"A PROSPECTIVE STUDY OF SENSORINEURAL HEARING LOSS AFTER MIDDLE EAR SURGERIES"

By

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Under the guidance of

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2014

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ABSTRACT

NEED FOR STUDY

Middle ear surgeries are done to correct the conductive hearing loss. Occasionally however, some of these surgeries may end up with sensorineural hearing loss, which is not called for. Sensorineural hearing loss is irreversible and its occurrence in middle ear surgeries must be taken seriously.

Thus this study is needed to determine the incidence, prognosis, possible causes and possible preventive measures for sensorineural hearing loss in patients following middle ear surgery.

OBJECTIVE

This study is done to assess the incidence, prognosis, possible causes and preventive measures of sensorineural hearing loss following middle ear surgery.

MATERIALS AND METHODS

All patients undergoing middle ear surgeries in the department of ENT, B.L.D.E.U'S Shri B. M. Patil Medical College Hospital & Research Centre from October 2011 to August 2013.

All patients undergoing middle ear surgeries will be subjected to pure tone audiometry pre operatively and bone conduction thresholds are specifically noted.

Post operatively 2^{nd} day, 7^{th} day, one month and two month later bone conduction threshold will be measured.

RESULT

Out of 153 ears, 50 underwent tympanoplasty, 78 underwent cortical mastoidectomy with tympanoplasty and 25 underwent modified radical mastoidectomy.

Incidence of development of sensorineural hearing loss was found significantly high in the immediate post operative period and most commonly with mastoidectomy procedures.

Out of 153ears, only 10 ears had persistent sensorineural hearing loss for higher frequency at the end of two month post operative follow up.

CONCLUSION

The incidence of development of sensorineural hearing loss after middle ear surgery in the immediate post operative period is high, but has tendency to recover spontaneously.

The development of sensorineural hearing loss is seen more for mastoidectomy procedures (cortical mastoidectomy and modified radical mastoidectomy) than tympanoplasty, the cause being attributable to noise trauma due to the drilling.

The sensorineural hearing loss is found for the higher frequency, 4 KHz. Contrary to the opinion[56] that the presence of a radical cavity leads to a deterioration in the bone conduction threshold, our results indicate that on an average no significant changes in bone conduction thresholds were found following cortical mastoidectomy or modified radical mastoidectomy.

CONTENTS

INTRODUCTION

Mild sensorineural hearing loss subsequent to middle ear surgery has till today been an important complication to middle ear surgery in spite of advances in surgical technique, operative instruments, monitoring devices and better treatment options.[5] Lack of proper knowledge about this problem is because of less reporting of exact magnitude of hearing loss on account of difficulty in measuring hearing threshold of patients in immediate postoperative periods as it may lead to postoperative infections and discomfort to the patients.

A study done in Royal Victoria Hospital, Belfast, Northern Ireland, a series of 3000 consecutive operations of tympanoplasty from 1960 to 1975 were reviewed in regard to the occurrence of sensorineural hearing loss as a consequence of the surgical procedure. Worsening of bone conduction thresholds by 10 dB through the frequencies 500 to 4000 cps, or a 10% reduction in speech discrimination scores were considered significant. Where as in transcanal tympanoplasty the incidence of cochlear damage was greater in ears when initially the ossicular chain was incomplete, by contrast in combined approach tympanoplasty the risk was greater when the chain was intact initially. It was concluded that cochlear trauma was usually due to 1) The hydraulic effect of excessive stapes manipulation during the removal of disease, and 2) The development of a perilymph fistula. The current methods of treating sensorineural hearing loss after tympanoplasty were enumerated and discussed. It was concluded that although those aimed at improving

labyrinthine circulation had theoretical backing, there is as yet little experimental or clinical evidence to support the claims of their protagonists.[1]

Another study done in University of Oulu, Finland, The majority of losses (57%) were limited to the frequency range of 4,000 to 8,000 Hz, and only six (0.3%) involved the most important area of speech frequencies. The results of special auditory tests were consistent with damage in the organ of Corti and are due to excessive movements of inner ear fluids, comparable to acoustic trauma. Certain surgical precautions are advocated, such as disarticulation of the incus before cleaning of the attic, dissection of the malleus in the direction of its handle, and conservative removal of granulations around the stapes.[2]

A study done in Hillel Jaffe Memorial Hospital, Hadera, Israel, Three types of mastoid surgery were evaluated: Radical mastoidectomy, modified radical mastoidectomy, and intact wall attico-mastoidectomy. The difference between the average three speech frequency preoperative bone conduction thresholds of the radical mastoidectomy group differed significantly when compared to the modified radical mastoidectomy or intact wall attico-mastoidectomy group..[3]

Another study done in Gentofte University Hospital, Hellerup, Denmark . Sensorineural hearing loss occurred in a total of 1.2% of cases: 0.5% became totally deaf, and 0.7% acquired a high tone loss, most often at 4 kHz only. [4.]

OBJECTIVE

This study is done to assess the incidence, prognosis, possible causes and preventive measures of sensorineural hearing loss following middle ear surgery.

REVIEW OF LITERATURE

HISTORICAL REVIEW

1000 BC - The early Egyptian healers had a large number of perspectives available for treatment of the ear like the use of the herbs and other extracts. It is of note that ear was often attributed to brain disease and efforts directed accordingly.[6]

400 BC - One of the earliest physicians, Hippocrates, recognized that a painful, discharging ear with fever was a life threatening condition and described classic Symptoms of otitis media. “Acute pain of the ear with continued, strong fever is to be dreaded, for there is danger that the man may become delirious and die,” Tools and techniques did not permit intervention or further work on chronic ear infection. Around the time Rafto described the tympanic membrane as a web like structure and as a part of the organ of hearing.

16th CENTURY - Surgery for mastoid infection was first proposed 4 centuries ago by Ambrose Pare on the young king Charles II of France who was dying with high fever and a discharging ear. The king’s bride, Mary, Queen of Scotland, agreed, but the king’s mother, Catherine de Medici, forbade the operation. The king died. This incident helped neither Pare’s reputation nor the fledgling specialty of otology. So another 100 years passed before the next recorded attempt at otologic intervention.

17th CENTURY - In 1640, Banzer published an account of a case of tympanic membrane repair. A pig's bladder was stretched across an ivory tube and placed in the ear. This marks a trend in the repp.ir of the drum, that of placing artificial membranes in the ear temporarily.

18th CENTURY - The first documented successful surgery for a mastoid infection was performed by Jean Petit of Paris. Shortly thereafter, in 1776, a Prussian surgeon named Jasser successfully performed a mastoid operation on a soldier with a draining ear. However, this new operation was discredited when Baron Berger, personal physician to the King of Denmark, persuaded a colleague to perform this procedure on Berger himself with a mistaken assumption that it would relieve his deafness and tinnitus. This operation led to sepsis and Berger's death, thus consigning the mastoid operation to obscurity for another century.

19th century - In 1853, Sir Oscar Wilde published a procedure for sepsis and suppuration of the ear. He described the post-auricular incision and removal of the mastoid cortex for purulent infections. This was the beginning of the modern era of otologic surgery. Nearly every operation that followed until today built upon this basic technique and expanded the indications and technique.

In 1873, Herman Schwartze published both indications and the procedure for removing the mastoid cortex and underlying air cells with mallet and chisel for acute mastoid infections. The art of using mallet and chisel

persisted for another 75 years. In the pre-antibiotic era, simple mastoidectomy became the mainstay in the treatment of acute mastoiditis and saved many lives.

Also, in 1873, von Troltsch and von Bergmann expanded the simple mastoidectomy of Schwartze to include the attic and antrum. This increased the success of mastoid surgery. Nearly all the surgical techniques in use today had been described, but advances remained slow for the next 50 years. No significant changes in the therapy of otologic disease occurred until the advent of the operating microscope and antibiotics in the 1950s.

Parallel to these developments were efforts to improve hearing. In 1853, Toynbee placed a rubber disc attached to silver wire over a perforation with hearing improvement. Yearly in 1863 improved hearing by placing a cotton ball over a perforation.

In 1877, Blake introduced the idea of placing a paper patch over the perforation, a practice that has stood the test of time. Roosa and Okneuff promoted healing of the drum by application of chemical cautery.

20 century - Surgeons were able to control disease with the techniques developed earlier. In the 1930s, antibioses helped to achieve dry ears by treating infection. Then, with the operating microscope, they became adept at examining the ear and developed instruments for manipulating the ear drum and ossicles.[6]

House, Sheehy and Glasscock developed techniques for creating a satisfactory onlay graft. Shea while performing stapedectomy, during a surgical misadventure discovered that vein graft could be satisfactorily placed under the drum to repair a tear. Storrs switched to fascia and Patterson et al determined the reasons for the success of fascia as a grafting material, the popularity and techniques of tympanoplasty can be attributed to the success of many other surgeons who have refined other's techniques.

SURGICAL AND TECHNOLOGICAL DEVELOPMENTS

Management of the Infected Ear

In 1906, the first conservative surgical procedures were described by Heath and Bryant. These were modifications of radical mastoidectomy, preserving the tympanic membrane and ossicles. These were not widely accepted due to complications.

In 1910, Bondy described the classic modified radical mastoidectomy but this did not become popular until the 1940s when it was reintroduced by Day and Baron.

Antibiotics and Instrumentation

In the 1930s, medical therapy of the ear was becoming popular with the availability of sulfonamide antibiotics. Instrumentation facilitated further development. Dental drills were used for mastoid exenteration while cautery helped control haemorrhage.

Operating Microscope

Holmgren a pioneer in fenestration Surgery for otoslerosis, Was the first otologist to use the binocular operating microscope. Lempert used the optic loupe.[6]

CONCEPT OF TYMPANOPLASTY

In 1863, a landmark discovery of the workings of the middle ear was made by Hermann von Helmholtz. His description of the middle ear transformer mechanism was essentially ignored. It was not understood until 90 years later. This work formed the foundation for all reconstructive middle ear surgery.

The concept of tympanoplasty is credited to Berthold who in 1878 was thought to have performed the first true tympanoplasty. He de-epithelialised the tympanic membrane by applying a court plaster to the membrane for 3 days, then removing it with the epithelium. A skin graft was then applied. In 1914, tympanoplasty was reintroduced by Schulhof and Valdez. In 1952, the procedure was publicized and popularized by Wullstein using split thickness skin grafts.

Zollner began his work in 1952. The work of these two surgeons integrated all previous work and formed the basis for modern otologic practice. They recognized the principles introduced by Helmholtz stating “A new tympanic membrane and an adequate tympanic cavity with intact ossicles are

necessary for the transformation of sound pressure upon the oval window as well as sound protection of the round window"[6]

Concurrently, stapes surgery was being changed radically. Kessel and Miot are credited with the first series of stapes mobilization and Blake and Jack with the first stapedectomies. Rosen reintroduced stapes mobilization in 1952 and in 1956, Shea performed the first modern stapedectomy with replacement by a prosthesis. The stability of the Zeiss operating microscope spurred further advances in middle ear surgery.

SENSORINEURAL HEARING LOSS AND MIDDLE EAR SURGERY

It has long been known that sounds of high intensity can inflict damage on the organ of hearing. As early as 1831 this was described in the lancet by Fosbroke.[7] In 1890, Habermann tested the hearing of 20 boiler makers, all of whom were found to be suffering from perceptive hearing defects. An autopsy was later performed on one of them, and microscopic examination of cochlea revealed that both organs of corti showed degeneration in the basal turn.

In the early years of the 20th century Wittmaack, Yoshii, and Rohr performed extensive experiments in various animal species to test the traumatic effect of loud sounds on the cochlea. Yoshii established that the first degenerative changes occurred in the organ of Corti, more specifically in the sensory cells. Rohr described the great variability of the auditory damage in various test animals, even when the same sound was offered, of the same intensity and duration. These observations have stood up as correct to this day.

With improved means to regulate intensity and pitch of sounds, there was an increasing interest in further investigation of the traumatic effect of loud sound on cochlea and in comparison of results. A standard work in this field, Das akustische trauma, was published in 1947 by Ruedi and Furrer. Other methods of investigation have evolved, such as behaviour audiometry in test animals, and methods to study the electrophysiology and histochemistry of the cochlea. Anatomy and pathological anatomy, however, continued to be dependent on material cut into sections, seen by the transmission electron microscope.[8,9,10] Engstorm et al have evolved a method which made the study of damaged cochlea possible by cutting the organ of Corti into sectors.[11]

Mild sensorineural hearing loss subsequent to middle ear surgery has till today been an important complication to middle ear surgery Inspite of advances in surgical technique, operative instruments, monitoring devices and better treatment options.

A study done in Royal Victoria Hospital, Belfast, Northern Ireland, a series of 3000 consecutive operations of tympanoplasty from 1960 to 1975 were reviewed in regard to the occurrence of sensorineural hearing loss as a consequence of the surgical procedure. Worsening of bone conduction thresholds by 10 dB through the frequencies 500 to 4000 cps, or a 10% reduction in speech discrimination scores were considered significant. Where as in transcanal tympanoplasty, the incidence of cochlear damage was greater in ears when initially the ossicular chain was incomplete, by contrast in combined approach tympanoplasty the risk was greater when the chain was

intact initially. It was concluded that cochlear trauma was usually due to 1) the hydraulic effect of excessive stapes manipulation during the removal of disease, and 2) the development of a perilymph fistula. The current methods of treating sensorineural hearing loss after tympanoplasty were enumerated and discussed. It was concluded that although those aimed at improving labyrinthine circulation had theoretical backing, there is as yet little experimental or clinical evidence to support the claims of their protagonists.[1]

Another study done in University of Oulu, Finland, a series of 1,680 chronic ear operations high-tone hearing losses occurred in 75 cases (4.5%). The majority of losses (57%) were limited to the frequency range of 4,000 to 8,000 Hz, and only six (0.3%) involved the most important area of speech frequencies. Some recovery occurred in all ears during the first three postoperative months. The results of special auditory tests were consistent with damage in the organ of Corti and are due to excessive movements of inner ear fluids, comparable to acoustic trauma. Certain surgical precautions are advocated, such as disarticulation of the incus before cleaning of the attic, dissection of the malleus in the direction of its handle, and conservative removal of granulations around the stapes.[2]

A study done in Hillel Jaffe Memorial Hospital, Hadera, Israel, the bone conduction threshold changes of 97 patients (100 ears) who underwent mastoid surgery were determined by comparing the last preoperative audiogram with the 1 year postoperative audiogram. Three types of mastoid surgery were evaluated: Radical mastoidectomy, modified radical mastoidectomy, and intact

wall atticomastoidectomy. The average three speech frequency preoperative bone conduction threshold was 17.4 dB (S.D. 11.5) in the radical mastoidectomy group, 10.1 dB (S.D. 9.6) in the modified radical mastoidectomy group, and 10.7 dB (S.D. 8) in the intact wall atticomastoidectomy group. The difference between the average three speech frequency preoperative bone conduction thresholds of the radical mastoidectomy group differed significantly when compared to the modified radical mastoidectomy or intact wall atticomastoidectomy group. The postoperative average three speech frequency bone conduction threshold did not change significantly following the three surgical procedures evaluated.[3]

Another study done in Gentofte University Hospital, Hellerup, Denmark .The incidence and characteristics of postoperative sensorineural hearing loss were analysed in 2,303 cases of chronic otitis and its sequel, representing our total series from 1965 to 1980. Sensorineural hearing loss occurred in a total of 1.2% of cases: 0.5% became totally deaf, and 0.7% acquired a high tone loss, most often at 4 kHz only. The incidence was highest in congenital malformations, granulating otitis and cholesteatoma, mastoidectomy (especially canal-down), and during the period from 1965 to 1974. The most common causes of anacusis were removal of cholesteatoma from the semicircular canal and removal of the fistula membrane. Different types of severe high tone loss are described and, in addition, 19 patients with mild high tone loss are discussed.[4.]

A study done in Baroda medical college, Baroda 80 patients who had undergone middle ear surgery and were on regular follow up were analysed for postoperative sensorineural hearing loss. 36 patients had no postoperative sensorineural hearing loss (45%), while 44 patients (55%) had suffered mild sensorineural hearing loss which was quite significant. Majority of the patients who suffered from hearing loss were those undergoing mastoidectomy (72%) compared to those undergoing plain tympanoplasty (38%).[5]

SURGICAL ANATOMY

The anatomy of the ear will be dealt with in relationship to:

1) External auditory canal
2) Tympanic membrane
3) Middle ear cleft
4) Eustachian tube

EXTERNAL AUDITORY CANAL

The external auditory canal is the only cul-de-sac in the human body. It is a short tube, 24mm in length in adults, open at one end and having the tympanic membrane at the other. Its outer one-third is cartilaginous (8mm) and inner two-thirds bony (16mm). The bony portion is formed by the tympanic and squamous part of the temporal bone, the narrowest part of the canal is the isthmus, 5mm lateral to the tympanic membrane. The floor of the canal dips deeply downwards and forwards to form the anterior recess. Another common finding in the meatus is the anterior canal wall bulge which has to be carefully tackled for successful myringoplasty. External auditory canal is 6 mm longer antero-inferiorly than postero-superiorly[12].

Important relations of the external auditory canal are**:**

Anteriorly - Gleniod fossa of temperomandibular joint

Parotid gland

Superiorly - Superficial temporal vessels

Auriculotemporal nerve

Middle cranial fossa

Posteriorly - Mastoid air cells

Vertical portion of facial nerve (deep)

NORMAL TYMPANIC MEMBRANE

The tympanic membrane separates the delicate structures of the middle and inner ear from the external environment. It is irregularly round and slightly conical in shape; the apex of the cone is located at the umbo, marking the tip of the manubrium. In the adult it is angulated approximately at 140° with respect to superior wall, 47° with the anterior wall and 127° with the posterior wall of the external auditory canal[12]. The vertical diameter along the manubrium is 8.5-10 mm, while the horizontal is 8-9 mm[13].

The anterior and posterior tympanic striae extend from the lateral process of malleus to the anterior and posterior tympanic spines respectively. The striae divide the tympanic membrane into the larger pars tensa below and the smaller, triangular pars flaccida (Shrapnell's membrane) above. The manubrium is firmly attached to the tympanic membrane at the umbo and the lateral process and is clearly visible (stria mallearis).

The thickened periphery of the pars tensa, the tympanic annulus (limbus), anchors the tympanic membrane in a groove known as the tympanic sulcus. The tympanic sulcus and annulus are absent superiorly in the area of

notch of Rivinus. The surgeon, when exposing the middle ear via a tympanomeatal flap approach, must elevate the tympanic annulus from the sulcus if perforation of the tympanic membrane is to be avoide[13]. It is the only membrane in the body that remains intact and closes an orifice[14].

Histology

The tympanic membrane consists of 3 layers, an outer ectodermal layer composed of keratinising squamous epithelium, an intermediate mesodermal fibrous layer and an inner endodermal mucosal layer.

The membrane is approximately 130µ thick, the outer squamous layer measuring about 30µ, the lamina propria l00µ and the mucosal layer about 1µ or less[6]. It weighs about 14 mg with a surface area of 85 mm (of which only 55 mm^2 is effective). Elasticity is close to that of rubber measuring 4.9 x 10^{-8} dynes/cm^2.

The outer epidermal layer is composed of

- Stratum corneum
- Stratum granulosum
- Stratum spinosum and
- Stratum basale.

The basal cells undergo cell division and migrate upward to replace lost and dying cells which are sloughed off as desquamated, cornified cell debris. Studies have shown the presence of epidermal growth factor (EOF) and

fibroblast growth factor (FGF) which are thought to promote healing of membrane perforations.

The epidermal layer also has migrating properties which are responsible for the self-cleansing ability of the ear. The epidermis of the human tympanic membrane migrates centrifugally from the umbo outward in a predominantly posterosuperior direction at about 131μ/day, with the umbo showing the greatest migration rate. There is also a centripetal movement of epithelium, thought to be central to healing of the tympanic membrane.

The epidermal layer also contains Langerhans' cells involved in immune response as antigen presenting cells (APCs), T-cells and mast cells. This suggests that the tympanic membrane is capable of an immune response and may play a significant role in middle ear defence.

The intermediate layer is also called lamina propria. The lamina propria of Shrapnell's membrane consists of a loose connective tissue network of collagen and elastic fibres with an external and internal network of blood vessels and nerves. The presence of elastic fibres accounts for the flaccidity of the Shrapnell's membrane. It lacks the fibrous layer of tensa. It also has abundant mast cells, the presence of which with their histamine content may play a role in the pathogenesis of middle ear effusion. Pars flaccida is thicker than tensa although lax.

In the pars tensa, the lamina propria has a sub epidermal loose connective tissue layer containing an internal network of blood vessels and

nerves and a fibrous layer made up of outer radial, inner circular and parabolic and transverse fibres in addition to the circular fibres. This unique fibrous arrangement is thought to play a role in the vibratory function of the tympanic membrane.

The outer and inner fibres are composed predominantly of collagen types II and III. In addition Procollagen I and III are seen in sub epidermal and sub mucosal layers. Procollagen type III is present throughout the fibrous layer. Antibodies to these are found in injured ear drums, which may suggest some form of autoimmunity to collagen was responsible for tympanosclerosis like lesions.

The loss of fibrous layer as a result of inflammation or atelectasis leads to a flaccid membrane. Drums that have healed spontaneously failed to show the presence of fibrous layer. Elastic fibers are rare in pars tensa.

Embryology

During the ninth week of intrauterine life the tympanic membrane is derived from the fusion of ectodermal meatal plugs from the first branchial cleft and endodermally derived first branchial pouch, known as tubotympanic recess. The mesenchyme between the meatal plate and epithelial cells of tympanic cavity form the middle fibrous layer. All these three structures constitute the tympanic membrane[6].

Blood Supply

It consists of external and internal plexus. The external plexus is formed from the tympanic branch of deep auricular artery, a branch of mandibular branch of internal maxillary artery. The deep auricular branch sends large manubrial (arteria manubrii) branches along Shrapnell's membrane and the manubrium and numerous radial branches into the tympanic membrane along its circumference. Angiography has shown that the malieal artery is the major blood supply of the posterior half of the drum, which is better perfused than the anterior half. The anterior half is apparently supplied by small radial branches from around the annulus. The internal plexus is derived from the stylomastoid branch of the postauricular artery. The venous supply closely follows the arterial distribution.

On the medial aspect of tympanic membrane the internal plexus arises in the anterior aspect, goes up to the pars flaccida and then descends along the manubrium to supply the tympanic membrane. Thus the vascular strip incision is put based on this context[6].

Innervation

The lateral surface is innervated by the auriculotemporal branch of the trigeminal nerve in the anterior half and the auricular branch of vagus nerve (Arnold's nerve) in the posterior half while the tympanic branch of the glossopharyngeal (Jacobson's nerve) supplies the medial portion of the drum.

THE MIDDLE EAR CLEFT

This includes - Tympanic cavity

- Opening of eustachian tube
- Mastoid air cell system.

The tympanic cavity is an irregular air filled cavity with four walls, a roof and a floor. It is divided into the following parts: [13]

- The hypotympanum - below the drum head
- The protympanum - anterior to the drum head
- The epitympanum - medial to the outer attic wall and above tympanic membrane
- The mesotympanum - medial to the tympanic membrane

The roof is formed by the tegmen tympani which is part of the petrous part of the temporal bone. The floor is bony and separates from the jugular bulb. The lateral wall is partly bony and partly membranous; the lateral epitympanic wall is wedge shaped and its lower bony wall is called scutum or shield of Leidy[13].

The 2 openings seen in the lateral wall are: [15]

1) Posterior canaliculus - for chorda tympani nerve and branch of stylomastoid artery

2) Petrotympanic fissure (Glasserian fissure) -this receives anterior malleolar ligament and transmits the anterior tympanic branch of maxillary artery to the middle ear.

The anterior wall has four openings[15] :

1) Canal for tensor tympani
2) Canal for eustachian tube
3) Hulguier canal for chorda tympani
4) Glasserian fissure - canal through which tympanic branch of internal caroticotympanic nerve and artery pass.

The medial wall separates middle ear from internal ear. The important structures are:

a) promontory with tympanic plexus
b) oval window
c) round window
d) facial nerve canal
e) processes cochleariformis
f) dome of lateral semicircular canal

The posterior wall has aditus, fossa incudis, pyramid, facial recess and sinus tympani.

The tympanic cavity measures 15mm vertically and anteroposteriorly. Mediolaterally it measures 6mm in epitympannum reduces to 2mm at umbo and 4mm in the hypotympanum[13].

Contents of tympanic cavity are:

- Ossicles —> Malleus, incus and stapes
- Muscles —> Tensor tympani and stapedius
- Nerves —> Chorda tympani and tympanic plexus mucosa and its folds and spaces

Ossicles

There are 3 in number namely:

Malleus

It is about 7.5mm in length. It has a head, neck and anterior and lateral (short) process and a handle. The head in the attic and articulates with the body of the incus. It weighs about 23mg. It is ovoid on cross section.

The lateral process contains a cartilaginous cap attached to the pars tensa. The pars propria splits to envelop the umbo and is firmly attached. Usually the manubrium lies midway between the anterior and posterior borders of tympanum, but may occupy a more anterior position. This anteriorly located malleus causes difficulty in repairs of anterior perforation of tympanic membrane. The tendon of tensor tympani is attached to the neck and manubrium of malleus. Normally, the medial pull of tensor tympani tendon is opposed by the elasticity of pars propria. With a large perforation of tympanic membrane, the unopposed pull causes medial displacement of inferior end of manubrium, almost reaching the promontory. In myringoplasty procedure, it

may be prudent to section the tensor tendon prior to manipulating the manubrium[13].

Incus

It is the largest of the auditory ossicles about 6 mm x 6 mm and weighing 27 mg. It consists of a body, long process, short process and a lenticular process. The body of incus rests in the epitympanum articulating with the head of the malleus. The long process parallels manubrium and ends in lenticular process to articulate with the head of stapes. The long process of incus is highly susceptible to osteitic resorption caused by chronic otitis media[13].

Stapes

It is the smallest and most medial link of the ossicular chain. It weighs around 2.3 mg and measures 3.26 mm x 2.99 mm x 1.41 mm. It consists of head, footplate (basis stapedis) and two crura. The anterior crus is more delicate and straight than the posterior. The footplate with annular ligament seals the oval window[13].

Blood supply

The malleus and incus are supplied by the ramus nutricia incudomallei, a branch of middle meningeal artery. Malleus also receives a branch from anterior tympanic artery.

The incus is also supplied by the incudal artery. The stapes is mainly supplied by two vessels, the first supplies the footplate and crura, derived from anterior tympanic artery anteriorly and plexus around the facial nerve posteriorly. The other group supplies the apex of crura, neck, head and incudo-stapedial joint with lower end of incus which arises from the plexus around facial nerve along the stapedius tendon[13].

Eustachian Tube

It is named after Bartolomeus Eustachio (1520-74). It connects the tympanic cavity with the nasopharynx, measuring 36 mm in adults, lateral third being bony and the rest fibro-cartilaginous. The two portions meet at an angle called isthmus, which is the narrowest part of the tube. The cartilage forming the medial part contributes to the medial, superior and upper part of lateral wall of the tube; the rest of the tube being completed by fibrous tissue. The Eustachian tube is wider, shorter and more horizontal in infants, thus permitting infection to travel easily from nasopharynx.[14]

Histology of middle ear

Respiratory epithelia line the mucosa of the middle ear cavity. Ciliated mucosa and goblet cells line the antero-inferior part, whereas the postero-superior area is covered with a richly vascularised cuboidal or flat epithelium. This latter epithelium is responsible for gas exchange. Variations in blood flow and permeability of blood vessels allow wide adaptations to normal fluctuations in gas pressure.

The mucous membrane lines the tympanic cavity like the peritoneum covering the viscera. These mucosal folds separate the middle ear into various compartments.

Anatomy of inner ear

In 1881 and 1884 Retzius[16] published an atlas of organ of hearing in vertebrates. His atlas presented marvellous drawings of the macroscopy and microscopy of inner ear, and has so far remained peerless. We cannot but feel the greatest admiration for the way in which Retzius, using the dissection techniques of his day and simple optical aids, managed to show details which modern investigators are today recording photographically with the aid of sophisticated electron microscopic equipment.

Cochlear structure under light microscope -

Because of its very complex shape the inner ear is called labyrinth[17]. It can be divided into membranous and a bony part or, in functional terms, into a vestibular and a cochlear part. The mammalian cochlea is indeed shaped like a snail shell. the central axis or the modulus contain the cochlear nerve and the spiral ganglion. The turns contain the cochlear duct or the scala media, which encloses the actual sense organ of Corti the cochlear duct, bisects a turn and separates the scala vestibule on the apical side from scala tympani on the basal side.

The cochlear duct is bounded on the outside by the upper portion of the spiral ligament, the inside of which is covered by stria vascularis. In the direction of the modiolus the duct is bounded by the bony spiral lamina on which the spiral limbus rests. The upper boundary is formed by Reissner's membrane, which extends between the spiral limbus and the spiral ligament. The lower boundary

is formed by the basilar membrane, which extends between the spiral ligament and the bony spiral lamina and is covered by basilar membrane cells on the side of the scala tympani. The organ of Corti rests on the basilar membrane and is spiral shaped.

On the outside the spiral limbus ends into two sharp extremities: the vestibular lip and the tympanic lip. These lips enclose the inner sulcus. From the vestibular lip the tectorial membrane extends across and is partly attached to the sensory cells and supporting cells of organ of Corti. From the inside out, the following component parts of the organ of Corti can be designated: to begin, the inner sulcus, which is lined with a layer of cubical epithelial cells known as inner sulcus cells. Next comes is a single row of border cells lying against the inner wall of the inner hair cells. The inner hair cells in turn are supported by the inner phalangeal cells, which rest against the inner pillar cells. The latter show an unmistakable fiber structure and constitute the inner boundary of the tunnel of Corti, of which the outer pillar cells form the outer boundary. Towards the spiral ligament one finds three rows of sensory cells: the outer hair cells, contained in Nuel's space and resting on Deiters cells. Each Deiters cells have phalangeal process which angulates and extends to the upper surface of the organ of Corti, where it broadens into a phalangeal plate. The surface of the sensory cells, jointly with that of the surrounding supporting cells, forms a thin, firm layer: the reticular membrane. Rows of stereocilia can be visualized near the hair cells by the surface specimen technique. The W-configuration of the stereocilia of the outer hair cells can also be distinguished.

After the third row of outer hair cells and the last row of Deiters cells, comes a ridge of supporting cells, the Hensen cells. Even further externally, the epithelium becomes cubical again, and much lower, the Claudius cells, which line the outer sulcus and continue in the spiral ligament.

Scanning Electron Microscopic Anatomy of the Organ Of Corti -

The sensory cells, or hair cells, are divided by the pillar cells into a single row of inner, and usually three rows of outer hair cells. The vestibular surface of these cells bears hairs or stereocilia. The pillar cells are likewise are divided into an inner and outer row. Together they form Corti's tunnel (canal of Corti). On the inside of inner hair cells lies a row of slender supporting cells whose surface is characterized by numerous microvilli. There cells are otherwise known as border cells[18]. Closer to the modiolus lies a row of oblong cells which are part of inner sulcus. There cells are clearly demarcated by the presence of microvilli. The other cells of the inner sulcus are flatter and of more variable shape. There nucleus is often visible on the surface. The inner sulcus cells are partly concealed by the shrunken tectorial membrane. Even closer to the modiolus, interdental cells form the surface of spiral limbus, and Reissner's membrane inserts here.

The inner pillar cells are virtually rectangular at the top, and on the surface separate the inner hair cells from the outer hair cells. The outer pillar cells are pear-shaped on the surface and separate the outer hair cells of the first row from each other and partly from those of the second row[16]. Over most of

the length of organ of Corti, there are three rows of outer hair cells, characterized by their regular arrangement and the W- configuration of their stereocilia. The outer hair cells are not only separated from each other by the heads of outer pillar cells, but also by the phalangeal plates of the first and second row of Deiters cells. These phalangeal plates are knuckle-shaped. Those of the third row of Deiters cells are polygonal and form a continuous row on the outside of the organ of Corti. This provides a sharp demarcation from the Hansen's cells. Unlike the sensory cells, the phalangeal plates and pillar heads show an abundance of microvilli. In the apical turn the Hansen's cell constitute a high dam, which gradually loses height in the direction of the round window. At the apex this dam takes an undulating course, and this also diminishes in basal direction. More externally is the outer sulcus, lined with Claudius cells; these are lower than Hansen's cells and resemble those of the inner sulcus. The outer sulcus changes into spiral prominence.

The stereocilia of an inner as well as of an outer hair cell of the guinea pig stand in three rows of varying length, like organ pipes. In the apical part, there are interconnections between the longest stereocilia of an inner hair cell[19]. However, the longest stereocilia of the outer hair cell also seem to be interconnected, at least in the upper two-thirds. The stereocilia of the inner and the outer hair cells are arranged in a W-configuration, this showing a much wider angulation in the inner than in the outer hair cell[20]. The shortest stereocilia of the inner hair cell are evidently thinner than the others, whereas the stereocilia of the outer hair cell all seem to be of the same thickness[19]. The

ends of the stereocilia of the outer cells are clubbed and packed close together, seemingly supporting each other and forming a single unit[20].

The combination of the stereocilia of a hair cell is reminiscent of a leaf spring design. Only the longest connect with the tectorial membrane, and bending of the stereocilia stimulates the sensory cell. When the longest stereocilia are bent in by a sound wave, there resistance will increase progressively and displacement will be only small, when they are bent out, there resistance will be considerably less owing to the presence of shorter ones[21,22]. This is in the direction of the basal body, which is regarded as the site of maximal sensitivity of cell membrane. The localization of the basal body is visible by a slight thickening of the surface of the outer hair cells.[23]

Nerve fibers extend through Corti's tunnel; the radiating tunnel fibers which ramify and extend between the outer pillars to the outer hair cells. These fibers are believed to be part of the afferent system, the Bundle of Rasmussen[24]. The outer hair cells are localized In Nuel's space and rest on Deiters cells. Each Deiters cell has a phalangeal process which broadens at the surface to form a phalangeal plate, always skipping every other cell so that the outer hair cell seems to be optimally spared.

The tectorial membrane seems to play an important role in the excitation of the sensory cells[25]. The mechanism of action has been a subject of discussion for decades[17,26,27]. This membrane can shrink in three directions; in height, in width and longitudinally, i.e. parallel to the axis of the cochlear duct.

It is made up of fibrillae and ground substance,[28] and can be divided into three zones: the limbal, the middle, and the marginal zone. The longest stereocilia of the outer hair cell are firmly attached to the tectorial membrane.[20,29]

THE SOUND SIGNAL

The signal perceived by the ear is the sound wave. The physical description of this phenomenon is called acoustics. The anatomical, biochemical, and physiological processes of hearing are called auditory processes.

The sound waves are the result of changes of the air pressure. Their frequency is measured in hertz (Hz). A tone is characterized by a sinusoidal wave of one frequency only. A tone is a rare exception in daily life. However it is used in audiometry to test patients' hearing. Music does not consist of single tones, but a collection of tones called sound, where the tones are usually harmonically related. Most acoustic events in daily life including speech are neither pure tones nor sounds, but include all the frequencies of the hearing range. This is called noise in acoustic terms. The magnitude of a sound wave is given by the amplitude of the sound pressure which is measured in Pascal, the sound energy passing a unit area (eg., the tympanic membrane)in a period of time is the sound intensity(also called acoustic power density)

The range of sound intensities which can be perceived by the ear – the dynamic range of the ear – is quite large. It starts at 10^{-16} W/sq.cm and reaches 10^{-4} W/sq cm . Noise at the pain threshold has the intensity higher by a factor

of 10^{12} than the hearing threshold. An audible sound intensity of 10^{-16} W/sq cm causes vibration within the cochlea of less than diameter of a hydrogen atom.

The decibel (dB) scale The wide dynamic range of the human ear results in extreme values of sound intensity and pressure. Thus, a logarithmic quantity- sound pressure level (SPL)- is used in physiology and medicine. It is measured in decibel, leading to values typically between 0 and 120 dB. The term “level” indicates that there is a logarithmic relation between the sound pressure. P_x, and a known relative sound pressure, P_o ($2x10^{-5}$ Pa, which is close to the hearing threshold).

The acronym SPL is included as a descriptor indicating that the sound pressure level is mean, and not some other logarithmic quantity. Thus only a few decibels represent a multiple of the sound pressure. When the sound pressure level is raised by 20 dB SPL, the sound pressure is increased 10 folds. When the sound pressure level is increased by 80 dB SPL, there is 10000 fold increase in sound pressure. Therefore, a patient with a hearing loss of 80 dB SPL requires a 10000 fold higher sound pressure to hear, compared to the healthy listener.

When a tone is generated at a particular sound pressure, the patient has a feeling of loudness. A raised sound pressure means that the perceived loudness is also increased and vice versa for a decrease. When the pitch of the stimulus is modulated at an unchanged sound pressure, the listener subjectively

perceives another pitch. At the same time, however, the subjectively perceived loudness also changes, although the sound pressure measured physically remains unchanged. Hence, the subjectively perceived loudness depends on frequency. At the same SPL tones between 2000 and 5000 Hz are perceived as louder than tones of higher or lower frequencies. Thus, if patient is to hear all pitches at the same loudness (isophonic), the sound pressure has to vary with the frequency. In this way, curves of equal loudness result (isophones). The convex aspect of the curves faces downwards and the curves are given in units called phon. At 1000 Hz isophon values are equal to the values of dB scale of the sound pressure level. Humans can hear frequencies between 20 Hz and 16 kHz, and loudness between 6 and 130phon.

The hearing threshold, in healthy people, every frequency is perceived starting from a characteristic minimum low SPL. This SPL is called hearing threshold. The hearing threshold for all pitches forms the lowest isophon. The hearing threshold depends on frequency; its lowest value is between 2 and 5 kHz.

In clinical practice, the measurement of the entire threshold curve would be prohibitively time- consuming because so many measurement points would be required. To overcome this problem, the hearing threshold in healthy adults at the main frequencies has been determined and is defined as 0 dB HL (hearing level); the hearing level can thus be determined by measuring at only 4-5 frequencies. Thus, the hearing threshold used clinically is characterized graphically by straight lines. This graphic display is a pure tone audiogram.

PHYSIOLOGY OF CONDUCTION OF SOUND

PHYSIOLOGY OF EXTERNAL EAR

The External Ear—the external ear forms a canal which leads the sound waves to tympanic membrane. The air on the both sides of the tympanic membrane is at body temperature. If this were not the case, then Brownian motion of the air would not be the same on the both sides of the tympanic membrane and this would lead to a stochastically audible movement of tympanic membrane[30].

The external ear canal forms a resonant cavity which results in a frequency dependent gain in the sound pressure at tympanic membrane[31]. The maximum gain of 20 dB SPL is achieved at a frequency around 2500 Hz. There is an additional resonance between 2 and 5 kHz which is produced only by the cavum of the auricle, called the resonance of the cavum conchae[32]. The hearing threshold is therefore lower (“improved”) in an open sound field than under conditions of headphone testing, since the auricle contributes to the total gain in sound pressure. The auricle contributes to the localization of the sound sources(but not to the evaluation of distance and movement). Spatial hearing relies in part on phase, level, and time differences of the sound signals of arriving at the two tympanic membranes. However, these differences are only important in lateral sound location, not in vertical location. Further modulation can be perceived by additional turning of the head, thus increasing the orientation in space[32].

TYMPANIC MEMBRANE

The movements of the tympanic membrane are studied by

- Attachment of small mirrors
- Sprinkling of fine silver particles and
- Use of an electrical probe (Bekesy)

The dispcement of the tympanic membrane are small; the amplitude of the vibration at the hearing threshold is around 10^{-10} m[33,34]. Bekesy found that the area of maximum vibration was near the lower end of the membrane. But according to Khanna and Tondorff there were two maxima of vibrations on either side of the manubrium. They suggested that the movement of malleus is less than the mass movement of the tympanic membrane.

PHYSIOLOGY OF MIDDLE EAR

In the ear, the impedance of cochlea is much higher than air. If sound waves were to hit the oval window directly, only 1% of the incident energy will be taken up, the rest being reflected back. However the middle ear transformer apparatus by its action as impedance transformer improves this considerably. In healthy ears, about 60% of sound energy is coupled to the cochlea.

Functions of the middle ear

- Sound pressure transformation
  - Ossicular leverage
  - Hydraulic action
- Acoustic separation

Ossicular Leverage

The vibrations of the ossicles can be measured by means of (a) mirrors attached to ossicles (b) stroboscopic light observation and (c) cinematography.

The malleus and incus vibrate as a combined unit rocking on a linear axis, running along the anterior ligament of malleus to the attachment of short process of incus in fossa incudis. At frequencies below about 1 kH the two ossicles appear to vibrate as a single mass. The axis of vibration passes through the column of the malleus, in front of the malleoincudal joint, to the base of the long incudal process. With sounds of moderate intensity the anterior end of the footplate of the stapes oscillates with greater amplitude than the posterior end. With high sound levels the mode of action changes to side to side rocking movements along the axis running through the footplate (longitudinally). The malieolar arm is longer than the incudal arm in the ratio of 1.3:1 to 1.15:1. The acoustic properties of the middle ear are largely determined by its two most important components, acoustic mass and stiffness. The middle ear is mass-controlled at high frequencies and stiffness – controlled at low frequencies[30,35].

Hydraulic Action

The effective vibrating area of the tympanic membrane is two-thirds of the anatomical area. The effective areal ratio between the tympanic membrane and oval window is 14:1. So the overall effective advantage for the sound pressure transmitter mechanism is 14 x 1.3=18.3.

In the normal ear the presence of the tympanic membrane, intact ossicular chain and the air containing-middle ear prevents the sound pressure waves from reaching the round window and opposing the outward movement of the round window membrane. This protection of the round window is lost where there is a large perforation of the tympanic membrane and this is one of the factors producing deafness.

The tympanic membrane is at its most efficient when the air pressures in the external auditory canal and the middle ear are equal. This is achieved by the Eustachian tube. The stapedius and tensor tympani muscles have a protective function. A contraction of middle ear muscles is induced at about 60 – 80 dB above hearing threshold. The contraction leads to an increase in ossicular stiffness. The increase in stiffness is accompanied by reduced sound transmission at low frequencies[36]. Middle ear muscle reflexes seem to play a minor role:

- In improving the signal-to-noise ratio, when a low frequency noise inhibits speech discrimination[37].
- As a protective mechanism against noise induced damage to cochlea.

The middle ear reflexes are obviously able to reduce the input to the cochlea at high SPL and low frequencies and to keep the signal at the constant level. This automatic gain control becomes effective at 20 dB above the reflex threshold at low frequencies[37]. Amplitude reduction at low frequencies assists discrimination of high frequency signals because they can be masked by the

low frequencies at high SPL. Such are protective mechanism of the middle ear muscles would, however, not be effective in impulse noise (eg.,gunshots)because of the muscles' long latency.

Inner Ear

The vibrations transmitted by the stapes produce displacement of the basilar membrane and shearing movements between the hair cells and the tectorial membrane. A stereocilia deflection initiates the sensory transduction of a hair cell. Each hair cell has 80 – 100 stereocilia at its upper end. They are arranged in rows with the sensory hairs in graded order of height: The closer to the modulus a row of stereocilia is located on a cell, the shorter they are. The tip of each sterocilium is linked to the next, taller or shorter sensory hair by a tip link[38]. When the hair cells are in resting state, the membrane potential of the IHC is -40 mV and that of OHC is -70 mV[39]. When the stereocilia of a hair cell is deflected, the resting membrane potential is changed[40]. This change is called receptor potential. The occurrence of the receptor potential reflects the transformation of a mechanical signal into an electrical signal. This is called mechanoelectrical transduction. The scala media, into which the stereocilia protrude, plays a key role in the generation of the receptor potential because it has two unique properties:

- The scala media is filled with an extracellular fluid, the endolymph, which is of an ionic composition unique within the body, in that it has an extremely high K^+ concentration of about 140 mol/l.

- Furthermore, the scala media is positively charged, resulting in an endolymphatic potential (EP) of +85mVthe high K^+ concentration and EP are both generated by the stria vascularis appropriately called the 'cochlea battery'[41].

When the stereocilia are deflected towards the longest stereocilium, ion channels open up due to the tip links[42,38], (tip link hypothesis) thus inducing opening of transduction channels, and therefore this direction is called excitatory direction[43]. The opposite direction of movement of stereocilia leads to the closure of transduction channels and hence, called, inhibitory direction or phase. The interplay between stereociliary deflections, transduction channel, depolarization, opening of basolateral K^+ channels, and repolarization has a very short time course. A cycle is completed within less than 1 ms.

Analysis of sound frequency in the cochlea

Frequency selectivity via the Travelling Waves of the Cochlear partition

The healthy ear can easily distinguish pitches when it hears the tones successively. The frequency selectivity threshold near 1 KHz is about 0.3%, 3Hz[44]. The frequency selectivity of the cochlea is produced by an elegant mechanism. The tonal vibrations induce upward and downward movement of the cochlear partition. They start immediately behind the stapes, but they are not restricted to this. The up and downward vibrations produce a wave which travels towards the apex of cochlea[45]. The travelling wave of cochlea, however, has an additional, important characteristic. Its amplitude does not decrease from the base to the apex of the cochlea. On the contrary, during its course the amplitude of the travelling wave is continuously amplified until it reaches the maximum, where after it decreases[45]. This amplification is particularly prominent at low and moderate SPL. For each pitch, the maximum of travelling wave is generated at a different location along the cochlea. The higher the frequency of the tone, the closer the travelling wave maximum is to stapes. As frequency decreases, the maximum of travelling wave moves closer to the apex of the cochlea. The corresponding pitch is called the *characteristic frequency* (CF) of that site. In the ear, this principle is called tonotopy of the travelling wave. As a result, at low SPL one single frequency leads to stimulation of a few hair cells only at the site of travelling wave maximum. Different pitches stimulate different hair cells along the cochlear partition. Therefore a complex

sound consisting of different pitches is separated out along the cochlear partition (frequency dispersion).

Passive movement of the Travelling Wave

The directional movement of the travelling wave from the base to the apex of the cochlea can be explained by a passive hydrodynamic mechanism, first described by George von Bekesy[45]. He was awarded Nobel Prize for his work. The cochlear partition is quite stiff at the cochlear base and increasingly flexible towards the apex mainly due to its increasing width towards apex[46]. However, the surface mass, of the cochlear partition is relatively constant along the cochlea. Therefore the spatial variation of the characteristic frequency of the cochlear partition is governed predominantly by the spatial variation of its stiffness.

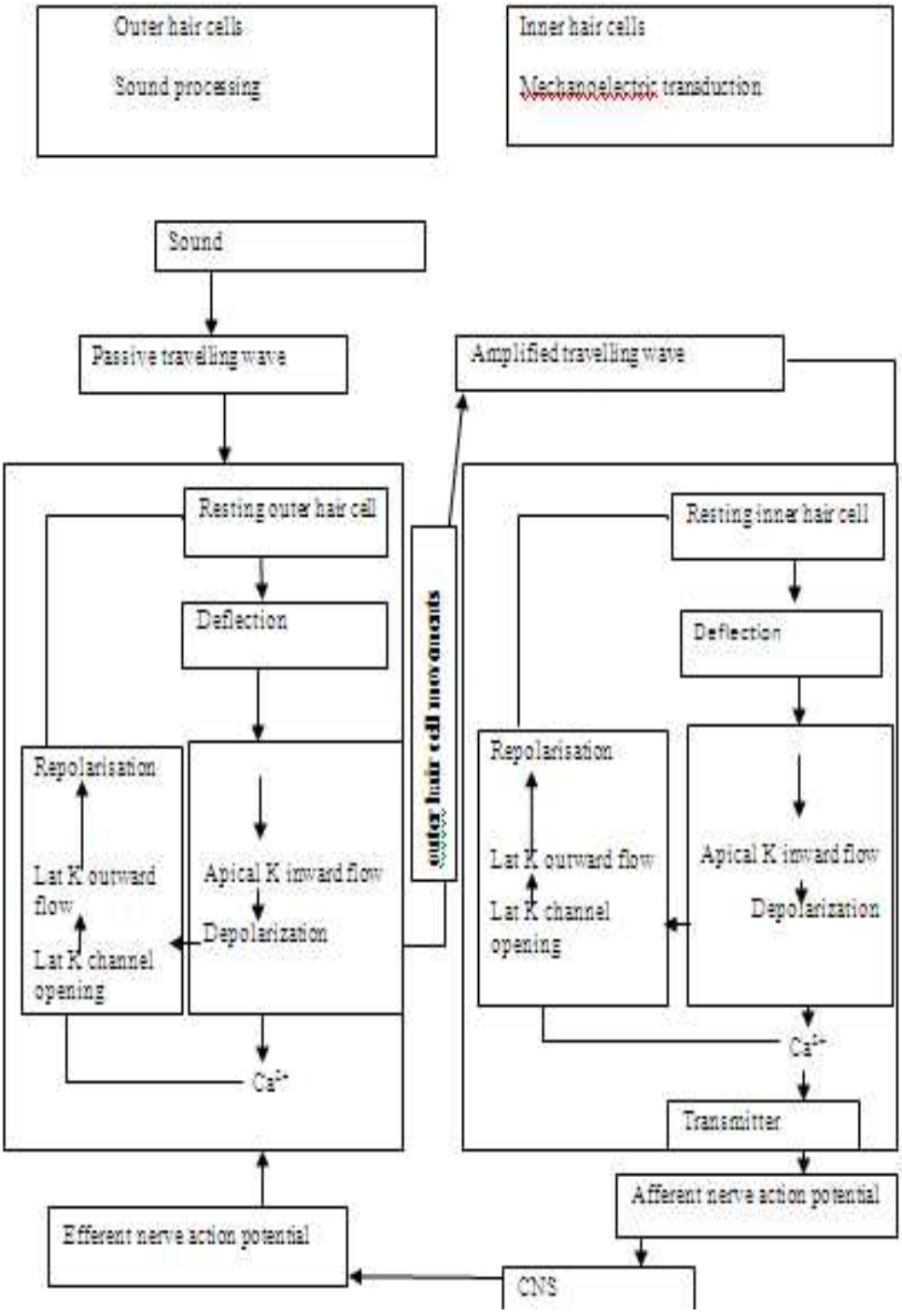
This passive hydrodynamic model, however, does not explain observable amplitude gain of the travelling wave and the exquisite frequency selectivity of the cochlea. An additional amplification mechanism is required to explain it[47,48].

Active Amplification of Travelling Wave by Outer Hair cells.

In healthy ear, OHC produce mechanical energy at low and moderate SPL, which can actively amplify the passive travelling wave by more than two orders of magnitude[49,50]. The required energy in the cochlear partition is assumed to be brought about by groups of OHC which undergo active cellular movement (length changes)[51]. These cells are located at and immediately basal

to the characteristic frequency site[52]. In this way, the organ of Corti can actively vibrate near the site of characteristic frequency. This process of passive travelling wave by OHC is called Cochlear Amplifier.

FLOW CHART SHOWING SOUND PROCESSING IN INNER EAR



Central Processing of auditory Information

The auditory nerve after a short course enters the medulla and terminates in the cochlear nucleus. Therefore, the cochlear nucleus is the first central location for processing information from the auditory periphery. Each fiber bifurcates upon entering the cochlear nucleus, sending one ascending branch rostrally to anteroventral cochlear nucleus and a descending branch caudally that innervates first the posteroventral cochlear nucleus and then the dorsal cochlear nucleus[53]. Each division contains a tonotopic representation of cochlea, with fibers tuned to low frequencies from the apical region of the cochlea represented ventrolaterally and high-frequency fibers from the basal region mapped dorsomedially. These are the sites of the first synapse. Second order neurons from the cochlear nuclei pass by different pathways to the superior olivary complex bilaterally. The presence of bilateral inputs means that superior olivary complex is the first station for processing of binaural information, providing cues for localization of sound source. Bilateral, inputs means that lesions above the cochlear nuclei do not produce unilateral deafness. The third order neurons pass up in the lateral lemniscus which is the next auditory station, projecting bilaterally to inferior colliculi. Lateral lemniscus play an integral role in the processing of binaural information, it is also a part of an acoustic- startle reflex pathway, with only 8 ms latency[54]. The inferior colliculus receives synaptic inputs from most ascending and descending auditory pathways and, therefore, is the first major site of neural integration. The central nucleus of inferior colliculus, is purely auditory processing centre, and is the main centre for spatial auditory integration. From

the inferior colliculus many fibers project to and relay in the medial geniculate body. The medial geniculate body is the obligatory thalamic relay between the auditory brainstem nuclei and the auditory cortex, receiving most of its ascending auditory input from the ipsilateral inferior colliculus and projecting mainly to ipsilateral cortical fields. The medial geniculate body receives massive reciprocal cortical input; it participates in the thalamocortical integration of multisensory information and in corticothalamic feedback.

Finally, the medial geniculate body neurons project to the auditory cortex. Auditory cortex is the principal target for ascending auditory pathways, mainly receiving ipsilateral thalamic input and contra lateral cortical input through the corpus callosum. The subdivision of the auditory cortex, in contrast to those of medial geniculate body, is extensively interconnected, with far greater intensity than between any one subdivision and any other cortical area. This observation led Winer et al[55] to propose that the auditory cortex may act primarily as a single system; in other words, it is the final place of sound recognition.

MATERIALS AND METHODS

1. SOURCE OF DATA:

All patients undergoing middle ear surgeries in the department of ENT, B.L.D.E.U'S Shri B. M. Patil Medical College Hospital & Research Centre from October 2011 to August 2013

2. METHOD OF COLLECTION OF DATA:

- Details of cases will be recorded including history and clinical examination with emphasis on detailed otoscopic examination and examination under microscope preoperatively and postoperatively.
- All patients undergoing middle ear surgeries will be subjected to pure tone audiometry pre operatively and bone conduction thresholds are specifically noted.
- Post operatively 2^{nd} day, 7^{th} day, one month and two month later bone conduction threshold will be measured.

3. INCLUSION CRITERIA:

All patients undergoing middle ear surgeries in BLDE University's Shri B. M. Patil Medical College Hospital and Research Centre from October 2011 to August 2013.

4. EXCLUSION CRITERIA:

- Patients who are undergoing middle ear surgeries for intracranial complications of middle ear disease.
- Any condition which causes progressive sensorineural hearing loss ie,
  - Age – 50 yrs
  - Meniere's disease
  - Professionals working in noisy environment
  - Hypothyroidism
  - Diabetes
  - Multiple sclerosis
  - Hypertension
  - Leukemia, polycythemia and other hyper viscosity conditions
  - Acoustic neuroma
  - Patients on ototoxic drugs.

5. SAMPLING

Time period of study: October 2011 to August 2013.

In a study conducted in Baroda Medical College, 55% of the study population had sensorineural hearing loss.[5] Considering 55% as a proportion at 95% confidence interval and allowable error of 15%, the calculated sample size is 139.

$$n = \frac{(1.96)^2 * p*q}{L^2}$$

So a minimum of 140 cases will be included in the study.

6. ANALYSIS

- Data will be presented with Mean ±SD.
- To find the incidence, causes and prognosis

  - Multiple regressions will be applied.

7. INVESTIGATIONS

Investigations or interventions required in this study are routine standardized procedures.

There is no animal experiment involved in this study.

These investigations are required as routine before taking any patient for surgery and

for routine postoperative follow-up:

- Complete blood count.
- Urine Albumin, sugar and microscopy.
- Random blood sugar, serum creatinine, blood urea.
- X-ray bilateral mastoid.
- Pure tone audiometry.

OBSERVATIONS

In our study 160 ears of CSOM were taken, of which 7 patients were lost during follow up. In 153 patients, primarily we have observed and analyzed data obtained on the following parameters.

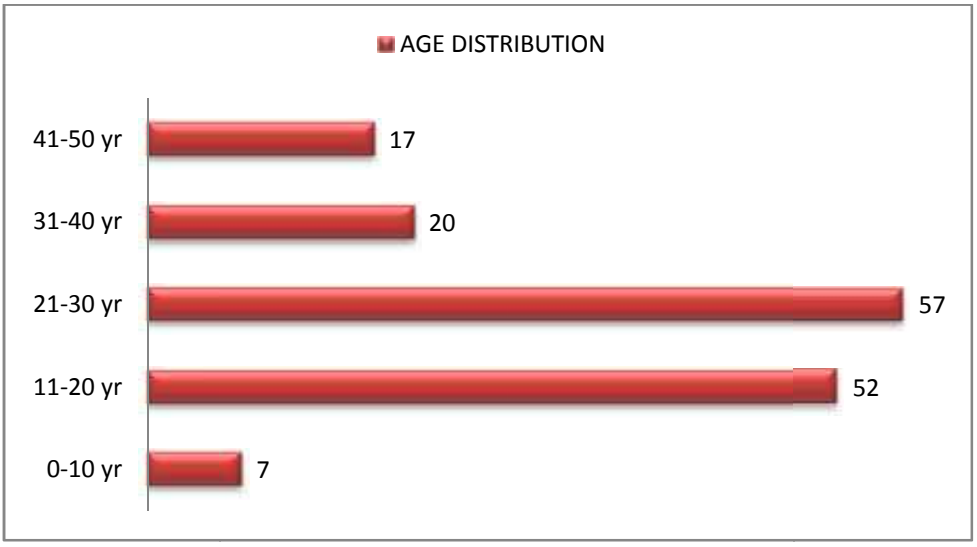
- Age Distribution.
- Sex Distribution.
- Types of middle ear surgical procedures.
- Bone conduction threshold changes and there relation with individual middle ear surgical procedures.

AGE DISTRIBUTION:

In our study, the age group ranged from 0 to 50 years. Our observations are as follows:

Table 1

Age group	Number of patients	Percentage
0 to 10	7	5
11 to 20	52	34
21 to 30	57	37
31 to 40	20	13
41 to 50	17	11
	153	100



In the present study, 7 ears (5%) were in the age group range of 0 to 10 years, 52 ears (34%) were in 11 to 20 years, 57 ears (37%) were in 21 to 30years, 20

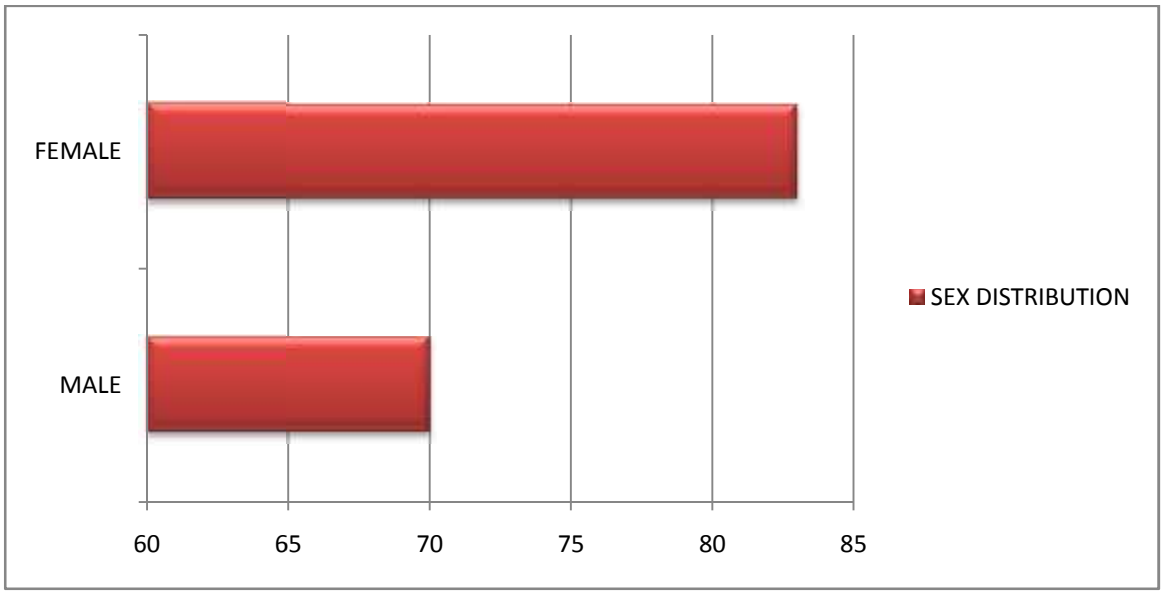
ears (13%) were in 31 to 40 years and 17 ears (11%) were in age group of 41 to 50 years respectively.

SEX DISTRIBUTION

In our study the sex distribution was also observed, the observations are as follows:

Table 2

Sex	No. of patients	Percentage
Female	83	54
Male	70	46



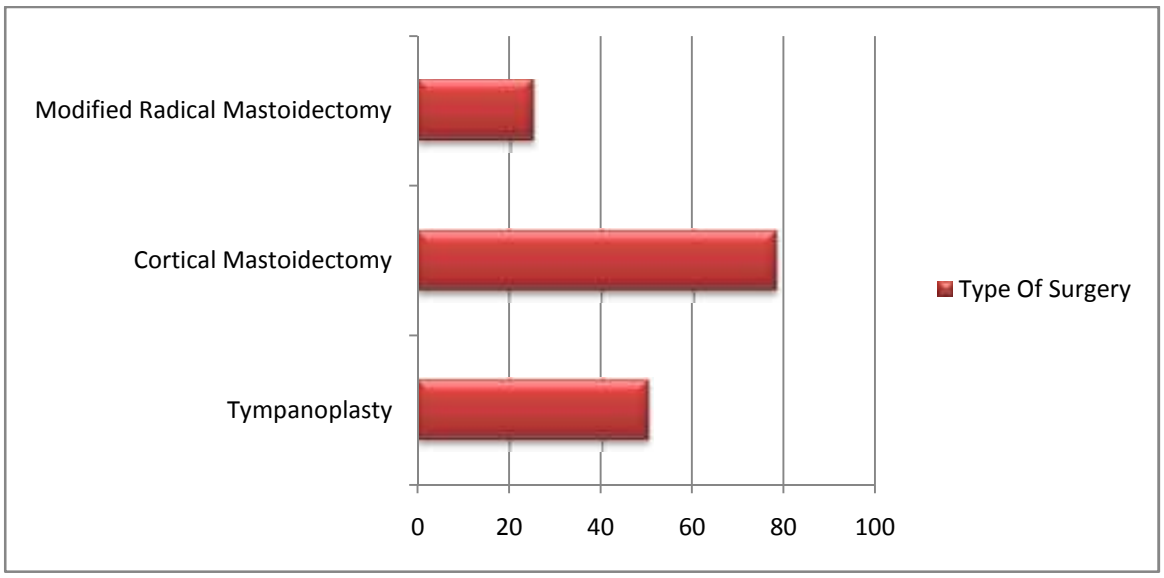
In the present study, 70 ears (46%) were of males and 83 (54%) were of females.

MIDDLE EAR SURGICAL PROCEDURES

In our study the surgical procedure distribution was also observed, the observations are as follows:

Table 3

Type of surgery	Number of ears	Percentage
Tympanoplasty	50	33
Cortical Mastoidectomy	78	51
Modified Radical Mastoidectomy	25	16



In this study,25 ears underwent modified radical mastoidectomy(16%),78 ears underwent cortical mastoidectomy with tympanoplasty(51%) and 50 ears underwent tympanoplasty(33%).

BONE CONUCTION THRESHOLD CHANGES AND THEIR RELATION TO MIDDLE EAR SURGERCAL PROCEDURES

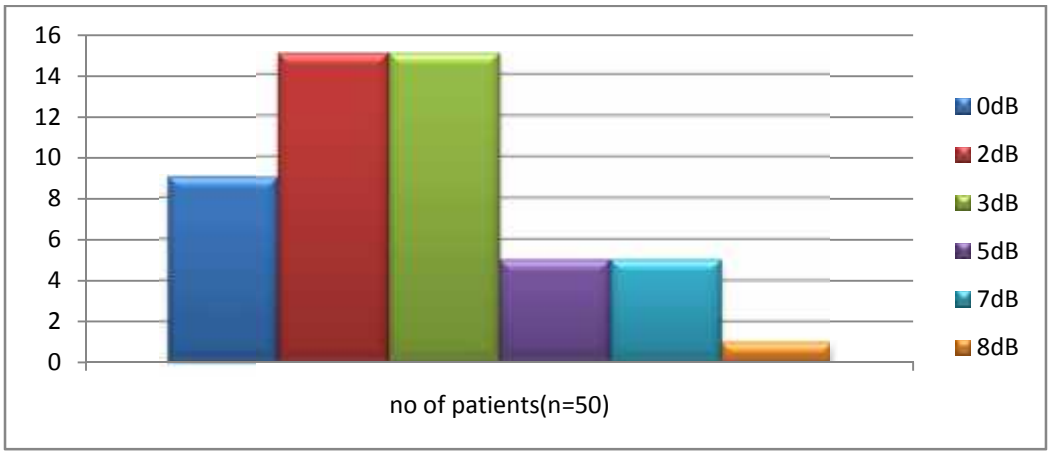
In this study, we have observed hearing loss in decibels for lower frequency, (500Hz, 1KHz, 2KHz) and post operative bone conduction threshold changes from the pre operative values were observed. Similar observation was also noted for high frequency (4 KHz).

The observations are as follows:

A1: tympanoplasty (0.5 + 1 + 2 KHz)

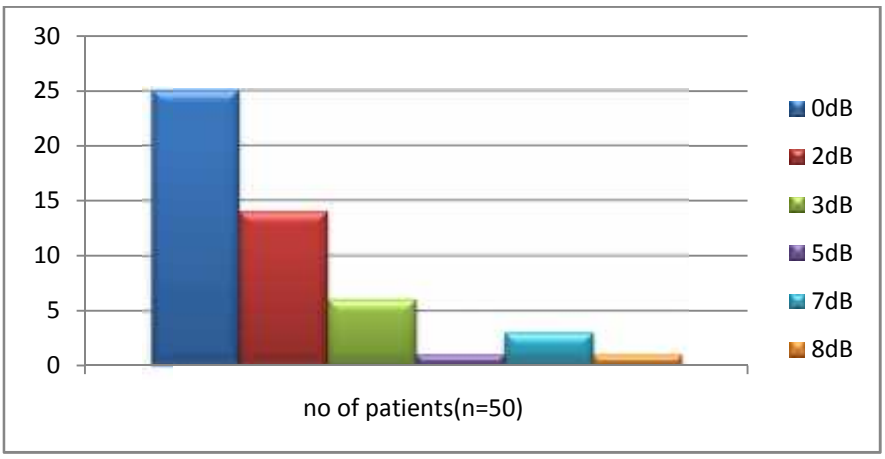
- **Day 2**

0dB	2dB	3dB	5dB	7dB	8dB
9	15	15	5	5	1



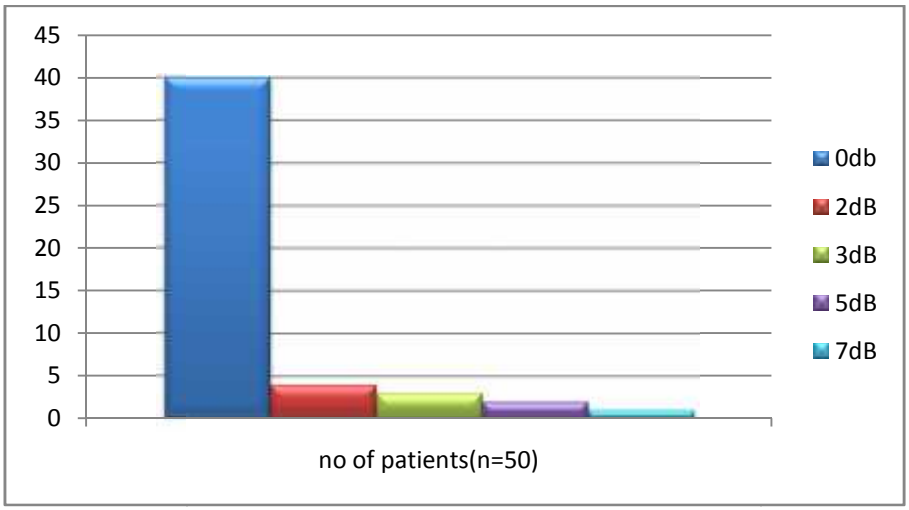
➢ **Day 7**

0dB	2dB	3dB	5dB	7dB	8dB
25	14	6	1	3	1



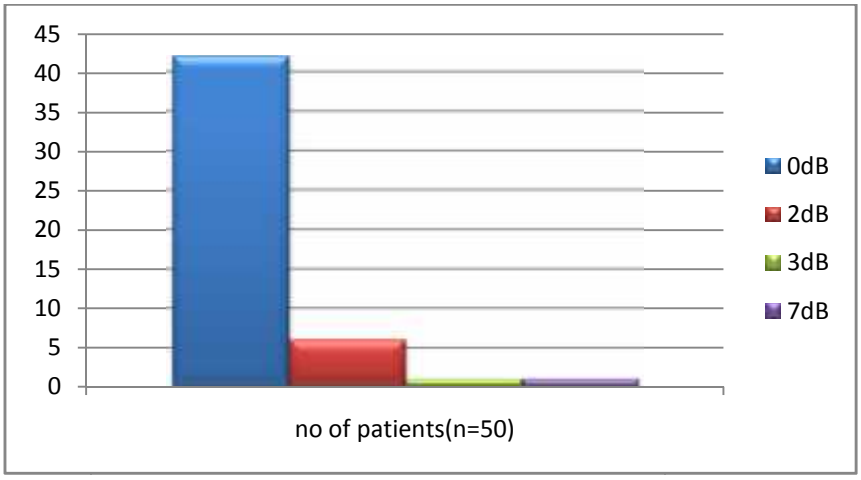
➢ **1 Month**

0dB	2dB	3dB	5dB	7dB
40	4	3	2	1



➢ **2 Months**

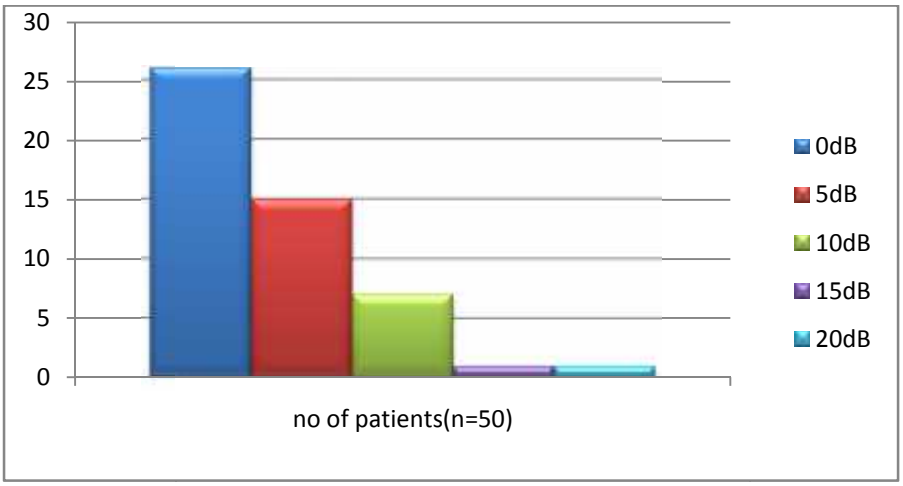
0dB	2dB	3dB	7dB
42	6	1	1



A2: Tympanoplasty (4KHz)

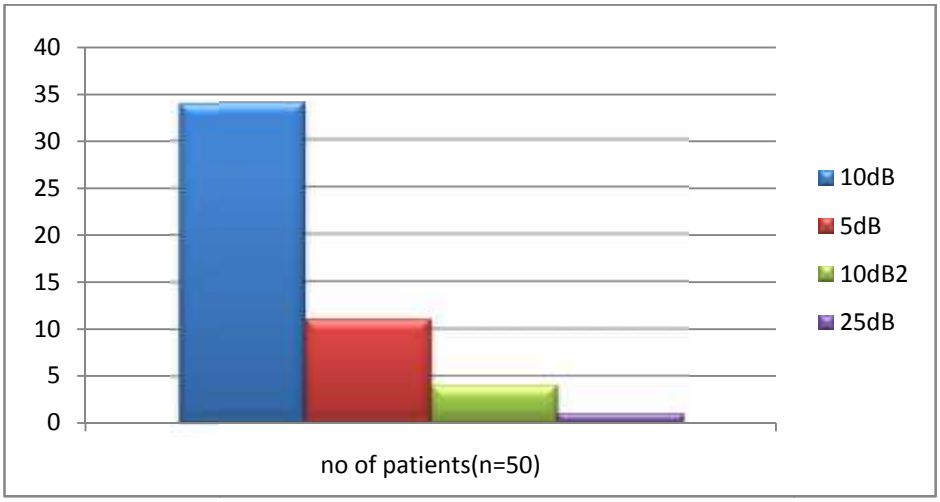
➢ **Day 2**

0 dB	5dB	10dB	15dB	20dB
26	15	7	1	1



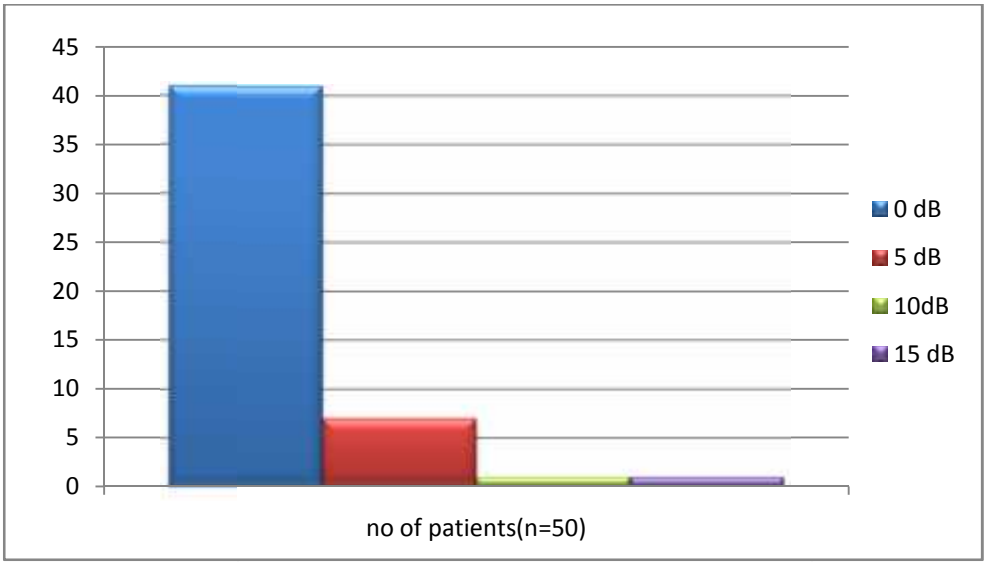
- **Day 7**

0dB	5dB	10dB	25dB
34	11	4	1



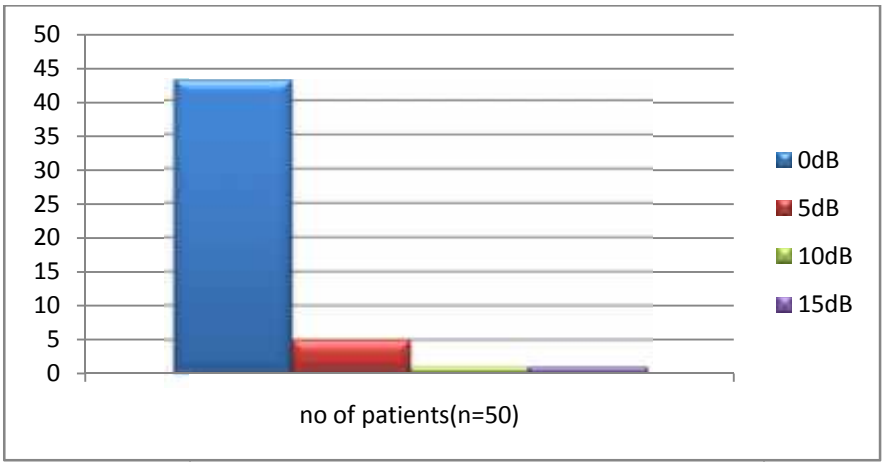
- **1 Month**

0dB	5dB	10dB	15dB
41	7	1	1



- **2 Month**

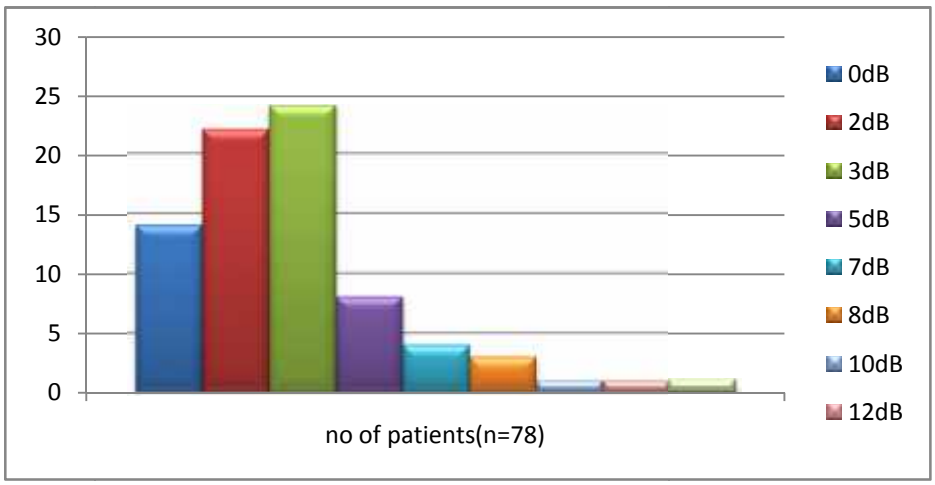
0dB	5dB	10dB	15dB
43	5	1	1



B1: Cortical mastoidectomy with tympanoplasty (.5 + 1 + 2 KHz)

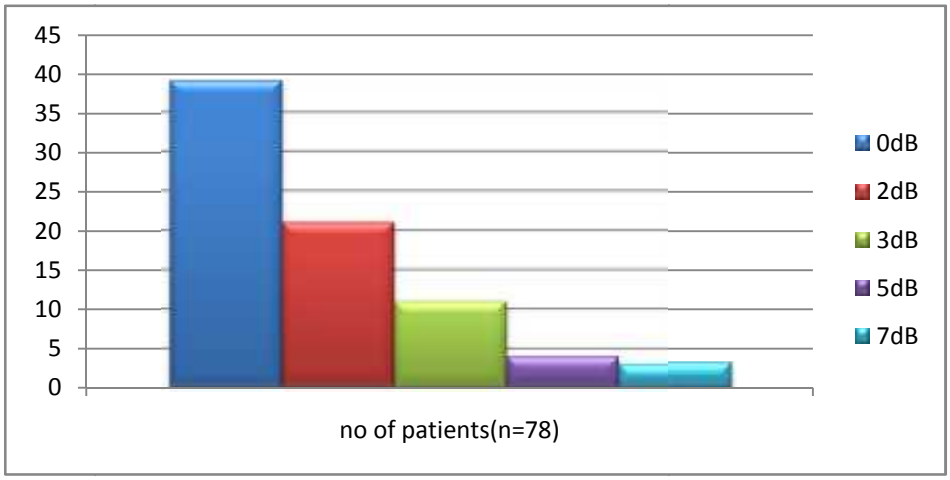
- **Day 2**

0dB	2dB	3dB	5dB	7dB	8dB	10dB	12dB	13dB
14	22	24	8	4	3	1	1	1



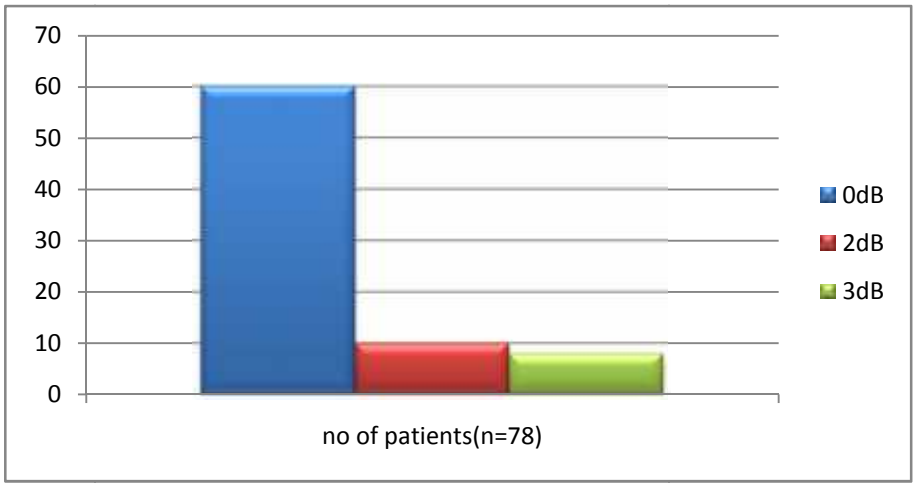
➢ **Day 7**

0dB	2dB	3dB	5dB	7dB
39	21	11	4	3



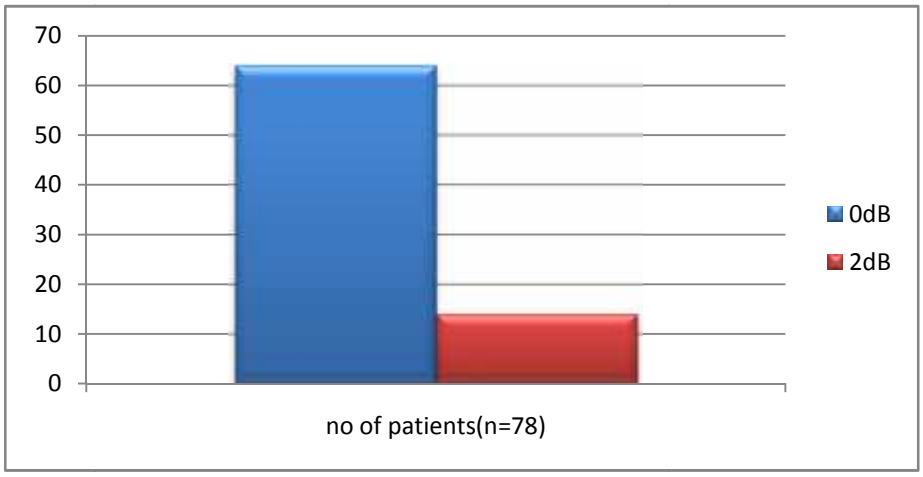
➢ **1 Month**

0dB	2dB	3dB
60	10	8



➢ **2 Month**

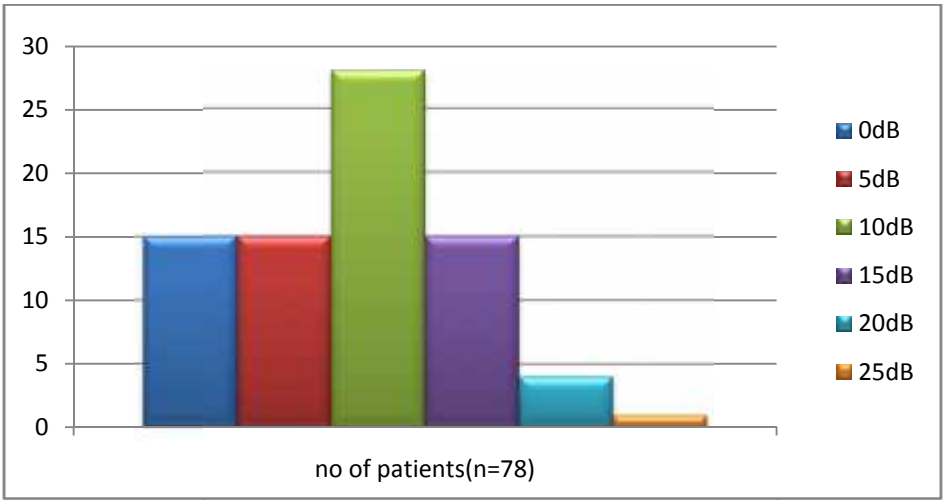
0dB	2dB
64	14



B2: Cortical mastoidectomy with tympanoplasty (4KHz)

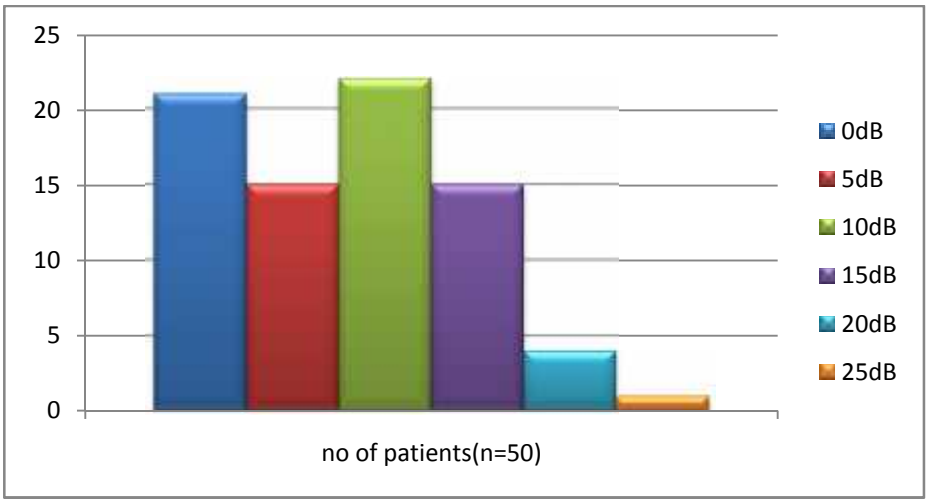
➢ **Day 2**

0dB	5dB	10dB	15dB	20dB	25dB
15	15	28	15	4	1



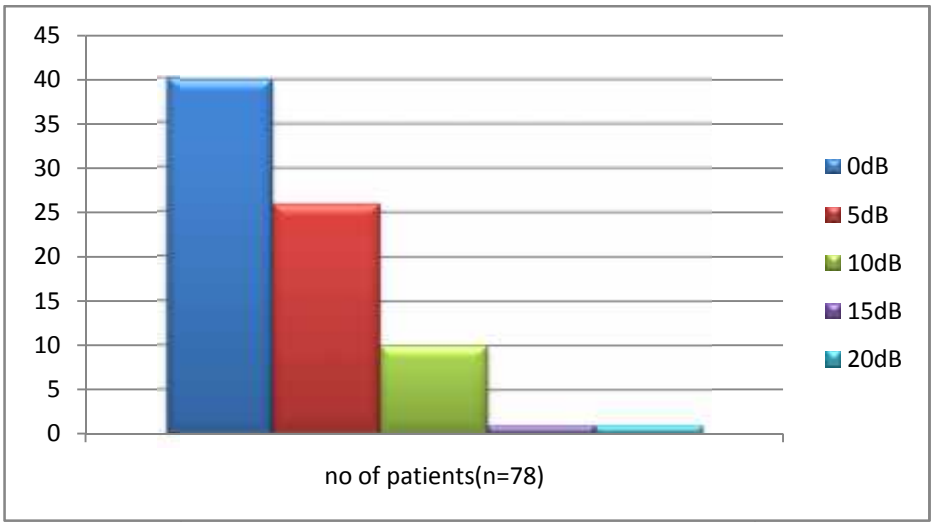
- **Day 7**

0dB	5dB	10dB	15dB	20dB	25dB
21	15	22	15	4	1



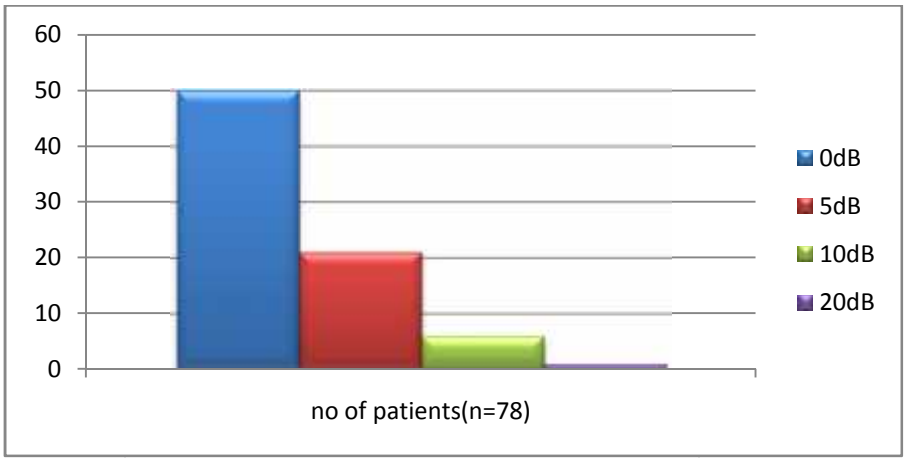
- **1 Month**

0dB	5dB	10dB	15dB	20dB
40	26	10	1	1



- **2 Month**

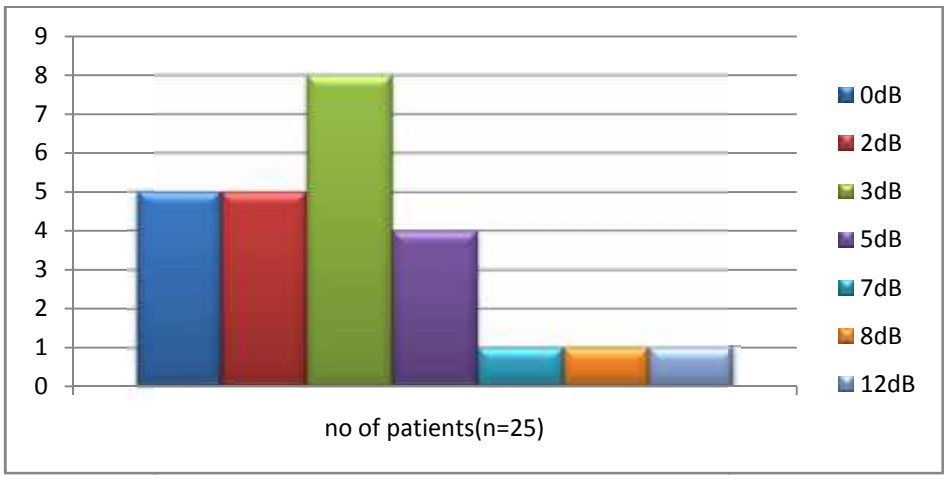
0dB	5dB	10dB	20dB
50	21	6	1



C2: Modified radical mastoidectomy (0.5 +1 + 2 KHz)

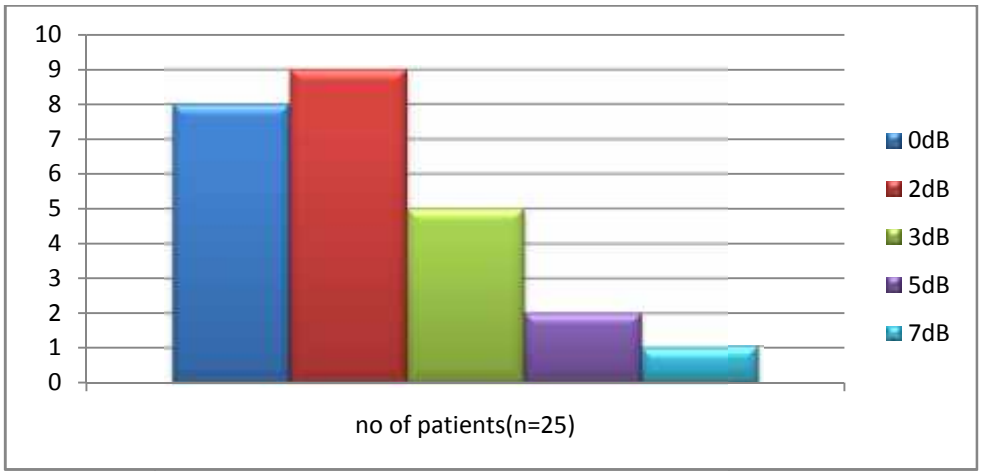
- **Day 2**

0dB	2dB	3dB	5dB	7dB	8dB	12dB
5	5	8	4	1	1	1



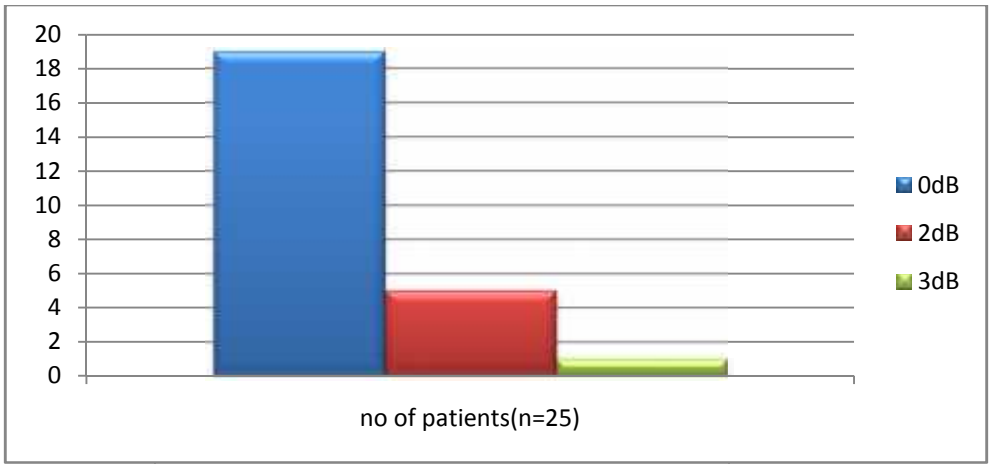
- **Day 7**

0dB	2dB	3dB	5dB	7dB
8	9	5	2	1



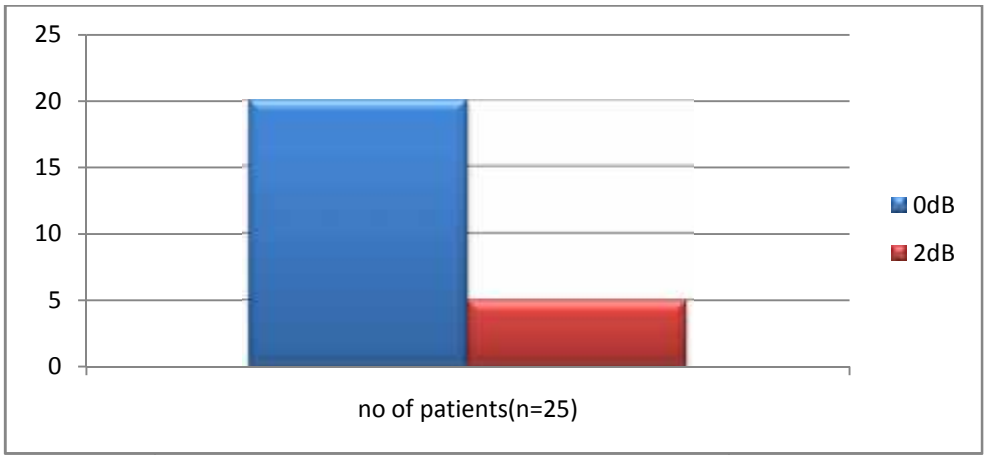
- **1 Month**

0dB	2dB	3dB
19	5	1



- **2 Month**

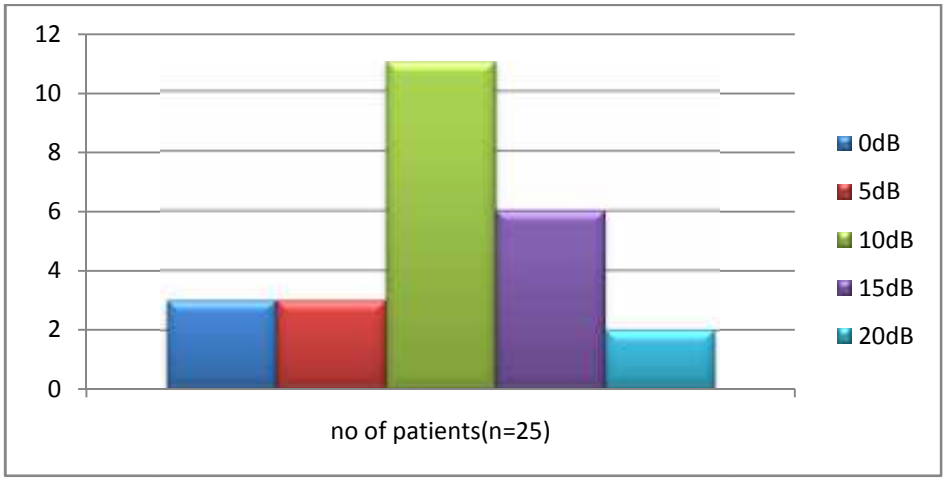
0dB	2dB
20	5



C2: Modified radical mastoidectomy (4 KHz)

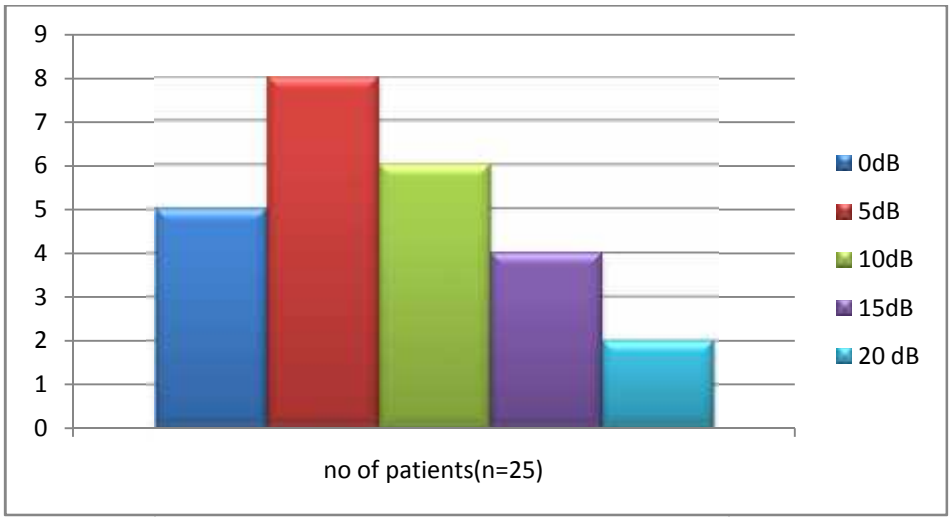
- **Day 2**

0dB	5dB	10dB	15dB	20dB
3	3	11	6	2



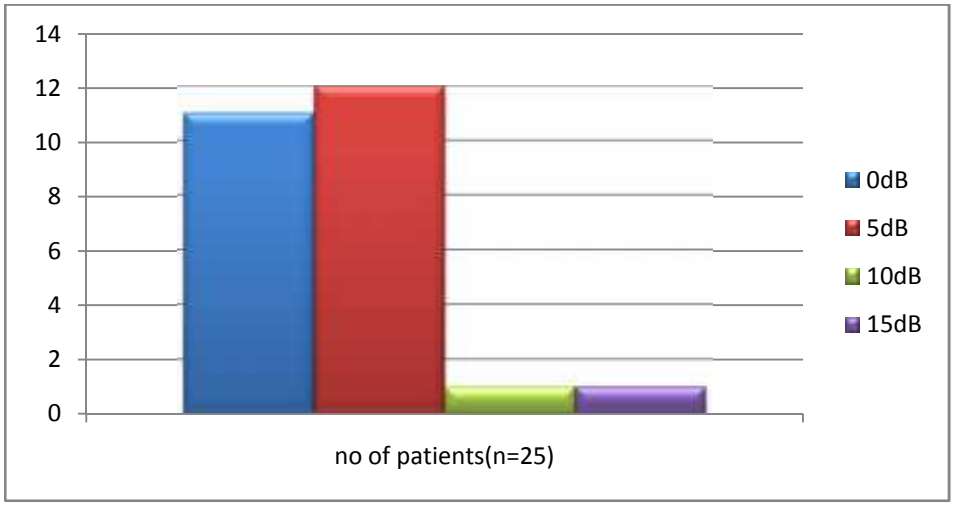
- **Day 7**

0dB	5dB	10dB	15dB	20dB
5	8	6	4	2



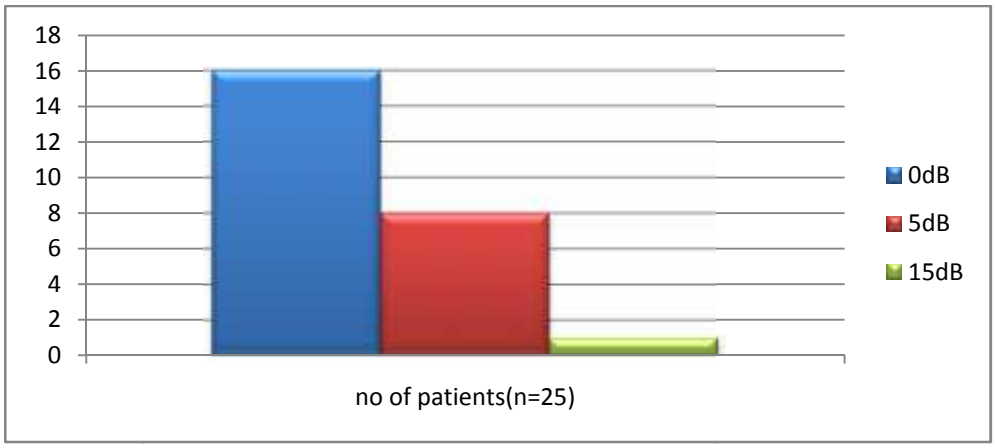
- **1 Month**

0dB	5dB	10dB	15dB
11	12	1	1



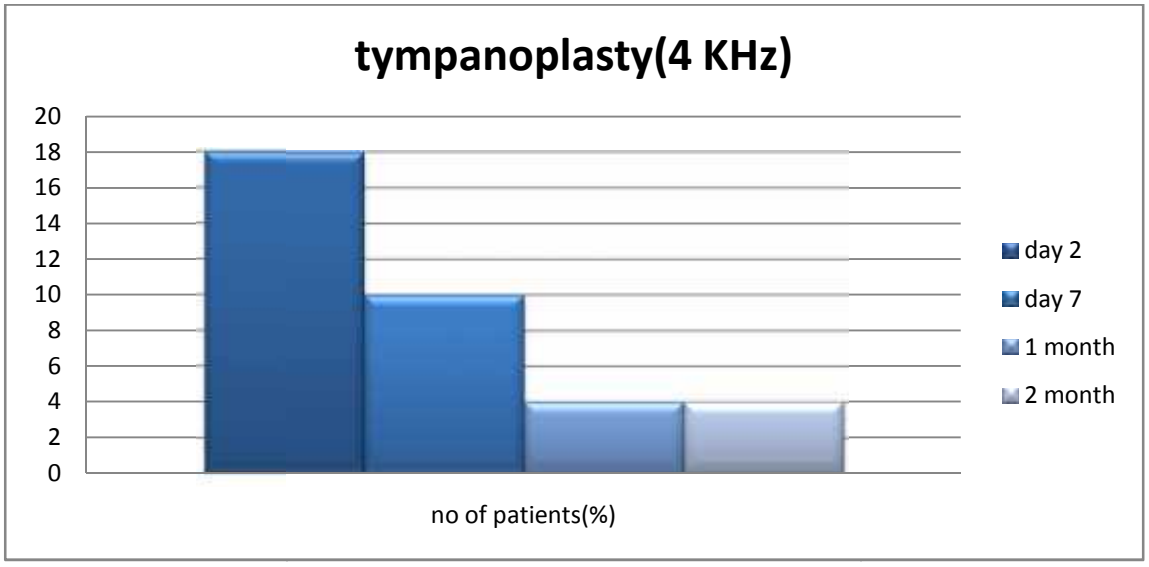
- **2 Month**

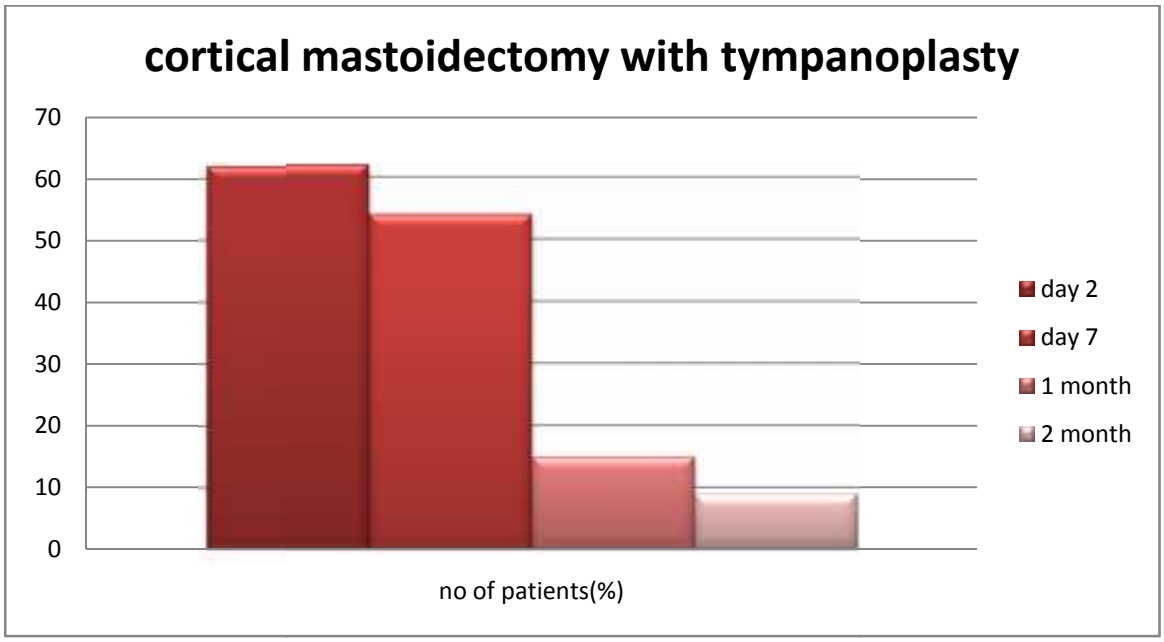
0dB	5dB	15dB
16	8	1

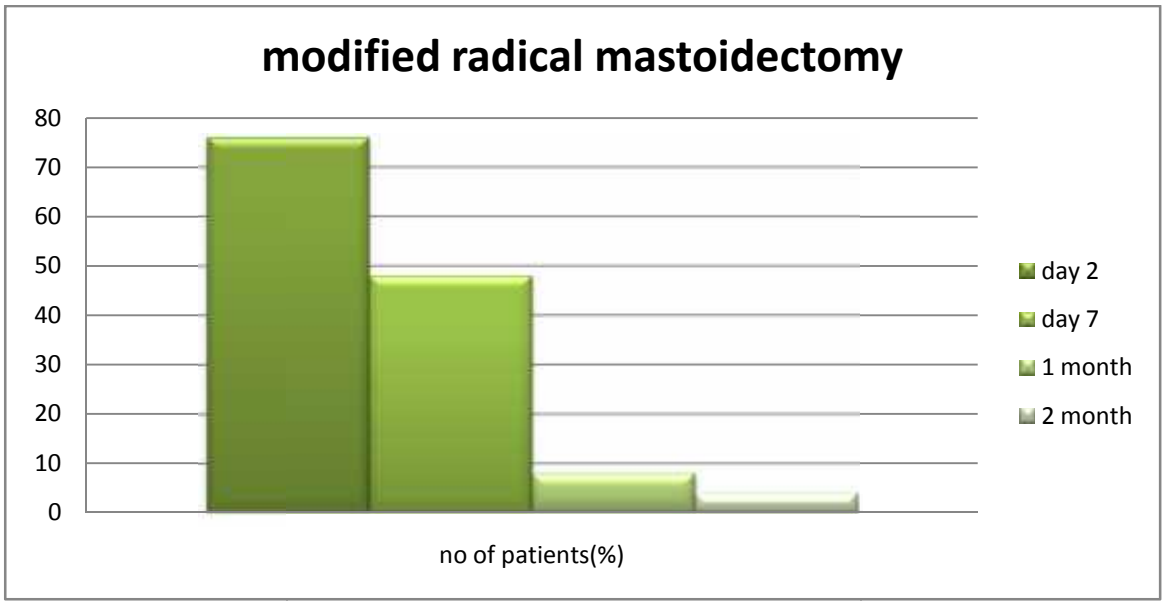


Thus, patients with more than 10 dB loss for various surgeries are:

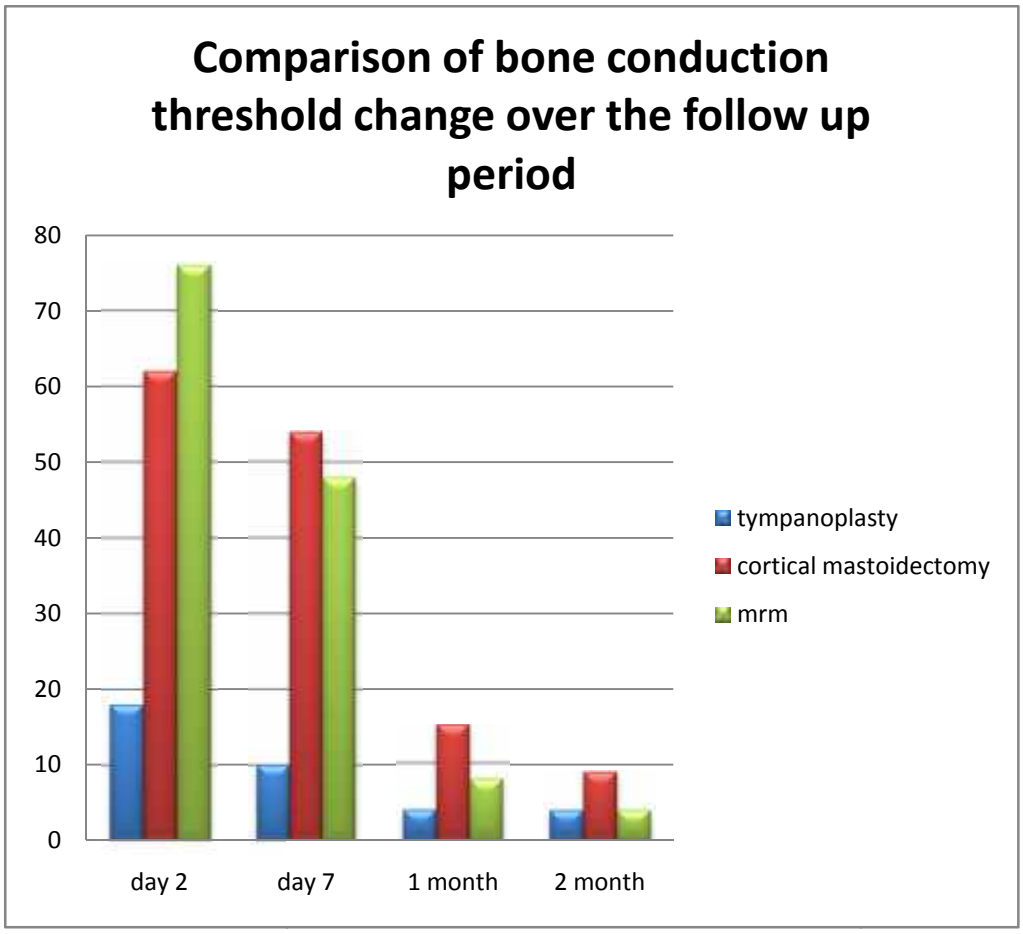
Surgery (n=153)	**Post op- Day 2**		**Post op – Day 7**		**Post op – 1 month**		**Post op – 2 month**	
	Low freq	High freq	Low freq	High freq	Low freq	High freq	Low freq	High freq
Tympanoplasty(n=50)	0	9 (18%)	0	5 (10%)	0	2 (4%)	0	2 (4%)
Cortical mastoidectomy(n=78)	3	48 (62%)	0	42 (54%)	0	12 (15%)	0	7 (9%)
Modified radical mastoidectomy(n=25)	1	19 (76%)	0	12 (48%)	0	2 (8%)	0	1 (4%)




cortical mastoidectomy with tympanoplasty
70
60
50
40
30
20
10
0
day 2
day 7
1 month
2 month
no of patients(%)


modified radical mastoidectomy
80
70
60
50
40
30
20
10
0
day 2
day 7
1 month
2 month
no of patients(%)

Summarized graphical representation of above observations is as follows



DISCUSSION AND ANALYSIS

The study was conducted from October 2013 to August 2013. In this study 160 ears of CSOM were taken, of which 7 patients were lost during follow up. Out of 153 ears were divided into three groups based on the surgical procedure the ears underwent. The surgical procedures were

A. Tympanoplasty
B. Cortical mastoidectomy with tympanoplasty
C. Modified radical mastoidectomy

Out of 153 ears, 50 underwent tympanoplasty, 78 underwent cortical mastoidectomy with tympanoplasty and 25 underwent modified radical mastoidectomy.

We considered increase in bone conduction threshold post operatively of more than 10 dB from the pre operative value as significant hearing loss. On post operative evaluation, we found that there was no considerable change in bone conduction threshold in the lower frequencies (500Hz, 1 KHz, 2 KHz).

For **4 KHz:**

In **tympanoplasty** out of 50 ears; on post operative day 2, there were 9(18%) with sensorineural hearing loss which showed an improvement to 5(10%), 2 (4%) and 2 (4%) over day 7, 1 month and 2 month respectively. Thus, at 2 months only 2 ears out of 50 operated ears of tympanoplasty had a permanent hearing loss.

	d	n	ni	p	q	nipbar	x-nibar	sqre	nipqbar	x2
2	9	41	50	0.18	0.82	4.5	4.5	20.25	4.095	4.945054945
7	5	45	50	0.1	0.9	4.5	0.5	0.25	4.095	0.061050061
1m	2	48	50	0.04	0.96	4.5	-2.5	6.25	4.095	1.526251526
2m	2	48	50	0.04	0.96	4.5	-2.5	6.25	4.095	1.526251526
Total	18	182	200							8.058608059

p bar	0.09
q bar	0.91
pqbar	0.0819

P value =.025< 0.044816< 0.05

Thus, Using chi square test for comparing more than two proportions it was found that there is no significant change of bone conduction threshold change for tympanoplasty over follow up days.

In **cortical mastoidectomy with tympanoplasty** out of 78 ears; at post operative day 2 there were 48(62%) with sensorineural hearing loss which showed an improvement to 42(54%),12(15%) and 7(9%) over the day 7,1 month and 2 month respectively. Thus, 7 ears out of 78 operated ears of cortical mastoidectomy with tympanoplasty had a permanent hearing loss.

	d	n	ni	P	Q	nipbar	x-nibar	sqre	nipqbar	x2
2	48	30	78	0.61538	0.38461	27.25	20.75	430.562	17.72995	24.28447
7	42	36	78	0.53846	0.46153	27.25	14.75	217.562	17.72995	12.2709
1m	12	66	78	0.15384	0.84615	27.25	-15.3	232.563	17.72995	13.11694
2m	7	71	78	0.08974	0.91025	27.25	-20.3	410.063	17.72995	23.12825
total	109	203	312							72.80056
	p bar	0.349359								
	q bar	0.650641								
	pqbar	0.227307								

P value =0.025> 1.07E-15<0.05

Thus, using chi square test for comparing more than two proportions it was found that there is significant bone conduction threshold change for the cortical mastoidectomy with tympanoplasty procedure over the post operative period.

In **modified radical mastoidectomy** out of 25 ears; there were 19(76%) with sensorineural hearing loss which showed an improvement to 12(48%), 2 (8%) and 1 (4%) over day 7,1 month and 2 month respectively. Thus, at 2 months only 1 ear out of 25 operated ears of modified radical mastoidectomy had a permanent hearing loss.

	d	n	ni	p	q	nipbar	x-nibar	sqre	nipqbar	x2
2	19	6	25	0.76	0.24	8.5	10.5	110.25	5.61	19.65241
7	12	13	25	0.48	0.52	8.5	3.5	12.25	5.61	2.183601
1m	2	23	25	0.08	0.92	8.5	-6.5	42.25	5.61	7.531194
2m	1	24	25	0.04	0.96	8.5	-7.5	56.25	5.61	10.02674
total	34	66	100							39.39394
	p bar	0.34								
	q bar	0.66								
	pqbar	0.2244								

P value =0.025 >1.43E-08 <0.05

Thus, using chi square test for comparing more than two proportions it was found that there is significant bone conduction threshold change for the modified radical mastoidectomy procedure over the post operative period.

By using chi square test,we found that there is no significant difference in the bone conduction threshold change caused by various surgical procedures by 2 months period.

Surgery	Not recovered	Recovered	Chi Square test
Tympanoplasty	2	48	X^2 =1.549 P value=0.461
Cortical mastoidectomy	7	71	
Modified radical mastoidectomy	1	24	

We found that out of 153 ears, 143(93.465%) had no post operative sensorineural hearing loss while 10 (6.535%) had suffered hearing loss at the end of 2 months for the high frequency (4KHz). Majority of the patients who suffered hearing loss were of cortical mastoidectomy with tympanoplasty (70%),tympanoplasty (20%) and modified radical mastoidectomy (10%) respectively.

SUMMARY

- This prospective study was performed in 153 patients aged between 6 yrs to 50 yrs attending BLDEA's Shri B. M. Patil Medical College, Hospital and Research Centre, Bijapur from October 2011 to August 2013.
- All patients were subjected to thorough history taking and examination to exclude those patients who were undergoing ear surgeries for intracranial complications and otosclerosis.
- All the patients were subjected to pure tone audiometry for assessment of hearing threshold before and after surgery.
- Out of 153 ears, 50 underwent tympanoplasty, 78 underwent cortical mastoidectomy with tympanoplasty and 25 underwent modified radical mastoidectomy.
- Incidence of development of sensorineural hearing loss was found significantly high in the immediate post operative period and most commonly with mastoidectomy procedures.
- Out of 153ears, only 10 ears had persistent sensorineural hearing loss for higher frequency at the end of two month post operative follow up.

CONCLUSION

- The incidence of development of sensorineural hearing loss after middle ear surgery in the immediate post operative period is high, but has tendency to recover spontaneously.
- The development of sensorineural hearing loss is seen more for mastoidectomy procedures (cortical mastoidectomy and modified radical mastoidectomy) than tympanoplasty, the cause being attributable to noise trauma due to the drilling.
- The sensorineural hearing loss is found for the higher frequency, 4 KHz.
- Contrary to the opinion[56] that the presence of a radical cavity leads to a deterioration in the bone conduction threshold, our results indicate that on an average no significant changes in bone conduction thresholds were found following mastoid surgery with cortical mastoidectomy or modified radical mastoidectomy.
- Studies have shown recoveries from noise induced hearing loss following middle ear surgeries, up to one year post operative period[57]. Hence, we recommend longer follow up period for better assessment.

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BLDEU'S SHRIB.M.PATILMEDICALCOLLEGE AND RESEARCH CENTER, BIJAPUR- 586103

SAMPLE INFORMED CONSENT FORM

BLDEU'S SHRI B. M. PATIL MEDICAL COLLEGE AND RESEARCH CENTER, BIJAPUR- 586103

TITLE OF THE PROJECT: **PROSPECTIVE STUDY OF SENSORINEURAL HEARING LOSS AFTER MIDDLE EAR SURGERY**

PG GUIDE: **DR. S. P. GUGGARIGOUDAR**
PROFFESSOR OF E.N.T.
B.L.D.E.U.'s Shri B. M. Patil Medical College, Hospital & Research Centre,
Bijapur, Karnataka.

PG STUDENT: **DR.RANI BABY**
P.G. Student,
Dept. of E.N.T.

PURPOSE OF RESEARCH

I have been informed that this is a study to assess the sensorineural hearing loss following middle ear surgery with its prognosis and preventive measures.

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.

RISK AND DISCOMFORTS:

I understand there is no risk involved and I will experience some pain and discomfort during my procedures performed. 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.

BENEFITS:

I understand that my participation in this study will help the investigator to assess sensorineural hearing loss occurring after middle ear surgery

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 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.

REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at any time. Dr. Rani Baby 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, if 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.

REFUSAL FOR 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. Rani Baby 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.

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. Rani Baby	Dr. S. P. Guggarigoudar
Date:	Date:
(Investigator)	(Guide)

STUDY SUBJECT CONSENT STATEMENT:

I confirm that Dr. Rani Baby 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

SCHEME OF CASE TAKING

1) Name: CASE NO:

2) Age: IP NO:

3) Sex: DOA:

4) Religion: DOS:

5) Occupation: DOD:

6) Residence:

7) CHIEF COMPLAINTS:

8) HISTORY OF PRESENTING ILLNESS:

9) PAST HISTORY:

- Diabetes mellitus
- Hypertension
- History of any drug intake
- Renal disease
- Jaundice

10) FAMILY HISTORY:

11) GENERAL PHYSICAL EXAMINATION:

Pallor:	present/absent
Icterus:	present/absent
Clubbing:	present/absent
Generalized Lymphadenopathy:	present/absent
Build:	Poor/Medium /Well
Nourishment:	Poor / Medium / Well

12) VITALS

PR:

BP:

RR:

Temp:

Weight:

13) OTHER SYSTEMIC EXAMINATION:

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

14) LOCAL EXAMINATION

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

- Tympanic membrane

Pars Tensa

Pars flaccida

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

- NOSE

- ORAL CAVITY

- OROPHARYNX

15) INVESTIGATION:

BLOOD: Hb
TC
DC
ESR
BT
CT
BLOOD UREA
SERUM CREATININE
RBS
X RAY: BILATERAL MASTOIDS
VERTEX PURE TONE AUDIOMETRY:

- Pre Operative
- Second day post operative
- Seventh day post operative
- One month post operative
- Two months post operative

URINE: Albumin
Sugar
Microscopy

16) FINAL DIAGNOSIS:

17) SURGERY AND INTRA OPERATIVE FINDINGS:

18) INFERENCE:

19) COMMENTS:

KEY TO MASTER CHART

SL	Serial No.
IP	In Patient
MRM	Modified radical Mastoidectomy
SNHL	Sensory Neural Hearing Loss
A	Absent
P	Present

Sl no	Name	Age	Sex	Date	Ip no	Diagnosis	Surgery	Surgery Date	Preoperative levels				Postoperative levels Day 2				Postoperative levels day 7				Postoperative levels 1 month				Postoperative level 2 months				SNHL
									Bone conduction threshold				Bone conduction threshold				Bone conduction threshold				Bone conduction threshold				Bone conduction threshold				
									0.5 KHz	1 KHz	2 Khz	4KHz	0.5 KHz	1KHz	2 KHz	4KHz	0.5 KHz	1 KHz	2 KHz	4 KHz	0.5KHz	1KHz	2KHz	4KHz	0.5KHz	1KHz	2KHz	4KHz	
1	Sudeep harijan	10	M	10/15/2011	21887	Rt CSOM (aa)	Rt MRM type 3 tympanoplasty	10/18/2011	15	10	15	15	15	20	15	25	15	15	15	25	15	10	15	15	15	10	15	15	A
2	Gundamma	15	F	11/2/2011	23164	Lt CSOM (tt)	Lt Cortical type 1 tympanoplasty	11/3/2011	10	15	10	10	10	15	10	20	10	10	10	20	10	10	10	15	10	10	10	10	A
3	Shilpa shri shail	13	F	11/2/2011	23161	Lt CSOM (tt)	Lt Cortical type 1 tympanoplasty	11/3/2011	0	5	0	0	0	10	0	20	5	10	0	20	5	10	0	10	0	5	0	5	A
4	Savita Patil	16	F	11/4/2011	23373	Lt CSOM (tt)	Lt Cortical type 1 tympanoplasty	11/5/2011	10	5	10	5	15	5	15	20	10	5	10	20	10	5	5	10	10	5	5	10	A
5	Sangamesh Patil	13	M	11/5/2011	23471	B/L CSOM(tt)	Rt Cortical type 1 tympanoplasty	11/8/2011	10	10	5	5	10	15	10	20	10	10	10	20	5	10	5	15	5	10	5	15	P
6	Swetha	15	F	11/9/2011	23699	Lt CSOM(tt)	Lt Cortical type 1 tympanoplasty	11/10/2011	10	10	5	5	10	10	5	15	5	5	5	15	5	5	5	10	5	5	5	10	A
7	Geeta Pattar	21	F	11/21/2011	24593	Rt CSOM (tt)	Rt Cortical type 1 tympanoplasty	11/22/2011	15	10	10	5	15	15	10	25	15	10	10	25	10	10	5	15	15	10	5	15	P
8	Mallikarjun	19	M	11/28/2011	25188	Rt CSOM (aa)	Rt MRM type 3 tympanoplasty	11/29/2011	10	15	10	15	10	15	15	20	10	15	10	20	10	10	10	15	10	10	10	15	A
9	Renuka	16	F	11/28/2011	25209	Rt CSOM (aa)	Rt MRM type 4 tympanoplasty	11/29/2011	10	15	10	10	10	10	15	20	10	10	15	15	10	10	10	10	10	10	10	10	A
10	Bismilla	15	F	1/4/2012	304	Lt CSOM (tt)	Lt type1 tympanoplasty	1/5/2012	5	5	5	10	5	20	10	25	5	15	5	20	10	10	5	15	5	5	5	5	A
11	Dadagouda Patil	18	M	1/10/2012	773	Lt CSOM (tt)	Lt type 1 tympanoplasty	1/11/2012	0	0	5	0	5	5	5	5	5	5	5	5	5	5	5	0	5	0	5	0	A
12	Kavita Kudli	35	F	1/30/2012	2406	Lt CSOM(aa)	Lt MRM type 4 tympanoplasty	1/31/2012	15	15	20	10	15	15	20	20	15	10	15	15	15	15	15	15	15	15	15	15	A
13	Shantabai	44	F	2/14/2012	3662	Rt CSOM(tt)	Rt Cortical type1 tympanoplasty	2/15/2012	15	10	10	10	15	10	15	20	15	15	10	20	15	10	5	10	15	10	10	15	A
14	Netra	18	F	3/3/2012	5133	B/L CSOM(tt)	Lt Cortical type 1 tympanoplasty	3/5/2012	5	5	0	10	5	5	0	5	5	5	0	5	10	5	0	5	5	5	0	5	A
15	Mahantesh	20	M	3/29/2012	7063	Rt CSOM(aa)	Rt MRM	3/30/2012	5	10	10	5	5	10	15	15	5	10	15	15	5	10	10	10	5	5	10	5	A
16	Riya	29	F	4/8/2012	7745	Lt CSOM (tt)	Lt type 1 tympanoplasty	4/9/2012	0	5	5	0	0	10	10	5	0	10	5	5	0	5	5	5	0	5	5	5	A
17	Subash	50	M	4/10/2012	7977	Lt CSOM (tt)	Lt Cortical type 1 tympanoplasty	4/11/2012	10	10	5	10	10	10	15	10	10	15	15	10	10	10	10	10	10	10	10	10	A
18	Chandrakala	34	F	4/16/2012	8428	Lt CSOM (tt)	Lt type 1 tympanoplasty	4/17/2012	15	5	5	5	15	5	10	15	15	10	10	15	15	10	10	20	15	10	10	20	P
19	Vivekanand	31	M	4/17/2012	8508	Rt CSOM(aa)	Rt MRM	4/18/2012	15	10	15	15	15	20	20	25	15	15	20	25	15	15	15	20	15	10	15	15	A
20	Ismail	15	M	4/18/2012	8594	Lt CSOM(tt)	Lt type 1 tympanoplasty	4/19/2012	5	0	0	5	5	0	10	5	5	0	0	0	5	0	0	0	5	0	5	0	A
21	Sudha hiramath	17	F	4/29/2012	9356	Rt CSOM (tt)	Rt Cortical type 1 tympanoplasty	4/30/2012	10	5	10	10	10	10	15	20	10	5	15	25	10	5	10	20	10	5	5	15	A
22	Uma Biradar	38	F	5/1/2012	9558	Lt CSOM(tt)	Lt type 1 tympanoplasty	5/2/2012	15	15	15	15	10	15	15	20	15	15	15	20	15	15	15	15	15	15	15	15	A
23	Sidamma	48	F	5/6/2012	9990	Lt CSOM (tt)	Lt Cortical type 1 tympanoplasty	5/7/2012	10	10	5	10	10	15	15	20	10	10	15	25	10	10	10	15	10	10	10	10	A
24	Parwati	26	F	5/9/2012	10146	Lt CSOM(tt)	Lt Cortical type 1 tympanoplasty	5/10/2012	0	5	5	0	0	5	20	15	0	5	20	20	0	5	15	10	0	5	5	5	A
25	Lakshmi	18	F	5/15/2012	10657	Lt CSOM(tt)	Lt type 1 tympanoplasty	5/16/2012	5	0	0	5	5	0	5	5	5	5	0	5	5	0	0	0	5	0	0	0	A
26	Shridevi Rathod	40	F	5/19/2012	11086	Lt CSOM (tt)	Lt type 1 tympanoplasty	5/21/2012	10	10	5	5	10	5	0	10	10	5	0	5	10	5	0	5	10	5	5	5	A
27	Pratiksha	6	F	5/29/2012	11912	Rt CSOM (tt)	Rt type 1 tympanoplasty	5/30/2012	5	0	5	5	5	0	0	0	0	0	0	0	5	5	0	0	0	0	0	0	A
28	Airavati Wadiyar	24	F	5/30/2012	11962	Lt CSOM(tt)	Lt Cortical type 1 tympanoplasty	5/31/2012	15	5	5	5	15	5	10	20	15	10	10	20	15	5	5	15	15	5	5	10	A
29	Kenchawwa	28	F	5/29/2012	11923	Rt CSOM (aa)	Rt Cortical type 3 tympanoplasty	6/1/2012	10	10	5	10	10	10	10	15	10	10	15	15	10	5	5	10	10	5	5	10	A
30	Sharanu	12	M	6/15/2012	13160	Rt CSOM(aa)	Rt MRM type3 tympanoplasty	6/16/2012	10	15	10	10	10	15	15	25	10	15	20	25	10	10	10	15	10	10	10	10	A
31	Vinod	16	M	6/19/2012	13459	Lt CSOM(tt)	Lt Cortical type 1 tympanoplasty	6/20/2012	15	10	10	10	10	10	15	10	10	10	10	10	10	5	10	10	10	10	10	10	A
32	Bhagyashree	15	F	6/21/2012	13686	Rt CSOM (tt)	Rt Cortical type 1 tympanoplasty	6/23/2012	5	5	5	10	5	10	15	15	5	5	15	15	5	5	10	10	5	10	5	10	A
33	Sangita	18	F	6/21/2012	13615	Rt CSOM (tt)	Rt Cortical type 1 tympanoplasty	6/22/2012	10	5	5	5	5	5	20	5	5	5	15	5	5	5	10	5	5	5	10	5	A
34	Basamma	17	F	6/25/2012	13951	Rt CSOM(tt)	Rt Cortical type 1 tympanoplasty	6/26/2012	0	5	0	0	0	5	0	5	0	0	0	0	0	0	0	0	0	0	0	0	A
35	Sangamma	17	F	6/29/2012	14253	Rt CSOM(tt)	Rt Cortical type 1 tympanoplasty	6/30/2012	10	10	15	5	10	15	15	10	10	10	15	5	10	10	10	5	10	10	10	5	A
36	Sheakh Modin	22	M	7/9/2012	15065	Lt CSOM (tt)	Lt type 1 Tplasty +Canalplasty	7/10/2012	10	5	5	10	5	5	5	15	10	5	5	10	10	5	5	10	10	5	5	10	A
37	Madevi	22	F	7/9/2012	1502	Lt CSOM(tt)	Left type 1 tympanoplasty	7/10/2012	10	5	5	5	5	5	10	5	5	5	5	5	5	5	5	5	5	5	5	5	A

					0																												
38	Rijwana Mujavar	21	F	7/10/2012	15112	Lt CSOM (tt)	Lt type 1 tympanoplasty	7/11/2012		5	5	0	10		5	10	5	20		5	5	5	15		5	5	5	10		5	5	5	10	A
39	Shridevi	19	F	7/14/2012	15493	Rt CSOM(aa)	Rt cortical type 3 tympanoplasty	7/16/2012		20	10	20	15		20	20	15	20		15	15	15	20		15	10	15	15		15	10	15	15	A
40	Sujata	21	F	7/16/2012	15615	B/L CSOM(tt)	Lt type 3 tympanoplasty	7/17/2012		10	5	5	5		10	10	5	10		10	5	5	5		10	5	0	5		10	5	0	5	A
41	Manjunath khed	20	M	7/25/2012	16337	B/L CSOM(tt)	Lt MRM type 3 tympanoplasty	7/26/2012		10	5	5	5		10	5	10	20		10	5	5	20		10	5	5	5		5	5	5	5	A
42	Satyma ashok	22	F	7/25/2012	16373	Lt CSOM(aa)	Lt MRM type 3 tympanoplasty	7/27/2012		10	15	10	10		10	15	15	25		10	15	10	10		10	15	10	10		10	10	10	10	A
43	Ravi	9	M	7/27/2012	16529	B/L CSOM(tt)	Lt type 1 tympanoplasty	7/28/2012		5	10	5	5		5	10	20	5		5	10	10	5		5	10	5	5		5	10	5	5	A
44	Shantawwa khed	22	F	7/16/2012	15661	Lt CSOM(tt)	Lt Cortical type 1 tympanoplasty	7/30/2012		0	0	5	0		5	0	0	5		5	0	5	5		0	0	0	0		0	0	0	0	A
45	Ratnappa	20	M	8/10/2012	17631	Rt CSOM (aa)	Rt MRM type 3 tympanoplasty	8/11/2012		10	15	15	10		10	20	25	20		10	15	20	20		10	10	15	15		10	10	15	10	A
46	Sharanappa B	29	M	8/24/2012	18837	B/L CSOM(tt)	Rt type 1 t plasty + Canalplasty	8/25/2012		5	5	5	10		5	5	15	5		5	5	5	5		5	5	5	5		5	5	5	5	A
47	Gurubai Hallad	35	F	8/31/2012	19487	Lt CSOM(tt)	Lt type 1 tympanoplasty	9/1/2012		0	5	5	0		0	5	5	5		0	5	0	0		0	5	0	0		0	5	0	0	A
48	Shivaram G	50	M	8/30/2012	19739	Lt CSOM(tt)	Lt type 1 tympanoplasty	9/5/2012		10	10	5	30		10	10	10	30		10	10	15	30		10	10	5	25		10	10	5	25	A
49	Pravin Patil	24	M	9/4/2012	19859	B/L CSOM(tt)	Rt type 1 tympanoplasty	9/5/2012		0	0	5	0		0	5	10	0		0	5	0	0		0	0	0	0		0	0	0	0	A
50	Sumabai Patil	49	F	9/4/2012	19831	Rt CSOM (tt)	Rt Cortical type 2 tympanoplasty	9/6/2012		10	10	15	15		10	10	15	10		15	15	10	25		10	15	10	15		10	15	10	15	A
51	Basalingawwa	50	F	9/8/2012	20222	Lt CSOM(tt)	Lt type 1 tympanoplasty	9/10/2012		15	15	15	15		15	15	25	25		15	15	20	25		15	15	15	15		15	15	15	15	A
52	Santosh Sajjan	24	M	9/19/2012	21148	Rt CSOM (tt)	Rt type 1 t plasty + Canalplasty	9/20/2012		15	10	5	10		15	10	15	15		15	5	10	15		10	5	10	15		10	5	10	15	A
53	Surekha P	25	F	9/21/2012	21313	Rt CSOM (tt)	Rt Cortical type 1 tympanoplasty	9/23/2012		5	10	5	5		5	10	5	15		5	10	5	15		5	10	5	10		5	10	5	10	A
54	Yallawa	50	F	9/23/2012	21453	Lt CSOM (tt)	Lt Cortical type 3 tympanoplasty	9/24/2012		10	15	20	20		10	15	20	30		10	15	15	25		10	15	15	25		10	15	15	25	A
55	Tahasim	17	F	10/5/2012	22590	B/L CSOM (tt)	Lt Cortical type 1 tympanoplasty	10/8/2012		5	5	5	5		5	10	5	15		5	5	5	15		5	5	5	10		5	5	0	5	A
56	Bandu Badiger	45	M	10/3/2012	22339	B/L CSOM(tt)	Rt type 1 tympanoplasty	10/9/2012		0	5	0	0		0	5	5	0		0	0	0	0		0	5	0	0		0	0	0	0	A
57	Shridevi S	12	F	10/1/2012	22098	Rt CSOM(tt)	Rt type 1 tympanoplasty	10/10/2012		10	10	15	10		10	10	0	5		10	10	5	5		10	5	5	10		10	5	5	10	A
58	Renuka Kannoli	16	F	10/17/2012	23684	B/L CSOM(aa)	Lt MRM type 3 tympanoplasty	10/18/2012		5	10	10	5		5	10	15	15		5	10	15	10		5	10	10	10		5	10	10	10	A
59	Nellawwa	35	F	10/18/2012	23695	Lt CSOM(tt)	Lt Cortical type 1 tympanoplasty	10/19/2012		10	5	10	10		10	10	15	20		10	5	15	25		10	5	10	25		10	5	10	20	P
60	Iramma	43	F	10/19/2012	23784	Rt CSOM(tt)	Rt type 1 tympanoplasty	10/20/2012		15	5	5	15		15	10	5	10		15	10	25	15		15	10	20	10		15	10	20	15	A
61	Gangabai joshi	42	F	10/26/2012	24321	Lt CSOM(aa)	Left MRM type 3 tympanoplasty	10/27/2012		10	10	15	15		10	15	30	20		10	15	25	30		10	15	15	30		10	15	15	30	P
62	Madev Karde	34	M	10/26/2012	24332	Lt CSOM(aa)	Lt MRM type 2 tympanoplasty	10/27/2012		10	10	10	10		10	15	15	10		10	15	15	15		10	10	10	15		10	15	10	10	A
63	Mahadevappa	26	M	10/28/2012	24482	Lt CSOM(aa)	Lt MRM type 3 tympanoplasty	10/29/2012		15	15	15	15		20	15	20	25		10	15	15	20		10	15	15	10		10	15	15	10	A
64	Sayabanna	6	M	10/28/2012	24511	Lt CSOM (tt)	Lt type 1 tympanoplasty	10/31/2012		10	10	5	5		10	10	10	5		10	10	10	5		10	5	5	5		10	10	5	5	A
65	Kalavati Karadi	26	F	10/31/2012	24759	Lt CSOM (tt)	Lt type 1 tympanoplasty	11/1/2012		10	5	5	5		10	5	5	5		10	10	5	5		10	0	0	0		10	0	0	0	A
66	Sanganna Sajjan	38	M	11/3/2012	25034	Rt CSOM (tt)	Rt type 1 tympanoplasty	11/5/2012		10	5	5	10		10	5	10	10		10	5	5	10		10	5	0	10		10	5	0	5	A
67	Kasturi	25	F	11/3/2012	25008	Rt COM (tt)	Rt type 3 t plasty+ atticotomy	11/5/2012		20	25	25	30		20	25	35	35		20	25	35	40		20	25	30	35		20	25	30	35	A
68	Priyanka Gadyal	18	F	11/10/2012	25709	Lt CSOM(tt)	Lt type 1 tympanoplasty	11/15/2012		15	5	5	5		10	5	5	5		10	5	5	5		10	0	0	5		5	0	0	5	A
69	Ramachandra	12	M	11/19/2012	26452	B/L CSOM (tt)	Rt cortical+ type 1 tympanoplasty	11/20/2012		0	0	0	0		0	0	5	10		0	0	0	10		0	0	0	0		0	0	0	0	A
70	Alfiya	10	F	11/23/2012	26987	Rt CSOM (tt)	Rt type 1 tympanoplasty	11/24/2012		5	10	10	15		5	10	15	15		5	10	10	15		5	10	5	15		5	10	5	15	A
71	Basavraj	19	M	11/28/2012	27576	B/L CSOM(tt)	Lt type 1 tympanoplasty	11/29/2012		20	20	15	0		20	25	25	5		20	20	15	5		20	20	15	0		20	20	15	0	A
72	Shivbasappa	30	M	11/28/2012	27577	B/L CSOM (tt)	Rt cortical+type 3 tympanoplasty	11/29/2012		0	0	5	0		0	5	5	10		0	0	10	10		0	0	5	0		0	0	5	0	A
73	Chandrakanth	25	M	12/1/2012	27876	Lt CSOM(tt)	Lt type 1 tympanoplasty	12/3/2012		0	5	5	0		0	5	10	5		0	5	5	5		0	0	0	0		0	0	5	0	A
74	Gurubai P	50	M	12/3/2012	27981	Lt CSOM(tt)	Lt type 1 tympanoplasty	12/4/2012		15	15	10	20		15	15	20	20		15	15	15	15		15	15	10	15		15	15	10	15	A
75	Geetawwa	15	F	12/5/2012	28445	B/L CSOM(aa)	Rt MRM type3 tympanoplasty	12/6/2012		15	10	5	10		15	10	15	25		15	10	10	25		15	10	10	15		15	10	10	15	A
76	Guru Mathapati	17	M	12/7/2012	28654	B/L CSOM(aa)	Lt MRM type 3 tympanoplasty	12/8/2012		0	0	0	0		0	0	0	10		0	0	0	0		0	0	0	0		0	0	0	0	A

77	Gurayya Ashok	17	M	12/7/2012	28690	B/L CSOM(aa)	Lt MRM type 3 tympanoplasty	12/8/2012		15	10	15	15		15	15	20	30		15	15	20	25		15	10	15	20		15	10	15	20	A
78	Shankerewwa	32	F	12/12/2012	29142	Lt CSOM(tt)	Lt Cortical+ type1 tympanoplasty	12/13/2012		5	5	10	5		10	25	25	30		15	15	10	10		5	5	10	0		5	5	10	0	A
79	Hirgappa	18	M	12/28/2012	30565	Lt CSOM(aa)	Lt MRM+ type4 tympanoplasty	12/29/2012		0	5	5	0		0	15	10	20		0	10	10	20		0	5	5	10		0	5	5	5	A
80	Siddamma	30	F	1/1/2013	36	Lt CSOM(tt)	Lt cortical+ type1 tympanoplasty	1/2/2013		5	5	10	10		5	10	15	20		5	5	15	20		5	5	10	10		5	5	10	10	A
81	Satish Shetti	13	M	1/3/2013	245	Rt CSOM (tt)	Rt cortical+ type 1 tympanoplasty	1/4/2013		0	0	0	5		0	5	5	10		0	5	0	5		0	5	0	0		0	5	0	5	A
82	Rafiq juned	47	M	1/6/2013	530	Lt CSOM(aa)	Lt MRM type 2 tympanoplasty	1/7/2013		10	15	15	20		10	15	25	35		10	15	20	30		10	10	15	25		10	10	15	25	A
83	Kallapa	30	M	1/6/2013	537	Lt CSOM(tt)	Lt Cortical+ type1 tympanoplasty	1/7/2013		10	15	10	10		10	10	15	10		10	15	10	10		10	15	10	10		10	15	10	10	A
84	Geeta Yadav	30	F	1/7/2013	626	Lt CSOM (tt)	Lt Cortical+type1 tympanoplasty	1/8/2013		0	0	0	0		0	5	10	15		0	0	15	15		0	0	0	5		0	0	0	0	A
85	Kannawa Chouri	18	F	1/9/2013	824	B/L CSOM (tt)	Lt type 1 tympanoplasty	1/10/2013		0	0	0	0		0	10	15	0		0	10	10	0		0	5	10	0		0	0	5	0	A
86	Sakkubai Talwar	27	F	1/10/2013	906	Lt CSOM (tt)	Lt cortical+ type3 tympanoplasty	1/15/2013		10	10	0	0		10	20	15	20		10	15	15	15		10	15	5	5		10	10	5	5	A
87	Sharanbasappa	10	M	1/15/2013	1308	Rt CSOM (aa)	Right MRM type3 tympanoplasty	1/16/2013		10	0	5	5		10	10	0	5		10	10	0	0		10	10	0	0		10	10	0	0	A
88	Ramappa rathod	34	M	1/15/2013	1350	Rt CSOM(tt)	Rt type 1 tympanoplasty	1/17/2013		10	5	15	20		10	15	25	20		10	15	25	20		10	10	15	20		10	5	15	20	A
89	Dundappa B	22	M	1/15/2013	1311	Lt CSOM (tt)	Lt Cortical+ type1 tympanoplasty	1/16/2013		10	10	15	15		10	15	20	30		10	10	15	25		10	10	15	20		10	10	15	20	A
90	Guru Madwale	25	M	1/17/2013	1513	Rt CSOM (tt)	Rt type 1 tympanoplasty	1/18/2013		0	0	0	0		0	0	10	5		0	0	5	5		0	0	0	5		0	0	0	0	A
91	Somashekar	18	M	1/17/2013	1506	Lf CSOM(tt)	Lt Cortical+ type1 tympanoplasty	1/18/2013		5	5	5	10		5	5	10	15		5	5	5	10		5	5	0	10		5	5	5	10	A
92	Somaraya	45	M	1/18/2013	1637	B/L CSOM(aa)	Lt MRM	1/21/2013		15	20	15	25		15	25	25	35		15	20	20	30		15	15	15	30		15	15	15	30	A
93	Mallikarjun	18	M	1/19/2013	1701	Lt CSOM(tt)	Lt Cortical+type1 tympanoplasty	1/21/2013		10	10	5	10		10	10	5	10		10	10	5	10		10	10	5	10		10	5	5	10	A
94	Mallapa Wadiar	21	M	1/19/2013	1703	Lt CSOM(tt)	Lt Cortical+type1 tympanoplasty	1/21/2013		5	10	5	5		5	10	10	10		5	10	5	15		5	10	5	15		5	10	5	15	P
95	Vani	30	F	1/22/2013	1925	Rt CSOM(tt)	Rt Cortical+type1 tympanoplasty	1/23/2013		10	15	20	15		10	15	25	25		10	15	20	25		10	15	20	20		10	15	20	20	A
96	Bharti Subas	19	F	1/25/2013	2266	Rt CSOM(tt)	Rt cortical+type 3 tympanoplasty	1/26/2013		15	5	10	0		10	5	20	0		10	5	20	0		10	5	10	0		10	5	10	0	A
97	Revati	18	F	1/25/2013	2231	Rt CSOM (tt)	Rt cortical+ type 1 tympanoplasty	1/26/2013		10	10	5	10		10	15	15	15		10	10	10	15		10	10	5	10		10	10	5	10	A
98	Mahantesh	25	M	1/29/2013	2570	Lt CSOM (tt)	Lt Cortical+ type1 tympanoplasty	1/30/2013		15	10	20	15		15	15	20	25		15	10	20	25		15	10	20	20		15	10	20	15	A
99	Chanagouda	30	M	1/29/2013	2572	Rt CSOM (tt)	Rt cortical+ type 1 tympanoplasty	1/30/2013		10	10	5	5		10	15	15	20		10	10	5	20		10	10	5	10		10	10	5	5	A
100	Mahadev Prabhu	25	M	1/29/2013	2574	Lt CSOM (tt)	Lt Cortical+ type1 tympanoplasty	1/30/2013		5	5	10	5		5	10	15	15		5	5	10	15		5	5	10	5		5	5	10	5	A
101	Parasuram	36	M	2/1/2013	2877	Lt CSOM (tt)	Lt Cortical+ type1 tympanoplasty	2/2/2013		10	10	15	10		10	15	15	25		10	15	15	25		10	10	15	15		10	10	15	15	A
102	Venkateshwar	30	M	2/1/2013	2874	Rt CSOM (tt)	Rt type 1 tympanoplasty	2/2/2013		10	10	15	15		10	15	20	15		10	10	15	15		10	10	15	15		10	10	15	15	A
103	Shobha	20	F	2/5/2013	3241	Lt CSOM (tt)	Left type 1 tympanoplasty	2/6/2013		15	10	20	15		15	15	35	35		15	15	35	40		15	10	25	25		15	10	20	25	P
104	Suresh	22	M	2/5/2013	3236	Rt CSOM (tt)	Rt cortical+ type 1 tympanoplasty	2/6/2013		5	10	10	5		5	10	20	20		5	10	15	20		5	10	10	10		5	10	10	5	A
105	Shankarappa	30	M	2/5/2013	3240	Lt CSOM (tt)	Lt Cortical+ type1 tympanoplasty	2/6/2013		10	10	20	10		10	15	25	20		10	10	20	20		10	10	20	10		10	10	20	10	A
106	Jatteppa	21	M	2/6/2013	3385	Rt CSOM(aa)+polyp	Rt MRM+type 3 tympanoplasty	2/7/2013		10	5	5	5		15	5	10	10		15	0	5	10		15	0	0	5		15	5	0	5	A
107	Vitthal	12	M	2/11/2013	3885	B/L CSOM(aa)	Lt MRM + type 3 tympanoplasty	2/12/2013		0	0	0	0		5	15	5	0		5	10	0	0		0	5	0	0		0	0	0	0	A
108	Shivamma T	26	F	2/12/2013	4000	Rt CSOM (tt)	Rt Cortical+type1 tympanoplasty	2/13/2013		5	0	0	0		5	0	10	0		5	0	10	0		5	0	0	5		0	0	0	0	A
109	Gururaj	20	M	2/12/2013	3972	Lt CSOM (tt)	Lt Cortical+ type1 tympanoplasty	2/13/2013		15	10	10	5		15	10	20	20		15	10	10	25		15	10	10	10		15	10	5	5	A
110	Padmavati	19	F	2/13/2013	4131	Lt CSOM (tt)	Left type 1 tympanoplasty	2/14/2013		0	0	0	0		0	5	0	0		0	5	0	0		0	0	0	0		0	0	0	0	A
111	Sadashiv	42	M	2/13/2013	4106	Rt CSOM (tt)	Rt Cortical type 1 tympanoplasty	2/14/2013		15	10	0	0		15	20	25	15		15	10	10	15		15	10	5	10		15	10	5	5	A
112	Saraswati	23	F	2/15/2013	4295	B/L CSOM (tt)	Rt type 1 tympanoplasty	2/16/2013		5	0	5	0		5	5	15	10		5	0	15	5		5	0	5	0		5	0	5	0	A
113	Priya Patil	18	F	2/15/2013	4324	Rt CSOM (tt)	Lt Cortical+ type1 tympanoplasty	2/16/2013		5	5	10	0		5	10	15	15		5	5	10	15		5	5	10	5		5	5	10	5	A
114	Rohini Tikoti	30	F	2/16/2013	4390	Rt CSOM (tt)	Rt type 1 tympanoplasty	2/18/2013		15	10	10	15		10	10	20	15		10	10	20	15		10	10	10	15		10	10	10	15	A
115	Mallamma Upase	45	F	2/18/2013	4600	Rt CSOM (aa)	Right MRM type3 tympanoplasty	2/19/2013		0	15	10	5		0	20	20	25		0	15	15	25		0	15	10	10		0	15	10	10	A
116	Shivannand	35	M	2/23/2013	5070	Rt CSOM (tt)	Lt Cortical+ type1 tympanoplasty	2/25/2013		5	5	5	0		5	5	10	10		5	5	5	5		5	5	5	0		5	0	5	0	A

117	Lata Gudadni	30	F	2/25/2013	5289	Rt CSOM (tt)	Rt type 1 tympanoplasty	2/26/2013		15	15	5	10		15	15	15	5		15	15	10	5		15	15	5	5		15	15	5	5	A
118	Shreedevi R	23	F	3/1/2013	5698	Lt CSOM (tt)	Lt Cortical+ type1 tympanoplasty	3/2/2013		5	5	5	5		0	0	5	0		0	0	5	5		5	0	0	0		0	0	0	0	A
119	Irappa Mudeapa	33	M	3/5/2013	6108	Lt CSOM (tt)	Lt Cortical+ type1 tympanoplasty	3/6/2013		15	10	15	20		15	15	25	30		15	10	15	25		15	10	15	20		15	10	10	20	A
120	Bouramma P	25	F	3/8/2013	6482	Rt CSOM (tt)	Rt type 1 tympanoplasty	3/9/2013		10	10	5	5		10	15	10	5		10	5	5	5		10	5	5	5		10	5	5	5	A
121	Shubham H	23	M	3/8/2013	6494	Lt CSOM (tt)	Lt Cortical+ type1 tympanoplasty	3/9/2013		5	10	10	5		5	15	15	15		5	10	10	15		5	10	10	10		5	10	10	10	A
122	Sachin B	27	M	3/9/2013	6551	Lt CSOM (tt)	Lt Cortical+ type1 tympanoplasty	3/11/2013		10	5	15	5		10	5	15	25		10	5	15	30		10	5	10	25		10	5	10	25	P
123	Shrikanth A	23	M	3/11/2013	6803	B/L CSOM (tt)	Left type 1 tympanoplasty	3/12/2013		0	0	0	0		0	5	10	0		0	5	5	0		0	5	0	0		0	5	0	0	A
124	Renuka Biradar	25	F	3/12/2013	6844	Rt CSOM (tt)	Rt cortical+ type 1 tympanoplasty	3/13/2013		5	0	5	0		10	10	15	10		5	5	5	10		5	5	0	5		0	0	0	0	A
125	Vijaykumar	20	M	3/12/2013	6832	B/L CSOM (tt)	Lt Cortical+ type1 tympanoplasty	3/13/2013		0	0	0	0		0	0	5	5		0	0	0	0		0	0	0	0		0	0	0	0	A
126	Laxmi bai	50	F	3/16/2013	7403	Lt CSOM (tt)	Lt cortical+ type3 tympanoplasty	3/18/2013		15	15	20	25		15	20	30	30		15	15	15	30		15	15	20	25		15	15	20	25	A
127	Sadashivappa	20	M	3/18/2013	7508	B/L CSOM (tt)	Lt type 1 tympanoplasty	3/19/2013		0	0	0	0		0	0	5	5		0	0	0	0		0	0	0	0		0	0	0	0	A
128	Somaray bibalagi	40	M	3/18/2013	7545	Rt CSOM (aa)	Rt MRM	3/20/2013		10	5	10	10		10	25	25	20		10	10	25	10		10	10	15	10		10	10	10	10	A
129	Ashok Mane	30	M	3/19/2013	7676	Rt CSOM (tt)	Rt type 1 tympanoplasty	3/20/2013		5	10	10	0		15	5	10	5		15	0	10	0		10	0	10	0		5	0	10	0	A
130	Mallikarjun	23	M	3/21/2013	7966	B/L CSOM(tt)	Left type 1 tympanoplasty	3/22/2013		10	5	5	5		10	10	15	5		10	5	5	5		10	5	5	5		10	5	5	5	A
131	Sagar	25	M	3/30/2013	8861	Lt CSOM (tt)	Lt Cortical+ type1 tympanoplasty	4/1/2013		10	10	0	5		15	15	10	15		10	10	5	15		10	10	0	10		10	5	0	5	A
132	Nayeem	28	M	4/2/2013	9155	Lt CSOM (tt)	Left type 1 tympanoplasty	4/3/2013		5	0	10	0		10	10	15	10		5	5	10	10		5	0	5	5		5	0	5	5	A
133	Bhuneshwari	17	F	4/4/2013	9359	Lt CSOM (tt)	Lt Cortical+ type1 tympanoplasty	4/5/2013		5	5	5	0		10	0	15	0		0	0	5	0		0	0	5	0		0	0	5	0	A
134	Jayashree	22	F	4/4/2013	9340	Rt COM (tt)	Rt cortical+ type 1 tympanoplasty	4/6/2013		5	0	5	0		10	10	20	10		5	5	20	10		5	0	15	5		5	0	10	10	P
135	Rafiq	25	M	4/26/2013	11574	Lt CSOM (tt)	Lt Cortical+ type1 tympanoplasty	4/27/2013		0	0	0	0		0	10	10	15		0	0	10	15		0	0	10	10		0	0	5	5	A
136	Savitha	21	F	5/8/2013	12645	B/L CSOM (tt)	Rt cortical+type 3 tympanoplasty	5/9/2013		0	5	0	0		0	5	10	0		0	5	5	0		0	5	0	0		0	5	0	0	A
137	Mallama Jinapur	30	F	5/9/2013	12667	Rt CSOM (tt)	Rt cortical+type 3 tympanoplasty	5/10/2013		5	5	5	0		5	5	15	10		5	5	5	5		5	5	5	5		5	0	0	5	A
138	Kaveri	20	F	5/13/2013	13035	Lt CSOM (tt)	Lt Cortical+ type1 tympanoplasty	5/14/2013		0	0	0	0		0	5	15	10		0	0	10	0		0	0	10	0		0	0	5	0	A
139	Devamma	25	F	5/15/2013	13299	B/L CSOM (tt)	Rt cortical+ type 1 tympanoplasty	5/16/2013		10	5	10	10		10	10	10	20		10	5	10	15		10	5	10	10		10	5	5	10	A
140	Asha Desai	32	F	5/16/2013	13343	Lt CSOM (tt)	Left type 1 tympanoplasty	5/17/2013		20	15	20	10		20	20	25	15		20	15	25	10		20	15	20	10		20	15	20	10	A
141	Rajeshwari A	13	F	5/17/2013	13447	B/L CSOM (tt)	Lt cortical+ type3 tympanoplasty	5/18/2013		10	5	5	5		10	5	15	5		10	5	10	5		10	5	15	5		5	5	15	5	A
142	Shivanand R	34	M	5/17/2013	13487	Lt CSOM (tt)	Lt Cortical+ type1 tympanoplasty	5/18/2013		15	10	5	5		10	10	10	5		10	10	5	5		10	10	5	5		10	10	5	5	A
143	Sharanppa B	21	M	5/18/2013	13598	Lt CSOM (tt)	Lt Cortical+ type1 tympanoplasty	5/20/2013		0	0	0	0		0	0	10	15		0	0	10	5		0	0	5	0		0	0	5	0	A
144	Anita S	35	F	5/21/2013	13891	Rt CSOM (tt)	Rt Cortical+type1 tympanoplasty	5/29/2013		5	15	10	15		15	15	25	25		15	10	20	25		10	10	20	20		10	10	15	20	A
145	Bharat Kumar	14	M	5/22/2013	13976	B/L CSOM (tt)	Lt Cortical+ type1 tympanoplasty	5/23/2013		0	0	0	0		0	5	0	0		0	0	0	0		0	0	0	0		0	0	0	0	A
146	Sushila Shenoor	25	F	5/27/2013	14470	B/L CSOM (tt)	Rt type 1 tympanoplasty	5/28/2013		15	10	0	0		15	10	5	5		10	10	5	0		10	10	0	0		10	10	0	0	A
147	Viresh	9	M	6/13/2013	15857	B/L CSOM (tt)	Lt cortical+ type3 tympanoplasty	6/14/2013		0	0	0	0		0	0	10	5		0	5	0	0		0	0	5	0		0	0	0	0	A
148	Sanabai	31	F	6/21/2013	17005	Lt CSOM (tt)	Lt Cortical+ type1 tympanoplasty	6/22/2013		10	10	15	5		10	15	15	15		10	10	10	20		10	10	15	10		10	10	15	10	A
149	Kashibai	30	F	6/21/2013	17021	Lt CSOM (tt)	Left type 1 tympanoplasty	6/22/2013		0	0	10	0		0	5	25	10		0	5	20	5		5	5	15	5		0	5	10	5	A
150	Savitha Balgari	18	F	6/22/2013	17092	Lt CSOM (tt)	Left type 1 tympanoplasty	6/24/2013		5	5	5	0		0	10	15	10		0	5	10	5		0	5	5	5		0	5	5	5	A
151	Tippawwa	40	F	6/25/2013	17346	Lt CSOM (tt)	Lt Cortical+ type1 tympanoplasty	6/26/2013		10	15	5	10		10	15	15	20		10	15	10	15		10	15	5	10		10	15	5	10	A
152	Julekha Sayaed	50	F	6/25/2013	17393	Lt CSOM (tt)	Lt Cortical+ type1 tympanoplasty	6/27/2013		25	15	10	15		25	20	25	30		20	15	20	25		25	15	15	25		25	15	15	25	P
153	Kavitha	17	F	7/1/2013	18014	Rt CSOM (tt)	Rt cortical+ type 1 tympanoplasty	7/3/2013		5	0	5	5		5	0	10	10		5	0	5	10		5	0	5	5		5	0	5	5	A