STUDY OF EUSTACHIAN TUBE FUNCTION IN NORMAL ADULTS AND THOSE WITH MIDDLE EAR DISEASE

Dissertation submitted to

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

CHENNAI

DECLARATION

I solemnly declare that the Dissertation entitled **" Study of Eustachian Tube Function in Normal Adults and Those with Middle Ear Disease "** was done by me at Coimbatore Medical College & Hospital during the period from December 2012 to November 2013 under the guidance and supervision of **Prof.Dr. V.Aravinthan, M.S. ENT , DNB.**

This dissertation is submitted to The Tamilnadu Dr. M.G.R Medical University towards the partial fulfillment of the requirement for the award of M.S. Degree(Branch IV) in Otorhinolaryngology.

Place : Coimbatore
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CERTIFICATE

This is to certify that this dissertation entitled "**STUDY OF EUSTACHIAN TUBE FUNCTION IN NORMAL ADULTS AND THOSE WITH MIDDLE EAR DISEASE**" submitted by **Dr. D.Vijay Babu** appearing for M.S. ENT (Branch IV) Degree Examination in April 2014 is a bonafide record of work done by him under my direct guidance and supervision in partial fulfillment of regulations of The Tamil Nadu Dr. M.G.R. Medical University, Chennai. I forward this to The Tamil Nadu Dr. M.G.R. Medical University, Chennai, Tamil Nadu, India.

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ABBREVIATIONS USED

AOM	Acute otitis media
COM	Chronic otitis media
CP	Central perforation
ET	Eustachian tube
ETF	Eustachian tube function
ETD	Eustachian tube dysfunction
TM	Tympanic membrane

CONTENTS

INTRODUCTION

Eustachian tube dysfunction is widely recognised as the triggering factor of middle ear disease. Though it happens to be the important factor for various middle ear problems, Eustachian tube function tests are not routinely done. Eustachian tube dysfunction predisposes the ear for chronic middle ear disease and it is a cause for concern to ENT Surgeon as the success rate of surgery is less when performed in ear with impaired tubal function. Eustachian tube function's role in successful outcome of surgeries for chronic otitis media has become a topic of interest in recent years.

There are various methods to test the functioning of Eustachian tube. Pneumatic otoscopy, Eustachian tube cathetarisation ,Valsalva manoeuvre, Politzer's test , Toynbee test ,Impedance audiometry ,Imaging ,Inflation-Deflation method and Sonotubometry are the methods available for testing the function of Eustachian tube. Testing the pressure regulation function of Eustachian tube by modern impedance audiometers is an non-invasive simple cost effective method for identifying Eustachian tube dysfunction.

Recent studies show that the success rates of tympanoplasty were found to be lesser when surgery was done in ears having poor tubal function compared to ears with normal Eustachian tube function. These studies have made surgeons realise the importance of Eustachian tube and testing the functioning of Eustachian tube.

Patient's with mucosal type of chronic otitis media (COM) are classified into healed COM ,inactive COM and active COM. Patients with tympanosclerosis and healed perforation are grouped under healed COM. In inactive mucosal COM there is a permanent perforation of the pars tensa but the middle ear mucosa is not inflamed. Active mucosal disease is where there is a permanent defect of the pars tensa with an inflammed middle ear mucosa which produces mucopus that may discharge. Testing the functioning of Eustachian tube in patients with COM and addressing the treatable causes of Eustachian tube dysfunction before tympanoplasty increases the success rate of the surgery.

This study was performed to find the functioning of Eustachian tube in normal adults and those with middle ear disease. This study was done in Department of ENT, Coimbatore Medical College Hospital ,Coimbatore in patients with normal tympanic membrane , in COM patients with dry central perforation, in patients with retracted tympanic

membrane and in patients with healed central perforation. Post operative patients who underwent myringoplasty and type 1 tympanoplasty were also tested for Eustachian tube function.

OBJECTIVES OF THE STUDY:

- To evaluate Eustachian tube function in normal adults .
- To test the functioning of Eustachian tube in patients with dry central perforation.
- To compare ET functioning of normal adults with patients having chronic otitis media.
- To find out whether it is important in healing of tympanic membrane in patients with central perforation.
- To evaluate its role in outcome of Myringoplasty and Type 1 Tympanoplasty.
- To evaluate its role in patients with retracted tympanic membrane.

REVIEW OF LITERATURE

The Eustachian tube otherwise known as pharyngotympanic tube connects the middle ear space with the Nasopharynx. The Eustachian tube (ET), middle ear, and mastoid cavity are spaces within the head, lined with respiratory epithelium and functioning in many ways like the respiratory sinuses. Consequently, the ET, ear, and sinuses share many similar types of mucosal physiology and pathology.

Three important physiologic functions of the ET are

(1) regulation of the middle ear pressure,

(2) protection of the middle ear and

(3) clearance of the middle ear space [1].

It is a well established fact that eustachian tube dysfunction (ETD) is an important factor in the causation and progression of chronic secretory otitis media[2]. ETD can also cause more invasive diseases. When the tubal mechanism fails, either in passive or active function, it leads to a series of events in the middle ear space that varies from a mild retraction to fulminate cholesteatoma.

HISTORY

Bartolomeus Eustachius, for whom the ET is named is the first researcher of the Eustachian tube in modern era .He was a 16th century anatomist who taught at the Collegia della Sapienza in Italy. Eustachius, studied all areas of the human body and described Eustachian tube in detail [3].

Further work was done by Antonio Valsalva, (1666-1723). Valsalva was a anatomist at Bologna and known for valsalva manoeuvre which tests the ET function by middle ear insufflation. Valsalva named the auditory tube, the Eustachian tube, and described its function in detail[4].

Adam Politzer is a pioneer in the field of otology and probably considered as the greatest otologist of the nineteenth century. One of his important inventions was a hand-held air bag that allowed insufflation of the middle ear space also known as politzerization, as the inventor termed it. For therapeutic purpose he developed a primitive middle ear ventilation tube [5].

EMBRYONIC DEVELOPMENT

The development of the eustachian tube and middle ear occurs as an out pouching of the pharynx that forms the tubotympanum and the pneumatised temporal bone.[6] The cartilaginous portion of the eustachian tube undergoes the majority of the growth seen in utero.[1] During development the ET provides a continuous sheet of epithelial cells to create the middle ear lining.[7]

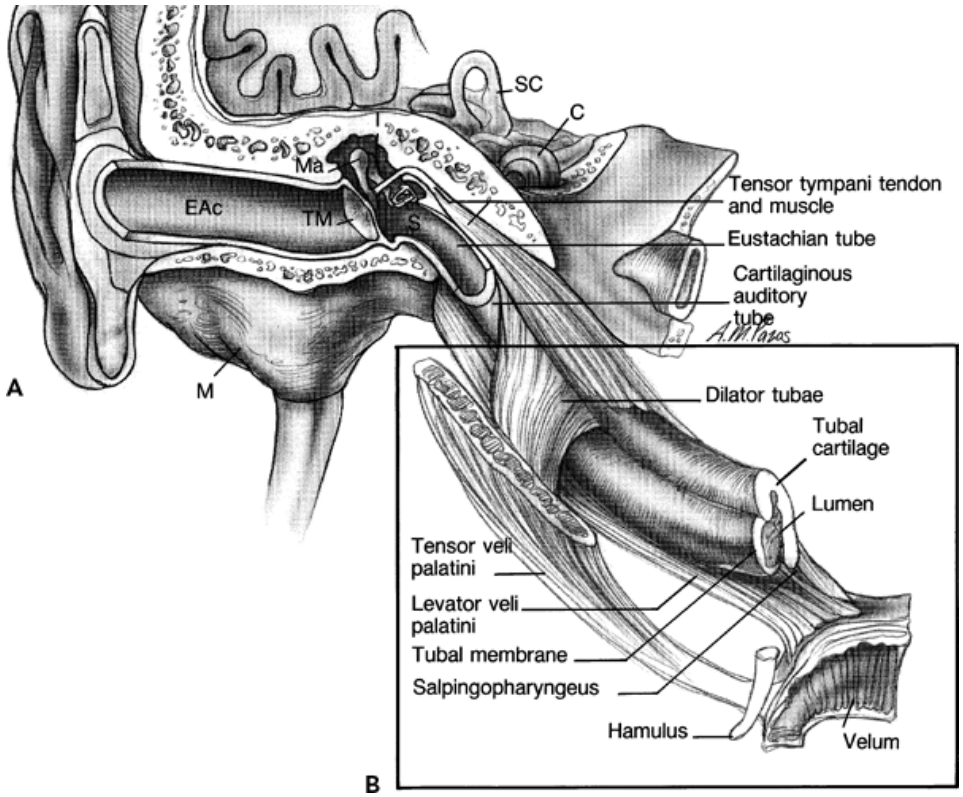
The main difference between mucosa of the the middle ear and the ET mucosa is that the epithelium of the ET differentiates into respiratory epithelium (pseudostratified ciliated columnar), whereas the middle ear epithelium does not. However, simple nonciliated cuboidal epithelium is present in the posterior part of the middle ear[8]. This distinction found in ET mucosa provides a more effective, inherent protective component. Unique characteristics of the ET include the presence of more mucus cells and accessory glands when compared with the middle ear mucosa [9].

Compared with adults, the position of the infant ET is 10 degrees from the Frankfort horizontal plane. This angle is different in adults whose tube is positioned at a 45 degrees.[1] Among other differences in adult and pediatric ETs, a less angled ET in the pediatric population has been thought to be responsible for the increased incidence of middle ear

pathology. However,some researchers have found that active muscle function, rather than passive clearance and impedance of the ET, is responsible for the decreased disease state seen in adults. [10,11\]

A smaller or partially obstructed ET does not necessarily correlate with the risk of disease or even active disease. Using an in vivo model, Sade and coworkers[12] in 2004 found that narrowing of the ET alone did not prohibit natural gas flow into the middle ear space with a swallowing manoeuvre. Although the narrowed ET was open for a brief period, this was sufficient to overcome a negative pressure.

ANATOMY



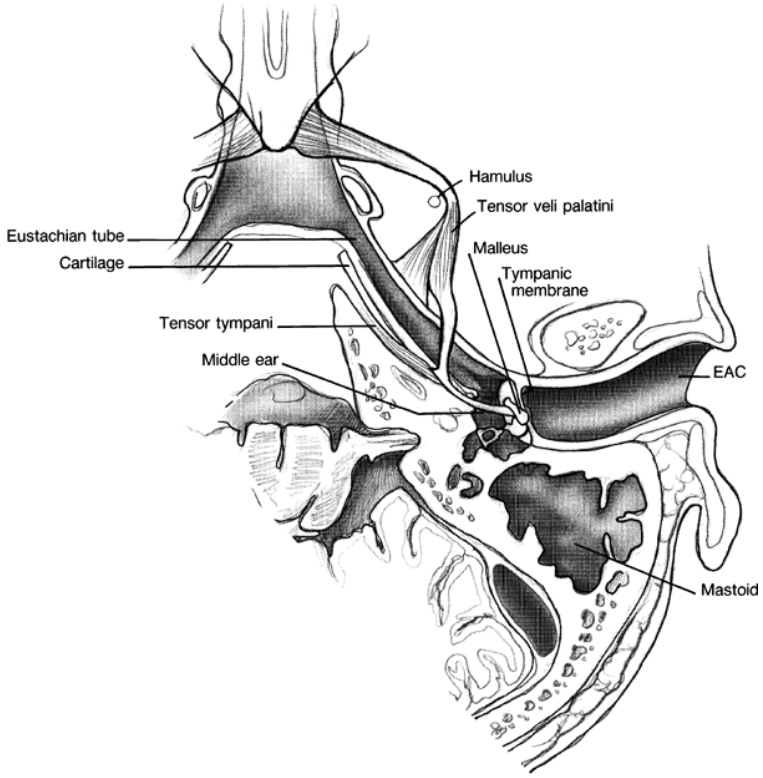
The length of the ET has been reported to be between 31 and 38 mm. [13] The normal orientation of the ET is downward, anterior, and with a medial rotation. With this positioning, the ET creates an angle of about 45 degrees and 30 to 40 degrees with the sagittal and horizontal planes, respectively .[9]

The Eustachian tube is made up of bone, cartilage, and fibrous tissue. The bony component is approximately 12 mm in length, whereas the cartilaginous is about 24 mm in length. This longer portion is described as a triangular plate of elastic fibrocartilage. The base of the tube forms the torus tubarius, which is posterior to the nasopharyngeal opening the eustachian tube [9].

Blood supply to the Eustachian tube and its supporting structures originates from the deep auricular branches of the internal maxillary artery, the ascending palatine artery and the ascending pharyngeal artery[14.]

Sensory and motor innervation of the Eustachian tube is supplied by a branch from the otic ganglion,the pharyngeal plexus from branches of the glossopharyngeal nerve and sphenopalatine nerve. Sympathetic branches innervate the Eustachian tube from the sphenopalatine ganglion, glossopharyngeal nerve, otic ganglion, petrosal nerves and the

carticotympanic nerve. Parasympathetic innervation is from the tympanic branch of the glossopharyngeal nerve [1,14].



MUSCLES OF THE ET

Four muscles are associated with the ET, which through a complex interaction; assist with equilibrating middle ear pressure. These muscles are:

1. Tensor veli palatini,
2. Levator veli palatini,
3. Salpingopharyngeus, and
4. Tensor tympani.

The tensor veli palatini (TVP) is a thin muscle lateral to the levator veli palatini. The medial portion of this muscle is the primary dilator of the Eustachian tube. The origin of the muscle arises from three locations. One origin is at the base of the medial pterygoid plate on the scaphoid fossa. The second is from the spina angularis of the sphenoid, and the third is from the lateral wall of the cartilaginous eustachian tube. The Tensor veli palatini muscle descends and inserts on a tendon at the pterygoid hamulus [9,15].

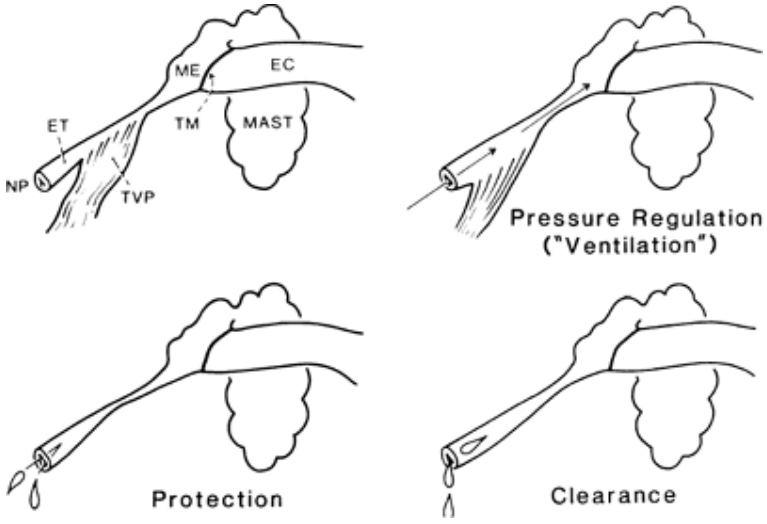
Levator veli palatini is a thicker muscle compared with the Tensor veli palatini and lies lateral to the choanae. Its origin is from two anatomic sites. The first is the inferior surface of the apex of the petrous part of temporal bone. The second origin is the medial lamina of the cartilage of the Eustachian tube. The muscle extends above the superior pharyngeal constrictor merging with the opposite levator muscle at midline [9,15].

Salpingopharyngeus originates from the inferior portion of the Eustachian tube extending downward joining the pharyngopalatinus muscle and assists with elevation of the pharynx and opening of the Eustachian tube with deglutition [9,15].

Tensor tympani is a large muscle encased in a bony canal above the osseous portion of the Eustachian tube. The tensor tympani origin involves three locations .The first is the cartilaginous portion of the ET, and the second is the greater wing of the sphenoid. The third is attachments to the bony canal in which the muscle travels. The tensor tympani insertion is at the manubrium of the malleus [9,15].

ANATOMIC FEATURES OF EUSTACHIAN TUBE	**IN THE INFANT, AS COMPARED WITH ADULT , IT IS**
Length of the tube	shorter
Angle of the tube	10 A^0 vs 45 A^0
Angle of tensor veli palatine muscle to cartilage	Variable vs Stable
Cartilage cell density	Greater
Lumen	Smaller area
Mucosal folds	Greater
Ostmann pad of fat	Wider
Cartilage volume & middle ear volume	Less
Elastin at hinge portion of cartilage	Less

FUNCTIONS OF THE EUSTACHIAN TUBE



Normally, the ET stays closed and opens when necessary to equalize pressure. Other functions include clearance of middle ear fluid while at the same time preventing nasopharyngeal secretions refluxing into the middle ear space.

1.Ventilation and regulation of middle ear pressure[16]

For normal hearing, it is essential that pressure on two sides of the tympanic membrane is equal. Negative or positive pressure in the middle ear affects hearing .Eustachian tube has to open periodically to equilibrate the air pressure in the middle ear with ambient pressure. Normally, the Eustachian tube remains closed and opens intermittently during swallowing, yawning and sneezing. Posture also affects the function; tubal opening is less efficient in recumbent position and during sleep due

to venous engorgement. Tubal function is also poor in infants and young children and thus responsible for more ear problems in that age group. It usually normalizes by the age of 7-10 years.[17-19]

2. Protective functions.

Abnormally, high sound pressures from the nasopharynx can be transmitted to the middle ear if the tube is open thus interfering with normal hearing. Normally, the eustachian tube remains closed and protects the middle ear against these sounds.[20]

A normal eustachian tube also protects the middle ear from reflux of nasopharyngeal secretions into the middle ear. This reflux occurs more readily if the tube is wide in diameter (patulous tube), short in length, (as in babies) or the tympanic membrane is perforated (cause for persistence of middle ear infections in cases of tympanic membrane perforations).

High pressures in the nasopharynx can also force nasopharyngeal secretions into the middle ear, e.g. forceful nose blowing, closed-nose swallowing as in the presence of adenoids or bilateral nasal obstruction.

3. Clearance or drainage

Mucous membrane of the Eustachian tube and anterior part of the middle ear is lined by ciliated columnar epithelium. The cilia beat in the direction of nasopharynx. This helps to clear the secretions and debris in

the middle ear towards nasopharynx. The clearance is further augmented by active opening and closing of the tube.

Gas exchange between the middle ear cleft and the nasopharynx through the fibrocartilaginous Eustachian Tube

The fibrocartilaginous Eustachian tube has a valve-like function: in its cartilaginous portion the mucosal surfaces are in apposition during the rest position. The tubal muscles must actively dilate the tubal valve in order to obtain adequate gas transfer into the middle ear cleft, so gas exchanges may occur.

At first, in normal conditions, healthy mucosa, in a normal daily situation, the fibrocartilaginous Eustachian tube provides an intermittent supply of gas, by transfer into the middle ear cleft. This is a regular, periodic, active process of gas transfer. Currently it is considered that, in normal conditions and situations, gas transfer via the fibrocartilaginous Eustachian tube is relatively poor, compared with the delicate and perfectly controlled gas exchanges between the middle ear cleft and blood compartment, via the mucosa.

The amount of gas introduced into the middle ear cleft is relatively poor: being approximately one micro-litre, five thousand times a day. It means 5 ml a day.The duration of the intermittent opening of the lumen

of the fibrocartilaginous Eustachian tube has been estimated to be about three to four minutes a day.

A second situation occurs during normal conditions, healthy mucosa, but exposed to an exceptional situation: altitude, flying, diving, etc., or during an accidental situation, such as an explosion, for example. In all these cases, the fibrocartilaginous Eustachian tube plays the leading role in balancing out the variations of pressure in the middle ear cleft. It is a highly sophisticated valve of security. It serves as a release valve for excessive positive pressure in the middle ear cleft and by means of muscular forces, opens to equalize excessive negative pressure in the middle ear cleft.

A third situation occurs when the mucosa undergoes an inflammatory process. The fibrocartilaginous Eustachian tube no longer balances the exchanges of gases between the middle ear cleft and the blood compartment. This means that with regard to the gas supply to the middle ear cleft, the main process occurs at the level of the middle ear cleft mucosa.

ETIOLOGY OF EUSTACHIAN TUBE DYSFUNCTION

Multiple causes of ET dysfunction exist. This varied differential includes infectious, allergic, mechanical (obstructive), environmental exposure, genetic, reflux, congenital, and iatrogenic causes.

Upper respiratory tract infection

Eustachian tube is commonly affected by viral upper respiratory tract infections (URIs).Healthy tubal function decreased the risk of complications resulting from URI.[21]

Chronic sinusitis

Chronic sinusitis is one of the most common cause for Eustachian tube dysfunction. Chronic sinusitis causes inflammation around the Eustachian tube opening in the Nasopharynx and over a period of time causes permanent change in mucociliary clearance and pressure regulation. Patients who had undergone endoscopic sinus surgery had relief of their otologic symptoms related to Eustachian tube dysfunction.[22]

Allergic rhinitis

Allergy induced changes in the epithelial lining of Eustachian tube and middle ear mucosa is well documented[23,24] .Allergic rhinitis with viral infections interact to enhance the physiologic responses of the middle ear and ET [25].

Adenoid hypertrophy

Adenoid enlargement obstructs the nasopharyngeal opening of the ET and it can also impair mucociliary clearance from the tube by means of metaplasia of ciliated epithelium to nonciliated epithelium and fibrosis of connective tissue associated with adjacent adenoid tissue [26].

Tobacco smoke

Two studies published by Agius and coworkers[27,28], confirmed that there was a decrease in ciliary beat frequency of the mucosa of the Eustachian tube in smokers compared with nonsmokers. This finding, however, has been tempered by work by Coggins and colleagues[29] and Antonelli and coworkers[30] who found passive tobacco smoke in the animal model to have little effect on otitis media. According to Dubin and coworkers [31], passive smoke does affect the Eustachian tube function, but may play only part of a role in causing middle ear disease.

Reflux

Gastro oesophageal reflux disease is one of the important cause of ET dysfunction and recent studies have confirmed it beyond doubt. White and coworkers[32] determined that not only exposure to gastric contents in the nasopharynx caused a significant ET dysfunction in an animal model, it also disabled middle ear pressure regulation and mucociliary clearance

of middle ear contents. Heavner and coauthors[33] in 2001 had previously published similar results in an animal model.

Cleft palate

Children with a cleft palate have an increased risk of middle ear pathology [34]. The incidence of ETD has been quoted as high as 79% in patients with cleft palate and cleft lip/palate [35].

Arnold and coworkers found patients with bilateral cleft palate to have a nearly horizontal course of the ET, possibly worsening symptoms. Although Tensor veli palatini muscle had a bony attachment on either side, the levator veli palatini muscle showed an abnormal course. This finding led the investigators to conclude that, during contraction, an aberrant obstruction of the ET may result.

Unfortunately, some cleft patients continue to have Eustachian tube dysfunction postoperatively and as they grow into adulthood. One third of the adults in a 2006 study by Gudziol and Mann [36] with cleft lip and palate had persistent tubal dysfunction.

Radiation

Treatment of nasopharyngeal malignancies with external beam radiation has detrimental effects on the surrounding structures, especially

the ET. Multiple investigators have found patients with early and late middle ear pathologies secondary to iatrogenic ET injury [37–39].

Reduced mastoid air cell system

The presence of a mastoid air cell system has been reported as an important criterion postoperatively to act as a pressure buffering system. However, this function is dependent on having healthy mastoid mucosa[40].

Nitrous oxide

A study by Teixeira and coworkers[41] in 2005 found that approximately one half of patients who received 50% nitrous oxide under general anesthesia, had Type C tympanograms postoperatively, compared with a Type A tympanogram preoperatively. This change in middle ear pressure is likely secondary to absorption of nitrous oxide, leaving a decrease in gaseous volume in the middle ear space.

EUSTACHIAN TUBE FUNCTION TESTS

Assessment can be started initially by taking a thorough history. A typical Eustachian tube dysfunction patient will complain of fullness or clogging of the ears, pain or discomfort, hearing loss, tinnitus, and dizziness. Most concerning to these patients is when these symptoms cannot be relieved by swallowing, yawning, or chewing.

Some researchers have reported that tests for evaluating ET function are not reliable [42]. However, most agree that there are objective and subjective assessments helpful in studying Eustachian tube dysfunction.

1.Pneumatic otoscopy

Using pneumatic otoscopy, an examiner can evaluate the mobility of the tympanic membrane. Stiffness or middle ear effusions are suggestive of Eustachian tube dysfunction.

2.Nasopharyngoscopy

Nasopharyngoscopy, represents another manner to visually inspect the posterior nasopharynx and proximal opening of the ET. Pathology such as adenoid hypertrophy or mucosal edema can be seen. It is well known that a rigid or flexible nasal endoscope allows the examiner to visualize the nasopharyngeal opening of the ET. Usually 30 degree or 70 degree rigid Hopkins rod endoscopes provide the best visualization. Other researchers have advocated using 0.8-mm flexible fiberscopes to evaluate beyond the isthmus of the ET and even into the middle ear cleft. [43,44]

3. Tympanometry

Tympanometry is based on a simple principle, i.e. when a sound strikes tympanic membrane, some of the sound energy is absorbed while the rest is reflected .A stiffer tympanic membrane would reflect more of sound energy than a compliant one. By changing the pressures in a sealed external auditory canal and then measuring the reflected sound energy, it is possible to find the compliance or stiffness of the tympano-ossicular system and thus find the healthy or diseased status of the middle ear.

Essentially, the equipment consists of a probe which snugly fits into the external auditory canal and. has three channels;

(i) to deliver a tone of 220 Hz,

(ii) to pick up the reflected sound through a microphone and

(iii) to bring about changes in air pressure in the ear canal from positive to normal and then negative. By charting the compliance of tympano-ossicular system against various pressure changes, different types of graphs called *tympanograms* are obtained which are diagnostic of certain middle ear pathologies.

Types of tympanograms

1.Type A Normal tympanogram.

2.Type As Compliance is lower at or near ambient air pressure. Seen in *fixation of ossicles*, e.g. otosclerosis or malleus fixation.

3.Type Ad High compliance at or near ambient pressure.Seen in ossicular discontinuity or thin and lax tympanic membrane.

4.Type B A flat or dome-shaped graph. No change in compliance with pressure changes. Seen in middle ear fluid or thick tympanic membrane.

5.Type C Maximum compliance occurs with negative pressure in excess of 100 mm of H_2O. Seen in retracted tympanic membrane

TYPE OF TYMPANOGRAM	DESCRIPTION
PEAKED	
A	Between +200 and -99 da pa
C_1	Between -100 and -199 da pa
C_2	Between -200 and -399 da pa
NON PEAKED	
B	No observable peak between +200 and -600 da pa

Tympanometry has also been used to find function of eustachian tube in cases of intact or perforated tympanic membrane . A negative or a positive pressure (- 200 or + 200 mm of H20) is created in the middle ear and the person is asked to swallow 5 times in 20 seconds. The ability to equilibrate the pressure indicates normal tubal function. The test can also be used to find the patency of the grommet placed in the tympanic membrane in cases of serous otitis media[45].

4. Valsalva test

The principle of this test is to build positive pressure in the Nasopharynx so that air enters the eustachian tube. To do this test, patient pinches his nose between the thumb and index finger, takes a deep breath, closes his mouth and tries to blow air into the ears. If air enters the middle

ear, the tympanic membrane will move outwards which can be verified by otoscope or the microscope. In the presence of a tympanic membrane perforation, a hissing sound is produced or if discharge is also present in the middle ear, cracking sound will be heard.

Failure of this test does not prove blockage of the tube because only about 65% of persons can successfully perform this test. This test should be avoided in the presence of atrophic scar of tympanic membrane which can rupture, and in the presence of infection of nose and nasopharynx where infected secretions are likely to be pushed into the middle ear causing otitis media.

5.Toynbee test

While valsalva tests positive pressure, Toynbee's manoeuvre causes negative pressure. It is a more physiological test. It is performed by asking the patient to swallow while nose has been pinched. This draws air from the middle ear into the nasopharynx and causes inward movement of tympanic membrane which is verified by the examiner otoscopically or with a microscope. Another variant of Toynbee test is equilibration of pressure generated by impedance audiometer by repeated swallowing which is an important method of testing tubal function in perforated ear drum[46].

6. Politzer test

In the Politzer test, one of the patient's nostrils is occluded with a rubber balloon as the examiner pinches the other nostril tightly. The patient elevates the palate by swallowing or phonating. The examiner then forces air into the closed nasal cavity from Politzer's bag. Air can be heard going into the middle space with an auscultation device. The examiner can also visually compare the tympanic membrane before and after the procedure to determine its relative patency [47].

7.**Radiological test**.

A radio-opaque dye, e .g . hypaque or lipoidal instilled into the middle ear through a pre-existing perforation, and X-rays taken should delineate the tube and any obstruction. The time taken by the dye to reach the nasopharynx also indicate its clearance function. This test is no longer popular now.

8.Catheterisation.

First nose is anaesthetised by topical spray of lignocaine and then a Eustachian tube catheter, the tip of which is bent, is passed along the floor of nose till it reaches the nasopharynx. Here it is rotated 90 degree medially and gradually pulled back till it engages on the posterior border of nasal septum. It is then rotated 180 degree laterally so that the tip lies

against the tubal opening . A Politzer's bag is now connected to the catheter and air insufflated. Entry of air in to the middle ear is verified by an auscultation tube.

The procedure of catheterization should be gentle as it is known to cause complications such as:

a) Injury to eustachian tube opening which causes scarring later.

b) Bleeding from the nose.

c) Transmission of nasal and nasopharyngeal infection into the middle ear causing otitis media.

d) (d) Rupture of atrophic area of tympanic membrane if too much pressure is used.

8. Saccharine or methylene blue test.

Saccharine solution is placed into the middle ear through a pre-existing perforation. The time taken by it to reach the pharynx and impart a sweet taste is also a measure of clearance function. Similarly, methylene blue dye can be instilled into the middle ear and the time taken by it to stain the pharyngeal secretions can be noted. Indirect evidence of drainage/clearance function is established when ear drops instilled into the ear with tympanic membrane perforation cause bad taste in throat.[42]

9.Bluestone's nine-step test.

Another method of measuring Eustachian tube function is an inflation-deflation test developed by Bluestone[48], although the applied middle-ear pressures are limited in magnitude. This test is currently used to test Eustachian tube function when the tympanic membrane is intact. The middle ear must be free of effusion.

The nine-step tympanometry procedure may be summarized as follows:

1. The tympanogram records resting middle-ear pressure.
2. Ear canal pressure is increased to +200 mm H_2O with medial deflection of the tympanic membrane and a corresponding increase in middle-ear pressure. The subject swallows to equilibrate middle-ear overpressure.
3. While the subject refrains from swallowing, ear canal pressure is returned to normal, thus establishing a slight negative middle-ear pressure (as the tympanic membrane moves outward). The tympanogram documents the established middle-ear under pressure.
4. The subject swallows in an attempt to equilibrate negative middle-ear pressure. If equilibration is successful, airflow is from the nasopharynx to the middle ear.

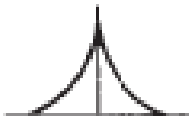
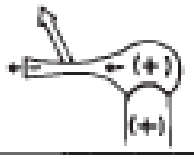
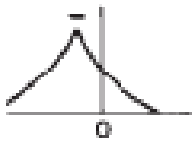
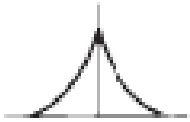
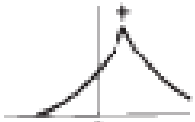
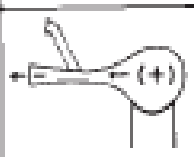
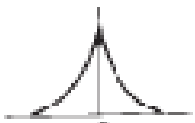
5. . The tympanogram records the extent of equilibration.

6. Ear canal pressure is decreased to -200 mm H2O, causing a lateral deflection of the tympanic membrane and a corresponding decrease in middle-ear pressure. The subject swallows to equilibrate negative middle-ear pressure; airflow is from the nasopharynx to the middle ear.

7. The subject refrains from swallowing while external ear canal pressure is returned to normal, thus establishing a slight positive pressure in the middle ear as the tympanic membrane moves medially. The tympanogram records the overpressure established.

8. The subject swallows to reduce overpressure. If equilibration is successful, airflow is from the middle ear to the nasopharynx.

9. The final tympanogram documents the extent of equilibration. The test is simple to perform, can give useful information regarding Eustachian tube function, and should be part of the clinical evaluation of patients with suspected Eustachian tube dysfunction. In general, most normal adults can perform all or some parts of this test, but even some normal children have difficulty in performing it. However, if any patient can pass some or all of the steps, Eustachian tube function is considered good.

STEP	ACTIVITY	MODEL	TYMPANOGRAM
1.	RESTING PRESSURE		
2.	INFLATION AND SWALLOW (x 3)		
3.	PRESSURE AFTER EQUILIBRATION		
4.	SWALLOW (x 3)		
5.	PRESSURE AFTER EQUILIBRATION		
6.	DEFLATION AND SWALLOW (x 3)		
7.	PRESSURE AFTER EQUILIBRATION		
8.	SWALLOW (x 3)		
9.	PRESSURE AFTER EQUILIBRATION		

10.Forced-Response Test

It is used in perforated tympanic membrane,and the middle ear should be free of inflammation. It enables the study of both passive and active responses of the Eustachian tube. The contractions of the tensor veli palatine muscle causes active response as it displaces the lateral walls from the cartilage-supported medial wall of the tube. It can be determined whether ET dysfunction is due to the material properties of the ET or due to a defective active opening mechanism.

During this test, the middle ear is inflated at a constant flow rate,forcing the Eustachian tube open. After forcefully opening the tube, the pump continues delivering a constant airflow maintaining a steady stream of air through the ET. Then the subject is instructed to swallow for assessment of the active dilatation of the tube.

The method is unique in that it eliminates the “mucous forces” in the Eustachian tube lumen that may interfere with the results of the inflation-deflation test when an attempt is made to assess the active opening mechanisms and the compliance of the tube. In this test, the passive resistance is assessed, and the active resistance is determined during swallowing. Patients with nonintact tympanic membranes as a result of chronic perforation or tympanostomy tubes can be distinguished

from apparently normal subjects with traumatic perforations of the tympanic membrane and negative otologic histories. The ratio of the passive and active resistance correctly differentiates a normally functioning Eustachian tube from an abnormally functioning one.

But van Heerbeek and colleagues[49,50] compared the forced response test results with the pressure equalization test (inflation- deflation) in children who had tympanostomy tubes inplace and concluded that the pressure equalization test was more reliable over time than the forced-response test because the latter showed a downward shift with repeated measurements.

11. Sonotubometry.

A final testing mechanism is sonotubometery. In this procedure, a sound source is applied to the nostril as a microphone in the external auditory canal records the transmitted sound. Sound levels are measured as the ET opens and closes. The tone is heard louder when the tube is patent. It also tells the duration for which the tube remains open. It is a non -invasive technique and provides information on active tubal opening. Accessory sounds produced in the nasopharynx, during swallowing, may interfere with the test results. The advantage of this

diagnostic test is the ability to evaluate the ET with or without an intact membrane under physiologic conditions[51].

MEDICAL MANAGEMENT OF ET DILATORY DYSFUNCTION

The treatment of ET dilatory dysfunction depends upon identification of the etiology. Most cases of tubal dysfunction are due to mucosal disease and can be improved if the underlying source of the inflammation can be identified and treated.

The two most common etiologies seen in adult patients have been laryngopharyngeal reflux and allergic disease. The etiologies in children are still under investigation but reflux and allergies appear to play an important role. Infectious and environmental irritants may play a larger role in young children.

Laryngopharyngeal reflux should be treated with dietary modifications such as avoiding large meals within two hours of bedtime, avoiding foods that relax the lower esophageal sphincter (caffeine, carbonated beverages) and that promote acid production. Consideration should be given to acid reducing agents such as proton pump inhibitors preferably twice daily and H2 blockers at bedtime may be added. Refractory cases can be managed by sleeping on an inclined bed, and ultimately, fundoplication may be considered.

A careful history and physical examination for any evidence of allergic disease should be undertaken. Avoidance of offending allergens can be effective, but may not always be possible. Oral second-generation antihistamines, nasal steroid sprays, nasal antihistamine or mast cell stabilizer sprays, leukotriene inhibitors, or combination therapy may be effective in reducing allergic manifestations. Refractory cases may be helped with immunotherapy.

A thorough search for nasal, sinus, and any systemic inflammatory conditions should be done and appropriate therapy administrated. Recurrent infections should prompt a search for underlying nasal or sinus disease, immunosuppression or immunodeficiency, or primary mucosal disorders (*e.g.*, Samter's triad, Wegener's disease, ciliary dysfunction). Granulomatous disease may be treated with immunosuppressants.

Anatomical obstructions are unusual causes of tubal dilatory dysfunction.Neoplasms, especially malignancy, should be suspected with unilateral dilatory dysfunction and the pathology may be nasopharyngeal carcinoma, lymphoma, chondrosarcoma, and others. Otitis media with effusion is the second most common presentation of nasopharyngeal carcinoma after a neck mass. The diagnosis of neoplasms is best done with contrast enhanced MRI or CT imaging. Other benign lesions that may cause tubal dilatory dysfunction may be adenoid hypertrophy, mucus

retention cysts, Thornwaldt's cysts, or synechiae from adenoidectomy and other surgical procedures.

A hypertrophic adenoid pad does not need to cover the tubal orifice at rest to cause significant functional obstruction. During the swallowing process, contraction of the pharyngeal constrictors can compress an otherwise non-obstructive adenoid into the posterior surface of the posterior cushion of the ET and force it anteriorly to close the tubal orifice at the time it should be dilating open.

Nguyen *et al.* demonstrated that adenoidectomy for relief of otitis media with effusion was most effective when adenoid tissue was in contact with the posterior cushions of the Eustachian tube orifices.[52] These findings would be consistent with the dynamic observations of pharyngeal constriction acting to push the adenoid into the posterior cushions as a mechanism causing tubal dilatory dysfunction.

SURGICAL MANAGEMENT OF EUSTACHIAN TUBE DILATORY DYSFUNCTION

In the event that thorough investigation of underlying medical conditions and maximal medical therapy fails to resolve otitis media or atelectasis, surgical intervention may be indicated.

Tympanostomy tubes are effective in the treatment of otitis media with effusion and may prevent retraction of the eardrum, atelectasis, and other sequelae of ET dilatory dysfunction. In the event that non-infectious middle ear drainage or inflammation persists despite a tube in place, it raises the suspicion of a primary mucosal disorder rather than just a Eustachian tube problem. Thick proteinaceous 'glue-like' effusions that repeatedly occlude the lumen of the ventilating tube often will respond to oral or topical steroids and may represent primary mucosal disease.

In cases of long-term persistent tubal dilatory dysfunction, repeated placement of ventilating tubes may be necessary. In such circumstances, larger flanged tubes, such as 'T' tubes or subannular semi-permanent tubes may be indicated. Longer duration tubes raise the risk of permanent perforation of the tympanic membrane and development of squamous epithelial ingrowth from the perforation margins.

Gates [53] demonstrated that adenoidectomy is effective in children in resolving medically refractory otitis media with effusion and Nguyen[52] noted that the benefit was most significant when the adenoid was in contact with posterior cushion or the ET orifice.

For adult cases of medically refractory ET dilatory dysfunction who have received multiple tympanostomy tubes, Eustachian tuboplasty (ETP) is now being investigated. It is indicated for otitis media with effusion or non-adherent atelectasis when tubes have been the only other effective therapy. ETP appears to be most effective when the underlying medical conditions have been brought under control but there is irreversible mucosal disease causing functional obstructive dysfunction of dilation.

The hypothesis for the procedure is that debulking of the posterior cushion, which contains a thicker mucosal and submucosal surface than the anterolateral wall, may facilitate the muscular efforts to dilate the valve open. Laser or microdebrider tuboplasty with removal of redundant edematous tissue along the posteromedial Eustachian tube orifice has been shown to be an effective alternative for patients with chronic otitis media with effusion who have received numerous prior tympanostomy tubes with or without significant atelectasis.[54,55,56]

The operation involves debulking of the luminal mucosa and submucosa of the posterior cushion down to the medial cartilaginous lamina from the free margin at the nasopharyngeal opening and extending proximally up to the valve. The extent of valve mucosa treated depends on preoperative evaluation with slow motion video endoscopy to reveal

the dynamics of the functional obstruction. Debulking of the luminal side of the medial cartilaginous lamina, the cartilage within the posterior cushion, may increase the effectiveness of the operation.

The open defect is allowed to heal by secondary intention, a process that usually takes about six weeks. For that reason, a temporary tympanostomy tube is customarily inserted at the time of surgery. The ETP operation may be more efficacious in less severe cases of otitis media, non-adherent atelectasis, and patients prone to baro-injury with flights and scuba diving.

Metson *et al.*[56] performed microdebrider ETP on adult patients simultaneously with endoscopic sinus surgery in twenty adult patients with otitis media with effusion, flat tympanograms, and sinus disease requiring surgical management. They noted that 70% improved subjectively and by tympanogram or pure tone average improvement of 10 dB or more.

Even after successful surgery, patients must remain vigilant about avoidance of offending agents or continuing medical therapy for their underlying condition. If they cease allergy or reflux precautions or therapy, the effusion may recur.

Long-term results and controlled studies need to be done to determine the ultimate role of the procedure in the treatment of refractory otitis media. Further research must be done on the physiology and pathophysiology of tubal function in children before tuboplasty surgery would be recommended for pediatric patients.

CHRONIC OTITIS MEDIA

Chronic otitis media is any structural change in the middle ear system associated with a permanent defect in the tympanic membrane (TM). Usually, but not always, there is associated inflammatory mucosal disease in the middle ear, which may also involve the mastoid cells. If there is persistent or intermittent otorrhea through a nonintact TM, the amplified designation“chronic suppurative otitis media” is preferred. The condition is considered “chronic” if the TM defect is present for a period greater than 3 months. Thus, a draining middle ear that is associated with a perforation from acute otitis media would not qualify for this diagnosis if it responds to treatment within 3 months. Histologically, COM is defined as irreversible mucosal changes within the middle ear cleft.

There are two types of chronic otitis media – mucosal or tubotympanic type & squamous or attico antral type .Patient's with mucosal type of chronic otitis media are classified into healed COM ,inactive COM and active COM. Patients with tympanosclerosis and healed perforation are grouped under healed COM. In inactive mucosal COM there is a permanent perforation of the pars tensa but the middle ear mucosa is not inflamed. Active mucosal disease is where there is a permanent defect of the pars tensa with an inflammed middle ear mucosa which produces mucopus that may discharge.

The other type of COM is squamous type (active and inactive).In inactive type there is retraction of the pars flaccida or pars tensa (usually posterosuperior) which has the potential to become active with retained debris .In active type there is retained squamous epithelial debris with inflammation.

PERFORATIONS OF THE TYMPANIC MEMBRANE

Perforations of the TM are described according to their anatomic location and are separated into two categories. Central perforations involve the pars tensa and are circumferentially surrounded by residual TM. The umbo of the malleus is used as a reference point to divide the pars tensa into four quadrants, allowing one to describe the location of the

perforation. Subtotal perforations describe large defects in which there is only a narrow rim of residual pars tensa near the annulus.

Central perforations are rarely associated with cholesteatoma and for this reason have generally been considered “safe” ears. However, serious complications in the setting of a central perforation, such as intracranial abscess, have been reported.[57] The term “tubotympanic disease” is sometimes used to describe COM with a central perforation. This term stems from the fact that the TM defect exposes both the middle ear mucosa and eustachian tube but generally does not produce inflammatory changes in the mastoid.

Marginal perforations have no remnant of Tympanic membrane adjacent to the bone of the posterior canal wall. As a result, the bony external canal wall, attic, antrum, and mastoid cells can be involved with inflammation. Hence, this condition has been referred to as Atticoantral disease. Retraction pockets produce marginal perforations and also occur in the pars flaccida where they are known to cause attic perforations. In addition, total perforations with complete loss of the pars tensa result from necrotizing otitis media.

CHOLESTEATOMAS

Cholesteatomas are retraction pockets or cysts lined with squamous cell epithelium and filled with keratin debris occurring within the pneumatized spaces of the temporal bone. They are frequently associated with marginal perforations. The name is a misnomer as cholesteatoma is not a true neoplasm and does not contain cholesterol, but this familiar term retains wide popularity in clinical parlance.

Cholesteatomas have a propensity for growth, bone destruction, and chronic infection. Therefore, COM with cholesteatoma is considered an “unsafe” ear and generally requires surgical treatment.

Cholesteatomas are categorized as congenital or acquired. Congenital cholesteatomas appear as white pearly masses deep to normal, intact TMs. Prior history of otorrhea, perforation, or otologic procedures excludes this category.However, patients may have a prior history of uncomplicated acute otitis media.[58]

Acquired cholesteatomas are much more common and develop in the setting of a retracted or perforated TM. Primary acquired cholesteatomas arise from retracted but intact drumheads, most often within an attic. Secondary acquired cholesteatomas result from ingrowth

of squamous cell epithelium into the middle ear, usually through marginal perforations.

Cholesteatomas may also be described according to their anatomic location. Pars tensa cholesteatomas usually involve the posterosuperior quadrant of the TM and commonly cause erosion of the long process of the incus with discontinuity of the incudostapedial joint and conductive hearing loss. The disease frequently extends into the posterior tympanic spaces, facial recess, and sinus tympani.

Attic cholesteatomas arise from pars flaccida defects and tend to be associated with scutum defects owing to erosion of the outer wall of the epitympanum. The ossicles are frequently engulfed or eroded by the cholesteatoma, which may then extend into the mastoid antrum.

PATHOGENESIS OF CHRONIC OTITIS MEDIA

Chronic otitis media is an insidious process, and patients tend to present with long-standing disease.As a result, the etiology and natural course of this process remain obscure, although several credible theories have been advanced.

Traditionally, COM has been thought to follow a bout of acute otitis media (AOM) that resulted in TM perforation. However, this direct correlation has fallen out of favour for several reasons. First, AOM is one

of the most common childhood diseases. Comparatively speaking, COM is lesser in number. In addition, the majority of TM perforations secondary to AOM result in complete healing of the drumhead.[59] Second, whereas streptococcal otitis media, which causes necrotizing infections resulting in large perforations, is seldom seen today, the incidence of COM has remained constant.[60] Third, in a study of 200 patients with TM perforations, only 50% clearly recalled an acute, painful ear infection associated with the onset of otorrhea. Instead, 40% described the insidious onset of drainage or gradual hearing loss.

Although COM may not result directly from a single episode of AOM, it has been suggested that all cases of otitis media represent different stages in a continuum of events.[61–63] For example, histologic studies have demonstrated that persistent effusion in chronic secretory otitis media leads to degradation of the fibrous layer of the TM. Loss of the fibrous layer results in a weakened, atrophic, two-layered drumhead that is vulnerable to atelectasis or perforation and hence chronic middle ear disease.[59,64]

ATELECTATIC AND ADHESIVE OTITIS MEDIA

It is acknowledged that eustachian tube dysfunction plays an important role in the development of COM.[65] The eustachian tube serves to ventilate the middle ear so that pressure equalization occurs between this space and the surrounding environment.

In persisting eustachian tube dysfunction, especially as seen in Down syndrome and cleft palate, the middle ear space is continually exposed to negative pressure. As a result, the TM is retracted medially. In atelectatic or adhesive OM, the middle ear space is partially or completely obliterated. In long-standing atelectasis, patients are at risk for secondary acquired cholesteatomas.

Poor mastoid pneumatization is also associated with chronic middle ear disease.[65] Although pneumatisation is not completed until adulthood, the majority of the process takes place during the first 5 years of life. Infancy and early childhood infections occurring during this period are thought to prevent normal cellular development of the mastoid and thus lead to chronic middle ear disease. Temporal bone histopathologic studies also demonstrate that infection of a pneumatized cleft incites sclerosis, obliteration of air cells, and chronic middle ear disease in the setting of poor mastoid pneumatization.[59]

Ventilating tubes can be placed within an atelectatic TM in an attempt to equalize the pressure and allow the TM to return to its normal anatomic position. Fifty percent of patients with pressure equalization tubes experience at least one episode of otorrhea, and 3% will have symptoms persisting beyond 6 weeks.66 After extrusion of the tube, the majority of iatrogenic TM perforations will heal. Residual perforation rates are 2 to 3% for button or grommet tubes but as high as 47% for T tubes.[67,68] Continued eustachian tube dysfunction, persistent otorrhea, and ingrowth of squamous cell epithelium through the defect can prevent spontaneous healing.

CHRONIC OTITIS MEDIA (MUCOSAL OR TUBOTYMPANIC TYPE)

Recurrent infections of the middle ear generally result in irreversible mucosal changes. Histologic studies have shown that as the inflammatory process enters the chronic phase, there is a shift in cellular population from infiltrating leukocytes toward mononuclear cells such as macrophages, lymphocytes, and plasma cells.[69] These mononuclear cells secrete inflammatory mediators and growth factors that increase capillary permeability and lead to edema and hyperemia of the middle ear mucosa.

In chronic inflammation, the mucosa undergoes metaplasia from a single layer of ciliated cuboidal or columnar epithelium to mucosa resembling that of the respiratory tract with increased numbers of goblet and glandular cells. Consequently, there is an increase in the volume and viscosity of the mucus. These changes further overwhelm the already compromised mucociliary clearance capability of patients suffering from chronic middle ear disease.[69,70]

Granulation tissue consisting of vascular connective tissue with inflammatory infiltrates has been found to be the prominent pathologic feature of COM. Granulation tissue was identified in over 95% of the temporal bones studied from individuals with a history of COM.[71] Tympanosclerosis was present in 43%, cholesteatoma in 36%, and cholesterol granuloma in 21% of patients in one large histologic study of temporal bone pathology. A pathologic review of 800 temporal bones revealed that granulation tissue had both a higher prevalence and more generalized distribution when compared to cholesteatoma.[72] Both studies demonstrated identical pathologic changes within the middle ear cleft regardless of the presence of a Tympanic membrane perforation.

As granulation tissue matures, it becomes dense and fibrotic with decreased vascularity. This process leads to scarring and adhesions associated with the ossicular chain and Tymapnic membrane.[73]

Irreversible changes such as subepithelial edema and mucoperiosteal fibrosis occur deep to the epithelial lining.[74] As the inflammation persists, sclerosis, along with new bone formation, can cause a reduction in mastoid and antral pneumatization.

ET, MASTOID, AND CHOLESTEATOMA

Progression of cholesteatomas is affected by the status of mastoid.sometimes mastoid status found to be more important than ET dysfunction. Hasebe and coworkers [75] in 2001 compared three group of patients in different stages of tympanic membrane retraction. Three groups were as follows

1. Patients with a severe attic retraction pocket.
2. Patients with cholesteatoma who were conservatively treated,
3. Patients with cholesteatoma who needed surgery.

Though all three groups had Eustachian tube dysfunction, no significant difference in Eustachian tube function was identified among the three groups. Progression of the cholesteatoma appeared to be significantly associated with the ventilatory condition of the mastoid rather than the function of ET. Less aeration of mastoid system found in the surgery group compared with the nonsurgical group led to this observation [75].

When the ET fails to equalize pressure, a negative pressure in the middle ear space occurs. The mastoid is seen by many researchers as a buffer zone for the middle ear and tympanic membrane allowing some equalization of this abnormal pressure. Cinamon and Sade [76] developed a model to evaluate how pressure homeostasis of the middle ear can be maintained. They found that the worst "model" for adapting to these changes was a middle ear space with a small mastoid. The investigators proposed that this anatomic finding may lead to patients developing compensatory buffering mechanisms, such as retraction or fluid accumulation, which reduced middle ear volume .

Retraction pockets are well known to result from Eustachian tube dysfunction. Wolfman and Chole [77] in 1986 found cauterized Eustachian tubes of the Mongolian gerbil resulted in a progressive retraction in 75% of the animal in a 16-week period.

Examining the retracted tympanic membrane, Paparella and co-workers [78] described epithelial and subepithelial changes such as keratin accumulation,papillary growth, mucosal adhesion, irregular epithelium, and bone destruction. Although reasonable theories exist to describe why cholesteatomas occur, it is not entirely known what allows retraction pockets to evolve into cholesteatomas .

Cholesteatomas are known to arise more commonly in the pars flaccida.The reason for this may be that there is poorer aeration in this area, especially in the area of the tympanic isthmus. Kobayashi and colleagues [79] in 1994 compared computed tomography scans of 53 patients with retractions in the pars flaccida, including those with cholesteatoma. Their results found little association with this blockage and progression of a cholesteatoma.

SURGERY FOR CHRONIC OTITIS MEDIA

MYRINGOPLASTY

Myringoplasty is a procedure used to repair a tympanic membrane perforation, without the need to examine the middle-ear.

The procedure should be limited to patients who satisfy all of the following four criteria:

1. Relatively small central perforation of the tympanic membrane

2. Translucent tympanic membrane

3. No middle-ear disease is present or suspected

4. Hearing is within normal limits

When these conditions are not met, a tympanoplasty is indicated to facilitate the repair and to explore the middle ear.

TYMPANOPLASTY

A tympanoplasty, as opposed to only a myringoplasty, should be performed when there is a need to examine the middle ear, such as when there is conductive hearing loss that cannot be attributed to the size and position of the perforation (eg, ossicular discontinuity or fixation), when a retraction pocket is present, or when an occult middle-ear cholesteatoma is suspected.Additional indications would be when the perforation is large, when its location makes it difficult to repair using a myringoplasty procedure (eg, anterosuperior quadrant), or when it is a “marginal” perforation.

When the perforation is in the posterosuperior quadrant and is marginal, a tympanoplasty provides not only a higher success rate, but also an opportunity to inspect the ossicular chain and middle ear, especially the sinus tympani and facial recess, to rule out cholesteatoma.

CLASSIFICATION OF TYMPANOPLASTY (WULLSTEIN - 1956)

The classification of tympanoplasty based on middle-ear mechanics, consists of five types, each of which is based on the most lateral intact structure that remains connected to the inner ear:

Type I tympanoplasty - TM is grafted to an intact ossicular chain

Type II tympanoplasty - Malleus is partially eroded .TM is grafted to the incus

Type III tympanoplasty - Malleus and incus are eroded .TM is grafted to the stapes suprastructure.

Type IV tympanoplasty - Stapes suprastructure is eroded but foot plate is mobile. TM is grafted to a mobile foot plate .

Type V Tympanoplasty - TM is grafted to a fenestration in the horizontal semicircular canaL

According to J. Sade (1976), the classification of tympanic atelectasis has 4 grades:

Grade I Mild TM retraction

Grade II TM retraction in contact with incus or stapes

Grade III TM in contact with promontory wall (not adhered to it)

Grade IV TM adhered to promontory (adhesive otitis media)

MATERIALS AND METHODS

This study was done in the department of ENT, Coimbatore Medical College , Coimbatore from December 2012 to November 2013. It consists of 50 adults with normal tympanic membrane and 50 patients with chronic otitis media (inactive mucosal type). As a part of the study eustachian tube function was also evaluated in patients with retracted tympanic membrane ,healed central perforation and post operative patients who underwent myringoplasty or type 1 tympanoplasty . Age range was between 18 to 49 yrs.

Control group

50 adults who came to ENT outpatient department for hearing assessment with normal hearing and normal tympanic membrane were subjected to Eustachian tube function tests. Prior consent was obtained from them before Eustachian tube function tests.

Study group

100 adult patients who visited ENT outpatient department with middle ear disease were subjected to Eustachian tube function tests.\

Inclusion criteria

Chronic otitis media with dry central perforation including post operative patients with residual perforation

1. Retracted pars tensa of tympanic membrane (grade 1 & 2)
2. Healed central perforation
3. Post operative patients who underwent myringoplasty or type 1 tympanoplasty before 3 months

Exclusion criteria

1. Patients with active ear discharge & middle ear mucosal inflammation
2. Post operative patients who underwent surgery within three months
3. Retracted pars tensa of tympanic membrane (grade 3 & 4)
4. Patients with upper respiratory tract infection
5. Patients with squamous type of chronic otitis media
6. Patients with structural abnormality like cleft palate
7. Age below 18 years and above 49 years

A detailed history was recorded for all patients. Past surgical history was elicited in detail. Ear examination including tuning fork tests and video otoscopy was done for all patients. Other clinical examinations like nasal, oral cavity examination were done.

All patients were subjected to pure tone audiometry and graphical recording of their hearing threshold were made and pure tone average in both ears were recorded. Tympanometry was done in all patients.

Eustachian tube function was assessed by Valsalva test, pneumatic otoscopy (siegalization), nasopharyngoscopy & tympanometry.

Eustachian tube function was assessed with impedance audiometer with two tests. William's test was done in patients with intact tympanic membrane. Toynbee test was used in patients with perforated ear drum. In our study we used AMPLAID Audiometer for testing Eustachian tube function in perforated drum and MAICO 34 for testing Eustachian tube function in intact tympanic membrane.[80-85]

William's test

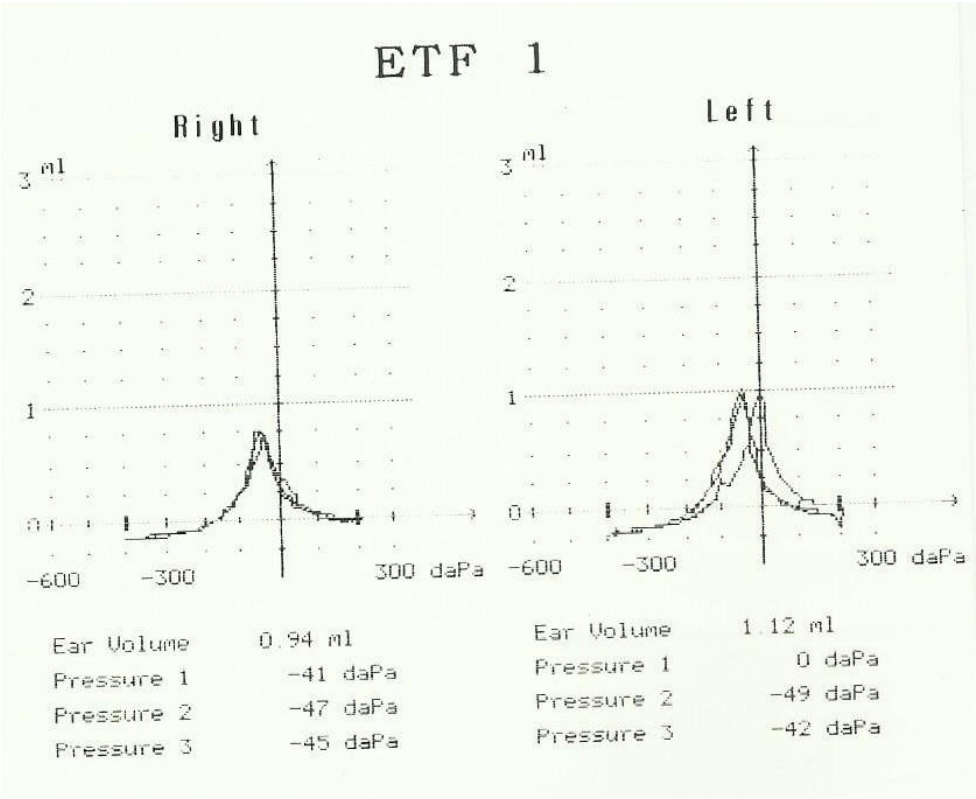
Middle ear pressure was measured at the start (resting pressure) , after patient swallows (with nose and mouth closed) and after Valsalva manoeuvre. Normal ambient middle ear is usually slightly negative.

Middle ear pressure becomes more negative on swallowing and becomes positive on Valsalva manoeuvre.

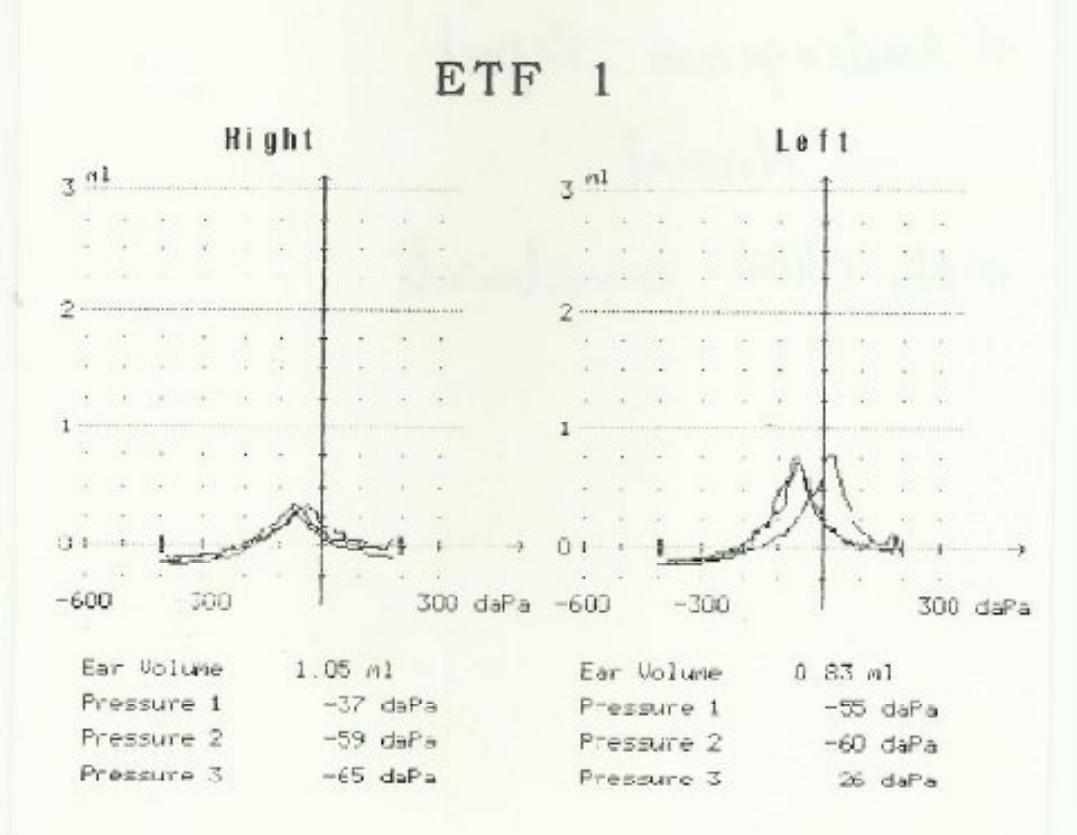
Interpretation :

1. **1.Partial impairment** : Middle ear pressure becomes negative on swallowing but it doesn't become positive on Valsalva or vice versa.
2. **Gross impairment** : Middle ear pressure doesn't change for both swallowing and Valsalva.

William's test printout showing gross impairment in right side and partial impairment in left side



William's test printout showing gross dysfunction in right side with normal tubal function in left side



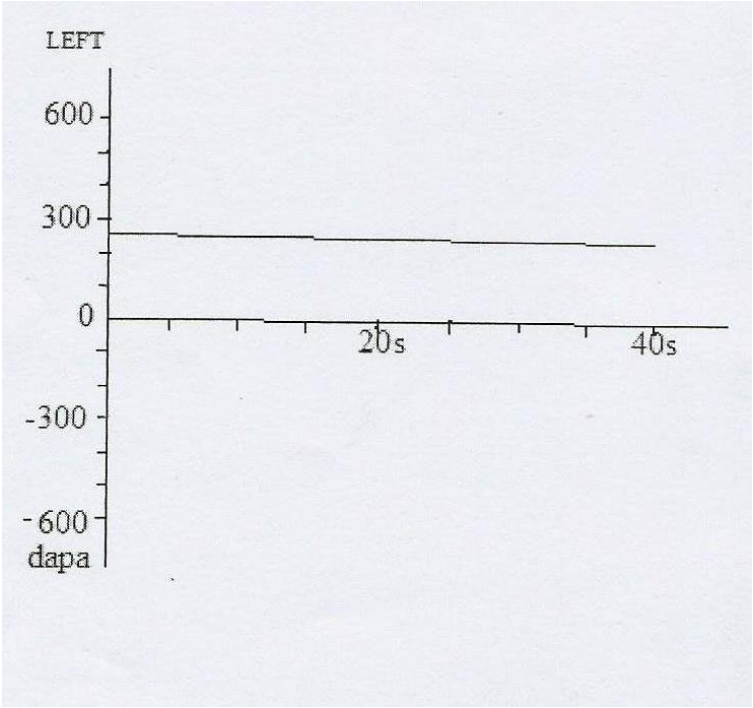
Toynbee's test :

A negative or positive pressure (- 250 or + 250 dapa) is created in the middle ear and the patient is asked to swallow 5 times . Change of air pressure in the middle ear is recorded each time when the patient swallows .The ability to equilibrate the pressure indicates normal tubal function. The test can also be used to find the patency of the grommet placed in the tympanic membrane in cases of serous otitis media.

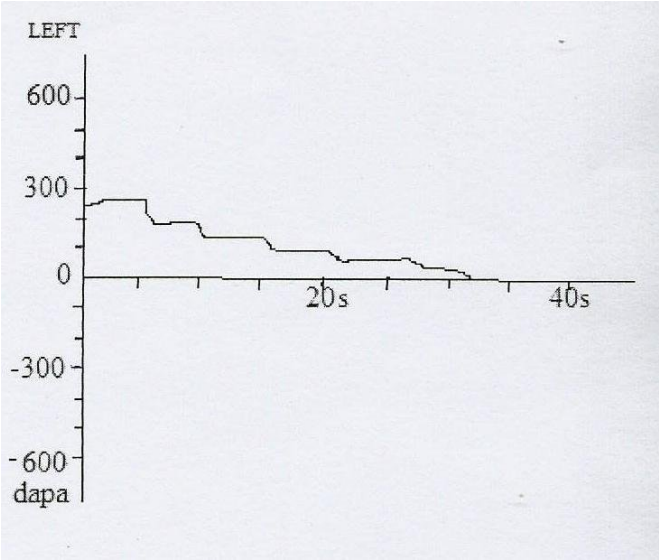
Interpretation :

1. **Normal Eustachian tube function -** The pressure built up by the impedance audiometer completely neutralized by repeated swallowing.
2. **Partial impairment** – Persisting of some residual pressure even after five swallows
3. **Gross impairment** - if the pressure built up by the impedance audiometer cannot be neutralized at all by repeated swallowing.

Toynbee‘s test showing gross impairment of tubal function



Toynbee's test showing normal tubal function in patient with dry perforation



RESULTS AND ANALYSIS

This study was performed to find the functioning of Eustachian tube in normal adults and those with middle ear disease. This study was done in department of ENT, Coimbatore Medical College & Hospital, Coimbatore in patients with normal tympanic membrane, chronic otitis media patients with dry central perforation, patients with retracted tympanic membrane and in patients with healed central perforation. Post operative patients who underwent myringoplasty and type 1 tympanoplasty were also tested for Eustachian tube function.

Eustachian tube function was assessed by Valsalva test, pneumatic otoscopy (siegalization), nasopharyngoscopy, tympanometry.

Eustachian tube function was assessed with impedance audiometer with two tests. William's test was done in patients with intact tympanic membrane. Toynbee test was used in patients with perforated ear drum. Control group was 50 normal adults in the age of 18 to 49 who were tested for Eustachian tube function. Out of 50 only one had partial ET dysfunction(2%).

Study group was 50 patients with dry central perforation of which 7 were post operative patients with failed tympanoplasty. Among 43 CSOM patients 26 had ET dysfunction (60.4 %). In 7 patients with residual Central perforation , 6 had ET dysfunction (85.71%) suggesting ET dysfunction as the important cause for failure of surgery. No significant age or gender difference in Eustachian tube dysfunction found.

As part of study 30 adults with retracted tympanic membrane and 8 patients with healed CP were tested for Eustachian tube function.In patients with retracted Tympanic membrane, ET dysfunction was identified in 27 patients (90 %) of which 21 had partial and 6 had gross dysfunction suggesting ET dysfunction as a major factor for chronic retraction.

In 7 out of 8 patients with healed tympanic membrane perforation ET function tests were normal suggesting that good Eustachian tube functioning plays a prominent role in the healing of tympanic membrane perforation.

12 patients who had undergone myringoplasty or type 1 tympanoplasty were tested for Eustachian tube functioning.10 patients (83 %) were found to have normal ET functioning while 2 had partial

tubal dysfunction suggesting that ET function tests are an important prognostic indicator for success of surgery.

Statistical analysis was done for all variables in the study using NCSS software . The data was analysed for mean, standard deviation, range and percentage.The chi-square and student's unpaired t tests were used for comparison between study and control groups.

Interpreting p-values was considered in the following manner.

Not Significant > 0.05

Significant < 0.05

Very Significant < 0.01

Highly Significant < 0.001

Table 1 :AGE DISTRIBUTION

Age in years	No.of normal adults	No.of patients with dry Central perforation
18 - 20	4	4
21 - 25	11	7
26 - 30	12	7
31 - 35	8	8
36 - 40	6	12
41 - 45	7	8
46 - 49	2	4
TOTAL	50	50

Line diagram showing age distribution of normal adults and patients with dry CP

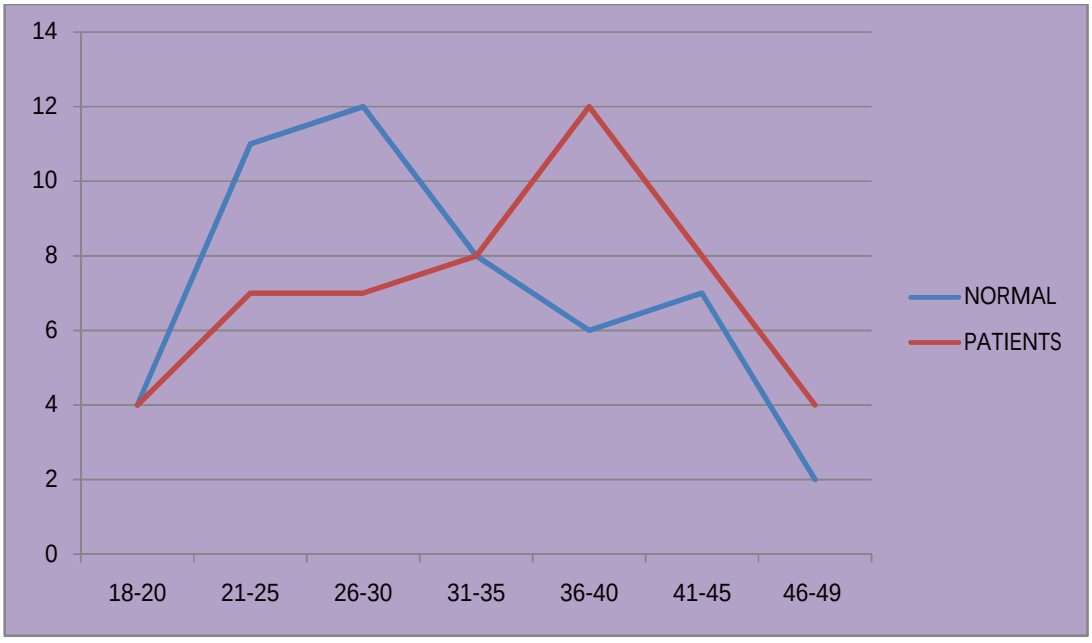


TABLE 2 :SEX DISTRIBUTION

SEX	Normal Adults	Patients With Dry Central Perforation
Male	26	29
Female	24	21

Bar diagram depicting the sex distribution of normal adults and patients with dry central perforation

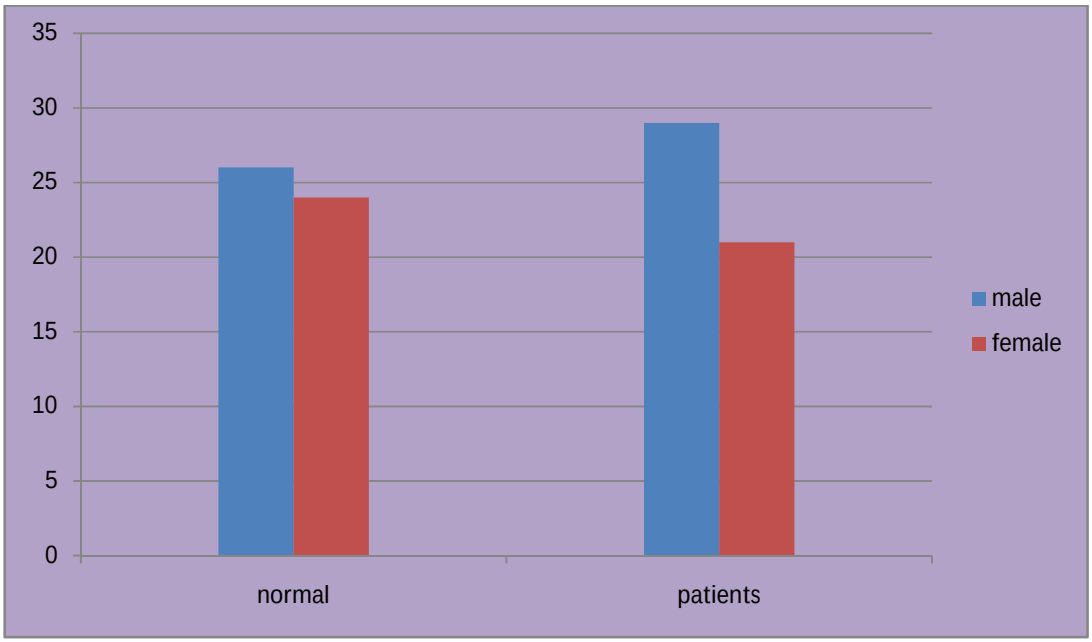


TABLE 3 : Age distribution of patients with retracted tympanic membrane ,patients with healed central perforation and post operative patients with neomembrane

Age (in years)	Retracted tympanic membrane	Healed central perforation	Post operative patients with neomembrane
18-20	5	1	1
21-25	3	2	2
26-30	7		2
31-35	5	1	4
36-40	4	2	1
41-45	4	2	1
46-49	2		1
Total	30	8	12

Line Diagram Showing Age Distribution Of Patients With Retracted Tympanic Membrane ,Patients With Healed Central Perforation And Post Operative Patients With Neomembrane

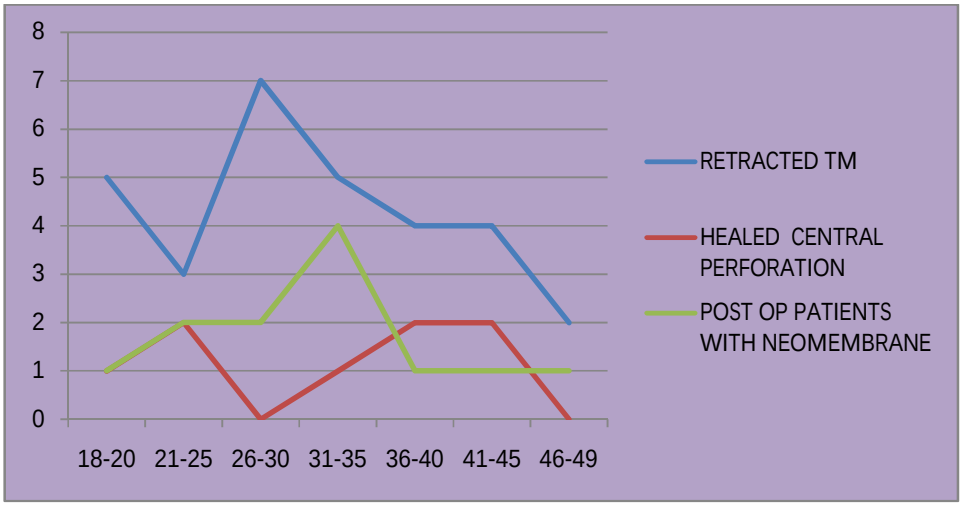


TABLE 4 : Sex distribution of patients with retracted tympanic membrane ,patients with healed central perforation and post operative patients with neomembrane

Sex	Retracted tympanic membrane	Healed central perforation	Post operative patients with neomembrane
Male	16	4	6
Female	14	4	6
Total	30	8	12

Bar Diagram Showing Sex distribution of patients with retracted tympanic membrane ,patients with healed central perforation and post operative patients with neomembrane

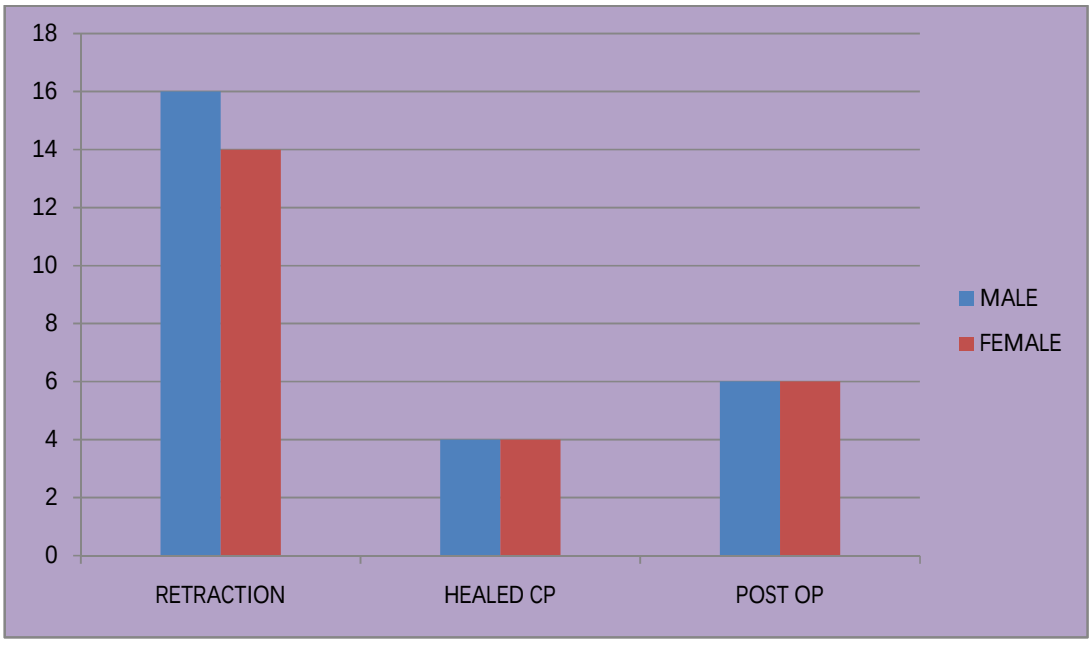


TABLE 5: TYMPANIC MEMBRANE STATUS

Tympanic Membrane Status	No.of patients
Central perforation	50
Healed Central perforation	8
Post op neomembrane	12
Retraction (grade 1 & 2)	30

Pie Chart Depicting The Tympanic Membrane Status In Patients With Middle Ear Disease

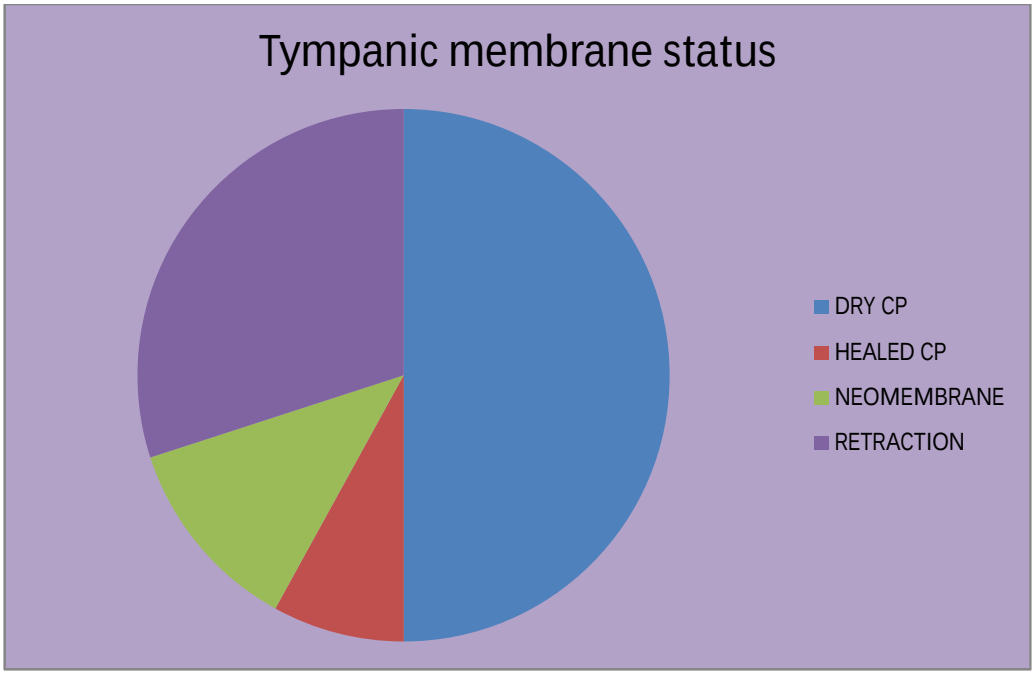


Table 6 : Surgical Status Of Patients With Chronic Otitis Media

Sl.no	Patients with COM	No of patients
1	Non Operated	43
2	Operated With Residual Central perforation Or Recurrent Disease	7

Pie chart showing surgical status of patients with chronic otitis media

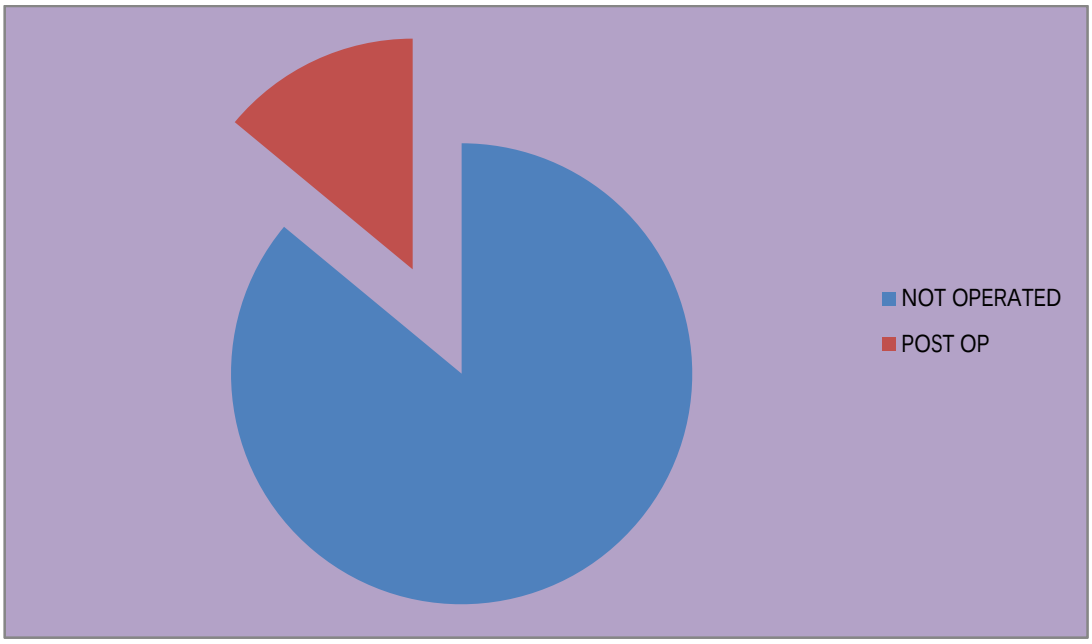


Table 7 : Eustachian tube function in patients with CSOM

		NORMAL TUBAL FUNCTION	PARTIAL ET DYSFUNCTION	GROSS TUBAL DYSFUNCTION
PATIENTS WITH DRY CP (NOT OPERATED)	43	17	14	12
POST OP PATIENTS WITH RESIDUAL CP	7	1	3	3
TOTAL	50	18	17	15

Bar diagram showing Eustachian tube function in patients with CSOM

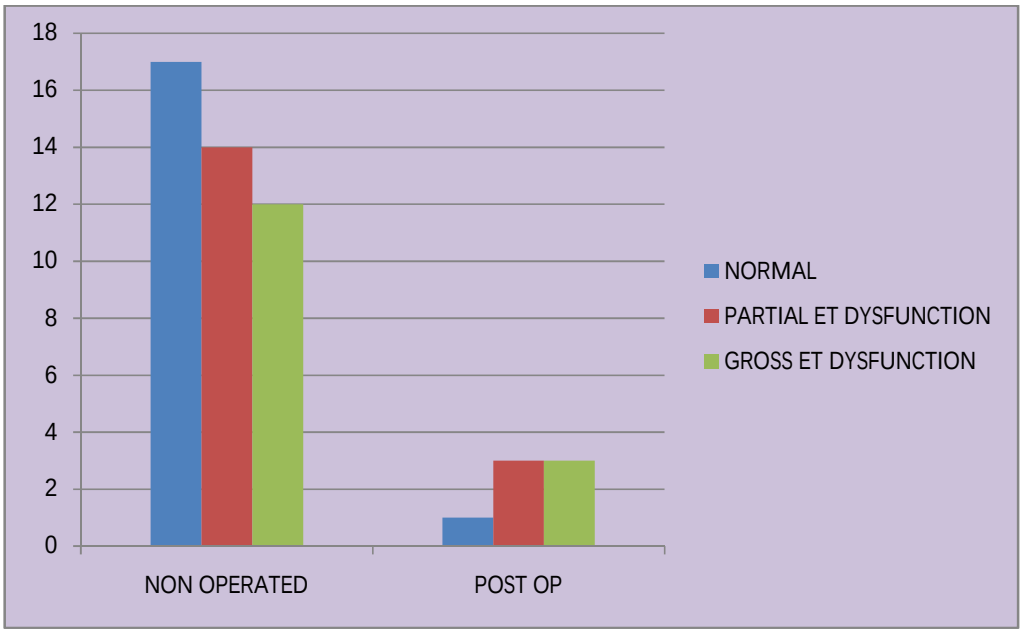


TABLE 8 Normal Adults Vs Patients With Dry Central Perforation

	TOTAL	Normal ETF	Eustachian tube dysfunction	Percentage (normal ETF)
NORMAL ADULTS	50	49	1	98%
PATIENTS WITH DRY CP	50	18	32	36 %

Chi-square with Yates correction

Chi squared equals 40.706 with 1 degrees of freedom. The two-tailed P value is less than 0.0001 . The association between COM and ETF is considered to be extremely statistically significant.

Bar Diagram comparing the Eustachain tube function of normal adults with CSOM patients with dry Central perforation

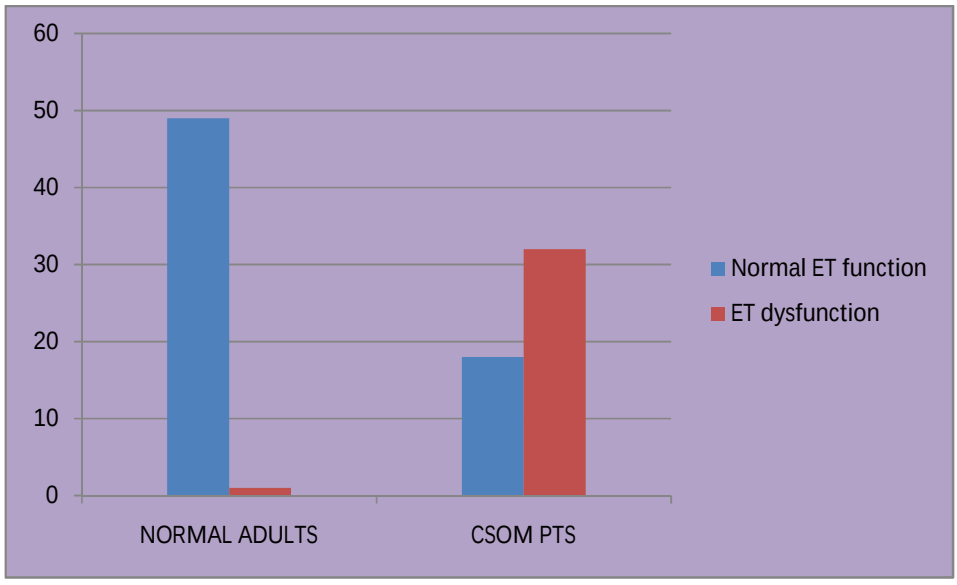


TABLE 9 : Eustachian tube function in patients with Retracted Tympanic membrane, Healed Central perforation, Post op patients

Sl.no	Patients	No of pts	Normal ETF	Eustachian tube dysfunction		Percentage (normal ETF)
				Partial	gross	
1	Retracted tympanic membrane	30	3	21	6	10.0%
2	Healed central perforation	8	7	1		87.5%
3	Postop patients	12	10	2		83.3 %

Bar Diagram showing Eustachian tube function in patients with retracted Tympanic membrane,healed Central perforation,post op patients

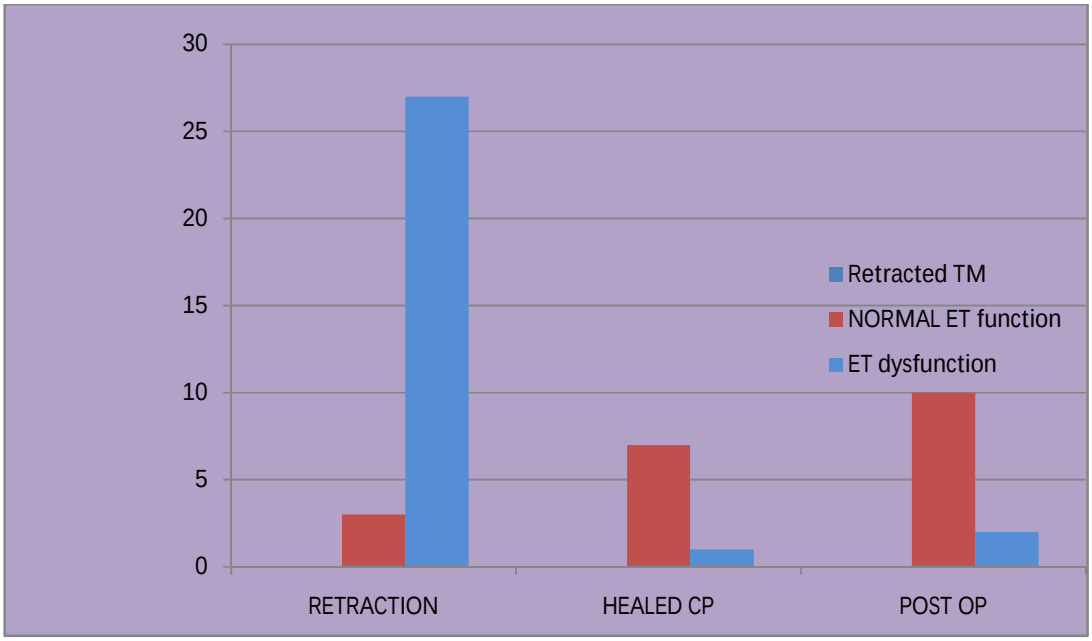


Table 10 : Eustachian tube function in patients with normal ear drum vs retracted membrane

PATIENTS	Normal tubal function	Eustachian tube dysfunction
NORMAL EAR DRUM	49	1
RETRACTED DRUM	3	27

Chi-square with Yates correction - Chi squared equals 60.015 with 1 degrees of freedom. The two-tailed P value is less than 0.0001 . The association between Eustachian tube function and Retraction of tympanic membrane is considered to be extremely statistically significant.

Bar Diagram Showing Eustachian tube function In Patients With Normal Ear Drum Vs Retracted Membrane

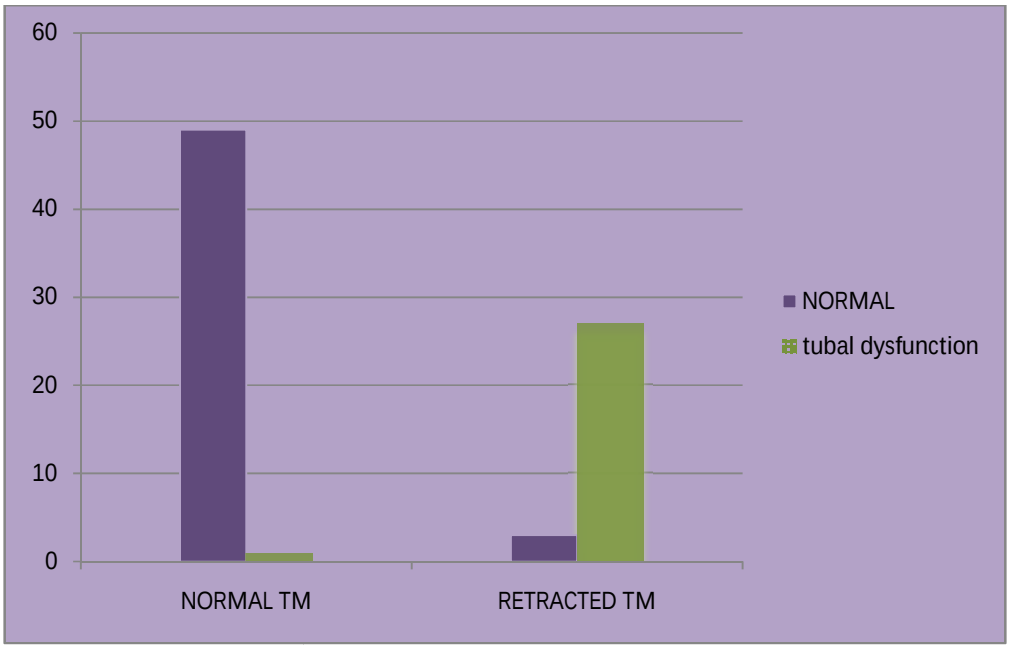


TABLE 11: Eustachian tube function in patients with recurrent CSOM vs patients with healed central perforation

PATIENTS	Normal Eustachian tube function	Eustachian tube dysfunction
RECURRENT CSOM	1	6
HEALED CP	7	1

Chi-square with Yates correction - Chi Squared equals 5.368 with 1 degrees of freedom. The two-tailed P value equals 0.0205 .The association between healing of central perforation and Eustachian tube function is considered to be statistically significant.

Bar diagram showing Eustachian tube function in patients with recurrent CSOM vs patients with healed central perforation

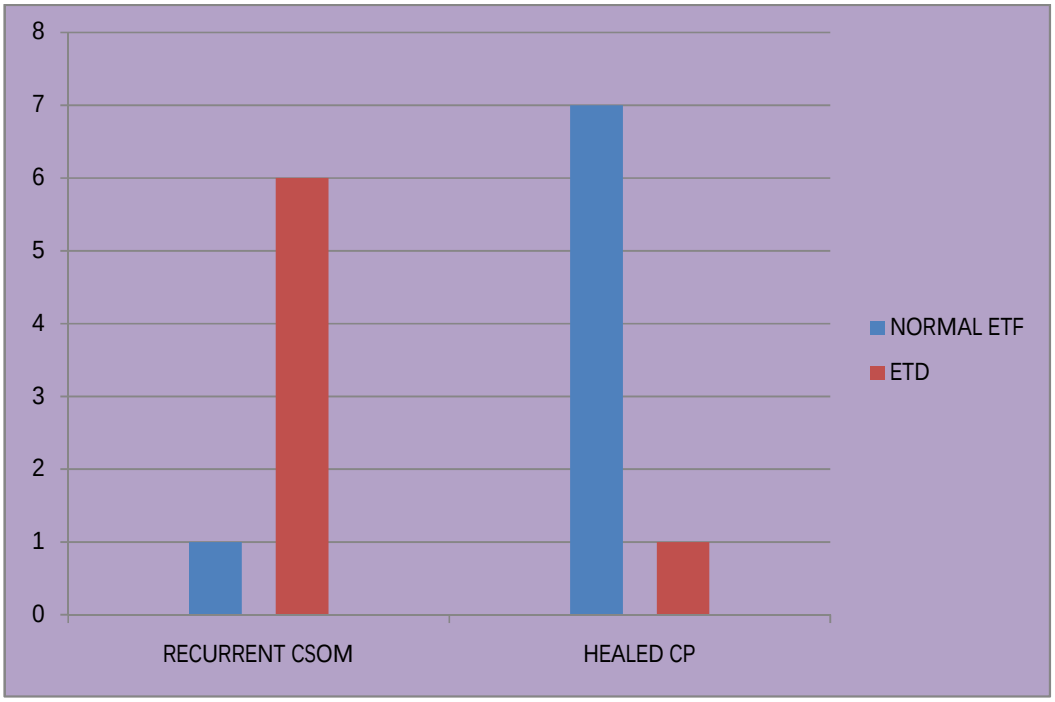
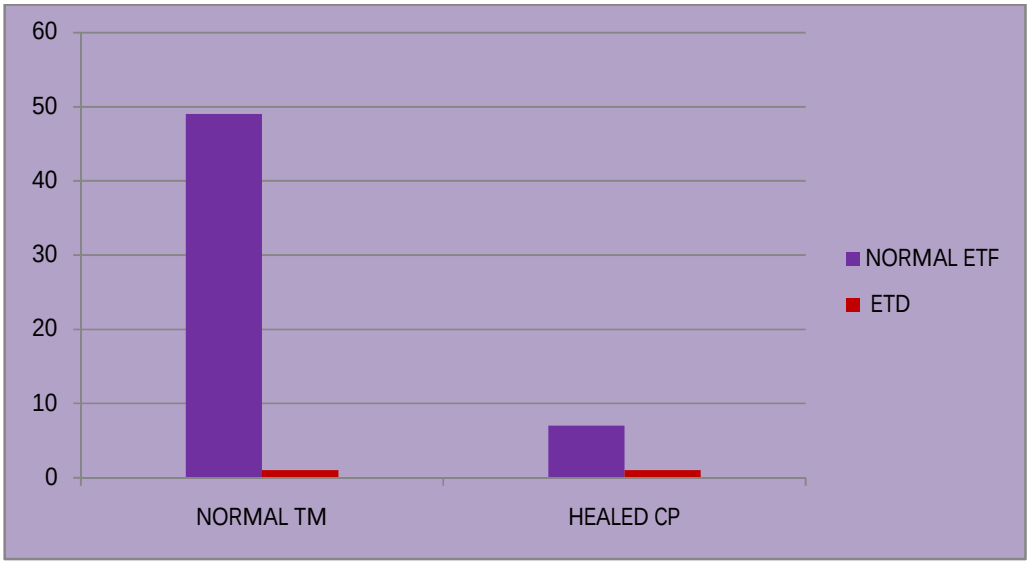


TABLE 12 : Eustachian tube function in normal tympanic membrane vs patients with healed central perforation

	Normal Eustachian tube function	Eustachian tube dysfunction
Normal Tympanic Membrane	**49**	**1**
Healed Central Perforation	**7**	**1**

Chi-square with Yates correction - Chi squared equals 0.219 with 1 degrees of freedom. The two-tailed P value equals 0.6400 . The p value is considered to be not statistically significant suggesting that the ETF in normal adults & patients with healed central perforation is similar.

Bar Diagram Showing Eustachian tube function In Normal Tymapnic membrane Vs Healed Central perforation



DISCUSSION

Eustachian tube function testing can be important tool for improving the success rate of tympanoplasty surgery. Earlier more importance was given for testing the anatomical patency of Eustachian tube. Merely testing the patency does not serve the purpose of identifying the physiological functioning of the tube. Otologists have started to test the functioning of Eustachian tube by testing the ability of the tube to regulate middle ear pressure. New impedence audiometers serve this purpose. In our study we used AMPLAID Audiometer for testing ETF in perforated drum and MAICO 34 for testing ETF in intact tympanic membrane.

Two tests were used. William's test for patients with intact tympanic membrane and Toynbee's test for patients with perforation.

Elner et al in 1976 classified patients into four groups based on their ability to equilibrate middle ear pressure.

Group 1 – ability to equilibrate both positive and negative pressure completely

Group 2 - ability to equilibrate both positive and negative pressure Partially

Group 3 - ability to equilibrate positive and but unable to equilibrate negative pressure

Group 4 –could not equilibrate both positive & negative

Cantekin et al[86] in 1979 studied the Eustachian tube function in 6 normal adults with traumatic perforation and in 5 adults with chronic perforation. He used inflation –deflation and forced response test for assessing ETF. Eustachian tube function was abnormal in chronic otitis media patients whereas it was normal in patients with traumatic perforation. In our study only 1 had partial tubal dysfunction out of 50 normal adults whereas 32 out of 50 CSOM patients had tubal dysfunction .There was no significant age or gender difference in tubal dysfunction identified.17 male (53%) and 15 female (47%) had tubal dysfunction in our study.

Cohn et al .[87] in 1979 tested tubal function by impedence audiometer in tympanoplasty patients. Success rate of surgery was only 69% in patients with gross tubal dysfunction and 75% in patients with partial tubal dysfunction. Patients with normal ETF had graft uptake of 95%.

Sen et al[88] in 1998 also used similar method of Toynbee's test in perforated drum. Success rate of surgery was only 66% in patients with gross tubal dysfunction and 80% in patients with partial tubal dysfunction.

El –Guindy in 1993 had a success rate of 95% in patients with normal ETF. Success rate was 90% if the tubal pathology was corrected before tympanoplasty. The rate was only 68% in patients who tubal dysfunction and was not corrected before tympanoplasty.

Anirban Biswas[89] in 1999 tested ETF using William's test in 34 post operative cases who were successfully treated by myringoplasty or type 1 typanoplasty. Out of 34 patients, 26 had perfectly normal function, 6 patients had partial impairment while only 2 had gross impairment. It compares with our study where only 2 out of 12 patients had partial dysfunction. Anirban Biswas tested ET function using Toynbee's test in 83 patients with failed tympanoplasty . 83% of the patients had tubal dysfunction in our study, we did ET function test for 7 patients with failed tympanoplasty. Out of 7 patients, only 1 patient had normal function, 3 had partial dysfunction and 3 had gross impairment (85.7 %) suggesting that ET dysfunction is an important cause for failure of surgery.

Shreyas s.joshi et al in 2012 and Priya et al in 2012 also came up similar results when used tympanometry as a prognostic indicator of myringoplasty and tympanoplasty.

In our study we had similar results .While the tubal function was normal in 7 of the 8 patients with healed perforation, Eustachian Tube function was impaired in 6 of the 7 patients with failed tympanoplasty. Eustachian Tube function was normal in 10 out of 12 patients with successful tympanoplasty.

SUMMARY

In our study, 50 normal adults and 50 Chronic otitis media patients with dry central perforation were tested for Eustachian tube function. In normal adults out of 50 only 1 had partial tubal dysfunction. In 50 Chronic otitis media patients with dry central perforation, 32 patients (64%) had Eustachian tube dysfunction and only 18 patients (36%) had normal function. On comparing Eustachian tube function in normal and Chronic otitis media patients, Eustachian tube dysfunction in Chronic otitis media patients is found to be statistically significant.

As a part of the study eustachian tube function was also evaluated in patients with retracted tympanic membrane ,healed central perforation and post operative patients who underwent myringoplasty or type 1 tympanoplasty 7 out of 8 [87.5%] patients with healed central perforation and 10 out 12 [83%] post tympanoplasty patients had normal tubal function suggesting that normal functioning of Eustachian tube function plays a major role in healing of the perforation and success of Tympanoplasty.

In 30 patients with Grade I & II retraction, Eustachian tube dysfunction was found in 27 patients. On comparing it with normal adults, association of tubal dysfunction with retraction was found to be statistically significant. There was no significant age or gender difference identified in patients with Eustachian tube dysfunction.

CONCLUSION

From our study it is evident that Eustachian tube dysfunction is an important factor that determines the outcome of chronic otitis media. In most patients with healed central perforation and in post operative patients who were successfully treated by surgery, the Eustachian tube function was found to be normal. ET function was found to be partially or grossly impaired in most patients with recurrent or residual CP. In most of the CSOM patients and patients with retracted ear drum also the impairment of Eustachian tube function was found to be statistically significant. Chronic sinusitis, allergic rhinitis and smoking were the important causes of ET dysfunction.

Mucosal disease is the leading cause of ET dysfunction and a careful search for the underlying aetiology can be effective in directing specific treatment to resolve or mediate the ear sequelae. When medical treatment is inadequate, surgical intervention may be indicated. Tympanostomy tubes have a long proven record of efficacy but repeated insertion may lead to tympanic membrane pathology. Adenoidectomy may be effective, especially in children or in adults with significant hypertrophy compromising the tubal dilation. Eustachian tuboplasty is a new treatment to widen the tubal orifice and aid the valve in dilation

when medical and conventional surgical procedures have proven inadequate. But the method of Eustachian tuboplasty is yet to be standardized. Further studies are needed before it finds universal acceptance.

Eustachian tube function testing by impedance audiometry is a simple non-invasive method of testing the Eustachian tube function and helps in identifying the tubal dysfunction. Patients with tubal dysfunction should be evaluated for underlying cause and treatable causes should addressed before proceeding for surgery as it increases the success rate of tympanoplasty.

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PROFORMA

NAME :

AGE :

SEX:

OCCUPATION:

ADDRESS:

SOCIOECONOMIC STATUS:

HOSPITAL OP/IP NUMBER:

PRESENTING COMPLAINTS :

1. EAR BLOCK

- SIDE
- DURATION
- ONSET
- AGGRAVATING / RELIEVING FACTORS

2.EAR DISCHARGE

- SIDE
- DURATION
- TYPE
- ONSET
- QUANTITY
- SMELL

- AGGRAVATING / RELIEVING FACTORS

3.HARD OF HEARING:

- ONSET
- SIDE
- DURATION
- PROGRESSIVE OR NOT
- FLUCTUATING OR NOT
- HISTORY OF EAR DISCHARGE
- HISTORY OF OTOTOXIC DRUGS
- HISTORY OF TRAUMA
- AUTOPHONY

4.OTALGIA

- SIDE
- DURATION
- ONSET
- AGGRAVATING / RELIEVING FACTORS

ASSOCIATED SYMPTOMS

- TINNITUS
- VERTIGO
- HEAD ACHE

- NASAL OBSTRUCTION
- POST NASAL DISCHARGE
- RECURRENT ATTACKS OF UPPER RESPIRATORY TRACT INFECTIONS

PAST HISTORY

- ALLERGY
- ASTHMA
- TRAUMA
- OTOTOXIC DRUGS
- PREVIOUS EAR SURGERY
- IRRADIATION
- HYPERTENSION
- DIABETES
- PULMONARY TB

PERSONAL HISTORY

- SMOKING
- ALCOHOLISM
- DIET
- BOWEL AND BLADDER HABITS

FAMILY HISTORY

- HARD OF HEARING
- HISTORY OF CONSANGUINOUS MARRIAGE

SOCIO ECONOMIC HISTORY

GENERAL EXAMINATION

TEMPERATURE

PULSE

BLOOD PRESSURE

PALLOR

ICTERUS

CLUBBING

CYANOSIS

EDEMA

GENERALISED LYMPHADENOPATHY

SYSTEMIC EXAMINATION

- CARDIOVASCULAR SYSTEM
- RESPIRATORY SYSTEM
- CENTRAL NERVOUS SYSTEM
- GASTROINTESTINAL SYSTEM

LOCAL EXAMINATION

EXAMINATION OF EAR : RIGHT LEFT

PINNA

PRE AURICULAR REGION

POST AURICULAR REGION

EXTERNAL AUDITORY CANAL

MASTOID REGION

TRAGAL SIGN

TYMPANIC MEMBRANE

- PARS TENSA
- PARS FLACCIDA
- HANDLE OF MALLEUS
- COLOUR
- CONE OF LIGHT
- RETRACTED OR NOT
- MOBILITY

TUNING FORK TESTS

- RINNE TEST
- WEBER TEST
- ABSOLUTE BONE CONDUCTION

THREE FINGER TEST

FACIAL NERVE

FISTULA SIGN

VESTIBULAR FUNCTION TESTS

EXAMINATION OF NOSE

- ANTERIOR RHINOSCOPY
- POST NASAL EXAMINATION

EXAMINATION OF THROAT

INVESTIGATIONS

Pure Tone Audiometry

Impedence Audiometry

Video otoscopy

Diagnostic Nasal Endoscopy

Pneumatic otoscopy

William's test in intact tympanic membrane

Toynbee's test in perforated ear drum

CT Scan of paranasal sinuses (in selected cases)

MASTER CHART – CONTROL GROUP (NORMAL ADULTS)

				HISTORY					TM			PTA			ETF		
S.No.	Name	Age	Sex	E B	E D	HOH	SURG	EAR	OTOSCOPY	MO	ETO	RT	LT	IMP	NL	PD	GD
1	BHUVANESWARI	24	F	-	-	-	-	BOTH	NL	+	NL	13	16	A	+	-	-
2	RAJENDRAN	29	M	-	-	-	-	BOTH	NL	+	NL	16	17	A	+	-	-
3	KAVITHA	23	F	-	-	-	-	BOTH	NL	+	NL	13	13	A	+	-	-
4	HARINI	19	F	-	-	-	-	BOTH	NL	+	NL	18	18	A	+	-	-
5	SURYA	31	F	-	-	-	-	BOTH	NL	+	NL	16	17	A	+	-	-
6	TINU MARIYA	22	F	-	-	-	-	BOTH	NL	+	NL	17	15	A	+	-	-
7	VELUSAMY	26	M	-	-	-	-	BOTH	NL	+	NL	13	13	A	+	-	-
8	PALANIYAPPAN	34	M	-	-	-	-	BOTH	NL	+	NL	15	15	A	+	-	-
9	BALAKRISHNAN	19	M	-	-	-	-	BOTH	NL	+	NL	13	15	A	+	-	-
10	KALAVATHI	25	F	-	-	-	-	BOTH	NL	+	NL	15	18	A	+	-	-
11	AKBAR	27	M	-	-	-	-	BOTH	NL	+	NL	13	15	A	+	-	-
12	SAKTHEESH	24	M	-	-	-	-	BOTH	NL	+	NL	17	16	A	+	-	-
13	ANNE MARIA	23	F	-	-	-	-	BOTH	NL	+	NL	16	16	A	+	-	-
14	PADMA	41	F	-	-	-	-	BOTH	NL	+	NL	13	13	A	+	-	-
15	SYED MOHAMMED	18	M	-	-	-	-	BOTH	NL	+	NL	15	13	A	+	-	-
16	ANGELINE	36	F	-	-	-	-	BOTH	NL	+	NL	13	13	A	+	-	-
17	BETHU	25	M	-	-	-	-	BOTH	NL	+	NL	13	15	A	+	-	-
18	RANJITH	38	M	-	-	-	-	BOTH	NL	+	NL	17	18	A	+	-	-
19	PRIYARANJINI	20	F	-	-	-	-	BOTH	NL	+	NL	16	17	A	+	-	-
20	SHAHUL HAMEED	24	M	-	-	-	-	BOTH	NL	+	NL	17	18	A	+	-	-
21	RAMATHAL	23	F	-	-	-	-	BOTH	NL	+	NL	16	17	A	+	-	-
22	CHITHRA	43	F	-	-	-	-	BOTH	NL	+	NL	16	13	A	+	-	-
23	SARAVANAKUMAR	33	M	-	-	-	-	BOTH	NL	+	NL	13	16	A	+	-	-
24	ANEESH	27	M	-	-	-	-	BOTH	NL	+	NL	17	16	A	+	-	-
25	JOSEPH	42	M	-	-	-	-	BOTH	NL	+	NL	16	16	A	+	-	-

MASTER CHART – CONTROL GROUP (NORMAL ADULTS)

				HISTORY					TM			PTA			ETF		
S.No.	Name	Age	Sex	E B	E D	HOH	SURG	EAR	OTOSCOPY	MO	ETO	RT	LT	IMP	NL	PD	GD
26	DEVIKA	44	F	-	-	-	-	BOTH	NL	+	NL	13	13	A	+	-	-
27	RAMYA	31	F	-	-	-	-	BOTH	NL	+	NL	15	16	A	+	-	-
28	KARAMATH	24	M	-	-	-	-	BOTH	NL	+	NL	18	22	C	-	+	-
29	JANARTHAN	47	M	-	-	-	-	BOTH	NL	+	NL	15	15	A	+	-	-
30	KRITHIKA	22	F	-	-	-	-	BOTH	NL	+	NL	12	15	A	+	-	-
31	MADHU	35	M	-	-	-	-	BOTH	NL	+	NL	15	13	A	+	-	-
32	SANJANA	28	F	-	-	-	-	BOTH	NL	+	NL	18	18	A	+	-	-
33	MARUTHU	37	M	-	-	-	-	BOTH	NL	+	NL	16	17	A	+	-	-
34	ELANGOVAN	29	M	-	-	-	-	BOTH	NL	+	NL	16	16	A	+	-	-
35	ELIZABETH	34	F	-	-	-	-	BOTH	NL	+	NL	13	16	A	+	-	-
36	MANIKANDAN	36	M	-	-	-	-	BOTH	NL	+	NL	17	16	A	+	-	-
37	KANNAN	26	M	-	-	-	-	BOTH	NL	+	NL	16	13	A	+	-	-
38	MARIMUTHU	33	M	-	-	-	-	BOTH	NL	+	NL	13	15	A	+	-	-
39	DIVYA	26	F	-	-	-	-	BOTH	NL	+	NL	18	18	A	+	-	-
40	GURUMANI	38	M	-	-	-	-	BOTH	NL	+	NL	16	13	A	+	-	-
41	SHAKTHIPRIYA	27	F	-	-	-	-	BOTH	NL	+	NL	15	16	A	+	-	-
42	ANUPRIYA	35	F	-	-	-	-	BOTH	NL	+	NL	17	18	A	+	-	-
43	SARASWATHY	29	F	-	-	-	-	BOTH	NL	+	NL	18	18	A	+	-	-
44	MOHAMED ISMAIL	48	M	-	-	-	-	BOTH	NL	+	NL	16	15	A	+	-	-
45	CHELLAKAVITHA	26	F	-	-	-	-	BOTH	NL	+	NL	12	13	A	+	-	-
46	VIJAYALAKSHMI	40	F	-	-	-	-	BOTH	NL	+	NL	13	15	A	+	-	-
47	HARISH	30	M	-	-	-	-	BOTH	NL	+	NL	15	16	A	+	-	-
48	DHANALAKSHMI	45	F	-	-	-	-	BOTH	NL	+	NL	16	16	A	+	-	-
49	CHELLAMUTHU	43	M	-	-	-	-	BOTH	NL	+	NL	18	17	A	+	-	-
50	GNANASEKAR	41	M	-	-	-		BOTH	NL	+	NL	15	15	A	+	-	-

MASTER CHART – STUDY GROUP (PATIENTS WITH COM)

				HISTORY					TM			PTA			ETF		
S.No.	Name	Age	Sex	E B	E D	HOH	SURG	EAR	OTOSCOPY	MO	ETO	RT	LT	IMP	NL	PD	GD
1	ANJUGAM	46	F	-	-	+	-	RT	CP	NA	NL	33	18	B	+	-	-
2	MARIYA	31	F	+	-	-	-	RT	CP	NA	NL	28	15	B	-	+	-
3	RAMAKRISHNAN	41	M	-	-	+	-	LT	CP	NA	NL	16	36	B	-	+	-
4	CHELLAKAVITHA	27	F	-	-	+	-	RT	CP	NA	NR	43	16	B	-	-	+
5	DURAI	21	M	+	-	-	-	LT	CP	NA	NL	13	23	B	-	+	-
6	VINEETA	33	F	-	-	+	-	RT	CP	NA	NL	33	16	B	+	-	-
7	RADIKA	36	F	-	-	+	-	LT	CP	NA	NL	13	38	B	+	-	-
8	THANGAPPAN	47	M	-	-	+	-	RT	CP	NA	NL	33	17	B	-	+	-
9	LORETTA	21	F	-	-	+	-	LT	CP	NA	NL	13	36	B	-	-	+
10	MUKESH	26	M	+	-	-	-	RT	CP	NA	NL	26	15	B	+	-	-
11	THAMARAI	34	F	-	-	+	-	LT	CP	NA	NL	36	38	B	-	+	-
12	ABU THAKEER	42	M	-	-	+	-	RT	CP	NA	NR	43	36	B	-	-	+
13	VETRISELVI	36	F	-	-	+	-	RT	CP	NA	NL	42	18	B	-	+	-
14	FATHIMA	49	F	-	-	+	-	LT	CP	NA	NL	15	35	B	-	+	-
15	VIGNESH	23	M	+	-	-	-	LT	CP	NA	NL	16	30	B	-	-	+
16	SURESH	28	M	-	-	+	-	LT	CP	NA	NL	16	43	B	-	+	-
17	SRIDIVYA	18	F	+	-	+	-	RT	CP	NA	NL	36	18	B	-	+	-
18	SETHUPATHI	22	M	-	-	+	-	LT	CP	NA	NL	16	53	B	+	-	-
19	ULAGANATHAN	35	M	-	-	+	-	RT	CP	NA	NL	46	38	B	-	+	-
20	VEERALAKSHMI	44	F	+	-	-	-	RT	CP	NA	NL	31	15	B	-	+	-
21	PUSHPARAJ	19	M	+	-	-	-	LT	CP	NA	NR	13	28	B	-	-	+
22	SALEEM	37	M	-	-	+	-	RT	CP	NA	NL	35	16	B	-	-	+
23	MOHAN	33	M	-	-	+	-	RT	CP	NA	NL	33	18	B	+	-	-
24	SUNDARAM	43	M	-	-	+	-	LT	CP	NA	NL	18	45	B	+	-	-
25	THIYAGARAJAN	40	M	-	-	+	-	RT	CP	NA	NL	46	36	B	+	-	-

MASTER CHART – STUDY GROUP (PATIENTS WITH COM)

				HISTORY					TM			PTA			ETF		
S.No.	Name	Age	Sex	E B	E D	HOH	SURG	EAR	OTOSCOPY	MO	ETO	RT	LT	IMP	NL	PD	GD
26	HARIHARAN	40	M	+	-	-	-	RT	CP	NA	NL	26	18	B	+	-	-
27	KALAIYARASU	19	M	-	-	+	-	LT	CP	NA	NL	15	33	B	-	-	+
28	ALAGAMMAL	41	F	-	-	+	-	LT	CP	NA	NL	13	36	B	-	-	+
29	KRISHNAN	37	M	-	-	+	-	RT	CP	NA	NL	43	15	B	-	+	-
30	SAMYUKTHA	27	F	-	-	+	-	RT	CP	NA	NL	45	18	B	+	-	-
31	ANURADHA	45	F	+	-	-	-	LT	CP	NA	NL	16	31	B	-	+	-
32	SIVA	23	M	-	-	+	-	RT	CP	NA	NL	43	38	B	+	-	-
33	KUPPUSAMY	39	M	-	-	+	-	RT	CP	NA	NL	38	16	B	+	-	-
34	SIMIONE	30	M	-	-	+	-	RT	CP	NA	NL	36	33	B	-	+	-
35	ILAVARASI	44	F	-	-	+	-	LT	CP	NA	NR	15	54	B	-	-	+
36	VASANTHI	47	F	-	-	+	-	RT	CP	NA	NL	46	15	B	+	-	-
37	MUTHUKUMAR	26	M	+	-	-	-	LT	CP	NA	NL	13	28	B	+	-	-
38	SENTHIL	37	M	-	-	+	-	RT	CP	NA	NL	31	31	B	-	-	+
39	MOHAIDEEN	31	M	-	-	+	-	LT	CP	NA	NL	33	36	B	+	-	-
40	KALARATHI	36	F	-	-	+	-	RT	CP	NA	NR	40	18	B	-	-	+
41	SELVAPEUMAL	18	M	-	-	+	-	LT	CP	NA	NL	13	32	B	+	-	-
42	ALVIN	22	M	-	-	+	-	LT	CP	NA	NL	16	51	B	-	-	+
43	SUGANTHI	34	F	-	-	+	-	LT	CP	NA	NL	13	35	B	+	-	-
44	VINOD	43	M	+	-	-	+	RT	CP	NA	NL	30	18	B	-	+	-
45	ANWAR	40	M	-	-	+	+	LT	CP	NA	NL	16	36	B	+	-	-
46	SHAKILA	35	F	-	-	+	+	LT	CP	NA	NL	15	41	B	-	-	+
47	ARUN KUMAR	38	M	-	-	+	+	RT	CP	NA	NL	38	18	B	-	-	+
48	VISHALI	24	F	+	-	-	+	LT	CP	NA	NL	16	30	B	-	+	-
49	DEVASAGAYAM	36	M	-	-	+	+	RT	CP	NA	NL	43	38	B	-	-	+
50	JAYANTHI	29	F	-	-	+	+	LT	CP	NA	NL	33	33	B	-	+	-

MASTER CHART - PATIENTS WITH RETRACTED TM,HEALED CP,POST OP PATIENTS

				HISTORY					TM			PTA			ETF		
S.No.	Name	Age	Sex	E B	E D	HOH	SURG	EAR	OTOSCOPY	MO	ETO	RT	LT	IMP	NL	PD	GD
1	PUGAZHENTHI	19	M	+	-	-	-	RT	RETRACTED	+	NL	18	20	C	-	+	-
2	ANBARASAN	31	M	+	-	-	-	LT	RETRACTED	+	NL	16	19	C	-	+	-
3	VIMALARANI	42	F	+	-	-	-	RT	RETRACTED	+	NL	20	18	A	+	-	-
4	AVIN KUMAR	20	M	+	-	-	-	LT	RETRACTED	+	NR	15	19	C	-	-	+
5	LEELAVATHI	26	F	-	-	+	-	BOTH	RETRACTED	+	NL	28	26	C	-	+	-
6	DHANYA JOBY	27	F	+	-	-	-	RT	RETRACTED	+	NR	20	18	C	-	-	+
7	SELVAKUMAR	43	M	+	-	-	-	LT	RETRACTED	+	NL	20	21	C	-	+	-
8	VENKATESH	38	M	-	-	+	-	BOTH	RETRACTED	+	NL	21	24	A	+	-	-
9	SADHANA	34	F	+	-	-	-	RT	RETRACTED	+	NL	20	16	C	-	+	-
10	BHARATHI	30	M	+	-	-	-	LT	RETRACTED	+	NL	15	21	C	-	+	-
11	SUJITHA	23	F	+	-	-	-	BOTH	RETRACTED	+	NL	23	21	C	-	-	+
12	GREESHMA	27	M	+	-	-	-	RT	RETRACTED	+	NL	20	16	C	-	+	-
13	SUDHARSHAN	33	M	+	-	+	-	LT	RETRACTED	+	NL	18	30	C	-	+	-
14	AISHWARYA	18	F	+	-	-	-	RT	RETRACTED	+	NL	18	15	C	-	+	-
15	MANIKANDAN	37	M	+	-	-	-	RT	RETRACTED	+	NL	23	16	A	-	+	-
16	DURGADEVI	37	F	-	-	-	-	LT	RETRACTED	+	NL	16	21	C	+	-	-
17	AMUL RAJULU	43	M	+	-	-	-	RT	RETRACTED	+	NL	18	13	C	-	+	-
18	SOWMIKA	32	F	+	-	-	-	LT	RETRACTED	+	NL	15	15	C	-	+	-
19	GOKUL	29	M	+	-	-	-	RT	RETRACTED	+	NR	23	15	C	-	-	+
20	DHARSHINISHREE	22	F	+	-	-	-	LT	RETRACTED	+	NL	16	20	C	-	+	-
21	SENTHILNATHAN	24	M	+	-	+	-	BOTH	RETRACTED	+	NL	24	26	A	-	-	+
22	PRATUKSHA	18	F	+	-	-	-	LT	RETRACTED	+	NL	16	20	C	-	+	-
23	JENNIFER	49	F	+	-	-	-	RT	RETRACTED	+	NL	18	16	A	-	+	-
24	RAMACHANDRAN	46	M	+	-	-	-	LT	RETRACTED	+	NL	13	21	C	-	+	-
25	LIBINA AUGUSTINE	29	F	+	-	-	-	LT	RETRACTED	+	NL	14	18	A	-	+	-

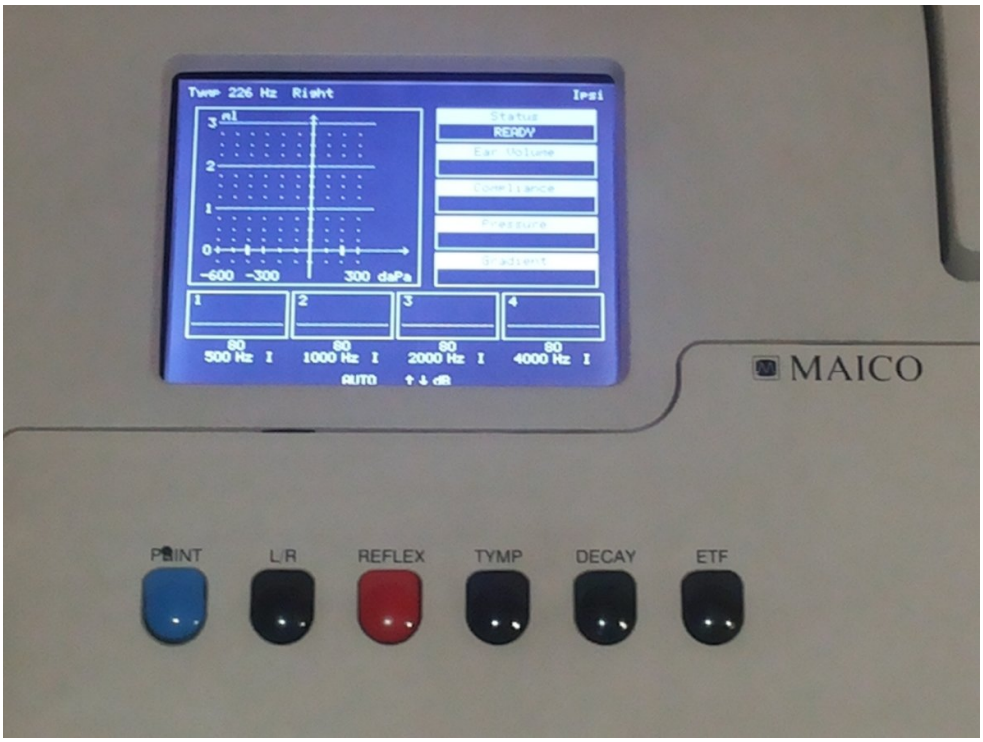
MASTER CHART - PATIENTS WITH RETRACTED TM,HEALED CP,POST OP PATIENTS

				HISTORY					TM			PTA			ETF		
S.No.	Name	Age	Sex	E B	E D	HOH	SURG	EAR	OTOSCOPY	MO	ETO	RT	LT	IMP	NL	PD	GD
26	SHARON	26	F	+	-	-	-	RT	RETRACTED	+	NL	18	16	C	-	+	-
27	MURUGESHAN	41	M	+	-	-	-	LT	RETRACTED	+	NL	15	16	A	-	+	-
28	JENNIFER	37	F	-	-	+	-	BOTH	RETRACTED	+	NR	21	24	C	-	-	+
29	SYED KEZAR	18	M	+	-	-	-	BOTH	RETRACTED	+	NR	18	21	C	-	+	-
30	SIVAKUMAR	35	M	+	-	-	-	RT	RETRACTED	+	NL	19	15	A	-	+	-
1	ANJU GRACE	20	F	-	-	-	-	LT	HEALED CP	+	NL	16	16	A	+	-	-
2	RADHA	25	F	-	-	-	-	RT	HEALED CP	+	NL	15	13	A	+	-	-
3	ARUMUGAM	38	M	+	-	-	-	RT	HEALED CP	+	NL	17	16	C	-	+	-
4	RAMYASHREE	23	F	-	-	-	-	LT	HEALED CP	+	NL	16	16	A	+	-	-
5	PERIYASAMY	45	M	+	-	-	-	RT	HEALED CP	+	NL	23	18	C	+	-	-
6	MALA	42	F	-	-	-	-	LT	HEALED CP	+	NL	16	15	A	+	-	-
7	GURUSAMY	39	M	-	-	-	-	LT	HEALED CP	+	NL	13	15	A	+	-	-
8	BASHEER	33	M	-	-	-	-	RT	HEALED CP	+	NL	17	18	A	+	-	-
1	MATHEW	28	M	-	-	+	+	LT	NEOMEMBRANE	+	NL	35	18	A	+	-	-
2	SHEELA	32	F	+	-	+	+	RT	NEOMEMBRANE	+	NL	21	45	C	-	+	-
3	IRFAN	19	M	-	-	-	+	LT	NEOMEMBRANE	+	NL	16	17	C	+	-	-
4	CHANDRAMOHAN	33	M	+	-	-	+	LT	NEOMEMBRANE	+	NR	18	24	C	-	+	-
5	NISHA KURIAKOSE	32	F	-	-	-	+	LT	NEOMEMBRANE	+	NL	13	13	A	+	-	-
6	DAMAYANTHI	43	F	-	-	+	+	RT	NEOMEMBRANE	+	NL	18	38	A	+	-	-
7	PALANINATHAN	47	M	-	-	-	+	RT	NEOMEMBRANE	+	NL	16	16	A	+	-	-
8	THULASI	38	F	-	-	-	+	LT	NEOMEMBRANE	+	NL	15	18	A	+	-	-
9	PATTUROSE	24	F	-	-	+	+	RT	NEOMEMBRANE	+	NL	18	46	C	+	-	-
10	NAVEEN	29	M	-	-	-	+	LT	NEOMEMBRANE	+	NL	16	18	A	+	-	-
11	JAYAPAL	21	M	-	-	-	+	LT	NEOMEMBRANE	+	NL	15	15	A	+	-	-
12	AARTHI	32	F	-	-	-	+	RT	NEOMEMBRANE	+	NL	18	12	A	+	-	-

KEY TO MASTER CHART

S.NO	Serial Number
M	Male
F	Female
HOH	Hard Of Hearing
EB	Ear Block
ED	Ear Discharge
TM	Tympanic membrane
MO	Mobilty Of Tympanic Membrane
ETO	Eustachian tube opening in nasopharynx
NL	Normal
NR	Narrow
PTA	Pure tone audiogram
RT	Right
LT	Left
IMP	Impedance audiometry
ETF	Eustachian Tube Function
PD	Partial dysfunction
GD	Gross dysfunction

MAICO 34 AUDIOMETER FOR EUSTACHIAN TUBE FUNCTION BY WILLIAM'S TEST

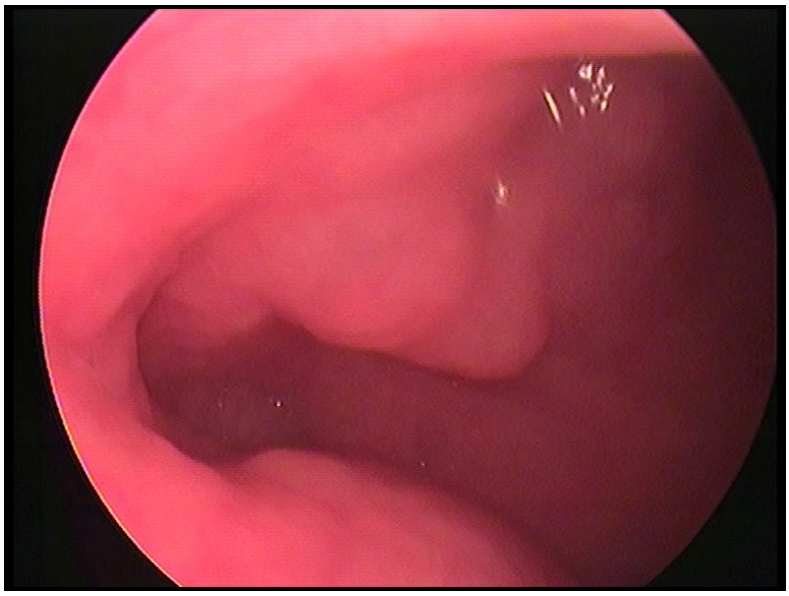


WILLIAM'S TEST MEASURING MIDDLE EAR PRESSURE WHILE THE PATIENT SWALLOWS WITH THE NOSE CLOSED

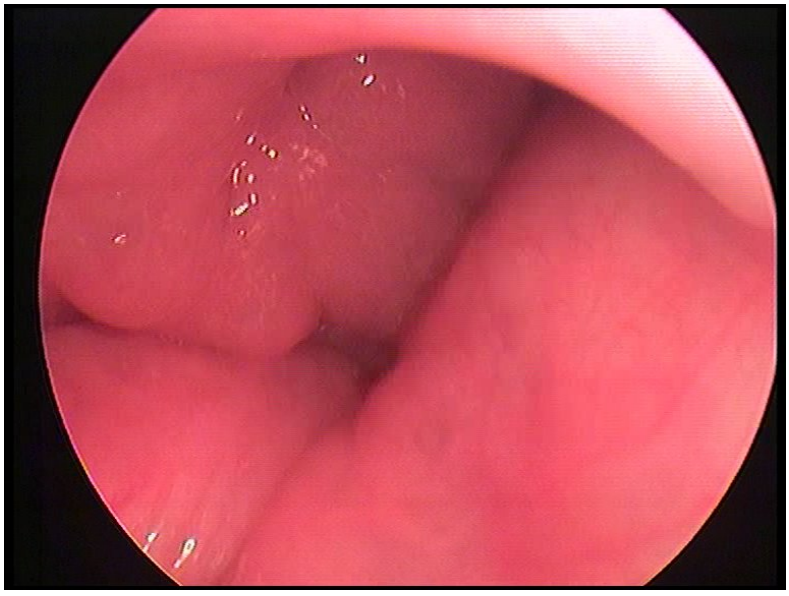
WILLIAM'S TEST MEASURING MIDDLE EAR PRESSURE WHEN THE PATIENT DOES VALSALVA MANOUEVRE

PNEUMATIC OTOSCOPY

RIGHT EUSTACHIAN TUBE OPENING IN OPEN STATE



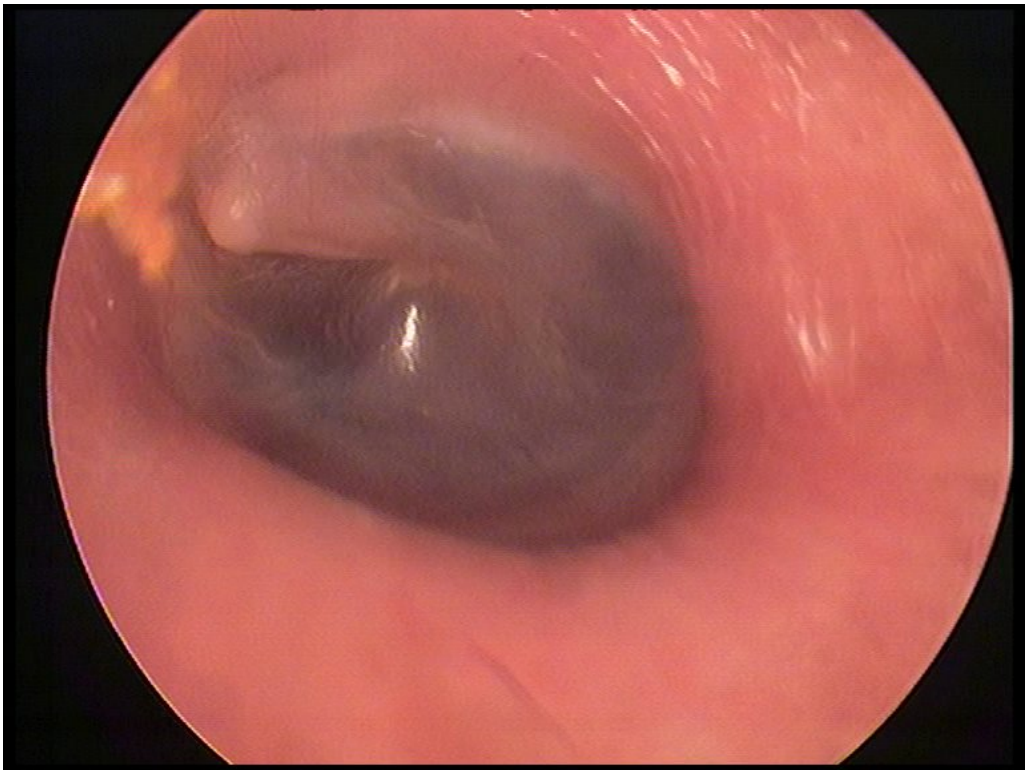
RIGHT EUSTACHIAN TUBE OPENING IN CLOSED STATE



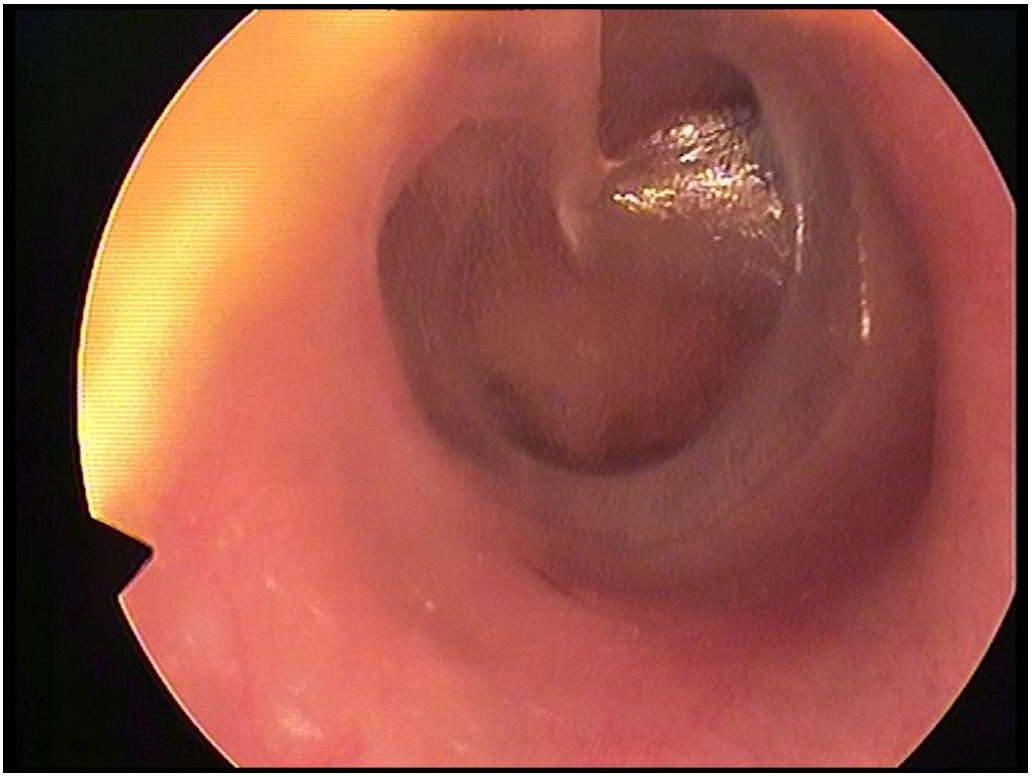
NORMAL RIGHT TYMPANIC MEMBRANE

DRY CENTRAL PERFORATION IN RIGHT CSOM PATIENT

RETRACTED LEFT TYMPANIC MEMBRANE



HEALED CENTRAL PERFORATION - RIGHT TYMPANIC MEMBRANE



TYMPANIC MEMBRANE – POST MYRINGOPLASTY

WILLIAM'S TEST PRINTOUT SHOWING NORMAL EUSTACHIAN TUBE FUNCTION IN BOTH EARS

MAICO MI 34

No. 1015238

Id No.:

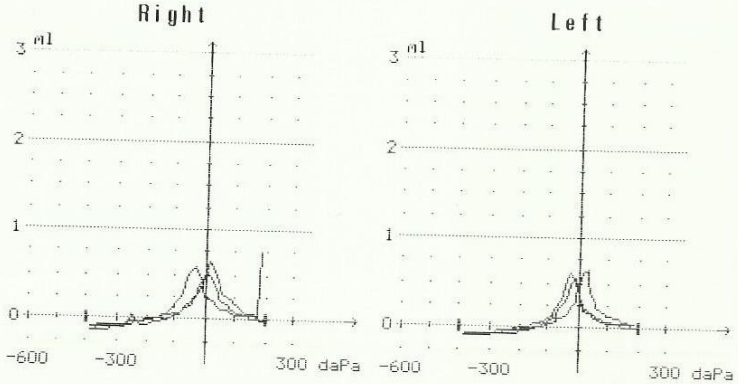
Date: 24/10/13

Name: Miss SURYA C AUDIOLOGIST (HNO)

Examiner:

Remarks:

ETF 1



	Right	Left
Ear Volume	0.88 ml	0.69 ml
Pressure 1	8 daPa	-18 daPa
Pressure 2	-37 daPa	-23 daPa
Pressure 3	0 daPa	24 daPa

WILLIAM'S TEST PRINTOUT SHOWING NORMAL EUSTACHIAN TUBE FUNCTION IN RIGHT EAR AND A FLAT CURVE IN PATIENT WITH LEFT CSOM

Id No.:

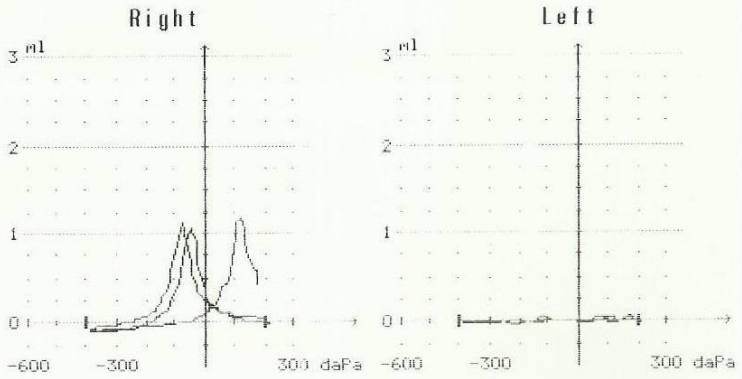
Date: 07.11.13

Name: MR. Balakrishnan

Examiner:

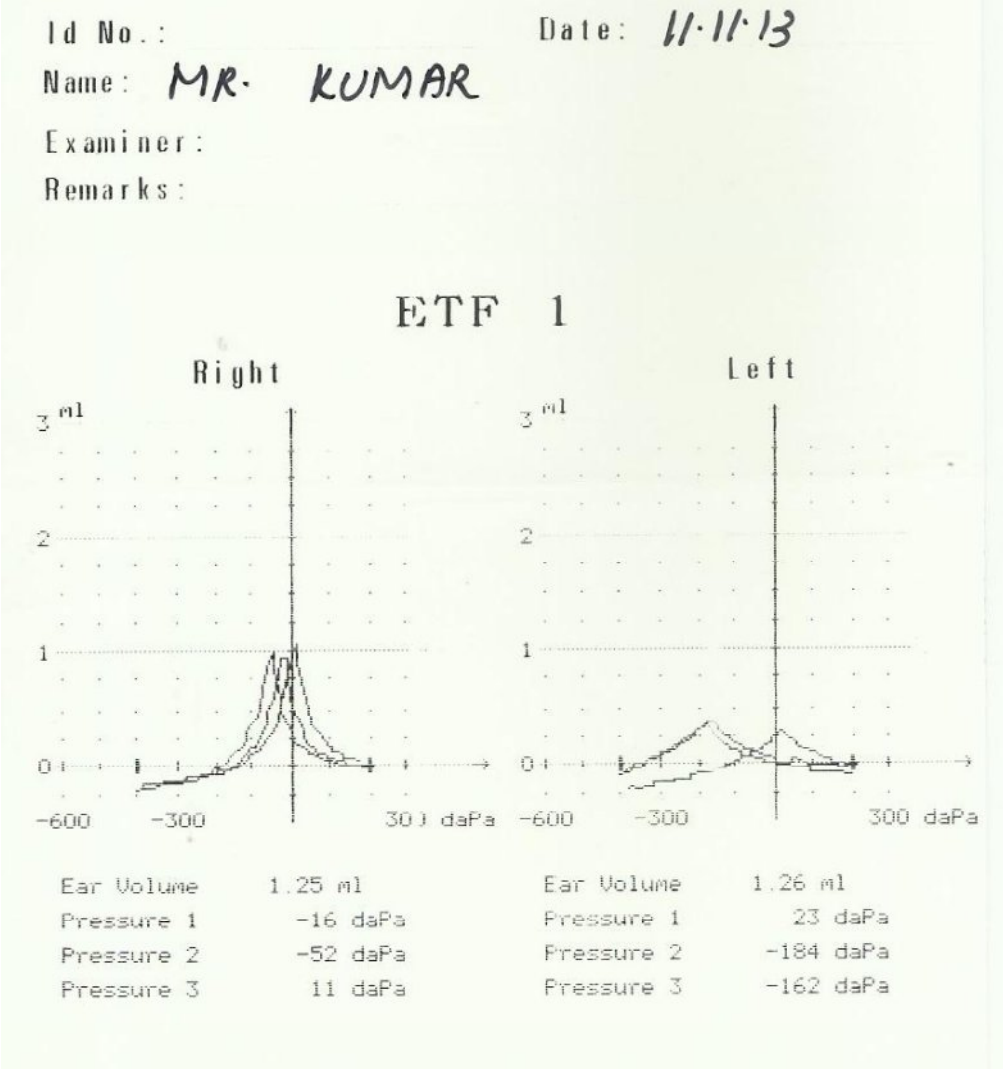
Remarks:

ETF 1

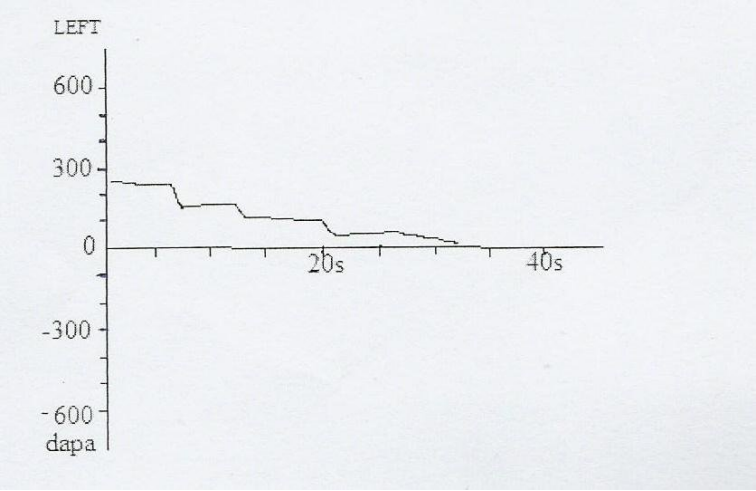


	Right	Left
Ear Volume	0.99 ml	2.44 ml
Pressure 1	-52 daPa	171 daPa
Pressure 2	-81 daPa	106 daPa
Pressure 3	121 daPa	69 daPa

WILLIAM'S TEST PRINTOUT SHOWING PARTIAL EUSTACHIAN TUBE DYSFUNCTION IN LEFT EAR WITH NORMAL FUNCTION IN RIGHT EAR



TOYNBEE TEST PRINTOUT SHOWING NORMAL EUSTACHIAN TUBE FUNCTION IN PATIENT WITH LEFT CSOM



TOYNBEE TEST PRINTOUT SHOWING PARTIAL EUSTACHIAN TUBE DYSFUNCTION IN PATIENT WITH LEFT CSOM