A DISSERTATION ON

CARTILAGE TYMPANOPLASTY IN ATELECTATIC EARS

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CERTIFICATE

This is to certify that this dissertation entitled " **CARTILAGE TYMPANOPLASTY IN ATELECTATIC EARS**" submitted by DR.Y. SEETHY to the faculty of OTORHINO LARYNGOLOGY, The TamilNadu Dr. M.G.R. Medical University, Chennai, in partial fulfilment of the requirement in the award of degree of M.S.Degree, Branch – IV (OTO - RHINO LARYNGOLOGY), for the March 2007 examination is a bonafide research work carried out by him under our direct supervision and guidance.

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This is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfilment of the requirement for the award of M.S.Degree,Branch IV(OTO - RHINO LARYNGOLOGY) degree Examination to be held in March 2007.

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CONTENTS

Section – A *Page No.*

1.	INTRODUCTION	1
2.	AIM OF THE STUDY	2
3.	REVIEW OF LITERATURE	3
4.	DEVELOPMENT OF MIDDLE EAR	10
5.	ANATOMY OF THE MIDDLE EAR CLEFT	13
6.	PHYSIOLOGY	30
7.	PATHOGENESIS OF RETRACTION	35
8.	ATELECTASIS OF MIDDLE EAR	40
9.	ADHESIVE OTITIS MEDIA	46
10.	MATERIALS & METHODS	48
11.	RESULTS	53
12.	DISCUSSION	57
13.	CONCLUSION	62

BIBLIOGRAPHY

MASTER CHART

INTRODUCTION

Middle ear effusion and Atelectasis with Eustachian tube dysfunction is a multifactorial multifaceted disease that manifest in the middle ear, mastoid and Eustachian tube.

Understanding the mechanism of this disease, the presentation and issues involved in its management allows for the most possible outcomes.

Surgery for the atelectatic tympanic membrane ranges from ventilation tube insertion to several others.

In this prospective study we used conchal cartilage graft to reverse atelectasis and to repneumatize the middle ear in atelectatic ears. Long term results has to be tested in time future.

AIMS AND OBJECTIVES

- A comprehensive study of cartilage perichondrium tympanoplasty in Stage III and stage IV atelectatic Ears.
- To analyse the stability of the cartilage
- To analyse the hearing improvement after the procedure

REVIEW OF LITERATURE

Sade. J. Avraham S, and Brown. M, (1982) studied about dynamics of atelectasis and retraction pockets In : Cholesteatoma and Mastoid Surgery (Proceedings of the IInd International conference, edited by J.Sade Amsterdam : Kigler, pp 267 – 282.

Sade. J and berco E, (1976) studied about Atelectasis and secretory otitis media (American journal of Otolaryngology).

Flishberg et al 1963; Miller 1965, have developed for assessing the function of the Eustachian tube and one of the most reliable is the inflation –deflation test. This is a method for examining the passage of the tube by adding to the pressure in the middle ear from the external ear through an eardrum perforation, a myringotomy, or a tympanostomy tube and examining whether the applied positive or negative middle ear pressure can be equalized by swallowing etc.

Takahashi et al 1995 observed that even a normal individual who is not otitis-prone sometimes has tubal dysfunction with an upper respiratory tract infection and needs several weeks to recover.

Bluestone et al 1972, in their study observed that the negative middle ear pressure equalizing function is impaired in almost all ears with otitis media with effusion.

A pathological condition of the Eustachian tube is a patulous tube with an excessively wide passage rather than stenosis, and this is often seen in ears with cholesteatoma and adhesive otitis media. The condition is called ‘eustachian tube closing failure” and is considered to be closely related to the pathogenesis of these conditions. (Magnuson 1978).

When the nose is bilaterally blocked, negative pressure is sometimes induced naturally in the middle ear by swallowing. This is called the “Toynbee phenomenon” and is also noteworthy as a unique pathological condition of the Eustachian tube which is actively and repeatedly causing negative middle ear pressure with every swallow during nasal allergy or upper respiratory tract infection, resulting in various middle ear diseases. (Sudo et al 1999).

Proud et al (1971), monitoring middle ear pressure for 24-36 h after ligating the eustachian tube in cats, failed to show a high negative pressure (over 90mm H2O) and Cantekin et al) (1980)

showed that the middle air pressure remained at approximately 50 mm H2O. when physiological respiration was maintained in dogs under general anesthesia.

Honjo 1988; Takahashi et al 1990, studied that the middle ear pressures in cats for several weeks by tympanograms after relatively abolishing tubal ventilatory functions by transecting the tensor veli palatine muscle and hamulus pterygoideus, but failed to find the frequent formation of high negative middle ear pressure or otitis media with effusion.

Bylander et al 1985, monitored the middle ear pressure of children with tubal dysfunction for more than 24 h by tympanogram, and found that their middle ears showed alternate positive and negative pressures during sleeping and waking, respectively.

Hergils et al 1985, found that the middle ear of many normal individuals showed positive pressure when they wokeup in the morning.

Buckingham et al 1985, through middle ear pressure monitoring of dogs under general anesthesia, demonstrated that the middle ear pressure varied according to their respiratory condition ;

positive pressure during hypoventilation and negative pressure during hyperventilation.

Gantekin et al (1980) showed experimentally that the speed and degree of middle ear pressure decrease in dogs depended upon the gas inflated into the middle ear.

Sade et al (1989) inflated various gases in to the middle ears of patients with atelectatic ears and found that the speed of normalization of the protruded eardrums after inflation varied according to the gases inflated.

Iwano et al, (1993) also reported that human middle ear pressure changed in relation to the gases inflated into the middle ear.

Sade et al 1995, pointed out that the middle ear gas composition is similar to that in the venous blood in humans, and later his colleagues (Levy et al 1995) directly detected inhaled inert gas in the middle ear. Thus the existence of a gas exchange function through the middle ear mucosa was confirmed.

Hergils et al (1985) and Iwano et al (1993) demonstrated in humans that applied negative and or positive middle ear pressures tend to approach atmosphere pressure if the person does not swallow.

Gates et al 1980, O'Neill et al 1985 observed that the gas exchange function in the middle ear is impaired by materials which increases the distance between the middle air cavity and the capillaries, such as edematous mucosa, granulation, or scars in the middle ear. Most typically, the function stops when the middle ear is filled with effusion or granulation without leaving any air space. In other words, an air space in the middle ear into which gases enter from the blood is necessary for gas exchange ; more precisely an air fluid interface is necessary..

Bylander et al 1985, observed that the sudden change in atmospheric pressure, and it cannot regulate the middle ear pressure at precisely atmospheric level because even a minute alteration in the blood gas composition (particularly carbon dioxide) for instance during sleeping, has a considerable influence on the middle ear pressure.

Bylander et al 1981, observed that the Eustachian tube appears to be too delicate to be relied upon as the only system controlling middle ear ventilation and pressure regulation.

Matsune et al 1996 showed that an important function of the Eustachian tube, the protection of the middle ear is mainly carried out by morphological features suchas submucosal lymphoid follicles.

Okubo 1993, reported that advocating the idea that gases are always produced in the middle ear (mastoid) and expelled through the Eustachian tube : in other words, that both ventilation and clearance are directed from the middle ear to the nasopharynx.

Neumann, Andreas, Schultz Coulon, Hans Jurgen, Jahnke, Klaus. In their article (Otology & Neurotology 24(1) 33-37 January 2003), one type III Tympanoplasty applying the Palisade Cartilage Technique reported that the palisade cartilage technique is suitable to manage difficult pathologic conditions in middle ear surgery. It was demonstrated that the palisade cartilage technique can be combined safely with titanium ossicular replacement prosthesis.

Yu LS, QiZM, the article on operative therapy of the adhesive otitis media showed that adhesive otitis media can be treated with cartilage tympanoplasty. The cartilage was a good material for reconstruction of the ear drum to the treatment of it.

Sade et al 1982, showed that the ventilating tube insertion is the commonest surgical procedure performed. This can arrest further progression in about 60% of grade 1 retraction pockets of pars tensa.

Srinivasan et al 2000 showed that the retraction is deemed amenable for complete excision, and it is their experience to perform this procedure in both ears at the same time as a day care procedure both in children and adults. And success rate is around 65% in retractions of grade 1 to 3.

Levinson 1987, characion et al 1992, Yung 1997 showed that the cartilage is considered to provide good re enforcement for the healing tympanic membrane. The reported recurrence rate of retraction with this procedure varies from 5% to 45%.

Desarda KK, Bhisegaonkar DA, Gill S Tragal perichondrium and cartilage in reconstructive tympanoplasty, Indian Otolaryngol Head Neck Surg, 2005 ; 57 : 9- 12, In their study, they strongly recommend the tragal pericondrium and cartilage composite graft in various tympanoplasty reconstructions. The hearing improvement within 15 dB of bone conduction has become almost a standard criterion for the analysis of surgical success.

DEVELOPMENT OF MIDDLE EAR

The cavity and lining of the middle ear cleft and Eustachian tube arise from the expanding first pharyngeal pouch with probably some contribution from medial edge of the second.

By the 4^{th} week stage the distal end lies against the ectoderm of the first pharyngeal groove and expands to form a flattened sac the precursor of the tympanic cavity. Mesenchyme grows between the Ectoderm and endoderm to form the third layer of the future tympanic membrane. The slit like space within the sac expands and as it reaches the developing ossicles and otic capsule the epithelium lining the sac is draped over the tympanic portion of the labrynths. The bodies of ossicles and their developing ligaments and muscular tendons so that a complex and variable networks of mucosal folds is formed. Pneumatisation of the meso and hypotympannum is complete at 8 months while the epitympanum and mastoid antrum has been developed by birth. The mastoid antrum which is an extension of epitympanum has started to develop in midfetal life. A few mastoid air cells are present in fetal life, but the bulk of their development takes place in infancy and childhood.

Development of Ossicles :

The outer lateral ends of the 1st (Meckel's) and 2nd (Reicherts) arch cartilage lie respectively, above and below the developing first pharyngeal pouch. Before these arch cartilages are fully defined,, condensations in the mesenchyme appear in this region at about 5th week. As development proceeds, the condensations from cartilage models which by 6th week are well defined as malleus, incus, stapes. By 5th week, the stapes can be recognized as a circular mass at the end of the precursor of Reicherts (2nd arch) cartilage. Approximately 2 weeks later, this becomes annular as it is pierced by (1st arch stapideal artery) and is now attached to the developing Reicharts cartilage by a membranous bar, the interhyale. At this time, the malleus and incus are developing from cartilage at the end of precursor of Meckels cartilage. A groove represents the site of the future incudomalleolar joint and the handle of malleus and long process of incus are already apparent. By 7th week the handle of malleus lies between the layers of the developing tympanic membrane.

The stapes continues to grow and its ring like shape is converted into the definitive arch like stapedial form. It seem likely that the foot plate of the stapes in formed primarly from the optic capsule, and that part of the stapedial ring which fuses with the otic capsule during ossifications usually regresses. In the adult, therefore, the stapedial arches are developed from 2^{nd} arch cartilage while the foot plate is part of the labrynthine capsule.

Frequently, regression of the base of the stapedial ring is incomplete so that a dual origin for the mature foot plate is possible.

Ossification in the stapedial cartilage starts from a single centre at 4-5 months and is followed by a complex pattern of resorption with the result that the crura and the adjoint head are eventually hollowed out.

The malleus and incus start ossifying at the 4 month stage and progress so rapidly that in the 25 week fetus they are already of adult size and forms.

ANATOMY OF THE MIDDLE EAR CLEFT

The middle ear cleft consists of the tympanic cavity, the eustachian tube and the mastoid air cell system. Included in this sections are the extension of the air cell system in to the anterior and posterior petrous apex.

The tympanic cavity is an irregular air filled space within the temporal bone and contains the auditory ossicles and attached muscles.

For descriptive purposes, the tympanic cavity may be thought of as a box with four walls, a roof and a floor. The corners are not sharp and therefore the precise localization of features lying at the edge of one wall may not be possible with this model.

The lateral wall of the tympanic Cavity :

The lateral wall of the tympanic cavity is part bony and part membranous. The tympanic membrane forms the central portion of the lateral wall, while above and below there is bone, forming the outer lateral walls of the epitympanum and hypotympanum

respectively. The lateral wall of the epitympanum also includes that part of the tympanic membrane lying above the anterior and posterior malleolar folds – the pars flaccida. This lateral epitympanic wall is wedge shaped in section and its lower bony portion is also called the outer attic wall or scutum (Latin – shield). It is thin and its lateral surface forms the superior portion of the deep part of the external meatus.

Three holes are present in the bone of the medial surface of the lateral wall of the tympanic cavity. The opening of the posterior canaliculus for the chorda tympani nerve is situated in the angle between the junction of the lateral and posterior walls of the tympanic cavity. It is often at the level of the upper end of the handle of the malleus. But a lower situation is very common. The opening leads into a small bony canal which descends through the posterior wall of the tympanic cavity. Near the tympanic opening, the chorda tympani lies anterior and lateral to the facial nerve ; it descends obliquely to join the nerve often at some point within the bone, but occasionally the channel remains separate and the two

nerves join outside the skull. A branch of the stylomastoid artery accompanies the chorda tympani into the tympanic cavity.

The petrotympanic (Glasserian) fissure opens anteriorly just above the attachment of the tympanic membrane. It is slit about 2 mm long which receives the anterior malleolar ligaments and transmits the anterior tympanic branch of the maxillary artery to the tympanic cavity. The chorda tympani enteres the medial surface of the fissure through a separate anterior (canaliculus canal of Huguier) which is short and is sometimes confluent with the fissure.

Tympanic Membrane :

The tympanic membrane emulates an irregular, the apex of which is formed by the umbo (at the tip of manubrium). The adult tympanic membrane is about 9 mm in diameter and subtends an acute angle with respect to the inferior wall of external auditory canal of 55 degree.

The fibrous annulus of tympanic membrane anchors in the tympanic sulcus. In addition, tymapanic membranefirmly attaches to

the malleus at the lateral process and at the umbo, between these two points, only a flimsy mucosal fold, the plica mallearis, connects tympanic to the malleus.

The tympanic membrane is separated into a superior par flaccida (shrapnells membrane) and an inferior pars tensa by the anterior and posterior tympanic stria which runs from the lateral process of the malleus to the anterior and posterior tympanic spines respectively.

The tympanic membrane is a trilaminar structure. The lateral surface is formed by squamous epithelium whereas medial layer is a continuation of the mucosal epithelium of the middle ear. Between these layers is a fibrous layer, as the pars propria. The pars propria at the umbo splits to envelop the distal tip of the manubrium.

The epidermis is divided into the stratum corneum, the stratum granulosum, the stratum spinosum and stratum basale. In man (Hentzer, 1969) the stratum corneum, which is the outermost layer, consists of between one and six compressed layers of almost acellular structures, without organelles but with recognizable membranes and intercellular junctions (desmosomes). The stratum

granulosum contains one to three layers of cells with smooth borders and interconnecting desmosomes. Keratohyaline granules and lamellar granules are present among occasional tonofilaments, but other cell constituents are lacking. The cells of the stratus spinosum, which are two or three layers deep, have prominent interdigitations with neighbouring cells to which they are bound by desmosomes. These cells contain bundles of tonofilaments with the mitochondrila and ribosomes also present, but have a high nucleus to cytoplasm ratio. The stratum basale, which is the deepest layer, consists of a single layer of cells separated from the lamina propria by a basement membrane. These cells have a polyhedral shape, and are elongated in a line parallel to the basement membrane. Occasionally prolongations of the deep surface of the cell extent down into the lamina propria. Nerve endings and melanin granules have not been seen in any of the cell layers of the epidermis.

The predominant feature of the lamina propria in both the pars tensa and the pars flaccida is the presence of collagen fibrils. In the pars tensa, the fibris closest to the epithelial layers are usually in direct contact with the basement membrane of the epidermal layer,

although in places a thin layer of loose connective tissue intervenes. These lateral fibres are radial in orientation, while the deeper ones are circular, parabolic and transverse. A loose connective tissue layer, containing fibroblasts, macrophages, nerve fibres (mainly unmyelinated) and many capillaries, lies between the deep layers of the lamina propria and the inner mucosal layer. Neither the capillaries nor the nerves appear to penetrate the basement membrane or enter the mucosal layer.

In the pars flaccida, the lamina propria is less marked, but it still contains collagen fibres although they appear to lie in an almost random orientation.

The mucosal epithelium of the pars tensa varies in height from a low simple squamous or cuboidal type to a pseudoostratified columnar epithelium. The adjoining cell borders have marked interdigitations with tight junctions between the apices of the cells facing the tympanic cavity. The free surface of the cells, ie. the surface facing the middle ear, possesses numerous microvilli and where the epithelium is cuboidal or columnar, cilia with the typical 'nine plus two' internal ultrastructure are found. These true cilia are

patchy in their distribution and a continuous sheet, such as that which covers the respiratory mucosa of, say the Eustachian tube, is not found. No goblet cells have been found in this layer, but in cells without cilia, secretory granules are present. The cytoplasm and nuclei of the cells are otherwise unremarkable. The mucosal layer is separated from the lamina propria by a basement membrane. In the pars flaccida, the overall picture is the same except that taller ciliated cells are not found.

Blood supply of the tympanic membrane :

The arterial supply of the tympanic membrane is complex and arises from branches supplying both the external auditory meatus and the middle ear. These two sources interconnect through extensive anastomoses, but the vessels are found only in the connective tissue layers of the lamina propria. Within this layer there appears to be a peripheral ring of articles connected by radial anastomoses, with one or two arteries that run down each side and around the tip of the malleus handle. The arteries involved include the deep auricular branch of the maxillary artery coming from the

external auditory meatus and from the middle ear, the anterior tympanic branches of the maxillary artery, twigs from the stylomastoid branch of the posterior auricular and probably several twigs from the middle meningeal artery.

The venous drainage returns to the external jugular vein, the transverse sinus, dural veins and the venous plexus around the Eustachian tube.

Nerve supply of the tympanic membrane :

The nerves, in the same way as the blood vessels, run in the lamina propria and arise from the auriculo temporal nerve supplying the anterior portion, from the auricular branch of the vagus, the posterior portion and from the tympanic branch of the glossopharyngeal nerve. The variations and overlap are considerable, but both the vascular supply and innervation are relatively sparse in the middle part of the posterior half of the tympanic membrane.

The medial wall of the tympanic cavity

The medial wall separates the tympanic cavity from the inner ear. Its surface possesses several prominent features and two openings. The promontory is a rounded elevation occupying much of the central portion of the medial wall. It usually has small grooves on its surface and these contain the nerves which form the tympanic plexus. Sometimes the grooves, especially the groove containing the tympanic branch of the glossopharyngeal nerve are covered by bone, with the consequence that small canals are present instead. The promontory covers part of the basal coil of the cochlea and in front merges with the anterior wall of the tympanic cavity.

Behind and above the promontoty is the fenestra vestibule (oval window) a nearly kidney shaped opening that connects the tympanic cavity with the vestibule but which in life is closed by the base of the stapes and its surrounding annular ligament. The long axis of the fenestra vestibule is horizontal, and the slightly concave border is inferior. The size of the fenestra vestibule naturally varies with the size of the base of the stapes, but on average it is 3.25 mm

long and 1.75 mm wide. Above the fenestra vestibule is the facial nerve and below is the promontory. The fenestra, therefore lies at the bottom of a depression or fossula that can be varying width depending on the position of the facial nerve and the prominence of the promontory.

The fenestra cochleae (round window), which is closed by the secondary tympanic membrane (round window membrane) lies below and a little behind the fenestra vestibule from which it is separated by a posterior extension of the promontory called the subiculum. Occassionally, a spicule of bone leaves the promontory above the subculum and runs to the pyramid on the posterior wall of the cavity. This spicule is called the ponticulus.

The roof of the tympanic cavity :

The tegmen tympani is the bony roof of the tympanic cavity, and separates it from the dura of the middle cranial fossa. It is formed in part by the petrous and part by the squamous bone ; and the petrosquamous suture line, unossified in the young, does not

close until adult life. Veins from the tympanic cavity running to the superior petrosal sinus pass through this suture line.

The floor of the tympanic cavity :

The floor of the tympanic cavity is much narrower than the roof and consists of a thin plate of bone which separates the tympanic cavity from the dome of the jugular bulb. Occasionally, the floor is deficient and the jugular bulb is then covered only by fibrous tissue and a mucous membrane. At the junction of the floor and the medial wall of the cavity there is a small opening that allows the entry of the tympanic branch of the glossopharyngeal nerve into the middle ear from its origin below the base of the skull.

The anterior wall of the tympanic cavity :

The anterior wall is rather narrow and comprises mainly the opening into the Eustachian tube. Above this is the canal for the tensor tympani tendon and medial to both of these runs the carotid artery as it turns from the middle ear segment forward into the carotid sinus before entering the skull in the cavernous sinus.

Eustachian tube :

The Eustachian tube extends approximately 3.5 mm from anterior aspect of the tympanic cavity to the posterior aspect of the nasopharynx and serve to ventilate, clear and protect the middle ear. The lining mucosa of the tube has an abundance of mucociliary cells important to its clearance function.

The tube is in the adult, about 36 mm long with the lateral, that is the bony middle ear segment, being approximately 12 mm long. It narrows down form its wide middle ear portion to a narrow part called the isthmus, which has an internal diameter of approximately 1 mm and a length of about 2 mm. A thin plate of bone forms the roof of the Eustachian tube and above this is the tensor tympani muscle. The carotid artery, which lies medially, is separated from the Eustachian tube also by a thin plate of bone.

The medial, cartilaginous portion of the tube is 24 mm long and the bulk of the walls of the tube is formed by a cartilaginous plate. This plate forms the posteromedial wall and the roof, with the anterolateral wall being formed by a mucosal and muscle sheet. The Eustachian tube opens in the nasopharynx behind and below the

posterior end of the inferior turbinate. The tensor palate muscle arises from the scaphoid bone and from along the whole length of the upper rim of cartilage that forms the roof of the cartilaginous Eustachian tube. From these two origins the muscle converges into a short tendon that turns medially around the hook of the hamulus and then spreads out within the soft palate to join the equivalent muscle from the other side of the skull base. The levator palate muscle contains some fibres that originate from the undersurface of the cartilaginous portion of the Eustachian tube. The tensor palate muscle is supplied by a branch of the mandibular nerve, whereas the levator is supplied from the pharyngeal plexus. In general, it is thought that on swallowing the tensor palati muscle contributes to the opening of the cartilaginous portion of the Eustachian tube, whereas the levator palati muscle which has a slower response, may contribute to middle ear ventilation.

The posterior wall of the tympanic cavity :

The posterior wall has its upper end an opening into the mastoid antrum. This opening is called aditus and leads back from

the posterior epitympanum (attic) into the antrum of the mastoid. Below this opening is a small depression in the posterior wall of the middle ear which houses the short process of the incus and its suspensory ligament. This depression, which is called the fossa incudis, lies above a small outgrowth of bone from the posterior wall called the pyramid. This houses the stapedius muscle and tendon, which inserts into the posterior aspect of the head of the stapes. The stapedius muscle itself curves downwards and posteriorly to run or just below the facial nerve canal.

The facial recess is a groove that lies between the pyramid and facial nerve and the annulus of the tympanic membrane. The sinus tympani lies deep to the pyramid and facial nerve and runs into the medial wall of the middle ear.

Middle Ear spaces :

The tympanic cavity is in continuity with the Eustachian tube anteriorly and with the mastoid air cells via the aditus and antrums. It is transversed by the ossicular chain and is lined with a mucosal epithelium. Planes extended from the tympanic annulus subdivide the tympanic cavity into a mesotympanic, hypotympanic, protympanic and posterior tympanic cavity. The epitympanam is above the plane of the anterior and posterior tympanic spines.

Anteriorly the mesotympanam is dominated by the bulge of the semicanal of the tensor tympani muscle, the tympanic orifice of the Eustachian true is immediately inferior to this bulge. Posteriorly the key anatomic features are the pyramidal eminance and lateral to it, chordal eminance.

The medial wall features three depressions. The sinus tympanic oval window niche and round window niche.

The sinus tympanic is defined by ponticulus superiorly, subiculum inferiorly, mastoid segment of the facial nerve laterally and the posterior semicircular canal medially.

The oval window nich occupied by the stapes foot plate, is located antero superior to the ponticulus. The round window niche can be found postero inferior to the promontory the bulge created by the basal turn of the cochlea.

Mucosa of tympano mastoid compartment :

The medial surface of the TM, tympanic cavity, mastoid air cells are all lined with mucosal epithelium reflecting their common heritage from the tubotympanic recess. Ciliated cells intermingle with secretory cells, and promontory in the hypotympannum and in the epitympanum. The mucociliary tracts thus formed act in convert with the mucociliary creation system of Eustachial tube.

Blood Supply :

Arteries supplying the walls and contents of the tympanic cavity arise from both internal and external carotid systems.

Artery	Branch of	Region supplied
Anterior tympanic	Maxillary artery	TM, Malleus, incus, anterior part of the tympanic cavity
Stylomastoid	Posterior auricular	Posterior part of tympanic cavity stapedius muscle
Mastoid	Stylomastoid	Mastoid aircells
Petrosal	Middle meiunged	Roof of mastoid, roof of epitympanum
Supe. Tympanic	Middle merurged	Malleus incus, inner tympanic
Inferior tympanic	Ascending pharyngeal	Mesotympannum
Tympanic branches	Internal carotid	Meso and Hypotympanum

PHYSIOLOGY

All biological gas pockets face two special problems : the need to overcome shrinkage or reduced pressure because of a net loss of gases into the surrounding circulation and the need to keep the inside of gas clean.

In the middle ear these two problems have been overcome by gas inhalation or ventilation through the Eustachian tube and by a mucociliary transport mechanism to clear mucus and debris from the middle ear towards nasopharynx.

Gas transfer from the nasopharynx into the middle ear :

Eustachian tube is 3-4 cm long and can be thought of as two cone like structures fused together by a narrow ring the isthmus.

The medial end that join Nasopharynx is collapsible. The lateral cone joining middle ear is bony and rigid.

The medial end opens 3-4 minutes per 24 hours and gas flows into middle ear.

Normally mucus flows through the floor of the tube. Air flows above it.

Under physiological and steady state conditions about 1-2 ml gas enter the middle ear every 24 hours.

This amount is equal to net amount of gas lost per day by diffusion from the middle ear cleft through the mucosa into the blood.

The negative middle ear pressure difference created between the middle ear and the nasopharynx depends on the size of the middle ear cavity which is turn depend on volume of middle ear cavity.

Air passes from Eustachian tube to middle ear by some active mechanism also. It is by the tensor palati contracts during swallowing or yawning it opens the collapsed medial end and by creating a new volume with a lower pressure than that found in the nasopharynx or in the middle ear.

Gas Diffusion between the middle ear and the circulation :

The middle ear lumen is separated from the blood by the middle ear epithelium the lining of the blood vessels and some connective tissue between them.

Gas diffuses passively from the blood vessels into the middle ear and vice versa according to the difference of partial pressures of the individual gases.

The gaseous steady state may change by

- an increase or decrease of the thickness of the middle ear lining, which correspondingly decreases or increases gas exchange at a given partial pressure difference
- an alteration in the blood flow
- a change in permeability of blood vessels.

SOUND CONDUCTING MECHANISM OF THE MIDDLE EAR

The transformer of the middle ear although working as a complex whole, may be divided into three stages.

1. That provided by the ear drum,
2. That provided by the ossicles
3. That provided by the difference in area between the TM and the stapes footplate

CATENARY LEVER :

The curved membrane of the drum head acted as a catenary lever, which, when stretched, exerts greater force upon its point of attachment. Because the fibrous annulus is immobile, sound energy applied to the tympanic membrane is amplified at its central attachment, the malleus.

OSSICULAR LEVER :

The malleus and incus acting as a unit rotating around an axis running between the anterior malleolar ligament and the incudal ligament. This lever ratio averages 1.3 to in human. The catenary and ossicular levers acting in concert provide an advantage of 2.3 more than twice that of the ossicular acting alone.

HYDRAULIC LEVER :

Sound pressure collected over the large area of the tympanic membrane and transmitted to the smaller footplate area results in an increase in force proportional to the ratio of the area.

A summary of the concepts of the middle ear transformer :

1.	Catenary lever	Force acting on TM / Force acting on malleus	2.0
2.	Ossicular lever	Force acting on malleus / Force acting on stapes	1.15
3.	Areal ratio	Area of TM / Area of footplate	21.0
4.	Total lever advantage	Force acting on foot plate / Force acting on TM	48.3 34.0db

Bone Conduction :

The internal component of bone conduction is due to the lag of the conduction apparatus in following the vibration of the skull, thus creating a relative movement of the stapes on oval window. This movement is important between 500 and 2000 Hz. Fixation or interruption of the ossicular chain reduces this energy transfer and causes falsely depressed scores on bone conduction testing.

The middle ear acts as a low – pass filter, allowing frequencies below the network resistance of 1000 Hz to pass while attenuating higher freqeuencies at a 16dB octave slope.

PATHOGENESIS OF RETRACTION

Patients with chronic secretory otitis media, chronic suppurative otitis media, atelectactic tympanic membrane, retraction pockets, and retraction pocket cholesteatomas may all be viewed as belonging to one family : the chronic Ear syndrome :

All such patients have had a lower than atmospheric pressure in their middle ear at same time.

This negative pressure is considered important in the pathogenesis of chronic ear syndrome.

The classic example of chronic ear syndrome is the Atelectatic drum which is typically retracted towards promontory.

- An atelectatic ear usually persists in the same position for weeks, months or years as it has acquired a stable state
- This new steady state is most often some where between the physiological state and total retraction. Only in a minority of ears is all gas lost from middle ear.
- Atelectatic ears may recover and return to their normal position spontaneously.

- Atelectatic ears may change in the other direction and reach a new steady state, reach a new steady state, with a gradual step wise progression towards a retraction pocket or retraction pocket cholesteatoma.
- A tympanic membrane that has at one time been indrawn and atelectatic may subsequently appear ballooned out, ie. anti atelectatic.

There are several theories in an attempt to explain gas deficiency in the middle ear

1. Ventilation deficiency – Eustachian tube dysfunction
2. Sniffing and increased Air flow through nasopharynx
3. Diffusion defects

VENTILATION DEFICIENCY

The most popular explanation for a lower middle ear pressure is that there is a relatively low gas supply through Eustachian tube.

In born or acquired obstruction or narrowing of the lumen or obstruction of the opening of Eustachian tube by Adenoids has often been suggested.

After the Eustachian tube obstruction theory was given up by some, but not all, a hypothesis suggesting a functional disorder was proposed.

The Eustachian tube muscles were considered to be inadequate thus causing a condition in which the presence of a negative middle ear pressure along with the positive atmospheric pressure blocked the opening of the tube and the ventilation of the middle ear.

SNIFF theory :

Maguson thought that the atelectatic condition was a result of middle ear gases being sucked air along the Eustachian tube by the patient themselves who were believed to sniff compulsively.

Sniffing may indeed lower the nasopharyngeal pressure and in the pressure of an excessively open (patulous) Eustachian tube this may induce sucking out of gas from the middle ear

EXCESS DIFFUSION

A third possibility that might explain the origin of the middle ear gas deficiency is an increased loss of gas through excessive diffusion into the surrounding tissues and blood despite steady state ventilation through Eustachian tube.

Nitrogen always has a higher pressure in the middle ear and therefore, steadily diffuses into the tissues and circulation only to be replenished by ventilation through Eustachian tube.

Under inflammatory conditions when the vascularity of mucosa increases many times perfusing increases and nitrogen clearance into the blood may exceed the normal rate.

This may result in an increased loss of nitrogen without a consistant increase in supply through Eustachians tube. Subsequently a negative middle ear pressure can result.

The mastoid acts as a physiological buffer for the middle ear. Most ears afflicted with the choronic ear syndrome, have a hypopneumatized acellular or scerotic mastoid.

EARS with well pneumatized mastoids rarely exhibit any of the chronic ear syndrome and probably rarely develop a negative middle ear pressure.

Reaction of the Middle Ear to Negative pressures :

When a negative pressure develops in the middle ear, the atmospheric pressure is the outside compresses the tympanic membranes at its weakest points, which there fore retract.

The pars flaccida retracts first because of its elasticity. When pars flaccida retracts to its maximum, a further decrease in middle ear pressure caused the relatively non fusible pass tensa to retract.

This compensating mechanism may not neutralize the entire negative pressure, but usually keep it a few mm H_2O below atmospheric as has been measured by Buckingham and ferrere and Sad et al.

If further gas depletion continues there are several consequences damage to the collagen skeleton of pars tensa will convert it an elastic membrane with properties similar to pars flaccida. This will allow for further retraction and deep atelectatic pockets.

ATELECTASIS OF MIDDLE EAR

Atelectasis of the middle ear is a sequela of otitis media, Eustachian tube dysfunction, or both. Retraction or collapse of the tympanic membrane is characteristic of the condition.

Collapse implies passivity (high negative middle ear pressure is absent) whereas retraction implies active pulling inward of the tympanic membrane, usually from negative middle ear pressure ; which is turn is due to Eustachian tube dysfunction. Middle ear effusion is usually absent when atelectasis is present. It may be acute or chronic, localized (with or without a retraction pocket) or generalized and mild moderate or severe.

Five stages for tympanic membrane retraction were described by Sade' in 1993. Stage I is a slight retraction of the TM, whereas stage II involves the drum touching the incus or stapes. In stage III the TM is inclined on the promontory; stage IV changes involve the drum adhering to the promontory while in stage V, the TM is adherent and may be perforated. Dornhoffer (2000) proposed a slight clinical modification as he diagnosed stage IV when the depth of the retraction cannot be adequately visualized by micro-otoscopy and

keratin debris accumulation or cholesteatoma formation should be suspected.

Atelectasis can be classified, graded and staged in relation to extend and duration as follows. A localized area of ateletasis of the tympanic membrane may or may not be a retraction pocket. Since the depth of the retraction may be mild moderate or severe. When localized (with or without a retraction pocket) it may be in 1 of the 4 quadrants of the pars tensa (ie. antero superior) antero inferior in the pars flaccida or in 2 or more of these anatomical sites. It is due to recurrent or chronic moderate to severe under pressures in the middle ear which are in turn due to Eustachian tube dysfunction.

In localized atelectasis with a retraction pocket 1 or more quadrant of the pars tensa is atelectasis but not in all quadrants.

The atelectasis can be mild, moderate or severe and acute or chronic when it is severe a retraction pocket is usually present.

A retraction pocket is characterized by a localized area of atelectasis of the TM in which there is indrawing of the membrane forming borders (ie. Edge or margin) most frequently at the site of

an osseous anatomic structure (eg notch of Rivinus or scutum) or the malleus.

A classification of a postero superior retraction pocket has been proposed by Sade in which the pocket is slightly retracted and self cleaning (stages) deeper still and needling cleansing (stage 2) deeper still and partly hidden and requiring excision (stage 3) or so deep the pocket can only be removed by exposing the scutum and the rest of the frame work (ie. Retraction pocket cholesteatoma stage 4). This staging system is helpful but not include duration or the presence or absence of adhesive changes which relate to reversibility or include other sites.

If persistant and progressive a retraction pocket can lead to sequalae commonly attributed to otitis media such as hearing loss, ossicular chain discontinuity and cholesteatoma.

Staging divides retraction pockets into retraction pockets that are acute (< 3 months duration) and those that are chronic (3 months or longer duration)

Key factors that affect the progression of the stages of a retraction pocket from stage 1 to stage 4 are as follows.

First in the relation to middle ear structures whether it approximates or is adherent to (ie. Adhesive otitis media), 1 or more osicles (ie. Incus, incudo stapedial joint, stapes head of malleus, incudomalleolar joint) or other middle ear structures such as promontory of the cochlea. Also considered is whether it expands with pressure ie. whether the entire pocket easily expands to normal positions. When the negative pressure applied with a pneumatic otoscope or with positive pressure whereas the patient is anaesthetized with N2O.

Third, one considers the extent to which the pocket is visualized ie. Whether the entire pocket is visualized or whether parts are not seen even after applied pressure, because the pocket extends beyond the visible portion of the middle ear spare or medial to other part of Tympanic membrane.

Finally one considers whether the pocket is self cleansing and free of infection ie. Whether epithelial debris crusting or purelent material present with in the pocket.

A proposed staging system that takes into account the key factors is as follows

Stage 1 A - an acute mild retraction pocket in which the membrane of the pocket neither approximates nor is adherent to any middle ear structures, the entire contents of the pocket are readily visible and the pocket is self cleansing.

Stage 1C - a chronic mild retractiton pocket is same as stage 1A, but is chronic.

Stage 2A - are acute moderate retraction pocket has a membrane that is applied to 1 or more middle ear structures but is not adherent expands with pressure has an extent that can be visualized and is self cleansing without infection.

Stage 2 C - chronic moderate, retraction pocket as stage 2A, but chronic

Stage 3A - acute severe retraction pocket TM is adherent to 1 or more middle ear structures the extent is visualized and is without infection and the pocket cannot be expanded with pressure.

Stage 3 C - a chronic severe retraction pocket as 3A.

Stage 4 A - an acute retraction pocket cholesteatoma has a tympanic membrane adherent to 1 or more middle ear structures and

the extent cannot be visualized or the pocket is not self cleansing or free of infection.

Stage 4C - a chronic retraction pocket cholesteatoma as stage 4A but chronic

Acute and chronic atelectasis that involves all 4 quadrants of pars tensa with or without involvement of pars flaccida are classified as follows.

Stage 1A - acute generalized mild atelectasis with the middle ear is well aerated.

Stage 1C - chronic generalized mild atelectasis is same as stage 1A but chronic.

Stage 2A - acute generalized severe atelectasis has a middle ear that is not aerated ie. There is no apparent middle ear space.

Stage 2C - chronic generalized severe ateleactasis same as 2A but chronic.

ADHESIVE OTITIS MEDIA

Adhesive otitis media is a result of healing following chronic inflammation of the middle ear and mastoid. The mucous membrane is thickened by proliferation of fibrous tissue, which frequently impairs movement of the ossicles, resulting in conductive hearing loss. The pathological process is a proliferation of fibrous tissue within the middle ear and mastoid termed fibrous sclerosis.when there are cystic spaces present, it is called fibrocystic sclerosis, and when thee is new bone growth in the mastoid, it is termed fibro-osseous sclerosis.

The staging of adhesive otitis media is as follows : In stage 1, the adhesive otitis media is within the middle ear or mastoid, or both and there is no functional deficit secondary to the adhesive changes, ie. Hearing loss ; the middle ear remains aerated. In stage 2, the adhesive otitis media is within the middle ear (with or without mastoid involvement)and there is mild hearing loss secondary to the adhesive pathology such as involvement of the ossicular chain (fixation or discontinuity, or both ; see ossicular discontinuity and Ossicular fixation), limitation of tympanic membrane compliance or

both but the middle ear remains aerated. Stage 3 is similar to stage 2, but there is maximum conductive hearing loss (secondary to ossicular pathology) and the absence of a middle ear space, both of which are due to the extensive adhesive otitis media.

Conductive Deafness in Tympanic membrane Atelectasis :

Atelectasis of the tympanic membrane occurring without a TM perforation (and in pressure of intact and mobile ossicles) can result in conductive hearing losses that vary in severity from negligible to 50 dB.

The conductive deafness can be explained an the basis of a reduction is ossicular coupling caused by as abnormality..

As long as the area outside round window remains aerated and is shielded from the sound pressure in the ear canal by the TM the conductive loss caused by the atelectasis should not exceed the amount of middle ear pressure gain is normal ears ie. air bone gap upto 25 dB If atelectasis result in invagination into the round window niche the protective effect of TM and middle ear space and round window niche is lost and larger air bone gap (40 – 50 dB) should result.

MATERIALS AND METHODS

In our Department we conducted a study on cartilage perichondrium tympanoplasty on 16 patients during a period from 2004-2006.

These patients presented to our hospital with different Ear symptoms e.g. hearing loss, autophony and discharges. Only cases in which tympanic membrane was adherent to the promontory ie. Stages III and IV were included in this study. Differentiation between stages was based on Dorn Hoffer's proposal (2000). The patients ranged in age from 13 to 35 years. All patients were subjected to full ENT history and examination.

Otologic assessment and documentation through photography were done through otoendoscopy.

Pure tone audiometry were done to calculate the average air bone gap both pre operatively and post operatively. Impedence Audiometry was done to confirm the diagnosis.

Methods :

Different operation techniques have been published depending on the form of the cartilage graft.

- Palisade technique
- Cartilage plate technique
- Cartilage island technique
- Cartilage tension ring (horseshoe)

Among these we used cartilage plate technique.

Technique

The procedure is done under either local or general anaesthesia. A post aural approach is generally adopted.

2% Lignocaine with epinephrine 1 : 1,00,000 is used for local infilteration.

Korners flap is elevated after putting incision at 6 'O clock and 12'O clock position. Then post auricular William wilde incision is made.

The conchal cartilage is harvested with intact perichondrium on both sides. The cartilage with attached perichondrium is dissected

from skin and soft tissue with a pair of sharp scissors in a plane superficial to perichondrium on both sides.

The cartilage is sliced to 15 mm length and 10 mm is width.

Two different techniques are used depending on whether the pathology is confined to the posterior segment or involves the whole middle ear.

In the first case, the anterior perichondrium is dissected off and prepared to graft a deficient TM. Using an angled knife, a perichondrium/ cartilage island flap is individually constructed to allow better fit in the middle ear according to the pathological findings.

In the second case, the whole cartilage is harvested, the anterior perichondrium is elevated in continuity with the posterior perichondrium on which a disc of cartilage is fashioned 8-9 mm in diameter and a strip of cartilage 2 mm wide is removed to accommodate the malleus handle.

The posterior segment should reach the malleus handle to guard against future retraction in the gap. The excised strip may

involve the whole islet length so that the anterior segment can be pushed laterally.

A tympanomeatal flap is elevated and the atrophic drum is carefully elevated off the bony sulcus, the promontory and middle ear structures with extreme caution to preserve the thin TM making certain to enter the middle ear beneath the annulus. Redundant tympanic membrane is removed, and the ossicular chain is inspected. If good movement exists between the incus and stapes, even in the presence of some erosion of the incudostapedial joint, the chain is left intact. In cases with immobilized malleus on the promontory, the tensor tympani tendon is cut to allow lateral displacement of malleus and to widen the mesotympanic space. Besides the depth, attic and posterior canal wall saucerization and removal of part of the posterior bony meatus enlarge the diameter of the middle ear. This is also mandatory to expose the fundus of a retraction pocket with its keratin debris and possible cholesteatoma. At this point a decision may have to be made as to expanding the dissection to include an atticotomy, atticoantrostomy, or a tympano-mastoidectomy.

The entire graft is placed in an underlay fashion, with the cartilage toward the promontory and the perichondrium adjacent to the tympanic membrane remnant, both of which are medial to the malleus. A drill can be used to sculpture the canal walls as necessary to accomplish a best-fit condition. An alternative method includes using two separate pieces of cartilage-perichondrial graft, one to obliterate the posterosuperior pars tensa quadrant and a separate piece for the attic blockage in cases with attic erosion, either suspected through radiology preoperatively or encountered intraoperatively. In cases, which need ossicular reconstruction, either a primary incus (auto or homograft) interposition is done or a secondary reconstruction using a suitable prosthesis is planned.

The anterior part of the middle ear is then packed with moistened Gel foam and the flap is repositioned. The external canal is filled with roller gauze socked antibiotic ointment.

RESULTS

During the 2-year period for which data were collected, 16 cartilageperichondrium tympanoplasties were performed for 16 patients with symptomatic stages III and IV atelectatic ears.

Type III group included 9 patients.

Seven patients were included among type IV group. The main presenting symptom among type III patients was aural discomfort Deafness was the presenting symptom in 3 patients whereas one patient had recurrent otorrhea. Concomitant deafness and offensive otorrhea were the presenting symptom among type IV patients

Of the surgeries performed, 12 were cartilage tympanoplasties only and 4 were combined with ossicular reconstruction.

The latter involved using autograft incus interposition in 3 cases, and femoral cortical bone homograft ossiculoplasty in one case.

Out of 16 patients operated grade III were 9 patients of these 3 were bilateral diseases. Preoperatively AB gap 30 ± 4 dB

All these 9 patients cartilage tympanoplasty was done after inspecting the ossicular chain mobility.

Post operatively 8 patients had hearing improvement and their AB gap closure was 14 $\pm$ 8 dB. One patient had no hearing improvement and had persistent AB gap of 30 dB.

Grade IV were 7 patients 3 patients AB was 40 dB $\pm$ 4 dB, 4 patients had 60 dB $\pm$ 4 dB.

Cartilage tympanoplasty was done for 3 patients after inspecting the ossicular chain mobility.

Among these patients with hearing loss of 60dB $\pm$ 4 dB, 3 patients had incus, necrosis 1 patients had ossicular fixation.

Post operatively 2 patients with cartilage tympanoplasty and 2 patient with cartilage tympanoplasty and ossiculoplasty had an AB gap closure. Remaining 2 patients had a persistent AB gap.

Table - 1

No.of patients	Grade of Retraction	Type of procedure	Average pre operative AB gap	Average post operative AB gap	Persistance of AB gap	% of success
9	III	Cartilage tympanoplasty	30 ± 6 dB	14 ± 8 dB (8patients)	1 patient (34 dB)	88%
3	IV	Cartilage tympanoplasty	40 ± 4 dB	14 ± 8dB) (2 patients)	1 patient (40 dB)	66%
4	IV	Cartilage tympano plasty and ossiculoplasty	60 ± 4 dB	20 ± 4 dB (2 patients)	2 patients (60 dB)	50%

Table -2

Diagnosis & type of procedure	No.of patients	Closure of air bone gap	Persistance of air bone gap
Atelectatic ear Cartilage tympanoplasty	9	8	1
Adhesive otitis media cartilage tympanoplasty	3	2	1
Adhesive otitis media cartilage tympanoplasty + ossiculo plasty	4	2	2

Table - 3

Grade of Retraction	**No.of patients**	**Percentage**
Grade III	**9**	**56 %**
Grade IV	**7**	**44 %**

DISCUSSION

Treatment of the atelectatic ear may be conservative or surgical. Conservative treatment includes a variety of maneuvers to increase pressure in the middle ear such as use of the auto-Valsalva maneuver or politzerization. Luntz and Sade' (1988) concluded that for this maneuver to be effective for the patient with an atelectatic ear, politzerization should be performed every hour, making it an impractical treatment. Moreover, Helms (1988) reported two cases of penetration of air into the middle cranial fossa through a defect in the tegmen tympani that caused major neurologic deficit.

Another commonly used treatment of an atelectatic ear involves the insertion of a ventilation tube. Many conflicting reports were found in the literature about the effectiveness of such procedure in treating atelectatic ears.

Tos et al., (1987) did not find a difference in the incidence of atelectasis in tympanic membranes of the same patient when the patient had myringotomy alone on one side and myringotomy with tube insertion on the other side. Pfaltz, (1988) had contended that a ventilation tube may prevent the progression of retraction pockets

into cholesteatoma. Buckley and Hinton (1991) reported an increased incidence of atelectasis in ears with previous insertion of ventilation tubes. Dialilian and Paparella (2000) concluded that tubes delay the progression of atelectasis but when extruded (which happens earlier in atelectatic ears), the disease will continue to progress.

Much of the controversy involves the validity of tympanoplasty as an effective measure to control middle ear atelectasis. Much of the confusion stems from a poor understanding of the underlying pathophysiologic conditions of the disease and lack of an accepted grading scheme for it. The literature contributes to the confusion by providing a wide variation in the reported incidence of this disease and the rate at which complications are seen to develop. The staging system adopted in this study follows that of Dornhoffer (2000) which presents a clinical modification of the original scheme of Sade' (1993). This modification allows proper preoperative staging with subsequent more precise patient selection and avoidance of unnecessary procedure as there is a clear differentiation between stages especially, III and IV.

The timing of intervention is another dilemma. Should a tympanoplasty be performed early in the disease as a prophylactic measure or later in the disease after the development of significant hearing loss or frank cholesteatoma? Early intervention is technically easier and complications are less. The argument against such concept lies in the possibility of performing an unnecessary surgery in an ear that potentially would have remained stable with time. On the other hand, late intervention puts the patient at an increased risk for much more extensive and often multiple surgical procedures with lesser hearing gain. Although most would not argue with the logic of surgical intervention in a type IV retraction because of the inability to rule out incipient cholesteatoma, surgical intervention with the type III retraction is more controversial. In this study, only symptomatic cases of type III and all cases of type IV were included. The main presenting symptom in type III patients was ear discomfort rather than deafness. This can be explained by the operative finding of myringo-incudo-stapediopexy or even myringo-stapediopexy which offer a reasonable acoustic gain. This was not the case in type

IV patients where otorrhea and deafness were the rule. The concept of the cartilageperichondrium graft is to create a stiff and structurally secure tympanic membrane that will resist the force of retraction (Levinson, 1993). Cartilage appears to be an ideal graft material in the atelectatic middle ear because it offers rigorous reconstruction with little or no detrimental effect on hearing when compared with more traditional material, such as fascia or perichondrium. It has been shown in both experimental and clinical studies that cartilage is well tolerated by the middle ear and long-term survival is the rule (Dornhoffer, 1997). Although it is similar to fascia in that it is mesenchymal tissue, its more rigid quality tends to resist resorption and retraction, even in the milieu of continued eustachian tube dysfunction (Dornhoffer, 2000). The only serious drawback is the possibility for development of cholesteatoma due to buried keratinized epithelium if the atrophic membrane cannot be elevated intact. Such complication cannot be detected early enough due to the thick opaque graft. It is recommended to follow up these cases by High Resolution Computed Tomography (HRCT) scan one year postoperatively. In this study, we applied the concept of

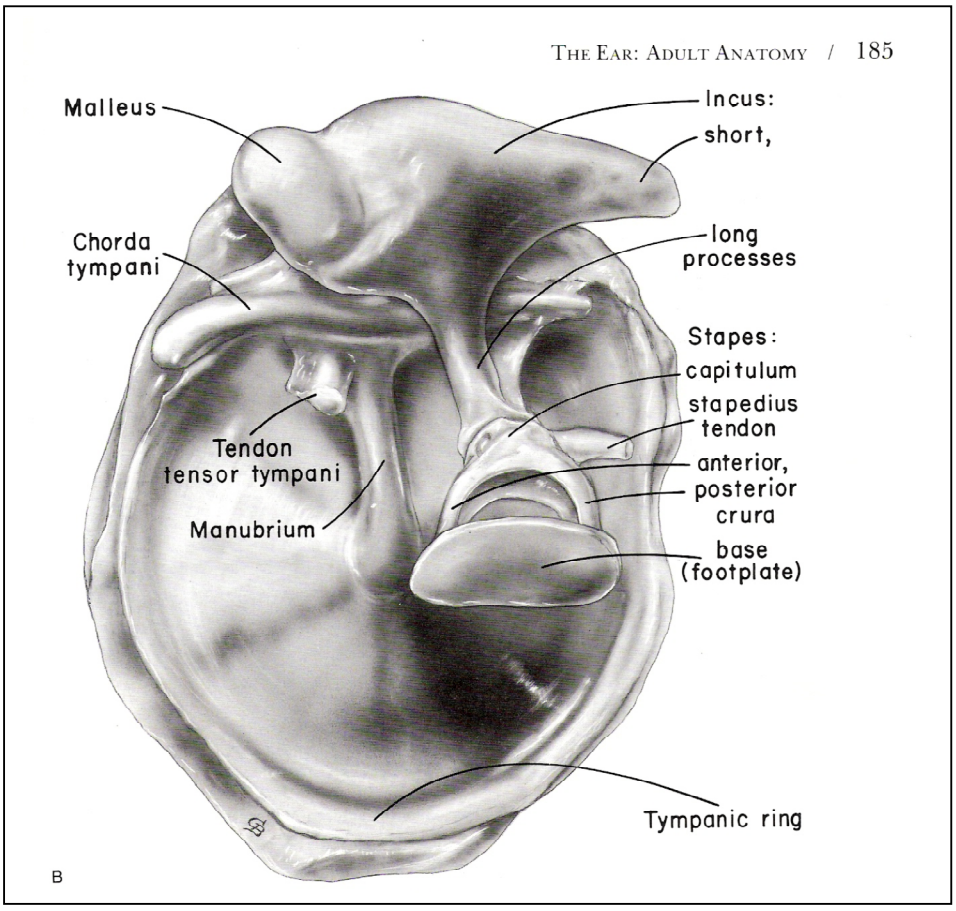
increasing the middle ear dimensions suggested by Djalilian and Paparella (2000) as a crucial step in treating middle ear atelectasis. By performing the posterior bony meatectomy, atticotomy, and cutting of the tensor tympani tendon, we increase the superior, posterior, and lateral dimensions.

The surgical technique used here appears to offer a viable alternative in the management of type III and IV atelectatic ears. The ultimate hearing result was quite encouraging, and hearing was improved in all patients. Otorrhea and ear discomfort were relieved. These results compare favorably with other reporters (Milewski, 1993, Harner, 1995, Dornhoffer, 2000).

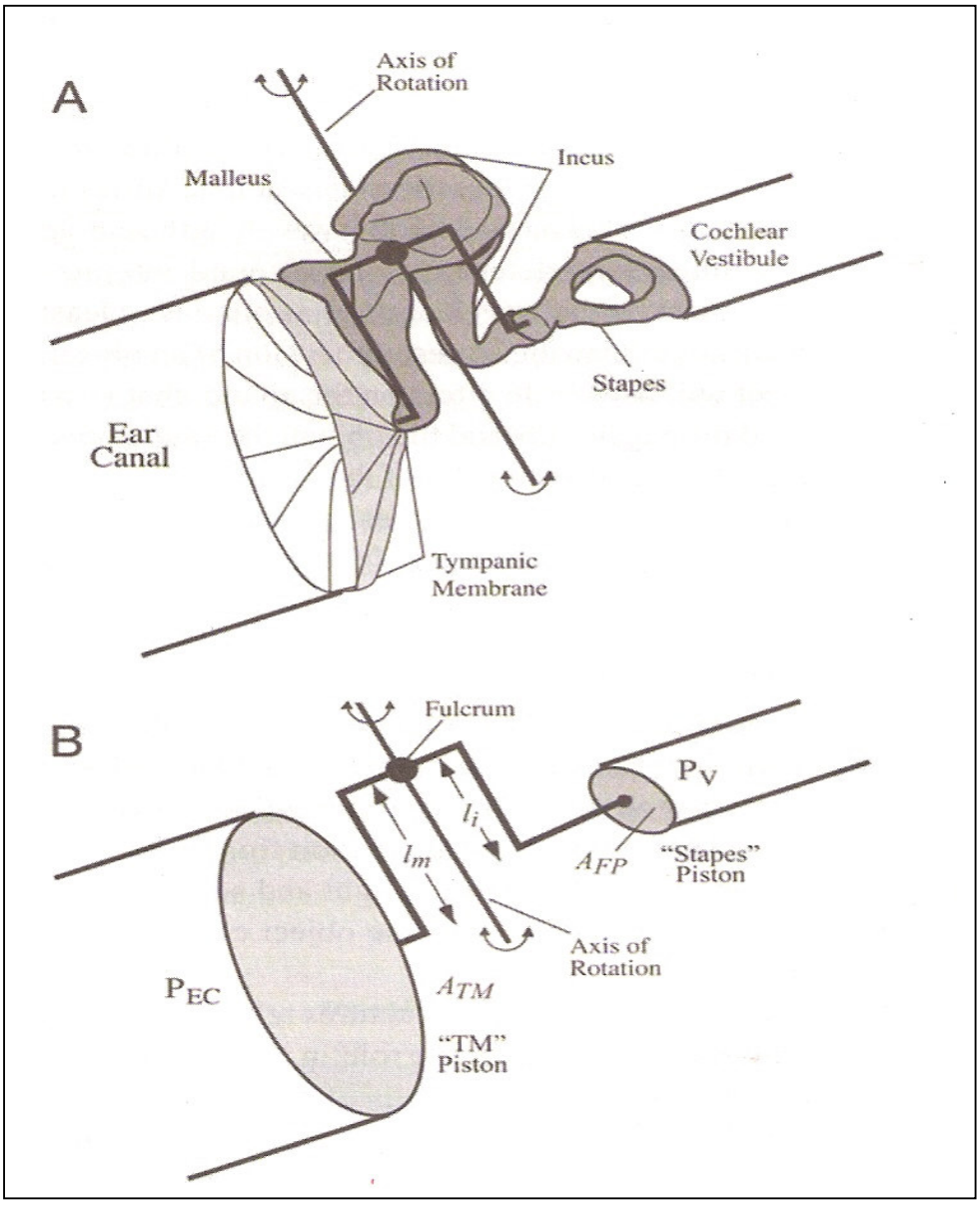
CONCLUSION

Management of types III and IV retraction though cartilageperichondrium tympanoplasty with or without ossicular reconstruction is a proven treatment modality, with successful results to hearing and complications. This technique provides for expansion of middle ear space, providing rigid support of the tympanic membrane and eliminating the possible risk of cholesteatoma.

TYMPANIC RING AND MEMBRANE –MEDIAL ASPECT

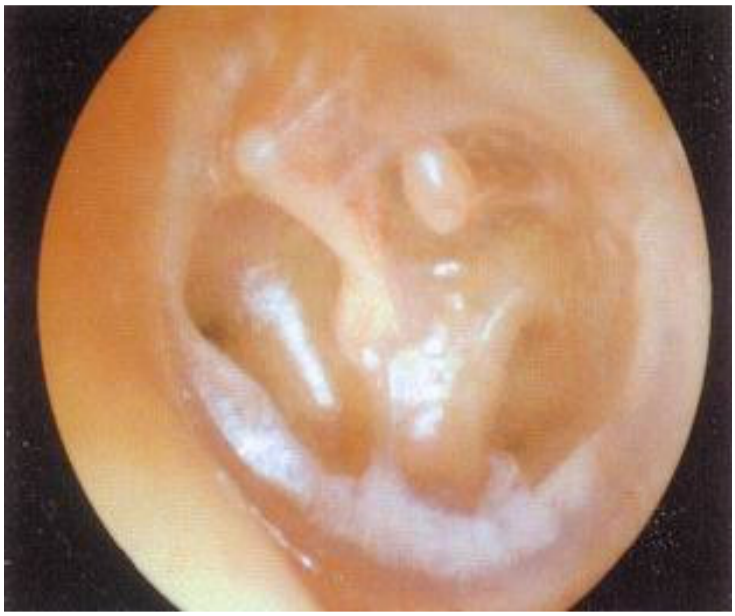


TYMPANO – OSSICULAR SYSTEM

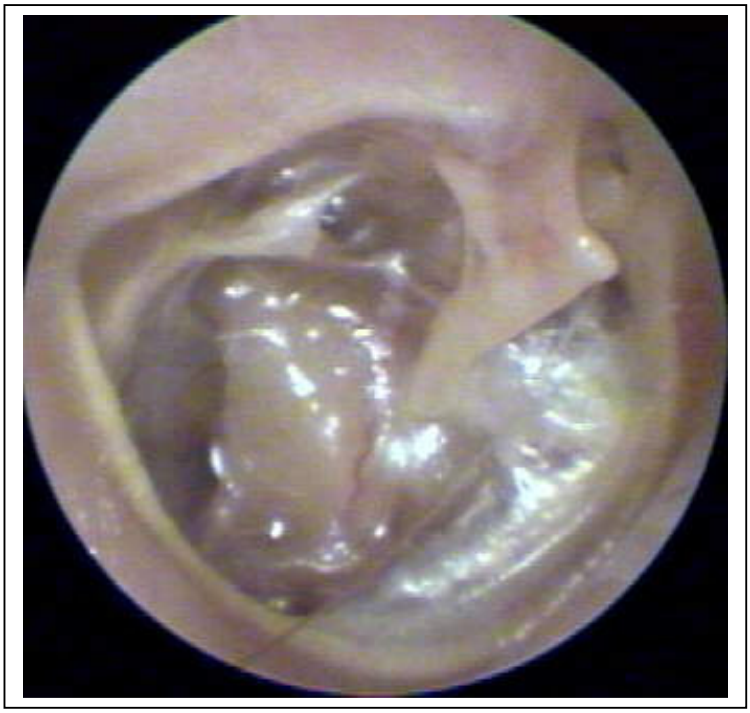


PRE OPERATIVE VIEW

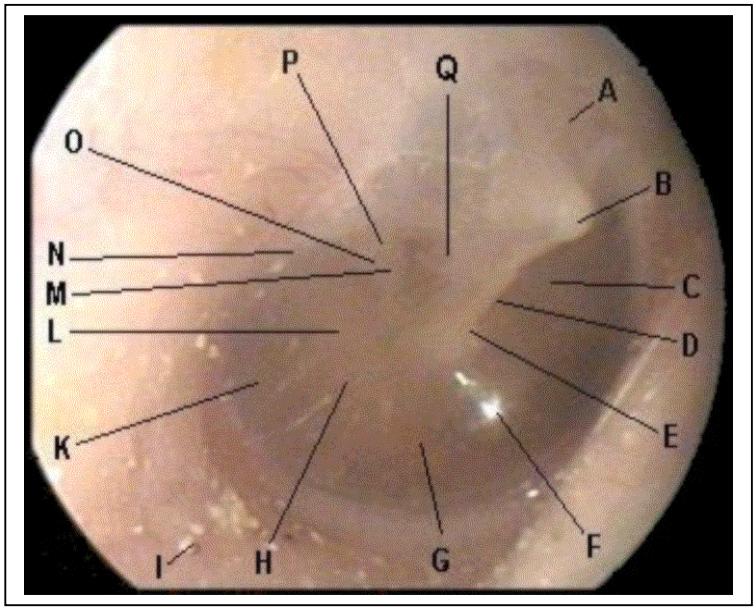
ATELECTATIC EAR DRUM



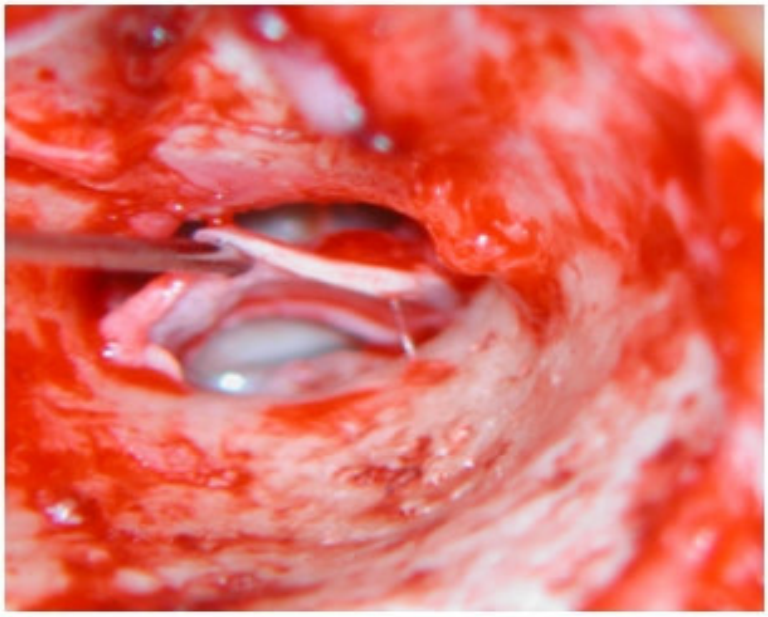
ATELECTASIS OF MIDDLE EAR



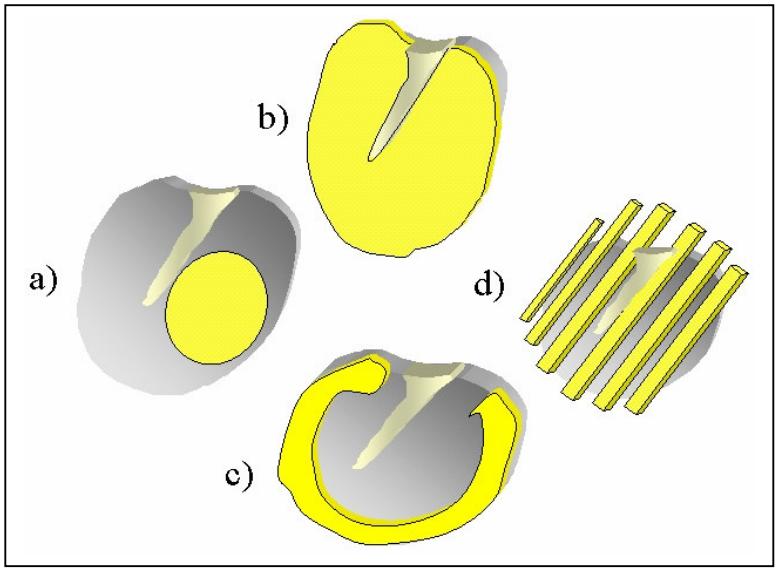
NORMAL TYMPANIC MEMBRANE



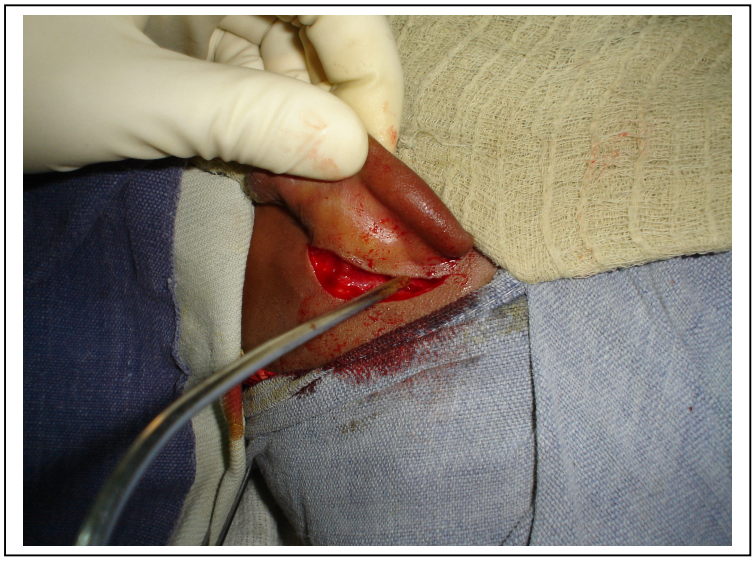
SURGICAL PROCEDURE



DIFFERENT FORMS OF CARTILAGE GRAFT

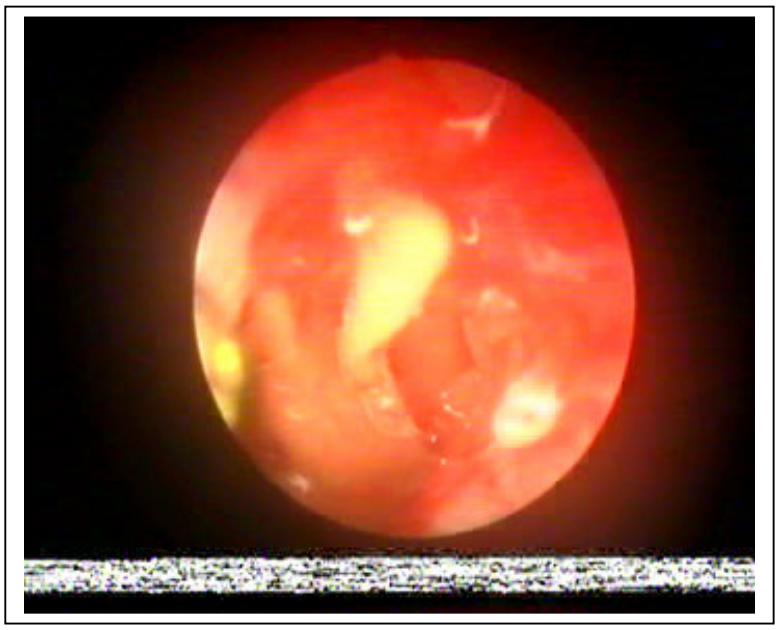


POST AURICULAR WILLIAM WILDE INCISION

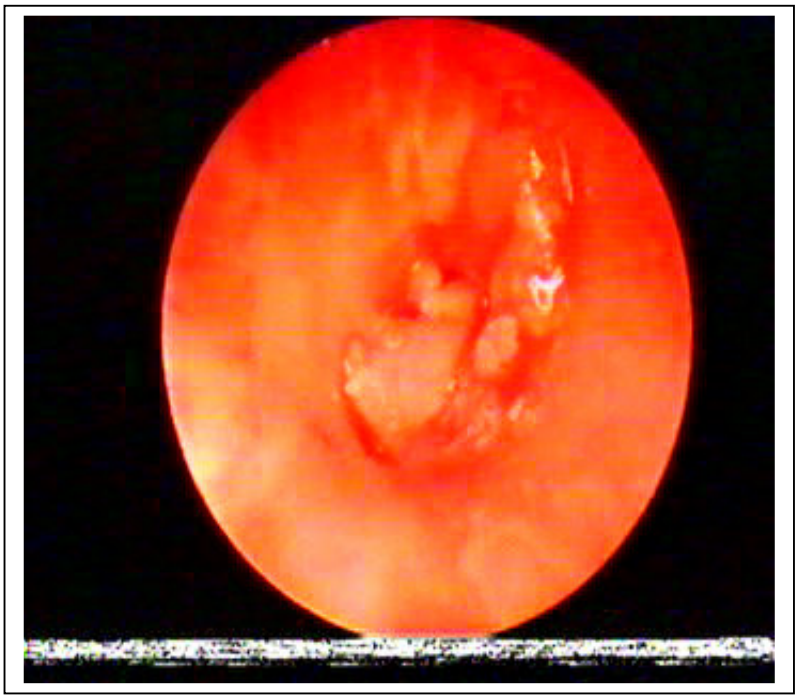


HARVESTING CONCHAL CARTILAGE

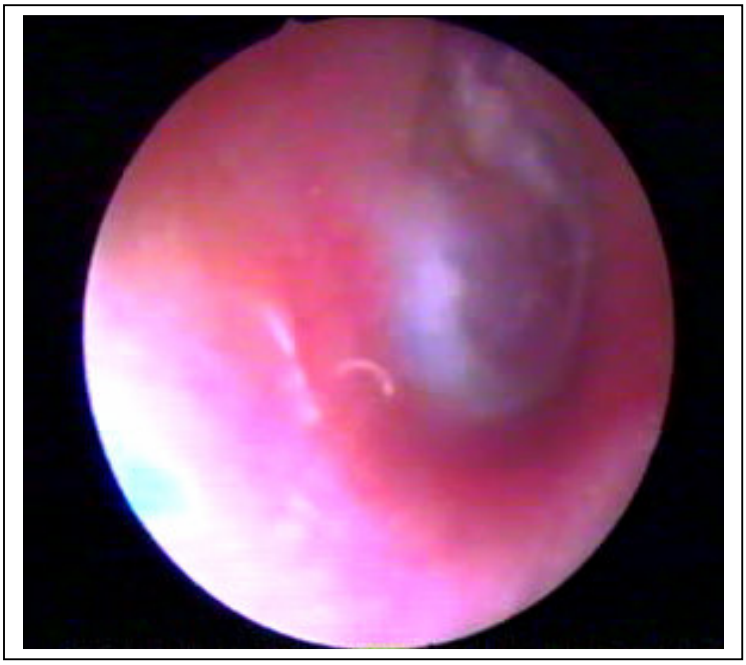
IMMEDIATE POST OPERATIVE VIEW



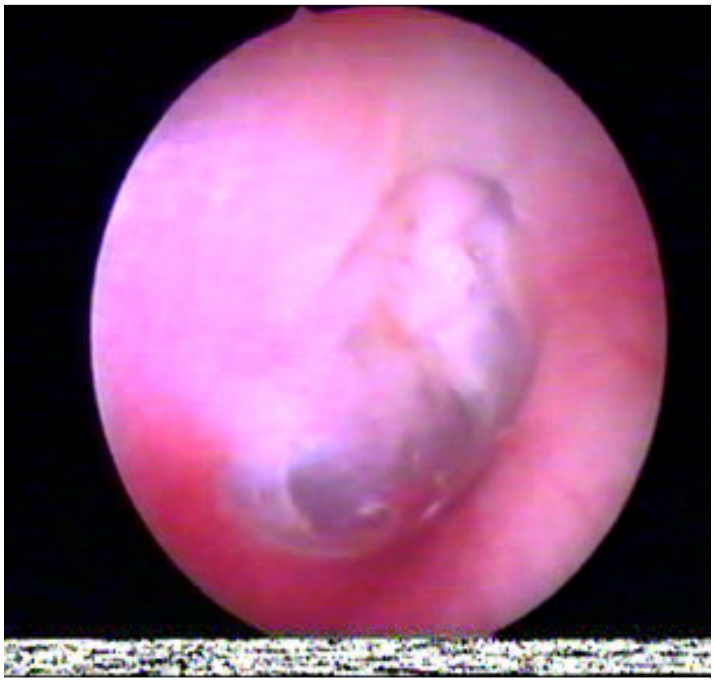
POST OPERATIVE VIEW AFTER 4 WEEKS



POST OPERATIVE VIEW AFTER 8 WEEKS



POST OPERATIVE VIEW AFTER 6 MONTHS

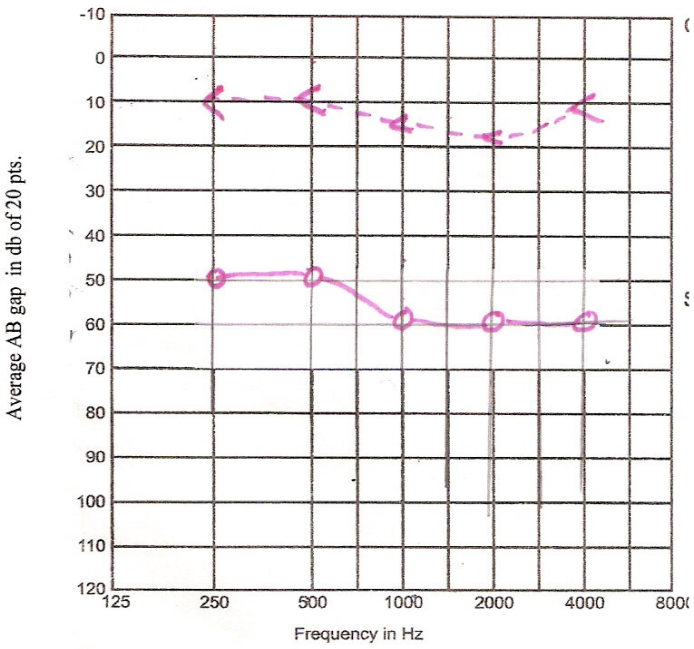


Department of ENT
Government Rajaji Hospital,
Madurai

PURE TONE AUDIOGRAM

Patient Name : Date :

Address : Age / Sex :



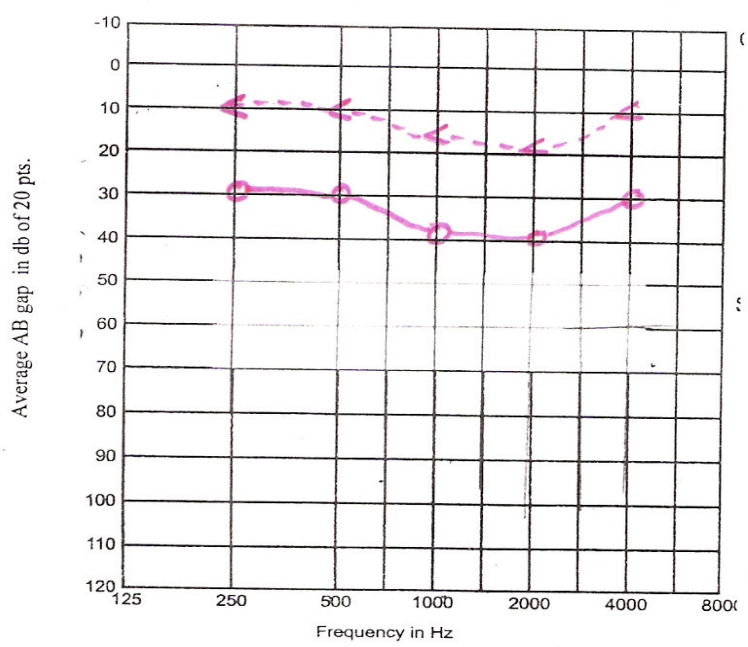
Preoperative Audiogram

Department of ENT
Government Rajaji Hospital,
Madurai

PURE TONE AUDIOGRAM

Patient Name : Date :

Address : Age / Sex :



Postoperative Audiogram

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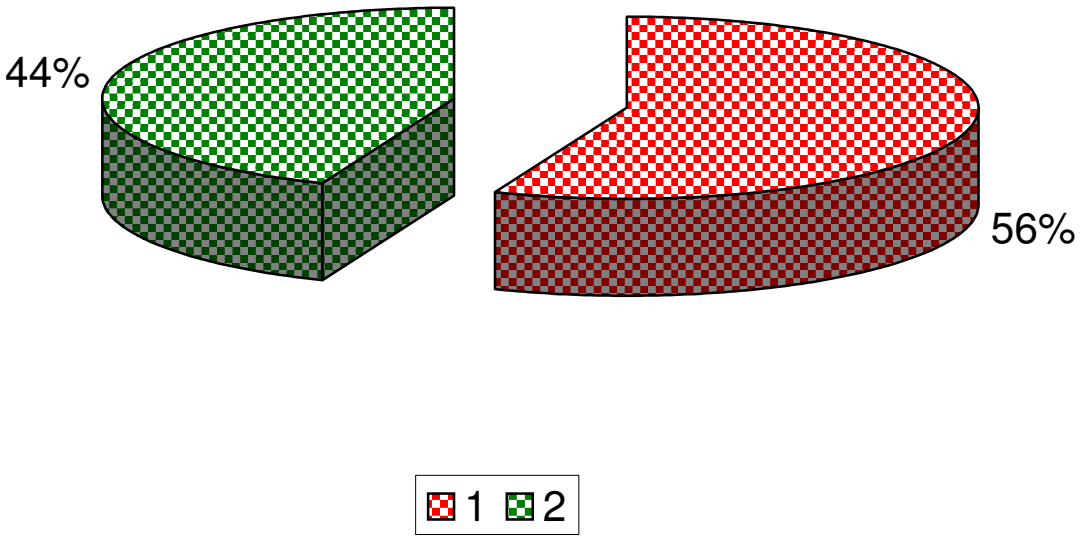
MASTER CHART

S.No	Name	Age	Sex	I.P.No.	Diagnosis	Procedure
1.	Jeyakumar	34	M	434966	Atelectasis Rt Ear	CT
2	Muthusamy	30	M	440735	Atelectasis Lt Ear	CT
3	Priyanka	16	F	443194	Atelectasis Rt Ear	CT
4	Surya devi	14	F	440304	Atelectasis Rt Ear	CT
5	Muthukumar	27	M	441779	Atelectasis Lt Ear	CT
6	Rajeshwari	17	F	449699	Atelectasis Rt Ear	CT
7	Valli	28	F	356411	Atelectasis Rt Ear	CT
8	Prasanth	13	M	349882	Atelectasis Rt Ear	CT
9	Ravi	17	F	462844	Atelectasis Rt Ear	CT
10	Pandiammal	20	F	462844	Adhesive otitis media Rt ear	CT
11	Mohamed anisha	15	F	464008	Adhesive otitis media Lt ear	CT
12	Chandrakumar	18	M	461661	Adhesive otitis media Rt ear	CT
13	Karthikraj	16	M	465455	Adhesive otitis media Lt ear	CT + OP
14	Jeyaram	30	M	361411	Adhesive otitis media Rt ear	CT + OP
15	Parameshwari	23	F	352264	Adhesive otitis media Lt ear	CT + OP
16	Palanichamy	30	F	381211	Adhesive otitis media Rt ear	CT + OP

CT - CARTILAGE TYMPANOPLASTY OP - OSSICULO PLASTY

GRADE OF RETRACTION



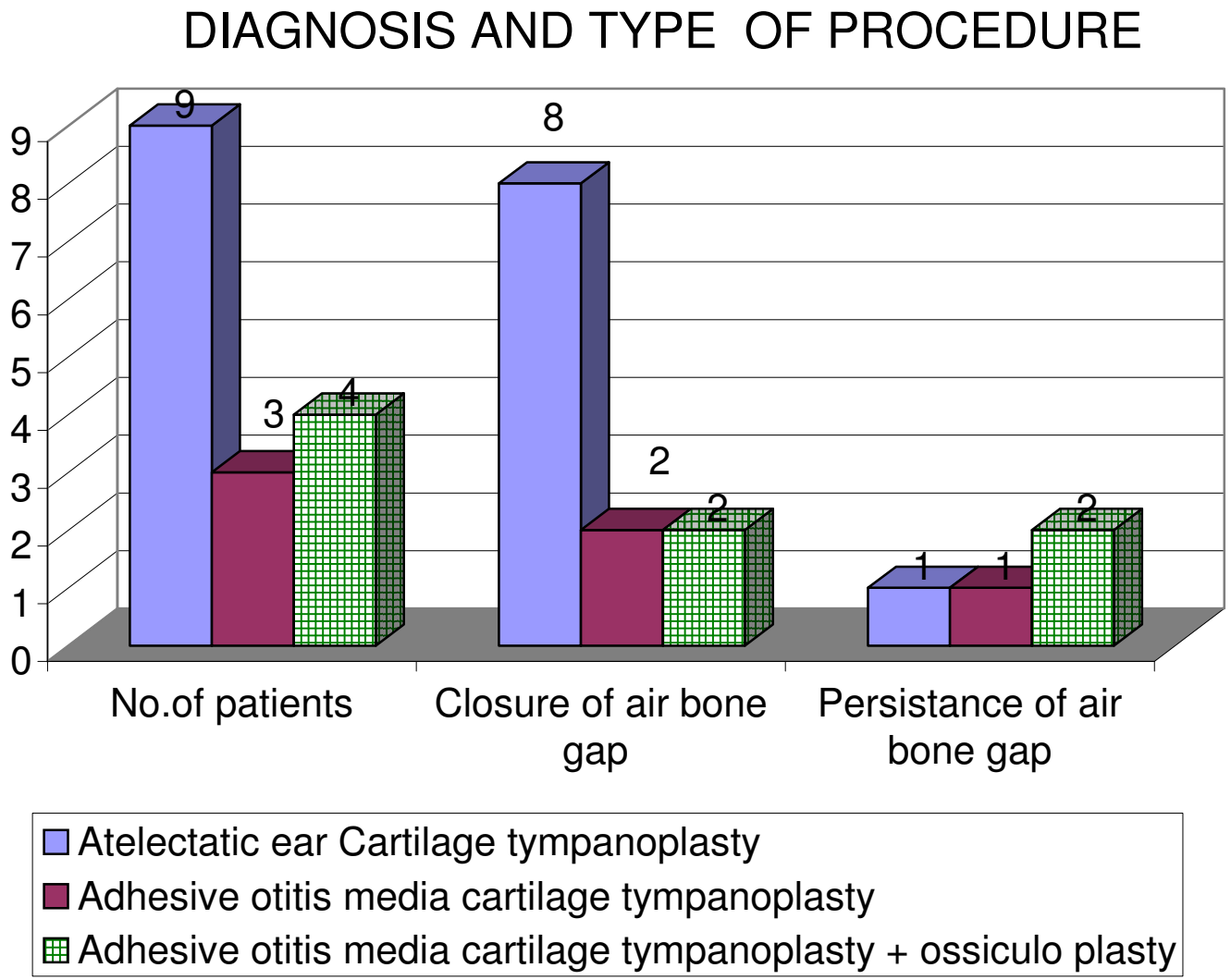

DIAGNOSIS AND TYPE OF PROCEDURE
9
8
3
4
2
2
1
1
2
0
1
2
3
4
5
6
7
8
9
No.of patients
Closure of air bone gap
Persistance of air bone gap
Atelectatic ear Cartilage tympanoplasty
Adhesive otitis media cartilage tympanoplasty
Adhesive otitis media cartilage tympanoplasty + ossiculo plasty

Table -2

Diagnosis & type of procedure	No.of patients	Closure of air bone gap	Persistance of air bone gap
Atelectatic ear Cartilage tympanoplasty	9	8	1
Adhesive otitis media cartilage tympanoplasty	3	2	1
Adhesive otitis media cartilage tympanoplasty + ossiculo plasty	4	2	2

Table - 3

Grade of Retraction	**No.of patients**	**Percentage**
Grade III	**9**	**56%**
Grade IV	**7**	**44%**