and periodic early screening for hearing loss in patients with essential hypertension which can improve their quality of life.

AIMS AND OBJECTIVES

1. To study the proportion, type and degree of hearing impairment among patients with essential hypertension.
2. To correlate the duration of hypertension and degree of hearing impairment in patients with essential hypertension.

REVIEW OF LITERATURE

ANATOMY OF THE EAR

HISTORY:

Greek physicians during 5th century BC knew tympanic membrane and middle ear. They assumed that the middle ear was the seat of hearing.

Galen described auditory nerve.

Vesalius in 1543 described malleus and incus.

Ingrassia in 1546 described stapes, oval and round window.

Fallopius named canal for facial nerve, cochlea and the labyrinth.

Eustachius described auditory tube.

Helmholtz in1868 initiated auditory physiology.

The ear plays an important role in hearing and communication system[9]. The ear functions as an early warning system by detecting and locating potentially life threatening environmental sounds. The ear plays an important role in balance system by detecting head movements so that eye can stay fixed. The ear is divided into external, middle and inner ear.

EXTERNAL EAR:

It consists of two components. The auricle or pinna and the external auditory canal, the pinna mainly collects and conducts the sound waves into the external auditory canal. It has medial and lateral surface. The lateral surface of the auricle has characteristic prominences and depressions which is different for each individual. This unique pattern is comparable to fingerprints and can

allow the identification of persons on the physiognomy of their auricles.[10] The auricle is made up of single sheet of yellow elastic cartilage and it is connected to temporal bone by three extrinsic ligaments, the anterior, superior and the posterior ligaments. There are six intrinsic ligaments, which connects various parts of the cartilaginous auricle.

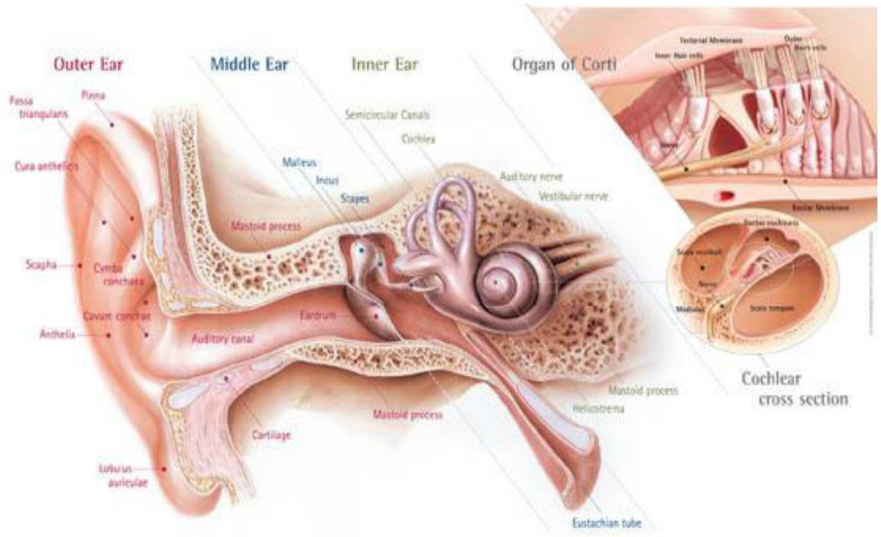

Figure 1- Showing coronal section of Ear (showing its 3 parts)

BLOOD SUPPLY:

Auricle is supplied by the branches of the external carotid artery. The posterior auricular artery, anterior auricular artery, branches of superficial temporal artery, and small branches from the occipital artery.

VENOUS DRAINAGE:

Drain into the corresponding internal and external jugular veins.

NERVE SUPPLY:

MOTOR: Facial nerve to the vestigial auricular muscles.

SENSORY: Greater auricular nerve (c2, 3) supplies medial surface and posterior part of lateral surface. Lesser occipital (c2, 3) supplies superior portion of medial surface. Auricular branch of vagus (Arnold's nerve) supplies concha, antihelix and emenentia concha. Auriculotemporal nerve branch of mandibular division of the trigeminal nerve supply tragus and crus of helix.

LYMPHATIC DRAINAGE:

The medial surface drains to the lymph nodes at the mastoid tip, tragus and upper part of anterior surface to pre auricular nodes, and rest from the auricle to upper deep cervical lymph nodes.

EXTERNAL AUDITORY CANAL:

External auditory canal extends from the concha of the auricle to the tympanic membrane. It is 'S' shaped and approximately 2.4 cms long. The lateral one third is cartilaginous and it is 8mm long and medial two thirds is bony and is 16mm long. In adults, cartilaginous portion runs slightly downwards and forwards. The bony meatus is directed medially downwards and forwards and is continuous with the walls of the bony meatus. The skin of the cartilaginous portion is thick and contains ceruminous and sebaceous glands, which secrete wax .The skin of the bony meatus is thin and has no hairs or gland. In neonates, as the tympanic bone is not developed there is virtually no bony external auditory meatus.

BLOOD SUPPLY:

The auricular branches of the superficial temporal artery and post auricular artery supplies the lateral portion of external auditory canal. Medial portion of the canal is supplied by deep auricular artery which is a branch of first part of the maxillary artery.

VENOUS DRAINAGE:

Veins drain into external jugular vein, maxillary vein and pterygoid plexus of veins.

LYMPHATIC DRAINAGE:

EAC drains into nodes at the mastoid tip, pre auricular nodes and upper deep cervical nodes.

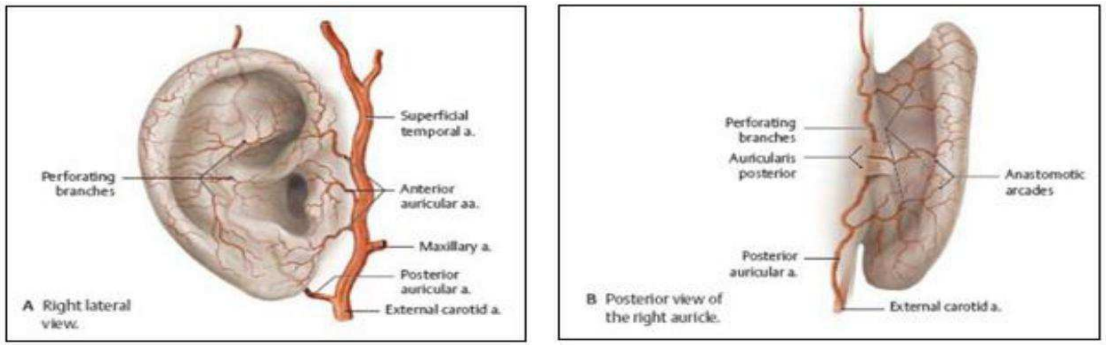

Fig.2. Showing blood supply of pinna

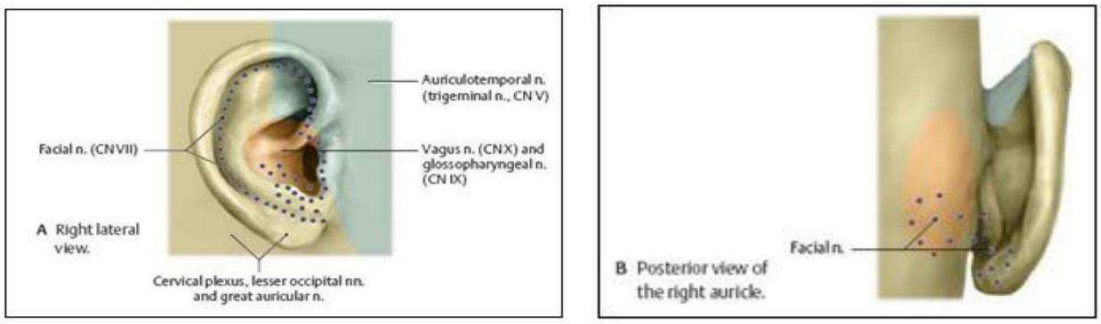

Fig.3.Showing nerve supply of Pinna

TYMPANIC MEMBRANE:

The adult tympanic membrane emulates an irregular cone and lies at the medial end of external auditory meatus. It is oval in shape, with a diameter of about 9mm and forms an acute angle with inferior wall of EAC. Most of the circumference is thickened to form fibrous annulus which sits in a groove called tympanic annulus. Tympanic membrane firmly attaches to the lateral process of malleus and umbo which is the tip of malleus. From the superior limits of the sulcus the annulus becomes fibrous bands which run centrally as anterior and posterior malleolar folds to the lateral process of malleus. The triangular portion above the malleolar folds is called the pars flaccida (shrapnel's membrane) and the portion below is called the pars tensa.

Tympanic membrane is trilaminar structure. The lateral or outer surface is formed by Squamous epithelium which is continuous with the skin of external auditory meatus. The medial or middle layer is the continuation of the mucosal epithelium of the middle ear, between these layers is a fibrous layer known as pars propria. Pars propria of pars tensa has radially oriented fibers in the outer layer and circular, parabolic and transverse fibers in the deeper layers.

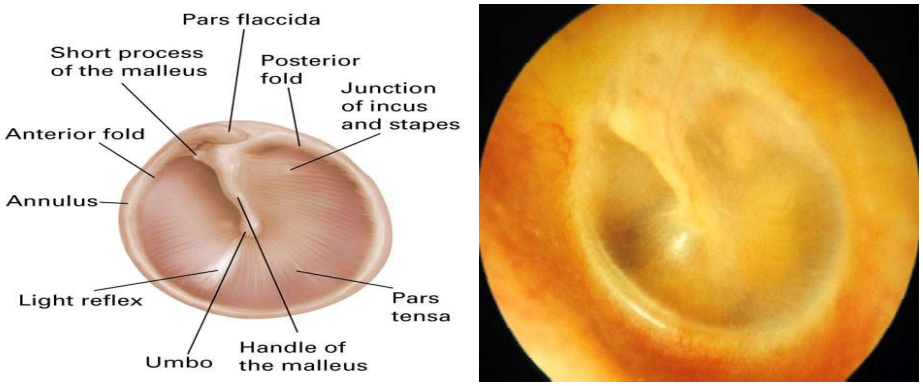

Figure 4 – Showing the diagram and endoscopic view of left tympanic membrane

BLOOD SUPPLY:

The epidermal vessels originate from deep auricular artery branch of maxillary artery coming from external auditory meatus whereas mucosal vessels arise from the anterior tympanic branches of the maxillary artery, the stylomastoid branch of the posterior auricular artery and probably from the middle meningeal artery.

NERVE SUPPLY:

Branches of auriculotemporal nerve, the auricular branch of the vagus and tympanic branch of the glossopharyngeal nerve supply the tympanic membrane.

MIDDLE EAR CLEFT:

It consists of tympanic cavity, the Eustachian tube and the mastoid air cell system.

TYMPANIC CAVITY:

The tympanic cavity is an irregular, air filled cavity within the temporal bone between the tympanic membrane laterally and osseous labyrinth medially. It contains auditory ossicles and their tendons that attach them to the middle ear muscles. Other structures including the tympanic segment of the facial nerve run along its walls to pass through the cavity. Tympanic cavity is divided in to three compartments: the epitympanum, (upper) the mesotympanum (middle) and hypo tympanum (lower).

LATERAL WALL:

The lateral wall is formed by bony lateral wall superiorly, the tympanic membrane centrally and the bony lateral wall of the hypo tympanum inferiorly. The lateral epitympanum wall is wedge shaped in section and its sharp inferior portion is also called the outer attic wall or scutum.

Three holes are present in the bone of medial surface of the lateral wall of the tympanic cavity. The petrotympanic fissure is a slit about 2mm long which opens anteriorly just above the attachment of tympanic membrane. It receives the anterior tympanic branch of maxillary artery to the tympanic cavity. The chorda tympani enters the medial surface of the fissure through a separate anterior canaliculus (canal of Huguier) which is sometimes confluent with the fissure.

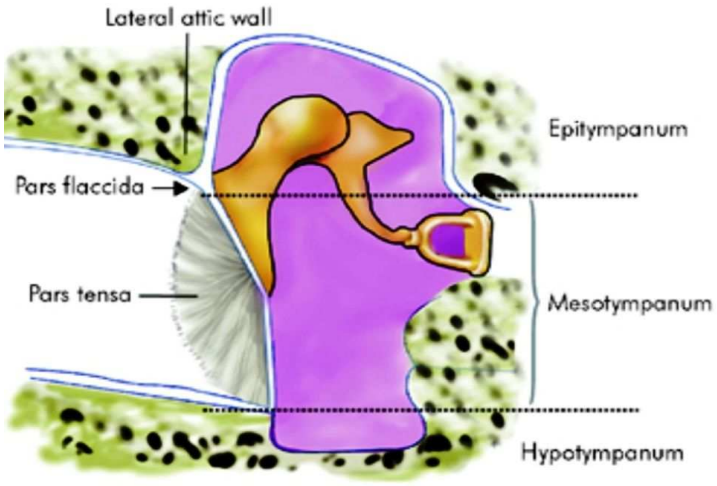

Figure 5 –Showing the tympanic cavity

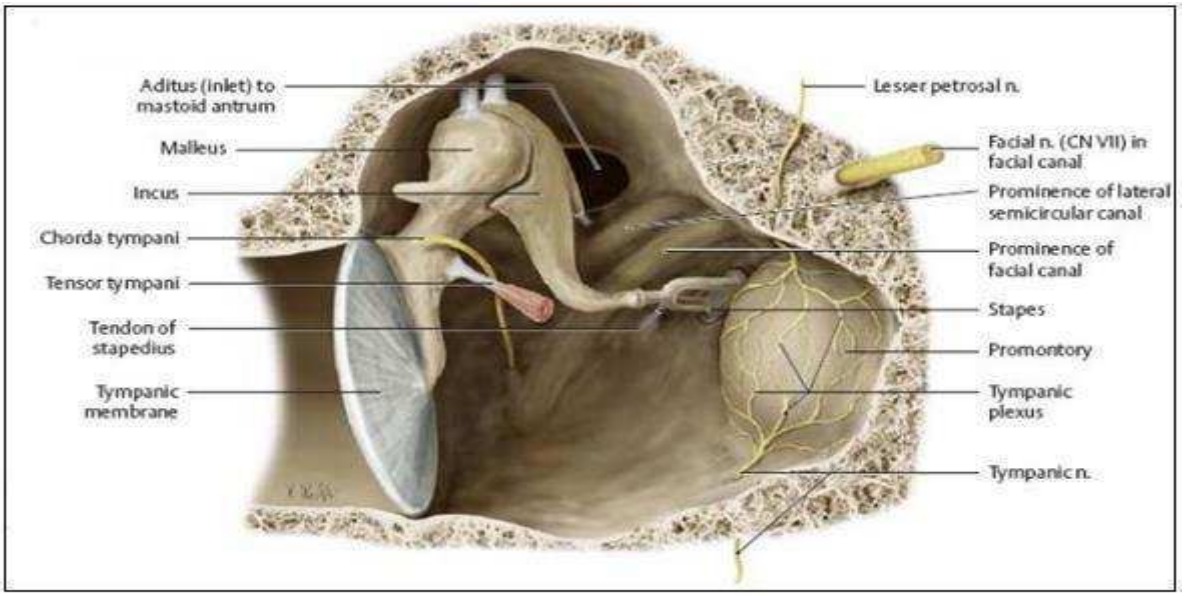

Figure 6 – Showing tympanic cavity anterior wall removed

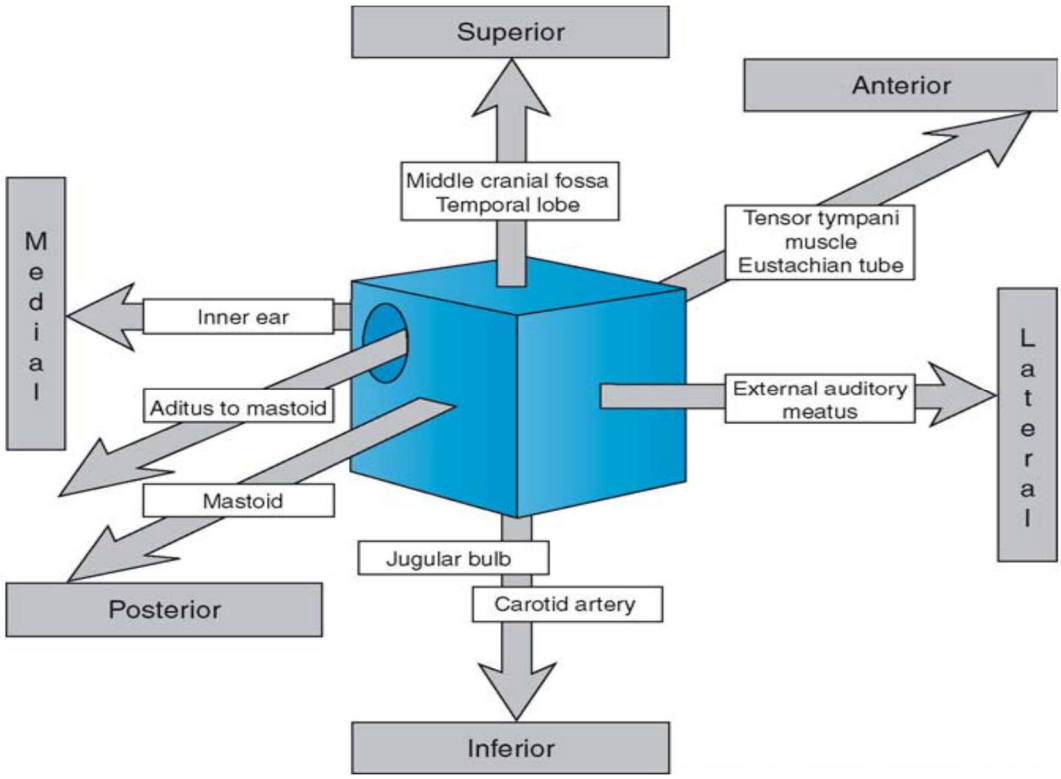

Figure 7- Showing relationship of the middle ear

ROOF:

The roof of the epitympanum is the tympanic cavity is the tegmen tympani, a thin bony plate that separates the middle ear space from the middle cranial fossa. It is formed by both petrous and squamous portion of the temporal bone.

FLOOR:

The floor of the tympanic cavity may consist of compact or pneumatized bone that separates hypo tympanum from the dome of jugular bulb. At the junction of floor and medial wall there is a small opening that allows the entry of tympanic branch of glossopharyngeal nerve into the middle ear from its origin below the base of skull.

ANTERIOR WALL:

The lower third of the anterior wall consists of a thin plate of bone covering the carotid artery. This plate is perforated by superior and inferior caroticotympanic nerves carrying sympathetic fibers to the tympanic plexus, and tympanic branches of the internal carotid artery. The middle third comprises the tympanic orifice of the Eustachian tube, which is oval and measures 5x2mm in size. The upper third is usually pneumatized and may house the anterior epitympanum sinus, a small niche anterior to the ossicular heads.

MEDIAL WALL:

The medial wall separates the tympanic cavity from the inner ear. The promontory is a rounded elevation occupying much of the central portion of the medial wall of the middle ear. It covers part of the basal coil of the cochlea and has grooves on its surface containing nerves which form tympanic plexus. Behind and above the promontory is the oval window, a kidney shaped opening that connects tympanic cavity with the vestibule of the inner ear and is closed by foot plate of stapes.

The round window niche lies below and a little behind the oval window niche from which it is separated by a posterior extension of promontory called subiculum.

POSTERIOR WALL:

Posterior wall is wider above than below and has in its upper part, a large irregular opening, the aditus ad antrum that leads back from the posterior tympanum into the mastoid antrum. The pyramid is a small conical projection with its apex pointed anteriorly which is below the fossa incudis and medial to the

opening of the chorda tympani nerve. This houses stapedius muscle and its tendon, which inserts into the posterior aspect of the head of stapes.

The facial recess is a groove which lies between the pyramid and facial nerve and the annulus of the tympanic membrane. It is bounded, medially by the facial nerve and laterally by the tympanic annulus with chorda tympani running obliquely through the wall between the two.

The sinus tympani, is the posterior extension of the mesotympanum and lies deep to both the promontory and the facial nerve.

CONTENTS OF TYMPANIC CAVITY:

The tympanic cavity contains two muscles, the tensor tympani muscle and stapedius muscle. Three ossicles: the malleus, incus and stapes, and chorda tympani nerve and tympanic plexus.

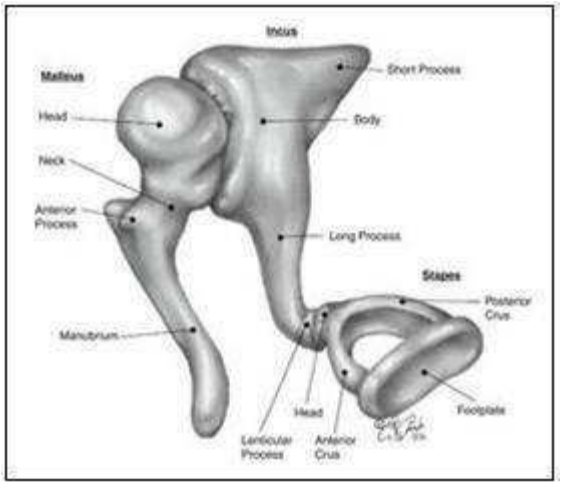

Figure 8- Showing the three ossicles

MALLEUS:

It is the largest of the three ossicles measuring up to 9mm in length. It comprises of a head, neck and handle or manubrium. The head lies in the epitympanum and is suspended by the Suspensory ligament, which runs upwards to the tegmen tympani. On the posteromedial aspect of the head, it articulates with body of incus by the way of synovial joint. The lateral process is a prominent landmark on the tympanic membrane and receives anterior and posterior malleolar folds from the tympanic annulus. The lateral process has a cartilaginous cap that imperceptibly merges with the lamina propria of the tympanic membrane.

INCUS:

The incus is the largest of the three ossicles. It has a body and three processes. The body lies in the epitympanum and is suspended by the superior incudal ligament that is attached to the tegmen tympani. The short process projects backwards from the body, to lie in the fossa incudis to which it is attached by the short Suspensory ligament. The long process descends into the mesotympanum and at its tip is a small medially directed lenticular process. This has sometimes been called the fourth ossicle, because of its incomplete fusion with the tip of long process. Lenticular process articulates with the stapes.

STAPES:

The stapes is the smallest of the three ossicles. Shaped like stirrup and consists of head, neck, the anterior and posterior crura and a footplate. Head of the stapes articulates with lenticular process of the incus. The two curare arises from the lower part of the neck and they join to form the footplate. It has a convex superior margin and anterior and posterior ends. The dimensions of footplate are 3mm long and 1.4mm wide, and lies in the oval window where it is attached to the bony margins by the annular ligament.

TENSOR TYMPANI MUSCLE:

It arises from the walls of the bony canal lying above the Eustachian tube, parts of the muscle also arise from the cartilaginous portion of the Eustachian tube and greater wing of sphenoid. The muscle passes backwards to lie in the medial wall enters the processes cochleariformis where it is held by a transverse tendon and turns right angle to pass laterally and gets inserted on to the medial aspect of the upper end of malleus. The muscle is supplied by mandibular nerve.

STAPEDIUS MUSCLE:

The stapedius muscle arises from the walls of the pyramid and from the downward curved continuation of this canal in front of descending portion of the facial nerve. Tendon emerges from the apex of the pyramid and gets inserted into the stapes. It is supplied by branch of the facial nerve.

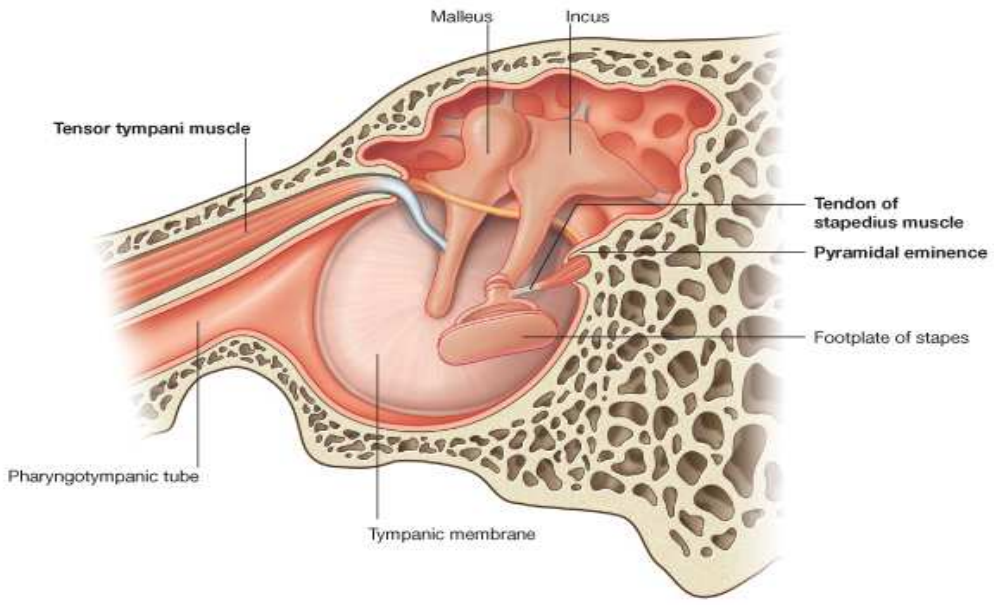

Figure 9- Showing the middle ear muscles

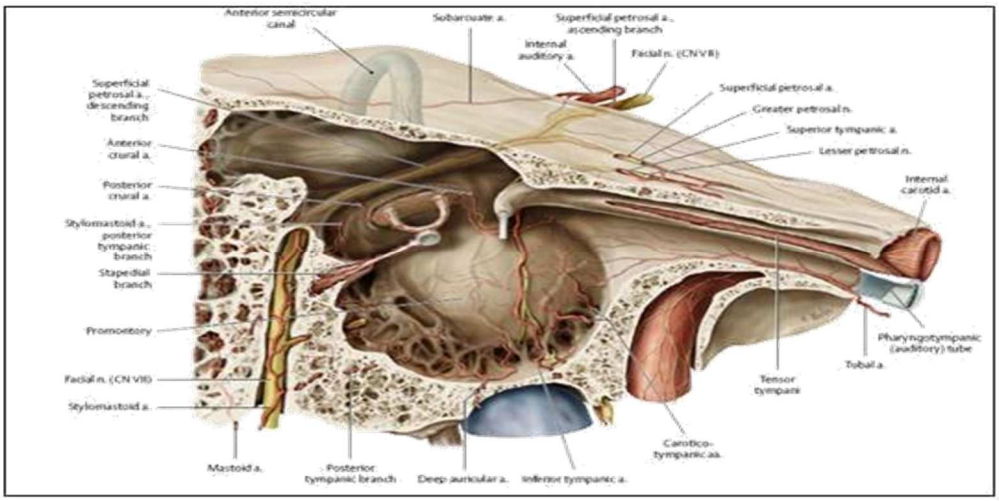

Figure 10 – Showing the tympanic Plexus

CHORDA TYMPANI NERVE:

Chorda tympani nerve a branch of facial nerve enters middle ear cavity from posterior canaliculus at the junction of lateral and posterior wall.

TYMPANIC PLEXUS:

The tympanic plexus, an important middle ear neural plexus which is formed by the tympanic branch of glossopharyngeal nerve (Jacobson's nerve) and caroticotympanic nerves, which arises from the plexus around the internal carotid artery, forms a network on the cochlear promontory of the medial wall of the middle ear.

MUCOSA OF THE TYMPANIC CAVITY:

The middle ear mucosa is essentially mucus secreting respiratory mucosa bearing cilia on its surface. Three distinct mucociliary pathways are present. The epitympanum, promontories and hypo tympanic which coalesces at the tympanic orifice of the Eustachian tube, these folds separate the middle ear spaces into compartments, as a result ventilation to epitympanum is ventilated via two small openings, the anterior and posterior tympanic isthmus.

The mucosal lining of the tympanum at the Eustachian end is respiratory epithelium to thin non glandular cuboidal epithelium at the anterior tympanum to Squamous epithelium at the mastoid end.

INNER EAR:

Inner ear is intricately shaped membranous tube which houses end organs of hearing and balance, within a bony tube – the labyrinth. The bony inner ear acts a transducer. It consists of cochlea. Vestibule and the three semicircular canals, these bony cavities contain clear fluid the perilymph.

The membranous labyrinth contains of cochlear duct, utricle and saccule, three semicircular canal and endolymphatic duct and sac.

The membranous spaces are filled with endolymph. The cochlear duct is the organ of hearing and three semicircular ducts, the utricle and saccule are the organs of balance.

THE VESTIBULE:

Vestibule is the central part of the bony labyrinth which contains oval window in its lateral part, it communicates anteriorly with the cochlea and posterosuperiorly with the semicircular canals.

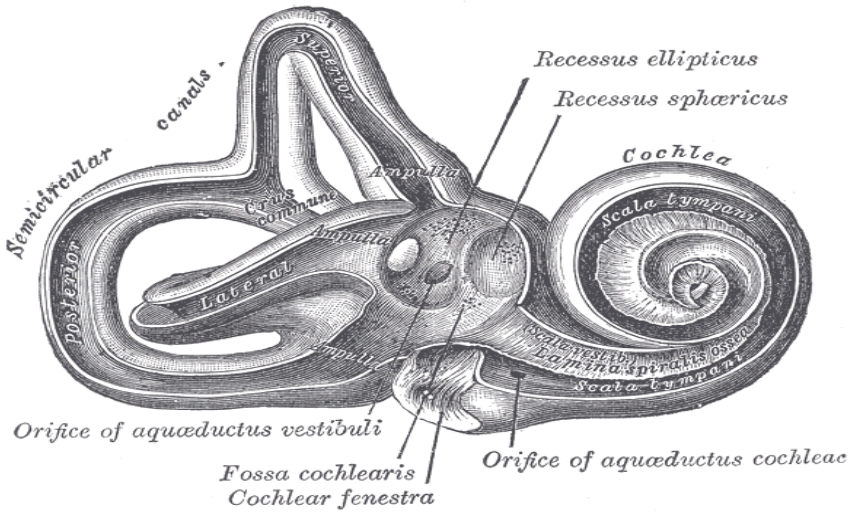

Figure 11- Showing the bony labyrinth

THE COCHLEA:

Cochlea comprises of $2^3/_4$ turns of spiral. It projects in an anterior direction from the vestibule. It is a bony structure that twists on itself around a central column of bone called the modiolus. Base of the modiolus is directed towards internal acoustic meatus and transmits vessels and nerves to the cochlea. Apex points towards the antero-superior part of the medial wall of the middle ear cavity. Oval window and round window are present in the walls of cochlea as bony defects which are covered by thin membranes. Extending laterally throughout the length of the modiolus is a thin lamina of bone called spiral lamina. Circling around the modiolus, and held in central position by its attachment to the lamina of modiolus, is the cochlear duct, which is a component of the membranous labyrinth.

SEMICIRCULAR CANALS:

Projecting from the posterosuperior direction from vestibule are the anterior, posterior and lateral semicircular canals. The canals form two-thirds of a circle connected at both ends to the vestibule and are oriented such that each canal is right angles to the other.

ORGAN OF CORTI:

The organ of corti runs spirally along the floor of the scala media situated on its lower boundary an acellular layer called the basilar membrane. The scala media is triangular in cross section, other boundaries are Reisseners membrane which runs obliquely with respect to the Basilar membrane. The lateral wall consists of striavascularis. It is composed of three layers of cells called marginal cells facing scala media, intermediate cells and the basal cells. These cells are covered by a layer of fibrocytes and connective tissue called the spiral ligament.

The organ of corti consists of special array of alternating sensory hair cells and supporting cells that are supported by the basilar membrane and overlaid by tectorial membrane. It consists of the following from lateral to medial side-Hensens cell, outer tunnel of corti, three to four layers of outer hair cells, Dieters cells with phalangeal process, spaces of Nuel, outer pillar cells, inner tunnel of corti, inner pillar cells, inner phalangeal cells and inner border cells. The cells of the lateral wall contain a variety of ion pumps, enzymes and transport proteins that are associated with haemostatic mechanisms for maintaining the ion composition of fluids of the cochlea. Recent evidence suggests that the hair cells, in concert with efferent innervations from brain are able to mechanoelectrically tune the output of sensory information from the Organ of Corti.[11]

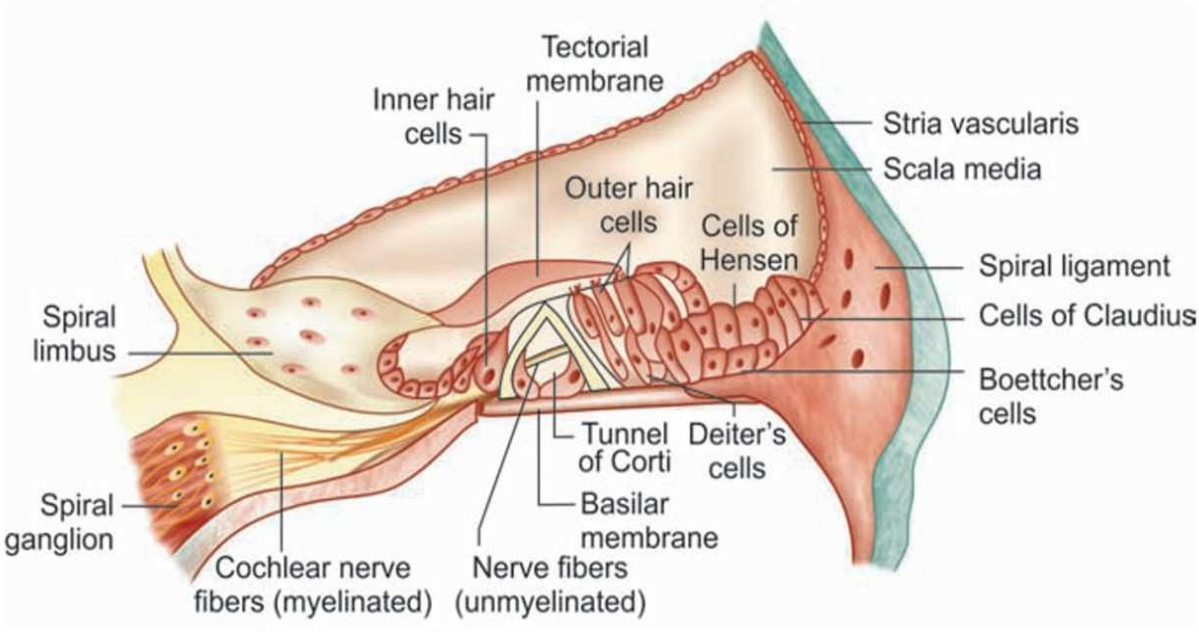

Figure 12- Showing the Organ of Corti

ENDOLYMPH:

Formed by stria vascularis and has high K+ and low Na+ resembling intracellular fluid. It has positive potential gradient (+50 to 120Mv) called the endolymphatic potential. This potential is due to Na+-K+ATPase in the marginal cells of striavascularis which maintains endolymphatic potential which is crucial for hearing.

PERILYMPH:

Perilymph site of production is controversial, it resembles extracellular fluid and it occupies the peri lymphatic space. Perilymph from scala vestibuli originates from plasma while perilymph from scala tympani originates both from plasma and cerebrospinal fluid.

SUPPORTING CELLS:

HENSEN'S CELLS:

Hensens cells form the lateral border of the Organ of Corti. They consist of several rows of tall columnar cells that increase in height towards the cochlear apex and their nuclei occur high in the cytoplasm. The Hensen cells which do not reach the endolymphatic space are called tectal cells.[12]

DEITER'S CELLS:

These cells support outer hair cells at the base and apex of the cochlea. They are attached to the basilar membrane and forms cup like process, around the basal pole of the outer hair cell. The cup like process is open at the base to allow attachment of the afferent and efferent nerve terminals to the outer hair cell body, a thin process containing microtubules and filaments, the phalangeal process, branches off the Dieter cell body and extends obliquely to the apical process of adjacent outer hair cells. This dumbbell-shaped apical pole joins together, to four different outer hair cells in a rigid plate like array called the reticular lamina[12].

INNER HAIR CELLS:

Forms single row.95% of afferent auditory nerves makes contact with inner hair cells. They are important in transmission of auditory impulses and detect the basilar membrane movements. They fit in to a groove called Henson's stripe. The cilia are driven by viscous drag of endolymph.

OUTER HAIR CELLS:

Arranged in 3-4 rows

Very few hair cells synapse with auditory nerves.

Inside of outer hair cells have -70Mv.

Receive efferent innervations from olivary complex.

Modulate the function of the inner hair cells.

Amplify the function of the basilar membrane vibration.

Increases the sensitivity and selectivity of the cochlea

Cochlear microphonics are derived from these cells.

STEREOCILIA:

The inner and outer hair cells contain apical Stereocilia that are crucial for hair cell transduction. Stereocilia are not true cilia but are long, stiff microvilli that extends from circular plate of the hair cell. The Stereocilia are graded in length across the rows with longest row being the most lateral.

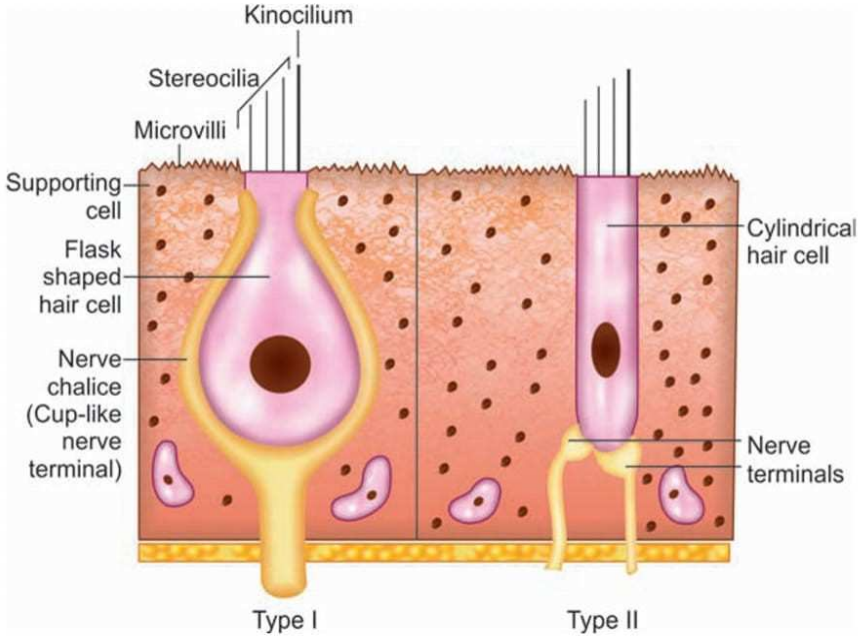

Figure 13- Showing the Hair Cells

INNER SULCUS:

The inner sulcus is a spirally directed open channel bounded by the lateral edge of the spiral limbus, by the medial edge of the Organ of Corti and apically by the tectorial membrane. Inner sulcus cells resemble Claudius cells by their cuboidal shape and vacant neoplasm. Their apical surface exhibits short microvilli and cells are connected together by tight junctions.[12]

SPIRAL LIMBUS:

The spiral limbus is a spirally directed shelf of vascularised connective tissue that overlies the medial region of the osseous spiral lamina. Reisseners membrane is attached at its most medial edge. Its lateral edge forms a wedge-shaped prominence, called the tooth of Huschke that extends over a channel formed

by the inner sulcus cells. The endolymphatic portion of the spiral limbus is covered by a thin extracellular matrix called the limbal portion of the tectorial membrane. Directly beneath the limbal portion of the tectorial membrane lie the epithelial cells called interdental or T cells. They are T shaped cells, whose apical surface forms broad plates across the apical surface of the spiral limbus. The connective tissue matrix of the spiral limbus contains fibroblast like cells, vascular elements and extra cellular filaments containing predominantly type II collagen.[12]

TECTORIAL MEMBRANE:

The tectorial membrane is an acellular, extracellular matrix that overlies the spiral limbus, inner sulcus and most importantly the organ of corti. It consists of primarily of fibrous materials and seems to be hydrated by endolymph. The tectorial membrane can be divided into six regions. The limbal layer, fibrous matrix, marginal band, covers net, Hensens stripe and Hardesty's or kimuras membrane. The limbal layer is thin and overlies the interdental cells of the spiral limbus. This layer expands out over the inner sulcus and forms a wedge-shaped main body called the fibrous matrix. It contains straight and unmatched 10-nm filament bundles of typeII collagen intermingled with a finer network of the filaments.

TypeII collagen seems to be predominant protein of tectorial membrane, it also contains lesser amounts of other collagen type V and IX and glycoproteins called tectorin.On the endolymphatic surface radial strands of material, the cover net, extends out towards the margin of the membrane in an oblique apical direction. The lateral margin of the membrane forms a discontinuous net of material called the marginal band. The portion of the tectorial membrane that faces the Organ of Corti is a thin poorly defined region of the material called Hardestys membrane, which overlies the outer hair cells. Hensens stripe forms a

prominent triangular shaped substructure that overlies the region containing the pillar cell head plate and inner hair cells.[12]

BLOOD SUPPLY OF THE INNER EAR:

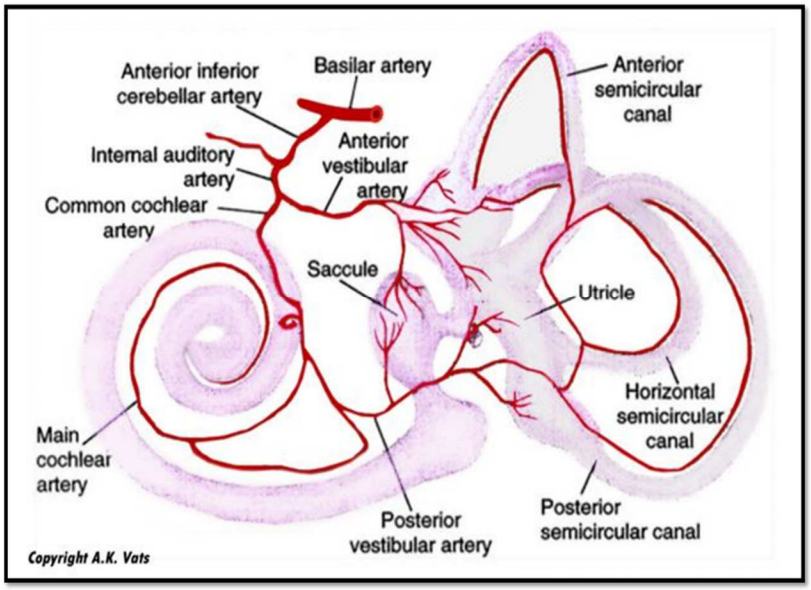

Figure 14- Showing the blood Supply of Inner Ear

Labyrinthine artery, a branch of the anterior inferior cerebellar artery enters the internal auditory meatus along with the vestibulocochlear nerve, supplies the inner ear. Labyrinthine artery branches into common cochlear artery and the anterior vestibular artery. Common cochlear artery branches into spiral modiolar artery and vestibulocochlear artery.

The Vestibulocochlear vascularises the basal turn of the cochlea. The spiral modiolar artery gives off two coiled branches that travel apically over the scala vestibuli and supply spiral ganglion and spiral limbus. The cochlea is drained by spiral modiolar vein, which originates as a confluence of the venules of the lateral wall, spiral limbus and the spiral ganglion.[12]

PHYSIOLOGY OF SOUND TRANSMISSION OF THE EAR:

Hearing Mechanism is mainly divided into mechanical conduction of sound through the tympanic membrane. Transduction of mechanical energy into electrical energy in the cochlea, and conduction of these electrical impulses into the brain, Pinna localizes and focuses the sound arriving from an arc 135^0 and concentrates the sound at the concha, increases sound pressure at the entrance of the ear canal by 6db, which then passes through the external auditory canal and strikes the tympanic membrane. Tympanic membrane vibrates, and the vibrations are transmitted to the ossicular chain to the foot plate of stapes. Movement of the foot plate of stapes induces pressure changes in the labyrinthine fluids which move the basilar membrane as result, hair cells are stimulated. Hair cells convert mechanical energy into electrical impulses which travels through the auditory nerve.

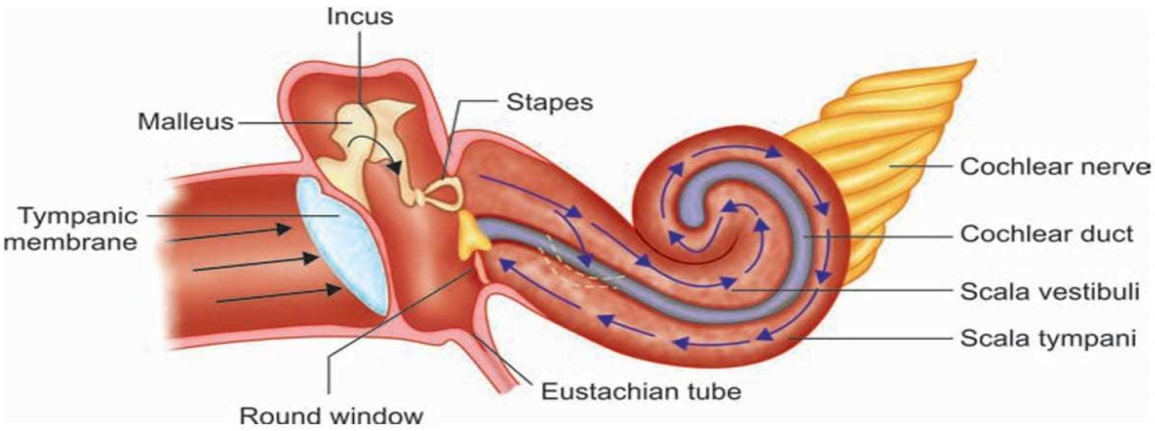

Figure 15- Showing the Sound transmission of the Ear

IMPEDENCE MATCHING:

Middle ear is the major transformer. The transformer mechanism is carried out by the ear drum (caternary lever), the ossicles (ossicular lever) and TM, and the foot plate of stapes (hydraulic lever). The middle ear transforms acoustic energy from air medium to the fluid medium. The energy is not lost due to impendence matching.

CATERNARY LEVER:

Attachment of tympanic membrane at the annulus tympanicus amplifies the energy at the annulus due to elastic properties of the stretched drum head fibers. As the annulus surrounding TM is immobile, sound energy is directed from the edges towards the centre of TM. It provides two-fold increases in sound pressure at the malleus.

OSSICULAR LEVER:

Lever ratio is the length of manubrium of malleus to the long process of incus which is 1.31:1. Malleus and the incus acts as a lever around an axis running between the anterior malleal ligament and the incudal ligament.

HYDRAULIC LEVER:

The effective vibratory area of tympanic membrane is55m, the Areal ratio is the area between TM and the foot plate of stapes (3.4mm), so the effective areal ratio is 17:1.

Thus, the combined transformer ratio of middle ear is about 22:1(17x1.3=22). This translates approximately to 25db.

INNER EAR:

Movement of stapes produces flow of perilymph in the scala vestibuli up to the helicotrema and down the scala tympani. Rapid vibrations produce movement of the basilar membrane which results in shearing force between tectorial membrane and hair cells. The distortion results in cochlear microphonics which triggers nerve impulses, which are conducted through the auditory pathway.[11]

TRANSDUCTION BY HAIR CELLS:

Stereocilia present on the apical surface of hair cells are mechanically rigid and are braced together with cross links so that they move as a stiff bundle. Therefore, as the bundle is deflected, the different rows of Stereocilia slide over one another. There are fine links running upwards from the tips of shorter Stereocilia on the hair cells, which join the adjacent taller Stereocilia of next row. Stereocilia are deflected in the direction of the tallest Stereocilia, the crosslinks are placed under tension, which opens the ion channels in the cell membrane.[11]

When the ion channels open, ions enter or leave the cell depending on the electrical and chemical gradients across the apical cell surface. The apical surface is faced by endolymph with high positive potential (+80mv) and high potassium concentration. Inside the cell there is negative potential of -70mv for outer hair cells. The potentials combine to give 125mv (inner hair cell) or 150mv (outer hair cell) of potential drop across the channel. When the ion channels open potassium from the endolymph is driven

inside as there is big potential gradient rendering the cell to be more positive inside. When the ion channels are shut during the opposite phase of sound waves cells become more negative, as K+ is the main ion in the endolymph it produces the transducer current. Potassium is automatically in equilibrium across the basal membrane of hair cells.

The energy for entire process comes from the striavascularis, which by ion pumping, stores the energy in the battery of the endolymph. This is the battery or resistance modulation theory of Davis.[11]

Radial shearing between the tectorial membrane and the reticular lamina and the vertical shearing movement of cochlear partition of hair cells are expected to have approximation of the same frequency tuning as the mechanical vibration of the basilar membrane.[11]

EXITATION OF PRIMARY AFFERENT ENDINGS:

Depolarization of the hair cells resulting from mechanoelectrical transduction leads to the release of neurotransmitters, which are taken up by dendritic process of afferent fibers. An amino acid, glutamate is released due to depolarization which is present at the base of inner hair cells. Neurotransmitter release occurs as there is K+ influx in to the cells, and is modulated by $Ca.^{2+}$

RESPONSES OF AUDITORY NERVE FIBERS:

Neurotransmitter released at the base gives rise to action potentials in the auditory nerve fibers. For low frequencies, the transmitter is released in packets which are concentrated during depolarization. A single auditory stimulus is excitatory, never inhibitory.

Transmitter release takes place in synchrony with the auditory stimuli. Action potential generation as a result of neurotransmitter release is also in synchrony with the individual cycles of the sound stimuli. This is known as phase locking. Phase locking is seen only at low frequencies and where the cyclic changes in hair cells intracellular potential are large.[11]

Phase locking represents that information regarding sound stimulus is transmitted up to the auditory nerve. For stimuli below 5 kHz, the timing of action potential in the auditory nerve is able to signal the details of temporal properties of sound waveform. This is known as temporal coding of stimuli.

The auditory nerve fiber codes information by frequency selectivity. Inner hair cells detect movement of the basilar membrane along the cochlear duct at one point. Mechanical response of the basilar membrane is sharply tuned.

Each auditory nerve fiber has characteristic frequency, the lowest sound pressure level for the criterion response. The tonotopic organization of mechanical travelling wave in the cochlea, the base codes for high frequency and apex codes for low frequencies, coding is based on frequency selectivity and is called place coding.

COCHLEAR NUCLEUS:

The cochlear nucleus is the relay station for all afferent auditory nerve fibers. The nuclei is bilateral, and is present at the pontomedullary junction lateral to entry of CNVIII. The cochlear nucleus divides into ventral cochlear nucleus (VCN) and dorsal cochlear nucleus (DCN). The VCN is classified into anteroventral cochlear nucleus (AVCN) and posteroventral cochlear nucleus (PVCN). The AVCN is divided again in to anterior and posterior division.

Each subdivision consists of specific cell types, highly heterogeneous between species that receive a complete topographic projection from the auditory nerve. Five neuronal classes are generally accepted on basis of cell morphology, spherical, globular, bushy cells, multipolar, octopus, pyramidal and granule cells.

The second order neurons form three main bundles, the ventral acoustic stria (VAS, trapezoid body), the intermediate acoustic stria (IAS, stria of Helde), and the dorsal acoustic stria (DAS,stria of Monakow).The VAS originates from spherical and globular cells, courses medially and rostrally across medulla to reach the lateral superior olive, the medial superior olive, the medial nucleus of trapezoid body, and the inferior colliculus. The IAS originates from the octopus cells of PVCN, projects bilaterally, ipsilaterally or contralaterally to the ventral nucleus of the trapezoid body. The lateral superior olive and the pre olivary region, which gives rise to olivocochlear bundle. The DAS is essentially a crossed pathway by which the cells in the DCN projects to the nuclei of the lateral lemniscus and central nucleus of the inferior colliculus.[12]

SUPERIOR OLIVARY COMPLEX:

Superior olivary complex (SOC) is located in the lower pons. The nuclear complex consists of the medial nucleus of the superior olive (MSO, the lateral nucleus of the superior olive (LSO), medial nucleus of the trapezoid body (MNTB), and the periolivary nuclei (PON).

LATERAL LEMNISCUS:

The lateral lemniscus is the major ascending pathway that connects cochlear nuclei and the superior olivary complexes to the inferior colliculus. There are three principal groups of cells, the ventral (VLL), intermediate (ILL) and the dorsal (DLL) nuclei of the lateral lemniscus.VLL receives fibers mainly from the contralateral VCN and a smaller contribution from the SOC and the DCN, it responds largely to the contralateral ear. The ILL receives from the contralateral VCN, the ipsilateral MNTB, and small fibers from the contralateral DLL.The DLL receives afferents from ipsilateral MSO and bilateral LSO with few fibers from contralateral VCN.The DLL on each side are interconnected by way of the comissure of probust. These fibers from DLL cross midline just below the medial longitudinal fasciculus to synapse in the dorsal nucleus of the opposite side.

INFERIOR COLLICULUS:

The inferior colliculi are bilateral, mesencephalic structures that form with superior colliculi part of the tectum of the brain. They serve as principal relay station for the ascending auditory pathway and process acoustic information from the lower brainstem to the medial geniculate body and then to the auditory cortex.

The inferior colliculi consists of three main cell groups, the central nucleus of the inferior colliculus (ICC), the external nucleus of the inferior colliculus or dorsal cortex (ICX) and the pericentral nucleus (ICP).

ICC is the largest and prominent nucleus of the IC. It receives most of the ascending information from the brain stem. It receives input from both the cochlear nuclei, SOC, nuclei of lateral lemniscus and the descending afferent fibers from the auditory cortex. The DCN projects directly to the ICC, whereas

indirect inputs project from the VCN through the SOC and the nuclei of lateral lemniscus. The efferent from IC travel to the medial geniculate body mainly by the way of brachium, and sends projections to the contralateral IC by way of the comissure of the inferior colliculus. Additional projections travel to the periaqueductal gray and deep layers of caudal superior colliculus, the contralateral inferior brachium and terminate in the parabranchial region, the interstial nuclei of inferior brachium and lateral part of the posterior thalamic group. Descending fibers project by means of the lateral lemniscus to the VLL.The medial PON, lateral PON, retroolivary region and contra lateral DCN.[12]

MEDIAL GENICULATE BODY:

The medial geniculate body (MGB), a relay station in the ascending auditory pathway intercalated between the fibers of the brachium of the IC and the auditory cortex. MGB is in the thalamus medial to the lateral geniculate body. The MGB is divided cytoarchitecturally into ventral, medial and dorsal. All the three subdivisions receive ascending projections from the nuclei of IC and descending fibers from the auditory cortex.[12]

AUDITORY CORTEX:

In human's primary auditory cortex (Brodmann's area 41and 42) is located in the upper bank of the temporal lobe and is surrounded by specific auditory and nonspecific association areas (Brodmann's areas 22 and 52).

The auditory cortex is divided into primary auditory cortex and association auditory regions that receive acoustic and other sensory inputs. The association areas connect the primary cortex to the frontal and the temporoparietal regions, concerned with language, speech, somaesthetic and vision areas.

The auditory cortex gives off three main descending pathways to the thalamus, midbrain and pons. Each auditory cortical area projects to the sources of its afferent fibers in the medial geniculate body. The primary auditory cortex also projects bilaterally to the central and

Ipsilateral to the pericentral nucleus of the ICC, and to the corpus striatum.[12]

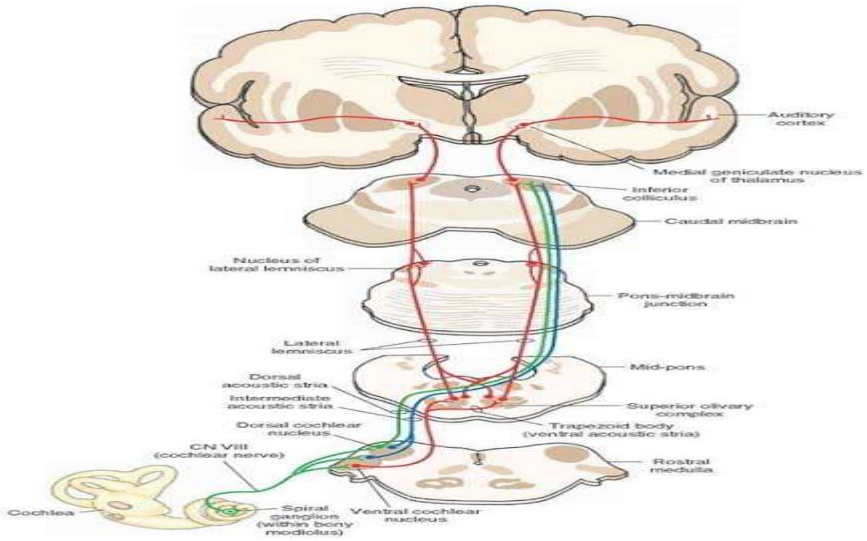

Figure 16-Showing the auditory pathway

THEORIES OF HEARING:

HELMHOLTZ PLACE THEORY (1857)/RESONANCE THEORY

Sound perception depends on where the component frequency produces vibrations, along the basilar membrane.

RUTHERFORDS TELEPHONE THEORY (1886)/FREQUENCY THEORY

This theory states that, all sounds were encoded to the brain by neurons firing at a rate that simulates the frequency of the sounds. Sound stimulates the entire basilar membrane to vibrate but the pitch of the sound is related to rate of firing of individual auditory nerve fibers. This theory had a major flaw as humans can hear up to 20.000Hz frequencies.

WEAVERS VOLLEY THEORY:

This theory states that, multiple neurons fire in a volley to later combine and equal the frequency of the original sound stimulus. Phase synchrony is only accurate up to 5000Hz, Volley theory cannot account for all frequencies.

BEKESYS TRAVELLING WAVE THEORY (1961):

Georg von Bekesy won Nobel Prize in 1961 for this theory which states that the sound stimulus produces vibration like a wave on to the basilar membrane, which starts from basal turn and proceeds to the apex of the cochlea. It increases in amplitude and reaches maximum and then dies off. Sound frequency is determined by the point of maximum amplitude.

HEARING SENSITIVITY LOSS:

The hearing sensitivity loss is the most common form of hearing disorder. It is characterized by a reduction in the sensitivity of auditory mechanism so that sounds need to be presented at higher intensity

than normal. The World health organization has estimated that278 million people suffer from a moderate to profound hearing loss. Hearing sensitivity loss is caused by abnormal reduction of sound being delivered to the brain by a disordered ear.[13]Hearing loss can be defined on the basis of site of the disease, severity, audiometric configuration and method of onset.[14]

'TYPES OF HEARING LOSS:

CONDUCTIVE HEARING LOSS: It results when the sound is not conducted through the outer and middle ear.

SENSORINEURAL HEARING LOSS: It results when the sensory or neural mechanisms within the cochlea are not functioning. It can also be caused by disorders of eighth nerve or auditory brainstem, such type of disorder is termed as retrocochlear disorders. When any structure in the Sensorineural mechanism is damaged, or lost its ability to transduce mechanical energy in to electrical energy is reduced, which results in changes in cochlear processing and a reduction in sensitivity of the cochlear receptors.

Sensorineural neural hearing loss has at least three important effects on hearing:

1. Reduction in cochlear sensitivity,

2. Reduction in frequency resolution,

3. Dynamic range of hearing mechanism.[15]

One of the features of Sensorineural hearing loss is recruitment (abnormal loudness growth). Loudness grows more rapidly than normal at intensity levels just above the threshold in an ear with Sensorineural

hearing loss resulting in reduced dynamic range from the threshold level to the discomfort level. Reduction in dynamic range and frequency resolution affects perception of speech. Extreme reduction in frequency resolution and dynamic range, can severely limit the usefulness of the residual hearing.[16]

MIXED HEARING LOSS: results due to structures of both the conductive mechanism and cochlea are involved.[14]

Configuration of the hearing loss: The configuration of the hearing loss refers to the degree and the pattern of hearing loss across the frequencies.[13]

Symmetrical versus asymmetrical: Symmetrical means, the degree and the configuration of the hearing loss are same in each ear.

Progressive versus sudden hearing loss: Hearing loss worsening over a period of time is progressive hearing loss. Sudden means that the loss happens acutely[13].

Fluctuating versus stable hearing loss

Bilateral versus unilateral hearing loss.[13]

TESTS TO ASSESS HEARING ACQUITY:

TUNING FORK TESTS:

Tuning fork examination comprises a part of routine and important component of neurotological examination and may be performed easily at the bedside or in the office.

RINNE'S TEST:

The Rinne tuning fork test was first described by Dr Adolf Rinne in 1835. The test has evolved to become the most widely used tuning fork test till today.[17] The test compares air conduction with the bone conduction.[18] The vibrating tuning fork is placed against the mastoid process (bone conduction) and comparing its loudness with that of the tuning fork just placed outside the ear canal (air conduction). Rinne test is performed ideally with a 512-Hz tuning fork. If the sound is perceived louder by bone conduction, then it indicates conductive hearing loss. If the sound is perceived louder by air conduction, then conductive hearing loss is not indicated.[17]

WEBER'S TEST:

The Weber test is performed by placing the vibrating tuning fork in the center of the patient's forehead or at the bridge of the nose, if the patient has difficulty with these locations, the mandible or the front teeth may be used, however the patient should tightly clench his or her teeth. The patient is asked if the sound is louder in one ear or is heard in the midline. The sound waves should be transmitted equally well to both the ears through the skull bone.

A unilateral Sensorineural hearing loss will cause the sound to lateralize to the ear with better cochlear reserve, however a unilateral conductive hearing loss will cause the sound to lateralize to the side with conductive pathology because the cochlea is intact bilaterally. Bone conduction causes the sound to be

heard better in the ear with conductive pathology as there is less background noise detected through air conduction. A midline Weber is referred as negative, while 'Weber right' and Weber left refers to the direction to which the sound is lateralized.[18]

ABSOLUTE BONE CONDUCTION TEST:

The absolute bone conduction test is also called as the Schwabach test. The test compares the patients hearing with the examiners hearing, and uses multiple tuning forks such as 256, 512, 1024 and 2048Hz tuning forks. The stem of the vibrating tuning fork is placed on the mastoid process of the patient, and then on the mastoid of the examiner, assuming that the examiner has normal hearing. If the patient hears the sound as long as the examiner then the test is said to be Schwabach normal. If the patient hears the sound longer than the examiner it is called Schwabach prolonged, if the patient hears the sound for less time than the examiner it is called Schwabach shortened.[18]

PURE TONE AUDIOMETER:

Pure tone audiometer is a simple, easy to use instrument and works on 230 volts AC supply and 15volts DC supply with a built-in voltage regulator circuit. The model has facilities for air, bone and tone decay tests. The basic elements of an audiometer are

Tone generator

Calibrator

Switch

Amplifier

Attenuator

Display

Ear phones are used to test hearing by air conduction, and a small vibrator is placed over the mastoid process to test the bone conduction. All audiometers incorporate a calibration circuit, which allows the output sound level to be set at each frequency. The signals presented to the subject by an audiometer are characterized by its frequency, sound pressure level and waveform which are all controlled.

PRINCIPLE:

The pure tone audiometry is used to establish hearing threshold sensitivity at discrete frequencies across the range, important for human communication. The American National Standards Institute (ANSI) S3.20-1973 defines the threshold of audibility as "the minimum effective sound pressure level of an acoustic signal producing an auditory sensation in a specified fraction of the trails". Audiometric threshold data are displayed on a graphic plot called an audiogram[19]. The unit of stimulus is the decibel (dB), a logarithmic unit of the ratio of the given sound pressure to the reference sound pressure. The reference sound pressure is the amount of pressure against the tympanic membrane just detected by normal human ear as sound defined as 0 dB hearing level (HL) [21]. Complete pure tone audiogram consists of air and bone conduction threshold curves for each ear.

The aim of performing this test is to rule out whether the subject has any hearing impairment, if there is hearing loss, whether it is conductive, sensorineural or mixed type of hearing loss. Two general methods are employed

1-Manual audiometry: referred to as conventional pure tone audiometry.2-Sweep frequency or discrete frequency automatic audiometry: referred as Bekesy-type audiometry.[20]

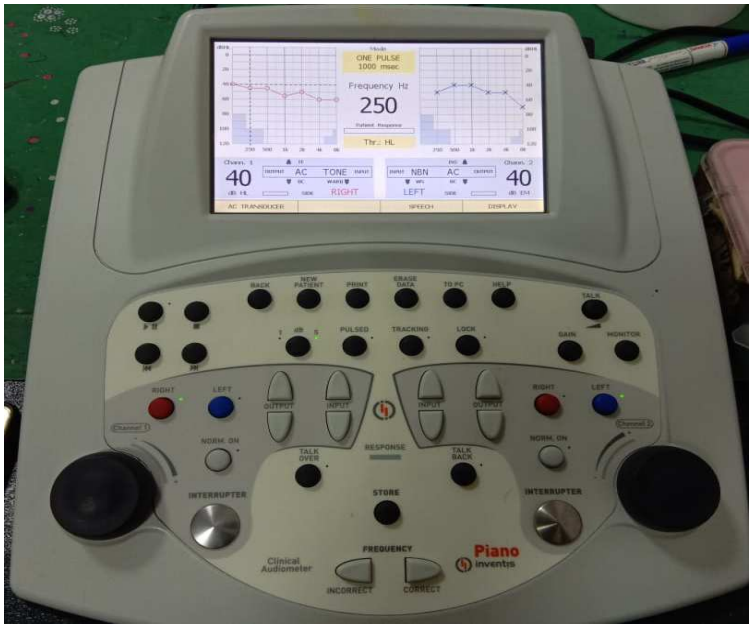

Figure 17: Showing pure tone audiometer

Pure tone audiometry is the most commonly used test for evaluating auditory sensitivity.

An audiometer is an electronic device which produces pure tones, the intensity of which can be increased or decreased to 5db steps. Air conduction thresholds are measured for 250, 500, 1000, 2000, 4000, 6000 and 8000 Hz.

Bone conduction thresholds are measured for 250, 500, 1000, 2000 and 4000 Hz.

The amount of intensity that has to be raised above the normal level is a measure of degree of hearing loss or impairment at that frequency. It is charted in the form of graph called audiogram.

The threshold of bone conduction is a measure of the cochlear function. The difference in the threshold air and bone conduction is a measure of the degree of hearing loss. The audiometer is calibrated so that the hearing of a normal person, both for air and bone conduction is kept at 0db and there is no A-B gap.

The patient is subjected to an otological examination to rule out any external and middle ear pathologies. He is taken to sound proof room for an audiometric testing. The method is based on American society for speech and hearing association (ASHA) guidelines for manual pure tone audiometry. Hearing sensitivity within the "speech frequency" region is summarized by calculating the pure tone average (hearing thresholds for 500, 1000, and 2000 Hz divided by three and reported in decibels[21]).

As the test is a subjective test patient is explained about the procedure in their own language.

Then the subject is made to wear ear phones during air conduction testing and a bone vibrator for bone conduction. He is instructed to raise his hand as soon he hears the sound regardless of how faint the tone is and stop responding as soon the tone goes off. The same procedure is done in the other ear.

DETERMINATION OF THRESHOLD OF HEARING:

Threshold is determined by the use of a version of the Hughson-Westlake ascending method in which sounds are initially presented well above threshold. This is called the "tone familiarization.'[22]

INTERPRETATION OF AUDIOGRAM:

NORMAL: When air and the bone conduction thresholds are between -10 to 25db.

CONDUCTIVE HEARING LOSS:

Raised air conduction threshold and normal bone conduction threshold, with a wide A-B gap of more than 15db.

SENSORINEURAL HEARING LOSS:

It is indicated by raised air and bone conduction thresholds (more than 25db).

MIXED HEARING LOSS:

Air and bone conduction thresholds are raised with an air bone gap of more than 15db.

Figure: 18: Showing pure tone audiogram test in progress

TABLE 1 - SYMBOLS IN AUDIOGRAM

Key to symbols on an audiogram		
	Right	Left
Air unmasked	0	X
Air masked	Δ	□
Bone unmasked	<	>
Bone masked	[	]
Sound field	S	S
Aided	A	A

DEGREE OF HEARING LOSS:

Normal=0-25dB

Mild =26-40dB

Moderate=41-55dB

Moderately severe=56-70dB

Severe=71-90dB

Profound=>90Db

CAUSES OF SENSORINEURAL HEARING LOSS[23]:

HERIDITORY DISORDERS:

Autosomal dominant: early onset degenerative hearing loss, otosclerosis.

Autosomal recessive: uncommon unless syndromal: Alports syndrome, Cruzons syndrome, Waardenburgs syndrome.

Inherited metabolic

X-linked

Mitochondrial

INFECTIONS:

Viral: acquired immunodeficiency syndrome. Ramsay hunt syndrome.

Microbial: syphilis, meningitis, Lyme disease, chronic suppurative otitis media.

Fungal

ISCHAEMIA:

Ischemic heart disease

Macro vascular disorders: arteriovenous malformations, ectasia, aberrant vessels

Stroke

Hypertension

Vasculitis

Coagulopathies

INFLAMMATION:

Neoplastic

Metabolic and endocrine disorders

Trauma and toxicity

Degenerative

Miscellaneous

Sudden Sensorineural hearing loss

ESSENTIAL HYPERTENSION:

Essential hypertension is also called primary hypertension, is a form of hypertension that by definition has no identifiable cause. It is a common type of hypertension affecting 95% of hypertensive patients. Prevalence of hypertension increases with age.[24]

The history of hypertension begins with the understanding of cardiovascular system with the work of physician William Harvey (1578-1657), who described the circulation of blood in his book 'De motu cordis". The English clergyman Stephen hales made first published measurement of blood pressure in 1973.[25,26,]

Description of hypertension as a disease came among others from Thomas young in 1808 and Richard Bright in 1836.[25]However Hypertension as a clinical entity was known after the invention of cuff-

based sphygmomanometer by scipione Riva-Roci in 1896.[27]This allowed blood pressure to be measured in the clinic. Nikolai Korotkoff in 1905 improved the technique and described Korotkoff sounds that are heard when the artery is auscultated with a stethoscope when the sphygmomanometer cuff is deflated.[26]

The concept of essential hypertension was introduced by physiologist Otto frank and described that there is no cause found for elevated blood pressure. The term malignant hypertension was coined by physicians from mayo clinic to describe a syndrome of very high blood pressure. Severe retinopathy and adequate kidney function which usually resulted in death within a year from strokes, heart failure or kidney failure.[28]

JOINT NATIONAL COMMITTEE ON PREVENTION, DETECTION, EVALUATION AND TREATMENT OF HIGH BLOOD PRESSURE (JNC)-7

The complete version of the joint national committee on Prevention, Detection, Evaluation, and treatment of high blood pressure (JNC 7) express the facts about guide to lowering high blood pressure, reference card from the JNC 7 for clinicians, blood pressure wallet card for patients, and palm application of the JNC7 recommendations.

Hypertension is an increasingly important medical and public health problem. The prevalence of hypertension increases with advancing age to the point where more than half of the people 60-69 years of age and approximately three-fourths of those 70 years of age and older are affected. Approximately thirty percent of adults are still unaware of their hypertension, more than forty percent of people with

hypertension are not on treatment and two-thirds of hypertensive patients are not being controlled to BP levels of <140/90mm of Hg.

The age-related rise in systolic blood pressure is primarily responsible for an increase in both incidence and prevalence of hypertension with increasing age. Investigators recently reported the life time risk of hypertension to be approximately 90% for men and women, who were nonhypertensive at 55or 65 years and survived to age 80-85.[29]even after adjusting for competing mortality, the remaining lifetime risks of hypertension were 86-90% in women and 81-83% in men.

Because of the new data on lifetime risk of hypertension and the impressive increase in the risk of cardiovascular complications associated with levels of BP previously considered to be normal, the JNC 7 report has introduced a new classification that includes the term pre hypertension for those with BPs ranging from 120-139mm Hg systolic and /or 80-89mmHg diastolic.

This new designation is intended to identify those individuals in whom early intervention by adaptation of healthy lifestyles could reduce BP, decrease the rate of progression of BP to hypertensive levels with age, or prevent hypertension entirely.

Pre hypertension is not a disease category rather it is to identify individuals at high risk of developing hypertension, so that both the patients and clinicians are alerted to intervene and prevent or delay the disease. Individuals with pre hypertension, who have diabetes or kidney disease, should be considered candidates for appropriate drug therapy if the life style modification fails to reduce their BP to 130/80 mm of Hg or less.

JNC 7 suggests that all people with hypertension (stages 1 and 2) to be treated. The treatment goal for individuals with hypertension and no other compelling conditions is less than140/90mmHg.The goal

for individuals with prehypertension and no compelling indications is to lower BP to normal levels with lifestyle changes.

Table-1: provides a classification of blood pressure for adults 18years and older is given below in the table. This classification is based on the average of two or more properly measured, seated, BP readings on each of two or more hospital visit.

Table-2: Blood pressure classification

BLOOD PRESSURE CLASSIFICATION	**SYSTOLIC BLOOD PRESSURE (mmHg)**	**DIASTOLIC BLOOD PRESSURE (mmHg)**
Normal	<120	<80
Pre hypertension	120-139	80-89
Stage-I Hypertension	140-159	90-99
Stage-II Hypertension	≥160	≥100

IMPORTANCE OF SYSTOLIC BLOOD PRESSURE:

Impressive evidence has accumulated to warrant greater attention to the importance of SBP as a major risk factor for cardiovascular disorders. Changing patterns of BP occurs with increasing age. The rise in SBP continues throughout life in contrast to diastolic blood pressure, which rises until approximately till 50 years and then level off over the next decade, and may remain the same or fall later in life.[30]

Diastolic hypertension predominates before the age 50, either alone or in combination with SBP elevation. The prevalence of systolic hypertension increases with age and above 50 years of age systolic hypertension represents the most common form of hypertension.

Number of important casual factors for hypertension have been identified, including excess body weight, excess dietary intake of sodium, reduced physical activity and excess alcohol intake.[31,32]To prevent BP levels from rising, primary preventive measures should be introduced to reduce these casual factors in the population, particularly in individuals with pre hypertension.

It is estimated that a 5mm Hg reduction of SBP in the population would result in 14 percent overall reduction in mortality due to stroke and 9 percent reduction in mortality due to CHD, and a 7 percent decrease in all- cause mortality.[32,33]

HEARING LOSS IN HYPERTENSIVE PATIENTS[7]:

Essential hypertension affects vast majority of hypertensive patients. Liu in 1988 assessed audiometric function of 32 individuals with hypertension and coronary heart disease and compared with normotensives

"Katz[34] says that all living cells in the human body depend on a proper supply of oxygen and nutrients in order to maintain their function, such supply are dependent on the structural integrity of the heart and blood vessels.[35]Hypertension is the most common vascular disorder which facilitates structural changes in the heart and blood vessels. High pressure in the vascular system may cause inner ear haemorrhage, which is supplied by the anterior inferior cerebellar artery, it divides in to anterior vestibular artery and cochlear artery and supply inner ear.[36] They cause progressive or sudden hearing loss.[34: 35]The circulatory system pathology may directly affect hearing in number of ways. One of the vascular pathophysiological mechanisms described is the Increase in blood viscosity, which reduces capillary blood flow and ends up reducing oxygen transport, causing tissue hypoxia thus causing hearing complaints and hearing loss in patients.[36]Moreover arterial hypertension may cause ionic changes in cell potentials, thus causing hearing loss[8]."

TINNITUS[37]:

HISTORICAL OVERVIEW:

The term tinnitus comes from the Latin word tinnire meaning to ring, and was introduced by pliny the Elder (AD23-79)[38].It was adopted in to the English language by Blanchard's physicians dictionary in the year 1693[39].however, a reference to tinnitus, as "ringing in the ears", dates back to the compendium of 1240, the first complete English medical text written by Gilbertus Angelicus (1180-1250)[39], and more recently to the seventeenth century in Francis bacons Sylva Sylvarum (1627)[40].

Tinnitus is defined as "a sound perceived for more than five minutes at a time in the absence of any external acoustical or electrical stimulation of the ear and not occurring immediately after exposure to loud noise.[41] phantom auditory perception.[42] or head noise.[43]

Tinnitus is described as subjective, audible only to the patient, or objective, audible to the examiner as well.

Relation to hearing impairment:

Tinnitus prevalence rises with increasing hearing loss[44] with74% of patients complaining of hearing loss having tinnitus.[45] In about 50% of population with self-reported tinnitus has been judged as normal.[46]

Pathophysiological overview:

The operating status of auditory system is maintained by a complex feedback mechanism which involves the afferent, ascending and the efferent descending pathways. The auditory pathway is also linked with other extra-auditory structures including reticular-serotonergic.[47] Somatosensory, hypothalamic and limbic systems allowing its integration with the central nervous system network.

The afferent pathway which provides an input to the proximal structures of the auditory system facilitates predominantly excitatory processes, while the parallel efferent pathway, which modulates acoustic information, facilitates predominantly inhibitory processes.

Mechanism of tinnitus:

It can be assumed that the alterations in spontaneous activity leading to tinnitus arise from changes in the balance between excitation and inhibition within the auditory system through different underlying mechanisms.

Abnormal afferent excitation at the cochlear level:

Mechanical tinnitus based on spontaneous cochlear oscillations.

Glutamate neurotoxicity

Modulation of NMDA and non-NMDA receptors

Abnormal ion channel conductance-calcium channel dysfunction

Efferent dysfunction/reduction of GABA effect

Alteration of spontaneous activity and tonotopic

Reorganization

Stress/psychological disorder

MANAGEMENT:

A similar management strategy applied in adults can also be used in children. According to some reports reassurance and counselling seem to play the most important part[48]. White noise generators are also useful, while hearing aids seem to be of limited value[48].Successful pharmacological treatment using intravenous has also been reported nevertheless there have been no controlled trails to verify proposed therapies and to indicate the most effective treatment of childhood tinnitus.

Rosen et al, in1962 carried out a study on hypertensive patients. There found a correlation between high blood pressure and hearing loss in high frequencies.[5]

Marchiori et al, conducted a study in 2006, involving 154 cases and 154 controls, aged 45 to 64 years found that there is significant association between blood pressure and hearing loss.[7]

Agarwal et al conducted a study in 2013, with 150 cases and 124 controls. The age ranged between 45 to 64 years. This study showed significant association between hypertension and increase in hearing threshold. The increase in hearing threshold was most marked among those with grade 3 hypertension particularly at higher frequencies.[49]

Mishra et al did a case control study in 2014. In this study there were 150 cases and 150 controls of both genders and they were in age group of 45 to 65 years. This study showed that hypertensive patients had five times risk of developing hearing loss than a normal person without hypertension.[3]

Mondolli et al conducted a study in 2009 to verify the relationship between systemic hypertension and hearing loss. In this study there were 392 patients of both genders, aged from45 to 60 years old. Their study showed an evident association between systemic arterial hypertension and hearing loss.[50]

According to yeoh LH hypertension is one of the causes of hearing loss.[51]

According to Baraldi GS et al, systemic arterial hypertension and hearing loss have important prevalence in the elderly population.[52]

Nazer et al.in a study carried out with controlled chronic hypertensive patients, without diabetes and intense exposure to intense noise and ototoxic drugs for at least 3years, observed that of 217 controlled chronic hypertensive patients presented with hearing alterations with varied audiogram profiles, the author stated that individual predisposing factors structural or metabolic may in isolated cases causes hearing loss which doesn't represent habitual development in chronic hypertensive patients.[53]

Brohem et al. assessed audio metrically 50 hypertensive patients with ages above 45 years in Brazil, and 62% of those had Sensorineural hearing loss.[54]

Rey et al. tested 59 patients with mean age of 75 years and found significant negative relation with hypertension.[55]

In case-controlled study carried at Kenya with 50 elderly individuals using absolute bone conduction test, Chen et al.observed a relation between hearing loss and arterial hypertension in this population[56].

Amstutz-Montadert et al.reported that in cases of early presbycusis[57],around 55 years of age, fast evolution of metabolic or vascular disorders, use of ototoxic drugs, nicotine and exposure to noise must be investigated, since all contribute to worsen cases of presbycusis.

MATERIALS AND METHODS

The present study entitled “A STUDY OF HEARING IMPAIRMENT AMONG PATIENTS WITH ESSENTIAL HYPERTENSION” is a descriptive cross sectional study conducted at a tertiary care centre.

Type of study : Descriptive cross sectional study

Study period : 6 months

Settings : Department of Otorhinolaryngology and Head and Neck surgery and

Hypertension clinic, Department of Medicine, of a tertiary care centre.

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Sample size:

Sample size in the present study was 120 essential hypertensive patients.

Type of study:

Descriptive cross-sectional study

INCLUSION CRITERIA: patients with essential hypertension

1. Patients in the age group of 20 to 60 years of both sexes.

2. The above criteria include patients with essential hypertension on medications as well as newly diagnosed patients.

3. Patients on only hypertensive medication.

EXCLUSION CRITERIA:

1. Family history of deafness

2. Aural discharge and deafness

3. Congenital deafness

4. Noise induced hearing loss

5. Previous otological surgery

6. Pregnant and lactating mothers

7. Obesity and diabetics

8. Alcoholics and smokers

9. Patients with renal disorders

10. Patients taking drugs for other associated illness with possible ototoxic effects.

METHODOLOGY

This study is based on the analysis of 120 patients with essential hypertension aged between 20 to 60 years attending the Department of Otorhinolaryngology and Heads and Neck surgery and Hypertension clinic of Department of Medicine in a tertiary care centre. All were screened for deafness using tuning fork tests and pure tone audiogram, along with general physical examination, ear nose, throat examination and laboratory tests after applying the inclusion and exclusion criteria and in those willing for giving informed consent.

All patients agreeing for taking part in the study were explained in detail about the procedure, and informed consent was obtained for the same. Detailed history is taken with emphasis on the hypertensive medications being taken (class of drugs, dosage, and duration of treatment).Detailed ear, nose and throat examination which includes otoscopic examination and tuning fork tests to rule out external and middle ear diseases.

Systemic examination (cardiovascular, neurological, renal, hepatobiliary, endocrine, loco motor system) are also carried out for the patients.

After clinical examination was done, patient's blood pressure was measured in sitting position using a manual sphygmomanometer. After which all patients were subjected to audiological evaluation. Air conduction and bone conduction tests were performed, pure tone average was calculated for three frequencies of 500Hz, 1000Hz, 2000Hz, for both the right and the left ear and plotted in a graph. Similarly, the air bone gap was calculated for the frequencies 500Hz, 1000Hz, 2000Hz and plotted in a graph.

The collected data were analysed with IBM SPSS Statistics for Windows, Version 23.0.(Armonk, NY: IBM Corp).To describe about the data descriptive statistics frequency analysis, percentage analysis were used for categorical variables and the mean & S.D were used for continuous variables. To assess the relationship between the variables Pearson's Correlation was used.To find the significance in categorical data Chi-Square test was used similarly if the expected cell frequency is less than 5 in 2×2 tables then the Fisher's Exact test was used. In all the above statistical tools the probability value 0.05 is considered as significant level.

RESULTS AND OBSERVATION

The collected data were analysed with IBM SPSS Statistics for Windows, Version 23.0.(Armonk, NY: IBM Corp).To describe about the data descriptive statistics frequency analysis, percentage analysis were used for categorical variables and the mean & S.D were used for continuous variables. To assess the relationship between the variables Pearson's Correlation was used.To find the significance in categorical data Chi-Square test was used similarly if the expected cell frequency is less than 5 in 2×2 tables then the Fisher's Exact was used. In all the above statistical tools the probability value .05 is considered as significant level.

Table 3: Age distribution

Age distribution		
	Frequency	Percent
Upto 30 yrs	2	1.7
31 - 40 yrs	22	18.3
41 - 50 yrs	33	27.5
51 - 60 yrs	63	52.5
Total	120	100.0
Mean ± SD = 50 ± 9 yrs		

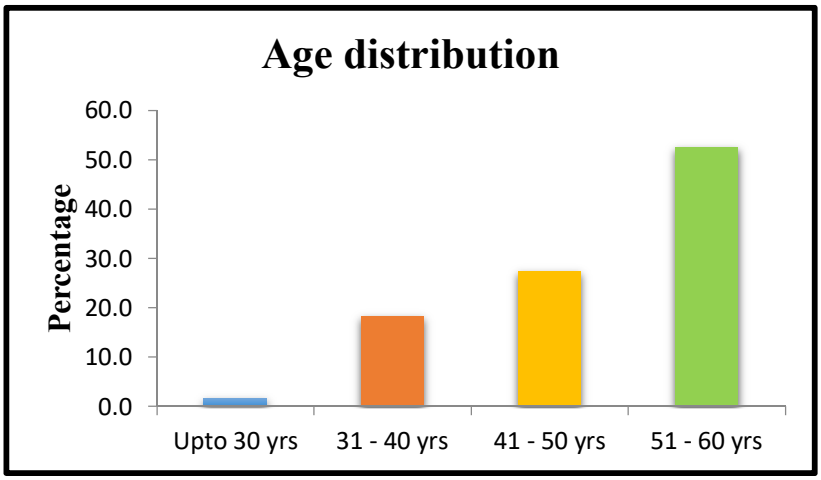


Figure 19

The above table shows Age distribution were <30 years is 1.7%, 31-40 years is 18.3%, 41-50 years is 27.5%, 51-60 years is 52.5%.

Table 4: Gender distribution

Gender distribution		
	Frequency	Percent
Female	53	44.2
Male	67	55.8
Total	120	100.0

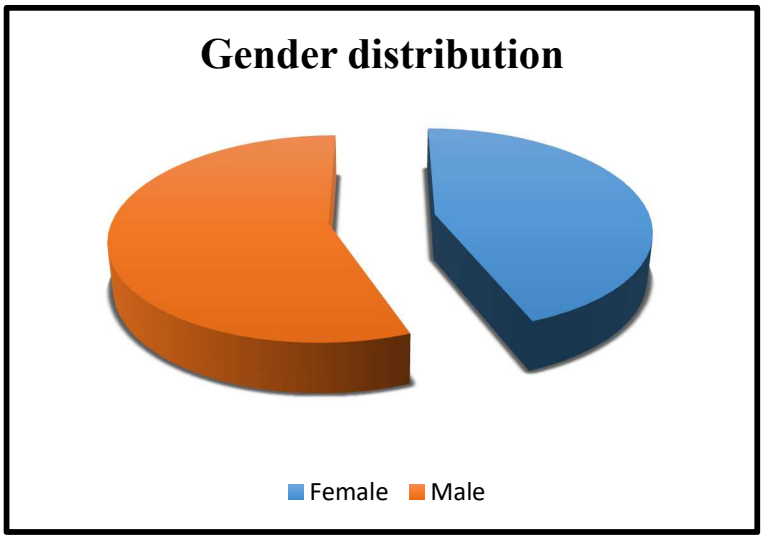


Figure 20

The above table shows Gender distribution where Female distribution was 44.2% and males were 55.8%. Males were predominant in our study.

Table 5: Comparison of Tinnitus between Gender by Pearson's Chi-Square test

			Gender		Total	$\chi 2$ - value	p-value
			Female	Male			
Tinnitus	Absent	Count	17	15	32	1.420	0.233 #
		%	32.1%	22.4%	26.7%		
	Present	Count	36	52	88		
		%	67.9%	77.6%	73.3%		
Total		Count	53	67	120		
		%	100.0%	100.0%	100.0%		
# No Statistical Significance at p > 0.05 level							

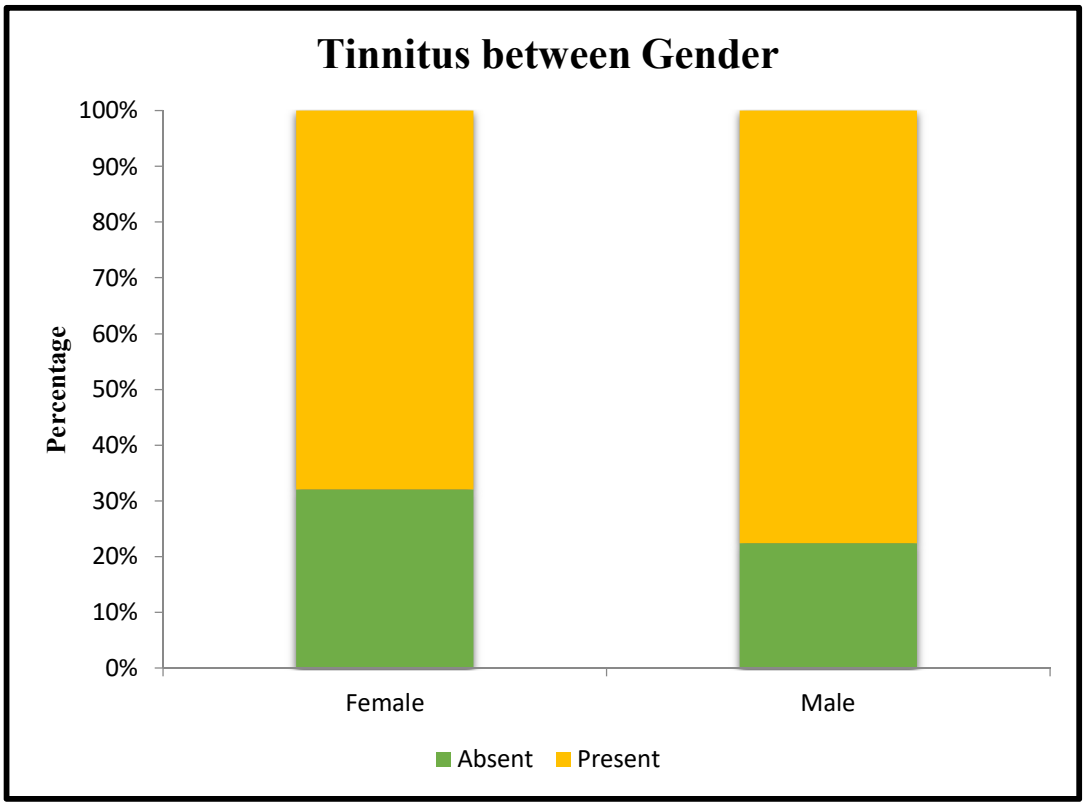


Figure 21

The above table shows comparison of Tinnitus between Gender by Pearson's Chi-Square test were $\chi 2=1.420$, $p=0.233>0.05$ which shows no statistical significance between Tinnitus and Gender. Symptom of tinnitus was present in 73.3% of our study population.

Table 6: Comparison of Hearing Loss between Gender by Pearson's Chi-Square test

			Gender		Total	$\chi 2$ - value	p-value
			Female	Male			
Hearing Loss	Absent	Count	30	28	58	2.600	0.107 #
		%	56.6%	41.8%	48.3%		
	Present	Count	23	39	62		
		%	43.4%	58.2%	51.7%		
Total		Count	53	67	120		
		%	100.0%	100.0%	100.0%		
# No Statistical Significance at p > 0.05 level							

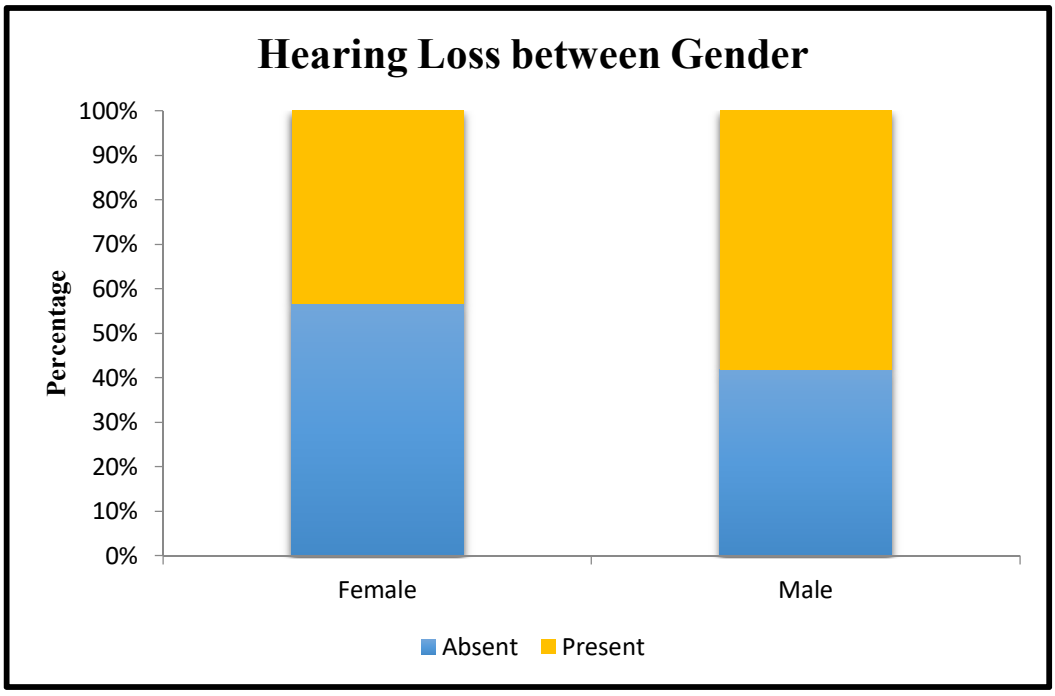


Figure 22

The above table shows comparison of Hearing Loss between Gender by Pearson's Chi-Square test were $\chi 2=2.600$, $p=0.107>0.05$ which shows no statistical significance between Hearing Loss and Gender. Symptom of hearing loss was present in 21.7% of our study population.

Table 7: Comparison of Vertigo between Gender by Fisher's exact test

			Gender		Total	χ 2 - value	p-value
			Female	Male			
Vertigo	Absent	Count	49	65	114	1.297	0.404 #
		%	92.5%	97.0%	95.0%		
	Present	Count	4	2	6		
		%	7.5%	3.0%	5.0%		
Total		Count	53	67	120		
		%	100.0%	100.0%	100.0%		
# No Statistical Significance at p > 0.05 level							

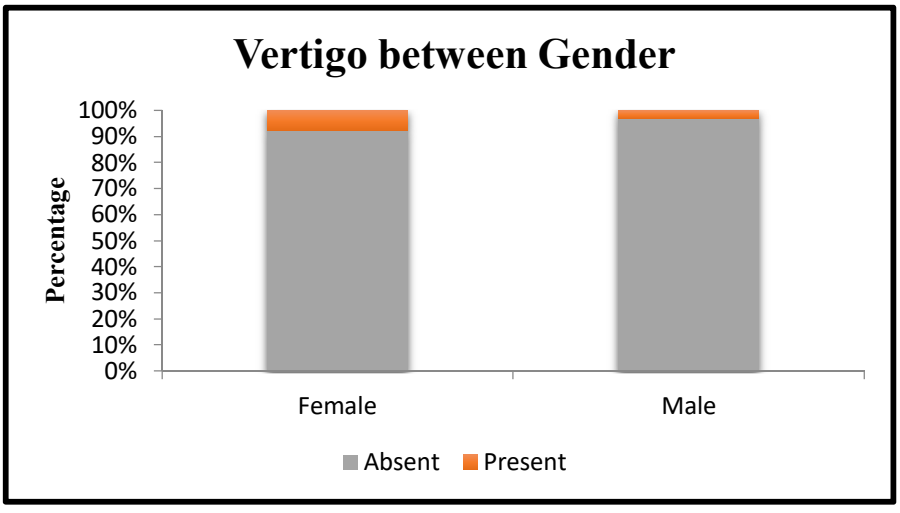


Figure 23

The above table shows comparison of Vertigo between Gender by Fisher's exact test were $\chi 2=1.297$, $p=0.404>0.05$ which shows no statistical significance between Vertigo and Gender.

Symptom of vertigo was present in 5% of our study population.

Table 8: Comparison of Other symptoms between Gender by Pearson's Chi-Square test

			Gender		Total	χ 2 - value	p-value
			Female	Male			
Other Symptoms	Absent	Count	42	57	99	0.696	0.404 #
		%	79.2%	85.1%	82.5%		
	Present	Count	11	10	21		
		%	20.8%	14.9%	17.5%		
Total		Count	53	67	120		
		%	100.0%	100.0%	100.0%		
# No Statistical Significance at p > 0.05 level							

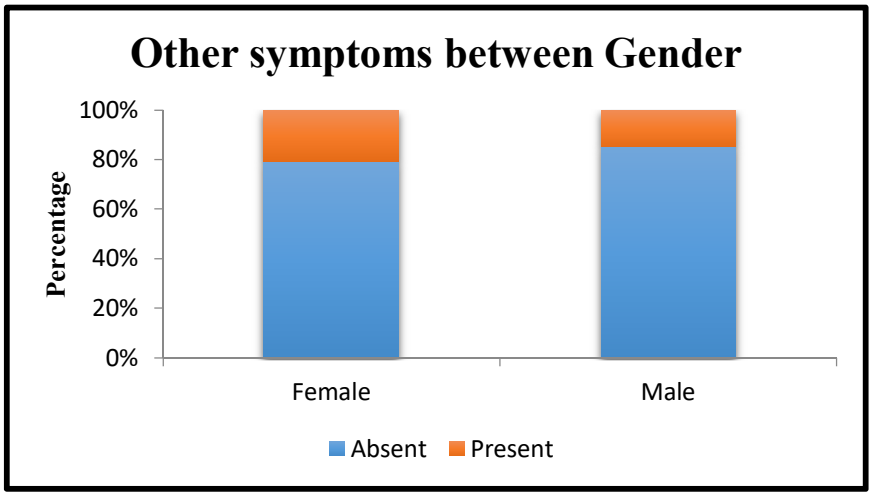


Figure 24

The above table shows comparison of Other symptoms between Gender by Pearson's Chi-Square test were $\chi 2=0.696$, $p=0.404>0.05$ which shows no statistical significance between Others and Gender.

Table 9: Comparison of Otoscopy between Gender by Fisher's exact test

			Gender		Total	$\chi 2$ - value	p-value
			Female	Male			
Otoscopy	Normal	Count	52	66	118	0.028	1.000 #
		%	98.1%	98.5%	98.3%		
	Abnormal	Count	1	1	2		
		%	1.9%	1.5%	1.7%		
Total		Count	53	67	120		
		%	100.0%	100.0%	100.0%		
# No Statistical Significance at p > 0.05 level							

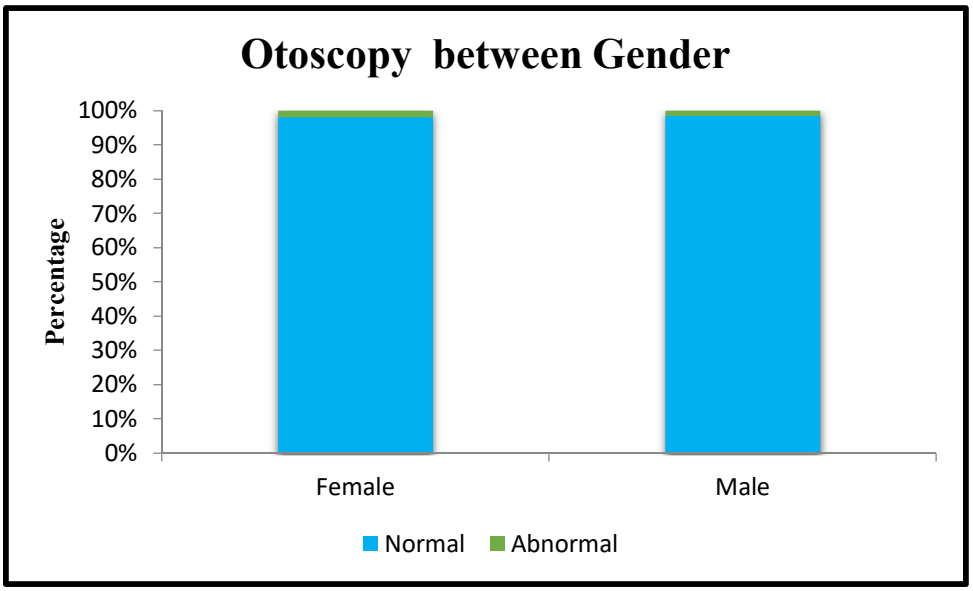


Figure 25

The above table shows comparison of Otoscopy between Gender by Fisher's exact test were $\chi 2=0.028$, $p=1.000>0.05$ which shows no statistical significance between Otoscopy and Gender. 98.3% of patients had normal otoscopic findings and 1.7% had abnormal finding like retracted TM.

Table 10: Comparison of TFT Right between Gender by Pearson's Chi-Square test

			Gender		Total	χ 2 - value	p-value
			Female	Male			
TFT Right	Normal	Count	37	40	77	1.315	0.251 #
		%	69.8%	59.7%	64.2%		
	Abnormal	Count	16	27	43		
		%	30.2%	40.3%	35.8%		
Total		Count	53	67	120		
		%	100.0%	100.0%	100.0%		
# No Statistical Significance at p > 0.05 level							

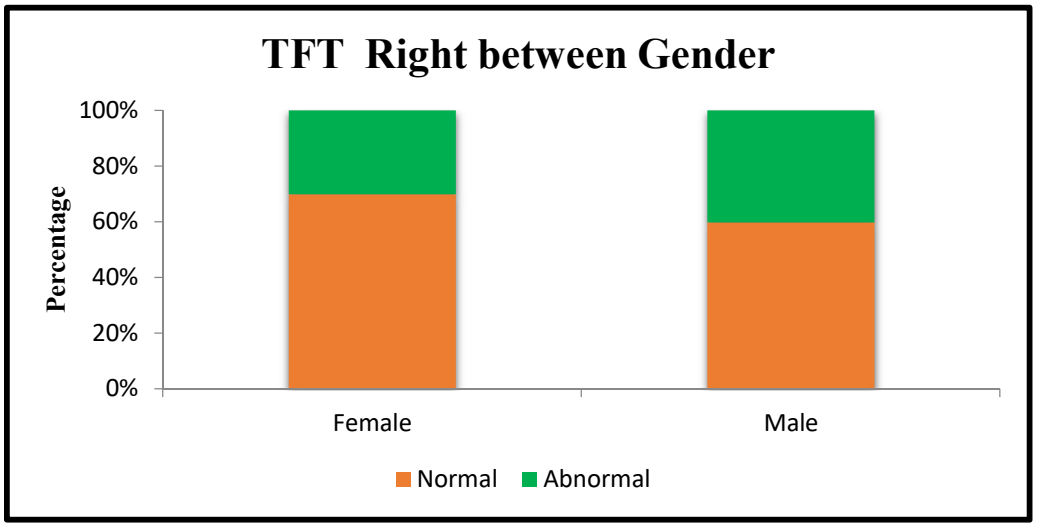


Figure 26

The above table shows comparison of TFT ABC Right between Gender by Pearson's Chi-Square test were $\chi 2=1.315$, $p=0.251>0.05$, which shows no statistical significance between TFT Right and Gender.

Table 11: Comparison of TFT Left between Gender by Pearson's Chi-Square test

			Gender		Total	$\chi 2$ - value	p-value
			Female	Male			
TFT Left	Normal	Count	37	39	76	1.464	0.226 #
		%	69.8%	59.1%	63.9%		
	Abnormal	Count	16	27	43		
		%	30.2%	40.9%	36.1%		
Total		Count	53	66	119		
		%	100.0%	100.0%	100.0%		
# No Statistical Significance at p > 0.05 level							

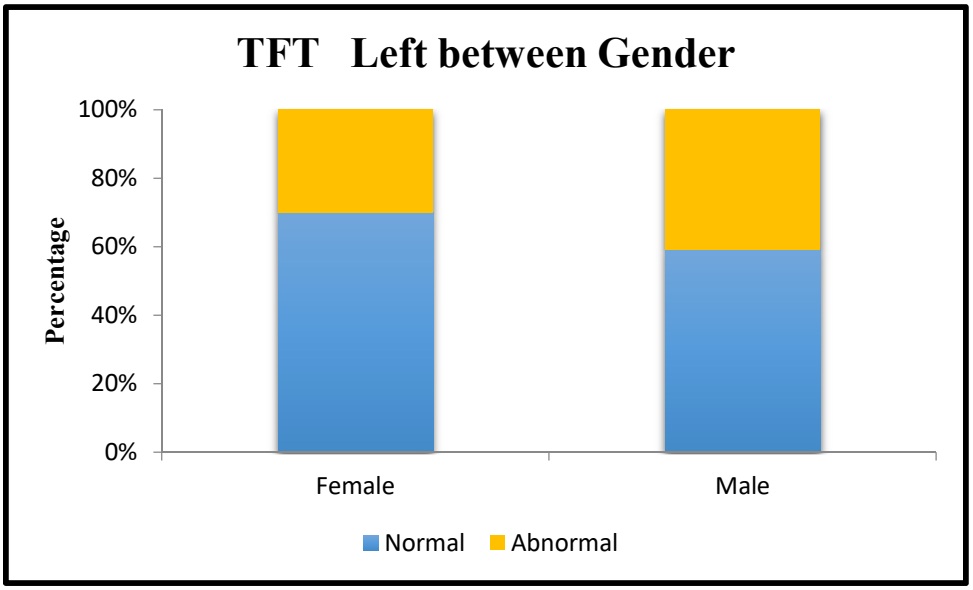


Figure 27

The above table shows comparison of TFT Left between Gender by Pearson's Chi-Square test were $\chi 2=1.464$, $p=0.226>0.05$, which shows no statistical significance between TFT Left and Gender.

Table 12: Comparison of PTA Right between Gender by Pearson's Chi-Square test

			Gender		Total	χ 2 - value	p-value
			Female	Male			
PTA Right	Normal	Count	13	19	32		
		%	24.5%	28.4%	26.7%		
	Mild	Count	23	21	44		
		%	43.4%	31.3%	36.7%		
	Moderate	Count	14	18	32		
		%	26.4%	26.9%	26.7%		
	Moderately severe	Count	2	2	4		
		%	3.8%	3.0%	3.3%		
	Severe	Count	0	3	3		
		%	0.0%	4.5%	2.5%		
	Profound	Count	1	4	5		
		%	1.9%	6.0%	4.2%		
Total		Count	53	67	120		
		%	100.0%	100.0%	100.0%		
# No Statistical Significance at p > 0.05 level							

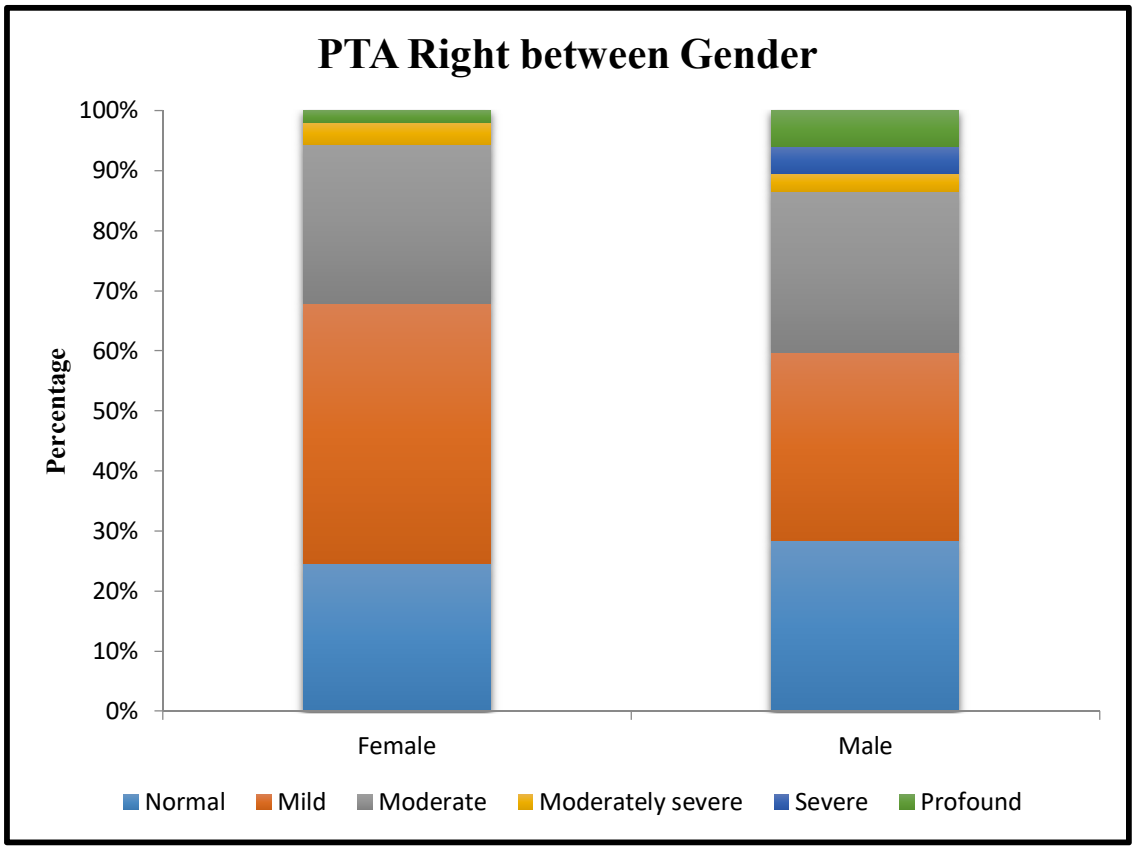


Figure 28

The above table shows comparison of PTA Right between Gender by Pearson's Chi-Square test were $\chi 2=4.950$, $p=0.422>0.05$, which shows no statistical significance between PTA Right and Gender. PTA showed varying degrees of hearing loss in right ear, with mild degree of hearing loss (36.7 %) as the most predominant category.

Table 13: Comparison of PTA Left between Gender by Pearson's Chi-Square test

			Gender		Total	$\chi 2$ - value	p-value
			Female	Male			
PTA Left	Normal	Count	11	18	29	5.257	0.385 #
		%	20.8%	26.9%	24.2%		
	Mild	Count	24	19	43		
		%	45.3%	28.4%	35.8%		
	Moderate	Count	10	21	31		
		%	18.9%	31.3%	25.8%		
	Moderately severe	Count	5	4	9		
		%	9.4%	6.0%	7.5%		
	Severe	Count	1	2	3		
		%	1.9%	3.0%	2.5%		
	Profound	Count	2	3	5		
		%	3.8%	4.5%	4.2%		
Total		Count	53	67	120		
		%	100.0%	100.0%	100.0%		
# No Statistical Significance at p > 0.05 level							

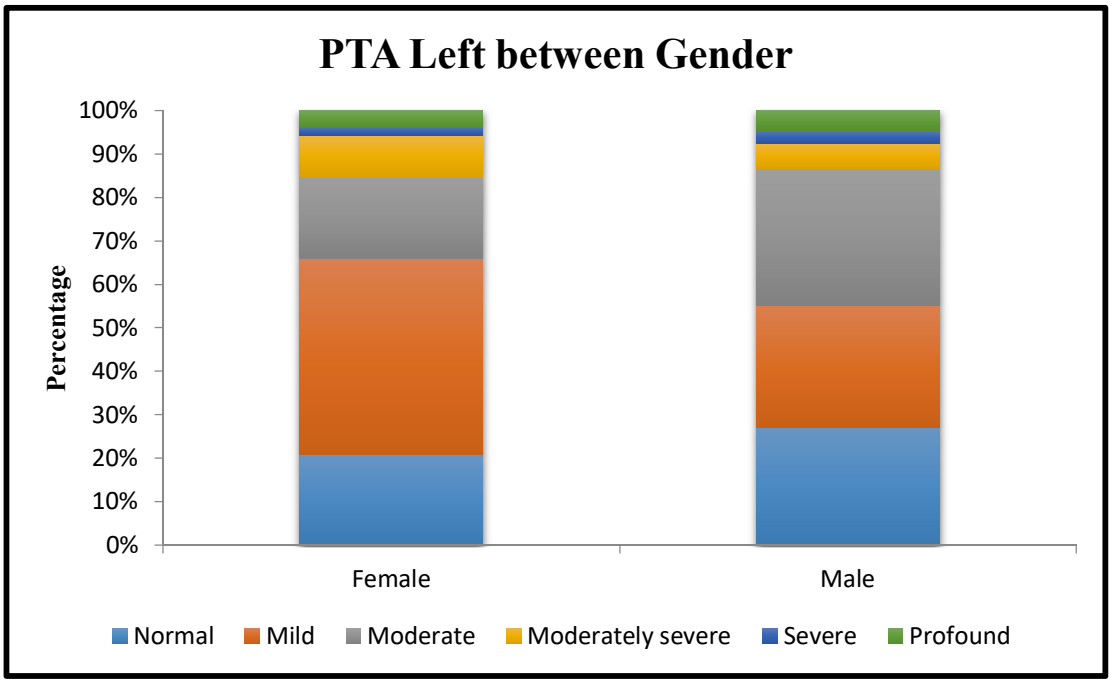


Figure 29

The above table shows comparison of PTA Left between Gender by Pearson's Chi-Square test were $\chi 2=5.257$, $p=0.385>0.05$, which shows no statistical significance between PTA Left and Gender. PTA showed varying degrees of hearing loss in left ear, with mild degree of hearing loss (35.8 %) as the most predominant category

Table 14: Comparison of Systolic BP between Gender by Fisher's exact test

			Gender		Total	χ 2 - value	p-value
			Female	Male			
Systolic BP	120 - 139	Count	52	65	117	0.146	1.000 #
		%	98.1%	97.0%	97.5%		
	140 - 159	Count	1	2	3		
		%	1.9%	3.0%	2.5%		
Total		Count	53	67	120		
		%	100.0%	100.0%	100.0%		
# No Statistical Significance at p > 0.05 level							

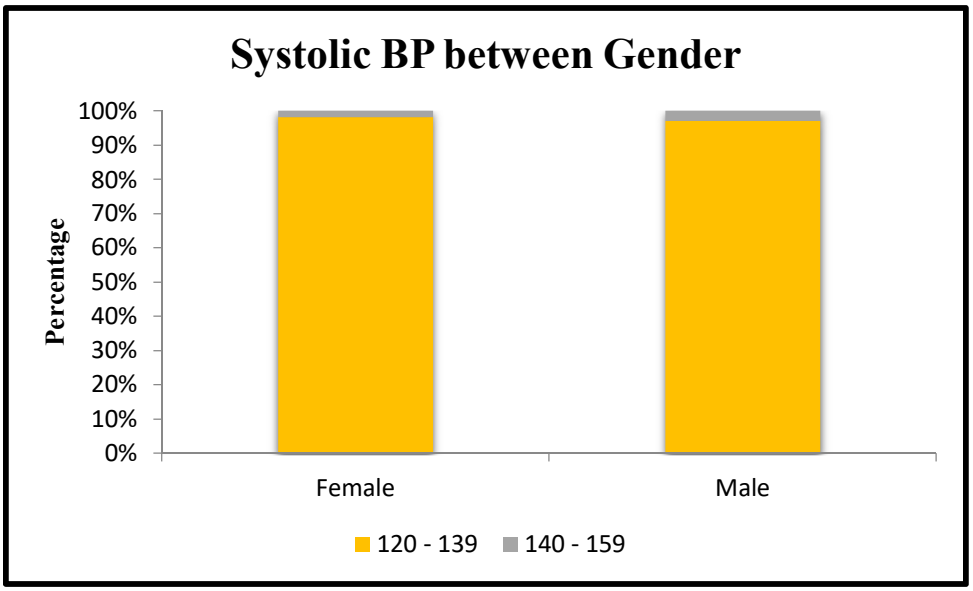


Figure 30

The above table shows comparison of Systolic BP between Gender by Fisher's exact test were $\chi 2=0.146$, $p=1.000>0.05$, which shows no statistical significance between Systolic BP and Gender.

Table 15: Comparison of Diastolic BP between Gender by Pearson's Chi-Square test

			Gender		Total	$\chi 2$ - value	p-value
			Female	Male			
Diastolic BP	80 - 89	Count	43	50	93	0.718	0.397 #
		%	81.1%	74.6%	77.5%		
	90 - 99	Count	10	17	27		
		%	18.9%	25.4%	22.5%		
Total		Count	53	67	120		
		%	100.0%	100.0%	100.0%		
# No Statistical Significance at p > 0.05 level							

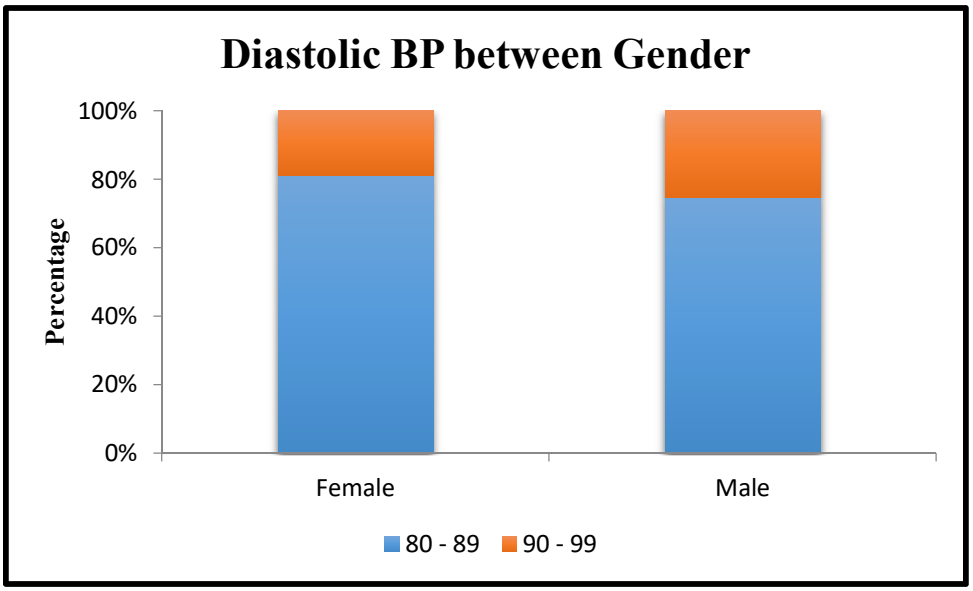


Figure 31

The above table shows comparison of Diastolic BP between Gender by Pearson's Chi-Square test were $\chi 2=0.718$, $p=0.397>0.05$, which shows no statistical significance between Diastolic BP and Gender.

Table 16: Comparison of Duration/Months between Gender by Pearson's Chi-Square test

			Gender		Total	$\chi 2$ - value	p-value
			Female	Male			
Duration/Months	< 10	Count	13	16	29	0.007	0.996 #
		%	24.5%	23.9%	24.2%		
	10-40	Count	33	42	75		
		%	62.3%	62.7%	62.5%		
	> 40	Count	7	9	16		
		%	13.2%	13.4%	13.3%		
Total		Count	53	67	120		
		%	100.0%	100.0%	100.0%		
# No Statistical Significance at p > 0.05 level							

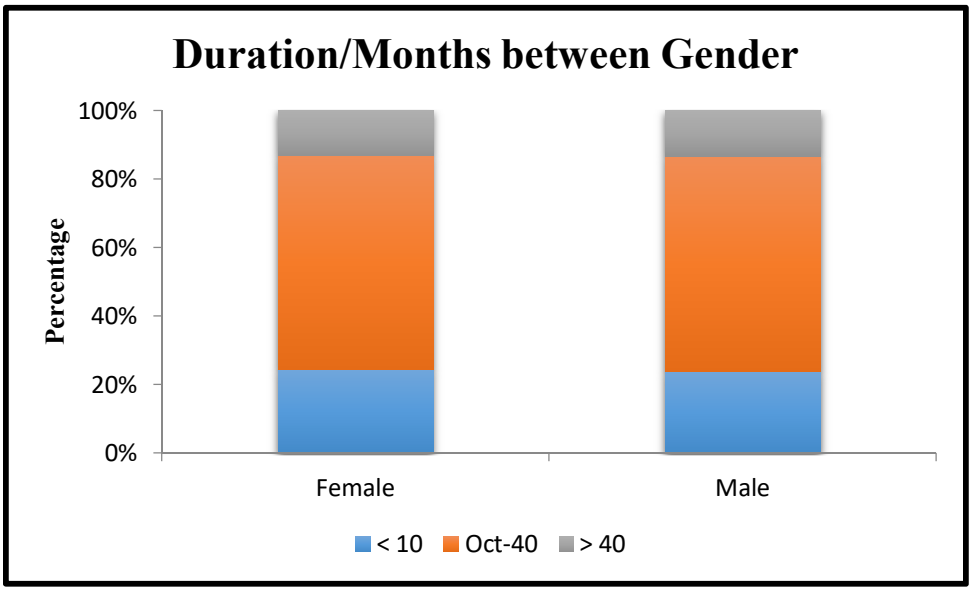


Figure 32

The above table shows comparison of Duration/Months between Gender by Pearson's Chi-Square test were $\chi 2=0.007$, $p=0.996>0.05$, which shows no statistical significance between Duration/Months and Gender. Most common was a duration of 10 – 40 months from onset of hypertension in both sexes.

TABLE 17: Comparison of duration and degree of hypertension with degree of hearing loss.

Duration of HTN	Degree of HTN	Degree of hearing loss	Laterality		Total
			Unilateral	Bilateral	
Short	Pre HTN	Mild	2	8	10
		Profound	0	2	2
		Total	**2**	**10**	**12**
	Stage I	Mild	0	2	2
		Moderate	0	1	1
		Total	0	3	**3**
Moderate	Pre HTN	Mild	0	15	15
		Moderate	0	17	17
		Moderately severe	0	5	5
		Severe	0	2	2
		Profound	1	4	5
		Total	**1**	**43**	**44**
	Stage I	Mild	0	8	8
		Moderate	0	8	8
		Moderately severe	0	1	1
		Profound	0	1	1
		Total	**0**	**18**	**18**
Long	Pre HTN	Mild	0	1	1
		Moderate	0	5	5
		Moderately severe	0	4	4
		Severe	0	2	2
		Profound	0	1	1
		Total	**0**	**13**	**13**
	Stage I	Mild	0	1	1
		Moderately severe	0	1	1
		Total	**0**	**2**	**2**

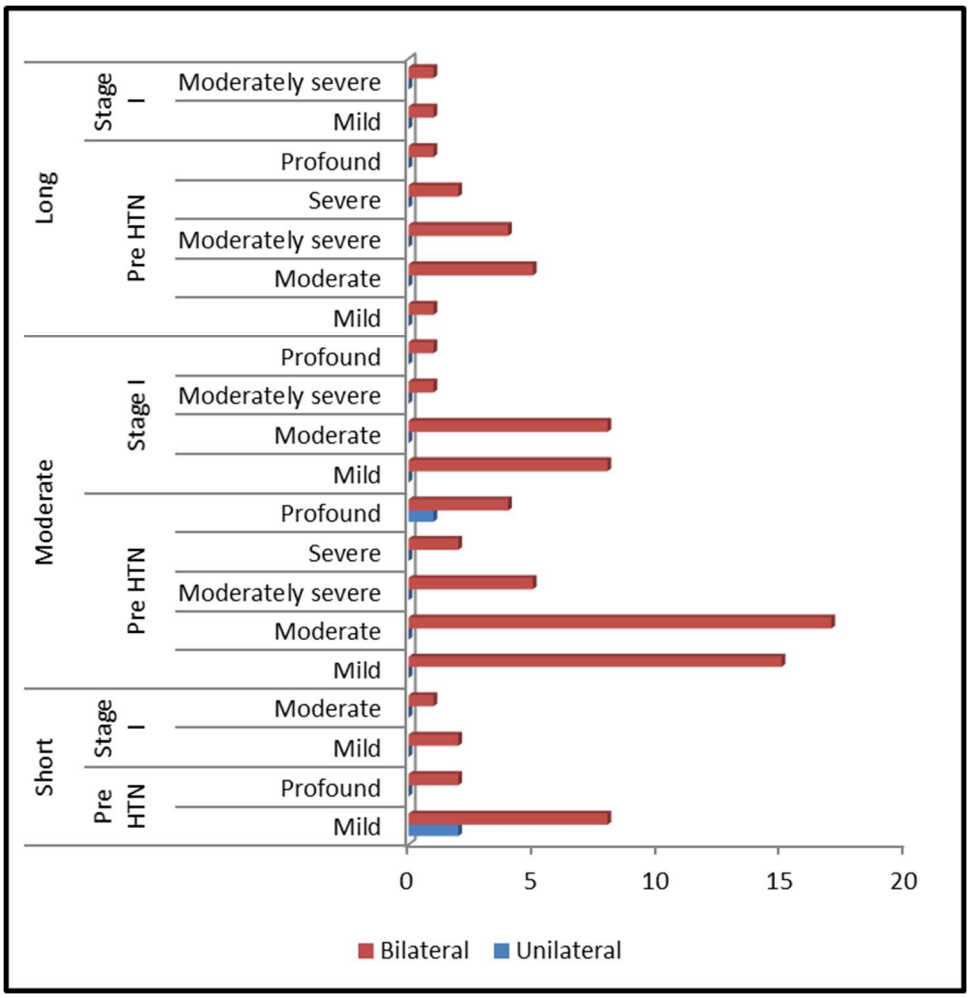


Figure 33

The above table shows the degree of hypertension with degree of hearing loss and the number of patients affected in each category. Patients in our study presented with varying duration of hypertension among which the moderate duration of hypertension i.e. between 10- 40 months was most common and degree of hypertension was most commonly in the pre hypertension category, i.e. SBP between 120 – 139mmHg and DBP between 80 – 89mmHg. Mild degree of hearing loss was the most common degree of hearing loss in our study.

TABLE 18: Comparison of duration of hypertension with degree of hearing loss in right ear.

			Duration/Months			Total	$\chi 2$ - value	p-value
			< 10	10 - 40	> 40			
PTA Right	Normal	Count	16	15	1	32	43.329	0.0005 **
		%	55.2%	20.0%	6.3%	26.7%		
	Mild	Count	10	30	4	44		
		%	34.5%	40.0%	25.0%	36.7%		
	Moderate	Count	1	25	6	32		
		%	3.4%	33.3%	37.5%	26.7%		
	Moderately severe	Count	0	3	1	4		
		%	0.0%	4.0%	6.3%	3.3%		
	Severe	Count	0	0	3	3		
		%	0.0%	0.0%	18.8%	2.5%		
	Profound	Count	2	2	1	5		
		%	6.9%	2.7%	6.3%	4.2%		
Total		Count	29	75	16	120		
		%	100.0%	100.0%	100.0%	100.0%		
** Highly Statistical Significance at p < 0.01 level								

The above table shows the comparison of duration of hypertension with degree of hearing loss in the right ear by Pearson's Chi-Square test with p value 0.0005< 0.01 which is highly significant. Based on pure tone audiogram report obtained in our study,it indicates that 73.3% of patients had sensorineural hearing loss in right ear. It suggests that duration of hypertension and degree of hearing loss has a definite association.

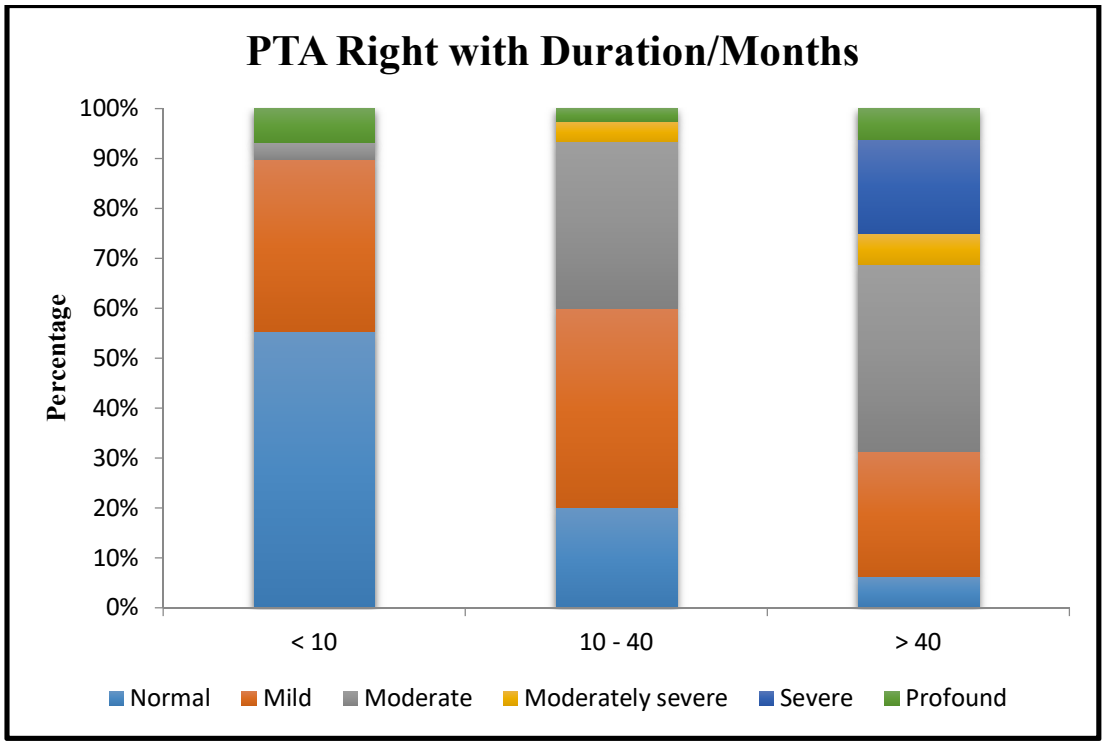


Figure 34

TABLE 19: Comparison of duration of hypertension with degree of hearing loss in left ear.

			Duration/Months			Total	$\chi 2$ - value	p-value
			< 10	10 - 40	> 40			
PTA Left	Normal	Count	15	13	1	29	39.117	0.0005 **
		%	51.7%	17.3%	6.3%	24.2%		
	Mild	Count	12	28	3	43		
		%	41.4%	37.3%	18.8%	35.8%		
	Moderate	Count	1	24	6	31		
		%	3.4%	32.0%	37.5%	25.8%		
	Moderately severe	Count	0	4	5	9		
		%	0.0%	5.3%	31.3%	7.5%		
	Severe	Count	0	2	1	3		
		%	0.0%	2.7%	6.3%	2.5%		
	Profound	Count	1	4	0	5		
		%	3.4%	5.3%	0.0%	4.2%		
Total		Count	29	75	16	120		
		%	100.0%	100.0%	100.0%	100.0%		
** Highly Statistical Significance at p < 0.01 level								

The above table shows the comparison of duration of hypertension with degree of hearing loss in the left ear by Pearson's Chi-Square test with p value 0.0005< 0.01 which is highly significant. •

Based on pure tone audiogram report obtained in our study indicates that 75.8% of patients had sensorineural hearing loss in left ear. It suggests that duration of hypertension and degree of hearing loss has a definite association.

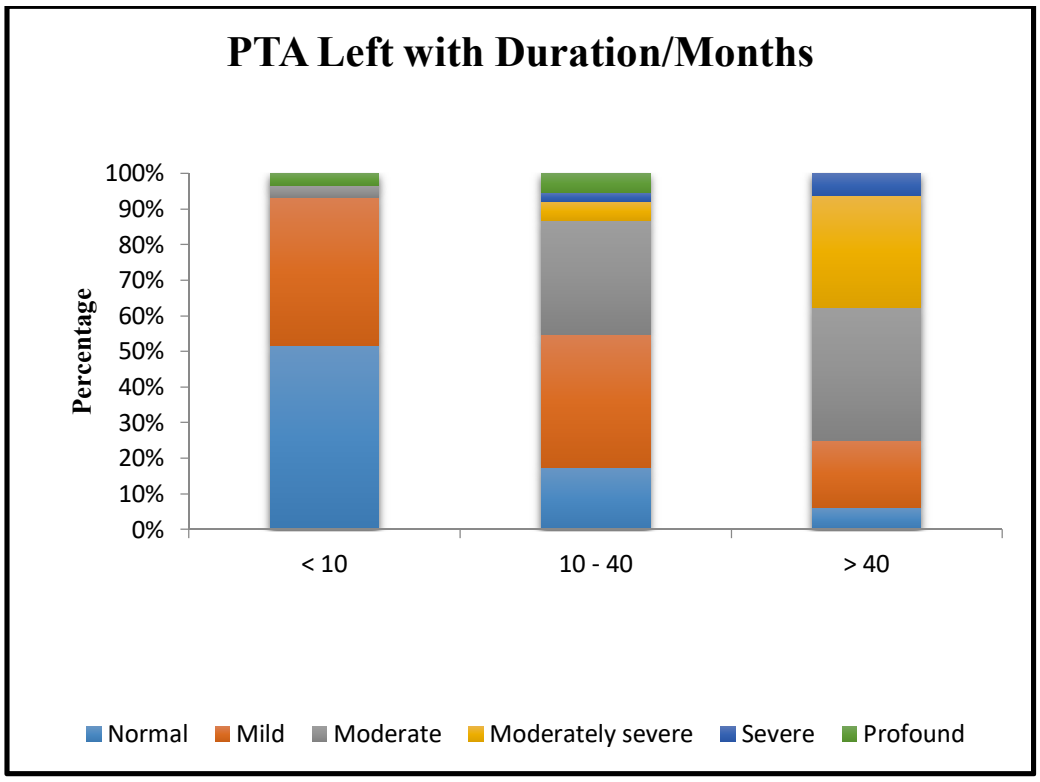


Figure 35

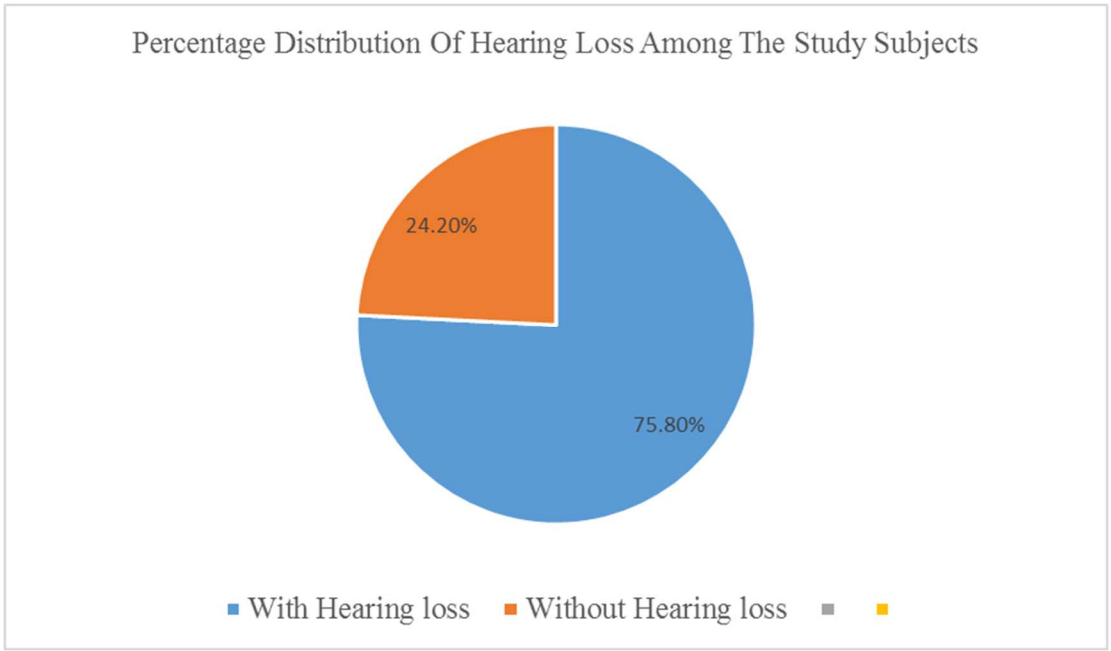


Figure 36

The above pie chart shows percentage distribution of hearing loss among the study subjects, 75.80 %(91) of subjects had hearing loss and 24.20 % (29) of subjects had no hearing loss.

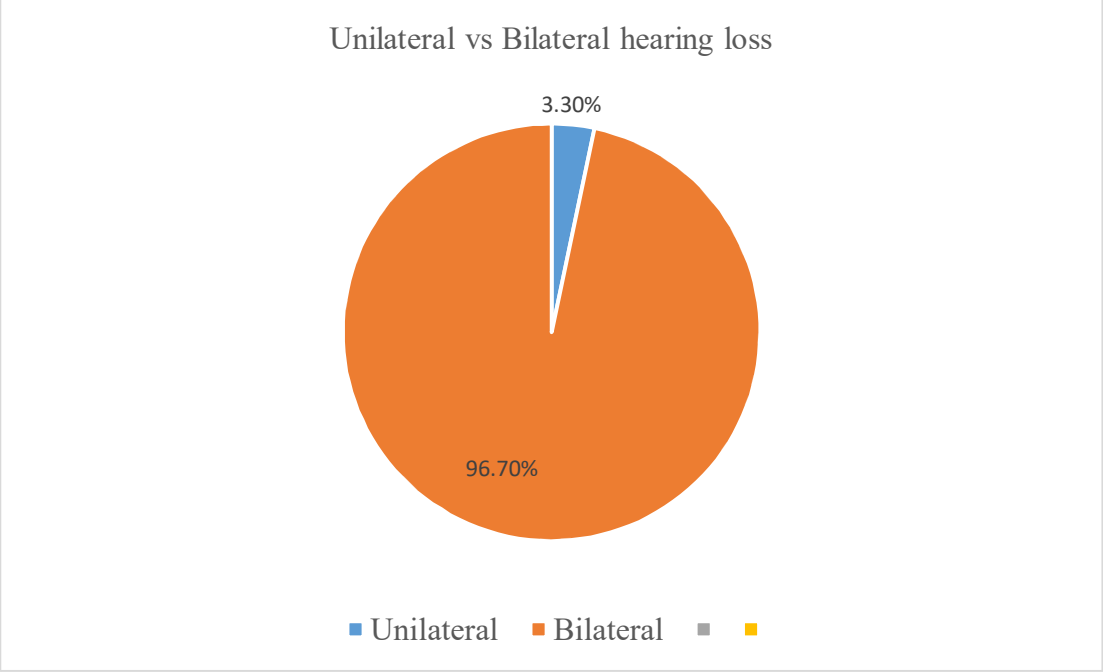


Figure 37

The above pie chart shows percentage distribution of unilateral vs bilateral hearing loss among the study subjects, 96.70% (88) of subjects had bilateral hearing loss and 3.30% (3) of subjects had unilateral hearing loss. Majority of the patients had bilateral hearing loss.

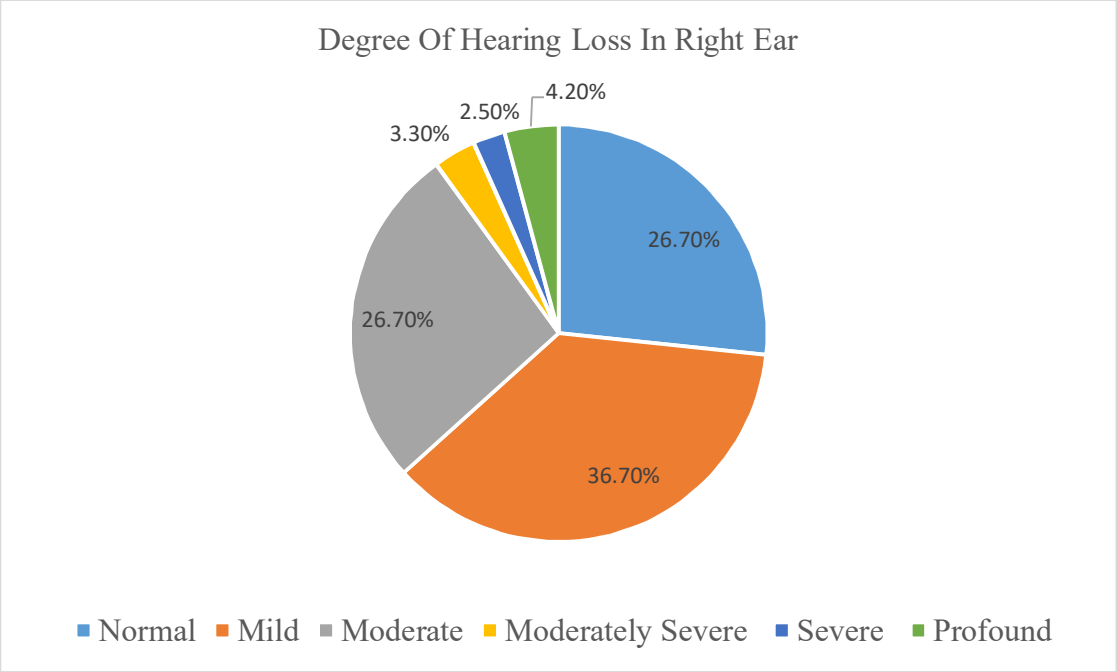


Figure 38

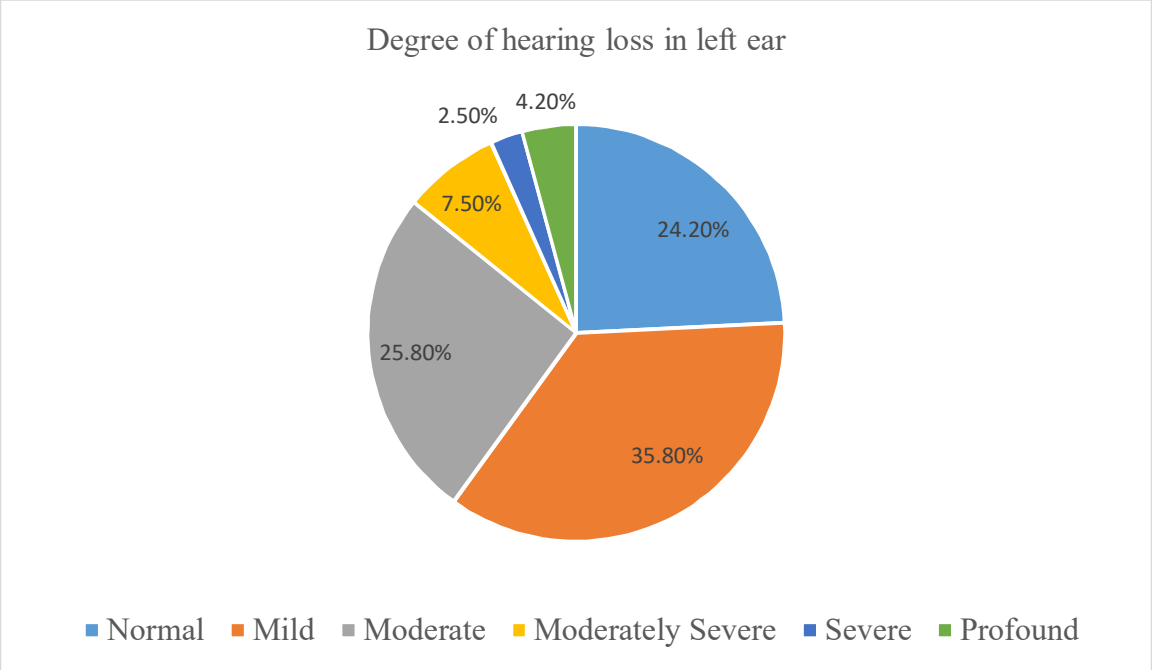


Figure 39

The above pie chart shows varying degree of hearing loss in both ears. In right ear, mild degree of hearing loss was 36.70% was the predominant type and in left ear, mild degree of hearing loss was 35.80% was predominant type.

Table 20: Correlation of PTA with SBP

PTA		SBP
Right	r-value	.548**
	p-value	.0005 **
	N	120
Left	r-value	.572**
	p-value	.0005 **
	N	120

The above table shows Correlation of PTA with SBP were Right r-value=0.548, p-value=0.0005<0.01, which shows highly statistical significance positive Correlation difference at p <0.01 level, whereas in Left r-value=0.572, p-value=0.0005<0.01, which shows highly statistically significant positive Correlation difference at p <0.01 level respectively.

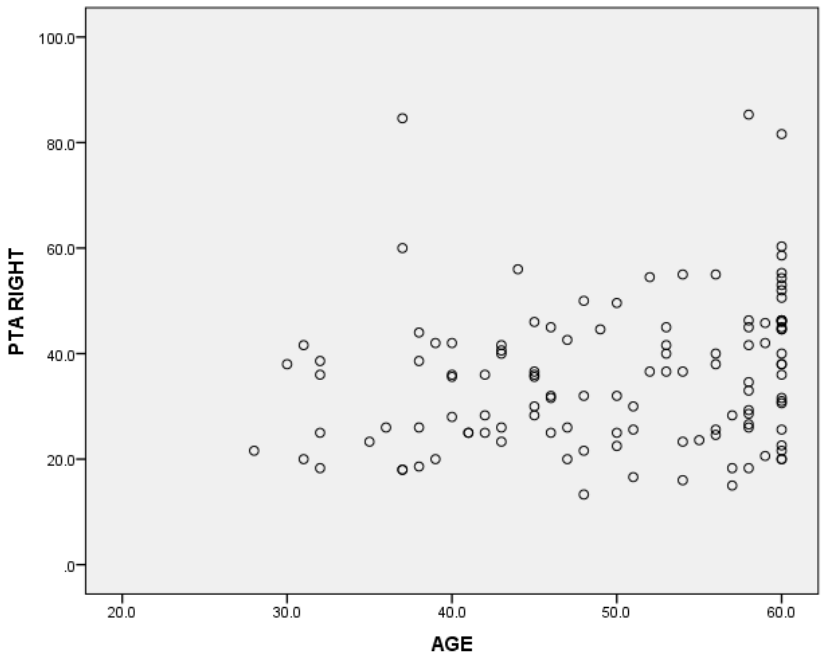
Table 21: Correlation of PTA with DBP

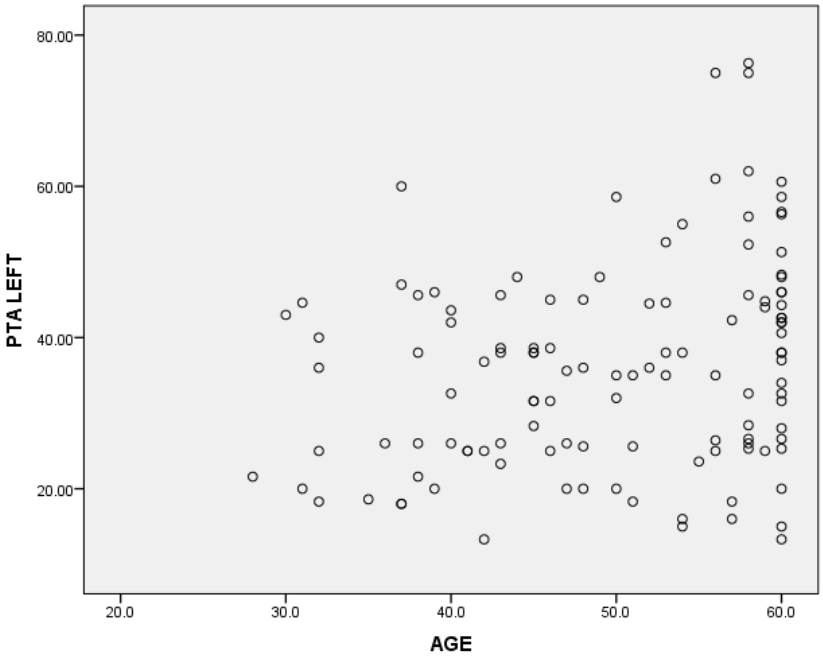
PTA		DBP
Right	r-value	.225*
	p-value	.014 *
	N	120
Left	r-value	.289**
	p-value	.001 **
	N	120

The above table shows Correlation of PTA with DBP were Right r-value=0.225, p-value=0.014<0.01, which shows statistical significance positive Correlation difference at p <0.05 level, whereas in Left r-value=0.289, p-value=0.001<0.01, which shows highly statistically significant positive Correlation difference at p <0.01 level respectively.

Table 22: Correlation of PTA with Age

PTA		Age
Right	r-value	.200*
	p-value	.028 *
	N	120
Left	r-value	.255**
	p-value	.005 **
	N	120


PTA RIGHT
100.0
80.0
60.0
40.0
20.0
.0
20.0
30.0
40.0
50.0
60.0
AGE


PTA LEFT
80.00
60.00
40.00
20.00
20.0
30.0
40.0
50.0
60.0
AGE

The above table shows Correlation of PTA with Age were Right r-value=0.200, p-value=0.028<0.01, which shows statistical significance positive Correlation difference at p <0.05 level, whereas in Left r-value=0.255, p-value=0.005<0.01, which shows highly statistical significance positive Correlation difference at p <0.01 level respectively.

Summary

- The Age distribution were <30 years is 1.7%, 31-40 years is 18.3%, 41-50 years is 27.5%, 51-60 years is 52.5%.
- The Gender distribution were Female is 44.2%, Male is 55.8%.
- The Tinnitus between Gender by Pearson's Chi-Square test were $\chi 2=1.420$, p=0.233>0.05 which shows no statistical significance between Tinnitus and Gender.
- The Hearing Loss between Gender by Pearson's Chi-Square test were $\chi 2=2.600$, p=0.107>0.05 which shows no statistical significance between Hearing Loss and Gender.
- The Vertigo between Gender by Fisher's exact test were $\chi 2=1.297$, p=0.404>0.05 which shows no statistical significance between Vertigo and Gender.
- The Other symptoms between Gender by Pearson's Chi-Square test were $\chi 2=0.696$, p=0.404>0.05 which shows no statistical significance between Others and Gender.
- The Otoscopy between Gender by Fisher's exact test were $\chi 2=0.028$, p=1.000>0.05 which shows no statistical significance between Otoscopy R and Gender.

- The TFT Right between Gender by Pearson's Chi-Square test were $\chi 2=1.315$, $p=0.251>0.05$, which shows no statistical significance between TFT ABC Right and Gender.
- The TFT Left between Gender by Pearson's Chi-Square test were $\chi 2=1.464$, $p=0.226>0.05$, which shows no statistical significance between TFT ABC Left and Gender.
- The PTA Right between Gender by Pearson's Chi-Square test were $\chi 2=4.950$, $p=0.422>0.05$, which shows no statistical significance between PTA Right and Gender.
- The PTA Left between Gender by Pearson's Chi-Square test were $\chi 2=5.257$, $p=0.385>0.05$, which shows no statistical significance between PTA Left and Gender.
- The Systolic BP between Gender by Fisher's exact test were $\chi 2=0.146$, $p=1.000>0.05$, which shows no statistical significance between Systolic BP and Gender.
- The Diastolic BP between Gender by Pearson's Chi-Square test were $\chi 2=0.718$, $p=0.397>0.05$, which shows no statistical significance between Diastolic BP and Gender.
- The Duration/Months between Gender by Pearson's Chi-Square test were $\chi 2=0.007$, $p=0.996>0.05$, which shows no statistical significance between Duration/Months and Gender.
- The comparison of duration of hypertension with hearing loss in the right ear by Pearson's Chi Square test were $\chi 2=43.329$, $p=0.0005<0.001$ which showed highly statistical significance .
- The comparison of duration of hypertension with hearing loss in the left ear by Pearson's Chi Square test were $\chi 2= 39.117$, $p=0.0005<0.001$ which showed highly statistical significance.
- The PTA with SBP were Right r-value=0.548, p-value=0.0005<0.01, which shows highly statistical significance positive Correlation difference at $p <0.01$ level, whereas in Left r-value=0.572, p-value=0.0005<0.01, which shows highly statistical significance positive Correlation difference at $p <0.01$ level respectively.

- The PTA with DBP were Right r-value=0.225, p-value=0.014<0.01, which shows statistical significance positive Correlation difference at p <0.05 level, whereas in Left r-value=0.289, p-value=0.001<0.01, which shows highly statistically significant positive Correlation difference at p <0.01 level respectively.
- The PTA with Age were Right r-value=0.200, p-value=0.028<0.01, which shows statistical significance positive Correlation difference at p <0.05 level, whereas in Left r-value=0.255, p-value=0.005<0.01, which shows highly statistically significant positive Correlation difference at p <0.01 level respectively.

DISCUSSION

Hearing loss is referred to as the silent, overlooked epidemic of the developing countries because of its invisible nature which prevents detection through routine clinical procedures. The association between essential hypertension and hearing loss in the age group between 20 to 60 years were studied. In the present study it showed that hearing loss can occur when they become hypertensive. The increase in duration of uncontrolled hypertension can cause hearing loss. Many environmental factors play vital role especially fatty diet, stress, family history, and raised cholesterol can cause hypertension. Methods have to be identified to prevent people from becoming developing hypertension. For hearing loss due to hypertension to be prevented, awareness and control of essential hypertension becomes important at an early age.

In the present study we have taken the age group from 20 to 60 years, among them mean age group was 50±9yrs years and were in fifth decade. In 120 essential hypertension patients who participated 55.8% were males and 44.2% were females. In both genders tinnitus was present in 73.3%, hearing loss in 51.7%, vertigo in 5% and other symptoms in 17.5%. which showed no statistical significance.

In our study based on clinical and audiometric evaluation all essential hypertensive patients having hearing loss had Sensorineural hearing loss. The Pure tone average for right ear was measure and among them 26.7% were within normal limits, mild-36.7%, moderate-26.7%, moderately severe-3.3%, severe-2.5%. For the left ear 24.2% was normal, mild-35.8%, moderate-25.8%, moderately severe-7.5%, severe-2.5%. Among the patients studied , based on pure tone audiogram report obtained

in our study indicates that 73.3% of patients had sensorineural hearing loss in right ear and 75.8% of patients had sensorineural hearing loss in left ear. As per the hearing loss there was predominance of mild and moderate degree hearing loss. But there was no statistical significance between PTA and gender.

In our study there were 24.2% patients with short duration of hypertension, 62.5% people were with moderate duration of hypertension and 13.3% were with long duration of hypertension, we used Chi square test and the p=0.996. Among 120 patients with essential hypertension 99% were taking anti-hypertensive medications. We used Fischer exact test and the p=1.000. .

In Our study, the correlation of PTA with systolic blood pressure in both ears with p= 0.0005<0.01 which shows highly statistical significance.

Study also showed positive correlation between diastolic blood pressure and PTA and the value of p was p=0.014<0.01 in right ear and in left ear p value 0.001<0.01 which is significant.

Patients in our study presented with varying duration of hypertension among which the moderate duration of hypertension i.e. between 10- 40 months was most common and degree of hypertension was most commonly in the pre hypertension category, i.e. SBP between 120 – 139mmHg and DBP between 80 – 89mmHg. Mild degree of hearing loss was the most common degree of hearing loss in our study. This study shows that there is a definite association between duration of hypertension and degree of hearing loss.

Rosen et al[5] stated that in a study carried out with hypertensive patients in the USA, there was a correlation between high blood pressure and hearing loss in high frequencies.

Bohem et al assessed audio metrically 50 hypertensive patients with ages above 45 years in Brazil and62% had sensorineural hearing loss.[54]

Marchiori et al[7] studied 552 patients, 137 were patients with arterial hypertension of both genders of which 88.32% of these hypertensive patients had Sensorineural hearing loss.

In a case-controlled study carried out in Kenya with 50 elderly individuals using ABR, Chen et al observed a relationship between hearing loss and arterial hypertension in this population.[56]

Agarwal et al[49] conducted a study in 2013, with 150 cases and 124 controls. The age ranged between 45 to 64 years. This study showed significant association between hypertension and increase in hearing threshold. The increase in hearing threshold was marked among those with grade 3 hypertension, particularly at higher frequencies.

Mishra et al[3] did a case-controlled study in 2014.in this study there were150 cases and 150 controls of both genders of the age group 45 to 60 years. Their study showed an evident association between systemic arterial hypertension and hearing loss.

Mondolli et al conducted a study in 2009 to verify the relationship between systemic arterial hypertension and hearing loss. In this study there were 392 patients of both genders, aged from 45 to 60 years old. Their study showed an evident association between systemic arterial hypertension and hearing loss[50].

According to Baraldi GS et al, systemic arterial hypertension and hearing loss have important prevalence in the elderly population.[52]

Luciano Lozza de Mores Marchiori; Eduardo de Almeida Rego Filho and Tiemo Matsuo conducted a study involving 154 cases and 154 controls, both genders, aged 45 to 64, and found that there is significant association between blood hypertension and hearing loss.

Micro circulatory insufficiency can result in hearing loss due to occlusion of the inferior cerebellar artery. Microangiopathy prevents oxygen and nutrient exchange at cochlear level thus damaging inner and outer hair cells which results in hearing loss.[58]

Tinnitus develops in some patients this may be due to metabolic abnormalities, high cholesterol and vascular problem like spasm, occlusion and hemorrhage.

There are many environmental factors such as noise, metabolic alterations, infections, trauma, inhaling of toxic substances; hereditary, ototoxic drugs can speed up the hair cell damage if they are associated in hypertensive patients.[59,60],

Hypertensive patients develop presbycusis at an early decade of life compared to normal people.

The present study emphasizes that gender has no statistically significant association with the hearing loss.

Hence in hypertensive patients hearing loss occurs due to the following reasons.

- Atherosclerosis of the inferior cerebellar artery which supplies cochlea and other vestibular parts, these are specialized vessels where the flow of blood normally should not be felt otherwise, they

will create tinnitus like noise. But when the vessels become thick the flow of blood becomes reduced and the rate of flow gets increased. So, it will affect oxygen and nutrient supply to the cochlea and thereby resulting in hearing loss.

- The rapidity of blood flow in the cochlea will affect cochlear dynamics. When the dynamics are affected the polarization and depolarization process gets affected and thereby resulting in hearing loss.

- In hypertensives when there is slow perfusion of potassium in to the perilymph or sodium perfusion in to endolymph slow progressive hearing loss occurs.

CONCLUSION:

- A descriptive cross-sectional study of hearing impairment among patients with essential hypertension attending otorhinolaryngology department and hypertension clinic of department of medicine was done.
- The mean age of patients in our study was found to be 50(±9) years. Most of the patients were in the 5th decade of life.
- The most common symptom in our study was tinnitus which was 73.3%. Symptom of hearing loss among patients with essential hypertension in our study is 51.7%. In our study the most common associated symptom was tinnitus and hearing loss. Vertigo was the next most common symptom.
- Patients in our study presented with varying duration of hypertension among which the moderate duration of hypertension i.e. between 10- 40 months was most common and degree of hypertension was most commonly in the pre hypertension category, i.e. SBP between 120 – 139mmHg and DBP between 80 – 89mmHg. Mild degree of hearing loss was the most common degree of hearing loss in our study.
- Among the patients studied , based on pure tone audiogram report obtained indicates that 75.8% of patients had sensorineural hearing loss.
- In our study, 96.70% (88) of subjects had bilateral hearing loss and 3.30% (3) of subjects had unilateral hearing loss. Majority of the patients had bilateral hearing loss.
- The degree of Sensorineural hearing loss was primarily in the mild category.
- This study shows that there is a definite association between duration of hypertension and degree of hearing loss.

- The systemic arterial hypertension is an independent risk factor for hearing loss. Therefore, health care providers need to be aware of the co-existence of the hearing loss with essential hypertension.

- There is a significant association of hearing loss in patients with essential hypertension. Therefore a balanced diet, exercise, medications and early screening will go a long way in its prevention.
- This study was an attempt to show importance of early routine screening for hearing loss in patients with essential hypertension and a need for starting preventive processes to minimize the degenerative mechanisms of the auditory system caused by circulatory problems and incorporate audiological referral as a routine practice for every patient with essential hypertension.

ANNEXURES

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STUDY PROFORMA

Date:

Patient Information:

Name	
Age	
Sex	
OPD No./IP No.	
Address	
Phone number	
Diagnosis	

History of Present illness:

Past History and medication history

Family History

Personal history

General Physical Examination:

BUILT AND NOURISHMENT:
PALLOR/CYANOSIS/CLUBBING/ICTERUS/LYMPHADENOPATHY/OEDEMA

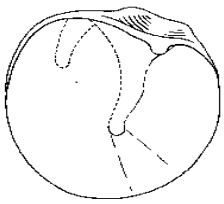
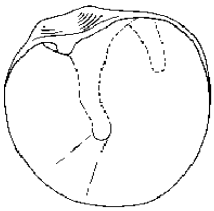
Weight(kg)/height(cms)	
Pulse rate	
Temperature (deg. Celsius)	
Respiratory rate (per min)	
Blood pressure(sitting position)	

Systemic Examination:

Cardiovascular system	
Respiratory system	
Per Abdomen	
Central nervous system	

Local examination: EAR

	RIGHT	**LEFT**
Pre auricular area		
Pinna		
Post auricular area		
EAC		

Tympanic membrane		
Tragal tenderness/ Mastoid tenderness		
Three finger test		
Facial nerve examination		
TFT Rinne Weber Absolute bone conduction		
Vestibular function tests Nystagmus Rhombergs Untenberger's Dix hallpike		

EXAMINATION OF NOSE:

External contour of the nose:

Vestibule of the nose

Anterior rhinoscopy

Posterior rhinoscopy

Air-way patency test:

Examination of Paranasal Sinuses

EXAMINATION OF THROAT

Examination of oral cavity:

Examination of oropharynx

Indirect laryngeal mirror examination

EXAMINATION OF NECK

	RIGHT	**LEFT**
Pure Tone Audiogram (PTA)		

PATIENT INFORMATION SHEET

TITLE: A STUDY OF HEARING IMPAIRMENT AMONG PATIENTS WITH ESSENTIAL HYPERTENSION

I , Dr.VINEETH. S, post graduate, MS in ENT, Government Stanley Medical College is going to undertake the study on the above mentioned topic.

Aim is A STUDY OF HEARING IMPAIRMENT AMONG PATIENTS WITH ESSENTIAL HYPERTENSION A STUDY OF HEARING IMPAIRMENT AMONG PATIENTS WITH ESSENTIAL HYPERTENSION .If you are willing to participate in this study you will be asked some questions regarding duration of your illness, treatment history ,family history regarding the illness. And you may need to undergo some non invasive tests such as PTA, Chest X ray, Blood pressure measurement.

This is A STUDY OF HEARING IMPAIRMENT AMONG PATIENTS WITH ESSENTIAL HYPERTENSION.I assure that all the information provided by you will be kept highly confidential and privacy is assured. Your identity won't be revealed to anyone. The study may be published in scientific journal, but your identity will not be revealed.

Your participation in this study is voluntary and you can withdraw from this at any point of time.

Signature/left thumb impression of the participant

INFORMED CONSENT

TITLE - A STUDY OF HEARING IMPAIRMENT AMONG PATIENTS WITH ESSENTIAL HYPERTENSION

The content of the information sheet dated _____ that was provided have been read carefully by me/explained in detail to me, in a language that I comprehend and fully understood the contents.

I confirm that I had the opportunity to ask questions. The nature and purpose of the study and its potential risks/benefits and expected duration of the study and other relevant details of the study have been explained to me in detail.

I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal right being affected.

I agree to take part in the above study

_____

(Signature/Left thumb impression)

Name of the Participant: _____

Son/Daughter/Spouse of _____

Complete postal address: _____

This is to certify that the above consent has been obtained in my presence.

_____ Date:

Signature of the principal investigator Place:

1)Witness – 1 2) Witness – 2

_____ _____

Signature: Signature:

Name: Name:

Address: Address:

PATIENT INFORMATION SHEET

தகவல் நகல்

TITLE: A STUDY OF HEARING IMPAIRMENT AMONG PATIENTS WITH ESSENTIAL HYPERTENSON

இந்த ஆய்வில் உங்களிடம் கேட்கும் கேள்விகளுக்கு முழு மனதுடன் பதில் அளிக்க வேண்டும். இந்த ஆய்வில் உங்களின் நாள்பட்ட நோய்கள், உடல்நலம் தொடர்பாக விவரங்கள் கேட்கப்படும்..

உங்களிடம் கேட்கும் கேள்விகளில் உங்களின் சுயவிபரம், குடும்பவிபரம், தொழில் விபரம் மற்றும் இதர விபரம் அடங்கும்.உங்களுக்கு பணம் எதுவும் அளிக்கபடாது என்பதை இதன் மூலம் தெறிவிக்கிறேன்.

உங்களின் விபரங்கள் எதுவும் மற்றவர்களுக்கு தெரிவிக்கப்படாது என்பதை உறுதியளிக்கிறேன். இந்த ஆய்வின் முடிவுகள் பத்திரிக்கைளில் பிரசுரிக்கபடலாம்,ஆனால் உங்களின் அடையாளம் எதுவும் காட்டப்படாது.

உங்களுக்கு விருப்பம் இல்லை என்றால் எப்போது வேண்டுமானாலும் இந்த ஆய்வில் இருந்து விலகிக்கொள்ளலாம். அதனால் உங்களுக்கு எந்த பாதிப்பும் இல்லை.

கையொப்பம்/ இடது பெருவிரல் ரேகை

INFORMED CONSENT

தகவல் தொடர்பு ஒப்புதல் படிவம்

TITLE: A STUDY OF HEARING IMPAIRMENT AMONG PATIENTS WITH ESSENTIAL HYPERTENSION

நான் தகவல் நகலில் கொடுக்கபட்டுள்ள முழு விவரங்களையும் கவனமாகப் படித்தேன். ஆய்வின் முழு விவரங்களையும் தமிழில் எனக்கு விளக்கமாக எடுத்துக் கூறப்பட்டது.

நான் இந்த ஆய்வின் விவரங்களை முழுமையாக புரிந்துகொண்டேன்.

ஆய்வில் பங்கு எடுக்கும் போது எற்படும் சாத்தியமான அபாயங்கள் மற்றும் பயன்களை நான் அறிந்துள்ளேன்.

மேலும், நான் எந்த ஒரு வேளையிலும் ஆய்வில் இருந்து திரும்ப முடியும் என்றும், அதன் பின்னர் நான் வழக்கம் போல் மருத்துவச் சிகிச்சைப் பெற முடியும் என்றும் புரிந்துக் கொள்கிறேன்.

நான் ஆய்வில் பங்கு கொள்ள பணம் எதையும் பெற முடியாது என்று அறிந்துள்ளேன்.

நான் இந்த ஆய்வில் பங்கு எடுப்பதன் மூலம் நான் என்ன செய்ய போகிறேன் என்று தெரியும்.

நான் இந்த ஆய்வில் என் முழு ஒத்துழைப்பையும் கொடுப்பேன் என்று உறுதியளிக்கின்றேன்.

ஆய்வில் பங்கேற்பவர் பெயர்:

சாட்சி:

பெயர் மற்றும் முகவரி: *பெயர் மற்றும் முகவரி:*

கையொப்பம்/ விரல் ரேகை *கையொப்பம்/ விரல் ரேகை*

ஆராய்ச்சியாளர் கையொப்பம் மற்றும் தேதி

ABBREVIATIONS

Sl. No.	Abbreviation	Stands for
1	WHO	World Health Organization
2	TM	Tympanic Membrane
3	EAC	External Auditory Canal
4	dB	Decibel
5	A-B gap	Air Bone gap
6	CN	Cranial Nerve
7	CHL	Conductive Hearing Loss
8	SNHL	Sensory Neural Hearing Loss
9	HL	Hearing Loss
10	AVCN	Anteroventral Cochlear Nucleus
11	DAS	Dorsal Acosutic Stria
12	DCN	Dorsal Cochlear nucleus
13	IAS	Internal Acosutic Stria
14	LSO	Lateral nucleus of superior olivary complex
15	MSO	Medial nucleus of superior olvary complex
16	SDT	Speech Detection Threshold
17	MNTB	Medial nucleus of trapezoid body
18	SRT	Speech reception threshold
19	PTA	Pure tone audiometry
20	SOC	Superior olivary complex
21	BP	Blood pressure
22	VAS	Ventral acoustic stria
23	VCN	Ventral cochlear nucleus
24	JNC	Joint national committee on hypertension
25	ASHA	American speech language And hearing association
26	SBP	Systolic blood pressure
27	DBP	Diastolic blood pressure
28	CN	Cranial nerve

ANTI PLAGIARISM CERTIFICATE

Curiginal

Document Information

Analyzed document	Dr Vineeth A study of hearing impairment among patients with essential hypertension.docx (D125574290)
Submitted	2022-01-20T09:58:00.0000000
Submitted by	Vineeth S
Submitter email	vineeth.vineeths@gmail.com
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Sources included in the report

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SA	**Tamil Nadu Dr. M.G.R. Medical University / DISSERTATION.docx** Document DISSERTATION.docx (D123175882) Submitted by: monisha.rm111@gmail.com Receiver: monisha.rm111.mgrmu@analysis.urkund.com	1
W	URL: https://www.jaypeedigital.com/eReader/chapter/9789351520801/ch1 Fetched: 2021-11-11T10:48:07.9430000	2

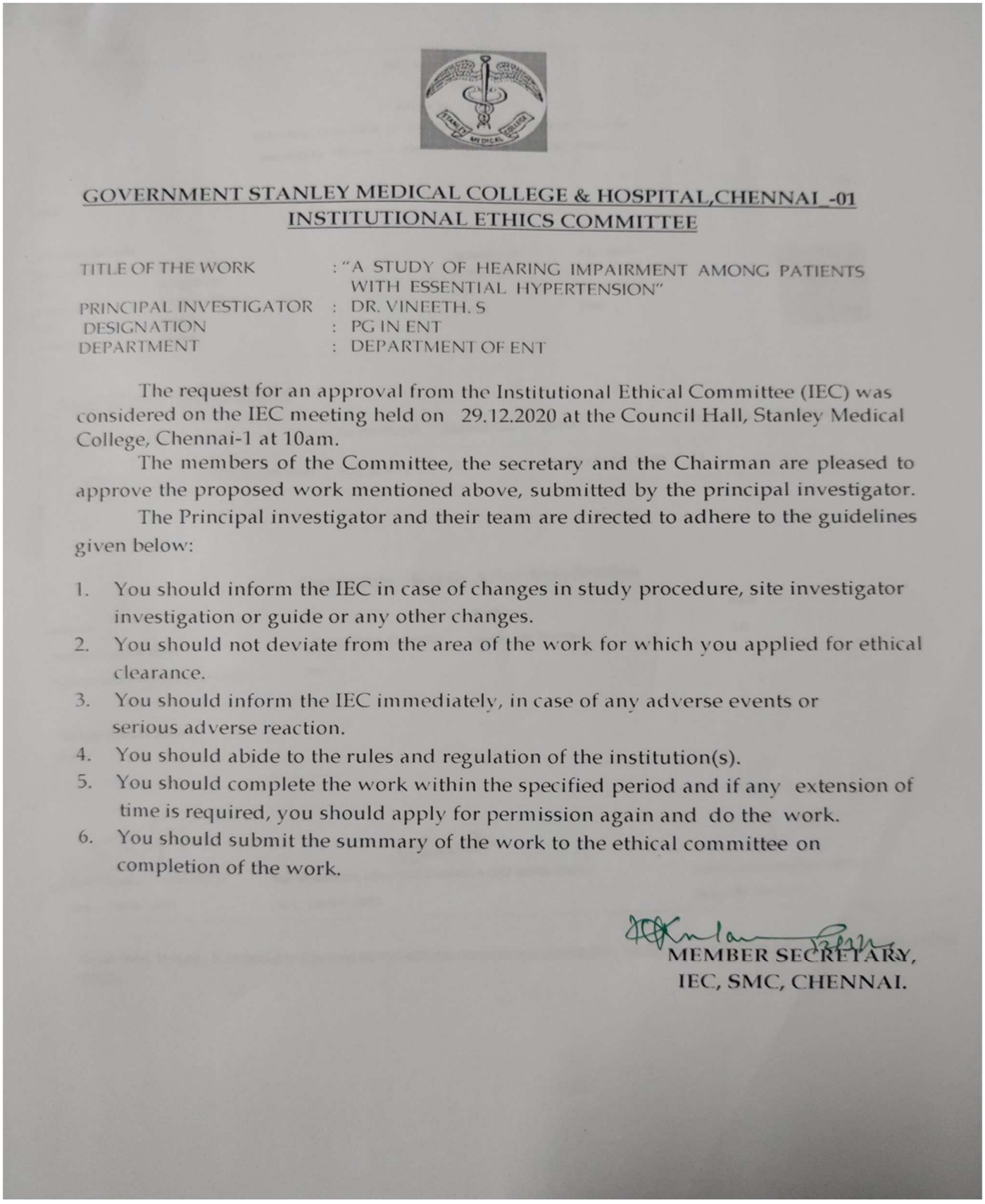
CERTIFICATE - II

This is to certify that this dissertation work titled "**A STUDY OF HEARING IMPAIRMENT AMONG PATIENTS WITH ESSENTIAL HYPERTENSION**" of the candidate **DR.VINEETH S** with registration Number **220420101007** for the award of degree of **M.S** in the branch of **OTORHINOLARYNGOLOGY.**

I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows …… percentage of plagiarism in the dissertation.

Guide & Supervisor sign with Seal.

ETHICAL COMMITTEE APPROVAL LETTER



GOVERNMENT STANLEY MEDICAL COLLEGE & HOSPITAL,CHENNAI -01
INSTITUTIONAL ETHICS COMMITTEE

TITLE OF THE WORK	:	"A STUDY OF HEARING IMPAIRMENT AMONG PATIENTS WITH ESSENTIAL HYPERTENSION"
PRINCIPAL INVESTIGATOR	:	DR. VINEETH. S
DESIGNATION	:	PG IN ENT
DEPARTMENT	:	DEPARTMENT OF ENT

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 29.12.2020 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.

MASTERCHART

Sl.NO.	OPD NO.	AGE	SEX	OCCUPATION	Symptoms_TINNITUS	Symptoms_HEARING LOSS	Symptoms_VERTIGO	Symptoms_OTHERS	OTOSCOPY R	OTOSCOPY L	TFT RINNE RIGHT	TFT RINNE LEFT	TFT WEBER	TFT ABC RIGHT	TFT ABC LEFT	AUDIOGRAM RIGHT	AUDIOGRAM LEFT	PTA RIGHT	PTA LEFT	Sys_BP	Dias_BP	DURATION_mths	TREATMENT_Yes
1	270301	60	F	HOUSE WIFE	1	0	0	0	0	0	1	1	1	2	2	SNHL	SNHL	46.3	51.3	132	92	6	1
2	270304	60	M	FARMER	1	0	0	0	0	0	1	1	1	1	1	WNL	WNL	22.6	15	120	80	48	1
3	270327	58	M	DRIVER	1	1	0	0	0	0	1	1	1	2	2	SNHL	SNHL	46.3	52.3	134	80	72	1
4	270352	60	M	COOLIE	1	1	0	0	0	0	1	1	1	2	2	SNHL	SNHL	55.3	44.3	126	80	60	1
5	281386	58	M	BARBER	1	1	0	0	0	0	1	1	1	2	2	SNHL	SNHL	85.3	76.3	130	84	72	1
6	280965	50	F	HOUSE WIFE	1	0	0	0	0	0	1	1	1	1	1	WNL	WNL	22.5	20	120	80	12	1
7	279746	52	F	HOUSE WIFE	1	1	0	0	0	0	1	1	1	2	2	SNHL	SNHL	54.5	44.5	130	80	60	1
8	279852	60	F	HOUSE WIFE	1	1	0	0	0	0	1	1	1	2	2	SNHL	SNHL	58.6	42	138	82	24	1
9	242903	58	M	SALESMAN	1	1	0	0	0	0	1	1	1	1	1	WNL	WNL	18.3	25.3	120	80	12	1
10	253882	57	M	LABOURER	1	1	0	0	0	0	1	1	1	1	1	WNL	WNL	28.3	42.3	132	86	24	1
11	295806	59	F	TEACHER	1	1	0	0	0	0	1	1	1	1	1	WNL	WNL	20.6	25	124	80	12	1
12	295168	56	M	SHOP KEEPER	1	0	0	0	0	0	1	1	1	1	1	WNL	WNL	24.6	25	128	80	16	1
13	295859	50	M	BARBER	1	1	0	0	0	0	1	1	1	2	2	SNHL	SNHL	49.6	58.6	130	80	36	1
14	295864	54	M	LABOURER	1	1	0	0	0	0	1	1	1	1	1	WNL	WNL	23.3	15	128	80	12	1
15	202730	60	M	SHOP KEEPER	1	1	0	0	0	0	1	1	1	2	2	SNHL	SNHL	81.6	56.3	136	80	60	1
16	202731	56	M	LABOURER	1	1	0	0	0	0	1	1	1	2	2	SNHL	SNHL	40	75	134	86	36	1
17	207352	53	M	SALESMAN	1	1	0	0	0	0	1	1	1	2	2	SNHL	SNHL	45	35	136	80	42	1
18	289290	60	M	LABOURER	1	0	1	0	0	0	1	1	1	2	2	SNHL	SNHL	44.6	40.6	128	86	24	1
19	207853	51	M	SALESMAN	0	0	0	1	0	0	1	1	1	1	1	WNL	WNL	25.6	25.6	120	80	6	1
20	207868	60	F	HOUSE WIFE	0	1	0	0	0	0	1	1	1	2	2	SNHL	SNHL	46.3	60.6	136	80	36	1
21	219506	60	F	HOUSE WIFE	1	1	0	0	0	0	1	1	1	2	2	SNHL	SNHL	40	56.6	126	80	12	1
22	219930	54	M	COOLIE	1	1	0	0	0	0	1	1	1	1	1	WNL	WNL	16	16	120	80	24	1
23	230671	60	M	LABOURER	1	1	0	0	0	0	1	1	1	1	1	WNL	WNL	25.6	26.6	126	80	12	1
24	231260	60	F	SHOP KEEPER	0	1	0	0	0	0	1	1	1	1	1	SNNL	WNL	31.6	25.3	126	80	24	1
25	230710	45	F	HOUSE WIFE	1	1	0	0	0	0	1	1	1	1	1	SNHL	SNHL	30	31.6	128	80	60	1
26	234646	60	F	HOUSE WIFE	1	1	0	0	0	0	1	1	1	1	1	SNHL	SNHL	36	32.6	138	90	36	1
27	237223	40	F	HOUSE WIFE	0	0	0	1	0	0	1	1	1	1	1	SNHL	SNHL	28	26	120	80	6	1
28	237654	52	F	HOUSE WIFE	1	0	0	0	0	0	1	1	1	1	1	SNHL	SNHL	36.6	36	130	90	8	1
29	255027	38	F	TEACHER	0	0	0	1	0	0	1	1	1	1	1	WNL	WNL	18.6	21.6	120	80	12	1
30	211527	48	F	HOUSE WIFE	0	0	0	1	0	0	1	1	1	2	2	SNHL	SNHL	50	45	140	90	36	1
31	230631	44	M	COOLIE	0	0	0	1	0	0	1	1	1	2	2	SNHL	SNHL	56	48	130	84	30	1
32	230646	60	F	HOUSE WIFE	0	0	1	0	0	0	1	1	1	2	2	SNHL	SNHL	46	46	120	80	24	1
33	230845	50	F	HOUSE WIFE	0	0	0	1	0	0	1	1	1	1	1	WNL	SNNL	25	35	126	80	7	1
34	227539	50	F	HOUSE WIFE	0	0	0	1	0	0	1	1	1	1	1	SNHL	SNHL	32	32	130	84	12	1
35	269748	60	F	HOUSE WIFE	1	0	0	0	0	0	1	1	1	1	1	SNHL	SNHL	30.6	31.6	130	80	9	1
36	263550	48	M	LABOURER	0	0	0	1	0	0	1	1	1	1	1	SNHL	SNHL	32	36	120	86	12	1
37	285918	57	M	SHOP KEEPER	1	0	0	0	0	0	1	1	1	1	1	WNL	WNL	18.3	18.3	130	90	36	1
38	185817	59	M	LABOURER	1	1	0	0	0	0	1	1	1	2	2	SNHL	SNHL	45.8	44.8	128	86	12	1
39	195301	28	M	BARBER	1	0	0	0	0	0	1	1	1	1	1	WNL	WNL	21.6	21.6	120	80	24	1
40	185432	31	M	COOLIE	0	0	0	1	0	0	1	1	1	1	1	WNL	WNL	20	20	128	80	12	1
41	196579	42	M	LABOURER	0	0	0	1	1	0	1	1	1	1	1	WNL	WNL	28.3	13.3	128	80	3	1
42	86623	60	M	SHOP KEEPER	0	0	0	1	0	0	1	1	1	1	1	WNL	WNL	20	28	120	80	2	1
43	224577	51	F	HOUSE WIFE	0	0	1	0	0	0	1	1	1	1	1	WNL	WNL	30	35	136	90	6	1
44	225310	42	F	HOUSE WIFE	0	0	0	1	1	0	1	1	1	1	1	WNL	WNL	25	25	128	86	5	1
45	225573	43	M	SALESMAN	0	0	0	1	0	0	1	1	1	1	1	WNL	WNL	23.3	23.3	130	80	6	1
46	239877	43	M	LABOURER	0	1	0	0	0	0	1	1	1	1	1	SNHL	SNHL	41.6	45.6	138	88	12	1
47	239938	58	M	SHOP KEEPER	1	0	0	0	0	0	1	1	1	2	2	SNHL	SNHL	34.6	45.6	138	86	24	1
48	245318	38	F	HOUSE WIFE	1	0	0	0	0	0	1	1	1	1	1	WNL	WNL	26	26	120	80	12	1
49	247230	46	F	HOUSE WIFE	0	0	1	0	0	0	1	1	1	1	1	SNHL	SNHL	31.6	31.6	130	80	24	1

50	247450	56	M	SALESMAN	1	1	0	0	0	0	1	1	1	2	2	SNHL	SNHL	55	61	130	80	46	1
51	294818	60	F	TEACHER	0	0	0	1	0	0	1	1	1	1	1	SNHL	SNHL	31	34	130	80	36	1
52	220042	60	M	LABOURER	0	1	0	0	0	0	1	1	1	2	2	SNHL	SNHL	50.6	42.6	138	86	14	1
53	220046	58	F	HOUSE WIFE	1	0	0	0	0	0	1	1	1	2	2	SNHL	SNHL	41.6	75	130	86	36	1
54	220078	47	F	HOUSE WIFE	1	1	0	0	0	0	1	1	1	1	1	SNHL	SNHL	42.6	35.6	126	80	24	1
55	281700	48	F	HOUSE WIFE	0	0	0	1	0	0	1	1	1	1	1	WNL	WNL	21.6	25.6	120	80	24	1
56	220295	47	M	LABOURER	0	0	1	0	0	0	1	1	1	1	1	WNL	WNL	20	20	120	80	9	1
57	420976	58	M	LABOURER	1	0	0	0	0	0	1	1	1	2	2	SNHL	SNHL	29.3	32.6	130	80	36	1
58	269577	58	F	HOUSE WIFE	0	1	0	0	0	0	1	1	1	1	1	SNHL	SNHL	33	62	138	86	46	1
59	211336	60	M	LABOURER	1	1	0	0	0	0	1	1	1	2	2	SNHL	SNHL	44.6	42	140	90	36	1
60	265815	38	M	BARBER	0	1	0	0	0	0	1	1	1	1	1	SNHL	SNHL	38.6	38	130	80	36	1
61	218317	60	M	SALESMAN	1	0	0	0	0	0	1	1	1	2	2	SNHL	SNHL	52	48	126	86	47	1
62	218895	53	F	HOUSE WIFE	1	1	0	0	0	0	1	1	1	2	2	SNHL	SNHL	36.6	52.6	134	80	46	1
63	226786	60	F	HOUSE WIFE	1	1	0	0	0	0	1	1	1	2	2	SNHL	SNHL	45	37	120	80	24	1
64	212850	60	F	HOUSE WIFE	1	1	0	0	0	0	1	1	1	2	2	SNHL	SNHL	46.3	42.6	134	86	36	1
65	212987	51	M	LABOURER	1	0	0	0	0	0	1	1	1	1	1	SNHL	SNHL	16.6	18.3	120	80	12	1
66	213280	45	F	HOUSE WIFE	1	1	0	0	0	0	1	1	1	2	2	SNHL	SNHL	35.6	31.6	128	86	26	1
67	214569	53	M	LABOURER	1	1	0	0	0	0	1	1	1	2	2	SNHL	SNHL	41.6	44.6	130	86	18	1
68	220704	32	F	HOUSE WIFE	1	0	0	0	0	0	1	1	1	1	1	WNL	WNL	18.3	18.3	120	80	24	1
69	218004	60	F	HOUSE WIFE	1	1	0	0	0	0	1	1	1	2	2	SNHL	SNHL	60.3	58.6	134	80	46	1
70	220696	35	F	HOUSE WIFE	0	0	0	1	0	0	1	1	1	1	1	WNL	WNL	23.3	18.6	120	80	12	1
71	249973	58	M	LABOURER	0	0	0	1	0	0	1	1	1	1	1	WNL	WNL	26.6	26.6	120	80	6	1
72	246786	58	M	SALESMAN	1	0	0	0	0	0	1	1	1	1	1	WNL	WNL	26	26	120	80	6	1
73	218093	45	M	SHOP KEEPER	0	0	0	1	0	0	1	1	1	1	1	WNL	WNL	28.3	28.3	128	80	6	1
74	265432	56	F	HOUSE WIFE	1	0	0	1	0	0	1	1	1	1	1	WNL	WNL	25.6	26.4	120	80	8	1
75	263873	48	M	SALESMAN	1	0	0	1	0	0	1	1	1	1	1	WNL	WNL	13.3	20	128	90	9	1
76	201889	60	M	LABOURER	1	1	0	0	0	0	1	1	1	2	2	SNHL	SNHL	54.3	48.3	142	90	36	1
77	285372	57	M	LABOURER	1	1	0	0	0	0	1	1	1	2	2	WNL	WNL	15	16	120	80	4	1
78	137803	55	M	SALESMAN	1	1	0	0	0	0	1	1	1	1	1	WNL	WNL	23.6	23.6	120	80	2	1
79	137903	37	M	SALESMAN	1	1	0	0	0	0	1	1	1	1		SNHL	SNHL	84.6	47	134	82	50	1
80	237944	37	M	LABOURER	1	1	0	0	0	0	1	1	1	1	1	SNHL	SNHL	60	60	130	90	36	1
81	237986	40	M	LABOURER	1	0	0	0	0	0	1	1	1	1	1	SNHL	SNHL	35.6	43.6	124	80	24	1
82	137843	37	M	STUDENT	1	0	0	0	0	0	1	1	1	1	1	WNL	WNL	18	18	128	80	4	1
83	214567	58	F	HOUSE WIFE	1	1	0	0	0	0	1	1	1	1	1	SNHL	SNHL	28.6	28.4	134	80	28	1
84	214578	60	F	HOUSE WIFE	1	1	0	0	0	0	1	1	1	2	2	SNHL	SNHL	38	38	130	90	46	1
85	246740	58	F	HOUSE WIFE	0	0	1	1	0	0	1	1	1	2	2	SNHL	SNHL	45	56	130	90	45	1
86	124910	60	F	HOUSE WIFE	1	0	0	0	0	0	1	1	1	1	1	WNL	WNL	21.6	13.3	12O	80	8	1
87	123388	45	F	TEACHER	1	1	0	0	0	0	1	1	1	1	1	SNHL	SNHL	46	38	130	80	24	1
88	223377	30	M	SALESMAN	1	1	0	0	0	0	1	1	1	1	1	SNHL	SNHL	38	43	136	90	12	1
89	223303	60	M	SHOP KEEPER	1	1	0	0	0	0	1	1	1	2	2	SNHL	SNHL	53	46	130	90	36	1
90	223308	60	F	HOUSE WIFE	1	1	0	0	0	0	1	1	1	1	1	SNHL	SNHL	38	38	120	80	24	1
91	274901	47	M	FARMER	1	1	0	0	0	0	1	1	1	1	1	WNL	WNL	26	26	126	80	7	1
92	137655	38	M	SHOP KEEPER	0	1	0	0	0	0	1	1	1	2	2	SNHL	SNHL	44	45.6	130	80	24	1
93	214565	45	M	SALESMAN	1	1	0	0	0	0	1	1	1	2	2	SNHL	SNHL	36	38	138	90	28	1
94	236120	42	F	CLERK	1	0	0	0	0	0	1	1	1	1	1	SNHL	SNHL	36	36.8	130	90	32	1
95	212456	43	M	FARMER	1	1	0	0	0	0	1	1	1	2	2	SNHL	SNHL	40.6	38.6	134	90	36	1
96	224361	40	F	HOUSE WIFE	1	0	0	0	0	0	1	1	1	1	1	SNHL	SNHL	36	32.6	120	80	24	1
97	237891	32	M	SHOP KEEPER	1	1	0	0	0	0	1	1	1	1	1	SNHL	SNHL	36	36	128	90	28	1
98	223379	41	F	HOUSE WIFE	1	0	0	0	0	0	1	1	1	1	1	WNL	WNL	25	25	120	80	16	1
99	212551	40	M	SALESMAN	1	1	0	0	0	0	1	1	1	2	2	SNHL	SNHL	42	42	130	80	32	1
100	135742	46	M	FARMER	1	1	0	0	0	0	1	1	1	2	2	SNHL	SNHL	32	38.6	136	80	36	1
101	232569	41	F	HOUSE WIFE	1	0	0	0	0	0	1	1	1	1	1	WNL	WNL	25	25	120	80	4	1
102	249840	37	M	SHOP KEEPER	1	0	0	0	0	0	1	1	1	1	1	WNL	WNL	18	18	128	90	6	1
103	276543	39	F	HOUSE WIFE	1	0	0	0	0	0	1	1	1	1	1	WNL	WNL	20	20	120	80	8	1
104	278655	43	M	FARMER	1	0	0	0	0	0	1	1	1	1	1	SNHL	SNHL	40	38	128	86	26	1
105	274912	39	F	SHOP KEEPER	1	1	0	0	0	0	1	1	1	2	2	SNHL	SNHL	42	46	130	80	31	1
106	113456	32	M	SALESMAN	1	1	0	0	0	0	1	1	1	1	1	SNHL	SNHL	38.6	40	128	90	32	1
107	224576	31	F	HOUSE WIFE	1	0	0	0	0	0	1	1	1	1	1	SNHL	SNHL	41.6	44.6	138	80	24	1
108	133451	46	M	FARMER	1	1	0	0	0	0	1	1	1	2	2	SNHL	SNHL	45	45	132	80	36	1

109	124567	45	M	SALESMAN	0	1	0	0	0	0	1	1	1	1	1	SNHL	SNHL	36.6	38.6	130	80	38	1
110	109873	46	F	HOUSE WIFE	1	0	0	0	0	0	1	1	1	1	1	WNL	WNL	25	25	120	80	8	1
111	123456	36	F	HOUSE WIFE	1	0	0	0	0	0	1	1	1	1	1	WNL	WNL	26	26	123	80	6	1
112	224678	32	M	BUSINESSMAN	1	0	0	0	0	0	1	1	1	1	1	WNL	WNL	25	25	128	90	4	1
113	234569	49	M	FARMER	1	1	0	0	0	0	1	1	1	1	1	SNHL	SNHL	44.6	48	130	90	35	1
114	245678	54	M	SALESMAN	1	1	0	0	0	0	1	1	1	2	2	SNHL	SNHL	55	55	136	90	40	1
115	233456	60	M	SHOP KEEPER	1	0	0	0	0	0	1	1	1	1	1	WNL	WNL	20	20	120	80	6	1
116	134256	59	F	HOUSE WIFE	1	1	0	0	0	0	1	1	1	1	1	SNHL	SNHL	42	44	130	90	36	1
117	234112	56	M	BUSINESSMAN	1	0	0	0	0	0	1	1	1	1	1	SNHL	SNHL	38	35	136	90	12	1
118	245678	53	M	FARMER	1	1	0	0	0	0	1	1	1	1	1	SNHL	SNHL	40	38	138	90	24	1
119	209854	54	F	HOUSE WIFE	0	1	0	0	0	0	1	1	1	1	1	SNHL	SNHL	36.6	38	128	80	22	1
120	212467	43	F	HOUSE WIFE	1	1	0	0	0	0	1	1	1	1	1	WNL	WNL	26	26	128	90	21	1