"A STUDY OF DRILL INDUCED HEARING LOSS IN THE CONTRALATERAL EAR FOLLOWING MASTOID SURGERY."

Submitted by

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Under the guidance of

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LIST OF ABBREVATIONS

%	Percentage
AOM	Acute otitis media
COM	Chronic otitis media
CSOM	Chronic suppurations otitis media
CSF	Cerebrospinal fluid
dB	Decibel
DPOAE	Distortion product otoacoustic emission
DP	Distortion product
EAC	External auditory canal
ET	Eustachian tube
F1	Frequency 1
F2	Frequency 2
Hz	Hertz
HL	Hearing Loss
IA	Interaural Attenuation
IHC	Inner hair cells
MRM	Modified Radical Mastoidectomy
NIHL	Noise induced hearing loss
OAE	Otoacoustic emission
OME	Otitis media with effusion
OSHA	Occupational Safety and Health Act
POD	Post-operative Day
PTA	Pure tone audiometry
PTS	Permanent threshold shift
ROC	Receiver operating characteristic
Rpm	Rotation per minute
SNHL	Sensorineural hearing loss
SPL	Sound Pressure Level
SOAE	Spontaneous otoacoustic emission
TA	Transcranial attenuation
TEOAE	Transient evoked otoacoustic emission
TTS	Temporary threshold shift

ABSTRACT

Background:

Chronic otitis media is the inflammation of the mucoperiosteal lining of the middle ear space and mastoid cavity. Mastoidectomy is considered to be the mainstay of treatment of COM. The usage of micro motor drill on the ear has effect on the contralateral ear due to the noise induced by the drill and the sound-conducting characteristic of the intact skull. The ipsilateral cochlea is exposed to a 100 dB sound during drilling, while the opposite cochlea to levels 5 to 10 dB lower. This in turn can lead to dysfunction of the outer hair cells, thus causing temporary or permanent hearing loss.

Aims and Objectives:

1. To identify the drill induced hearing loss in the contralateral ear, by transient evoked otoacoustic emissions. following mastoidectomy.
2. To identify the relation between the type of burr tip used and the amount of hearing loss.
3. To identify that hearing loss is found to be more, if drilling is done for a longer duration of time.

Methodology:

It is a hospital – based prospective study, from November 2018 to April 2020. A total of 63 patients that underwent mastoidectomy were included in this study, with age ranging from 8 to 50 years. For each patient a thorough clinical history was taken with a detailed otologic examination was done, to make sure the contralateral ear was normal. Routine blood investigations along with a x-ray mastoid was done for all patients. Each patient was subjected to a pre-operative PTA and TEOAE. PTA was repeated on POD-1 and POD-7. TEOAE was done on POD-1,3 and 7 for each patient. If any changes in TEOAE readings are detected, a repeat OAE was done until normal values were obtained. A repeat TEOAE will be done on day 15, 30, 60 and 90 postoperatively, if required. TEOAE was recorded at 1000, 2000, 3000 and 4000 Hz. Intraoperatively, the type of surgery, the type of burr

tip used as well as the individual drilling time for each type of drill bit was recorded, using a stopwatch.

Results:

Out of the 63 patients enrolled in this study, 30 patients (47.6%) developed transient and temporary SNHL on POD-1 and 7 patients (21.2%) had impaired hearing by POD-3. 4 patients (10.8 %) recovered by POD-3, 19 patients (51.4 %) had normal TEOAE readings by POD-7, 10 patients (27.0 %) were normal by POD-15, while the remaining 4 (10.8 %) recovered by POD-30. It was observed that higher frequencies of 3000 Hz and 4000 Hz were more commonly affected. All patients recovered by POD-30. No change was detected on pre and post-operative PTA. It was also detected that drilling with a cutting burr for an average of 45.4 minutes resulted in drill induced hearing loss, whereas when used for an average of 37.5 minutes, normal TEOAE readings were achieved. It was established that usage of a diamond burr for an average of 13.8 minutes resulted in hearing loss in the contralateral ear. However, no hearing impairment was noted when drilling was done for an average of 10.8 minutes.

Conclusion:

Thus, drilling with a cutting burr for more than 40.2 minutes, can lead to hearing loss. This has a sensitivity of 70% and a specificity of 69%. On the other hand, drilling with a diamond burr for over 12.5 minutes, can lead to hearing impairment, with a sensitivity of 68% and a specificity of 65%. The drill is not only a source of noise but is also a strong vibration generator, and a strong oscillation is transmitted into the cochlea. Thus surgeons should pay more attention to the vibrations and lessen the intensity of drill induced hearing loss by an appropriate selection of burrs and drills, thus minimizing the vibrations of the temporal bone.

Key Words: Transient evoked otoacoustic emissions, Drill induced hearing loss.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

The internal anatomy of the ear is made up of a number of extremely tiny, delicate, and interlocking anatomical structures that are surrounded by bone and muscle. In particular, the mastoid portion of the temporal bone lies behind the ear and serves as a solid, normally impenetrable, barrier protecting the internal ear.

This bony barrier has made it particularly challenging to access the anatomical structures within the internal ear. This has been accomplished by drilling through the mastoid bone and thus removing it. This surgical procedure is known as mastoidectomy. This surgical technique has been the mainstay of ear surgery up until the turn of the 20th century.[1,2] For most of the ear surgeries performed today, the main objective is either hearing preservation or improving the hearing. However the surgical instruments required in mastoidectomy, can generate vibrations and noise, thus damaging the ear.

It is known that hearing loss can occur due to acoustic trauma. It has been suggested that an audiometry test is insufficient for the early detection of noise-induced cochlear damage. This can be effectively assessed by otoacoustic emissions[1,2]. In addition, a damaged outer hair cell is one of the first findings of sensorineural hearing loss.[3,4] Therefore, otoacoustic emissions are used for the early detection of noise-induced damage in the inner ear.[5-7]

The diagnosis of chronic otitis media, implies a permanent abnormality of the pars tensa or flaccida, with a worldwide prevalence of 65–330 million people. It may be unilateral or bilateral, but most of the cases are unilateral.[5-7]

Mastoidectomy is the mainstay of the treatment which may be either an intact canal wall mastoidectomy or a canal wall down mastoidectomy. The exposure of the ear to a high level of noise can lead to sensorineural hearing loss. Bone drilling and suction are essential in ear surgery, but it exposes both the cochleae, to the heat and noise generated by a high speed drill.

Drilling the bone during mastoidectomy can lead to sensorineural hearing loss in the healthy contralateral ear as well as in the operated ear because of undesired acoustic trauma.[8]

During drilling the exposed cochlea is subjected to noise levels of more than 90 dB, while the contralateral cochlea to 80-85 dB and above. Vibration of temporal bone may lead to cochlear damage, and both the drill as well as the suction generated noise and vibration may have an additive effect in damaging the cochlea.

Cutting burrs produce more noise as compared to the diamond burr. Variables such as rotation speed of burr, type of burr, burr size and site of drilling have been investigated in isolated temporal bones, cadavers and animal models. Heat generated by the rotating burr specially while drilling near the vestibule can also lead to SNHL. The variations in drill parameter and the duration of drilling determine the extent of noise, vibration and heat generation. [9-11]

Exposure to high levels of noise is known to be harmful to the ear. Noise exposure to sound generated by the drill may result in a transient hearing deficit or permanent hearing impairment.

The probable causes of postoperative hearing loss in a patient undergoing middle ear surgery are, noise due to the drill, continuous suction irrigation, vibrations, inner ear injury and manipulation of ossicles.[9-11]

It has been found that the noise level is 100 dB in the operated ear, while 90–95 dB in the contralateral ear during the drilling procedure in mastoidectomy.[12] In addition, in the study by Tos *et al.*[13], which was conducted on cadavers, they reported that a noise of 114–128 dB was produced in association with instruments used in middle ear surgery.

Studies using pure tone audiometry and OAE for analysis, revealed a number of variations in the findings. Hence the present study was done at our tertiary care centre to identify the drill induced hearing loss in the contralateral ear, by transient evoked otoacoustic emission, after mastoidectomy and assess the relation between the type of burr tip used, duration of time and the amount of hearing loss.

AIMS AND OBJECTIVES

1) To identify the drill induced hearing loss in the contralateral ear, by transient evoked otoacoustic emission, after mastoidectomy.

2) To identify the relation between the type of burr tip used and the amount of hearing loss.

3) To identify that hearing loss is found to be more, if drilling is done for a longer duration of time.

REVIEW OF LITERATURE

HISTORY:

Chronic and suppurative infections of the mastoid have been described as long ago as the ancient Greece. However, it was not until mid-17th century when Riolan the Younger described the first trephination procedure of the mastoid. The subsequent 200 years did not produce many significant advances until Fielitz and Petit reported multiple cases of mastoid trephinations for acute abscesses in the late 18th century. These procedures fell out of favour for more than 100 years until Schwartze and Eysell popularized the cortical mastoidectomy in 1873. It was effective for draining acute infections; however, it did little to treat chronic infections of the ear.[14]

In 1890, Zaufal[15] described the first radical mastoidectomy by removing the superior and posterior ear canal, the tympanic membrane, and the ossicles in an attempt to eliminate infection, externalize disease, and create a dry ear. Bondy revised this technique by leaving the uninvolved middle ear alone and exteriorizing the epitympanum.[16]

The introduction of the Zeiss otologic operating scope in 1953 made precise dissection possible. Soon thereafter, Wullstein described the first attempts at reconstruction of the tympanic membrane via tympanoplasty.[17]

Five years later, William House introduced the intact canal wall mastoidectomy.[18] Since then, multiple variations of the mastoidectomy have been described.

EMBRYOLOGICAL DEVELOPMENT:

The ear is a highly specialized structure in the body, which is required for hearing as well as balance. It can be divided into 3 parts:

1) External ear

2) Middle ear

3) Inner ear

External Ear:

The external auditory meatus arises from the first pharyngeal cleft. It begins as an invagination of ectoderm that extend inwards. By the 5th week of embryonic development, there is proliferation of the ectodermal cells, thus forming a meatal plug that fills the entire lumen. By the 10th week, a disk-like structure if formed, which later comes in contact with the primordial malleus medially, contributing to the future formation of the tympanic membrane. The external auditory meatus is completely patent and expands to its complete form by the eighteenth week.[19]

By the end of the 4th week of development, the auricle develops from 6 mesenchymal proliferations known as Hillocks of His, which are derived from the first and second pharyngeal arches. The first three auricular hillocks emerge from the first pharyngeal arch and give rise to the tragus, helix, and cymba concha. The last three auricular hillocks arise from the second pharyngeal arch and give rise to the concha, antihelix, and antitragus.[19]

Middle Ear:

The tympanic cavity and eustachian tube originate from an extension of the endoderm of the first pharyngeal pouch called the tubotympanic recess. During the 5th week, the tubotympanic recess extends laterally until it reaches the floor of the first pharyngeal cleft. The endoderm of the tubotympanic recess and the ectoderm of the first pharyngeal cleft are adjacent to one another at this point, with a fibrous layer derived from mesenchyme called the lamina propria sandwiched in between. Resulting in the formation of a trilaminar tympanic membrane made up of three separate germ layers consisting of ectoderm, mesoderm, and endoderm. The ventral portion of the tubotympanic recess develops into the eustachian tube. The eustachian tube demonstrates the most growth during weeks 16 to 28 of the fetal period.[20]

Ossicles:

The cartilage origin of the three middle ear ossicles arises from neural crest-derived mesenchyme of the first and second pharyngeal arches. The malleus and incus develop from Meckel's cartilage of the first pharyngeal arch, while the stapes arises from Reichert's cartilage of the second pharyngeal arch. As the tympanic cavity develops, the cartilages ossify via the process of endochondral ossification, which continues throughout the entire fetal period. During the 8th and 9th months of fetal life, the mesenchyme holding the ossicles in place undergoes resorption via programmed cell death resulting in an air-filled tympanic cavity at birth.[21]

Inner Ear:

The inner ear originates from the invagination of the otic placodes during the fourth week of development. The otic placodes are sensory placodes, which are a series of transiently thickened surface ectodermal patches that form pairs in the head region. They are located behind the second pharyngeal arch and give rise to the otic pits by invaginating into the mesenchyme during the fourth week of development. Towards the end of the fourth week, the otic pits break off from the surface ectoderm to form a hollow piriform shaped structure lined with columnar epithelium called the otic vesicle. At this point, the otic vesicle lies beneath the surface ectoderm enveloped in the mesenchyme, forming the otic capsule.

The otic vesicle differentiates to form all the components of the membranous labyrinth and ultimately gives rise to the inner ear structures associated with hearing and balance. The otic vesicle divides into a dorsal utricular portion and ventral saccular portion, with the dorsal utricular portion giving rise to the vestibular system and the ventral saccular portion giving rise to inner ear structures involved in hearing. The ventral saccular portion develops into the cochlear duct and saccule. The dorsal utricular portion forms into the utricle, semicircular canals, and endolymphatic tube.[22]

Saccule and Cochlea:

In the 6th week of development, the ventral saccular component of the otic vesicle penetrates the surrounding mesenchyme in a spiralling fashion. It completes two and a half turns to form the cochlear duct by the end of the 8th week. At this point, the saccule connects to the utricle via the ductus reuniens and mesenchyme surrounds the entire cochlear duct.[23]

The mesenchyme surrounding the cochlear duct forms cartilage. During the tenth week of development, this cartilaginous shell undergoes vacuolization to create the two perilymphatic spaces of the cochlea, the scala vestibule, and the scala tympani. Two membranes separate the cochlear duct proper, which is also known as the scala media, from the scala tympani and scala vestibule. The basilar membrane demarcates the scala media from the scala tympani, while the vestibular membrane separates the scala media from the scala vestibule.[23]

Organ of Corti:

The Organ of Corti is located within the scala media of the cochlear duct and resides on the basilar membrane. It is composed of mechanosensory cells and supporting cells. The arrangement of the mechanosensory cells is as outer and inner hair cells along rows. The outer hair cells are separated by supporting cells and form three rows, while the inner hair cells form a single row. The tectorial membrane covers these mechanosensory hair cells and, in combination with each other, constitute the organ of Corti. Shifting of the tectorial membrane in response to endolymph fluid motion displaces the stereocilia of sensory hair cells. Stereocilia displacement results in the generation of impulses that transmit to the spiral ganglion and reach the central nervous system via the auditory fibres of the vestibulocochlear nerve. The capsular cartilage that surrounds the membranous labyrinth becomes ossified between 16 and 23 weeks of gestation to form the true bony labyrinth.[23]

The Utricle and Semicircular Canals:

The utricle and semicircular canals are the organs of balance and originate from the dorsal utricular portion of the otic vesicle. During the 6th week of development, three flattened outpouchings of epithelium extend from the dorsal utricular portion of the membranous labyrinth that eventually give rise to the semicircular canals.[24]

One end of each of the semicircular canal dilates to form the crus ampullare, while the other end, the crus nonampullare, does not dilate. The dilated ampulla consists of sensory hair cells that form a crest called the crista ampullaris. Similar sensory areas form in the walls of the saccule and utricle. The crista ampullaris senses changes in angular acceleration and is the sensory organ of rotation. Impulses generated in the sensory cells of the crista ampullaris reach the brain via the vestibular fibres of the vestibulocochlear nerve.[24]

Hair cells:

The utricle and saccule are otolith organs located in the vestibule that detect movement in different planes. The utricle and saccule consist of sensory areas called maculae comprised of supporting cells and hair cells covered in a gelatinous acellular matrix called the otolithic membrane. The crista ampullaris of the semicircular ducts have a sensory epithelium similar to that of the macula, also consisting of hair cells and supporting cells. The hair cells of the cristae project into a gelatinous material called the cupula, which does not contain otoliths, and serve to detect rotational acceleration.[25]

The Organ of Corti also consists of two groups of hair cells: inner hair cells and outer hair cells. The inner hair cells account for approximately 95% of the sensory input into the auditory system and arrange in one line along the entire basilar membrane. The outer hair cells account for about 5% of sensory input and serve primarily as acoustical pre-amplifiers. The outer hair cells receive efferent input and contract when stimulated, resulting in amplified sound waves. It also consists of supporting

cells, namely the Hensen cells, Corti pillars, the Deiters cells, and the Claudius cells. The supporting cells play essential roles in the function and maintenance of the inner ear and primarily serve structural and homeostatic functions.[25]

SURGICAL ANATOMY OF EAR:

Tympanic Membrane:

It forms the partition between the external acoustic meatus and the middle ear. It is obliquely set and as a result, its posterosuperior part is more lateral than its antero-inferior part. It is 9-10 mm long, 8 mm wide and 0.1 mm thick.[26] It is divided into 2 parts:

(a) Pars tensa:

It forms most of the tympanic membrane. Its periphery is thickened to form a fibrocartilaginous ring called the annulus tympanicus or Gerlach's ligament. The central part of pars tensa is tented inwards at the level of tip of Malleus and is called the umbo. A bright cone of light can be seen radiating from the tip of Malleus to the periphery in the anteroinferior quadrant.[26]

(b) Pars flaccida:

This is situated above the lateral process of Malleus between the Notch of Rivinus and the anterior and posterior malleal folds. It is not taut and may appear slightly pinkish.[26]

The Middle Ear:

The middle ear extends much beyond the limits of tympanic membrane which forms its lateral boundary and is divided into:

(a) Epitympanum or the attic

(b) Mesotympanum

(c) Hypotympanum

The middle ear cavity is compared to a six-sided box which consists of a roof, a floor, a medial wall, a lateral wall, an anterior wall and a posterior wall.[26]

The roof is formed by a thin plate of bone called the tegmen tympani. The floor consists of a thin plate of bone which separates the tympanic cavity from the jugular bulb.[26]

The anterior wall comprises of a thin plate of bone, which separates the mastoid cavity from the internal carotid artery. It has two openings: the lower opening is for the eustachian tube while the upper one for the tensor tympani muscle.[26]

The posterior wall consists of a bony projection called the pyramid, which provides attachment to the stapedius tendon. The facial nerve runs in the posterior wall just behind the pyramid.[26]

The medial wall is formed by the labyrinth. It consists of a bulge called the promontory which is formed by the basal coil of the cochlea. It also comprises of the oval window, above which lies the footplate of stapes and the round window, which is covered by the secondary tympanic membrane. Just anterior to the oval window, lies a hook like projection called the processus cochleariformis. The lateral wall is formed largely by the tympanic membrane and to a lesser extent by the bony outer attic wall called scutum.[26]

Mastoid Antrum:

It is a large air containing space in the upper part of mastoid cavity that communicates with the attic through the aditus. The lateral wall of antrum is formed by a plate of bone which is on an average 1.5cm thick in the adults. It is marked externally, on the surface of the mastoid by suprameatal triangle, also known as the Macewen's triangle.[26]

Inner Ear:

The inner ear lies in the temporal bone. It is also called the labyrinth and consists of two parts: -

1. The osseous periotic labyrinth
2. The membranous otic labyrinth

The osseous labyrinth:

It comprises of three main parts: the vestibule, the 3 bony semicircular canals and the bony cochlea.

The vestibule:

This is a small ovoid bony chamber, about 5 mm in length. It is placed between medial wall of the middle ear and the outer part of the internal auditory canal.[26]

The fenestra ovale, in the lateral wall of the vestibule, is separated from the middle ear by the footplate of stapes and its annular ligament. A small aperture in the posterior part of the medial wall of the vestibule leads into the aqueduct of the vestibule, which is a small canal which passes backwards to the posterior surface of the petrous bone, where it opens under the dura.[26]

The bony semicircular canals:

These open into the posterior part of the vestibule by five round apertures. The two vertical canals join posteriorly to form the crus commune. The superior semicircular canal lies almost transverse to the long axis of the petrous bone. Its highest point lies beneath the arcuate eminence, on the anterior surface of the petrous.[26]

The posterior semicircular canal lies in a plane parallel to the posterior surface of petrous bone.[26]

The horizontal semicircular canal lies in the angle between the superior and posterior canals. It makes a bulge on the medial walls of the attic, the aditus and the antrum.[26]

The bony cochlea:

It lies in front of the vestibule and resembles a snail shell in shape. It coils for 2 ¾ turns, i.e. a distance of about 35 mm around a central bony axis called the modiolus. The modiolus is thick at the base, but rapidly tapers towards its apex. The osseous labyrinth is lined throughout with a delicate endosteum, and contains perilymph fluid, in which the membranous labyrinth is situated.[26]

The membranous labyrinth:

It comprises of a continuous series of communicating sacs and ducts within the bony labyrinth. It consists of: the saccule and utricle, in the bony vestibule; the three membranous semicircular ducts, in the bony canals; and the ductus cochlearis. The membranous labyrinth contains endolymph fluid.[26]

The saccule and utricle:

The utricle occupies a depression on the upper wall of the vestibule. On the other hand, the saccule is smaller and lies in a depression below and in front of the utricle. These two sacs communicate indirectly by means of a slender membranous tube called the endolymphatic duct. This duct occupies the bony aqueduct of the vestibule and divides into two branches which separate to open respectively into the utricle and the saccule.[26]

The endolymphatic duct has an initial dilatation, known as the sinus, before it narrows to enter the bony aqueduct. The aqueduct enlarges beyond the isthmus of the duct; and in this expanded portion, the duct is surrounded by vascular connective tissue which forms the proximal part of the endolymphatic sac. The relatively smooth distal part of the sac is contained within the dura mater covering the posterior surface of the petrous pyramid, where it ends in close proximity to the sigmoid

sinus. A short, narrow tube, called the ductus reuniens, connects the saccule with the duct of the cochlea.[26]

The vestibular receptor organs:

A special sensory epithelium, known as the crista, is found in each ampulla, and is supplied by a branch of the vestibular division of the eighth cranial nerve.[26]

In the utricle and saccule, also consists of a patch of specialized epithelium, called the macula. The epithelium of these receptor organs contains three basic structures: sensory cells or the hair cells; the supporting cells; and a gelatinous substance secreted by the supporting cells.[26]

The vestibular cells are of two types: the type 1 cell, is round and flask- shaped. It is surrounded by a nerve chalice; and the type 2 cell, which is cylindrical and has no chalice.[26]

THE MECHANISM OF SOUND CONDUCTION:

The dynamics of the tympanic membrane, was studied by Dalimann and Bekesy. From their researches we know that the tympanic membrane vibrates in a manner quite different from that of a simple stretched elastic membrane. Bekesy used an electrical probe to measure the linear displacement of the membrane. This method has the advantage of allowing normal vibrations to be measured, uninfluenced by the loading effects due to the weight of optical reflecting devices.[27]

The transformer mechanism of the middle ear:

In a normal middle ear, a considerable degree of impedance matching is done, so that, while the amplitude is greatly reduced at the oval window as compared to the amplitude at the tympanic membrane, the force of the vibrations at the oval window is increased in the same proportion. This desirable effect depends on:

1. **The ossicular chain lever ratio:**

The malleus and incus jointly act as a lever, pivoting upon the axis of rotation. The malleolar arm is longer than the incudal arm in the ratio of 1.3:1. The expected lever ratios are in fact operative in the presence of sound energy has been shown experimentally by Wever and Lawrence.[27-28]

2. **The areal ratio of the tympanic membrane and oval window:**

There is a hydraulic effect between these two structures, thus increasing the force of the vibrations at the oval window. If it is assumed that the sound energy accepted by the tympanic membrane reaches the oval window undiminished, the increase in force will be in the same ratio as the ratio between the effective area of the membrane and the oval window. On the basis of the equal displacement contours of Bekesy, it can be deduced that the effective area for the tympanic membrane is two third of the anatomical area. The effective areal ratio between these two structures is 14:1.[27-28]

The overall ratio for the middle ear is the product of the ossicular chain lever ratio and the areal ratio between the tympanic membrane and the oval window. This gives an approximate figure of 18.3. By definition the impedance transformation ratio is the square of this figure and is therefore 336. We have already seen that the ratio of acoustic impedance of air and water is 3880, hence it is evident that impedance matching due to the middle ear is lesser than what is ideally required. [27-28]

3. Phase differential between oval and round window.

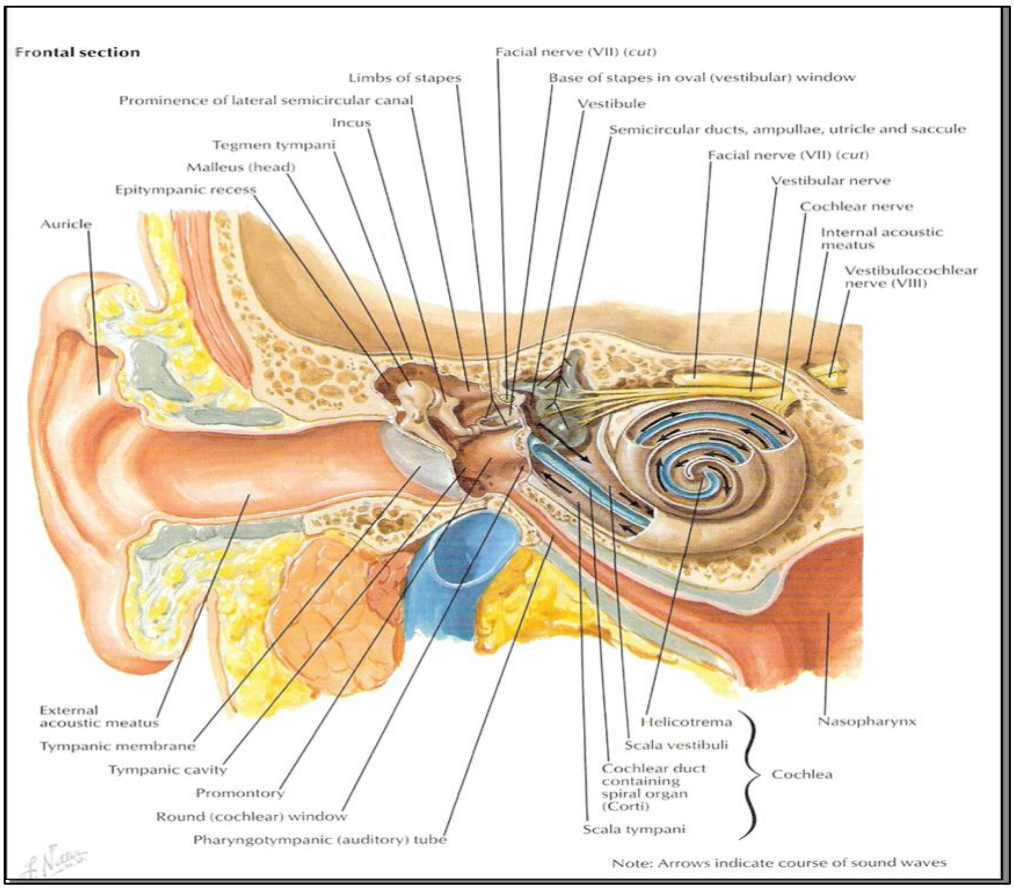

Figure 1: Section of ear and its relationship with other structures.

The ossicles:

I. **Malleus (Hammer):**

Malleus is the largest of the ossicles. It comprises of a head, a neck and three processes (anterior process, lateral process and the handle) arising from below the neck. The overall length of the malleus ranges from 7.5 mm to 9 mm. The head of the malleus has a saddle-shaped facet on its posteromedial surface to articulate with the body of the incus. The lateral process is a prominent landmark on the tympanic membrane and receives the anterior and posterior malleolar folds from the tympanic annulus.[9-11, 29-34]

II. **Incus (Anvil):**

Incus articulates with the malleus and has a body and two processes. The body lies in the epitympanum and has a cartilage covered facet corresponding to that on the malleus. The short process projects backwards from the body to lie in the fossa incudis. The long process descends into the mesotympanum posteriorly and medially to the handle of the malleus. The tip of the incus is small and is medially directed forming the lenticular process which articulates with the stapes.[9-11, 29-34]

III. **Stapes (Stirrup):**

The stapes consists of a head, a neck, two crura and a footplate. The head points laterally and has a small cartilage covered depression for articulation with the lenticular process of the incus. The stapedius tendon inserts into the posterior part of the neck and upper portion of the posterior crus.[9-11, 29-34]

The ossicular chain:

The mode of vibration of the various elements of the ossicular chain can be deduced from their dimensions and from the arrangements from their ligamentous supports and joint surfaces.[27]

The malleus and incus vibrate as a combined unit, rocking on a linear axis which runs from the anterior ligament of the malleus to the attachment of the short process of the incus in the fossa incudis. When reciprocating movements of the conducting system take place the mass of the body of the incus and the head and neck of the malleus, help to balance the mass of the drumhead, malleus handle, long process of incus and stapes which lies below it. The stapes often imagined to move in and out in the oval window niche with the simple movement of a piston.[27]

Sound conduction mechanism of inner ear:

Cochlea:

The scala media or the cochlear duct is triangular in shape and contains endolymph. The basilar membrane forms the horizontal limb of the triangle, the superior limb is formed by the Reissner's membrane, while on the vertical side, the stria vascularis and the spiral ligament is present.[9-11, 29-34]

The scala vestibuli and scala tympani contain perilymph. All of the structures of the cochlear duct and, particularly, the basilar membrane have a morphologic gradient, whereby the width of the basilar membrane is the narrowest at the basal end and widest at the apex.[9-11, 29-34]

The spiral ligament and the epithelial elements in the Organ of Corti, determine the location of maximal stimulation of the basilar membrane and inner hair cells by a given tone or frequency that is introduced to the inner ear. In this way, high frequencies are located at the base and low frequencies at the apex.[9-11, 29-34]

Vibration from the stapes accounts for the transfer of acoustic energy from the oval window to the hair cells. These vibrations produce a flow of perilymph up the scala vestibuli, through the helicotrema and down the scala tympani to the round window membrane. With vibrations, movement of basilar membrane occurs, which sets up a shearing force between the tectorial membrane and hair cells. The distortion of hair cells gives rise to cochlear microphonics which triggers the nerve impulses.[35]

Cochlear fluids:

The cochlear fluids are recognized as an essential part of the sound conducting mechanism, as indeed are the basilar membrane and all the vibrating structures within the cochlea. The mechanical loading on the inner surface of the stapes footplate is provided by the vibratory system between the

fluids and the cochlear membranes. This acoustic impedance is an essential component in the conducting mechanism to which the external ear is matched.[27]

Organ of Corti:

The Organ of Corti is a complex sense organ that contains inner and outer hair cells, as well as supporting cells which rest on the basilar membrane. The ciliated ends of these hair cells protrude into a covering structure, known as the tectorial membrane. The apical portion of the hair cells are anchored to the cuticular plate via the stereocilia (100 to 150 per cell).[9-11, 29-34]

The stereocilia of the outer hair cells are in contact with the tectorial membrane, whereas the stereocilia of the inner hair cells lie free in the endolymphatic space inferior to the tectorial membrane. There are a single row of inner hair cells and three to five rows of outer hair cells. The Organ of Corti contains approximately 15,500 hair cells, with about 3,500 of them being inner hair cells and 12,000 being outer hair cells.[9-11, 29-34]

The stereocilia present on the apical surface of the hair cells are mechanically rigid, and are faced together with cross links so that they move as a stiff bundle. [35]

Therefore, when a bundle is deflected by the movement of fluid, the different rows of stereocilia could be expected to slide relative to one another. There are fine links running upwards from the tips of the shorter stereocilia on the hair cell, which join the adjacent taller stereocilia of the next row. When the stereocilia are deflected in the direction of the tallest stereocilia, the links are stretched, opening the potassium and calcium channels in the cell membrane.[35]

When the stereocilia are deflected in the opposite direction, the tension is taken off the links and the channels close. This hypothesis is consistent with the present electro-physiological evidence from hair cells.[35]

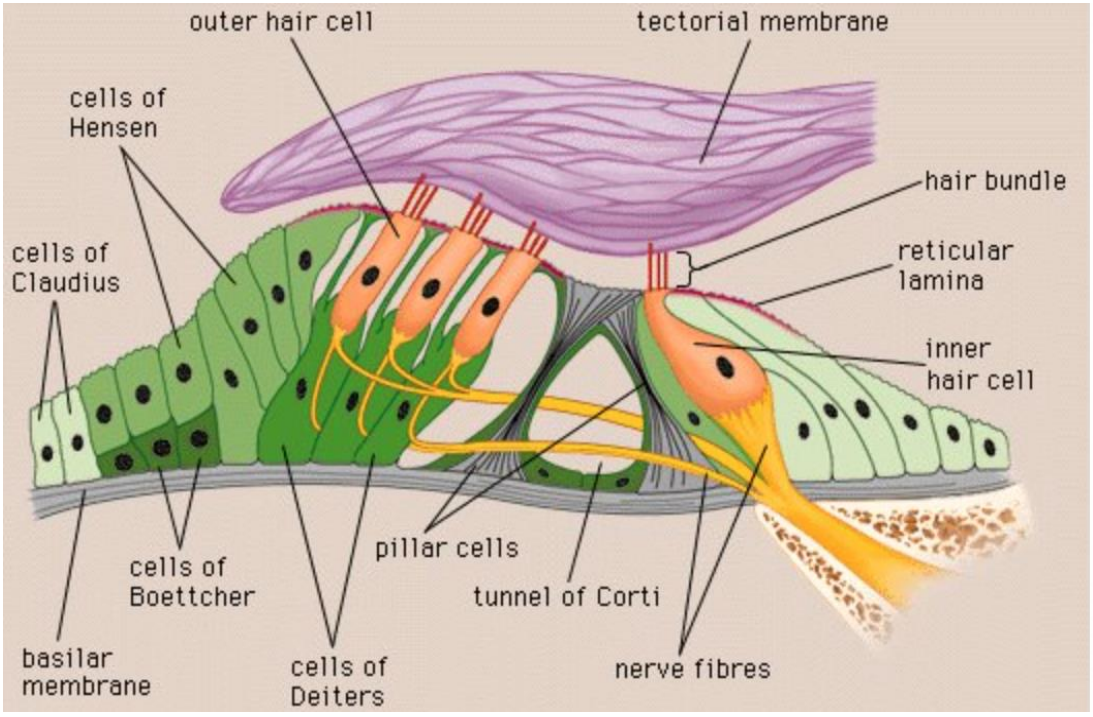

Figure 2: Organ of Corti.

Innervation:

The Organ of Corti is innervated by two types of nerve fibres. Afferent fibres from the auditory portion of the VIII cranial nerve, conduct impulses from the hair cells to the brain. Efferent fibres of the olivocochlear bundles conduct nerve impulse from the brain to the hair cells.[9-11, 29-34]

Afferent Innervation:

The auditory portion of the VIII cranial nerve, provides the afferent innervations of the inner and outer hair cells. The cell bodies of the cochlear nerve are located in the modiolus within Rosenthal's canal. Here, they form the spiral ganglion. The axon of each ganglion cell extends to the synapse in the cochlear nucleus.[9-11, 29-34]

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The peripheral process ends as a dendrite beneath the hair cells. The spiral ganglion in the human cochlea is composed of 2 types of neurons. Approximately 9% of the 30,000 ganglion cells are referred to as the type I cells. They have large bipolar cell bodies and are surrounded by myelin sheath. The remaining 5% of ganglion cells are the type II cells and have small pseudomonopolar cell bodies.[9-11, 29-34]

Efferent Innervation:

The efferent innervation of the hair cells is provided by the olivocochlear bundle. The cell bodies of the olivocochlear bundle are located in the brain stem within the superior olivary complex, primarily in the region surrounding the lateral, medial and superior salivary nuclei.[9-11, 29-34]

Acoustic Nerve:

The acoustic nerve consists of two distinct sets of fibers, the cochlear fibers and the vestibular fibers. The peripheral part of the cochlear and the vestibular nerve join to form the common acoustic nerve in the internal auditory canal. It transfers impulses from the Organ of Corti to the auditory cortex. The receptor cells are hair cells that lie along the entire length of the Organ of Corti in the cochlear duct.[9-11, 29-34]

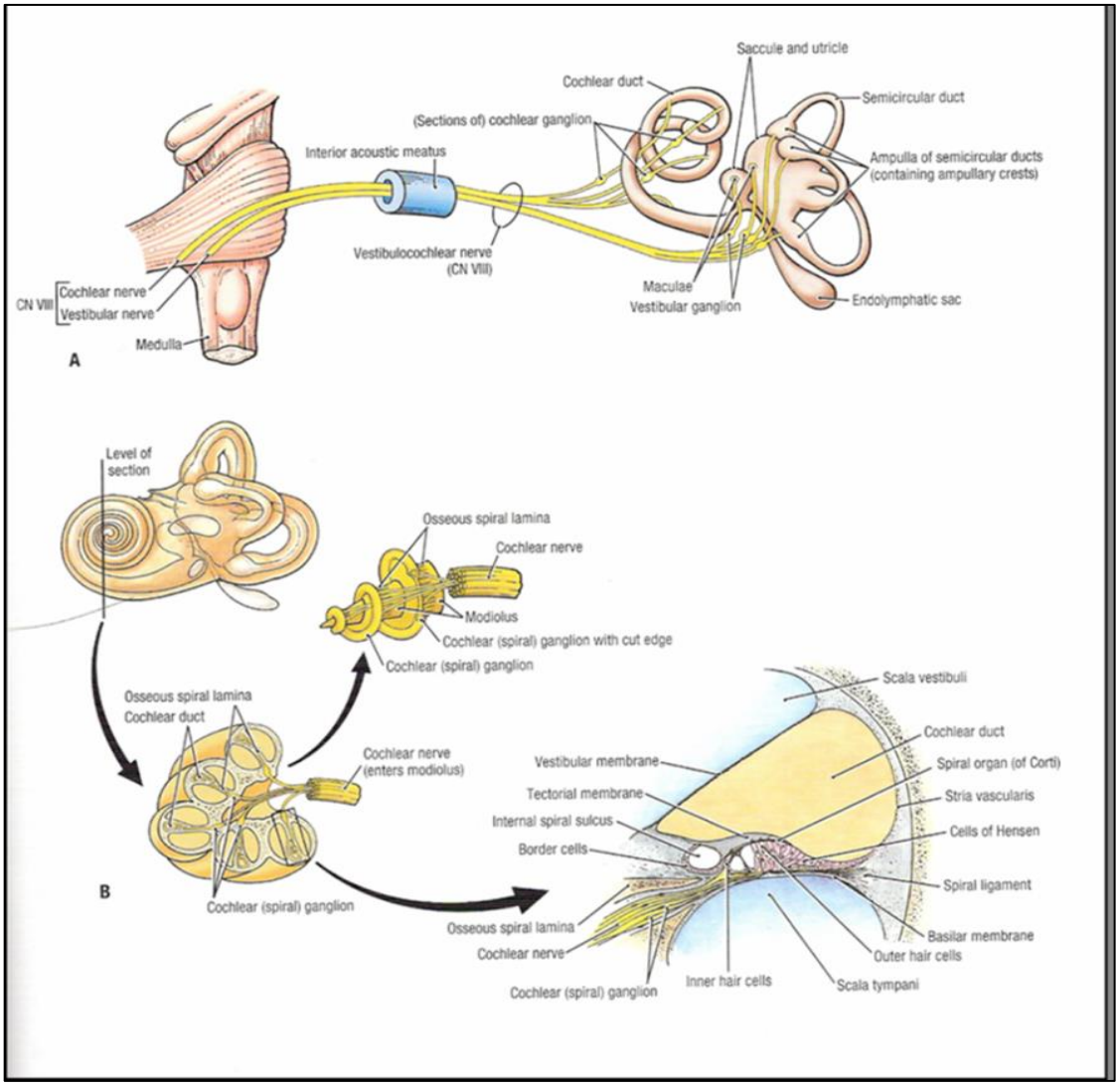

Figure 3: Structure of inner ear and inner ear innervation.

Otoacoustic emissions and the inner ear:

Unlike other sensory receptor systems, the inner ear appears to generate signals of the same type as it is designed to receive. These sounds, called otoacoustic emissions (OAEs), have long been considered as by-products of the cochlear amplifier, the process that makes cochlear mechanics active by adding mechanical energy at the same frequency as a stimulus tone in a positive feedback process. This feature of the inner ear is one of the most important distinctions from other sensory receptors.

In no other system is there such a profound collective reciprocal action of a population of sensory cells and supporting structures. In the eye, for example, the sensitivity of individual

photoreceptors is essentially determined by the light absorption properties of the visual pigment, the rest of the eye serves passively to couple the light entering the pupil to the array of photoreceptors. The chemistry of the photopigments seems designed such that, light absorption is virtually irreversible, thus, is no apparent way for the eye to operate like the inner ear, where the receptor cells interact with accessory structures in a feedback loop to enhance sensitivity.[35]

MODES OF SOUND TRANSMISSION:

There are two transmission pathways by which physical sound waves can be transformed into mechanical vibrations, which in turn stimulate the inner ear.

(a) Air conduction:

It is the process by which an acoustic signal travels through the structures of the outer and middle ear and arrives at the cochlea.[35]

(a) Bone conduction:

It's the process by which an acoustic signal vibrates the bones of the skull to stimulate the cochlea. Skull bone vibration can be a result of acoustic or mechanical stimulation of the skull.[35]

Air conduction pathway is the primary transmission pathway for reception of information about the acoustic environment by a person with normal hearing. The external and middle ear mechanisms are designed to channel and enhance acoustic information, optimize its conversion into mechanical vibrations of the ossicular chain, and deliver it to the mechanoneural converter of the cochlea. The bone conduction pathway bypasses the external and the middle ear mechanisms, resulting in suboptimal sound transmission to the cochlea.[35]

The main difference between hearing through air conduction and bone conduction is the manner in which the cochlea receives its stimulation. In the air conduction process, sound energy travels in a unidirectional manner, down the EAC vibrates the tympanic membrane, travels across the ossicular chain and creates movement of the stapes against the oval window.[35]

In bone conduction, the bones of the skull vibrate and, depending on the direction of stimulation, the stapes remains steady or vibrates with some time lag due to inertia. The vibrations of the skull lead to vibration of the fluids in the cochlea. Neural impulses produced within the cochlea are sent to the brain to be interpreted as sound.[35]

Bone conduction mechanism:

In 1966, Tonndorf identified mechanisms of bone conduction, at operate at both high as well as low frequencies.[36] These mechanisms are as follows:

(1) Inertial mode:

In this the whole skull vibrates as a unit, making oscillatory movements in the direction of an acting force.[36]

(a) Inertial inner ear mechanism:

In this, the vibrations of the skull are transmitted directly to the inner ear through the vibrations of the temporal bone surrounding the ear (osseous pathways).[36]

(b) Inertial middle ear mechanism:

Mechanism by which vibrations from the skull cause relative movements of the ossicular chain due to differences in inertia of the individual as well as the bones (osseotympanic pathways).[36]

Guild *et al.* hypothesized that the osseous pathway connecting the medial part of the posterior wall of the EAC to the lateral aspect of the horizontal semicircular canal is the most important temporal bone inertial pathway to the inner ear fluids.[37]

(2) Compressional mode:

Here the skull is divided into a number of parts that vibrate in opposite directions, creating pulsating movements of the bony structure.[36]

(a) Compressional inner ear mechanism:

Mechanism by which the compressional vibration of the temporal bone moves the cochlear fluids (osseous pathways).[37]

(b) Compressional outer ear mechanism:

Mechanism by which vibration from the osseous portion of the EAC is radiated back to the inner ear along the air conduction pathway (osseotympanic pathways).[37]

CHEMISTRY OF THE COCHLEAR FLUIDS:

Perilymph has a composition much like that of extracellular fluids while on the other hand, endolymph has a remarkably high potassium concentration and a low sodium content, similar to that of intracellular fluids.

The ratio between the high potassium levels and the low sodium levels leads to the following:

1. The walls of the scala media present a barrier to the passage of electrolyte ions.
2. The positive endolymphatic potential cannot be due to electrolyte concentrations because these would result in a negative voltage.
3. The hair cells and non-medullated nerve fibres of the Organ of Corti cannot be bathed in endolymph. This obeys the neurophysiological fact that nerve action potentials cannot arise in the presence of high potassium concentration outside the polarized membrane of nerve cells.

On electron microscopic evidence, Engstrom believes that the Tunnel of Corti is completely closed in every direction, and that the cortilymph is an intra epithelial accumulation of intracellular fluid. Other evidence indicates that the cortilymph is chemically similar to perilymph and that it is probably derived from the scala tympani. Schuknecht and Seifi demonstrated in a cat, the presence of minute openings in the osseous spiral lamina which led from scala tympani to the habenula perforata and Organ of Corti.[38]

Axelsson believed that nearby vessels, in the basilar membrane and the tympanic leak may be of some importance in the formation and absorption of the cochlear fluids.[39]

Naftakin and Harrison suggested that endolymph is derived from perilymph across Reissner's membrane and is reabsorbed by stria vascularis. They account for the reversed electrolyte concentration by suggesting that the stria vascularis is selective, leaving potassium in the endolymph at a raised concentration. This point is reinforced by analogy with similar tonic adjustment by renal tubules, and the possible influence of aldosterone upon these processes in the inner ear is raised.[40]

CHRONIC SUPPURATIVE OTITIS MEDIA:

CSOM remains one of the most common childhood infectious diseases worldwide, affecting diverse racial and cultural groups both in developing and industrialized countries. It involves considerable morbidity and can cause intracranial as well as extracranial complications.[41]

CSOM can be defined as a chronic inflammation of the middle ear and mastoid mucosa in which the tympanic membrane is not intact (perforation or tympanostomy tube) and discharge (otorrhea) is present.[42-44] There is, however, no consensus about the duration of the symptoms.

The World Health Organisation[45] defines CSOM as ''otorrhea through a perforated tympanic membrane present for at least 2 weeks'', while others define 'chronic' as symptoms persisting for more than 6 weeks.[46-49]

Since it is accepted that CSOM is preceded by incompletely or unsuccessfully treated acute otitis media treated,[50-52] these variations in the definition of duration of symptoms suggest that the transition from otorrhea as a sign of AOM to that of CSOM is not clearly established.

CSOM should be distinguished from tympanostomy tube otorrhea, which is the most common complication of tympanostomy tube placement.[52-53] At the same time, CSOM should be distinguished from chronic otitis media with effusion, in which no perforation or active infection is present, as well as from a chronic perforation of the tympanic membrane, in the absence of middle ear infection.[54]

Eustachian tube dysfunction:

The eustachian tube has three important functions with respect to the middle ear: ventilation, protection, and clearance. Both endogenous and exogenous factors can impair these functions and therefore cause otitis media.[55,56]

When a perforation of the tympanic membrane is present, the middle ear ‘‘gas cushion’’ is lost, resulting in reflux of nasopharyngeal secretions through the ET and subsequent contamination of the middle ear with potential respiratory pathogens.[56]

Infants and young children are especially at risk for such a reflux because their ET are short, horizontal, and floppy.[57] Reduced ciliary function of the middle ear and ET mucosa has been associated with impairment of clearance of the middle ear secretions and may, therefore, facilitate the progression from AOM and OME into CSOM.[58,59] The gastroesophageal reflux may also contribute to the eustachian tube dysfunction and consequent middle-ear infection.[60,61]

Classification of CSOM:

1) **Mucosal type (Tubotympanic):** Perforation of the pars tensa with an intact annulus. It is of 2 types: active and inactive.

2) **Squamosal type (Atticoantral):** Perforation in the posterosuperior quadrant involving the annulus or an attic perforation or a retraction pocket present in the pars flaccida. Can be of 2 types: active and inactive.

RESONANCES OF THE HEAD:

The first theory of human skull vibrations was proposed in 1932 by Bekesy who considered the skull to be a thin sphere that is able to vibrate in several different modes because of the distributed elasticity and density of its structure.[62]

Low frequencies have longer wavelengths than high frequencies. Therefore, frontal excitation of the skull at low frequencies whose wavelengths are larger than the dimensions of the head makes the elements of the skull move together as a single vibrating body (inertial vibrations). However, this does not occur for high frequencies.[62]

At high frequencies, the back of the head gradually begins to lag behind the vibration of the forehead because of the difference in inertia produced by various parts of the head. This lag facilitates development of standing waves in the skull bones, which divide the skull into several vibrating elements. The vibration mode of the skull changes at approximately 600 Hz. Thus, above 600 Hz, the skull can no longer be considered as a single vibrating object (lumped system) but as a system of small masses connected together by elastic links (distributed system).[62]

Bekesy identified the first natural resonance of the head to be around 800 Hz. In this mode of vibration, the head vibrates as a front-back oriented dipole. Above 800 Hz, the unidirectional front-back compressional mode of head vibration gradually changes into the second compressional mode where the head begins to vibrate as two out-of-phase pairs of elements moving along the medial and lateral axes in such a way that the skull interchangeably elongates and widens. The second natural resonance corresponding to this mode of vibration was reported by Bekesy to be around 1600 Hz.[62]

INTERAURAL ATTENUATION:

When acoustic signals are delivered to the ears, the head provides some degree of isolation between them. This isolation is referred to as the interaural attenuation.[63]

In the case of bone-conducted sounds, the term interaural attenuation is replaced by the term transcranial attenuation, reflecting cranial rather than aural stimulation. If the vibratory signal is delivered through a vibrator placed in the median plane of the skull, the TA is practically zero because of symmetrical attenuation effects on the sound reaching both cochleae.[63]

If the vibratory signal is delivered through a location on the side of the head, the TA is not zero because of the differential attenuation of the sound produced by the structures of the head as it reaches each of the cochlea. Stenfelt and Goode reported that the TA for a vibrator placed on the side of the head is less than 5 dB in the 250 to 500 Hz range but increases with frequency to about 15 to 20 dB in the 2000 to 4000 Hz range.[63]

TYPES OF HEARING LOSS:

Hearing loss falls into three broad categories: conductive hearing loss, sensorineural hearing loss and mixed hearing loss.

SNHL, can be attributed to problems within the inner ear, primarily the cochlea and associated hair cells or the vestibulocochlear nerve. It can be caused by either intrinsic factors such as genetic aberrations resulting in congenital abnormalities, or extrinsic factors such as inner ear infections; ototoxic drugs such as aminoglycosides and cisplatin; or exposure to high noise levels both over an extended period of time such as in an industrial workplace, prolonged use of headphones or a single discrete event such as a blast of noise from equipment, gun shots, or bomb blasts.[64]

Noise induced hearing loss:

Noise is referred to as an intense sound capable of producing damage to inner ear. A possible definition of NIHL is permanent damage to the outer hair cells of the cochlea resulting in reduction of the amplification ability of the cochlea.[64]. NIHL may be temporary and is described as temporary threshold shift although strict definition regarding duration is not available and may be from hours to days.[65]

The hearing loss may be permanent and this is described as permanent threshold shift. A PTS may occur following repeated TTS, or following a single episode of noise exposure. The term 'acoustic trauma' has, however, been utilized to describe the situation where a single exposure to an intense sound leads to an immediate hearing loss.[66] Most of the studies consider that TTS is associated with metabolic changes in the cochlea, whereas PTS occurs due to structural changes in cochlea.[67]

The Occupational Safety and Health Standards have made guidelines that state, an employee should not be exposed to sound levels of more than 90 dB averaged over 8 working hours for five days a week. As doubling of sound energy leads to a change in the sound pressure level by 3 dB, OSHA has recommended reducing time by half for every 3 dB increase in SPL for continuous sound exposure.[68]

Various studies have shown that the sound produced during drilling is in the range of 100-125dB.[69-71] A similar level of sound reaches the opposite ear during drilling as the interaural attenuation for bone conduction is 0 to 5 dB. The mean time of drilling is around 30 minutes. This is considered to be higher which is more than the prescribed safety limits as per OSHA guidelines.[72]

Hearing Loss by powered surgical instruments:

Hearing loss as related to powered surgical instruments has primarily been studied from two perspectives: noise levels (air conducted) and vibrations (bone conducted). Such a type of hearing loss is measured by the degree to which the hearing threshold sensitivity has risen and is classified as

either a permanent threshold shift or a temporary threshold shift. Most sensorineural hearing loss caused by powered surgical instruments fortunately falls into the temporary threshold shift category.[73]

Apart from these two types of hearing loss caused by powered surgical instruments, physical contact of an instrument with the ossicular chain results in vibrations which are transmitted to the cochlea. The subsequent damage to the cochlea generally results in permanent hearing loss. However, this hearing loss can be attributed to surgeon error rather than conventional use of the surgical instrument itself.[73]

Powered surgical instruments cause vibrations of the skull, thus causing SNHL. However, in accordance to this, the occupational standards are not widely classified as compared to the damage caused by hand-held heavy equipment on the circulatory system.[74] According to Seki *et al.*[75] and Miyasaka [76], these vibrations alter the morphology, specifically the permeability of the capillaries present in the stria vascularis. Vibration induced hearing loss has been associated more with the use of surgical drills, than in comparison to ultrasonic devices. [77-79]

PURE TONE AUDIOMETRY:

Pure tone audiometry is a type of hearing test by which the hearing acuity or hearing threshold of a subject for pure tone sounds of various frequencies, can be assessed.

The hearing threshold as defined as the lowest sound pressure level at which, a person gives a predetermined percentage of correct responses on repeated trials. The result when plotted graphically is called a pure tone audiogram.[80]

Procedure:

Tones of different frequencies and decibels is presented to the subject for 1 to 3 seconds with intervals of 1 to 3 seconds between each presentation. The subject responds as soon as he hears the

sound, for example by raising the finger or pressing a button, which lights a signal on the audiometer panel, and maintains the response as long as the sound is heard.

Method to assess air conduction:

Carhart and Jerger, modified the conventional technique for assessment of air and bone conduction thresholds.[80]

(a) The better ear is tested first. Various frequencies ranging from 125 Hz to 8000 Hz is presented to the subject. If the patient hears the sound, the tone is decreased by steps of 10 dB until patient stops hearing. Once this stage is reached the tone is raised by 5 dB. If the patient hears this tone, the sound is again decreased by 10 dB. If he does not hear it, the tone is again raised by 5 dB. In this way by several threshold crossings, the exact hearing threshold is obtained when one gets at least 3 out of 5 responses correct.[80]

(b) The second ear is tested in a similar manner. The faintest audible intensity as established above is recorded against the test frequency on a standard audiogram chart as the threshold intensity. By convention, the symbols ‘o’ and ‘x’ are used to represent the air conduction thresholds for the right and left ear respectively.[80]

Technique to assess air conduction:

Bone conduction thresholds are obtained in an identical manner to those described for air conduction, but the sound stimulus is produced by a bone vibrator which is placed on the mastoid process and held firmly, by means of a head band.[80]

Measurements are restricted to a frequency range of 250 to 4000 Hz. The subject is instructed to respond to sound regardless of the side on which the sound is actually heard. It must be emphasized

that without the use of masking it is not possible to determine the ear that is responsible for the detection of the 'non-masked' bone conduction threshold. By convention, the symbols ' < ' and ' > ' are used to denote the bone conduction thresholds for the right and left ear respectively.[80]

OTOACOUSTIC EMISSIONS:

Generation of otoacoustic emissions:

OAEs are sounds generated within the normal cochlea, either spontaneously or in response to acoustic stimulation. The measurement of OAEs were reported first by David Kemp in 1978. It is also known as Kemp echoes and cochlear echoes. Otoacoustic Emissions are believed to reflect the activity of active biological mechanisms within the cochlea responsible for the exquisite sensitivity, sharp frequency selectivity and the wide dynamic range of the normal auditory system.[81]

When sound is used to elicit an emission, it is transmitted through the external ear, where the auditory stimulus is converted from an acoustic signal to a mechanical signal at the tympanic membrane and is transmitted through the middle ear ossicles; the stapes footplate moves at the oval window, causing a traveling wave in the fluid-filled cochlea. The cochlear fluid's traveling wave moves the basilar membrane; each portion of the basilar membrane is maximally sensitive to only a limited frequency range.[81]

The arrangement is a tonotopic gradient. Regions closest to the oval window are more sensitive to high-frequency stimuli. Regions further away are most sensitive to lower-frequency stimuli. Therefore, for OAEs, the first responses returned and recorded by the probe microphone emanate from the highest-frequency cochlear regions because the travel distance is shorter. Responses from the lower-frequency regions, closer to the cochlear apex, arrive later.[81]

When the basilar membrane moves, the hair cells are set into motion and an electromechanical response is elicited, while an afferent signal is transmitted and an efferent signal is emitted. The

efferent signal is transmitted back through the auditory pathway, and the signal is measured in the EAC.[81]

The OHCs are located in the Organ of Corti on the basilar membrane. These hair cells are motile, thus an electrochemical response elicits a motoric response. The 3 rows of outer hair cells have stereocilia arranged in a W formation. The stereocilia are linked to each other and, therefore, move as a unit, and is believed to be involved in OAE generation.[81]

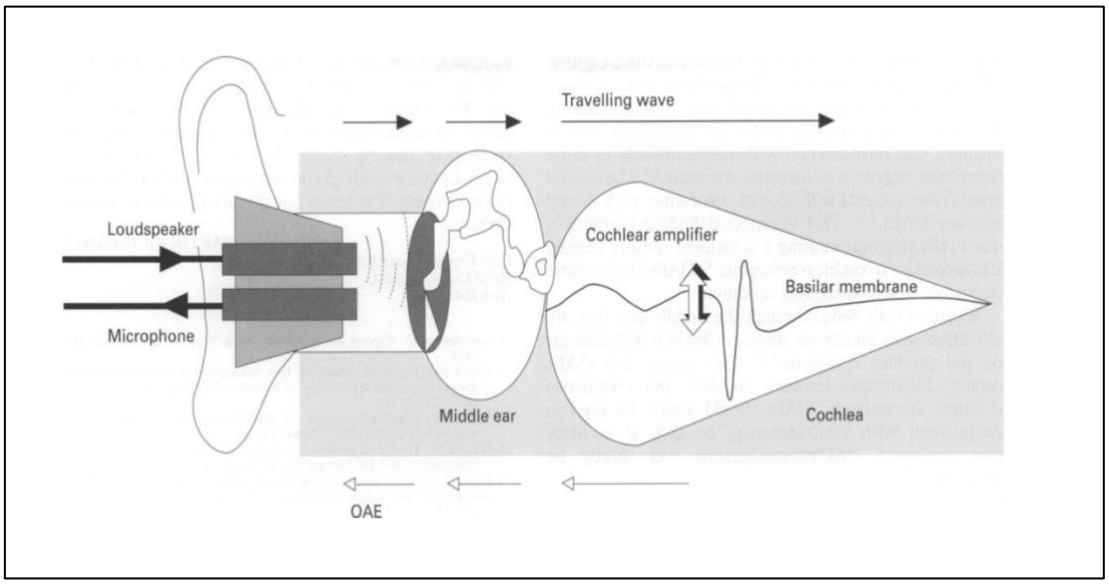

Figure 4: Physiology of OAE.

Recording of otoacoustic emissions:

The OAEs are on the order of -10 to 20 dB SPL. It is recorded with the help of a sensitive microphone that is coupled to a specialized computer which enhances the low level OAEs and reduces the unwanted signals in a process called signal averaging. This microphone is placed in the ear canal of the subject.[81]

Since the measurement of OAEs can be done in a few minutes, it is very popular in routine clinical use. It is a non-invasive, sensitive and objective test to assess functioning of outer hair cells.[81]

Absence of OHCs is associated with lack of OAEs, supporting the hypothesis that OHCs are responsible for OAE generation. OAEs are normally very stable with time and are valuable as a

sensitive monitor of changes in cochlear, especially in relation to sudden hearing loss, Ménière's disease and NIHL.[81]

Outer hair cells Vs Inner hair cells:

OAE is a preneural phenomenon. It can be measured even when the eighth nerve is severed. Otoacoustic emissions, which are particularly evoked by a low stimulus is vulnerable to acoustic trauma, hypoxia and ototoxic agents, thus causing hearing loss and damage to the OHC. On the other hand, OAE's do not appear to be vulnerable to selective loss of IHC.[82]

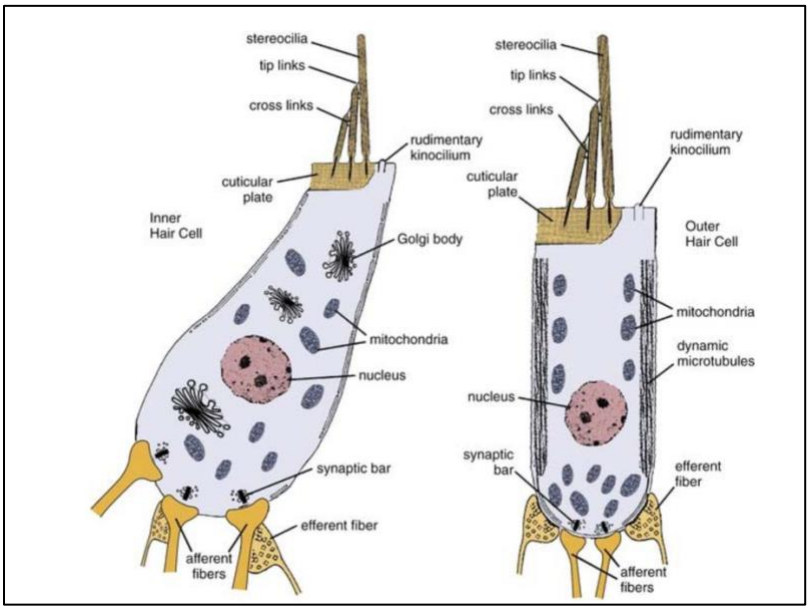

Figure 5: Structure of inner and outer hair cells.

Classification of otoacoustic emissions:

The two general categories of OAEs are spontaneous and evoked. The evoked type of OAE is determined by type of stimulus used. This includes:

(a) Transient evoked otoacoustic emissions.

(b) Distortion product otoacoustic emissions.

(c) Stimulus frequency otoacoustic emissions.

Spontaneous otoacoustic emissions:

SOAEs are continuous narrow-band signals emitted by about 50% of individuals, in the absence of any external stimulus.[83]

SOAEs are recorded in the ear canal with a probe consisting of a sensitive microphone with a low internal noise. The presence of OAEs is confirmed by the appearance of spikes that have an amplitude of 10-15 dB, at one or more frequencies.[84] They are generally absent in frequency regions with SNHL. However the clinical value of SOAEs is limited, as normal ears do not invariably produce them.

Transient evoked otoacoustic emissions:

TEOAEs are also known as click-evoked otoacoustic emissions.. TEOAEs are recorded in response to abrupt stimuli, such as a click or tone-burst. A click of short duration (0.1ms) has a broad spectrum. Such a transient stimulus activates the cochlea simultaneously across a wide frequency region. If the cochlea, specifically OHCs, are normal, then a robust and normal amplitude TEOAE should be recorded from low frequencies up to about 5000 Hz.[83,84]

Thus TEOAEs are obtained by using a synchronous time domain averaging technique. The recording of TEOAEs require a probe, which in turn consists of a sensitive low noise miniature microphone and a miniature sound source to deliver the stimulus. Responses to several stimuli are averaged to improve signal-to-noise ratio.[83,84]

Clinically, two response properties of TEOAE are quantified and described, namely the temporal waveform and the spectral waveform. The earliest portion of the temporal waveform reflect TEOAE activity produced by the basal region of the basilar membrane, whereas the later portion of the waveform arise from more apical region. Spectral waveform, which is in frequency domain, reveals the presence of TEOAE activity, above the noise floor, across a frequency region of 0 to 5000 Hz.[83,84]

For more precise analysis, the amplitude-noise difference should be calculated at individual frequencies. Any reduction in the TEOAE signal-to-noise ratio will reflect cochlear dysfunction.[83,84]

Most of the studies related to OAEs, are focused on click-evoked emissions. This is because they provide broad band, cochlea wide information. In presence of hearing loss, TEOAEs have been shown to decrease in incidence as the hearing threshold increases. Generally if hearing loss exceeds 40-50 dB, an emission cannot be evoked to a transient stimulus.[83]

TEOAEs also decrease in magnitude with age in a normal hearing individual. They are extremely robust in normal hearing, full-term new-born babies.[83,84]

Distortion product evoked otoacoustic emissions:

DPOAEs are elicited by the simultaneous presentation of two pure tones known as primaries which are closely spaced in frequency. These primaries are referred as f1 and f2 where f1 is the lower frequency and f2 is the higher. These primaries are separated by a ratio defined as f2/f1. A DPOAE will not be generated if these two primaries are far apart or too close together. A number of studies have suggested that a f2/ f1 ratio of 1.22, produces robust DPOAEs in most normal ears.[84]

The most appropriate stimulus intensity for evoking DPOAE is in the range of 50 to 70 dB SPL. Experimental evidence suggests that DPOAE amplitude is slightly larger and sensitivity to cochlear dysfunction is enhanced, when the intensity of f2 primary (L2) is lower than that of the f1 primary (L1).[84]

A clinical DPOAE device performs spectral analysis of energy in the ear canal following the stimulus and specifically searches for a peak in energy around this frequency region. This energy is calculated in dB and compared to noise in the same frequency region, which is always present in ear canal. Clinical studies have confirmed that the region of cochlea that is activated with this stimulus arrangement is closer to f2.[84]

The most commonly used format of distortion product measurement is the DP gram. In this the frequency is changed while the levels are kept constant. For each stimulus frequency the corresponding noise floor in the region of the DP frequency (2f1-f2) is plotted. To be considered as a valid DPOAE, the DP amplitude must exceed the noise floor value by at least 3 dB.[84] Chan *et al*, found that the screening criteria of 1500 and 2000 Hz, with a signal-to-noise ratio of > 0 or 3 dB, yielded high sensitivity and specificity in indicating possible occupational hearing loss in noise exposed individuals.[85]

Interpretation:

(a) The presence of robust evoked otoacoustic emissions across the speech frequency range (1000 to 4000 Hz) indicates a useful degree of normal function in both the middle ear and cochlea and further denotes that speech and language development will not be greatly impeded by peripheral auditory dysfunction.

(b) The absence of OAEs without middle ear pathology or acoustic obstruction strongly indicates sensory transmissive hearing loss.

(c) Depending on the type and intensity of stimulation, OAEs can reveal threshold elevations as small as 20 dB HL.

(d) The superior capacity of TEOAE to predict NIHL has been reported by Lapsley M *et al.*[86] This is due to the fact, that TEOAE responses originate from the entire length of the basilar membrane.[87] Whereas, DPOAE reflects the functioning of only limited population of OHC tuned to specific stimulus f2 frequency.[88] Thus, minor cochlear damage at numerous regions of the

basilar membrane might not be revealed by the frequency specific DPOAE but will have a significant impact on the broader tuned TEOAE response.

MICRO MOTOR DRILL:

The drill plays an important role in the development of hearing loss following mastoidectomy, as the efficiency of its main components, i.e. the drill system and the drill bits, will determine ease, safety, the time required and the noise produced during bone removal.

The drill should be light weight, generate high speed with minimum torque of the hand piece on initiation and cessation of drilling. Features like forward and reverse mode for burr movement and variable speed adjustment are useful. The drill speeds are variable in most drill systems, ranging up to 40,000 rpm. Higher the speed, more efficient is the bone removal with less pressure on the burr. But higher speeds can also make the drill run on the surface causing unintended collateral damage. Slower speeds are preferable during the final bone removal over delicate structures.

Schuknecht and Tondorff, along with Paulsen and Vietor concluded that bone conducted noise was of no importance for the development of high tone loss in patients, whereas air conducted noise would be dangerous for the surgeon.[89,90]

A study was conducted by Holmquist J *et al.* on the sound intensities produced by drilling in ear surgery. They established that the peroperative sound levels can reach up to 125 dB with mean sound level of 100 dB. The short process of incus is most susceptible to trauma from burr.[70]

A study conducted by Jiang D *et al.* suggests that drilling on the ossicular chain can produce vibratory force that is analogous with noise levels known to produce acoustic trauma. For the same type of burr, larger the diameter, greater is the vibratory force. The cutting burr creates a greater vibratory force than the diamond burr. The cutting burr produces more high frequency than lower frequency vibratory energy. The equivalent noise levels generated ranged from 93 to 125 dB SPL according to the burr used.[71]

MASTOIDECTOMY:

The goal of any ear surgery is to create a dry, safe ear and preserve or restore hearing as much as possible. A mastoidectomy is performed to help eradicate disease and gain access to the antrum, attic and middle ear. It also increases the air containing space in the middle ear cavity, thus allowing the middle ear to accommodate changes in pressure without causing a tympanic membrane retraction.

Absolute indications include cholesteatomas or tumours that extend into the mastoid bone.

Relative indications include:

- History of profuse otorrhea.
- Previous tympanoplasty failure.
- Secondary acquired cholesteatoma.
- A tympanic membrane perforation that cannot be corrected without further exposure provided by a mastoidectomy.

Although surgeons remain divided on the utility of the mastoidectomy in primary cholesteatoma surgery and tympanic membrane perforation repairs, most agree to its usefulness in revision cases after graft failure.[91]

Simple mastoidectomy:

A simple or cortical mastoidectomy involves removing the mastoid cortex and the underlying air cells. Dissection may be superficial or may proceed to the mastoid antrum. It is used to unroof the mastoid cortex and drain the coalescent mastoiditis with a subperiosteal abscess.[91]

Intact canal wall or complete mastoidectomy:

The canal wall up mastoidectomy involves removal of mastoid air cells lateral to the facial nerve and otic capsule bone while preserving the posterior and superior external auditory canal walls. This

technique affords access to the epitympanum while maintaining the natural barrier between the external auditory canal and mastoid cavity.[91]

In paediatric patients, this approach is preferred generally to avoid the long term problems associated with canal wall down procedures. This approach can be combined with a facial recess dissection for:

- Better exposure of the posterior mesotympanum around the oval and round window.
- Better visualization of the tympanic segment of the facial nerve.
- Better middle ear aeration.[91]

Modified radical mastoidectomy:

Although the classic description of a modified radical mastoidectomy is the atticotomy described by Bondy, most surgeons currently use the term to describe a canal wall down mastoidectomy.

There are both preoperative and intraoperative indications to remove the posterior meatal wall. Preoperative indications for a MRM include:

1. Disease involving the only hearing ear.
2. Cases where regular follow up is difficult.

Some surgeons advocate a canal wall down after multiple failed attempts at canal wall intact surgery.[92] The decision to remove the canal wall is made intraoperatively when one of the following is encountered:

1. Unreconstructible posterior meatal wall defect.
2. Labyrinthine fistula where the matrix cannot be resected primarily.
3. A low-lying dura, thus limiting access to the epitympanum.

Radical mastoidectomy:

A radical mastoidectomy is performed in patients with irreversible middle ear disease, unresectable cholesteatoma or tumours.

In this procedure, mastoid air cells are exteriorized and the posterior canal wall is removed. The middle ear along with the mastoid cavity is made into a large single cavity, with no attempt at reconstruction. The eustachian tube is occluded and both the malleus and the incus are removed.[92]

Canal wall-up procedures do not require regular debridement, and the hearing outcome is slightly better when compared to that of canal wall-down procedures.[93]

Mastoidectomy complications:

a) Facial nerve injury:

Injury to the epineurium or nerve sheath usually results in no long-term consequences.[94] If less than 40% of the nerve is injured and facial muscle contraction can be elicited with minimal stimulation of the proximal segment of the nerve, nerve grafting is not required. However, if more than 50% of the nerve is injured, superior results may be achieved through nerve grafting.[95]

Immediate facial paralysis in the postoperative period requires prompt evaluation. If paralysis persists beyond 4 hours, prompt re-exploration of the nerve is required. Conservative management with steroids, antibiotics, and antivirals is warranted in all cases of delayed facial paralysis.[96]

b) Infection:

Postoperative infections occur in 2-5% of all mastoidectomies. Infection may be the result of wound infection or continued chronic ear disease. Routine prophylaxis may not necessarily reduce postoperative infection rates.[97] Perichondritis may occur in approximately 1% of canal wall down procedures; therefore, perioperative antibiotics are used routinely in these procedures.[91]

c) Hearing loss:

SNHL may be the result of removal of cholesteatoma over labyrinthine fistulas or inadvertent contact between the drill and ossicular chain during dissection. Labyrinthitis may also lead to SNHL. Drill injuries usually result in a high-frequency sensorineural hearing loss.[91]

d) Vertigo:

Labyrinthine fistulas and injuries during mastoid surgery may alter the vestibular responses of an ear.[91]

e) Intracranial injury:

Exposure of the dura, thus leading to CSF otorrhea, may be seen in cases with tegmen erosion. Repair is generally through layered closure with soft tissue support including muscle and fascia grafts.[91]

f) Bleeding:

In MRM and radical mastoidectomy, postoperative bleeding is more due to increase in the soft tissue dissection; however, blood drains through the meatus and there is little risk for hematoma formation. Injury to large vascular structures like the sigmoid sinus, the jugular bulb and large emissary veins mandates immediate assessment. Bleeding often is controlled easily with gel foam and gentle pressure.[91]

DRILL INDUCED HEARING LOSS DETECTED BY PTA:

Domanech J *et al.* piloted a study, that demonstrated sensorineural high frequency hearing loss after drill generated acoustic trauma in tympanoplasty. Twenty-four patients with normal bone conduction audiometric thresholds scheduled for tympanoplasty were assessed, using a high

frequency audiometer, which can measure hearing frequencies up to 20,000 Hz. It was concluded that drilling of the temporal bone could impair the hearing level at high frequencies in a significant number of patients.[98]

In large clinical series, SNHL following middle ear surgery has been demonstrated in 1.2% to 4.5% of patients. It was suggested in these studies that drill generated noise is transmitted via the bone, and not via the ossicles, thus affecting higher frequencies.[99]

Singh A *et al.* studied the inherent risk of inner ear damage following middle ear surgeries in 60 patients. Bone conduction thresholds at different frequencies were recorded by a pure tone audiometer both pre-operatively and post-operatively. The over-all results showed 1 case (1.67 %) with severe SNHL of more than 25 dB and 10 cases (16.66 %) with mild to moderate SNHL including three cases of temporary threshold shift. In majority of the cases (11.67%) frequencies of 2000 and 4000 Hz were involved.[100]

Kylen and Arlinger performed drilling experiments on temporal bones in intact skulls. They also measured a noise level of 100 dB in the ipsilateral cochlea and that of 90 to 95 dB in the contralateral side, during drilling. They also stated that variations in rotation speed as well as the site of drilling did not appear to influence the noise level. The authors concluded that bone conducted noise trauma might be responsible for the high tone loss.[12]

DRILL INDUCED HEARING LOSS DETECTED BY PTA AND OAE:

Migrov L *et al.* conducted a study to determine the changes occurring in the outer hair cell function, in response to drill noise. Drill-induced noise can cause reversible changes in DPOAE in the non-operated ear. The OHC function is diminished post-operatively and persists for 1 month.[10]

Vashishth A *et al.* piloted a study on 30 patients with unilateral COM. On analysing the otoacoustic emissions, statistically significant changes were observed in DPOAE at a high frequency, while changes in TEOAE were noted at both low and high frequency ranges. High frequency pure tone audiometry also showed significant changes in air conduction thresholds.[11]

A retrospective study on temporary hearing deficits after ear surgery was done by Schick B *et al*. A total of 393 patients were assessed at hearing frequencies ranging from 500 to 4000Hz. They were evaluated daily for the first 4 days after surgery and then directly after 3 weeks. They stated that a slight temporary threshold shift is present at 2000Hz and 4000Hz, following ear surgery.[31]

Hattenbrink KB conducted a prospective study to assess the reaction of the cochlea after the trauma of middle ear surgery. For this purpose the bone conduction of 50 patients was tested every day, beginning on the first post-operative day. He concluded that excessive drilling may result in a temporary threshold shift, which resolved at the time of unpacking the ear.[32]

Karatas E *et al.* assessed the hearing of 22 patients, following mastoid surgery. Pure tone audiometry and otoacoustic emissions were utilized for baseline evaluation. OAEs were repeated during the immediate postoperative period and daily up to the 6th post-operative day. The amplitude of the OAEs in the contralateral ear was affected immediately after surgery and progressive improvement was detected with full recovery by 72-96 hours. None of the patients had permanent deterioration in OAE amplitudes. Thus, burrs used during mastoid surgery can cause temporary hearing threshold changes in the contralateral ears.[33]

In 2015, Baradaranfar MH *et al.*[101] conducted a prospective study, assessing the hearing threshold recovery in the contralateral ear following mastoidectomy. PTA and DPOAE were performed in all contralateral ears before and 6, 24, 48, 72 and 96 hours after surgery. Post-operatively, the survival rates at frequencies of 3000 Hz, 4000 Hz, 6000 Hz, and 8000 Hz were 44.4%, 36.4%, 51.7%, and 47.4%, 24 hours after surgery; 11.1%, 9.1%, 10.3%, and 13.2%, 48 hours after surgery; and 0%, 0%, 3.4%, and 2.6%, 72 hours after surgery, respectively. All patients had normal PTA and OAE readings by POD-4. The authors therefore concluded that high-frequency hearing loss usually occurs following mastoid surgeries that is mainly temporary and reversible after 96 hours.

Özdamar K *et al.*[102] piloted a comparative study in 2015, demonstrating the inner ear damage caused by drilling. The authors observed that, among the patients that underwent mastoidectomy, the DPOAE values on post-operative day 1, 2, 3 and 4 were significantly lower, at a frequency of 4000 Hz. They established that the drilling done in mastoid surgeries can damage the healthy contralateral ear. This damage can be determined by otoacoustic emissions in the early period.

In the year 2016, Abtahi SH *et al.*[103] conducted a prospective clinical study on 23 patients and assessed their hearing using PTA, TEOAE and DPOAE. Patients were evaluated on post-operative day 1 and 7. On POD-1, a significant decrease in PTA, TEOAE and DPOAE was noted. Lower frequencies were affected in PTA, however on OAE, changes were noted at both high and low frequency ranges. All patients recovered by post-operative day 7.

Jerath V *et al.*[104], in the year 2018, studied the effect of drill noise on contralateral hearing after mastoidectomy in 25 patients, using PTA and TEOAE. The average duration of drilling was 30 minutes. They established no change in the bone conduction threshold of the contralateral ear, on PTA. However, there was significant worsening of TEOAE readings at all frequencies (500 to 4000

Hz). They further concluded that a larger study group with a longer follow up period was required to determine whether hearing loss is permanent or temporary.

In 2018, Badarudeen S *et al.*[105] conducted a prospective study to determine the effect of drilling on DPOAE in the normal ear following mastoid surgeries. The authors found significant deterioration in DPOAE amplitudes during the immediate post-operative period. However, no correlation between the duration of drilling and the change in amplitudes was obtained.

MATERIAL AND METHODS

A hospital based prospective study was conducted with 63 patients to identify the drill induced hearing loss in the contralateral ear by transient evoked otoacoustic emission after mastoidectomy and to identify whether hearing loss is more if drilling is done for a longer duration of time.

Study design: A hospital based prospective study.

Study Duration: From November 2018 to April 2020.

Study area: The study was done at our tertiary care centre in the department of Otorhinolaryngology, BLDE (Deemed to be University) Shri B. M. Patil Medical College Hospital and Research Centre, Vijayapura, on attending OPD/IPD.

Study population: All patients attending OPD/IPD at our tertiary care centre in the department of Otorhinolaryngology, for mastoid surgery, irrespective of ear disease and who fulfilled the inclusion criteria.

Sample size: 63 patients

With reference to the study of Patil A *et al.*[106], the anticipated proportion of SNHL is 65%. The minimum sample size is 62 patients with 90% confidence interval and 10% absolute error.

Sample size was calculated using the formula:

$$n = \frac{z^2\, p*q}{d^2}$$

Where Z= Z statistic at α level of significance

d^2 = Absolute error

p = Proportion rate

q = 100-p

Hence, sample size should be a minimum of 62 patients.

Inclusion Criteria:

1) Age between 8-50 years.
2) Opposite ear should be normal.
3) Any type of mastoid surgery, irrespective of ear disease.

Exclusion Criteria:

1) Previously operated ear cases.
2) Patients with pre-existing sensorineural hearing loss.
3) Impaired Otoacoustic emission test on contralateral ear.
4) Patient with upper respiratory tract infection.
5) Patients with immune compromised status like renal failure, diabetes, hypertension.
6) Habits of smoking.
7) Patients on ototoxic drugs.
8) Ear surgeries for CSOM where drilling of the mastoid is not required.

Methodology:

The study was done at our tertiary care centre in the department of Otorhinolaryngology, BLDE (Deemed To Be University) Shri B. M. Patil Medical College Hospital and Research Centre, Vijayapura, after due permission and approval from the Institutional Ethics Committee and Review Board and after taking written informed consent from the patients.

Once the patients were enrolled for the study, a thorough history and physical examination was done as per proforma. An informed consent was taken in writing from patients or patient's attendant.

The following Investigations were carried out

- Blood examination: – Complete blood count
- Urine examination:- Albumin, sugar, microscopy
- HIV rapid & HBsAg Spot
- Pure Tone Audiometry (pre operatively and post operatively).
- Transient Evoked Otoacoustic Emission (pre and post operatively).
- Bilateral X ray mastoid.
- RBS, Blood Urea, Serum Creatinine, Chest X-ray and ECG.

Pre-operative examination of the patient including the complete clinical history with a healthy contralateral ear.

Detailed examination of the patient with emphasis on otoscopic findings and examination under microscope was done to see the status of tympanic membrane on the diseased side, while opposite ear was completely disease free (otoscopic findings, audiometry tests, x-ray mastoid and OAE).

The patient was subjected to investigations such as urine routine and blood routine investigations. A bilateral mastoid x-ray was also taken. A preoperative pure tone audiometry was done for each patient, and then repeated on post-operative day 1 and 7.

A preoperative transient evoked otoacoustic emission test and post-operative TEOAE (Echo lab OAE, ECL, model number-14028) on day 1, 3 and 7 was done for all patients. If any changes in TEOAE readings are detected, a repeat OAE was done until the readings were normal. A repeat TEOAE was done on day 15, 30, 60 and 90 postoperatively, if required.

TEOAE (Echo lab OAE, ECL, model number-14028) was measured at 1000 Hz, 2000 Hz, 3000 Hz and 4000 Hz. OAE testing was done in a sound proof room and by the same examiner.

The same drill machine (Marathon micro motor ECO 450, max speed 45000 rotations/minute) was used for all patients. Fresh burr tips of the same company, was used for each patient. Two types of burr tips was used cutting (D+Z Germany, stainless steel, of size 1 mm to 6 mm) and diamond burr tip (D+Z Germany, stainless steel, of size 1 mm to 6 mm).

Intraoperatively, the type of burr tip used, as well as the individual drilling time for each type of burr were recorded using a stopwatch.

STATISTICAL ANALYSIS

All characteristics were summarized descriptively. For continuous variables, the summary statistics of mean ± standard deviation (SD) were used. For categorical data, the number and percentage were used in the data summaries and diagrammatic presentation. Chi-square (χ^2) test was used for association between two categorical variables.

The formula for the chi-square statistic used in the chi square test is:

$$\chi_c^2 = \sum \frac{(O_i - E_i)^2}{E_i}$$

The subscript "c" is the degree of freedom. "O" is observed value and E is expected value.

C = (number of rows - 1) x (number of columns - 1)

The difference of the means of analysis variables between two independent groups was tested by unpaired t test.

The t statistic to test whether the means are different can be calculated as follows:

$$t = \frac{(\overline{x_1} - \overline{x_2}) - (\mu_1 - \mu_2)}{\sqrt{\frac{s_1^2}{n_1} + \frac{s_2^2}{n_2}}}$$

where $\bar{x}_1$ = mean of sample 1

$\bar{x}_2$ = mean of sample 2

n_1 = number of subjects in sample 1

n_2 = number of subjects in sample 2

s_1^2 = variance of sample 1 = $\frac{\Sigma(x_1 - \bar{x}_1)^2}{n_1}$

s_2^2 = variance of sample 2 = $\frac{\Sigma(x_2 - \bar{x}_2)^2}{n_2}$

ROC analysis for sensitivity- specificity was done to check relative efficiency.

sensitivity or true positive rate (TPR)

eqv. with hit rate, recall

$$TPR = TP/P = TP/(TP + FN)$$

specificity (SPC) or true negative rate

$$SPC = TN/N = TN/(FP + TN)$$

precision or positive predictive value (PPV)

$$PPV = TP/(TP + FP)$$

negative predictive value (NPV)

$$NPV = TN/(TN + FN)$$

If the p-value was < 0.05, then the results were considered to be statistically significant otherwise it was considered as not statistically significant. Data were analysed using SPSS software v.23 (IBM Statistics, Chicago, USA) and Microsoft office 2007.

OBSERVATIONS AND RESULTS

A hospital-based prospective study was conducted with 63 patients to identify the drill induced hearing loss in the contralateral ear by transient evoked otoacoustic emission after mastoidectomy, to identify the relation between the type of burr used and the amount of hearing loss as well as to identify whether hearing loss is more if drilling is done for a longer duration of time.

SEX DISTRIBUTION:

Out of the 63 patients, enrolled in this study, 33 (52.4%) were females and 30 (47.6%) were males. The male to female ratio was 1:1.10.

GENDER	NO. OF PATIENTS	PERCENTAGE
Female	33	52.4
Male	30	47.6
Total	63	100.0

Table 1: Distribution of patients according to gender.

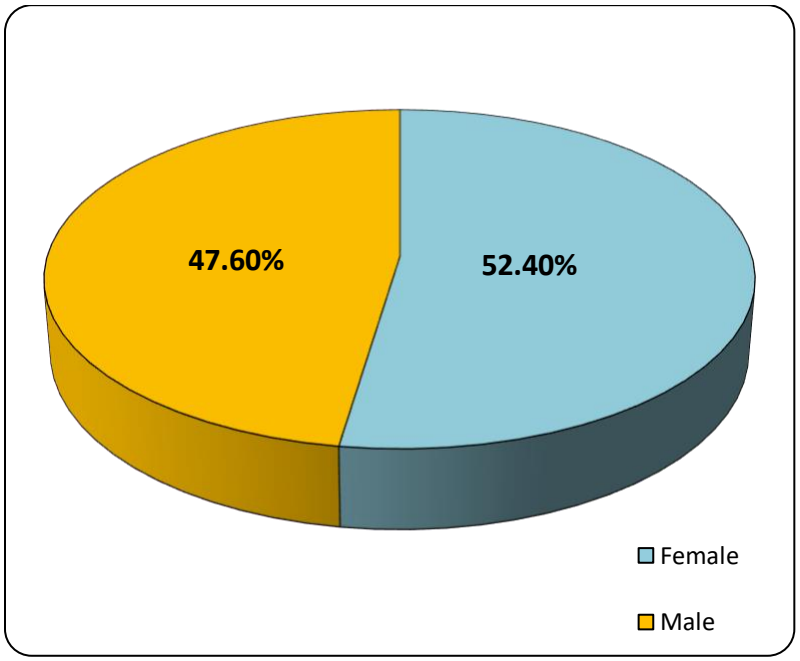

Figure 6: Distribution of patients according to gender.

AGE DISTRIBUTION:

The present study, included patients ranging from 8 to 50 years of age, with a mean age (± SD) of 30.2 years (12.10). The most commonly affected age group was 31 to 40 years, which included 17 (27%) patients. This was closely followed by patients belonging to the age group of 11 to 20 years and 41 to 50 years with 16 (25.4%) and 15 (23.8%) patients in each group respectively.

AGE (YEARS)	NO. OF PATIENTS	PERCENTAGE
08 – 10	2	3.2
11 – 20	16	25.4
21 – 30	13	20.6
31 – 40	17	27.0
41 – 50	15	23.8
Total	63	100.0

Table 2: Distribution of patients according to age (years).

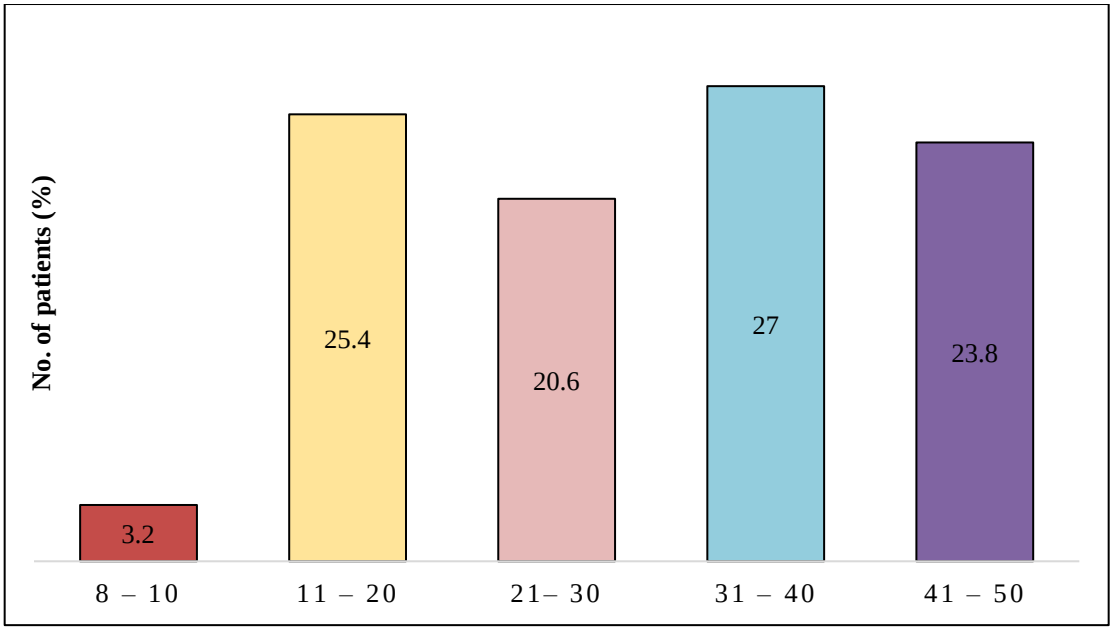

Figure 7: Distribution of patients according to age (years).

DISTRIBUTION BY DIAGNOSIS:

Out of the 63 patients in our study, 26 patients had a squamosal type of chronic otitis media, out of which 16 patients (25.6%) had an active disease, and 10 patients (15.9%) had an inactive type. The remaining 37 patients were suffering from a mucosal type of COM, 23 (36.5%) of which had an inactive disease and 14 patients (22.2%) were diagnosed with an active type.

DIAGNOSIS	NO. OF PATIENTS	PERCENTAGE
Active COM (Squamosal)	16	25.4
Inactive COM (Squamosal)	10	15.9
Active COM (Mucosal)	14	22.2
Inactive COM (Mucosal)	23	36.5
Total	63	100.0

Table 3: Distribution of patients according to diagnosis.

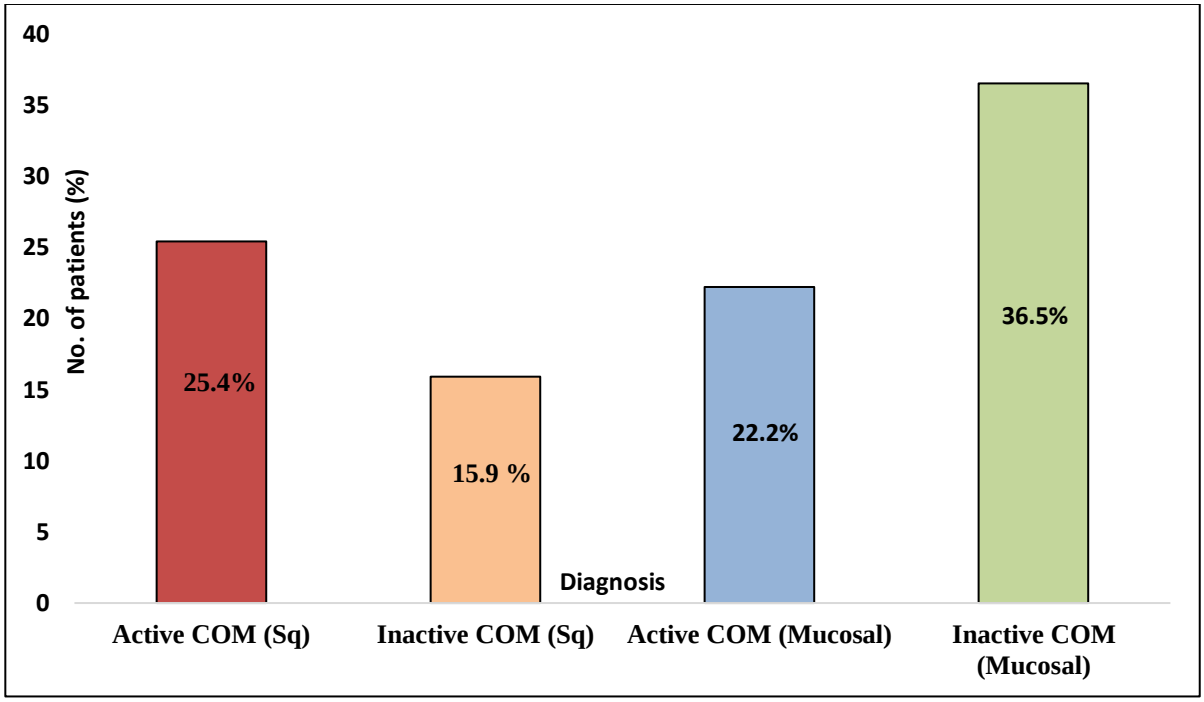

Figure 8: Distribution of patients according to diagnosis.

DISTRIBUTION BY TYPE OF SURGERY:

Mastoidectomy was done for all patients in this study. Cortical mastoidectomy was the treatment of choice for 35 cases (53%), while modified radical mastoidectomy was preferred for the remaining 28 patients (44%).

TYPE OF SURGERY	NO. OF PATIENTS	PERCENTAGE
Cortical mastoidectomy	35	53
Modified radical mastoidectomy	28	44
Total	63	100

Table 4: Distribution of patients according to type of surgery.

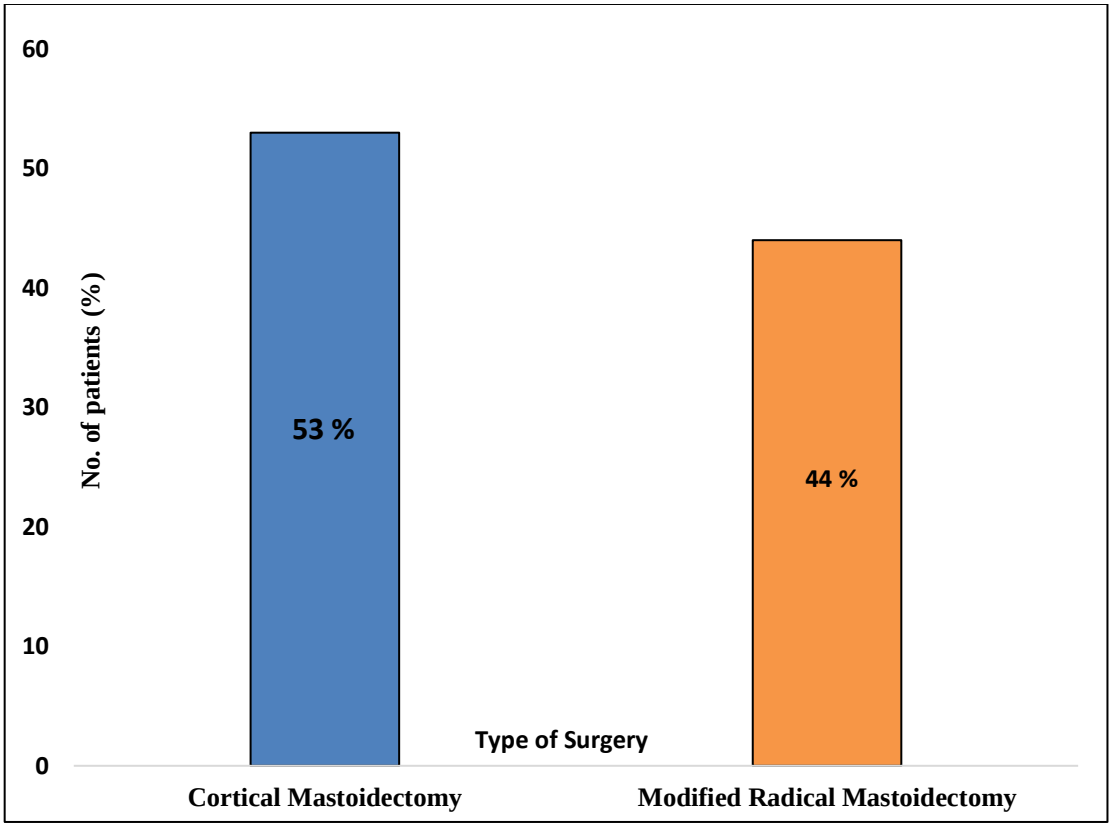

Figure 9: Distribution of patients according to type of surgery.

DISTRIBUTION BY TIME OF DRILLING:

For each case of mastoidectomy, various sizes of cutting as well as diamond burrs was used. The average time of drilling with a cutting burr was 42.1 minutes and that for a diamond burr was 12.5 minutes. The mean total duration of drilling was 54.7 minutes.

TOTAL	MEAN	SD
CUTTING BURR	42.1	10.3
DIAMOND BURR	12.5	4.5
TOTAL TIME	54.7	12.9

Table 5: Distribution of mean time of drilling.

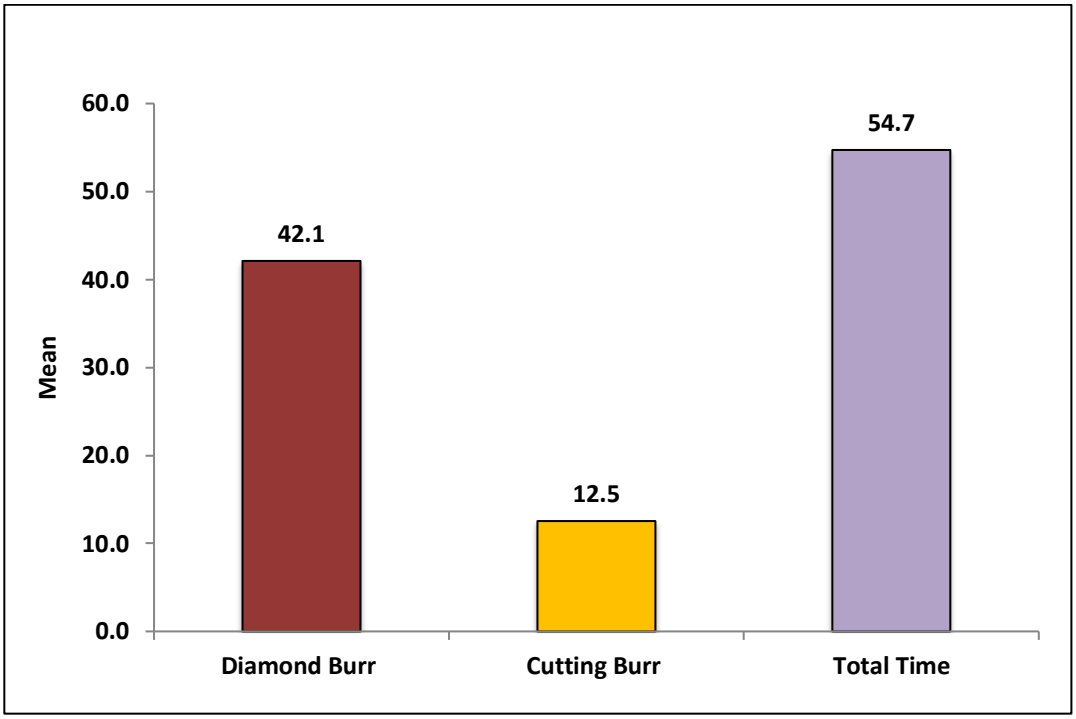

Figure 10: Distribution of mean time of drilling.

DISTRIBUTION BY CUTTING BURR DURATION:

The cutting burr was used for a longer duration of the time in MRM than in cortical mastoidectomy. The average time of drilling with a cutting burr in the case of MRM was 53.4 minutes. While on the other hand, it was 36.7 minutes, for cortical mastoidectomy.

TYPE OF SURGERY	CUTTING BURR		p VALUE
	MEAN	SD	
Cortical mastoidectomy	36.7	7.5	<0.001*
Modified radical mastoidectomy	53.4	9.4	

Note: * significant at 5% level of significance (p<0.05)

Table 6: Mean cutting Burr (min) according to type of surgery.

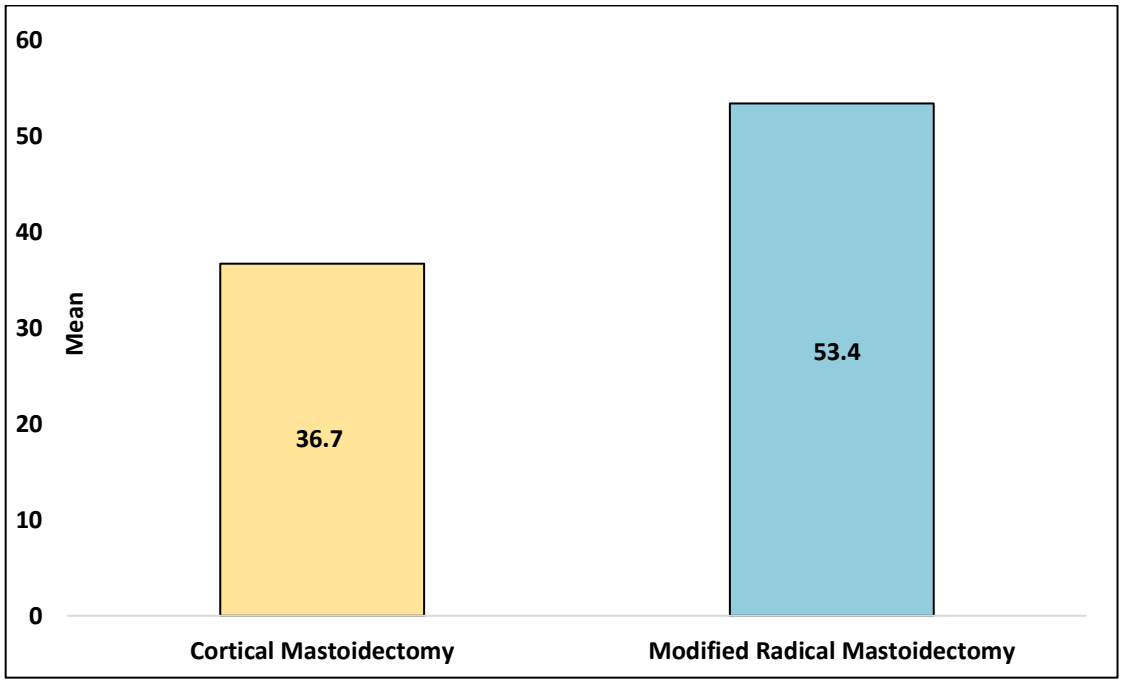

Figure 11: Mean cutting burr (min) according to type of surgery.

DISTRIBUTION BY DIAMOND BURR DURATION:

The diamond burr was used for a longer period of time in MRM than in cortical mastoidectomy. The mean time of drilling with a diamond burr in modified radical mastoidectomy was 15.6 minutes and for cortical mastoidectomy was 11.0 minutes.

TYPE OF SURGERY	DIAMOND BURR		p VALUE
	MEAN	SD	
Cortical mastoidectomy	11.0	3.6	0.004*
Modified radical mastoidectomy	15.6	4.9	

Note: * significant at 5% level of significance ($p<0.05$)

Table 7: Mean diamond burr (min) according to type of surgery.

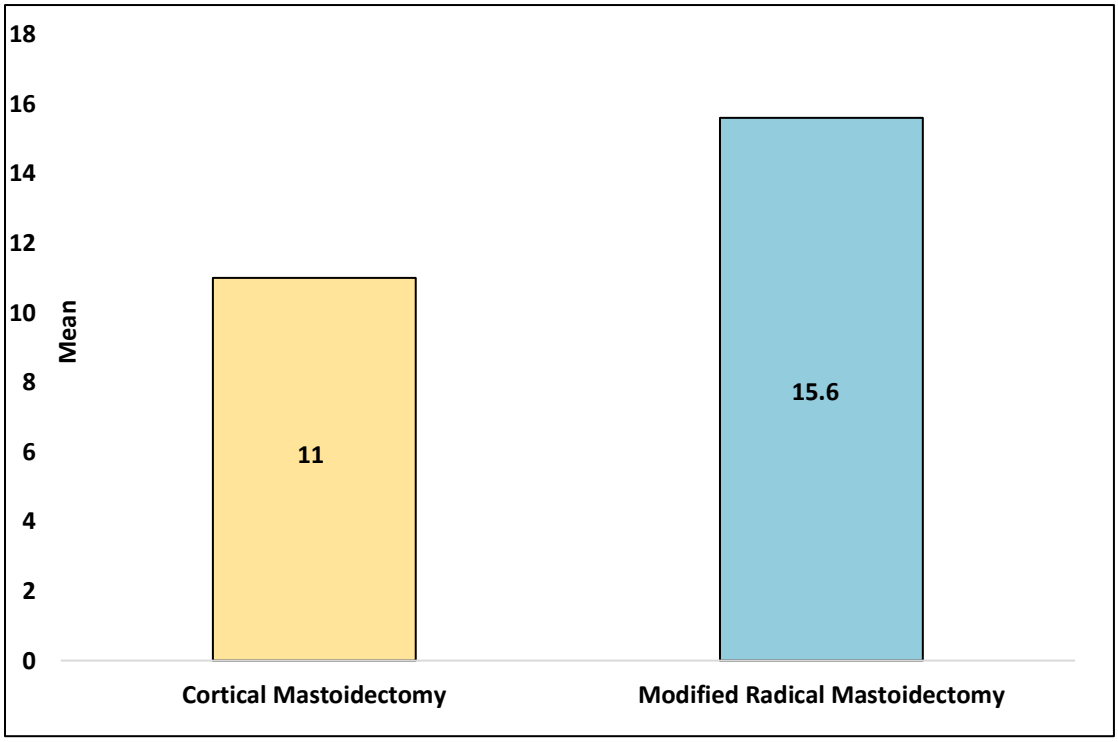

Figure 12 : Mean diamond burr (min) according to type of surgery.

DISTRIBUTION BY TOTAL DRILLING TIME:

The mean drilling time varied from 47 to 66 minutes, depending on the type of surgery. The duration of drilling was found to be more in the case of modified radical mastoidectomy (66.3 minutes) when compared to cortical mastoidectomy (47.8 minutes).

TYPE OF SURGERY	TOTAL TIME		p VALUE
	MEAN	SD	
Cortical mastoidectomy	47.8	9.2	<0.001*
Modified radical mastoidectomy	66.3	11.7	

Note: * significant at 5% level of significance (p<0.05).

Table 8: Mean total time (min) according to type of surgery.

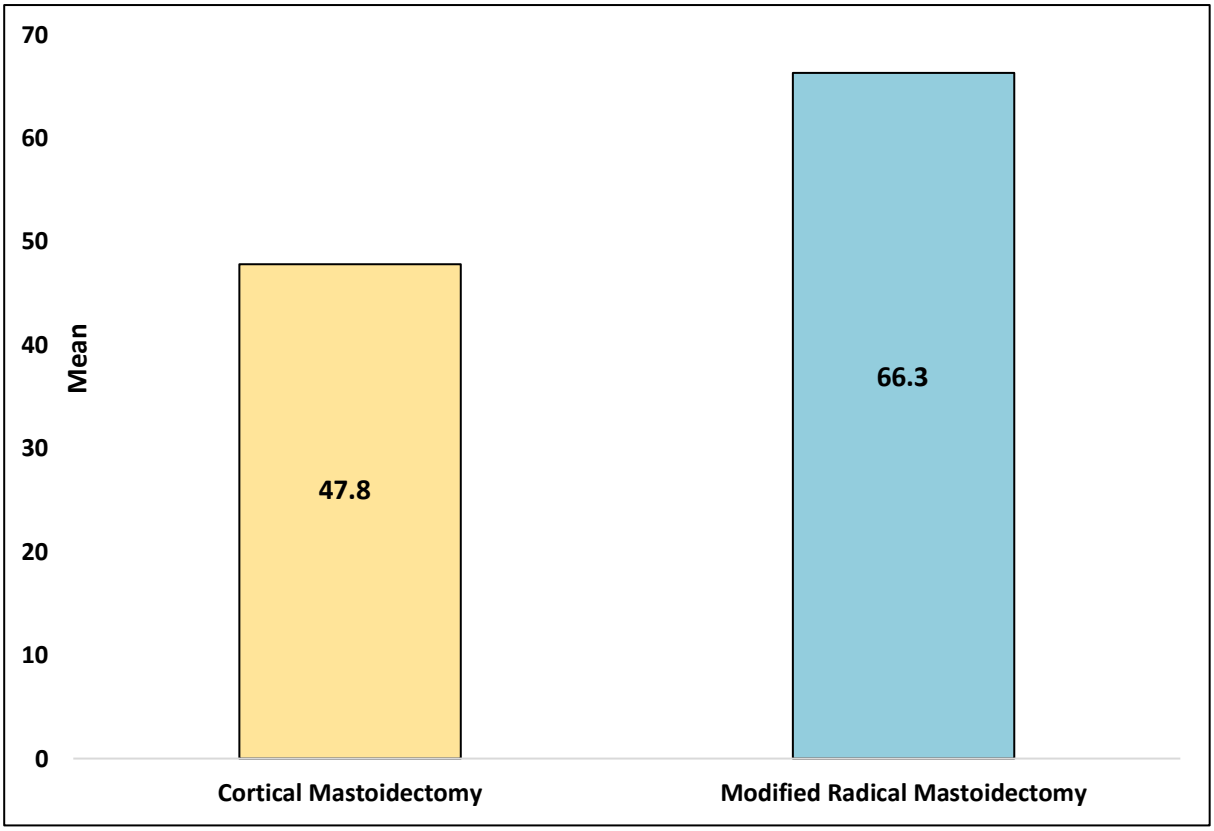

Figure 13: Mean total time (min) according to type of surgery.

DISTRIBUTION BY HEARING LOSS:

Out of the 63 patients enrolled in this study, 26 patients (41.3%) did not develop hearing loss following mastoidectomy. However, it was observed that 37 patients (58.7%) had abnormal TEOAE readings and suffered from temporary drill induced hearing loss in the contralateral ear. On pure tone audiometry, no changes were observed on post-operative day 1 and 7.

HEARING LOSS IN TOTAL	N	%
NORMAL	26	41.3
HEARING LOSS	37	58.7
TOTAL	63	100

Table 9: Distribution of cases with hearing loss.

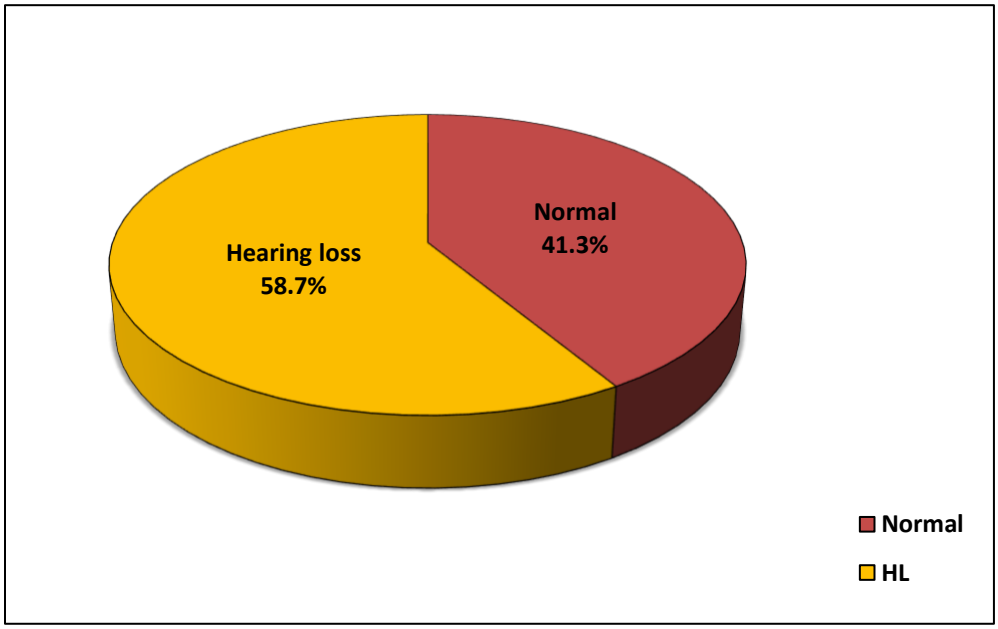

Figure 14: Distribution of cases according to hearing loss in total.

DISTRIBUTION BY RECOVERY:

Out of the 37 cases that developed transient hearing loss, 4 (10.8%) recovered by post-operative day 3, 19 (51.4%) patients were normal by POD 7, 10 (27.0%) cases showed no abnormality by post-operative day 15 and the remaining 4 patients (10.8%) recovered by post-operative day 30.

DAY OF RECOVERY	NUMBER OF PATIENTS (TOTAL 37)
Post-operative day 3	4 (10.8%)
Post-operative day 7	19 (51.4%)
Post-operative day 15	10 (27.0%)
Post-operative day 30	4 (10.8%)

Table 10: Distribution of cases according to day of recovery.

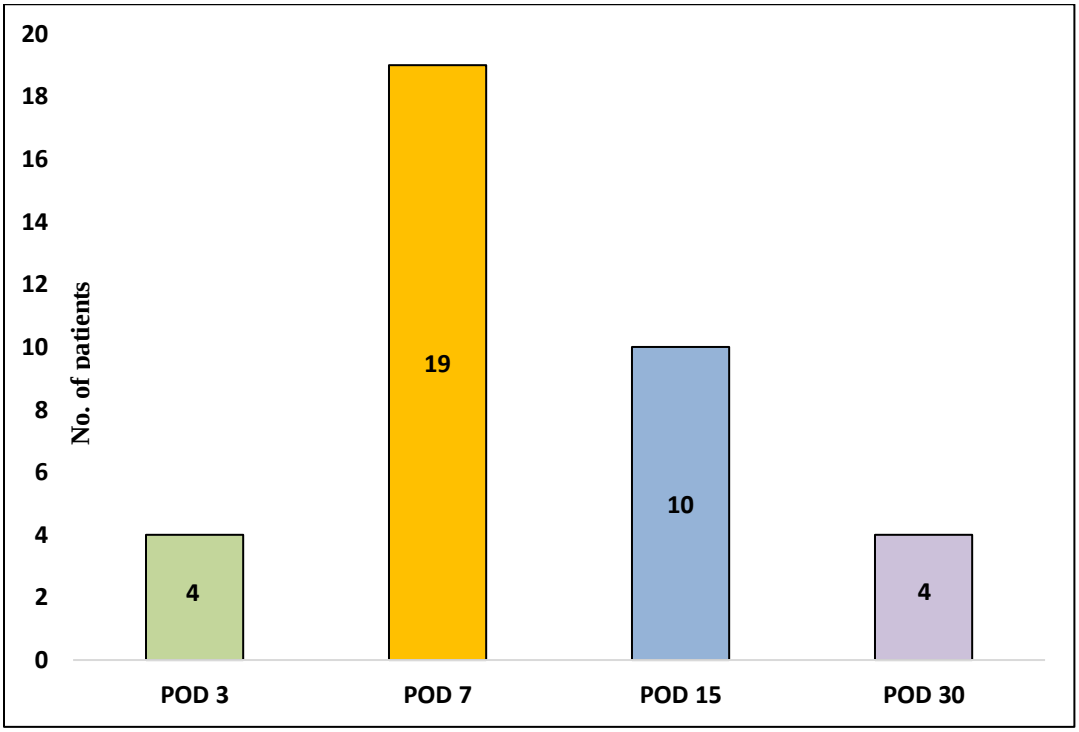

Figure 15: Distribution of cases according to day of recovery.

DISTRIBUTION BY FREQUENCY:

TEOAE, was done for all patients on post-operative day 1, 3 and 7. On post-operative day 1, 26 cases (41.3%) had abnormal TEOAE readings at 3000Hz, while 30 patients (47.6%) had deranged values at 4000Hz. Similar findings were observed on post-operative day 3, where 31 patients (49.2%) were affected at 3000Hz and 33 patients (52.4%) had abnormal readings at 4000Hz. Out of the 37 patients affected, 23 recovered by post-operative day 7. Among the remaining 14 patients, 9 patients presented with hearing loss at both 3000 and 4000Hz, while 5 cases developed abnormal TEOAE readings at 4000Hz only.

Thus, it was observed that, there were significant worsening in values of TEOAE at higher frequencies of 3000 and 4000Hz, in comparison to lower frequencies (1000 to 2000 Hz).

PARAMETERS		**TRANSIENT EVOKED OTOACOUSTIC EMISSION**							
		1000Hz		**2000Hz**		**3000Hz**		**4000Hz**	
		N	**%**	**N**	**%**	**N**	**%**	**N**	**%**
POD1	Passed	62	98.4	61	96.8	37	58.7	33	52.4
	Refer	1	1.6	2	3.2	26	41.3	30	47.6
POD3	Passed	59	93.7	55	87.3	32	50.8	30	47.6
	Refer	4	6.3	8	12.7	31	49.2	33	52.4
POD7	Passed	62	98.4	60	95.2	54	85.7	49	77.8
	Refer	1	1.6	3	4.8	9	14.3	14	22.2
TOTAL		63	100	63	100	63	100	63	100

Table 11: Number of referred cases according to TEOAE.

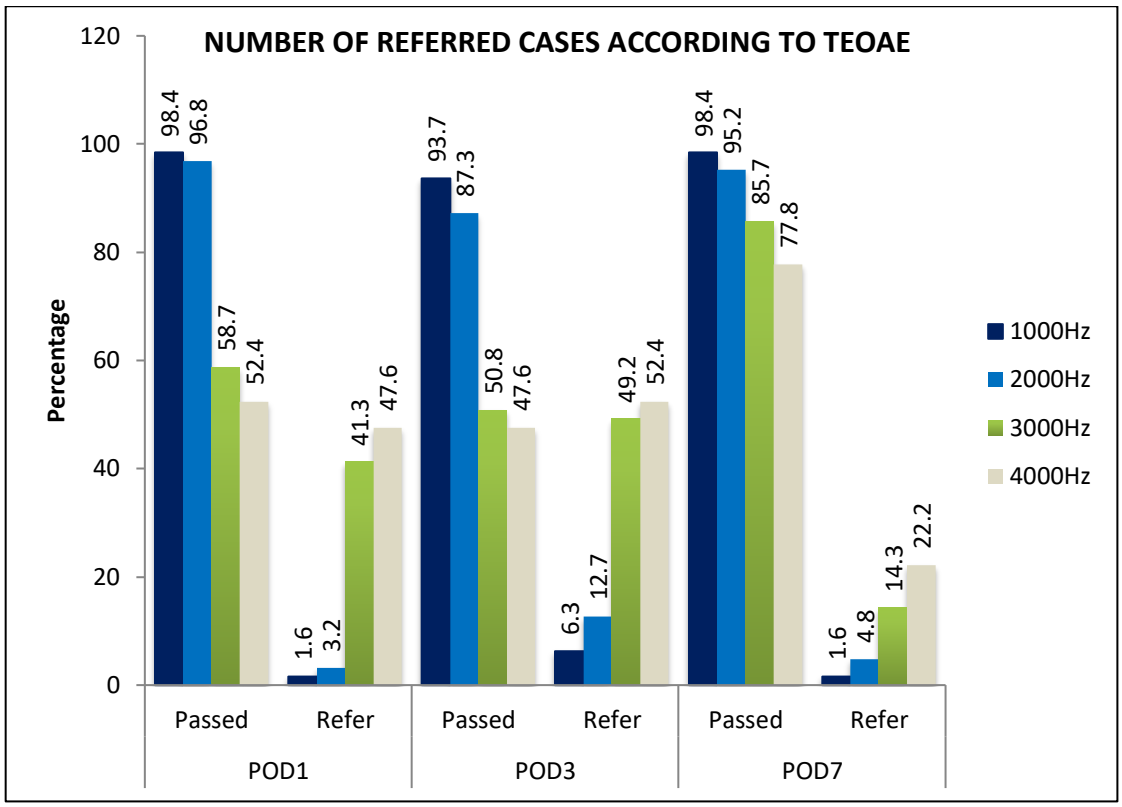

Figure 16: Number of referred cases according to TEAOE.

DISTRIBUTION BY HEARING LOSS ON POST-OPERATIVE DAY 1:

Out of the 63 cases included in this study, 30 patients (47.6%) developed hearing loss on post-operative day 1.

HEARING LOSS ON POD 1	N	%
NORMAL	33	52.4
HEARING LOSS	30	47.6
TOTAL	63	100

Table 12: Distribution of cases according to hearing loss on POD-1

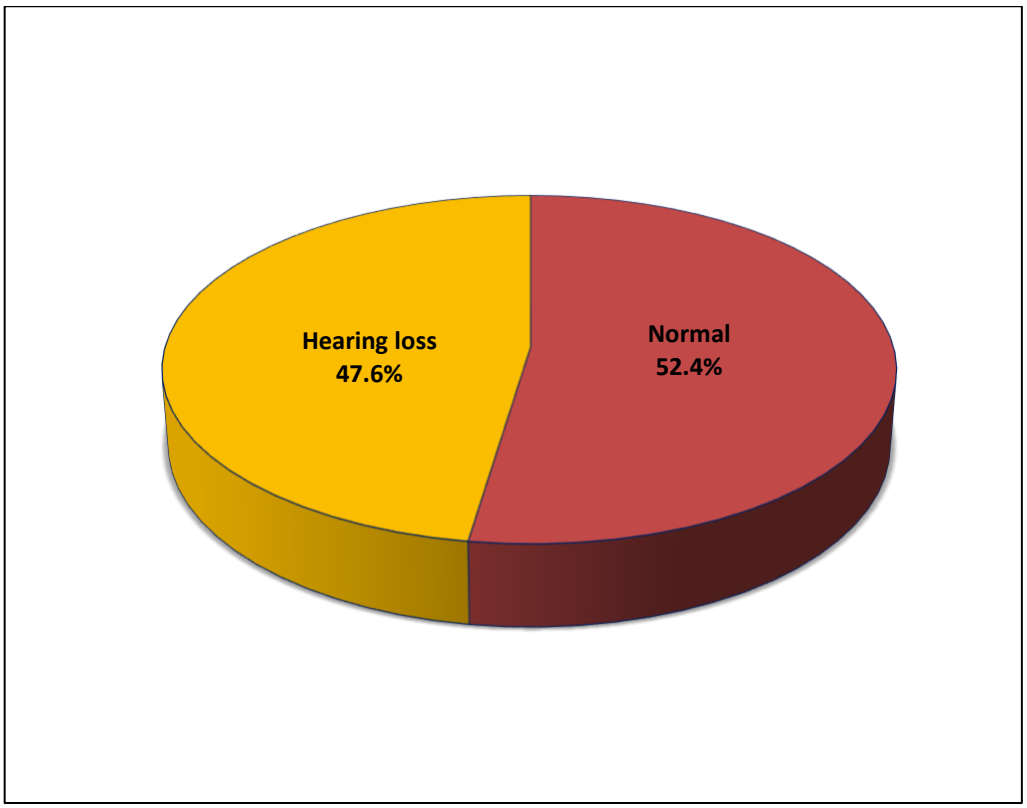

Figure 17: Distribution of cases according to Hearing loss on POD-1.

DISTRIBUTION BY HEARING LOSS ON POST-OPERATIVE DAY 3:

By POD-3, it was observed that 33 out of 63 patients (52.4%) suffered from drill induced hearing loss in the opposite ear. Out of these 33 cases, 7 patients developed hearing loss on POD-3, while the remaining 26 cases had developed hearing impairment on post-operative day 1. Out of the 37 patients that developed hearing loss, 4 cases (6.3%) recovered by POD-3.

HEARING LOSS AT POD 3	N	%
NORMAL	26	41.3
RECOVERED	04	6.3
HEARING LOSS	33	52.4
TOTAL	63	100

Table 13: Distribution of cases according to hearing loss on POD-3

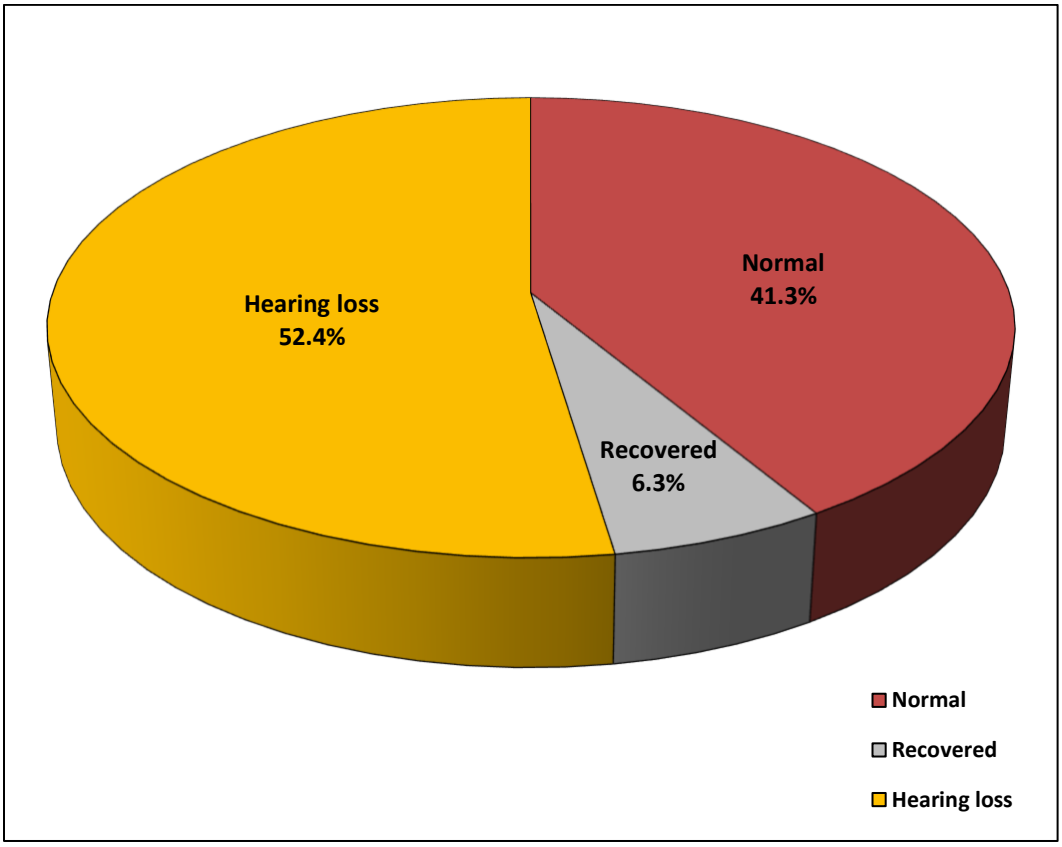

Figure 18: Distribution of cases according to hearing loss on POD-3.

DISTRIBUTION BY HEARING LOSS ON POST-OPERATIVE DAY 7:

On post-operative day 7, 23 out of 37 cases (36.5%) have normal TEOAE values, whereas, 14 patients (22.2%) have abnormal TEOAE readings.

HEARING LOSS ON POD 7	N	%
NORMAL	26	41.3
RECOVERED	23	36.5
HEARING LOSS	14	22.2
TOTAL	63	100

Table 14: Distribution of cases according to hearing loss POD-7

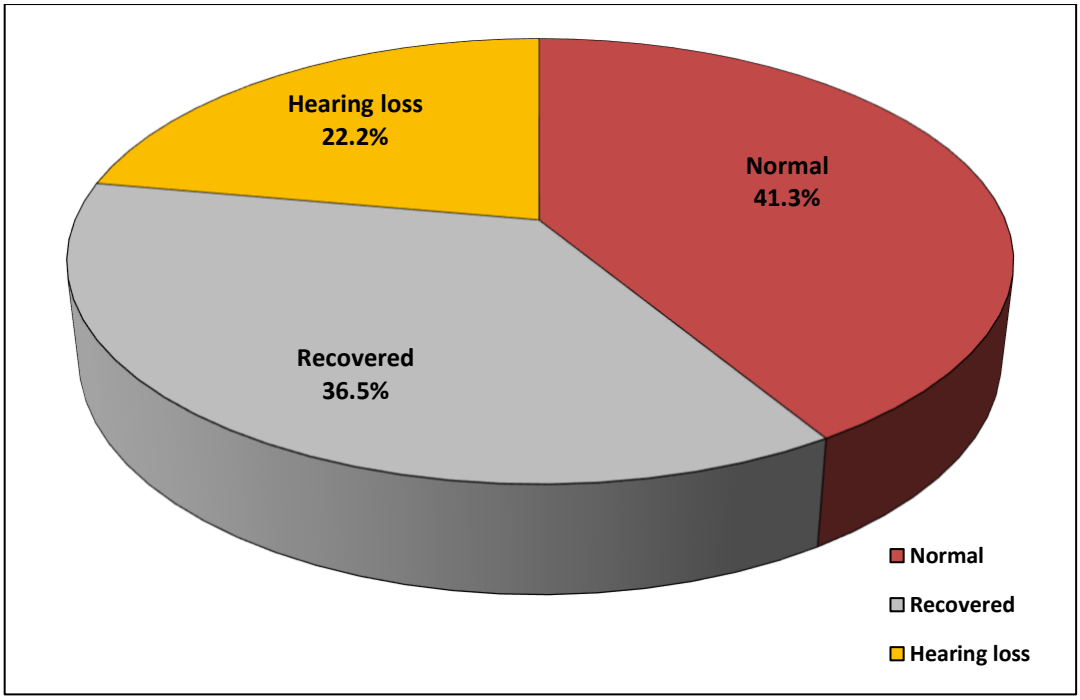

Figure 19: Distribution of cases according to hearing loss on POD-7

TRANSITION OF HEARING LOSS FROM POST-OPERATIVE DAY 1 TO POST-OPERATIVE DAY 3:

Thirty-three cases developed hearing impairment on POD-3. Out of which, only 7 patients (21.2%) developed drill induced hearing loss on post-operative day 3, whereas the remaining 26 cases (86.7%) developed decreased hearing by POD-1. It was also observed that out of a total of 37 patients that developed hearing loss, 4 cases (13.3%) recovered by POD-3.

POD 1	POD 3				p VALUE
	NORMAL		HEARING LOSS		
	N	%	N	%	
NORMAL	26	78.8%	7	21.2%	<0.001*
HEARING LOSS	4	13.3%	26	86.7%	
TOTAL	30	47.6%	33	52.4%	

Note: * significant at 5% level of significance ($p<0.05$)

Table 15: Transition of hearing loss from POD-1 to POD-3

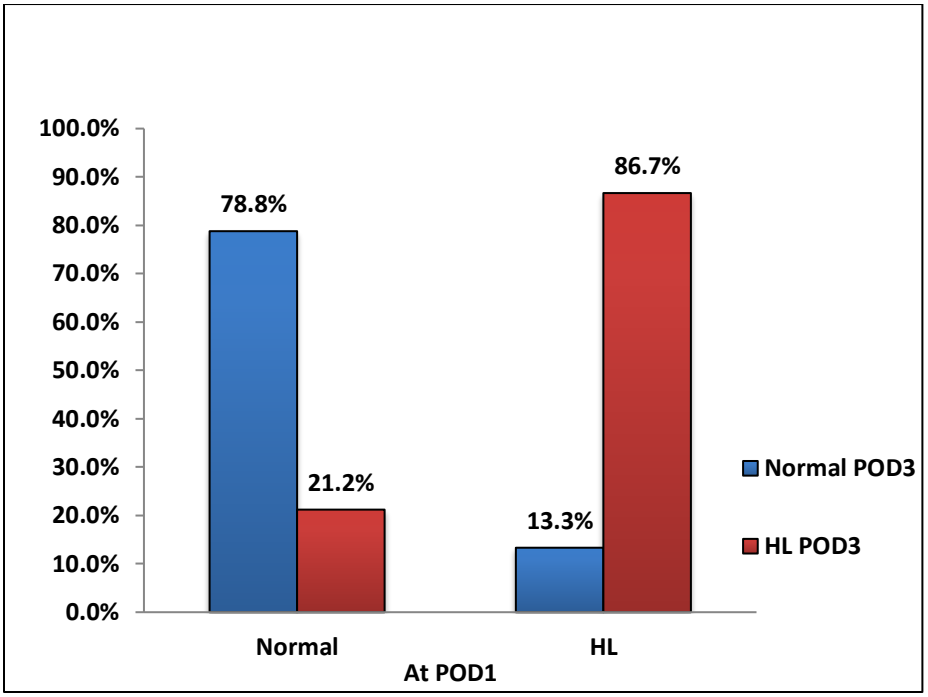

Figure 20: Transition of hearing loss from post-operative day 1 to day 3.

TRANSITION OF HEARING LOSS FROM POST-OPERATIVE DAY 3 TO POST-OPERATIVE DAY 7:

On POD-3, 30 cases (100%) had normal TEOAE values, out of which 4 patients recovered on post-operative day 3 and 26 patients did not develop hearing loss following mastoidectomy. On POD-7, 14 patients (42.4%) suffered from hearing loss, which developed on post-operative day 1 and 3. It was also observed that 19 cases (57.6%) recovered by POD-7.

POD 3	POD 7				p VALUE
	NORMAL		HEARING LOSS		
	N	%	N	%	
NORMAL	30	100.0%	0	0.0%	<0.001*
HEARING LOSS	19	57.6%	14	42.4%	
TOTAL	49	77.8%	14	22.2%	

Note: * significant at 5% level of significance ($p<0.05$)

Table 16: Transition of hearing loss from POD-3 to POD-7.

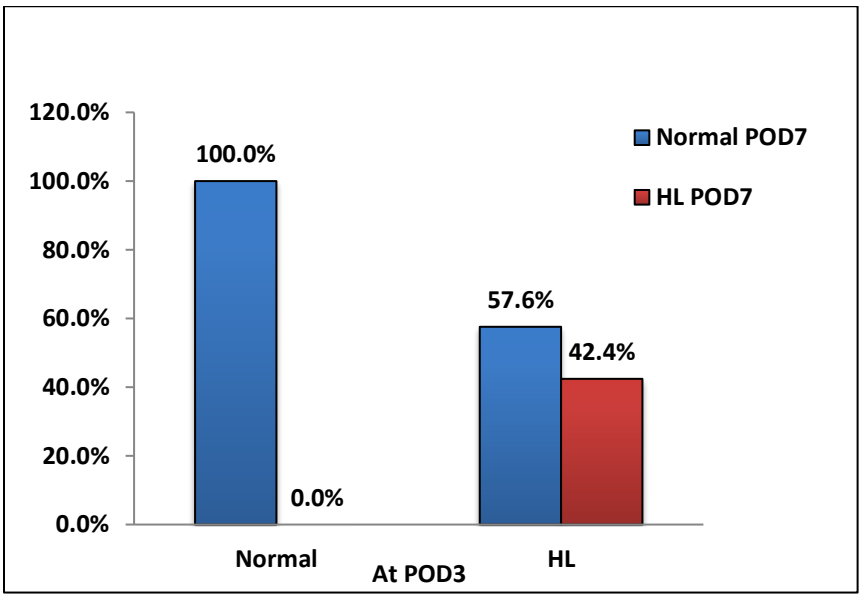

Figure 21: Transition of hearing loss from POD-3 to POD-7.

TRANSITION OF HEARING LOSS FROM POST-OPERATIVE DAY 7 TO POST-OPERATIVE DAY 15:

On POD-7, 49 cases (100%) had normal TEOAE readings, out of which 4 patients recovered on POD-3, 19 cases had normal TEOAE values on POD-7and 26 patients did not develop drill induced hearing loss following mastoidectomy.

POD-15, 4 patients (28.6%) suffered from hearing impairment, which developed on post-operative day 1 and 3. It was also observed that 10 cases (71.4%) recovered by POD-15.

POD 7	POD 15				p VALUE
	NORMAL		HEARING LOSS		
	N	%	N	%	
Normal	49	100.0%	0	0.0%	<0.001*
Hearing Loss	10	71.4%	4	28.6%	
Total	59	93.7%	4	6.3%	

Note: * significant at 5% level of significance (p<0.05)

Table 17: Transition of Hearing loss from POD-7 to POD-15.

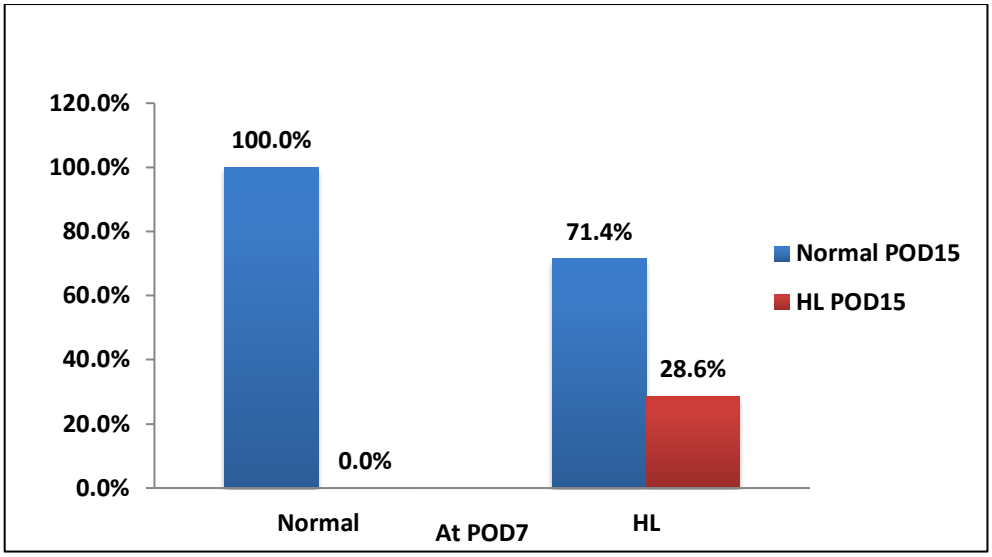

Figure 22: Transition of Hearing loss from POD-7 to POD-15.

TRANSITION OF HEARING LOSS FROM POST-OPERATIVE DAY 15 TO POST-OPERATIVE DAY 30:

On POD-15, 59 cases (100%) had normal TEOAE readings, out of which 4 patients recovered on POD-3, 19 cases had abnormal TEOAE values on POD-7, 10 patients showed normal TEOAE readings on post-operative day 15 and 26 patients did not develop drill induced hearing loss in the opposite ear.

It was observed, that the remaining 4 cases (100%) recovered by POD-30. Thus, no patient suffered from drill induced hearing loss beyond POD-30.

POD 15	POD 30				p VALUE
	NORMAL		HEARING LOSS		
	N	%	N	%	
Normal	59	100.0%	0	0.0%	-
Hearing loss	4	100.0%	0	0.0%	
Total	63	100.0%	0	0.0%	

Table 18: Transition of hearing loss from POD-15 to POD-30.

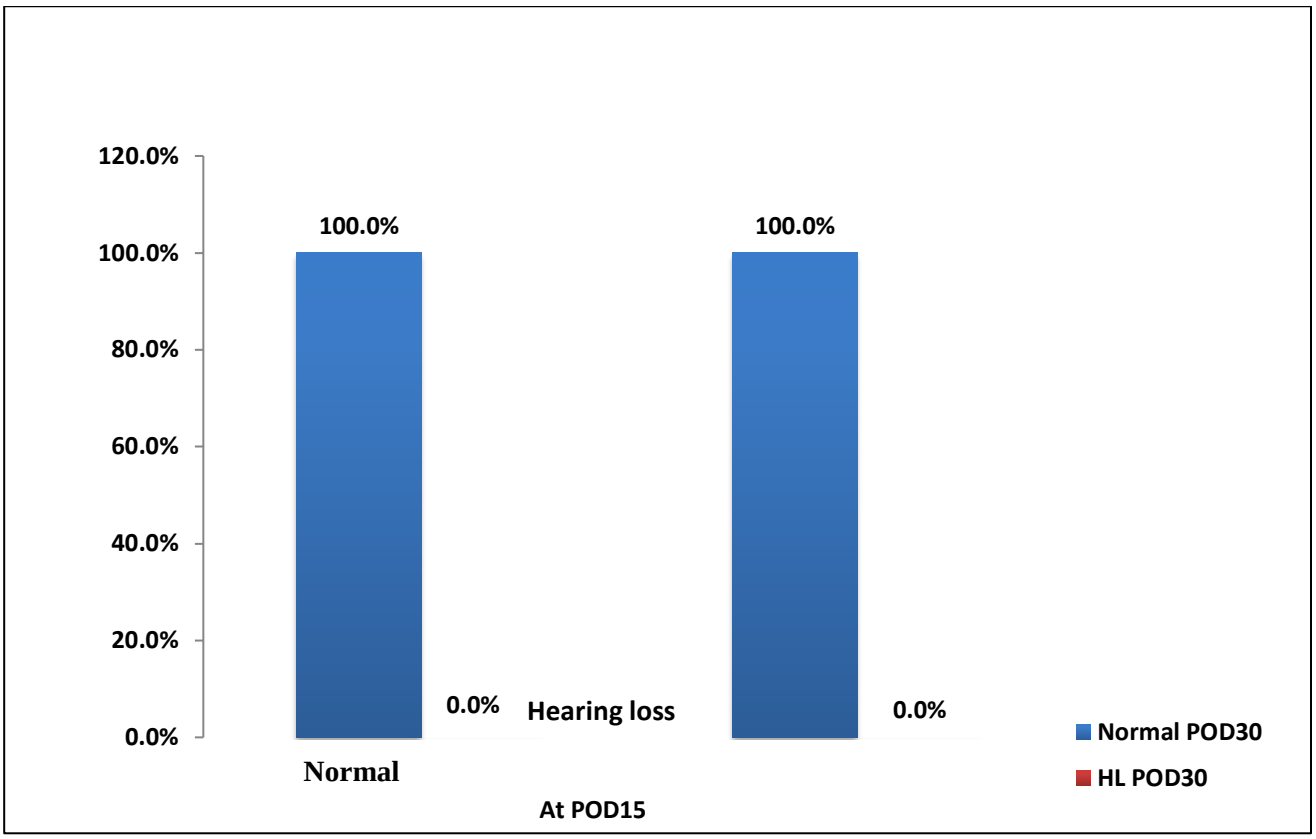

Figure 23: Transition of hearing loss from POD-15 to POD-30.

DRILLING WITH CUTTING BURR:

In this study, it was observed that drilling with a cutting burr for an average of 45.4 minutes resulted in drill induced hearing loss, whereas when used for an average of 37.5 minutes, normal TEOAE readings were achieved.

PARAMETERS	NORMAL		HEARING LOSS		p VALUE
	MEAN	SD	MEAN	SD	
Cutting Burr (min)	37.5	10.3	45.4	9.1	0.002*

Table 19: Mean cutting burr (min) between normal and hearing loss.

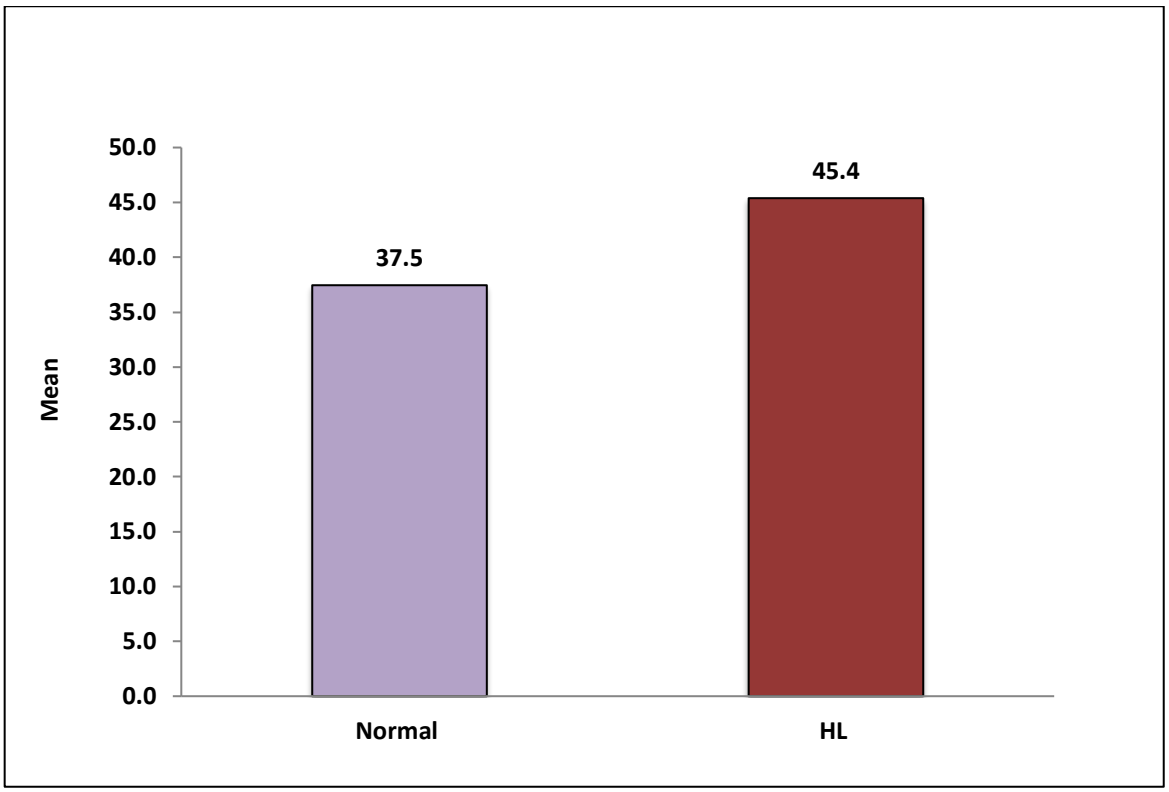

Figure 24: Mean cutting burr (min) between normal and hearing loss

DRILLING WITH DIAMOND BURR:

It was detected that usage of diamond burr for an average of 13.8 minutes resulted in hearing loss in the contralateral ear. However, no hearing impairment was noted when drilling was done for an average of 10.8 minutes.

PARAMETERS	NORMAL		HEARING LOSS		p VALUE
	MEAN	SD	MEAN	SD	
Diamond Burr (min)	10.8	4.0	13.8	4.4	0.009*

Note: * significant at 5% level of significance ($p<0.05$)

Table 20: Mean diamond burr (min) between normal and hearing loss.

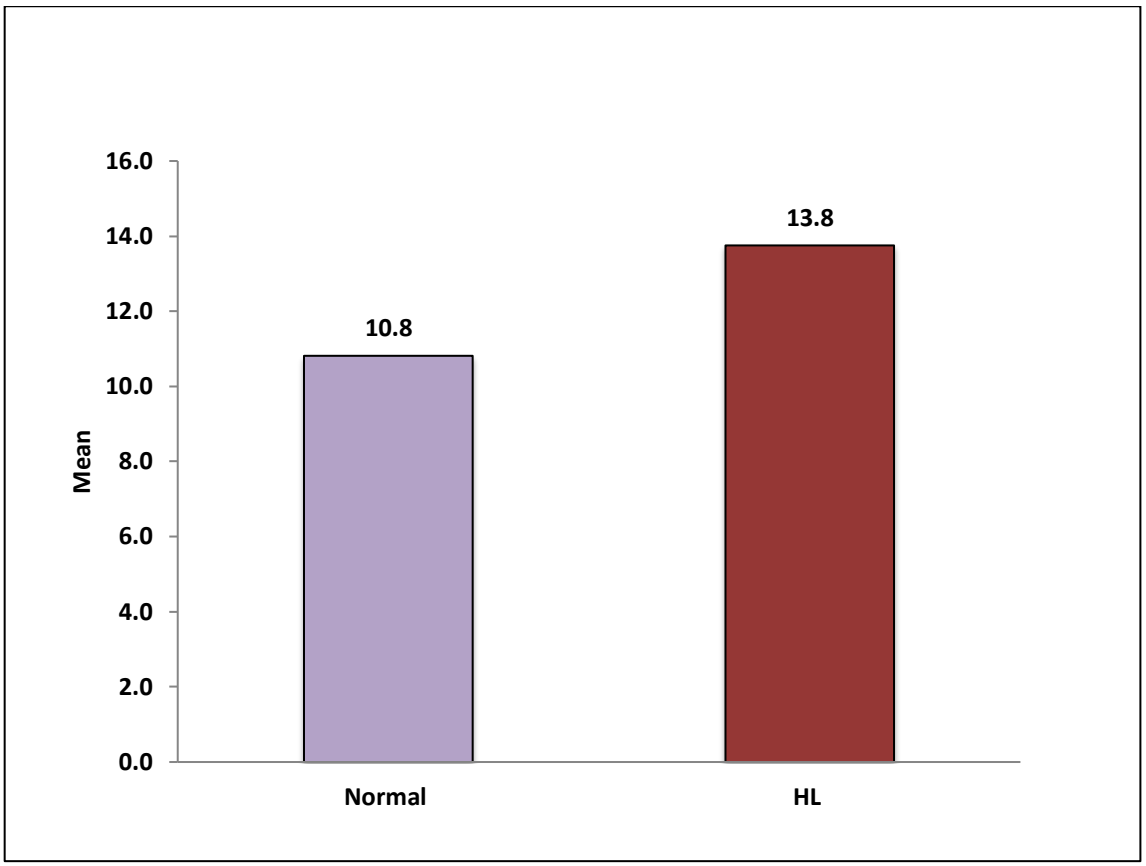

Figure 25: Mean diamond burr (min) between normal and hearing loss.

PREDICTORS OF HEARING LOSS:

The Receiver Operating Characteristic (ROC) curve is defined as a plot of test sensitivity on the y axis versus its specificity on the x coordinate. It is an effective tool for evaluation of both quality as well as performance of a test, by means of sensitivity and specificity.

In this study, it was observed that drilling with a cutting burr for more than 40.2 minutes, resulted in hearing loss, with sensitivity of 70% and specificity of 69%. On the other hand, drilling with a diamond burr for over 12.5 minutes, led to hearing impairment, with sensitivity of 68% and specificity of 65%.

PARAMETERS	AREA UNDER THE CURVE	STD. ERROR	p VALUE	95% CONFIDENCE INTERVAL	
				LOWER	UPPER
Cutting Burr	0.723	0.068	0.003*	0.59	0.86
Diamond Burr	0.705	0.066	0.006*	0.58	0.83

Note: * significant at 5% level of significance ($p<0.05$)

Table 21: ROC analysis of study parameters in predicting hearing loss.

PARAMETERS	CUT-OFF VALUE	SENSITIVITY	SPECIFICITY
Cutting Burr	40.2	70%	69%
Diamond Burr	12.5	68%	65%

Table 22: Sensitivity and specificity of cutting and diamond burr.

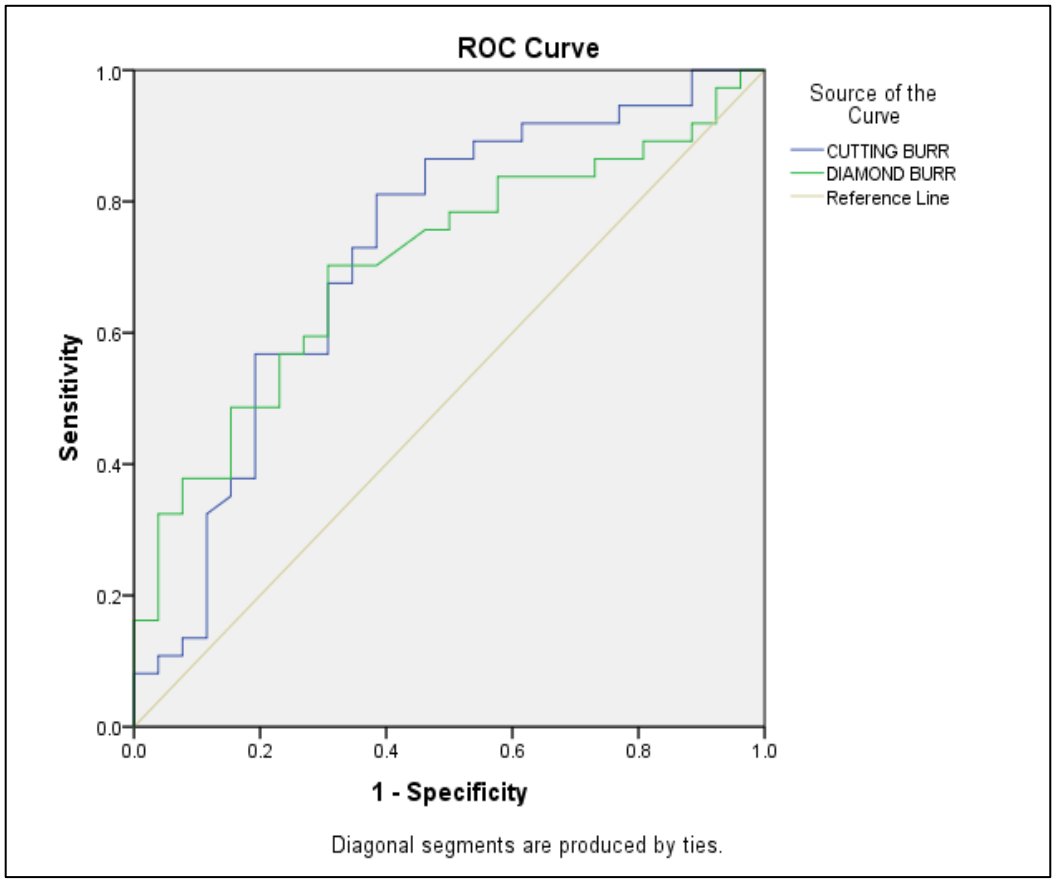

Figure 26: ROC curve of study parameters in predicting hearing loss

IMAGES

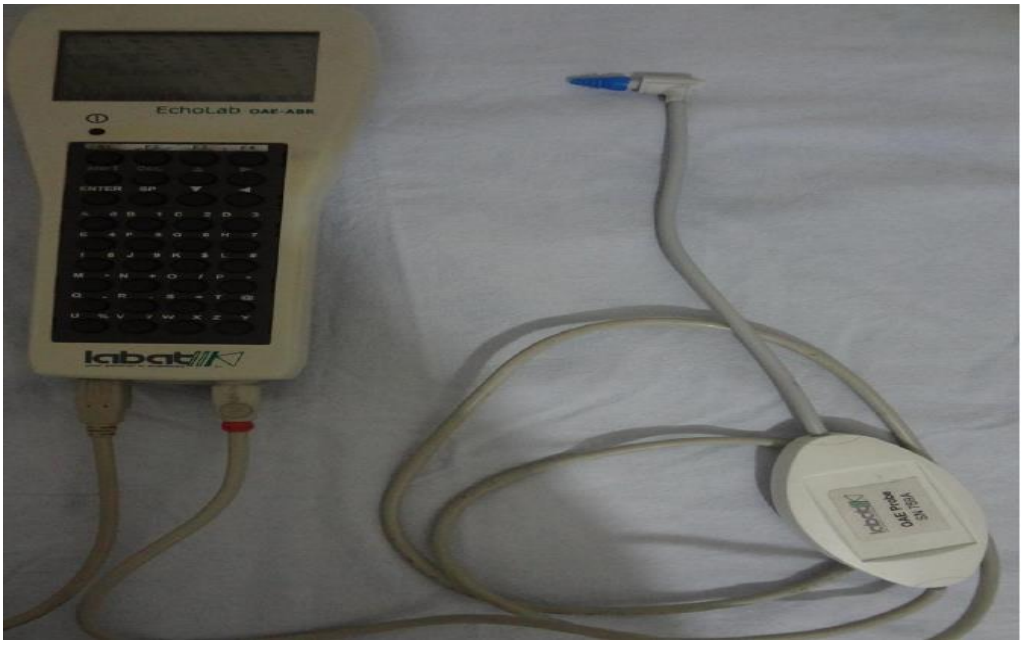

Figure 27: Otoacoustic emission machine

Figure 28: Otoacoustic emission machine connected to laptop

Figure 29: Pre-operative hearing assessment OAE measurement.

Figure 30: Pre-operative hearing assessment by PTA

Figure 31: Micromotor drill with hand-piece, foot paddle and control box.

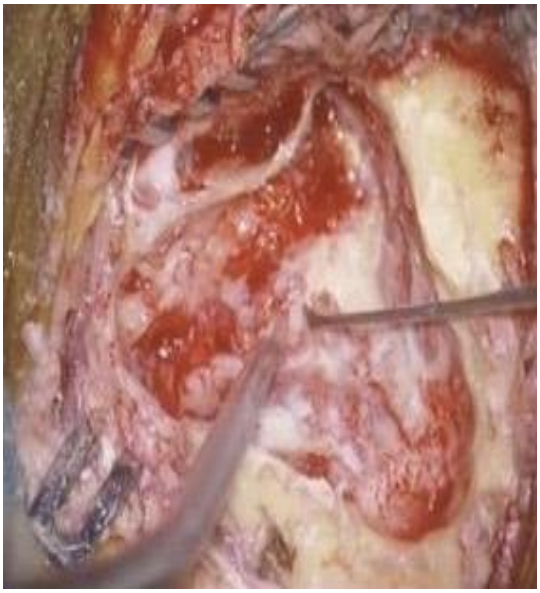

Figure 32: Intra-operative image of modified radical mastoidectomy.

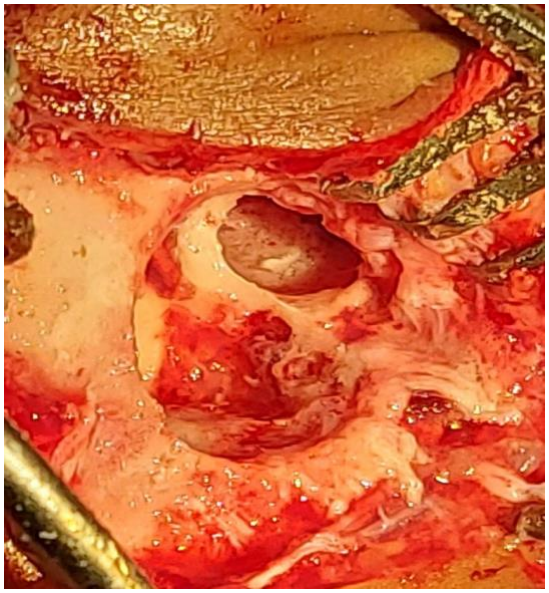

Figure 33: Intra-operative image during mastoid surgery.

DISCUSSION

A hospital-based prospective study was conducted with 63 patients to identify the drill induced hearing loss in the contralateral ear by transient evoked otoacoustic emission after mastoidectomy, to identify the relation between the type of burr used and the amount of hearing loss as well as to identify whether hearing loss is more if drilling is done for a longer duration of time. Ear surgery is associated with a risk of SNHL, which is transient and in most cases reversible.

Drilling during mastoid surgery forms the major part of the noise exposure during surgery and noise produced during drilling is transferred to both cochleae by bone conduction. The sound intensity produced by drilling is estimated to be >100 dB. Since interaural attenuation by bone conduction is minimal (0–5 dB) and drill induced noise can cause hearing loss to the contralateral ear.[13]

In the present study, out of the 63 patients enrolled, 33 (52.4%) were females and 30 (47.6%) were males. The male to female ratio was 1:1.10. The age of the patients ranged from 8 to 50 years, with a mean age (± SD) of 30.2 years (12.10). The most commonly affected age group was 31 to 40 years, which included 17 (27%) patients. This was closely followed by patients belonging to the age group of 11 to 20 years and 41 to 50 years with 16 (25.4%) and 15 (23.8%) patients in each group respectively. This is similar to the studies of Baradaranfar MH *et al.* [101], Abtahi SH *et al.* [103], Jerath V *et al.* [104] and Patil A *et al.* [106].

Baradaranfar MH *et al.* [101] assessed the hearing threshold recovery in the contralateral healthy ear following mastoid surgery. His study included 28 patients with a mean age of 35.57 ± 11.61 years (median 32.5 years, range 16 to 62 years); out of which 46.4% were males. Abtahi SH *et al.*[103] piloted a clinical study that evaluated the effect of drill-generated noise on hearing loss This consisted of 23 patients; with a mean age of 35.52 ± 9.4 years (range: 17–49 years). Among these patients, 52.2%

were male, and 47.8% were female.

Jerath V *et al.* [104] conducted a hospital-based prospective, observational pilot study assessing whether noise produced during drilling causes any hearing loss in the contralateral ear and also assessing the severity of hearing loss. His study included 25 patients, of which 16 were males and 9 were females. Their age ranged from 18 to 51 years with the mean age of 35.12 years. A cross sectional study to assess the effects of mastoid drilling on the hearing of 80 patients was conducted by Patil A *et al.*[106] His study comprised of patients ranging from 15 to 60 years, out of which 60% were females and 40% were males.

Out of the 63 patients in our study, 26 patients had a squamosal type of COM, out of which 16 patients (25.6%) had an active disease, and 10 patients (15.9%) had an inactive type. The remaining 37 patients were suffering from a mucosal type of COM, 23 (36.5%) of which had an inactive disease and 14 patients (22.2%) were diagnosed with an active type. This is comparable to the study of Patil A *et al.* [106] Patil A *et al.* [106] conducted a cross sectional study and established that 40 cases had a central perforation, 25 were diagnosed with a subtotal perforation, 7 had a total perforation, 5 were detected with an attic retraction pocket and the remaining 3 had an intact membrane.

Mastoidectomy was done for all the patients in this study. Cortical mastoidectomy was the treatment of choice for 35 cases (53%), while modified radical mastoidectomy was preferred for the remaining 28 patients (44%). This is concordant to the studies of Jerath V *et al.* [104], Patil A *et al.*[106] and Singh V *et al.*[107].

Jerath V *et al.* [104] concluded that majority of patients underwent cortical mastoidectomy and

tympanoplasty for central perforations of the pars tensa (64%). Patil A *et al.*[106] conducted a cross sectional study and detected that 24 patients underwent tympanoplasty, 40 patients underwent cortical mastoidectomy with tympanoplasty and rest 16 patients underwent MRM with tympanoplasty. A clinical observational prospective study was conducted by Singh V *et al.*[107]. The authors concluded that most of the patients (54.9%) underwent cortical mastoidectomy, while for the remaining 45.1%, modified radical mastoidectomy was the treatment of choice.

In our study, both cutting and diamond burrs of different sizes were used for all cases of mastoidectomy. The average time of drilling with a cutting burr was 42.1 minutes and that for a diamond burr was 12.5 minutes. The mean total duration of drilling was 54.7 minutes. It was established that, both cutting and diamond burrs were used for a longer duration of time in modified radical mastoidectomy than in cortical mastoidectomy.

The average time of drilling using a cutting burr and diamond burr in the case of MRM was 53.4 minutes and 15.6 minutes respectively. On the other hand, for cortical mastoidectomy, the cutting burr was used for an average of 36.7 minutes, and that with a diamond burr was done for 11.0 minutes. The mean drilling time varied from 47 to 66 minutes, depending on the type of surgery. The duration of drilling was found to be more in the case of MRM (66.3 minutes) when compared to cortical mastoidectomy (47.8 minutes). This is consistent with the studies of Migirov L *et al.*[10], Baradaranfar MH *et al.*[101], Jerath V *et al.* [104], Kylén P *et al.*[108] and Goyal A *et a.* [109].

Jerath V *et al.*[104] detected that the average drilling time was 30 min. Baradaranfar MH *et al.*[101] established that the mean time of drilling was 56.48 ± 12.70 min (median 55 min, range 40-90 min). A comparative prospective study on the influence of drilling in the non-operated ear was done by Migirov L *et al.*[10], in which the duration of drilling ranged from 60 to 90 minutes (mean 72

minutes). Kylén P *et al.*[108] piloted a study on peroperative temporary threshold shift in ear surgery. They concluded that large cutting burrs produced more noise than fine small cutting burrs and the noise level was reduced when equivalent-sized diamond burrs were used. Goyal A *et al.*[09] reported the mean drilling time as 53.93 min, while the average drilling duration using the cutting burr and diamond burr were 28.63 min (SD ± 13.687) and 24.63 min (SD ± 15.897) respectively.

Out of the 63 patients included in this study, 26 patients (41.3%) did not develop hearing loss following mastoidectomy. However, it was noted that 37 patients (58.7%) had abnormal TEOAE readings and suffered from temporary drill induced SNHL in the contralateral ear. On pure tone audiometry, no changes were observed on post-operative day 1 and 7. This is in concordance to the studies of Migirov L *et al.*[10], Baradaranfar MH *et al.* [101] Patil A *et al.* [106], Singh V *et al.* [107], Goyal A *et al.* [109], Konopka W *et al.* [110], Sliwinska-Kowalska N *et al.* [111] and Paksoy M *et al.* [112]

Migirov L *et al.* [10] stated that there was a significant decrease in DPOAE readings in the contralateral ear following mastoidectomy in majority of the patients. Baradaranfar MH *et al.* [101] detected that there was a significant difference in hearing level the heating levels 24 hours after surgery at frequencies of 3000-8000 Hz, on both PTA and DPOAE. Patil A *et al.*[106] evaluated 80 patients and assessed for drill induced hearing loss in the opposite ear. They came to the conclusion that there was no significant change on pure tone audiometry, in the contralateral ear.

Singh V *et al.*[107] observed that among the 62 patients that underwent mastoidectomy, 30 cases (49%), had abnormal DPOAE values, in the immediate post-operative period. However, all patients recovered by post-operative day 7. Goyal A *et al*.[109] detected that there was a substantial decrease in TEOAE and DPOAE reading in the opposite ear, following mastoid surgeries. However, no statistically significant changes were noted on pure tone audiometry.

Konopka W *et al.*[110] found 2 dB decrease in TEOAE readings, but the only significant changes in audiometric thresholds were at 10 and 12 kHz. Sliwinska-Kowalska N *et al.*[111] established that although there was no change in the PTA of noise-exposed patients a constant and gradual decrease in TEOAE was noted. Paksoy M *et al.*[112] concluded that 67% of patients (41/61) showed hearing impairment following mastoidectomy. Goyal A *et al*.[109] detected that there was a substantial decrease in TEOAE and DPOAE reading in the opposite ear, following mastoid surgeries. However, no statistically significant changes were noted on pure tone audiometry.

In the current study, out of the 37 cases that developed transient hearing loss, 4 (10.8%) recovered by post-operative day 3, 19 (51.4%) patients were normal by post-operative day 7, 10 (27.0%) cases showed no abnormality by post-operative day 15 and the remaining 4 patients (10.8%) recovered by post-operative day 30. These finding was consistent with the studies of Migirov L *et al*.[10], Baradaranfar MH *et al.* [101], Abtahi SH *et al.* [103] and Singh V *et al*.[107].

In the study conducted by Migirov L *et al*.[10], it was established that deterioration of DPOAE amplitudes were noted immediately after surgery, with progressive improvement within 72 to 96 hours. However complete recovery was observed by 4 weeks. Baradaranfar MH *et al.*[101] observed that majority of the cases had a recovery time of 3 to 4 days. Patients developed a transient and reversible hearing loss, at frequencies above 2000Hz. Abtahi SH *et al.*[103] concluded that most of the patients developed hearing loss by post-operative day 1, however had normal TEOAE and DPOAE readings by post-operative day 7. However, Singh V *et al.*[107] observed that out of the 30 patients that had an absent DPOAE in the immediate post-operative period, 10 patients recovered within 1 hour of surgery, 12 patients (60%) regained outer hair cell function by post-operative day 1, while the remaining 8 were normal by post-operative day 7.

In our study, TEOAE was done for all patients on post-operative day 1,3 and 7. On post-operative day 1, 26 cases (41.3%) had abnormal TEOAE readings at 3000Hz, while 30 patients (47.6%) had deranged values at 4000Hz. Similar findings were observed on post-operative day 3, where 31 patients (49.2%) were affected at 3000Hz and 33 patients (52.4%) had abnormal readings at 4000Hz. 23 out of the 37 patients affected, recovered by post-operative day 7. Among the remaining 14 patients, 9 patients presented with hearing loss at both 3000 and 4000Hz, while 5 cases developed abnormal TEOAE readings at 4000Hz only.

Hence, it was established that there was significant worsening in TEOAE readings at higher frequencies of 3000 and 4000Hz, than in lower frequencies of 1000 and 2000 Hz. Similar observations were noted in the studies of Baradaranfar MH *et al.*[101]. Baradaranfar MH *et al.*[101] concluded that drill induced hearing loss was more at frequencies higher than 2000Hz. On the contrary, Abtahi SH *et al.*[103] observed a significant difference in DPOAE and TEOAE values, at low frequencies of 500 to 2000Hz as well as at higher frequencies of 4000 and 8000 Hz.

Out of the 63 cases included in this study, 30 patients (47.6%) developed hearing loss on post-operative day 1, while the remaining 7 cases developed impaired hearing by POD-3. This is in concordant to the studies of Abtahi SH *et al.*[103] and Jerath V *et al.*[104] On post-operative day 1 Abtahi SH *et al.*[103] observed that majority of the patients developed significant changes in PTA at lower frequencies and abnormal OAE values at higher as well as lower frequencies. Jerath V *et al.*[104] found a significant drop in TEOAE values on post-operative day 1.

In our study, it was detected that by POD-3, 33 out of 63 patients (52.4%) suffered from drill induced hearing loss in the opposite ear. Thirty cases had normal TEOAE values, out of which 4 patients (6.3%) recovered on post-operative day 3 and 26 patients did not develop hearing loss

following mastoidectomy.

On post-operative day 7, 23 out of 37 cases (36.5%) had normal TEOAE values, whereas, 14 patients (22.2%) have abnormal TEOAE readings. It was also observed that 19 cases (57.6%) recovered by post-operative day 7. This is comparable to the study done by Özdamar K *et al.*[102], Abtahi SH et al.[103], Jerath V *et al.*[104] and Singh V *et al.*[107]

Özdamar K *et al.*[102] assessed the inner ear damage in the contralateral ear, following mastoidectomy. The authors stated that a statistically significant decrease in DPOAE values at 4000Hz was detected on POD-4. Abtahi SH *et al.*[103] stated that all patients with drill induced hearing loss in the normal ear, recovered by POD-7. Jerath V *et al.*[104] and Singh V *et al.*[107] also made similar observations. However, Baradaranfar MH *et al* [101] stated that all patients with hearing loss following mastoidectomy recovered within 72 to 96 hours.

On post-operative day 15, 4 patients (28.6%) suffered from hearing impairment, which developed on post-operative day 1 and 3. It was also established that on post-operative day 15, 59 cases had normal TEOAE readings, out of which 4 patients recovered on post-operative day 3, 19 cases had abnormal TEOAE values on post-operative day 7, 10 patients (71.4%) showed normal TEOAE readings on post-operative day 15 and 26 patients did not develop drill induced hearing loss in the opposite ear. It was observed, that the remaining 4 cases (100%) recovered by post-operative day 30. Thus, no patient suffered from drill induced hearing loss beyond post-operative day 30. This is similar to the study done by Migirov L *et al.* [10] The authors established that a decrease in DPOAE amplitude was noted immediately after surgery, with progressive improvement within 72 to 96 hours. However complete recovery was observed by 4 weeks.

In this study, it was observed that drilling with different sizes of cutting burr for a mean duration of 45.4 minutes resulted in drill induced hearing loss, whereas when used for an average of 37.5 minutes, normal TEOAE readings were achieved. It was also detected that drilling with a diamond burr for an average of 13.8 minutes resulted in hearing loss in the contralateral ear. However, no hearing impairment was noted when drilling was done for a mean time duration of 10.8 minutes.

Burr types and sizes can affect the acoustic trauma levels. Kylén P *et al.*[113] study tested three different types of cutting burrs. The 6 mm cutting burrs developed a noise level of 88 to 108 dB. The use of a 4 mm burr resulted in a reduction of 1 to 6 dB, while drilling with a 2 mm burr caused a decrease of 5 to 16 dB. The mean noise level produced by the diamond burrs is 5 to 11 dB lower than that produced by the cutting burrs.

It was suggested that all drills emitted noise exceeding 85 dB. The pneumatic drill can reach a noise level of 114 dB, while the shielded self-propelled drill almost complied with a noise level of 85 dB. By isolating the operator from the self-propelled drill, many believe that complications arising from both vibration as well as noise exposure can be solved [114]. Da Cruz MJ *et al.* [115] established that reversible drill-related outer hair cell dysfunction was seen in 16.7% of the operated ear cases.

The Organ of Corti can be completely examined with a scanning electron microscope. It was found that a drill with the lowest rotations per minute but the highest torque can produce the highest noise intensities, thus causing trauma to the ears. Extremely high-speed drills have a less detrimental effect on the Organ of Corti than the low speed drill. Therefore, it is advised to refrain from using low speed drills in prolonged operations. [99]

In the present study, we observed that drilling with a cutting burr for more than 40.2 minutes, resulted in hearing loss, with sensitivity of 70% and specificity of 69%. On the other hand, drilling with a diamond burr for over 12.5 minutes, led to hearing impairment, with sensitivity of 68% and specificity of 65%.

CONCLUSION

Following mastoidectomy, drill induced hearing loss, especially at frequencies higher than 2000 Hz may occur. This transient and reversible type of SNHL recovers within 7 days in majority of the patients. However, in a handful of cases, the hearing loss may be prolonged (1 month post operatively).

The clinical impact of the drill induced hearing loss varies. Some patients with small amount of hearing changes are completely asymptomatic, while others complain of tangible effects.

Also it has been observed that drilling with a cutting burr for more than 40.2 minutes, can lead to hearing loss. This has a sensitivity of 70% and a specificity of 69%. On the other hand, drilling with a diamond burr for over 12.5 minutes, can lead to hearing impairment, with a sensitivity of 68% and a specificity of 65%.

The drill is not only a source of noise but is also a strong vibration generator, and a strong oscillation is transmitted into the cochlea. Thus surgeons should pay more attention to the vibrations and lessen the intensity of drill induced hearing loss by an appropriate selection of burrs and drills, thus minimizing the vibrations of the temporal bone.

SUMMARY

A hospital based prospective study was conducted with 63 patients to identify the drill induced hearing loss in the contralateral ear, following mastoidectomy by transient evoked otoacoustic emission and to identify whether hearing loss is more if drilling is done for a longer duration of time. The following observations were noted:

1) Out of the 63 patients, enrolled in this study, 33 (52.4%) were females and 30 (47.6%) were males. The male to female ratio was 1:1.10.

2) The present study, included patients ranging from 8 to 50 years of age, with a mean age (± SD) of 30.2 years (12.10). The most commonly affected age group was 31 to 40 years, which included 17 (27%) patients. This was closely followed by patients belonging to the age group of 11 to 20 years and 41 to 50 years with 16 (25.4%) and 15 (23.8%) patients in each group respectively.

3) Out of the 63 patients in our study, 26 patients had a squamosal type of COM, out of which 16 patients (25.6%) had an active disease, and 10 patients (15.9%) had an inactive type. The remaining 37 patients were suffering from a mucosal type of COM, 23 (36.5%) of which had an inactive disease and 14 patients (22.2%) were diagnosed with an active type.

4) Mastoidectomy was done for all the patients in this study. Cortical mastoidectomy was the treatment of choice for 35 cases (53%), while modified radical mastoidectomy was preferred for the remaining 28 patients (44%).

5) Cutting and diamond burrs of various sizes were used in every case of mastoidectomy. The

average time of drilling with a cutting burr was 42.1 minutes and that for a diamond burr was 12.5 minutes. The mean total duration of drilling was 54.7 minutes.

6) Both cutting and diamond burrs were used for a longer duration of time in MRM than in cortical mastoidectomy. The average time of drilling using a cutting burr and diamond burr in the case of MRM was 53.4 minutes and 15.6 minutes respectively. On the other hand, for cortical mastoidectomy, the cutting was used for 36.7 minutes, and that with a diamond burr was 11.0 minutes

7) The mean drilling time varied from 47 to 66 minutes, depending on the type of surgery. The duration of drilling was found to be more in the case of modified radical mastoidectomy (66.3 minutes) when compared to cortical mastoidectomy (47.8 minutes).

8) Out of the 63 patients enrolled in this study, 26 patients (41.3%) did not develop hearing loss following mastoidectomy. However, it was observed that 37 patients (58.7%) had abnormal TEOAE readings and suffered from temporary drill induced hearing loss in the contralateral ear. On pure tone audiometry, no changes were observed on post-operative day 1 and 7.

9) Out of the 37 cases that developed transient hearing loss, 4 (10.8%) recovered by post-operative day 3, 19 (51.4%) patients were normal by post-operative day 7, 10 (27.0%) cases showed no abnormality by post-operative day 15 and the remaining 4 patients (10.8%) recovered by post-operative day 30.

10) It was observed that there was significant worsening in TEOAE readings at higher frequencies of 3000 and 4000Hz, than in lower frequencies (1000 to 2000 Hz).

11) Out of the 63 cases included in this study, 30 patients (47.6%) developed hearing loss on post-operative day 1, while 7 patients had abnormal TEOAE readings on POD-3.

12) By POD-3, it was observed that 33 out of 63 patients (52.4%) suffered from drill induced hearing loss in the opposite ear. Out of the 37 patients that developed hearing loss, 4 cases (6.3%) recovered by post-operative day 3.

13) On post-operative day 7, 23 out of 37 cases (36.5%) have normal TEOAE values, whereas, 14 patients (22.2%) have abnormal TEOAE readings. It was also observed that 19 cases (57.6%) recovered by post-operative day 7.

14) On post-operative day 7, 49 cases had normal TEOAE readings, out of which 4 patients recovered on post-operative day 3, 19 cases had normal TEOAE values on post-operative day 7 and 26 patients did not develop drill induced hearing loss following mastoidectomy.

15) It was observed that 10 cases (71.4%) recovered by post-operative day 15.

16) On post-operative day 15, 59 cases had normal TEOAE readings, out of which 4 patients recovered on post-operative day 3, 19 cases had abnormal TEOAE values on post-operative day 7, 10 patients showed normal TEOAE readings on post-operative day 15 and 26 patients did not develop drill induced hearing loss in the opposite ear.

17) It was observed, that the remaining 4 cases (100%) recovered by post-operative day 30. Thus, no patient suffered from drill induced hearing loss beyond post-operative day 30.

18) It was established that drilling with a cutting burr for an average of 45.4 minutes resulted in drill induced hearing loss, whereas when used for an average of 37.5 minutes, normal TEOAE readings were achieved.

19) It was detected that usage of diamond burr for an average of 13.8 minutes resulted in hearing loss in the contralateral ear. However, no hearing impairment was noted when drilling was done for an average of 10.8 minutes.

20) We concluded that drilling with a cutting burr for more than 40.2 minutes, resulted in hearing loss, with sensitivity of 70% and specificity of 69%.

21) Drilling with a diamond burr for over 12.5 minutes, led to hearing impairment, with sensitivity of 68% and specificity of 65%.

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ANNEXURE I

ETHICAL CLEARANCE CERTIFICATE

B.L.D.E (Deemed to be University)
SHRI.B.M.PATIL MEDICAL COLLEGE HOSPITAL & RESEARCH CENTRE
VIJAYAPUR – 586103

IEC/NO:286/2018
17-11-2018

INSTITUTIONAL ETHICAL COMMITTEE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 13-11-2018 at 03-15 PM scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected and revised version synopsis of the Thesis has accorded Ethical Clearance.

Title : A Study of drill induced hearing loss in the contralateral ear following mastoid surgery.

Name of P.G. Student : Dr Ashima Kumar
Department of Otorhinolaryngology,

Name of Guide/Co-investigator: Dr R N Karadi, Professor & HOD of Otorhinolaryngology,

DR RAGHAVENDRA KULKARNI
CHAIRMAN
Institutional Ethical Committee
BLDE(Deemed to be University) Shri B.M. Patil
Medical College, Vijayapur-586103.

Following documents were placed before E.C. for Scrutinization:

1) Copy of Synopsis/Research Project
2) Copy of informed consent form.
3) Any other relevant documents.

ANNEXURE – II

PROFORMA

1) NAME: 2) CASE NO:

3) AGE: 4) IPD NO:

5) SEX: 6) DOA:

7) RELIGION: 8) DOS:

9) OCCUPATION: 10) DOD:

11) RESIDENCE:

12) CHIEF COMPLAINTS:

13) HISTORY OF PRESENTING ILLNESS:

14) PAST HISTORY:

- Diabetes mellitus
- Hypertension
- History of any previous surgery.
- Usage of Ototoxic drugs

15) FAMILY HISTORY:

16) GENERAL PHYSICAL EXAMINATION:

Pallor: Present/Absent

Icterus: Present/Absent

Clubbing: Present/Absent

Generalized Lymphadenopathy: Present/Absent

Build: Poor/Medium /Well

Nourishment: Poor / Medium / Well

17) VITALS

PR:

BP:

RR:

Temp:

18) OTHER SYSTEMIC EXAMINATION:

- Respiratory System
- Cardiovascular System
- Central Nervous System
- Per Abdomen examination

19) LOCAL EXAMINATION

- EAR Right Left
  - Pinna
  - Pre auricular area
  - Post auricular area
  - Infra auricular area
  - External auditory canal

Right Left

- Tympanic membrane

Pars Tensa

Pars flaccida

- Mastoid Tenderness
- Fistula sign
- Tragal Tenderness
- Facial nerve function
- Nystagmus
- Tuning Fork test

  Rinnes

  Webers

  ABC

- NOSE

- ORAL CAVITY

- OROPHARYNX

20) INVESTIGATION:

BLOOD ROUTINE: URINE ROUTINE:

X RAY: BILATERAL MASTOIDS

PURE TONE AUDIOMETRY:

Preoperative: Air bone gap:

dB loss:

Postoperative day 1: Air bone gap:

dB loss:

Postoperative day 7: Air bone gap:

dB loss:

TRANSIENT EVOKED OTOACOUSTIC EMISSION

Pre-operative: 1000Hz:

2000Hz:

3000Hz:

4000Hz:

Post-operative:

Day 1: 1000Hz:

2000Hz:

3000Hz:

4000Hz:

Day 3: 1000Hz:

2000Hz:

3000Hz:

4000Hz:

Day 7: 1000Hz:

2000Hz:

3000Hz:

4000Hz:

If required TEOAE readings on

Day 15: 1000Hz:

2000Hz:

3000Hz:

4000Hz:

Day 30: 1000Hz:

2000Hz:

3000Hz:

4000Hz:

Day 60: 1000Hz:

2000Hz:

3000Hz:

4000Hz:

Day 90: 1000Hz:

2000Hz:

3000Hz:

4000Hz:

21) FINAL DIAGNOSIS:

22) SURGERY: Intra operative findings

Type of surgery:

Duration of drilling: Total time:

Diamond burr:

Cutting burr:

Type of drill machine used

23) INFERENCE:

24) COMMENTS:

ANNEXURE –III

INFORMED CONSENT FORM

BLDE (deemed to be university)

SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL AND

RESEARCH CENTRE, VIJAYAPURA- 586103.

TITLE OF THE PROJECT

"A STUDY OF DRILL INDUCED HEARING LOSS IN THE CONTRALATERAL EAR FOLLOWING MASTOID SURGERY"

PG STUDENT - Dr. ASHIMA KUMAR
DEPARTMENT OF
OTORHINOLARYNGOLOGY

PG GUIDE - Dr. R.N. KARADI
PROFESSOR
DEPARTMENT OF
OTORHINOLARYNGOLOGY
BLDE (Deemed To Be University)
SHRI B. M. PATIL MEDICAL COLLEGE
HOSPITAL AND RESEARCH CENTRE,
VIJAYAPURA – 586103

All aspects of this consent form are explained to the patient in the language understood by him/her.

1) PURPOSE OF RESEARCH:

I have been informed about this study. I have also been given a free choice of participation in this study.

2) PROCEDURE:

I am aware that in addition to routine care received I will be asked series of questions by the investigator. I have been asked to undergo the necessary investigations and treatment, which will help the investigator in this study.

3) RISK AND DISCOMFORTS:

I understand that I may experience some pain and discomfort during the examination or during my treatment. This is mainly the result of my condition and the procedure of this study is not expected to exaggerate these feelings that are associated with the usual course of treatment.

4) BENEFITS:

I understand that my participation in this study will help to improve survival of the patient and will bring about a better outcome.

5) CONFIDENTIALITY:

I understand that the medical information produced by this study will become a part of Hospital records and will be subject to the confidentiality and privacy regulation. Information of a sensitive personal nature will not be a part of the medical records, but will be stored in the investigator's research file and identified only by a code number. The code-key connecting name to numbers will be kept in a separate location. If the data are used for publication in the medical literature

or for teaching purpose, no name will be used and other identifiers such as photographs and audio or videotapes will be used only with my special written permission. I understand that I may see the photographs and videotapes and hear the audiotapes before giving this permission.

6) REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at any time. Dr. ASHIMA KUMAR is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of the study, which might influence my continued participation.

If during the study, or later, I wish to discuss my participation in or concerns regarding this study with a person not directly involved, I am aware that the social worker of the hospital is available to talk with me. A copy of this consent form will be given to me to keep for careful reading.

7) REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and that I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice to my present or future care at this hospital. I also understand that DR. ASHIMA KUMAR may terminate my participation in the study after she has explained the reasons for doing so and has helped arrange for my continued care by my own physician or physical therapist, if this is appropriate.

8) INJURY STATEMENT:

I understand that in the unlikely event of injury to me resulting directly from my participation in this study, if such injury were reported promptly, the appropriate treatment would be available to me, but no further compensation would be provided. I understand that by my agreement to participate in this study I am not waiving any of my legal rights.

I have explained to _____the purpose of the research, the procedures required and the possible risks and benefits to the best of my ability in patient's own language.

_____ _____

Dr. ASHIMA KUMAR Date

(Investigator)

STUDY SUBJECT CONSENT STATEMENT

I confirm that DR. ASHIMA KUMAR has explained to me the purpose of research, the study procedures that I will undergo, and the possible risks and discomforts as well as benefits that I may experience in my own language. I have read and I understand this consent form. Therefore, I agree to give consent to participate as a subject in this research project.

_____ _____

Participant / Guardian Date

_____ _____

Witness to signature Date

ANNEXURE IV

KEY TO MASTERCHART

S. No	Serial Number
(R)	Right
(L)	Left
F	Female
M	Male
AS	Active squamosal
IS	Inactive Squamosal
AM	Active Mucosal
IM	Inactive mucosal
COM	Chronic otitis media
MRM	Modified radical mastoidectomy
CM	Cortical mastoidectomy
TP	Tympanoplasty
T -I TP	Type I Tympanoplasty
T-II TP	Type II Tympanoplasty
T-III TP	Type III Tympanoplasty
T-IV TP	Type IV Tympanoplasty

S.NO	NAME	AGE (Y)	SEX	DIAGNOSIS	TYPE OF SURGERY	CUTTING BURR (MIN)	DIAMOND BURR (MIN)	TOTAL TIME (MIN)	PTA POD1/ POD7	TRANSIENT EVOKED OTOACOUSTIC EMISSION																			
										POD1				POD3				POD7				POD15				POD30			
										1000 Hz	2000 Hz	3000 Hz	4000 Hz	1000 Hz	2000 Hz	3000 Hz	4000 Hz	1000 Hz	2000 Hz	3000 Hz	4000 Hz	1000 Hz	2000 Hz	3000 Hz	4000 Hz	1000 Hz	2000 Hz	3000 Hz	4000 Hz
1	BORAMMA PUJERI	35	F	(R) AS COM	(R) MRM	34	6.7	40.7	S	P	P	P	P	P	P	P	P	P	P	P	P	NR				NR			
2	HANAMATH METI	42	M	(R) IM COM	(R) CM + T I TP	24.97	1.67	26.64	S	P	P	P	P	P	P	R	R	R	R	R	R	P	P	R	P	P	P	P	P
3	AISHWARYA MANGOND	14	F	(L) AM COM	(L) MRM + T III TP	55.07	3.17	58.24	S	P	P	P	P	P	P	P	P	P	P	P	P	NR				NR			
4	SUGALAWWA UKALI	11	F	(L) IS COM	(L) MRM + T III T-PLASTY	20.03	13.38	33.41	S	P	P	P	P	P	P	P	P	P	P	P	P	NR				NR			
5	SUDHA NATIKA	28	F	(R) AS COM	(R) MRM + TIII T-PLASTY	38.03	1.43	39.46	S	P	P	P	P	P	P	P	P	P	P	P	P	NR				NR			
6	SUDEEP DODAMANI	37	M	(R) AS COM	(R) MRM	57.13	12.06	69.19	S	P	P	R	R	P	P	R	R	P	P	P	P	NR				NR			
7	SHIVAPPA BIRADAR	30	M	(R) IS COM	(R) CM + T IV TP	39.2	5.63	44.83	S	P	P	R	R	P	P	R	R	P	P	P	P	NR				NR			
8	JAYASHREE KAMBLE	15	F	(R) IS COM	(R) MRM + T IV TP	57.2	17.72	74.92	S	P	P	R	R	P	P	R	R	P	P	P	P	NR				NR			
9	VIJAYLAXMI KUMBHAR	25	F	(L) IM COM	(L) CM + TYPE I TP	32.2	12.57	44.77	S	P	P	R	R	P	P	R	R	P	P	P	P	NR				NR			
10	RAMU HUGAR	9	M	(R) IM COM	(R) CM + T I TP	25.2	8.78	33.98	S	P	P	P	P	P	P	P	P	P	P	P	P	NR				NR			
11	BASALINGAMMA M	18	F	(R) IS COM	(R) MRM + T IV TP	32.28	17.85	50.13	S	P	P	P	P	P	P	P	P	P	P	P	P	NR				NR			
12	KALAVATI MALI	31	F	(R) AS COM	(R) MRM + T III TP	51.2	17.68	68.88	S	P	P	R	R	P	P	P	P	P	P	P	P	NR				NR			
13	PRIYANKA PAWAR	17	F	(L) AM COM	(L) CM + TYPE I TP	28.7	11.07	39.77	S	P	P	P	P	P	P	P	P	P	P	P	P	NR				NR			
14	TUKARAM KABMLE	45	M	(L) AM COM	(L) CM + TYPE III TP	24.86	7.18	32.04	S	P	P	R	R	P	P	R	R	P	P	P	P	NR				NR			
15	BASSAYA KAMBLE	36	M	(R) IS COM	(R) MRM + T III TP	48.52	15.3	63.82	S	P	P	P	P	P	P	R	R	P	P	R	R	P	P	P	P	NR			
16	PRADEEP CHAVAN	9	M	(R) IS COM	(R) MRM + T III TP	57.17	14.02	70.19	S	P	P	P	P	P	P	P	P	P	P	P	P	NR				NR			
17	KRISHNA	20	M	(L) IS COM	(L) MRM + T IV TP	58.2	17.53	75.73	S	P	P	R	R	P	P	R	R	P	P	P	P	NR				NR			
18	SUJATHA PRABHU	38	F	(R) IS COM	(R) MRM + T IV TP	51.3	20.52	71.82	S	P	P	P	P	P	P	R	R	P	P	R	R	P	P	P	P	NR			
19	PRAJWAL KULKARNI	11	M	(L) IM COM	(L) CM + T I TP	21.28	8.7	29.98	S	P	P	P	P	P	P	P	P	P	P	P	P	NR				NR			
20	RENUKA KUMBHAR	28	F	(R) AM COM	(L) CM + T III TP	38.17	12.12	50.29	S	P	P	R	R	P	P	R	R	P	P	P	P	NR				NR			
21	MANJUNATH	45	M	(R) AS COM	(R) MRM + TIII TP	54.3	13.83	68.13	S	P	P	R	R	P	R	R	R	P	P	P	R	P	P	P	P	NR			
22	ALLABI K	50	F	(L) IM COM	(L) CM + T III TP	34.22	12.8	47.02	S	P	P	R	R	P	P	R	R	P	P	R	R	P	P	P	P	NR			

S.NO	NAME	AGE (Y)	SEX	DIAGNOSIS	TYPE OF SURGERY	CUTTING BURR (MIN)	DIAMOND BURR (MIN)	TOTAL TIME (MIN)	PTA POD1/ POD7	TRANSIENT EVOKED OTOACOUSTIC EMISSION																			
										POD1				POD3				POD7				POD15				POD30			
23	SANGEETHA TAMASHETTI	11	F	(L) AM COM	(R) MRM + T III TP	51.2	12.37	63.57	S	P	P	P	P	P	P	P	P	P	P	P	P	NR				NR			
24	SANGAMESH QUATI	12	M	(R) AM COM	(R) MRM	49.28	16.08	65.36	S	P	P	P	P	P	P	P	P	P	P	P	P	NR				NR			
25	HEENA KOUSAR	19	F	(L) IM COM	(L) CM + TYPE I TP	42.3	7.33	49.63	S	P	P	P	P	P	P	P	P	P	P	P	P	NR				NR			
26	AMUL	12	M	(L) AS COM	(L) MRM + T IV TP	56.7	12.07	68.77	S	P	P	P	P	P	P	P	P	P	P	P	P	NR				NR			
27	JYOTHI UMADI	34	F	(R) IM COM	(R) CM + T I TP	43.23	11.08	54.31	S	P	P	P	R	P	P	R	R	P	P	P	P	NR				NR			
28	LAXMI A	16	F	(R) AM COM	(R) CM + T I TP	23.67	9.6	33.27	S	P	P	P	P	P	P	P	P	P	P	P	P	NR				NR			
29	SHANTABAI YADAV	37	F	(R) AS COM	(R) MRM + T III TP	36.67	14.48	51.15	S	P	P	P	P	P	P	P	P	P	P	P	P	NR				NR			
30	MAHADEVI KUMBHAR	45	F	(L) IM COM	(L) CM + T III TP	37.25	12.7	49.95	S	P	P	R	R	P	P	R	R	P	P	P	P	NR				NR			
31	SHIVASHANKAR D	21	M	(L) IM COM	(L) CM + TYPE I TP	42.25	8.4	50.65	S	P	P	P	P	P	P	P	P	P	P	P	P	NR				NR			
32	CHANDRASHEKAR N	31	M	(R) IS COM	(R) MRM + T III TP	42.5	17.23	59.73	S	P	P	R	R	R	R	R	R	P	R	R	R	P	P	P	P	NR			
33	BASANNA PATIL	33	M	(L) IM COM	(R) CM + T I TP	37.2	8.8	46	S	P	P	P	P	P	P	P	P	P	P	P	P	NR				NR			
34	VITHAL KAMATH	41	M	(R) AS COM	(R) MRM + T III TP	52.28	18.83	71.1	S	P	P	P	P	R	R	R	R	P	P	R	R	P	P	P	P	NR			
35	RAMAPPA BIRADAR	27	M	(L) IM COM	(L) CM + T III TP	37.57	12.12	49.69	S	P	P	P	P	P	P	P	P	P	P	P	P	NR				NR			
36	SHIKANDER PATRE	25	M	(R) AM COM	(R) MRM + T IV TP	54.33	18.67	73	S	P	P	R	R	P	P	P	R	P	P	P	P	NR				NR			
37	SIDAMMA YALAGI	45	F	(R) IM COM	(R) CM + T I TP	41.4	8.67	50.07	S	P	P	R	R	P	R	R	R	P	P	P	R	P	P	P	P	NR			
38	RENUKAMMA PATIL	40	F	(R) AM COM	(R) CM + T III TP	39.4	15.13	54.53	S	P	P	R	R	P	P	R	R	P	P	P	P	NR				NR			
39	PRAKASH PATIL	38	M	(R) IM COM	(R) CM + T III TP	41.28	9.4	50.68	S	P	R	R	R	P	P	R	R	P	P	P	P	NR				NR			
40	SUNANDA BIRADAR	41	F	(R) AS COM	(R) MRM + T IV TP	53.33	18.08	71.41	S	P	P	R	R	R	R	R	R	P	P	P	R	P	P	P	R	P	P	P	P
41	ARAVIND KAMATH	29	M	(L) IM COM	(L) CM + TYPE I TP	42.3	12.4	54.7	S	P	P	P	P	P	P	P	P	P	P	P	P	NR				NR			
42	SANTOSH LONI	25	M	(R) AS COM	(R) MRM + TIII TP	52.47	12.57	65.04	S	P	P	R	R	P	P	P	P	P	P	P	P	NR				NR			
43	KAMALA AJANAL	47	F	(R) AM COM	(R) CM + T I TP	49.12	13.75	62.87	S	P	P	P	R	P	P	R	R	P	P	P	P	NR				NR			
44	KASTURI BIRADAR	49	F	(R) IM COM	(R) CM + T III TP	41.33	6.63	47.96	S	R	R	R	R	P	P	R	R	P	P	R	R	P	P	P	R	P	P	P	P
45	SHREESHAIL HUGAR	43	M	(L) AS COM	(L) MRM + T III TP	48.42	17.67	66.09	S	P	P	P	P	P	P	R	R	P	P	P	R	P	P	P	P	NR			
46	SUDHIR KARAPE	39	M	(R) IM COM	(R) CM + T I TP	37.47	16.67	54.14	S	P	P	P	R	P	P	P	P	P	P	P	P	NR				NR			

S.NO	NAME	AGE (Y)	SEX	DIAGNOSIS	TYPE OF SURGERY	CUTTING BURR (MIN)	DIAMOND BURR (MIN)	TOTAL TIME (MIN)	PTA POD1/ POD7	TRANSIENT EVOKED OTOACOUSTIC EMISSION																			
										POD1				POD3				POD7				POD15				POD30			
47	BAPUGOUDA BIRADAR	29	M	(R) AS COM	(R) CM + T III TP	50.2	13.42	63.62	S	P	P	P	P	P	P	R	R	P	P	P	P	NR				NR			
48	MAHADEVI PATIL	36	F	(L) IS COM	(R) MRM + T IV TP	56.33	18.53	74.86	S	P	P	R	R	P	P	P	R	P	P	P	P	NR				NR			
49	SUMAYA K	16	F	(R) IM COM	(R) CM + T I TP	32.67	11.47	44.14	S	P	P	P	P	P	P	P	P	P	P	P	P	NR				NR			
50	RAJASHEKHAR K	39	M	(R) IM COM	(R) CM + T I TP	42.22	9.4	51.62	S	P	P	R	R	P	P	R	R	P	P	P	P	NR				NR			
51	JAYASHREE RAJAPUT	49	F	(L) AS COM	(R) MRM + T III TP	58.75	19.37	78.12	S	P	P	R	R	P	R	R	R	P	P	R	R	P	P	P	P	NR			
52	ANIL S. METRI	31	M	(L) AS COM	(L) MRM + T IV TP	48.42	16.53	64.95	S	P	P	P	R	P	P	R	R	P	P	P	P	NR				NR			
53	AMARESH R	39	M	(L) AM COM	(L) CM + T III TP	38.21	14.42	52.63	S	P	P	R	R	P	P	R	R	P	P	P	P	NR				NR			
54	TAMANNA H	33	F	(R) IM COM	(R) CM + T I TP	54.17	13.7	67.87	S	P	P	P	P	P	P	R	R	P	P	P	P	NR				NR			
55	SIDDALINGAPPA Y	48	M	(L) AM COM	(L) CM + T III TP	36.37	7.5	43.87	S	P	P	R	R	R	R	R	R	P	R	R	R	P	P	P	R	P	P	P	P
56	GANGAVVA BAGOJI	45	F	(L) IM COM	(L) CM + TYPE I TP	38.38	17.58	55.96	S	P	P	R	R	P	P	P	P	P	P	P	P	NR				NR			
57	BASAPPA BIRADAR	48	M	(R) AS COM	(R) MRM + T IV TP	49.2	14.83	64.03	S	P	P	R	R	P	R	R	R	P	P	P	R	P	P	P	P	NR			
58	AMAR KAMBLE	30	M	(L) AM COM	(L) CM + T III TP	34.69	15.5	50.19	S	P	P	P	P	P	P	P	P	P	P	P	P	NR				NR			
59	SHIPLA HUGAR	28	F	(L) IM COM	(L) CM + TYPE I TP	35.2	13.67	48.87	S	P	P	P	P	P	P	P	P	P	P	P	P	NR				NR			
60	APARNA CHAVAN	20	F	(L) IM COM	(L) CM + T I TP	38.68	7.2	45.88	S	P	P	P	P	P	P	P	P	P	P	P	P	NR				NR			
61	SHRUTHI HIREMATH	17	F	(L) AM COM	(L) CM + T III TP	32.67	17.2	49.87	S	P	P	P	P	P	P	P	P	P	P	P	P	NR				NR			
62	BHAYASHREE KALLI	22	F	(R) IM COM	(R) CM + T I TP	27.69	9.35	37.04	S	P	P	P	P	P	P	P	P	P	P	P	P	NR				NR			
63	SHRIDEVI PATIL	19	F	(L) AS COM	(L) MRM + T III TP	41.2	8.25	49.45	S	P	P	P	P	P	P	P	P	P	P	P	P	NR				NR			