"A PROSPECTIVE STUDY TO ASSESS THE ROLE OF AGE AS A FACTOR IN HEALING OF TYMPANIC MEMBRANE FOLLOWING MIDDLE EAR RECONSTRUCTIVE SURGERIES"

By

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Dissertation submitted to B.L.D.E. University, Bijapur

In partial fulfillment
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MASTER OF SURGERY

IN

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Under the guidance of

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2011

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LIST OF ABBREVIATIONS USED

CSOM	:	Chronic suppurative otitis media
CM	:	Cortical mastoidectomy
dB	:	Decibel
KHz	:	KiloHertz
PTA	:	Pure tone audiometry
TYM	:	Tympanoplasty

ABSTRACT

Background: Age is known to affect all phases of healing Though it is suggested by many it is not properly documented in healing of tympanic membrane with age

Objective: This study was carried out to assess the role of age as a factor in healing of tympanic membrane following middle ear reconstructive surgeries.

Methods: 71 patients were studied and audiograms were recorded pre-operatively and post-operatively 3rd and 7th week Comparisons were made between two age groups below and above 40 years.

Results: Pre operative and post operativeA-B gap were compared by using-t test and chi-square test. Using chi-square test , the p values (0.712) was insignificant between the two age groups

Interpretation and conclusion: In this study age was not a significant factor affecting healing of tympanic membrane following middle ear reconstructive surgeries.

Key words: age; pure tone audiometry

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

While the general principles of wound healing can be applied to the tympanic membrane, there are some variations that occur secondary to the eardrum's unique anatomy and function. The three levels of the tympanic membrane are the epithelial outer layer, the fibrous middle layer, and the mucosal inner layer. The blood supply is radially arranged along the edges (forming the vascular ring) and the manubrium.

The pars tensa, which represents the bottom 80% of the eardrum's structure, is composed mainly of collagen II running in parallel sheets, which is unique. The pars flaccida, which is the superior aspect of the drum, is made up primarily of collagen I; it also has multiple three-dimensional areas of collagen. Basically, a tympanic membrane perforation closes by a process of circumferential epithelial proliferation followed by connective tissue growth. Like most healing tissues, tympanic membranes heals more effectively if the local environment is free of infection, if there is a rich blood supply and oxygen level.

It has commonly been reported that healing slows down with increasing age and is known to affect all phases of healing. With increasing age, there is a decrease in inflammatory response and reduction in the rate of epithelialisation. . Clinical experience with elderly patients tends to support this belief. Studies of hospitalized surgical patients show a direct correlation between older age and poor wound healing outcomes such as wound dehiscence and incisional hernia.

In healthy human volunteers there was a significant delay of 1.9 days in the epithelialization of superficial skin defects in those older than 70 years of age when

compared to younger volunteers. Thus, although wound collagen synthesis does not seem to be impaired with advanced age, noncollagenous protein accumulation at wounded sites is decreased. Thus it remains controversial whether aging delays wound healing in humans.

The aim of this study is to examine the effects of age on healing of tympanic membrane following middle ear surgeries .

AIM AND OBJECTIVES

To assess the role of age as a factor in the healing of tympanic membrane following middle ear reconstructive surgeries

REVIEW OF LITERATURE

Ercan Pinar, Kerim Sadullahoglu,Caglar Calli ,Semih Oncel conducted a study on 231 patients who underwent tympanoplasty between 2002 and 2007. Prognostic factors such as age,sex, presence of systemic disease,location and size of perforation,duration of dry period,presence of myringosclerosis,presence of septal and conchal pathology,operation time,status of opposite ear and middle ear index were investigated. Pediatric age group (11 to 16 years) included 42 patients and adult group (17 to 58 years) had 189 patients .Each patient underwent audiological evaluation preoperatively and 6 months postoperatively.This study did not find a relationship between success rate of surgery and patient age.[1]

Aaron C Lin and Anna H Messner reviewed the role of age and other factors on success of tympanoplasty in children . This study concluded that success of pediatric tympanoplasty is not a matter of age ,but a matter of patient selection.No one variable determines outcome. Clearly some factors studied are age related, but age in itself should not be an indication or contraindication to treatment.[2]

Sckolnick et al in a retrospective study evaluated 604 children aged 1 to 18 years who underwent myringoplasty from 2000 to 2005. Two analyses were performed to assess the effect of patient age on perforation closure. This study revealed non linear relationship between age and outcome. The rate of successful outcome goes down with each increasing age from I year until 9 years .However after 9 years ,the success rate increases with each year of age[3].

Shrestha S ,Sinha B K conducted a study on 50 patients who underwent myringoplasty. Majority of patients were in age group of 15-25 years. The result showed

that age and sex did not seem to have any bearing on site of perforation ,preoperative hearing level or post operative hearing improvement.[4]

Ilana Fukuchi et al conducted a study during 2004 to assess age and other factors influencing tymanoplasty results Average age of patients was 13 to 56 years.Age is not a factor that alters tympanoplasties success rates .The population of present study was 13 to 56 years ; therefore in this sample there were no representatives from geriatric and pediatric populations,thus not being able to correlate age as a significant success factor for surgery.[5]

Roberto Albera ,Vittorio Ferrero,Michelangelo Lacilla,Andrea Canale evaluated importance of various prognostic factors to risk of tympanic reperforation. Study was conducted on 212 patients aged 3 to 73 years..Age is usually considered a major prognostic factor ,and some authors suggest not performing myringoplasty before patient is 8 or 12 years,on the assumption that in younger patients recurrent otitis media is more frequent ,the Eustachian tube function is not yet mature and the tympanic perforation allows middle ear ventilation. The good results obtained in this study made the authors conclude that myringoplasty can be carried out in younger patients ,if at least over 3 years and surgical approach is most predictive factor of success following surgery.[6]

William Collins, Fred Telischi, Thomas Balkany, Craig Buchman conducted a study on 72 patients aged 3 to 18 years to assess the prognostic value of different variables on outcome of pediatric tympanoplasty. . The youngest patient in this series was aged 3 years at the time of surgery with successful healing of tympanic membrane.[7]

N Umapathy ,P J Dekker conducted a retrospective analysis of 100 children aged 4 to 14 years who underwent myringoplasty. This study concluded that myringoplasty in children

as young as 4 years of age can result in an intact tympanic membrane and improved hearing.[8]

Fadl A Fadl conduted a retrospective study of 97 patients who underwent tympanoplasty. Factors presumed to influence outcome like age,sex, affected ear, middle ear status, perforation size, surgical technique were considered. Age of patients included in this study ranged from 11-45 years. Age factor did not affect the success in this study.[9]

Radpour S conducted a study on the outcome of tympanoplasty in geriatric patients Although some reports suggest that hearing results were not good in patients older than 60,.the instance of graft failure in these patients did not differ greatly by age. The study concluded that the presence of coexisting disease is more important than age itself, although physiologic age is more important than chronological age.[10]

Emmett J R conducted a study to compare success of tympanoplasty in young and old .The results of 163 consecutive type I tympanoplasties performed on patients aged 20 to 40 was compared with those of 97 consecutive type I tympanoplasties performed on patients aged 65 and older. The tymanoplasties were performed over a 5 year period .The study found no difference in the success or failure of the graft- take rates between the two groups. This study concluded that age is not a factor in the success or failure of healing following tympanoplasty surgery.[11]

Jeffrey T Vrabec, Ronald W Deskin, James J Grady conducted a study to assess whether preoperative condition or surgical technique may influence the success of tympanoplasty in pediatric population.This study showed an association of greater success with advancing age. The most common explanation for this relationship is the greater incidence of recurrent otitis media in the younger child.[12]

Francoise Denoyelle, Gilles Roger, P.Chauvin, Erea Noel Garabedian conducted a retrospective study of factors influencing myringoplasties in 188 children between 4 to 17 years of age. Clinical, preoperative and postoperative audiological assessment was done for each patient. This study concluded that age of the patient did not influence the postoperative outcome. Inflammation of middle ear mucosa and a pathologic contralateral ear independently influence the risk of an abnormal postoperative tympanic membrane[13].

Brian W Blakley ,Steve Kim, Mary Van Camp conducted a study in 124 patients to assess if preoperative hearing affects postoperative results. Audiograms were performed before and after surgery at 8 weeks. Age was not found to affect postoperative hearing results.[14]

Yogesh Bajaj, A S Bais, Bakul Mukherjee conducted a prospective study on 45 children aged 5 to 14 years undergoing tympanoplasty. Children were divided into 3 age groups – five to eight, nine to eleven and 12 to 14 years. In this study success rate was maximum in five to eight age group. The authors concluded that one should proceed with tympanoplasty regardless of the patients age.[15]

Caylon et al conducted a study on 51 pediatric myringoplasty cases. Factors known to influence hearing results like age, size and site of perforation , condition of ear, status of cotralateral ear and grafting materials were considered. Patients were divided into 2 groups : ten years and younger and 11 to 16 years groups . The failure rate of age groups in this study also reflects that child age is not an influencing factor.[16]

Tai CF, Ho KY , Juan KH conducted a study to evaluate the influence of age on the prognosis of type 1 tympanoplasty. 100 patients aged 16-65 years were assessed

during a 4 year period . There was no statistically significant difference in hearing gain between various age groups and the preoperative hearing thresholds also increased with advancing age. In conclusion, although type 1 tympanoplasty offered the patients a similar hearing gain among the different age groups ,from the point of view of social function ,it offered younger people a better chance of hearing than the elderly and a higher surgical success rate.[17]

Silva Albu, Gregorio Babighian, Franco Trabalzini conducted a a retrospective study on 544 patients to assess the prognostic value of pathologic and technical variables influencing the functional outcome of tympanoplasty. The patients ranged in age from 8-70 years. This study concluded that middle ear pathologic condition affects functional outcome of tympanoplasty[18].

Gillian S Aschcroft, Michael A Horan, Mark J Ferguson conducted a study to assess the effects of ageing on wound healing. The process of cutaneous wound repair is controlled by growth factors in an autocrine and paracrine fashion . A delay in the appearance of plateletderived growth factor (PDGF) A and B isoforms ,and PDGF –a and β receptors was evident with increasing animal age,paralleled by a similar finding for epidermal growth factor(EGF) and EGF receptor .The quantity and distribution patterns of the various growth factors and their receptors may explain the age –related differences in wound healing speed and quality ,and possibly suggest new therapeutic targets for manipulating wound healing in the aged[19].

Ludwig Podoshin, Milo Fradis, Shelton Malatskey,Jacob Ben-David conducted a study to identify factors that could influence the success of tympanoplasty in children .Patients aged 9-14 years were assessed clinically and audiologically over a 3 year period

following surgery. This study concluded that the success rate of myringoplasty in children is nearly equal to the widely accepted rate in adults of 92% .[20]

Michel Gersdorff, Pierre Garin , Monique Decat,Miguel Juantegui conducted a study to determine long term results of tympanoplasty in adults and children. The mean hearing improvement was poorer in children than in adults. This study confirms that the results of tympanoplasty are less satisfactory in children than in adults ,because of higher incidence of upper respiratory infections in children leading to acute otitis media or otitis media with effusion ,and because of unpredictability of Eustachian tube function.[21]

Aoyagi et al conducted a study to assess the effects of aging on hearing results in tympanoplasty. 642 patients were assessed pre and postoperatively. Averaged air and bone conduction thresholds in patients were appreciably poorer in younger patients and increased with age, compared with physiological hearing impairment in old age (presbyacusis). Means of air bone gap were almost the same in each age group, though hearing thresholds in individual patients were widely distributed. This was more dominant in patients who had undergone type III or IV tympanoplasty than those with type I tympanoplasty , and in patients with chronic suppurative otitis media than with cholesteatoma. Thus this study concluded that patients with chronic ear disease should be recommended to undergo tympanoplasty at an early age.[22]

Van de Kerkhof PC ,Van Bergen B, Spruijit K, Kuiper JP conducted a study to assess age related changes in wound healing . Many of the processes involved in wound healing are impaired in elderly. However, in elderly patients not suffering from concomitant diseases, the rate of wound healing is normal or only slightly reduced. Various ‘systemic factors’ (endocrine and haematological diseases, nutritional

deficiencies and medications) and 'regional disorders' (vascular and neural diseases) may impair wound healing. These complicating conditions occur more frequently in aged subjects.[23]

Gerstein AD, Philips TJ, Rogers GS, Gilchrest BA conducted a study to assess age related differences in healing. Although the elderly can heal most wounds ,they have a slower healing process ,and all phases of wound healing are affected. The inflammatory response is decreased or delayed ,as is the proliferative response. Remodelling occurs,but to a lesser degree.[24]

Glenn Isaacson in his study of tympanoplasty in children discussed factors affecting success of tympanoplasty . There is a sharp disagreement over the optimal age for tympanoplasty.. From a practical standpoint ,most otologists prefer to operate on a dry perforation in a child over 6 years of age with no otitis in the other ear and would consider a pretympanoplasty adenoidectomy if demonstrable adenoiditis were a frequent complaint.[25]

In the recommendation for guidelines for reporting hearing results in middle ear and mastoid surgery it has been suggested that pre and postoperative air conduction levels ,averaged in the three speech hearing frequencies (500,1000, and 2000 HZ) and air bone gap be considered as the baselines for determining postoperative hearing results.[26]

Ophir et al conducted a study to assess the long term results of tympanoplasty in children of 5-8 and 9-12 years age groups . Results of postoperative hearing ,analysed by calculating the postopeoperative air bone gap and by speech audiometry,were similar in the two age groups . It concluded that myringoplasty has a good chance of success in children ,regardless of age.[27]

Warren Y Adkins ,Charleston SC, Benjamin White conducted a study to assess the factors influencing tympanoplasty .40 adults and 25 children were analysed. Age of patients ranged from 4-67 years. This study concluded that neither the age of patient nor the duration of dry ear had a significant bearing on success. There was a definite relationship between the size of perforation and the likelihood of success.[28]

Surgical Anatomy Of Middle Ear Cleft

The middle ear cleft consists of the eustachian tube, the tympanic cavity (middle ear), the aditus ad antrum and the pneumatic system of the temporal bone.

The ear is the first organ of special senses to become differentiated in man. The middle ear, however is not completely formed at birth.

The anatomy of middle ear cleft is complex. It contains sound conducting apparatus and it is related to important and sensitive neighbouring structures.

The Tympanic membranes

It forms the partition between the external acoustic meatus and the middle ear. It is obliquely set and as a result, its postero-superior part is more lateral than its antero-inferior part. It is 9-10mm long, 8mm wide and 0.1mm thick.

Tympanic membrane can be divided into two parts:

Pars tensa: It forms most of the tympanic membrane. Its periphery is thickened to form a fibrocartilagenous ring called ***Annulus tympanicus,*** which fits in the tympanic sulcus. The central part of pars tensa is tented inwards at the level of tip of malleus and is called

the umbo. A bright cone of light can be seen radiating from the tip of malleus to the periphery in the anteroinferior quadrant.

Pars flacida: This is situated above the lateral process of malleus between the notch of rivinus and the anterior and posterior malleal folds. It is not taut and may appear slightly pinkish.

Layers Of Tympanic membrane

Tympanic membrane consists of three layers.

1. Outer epithelial layer (which is continuous with the skin of the external acoustic meatus).
2. Inner mucosal layer (which is continuous with the mucosa of the middle ear).
3. Middle fibrous layer (which encloses the handle of malleus and has three types of fibres- the radial, circular and parabolic).

The Middle ear :

The middle ear extends much beyond the limits of tympanic membrane which forms its lateral boundary and is divided into: 1) *Mesotympanum* (lying opposite to the pars tensa). 2) *Epitympanum* or *attic* (lying medial to sharpnell's membrane and bony attic wall). 3) *Hypotympanum* (line below the level of pars tensa). The portion of the middle ear around the tympanic orifice of the eustachian tube is sometimes called as *the protympanum.*

Middle ear can be likened to a six sided box with a roof, floor, medial, lateral, anterior and posterior walls.

The roof is formed by a thin plate of bone called *tegmen tympani.* It extends posteriorly to form roof of aditus ad antrum. It separates tympanic cavity from middle cranial fossa.

The floor is a thin plate of bone which separates tympanic cavity from the jugular bulb. Sometimes it is congenitally deficient and the jugular bulb may then project into the middle ear, separated from the cavity by the mucosa only.

The anterior wall has a thin plate of bone, which separates cavity from internal carotid artery. It has two openings in its upper portion, the lower opening for eustachian tube and the upper one for tensor tympani muscle.

The posterior wall is close to mastoid air cells. It presents a bony projection called pyramid, through the summit of which appears the stapedius tendon to get attached to the neck of the stapes. Aditus, an opening through which attic communicates with antrum, lies above the pyramid. Facial nerve runs in the posterior wall just behind the pyramid. Facial recess is a depression in the posterior wall, lateral to the pyramid. It is bounded medially by the vertical part of facial nerve, laterally by the chorda tympani and above by fossa incudis. Surgically, facial recess is important as direct access can be made through this into middle ear, without disturbing posterior meatal wall (Intact canal wall technique).

The medial wall is formed by the labyrinth. It presents a bulge called *promontory* which is due to basal coil of cochlea; oval window into which is fixed the footplate of stapes, round window/ fenestra cochlea which is covered by secondary tympanic membrane. Above the oval window is the canal for facial nerve. Its bony covering may sometimes be dehiscent and the nerve may lie exposed making it vulnerable to injuries or infections. Above the canal for facial nerve is the prominence of lateral semicircular canal. Just anterior to the oval window, the medial wall presents a hook like projection called *processus cochleariformis.* The tendon of the tensor tympani takes a turn here to get attached to the neck of malleus. The cochleariform process also marks the level of the genu of the facial nerve. Medial to the pyramid is a deep recess called *sinus tympani* which is bounded by the subiculum below and ponticulus above.

The lateral wall is formed largely by the tympanic membrane and to a lesser extent by the bony outer attic wall called *scutum.* Tympanic membrane is semi transparent and forms a window into the middle ear.

The Eustachian [Pharyngo tympanic] tube:

It connects the tympanic cavity with the nasopharynx. In the adult it is 36mm long. Its lateral one third (12mm) is bony while its medial two thirds (24mm) is fibrocartilagenous. These two portions meet at an angle called isthmus which is the narrowest part of the tube. The cartilage forming the medial part of the tube contributes to the medial, superior and upper part of the lateral wall of the tube. The rest of the tube being completed by fibrous tissue. At the pharyngeal end the cartilage of the tube raise an

elevation called *torus tubaris*. The eustachian tube is wider, shorter and more horizontal in infants, thus permitting infections to travel easily from the nasopharynx.

The mucosa of tympanic cavity:

The middle ear mucosa is to some degree a respiratory mucosa carrying cilia on its surface and being able to secrete mucous. The extent of the mucociliary epithelium varies in the normal middle ear. It is more widespread in the young and it ends at the line of facial nerve in all ages. Above the facial nerve i.e. in the epitympanum and mastoid, a flat non-ciliated epithelium, with only a very occasional mucus producing cell is found. The mucous comes from the goblet cells and the mucous gland which are a collection of mucous producing cells linked to the surface by a short duct. In the middle ear, the glands are sometimes absent. In those ears where they are present, they tend to be clustered around the orifice of the eustachian tube. Goblet cells eject mucous directly into the middle earspace and are in highest concentration close to the eustachian tube opening. The presence of the goblet cells and mucous glands is indicative of the potential ability of the middle ear mucosa to undergo changes typical of respiratory epithelium.

The mucous membrane lines the bony walls of the tympanic cavity and it extends to cover the ossicles and their supporting ligaments in much the same way as the peritoneum covers the abdominal viscera.

The mucosal folds also cover the tendons of the two infratympanic muscles and carry the blood supply to and from the contents of tympanic cavity. These folds separate the middle ear space into compartments and the epitympanic space is only connected the

mesotympanum by way of two small openings between the various mucosal folds, the isthmus tympani anticus and isthmus tympani posticus.

Mastoid antrum:

It is a large air containing space in the upper part of mastoid and communicates with attic through the aditus. Its roof is formed by the tegmen antri which separates it from the middle cranial fossa. The lateral wall of antrum is formed by a plate of bone which is on an average 1.5cm thick in the adults. It is marked externally, on the surface of the mastoid by suprameatal (MC Ewen's) triangle.

Aditus ad antrum:

Aditus is an opening through which the attic communicates with the antrum. The bony prominence of the horizontal canal lies on its medial side while the fossa incudis to which is attached the short process of incus lies laterally. Facial nerve's course is just below the aditus.

The mastoid process:

The mastoid process lies behind the tympanic portion of the temporal bone . The stylomastoid foramen with facial nerve lies on the lateral surface immediately behind the tympanic ring. Its development begins at the end of first year of life and forms a definite elevation at the end of second year and achieves its definitive size at puberty.

20% of the mastoid becomes honeycombed with air spaces (cellular type). In some persons marrow persists within the bony structure of the mastoid (diploeic type)

and in others aircells and marrow spaces are completely absent (acellular, sclerotic or ivory type).

Pneumatic system of temporal bone:

In the majority of adult population a more or less extensive system of interconnecting air filled cavities arise from the wall of the mastoid antrum and sometimes even from the walls of the epitympanum and mesotympanum. These aircells like the mastoid itself are lined with a flattened nonciliated squamous epithelium.

The process of pneumatisation begins with the resorption of mesenchyme early in the third fetal month. Resorption of mesenchyme progresses very rapidly during the first two months of infancy. It is practically complete in the middle ear by the sixth month and in the mastoid antrum by the first birthday. Pneumatisation of the petrous apex begins about 3rd or 4th year and may continue into early adult life

The compartments and folds of the middle ear:

During 3rd to 7th month of intrauterine life, four endothelially lined sacs originate from the first pharyngeal pouch to tubotympanic recess. The four sacs are saccus medius, anticus, posticus and superior. The connecting layers of the sacs later forms the mucosal folds and suspensory ligaments of the ossicles.

Saccus medius: saccus medius develops in the later life to epitympanum. Occasionally saccus anticus also contributes to form the epitympanum.

Prussak's space: the medial most saccule of the saccus medius forms the prussak's space. It lies between the neck of the malleus and sharpnell's membrane laterally.

Boundaries: Medially by the neck of malleus, laterally by sharpnell's membrane, superiorly by fibres of lateral malleolar fold and inferiorly by lateral process of malleus.

Superior incudal space: Is also formed by medial most saccule. This space lies lateral to incus body and above head of malleus.

Isthmus tympani anticus: It runs between stapes and tensor tympani tendon, connecting the anterior mesotympanum and anterior attic.

Isthmus tympani posticus: It passes between the short process of the incus and the stapes tendon to the posterior attic and aditus.

Anterior pouch of Von Troltsch: lies between tympanic membrane and anterior malleolar folds.

Posterior pouch of Von Troltsch: lies between tympanic membrane and posterior malleolar folds.

The inferior edge of the fold contains chorda tympani nerve. This pouch is open inferiorly to posterior mesotympanum.

The contents of tympanic cavity

Muscles - Tensor tympani and stapedius

Nerves - Chorda tympani and tympanic plexus

Ossicles - Malleus, incus, stapes Air

Intratympanic muscles

There are two muscles – Tensor tympani and Stapedius. The former attaches to the neck of the malleus and tenses the tympanic membrane while the latter attaches to the neck of the stapes and helps to dampen very loud sounds thus preventing noise trauma to the inner ear.

Nerves:

Chorda tympani: this branch of the facial nerve enters the tympanic cavity from the posterior canaliculus at the junction of the lateral and posterior walls. It runs across the medial surface of the tympanic membrane between the mucosal and fibrous layers and passes medial to the upper portion of the handle of the malleus above the tendon of the tensor tympani to continue forwards and leave by way of the anterior canaliculus.

The chorda tympani subsequently joins the lingual nerve to supply the anterior $2/3^{rd}$ of taste buds of the tongue.

The Tympanic plexus: tympanic plexus is formed by the tympanic branch of the glossopharyngeal nerve (Jacobson's nerve) and by the caroticotympanic nerves which arise from the sympathetic plexus around the Internal carotid artery. The nerves form a plexus on the promontory and provide the following:

1) Branches to the mucous membrane lining the tympanic cavity, eustachian tube, mastoid antrum and air cells.
2) A branch joining the greater superficial petrosal nerve.
3) The lesser superficial petrosal nerve, which contains all the parasympathetic fibres of 9^{th} cranial nerve. The nerve is really a continuation of the tympanic branch of glossopharyngeal nerve, which passes through the foramen ovale to the otic ganglion. From this, postganglionic secretomotor fibres are relayed to the parotid gland via the auriculotemporal nerve.

ossicles:

Malleus

Malleus is the largest of the ossicles. It comprises of head, neck and three processes arising from below the neck. The overall length of the malleus ranges from 7.5mm to 9mm.

The head lies in the epitympanum and has on its posteromedial surface an elongated, saddle shaped, cartilage covered facet for articulation with the incus. This surface is constricted near its middle and the smaller inferior portion at the joint surface lies nearly at right angles to the superior portion.

This projecting lower part is a cog or spur of the malleus. Below the neck of the malleus, the bone broadens and gives rise to petrotympanic fissure, the lateral process which receives the anterior and posterior malleolar folds form the tympanic annulus and the handle.

The handle or manubrium is cresentric and concave laterally. The lower horn of cresent terminates flatly at the umbo. The upper horn projects into the lumen of the external meatus as the lateral process. The handle runs downwards, medially and slightly backwards between the mucosal and fibrous layers of the tympanic membrane on the deep medial surface of the handle, near its upper end, is a small projection into which the tendon of the tensor tympani muscle inserts. Additional support for the malleus comes from the superior ligament which runs from the head of the tensor tympani.

Incus

The incus articulates with the malleus and has a body and two processes. The body lies in the epitympanum and has a cartilage covered facet corresponding to that on the malleus. The short process projects backwards from the body to lie in the fossa incudis to which it is attached by a short ligament. The long process descends into the mesotympanum behind and medial to the handle of the malleus, and its tip is small medially directed lenticular process which articulates with the stapes.

Stapes

The stapes consists of head, neck, two crura (limbs) and a base or footplate. The head points laterally and has a small cartilage covered depression for articulation with the lenticular process of the incus. The stapedius tendon inserts into the posterior part of the neck and upper portion of the posterior crus. The two crura arise from the broader lower part of the neck, and the anterior crus is thinner and less curved than the posterior crus. Both are hallowed out on their concave surfaces, they join the footplate. The foot plate has a convex superior margin, straight inferior margin, curved anterior and posterior ends.

Relations of the middle ear cleft

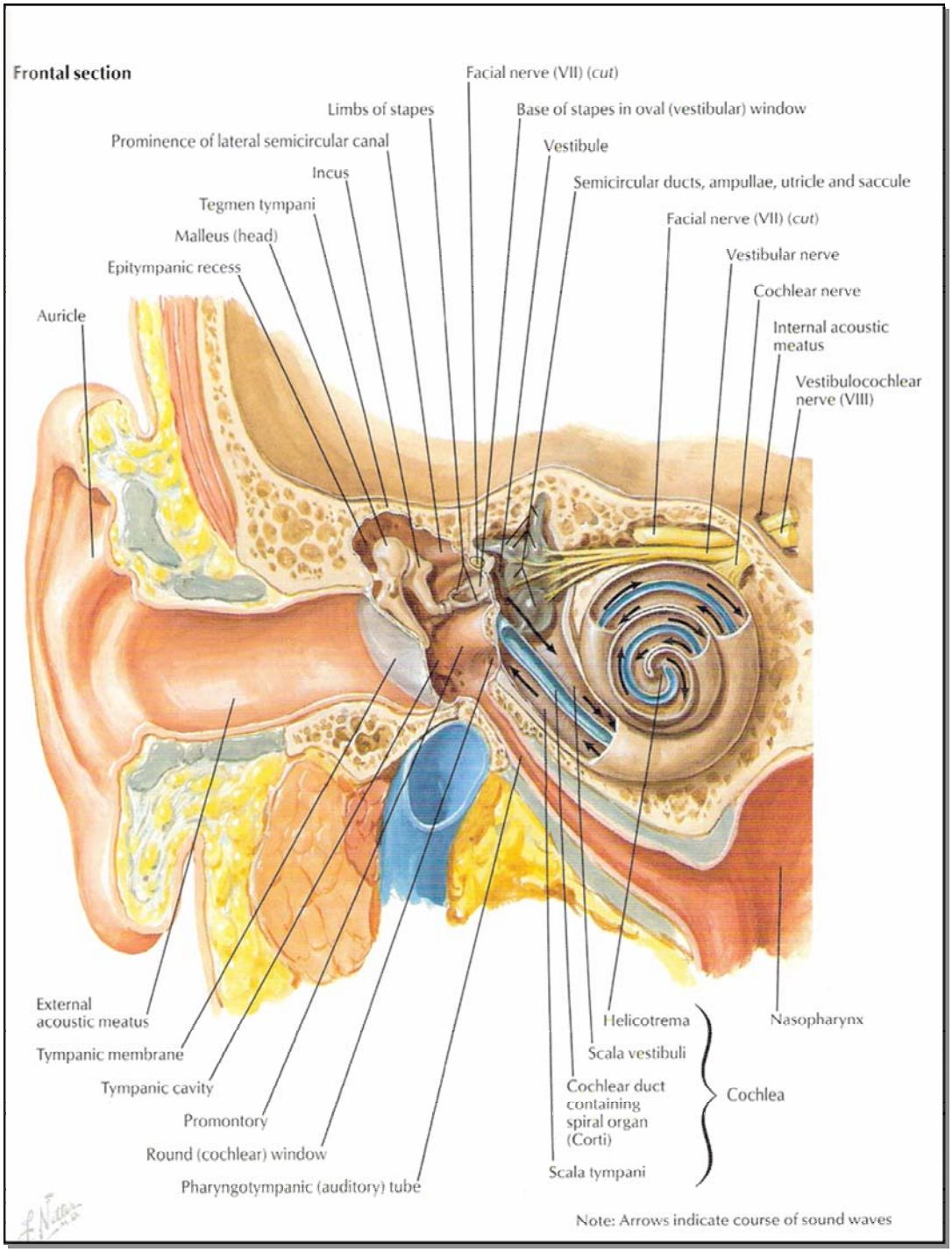
The external auditory canal is separated from the cavity by the tympanic membrane. The inner ear is separated from the cavity by its medial wall. The temporal lobe of the brain, in the middle cranial fossa, is separated from the tympanic cavity by the

thin tegmen tympani which is formed partly by the petrous and partly by the squamous portion of the temporal bone. It is separated from the mastoid antrum by the tegmen antri. The carotid canal containing the Internal carotid artery is separated from the lower part of the tympanic cavity, anteriorly by a plate of bone which merges with the floor of the cavity. The jugular bulb is closely related to the bony floor of the cavity which may be deficient in part . The glossopharyngeal, vagus and accessory cranial nerves emerge from the skull through the jugular foramen alongside and just medial to the jugular bulb.

The ganglion of trigeminal nerve (gasserian ganglion) lies in a shallow depression (meckel's cave) on the anterior surface of the petrous apex, between two layers of dura.

The abducent cranial nerve, the motor nerve to the lateral rectus muscle of the eye, runs along the posterior surface of the petrous apex, in the posterior cranial fossa on its way to Dorello's canal. The canal is formed by the petroclinoid process of the sphenoid bone and the posterior clinoid process of the sphenoid bone.

The facial cranial nerve is related to the middle ear cavity in the horizontal and vertical tympanic portions. The bony covering (the fallopian canal) may be very thin or totally deficient in parts, the nerve being covered only by the tympanic mucoperiosteum in as many as 10% or more of temporal bones.



Anatomy of middle ear

ANATOMICAL CHANGES IN EAR AS AGE ADVANCES

The middle ear structures undergo anatomical changes with age,although they have little effect on the physiology of the middle ear.There is little evidence of substantial stiffening of middle ear transmission system with age.

The tympanic membrane,ossicular chain, articular cartilages at the surface of incudo-malleolar and incudo-stapedial joints and the middle ear cartilages are susceptible to minor age-related changes.The tympanic membrane appears to become stiffer, thinner and less vascular with increased age. In becoming more translucent with age,selected landmarks appear more visible during otoscopic examination.

Arthritic changes in middle ear have been observed in individuals over 30 years of age,increasing in frequency and severity with age.The arthritic changes include thinning and calcification of incudo-malleolar and incudo-stapedial joint.Additional age-related changes include atrophy and degeneration of fibres of middle ear muscles and ossicular ligaments and ossification of ossicles.Finally ,calcification of cartilaginous support of eustachian tube and atrophy of the musculature has been reported in older adults.The age-related decline in muscle function may interfere with opening of Eustachian tube especially during swallowing.

While these changes do not appear to impact on pure tone and bone conduction thresholds,they may account for age-effects appearing in selected studies on Eustachian tube function.

Ventilation of middle ear is an essential predictor of the functional results following middle ear reconstruction.It is a complex and dynamic process depending on a number of factors,most important of which include the functional status of Eustachian

tube,the degree of pneumatization of mastoid air cells and the condition of middle ear mucosa.The role of mastoid pneumatisation is not exactly known.But it forms an air reservoir and acts as a surge tank to minimize pressure fluctuation.

The inner ear undergoes dramatic changes with age with corresponding effects on pure tone thresholds. While both outer and inner hair cells tend to degenerate with age, the outer hair cells are more vulnerable than inner hair cells and most likely account for the typical decline in hearing with age. Hair cell loss at basal turn of cochlea produces hearing loss in high frequencies, which typifies presbycusis. The ageing process is also associated with loss of spiral ganglion cells and average number of fibres in cochlear nerve [33].

Applied Physiology:

In man hearing becomes the vital basis for acquisition of speech and language.

Mechanism of hearing can be broadly divided into

1) Mechanical conduction of sound.
2) Transduction of mechanical energy to electrical impulses (sensory system of cochlea)
3) Conduction of electrical impulses to the brain (neural pathways).

Physiology of sound conduction:

A sound signal in the environment is collected by the pinna, passes through external auditory canal and strikes the tympanic membrane. Vibrations of the tympanic membrane are transmitted through the ossicular chain to the cochlea.

Impedance matching mechanism of middle ear:

When air conducted sounds travels to the cochlear fluids, the loss of sound energy that occurs is compensated by impedance matching or transformer action of the middle ear. It is accomplished by

1) Ossicular-level chain ratio: It is 1.3:1 between handle of malleus and long process of incus.
2) The ratio of area of tympanic membrane and the oval window: The effective area ration is 14:1.
3) Phase differential between oval and round window.

There are two essential physiological processes in Cochlea namely transmission and transduction. The former accounts for the transfer of acoustic energy from the oval window to the hair cells, while the latter is the process by which this sound energy pattern is converted at the organ of corti into action potentials in the auditory nerve.

Vibrations of the stapes produce a flow of perilymph up the scala vestibuli, through the helicotrema and down the scala tympani to the round window membrane. With vibrations, movement of basilar membrane occurs, which sets up a shearing force between the tectorial membrane and hair cells. Higher frequencies are represented in the basal turn of cochlea and progressively lower ones towards the apex. The distortion of hair cells gives rise to cochlear microphonics which triggers the nerve impulses. Auditory

nerve action potential is the algebraic sum of the neural discharges in the whole of the cochlear nerve. Each nerve fibre has an optimum stimulus frequency for which the threshold is lowest.

Transduction by hair cells:

The individual stereocilia on the apical surface of the hair cell are mechanically rigid, and are faced together with cross links so that they move as a stiff bundle. Therefore, when a bundle is deflected, the different rows of stereocilia could be expected to slide relative to one another. There are fine links running upwards from the tips of the shorter stereocilia on the hair cell, which join the adjacent taller stereocilia of the next row. When the stereocilia are deflected in the direction of the tallest stereocilia, the links are stretched, opening ion channels in the cell membrane.

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

The stimulus is coupled to the stereocilia by means of the shear relative motion between the tectorial membrane and the reticular lamina. As the laminar membrane and the organ of corti are driven upwards and downwards by a sound stimulus, the stereocilia are moved away from and towards the modiolus. Because the tallest stereocilia are situated on the side of the hair cell farthest away from the modiolus, an upward movement of the basilar membrane is translated in the movement of the stereocilia in the direction of the tallest. The effective direction of the shear between the tectorial membrane and the reticular lamina is therefore radial across the cochlear duct. This is the direction associated with opening of the ion channels. In both inner and outer hair cells

the tip links are organized in such a way that they run in a direction most suited for picking up radial shear.

When the channels on the stereocilia are open, ion will enter or leave the cell depending on the electrical and the chemical gradients across the apical cell surface. It appears that the ion channels are rather large and non-selective, so that, for instance, Na^+, K^+ and Ca^{++} will enter with nearly the same efficacy. Under the generally accepted position, the apical surface of the hair cells is faced by endolymph with a high positive potential (± 80mV) and a high K^+ concentration. Inside the cell there is a negative intracellular potential, which is –45 mV for inner hair cells and –70mV for outer hair cells. The potential combine to give 125mV (inner hair cells) or 150mV (outer hair cells) of potential drop across the channel, When the channels are open K^+ from the endolymph will tend to be driven into the cell by this big potential gradient, thus making the cell more positive inside. When the channels are completely shut off, the cells will become more negative. Most of the transducer current may be carried by K^+, as this is the predominant ion in the endolymph. However, it is possible that some of the current is carried by some other ions, such as Ca^{++}. The energy from the whole process comes from the stria vascularis, which by ion pumping, stores energy in the 'battery' of the endolymph. This is the 'battery' or 'resistance modulation' theory of Davis (1965) as it appears in the light of modern evidence.

Neural pathways:

Hair cells get innervation from bipolar cells of spiral ganglion. Central axons of these cells collect to form cochlear nerve which goes to central and dorsal cochlear nuclei. From there, both crossed and uncrossed fibers travel to the superior olivary complex, lateral leminiscus, inferior colliculus and medial geniculate body and finally reach the auditory cortex of the temporal lobe.

THEORIES OF HEARING:

1. **Travelling wave theory of Von Bekesy:-**

A sound wave depending on its frequency reaches maximum amplitude on a particular place o the basilar membrane and stimulates that segment. If vibrations of the basilar membrane are observed, a travelling wave is seen to start at the base of the cochlea and progress toward the helicotrema with increasing amplitude to a region of maximum displacement, the position of which depends upon the frequency. Higher frequencies are presented in the basal turn of the cochlea and the progressively lower tones towards the apex.

2. **Helmholtz's resonance place theory:-**

Suggests that the frequency analysis by the ear was due to the fact that each pitch would cause resonant vibration of its own particular place on the basilar membrane. This theory is disproved because basilar membrane cannot act as a resonator.

3. **Rutherford's telephone theory:-**

This theory suggests that pitch perception is based on the rate of firing of individual nerve fibres.

4. Wever's volley theory:-

This postulates that high frequencies are perceived by place alone in the basal turn, low frequencies (below 1000) stimulate nerve action potentials at a rate equal to the stimulus frequency, while intermediate frequencies are presented in the auditory nerve by asynchronous discharge in groups of neurons whose combined activity represents the frequency of the stimulus.

WOUND HEALING

Wound healing is the restoration of tissue continuity after injury. It involves wound closure and restoration of function to the damaged tissue. The process of wound healing can be arbitrarily divided into three main overlapping and inter-related phases:

1. Inflammation
2. Proliferation
3. Remodelling

The inflammatory phase is marked by platelet accumulation ,coagulation and leukocyte migration into the wound bed.

The proliferative phase is characterised by

- Re-epithelialization : restoring cutaneous barrier.
- Angiogenesis :the neovasculature supplying much of the nutrition required for healing during this phase.

- Fibroplasia :forming the collagenous and noncollagenous matrix for the dermal component of the wound .

- Wound contraction :reducing wound size and thus need for scar tissue.

The remodelling phase takes place over a period of months ,during which the dermis responds to injury with a dynamic continuation of collagen synthesis and degradation ,and the once highly vascular granulation tissue undergoes a process of devascularization as it matures into less vascular scar tissue.[30]

HEALING OF TYMPANIC MEMBRANE

In a perforation of healthy tympanic membrane,the healing process starts with the migration of the keratinising squamous epithelium. The advancing squamous epithelium is guided by a spur,protruding like a needle. Below the epithelium,a loose connective tissue,rich in active fibroblasts is formed.This is a mechanism completely different from normal skin wound healing,in which the closing squamous epithelium will migrate on a bed of granulation tissue.

At the time of closure,the multilayered migrating epithelium and the connective tissue meet each other roughly simultaneously.The squamous epithelium then starts to diminish in thickness and the major thickness of healed area will be due to an increased unorganized connective tissue layer.

One matrix substance has been of certain interest in general wound healing and in particular in healing of tympanic membrane: glycosaminoglycan hyaluronan.In the normal pars tensa,there is hardly any detectable hyaluronan.However ,within the first 2-3 days after a tympanic membrane perforation , is a heavy accumulation of hyaluranon in perforation borders.The intense presence of hyaluranon will persist in advancing borders until the tympanic membrane is closed at 10-12 days.[34]

FACTORS AFFECTING HEALING

1. Intrinsic factors
    a) local
    b) systemic
2. Extrinsic factors

Local intrinsic factors

i) Blood supply

Adequate blood supply is essential in all phases of healing and the reestablishment of a micro-ciculation in wounds with angiogenesis is essential to re-establish the structure and function of the original tissue. Impaired blood supply in wounds due to atherosclerosis seen with normal ageing is known to impair wound healing.

ii) **Changes in oxygen tension**

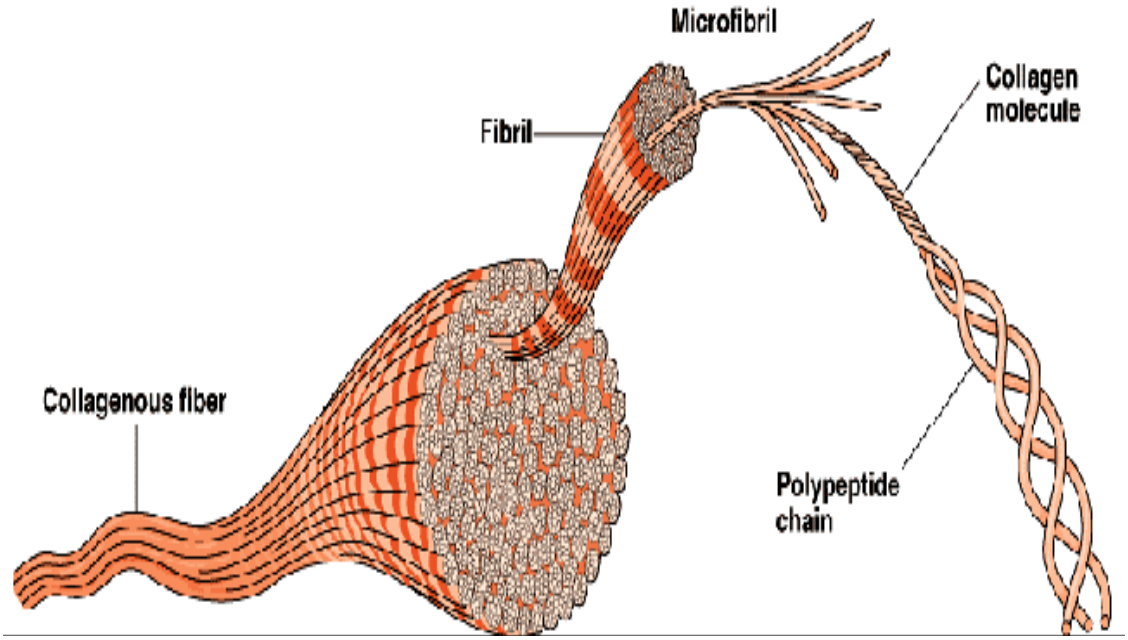
variations in oxygen tension can alter fibroblast proliferation and therefore collagen production. Free radicals are bi-products of oxygen metabolism known to have detrimental effect on healing.

Systemic intrinsic factors

i) Ageing

Ageing affects all stages of wound healing. The onset of inflammatory phase of repair is delayed and lasts longer. Cell proliferation and metabolism decreases . The rate of capillary growth into the wound bed and mast cell numbers decrease with age. The diminished blood flow causes decreased clearance of metabolites and foreign materials ,and increased tissue hypoxia which delays healing.[30]

Wound remodelling is also affected by age due to decreased fibroblast activity . There is also a reduction in the amount of collagen organization and cross-linkage which results in decreased tensile strength.



Normal collagen structure

ii) Diabetes mellitus

Pathology affecting healing in diabetes can be explained by the following :

1) The structure and function of proteins in patients with diabetes is altered.

2) The thickened basement membrane in microcirculation reduces local blood flow

3) Glycosylated proteins cause changes in cellular functions or generate free radicals.

4) Non enzymatic glycosylation and sulphonation also leads to reduction of the ionic change of thickened basement membrane which allows passage of highly charged molecules such as albumin to cross the membrane leading to altered cellular nutrition.

5) Hyperglycemia also leads to osmotic diuresis,leading to decreased perfusion and oxygenation.

6) Hyperglycemia reduces the activity of leukocytes and macrophages which consequently leads to lowered fibroblast production and collagen synthesis.

iii) Renal Disease

Wound failure is increased in patients with uraemia. Uraemia may affect either collagen polymerisation or fibroblast proliferation ,suggesting that renal failure results in a general inhibition of cell proliferation. The effect of uraemia on wound healing may be related to the associated malnutrition and anaemia seen in such patients and should always be taken into account when assessing the wound healing potential as single factors do not usually occur in isolation.

iv) Jaundice

Due to the production of excessive ammonia in hepatic failure, a defiency of the amino acids ornithine and arginine can occur. In such a state ornithine becomes rate limiting for urea cycle function, leading to decreased urea synthesis which in turn affects collagen synthesis.

Extrinsic factors affecting healing

i) Nutrition

Vitamin B and C have attracted the greatest attention in wound healing due to their essential role in collagen synthesis. In addition to iron defiency, sodium, potassium, calcium, magnesium and zinc may all affect the wound healing process. Patients who are zinc deficient have reduced rates of epitheliasation, decreased wound strength, and reduced collagen synthesis. Zinc deficiency has been implicated as a factor in failure of wound healing. Copper is required for cross-linkage of collagen. Iron is also necessary for collagen synthesis.

Anaemia may impair healing through reducing oxygen transportation ,although wound repair is not affected until packed cell volume drops to levels of 15.3%

In addition in malnourished patients,decreased inflammatory responses may occur with a consequent slowing of normal cellular response to trauma,with lower levels of cytokine production and impaired cellular migration and proliferation. ECM synthesis and deposition may be affected decreasing tensile strength of wounds.

ii) Smoking

Smoking adversely affects healing. Nicotine inhibits epitheliasation. Carbon monoxide and hydrogen cyanide also have detrimental effects on healing.

iii) Infection

Infection is invasion of the body by micro-organisms capable of causing disease or the reaction of body tissues to toxic products produced by micro-organisms.

iv) Drugs

Corticosteroids : steroids have a known inhibitory effect on healing process especially in the inflammatory stage of healing .Studies have reported inhibition of collagen synthesis due to effect on growth and metabolism of fibroblasts.

Immunosuppressive**s:** are thought to interfere with wound healing in two ways

a) interfering with mitosis (alkylators)

b) interfering with protein synthesis (antimetabolites)

MATERIALS AND METHODS

1. SOURCE OF DATA

All patients undergoing middle ear reconstructive surgery in the department of ENT in B.L.D.E.A's Shri B.M.Patil Medical College Hospital & Research Centre.

2. METHOD OF COLLECTION OF DATA

1) Preoperatively: history,clinical examination with emphasis on detailed otological examination, examination under microscope and audiometry.
2) Postoperatively: history,clinical examination and audiometry at 3rd and 7th week.
3) The patients are divided into 2 groups and comparisons made between them

   Group I: patients upto 40 years

   Group II:patients above 40 years

3. INCLUSION CRITERIA

All patients undergoing middle ear reconstructive surgeries in BLDEA's Shri B. M. Patil Medical College Hospital and Research Center, Bijapur.

4. EXCLUSION CRITERIA

- Patients with diabetes mellitus or hypertension
- Patients with clinical signs and symptoms of anaemia
- Patients with renal disease or liver disease
- Patients with pulmonary tuberculosis
- Patients with hypoproteinemia
- Patients with tympanosclerosis,cholestatoma,and granulation polyps.
- Patients with blocked aditus on cortical mastoidectomy.

- Patients with sinusitis or atrophic rhinitis.
- Patients with history of smoking
- Patients with history of intake of drugs like corticosteroids or immunosupressants
- Patients with actively discharging ears

5. SAMPLING

Time period of study : October 2008 to april 2010

Sample size :with prevalence of 46% [29] and allowable error of 25% the calculated sample size n=71 using statistical formula $n=4pq/l^2$

Statistical analysis

1. diagrammatic representation
2. mean+SD
3. statistical tests like 't' test and 'Z' test and Chi-square test

METHODS

Patients who presented to BLDEA's Shri.B.M.Patil Medical College Hospital and Research Centre, Bijapur were subjected to the study.

A thorough clinical history was taken for duration of otorrhoea, frequency of otorrhoea, hearing loss, duration and nature of previous treatment.

All cases were subjected to detailed examination which included general physical examination, careful examination of ear, nose and throat. Otoscopic, examination and

tuning fork tests were performed. Ear was examined under microscope for detailed evaluation.

Hearing assessment done with Elkon EDA 3N3 MULTI audiometer.The hearing threshold for pure tone audiometer was determined in a sound treated room at frequencies ranging from 125-8000 Hz for air conduction and 250-4000 Hz for bone conduction.

ELKON PURE TONE AUDIOMETER

Interpretation of Audiogram

Air conduction threshold and bone conduction threshold were noted at 500, 1000 and 2000 Hz and the three frequency average air threshold minus the same three frequency bone threshold was used to calculate air bone gap.

Pure Tone Audiometry:

Pure tone audiometry is as much a science as it is an art for ascertaining the hearing threshold of a subject for pure tone sounds of various frequencies. The hearing threshold as defined by International Standard Organisation (ISO) is "the lowest sound pressure level, at which under specified conditions, a person gives a predetermined percentage of correct responses on repeated trials". The result when plotted graphically is called a 'pure tone audiogram'.The 'decibel' is the unit by which the intensity of sound is measured.If measurements is done in units of pressure ,then this calculated minimum is 0.00024 dynes/sqcm. To facilitate calculations ,the term Bel was introduced .Bel is defined as a ratio expressed in logarithm(with base 10) which tells us how many times the sound that we are measuring is stronger or weaker than reference sound. To simplfy calculations still further the term decibel was coined.

1 Bel =10 decibels.

The reference intensity level, which is designated '0' dB at each frequency is the mean value of the minimal audible threshold of pure tones in a group of healthy, normally hearing, young adults, in accordance with standards set by the (ISO).

Procedure:

The patient is instructed about the procedure in detail. The examiner should be able to observe the subject, but care should be taken to provide no visual clues to the subject, as the examiner operates the audiometer. The head phones are placed over the subject`s ears, so that the center of each transducer is at the ear. Women should remove ear-rings. The subject is instructed that the aim of the test is to establish the least sound that he/she can hear at each of the several frequencies.

Tones should be presented for 1 to 3 seconds with the intervals of 1 to 3 seconds between each presentation. It is important to randomize the intervals and to avoid presenting the tones in a rhythmic fashion to facilitate the recognition of true response. The subject responds as soon as he hears the sound, for example by raising the finger or pressing a button, which lights a signal on the audiometer panel, and maintains the response as long as the sound is heard.

Technique of air conduction test:

A method of air conduction threshold assessment by conventional Hughson-Weslake technique slightly modified by Carharts and Jerger is described below. [32]

The better ear is tested first. The various frequencies are presented in the following order; 1000, 2000, 4000, 8000, repeated again followed by 500, 250, 125 Hz. For given frequency, the initial presentation should be at an arbitrarily presumed suprathreshold level, to allow easy recognition and identification. If patient hears then the tone is decreased by 10 dB steps until patient stops hearing. Once this stage is reached the tone is raised by 5 dB. If the patient hears this tone, the sound is again decreased by 10 dB. If he does not hear it, the tone is again raised by 5 dB. In this way by several threshold crossings, the exact hearing threshold is obtained when one gets atleast 3 out of 5 responses correct. Though threshold is defined as 'the lowest intensity heard on 50 percent of occasions of repeated crossing', but in clinical practice, this is not usually possible on clinical audiometer where graduations are in 5 dB. The second ear is tested in a similar manner. The faintest audible intensity as established above is recorded against the test frequency on a standard audiogram chart as the threshold intensity. By

convention, the symbols ‘o’ and ‘x’ are used for air conduction thresholds for the right and left ears respectively. If the maximum sound intensity of the audiometer at a given frequency cannot be heard, this is indicated by a downward pointing arrow at the level of the maximum output on the appropriate frequency line.

Technique of bone conduction test:

Bone conduction thresholds are obtained in an identical manner to those described for air conduction, but the sound stimulus is produced by a bone vibrator placed on the mastoid process and held firmly, by means of a head band. Care is taken to remove any intervening hair . Contact with the cartilaginous external meatus or pinna is also avoided during the test as these structures may carry air conducted sounds. The vibrator should be placed on the mastoid process of the ear . Measurements are restricted to the frequency range 250 to 4000 Hz and calibration standards do not generally give data for stimuli outside this range. The test is commenced at 1000 Hz, followed by 2000, 4000, 500 and 250 Hz. The subject is instructed to respond to sound regardless of the side on which the sound is actually heard. It must be emphasized that without the use of masking it is not possible to determine the ear that is responsible for the detection of the ‘non-masked’ bone conduction threshold.

Bone conduction thresholds cannot be established with such precision as air conduction thresholds, as the threshold of tactile sensation, particularly at low frequencies may be more acute than audition leading to erroneous results. In addition, as the output level of bone vibrations rises the distortion increases and the threshold measured may relate to second or third harmonics, rather than the fundamental frequency. Care must therefore be taken not to exceed the maximum output level for the particular

audiometer in use. At higher frequencies (2000 - 4000), air borne sound radiated by bone vibrator may result in errors. Hence, an essential requirement of bone conduction testing is the exclusion of the non-test ear by an efficient marking sound so that all threshold levels may be reliably attributed to the tested ear. The symbol '>' represents unmasked bone conduction in left ear and the symbol '<' represents unmasked bone conduction in right ear.↓ indicates no response.

Masking:

Masking is the phenomenon by which one sound impairs the perception of another. The most effective sound to mask a pure tone is a narrow band of noise with the central frequency equal to the test tone. In commercially available audiometers the masking band is automatically selected upon selection of the test tone frequency.

Masking Is mandatory :

1. In bone conduction studies, the non test ear should always be masked,especially if A-B gap is 10 dB or more in non-test ear.

2. In air conduction studies.-masking of non-test ear should be done whenever test tones of more than 45dB are used during air conduction audiometry.

These requirements for masking may be readily understood considering certain facts regarding the transmission of air and the bone conduction sounds across the head. An air conducted sound is transmitted across the skull with an internal attenuation of about 50 dB; while the attenuation for a bone conducted sound is negligible. Hence, in

this later condition an apparent threshold level may be a record of the sensitivity of the cochlea not under test.

Central masking

This refers to the inability of the brain to distinguish sounds of very different loudness, even when they are heard in opposite ears. This effect is most commonly apparent at the higher masking levels .

Cross-masking

Once masking levels corresponding to the beginning of the plateau at a particular masking function have been reached, additional increases in masking level further raise the threshold of the non-test ear. This may not be apparent initially, since the test ear pure tone threshold has been reached and may not be adversely affected by the noise (apart from any central masking effects). However if at some stage the masking level becomes sufficiently high, it may be capable of providing a masking effect in the test cochlea itself even though the noise is attenuated by transcranial attenuation. This is known as crossmasking.

Hoods plateau method of masking:[32]

Air conduction:

1) The unmasked threshold of the ear is ascertained. If it is thought that there is a possibility of cross-hearing, then a masking sound is introduced into the non-test ear at 10 or 15dB above air conduction threshold level of the non-test ear.
2) The tone is presented to the test ear (at unmasked threshold). If the patient gets the tone, masking will not be required. But if the patient does not get the tone, it

indicates adequate masking will be required to get the actual threshold of the test ear and third step is then started.

3) The test tone is raised by 5dB, if patient hears the tone, then next (4^{th}) step is started. If patient does not get the tone, then tone is raised by 5dB steps till the tone is heard, and then the fourth step is started.
4) The masking level is raised by 5dB in the non-test ear. Tone is again presented to the test ear to see whether the patient is getting the tone or not. If the patient is still getting the tone, it indicates actual hearing threshold. If patient does not get the tone, it indicates that actual threshold of the test ear has not been reached as yet.
5) The test tone is raised by 5dB or in 5dB steps till it is heard. Once the test tone is heard the masking level is raised by 5dB and it is checked whether test tone is still being heard. This process of alternately increasing the masking noise and test tone is continued till a time comes when patient will continue to hear the tone inspite of increasing the masking noise by 2 or 3 steps of 5 dB each. This tone level is the actual hearing threshold level of the test ear.

Bone conduction:

This is similar to air conduction masking.

1) Unmasked bone conduction threshold of test ear is ascertained.
2) A masking sound is introduced into the non-test ear at a level of 15 dB above air conduction threshold for the non-test ear.
3) Tone is given by bone conduction to see whether it is being heard or not. If heard, it indicates that masking threshold is correct. If not heard, then tone and masking

sound are increased in 5 dB steps alternately till masking sound level can be increased by 2-3 steps of 5 dB each without requiring any increase in tone sound for the tone sound to be heard. This level of the tone is the actual hearing threshold level of the test ear by bone conduction.

RESULTS

The study was conducted on 71 patients, who underwent ear surgery for CSOM at Shri B.M. Patil Medical college and Research Centre Bijapur, from October 2008 to april 2010. Patients hearing results following middle ear surgery were compared based on age. Comparisons were made between two groups: patients less than 40 yrs and above 40 years.

Observations of the study are described under the following headings:

TABLE -1: AGE DISTRIBUTION

AGE OF PATIENTS [n=71]		
AGE (years)	**NUMBER OF PATIENTS**	%
≤40	40	56
> 40	31	44

PIE CHART

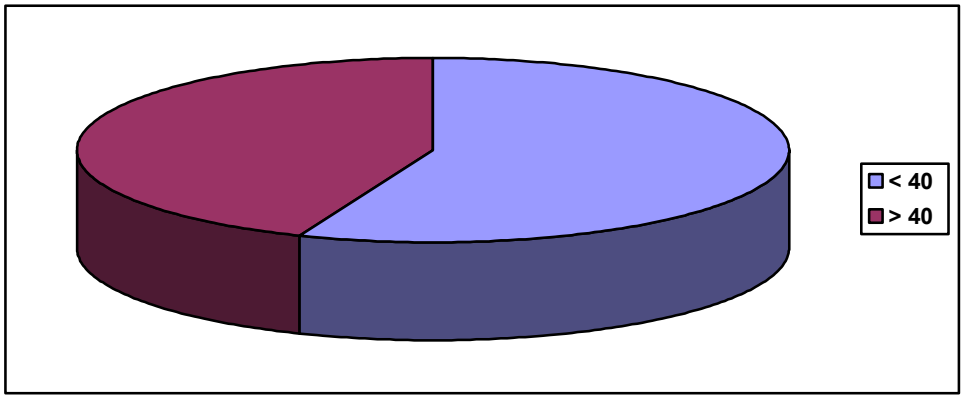


TABLE-2 SEX DISTRIBUTION

Sex distribution		
SEX	**NUMBER OF PATIENTS**	**PERCENTAGE**
Males	27	38%
Females	44	62%

GRAPH-2

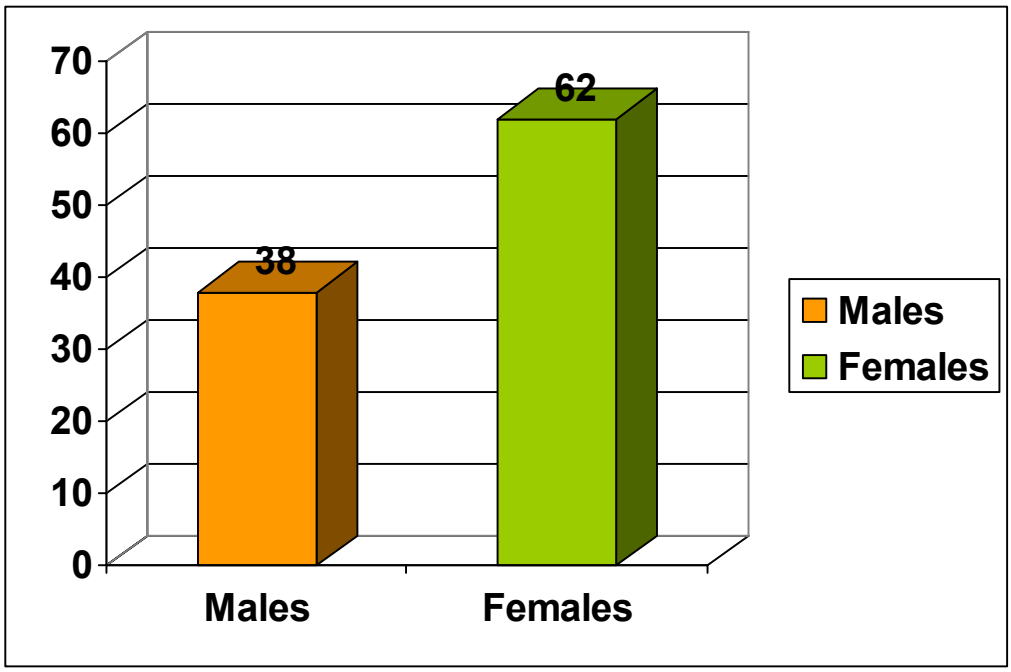


TABLE-3 : PRE-OPERATIVE DIAGNOSIS

Pre-Operative Diagnosis [n=71]		
DIAGNOSIS	**NUMBER OF PATIENTS**	**PERCENTAGE**
LEFT CSOM	27	38%
RIGHT CSOM	18	25%
BILATERAL CSOM	26	37%

GRAPH-3

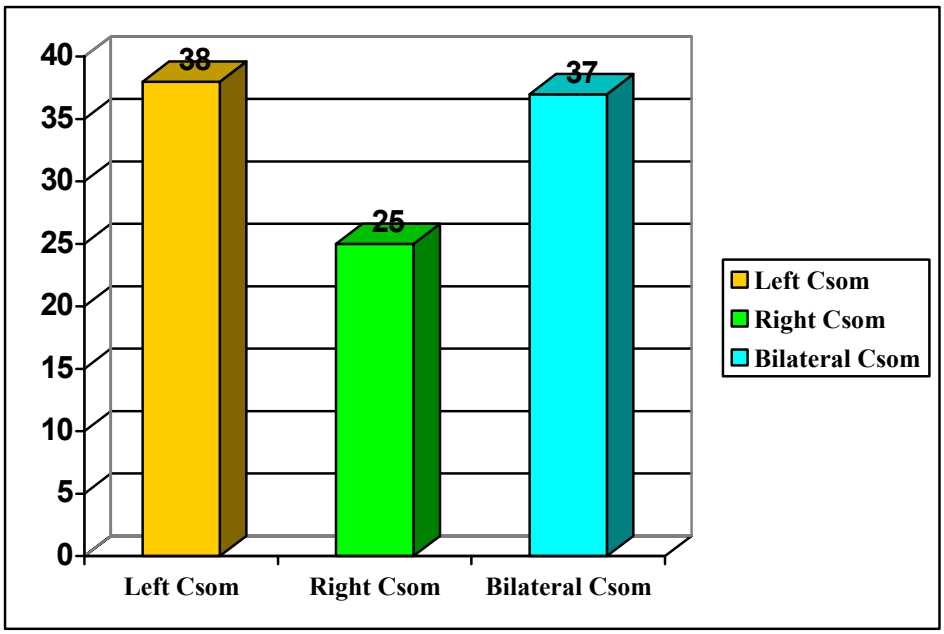


TABLE-4 SURGICAL PROCEDURES PERFORMED

Surgical procedures performed [n=71]		
SURGICAL PROCEDURE	**NUMBER OF PATIENTS**	**PERCENTAGE**
Tympanoplasty	63	89%
Cortical mastoidectomy with Tympanoplasty	8	11%

GRAPH-4

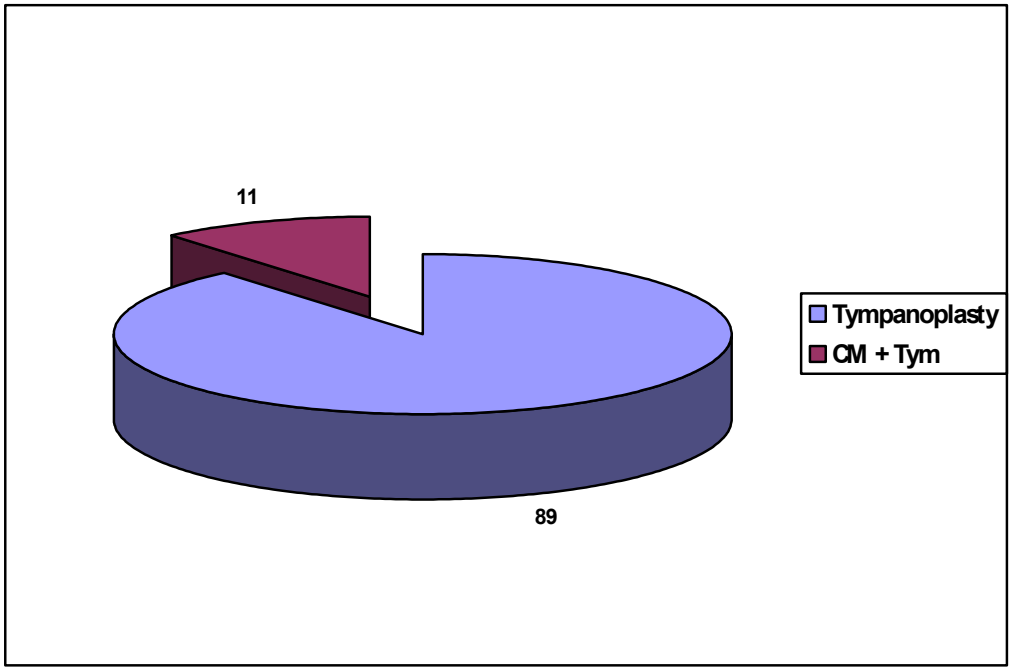


TABLE 5 : COMPARISON BETWEEN PREOPERATIVE & POSTOPERATIVE RESULTS

Variable	**Mean ± SD**	**(SE)**	**Paired t test**
Preoperative Vs Postop 3rd week	28.443 ± 6.932 22.743 ± 7.278	0.83 0.87	t = 15.32 p = 0.000 HS
Postoperative 3rd week Vs Postop 7th week	22.743 ± 7.278 17.789 ± 7.691	0.87 0.92	t = 13.15 p = 0.000 HS
Preoperative Vs Postop 7th week	28.394 ± 6.894 17.789 ± 7.691	0.82 0.92	t = 18.3 p = 0.000 HS

Standard deviation is a widely used measure of variability. It shows how much variation is there from average(mean or expected value). Low standard deviation indicates that the data points tend to be very close to the mean whereas high standard deviation indicates that the data is spread out over a large range of values.

Uses of standard deviation

- It summarizes the deviations of a large distribution from mean in one figure.
- Indicates whether the variation of difference of an individual from the mean is by chance .
- Helps in finding the standard error which determines whether the difference between means of two similar samples is by chance or real.

Standard Error of mean is the index of variability. It indicates how well the mean of a sample estimates the mean of a population. It is defined as Standard deviation / √n, where n is the sample size.

$$SEM=SD/\sqrt{n}$$

Probability may be defined as the relative frequency or probable chances with which an event is expected to occur on an average or in long run. Probability is usually expressed by the symbol 'p'. It provides the basis for all the tests of significance.

Tests of significance

These tests are mathematical methods by which the probability or relative frequency of an observed difference ,occurring by chance is found. Common tests in use are 'Z'test, 't'test and chi-square test. The first two tests express the difference observed in terms of standard error.

The ratio of observed difference between two means of small samples to the SE of difference in the same is denoted by the letter't'.

Probability 'p' of occurrence of any calculated value of 't' is determined by comparing it with the value given in the row of table corresponding to the degree of freedom derived from the number of observations in the samples under study.

The Paired-Samples T Test procedure is used to test the hypothesis of no difference between two variables. The data may consist of two measurements taken on the same subject or one measurement taken on a matched pair of subjects.

In this study Paired-Samples T Test procedure is used to test the significance difference between the two treatments (pre-post), given on the same patient. Here p value

is the probability of t- statistic. If p –value is < 0.05 then reject the hypothesis otherwise accept the hypothesis that there is no significant difference between the two treatment means

Postoperative hearing improvement between the two age groups was evaluated by air- bone gap closure. The results were catergorised into the following groups:[31]

i) Group A: remarkable improvement in which air –bone gap closure is 0-10 dB.

ii) Group B: moderate improvement in which air –bone gap closure is 11-20dB.

iii) Group C: slight improvement indicates air –bone gap closure of 21-30dB.

Iv) Group D: no improvement indicates air –bone gap of more than 31dB.

TABLE 6 A – B gap at 3rd week following surgery

Age (Yrs)	No of Patients	A (0-10 dB)		B (11-20 dB)		C (21-30 dB)		D (> 31 dB)		E (0-30 dB)	
		No	%	No	%	No	%	No	%	No	%
≤ 40	40	0	0	20	50	12	30	8	20	32	80
> 40	31	0	0	18	58	11	36	2	6	29	93
Total	71	0	0	38	54	23	32	10	14	61	86
		Excellent		Good		Fair		Failure		Satisfactory	

Using proportion of patients with a postoperative A-B gap of 30 dB as the criterion ,86% of patients achieved their A-B gap closer within 30 dB.

TABLE 7 Hearing results at 3rd postoperative week.

Age (years)	Mean A-B gap (dB) ±SD	p Value
≤40	23.08±7.57	t=0.12 p=0.903 NS
>40	22.87 ±6.48	

No statistically significant difference between the two age groups at 3rd post operative week.

TABLE 8 A **– B gap at 7 week following surgery**

Age (Yrs)	No of Patients	A (0-10 dB)		B (11-20 dB)		C (21-30 dB)		D (> 31 dB)		E (0-30 dB)	
		No	%	No	%	No	%	No	%	No	%
≤ 40	40	14	35	13	32	11	28	2	5	38	95
>40	31	8	26	15	48	7	23	1	3	30	97
Total	71	22	31	28	39	18	26	3	4	68	96
		Excellent		Good		Fair		Failure		Satisfactory	

Using proportion of patients with a postoperative A-B gap of 30 dB as the criterion ,in this study 96% of patients achieved their A-B gap closer within 30 dB at 7 th postoperative week .

TABLE : 9 Hearing results at 7thpostoperative week.

Age (years)	Mean A -B gap (dB) ± SD	p Value
≤40	17.80 ± 7.85	t=0.15 p=0.879 NS
>40	18.06 ± 6.67	

There was no statistically significant difference between the results in two age groups at the end of the 7th postoperative week.

TABLE 10 Success in relation to age

Age	Success (closure of TM)		Failure	
	No	%	No	%
≤40 Years	38 / 40	95%	2 / 40	5 %
> 40 Years	30 / 31	96.7%	1 / 31	3 %

p value= 0.712

Differences not significant on Chi-square test ($p > 0.05$)

Hence, from the above results it can be concluded that age did not significantly influence the results following middle ear surgery.

DISCUSSION

Despite a rapidly increasing proportion of elderly subjects in society,little is known regarding the effects of ageing on wound healing. Most studies claiming impaired wound healing with age have invariably been carried out on aged subjects with associated pathology,thus raising doubts about results ranging from the assessment of wound dehiscence rates and tensile strength to invitro fibroblast and keratinocyte studies[19].This study exclusively studies effect of age as an independent factor in healing of tympanic membrane following middle ear reconstructive surgeries.

There are different criteria for assessing hearing after chronic ear surgery such as social hearing method,hearing gain method and mean A-Bgap for each frequency but none are universally accepted method. The standard method of comparing postoperative air conduction to preoperative bone conduction appears most frequently in literature[4]. Thus, this method has been used for calculating hearing results in this study.

In a study by Radpour S, the instance of graft failure following middle ear surgery did not differ greatly by age. The risk of failure was higher in patients with concomitant diseases ,such as coronary artery disease, diabetes mellitus, hypertension,renal disease, cerebral vascular disease and anaemia[10]. This study has excluded patients with any of these coexisting conditions. Hence, outcome of surgery was not found to be different in any age group.

In a study by Aoyagi et al, the effect of aging on the preoperative and postoperative hearing results of tympanoplasty were assessed .Means of air bone gap were almost the same in each age group.[22] Similar results were found in this study.

In a study by Vrabec et al, the authors emphasised that age is unimportant. The basis for better surgical results lies in patient selection. Selecting patients for surgery is done on a case by case basis. Each presents with a unique combination of anatomic deficits , impaired function , unrelenting infection , and expectations for improvement . The goal of surgeon is to produce the best possible result for each. A categorical refusal to operate on a patient because of age would exclude some from significant benefit.[12]

SUMMARY

The effect of age as a factor in healing of tympanic membrane following middle ear surgeries was looked into by this prospective study on 71 patients who presented with CSOM to department of ENT in BLDEAs Shri B M Patil Medical college between October 2008 to April 2010. All these cases had a dry central perforation without any evidence of cholesteatoma. Comparisons were made between 2 groups of patients ,one below and the other above 40 years..

The results led to the conclusion that rate of success following surgery is likely not a matter of age but a matter of careful patient selection. No one variable determines the outcome. Clearly, some factors studied are age related, but age in itself should not be an indication or contraindication to treatment.

CONCLUSION

This study confirms that age as an individual factor had no significant role on the outcome of surgery. The presence of coexisting disease is more important than age itself. Hence ,careful patient selection was most likely to increase the rate of an intact tympanic membrane with improvement in hearing.

Therefore it was concluded that age is not a factor in success or failure of healing of tympanic membrane following middle ear reconstructive surgery.

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PROFROMA

CASE RECORD

1) Name: CASE NO:

2) Age: IP NO:

3) Sex: DOA:

4) Religion: DOS:

5) Occupation: DOD:

6) Residence:

7) Chief complaints

8) History of presenting illness

9) Past History:

1. Diabetes mellitus
2. Hypertension
3. History of any drug intake
4. History of jaundice or renal disease.

10) General Physical Examination

Pallor	present/absent
Icterus	present/absent
Clubbing	present/absent
Generalized Lymphadenopathy	present/absent

Build	Poor/Moderate /Well
Nourishment	Poor / Moderate/ Well

11) Vital signs

PR:	Temp:
BP:	Weight:
RR:	

12) Systemic Examination:

i. Respiratory System
ii. Cardiovascular System
iii. Central Nervous System
iv. Per abdomen

13) Local examination

Ear:	Right	Left
1. Pinna		
2. Pre-auricular area		
3. Post-auricular area		
4. Infra-auricular area		
5. External auditory canal		

6. Tympanic membrane

    Pars tensa

    Pars flaccida

7. Mastoid tenderness

8. Facial nerve functioning

9. Nystagmus

10. Tuning Fork test

    a. Rinnes test

    b. Webers test

    c. ABC test

Nose:

1. Extenal Appearance

2. Cold Spatula test

3. Anterior rhinoscopy

4. Posterior rhinoscopy

5. Paranasal sinus tenderness

Throat:

1. Oral cavity
2. Oropharynx
3. IDL Examination

14) Investigation:

Blood: Hb %

TC

DC

ESR

BT

CT

HbsAg

HIV

BLOOD UREA

SERUM CREATININE

RBS

X-ray :Bilateral Mastoids

Urine: Albumin, Sugar, Micro

15) Pure tone audiometry

16) Final Diagnosis:

17) Treatment:

Medical**:** Aural toilet

Antibiotics-systemic & local

Decongestants

Surgical**:** Anaesthesia: GA/LA with sedation

Incision: Post aural/ Endaural/ Permeatal

Mastoid Exploration: done/not done

Operative findings

Ossicular chain: intact/ disrupted

Graft: Temporalis fascia/ vein/ perichondrium

Graft placed: inlay/ interlay/ onlay

18) Inference:

19) Comments:

B. L. D. E.A'S, BIJAPUR

SAMPLE INFORMED CONSENT

PURPOSE OF RESEARCH:

I have been informed that this is a study to assess the role of age as a factor in healing of tympanic membrane following middle ear reconstructive surgeries . I have also been given a free choice of participation in this study.

PROCEDURE:

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

RISK AND DISCOMFORTS:

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

BENEFITS

I understand that my participation in this study will help to assess role of age as a factor in healing of tympanic membrane following middle ear reconstructive surgeries.

CONFIDENTIALITY

I understand that medical information produced by this study will become part of Hospital records and will be subject to the confidentiality and privacy regulation . Information of a sensitive personal nature will not be a part of medical record, but will be

stored in investigators research file and identified only by a code number. The code key connecting name two numbers will be kept in a separate secured location.

If the data are used for publication in the medical literature and for teaching purposes no names will be used and other identities such as photographs, audio and video tapes will be used only with my special written permission. I understand I may see the photographs and the video tapes and have the audio tapes before giving this permission.

REQUEST FOR MORE INFORMATION:

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

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

REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and that i may refuse to participate or may withdraw my consent and discontinue participation in the study at any time without prejudice to my present or future care at this hospital. I also understand that Dr Preety Ninan may terminate my participation in this study at any time after she has

explained the reasons for doing so and has helped arrange for my continued care by my own physician or physical therapist, if this is appropriate.

INJURY STATEMENT:

I understand that in unlikely event of injury to me resulting directly from my participation in this study, if such injury were reported promptly, then medical treatment will be available to me, but no further compensation would be provided.

I understand that by my agreement to participate in this study I am not waiving any of my legal rights.

I confirm that Dr Preety Ninan has explained to me the purpose of research, the study procedure that I will undergo, and the possible risk and discomforts as well as benefits that I may experience in my own language. I have read and I understand this consent form . Therefore I agree to give consent to participate as a subject inthis research project.

Participant / Guardian Date

Witness to signature Date:

Key to Master Chart

A – B gap	:	Air Bone gap
B/ L	:	Bilatral
CSOM	:	Chronic Suppurative otitis media
CM	:	Cortical Mastoidectomy
dB	:	Decibel
F L	:	Failure
f	:	Female
Khz	:	Kilohertz
Lt	:	Left
m	:	Male
Rt	:	Right

MASTER CHART

Sl no	Name	Age	Sex	Date	Ip no	Diagnosis	Treatment	Preoperative				Postoperative 3rd week				Postoperative 7TH week				
								Air conduction threshold			A-B gap	Air conduction threshold			A-B gap	Air conduction threshold			A-B gap	
								0.5 Khz	1 khz	2 Khz	(mean)	0.5 Khz	1Khz	2 Khz	(mean)	0.5 Khz	1 Khz	2 Khz	(mean)	
1	Kamala	55	f	21/10/08	14372	Lt CSOM	Lt type 1 tympanoplasty	35	30	40	30	25	20	30	20	25	20	25	15	
2	Sujata	19	f	7/11/2008	15087	Rt CSOM	Rt type 1 tympanoplasty	40	30	35	30	35	30	25	22	30	25	25	15	
3	Pulabai	55	f	24/11/08	15892	B/L CSOM	Rt CM+type 1 tympanoplasty	50	45	35	30	45	45	30	25	40	40	25	20	
4	Drakshyani	46	f	19/12/08	16475	Lt CSOM	Lt CM+type 1 tympanoplasty	40	35	40	30	40	40	35	30	45	40	45	35	FL
5	Vijaylakshmi	42	f	2/1/2009	70	Lt CSOM	Lt type 1 tympanoplasty	30	35	30	20	25	25	30	15	20	25	20	10	
6	Ravindra	65	m	7/1/2009	219	B/L CSOM	Lt CM+ type 1 typanoplsty	40	35	35	30	30	30	25	24	30	25	25	20	
7	Renuka	30	f	23/1/09	222	Rt CSOM	Rt type 1 tympanoplasty	50	40	45	35	45	40	40	30	40	35	40	25	
8	Sangeeta	20	f	11/1/2009	456	Rt CSOM	Rt type 1 tympanoplasty	30	25	30	20	30	40	30	30	40	45	35	35	FL
9	Gourawwa	55	f	23/1/09	885	Lt CSOM	Lt type 1 tympanoplasty	30	35	25	25	25	30	20	20	20	25	15	15	
10	Ramesh	20	m	23/1/09	919	Rt CSOM	Rt type1 tympanoplasty	30	25	30	20	20	20	25	15	15	20	20	10	
11	Shakuntala	58	f	13/3/09	2172	Rt CSOM	Rt type 1 tympanoplasty	40	30	30	25	30	20	25	20	25	20	15	15	
12	Iranna	18	m	25/2/09	2245	B/L CSOM	Lt type 1 tympanoplasty	35	35	30	20	25	35	25	15	20	25	25	10	
13	Shobha	24	f	27/3/09	3565	B/L CSOM	Rt type 1 tympanoplasty	55	45	50	35	45	40	40	30	40	40	35	25	
14	Poornima	10	f	7/4/2009	4103	B/L CSOM	Lt type 1 tympanoplasty	40	35	35	40	35	35	30	35	35	30	25	20	
15	Siddaraya	50	m	13/4/09	4286	Lt CSOM	Lt type 1 tympanoplasty	50	40	40	35	40	35	35	30	35	30	25	25	
16	Mahadevi	45	f	6/5/2009	4004	Lt CSOM	Lt type 1 tympanoplasty	35	30	35	25	25	25	30	18	20	15	20	10	
17	Ningappa	52	m	2/5/2009	5051	Lt CSOM	Lt type 1 tympanoplasty	30	30	35	30	25	20	25	20	20	20	15	20	
18	Haziya	32	f	4/5/2009	5145	Lt CSOM	Lt type 1 tympanoplasty	40	30	35	40	35	25	35	32	30	25	30	25	
19	Mahadeveppa	47	m	20/5/09	5933	Lt CSOM	Lt type 1 tympanoplasty	30	25	35	25	25	20	25	20	20	15	25	10	
20	Sharada	56	f	26/5/09	6296	B/L CSOM	Rt type 1 tympanoplasty	35	30	35	20	30	30	25	15	25	25	20	10	
21	Veeresh	20	m	26/5/09	6259	B/L CSOM	Rt type1 tympanoplasty	30	25	35	20	25	25	20	15	20	25	15	10	
22	Sunanda	25	f	30/5/09	6525	B/L CSOM	Rt CM+type 1 tympanoplasty	45	50	40	25	40	40	35	20	35	40	35	15	
23	Mallawa	55	f	9/6/2009	7022	B/L CSOM	Rt type 1 tympanoplasty	30	35	35	20	30	25	30	15	25	30	25	15	
24	Vittal	70	m	10/6/2009	7092	Lt CSOM	Lt type 1 tympanoplasty	50	50	45	25	40	45	35	20	35	35	35	15	
25	Shwetha	15	f	8/7/2009	8679	B/L CSOM	Lt type 1 tympanoplasty	35	35	40	25	30	30	30	15	25	20	25	10	

26	Bhagyashree	17	f	9/7/2009	8772	B/L CSOM	Lt type 1 tympanoplasty	45	40	40	30	40	35	35	22	35	35	30	20	
27	Shivamma	22	f	28/7/09	9942	Rt CSOM	Rt type 1 tympanoplasty	40	35	35	30	30	30	25	20	25	20	15	10	
28	Siddappa	42	m	2/8/2009	9984	Rt CSOM	Rt CM +type 1 tympanoplasty	30	25	30	20	20	25	25	15	20	15	20	10	
29	Sharanawa	65	f	11/8/2009	10837	Lt CSOM	Lt type 1 tympanoplasty	40	35	40	35	35	40	35	35	30	30	25	25	
30	Shanta	48	f	19/8/09	11378	Lt CSOM	Lt type 1 tympanoplasty	35	40	40	25	30	40	35	20	30	35	35	20	
31	Anitha	21	f	20/8/09	11438	Rt CSOM	Rt type1 tympanoplasty	45	40	35	30	45	50	35	35	45	40	40	32	FL
32	Iramma	42	f	8/9/2009	12645	Lt CSOM	Left type 1 tympanoplasty	35	30	30	25	25	20	25	15	20	20	15	10	
33	Somayya	25	m	21/9/09	13888	Rt CSOM	Rt CM+type1 tympanoplasty	35	30	35	30	25	25	30	25	30	20	15	20	
34	Mallikarjun	46	m	25/9/09	14125	Lt CSOM	Left type 1 tympanoplasty	40	35	35	30	35	35	25	22	25	25	20	15	
35	Siddappa	22	m	4/10/2009	14497	Lt CSOM	Lt type 1 tympanoplasty	35	30	25	20	30	30	20	15	25	20	15	10	
36	Sujata	25	f	23/10/09	15733	B/L CSOM	Rt type1 tympanoplasty	45	50	55	35	45	50	50	35	40	45	45	30	
37	Laksmibai	63	f	27/10/09	15960	Rt CSOM	Rt type 1 tympanoplasty	50	40	40	40	35	40	35	30	25	30	30	25	
38	Vittal	21	m	28/10/09	16049	Lt CSOM	Lt CM+ type 1 tympanoplasty	35	35	30	25	30	25	25	20	20	20	15	15	
39	Mahantesh	31	m	30/10/09	16228	Rt CSOM	Rt type 1 tympanoplasty	40	35	40	30	30	25	30	20	25	20	25	20	
40	Sangamma	17	f	6/11/2009	16591	B/L CSOM	Rt type III tympanoplasty	25	30	30	20	20	25	20	12	20	20	20	10	
41	Muttana	25	m	9/11/2009	16738	B/L CSOM	Rt type 1 tympanoplasty	45	35	25	38	40	30	25	31	35	30	25	30	
42	Vimala	42	f	12/11/2009	16969	Lt CSOM	Lt type 1 tympanoplasty	35	30	30	25	30	25	25	20	25	20	20	15	
43	Basalingamma	42	f	15/11/09	17111	B/L CSOM	Rt type 1 tympanoplasty	45	45	55	40	45	40	55	40	40	45	50	30	
44	Baburao	53	m	16/11/09	17112	Rt CSOM	Rt Atticotomy+type 1 tympanoplasty	45	50	35	35	35	40	30	28	30	30	20	20	
45	Anand	50	m	17/11/09	17213	Lt CSOM	Lt type 1 tympanoplasty	35	30	30	25	25	20	25	20	15	20	20	15	
46	Mahadevi	58	f	20/11/09	17387	Lt CSOM	Lt type 1 tympanoplasty	35	40	40	30	30	35	40	25	30	40	40	25	
47	Bhagyarathi	65	f	30/11/09	17912	Rt CSOM	Rt type1 tympanoplasty	55	55	45	35	45	40	40	30	40	40	40	25	
48	Ambuja	20	f	15/12/09	18733	B/L CSOM	Rt type1 tympanoplasty	50	40	25	40	45	35	25	35	35	35	25	30	
49	Umadevi	28	f	17/12/09	18848	B/L CSOM	Lt type 1 tympanoplasty	55	55	50	35	50	55	50	35	45	50	45	30	
50	Ningawwa	60	f	18/12/09	18355	Lt CSOM	Lt type 1 tympanoplasty	50	45	40	40	40	35	30	30	35	25	30	25	
51	Basamma	30	f	31/12/09	19526	B/L CSOM	Lt type 1 tympanoplasty	35	25	25	20	30	25	25	15	25	20	20	10	
52	Gradison	14	m	5/1/2010	260	B/L CSOM	Lt type 1 tympanoplasty	50	40	35	25	40	30	30	18	35	30	20	10	
53	Anitha	28	f	8/1/2010	438	Rt CSOM	Rt type 1 tympanoplasty	40	35	25	25	30	25	15	20	20	15	15	15	
54	Lalitha	30	f	15/1/10	766	Lt CSOM	Lt type 1 tympanoplasty	45	50	40	42	30	30	20	30	25	20	20	20	
55	Sunitha	25	f	18/1/10	936	B/L CSOM	Rt type1 tympanoplasty	45	45	50	25	35	40	40	20	25	30	25	10	
56	Vidya	16	f	20/1/10	1123	Rt CSOM	Rt type1 tympanoplasty	40	35	35	30	30	25	25	20	25	20	15	15	
57	Prasanth	52	m	25/1/10	1324	Lt CSOM	Lt type 1 tympanoplasty	35	35	40	30	25	20	20	20	20	15	15	15	

58	Basangouda	42	m	3/2/2010	1753	B/L CSOM	Rt type 1 tympanoplasty	35	40	40	20	30	30	35	15	30	30	30	10	
59	Muttana	11	m	6/2/2010	1991	Lt CSOM	Lt type 1 tympanoplasty	35	35	30	20	30	25	25	15	25	20	20	10	
60	Sunanda	55	f	14/2/10	2451	Rt CSOM	Rt type1 tympanoplasty	45	40	35	30	40	40	40	30	35	40	30	25	
61	Heena	20	f	23/2/10	3239	B/L CSOM	Rt type1 tympanoplasty	55	50	50	40	50	45	40	35	40	40	30	25	
62	Mallapa	30	m	28/2/10	3686	B/L CSOM	Lt type 1 tympanoplasty	45	35	35	25	25	20	20	18	20	15	20	15	
63	Jayshree	27	f	9/3/2010	4279	Lt CSOM	Lt type 1 tympanoplasty	40	30	35	22	30	25	25	15	25	25	25	10	
64	Ambawwa	25	f	11/3/2010	5161	Rt CSOM	Rt type1 tympanoplasty	35	30	30	25	25	25	20	15	20	15	15	10	
65	Jyoti	17	f	22/3/10	4279	Lt CSOM	Lt type 1 tympanoplasty	40	30	20	27	25	25	30	22	20	20	25	15	
66	Prakash	42	m	23/3/10	5827	B/L CSOM	Rt type1 tympanoplasty	40	35	35	25	35	35	30	22	30	25	25	15	
67	Prasad	19	m	23/3/10	5948	B/L CSOM	Rt type1 tympanoplasty	30	25	25	20	25	20	20	15	20	15	20	10	
68	Prema	24	f	23/3/10	6297	Rt CSOM	Rt type1 tympanoplasty	55	50	45	35	50	40	35	30	45	40	30	25	
69	Mruntjay	12	m	25/3/10	6391	Lt CSOM	Lt type 1 tympanoplasty	50	40	35	40	30	35	25	30	30	25	20	25	
70	Husenbasha	25	m	26/3/10	6619	Lt CSOM	Lt type 1 tympanoplasty	45	40	30	32	30	25	20	25	25	20	15	20	
71	Sachin	9	m	29/3/10	6917	B/L CSOM	Rt CM+type 1 tympanoplasty	30	30	30	20	10	15	20	16	10	10	15	10	