DISSERTATION ON

THE ROLE OF MIDDLE EAR RISK INDEX ON THE OUTCOME OF TYMPANOPLASTY

Dissertation submitted to

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

In partial fulfillment of the regulations

for the award of the degree of

M.S. IN OTORHINOLARYNGOLOGY

BRANCH – I

THANJAVUR MEDICAL COLLEGE,

THANJAVUR - 613 004

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

CHENNAI - 600 032

APRIL -2016

CERTIFICATE

I certify that the Dissertation titled THE ROLE OF MIDDLE EAR RISK INDEX ON THE OUTCOME OF TYMPANOPLASTY submitted by Dr.N.GITANJALI, for Degree of Master of Surgery (Otorhinolaryngology) to The Tamilnadu Dr.M.G.R. Medical University, Chennai is the result of original research work undertaken by her in the department of ENT AND HEAD & NECK SURGERY, Thanjavur Medical College, Thanjavur.

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DECLARATION

I hereby declare that the dissertation titled **THE ROLE OF MIDDLE EAR RISK INDEX ON THE OUTCOME OF TYMPANOPLASTY,** a clinical study submitted by me is a result of original work carried out by myself under the guidance of **Prof.Dr.T.Ramanathan, M.S.,D.L.O., Head of the Department Otorhinolaryngology and Head and Neck, Thanjavur** Medical College, Thanjavur. I further declare that the result of research has not been submitted previously by myself or other persons in any conferences or journals.

Dr.N.GITANJALI.

ACKNOWLEDGEMENTS

I thank The Dean, Prof ***Dr.Singaravelu M.D., DCH.,*** Thanjavur Medical College for permitting me to use the clinical material of this hospital for my study.

I express my deepest gratitude and heartfelt thanks to my mentor and guide ***Dr.T.Ramanathan,M.S.,D.L.O., The Professor and Head of the Department, Department of Otorhinolaryngology and Head and Neck Surgery, TMC, Thanjavur*** for his invaluable guidance, support and for his constant encouragement and inspiring views on this study. I consider it as discrete privilege to have had him as my guide. I shall always remain indebted to him.

My sincere thanks to ***Professor Dr.G.Gandhi M.S.,D.L.O., Department of Otorhinolaryngology and Head and Neck Surgery, TMC, Thanjavur*** for his blessings and constant support.

I am grateful to ***Dr.B.Ganesh kumar., M.S.,D.L.O., Senior Assistant Professor, Department of Otorhinolaryngology and Head and Neck Surgery, TMC, Thanjavur*** for his constant support and willingness to help all the times.

I am grateful to ***Dr. K. Ramesh Babu,M.S(ENT) Senior Assistant Professor, Department of Otorhinolaryngology and Head and Neck Surgery, TMC, Thanjavur*** for his constant support and encouragement for this study.

I thank ***Dr Amirthagani., M.S, (ENT)*** and ***Dr. PrincePeterDoss., M.S, (ENT) , Assistant Professors, Department of Otorhinolaryngology and Head &Neck Surgery*** for their strong motivation in my studies.

My gratitude goes to all of my colleagues and friends for their constant support and valuable help.

I would always be grateful and thankful to all the patients who took part in this study, without whom this effort would never have seen the light of the day.

I thank the almighty, my family and all the well wishers for guiding me in the right path all my life.

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...THE ROLE OF MIDDLE EAR RISK INDEX ON THE OUTCOME.....

...OF TYMPANOPLASTY.....

submitted by Dr. .....N. GITANJALI..... **of**

Dept. of .....ENT..... **Thanjavur Medical College, Thanjavur**

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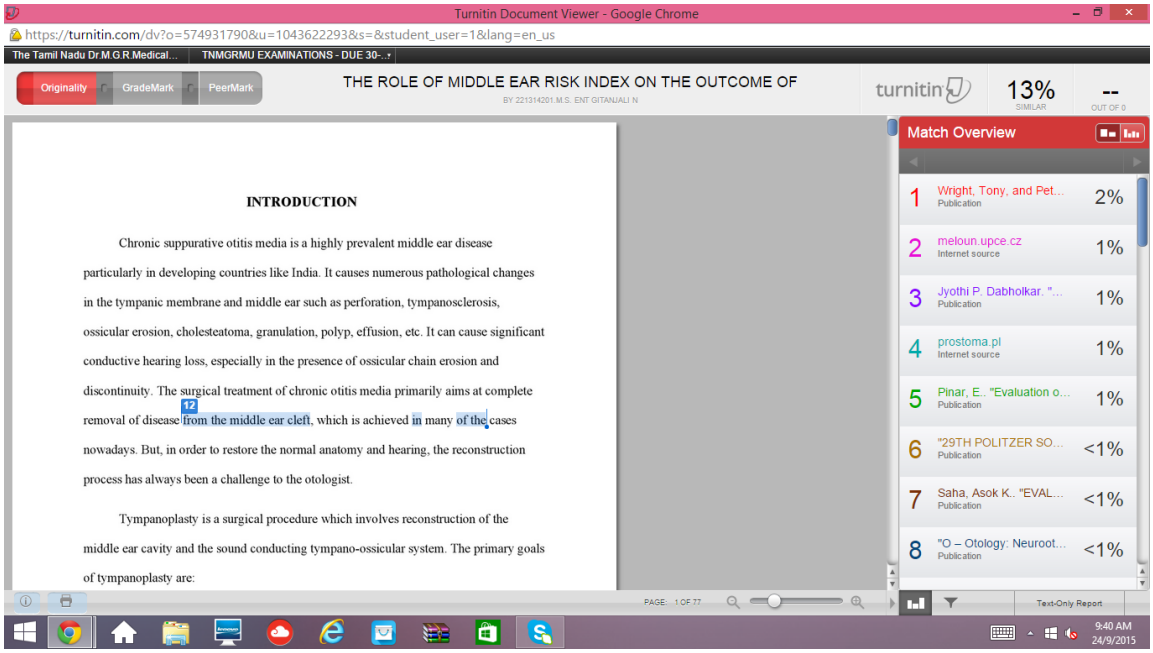
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INTRODUCTION
Chronic suppurative otitis media is a highly prevalent middle ear disease particularly in developing countries like India. It causes numerous pathological changes in the tympanic membrane and middle ear such as perforation, tympanosclerosis, ossicular erosion, cholesteatoma, granulation, polyp, effusion, etc. It can cause significant conductive hearing loss, especially in the presence of ossicular chain erosion and discontinuity. The surgical treatment of chronic otitis media primarily aims at complete removal of disease from the middle ear cleft, which is achieved in many of the cases nowadays. But, in order to restore the normal anatomy and hearing, the reconstruction process has always been a challenge to the otologist.
Tympanoplasty is a surgical procedure which involves reconstruction of the middle ear cavity and the sound conducting tympano-ossicular system. The primary goals of tympanoplasty are:
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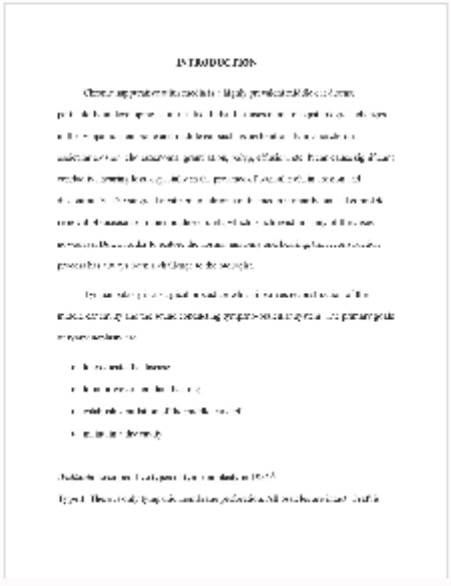
Submission title:	THE ROLE OF MIDDLE EAR RISK IN..
File name:	Full_dissertation.docx
File size:	547K
Page count:	77
Word count:	13,503
Character count:	73,482
Submission date:	24-Sep-2015 06:05PM
Submission ID:	574931790



INTRODUCTION

ABBREVIATIONS

- **CSOM -- CHRONIC SUPPURATIVE OTITIS MEDIA.**
- **MERI -- MIDDLE EAR RISK INDEX**
- **TT -- TUBO TYMPANIC.**
- **AA -- ATTICO ANTRAL.**
- **CT -- COMPUTERISED TOMOGRAM.**
- **HL -- HEARING LOSS.**
- **CHL -- CONDUCTIVE HEARING LOSS.**
- **SNHL -- SENSORINEURAL HEARING LOSS.**
- **A-B GAP -- AIR - BONE GAP**
- **DNE -- DIAGNOSTIC NASAL ENDOSCOPY.**
- **DNS -- DEVIATED NASAL SEPTUM.**
- **L -- LEFT SIDE.**
- **R -- RIGHT SIDE.**
- **MRM -- MODIFIED RADICAL MASTOIDECTOMY.**
- **PNS -- PARANASAL SINUS.**
- **FESS -- FUNCTIONAL ENDOSCOPIC SINUS SURGERY.**
- **B/L -- BILATERAL.**

- **M -- MALLEUS**
- **I -- INCUS**
- **S -- STAPES**
- **C/S -- CULTURE AND SENSITIVITY**

CONSENT FORM

I _____ hereby give consent to participate in the study conducted by **DR GITANJALI.N.,** Post graduate in the Department of Otorhinolaryngology, Thanjavur Medical College & Hospital, Thanjavur – 613004 and to use my personal clinical data and result of investigation for the purpose of analysis and to study the nature of disease. I also give consent for further investigations.

Place :

Date :

Signature of participant

CONTENTS

ABSTRACT

THE ROLE OF MIDDLE EAR RISK INDEX ON THE OUTCOME OF TYMPANOPLASTY.

AUTHOR: Prof. Dr. T.Ramanathan, **Dr. N.Gitanjali.**

Thanjavur Medical College, Thanjavur-613004.

AIMS AND OBJECTIVES: To determine the MERI score and categorise the patients into mild, moderate and severe MERI and study the relation between MERI and outcome of tympanoplasty.

BACKGROUND:

The results of tympanoplasty depend to a large extent on the severity of disease in the middle ear which is present preoperatively. The Middle Ear Risk Index combines these factors in the middle ear into a numerical value to assess the prognosis of tympanoplasty. The present study was done to assess the prognostic value of MERI index on the outcome of tympanoplasty.

MATERIALS AND METHODS:

The study consisted of 50 patients undergoing tympanoplasty for mucosal or squamous type of CSOM with hearing loss. The MERI score was

calculated. The patients were categorised into those with mild, moderate and severe MERI. The hearing benefit was calculated from the pre and post operative air-bone gap. The graft status was assessed. The relation between MERI score and graft status and between MERI score and hearing benefit was assessed by T test and Chi-Square test respectively.

RESULTS:

The overall graft uptake was 76%. Patients with a high MERI score have lower rate of graft uptake which is statistically significant with a P value of 0.031. Also, patients with mild MERI had greater hearing benefit and those with severe MERI had lesser hearing benefit postoperatively which is statistically significant with a P value of 0.041. The mean hearing benefit was 9.90 dB.

CONCLUSION:

MERI score is a prognostic tool to predict the outcome of tympanoplasty. It has an inverse relation with graft uptake and hearing benefit. Based on MERI score, the chances for surgical success and hearing benefit should be explained to the patient.

KEYWORDS: Middle Ear Risk Index. Tympanoplasty, Hearing benefit, Graft uptake.

INTRODUCTION

Chronic suppurative otitis media is a highly prevalent middle ear disease particularly in developing countries like India. It causes numerous pathological changes in the tympanic membrane and middle ear such as perforation, tympanosclerosis, ossicular erosion, cholesteatoma, granulation, polyp, effusion, etc. It can cause significant conductive hearing loss, especially in the presence of ossicular chain erosion and discontinuity. The surgical treatment of chronic otitis media primarily aims at complete removal of disease from the middle ear cleft, which is achieved in many of the cases nowadays. But, in order to restore the normal anatomy and hearing, the reconstruction process has always been a challenge to the otologist.

Tympanoplasty is a surgical procedure which involves reconstruction of the middle ear cavity and the sound conducting tympano-ossicular system. The primary goals of tympanoplasty are:

- to eradicate the disease
- to improve or maintain hearing
- establish ventilation of the middle ear cleft
- maintain a dry cavity

Wullstein described five types of tympanoplasty in 1956[1]:

Type 1: There is only tympanic membrane perforation. All ossicles are intact. Graft is placed in contact with the malleus handle. It is also known as myringoplasty.

Type 2: Malleus is eroded. The graft is placed over the incus (incudopexy) or remanant of malleus.

Type 3: Both malleus and incus are absent. The graft is placed over an intact mobile stapes. It is called *myringostapediopexy* or *columella tympanoplasty*. It produces a shallow middle ear and columella effect.

Type 4: Here suprastructure of the stapes is eroded, but the footplate is mobile. Here the footplate is left exposed to the sound waves and graft is placed to shield the round window. A small middle ear (cavum minor) is thus created.

Type 5: There is fixation of stapes footplate but with a functional round window. A window is created on the lateral semicircular canal and it is covered with a graft. It is called ***fenestration operation***. The sound waves reach the inner ear through the lateral semicircular canal.

Considerable research has been done and several materials have been used for the reconstruction of tympanic membrane and ossicular chain to improve the sound conduction and impedance matching. But optimal results depend not only on the reconstruction material but also the severity of the middle ear disease which is present preoperatively. Hence various factors in the middle ear cleft and tympanic membrane are used to assess the disease severity and thereby predict the outcome of surgery. ***Belluci*** developed a grading system based on the severity of ear discharge and associated craniofacial anomalies such as cleft palate. ***Wullstein*** and ***Austin*** proposed a five part system based on ossicular defects[2,3]. ***Black*** introduced the **SPITE** system (Surgical,

Prosthetic, Infection, Tissues and Eustachian tube). ***Kartush*** classified the factors into intrinsic (middle ear and Eustachian tube pathology) and extrinsic (the factors under surgeon's control such as surgical technique, the graft and prosthesis used)[4].

The Middle Ear Risk Index (MERI) was developed by ***Becvarovski and Kartush***. It combines the preoperative and intraoperative factors into a numerical value for assessing prognosis of tympanoplasty. The factors included are otorrhoea, tympanic membrane perforation, presence or absence of cholesteatoma, ossicular status, presence of middle ear granulation or effusion and history of previous middle ear surgery. Each patient is assigned a numerical score based on the risk factors. The total score is 12. Based on MERI score, the patients are classified as mild disease (1-3), moderate disease (4-6) and severe disease (7- 12). It was modified in 2001. Smoking was added as a risk factor[5]. The presence of cholesteatoma and granulation or effusions were given an additional score of 1 each. MERI score helps to predict the outcome of surgery in terms of success or failure. It helps intraoperatively to decide whether to proceed with canal wall up or canal wall down mastoidectomy and whether reconstruction can be done as primary or secondary reconstruction. It also helps to compare the hearing improvement by using different types of prosthesis in ossiculoplasty.

With increasing numbers of tympanoplasty procedures being performed nowadays, it is important to predict the outcome of surgery and give proper counseling for the patient. This avoids untoward expectations from the side of the patient. The aim of our study is to stratify the patients based on their MERI scores and assess the outcome of

tympanoplasty. Becvarovski and Kartush summarised their results based on otoscopic findings only ie, uptake or rejection of graft. But in our study, we have also evaluated the functional outcome (hearing benefit) by pure tone audiometry based on preoperative and postoperative air-bone gap, in addition to the graft uptake.

MIDDLE EAR RISK INDEX (2001)

RISK FACTOR	***FINDING***	***RISK VALUE***
Otorrhea	Dry	0
	Occasionally Wet	1
	Persistently Wet	2
	Wet with cleft palate	3
Perforation	Absent	0
	Present	1
Cholesteatoma	Absent	0
	Present	2
Ossicular Chain	Malleus, Incus and Stapes present	0
	Defect of Incus	1
	Defect of Incus and Stapes	2
	Defect of Incus and Malleus	3
	Defect of Malleus, Incus and Stapes	4
	Ossicular head fixation	2
	Stapes fixation	3
Middle ear Granulation / Effusion	No	0
	Yes	2
Previous surgery	None	0
	Staged	1
	Revision	2
Smoker	No	0
	Yes	2

AIMS AND OBJECTIVES

- To determine the Middle Ear Risk Index in patients with chronic suppurative otitis media undergoing tympanoplasty

- To categorise the patients into mild, moderate and severe disease based on MERI score

- To study the relation between MERI score and success of tympanoplasty

EMBRYOLOGY AND ANATOMY

EMBRYOLOGY OF MIDDLE EAR CLEFT AND TYMPANIC MEMBRANE

The three layers of the tympanic membrane develop from the following:

1. The outer epithelial layer from the ectoderm of first branchial cleft

2. The middle fibrous layer from the mesoderm between the first branchial cleft and the tubotympanic recess

3. The inner mucosal layer from the endoderm of tubotympanic recess

The middle ear cleft develops from the endoderm of tubotympanic recess, which develops from first pharyngeal pouch. The Eustachian tube develops from the primitive pharynx during the second month. The development of *Meckel's cartilage* begins around sixth week of embryonic life. It develops from the mesoderm of first branchial arch. It gives rise to malleus and incus. *Reichert's cartilage* develops from the of second branchial arch and gives rise to the head, neck and crura of stapes. The stapes footplate has a dual origin. The outer layer develops from Reichert's cartilage and the inner endosteal layer develops from the otic capsule. Initially the middle ear cavity is filled with mesenchyme and the ossicles are embedded in it. This mesernchyme gradually gets absorbed during the later part of fetal life.

From the Eustachian tube, an endoderm lined pouch grows into the middle ear cleft. From this four sacs, *saccus anticus, saccus posticus, saccus medius* and *saccus*

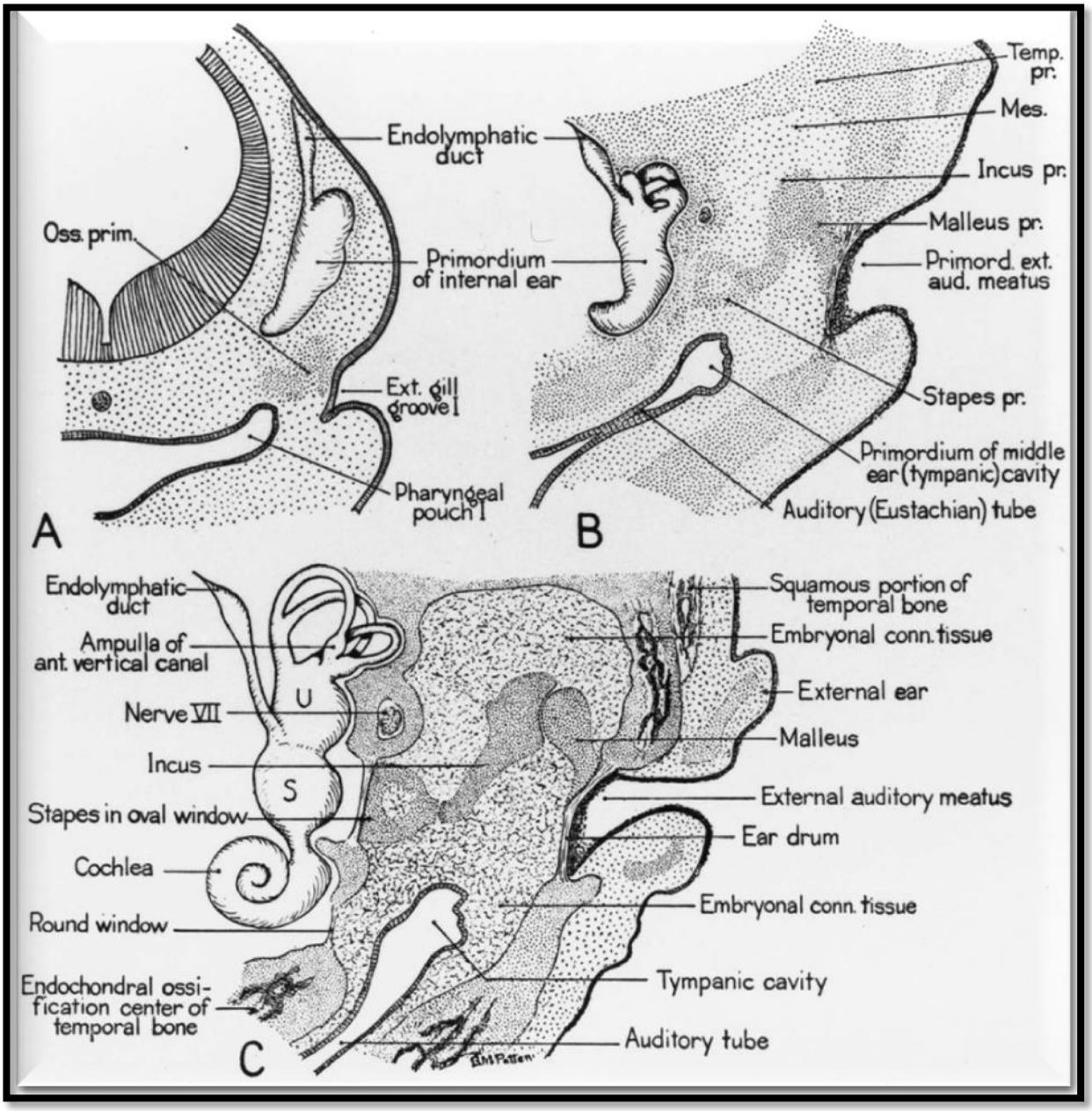
superior are seen to arise. The middle ear mucosal folds develop where the pouches contact each other. The mesodermal remanants in between these folds form the blood vessels. The saccus anticus gives rise to the anterior pouch of von Troltsch. The saccus superior gives rise to the posterior pouch of von Troltsch and inferior incudal space. The attic develops from the saccus medius. The saccus posticus gives rise to round window niche, oval window niche and sinus tympani.

The development of these sacci help to form a tympanic diaphragm in the middle ear cleft which separates the middle ear into anteroinferior and posterosuperior parts. They also lead to the development of two different types of epithelium, both of which have specific functions, in anteroinferior and posterosuperior compartments.

The temporal bone develops from the following:

- petromastoid
- squama
- ostympanicum
- styloid process

The petromastoid gives rise to petrous apex, tegmen tympani, lateral wall of eustachian tube and floor of midle ear. The squamous part gives rise to lateral wall of mastoid antrum. The os tympanicum give rise to external auditory meatus and tympanic sulcus. The styloid process develops from two centres in Reichert's cartilage. The prominence of mastoid process which develops from petrous and squamous parts can be identified from second year after birth.


Endolymphatic duct
Oss. prim.
Primordium of internal ear
Ext. gill groove I
Pharyngeal pouch I
A
Temp. pr.
Mes.
Incus pr.
Malleus pr.
Primord. ext. aud. meatus
Stapes pr.
Primordium of middle ear (tympanic) cavity
Auditory (Eustachian) tube
B
Endolymphatic duct
Ampulla of ant. vertical canal
Nerve VII
Incus
Stapes in oval window
Cochlea
Round window
Endochondral ossification center of temporal bone
U
S
C
Squamous portion of temporal bone
Embryonal conn. tissue
External ear
Malleus
External auditory meatus
Ear drum
Embryonal conn. tissue
Tympanic cavity
Auditory tube

ANATOMY

MIDDLE EAR CLEFT

The following structures constitute the middle ear cleft:

- The tympanic cavity
- Pharyngotympanic (eustachian) tube
- Aditus
- Mastoid antrum
- The other air cells comprising the pneumatic system of temporal bone.

HISTOLOGY

The mucosal lining is respiratory epithelium. The cells in the middle ear are predominantly cuboidal, 2 or 3 layered. Ciliated columnar epithelium is found in the Eustachian tube and anteroinferior part of the tympanic cavity. Here there is pseudostratified epithelium with underlying dense connective tissue. Its main function is clearance of secretions via the Eustachian tube. Posteriorly and superiorly, the epithelium is flattened and single layered. The underlying

connective tissue is loose. The distance between the blood vessels and basement membrane of the mucosa is lesser in posterosuperior part than anteroinferior part. Posterosuperior part is mainly devised for the function of gas exchange[6].

TYMPANIC CAVITY

It is an air filled, biconcave disc shaped space within the temporal bone between

the external ear and the labyrinth. It measures 15mm superoinferiorly, 13mm anteroposteriorly and transversely it is 4mm in lower part, 2mm in the centre and 6mm in the upper part. It is divided into three regions - the attic or epitympanum above the level of anterior and posterior malleolar folds, the hypotympanum below the level of lower part of tympanic sulcus and the mesotympanum between the above two. The protympanum is the region around the Eustachian tube orifice in the anterior wall.

TYMPANIC MEMBRANE

The tympanic membrane which forms the lateral wall is an oblique disc placed at an angle of 55° with the floor. It measures 8×10mm. Umbo is the point of maximum convexity (towards the middle ear). The two cell layered outer epithelium is continuous with that of external auditory canal and has centrifugal migratory property. The fibrous layer in pars tensa has stratum radiatum directed from malleus handle towards annulus and stratum circulare arranged concentrically. The fibres in pars flaccida are randomly arranged. This layer has numerous capillaries and nerves passing through it. The one layered inner mucosal layer is continuous with the mucosa of tympanic cavity.

The fibrocartilaginous annulus (***Gerlach's ligament***) fits into the tympanic sulcus, which is lacking superiorly at the notch of Rivinus. In addition, the ear drum attached firmly to the lateral process of malleus and at the umbo. Above the Shrapnel's membrane is the scutum or outer attic wall which is easily eroded by attic cholesteatoma.

WALLS OF THE MIDDLE EAR

The lateral wall is formed by

- the bony lateral attic wall superiorly
- lateral wall of hypotympanum inferiorly
- the tympanic membrane between the above two.

Scutum is the inferior part of the lateral wall of the epitympanum. It is thin and easily eroded by attic cholesteatoma.

The tegmen tympani, forming the roof of middle ear separates it from the middle cranial fossa dura. It is formed by petrous and squamous parts of temporal bone. The suture line between these two parts carries veins to superior petrosal sinus and can serve as a pathway of spread to middle cranial fossa. The *jugular wall* or floor separates the hypotympanum from the dome of the jugular bulb. ***Jacobson's nerve*** enters through the inferior tympanic canaliculus at the junction of floor and medial wall.

The *medial or labyrinthine wall* forms the surgical floor of middle ear. The promontary, the bulge produced by the basal turn of cochlea, occupies much of the central part of medial wall. The promontary has grooves on its surface for the tympanic plexus and tympanic branch of glossopharyngeal nerve. Posterosuperior to the promontary is the kidney shaped fenestra ovale whose niche measures 3.25×1.75×3mm[7]. It opens into the vestibule of labyrinth and is closed by the stapes footplate and the annular ligament. Posteroinferior to the promontary is the triangular fenestra rotundum whose niche faces posteriorly and measures 2.3×1.9mm. It communicates with scala tympani at the basal turn of cochlea. It is at right angles to oval window and is well protected by the promontary. It is closed by secondary tympanic membrane.

Subiculum is the bony ridge extending from the promontary which separates the two windows. *Ponticulus* is another ridge of bone which runs from the medial towards the posterior wall of the tympani cavity. The ***sinus tympani*** is a deep recess between the ponticulus and the subiculum. Lateral to the sinus tympani is the facial recess in the posterior wall. Fallopian canal runs between the two recesses. Superior to the oval window lies the tympanic portion of facial nerve and further above it lies the dome of horizontal semicircular canal. The anterior end of tympanic segment of facial nerve is marked by processus cochleariformis, where the tendon of tensor tympani takes a turn.

The ***mastoid wall (posterior wall)*** has an opening, aditus ad antrum in its upper part which is the communication between posterior epitympanum and mastoid antrum. Inferior to aditus lies a small depression, the fossa incudis lodging the short process of incus along with its suspensory ligament. Further below lies a conical projection called the pyramid. The tendon of stapedius emerges from its apex, which points anteriorly. Posterior to the pyramid runs the mastoid segment of facial nerve.

The anterior wall is narrow due to the convergence of medial and lateral walls. It is known as ***carotid wall*** because it is separated by a thin plate of bone from the carotid artery in the lower third. The sympathetic plexus around the carotid artery carried by the superior and inferior caroticotympanic nerves as well as tympanic branch of internal carotid artery perforate this wall at its lower third. The middle third presents the oval Eustachian tube orifice. The canal for tensor tympani lies superior to Eustachian tube orifice. The anterior epitympanic sinus, a site of residual cholesteatoma, is situated

in the pneumatised upper one third. The chorda tympani nerve exits at the canal of ***Huguier***, which is located at the medial end of petrotympanic fissure after passing between the fibrous and mucosal layers of tympanic membrane. The 2mm long petrotympanic fissure ***(Glaserian fissure)*** transmits the anterior tympanic branch of maxillary artery and anterior ligament of malleus.

CONTENTS OF MIDDLE EAR

The tympanic cavity contains air, the three ossicles, tensor tympani and stapedius muscles, chorda tympani and tympanic plexus of nerves.

The air in the middle ear is nitrogen rich and is supplied by the Eustachian tube.

OSSICLES

The ***malleus***, the largest ossicle, is about 9mm long and is *hammer* shaped. It has head or capitulum, neck, manubrium or handle, anterior and lateral processes. The capitulum articulates with the body of incus on its posteromedial aspect forming a synovial joint in the attic. The lateral process has a cartilaginous cap and gives attachment to anterior and posterior malleolar folds. The manubrium is directed downwards, medially and backwards and runs between the fibrous layer and mucosal layer of tympanic membrane. The malleus is suspended by the anterior ligament which is attached to the anterior tympanic wall, superior ligament attached to tegmen, lateral ligament attached to the Notch of Rivinus.

The *anvil* shaped ***incus*** has a body and short process which lie in the attic, a long process and lenticular process (known as the fourth ossicle due to incomplete ossification), which articulates with the head of stapes. The fossa incudis lodges the short

process. ***Stapes***, the smallest ossicle, is *stirrup* shaped. It has head, neck, anterior and posterior crura and footplate. The posterior surface of neck receives insertion of stapedius tendon. The posterior crus is thicker and more curved than the anterior crus. The footplate, which is horizontally placed, is attached to the bony margins of the fenestra vestibuli by the annular or stapediovestibular ligament.

MUSCLES

The bipennate *tensor tympani* muscle originates from the walls of eustachian tube and greater wing of sphenoid passes backwards and is inserted just below the neck of malleus. It is supplied by medial pterygoid branch of mandibular nerve. The bulky *stapedius* arises from the cavity within the pyramid and the tendon, coming out of its apex, is inserted into neck of stapes. It is supplied by the facial nerve.

MIDDLE EAR MUCOSA

The mucosa of middle ear is thrown into folds which stretch across the tympanic cavity. The carry blood vessels to various stuctures. They almost completely separate the mesotympanum from the epitympanum except at ***isthmus tympani anticus*** and ***isthmus tympani posticus***. Thus the ventilation of attic is through the isthmus tympani. Attic is divided into a larger posterior and smaller anterior compartment by superior malleolar fold. The tensor tympani fold lies between the anterior attic space and anterior mesotympanum and prevents spread of cholesteatoma across these. The posterior compartment is divided into medial and lateral compartments (medial and superior incudal space respectively) by superior incudal fold. Lateral incudal fold prevents spread of disease from posterosuperior marginal perforation into attic. ***Prussak's space*** is bound

by pars flaccida, neck of malleus and lateral malleolar fold. It is the site of formation of primary acquired cholesteatoma.

BLOOD SUPPLY

The malleus and incus are supplied by malleolar and incudal arteries respectively, which are branches of anterior tympanic artery. The stapes is supplied by the vascular plexus in promontary and from facial canal. The *incudo stapedial joint* is supplied by three groups of vessels which anastamose here - vessels which descend down the incus, vessels which pass from mucosa covering stapedius tendon and those that climb up the stapes crura. Because of tenuous blood supply, the tip of long process of incus is more prone for osteonecrosis.

MASTOID

The ***mastoid antrum*** is the largest and the most constant mastoid air cell located in the petrous part of temporal bone. It is ventilated from the middle ear through aditus ad antrum. It has a volume of 2ml. Its roof is formed by tegmen antri, which separates it from middle cranial fossa. Medially, it is related to posterior semicircular canal. It is well developed at birth. The groove for posterior belly of digastric is located in the base of mastoid process. The digastric ridge divides the tip cells into medial and lateral tip cells. It is also a landmark for facial nerve as the nerve exits at the stylomastoid foramen at the anterior end of this ridge. The surface landmark for antrum, the ***Mac Ewen's or suprameatal triangle*** is bound by suprameatal crest, posterosuperior canal wall and a tangential line drawn from the suprameatal crest which cuts the second line. The antrum is lined by a layer of flattened epithelium, devoid of

cilia, goblet cells or mucous glands.

The mastoid air cells are numerous, extending upto mastoid tip inferiorly, retrofacial region medially, sinodural angle posteriorly, and anteriorly upto petrous apex and arch of zygoma. They are perilabyrinthine (supralabyrinthine and infralabyrinthine) cells, peritubal cells, retrofacial cells, perisinus cells, zygomatic cells, tegmen cells, squamous cells, medial and lateral tip cells, petrous cells and marginal cells. These air cells are not well aerated in diseased individuals. *Pneumatisation* of temporal bone begins with the entry of air at birth through the eustachian tube. This occurs by resorption of mesenchyme and haematopoietic bone marrow. According to the type of pneumatisation, the mastoid can be cellular, diploeic or sclerotic. Pneumatisation of petrous apex begins after a few years and continues into early adult life. The extent of pneumatisation depends on heredity, environment, nutrition, infection and eustachian tube function. The petrous apex is the most inaccesible area sugically.

EUSTACHIAN TUBE

The ***pharyngotympanic or eustachian tube***, which is 36mm long, connects the middle ear with the nasopharynx. It runs through the petrous and squamous parts of temporal bone. The main function is ventilation of the middle ear cleft. It has a cartilaginous (medial two third) part and a bony (lateral one third) part. The junction of these two parts, the isthmus is the narrowest region, where the diameter is 0.5mm. The bony part is directed anteromedially and is triangular in cross section. It opens into the anterior wall of the middle ear. It further has a posterolateral labyrinthine part and anteromedial carotid part. The cartilaginous part opens into the lateral wall of

nasopharynx. It has dynamic movement[8]. Ostmann pad of fat in inferolateral aspect prevents excess opening of the tube. The lining epithelium is respiratory, with the number of cilia and goblet cells reducing near the middle ear end.

PHYSIOLOGY

The sound conducting system extends from the pinna to the organ of corti. The pinna acts as a sound collector and the external auditory canal transmits it to the drumhead. The middle ear helps in impedance matching between the air in the external auditory canal and cochlear fluids. It transmits sound preferentially to the oval window thereby maintaining phase difference between the oval and round windows. This is necessary for movement of cochlear fluids. It also protects the cochlea from loud sounds.

ROLE OF TYMPANIC MEMBRANE

The radial and circular fibres of the middle fibrous layer are arranged in a complex manner. Though the membrane has concavity towards the external auditory canal due to pull of malleus at the umbo, it has convexity in each segment from annulus to the malleus handle. Hence there is ***buckling of the membrane*** in response to sound. This in turn helps in *impedance matching* when the sound is absorbed by the middle fibrous layer and transmitted to the handle of malleus. This improves the impedance value by a factor of 4 and increases the force of transmission[9]. The intact tympanic membrane also ensures preferential conduction of sound to the oval window through the ossicular chain. At frequencies above 6 kHz, there is reduction in transmission as the vibration breaks up into multiple zones.

OSSICULAR CHAIN

The air in middle ear offers a low friction environment for the vibration of ossicles. The ligaments suspending the malleus and incus reduces their mass and inertia. The malleus and incus vibrate together as a single unit. The axis of vibration runs from the anterior ligament of malleus to the short process of incus. The mass of the structures in the attic (head and neck of malleus and body of incus) balances the other structures below (tympanic membrane, malleus handle, long process of incus and stapes). The stapes moves like a *piston* in and out of the oval window. The movement varies with different intensities. The annular ligament fibres are longer at the anterior than posterior part. Hence anterior part of the footplate has greater amplitude of vibration than the posterior end. At higher intensities, there is side to side rocking movement. Thus the amount of displacement of labyrinthine fluids is lesser in the latter mode than the former. This in turn is a mechanism to protect the labyrinth against high intensity sounds.

During evolution, the transition from aquatic to terrestial life necessitated an apparatus to overcome the difference in impedance between air and perilymph. Much of the sound waves will be reflected at the interface between if the two media have different impedances. This causes loss of sound energy. The impedance of air is 430 $N \cdot s/m^3$. The impedance of cochlear fluids is 1.5 $N \cdot s/m^3$ at 1kHz. this should cause a loss of 60 dB. This is prevented by the presence of a mobile drumhead, an air filled middle ear and the arrangement of the ossicular chain, which helps in impedance matching.

TRANSFORMER MECHANISM OF MIDDLE EAR

This ensures that, when the amplitude is reduced as sound is transmitted from the drumhead to the oval window, the force of vibration is increased in the same proportion. It is achieved by:

1. ***The lever ratio:***

The malleus and incus vibrate together as a single unit like a lever pivoting upon the axis of rotation. The handle of malleus is longer than the long process of incus. Thus the geometrical length of malleus to incus is 2.1:1. This increases the impedance by 4.4times.

2.***The areal ratio***:

The area of the tympanic membrane is 60mm^2 while the area of the footplate of stapes is 3.2mm^2. Hence the pressure on the footplate is increased by 18.75 times.

This calculation is done for frequency of 1kHz. The sound transmission is lower at high and low frequencies. At low frequencies, the movement of the tympanic membrane and footplate is reduced. At higher frequencies, the vibration of tympanic membrane breaks up into multiple zones. Also, the effective area of vibration of tympanic membrane is reduced. A relative motion develops between the malleus handle and long process of incus. This reduces the lever ratio.

INTRATYMPANIC MUSCLES

High intensity sounds (90dB above threshold) causes reflex contraction of tensor tympani and stapedius and reduces sound transmission to protect the inner ear. The reflex arc travels through the cochlear nuclei to the superior olivary body and then to motor nuclei of fifth and seventh cranial nerves. The stapedius pulls stapes away from oval window and tensor tympani pulls the drumhead inwards. This reflex stiffens the middle ear mechanism and dampens hearing. The intensity of stimulus is lower for ipsilateral stimuli than contralateral stimuli. The threshold is least for bilateral stimuli. The stapedius is more significant compared to tensor tympani. The stapedius has a latency period of 10ms.

The ***acoustic reflex*** is an effective attenuator especially for lower frequencies, which cause more damage to the cochlea. It also helps in selectively augmenting the middle and high speech frequencies by masking the low frequency noise. The muscles also support the ossicles and stabilise them. Tensor tympani contraction is said to cause Eustachian tube opening.

PHASE DIFFERENCE

For effective movement of hair cells, the oval and round windows must vibrate in reciprocal phases to each other. The sound is preferentially conducted to the oval window through the ossicles. The round window acts as a relieving point and only negligible part of the sound reaches the round window directly.

BONE CONDUCTION

Hearing by bone conduction is by the following mechanisms

1. *Inertial or translatory mechanism:*

Sound waves cause vibration of skull bones. But due to inertia, the three ossicles lag behind the skull in vibration. This causes vibration of stapes footplate with respect to the oval window.

2. *Compressional mechanism*:

Vibration of skull in response to sound causes vibration of the bony labyrinth. The alternate compression and decompression causes movement of cochlear fluids.

3. *Effect of mandible:*

Mandible due to its inertia, lags behind the vibrations of skull. The head of the mandible produces vibrations in the external auditory canal. These are further transmitted by the air conduction route to the cochlea.

At low frequencies, the skull bones vibrate as a whole. The inertial mechanism mainly accounts for hearing. At higher frequencies, there is bending of skull bones. Hence compressional mechanism plays a significant role at high frequencies.

ACOUSTIC COUPLING

Usually the sound is transmitted by the ***tympano-ossicular system*** to the inner ear. This is termed ossicular coupling. The movement of tympanic membrane creates a sound pressure within the middle ear cavity. Due to spatial separation of the oval and round windows, their sound pressures are not the same. This causes a small measurable difference in sound pressures at the two windows which causes movement of labyrinthine fluids. This is called acoustic coupling. This is not significant in normal individuals. But it contributes to hearing when the ossicular coupling is affected.

JOINTS IN MIDDLE EAR

The two joints between the ossicles, the incudomalleal and the incudostapedial joints increase flexibility of ossicles helping them to withstand large pressure differences across the drumhead preventing damage to the ear. They also allow independent control of ossicles. The stapedius contraction causing movements of stapes has little effect on the other ossicles due to incudostapedial joint. Similarly, the tensor tympani has little effect on malleus due to incudomalleal joint.

PATHOPHYSIOLOGY

A variety of pathological changes occur in the tympanic membrane and middle ear as a result of chronic suppurative otitis media such as tympanic membrane perforation, retraction pocket, cholesteatoma, granulation, middle ear effusion, ossicular necrosis, polyps, etc.

MIDDLE EAR MUCOSA

There is thickening of the mucosa due to edema, submucous fibrosis and infiltration with inflammatory cells. The height of ciliated cells and number of *mucous secreting cells* increases with more mucous glands in submucosa. Thus the thick mucus plugs the Eustachian tube orifice which leads to negative pressure and retraction pockets. Also, the inflammatory process reduces the distance between the blood vessels and the mucosa further due to an increase in both the number and size of the blood vessels in the posterosuperior part than anteroinferior part. Hence typanosclerosis is common in anteroinferior part.

PERFORATION

Perforations arise secondary to acute otitis media or trauma. Sometimes deepening of retraction pocket and contact with edematous mucosa or granulation tissue can cause perforation. In recurrent attacks of acute otitis media, the tympanic membrane undergoes necrosis due to ischaemia secondary to endarteritis. Also, enzymes in granulation tissue break down collagen in the tympanic membrane increasing the size of

perforation. There are several factors which prevent healing of the perforation. If the patient has persistent infection and discharge, the perforation serves as the drainage pathway of the discharge. Eustachian tube obstruction prevents closure of the perforation. Too large perforations cannot close spontaneously by ingrowth of cells from the margins of the perforation. The squamous epithelium on the lateral aspect can grow medially. Once it is epithelialised completely, it does not close and becomes a permanent perforation.

A dry perforation can occur in pars tensa or pars flaccida. The middle ear mucosa is normal. The margins of perforation may be epithelialised or thickened due to proliferation of fibrous tissue. Sometimes the epithelial cells in the margin of the perforation can migrate into middle ear and cause formation of cholesteatoma. Also, a perforation in pars flaccida is always associated with cholesteatoma. If a polyp or granulation tissue protrudes from attic perforation, it indicates the presence of an infected cholesteatoma.

EAR DISCHARGE

The discharge can be continuous or intermittent. Entry of water through the perforation or respiratory infections can increase the amount of discharge. Persistent discharge which does not reduce after antibiotic therapy may be due to mastoid reservoir with inflammation of the entire middle ear cleft. A wide variety of Gram positive and Gram negative organisms are implicated in CSOM. The most commonly isolated organisms are Pseudomonas aeruginosa, Proteus and Staphylococcus aureus. The usual

portal of entry is perforation in tympanic membrane. The mucus secreted by middle ear serves as a culture medium for the bacteria. In patients with craniofacial anomalies such as cleft palate, the discharge is persistent. In such patients, the tensor veli palatini muscle is not well developed. This causes Eustachian tube dysfunction leading to infection.

RETRACTION POCKETS

Retraction pockets develop due to negative pressure in middle ear secondary to eustachian tube dysfunction. The chronic inflammation process can lead to adhesions between the retraction pocket and middle ear structures such as ossicles, promontary, etc. This results in adhesive otitis media. They also lead to cholesteatoma formation. Retraction pockets are common in the posterosuperior quadrant. The ventilation of attic is only through isthmus tympani anticus and isthmus tympani posticus. When one of these is blocked, retraction pocket arises from the posterosuperior part of the pars tensa. When both are blocked, retraction pocket arises from pars flaccida. Hence an *atticotomy* should be done in case of impaired ventilation to the posterosuperior part. In some cases, the middle ear mucosa is replaced by keratinising squamous epithelium but there is no accumulation of keratin debris. However it does not lead to cholesteatoma formation and is not an indication for surgery. This is called ***Epidermization***. It is an advanced type of retraction.

CHOLESTEATOMA

Choesteatoma is accumulation of trapped squamous epithelium along with the

desquamated keratin debris. Its central core consists of keratin material. The matrix is formed by squamous epithelium. The subepithelial connective tissue surrounding it has inflammatory cells, fibroblasts and blood vessels. It has a tendency to erode the surrounding structures most commonly the ossicles and scutum. It damages the lining mucosa and causes inflammatory cell infiltration with osteoclastic bone resorption. The enlarging cholesteatoma exerts pressure on bone leading to ischemia and erosion. ***Bacterial biofilms*** are implicated in cholesteatoma formation. The bacterial cell wall has *lipopolysaccharide*, which triggers osteoclastic proliferation. Hence cholesteatoma with infection causes more ossicular necrosis than cholesteatoma without infection.

Cholesteatoma is classified as congenital and acquired cholesteatoma. Acquired cholesteatoma is further classified as primary and secondary. Primary acquired cholesteatoma is not associated with middle ear infection. Secondary

acquired cholesteatoma is associated with infection of the middle ear cleft.

There are various theories of formation of aquired cholesteatoma. ***Metaplasia theory of Sade*** states that cholesteatoma is due to squamous metaplasia of middle ear mucosal epithelium. ***Immigration theory*** states that squamous epithelium from the edge of perforation migrates into the middle ear. ***Basal hyperplasia*** theory proposed by Ruedi states that, due to inflammation, activity of basal cells increases and this cellular proliferation breaks past the intact tympanic membrane into the middle ear. Iatrogenic or ***implantation theory*** proposed by *Wullstein and Mckennan* and Cole states that, due to trauma or surgery, cholesteatoma in canal skin is accidentally implanted into the middle

ear. According to retraction pocket theory, there is retraction of tympanic membrane due to Eustachian tube malfunction. This weakens the middle fibrous layer, which causes hyperplastic epidermal growths into the middle ear leading to development of cholesteatoma.

The most common site of cholesteatoma is the attic. It develops secondary to retraction of pars flaccida with accumulation o squamous cells within the retraction pocket leading to cholesteatoma formation. It can extend into the antrum via the aditus, facial recess, sinus tympani and posterior mesotympanum. Cholesteatomas arising from pars flaccida region cause erosion of scutum and medialise the malleus and incus. But pars tensa cholesteatomas (arising from posterosuperior quadrant) cause erosion of long process of incus and stapes and lateralise the malleus and incus. They can spread into round window niche, sinus tympani and facial recess. The main complications of cholesteatoma are erosion of the bony canal of facial nerve, the dura of middle and posterior cranial fossa and horizontal semicircular canal.

Cholesteatomas are commonly found in children with cleft palate (probably due to eustachian tube dysfunction). Cholesteatoma occurs in children with well pneumatised mastoid. They tend to be more aggressive in children than in adults. Immature Eustachian tube function can lead to retraction pocket and hence cholesteatoma formation. Children have greater levels of growth factors than

adults. This accelerates growth of cholesteatomas. Recent studies also show that there is increased rate of keratinocyte proliferation in children than adults. ***Cholesterol***

granuloma is due to accumulation of multinucleate giant cells in response to cholesterol crystals from degraded blood. It is formed when the mastoid air cells are blocked due to inflammation of mucosa.

GRANULATION

Chronic inflammation causes the edematous hyperemic middle ear mucosa to form polyps. The polyps can be limited to middle ear or protrude through perforation into external auditory canal. Polyp is usually lined by ciliated

columnar epithelium. Rarely this epithelium can undergo squamous metaplasia.

Bacterial toxins and other inflammatory mediators act on the oedematous middle ear mucosa leading to rupture of basement membrane. This causes prolapse of the underlying lamina propria. Infiltration of inflammatory cells in this tissue causes angiogenesis and further growth of granulation tissue. The most common sites of granulation tissue formation are attic and round window niche. Sometimes the granulation tissue can block the aditus and impair ventilation of mastoid.

OSSICLES

The pathology affecting the bony structures ie, ossicles, mastoid and bony labyrinth is termed osteitis. The chronic inflammation causes osteoclastic bone resorption. This can cause discontinuity of ossicles, labyrinthine fistula and vital structures such as dura may be exposed. There is hyperaemia, angiogenesis and histiocytic infiltration in the ossicles. The most commonly eroded ossicle is incus,

followed by stapes and malleus. The *lenticular process* at incudostapedial joint is more vulnerable to undergo ossicular resorption due to its sparse blood supply. Other structures commonly undergoing necrosis are long process of incus, stapes head and crura due to their delicate nature. Handle of malleus and body of incus rarely undergo ossicular necrosis. Bony resorption and new bone formation in mastoid due to osteitis can cause formation of sclerotic mastoid.

Ossicular fixation is the result of ***tympanosclerosis*** of ossicles. It commonly involves malleus head or body of incus in the epitympanum or the footplate of stapes. It can also occur due to adhesions between ossicles and tympanic membrane. Also, osteitis may be associated with neo-osteogenesis which causes fixation. Smoking (both passive and active) affects healing after tympanoplasty. It has local and systemic effects. It affects mucociliary clearance in middle ear mucosa.

SMOKING

Smoking (both passive and active) affects healing after tympanoplasty. It has local and systemic effects. It affects mucociliary clearance in middle ear mucosa. Nicotine causes vasoconstriction and promotes thrombosis. It also reduces oxygen carrying capacity of blood which reduces oxygenation to the graft. Thus there is impaired blood supply to the graft. It also causes Eustachian tube dysfunction. It increases susceptibility to infection. Smoking is associated with diseases like bronchitis, asthma, etc. Chronic cough due to these diseases can disturb the graft.

HEARING IN A DISEASED MIDDLE EAR

In the absence of perforation when the tympanic membrane is intact, with erosion of ossicles, ossicular coupling doesnot occur and sound is transmitted by acoustic coupling. The difference between ossicular coupling and acoustic coupling is 60 dB. So the patient has 60 dB loss of the conductive type. Sometimes the gap between the ossicles is *bridged* by connective tissue or cholesteatoma. in such cases the patient has loss less than 60 dB. But when there is perforation in addition to erosion of malleus and incus, sound waves can directly reach the oval window via the perforation. Hence the loss is around 40 dB and not 60 dB.

Fixation of footplate of stapes causes a variable degree of conductive hearing loss based on the degree of fixation. Similarly, fixation of malleus affects hearing to a variable extent. Anterior malleal ligament fixation causes loss less than 10 dB. But malleus fixation associated with fibrous tissue deposition in attic can cause greater hearing loss.

Perforation of tympanic membrane affects hearing to a variable extent depending on the site and size of perforation. Perforation causes loss of difference in sound pressure across the two sides of tympanic membrane, which in turn affects *ossicular coupling*. Hearing loss due to perforation mainly affects low frequencies. Larger perforation causes greater loss. Perforations in patients with sclerotic mastoid cause greater hearing loss than those with well pneumatised mastoid. Also, during active infection, the volume of air in middle ear and mastoid is greatly reduced than in a dry ear.

Hence hearing is more worse during active infection. Posterior perforations are said to cause more loss than anterior perforations. This is due to direct exposure of the round window to the sound waves which reduces the phase difference between the two windows[10]. On the other hand, a small anterior perforation may not produce hearing deficit.

Middle ear effusion (otitis media with effusion) is due to chronic inflammation and eustachian tube dysfunction. The tubal dysfunction causes negative pressure in middle ear which causes outpouring of transudate from the mucosa. This fluid does not drain due to block in the eustachian tube and hence accumulates. But recent studies show that the main cause of effusion is chronic inflammation secondary to infection. The bacteria produce biofilms and survive on the surface on mucosa and hence the cultures are negative. They trigger infiltration of inflammatory cells in the submucosa. This causes increased mucous secretion and also increase in the number of goblet cells which produce thick tenacious mucous. It can cause hearing loss of the conductive type with loss of upto 35 dB. This affects the ossicular coupling by two mechanisms. First, it causes mass loading of the surface of tympanic membrane by fluid. Second, it reduces the middle ear air space.

Adhesive otitis media and ***atelectasis*** of tympanic membrane in the presence of an intact ossicular chain can cause a loss of upto 50 dB. Here, tympanic membrane mobility is affected which causes lesser transmission. Also, if there is severe atelectasis causing tympanic membrane to invaginate into round window niche, there is

greater loss. It can cause pressure necrosis of ossicles.

Third window lesions can cause dissipation of sound away from the cochlea and hence hearing loss. It can be due to dehisence of superior or other semicircular canals, large vestibular aqueduct, Paget's disease, etc. The location of the third window is significant in pathogenesis of hearing loss. A window on the scala vestibuli side causes loss. But a window on the scala tympani side does not cause loss. In fact, it can improve hearing.

Patients with CSOM can also have sensorineural hearing loss. This is due to absorption of bacterial toxins across the round window membrane into the inner ear which causes inner ear damage.

REVISION SURGERY

Revision surgery has *poorer chance of success* than primary repairs. The failure of the primary surgery is due to chronic disease process which in turn can affect the outcome of revision surgery also. controlling the infection and restoration of ventilation of middle ear are necessary before the revision procedure. Hence staging the surgery if there is extensive disease is a better option to improve the outcome. The decision for staging the procedure is taken at the time of initial surgery if the disease is extensive. During the second stage surgery, ossicular reconstruction is done and the main sites of disease recurrence such as facial nerve, stapes and sinus tympani are examined for recurrence.

HEARING PATHOPHYSIOLOGY

When there is extensive middle ear granulation or cholesteatoma eroding all ossicles along with perforation, the entire middle ear mechanism is lost. Both the oval and round windows are equally exposed to sound waves. Such a patient will have a hearing loss of around 60dB. Some hearing is still preserved because the round window niche is deeply situated. Also, the labyrinthine vessels can yield more in scala vestibuli than scala tympani. This difference helps in movement of hair cells. If the tympanic membrane, malleus and incus are absent, good amount of hearing still exists if the round window is protected from direct exposure to sound. This causes sound to be conducted preferentially to the oval window. Thus phase difference between the two windows is maintained. the loss here is only 25 dB instead of 40 to 60 dB. This is the principle of hearing reconstruction in type 4 tympanoplasty. It is called ***round window baffle effect***.

If the malleus and incus are absent and the tympanic membrane is in direct contact with stapes, the lever ratio is lost but the areal ratio is maintained. This preserves considerable hearing. Similarly when stapes is also eroded, if we place a synthetic rod between tympanic membrane and oval window, good hearing is maintained. This mimics the 'columella' or the single ossicle in birds. Hence it is called ***columella effect***. In case of fixation of footplate of stapes, sound conduction mainly occurs by round window.

PATHOPHYSIOLOGY OF GRAFT UPTAKE

A variety of grafts are used for reconstruction of tympanic membrane

perforations, most commonly ***temporalis fascia***. Postoperatively, middle ear mucosa lines the graft on the medial aspect and squamous epithelium on the lateral aspect. The graft itself forms the middle fibrous layer. Though there is no such arrangement of radial and circular fibres in graft as in normal tympanic membrane, it can still function well. The tympanic membrane repair starts 12 hours after surgery while the granulation tissue starts appearing after 36 hours[11].

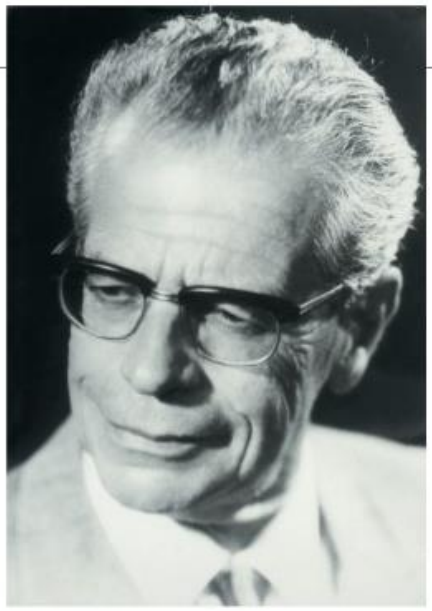
Grafts used for ossicular reconstruction are autograft ossicles, bone from mastoid cortex, cartilage, synthetic materials, etc. In autograft ossicles, the nonviable bone is postoperatively gradually replaced by new bone by creeping substitution. ***Creeping substitution*** occurs slowly in cortical bone grafts compared to ossicles. Cartilage grafts are not very stiff and undergo resorption over time. Synthetic graft materials stimulate a foreign body giant cell reaction that helps in graft survival.

HISTORICAL REVIEW

The first attempt to close a perforated tympanic membrane was done by **Marcus Banzer** in 1640. He used an ivory tube, stretched a pig's bladder across it and placed it over the perforation. In 1853, **Toynbee** used a rubber disc attached to a silver wire and placed it over the perforation and showed hearing improvement. In 1878, **Berthold** performed the first true tympanoplasty. He applied a plaster against the perforated tympanic membrane for 3 days to and de-epithelialized it while removing the plaster. He then placed a skin graft over the defect[12].

Surgeries for reconstruction of the middle ear apparatus faced much opposition due to lack of antibiotics, sterlization techniques, and microscopes. **Nylen** in 1921 introduced the monocular operating microscope[13]. Holmgren introduced binocular operating microscope in 1922 and also used aseptic techniques. In early 1950s, **Wullstein and Zollner** introduced tympanoplasty and used overlay graft[14,15]. Wullstein coined the term tympanoplasty in 1953. He described five types of tympanoplasty in 1956. Subsequently, various graft materials were used. Temporalis fascia graft was used by Heermann in 1961[16]. Ossiculoplasty was first described by Hall and Rytzner in 1957 where they used the patient's remanant ossicles. Cartilage was first used for tympanoplasty in 1973. Subsequently, various prosthesis were developed to improve hearing in tympanoplasty.

Wullstein



Fritz Zöllner, Otologist in Freiburg

Various factors in the middle ear which affect the outcome of surgery, have been analysed. ***Bellucci*** in 1973 classified based on the state of infection (otorrhoea and nasopharyngeal anomalies). He proposed that patients with higher Bellucci score have a greater risk for suppuration following tympanoplasty as persistent infection ultimately destroys the surgical repair[17]. But it didnot include other factors such as ossicular status, granulation, cholestestoma, etc. which will also influence the outcome of surgery.

Austin in 1972 classified four groups ***A to D*** based on the presence or absence of malleus handle and stapes suprastructure in cases where the incus was partially or completely eroded. In some patients, though all ossicles are intact, malleus head is fixed to attic by ankylosis or there may be stapes fixation. *Kartush* in 1994 added these two groups E and F respectively to Austin's classification. Another category, "nonclassifiable"

was added by Moretz in1998 to include rare conditions such as congenital anomalies and lateralised drumhead.

Black in 1992 introduced the **SPITE** system[18]. It had 12 important factors which were grouped under five group. The factors are SURGICAL (the complexity of surgery and whether repair of tympanic membrane and scutum are needed), PROSTHETIC (erosion of malleus with or without stapes, with ≥50dB air bone gap), INFECTION (active ear discharge or myringitis), TISSUE (poor general condition, mucosal abnormalities), EUSTACHIAN TUBE DYSFUNCTION (glue ear or retracted tympanic membrane). But he performed only univariate analysis and not multivariate analysis which avoids confounding factors. Albu et al proposed that the presence of malleus handle and mucosal pathology are important factors determining the outcome. ***Dornhoffer*** proposed ***OOPS (Ossicular Outcomes Parameters Staging)***. It includes ear discharge, ossicular status, status of middle ear mucosa and type of surgery. He concluded that mucosal status and otorrhoea contributed more to the outcome of surgery than pathology such as cholesteatoma[19].

REVIEW OF LITERATURE

In 1980 **James L. Sheehy and Robert G Anderson** did a retrospective study of 472 patients who had undergone tympanoplasty during an 11 year period at the otologic medical group[20]. They found a statistically significant relationship between the *perforation size and the amount of hearing loss* but no relationship between amount of hearing loss and tinnitus. The surgery was successful in terms of graft uptake in over 97% of the cases and reduced conductive deafness to 10 dB or less in 88%.

Warren Y. Adkins, Benjamin white, and Charlston SC in 1984 conducted a 5 year study on type 1 tympanoplasty with underlay technique using temporalis fascia graft. Patients with history of previous tympanoplasty were excluded. Overall success rate was 89%. They concluded that age of the patient, the period of dryness and the presence of infection at the time of surgery had no influence on the success[21].

In 2002, ***Karthush, Jack M***. Michaelides, Elias M. Becvarovski, Zoran La Rouere and Michael J. conducted studies on 120 cases of type 1 tympanoplasty. There was 100% graft uptake. The average improvement in air-bone gap was 5.3 dB, and the improvement in speech reception threshold was 5.9 dB[22].

In 2006 ***Debora Bunzen***, Alexandre Campos, Fabiana Sperandio, Silvio Caldas Neto identified several factors which influenced the final results of the tympanoplasty. They stated that presence of ear discharge preoperatively and alterations in the middle ear mucous did not change the final surgical result. Perforations occupying less than 50% of the tympanic membrane surface yielded better results than subtotal and

total perforations. Out of the 94 patients who underwent surgery, 29 were men and 68 were women with average age of 25.16 years. In patients with healthy middle ear mucosa there was 80% success rate[23].

In 2007 ***Emir, Hatice***; Ceylan, Kursat, Kizilkaya et al. did retrospective studies on 607 patients who underwent type 1 tympanoplasty between January 1997 and December 2004. Based on their statistical analysis, they concluded that *male gender, younger age, smaller perforations and experienced surgeons* were good prognostic factors for success of tympanoplasty[24].

In **2007 Jyothi P. Dabalkar**, Krishna Vora and Abhik Sikdar said that ***temporalis fascia graft*** achieved an uptake of 84% and satisfactory improvement in hearing in 76% of the patients. ***Tragal perichondium*** achieved a success rate of 80% graft uptake and 75% hearing gain. Their study group had 50 patients of which, 33 were males and 17 were females. The age group of patients was between 13 to 56 years. The study included those with unilateral safe perforation and pure conductive hearing loss. Patients with active discharge were initially treated conservatively and were included in the study when their ear became dry for at least 6 weeks[25].

Khalid Almazrou et al used MERI as a tool to evaluate *paediatric ossiculoplasty* between 1995 to 2005. They used a variety of autologous and alloplastic prosthesis and concluded that MERI is not a useful tool for predicting the outcome of ossiculoplasty in children. They stated that children have poorer hearing outcome following ossiculoplasty than adults[26].

Min Beom Kim et al conducted a retrospective study between 1997 to 2005. They compared the air bone gap closure between patients undergoing *canal wall up and canal wall down mastoidectomy*. All the subjects underwent ossiculopalsty as second procedure following canal wall up or canal wall down mastoidectomy. They concluded that there was no statistical significance between both the groups[27].

Pinar E et al studied the role of middle ear risk index and other factors such as age, sex, systemic diseases, site and size of perforation, period of dryness, presence of myringosclerosis, nasal pathology, status of opposite ear and type of surgery on the outcome of tympanoplasty. It was conducted between 2002 and 2007. They concluded that **low MERI scores**, smaller perforation, healthy opposite ear, absence of myringosclerosis and more than 3 months dryness were **good prognostic** factors[28].

Uygar Levent Demir et al did a study on whether the Middle Ear Disease or the reconstruction material determined the functional outcome in ossiculoplasty. It was done as a retrospective study between 2007 to 2010. The patients were classified into mild, moderate and severe MERI. They stated that success was mainly based on the status of middle ear ie, MERI score. But, within the same risk group, different reconstruction materials had different outcomes[29].

Sevim Aslan Felek published his results on the prognostic value of MERI in type 2 ossiculoplasty in 2010 based on a 12 year retrospective analysis. He classified the patients into low, moderate and high risk based on MERI and compared the outcome of different ossicular prostheses. He stated that MERI is a valuable tool to make good

patient selection and judge the risks[30].

Ankush Sayal et al studied Hearing Results of Tympanomastoidectomies Using Titanium Prostheses in patients with mild, moderate and severe MERI from September 2009 to December 2011. They concluded that in developing countries such as India, as we have limited resources, decision regarding ossicular reconstruction should be made taking into account MERI scores. They also stated that in cases having severe MERI it is advisable to stage the *surgery* and do ossicular reconstruction in second sitting[31].

Sushil Jha et al between 2007 to 2009 did a comparative study of ossiculoplasty by using various graft materials. He combined the preoperative and intraoperative risk factors into a numerical value, the MERI. He calculated and compared the hearing improvement for various graft materials in ossiculoplasty for patients falling under the same MERI group[32].

In 2009, **Viktor Chrobok** et al studied the *Prognostic Factors for Hearing Preservation in Surgery of Chronic Otitis Media*. They compared the MERI score of patients with hearing benefit after surgery. They also analysed the individual risk factors in the middle ear which affect the outcome of surgery. They concluded that MERI is a significant prognostic factor for predicting the outcome. Patients with a higher MERI had a more severe impairment of air and

bone conduction hearing threshold pre-op and post-op compared to patients with a lower MERI score. They stated that cholesteatoma, perforation, ossicular erosion, and previous surgery were negative prognostic factors[33].

Rakesh Saboo et al in 2014 did studies on the role of MERI score on the outcome of tympanoplasty. They concluded that patients with mild MERI had maximum graft uptake while those with severe MERI had greater failure rates. They also assessed Eustachian tube function in their patients and said that normal Eustachian tube function was a prerequisite for success of tympanoplasty[34].

ABSHIRINI et al studied the prognostic factors in tympanoplasty. They also concluded that patients with lower MERI score had higher rates of success. They evaluated other factors and stated that size of perforation, healthy opposite ear, more than 3 months dry period, absence of myringosclerosis, intact canal wall technique were factors that were associated with higher success rate. Prediction of the outcome before surgery helps to confer the patient before surgery[35].

MATERIALS AND METHODS

This study is a prospective study which was conducted in the Department of Otorhinolaryngology and head and neck surgery, Thanjavur medical college hospital, Thanjavur, Tamil Nadu from NOVEMBER 2013 to JUNE 2015. The study group comprises 50 patients with chronic suppurative otitis media both mucosal and squamous type with hearing loss.

INCLUSION CRITERIA:

All patients with CSOM both mucosal and squamous type planned for tympanoplasty with or without mastoidectomy

EXCLUSION CRITERIA: Patients with

1. Systemic diseases
2. Otomycosis
3. Other septic foci which can influence the outcome of tympanoplasty

Detailed history was obtained from the patient such as the nature of ear discharge, the period of dryness, hearing loss, other medical illness, history of smoking, previous ear surgery and long term use of ototoxic drugs. Otoscopic examination was done to find the presence or absence of perforation, granulation tissue and cholesteatoma. Tuning fork tests were done to find the type of hearing loss. Patients with conductive hearing loss alone were taken for the study. Those with sensorineural and mixed hearing loss were excluded from the study. Examination of nose and paranasal nasal sinuses and throat was done to rule out septic foci such as

adenotonsilltis and sinusitis. In such cases, the patients underwent surgery to eliminate septic foci prior to mastoidectomy.

INVESTIGATIONS

Basic investigations such as complete blood counts were done prior to surgery. Aural swab culture sensitivity and CT temporal bone were taken. Otoendoscopy and otomicroscopy were done to confirm the otoscopic findings and also in large perforations, the middle ear mucosa, any polypoidal changes in middle ear, the ossicles, and attic were inspected. Pure Tone Audiometry was done by ***Hughson and Westlake method modified by Carhart and Jerger***. The type and degree of hearing loss was noted. The mean air-bone gap was measured from the air and bone conduction thresholds at 0.5 kHz, 1 kHz, 2 kHz, and 3 kHz. The middle ear risk index was calculated. The patients were stratified into those with mild(0-3), moderate(4-6) and severe(≥7) MERI.

PROCEDURE

The type of tympanoplasty and mastoidectomy was decided intraoperatively based on the extent of disease in middle ear and mastoid. Temporalis fascia graft was used for all patients. The mastoid cortex was drilled using electrical burr and any granulation, cholesteatoma or serous effusion were removed. For ossiculoplasty, the eroded ossicles were reshaped and used. Septal cartilage allograft or grommet were also used. The temporalis fascia graft was placed by underlay technique. Intravenous

antibiotics and the analgesics were given in the immediate postoperative period. Suture removal was done on 7th post operative day. Postoperatively the patients were advised steam inhalation, bubble gum chewing and balloon blowing to maintain aeration of middle ear and mastoid air cells. They were prescribed antibiotics and medicated ear drops. After discharge the patients were reviewed once in 15 days for 3 months and once a month for another 3 months.

The outcome of surgery was assessed based on graft status and pure tone audiometry. Graft status was analysed by otoscopy.

a) Successful - the healed graft with proper middle ear aeration.
b) Atelectatic graft.
c) Graft failure or perforation of graft.

Pure tone audiometry was done at the end of one month and 3 months. The mean air bone gap was calculated (from the air bone gaps at 0.5 kHz, 1 kHz, 2 kHz and 3 kHz). Based on the preoperative and postoperative air bone gaps, the hearing benefit was calculated. The hearing benefit in patients with mild, moderate and severe MERI were analysed.

Pre operative PTA of a patient

GCP—177/5—10,000 Cps. 3-7-2008(P4-1)

DEPARTMENT OF OTORHINOLARYNGOLOGY

THANJAVUR MEDICAL COLLEGE, THANJAVUR

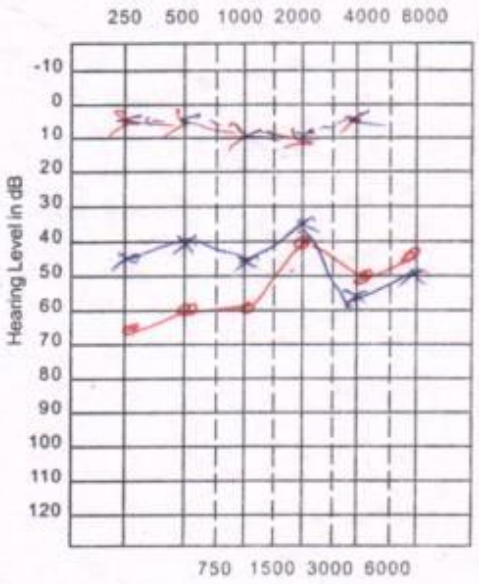
AUD No. : 969/15

Patient Name : Manikandan Age/Sex : 14/M

Address : S/O. Karunanithi
Orathanadu Rd.

Date : 11 - 06 - 2014
OP/IP No. : 013379
Occupation :
Unit : ENT / IP.

AUDIOGRAM
FREQUENCY IN Hz.



	Rt. EAR RED	Lt. EAR BLUE
AIR CONDUCTION UNMASKED	O	X
MASKED	△	□
BONE CONDUCTION UNMASKED	⊏	⊐
MASKED	⌜	⌝
FREE FIELD	S	
Above symbol with arrow indicates no response		

IMPEDANCE AUDIOMETRY
TYMPANOMETRY

	Rt. Ear	Lt. Ear
Peak Middle Ear pressure in mm H_2O		
Static compliance in cc		
Cavity volume in cc		
Type		

SPEECH AUDIO METRY

	Rt. Ear	Lt. Ear
RINNE		
WEBER		
PTA	53	40

	Rt. Ear	Lt. Ear
SRT		
SDS		
UCL		

REFLEX THRESHOLDS

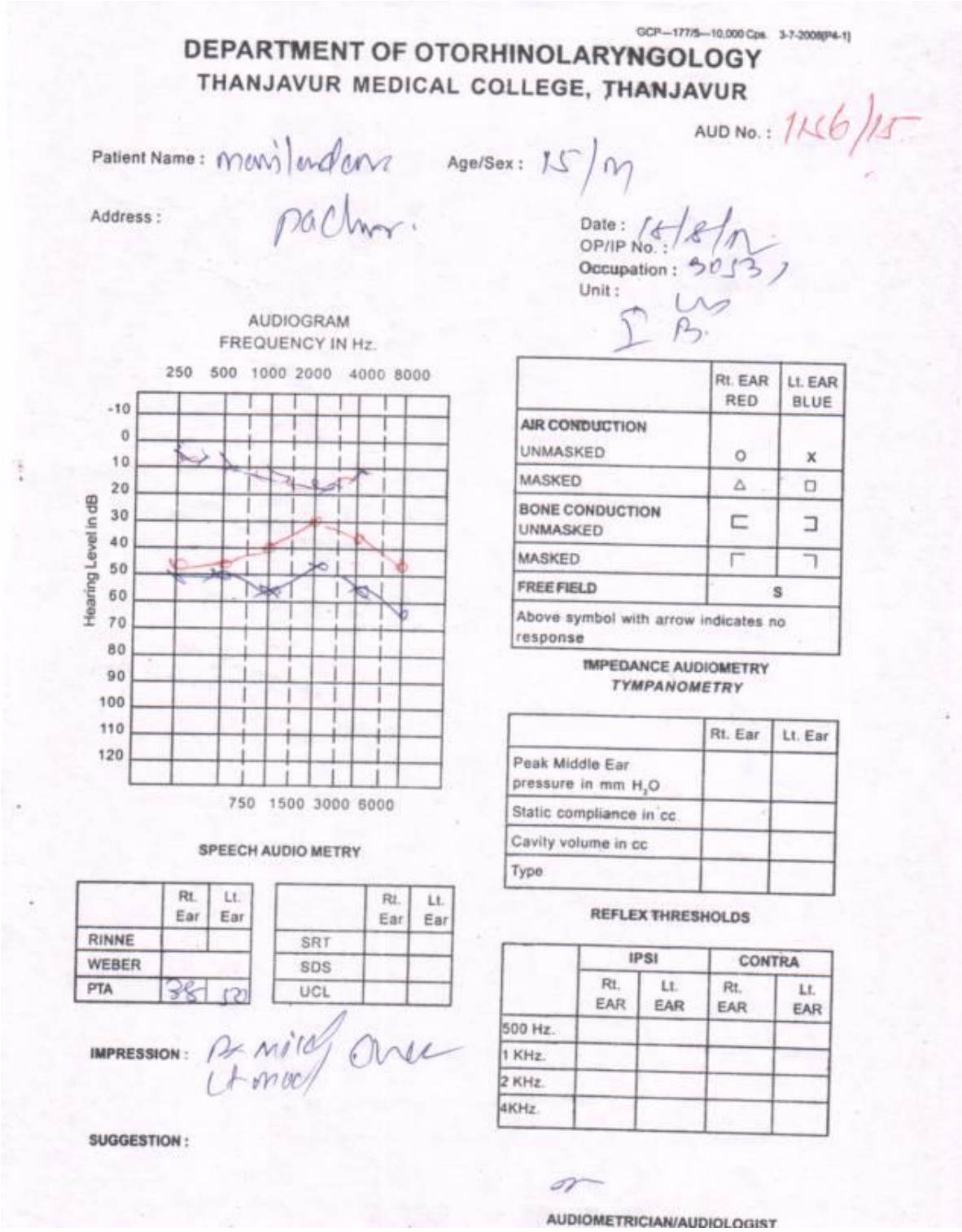
	IPSI		CONTRA	
	Rt. EAR	Lt. EAR	Rt. EAR	Lt. EAR
500 Hz.				
1 KHz.				
2 KHz.				
4KHz.				

IMPRESSION : Rt moderate conductive Hearing loss

SUGGESTION : Lt mild conductive Hearing loss

AUDIOMETRICIAN/AUDIOLOGIST

Post operative PTA of the same patient showing reduction in air - bone gap.



GCP—177/5—10,000 Cps. 3-7-2008[P4-1]

DEPARTMENT OF OTORHINOLARYNGOLOGY
THANJAVUR MEDICAL COLLEGE, THANJAVUR

AUD No. : 1126/15

Patient Name : manilandan Age/Sex : 15/M

Address : pachur.

Date : 14/8/15

OP/IP No. :

Occupation : Boy

Unit :

AUDIOGRAM
FREQUENCY IN Hz.

250 500 1000 2000 4000 8000

-10 0 10 20 30 40 50 60 70 80 90 100 110 120

Hearing Level in dB

750 1500 3000 6000

	Rt. EAR RED	Lt. EAR BLUE
AIR CONDUCTION UNMASKED	O	X
MASKED	△	□
BONE CONDUCTION UNMASKED	⊏	⊐
MASKED	⌜	⌝
FREE FIELD	S	
Above symbol with arrow indicates no response		

IMPEDANCE AUDIOMETRY
TYMPANOMETRY

	Rt. Ear	Lt. Ear
Peak Middle Ear pressure in mm H_2O		
Static compliance in cc		
Cavity volume in cc		
Type		

SPEECH AUDIO METRY

	Rt. Ear	Lt. Ear
RINNE		
WEBER		
PTA	35	50

	Rt. Ear	Lt. Ear
SRT		
SDS		
UCL		

REFLEX THRESHOLDS

	IPSI		CONTRA	
	Rt. EAR	Lt. EAR	Rt. EAR	Lt. EAR
500 Hz.				
1 KHz.				
2 KHz.				
4KHz.				

IMPRESSION : B/L mild CHL

SUGGESTION :

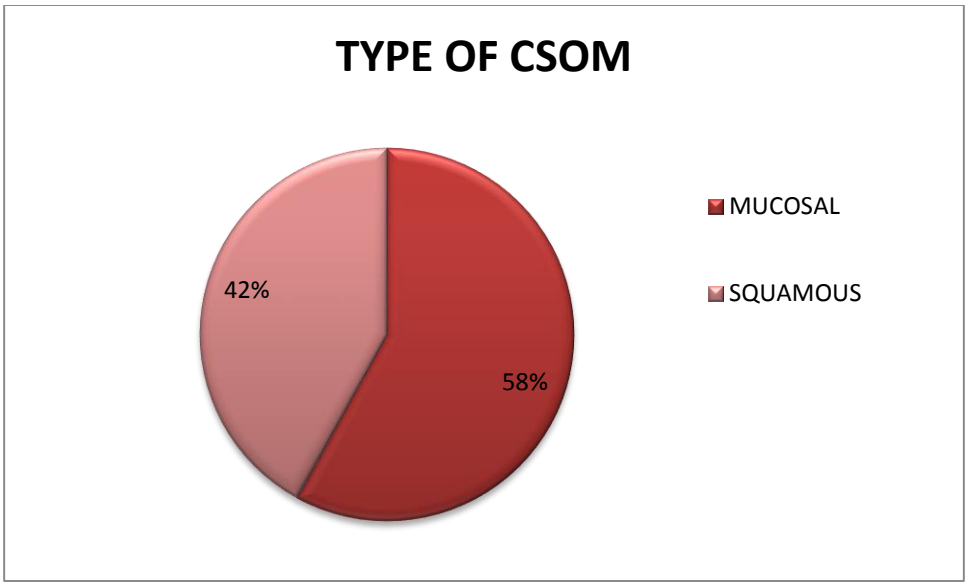
AUDIOMETRICIAN/AUDIOLOGIST

RESULTS AND ANALYSIS

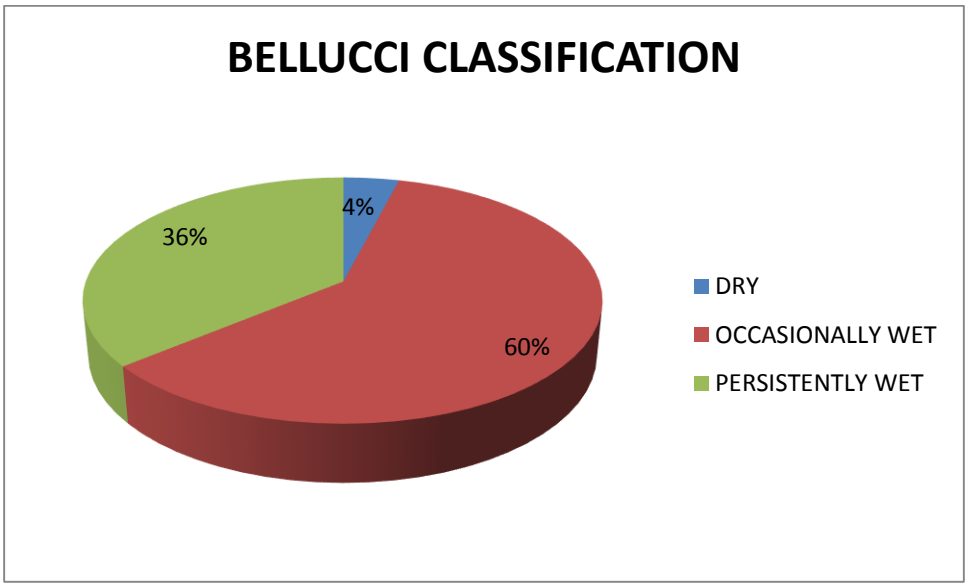
This study was conducted in the department of *ENT AND HEAD & NECK SURGERY, THANJAVUR MEDICAL COLLEGE AND HOSPITAL, THANJAVUR, TAMILNADU* for a period of two years from NOVEMBER 2013 to JUNE 2015. The study group consists of 50 patients with chronic suppurative otitis media of both mucosal and squamous type.

AGE	NO. OF PATIENTS	PERCENTAGE
0 - 10 YEARS	3	6
11 - 20 YEARS	18	36
21 - 30 YEARS	16	32
31 - 40 YEARS	9	18
41 - 50 YEARS	4	8

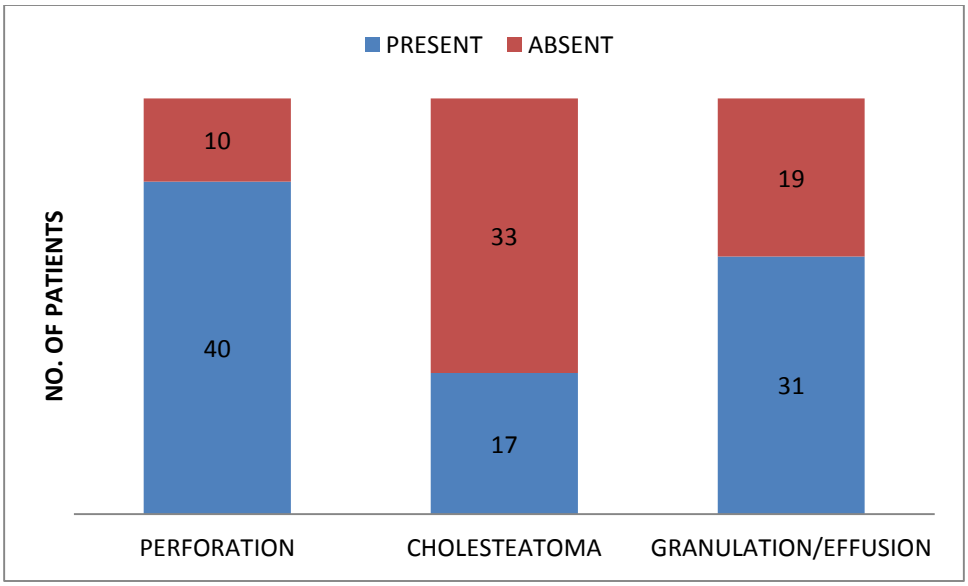
According to this study, the most common age group affected is 11 to 20 years followed by 21 - 30 years. The study comprises 19 males and 31 females. Thus females are commonly affected (60 %). 29 patients belong to mucosal or tubotympanic type of CSOM and 21 patients belong to the squamous type or the atticoantral type.



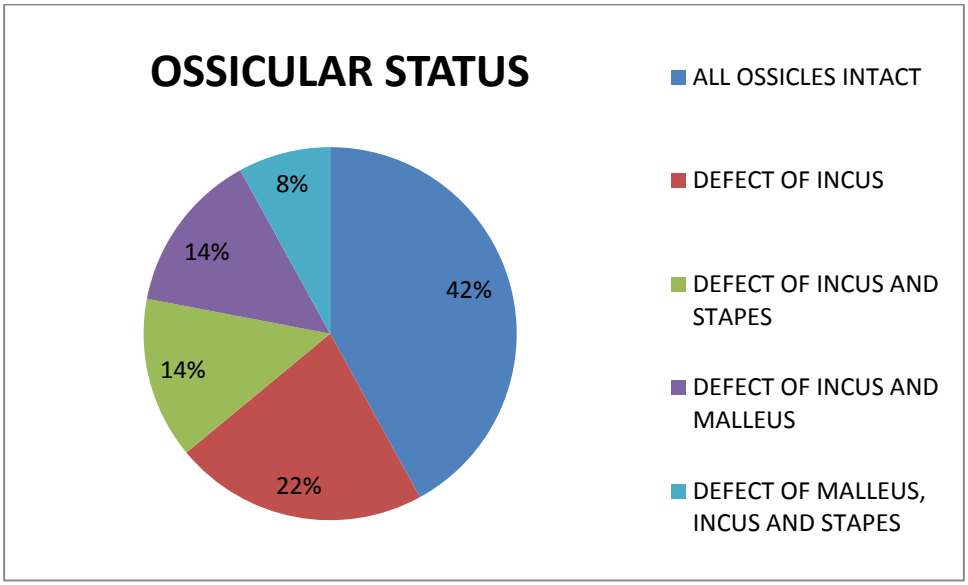
2 patients had dry ear, 30 patients had occasionally wet ear and 18 patients had persistently wet ear according to Bellucci classification of otorrhoea.



Thus most patients had occasionally wet ear (Bellucci Classification 1).

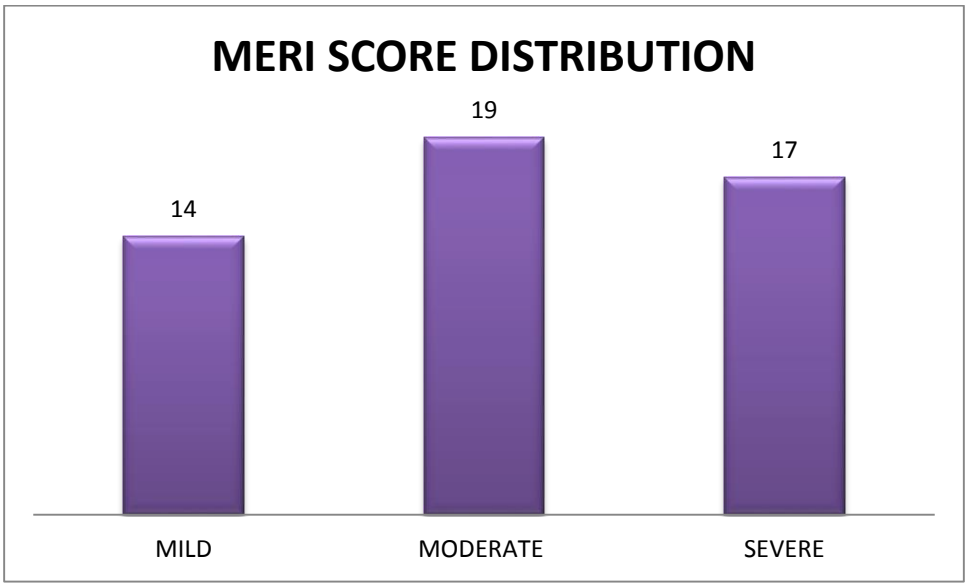


40 patients had perforation, 17 had cholesteatoma and 31 had granulation / effusion. The following chart shows the ossicular status of the study group according to Austin-Kartush classification.



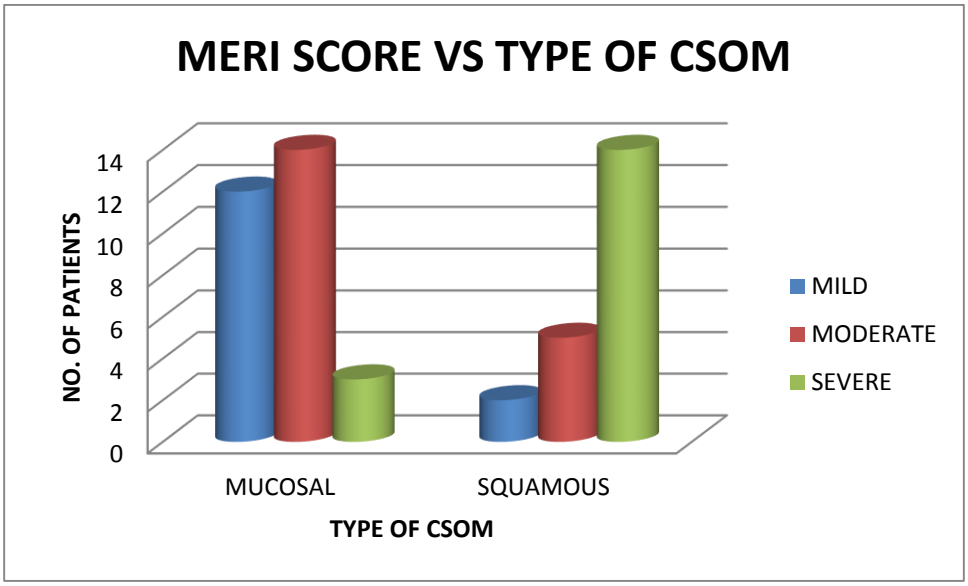
There were 3 patients with history of active or passive smoking. There was no case of revision or staged procedure.

The study group comprises 14 patients with mild (1-3) MERI score, 19 patients with moderate (4-6) MERI score and 17 patients with severe (≥ 7) MERI score. Of this, majority of patients with severe MERI had squamous type CSOM (14 patients). Among mucosal type, very few patients (3 patients) had severe MERI.



MERI SCORE	NO. OF PATIENTS
MILD	14
MODERATE	19
SEVERE	17

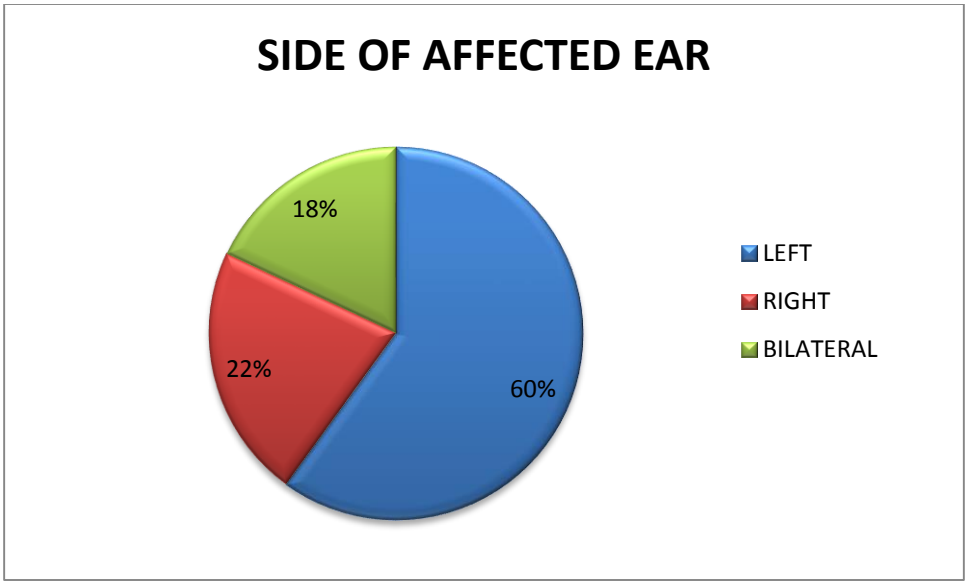
	MILD	MODERATE	SEVERE
MUCOSAL	12	14	3
SQUAMOUS	2	5	14



The above graph shows patients with squamous type CSOM have greater MERI scores.

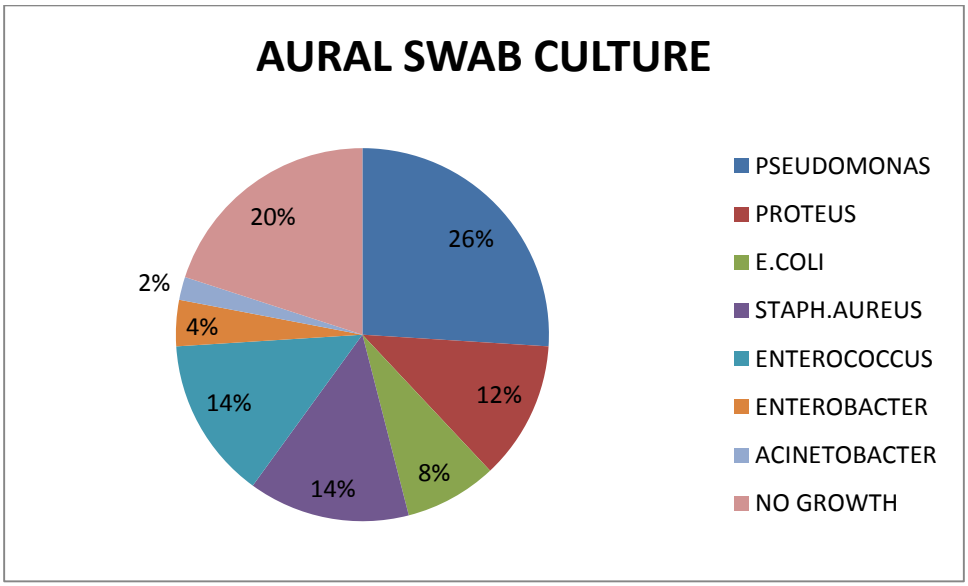
33 patients were operated for left sided disease and 17 patients were operated for right sided disease. Of this 3 patients operated for left side and 6 patients operated for right side had bilateral disease. Thus left ear pathology is more common in our study group.

SIDE AFFECTED	FREQUENCY	PERCENTAGE
LEFT	30	60
RIGHT	11	22
BILATERAL	9	18



The most common organism isolated in aural swab culture was Pseudomonas (26%). The other organisms were Proteus, Escherichia coli, Staphylococcus auerus, Enterococcus, Enterobacter and Acinetobacter. No growth was obtained in 10 patients.

ORGANISM	NO. OF PATIENTS	PERCENTAGE
PSEUDOMONAS	13	26
PROTEUS	6	12
E. COLI	4	8
STAPHYLOCCUS AUREUS	7	14
ENTEROCOCCUS	7	14
ENTEROBACTER	2	4
ACINETOBACTER	1	2
NO GROWTH	10	20

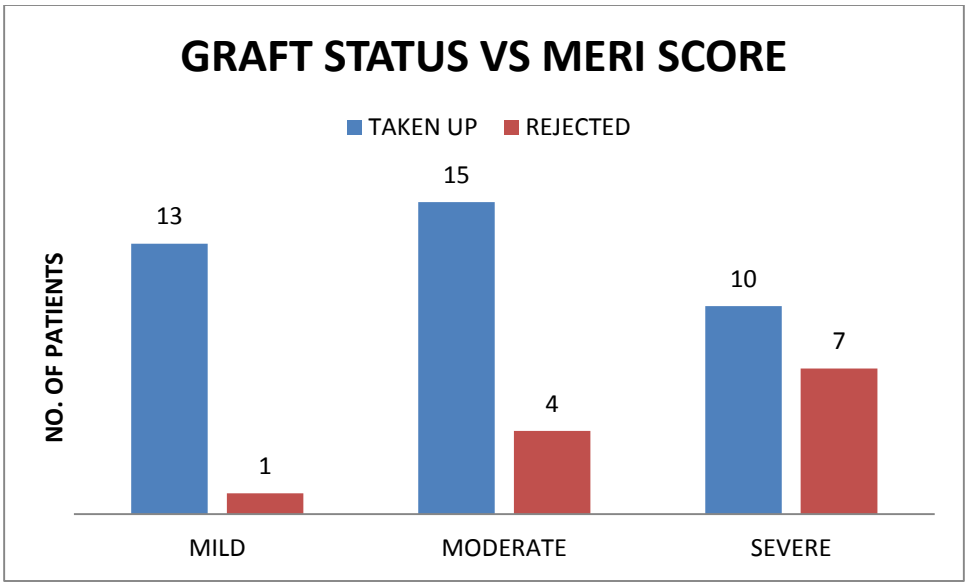


	MILD	MODERATE	SEVERE
TAKEN UP	13	15	10
REJECTED	1	4	7

The data in the table indicates that when the MERI score is mild graft is taken up by 13 patients and rejected for only 1 patient. When the MERI score is moderate the graft is taken up by 15 patients and rejected for only 4 patients when the MERI score is high the chances of graft taken up among patients is low & rejection rate is high .

F	df1	df2	Significance
2.299	10	39	.031

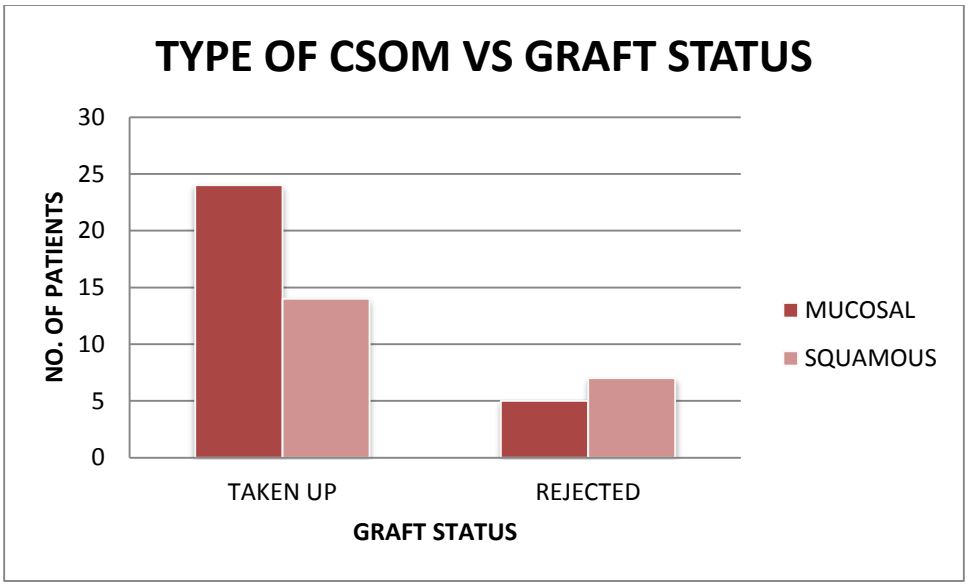
It may be noted from the above table that the p value of .031 is lower than alpha value at 5% level of significance by doing the T Test. Thus there is an inverse relation between the MERI score and the rate of graft uptake. Therefore higher the MERI score, lower is the rate of graft taken up in patients and patients with lower MERI have higher rate of graft uptake.



The graft is taken up for 38 patients (76%) and rejected for 12 patients (24%). Thus the overall success rate of tympanoplasty is 76 % according to graft status. Among those with mild MERI, graft is taken up for 13 patients and rejected only for 1 patient. Similarly, among those with severe disease, there is higher graft rejection rate (7 patients).

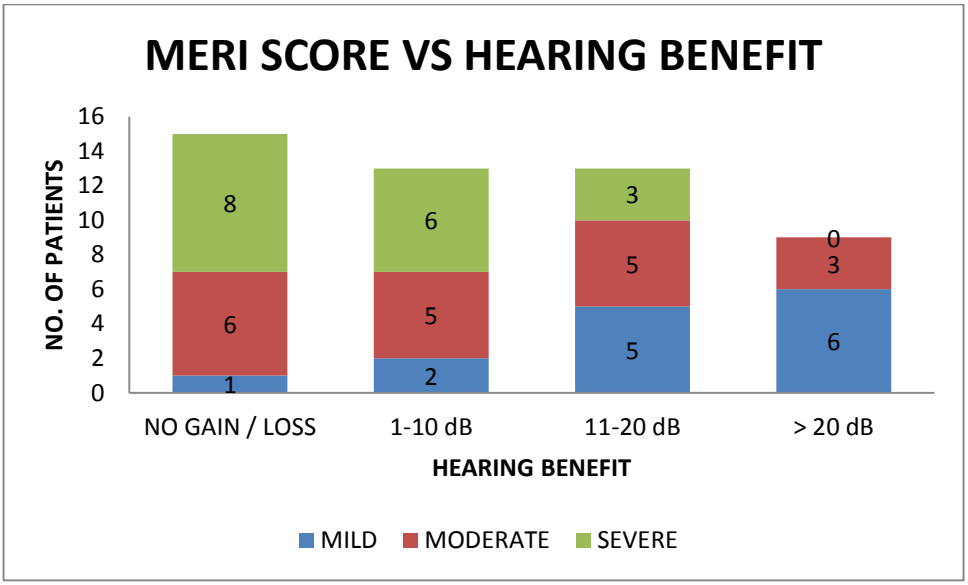
	TAKEN UP	REJECTED
MUCOSAL	24	5
SQUAMOUS	14	7

The patients with mucosal disease have higher graft uptake (82.7%). The patients with squamous type CSOM have graft uptake rate of 66.6% and graft rejection rate of 33.3%.



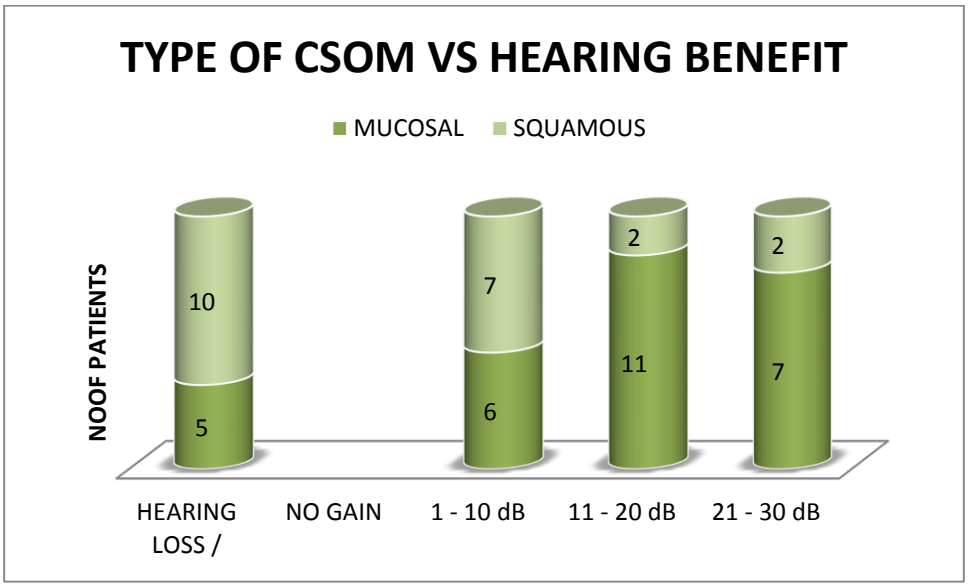
35 patients have obtained hearing benefit postoperatively according to this study. Thus this study group has 70% hearing benefit. The data in the table indicates that when the MERI score is mild, hearing benefit is observed for 13 patients and hearing loss / no gain for only 1 patient. When the MERI score is moderate, hearing benefit is observed for 13 patients and hearing loss / no gain for 6 patients. When the MERI score is high, the chances of hearing benefit is observed for only 9 patients and hearing gain / loss no for 8 patients . By doing Chi-Square test, the resultant p value of .041 is significant at 5 % level of significance. Therefore higher the MERI score, lower is the patient's chances of hearing to improve. Similarly, patients with low MERI scores have greater hearing benefit after surgery. The mean score of hearing benefit among patients is 9.90 dB.

	HEARING LOSS / NO GAIN	1 -10 dB	11 - 20 dB	> 20 dB
MILD	1	2	5	6
MODERATE	6	5	5	3
SEVERE	8	6	3	0



The patients with mucosal type CSOM have obtained greater hearing benefit than patients belonging to the squamous type.

	MUCOSAL	SQUAMOUS
HEARING LOSS / NO GAIN	5	10
1 - 10 dB	6	7
11 - 20 dB	11	2
21 - 30 dB	7	2



DISCUSSION

Chronic suppurative otitis media is a very common Otorhinolaryngeal problem worldwide, especially in developing countries. Around **7.8%** of the Indians suffer from this infection according to WHO report, 2004[36]. It is more common in rural areas than urban areas and is associated with poor hygiene, illiteracy and is common among the middle and low income groups. Inspite of the availability of wide range of antibiotics, better surgical techniques and newly developed prosthetic materials we are still not able to reach 100% successful outcomes in tympanoplasty in terms of graft uptake and hearing benefit. This is due to the extent of pathology in the middle ear and mastoid which affects the outcome. Hence these are summarised and assigned a numerical value, the MERI index, which helps us to identify the extent of disease and thereby predict the outcome of surgery. Hence this study was done to find if MERI score can be effectively used as a tool for this purpose.

According to the study conducted by *Shrikrishna BH* et al, non cholesteatomatous CSOM is common in the age group of **10 to 30 years**. This is in comparison with our study, which shows that the most common age group affected is 11 to 20 years (36%) followed by 21 to 30 years (32%). Thus 68% of our study group belongs to 11 to 30 years of age[37]. *Bijan Basak* et al also stated that CSOM (both tubotympanic and atticoantral type is common in the age group 11 to 30 years[38]. According to Glasscock, though otitis media is a disease which occurs commonly in paediatric age group, the mean age at which the disease manifests is 20-29 years. The

infection is most commonly *acquired in childhood* as acute suppurative otitis media is common in children. This condition persists in early and middle adulthood, unless treated. Hence the incidence is more common in young population. Also, trauma, one of the causes of the initial episode, is common in young adults. Non healing of the traumatic perforation leads to CSOM.

In our study, females are more commonly affected than males (60%). According to the study conducted by Bijan Basak et al there is **female predominance**[38]. However, according to the study conducted by *Abhinav. D*. et al, the disease is more common in males[39]. Lack of proper nutrition and reduced literacy rate (and hence poor hygienic practices) among females may contribute to higher incidence in females. Also, in our study, mucosal type CSOM is more common in females and squamous type CSOM is more common in males.

The mucosal type constitutes 58% and squamous type constitutes 42% in this study. The squamous type is more prone for complications. But in our study, two patients presented with complications one with facial nerve palsy and the other with temporal lobe abscess. The patient with facial nerve palsy had squamous type disease and the patient with temporal lobe abscess had mucosal disease. Both underwent treatment of the complication followed by tympanoplasty.

There were 14 patients with mild MERI score, 19 patients with moderate MERI score and 17 patients with severe MERI score. Of this, majority of patients with severe MERI had squamous type CSOM. Among mucosal type, very few patients had

severe MERI. Squamous type is associated with greater extent of disease in middle ear cleft as cholesteatoma and granulation tissue erode the ossicles and other structures. Thus **squamous type** is associated with greater middle ear destruction and **higher MERI**. More patients were operated for left sided disease (60%) than right side (22%) and 18% had bilateral disease. There is no association between the side operated and the outcome of surgery in literature.

In this study, the most common organism isolated in aural swab culture was ***Pseudomonas*** (26%). No growth was obtained in 20% patients. The works of *Harvinder kumar* et al shows that Pseudomonas aeruginosa is the most common organism which correlates with our study[40]. This organism is especially implicated in the formation of biofilms. Also in his study, Staphylococcus is the second commonly isolated bacteria. In our study, both Staphylococcus aureus and Enterococcus are the next common bacteria and constitute 14% each. According to the studies done by *A Srivastava. et al and Prakash M* et al, **Staphylococcus aureus** is the most common organism followed by Pseudomonas Aeruginosa[41]. The patients with no growth had dry ear or mucoid secretions from middle ear. The antimicrobial agent of choice against Pseudomonas aeruginosa is ciprofloxacin followed by ceftazidime.

The organisms usually enter through the tympanic membrane perforation. Sometimes it can be due to infection through the Eustachian tube. For all the patients without complication middle ear suction clearance is very helpful to reduce the load of infection[42]. Poor hygiene, overcrowding, and antibiotic abuse leads to bacterial infection

and antibiotic resistance. Chronic use of antibiotic ear drops leads not only to bacterial resistance but also ototoxicity and sensorineural hearing loss.

According to Bellucci's classification of otorrhoea, in this study, 4% had dry ear, 60% had occasionally wet ear and 36% had persistently wet ear. There is no patient belonging to wet with cleft palate group. Both patients with dry ear had successful graft uptake. Among those with persistently wet ears, 33% had graft failure while 20% graft rejection rate was found in occasionally wet ears. The study conducted by *John L. Dornhoffer* et al shows that Bellucci classification was not statistically significant overall. However, there was a clear trend towards worse results with Bellucci classifications 1, 2, and 3[43]. Thus otorrhoea alone may not be significant as there are other middle ear factors which can influence the outcome. However, more the severity of otorrhoea, worser is the outcome. Hence appropriate antibiotics should be started based on culture and sensitivity reports to reduce the ear discharge. Studies show that if there is a minimum interval of three months without otorrhea, the success rate of surgery increases to more than 30% when compared to the cases that underwent surgery in an infected site[44]. Thus it is preferable to *make the ear dry preoperatively to achieve best results.*

In our study, success rate has been explained with reference to two different entities - graft status and hearing benefit. The overall success rate of tympanoplasty is 76 % according to graft status. Grafts which are rejected or perforated are taken as failures, which is 24%. *Manpreet Kaur* et al did studies on comparison of graft uptake between tympanoplasty alone and tympanoplasty combined with cortical mastoidectomy in non

cholesteatomatous chronic suppurative otitis media in patients with sclerotic bone. They concluded that graft uptake was 76% in patients who underwent tympanoplasty and 88% in tympanoplasty combined with cortical mastoidectomy[45]. But the studies conducted by *Andersen SA,* Aabenhus K, Glad H, Sorensen MS show that graft uptake rate after type 1 tympanoplasty was 86.6% at the end of 12 months[46]. Thus different studies show different graft uptake rates depending on the type of surgery and middle ear status. The main cause for graft failure in our study group was infection (especially upper respiratory infection) in the immediate postoperative period.

Harikrishnan et all studied the factors improving the outcome of tympanoplasty. He said that there is a good hearing benefit and tympanoplasty results if the patients have proper follow up for two years. They also concluded that preoperative audiological evaluation is necessary to assess the prognosis and also for medicolegal point of view[47].

Our study concluded that there is higher graft rejection rate for patients with severe MERI scores and vice versa. Studies conducted by *Viktor Chrobok* et al shows that patients with a generally lower MERI had better pre-op and post-op air and bone conduction than patients with a higher MERI score[33]. They concluded that cholesteatoma, tympanic membrane perforation, status of the ossicular chain, history of previous surgery and the overall sum of the MERI were highly significant pre-op negative prognostic factors influencing the outcome of tympanoplasty. Thus our study coincides with the findings of Viktor Chrobok's study.

In our study, the choice of treatment (whether the patient undergoes simple or modified radical mastoidectomy and the type of tympanoplasty) was done based on the extent of disease in the middle ear and mastoid. For patients with limited disease in the attic, atticotomy with tympanoplasty was done. For dry central perforation, mastoidectomy was not done and only type 1 tympanoplasty was done. Ossiculoplasty was done based on the extent of ossicular erosion. The remaining ossicles were reshaped and kept as ossicular graft for most patients. For 5 patients, septal cartilage allograft was used. For two patients, grommet was reshaped and kept as ossicular graft.

In patients with incus erosion, ossicular prosthesis was placed between stapes head and handle of malleus. In patients with erosion of stapes, ossicular graft was placed between the footplate of stapes / oval window and malleus. In case of malleus and incus erosion, ossicular graft was placed between stapes and tympanic membrane.

Proper ventilation of the middle ear is necessary for successful graft uptake. Hence postoperatively patients were advised valsalva manouvre, steam inhalation and bubble gum chewing to ensure middle ear aeration. At the same time they were advised not to blow forcefully to avoid displacement of the ossicular graft.

Success of hearing reconstruction procedure also depends on the preoperative ossicular status. An intact ossicular system with only a perforation in the tympanic membrane gives the best results. The most common ossicle to be eroded is the **long process of incus** due to the nature of blood supply to the incudostapedial joint. In our study, there is erosion of incus in 29 patients (58%). According to the studies conducted by *Ghodrat Mohammadi,* incus is the most commonly eroded ossicle. In his

study, total erosion of the incus is more common than partial erosion[48]. However, several studies state that the **presence of malleus handle is an important factor** which affects the outcome. Hence defect of malleus is given a higher MERI score than defect of incus or stapes. The studies conducted by *Kevin F. Wilson, Nyall R. London and Clough Shelton* show that the presence of handle of malleus preoperatively gives a better postoperative hearing outcome. They said that the presence of an intact stapes did not affect initial hearing outcome. However, patients with an intact stapes had better long term hearing outcomes than those with erosion of stapes[49]. Ossicular fixation is less common in CSOM compared to ossicular necrosis. In our study, there was no case of ossicular fixation.

Manas Ranjan Rout et al analysed the ossicular changes in tubotympanic type of CSOM. In his study, 37% patients had ossicular pathology. He said that ossiular erosion is less common in mucosal than squamous type of CSOM[50]. In our study, 11 patients belonging to the mucosal type had ossicular necrosis.

Granulation tissue and cholesteatoma should be dissected away from the underlying bone using fine hooks and small gauze balls. this prevents chances for remanants and hence recurrence. **Alpha chemotrypsin enzyme** can be used to dissolve the granulomatous layer deep to the matrix of cholesteatoma[51].

Veysel Yurttafl et al stated that the presence of *granulation in middle ear had a negative effect on the hearing improvement after tympanoplasty*. His study concluded that graft uptake rate was only 44.4% in patients with extensive middle ear granulation tissue. He advocated mastoidectomy in addition to tympanoplasty for all

patients with active middle ear infection to remove granulation tissue from middle ear and mastoid cavity[52]. Granulation in middle ear predisposes to ossicular necrosis. *Da Costa and Paparella* found out that ossicle changes developed in 96% of granulation tissue[53]. We observed that *preoperative instillation of antibiotic steroid ear drops* is very useful to reduce middle ear granulations if present. Also incomplete removal of granulation tissue leads to recurrence and failure of graft uptake.

Smoking is asssociated with reduced graft uptake. *Zoran Becvarovski* stated that *delayed failure* of the graft was more commonly seen in smokers(60%) than non-smokers(20%)[5]. According to *Lin Y C* et al, the frequency of failure in tympanoplasty was significantly higher for smokers than for nonsmokers. They recommended preoperative and postoperative cessation of smoking for individuals scheduled for tympanoplasty[54]. *Said M. Said Al-Jaaf* et al also said that success of tympanoplasty is higher in non smokers than smokers[55].

The patients without tympanic membrane perforation had better graft uptake in the absence of other significant middle ear pathology. Many studies have concluded that the rate of graft uptake is lesser with anterior perforations than posterior perforations. This is due to lesser blood supply to anterior part of the drumhead and lesser surgical access to the anterior part. *Frade Gonzalez C* said that there is better success rate in posterior perforations than subtotal perforations[56]. Onal's study also stated that graft uptake is better in posterior than anterior perforations and smaller perforations have higher uptake of graft than larger perforations[57]. However some studies state that the size of the perforation is not as significant as the location of the perforation. There is also

a view suggesting that hearing loss due to a small perforation is small and is mainly due to other factors. Hence patients have smaller improvement in hearing after closure of such perforations. According to the studies of *Asok K. Saha*, D. M. Munsi, S. N. Ghosh the presence of bilateral perforations lowers the success rate. They said that the status of non-operated ear is a significant factor for success rate. The status of contralateral ear is an important deciding factor for surgery especially in children. If contralateral ear is affected, adenoidectomy should be done and tympanoplasty is avoided until seven years of age[58].

Cholesteatoma is associated with reduced rate of graft uptake and hearing benefit. Generally canal wall down procedure is done for extensive cholesteatoma which is associated with lesser hearing benefit. There is higher rate of recurrence especially if there is cholesteatoma in inaccesible sites such as sinus tympani, fallopian canal, etc. This in turn affects the outcome. *Stankovic* stated that recurrence of cholesteatoma is one of the causes for diminished postoperative hearing[59]. In patients with extensive cholesteatoma, it is advisable to stage the procedure. Cholesteatoma recurrence is more than twice common in children than adults according to *Stankovic M*[60]. The choice of surgery whether to go for open or closed canal wall technique is base on the type and extent of disease. A closed technique is better for attic and sinus cholesteatomas, while in pars tensa cholesteatomas, an open technique is preferable. Also *closed technique is preferable in children.*

A.Sethi et al studied on the correlation between pneumatisation of mastoid

and success of surgery. He stated that there is no significant relation between pneumatisation of mastoid and success in terms of graft uptake and hearing benefit[61].

In our study, hearing benefit is observed in 70% patients. Hearing benefit is assessed by various methods. According to Belfast rule of thumb, postoperative hearing benefit is significant if air conduction threshold in speech frequency range is ≤ 30 dB or if interaural difference is ≤ 15 dB. Some authors define success as postoperative air-bone gap within 20 dB. In some studies, only the air conduction threshold is taken into account. In our study, we have measured the average air - bone gap closure at speech frequencies (500Hz, 1 kHz, 2 kHz and 3 kHz) based on the guidelines given by the committee on hearing and equilibrium, American academy of otolaryngology head and neck surgery.

According to the studies conducted by *Asok K Saha* et al type l tympanoplasty with simple mastoidectomy results excellent surgical success rate (100%) but gives less improvement of hearing (closure of A-B gap= 3.3db). In type I tympanoplasty alone (without mastoidectomy) surgical success rate drops to 80-75% but is offers more improvement of hearing (closure of A-B gap = 6.708 db)[58]. *Siddharth Nirwan and K. G. Somashekara* calculated hearing benefit based on pre and postoperative air - bone gap. They concluded that postoperative hearing was better in patients with tubotympanic disease than those operated with atticoantral disease, in whom autologous incus was sculpted and used as ossicular graft compared to autologous conchal cartilage and where mastoidectomy with tympanoplasty were performed than tympanoplasty

alone. Also, better hearing gain was obtained in the cortical mastoidectomy group as compared to modified radical mastoidecomy group[62].

SUMMARY

This prospective study was conducted in our department for a period of two years with an objective to find the role of middle ear risk index on the outcome of tympanoplasty in terms of graft uptake and hearing benefit.

- The study group comprises 50 patients, of which 19 were males and 31 were females. Most of them belong to the lower middle class, with malnutrition and poor literacy.
- 68% of our study group belongs to 11 to 30 years of age.
- Pseudomonas aeruginosa is the most common pathogen isolated from our study group.
- Most patients with severe MERI have squamous type CSOM (14 patients). Among mucosal type, very few patients (3 patients) have severe MERI.
- MERI is a useful tool to ascertain the prognosis of tympanoplasty.
- Higher the MERI score, lower is the rate of graft taken up in patients and patients with lower MERI have higher rate of graft uptake.
- Higher the MERI score, lower is the patient's chances of hearing to improve. Similarly, patients with low MERI scores have greater hearing benefit after surgery. The mean score of hearing benefit among patients is 9.90 dB.
- For patients with higher MERI score, it is advisable to stage the surgical procedure.

- MERI score helps us to determine whether to proceed with canal wall up or down mastoidectomy.
- It helps to stratify the patients into similar risk groups which in turn can be used to compare the efficacy of different surgical techniques or prosthetic materials.

CONCLUSION

- MERI score is a useful measure of the extent of disease in the middle ear.
- MERI is a good prognostic factor for hearing benefit after surgery.
- Based on the MERI score, the goals of surgery should be determined and the extent to which surgical success and hearing benefit can be obtained should be explained to the patient.
- Attempts should be made to reduce the middle ear disease whenever possible (otorrhoea and granulation) preoperatively to improve the success rate of tympanoplasty.

BIBLIOGRAPHY

1. Aristides Athanasiadis-Sismanis. Tympanoplasty: Tympanic membrane repair. A. In: Julianna Gulya, Lloyd B. Minor, Dennis S. Poe. (eds) *Glasscock-Shambaugh Surgery of the ear.* 6th Edn. Connecticut: People's medical publishing house; 2010. p.466.

2. Wullstein H. Focal infection as etiological factor of hard of hearing. Z Laryngol Rhinol Otol 1952;31:541-5.

3. Austin D. Reporting results in Tympanoplasty. Am J Otol 1985;6:85-8.

4. Kartush JM. Ossicular Chain Reconstruction: capitulum to alleus. Otolaryngol Clin North Am 1994;27:689-715.

5. Becvarovski Z, Kartush JM. Smoking and tympanoplasty: implications for prognosis and the Middle Ear Risk Index (MERI). Laryngoscope 2001;111:1806-11.

6. Bernard M. P. J. Ars, Nicole M, Ars-Piret. Morphofunctional Partition of the Middle Ear Cleft. The Mediterranean Journal of Otology. Mediterr J Otol 2007;3:31-39.

7. Tony Wright, Peter Valentine. The anatomy and embryology of external and middle ear. In: Michael Gleeson. (ed) Scott Brown's Otorhinolaryngology, Head and Neck Surgery 7th edn. London: Hodder Arnold;2008. pp3111.

8. Hopf J, Linnarz M, Gundlach P et al. Microendoscopy of the Eustachian tube and the middle ear. Indications and clinical application. Laryngorhinootologie1991;70:391-4.

9. Tony Wright. Applied anatomy and physiology of the ear. In: Harold Ludman, Tony Wright. Diseases of the ear. 6th edn. London: Hodder Arnold; 2006. pp32-47.

10. Vijayshree Nahata, Chandrakant Y Patil, Rashmi K Patil, Gaurav Gattani, Ashish Disawal, Amitava Roy. Tympanic membrane perforation: Its correlation with hearing loss and frequency affected - An analytical study. Indian Journal of otology. 2014;20 (1):10-15.

11. Abhishek Maheshwari, Rajlaxmi Panigrahi, Shruti Mahajan. Comparison of Temporalis Fascia with Tragal Cartilage-Perichondrium (Composite Graft) as a Grafting Material in Type I Tympanoplasty: A Prospective Randomized Study. International Journal of Scientific Study. April 2015; 3(1):29-34.

12. Berthold E. Uebermyringoplastik. Wier Med Bull. 1878;1:627.

13. Nylen CO. The microscope in aural surgery, its first use and later development. Acta Otolaryng, Suppl 1954;116:226-40.

14. Wullstein H. The restoration of the function of the middle ear in chronic otitis media. Ann Otol Rhinol Laryngol. 1971;80(2):210–217.

15. Zollner F. The principles of plastic surgery of the sound-conducting apparatus. J Laryngol Otol.1955;69:637. doi: 10.1017/S0022215100051240.

16. Heermann H. Tympanoplasty with fascial tissue taken from the temporal muscle after straightening the anterior wall of the auditory meatus. HNO 1961;9:136-7.

17. R J Bellucci. Selection of Cases and Classification of Tympanoplasty Otolaryngologic Clinics of North America (Impact Factor: 1.34). 11/1989; 22(5):911-26.

18. Black B. Ossiculoplasty prognosis: the spite method of assessment. Am J Otol. 1992 Nov;13(6):544–551.

19. Dornhoffer JL, Gardner E. Prognostic factors in ossiculoplasty: a statistical staging system. Otology and Neurotology. 2001;22:299–304.

20. James L, Sheehy, Robert G.Andrerson. Myringoplasty - A Review of 472 cases. Ann otol 89; 1980;331-334.

21. Warren Y. Adkins, Benjamin White, Charleston SC. Type 1 tympanoplasty : influencing factors. The Laryngoscope July 1984; 916-919.

22. Karthush, Jack M. Michaelides, Elias M. Becvarouski, Zoran La Rouere. Michael. Over-under tympanoplasty. The American laryngological Rhinological and Otological society inc. May 2002; 112(5):802-7.

23. Debora Bunzen, Alexandre Campos, Fbiana Sperandio, Silvio Caldas Neto. Intraoperative findings influence in Myringoplasty anatomical result. 2006. 10(4):391-5.

24. Emir, Hatice, Ceylan, Kursat, Kizilkaya, Zeynep, Gecmen, Hakan, Uzunkulaoglu, Hakki Samin, Erdal. Influencing factors on Type 1 tympanoplasty. Europeon archives of Oto-Rhino-Laryngology. June 2007;264(6): 595-599.

25. Jyothi P Dabalkar, Krishna Vora, Abhik Sikdar**.** Comparative study of underlay tympanoplasty with temporalis fascia and tragal perichondrium. Indian J Otolaryngol Head and Neck surgery. April-June 2007. 59:116-9.

26. Khalid Almazrou, Munahi Alqahtani, Mohamed Alshehabi. Middle ear risk index as a prognostic factor in pediatric ossicular reconstruction. Indian journal of otology. 2013; 19(1):23-26.

27. Min-Beom Kim et al. Hearing Outcomes According to the Types of

Mastoidectomy: A Comparison between Canal Wall Up and Canal Wall Down Mastoidectomy. Clin Exp Otorhinolaryngol. 2010 Dec; 3(4): 203–206.

28. Pinar E, Sadullahoglu K, Calli C, Oncel S. Evaluation of prognostic factors and middle ear risk index in tympanoplasty. Otolaryngol Head Neck Surg. 2008 Sep;139(3):386-90.

29. Uygar Levent Demir, Sait Karaca, Omer Afsin Ozmen, Fikret Kasapoglu, Hakan Coskun, Oguz Basut. Is it the Middle Ear Disease or the Reconstruction Material That Determines the Functional Outcome in Ossicular Chain Reconstruction? Otology & neurotology 03/2012; 33(4):580-5.

30. Sevim Aslan Felek, Hatice Celik, Ahmet Islam, Atilla H. Elhan, Munir Demirci, Erdal Samim. Type 2 ossiculoplasty: prognostic determination of hearing results by

middle ear risk index. American journal of Otolaryngology head and neck medicine and surgery.2010;3(5):325-331.

31. Ankush Sayal, Virangna Taneja, Achal Gulati. Preliminary Hearing Results of Tympanomastoidectomies Using Titanium Prostheses: Scenario in a Developing Country. International Journal of Otolaryngology and Head & Neck Surgery. 2013;2:195-200.

32. Sushil Jha, Keyur Mehta, Viral Prajapati, Deepak Patel, Pramod Kharadi. A Comparative Study of Ossiculoplasty by Using Various Graft Materials. NJIRM. 2011;2(4):53-55.

33. Viktor Chrobok, Arnost Pellant, Milan Meloun, Karel Pokorny, Eva Simáková, Petra Mandysová. Prognostic Factors for Hearing Preservation in Surgery of Chronic Otitis Media. Int. Adv. Otol. 2009; 5:(3) 310-317.

34. Rakesh Saboo, Amit Modwal, Priyanjal Gautam. Tubotympanic CSOM: clinical profile of perforation, eustachian tube function and MERI score as parameter for outcome of tympanoplasty. Transworld Medical Journal. 2(2):154159.

35. Hassan Abshirini, Nader Saki, Soheila Nikakhlagh, Malihe Yavari. Evaluation of Prognostic Factors in Tympanoplasty. Biosci., Biotech. Res. Asia. 2015;12(1):37-41.

36. Jose Acuin. Chronic suppurative otitis media Burden of Illness and Management Options. Geneva: World Health Organisation;2004. Child and Adolescent Health and Development Prevention of Blindness and Deafness.

37. Shrikrishna BH, Jyothi AC, Sanjay G, Sandeep Samson G. Age and Gender Differences in the Incidence of Non-Cholesteatomatous Chronic Suppurative Otitis Media. International Journal of Research in Pharmaceutical and Biomedical Sciences. 2013;4(4):1172-4.

38. Bijan Basak, Ganesh Chandra Gayen, Munmun Das (Sarkar), Gautam Dhar, Ritam Ray, Atanu Kumar Das. Demographic profile of CSOM in a rural tertiary care hospital. IOSR Journal Of Pharmacy. June 2014;4(6):43-46.

39. Abhinav D Wankar, Sanjiv Golhar. To Determine Prevalence of Chronic Suppurative Otitis Media with Reference to Unsafe Otitis Media in Primary School Going Children of Rural Setup of Wardha District.Global Journal of Medical research: J Dentistry and Otolaryngology. 2014;14(1):27-35.

40. Harvinder kumar et al. Bacterial & fungal study of 100 cases of CSOM. Journal of clinical & diagnostic research. nov 2011(suppl-1);5(6):1224-7.

41. Prakash M et al. Bacteriological profile and their antibiotic susceptibility pattern of cases of chronic suppurative otitis media. Asian journal of pharmaceutical & clinical research. 2013;6(3).

42. Shamweel Ahmed. Antibiotics in chronic Suppurative Otitis media: A bacteriological Study. Egyptian Journal of Ear, nose , throat & allied sciences. Nov 2013;14(3):191-4.

43. John L. Dornhoffer, Edward Gardner. Prognostic Factors in Ossiculoplasty: A Statistical Staging System. Otology & Neurotology 2001;3(22):299–304.

44. Gersdorff M, Garin P, Decat M, Juantegui M. Myringoplasty: long-term results in adults and children. Am J Otol 1995;16(4):215-8.

45. Manpreet Kaur et al. Comparative Evaluation between Tympanoplasty Alone & Tympanoplasty Combined With Cortical Mastoidectomy in NonCholesteatomatous Chronic Suppurative Otitis Media in Patients with Sclerotic Bone. IOSR Journal of Dental and Medical Sciences. Jun. 2014;13(6):40-45.

46. Andersen SA, Aabenhus K, Glad H, Sorensen MS. Graft take-rates after tympanoplasty: results from a prospective ear surgery database. Otol Neurotol. 2014 Dec;35(10):e292-7.

47. P.Harikrishnan et al. Clinical study of influence of prognostic factor on the outcome of tympanoplasty surgery. IOSR journal of Dental & medical sciences. 2013;5(6):41-45.

48. Ghodrat Mohammadi, Masoud Naderpour, Mehrnoosh Mousaviagdas. Ossicular Erosion in Patients Requiring Surgery for Cholesteatoma. Iran J Otorhinolaryngol. 2012; 24(68): 125–8.

49. Kevin F. Wilson, Nyall R. London, Clough Shelton. Tympanoplasty with intact canal wall mastoidectomy for cholesteatoma: Long-term hearing outcomes. The Laryngoscope. 2013;123(12) :3168–3171.

50. Manas Ranjan Rout, Pakeer Das, Deeganta Mohanty, Venkateswar Rao, Karri Susritha, Bolla Eswari Siva Jyothi. Ossicular chain defects in safe type of chronic suppurative otitis media. Indian journal of otology.2014;20(3):102-5.

51. Bernard Ars, Nicole Ars-Piret. Tympano-Ossicular, Allograft Tympanoplasty: A Manual of Techniques. Tympano-ossicular allografts and middle ear pathology Newyork: Kugler publications; 1993.8:49.

52. Veysel Yurttafl, Ahmet Ural, Ahmet Kutluhan, Kaz›m Bozdemir. Factors that may affect graft success in tympanoplasty with mastoidectomy. ENT Updates 2015;5(1):9–12.

53. Da Costa SS, Paparella MM, Schachern PA, Yoon TH, Kimberley BP. Temporal bone histopathology in chronically infected ears with intact and perforated tympanic membranes. Laryngoscope 1992; 102: 1229-36.

54. Lin YC, Wang WH, Weng HH. Predictors of surgical and hearing long-term results for inlay cartilage tympanoplasty. Arch Otolaryngol Head Neck Surg. 2011 Mar;137(3):215-9.

55. Said M. Said Al-Jaaf, Arsalan A, Mustafa Shem, Lana Abdulrazak Dabbagh.Zanco J. Myringoplasty [Risk factors and the effect of post operative follow up on the outcome of success rate in Erbil / Kurdistan of Iraq Med. Sci. 2010;14(3):1-9.

56. Frade Gonzalez C, Castro Vilas C, Cabanas Rodriguez E, Elhendi W, Vaamonde Lago P, Labella Caballero T. Prognostic factors influencing anatomic and functional outcome in myringoplasty. Acta Otorhinolaryngol Esp. 2002 Dec; 53(10):729-35.

57. Onal K, Uguz MZ, Kazikdas KC, Gursoy ST, Gokce H. A multivariate analysis of otological, surgical and patient related factors in determining success in myringoplasty. Clin Otolaryngol. 2005 Apr ;30(2):115-20.

58. Asok K. Saha, D. M. Munsi, S. N. Ghosh. Evaluation of improvement of hearing in type I tympanoplasty & its influencing factors. Indian Journal of Otolaryngology and Head and Neck Surgery. July 2006;58(3):253-7.

59. Stankovic. Audiologic results of surgery for cholesteatoma: short- and long- term follow-up of influential factors. Otol Neurotol. 2008 Oct;29(7):933-40.

60. Stankovic M. Follow-Up of Cholesteatoma Surgery: Open versus Closed Tympanoplasty. ORL 2007;69:299–305.

61. A.Sethi, I. Singh, A.K.Agarwal, D.Sareen. Pneumatisation correlated to myringoplasty and tubal function. Indian journal of otolaryngology and head and neck surgery. 2005;57(4):283-6.

62. Siddharth Nirwan, K. G. Somashekara. A study of hearing improvement after tympanoplasty by means of pure tone audiometry. Journal of evidence based medicine and healthcare. 2015;2(2):113-122.

PROFORMA

NAME

AGE/SEX

OP/IP NUMBER

ADDRESS WITH PHONE NO.

CHIEF COMPLAINTS

HISTORY OF PRESENTING ILLNESS

1. EAR DISCHARGE

- Side
- Duration
- Dry / Occasionally wet/ Persistently wet / Wet with cleft palate
- Nature
- Amount
- Whether foul smelling
- Whether blood stained
- Aggravating / Relieving factors
- Period of dryness

2. HARD OF HEARING

- Side
- Duration
- Onset
- Progressive / Static / Fluctuant

3. EAR PAIN

Side Duration

4. Tinnitus

5. Giddiness

6. Nasal obstruction

Side Duration

7. Sneezing

8. Nasal discharge

9. Headache

10.Throat pain

11.Pain during swallowing

PAST HISTORY

- Trauma
- Cleft palate

PERSONAL HISTORY

Smoking

TREATMENT HISTORY

- Previous surgeries
- Ear drops / Ototoxic drugs

GENERAL EXAMINATION

VITALS

PR BP

SYSTEMIC EXAMINATION

CVS RS

ABDOMEN CNS

EXAMINATION OF ENT RIGHT LEFT

EAR

Preauricular region

Pinna

Postauricular region

External Auditory Canal

Tympanic membrane

Perforation Present / Absent

Cholesteatoma Present / Absent

Attic

Middle ear mucosa

Facial nerve

Mastoid tenderness

Fistula Test

TUNING FORK TEST RIGHT LEFT

Rinne

256 Hz

512 Hz

1024 Hz

Weber

ABC

NOSE AND PARANASAL SINUSES

THROAT

INVESTIGATIONS

- Blood investigations
- Aural swab culture and sensitivity
- Pure Tone Audiogram
- Impedance Audiometry
- CT Temporal Bone
- Otoendoscopy and Otomicroscopy

MIDDLE EAR FINDINGS

Granulation / Effusion / Cholesteatoma

OSSICLES

- Malleus - Intact / Eroded
- Incus - Intact / Eroded
- Stapes - Intact / Eroded
- Ossicle head fixation
- Stapes fixation

MERI SCORE

PROCEDURE DETAILS

POST OPERATIVE FOLLOWUP

1st visit

2nd visit

3rd visit

CONDITION OF GRAFT

POSTOPERATIVE PTA

HEARING BENEFIT

S. NO	NAME	AGE	SEX	SIDE	MUCOSAL / SQUAMOUS	DISCHARGE	SMOKING (ACTIVE/ PASSIVE)	TYMPANIC MEMBRANE	GRANULATION / EFFUSION	PERFORATION	CHOLESTEATOMA	AURAL SWAB C/S	OSSICLES	PREOP A-B GAP	PROCEDURE	POST OP A-B GAP	MERI SCORE	HEARING BENEFIT	GRAFT STATUS
1	JEYARANI	43	F	LEFT	MUCOSAL	OCCASIONALLY WET	NO	MODERATE CENTRAL PERFORATION IN ANTEROINFERIOR AND POSTEROINFERIOR QUADRANTS	NO	YES	NO	NO GROWTH	M+ I+ S+	28	LEFT SIMPLE MASTOIDECTOMY WITH TYPE 1 TYMPANOPLASTY	16	2	12	TAKEN UP
2	FLORAMARY	29	F	LEFT	MUCOSAL	OCCASIONALLY WET	NO	MODERATE CENTRAL PERFORATION IN ANTEROINFERIOR AND POSTEROINFERIOR QUADRANTS	NO	YES	NO	PSEUDOMONAS	M+ I+ S+	20	LEFT SIMPLE MASTOIDECTOMY WITH TYPE 1 TYMPANOPLASTY	15	2	5	TAKEN UP
3	LALITHA	34	F	LEFT	MUCOSAL	OCCASIONALLY WET	NO	MODERATE CENTRAL PERFORATION IN ANTEROINFERIOR QUADRANT	YES (GRANULATION)	YES	NO	PSEUDOMONAS	M+ I- S-	34.5	LEFT MRM WITH TYPE 3 TYMPANOPLASTY	30.5	6	4	TAKEN UP
4	DHIVYA	20	F	LEFT	MUCOSAL	OCCASIONALLY WET	NO	MODERATE CENTRAL PERFORATION IN ANTEROINFERIOR QUADRANT	NO	YES	NO	PROTEUS	M+ I- S+	38.75	ENDOSCOPIC TYPE 3 TYMPANOPLASTY	38.25	3	NO	REJECTED
5	MALARKODI	30	F	RIGHT	MUCOSAL	DRY	NO	LARGE CENTRAL PERFORATION	YES (GRANULATION)	YES	NO	NO GROWTH	M+ I- S+	50.25	RIGHT MRM WITH TYPE 3 TYMPANOPLASTY	21.25	4	29	TAKEN UP
6	HARIKRISHNAN	15	M	RIGHT	SQUAMOUS	PERSISTENTLY WET	NO	RETRACTION POCKET WITH CHOLESTEATOMA IN POSTEROSUPERIOR QUADRANT	NO	NO	YES	NO GROWTH	M- I- S+	54	RIGHT MRM WITH TYPE 3 TYMPANOPLASTY WITH FACIAL NERVE DECOMPRESSION	38	7	16	TAKEN UP
7	KUMARESAN	22	M	RIGHT	MUCOSAL	OCCASIONALLY WET	YES	SUBTOTAL PERFORATION	YES (GRANULATION)	YES	NO	PSEUDOMONAS	M+ I+ S+	35	RIGHT SIMPLE MASTOIDECTOMY WITH TYPE 1 TYMPANOPLASTY	35.5	6	NO	PERFORATED
8	GUNASEKARAN	40	M	LEFT	MUCOSAL	OCCASIONALLY WET	NO	LARGE CENTRAL PERFORATION	YES (GRANULATION)	YES	NO	PSEUDOMONAS	M+ I+ S+	32.5	LEFT SIMPLE MASTOIDECTOMY WITH TYPE 1 TYMPANOPLASTY	17.5	4	15	TAKEN UP
9	VEERAMMAL	33	F	LEFT	SQUAMOUS	OCCASIONALLY WET	NO	GRANULATION TISSUE IN POSTEROSUPERIOR QUADRANT	YES (GRANULATION)	NO	YES	STAPHYLOCCUS AUREUS	M- I- S-	18.75	ENDOSCOPIC ATTICOTOMY WITH TYPE 3 TYMPANOPLASTY	20	9	NO	REJECTED
10	SRIVIDHYA	6	F	LEFT	SQUAMOUS	OCCASIONALLY WET	NO	RETRACTED AND ADHERED TO PROMONTARY	YES (GRANULATION)	NO	YES	ENTEROBACTER	M+ I- S+	40.5	LEFT MRM WITH TYPE 3 TYMPANOPLASTY	27.5	6	13	TAKEN UP
11	MANIMARAN	15	M	RIGHT	MUCOSAL	OCCASIONALLY WET	NO	LARGE CENTRAL PERFORATION	YES (GRANULATION AND EFFUSION)	YES	NO	STAPHYLOCCUS AUREUS	M+ I+ S+	38.75	RIGHT ENDOSCOPIC TYPE 1 TYMPANOPLASTY	42	4	NO	REJECTED
12	CHANDRA	28	F	RIGHT	MUCOSAL	PERSISTENTLY WET	NO	LARGE CENTRAL PERFORATION	YES (GRANULATION)	YES	NO	ESCHERICHIA COLI	M+ I- S+	28.75	RIGHT MRM WITH TYPE 3 TYMPANOPLASTY	23.75	6	5	TAKEN UP
13	GANDHIMARY	40	F	LEFT	MUCOSAL	PERSISTENTLY WET	NO	LARGE CENTRAL PERFORATION	YES (GRANULATION)	YES	NO	STAPHYLOCCUS AUREUS	M- I- S+	41.25	LEFT MRM WITH TYPE 3 TYMPANOPLASTY	45	8	NO	REJECTED
14	SHARMILA	18	F	LEFT	MUCOSAL	OCCASIONALLY WET	NO	MODERATE CENTRAL PERFORATION IN ANTEROINFERIOR AND POSTEROINFERIOR QUADRANTS	NO	YES	NO	PSEUDOMONAS	M+ I+ S+	32.75	LEFT SIMPLE MASTOIDECTOMY WITH TYPE 1 TYMPANOPLASTY	8.75	2	24	TAKEN UP
15	MADHAVAN	8	M	LEFT	SQUAMOUS	PERSISTENTLY WET	NO	RETRACTION POCKET WITH CHOLESTEATOMA IN POSTEROSUPERIOR QUADRANT	NO	NO	YES	PROTEUS	M+ I- S-	35	LEFT MRM WITH TYPE 3 TYMPANOPLASTY	35	6	NO	PERFORATED
16	SUGANYA	22	F	LEFT	SQUAMOUS	PERSISTENTLY WET	NO	CHOLESTEATOMA IN POSTEROSUPERIOR QUADRANT WITH SMALL CENTRAL PERFORATION IN ANTEROINFERIOR QUADRANT	YES (GRANULATION)	YES	YES	PSEUDOMONAS	M- I- S-	56.25	LEFT MRM WITH TYPE 3 TYMPANOPLASTY	59.5	11	NO	REJECTED
17	INBAM	42	F	RIGHT	SQUAMOUS	OCCASIONALLY WET	NO	SUBTOTAL PERFORATION WITH GRANULATION IN POSTEROSUPERIOR QUADRANT	YES (GRANULATION)	YES	NO	NO GROWTH	M+ I- S-	62	RIGHT MRM WITH TYPE 3 TYMPANOPLASTY	62	6	NO	TAKEN UP
18	MANIGANDAN	14	M	RIGHT	SQUAMOUS	OCCASIONALLY WET	NO	MEMBRANE ADHERED TO PROMONTARY, GRANULATION IN POSTEROINFERIOR QUADRANT	YES (GRANULATION)	NO	NO	ENTEROCOCCUS	M+ I+ S+	43.75	RIGHT SIMPLE MASTOIDECTOMY WITH TYPE 1 TYMPANOPLASTY	22.75	3	21	TAKEN UP
19	NAGARAJAN	29	M	LEFT	MUCOSAL	OCCASIONALLY WET	NO	LARGE CENTRAL PERFORATION	NO	YES	NO	PSEUDOMONAS	M+ I+ S+	30.25	LEFT SIMPLE MASTOIDECTOMY WITH TYPE 1 TYMPANOPLASTY	6.25	2	24	TAKEN UP
20	PUSHPALATHA	40	F	LEFT	MUCOSAL	OCCASIONALLY WET	NO	LARGE CENTRAL PERFORATION	YES (GRANULATION)	YES	NO	STAPHYLOCCUS AUREUS	M+ I+ S+	48.75	LEFT SIMPLE MASTOIDECTOMY WITH TYPE 1 TYMPANOPLASTY	26.75	4	22	TAKEN UP
21	RAMYA	18	F	LEFT	MUCOSAL	OCCASIONALLY WET	NO	SMALL CENTRAL PERFORATION IN ANTEROINFERIOR QUADRANT	NO	YES	YES	ENTEROCOCCUS	M- I- S+	31.25	LEFT MRM WITH TYPE 3 TYMPANOPLASTY	13.25	7	18	TAKEN UP
22	DIVYADARSHINI	11	F	RIGHT	SQUAMOUS	PERSISTENTLY WET	NO	AURAL POLYP	YES (GRANULATION)	YES	YES	STAPHYLOCCUS AUREUS	M+ I- S-	49	RIGHT MRM WITH TYPE 3 TYMPANOPLASTY	45	9	4	TAKEN UP
23	BHUVANESWARI	30	F	LEFT	MUCOSAL	OCCASIONALLY WET	NO	SUBTOTAL PERFORATION	NO	YES	NO	NO GROWTH	M+ I+ S+	32.5	LEFT SIMPLE MASTOIDECTOMY WITH TYPE 1 TYMPANOPLASTY	17.5	2	15	TAKEN UP
24	ARUNADEVI	15	F	LEFT	MUCOSAL	OCCASIONALLY WET	NO	MODERATE CENTRAL PERFORATION INVOLVING POSTEROINFERIOR QUADRANT	YES (GRANULATION)	YES	NO	PSEUDOMONAS	M+ I+ S+	25	LEFT SIMPLE MASTOIDECTOMY WITH TYPE 1 TYMPANOPLASTY	16	4	9	TAKEN UP
25	YOGESH	11	M	LEFT	SQUAMOUS	PERSISTENTLY WET	NO	ATTIC PERFORATION	YES (GRANULATION)	YES	NO	ENTEROCOCCUS	M- I- S+	35	LEFT MRM WITH TYPE 3 TYMPANOPLASTY	26	8	9	TAKEN UP

26	ANANDRAJ	24	M	LEFT	SQUAMOUS	PERSISTENTLY WET	YES	ATTIC PERFORATION	YES (GRANULATION)	YES	YES	PROTEUS	M+ I- S+	42.5	LEFT MRM WITH TYPE 3 TYMPANOPLASTY	44.75	10	NO	REJECTED
27	JAYARANI	37	F	RIGHT	MUCOSAL	OCCASIONALLY WET	NO	MODERATE CENTRAL PERFORATION INVOLVING POSTEROINFERIOR AND ANTEROINFERIOR QUADRANTS	NO	YES	NO	NO GROWTH	M+ I+ S+	35.25	RIGHT SIMPLE MASTOIDECTOMY WITH TYPE 1 TYMPANOPLASTY	14.25	2	21	TAKEN UP
28	KARTHIKEYAN	11	M	LEFT	SQUAMOUS	PERSISTENTLY WET	NO	POSTEROSUPERIOR MARGINAL PERFORATION	NO	YES	YES	ENTEROCOCCUS	M+ I- S+	38.75	LEFT MRM WITH TYPE 3 TYMPANOPLASTY	31.75	6	7	TAKEN UP
29	TAMILALAGI	27	F	RIGHT	MUCOSAL	PERSISTENTLY WET	NO	SMALL CENTRAL PERFORATION IN ANTEROINFERIOR QUADRANT	YES (GRANULATION)	YES	NO	ENTEROCOCCUS	M+ I+ S+	34.5	RIGHT SIMPLE MASTOIDECTOMY WITH TYPE 1 TYMPANOPLASTY	17.5	5	17	TAKEN UP
30	SURYA	18	M	RIGHT	SQUAMOUS	PERSISTENTLY WET	NO	ATTIC PERFORATION	YES (GRANULATION)	YES	YES	PROTEUS	M+ I+ S+	31	RIGHT SIMPLE MASTOIDECTOMY WITH TYPE 1 TYMPANOPLASTY	28	7	3	TAKEN UP
31	SARAVANAN	19	M	LEFT	SQUAMOUS	PERSISTENTLY WET	NO	GRANULATION TISSUE IN POSTEROSUPERIOR QUADRANT	YES (GRANULATION)	NO	NO	ESCHERICHIA COLI	M+ I- S-	54.5	LEFT MRM WITH TYPE 3 TYMPANOPLASTY	44.5	8	10	TAKEN UP
32	RAVIVARMAN	15	M	LEFT	SQUAMOUS	OCCASIONALLY WET	NO	GRANULATION TISSUE IN POSTEROSUPERIOR QUADRANT	YES (GRANULATION)	NO	YES	ESCHERICHIA COLI	M- I- S+	48.25	LEFT MRM WITH TYPE 3 TYMPANOPLASTY	50.75	8	NO	TAKEN UP
33	DHANALAKSHMI	48	F	LEFT	MUCOSAL	OCCASIONALLY WET	NO	LARGE CENTRAL PERFORATION	NO	YES	NO	NO GROWTH	M+ I+ S+	33	LEFT SIMPLE MASTOIDECTOMY WITH TYPE 1 TYMPANOPLASTY	21	2	12	TAKEN UP
34	ABBAS	22	M	LEFT	SQUAMOUS	PERSISTENTLY WET	NO	AURAL POLYP	YES (GRANULATION)	YES	YES	ACINETOBACTER	M- I- S-	72.75	LEFT MRM WITH TYPE 3 TYMPANOPLASTY	74.25	11	NO	REJECTED
35	KARTHIGA	21	F	RIGHT	MUCOSAL	OCCASIONALLY WET	NO	SMALL CENTRAL PERFORATION IN ANTEROINFERIOR QUADRANT	NO	YES	NO	PSEUDOMONAS	M+ I- S+	37.25	LEFT MRM WITH TYPE 3 TYMPANOPLASTY	19.25	3	18	TAKEN UP
36	KAVITHA	16	F	LEFT	MUCOSAL	OCCASIONALLY WET	NO	MODERATE CENTRAL PERFORATION IN ANTEROINFERIOR QUADRANT	YES (GRANULATION)	YES	NO	PROTEUS	M+ I+ S+	29	LEFT SIMPLE MASTOIDECTOMY WITH TYPE 1 TYMPANOPLASTY	17	4	12	TAKEN UP
37	RAJENDRAN	32	M	RIGHT	SQUAMOUS	PERSISTENTLY WET	NO	MODERATE CENTRAL PERFORATION WITH RETRACTION POCKET	NO	YES	NO	PSEUDOMONAS	M+ I+ S+	38	RIGHT SIMPLE MASTOIDECTOMY WITH TYPE 1 TYMPANOPLASTY	16	3	22	TAKEN UP
38	JEEVITHA	24	F	LEFT	MUCOSAL	OCCASIONALLY WET	NO	MODERATE CENTRAL PERFORATION IN ANTEROINFERIOR AND POSTEROINFERIOR QUADRANTS	NO	YES	NO	STAPHYLOCCUS AUREUS	M+ I+ S+	37.5	LEFT SIMPLE MASTOIDECTOMY WITH TYPE 1 TYMPANOPLASTY	14.5	2	23	TAKEN UP
39	PARTHIBAN	27	M	LEFT	MUCOSAL	OCCASIONALLY WET	NO	SUBTOTAL PERFORATION	NO	YES	YES	PROTEUS	M+ I- S+	43.25	LEFT MRM WITH TYPE 3 TYMPANOPLASTY	17.25	5	26	TAKEN UP
40	SUNDARAMBAL	47	F	LEFT	SQUAMOUS	PERSISTENTLY WET	NO	CHOLESTEATOMA IN POSTEROSUPERIOR QUADRANT	YES (GRANULATION)	NO	YES	ESCHERICHIA COLI	M- I- S+	63.5	LEFT MRM WITH TYPE 3 TYMPANOPLASTY	58.5	9	5	TAKEN UP
41	LAKSHMI	21	F	LEFT	MUCOSAL	OCCASIONALLY WET	NO	MODERATE CENTRAL PERFORATION IN ANTEROINFERIOR AND POSTEROINFERIOR QUADRANTS	NO	YES	NO	PSEUDOMONAS	M+ I+ S+	26	LEFT SIMPLE MASTOIDECTOMY WITH TYPE 1 TYMPANOPLASTY	18	2	8	TAKEN UP
42	TAMILSELVAN	12	M	RIGHT	SQUAMOUS	PERSISTENTLY WET	NO	GRANULATION TISSUE IN POSTEROSUPERIOR AND ANTEROSUPERIOR QUADRANT	YES (GRANULATION)	NO	NO	NO GROWTH	M+ I- S-	49.5	RIGHT MRM WITH TYPE 3 TYMPANOPLASTY	51.25	6	NO	TAKEN UP
43	SUNDARI	28	F	LEFT	MUCOSAL	OCCASIONALLY WET	NO	SUBTOTAL PERFORATION	YES (GRANULATION)	YES	NO	ENTEROCOCCUS	M+ I- S+	35	LEFT MRM WITH TYPE 3 TYMPANOPLASTY	19	7	16	TAKEN UP
44	PUNITHA	33	F	RIGHT	MUCOSAL	OCCASIONALLY WET	NO	SMALL CENTRAL PERFORATION IN ANTEROINFERIOR QUADRANT	YES (GRANULATION)	YES	NO	PSEUDOMONAS	M+ I+ S+	25.5	RIGHT SIMPLE MASTOIDECTOMY WITH TYPE 1 TYMPANOPLASTY	16.5	4	9	TAKEN UP
45	ANITHA	18	F	LEFT	MUCOSAL	DRY	NO	MODERATE CENTRAL PERFORATION IN ANTEROINFERIOR AND POSTEROINFERIOR QUADRANTS	NO	YES	NO	NO GROWTH	M+ I+ S+	32.75	LEFT TYPE 1 TYMPANOPLASTY	17.75	1	15	TAKEN UP
46	LOKESH	9	M	LEFT	SQUAMOUS	PERSISTENTLY WET	NO	AURAL POLYP	YES (GRANULATION)	YES	NO	ENTEROBACTER	M- I- S+	54	LEFT MRM WITH TYPE 3 TYMPANOPLASTY	46	8	8	TAKEN UP
47	AROKYAMARY	25	F	RIGHT	MUCOSAL	PERSISTENTLY WET	NO	SUBTOTAL PERFORATION	NO	YES	NO	PSEUDOMONAS	M+ I- S+	49.25	RIGHT MRM WITH TYPE 3 TYMPANOPLASTY	57.25	4	NO	REJECTED
48	RAVIKUMAR	19	M	LEFT	MUCOSAL	OCCASIONALLY WET	NO	POSTEROSUPERIOR RETRACTION POCKET	YES (GRANULATION)	NO	YES	STAPHYLOCCUS AUREUS	M+ I- S+	53	LEFT SIMPLE MASTOIDECTOMY WITH TYPE 2 TYMPANOPLASTY	35	6	18	TAKEN UP
49	VASANTHI	35	F	LEFT	SQUAMOUS	OCCASIONALLY WET	YES	POSTEROSUPERIOR CHOLESTEATOMA WITH ANTEROINFERIOR PERFORATION	YES (GRANULATION, EFFUSION)	YES	YES	NO GROWTH	M+ I- S-	69.75	LEFT MRM WITH TYPE 3 TYMPANOPLASTY	71.25	10	NO	REJECTED
50	KANAGA	13	F	RIGHT	SQUAMOUS	OCCASIONALLY WET	NO	ATTIC PERFORATION	YES (GRANULATION)	YES	YES	ENTEROCOCCUS	M- I- S-	72	RIGHT MRM WITH TYPE 3 TYMPANOPLASTY	71.25	10	NO	REJECTED