# "CAN INFLATION-DEFLATION TEST PREDICT ADITUS PATENCY IN TUBOTYMPANIC DISEASE? A PROSPECTIVE STUDY"

**By**

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Dissertation submitted to

**SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION & RESEARCH, TAMAKA, KOLAR, KARNATAKA**

in partial fulfilment
of the requirements for the degree of
**MASTER OF SURGERY**

in

**OTORHINOLARYNGOLOGY**

under the guidance of

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I hereby declare that this dissertation entitled

**"CAN INFLATION-DEFLATION TEST PREDICT ADITUS PATENCY IN TUBOTYMPANIC DISEASE? A PROSPECTIVE STUDY"**

is a bonafide and genuine research work carried out by me under the guidance of **DR. VINAY BABU S. MBBS, MS**

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Date: Signature of the Candidate

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## List of abbreviations (in alphabetical order)

| | | |
|---|---|---|
| **1** | **CSOM** | Chronic Suppurative Otitis Media |
| **2** | **CHL** | Conductive Hearing Loss |
| **3** | **EAC** | External Auditory Canal |
| **4** | **ET** | Eustachian tube |
| **5** | **ETF** | Eustachian tube function |
| **6** | **HRCT** | High Resolution Computed Tomography |
| **7** | **IDL** | Indirect Laryngoscopy |
| **8** | **IDT** | Inflation-Deflation Test |
| **9** | **MHL** | Mixed Hearing Loss |
| **10** | **PNS** | Paranasal sinus |
| **11** | **TTD** | Tubotympanic Disease |

# ABSTRACT

## Background and objectives

Chronic suppurative otitis media (CSOM) of tubotympanic type (TTD) is common at R.L.Jalappa Hospital. Most patients are from poor socio-ecomonic background. Nearly, a third of these patients will require surgery as definitive treatment. However, there is no consensus on the surgical options. Currently, TTD is treated by either tympanoplasty alone or tympanoplasty with adjuvant cortical mastoidectomy.

Obstruction of the aditus is cited as the indication for adjuvant cortical mastoidectomy during tympanoplasty. At present, there is no accurate investigation for preoperative evaluation of status of aditus ad antrum. The association between aeration of mastoid, patency of aditus and eustachian tube function (ETF) is well known. Our objective was to determine if Eustachian tube function as assessed by inflation-deflation test (IDT), would help predict aditus patency preoperatively so unnecessary cortical mastoidectomy can be avoided.

## Materials and Methods

This prospective study was undertaken at R.L.Jalappa Hospital, Kolar, between December 2010 and October 2012 on 60 patients with CSOM (TTD) above the age of 12yrs. All patients were subjected to preoperative inflation-deflation test to assess Eustachian tube function. The tubal function was graded as adequate if $\geq$ 50% of applied negative pressure was equilibrated and poor if equilibration was less than 50%.

A standard tympanoplasty with cortical mastoidectomy was done and patency of aditus checked. Results of the IDT were then correlated with the status of aditus intraoperatively.

## Results and Interpretation

80% of our patients showed adequate tubal function on IDT while 20% had poor function. Aditus was found patent in 63.3% of patients and blocked in 36.7%.

Correlation of IDT results with intraoperative status of aditus revealed good correlation in 70% patients: adequate ETF and patent aditus was seen in 56.7% and poor ETF with blocked aditus in the rest (13.3%).

No correlation between IDT and aditus patency was found in 30% with adequate ETF & blocked aditus group comprising 23.3% and poor ETF & patent aditus group contributing to 6.7% of patients. The sensitivity of the IDT in predicting aditus patency was 89.5% but the specificity was low at 36.6%. Positive predictive value of the test was 70.8%, while the negative predictive value was 66.6%.

## Conclusion

It can be concluded from our study that cortical mastoidectomy could probably be avoided when the Eustachian tube function is adequate. But when the Eustachian tube function is poor mastoidectomy would be recommended in order not to miss the small group of patients with blocked aditus.

**Key Words**: **Eustachian tube function, inflation-deflation test, aditus patency**.

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# INTRODUCTION

Chronic suppurative otitis media (CSOM) of tubotympanic type (TTD) is a common presentation at R. L. Jalappa Hospital . Most patients are from poor socio-economic background. Nearly one third of all patients with CSOM will require surgery as a definitive treatment.[1] However, there is no consensus on the surgical options. Currently, TTD is treated by either tympanoplasty alone or tympanoplasty with adjuvant cortical mastoidectomy.[2]

Mastoid antrum and air cells, are a reservoir of gas and act as buffer for pressure changes in the middle ear. Aditus ad antrum connects middle ear with the mastoid. Blockage of aditus by granulation tissue or mucosal swelling impedes ventilation of mastoid air cell system affecting its buffering capacity.[2,3,4] Obstruction of aditus is cited as the justification for an adjuvant cortical mastoidectomy during tympanoplasty.[3]

At present, there is no accurate investigation for preoperative evaluation of status of aditus. Although, high resolution computed tomography (HRCT) of temporal bone can provide information regarding aditus patency there is a universal reluctance among otologists to consider preoperative HRCT.[5] Access to computerized tomography scanning, exposure to radiation, cost factors may be deterrents, hence the need for an alternate investigation.

The association between aeration of mastoid, patency of aditus and eustachian tube function is well known.[3] Hence, we propose to determine if Eustachian tube

function (ETF) as assessed by inflation-deflation test, would help predict aditus patency preoperatively. Thus, a simple, non invasive, inexpensive investigation could avoid an adjuvant surgical procedure (cortical mastoidectomy) which involves significant operating time, cost and morbidity.

# OBJECTIVE OF THE STUDY

To assess the eustachian tube function using inflation - deflation test and to correlate the results of inflation- deflation test with operative findings regarding aditus ad antrum patency in tubotympanic disease.

# REVIEW OF LITERATURE

## HISTORICAL REVIEW

The Eustachian tube is named after Bartolomeus Eustachus (1520-1574), who at one time held the chair of anatomy at Rome.[6] His book "Epistola de Auditus Organis" appeared in 1562, & is probably the earliest work to deal exclusively with the ear. He was one of the first to describe the structure, course and relations of Eustachian tube with accuracy and he added that Eustachian tube acted like a janitor of middle ear cleft.

Another early description came from Groningen in 1572. Du Verney (1648-1730) was the first to recognize the function of Eustachian tube. He stated in 1683, that the Eustachian tube was not a passage for breathing or of hearing, but one through which air in the tympanum was renewed.

The Eustachian tube was known to the Greeks, and was mentioned by Aristotle, but Eustachius was the first to describe its anatomy with accuracy. He divided it into bony and cartilaginous parts, the cartilaginous part lined with mucus membrane similar to that of nasal cavity. He believed that the tube was normally open.

In 1704, Antonio Valsalva (1665-1723), was the first to recognise the importance of tensor veli palatini muscle in opening of the eustachian tube. His name

is associated with a now time honoured technique of forcing air into the tympanum from the nasopharynx called the valsalva manoeuvre.

Valsalva described this manoeuvre in his treatise "Treachus De Aure Humena." He originally described its use as a therapeutic measure to remove pus from the ear, in cases of otitis media with a perforated ear drum. Subsequently it was used for inflation of the middle ear in cases of obstruction of the Eustachian tube.

In 1853, Toynbee demonstrated that the Eustachian tube was usually closed, and that swallowing was the mechanism that opened the tube. He introduced the Toynbee's manoeuvre to test the patency of the eustachian tube.

In 1883, Adam Politzer made important contributions in connecting the role of Eustachian tube in middle ear pathology.[7]

Physiology of the middle ear was first described by Helmholtz, in 1868.[8] The potent seriousness of ear suppuration was appreciated by Hippocrates.[9] The first recorded successful mastoid operation for the relief of aural suppuration was in 1774, by Jean Petit of Paris, followed shortly thereafter by Jasser (1776) and Baron Bergen, a personal physician to the King of Denmark.

In 1853, Sir William Wilde of Dublin, introduced his famous postaural incision for suppuration of the ear with post aural abscess. It was Schwartze, in 1873 who described the simple mastoid operation.[10] In 1868, Helmholtz defined clearly the mechanics of middle ear and laid ground work for the principles of tympanoplasty. The Monocular Operating Microscope was introduced in 1921, by Nylen and the

Binocular microscope by Holmgren, in 1922. Landmark studies were conducted by Bekesy, Juers & Davis and Walsh and shed light on how the normal and pathologic middle ears work.

Zollner, in 1951, and Wullstein, in 1952, introduced the tympanoplasty techniques to reconstruct the sound conducting apparatus of the middle ear, that had been impaired or destroyed by suppurative disease of middle ear. Heerman, in 1961, proposed temporalis fascia as a graft.[11]

# ANATOMY OF MIDDLE EAR CLEFT

The middle ear is part of a system of contiguous organs, including the nose, nasopharynx, eustachian tube, middle ear and mastoid. Respiratory mucosa is continuous throughout the system. Thus, signs and effects of inflammation , infection, or obstruction in one area are likely to be reflected in other areas.[12]

Middle ear cleft consists of the tympanic cavity, eustachian tube, the aditus ad antrum, mastoid antrum and the pneumatic system of the temporal bone.

## THE MIDDLE EAR ( TYMPANIC CAVITY)

The middle ear cavity is an irregular, air-filled space within the temporal bone between the tympanic membrane laterally and the osseous labyrinth medially. The cavity has lateral and medial walls, a roof and a floor and anterior and posterior walls.

### The lateral wall

The lateral wall consists mainly of tympanic membrane and partly of bone above and below the membrane. Accordingly, the cavity is divided into three parts:

a. the attic or epitympanum - is the upper compartment of middle ear above the horizontal part of facial nerve. It contains the main bulk of incus and malleus.
b. the mesotympanum (or the tympanum proper) - medial to the tympanic membrane.
c. the hypotympanum - below the level of tympanic membrane.

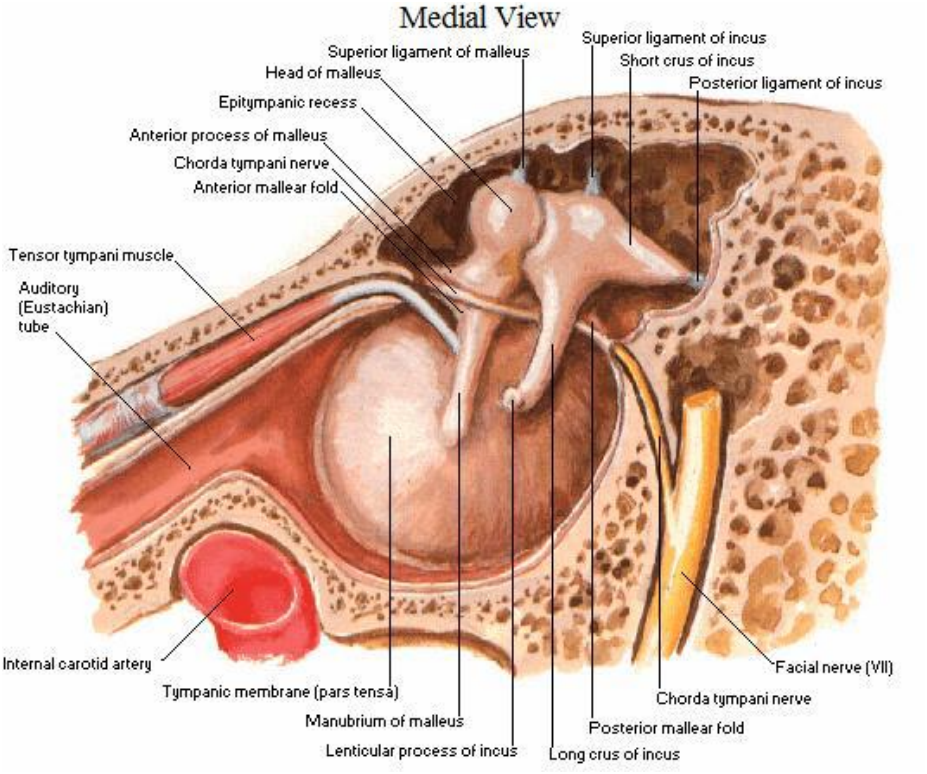



Fig. 1: Medial view of the Middle Ear

**The medial wall**

It separates the tympanic cavity from the inner ear and is distinguished by the presence of promontory, a smooth rounded bony projection covering the basal turn of the cochlea. Behind and above the promontory is the oval window, a nearly kidney-shaped opening that connects the tympanic cavity with the vestibule, but in life is closed by footplate of the stapes and its surrounding annular ligament. Its size varies with the size of the footplate, but on average it is 3.25 mm long and 1.75 mm wide. Above the oval window is the facial nerve and below is the prominence of the promontory.

The round window niche lies below and a little behind the oval window niche from which it is separated by a posterior extension of the promontory called the subiculum. Occasionally, another ridge of bone, the ponticulus, leaves the promontory above the subiculum and runs to the pyramid on the posterior wall of the cavity. The round window niche is most commonly triangular in shape, with anterior, posterosuperior and posteroinferior walls. The latter two meet posteriorly and lead to the sinus tympani. The round window membrane is usually out of sight, obscured by the overhanging edge of the promontory forming the niche and mucosal folds within it. The membrane is roughly oval in shape, about 2.3 x 1.9 mm in dimension and lies in a plane at right angles to the plane of the stapes footplate.

The facial nerve canal (or Fallopian canal) runs above the promontory and oval window in an anteroposterior direction. It has a smooth rounded lateral surface that often has micro dehiscences and when the bone is thin or the nerve exposed by disease, there are two or three straight blood vessels clearly visible along this line of nerve. The facial nerve canal is marked anteriorly, by the processus cochleariformis, a curved projection of bone, concave anteriorly, which houses the tendon of the tensor tympani muscle as it turns laterally to the handle of the malleus. Behind the oval window, the facial canal starts to turn inferiorly as it begins its descent in the posterior wall of the tympanic cavity.

**The roof**

The roof of the middle ear cavity, the tegmen tympani separates it from the middle cranial fossa.

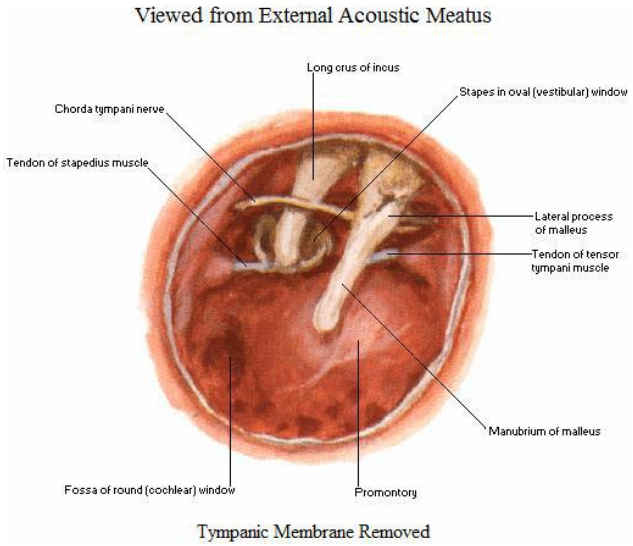



Fig.2: Medial wall of middle ear viewed from external acoustic meatus

**The floor**

The floor of the tympanic cavity is also a plate of bone, separating the cavity from the jugular bulb. Occasionally, when it is dehiscent, the jugular bulb encroaches on the cavity and the bulb may be injured in surgical procedures on the middle ear.

**The anterior wall**

It presents four openings from above downwards:

1. the small orifice through which the chorda tympani escapes from the middle ear (canal of hugier)
2. the canal for tensor tympani.
3. orifice of the eustachian tube.
4. the GLASSERIAN fissure containing the tympanic artery and anterior ligament of malleus.

The tensor tympani muscle is contained in its own bony canal above the osseous portion of the auditory tube and the adjoining part of the greater wing of the sphenoid bone. This muscle ends in a slender tendon, makes a sharp bend at the cochleariform process and inserts into the manubrium of the malleus.

**The posterior wall**

It presents an opening to the aditus ad antrum which leads backwards from the epitympanum into the mastoid antrum. Below this is the pyramidal eminence, containing the stapedius muscle.

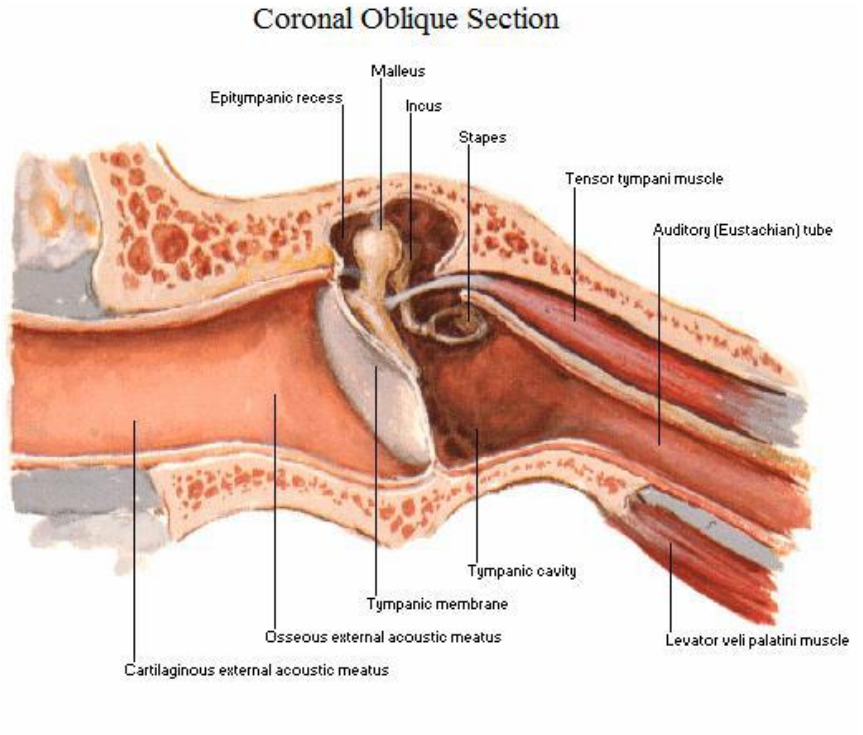



Fig.3 Coronal Oblique view of Middle ear showing insertion of tensor tympani into manubrium of malleus

The medial wall of the posterior mesotympanum is divided into discrete bony pockets by the ponticulus and the subiculum. Below the ponticulus on the posterior

aspect of the tympanum (between the labyrinthine capsule and the styloid complex) is the sinus tympani; above this area and the facial nerve is an area known as the facial recess.

## The contents of the middle ear cavity

The contents of the middle ear cavity are: 1. air which fills the cavity 2. the auditory ossicles (malleus, incus and stapes) 3. the muscles- tensor tympani and stapedius 4. the facial and chorda tympani nerves 5. the tympanic plexus of nerves.

### The malleus

It is the largest of the 3 ossicles measuring upto 9mm in length. It comprises a head, neck and handle or manubrium. The head lies in the epitympanum and is suspended by the superior ligament, which runs upward to the tegmen tympani. The head of the malleus has a saddle-shaped facet on its postero-medial surface to articulate with the body of the incus by way of a synovial joint. Below the neck of the malleus, the bone broadens and gives rise to the lateral process, the anterior process and the handle. The lateral process is a prominent landmark on the tympanic membrane and receives the anterior and posterior malleal folds from the tympanic annulus. The chorda tympani crosses the upper part of the malleus handle on its medial surface above the insertion of the tendon of tensor tympani, but below the neck of the malleus itself. The neck of the malleus connects the handle with the head. A slender anterior ligament arises from the anterior process to insert into the petrotympanic fissure. The handle runs downwards, medially and slightly backwards between the mucosal and fibrous layers of the tympanic membrane. On the deep,

medial surface of the handle, near its upper end, is a small projection into which the tendon of the tensor tympani muscle inserts.

**The Incus**

It consists of a body and 2 processes, known as the short and long process. On the anterior surface, there is a facet that articulates with the malleus. The short process is attached into the fossa incudis by the posterior ligament of the incus. The long process ends at the lenticular process which in turn articulates with capitulum of stapes.

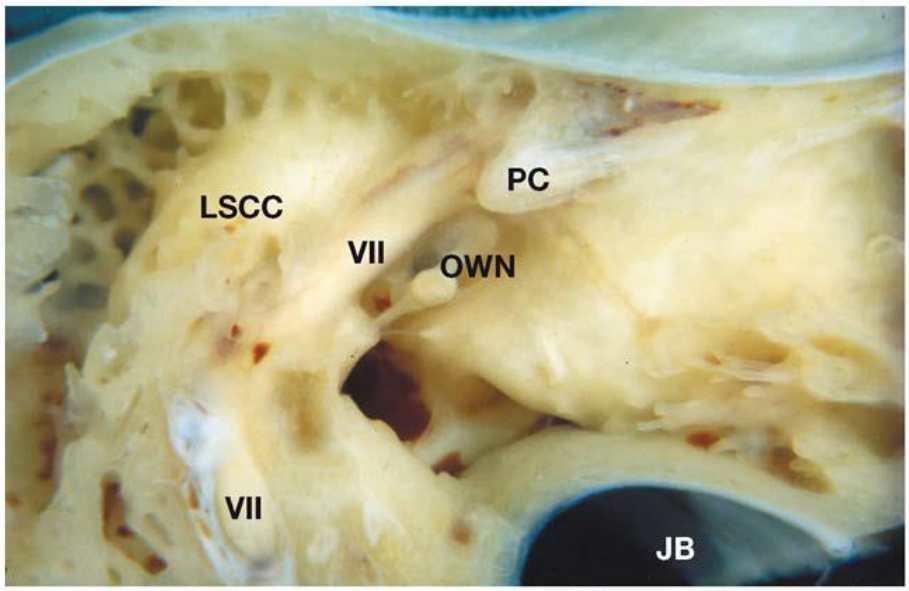



Fig.4. A specimen cut to show the medial wall of the tympanic cavity. The processus cochleariformis marks the anterior portion of the intratympanic portion of the facial nerve, which passes between the lateral semicircular canal (LSCC) and oval window nich (OWN). Note the jugular bulb (JB) in the floor of the tympanic cavity.

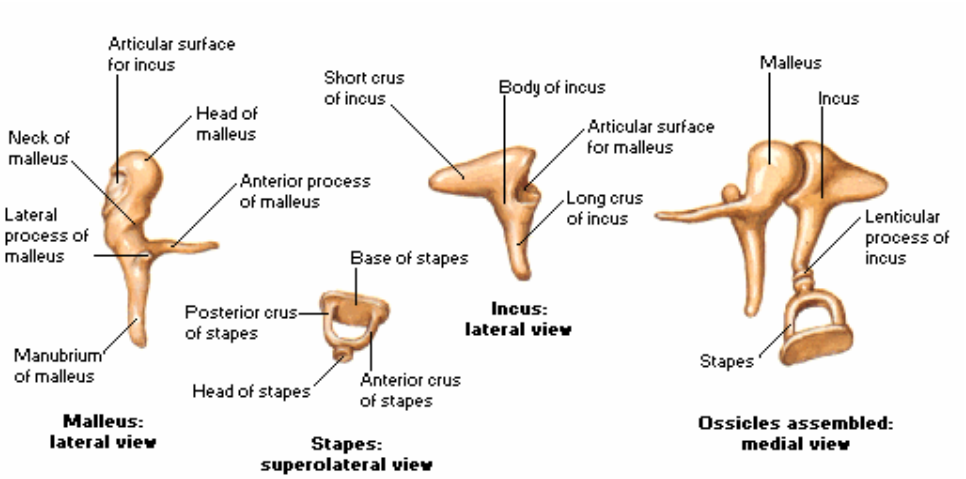



Fig.5 showing Auditory Ossicles

**The Stapes:** It is the smallest bone in the body. It has a head, neck, anterior and posterior crura and a foot plate-held in the oval window by the annular ligament. The stapedius tendon is inserted into the posterior surface of the neck.[13]

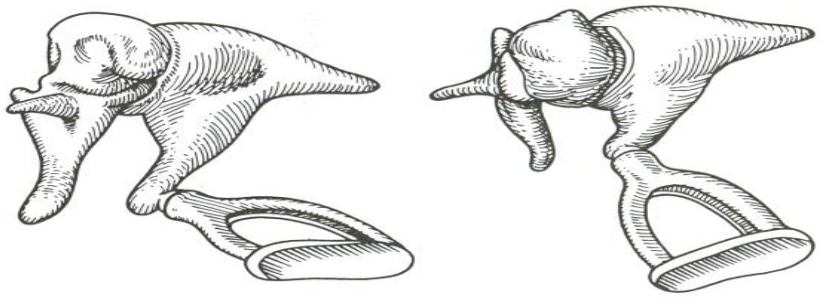


Fig.6. Middle-ear ossicles viewed from medial to lateral (left) and from above (right).

**Mucosa –** The mucous membrane of the middle ear and mastoid is continuous with that of the nasopharynx via the Eustachian tube. This membrane covers all structures within the middle ear, including the ossicles, vessels, and nerves. Examination of cells of the mucous membrane within the tympanic cavity reveals a gradual change from

tall, columnar cells with interspersed goblet cells to shorter cuboid cells at the posterior portion of the promontory and aditus ad antrum.[12]

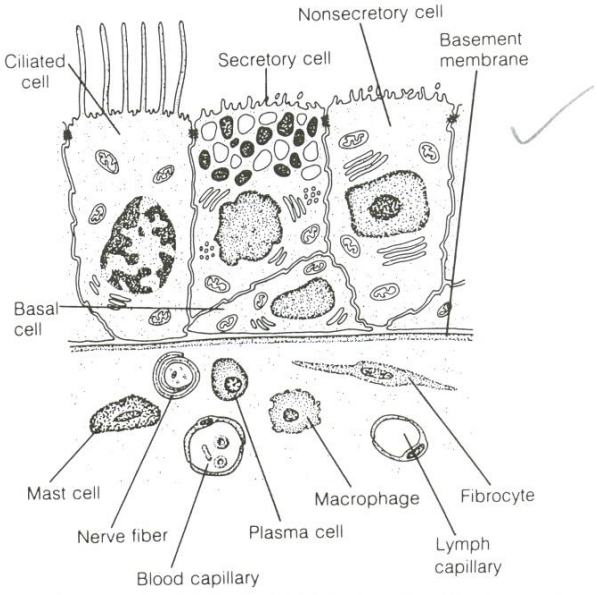



Fig.7: Mucosa of the middle ear.

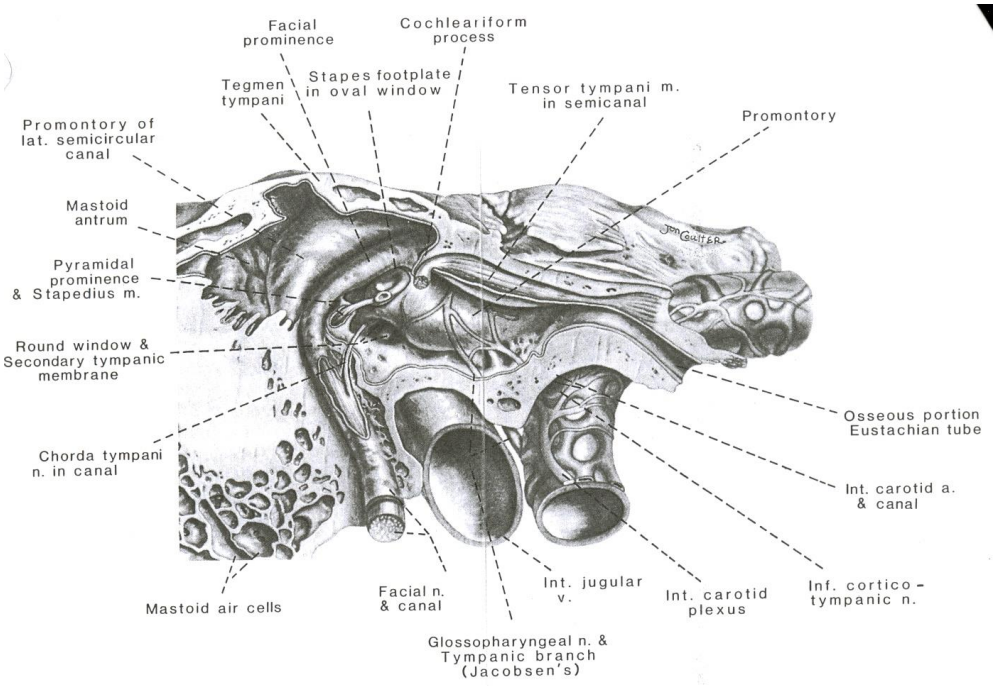



Fig.8: The innervations of the middle ear as depicted from lateral to medial

**Nerve Supply**

The tympanic cavity and contained structures are innervated by branches of the tympanic plexus of nerves. Jacobson nerve, a branch of the glossopharyngeal nerve, enters the cavity through its floor, divides, and ramifies about the promontory to contribute to the plexus. The tympanic plexus has connections to the ventral subnucleus of ipsilateral nucleus of the solitary tract within the brain stem, which has been postulated to provide sensory input from middle ear chemoreceptors, baroreceptors, or both, and thus is related to middle ear aeration.

Sympathetic innervations to the plexus is provided by the superior and inferior caroticotympanic nerves and parasympathetic fibres by the smaller superficial petrosal nerve.

**Chorda Tympani Nerve** – arises from the sensory part of the descending facial nerve. It enters the cavity through the iter chordae posterior, traverses the cavity by crossing the manubruium of the malleus and the long process of the incus, and exits via the iter chordae anterior.

The tensor tympani muscle receives its innervation from the trigeminal nerve. The Stapedius muscle derives its innervations from the facial nerve.[12]

**MASTOID ANTRUM**

The mastoid antrum is an air filled sinus within the petrous part of the temporal bone. It communicates with the middle ear by way of the aditus and has mastoid air cells arising from its walls. The antrum, but not the air cells is well

developed at birth and by adult life has a volume of about 1 ml, being 14mm from front to back, 9mm from top to bottom and 7mm from side to side.

The medial wall of the antrum is related to the posterior semicircular canal and more deeply and inferiorly is the endolymphatic sac and the dura of the posterior cranial fossa. The roof forms part of the middle cranial fossa and separates, the antrum from the temporal lobe of the brain. The posterior wall is formed mainly by the bony covering of the sigmoid sinus. The lateral wall is part of the squamous portion of the temporal bone and increases in thickness during life from about 2mm at birth to 12-15mm in adult.

The floor of the mastoid antrum is related to the digastric muscle laterally and sigmoid sinus medially. The anterior wall has the aditus in its upper part while lower down the mastoid segment of facial nerve passes in its descent to the stylomastoid foramen.

**ADITUS AD ANTRUM**

This is a large irregular opening leading from the posterior epitympanum into the air filled space of the mastoid antrum. On the medial wall is the prominence of the lateral semicircular canal. Below and slightly medial to this is the bony canal of the Facial nerve. The short process of the incus lies on its floor.

## MASTOID AIR CELLS

These are irregular air filled cavities communicating with each other and with the mastoid antrum. The distribution and number of air cells communicating with the mastoid antrum is extremely variable.

Temporal bones may be classified as follows:

Pneumatized – when air cells are well developed.

Diploic (mixed) - When air cells are interspersed with marrow containing spaces.

Sclerotic (acellular) – When the bone is hard and dense with no air spaces beyond the mastoid antrum.[14]

80% of adult bones are pneumatised. The air cells form in both the petrous and squamous parts of the mastoid and when well developed they may be classified according to their anatomical location into the following groups: 1.zygomatic 2. Tegmen 3. Sinodural 4. Marginal or perisinus 5. Periantral cells 6. apical 7. Retrofacial 8. Perilabyrinthine 9. Tip cells 10. Peritubal cells.

## CORTICAL MASTOIDECTOMY

A simple or cortical mastoidectomy is an operation performed to remove disease from the mastoid antrum and air cell system (when present) and the aditus ad antrum, with preservation of an intact posterior bony external auditory canal wall, without disturbing the existing middle ear contents.

**Complications**:

- facial nerve injury, hearing loss, infection, injury to dura, bleeding

## EUSTACHIAN TUBE (ET)

The eustachian tube connects the middle ear and the nasopharynx. ET dysfunction has been implicated in the cause of chronic otitis media and hence the study of anatomy and function of this area is important in understanding disease and disorders that affect middle ear and mastoid.

In adults, the tube lies at an angle of 45 degrees in relation to the horizontal plane, whereas in infants this inclination is only 10 degrees. The tube is longer in adult than in infants and young child and its length varies with race. The usual range of length reported is 31-38mm. It is generally accepted that the posterior third [11-14mm] of the adult tube is osseous and the anterior two- thirds [20-25mm] is composed of membrane and cartilage.

The osseous eustachian tube (protympanum) lies completely within the petrous portion of the temporal bone and is directly continuous with the anterior wall of the superior portion of middle ear. The juncture of the osseous tube and the epitympanum lies 4mm above the floor of the tympanic cavity. This relationship, is of some importance in the functional clearance of the middle ear fluids. The lumen is roughly triangular, measuring 2-3mm vertically and 3-4mm horizontally. The healthy osseous portion is open at all times in contrast to the fibro cartilaginous portion, which is closed at rest and open during swallowing or when forced open, such as during the valsalva manoeuver. The osseous and cartilaginous portions of the eustachian tube meet at an irregular bony surface and form an angle of 160 degrees with each other.

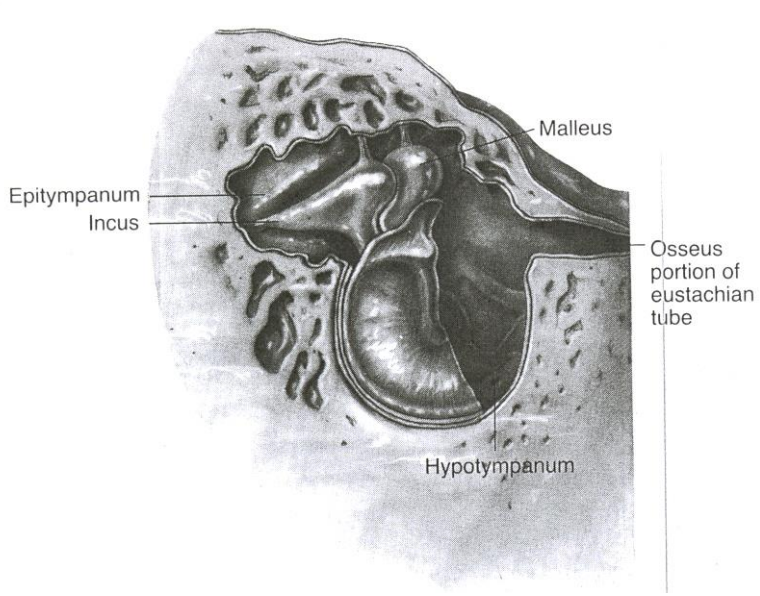



Fig.9a: The anatomy of the aural portion of the Eustachian tube as viewed from the external canal. Note that the orifice of the Eustachian tube is relatively high in the middle ear.

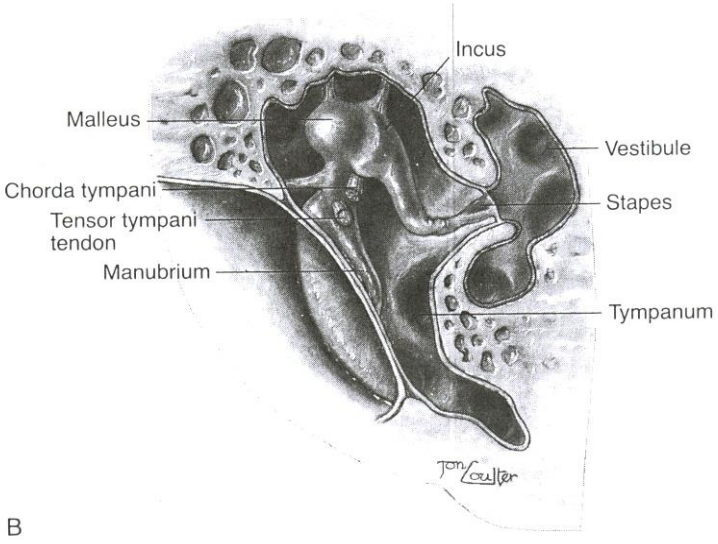



Fig 9b: Coronal section through middle ear.

The cartilaginous tube courses anteromedially and inferiorly, angled in most cases 30-40 degrees through the transverse plane and 45 degrees to the sagittal plane.

The tube is closely applied to the basal aspect of the skull and is fitted to a sulcus tubae between the greater wing of the sphenoid bone and the petrous part of the temporal bone. The cartilaginous tube is firmly attached at its posterior end to the osseous orifice by fibrous bands and usually extends some distance (3mm), into the osseous portion of the tube. At its inferomedial end it is attached to the tubercle on the posterior edge of the medial pterygoid lamina.

The cartilaginous portion is completed laterally and inferiorly by veiled membrane which serves as a site for the attachment of the dilator tubae, tensor veli palatini muscle. The tubal lumen is shaped like two cones joined at the apexes. The junction of the cones is the narrowest point of the lumen and has been named the isthmus, and its position is usually described as at or near the junction of the osseous and cartilaginous portions of the tube. The lumen at this point is approximately 2mm high and 1 mm wide. From the isthmus, the lumen expands to approximately 8-10mm in height and 1-2 mm in diameter at the pharyngeal orifice. Tubal cartilage increases in mass from birth to puberty and this development has physiologic implications.

The cartilaginous tube has a thickened anterior fibrous investment that presses against the pharyngeal wall to form a prominent fold, the torus tubarius, which measures 10-15mm in thickness. The mucosal lining of the ET is continuous with that of the nasopharynx and middle ear and is characterized as respiratory epithelium. Mucous glands predominate at the nasopharyngeal orifice, and there is graded change to a mixture of goblet, columnar and ciliated cells near the tympanum.

Muscles associated with the ET:

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

Usually the eustachian is closed; it opens during such actions as swallowing, yawning, or sneezing and thereby permits the equalization of middle ear and atmospheric pressures. The medial bundle of the tensor veli palatini muscle is called the dilator tubae muscle and is responsible for active dilatation of the tube.[12]

**Relations of Eustachian Tube**

Anteromedially, the tensor veli palatini (tensor palate) separates the tube from Otic ganglion, mandibular nerve and its branches, chorda tympani and middle meningeal artery.

Tensor palati is also related superiorly to the bony portion of the tube, from which it is separated by a thin plate of bone. Carotid artery lies medial to the Eustachian tube. The salpingopharyngeus muscle is attached to the inferior aspect of cartilage of the tube near its pharyngeal opening. Posteromedially, the eustachian tube is related to the petrous part of the temporal bone and to the levator veli palatini, which arises partly from its medial lamina.

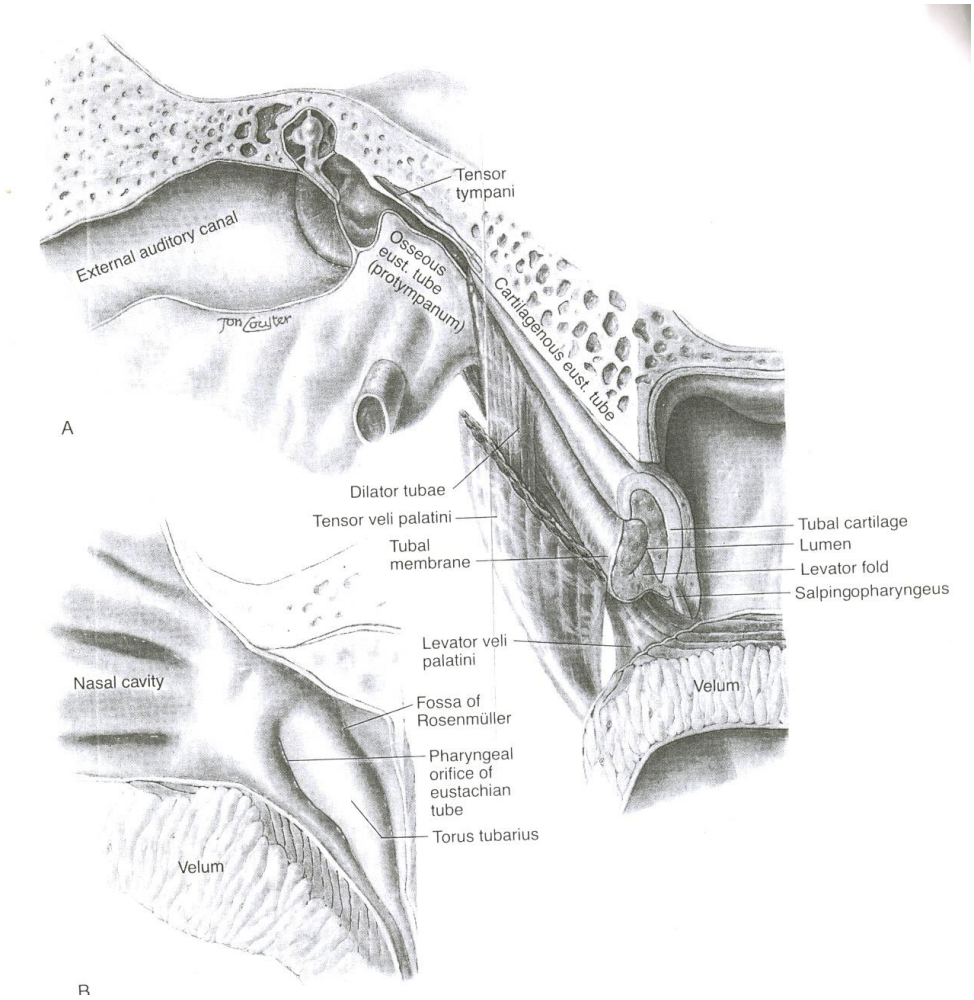



Fig.10: A. Illustration of complete dissection of Eustachian tube and middle ear. Especially evident are the relations of the Eustachian tube and the middle ear.

B. Appearance of the nasopharyngeal orifice of the Eustachian tube. Note the large torus tubarius and its inferior continuation at the salpingopharyngeal fold.

The Pharyngeal opening of the eustachian tube lies in the lateral wall of nasopharynx 10-12.5mm behind & a little below the posterior end of the inferior nasal concha. It is triangular in shape and is bounded above and behind by the tubal elevation which is a firm prominence provided by the underlying pharyngeal musculature and of cartilage of Eustachian tube. A vertical fold of mucus membrane, called salpingopharyngeal fold, stretches from the lower part of tubal elevation

downwards in the lateral wall of nasopharynx . It contains the salpingopharyngeus muscle. A second and smaller fold, the salpingopalatine fold, stretches from the anterosuperior part of the elevation to the soft palate. The levator veli palatini, as it enters the soft palate, produces an elevation of mucus membrane immediately below the pharyngeal opening of the tube. The pharyngeal recess lies behind the tubal elevation.

**Blood supply**

Blood supply of Eustachian tube is derived from :

1. Ascending pharyngeal artery
2. Middle meningeal artery and
3. Artery of the pterygoid canal

**Nerve Supply**

Eustachian tube is supplied by branches from the tympanic plexus of nerves and by the pharyngeal branch of the pterygopalatine ganglion.

The muscles that act to open the Eustachian tube are the tensor tympani, tensor veli palatini, levator veli palatini and salpingopharyngeus. The tensor palatine and the tensor tympani muscles have a common embryological origin and a common nerve supply from a branch of the trigeminal nerve, their action on the Eustachian tube is synergistic.[12]

**Tensor veli palatini**

This muscle is situated on the lateral aspect of the cartilaginous portion of the tube. It originates from :

1. Spine of the sphenoid
2. Scaphoid fossa and
3. Lateral lamina of the tubal cartilage and the adjacent membranous part.

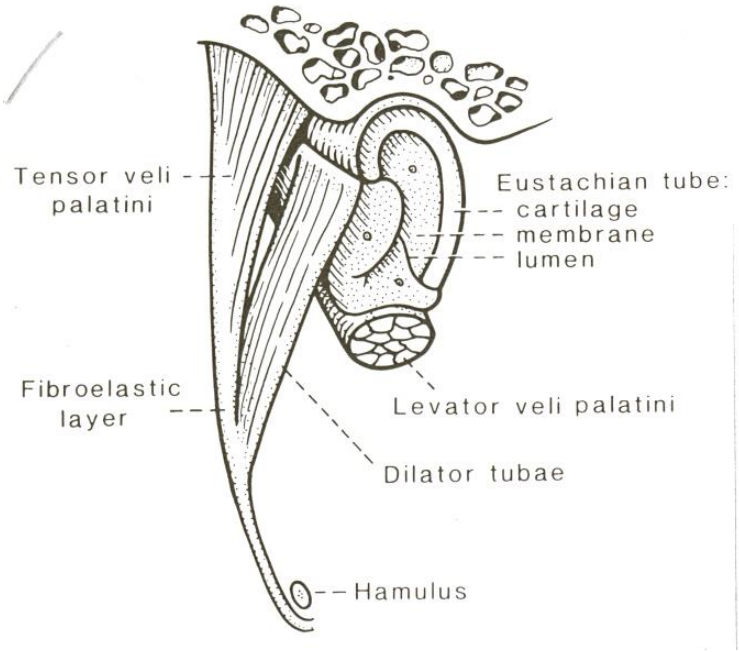



Fig.11: Diagrammatic representation of the relationship between the superficial muscle bundle (tensor veli palatini) and the deep bundle (dilator tubae) to the lateral wall of the Eustachian tube.

The flattened triangular muscle runs downwards and forward and becomes tendinous. The tendon winds round the pterygoid hamulus to be inserted radially into the soft palate and the palatine bone.

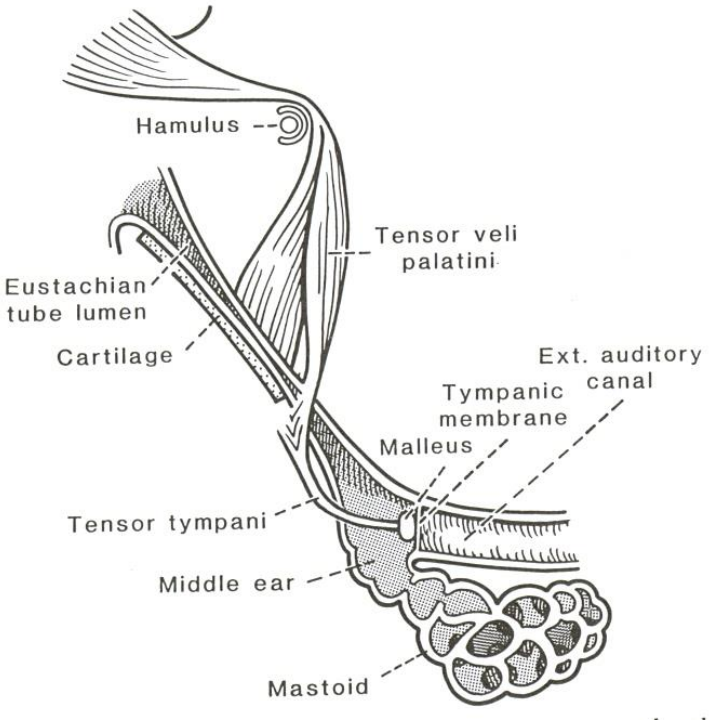



Fig. 12: Diagrammatic representation of the tensor veli palatini muscle attachment along the lateral wall of the Eustachian tube, its course around the hamulus of the pterygoid bone, and its attachment into the posterior margin of the hard palate.

Rood & Doyle have shown that the tensor veli palatini actually consists of two groups of muscle fibres - a medial group called dilator tubae and a lateral group called tensor veli palatine. The latter does not have an eustachian tube attachment, but is continuous anteriorly with the tensor tympani. The dilator tubae originates from the entire membranous wall of the posterior one third to one half of the tube and is considered an active tubal dilator.[15]

**Tensor tympani muscle** - is a bipinnate muscle, the medial group of fibres originates from (1) the cartilaginous portion of the tube and (2) the adjoining part of the sphenoid.

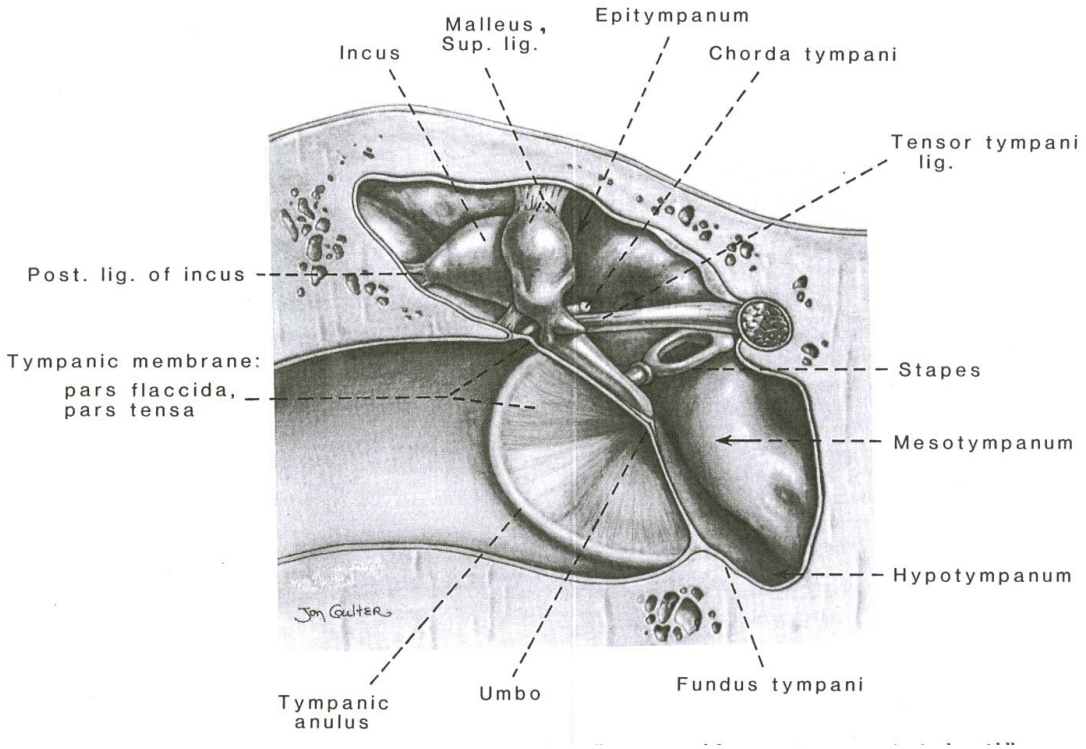



Fig.13: The tensor tympani inserts into the manubrium of the malleus as viewed from anterior to posterior in the middle ear.

The lateral group arises as a continuation of the most inferoposterior fibres of the tensor veli palatini, which lack bony attachment, but rather extend lateral to the tube to join the tendon of the medial fibres. The muscle is contained in a bony canal situated above the osseous part of the tube. It passes posteriorly forming a tendon which turns, and after crossing the tympanic cavity is inserted into the head of malleus.[12]

## Physiology of Eustachian Tube

The eustachian tube performs 3 important physiologic functions with respect to the middle ear:

1. **Ventilation** - The most important function of the Eustachian tube is to maintain air pressure equality on both sides of the tympanic membrane. The continuous absorption of oxygen through the mucosal lining in the middle ear space together with the variation of ambient barometric pressure constitute the basic need for intermittent air exchange through the eustachian tube. This is accomplished by opening the tube and admitting air either from or into the middle ear space.[16,17]

   Regulation of middle ear pressure is primarily caused by contraction of tensor veli palatini during swallowing and yawning. This allows opening of eustachian tube and passive exchange of air between middle ear and nasopharynx. Airflow is intermittent lasting around 0.4sec once every minute and occurs in both directions, based on pressure difference between middle ear and atmosphere. During majority of time, pharyngeal opening of ET remains closed.[12]

2. **Clearance of Secretions** – This is an active process based on mucociliary lining of tube. Movement of material from middle ear into nasopharynx has been shown in human models. Embryologically, eustachian tube forms by invagination of nasopharyngeal mucosa, with structure of epithelium and submucosal layer similar to other parts of respiratory tract. A variety of irritant factors like environmental pollutants, bacterial and viral infection, irradiation affect function of this epithelium. Studies have shown ciliated cells and goblet cells are more populated along the floor of Eustachian tube and increase in density from tympanic to nasopharyngeal opening. The activity of these cells has been shown to move secretions actively out of middle ear. Sando et al also found more mucosal folds along floor of Eustachian tube which led them to

theorize that floor was more important for secretions clearance and that roof of eustachian tube was involved in ventilation of middle ear.

3. **Protection of middle ear** - A closed Eustachian tube pharyngeal orifice protects middle ear from sound generated in respiratory tract and from secretions entering from nasopharynx. Presence of intact middle ear mucosa and mastoid, which provides a "cushion" of air behind the tube, also aid in preventing secretions from entering Eustachian tube orifice. Though closure of Eustachian tube orifice is largely passive, dysfunction of process of active dilation can also cause difficulties.

Several anatomical features of Eustachian tube have been shown to contribute to mechanical protection of Eustachian tube. Though opening of eustachian tube is largely an active process based on contraction of tensor veli palatini, closure is passive. In mid portion of eustachain tube, elastin is present between medial and lateral lamina. Sando et al have hypothesized that this allows the lateral lamina to close the roof of eustacian tube after contraction of this muscle. Protection of the tube floor may come from presence of ostmann's pad of fat which exerts pressure on orifice when tensor veli palatini relaxes. On cross sectional analysis, this fat pad is located between Eustachian tube and tensor veli palatini.

**Role of mastoid**

The presence of air in the mastoid acts as buffer for middle ear. A well pneumatised mastoid has capacity to offset any middle ear pressure changes more than a contracted, poorly pneumatised mastoid.[4]

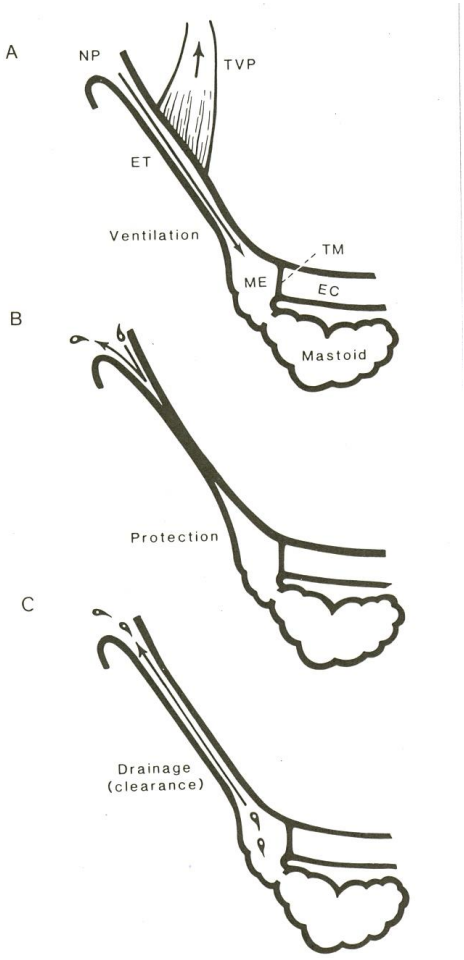



Fig.14: Three physiologic functions of the Eustachian tube in relation to the middle ear. Np, nasopharynx; ET, Eustachian tube; TVP, tensor veli palatini muscle; ME, middle ear; TM, tympani membrane; EC, external canal.

## Middle ear pressure

Due to pressure differential between air in the middle ear space and that of the surrounding tissue, a small amount of oxygen and nitrogen is continuously absorbed through the mucosal lining. This corresponds to a decrease in the middle ear pressure of approximately 50mm $H_2O$ per hour if the tube is closed. A slight negative pressure develops in the middle ear which is normally neutralised by tubal opening during deglutition.

Deglutition occurs approximately once per minute in the awake subject and once every five minutes during sleep. Though deglutition may occur, the Eustachian tube does not necessarily open during every swallow.[18]

Most studies have shown resting middle ear pressures of between 50 and -50 mm $H_2O$.[19,20] A pressure outside this range does not necessarily mean the patient has ear disease.[12]

Depending on the construction of the cartilaginous support, the loose arrangement of the peritubal tissue and the rugose mucosa, the lumen of the cartilaginous portion of the eustachian tube is collapsed. This closure force is determined by a series of so called tissue factors, namely :

1. The elasticity of the cartilage
2. The venous and
3. The properties of the mucus membrane

which are therefore the main factors in determining the middle ear pressure.

## Tubal opening

As a result of muscular activity, the cartilaginous portion of the tube opens. The tensor veli palatini is considered the main dilator, while the levator veli palatini muscle, forming part of the bottom of the tube, supports the opening. Contraction of the tensor veli palatini produces dilatation of the tubal lumen as well as its orifice by anterior displacement of the fibromembranous wall of the tube from the cartilaginous wall. However, if the middle ear pressure exceeds + 100 to +150 mm of $H_2O$ the eustachian tube opens spontaneously, without any muscular activity. This was shown

by Armstrong and Hien (1937) and King P.F. (1965) who demonstrated passive eustachian tube opening at a middle ear pressure of +200 mm of $H_2O$.[6] Perlman produced similar results at +270 mm of $H_2O$.[18]

With increasing environmental pressures, passive opening of the eustachian tube is prevented by "flutter valve" mechanism. Contraction of the tubal muscles during swallowing and yawning is necessary then to equalize the middle ear pressure with ambient pressure. However, if the pressure differential is allowed to build up, it progressively becomes more difficult to open till the occlusive force is greater than that which can be exerted by the muscles, which normally open the eustachian tube.[21] The importance of small air pressure differences across the tube to effect complete separation of the mucosal surfaces has been reported in literature. In pressure chamber experiments performed by Inglestedt et al in 1967, it was shown that the tube did not open upon swallowing until chamber reached a pressure of +150mm of $H_2O$. This demonstrates the effects of step by step increases of negative pressure in middle ear. Not until the middle ear pressure reaches -150 mm of $H_2O$ is the subject able to change pressure by swallowing.

## Tubal closing mechanism

In contrast to opening, closure of eustachian tube is exclusively a passive phenomenon; when the muscles relax and the static pressure is insufficient to keep the tubal walls separated, the lumen collapses.

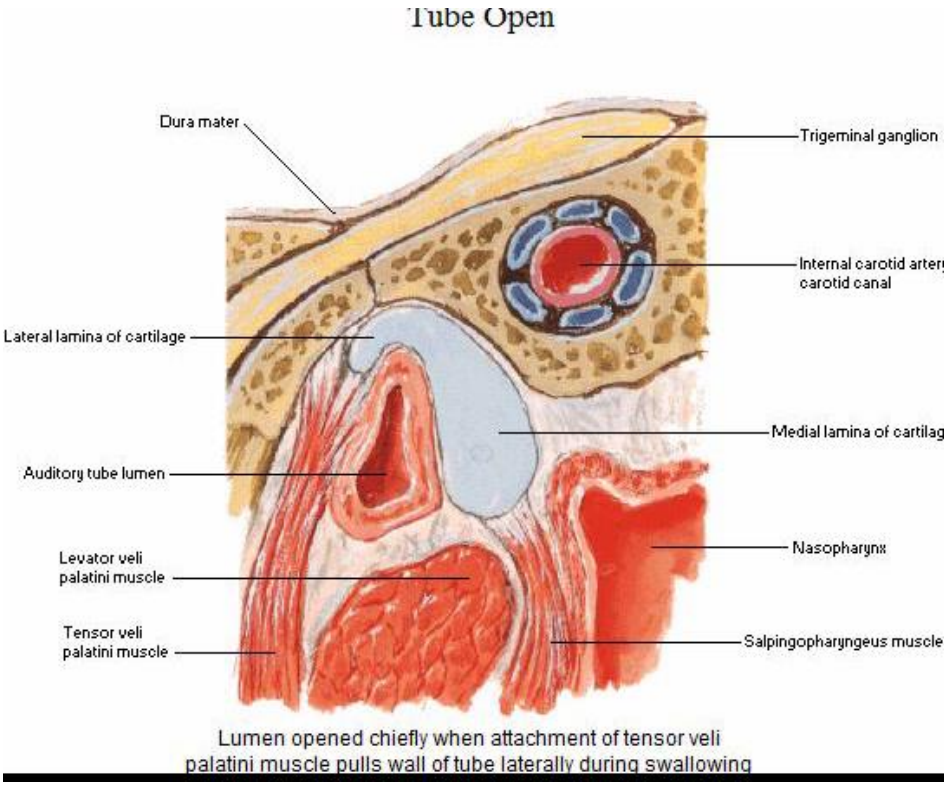



Fig. 15: Eustachian tube open

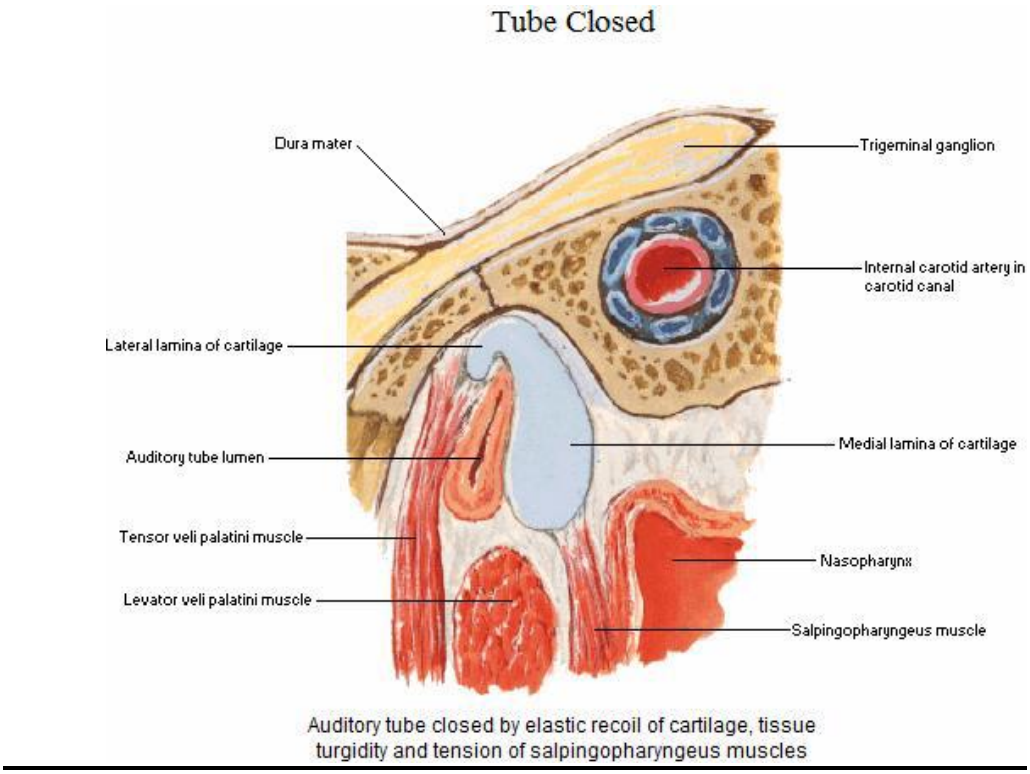



Fig.16: Eustachian tube closed

Aschan, using radiographic techniques, has shown that the closure of the eustachian tube starts at the nasopharyngeal end. In this way, it is possible that small volume of air is forced into the middle ear during closure of the tube, and this may explain the presence of slight positive pressure under normal conditions in many ears.[20]

## Pathophysiology of eustachian tube dysfunction

Abnormal Eustachian tube function appears to be the most important factor in the pathogenesis of middle ear disease.[22] Eustachian tube fails to open either from physical or physiological obstruction causing negative middle ear pressure development. Physical obstruction is usually due to hypertrophy of mucosa lining the tube and secondary to infectious etiology. Obstruction at middle ear opening can be due to hypertrophied middle ear mucosa.

Physiologic obstruction arises from failure of muscles involved to open the Eustachian tube, a theory supported by several studies showing that young children commonly have difficulties with active opening.[12]

Second factor important in regulation of middle ear pressure is based on gas exchange in middle ear and mastoid. Regulation of gas exchange between middle ear and middle ear mucosa circulation is not well understood. Tissue diffusion of gases is dependent on thickness of middle ear mucosa, rate of perfusion of mucosa, permeability of blood vessels and partial pressure of gas in middle ear and blood. The fact that middle ear gases are more similar to venous blood than atmospheric air suggests that this regulation is critical in understanding middle ear dynamics.

Gas exchange occurs passively and therefore can be impaired by materials which increase the distance between the middle ear cavity and the capillaries, such as oedematous mucosa, granulation, or scars in the middle ear.[23]

## EUSTACHIAN TUBE FUNCTION TESTS

One of the factors necessary for successful middle ear surgery is good preoperative eustachian tube function. Poor outcomes have been reported after tympanoplasty in patients with eustachian tube dysfunction.[24] Therefore, various methods have been devised to test eustachian tube function preoperatively to predict success in tympanoplasty.[25]

Until about 1960, most tests of the ventilatory function of the eustachian tube were in reality only assessments of the tubal patency. Some of the methods used are:

1. Valsalva test
2. Toynbee test
3. Politzer test
4. Catheterization of Eustachian tube
5. Manometry
6. Sonotubometry
7. Tympanometry
8. Trans - eustachian tube endoscopy to directly assess Eustachian tube status
9. Assessment of mucociliary function of eustachian tube using dye studies with fluorescein and saccharin.[25,26,27]

The first quantitative tubal function study performed by intratympanic manometry was the systematically conducted inflation-deflation test (Ingelstedt and Ortegren, 1963). Later, numerous investigators employed the same technique to determine tubal function (Miller, 1965; Holmquist, 1969a).The next improvement in this technique was the addition of a flow meter to the manometric system to involve pressure-flow relationships during Eustachian tube function testing ( Flisberg, 1966). The evaluation of tubal function was limited to the assessment of active function (owing to the contractions of the tensor veli palatini muscle) until Bluestone and co-workers (1972) introduced a modified inflation-deflation test by which passive function could also be described by variables such as forced opening pressure and closing pressure of the tube.[12]

Flisberg Aspiration test was a quantitative test of Eustachian tube function used by Holmquist in 72 patients with central perforation. 75% success rate was seen after tympanoplasty in patients with good tubal function whereas only 10% with hypofunction showed success.[28]

MacKinnon, in 1970, measured Eustachian tube function using modified Flisberg test in 80 patients by testing their ability to equilibrate applied pressure by swallowing. Residual pressure after swallowing of 0-100mm $H_2O$ was considered as good function, 100-200 mm $H_2O$ as moderate function and >200 mm $H_2O$ as poor function. Successful outcome was seen in 80% of patients with good or moderate function and in only 29% with poor function.

In 1993, Gimenez and Marco Algarra, studied preoperative Eustachian tube mucociliary clearance function by instilling sodium saccharine solution in the middle ear. The time required for patient to taste the solution was measured. 52 patients were reviewed and normal transport time was seen in 50% of patients with successful outcome and in only 22% of failures.[29]

**Inflation- Deflation five swallow tympanometry Test**

This test first reported by Flisberg et al in 1963, tests active function of the eustachian tube. It is a simple, non-invasive and reproducible test, that can be performed in all patients with perforated eardrums such as those with CSOM and those who have undergone myringotomy and grommet insertion for otitis media with effusion.[3]

The test is done with the patient seated comfortably. Otoscopic examination is done before the test to confirm dryness of external and middle ear.[24] The external auditory canal is sealed with a rubber ear tip. The middle ear is inflated, that is, positive pressure of +200 mm $H_2O$ is applied. The patient is then instructed to equilibrate middle ear pressure actively by swallowing. He/she then performs five consecutive swallows with an interval of 10 seconds in between to prevent strain on the pharyngeal muscles (active tubal function). The patient swallows "dry". The residual pressure remaining in the middle ear, if any, is recorded.

Comparison of dry and wet swallowing on equilibration of middle ear pressure by Adali has shown dry swallowing to be more effective. Although wet swallowing is easier for patients, the test may have to be repeated if the results are poor.[30]

The deflation phase of the test is then performed by applying -200mm$H_2O$ pressure to the middle ear and the patient is again instructed to swallow. The residual pressure remaining in the middle ear after attempts to equilibrate by five swallows is noted and recorded.

Even though the inflation-deflation test of eustachian tube is not strictly physiologic, the results are helpful in differentiating normal from abnormal function.[12] If positive and negative pressures are equilibrated by swallowing, the function of the eustachian tube is considered normal. Low opening pressure eg: <100 mm $H_2O$ may indicate semi-patulous eustachian tube whereas a patulous eustachian tube may not allow even a modest positive pressure to be maintained within the middle ear.[12]

Applied negative pressure equilibration is usually complete with normal Eustachian tube function. Partial or even absence of equilibration of any applied negative pressure may not indicate poor tubal function as even a normal Eustachian tube can lock with rapid application of negative pressure. On the other hand, the tube may have increased compliance and may collapse in response to negative pressure causing functional tubal obstruction.[12]

Takahashi considers Eustachian function as good if more than half of applied positive middle ear pressure or any applied negative pressure is equilibrated.[31]

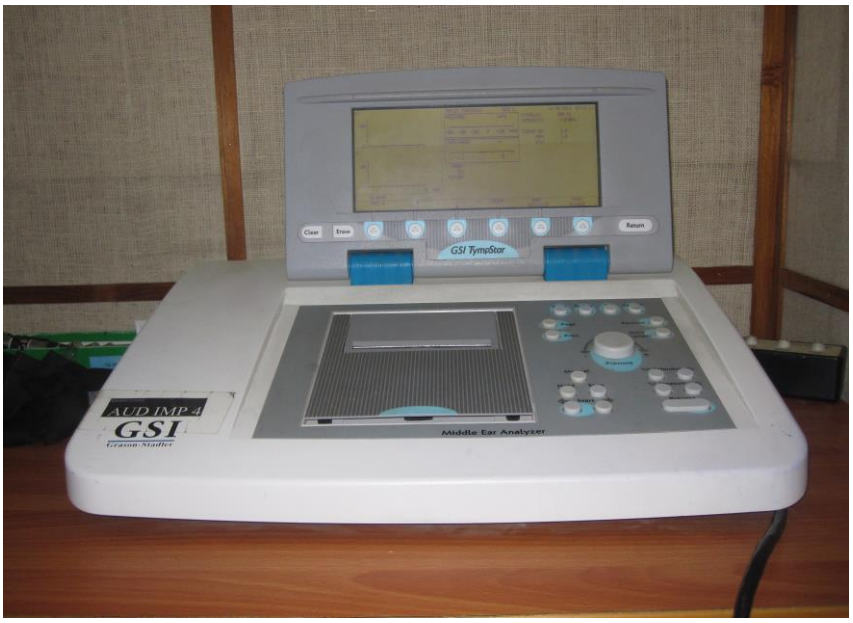



Fig.17: AUD IMP 4, GSI TYMPSTAR is the tympanometer used to assess Eustachian tube function using inflation-deflation test.

Fig. 18: Inflation–Deflation test in progress.

Mc Curdy, in 1980, observed that over half of patients with negative otologic history and normal otologic examination with type A tympanograms were unable to equilibrate applied negative and positive pressure. He suggested that such inability might not indicate clinically significant tubal dysfunction. He postulated that as with other biologic systems eustachian tube also exhibits individual variability in ventilatory function.[22]

Study by Swarts et al to establish normative data for inflation and deflation tests in adults without middle ear disease concluded that eustachian tube function can be considered to be good if approximately 83% of applied pressure was equilibrated during inflation test and 69% equilibration was present during deflation test.[32] Patulous eustachian tube is less compliant than normal eustachian tube. Sakakihara et al have shown them to be unable to equilibrate negative middle ear pressure by swallowing in 50% of patients.[33]

Applied negative and positive middle ear pressures can approximate atmospheric pressures without swallowing. This has been explained by gas exchange occurring via middle ear mucosa and mastoid air cell system. This may the reason for good outcome after tympanoplasty in patients with poor eustachian tube function.[34]

In a pilot study by Kurien et al in 2009, 80 patients with CSOM with central perforation were evaluated using inflation-deflation test followed by tympanoplasty and cortical mastoidectomy to assess aditus patency. Of a total of 80 patients, 31 had dry ears and 49 had discharging ears. The study showed that inflation - deflation test had a sensitivity of 93% in predicting aditus patency in dry ears and sensitivity of

72% in discharging ears. The specificity in both dry and discharging ears was 67%. It was concluded that a patent eustachian tube as assessed by inflation-deflation test is a good predictor of aditus patency in patients with CSOM with dry central perforations but less so in patients with discharging ears.[3]

Another factor which has been shown to affect the outcome of tympanoplasty is the presence of granulations or thickened mucosa within the mastoid. The mastoid air cell system plays an important role in the non-immunological defence of the middle ear. It produces secretions that clean the middle ear of bacteria and viruses and get transported to the nasopharynx via the two openings of the ear, the aditus ad antrum and the ostia of the eustachian tube, by the mucociliary system and gravity.[35]

For ventilation and pressure regulation within the middle ear, gas exchange not only via the eustachian tube but also via middle ear mucosa and mastoid air cell system is important. A diseased mucosa can act as a barrier to normal gas exchange and can also block the ET directly. As transmucosal gas exchange occurs passively between middle ear cavity and mucosal capillaries, such mucosal exchange is impaired by inflammatory changes in the middle ear mucosa and mastoid.[24]

Blockage of the aditus by granulation tissue or thickened mucosa can impede ventilation of the mastoid. It can adversely affect graft take up rate or produce retraction of tympanic membrane following tympanoplasty, if aerating mastoidectomy has not been performed simultaneously.[3] Therefore, if aditus patency could be determined preoperatively the decision to do mastoid clearance can be made before the operation.

# MATERIALS AND METHODS

## SOURCE OF DATA

All patients with CSOM of tubotympanic type undergoing tympanoplasty and cortical mastoidectomy at R.L. Jalappa Hospital and Research Centre, Tamaka, Kolar, from December 2010 to October 2012 have been included in the study.

## INCLUSION CRITERIA

All patients aged 12 years and above diagnosed with CSOM tubotympanic disease and advised to undergo tympanoplasty with cortical mastoidectomy were included in the study.

## EXCLUSION CRITERIA

The following patients were excluded from the study:

- Patients with past history of middle ear and mastoid surgery
- Patients with middle ear disease associated with cholesteatoma
- Patients with cleft palate
- Patients with past history of fracture of temporal bone/skull base
- Patients with sinusitis

## METHOD OF COLLECTION OF DATA

This is a prospective study in which all patients diagnosed with chronic suppurative otitis media - tubotympanic type in the department of Otorhinolaryngology and advised to undergo tympanoplasty with cortical mastoidectomy have been included. All patients underwent pure tone audiogram and X-ray mastoid preoperatively. All of them were subjected to a preoperative inflation-deflation test.

The inflation–deflation five swallow tympanometry test was done to assess the eustachian tube function.

- The patient was seated comfortably and the external auditory canal was sealed with a rubber ear tip.
- The middle ear was inflated to +200 mm$H_2O$ pressure and the patient was instructed to equilibrate middle ear pressure actively by swallowing.
- He/She was asked to perform five consecutive swallows with an interval of 10 seconds in between to prevent strain on the pharyngeal muscles (active tubal function). The residual pressure remaining in the middle ear, if any, was noted and recorded. The deflation phase of the test was then performed by applying -200 mm$H_2O$ pressure to the middle ear and the patient was again instructed to swallow. The residual pressure remaining in the middle ear after attempts to equilibrate by five swallows was noted and recorded. After 5 swallows, if patient was able to equilibrate 50% (0-99 mm $H_2O$) of applied pressure during inflation and deflation the eustachian tube was considered to be functioning adequately and residual pressure of 100 to 200 mm $H_2O$ was taken as poor Eustachian tube function as per the grading system by Mc Curdy[22].

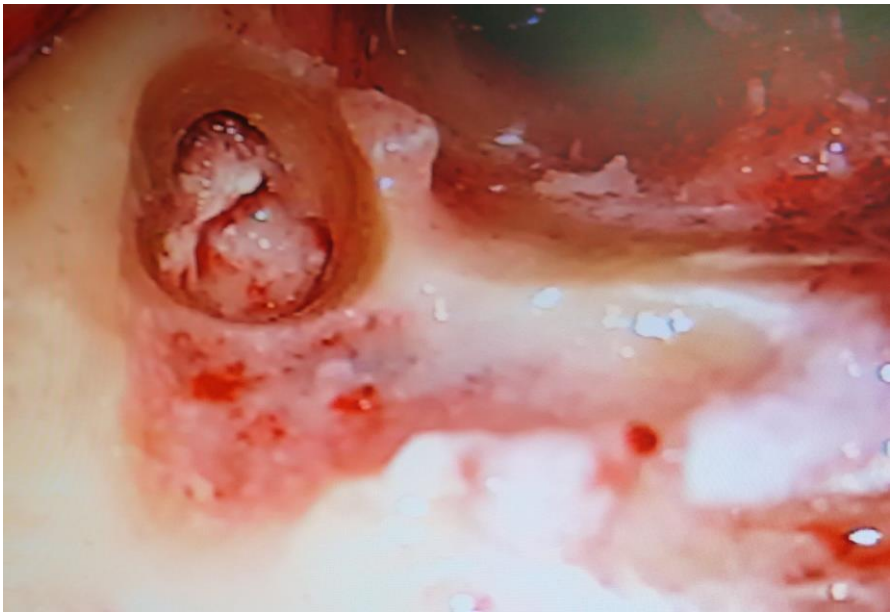


Fig.19: Cortical Mastoidectomy in a contracted mastoid with anteposed Sigmoid sinus

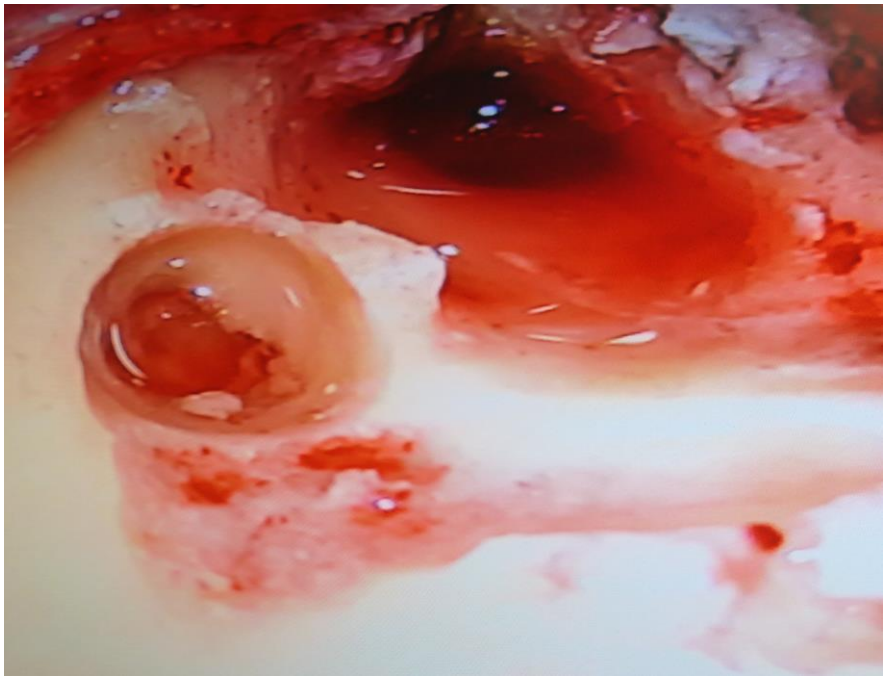


Fig 20: Patent aditus with free flow of saline from mastoid antrum to mesotympanum via aditus ad antrum

A standardized tympanoplasty with cortical mastoidectomy was then performed during which the patency of the aditus was assessed. The mastoid air cell system and the aditus were first inspected for presence of granulations or thickened mucosa. Then, irrigation of aditus with saline was done to check for patency. Free flow of saline from mastoid antrum to mesotymanum through the aditus ad antrum was recorded as "patent" and no flow observed was recorded as "blocked". Results of inflation-deflation test were correlated with intraoperative findings of aditus patency.

# OBSERVATIONS & RESULTS

In our study, a total of 60 patients varying in age from 16 to 77years were studied. Of these, 26 patients were male and 34 were females.

## AGE DISTRIBUTION

In our study of 60 patients, age of the patients varied from 16yrs to 77 years. 23 patients were in the age group of 21-30yrs constituting the majority (38.3%). There were 3 patients (5%) above 60 years, the oldest patient was 77years old. The average age at presentation was 32.4yrs.

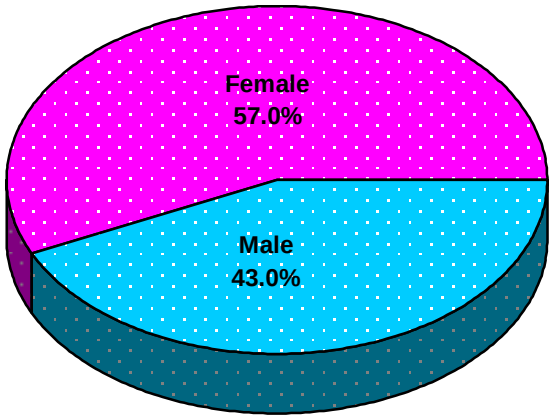
**Table 1: Age distribution**

| Age in years | Number of patients | % |
|---|---|---|
| 11-20 | 14 | 23.3 |
| 21-30 | 23 | 38.3 |
| 31-40 | 10 | 16.7 |
| 41-50 | 6 | 10.0 |
| 51-60 | 4 | 6.7 |
| >60 | 3 | 5.0 |
| Total | 60 | 100.0 |

Mean ± SD: 32.40±14.02

## SEX DISTRIBUTION

In the present study, male to female ratio was 1 : 1.2 with 26 males (43%) and 34 females (57%).




**Graph 1: Sex Distribution**

## SYMPTOMS

Out of 60 patients, 58 patients had history of otorrhea and two patients presented with otalgia only. History of otorrhea was present equally on both sides. Patients with otorrhea at presentation were treated medically and were included in the study when the ears had become dry. All patients had dry ears during assessment of eustachian tube function.

History of otalgia was present in 7 patients (11.7%) . 2 patients had otalgia as the presenting complaint with no history of otorrhea. 46 patients had history of

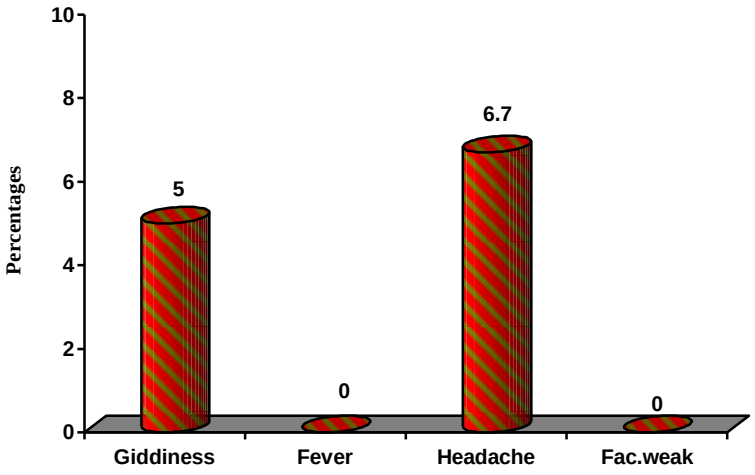
hearing loss. Most patients had hearing loss of 1-5 years duration. Tinnitus was present in 25%.

**Table 2: Symptoms**

| Symptoms | Number of patients (n=60) | % |
|---|---|---|
| Otorrhea | 58 | 96.5 |
| Otalgia | 7 | 11.6 |
| Hearing Loss | 46 | 76.6 |
| Tinnitus | 15 | 25.0 |

## ASSOCIATED SYMPTOMS

None of the patients presented with fever or facial weakness. Giddiness was complained of by 3 patients (5%) while headache was present in 4 patients(6.7%)




**Graph 2: Associated Symptoms**

## SIZE OF PERFORATION

Of the total 60 patients, 31.7% had subtotal perforation followed closely by large perforations seen in 17 patients (28.3%). Medium sized perforations were seen in 15 patients (25%) and small perforations were present in 9 patients (15%) operated upon following inflation - deflation test.

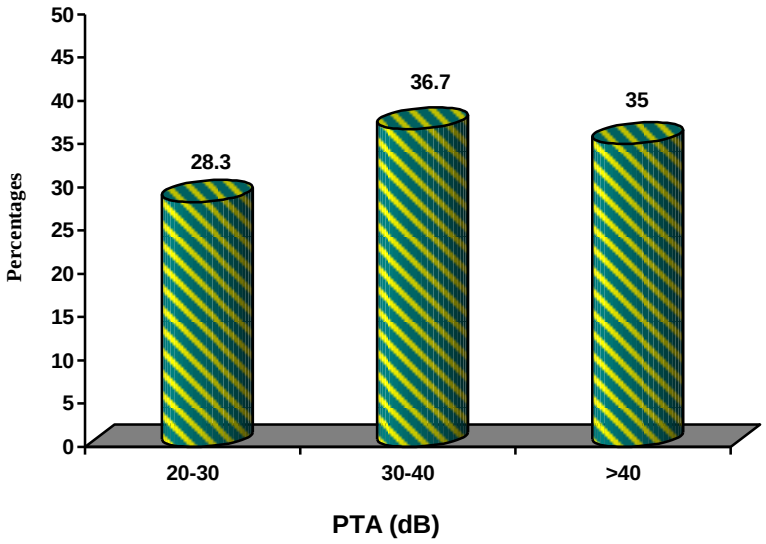
**Table 3: Perforation Size**

| Tympanic Membrane perforation | Number of patients (n=60) | % |
|---|---|---|
| • Subtotal perforation | 19 | 31.7 |
| • Large perforation | 17 | 28.3 |
| • Medium perforation | 15 | 25.0 |
| • Small perforation | 9 | 15.0 |

No signs of complication were seen in any of the patients.

## PURE TONE AVERAGE (PTA)

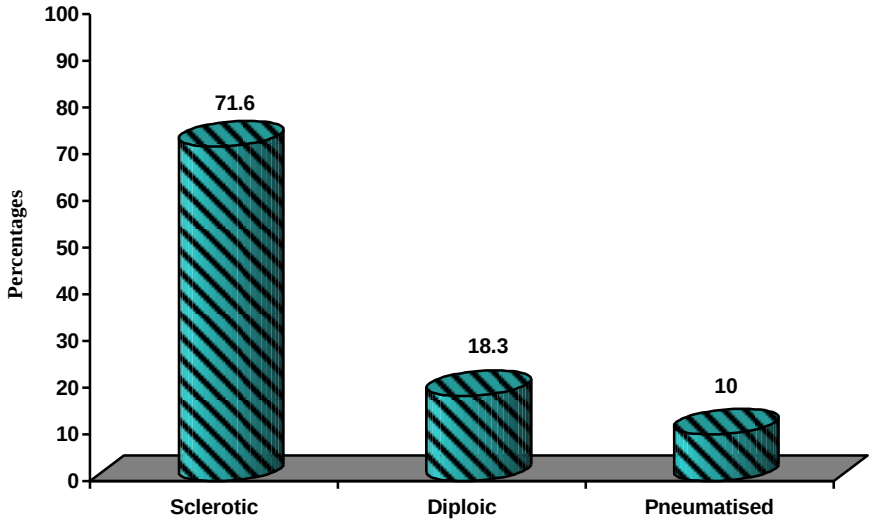
Majority of the patients (72%) had hearing loss above 30dB. 17 patients had PTA in the range of 20-30 dB.




**Graph 3: Pure Tone Average**

## X-RAY MASTOID FINDINGS

Majority (71.6%) of patients had sclerotic mastoids accounting for 43 of 60 patients. 11 patients (18.3%) had diploic mastoid and 6 patients (10%) had pneumatised mastoid




**Graph 4 : X- Ray Mastoid findings**

**INFLATION-DEFLATION TEST**

A pressure of 200 mm$H_2O$ was applied during inflation and deflation test. If negative residual pressure after 5 swallows was between 0 and 99mm $H_2O$, eustachian tube function (IDT) was taken as adequate and residual pressure of ≥100 mm $H_2O$ was taken as poor result.

**Table 4: Results of Inflation –deflation test**

| IDT | Number of patients (n=60) | % |
|---|---|---|
| Adequate | 48 | 80 |
| Poor | 12 | 20 |

Out of 60 patients, 48 were able to equilibrate at least 50% of applied negative pressure while 12 patients were unable to equilibrate even 50% of applied negative pressure with 5 swallows. Therefore, 80% of patients had adequate eustachian tube function as assessed by inflation- deflation test while 20% had poor eustachian tube function.

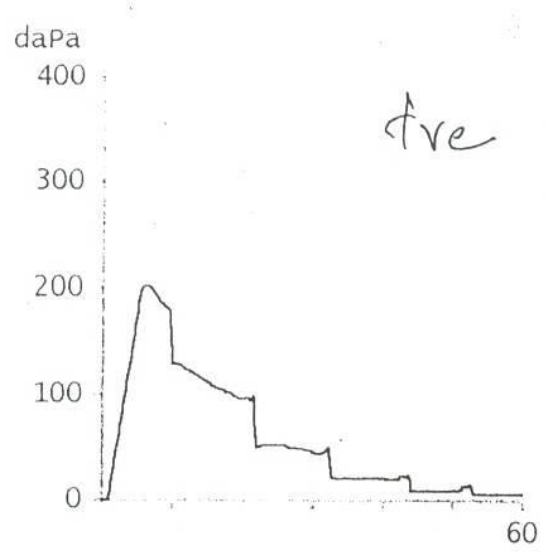



Fig.21 Inflation phase of inflation-deflation test. Step wise reduction of +200 mm $H_2O$ applied positive pressure seen with complete equilibration of applied pressure by 5 swallows in 60 seconds.

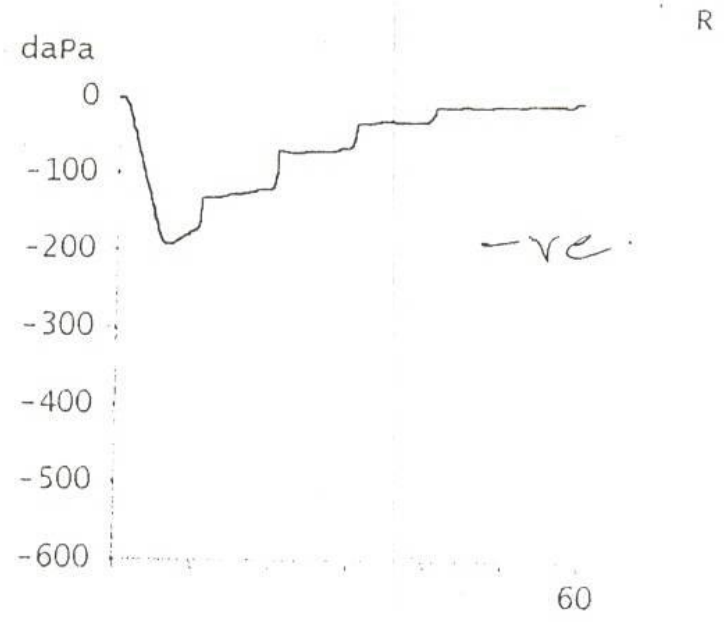



Fig.22 Deflation phase of inflation-deflation test. Step wise reduction of applied negative pressure of -200mm$H_2O$ with 5 swallows in 60 seconds with complete equilibration of applied pressure indicating good Eustachian tube function.

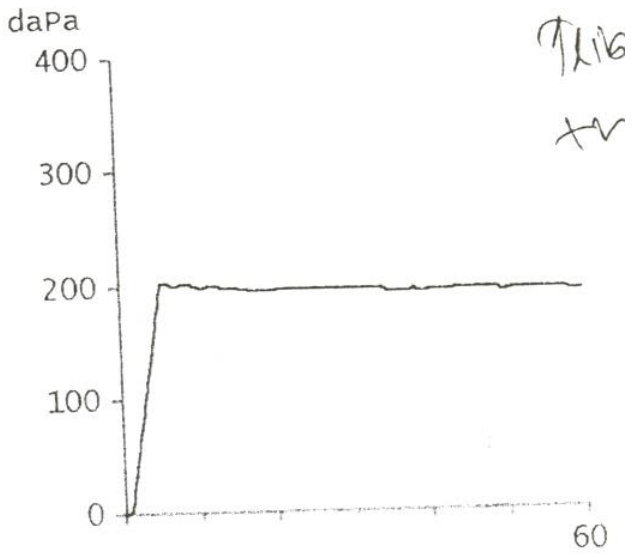



Fig. 23 Inflation phase of inflation-deflation test(IDT) showing no equilibration of applied positive pressure of +200mm $H_2O$ with 5 swallows in 60 seconds (residual pressure of 200mm $H_2O$) indicating poor eustachian tube function.

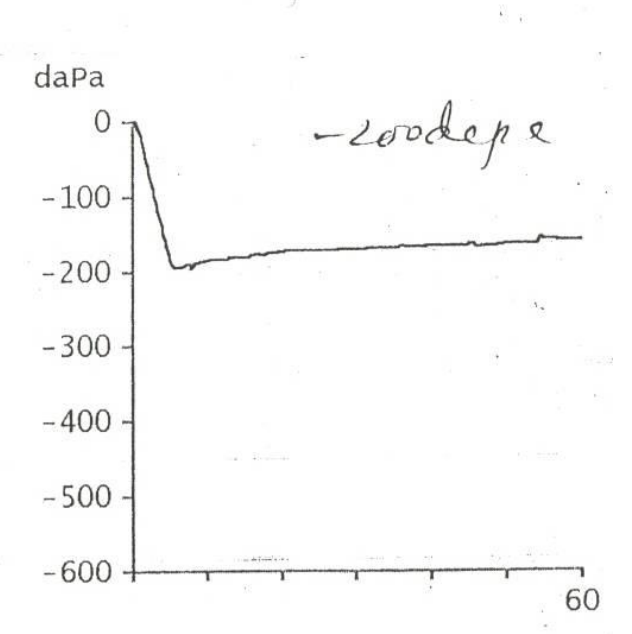



Fig. 24: Deflation phase of inflation-deflation test. No step-ladder pattern seen. No equilibration of applied negative pressure of -200mm $H_2O$ with 5 swallows in 60 seconds indicating poor Eustachian tube function.

## INTRAOPERATIVE FINDINGS OF ADITUS PATENCY

38 patients (63.3%) were found to have patent aditus with free flow of saline intra operatively and blockage of aditus was seen in 22 patients (36.7%).

**Table 5: Intra-operative assessment of aditus patency**

| Aditus | Number of patients | % |
|---|---|---|
| Patent | 38 | 63.3 |
| Blocked | 22 | 36.7 |
| Total | 60 | 100.0 |

## CAUSES OF ADITUS BLOCK

22 of 60 patients had a blocked aditus intraoperatively. The cause of aditus block was hypertrophied mucosa in most patients (59.1%). Oedematous mucosa was seen in 22.7% and tympanosclerotic patch and mucosal polyp were seen in 2 patients each (9.1% each).

**Table 6: Causes of Aditus Block**

| Cause of Aditus Block | Number of patients (n=22) | % |
|---|---|---|
| Hypertrophied mucosa | 13 | 59.1 |
| Oedematous mucosa | 5 | 22.7 |
| Tympanosclerotic patch | 2 | 9.1 |
| Mucosal polyp | 2 | 9.1 |

## CORRELATION BETWEEN IDT AND ADITUS PATENCY

Our study showed, in dry ears, the IDT had a sensitivity of 89.47% in predicting aditus patency. The specificity was low at 36.36%. The positive predictive value of the test was calculated to be 70.83% and the negative predictive value was 66.66%.

**Table 7: Correlation of inflation-deflation test with aditus patency (n=60)**

| IDT | ADITUS PATENT | ADITUS BLOCKED | TOTAL |
|---|---|---|---|
| ADEQUATE ETF | 34 | 14 | 48 |
| POOR ETF | 4 | 08 | 12 |
| TOTAL | 38 | 22 | 60 |

## CONDITION OF MIDDLE EAR MUCOSA

Middle ear mucosa was normal in 47 of 60 patients (78.3%). 13 patients (21.7%) showed abnormal mucosa.

**Table 8: Condition of middle ear mucosa**

| Middle ear mucosa | Number of patients(n=60) | % |
|---|---|---|
| Normal | 47 | 78.3 |
| Abnormal | 13 | 21.7 |

## CAUSES OF ABNORMAL MIDDLE EAR MUCOSA

**Table 9: Cause of abnormal middle ear mucosa**

| Causes of abnormal middle ear mucosa | Number of patients (n=13) | % |
|---|---|---|
| 1. Hypertrophied mucosa | 5 | 38.4 |
| 2. Tympanosclerotic patch | 4 | 30.8 |
| 3. Oedematous mucosa | 3 | 23.1 |
| 4. Mucus in middle ear folds | 1 | 7.7 |

Hypertrophied mucosa was present in 5 patients (38.4%), Tympanosclerosis in 4 patients and oedematous mucosa was seen in 3 patients (23.1%). One patient had mucus in the middle ear folds (7.7%).

## CORRELATION OF X-RAY MASTOID FINDING WITH ADITUS PATENCY

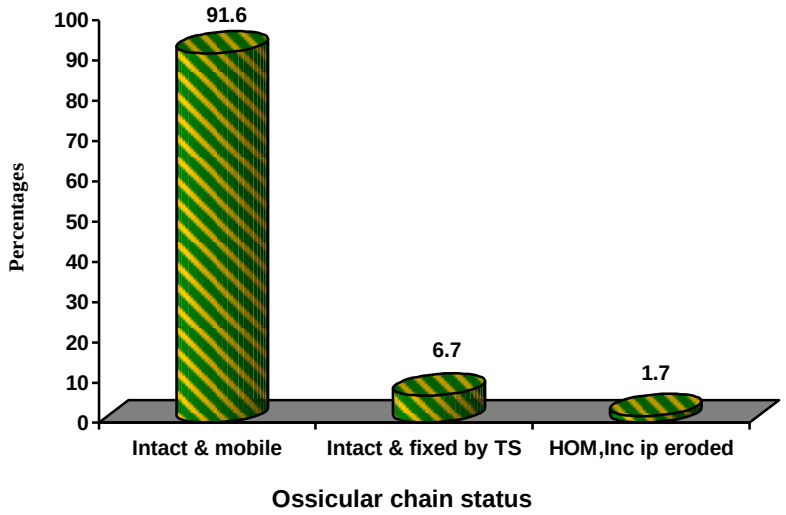
Of a total of 60 patients, mastoid was found to be pneumatised in 10%, diploic in 18.3% and sclerotic in 71.6%. In all 6 patients (100%) with pneumatised mastoid, aditus was found to be patent. Diploic mastoids showed patent aditus in 45.5% and blocked aditus in 54.5% patients while 63% patients with sclerotic mastoids on x-ray had patent aditus, 37.2% patients were found to have aditus blockage.

**Table 10: Correlation of X-ray mastoid with Aditus patency**

| MASTOID | ADITUS PATENT | ADITUS BLOCKED | TOTAL |
|---|---|---|---|
| Pneumatic | 6 | - | 6 |
| Diploic | 5 | 6 | 11 |
| Sclerotic | 27 | 16 | 43 |
| Total | 38 | 22 | 60 |

## OSSICULAR CHAIN STATUS

Ossicular chain was intact and mobile in majority 91.6% of patients. Ossicular chain was intact but fixed in four patients by tympanosclerosis. Erosion of handle of malleus and lenticular process of incus was seen with ossicular discontinuity in one patient (1.7%).




**Graph 5: Ossicular Chain Status**

# DISCUSSION

Eustachian tube plays an important role in maintaining normal aeration of the middle ear. Abnormal Eustachian tube function appears to be the most important factor in the pathogenesis of middle ear disease.[12,36] Currently, Chronic suppurative otitis media (CSOM) of tubotympanic type (TTD) is treated by either tympanoplasty alone or tympanoplasty with adjuvant cortical mastoidectomy.[2]

There remains no consensus of opinion regarding the optimum treatment of patients with otitis media of tubotymanic type. Aerating mastoidectomy can be beneficial in patients with CSOM (TTD) with aditus block or epitympanic or mastoid disease. Restoring the connection between the middle ear & mastoid & by opening up the mastoid by cortical mastoidectomy, pressure buffer can be re-created.[2]

Cortical mastoidectomy, although commonly performed, may be associated with complications such as injury to the tegmen, sinus plate, lateral semicircular canal and facial nerve palsy. These complications, although rare, can be quite harmful to the patient and embarrassing to the surgeon. Moreover, the addition of this procedure to tympanoplasty adds to the operating time, with additional financial burden on the patient.

Obstruction of aditus ad antrum is cited as the indication for an adjuvant cortical mastoidectomy during tympanoplasty. At present, there is no accurate investigation for preoperative evaluation of status of aditus ad antrum.[3] Although, high resolution computed tomography (HRCT) of temporal bone can provide information regarding aditus patency there is a universal reluctance among otologists

to consider preoperative HRCT. Access, cost factors and exposure to radiation may be the deterrents.[5] The association between aeration of mastoid, patency of aditus and eustachian tube function (ETF) is well known.[3] Hence, we propose to determine if eustachian tube function (ETF) as assessed by inflation-deflation test (IDT), would help predict aditus patency preoperatively.

Children less than 12yrs were not included in the study. Studies have reported that the eustachian tube function even in apparently otologically normal children is not as good as in adults (which would contribute to the higher incidence of middle ear disease in children).[12] Some groups recommend avoiding surgery before the age of 7 years, while others recommend waiting until the patient is older than 12years.[37] A meta-analysis of these articles indicates a greater success rate with increased age in the healing of tympanic membrane following tympanoplasty in children.[29]

In our study, 58 of the 60 patients had history of otorrhea. Patients with otorrhea at presentation were treated medically and were included in the study when the ears had become dry. 12% of patients had history of otalgia. Of these, 2 patients presented with otalgia only with no history of otorrhea. Hearing loss was present in 46 patients with most patients complaining of reduced hearing of 1-5 yrs duration. Tinnitus was present in 25% patients for average duration of 1 year.

Most patients (32%) had subtotal perforation. None of the patients had any signs of complications. 72% had hearing loss above 30dB.

Several studies support the contention that mastoid system functions as a pressure buffer.[4,23,12] Sade reports a correlation between mastoid pneumatization & middle ear aeration. 6 patients had pneumatised mastoids in our study.[38] Patency of the aditus was found in100% of our patients with pneumatised mastoids.

Poor mastoid pneumatization is correlated with a number of pathological conditions, and a healthy middle ear space depends to a certain extent on an open mastoid air cell system.[38] 43 patients (68%) had sclerotic mastoid air cell system in our study. This probably contributed to the chronic otitis media (COM) in this patient group.

Aditus was patent in 27 patients (63%) with sclerotic mastoids on radiographic studies while 16 (37.2% ) patients with sclerosed mastoids were found to have aditus blockage. This is similar to Kurien's study where 66.3% of sclerosed mastoids had patent aditus.

Various tests have been employed to evaluate Eustachian tube function. In our study, inflation-deflation five swallow tympanometry test was used to assess ETF as it enables us to assess active opening function of the ET in the presence of perforation or myringotomy.[12]All our patients had dry ears during assessment of Eustachian tube function. In contrast to our study, Kurien et al included both dry and discharging ears in their study and inflation-deflation (Forced Response) test was used to evaluate ETF.

Patients in our study were instructed to swallow "dry" because studies have shown "dry" swallowing to be more effective than "wet" swallowing, although it is more difficult.[30]

Results of Eustachian tube function have been reported in various ways by different authors. In our study, results of negative pressure equilibration were used to assess Eustachian tube function because Sato et al (1990) found that the outcome following surgery correlated better with the results of negative pressure equalization than with the results of positive pressure equalization. They recommended the use of latter test in the prediction and prognosis of successful tympanoplasty.[39]

According to criteria reported by Takahashi, if they could equalize more than half of applied positive middle ear pressure by swallowing, or if they could equalize any of the applied negative pressure after several swallows, those functions were judged as good. Otherwise, those functions were judged as poor.[40]

Various studies have used various residual pressures following swallowing to grade tubal function. While some of these studies use only the negative pressure and grade the tubal function into 3 grades, others have used positive and negative pressures to grade tubal function into 4 groups.[36,41]

In our study. equilibration of 50% of applied negative pressure (from -200 to -100mm $H_2O$) by 5 swallows, as Mc Curdy has made use of, was used to differentiate adequate and poor functions of Eustachian tube.[22] Therefore, in our study, a negative pressure of -200 mm $H_2O$ was applied to middle ear and tubal function was

considered adequate if patient could equilibrate this pressure to a range of 0-99 mm $H_2O$ (50%) with 5 swallows.

48 (80%) patients were able to equilibrate 50% of the applied negative pressure (from -200 to -100 mm $H_2O$), while 12 (20%) patients had residual negative pressure of more than -100 mm $H_2O$. This is in comparison with Elner's study, where by using applied pressure of ±100 mm H2O, 95% of patients could equilibrate positive pressure and 93% could equilibrate negative pressure.[43] However, in another study by Pandey, using forced response modified inflation- deflation test, only 52% of patients could equalize the applied negative pressure of -200mm $H_2O$.[36]

Aditus was patent in 38 patients (63%) and blocked in 22 patients (37%). Aditus mucosa was hypertrophied in 13 patients, oedematous in 5 patients, tympanosclerotic patch was found in 2 and mucosal polyp in 2 patients.

Ossicular chain was intact and mobile in 91.6% of patients. Fixity of ossicles with tympanosclerosis was seen in 4, while one patient had erosion of handle of malleus and lenticular process with ossicular discontinuity. Middle ear mucosa was normal in 47 of 60 patients (78.3%) and 13 patients (21.7%) showed abnormal mucosa. Of the 13 patients with abnormal middle ear mucosa, hypertrophied mucosa was present in 5 patients (38.4%), tympanosclerosis in 4 patients (30.8%) and oedematous mucosa was seen in 3 patients (23.1%). One patient had mucus in the middle ear folds (7.7%).

**Correlation of Inflation- deflation test with aditus patency**

In our study, Eustachian tube function was adequate as assessed by inflation-deflation test and aditus was patent in 34 patients. Therefore, inflation-deflation test was able to predict aditus patency in 34 of 60 patients.

Among the 22 patients with blocked aditus, the inflation-deflation test showed poor eustachian tube function in 8 patients and adequate function in 14 patients. Therefore, inflation-deflation test was able to predict aditus block in 36.36% of patients only.

Gaseous exchange occurs through middle ear mucosa and Eustachian tube. A diseased mucosa can act as a barrier to normal gas exchange and can also block the Eustachian tube. Gas exchange in Eustachian tube is more impaired than exchange via the mucosa in the presence of inflammation.[24]

However, in our study, all patients had dry ears at the time of surgery (no inflammation). Therefore, Eustachian tube function may have been adequate as found on inflation-deflation test even in the presence of blockage of aditus which was due to hypertrophied mucosa.

Studies have also demonstrated that in humans applied pressures to middle ear tend to approach atmospheric pressure even without swallowing.[23]

In our study, inflation-deflation test correlated with aditus patency in 42 patients (34 patients with adequate ETF had patent aditus and 8 patients with poor ETF had blocked aditus). 4 patients with poor eustachian tube function were found to have patent aditus intraoperatively. These patients had residual pressure of ≥150 mm $H_2O$ with none of the patients being able to equilibrate more than 50 mm $H_2O$ of applied negative pressure.

The inability of these patients to equilibrate negative pressure when the aditus was patent could possibly be due to the following reasons:

- Some of the eustachian tubes being judged as having poor function may be able to equalize middle ear pressure with more than 5 swallows & this may be enough for the middle ear pressure regulatory function.[31]
- The Eustachian tube does not necessarily open with every swallow.[27]
- Locking phenomenon: The speed of application of positive and negative pressure is an important variable in testing ETF with the inflation-deflation test. The faster the positive pressure is applied, the higher the opening pressure is. During the deflation phase of the study, the faster the negative pressure is applied, the more likely it is that the locking phenomenon will occur.[12]

Studies have demonstrated that asymptomatic patients (except for aural fullness) may not always have adequate ventilatory function of Eustachian tube on IDT because the pressure changes applied in IDT are not physiological and are too fast. So, some Eustachian tubes may be able to cope with applied negative pressure while some may not. Such inadequately ventilating Eustachian tubes may not equilibrate

rapid pressure changes during flight or aquatic activity where the Eustachian tube is likely to get locked and may not open.[22]

In comparison with study conducted by Kurien et al in which Eustachian tube function as assessed by inflation- deflation (Forced response) test showed a sensitivity of 93% and specificity of 67% in dry ears, our study showed the inflation-deflation test (IDT) had a sensitivity of 89.5% in predicting aditus patency. The specificity of the test was low at 36.36%. Positive predictive value of the test was calculated to be 70.83%, while the negative predictive value was 66.66%.

Bluestone suggested that the forced response test result was more indicative of the active function of the Eustachian tube than is the inflation- deflation test outcome.[12]

# CONCLUSION

1. In our study, the sensitivity of the IDT in predicting aditus patency was 89.5% with positive predictive value of 70%. Therefore, cortical mastoidectomy could probably be avoided in patients who have adequate eustachian tube function indicating patent aditus.

2. The specificity of the IDT in predicting aditus patency was 36.6%, implying that majority of patients with poor ETF also had patent aditus. However, cortical masotidectomy may still be advised in these patients in order not to miss the small group of patients with blocked aditus.

3. A study with a larger sample size may help in further validating the role of inflation - deflation test (IDT) in predicting aditus patency.

# SUMMARY

Chronic suppurative otitis media (CSOM) of tubotympanic type (TTD) is a common presentation at R. L. Jalappa Hospital. Most patients are from poor socio-ecomonic background. Nearly a third of these patients will require surgery as definitive treatment. However, there is no consensus on the surgical options. Currently, TTD is treated by either tympanoplasty alone or tympanoplasty with adjuvant cortical mastoidectomy.

Obstruction of the aditus is cited as the indication for adjuvant cortical mastoidectomy during tympanoplasty. At present, there is no accurate investigation for preoperative evaluation of status of aditus ad antrum. The association between aeration of mastoid, patency of aditus and Eustachian tube function is well known. The purpose of this study was to determine if ETF as assessed by inflation-deflation test (IDT), would help predict aditus patency preoperatively, so unnecessary cortical mastoidectomy can be avoided.

This prospective study was undertaken at R. L. Jalappa Hospital, Kolar, between December 2010 and October 2012. 60 patients with CSOM (TTD) above the age of 12yrs were included. Most of our patients belonged to the age group of 21-30years of which 57% were females. All patients were subjected to inflation-deflation test to assess eustachian tube function. The tubal function was graded as adequate ETF if $\geq$ 50% of applied negative pressure was equilibrated and poor if equilibration was less than 50%.

80% of our patients showed adequate tubal function on IDT while 20% had poor function. A standard tympanoplasty with cortical mastoidectomy was done and patency of aditus checked. Results of the IDT were then correlated with the status of aditus intraoperatively. Aditus was found patent in 63.3% of patients and blocked in 36.7%.

The sensitivity of the inflation-deflation test in predicting aditus patency was 89.5% . The specificity of the test was low at 36.6% with positive predictive value of 70.8% and negative predictive value of 66.6%.

From our study, it can be concluded that while cortical mastoidectomy may be avoided in patients with adequate Eustachian tube function, it would be advisable to consider it in patients with poor Eustachian tube function.

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# PROFORMA OF THE CASE SHEET

## I PERSONAL DETAILS

Name | Age | Sex

Address | DOA | DOD

Occupation | Hospital No.

## II PRESENTING COMPLAINT

Ear Discharge: Y/N R/L/B Duration

Ear ache : Y/N R/L/B Duration

Decreased Hearing : Y/N R/L/B Duration

Giddiness : Y/N Fever: Y/N Headache : Y/N

Tinnitus: Y/N R/L/B Duration

Facial Weakness: Y/N R/L/B Duration

## III HISTORY OF PRESENT ILLNESS

### Ear discharge

Onset: Type: intermittent/continuous Quantity: scanty/profuse

Quality: mucoid/ mucopurulent/purulent/bloody Odour: odorless/foul smelling

Any precipitating factors: cold/ pharyngitis/ tonsillitis

### Earache:

Onset: insidious/sudden Location: Preauricular/postauricular/intra-auricular

Character: dull /moderate/severe Intermittent/continuous

Decreased Hearing:

Onset: insidious/sudden
Progressive/Nonprogressive

Does hearing improve with otorrhea

Associated Symtoms

Swelling around the ear: Y/N Headache Giddiness

Fever Tinnitus

IV PAST HISTORY

Was otorrhea in the past ever associated with vertigo/ headache / vomiting

h/o features suggestive of chronic sinusitis/ chronic tonsillitis/ adenoid hypertrophy

DM/HTN/TB/BA

V FAMILY HISTORY

Did any member of the family have chronic discharging ear or any complications of chronic discharging ear

Any h/o TB or any chronic illness in the family

VI PERSONAL HISTORY

H/o swimming: Y/N Loss of appetite: Y/N Disturbed sleep: Y/N

Bowel/ bladder disturbances: Y/N Smoking: Y/N Alcohol: Y/N

VII GENERAL PHYSICAL EXAMINATION

Built: poor/moderate/well Nutritional status: poor/satisfactory

Temperature Pulse BP RR

Pallor:Y/N Icterus:Y/N Clubbing:Y/N Edema:Y/N Lymphadenopathy:Y/N

VIII SYSTEMIC EXAMINATION

Central Nervous System : Higher mental functions

Cranial Nerves

Respiratory System

Cardiovascular System

Per Abdomen

IX ENT EXAMINATION

EAR

| Sl No. | EAR | Right | Left |
|---|---|---|---|
| 1. | Pinna: deformity/ scar | | |
| 2. | Preauricular area | | |
| 3. | Post auricular area | | |
| 4. | External auditory meatus | | |
| 5. | Tympanic membrane:<br>Perforation:<br>Granulations<br>Polyp<br>Pulsatile discharge<br>Condition of tympanic remnant | | |
| 6. | Status of middle ear mucosa | | |
| 7. | Clinical tests of Hearing<br>• Rinne's test<br>• Weber's test<br>• Absolute bone conduction | | |
| 8. | Vestibular function tests<br>Spontaneous nystagmus<br>Fistula test | | |
| 9. | Facial nerve | | |

NOSE & PNS

External Nose

Anterior Rhinoscopy

Posterior Rhinoscopy

PNS tenderness

THROAT

Oral cavity & Oropharynx

IDL

NECK

XII CLINICAL DIAGNOSIS

Tubotympanic disease active/quiescent/inactive with CHL/MHL

XIII INVESTIGATIONS

Examination under microscope

Aural Swab – for culture & sensitivity

Routine blood/urine examination

X-ray Mastoids: Lateral Oblique View – Pneumatic/ sclerotic/ Diploeic

Pure Tone Audiometry: CHL/MHL Pure tone average

Inflation- Deflation Test: Eustachian tube function – adequate/poor

XIV TREATMENT

Surgical - Tympanoplasty with Cortical mastoidectomy

Intraoperative findings:

a. Aditus : patent/blocked
b. Cause of aditus block when present
c. Mastoid aeration: pneumatised/ diploeic/sclerotic
d. Mastoid disease when present
e. Middle ear mucosa
f. Status of ossicular chain : M± / I± / S±

# KEY TO MASTER CHART

O – Otorrhea

OT – Otalgia

HL - Hearing Loss

T – Tinnitus

G- Giddiness

F- fever

H- headache

FW- Facial weakness

MT – mastoid tenderness

TM perf – Tympanic membrane perforation

FS – Fistula sign

FN-Facial nerve

X- x-ray mastoid

IDT- Inflation-Deflation test

A- Aditus

CAB – cause of aditus block

MM- middle ear mucosa

O-ossicular chain status

Ad- adequate

C – childhood

m- months

y – years

Lg- large

N- normal

Sm- small

Mm- medium

ST- subtotal

Pn- pneumatised

S-sclerotic

D- diploic

P-patent

PTA- pure tone average

Bl- block

TS – Tympanosclerotic patch

Inc – Incus

Lp – long process

erod – eroded

N' - negative residual pressure (deflation Phase)

Oe mucosa – oedematous mucosa

HOM – handle of malleus

Muc poly – mucosal polyp

W – weeks

Mal –malleus

i & m – intact & mobile

M- male

F- female

# MASTER CHART

| Sl. No | NAME | Hospital Number | Age | Sex | Symptoms | | | | | | | | | | | | Signs | | | | | PTA | M | | I | A | CAB | MM | OS |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | O | | OT | | HL | | T | | G | F | H | FW | MT | TM perf | | FS | FN | | R | L | | | | | |
| | | | | | R | L | R | L | R | L | R | L | | | | | | R | L | | | | | | | | | | |
| 1 | Khaja Hussein | 675568 | 27 | M | - | - | + | - | 1y | - | - | - | - | - | - | - | - | Lg | N | - | N | 35 | Pn | S | Ad | P | - | N | Intact & mobile |
| 2 | Srinivas C | 616998 | 47 | M | 18m | - | - | - | 6m | - | + | - | - | - | - | - | - | Sm | N | - | N | 28.6 | S | S | Ad | P | - | N | Intact & mobile |
| 3 | Mohammed H.S. | 642478 | 40 | M | 6m | - | + | - | 6m | - | + | - | - | - | - | - | - | Mm | N | - | N | 31.6 | Pn | Pn | Ad | P | - | N | Intact & mobile |
| 4 | Shyamalamma | 695525 | 42 | F | 1y | C | - | - | 1y | 1y | - | - | - | - | - | - | - | Mm | Lg | - | N | 38.3 | S | S | Ad | P | - | N | Intact & mobile |
| 5 | Rajesh H.K. | 684743 | 26 | M | 10y | - | - | - | 6m | - | + | - | - | - | - | - | - | Mm | N | - | N | 25 | S | Pn | Poor | P | - | H mucosa | Intact & mobile |
| 6 | Yarab Baba | 729833 | 18 | M | 13y | 13y | - | - | 6m | 6m | - | - | - | - | - | - | - | Lg | Sm | - | N | 26.6 | D | D | Poor | P | - | N | Intact & mobile |
| 7 | Lakshmidevamma | 649577 | 55 | F | - | 6m | - | - | - | 6y | - | + | - | - | - | - | - | N | Mm | - | N | 65 | Pn | S | Ad | Bl | H mucosa | H mucosa | Intact & mobile |
| 8 | Radhamma | 661786 | 30 | F | 1y | - | - | - | - | - | - | - | - | - | - | - | - | Mm | N | - | N | 23 | Pn | Pn | Ad | P | - | N | Intact & mobile |
| 9 | Krishnappa K.M. | 737502 | 77 | M | 8y | 8y | - | - | - | 1y | - | - | - | - | - | - | - | N | Mm | - | N | 66 | S | S | Ad | P | - | N | Intact & mobile |
| 10 | Karthik A | 736739 | 18 | M | C | - | - | - | 3y | - | - | - | - | - | - | - | - | Lg | N | - | N | 26.3 | S | S | Ad | P | - | oedematous | Intact & mobile |
| 11 | Lakshman T | 632271 | 38 | M | C | - | - | - | 1y | - | - | - | - | - | - | - | - | ST | N | - | N | 36 | S | Pn | Poor | Bl | TS | N | Intact & mobile |
| 12 | Shadika D | 741768 | 25 | F | C | - | - | - | 2y | - | + | - | - | - | - | - | - | ST | N | - | N | 35 | D | Pn | Ad | Bl | Muc poly | N | Intact & mobile |
| 13 | Girija | 806781 | 30 | F | 2y | 2w | + | - | 1y | - | + | - | 1m | - | - | - | - | ST | Sm | - | N | 38.3 | S | S | Ad | p | - | N | Intact & mobile |
| 14 | Wasim Akram | 684467 | 19 | M | C | C | - | - | - | - | - | - | - | - | - | - | - | Lg | ST | - | N | 28.3 | S | S | Ad | P | - | N | Intact & mobile |
| 15 | Lakshmappa M | 738910 | 24 | M | - | 10y | - | - | - | 8m | - | - | - | - | - | - | - | N | Lg | - | N | 30 | S | S | Ad | P | - | N | Intact & mobile |
| 16 | Nagarathnamma C | 777997 | 30 | F | 18m | - | + | - | 1y | - | - | - | - | - | - | - | - | ST | N | - | N | 26.6 | D | Pn | Ad | P | - | N | Intact & mobile |
| 17 | Nagalingappa | 685745 | 24 | M | 3y | C | - | - | - | 1y | - | - | - | - | - | - | - | N | Lg | - | N | 21.6 | D | D | Poor | P | - | N | Intact & mobile |
| 18 | Ashwathi PR | 800462 | 16 | F | - | 1y | - | - | - | - | - | - | - | - | - | - | - | N | Mm | - | N | 38.3 | Pn | S | Ad | P | - | N | Intact, Inc Lp erod |
| 19 | Arthi Devi K | 793344 | 30 | M | 2y | - | - | - | 18m | - | - | - | - | - | - | - | - | Lg | N | - | N | 40 | S | S | Ad | P | - | N | Intact & mobile |
| 20 | Susheelamma | 769463 | 40 | F | - | 3m | - | - | - | 2m | - | - | - | - | - | - | - | N | Lg | - | N | 33.3 | Pn | Pn | Ad | P | - | N | Intact & mobile |
| 21 | Geethashree V | 783351 | 17 | F | 15y | - | - | - | - | - | - | - | - | - | - | - | - | Lg | N | - | N | 45 | S | Pn | Ad | Bl | H mucosa | N | Intact & mobile |
| 22 | Sugunamma | 785056 | 20 | F | 13y | - | - | - | 7y | - | - | - | - | - | - | - | - | ST | N | - | N | 40 | D | Pn | Ad | Bl | Oe muco | N | Intact & mobile |
| 23 | Meenakshi | 799085 | 41 | F | 7m | - | - | - | 7m | - | + | - | - | - | - | - | - | Lg | N | - | N | 38.3 | S | Pn | Ad | Bl | H mucosa | N | Intact & mobile |
| 24 | Nalina | 805634 | 21 | F | - | 1y | - | - | - | 1y | - | - | - | - | - | - | - | N | Lg | - | N | 40 | S | S | Ad | P | - | N | Intact & mobile |
| 25 | Sampath kumar | 699193 | 22 | M | - | 5y | - | - | - | - | - | - | - | - | - | - | - | N | Sm | - | N | 26 | D | S | Poor | Bl | Oe muco | Oedematous | Intact & mobile |
| 26 | Yelasappa B.N. | 740943 | 55 | M | - | 1y | - | - | - | 1y | - | - | - | - | - | - | - | N | Lg | - | N | 36.6 | Pn | D | Poor | Bl | H mucosa | N | Intact & mobile |
| 27 | Shashikala | 776105 | 36 | F | 2m | C | - | - | - | - | - | - | - | - | - | - | - | Lg | ST | - | N | 50 | S | S | Ad | P | - | N | HOM, Lp eroded |
| 28 | Murugeshwari K | 770362 | 38 | F | 6m | - | - | - | 6m | - | - | - | - | - | - | - | - | Mm | N | - | N | 28.3 | S | D | Poor | P | - | N | Intact & mobile |
| 29 | Guru singh | 703034 | 19 | M | 4y | 4y | - | - | 6m | 6m | - | - | - | - | - | - | - | Lg | Lg | - | N | 46.3 | S | S | Ad | Bl | H mucosa | N | Intact & mobile |

| | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 30 | Shobha M | 736200 | 23 | F | 10y | - | - | - | 3m | - | - | - | - | - | - | - | - | Sm | N | - | N | 55 | D | Pn | Poor | Bl | TS | N | Intact & fixed by TS |
| 31 | Khadar Pasha | 768060 | 30 | M | 12y | 12y | - | - | 3m | 3m | - | - | - | - | - | - | - | Lg | ST | - | N | 40 | S | S | Poor | Bl | H mucos | N | Intact & mobile |
| 32 | Varalakshmamma | 704757 | 48 | F | 3y | - | - | - | 1y | 1y | - | + | - | - | - | - | - | Sm | N | - | N | 60 | S | S | Ad | P | - | N | Intact & mobile |
| 33 | Gopala Krishnaiah | 773458 | 61 | M | 10y | - | - | - | 2m | - | - | - | - | - | - | - | - | ST | N | - | N | 55 | S | D | Ad | P | - | N | Intact & mobile |
| 34 | Madhusudhan M.N | 706549 | 19 | M | 3y | 2y | - | - | 2y | - | - | - | - | - | - | - | - | ST | Mm | - | N | 41.6 | S | Pn | Ad | Bl | H mucosa | N | Intact & fixed |
| 35 | Mohammed Abrar | 699570 | 13 | M | 11y | 11y | - | - | 6y | 6y | - | - | - | - | - | - | - | ST | ST | - | N | 53.3 | S | S | Ad | Bl | H mucosa | N | Intact & mobile |
| 36 | Indramma C | 768416 | 35 | F | - | 3w | - | - | - | - | - | - | - | - | - | - | - | N | Mm | - | N | 46.6 | S | S | Ad | P | - | N | Intact & mobile |
| 37 | R.Shantharaj | 722209 | 39 | M | 3m | 10y | - | - | - | - | - | - | - | - | - | - | - | Mm | Lg | - | N | 32 | D | D | Ad | Bl | H mucosa | N | Intact & mobile |
| 38 | Ashwini | 724511 | 20 | F | 10y | - | + | - | 1y | - | - | + | - | - | - | - | - | Mm | N | - | N | 33.3 | S | Pn | Ad | P | - | N | Intact & mobile |
| 39 | Parvathamma | 724976 | 24 | F | - | 1m | - | - | - | 1m | - | + | - | - | - | - | - | N | Sm | - | N | 40 | P | S | Ad | P | - | N | Intact & mobile |
| 40 | Jyothi | 737295 | 30 | F | - | - | - | + | - | - | - | + | - | - | - | - | - | N | Sm | - | N | 26.6 | P | S | Ad | P | - | N | Intact & mobile |
| 41 | Jayanthi J | 704081 | 38 | F | 1y | - | - | - | - | - | - | - | 2w | - | - | - | - | Mm | N | - | N | 40 | S | S | Ad | P | - | N | Intact & mobile |
| 42 | Byramma | 609433 | 70 | F | - | 20y | - | - | 20y | 2y | - | - | - | - | - | - | - | N | ST | - | N | 75 | S | S | Ad | P | - | N | Intact & fixed by TS |
| 43 | Arthi G | 759235 | 35 | F | 6y | 6m | - | - | 1m | 1m | - | - | - | - | - | - | - | Sm | Sm | - | N | 26.3 | S | S | Ad | P | - | N | Intact & mobile |
| 44 | Nagarathnamma | 722740 | 60 | F | 3y | 3y | - | - | - | - | - | - | - | - | - | - | - | ST | ST | - | N | 50 | S | S | Ad | Bl | Oe muco | N | Intact & mobile |
| 45 | Nabeesa A | 763283 | 23 | F | - | 2y | - | - | - | 1y | - | - | - | - | - | - | - | N | Mm | - | N | 43.3 | P | D | Ad | P | - | N | Intact & mobile |
| 46 | Malini S | 755888 | 19 | f | - | 1m | - | - | - | 1m | - | + | - | - | - | - | - | N | Mm | - | N | 30 | P | P | Ad | P | - | N | Intact & mobile |
| 47 | Srinivas B | 713285 | 42 | M | - | 2y | - | - | - | 2y | - | - | - | - | - | - | - | N | Mm | - | N | 58.3 | P | S | Ad | Bl | H mucosa | N | Intact & mobile |
| 48 | Naveen Kumar | 754154 | 24 | M | - | C | - | - | - | C | - | - | - | - | - | - | - | N | ST | - | N | 46.6 | D | D | Ad | Bl | Oe muco | N | Intact & mobile |
| 49 | Sukumar S | 748851 | 23 | M | - | 10y | - | - | - | 3y | - | + | - | - | - | - | - | N | ST | - | N | 51 | P | S | Ad | P | - | N | Intact + fixed |
| 50 | Shaheen Taj | 756036 | 20 | F | 14y | - | - | - | 2y | - | - | - | - | - | - | - | - | Lg | N | - | N | 45 | S | P | Poor | Bl | Muc poly | N | Intact & mobile |
| 51 | Ruby Ayesha | 757120 | 19 | F | C | C | - | - | 1y | 1y | + | - | - | - | - | - | - | ST | ST | - | N | 35 | S | S | Ad | Bl | H mucosa | N | Intact & mobile |
| 52 | Manjunath M | 696291 | 20 | M | - | 2m | - | - | - | - | - | - | - | - | - | - | - | N | Sm | - | N | 26.6 | S | S | Ad | P | - | N | Intact & mobile |
| 53 | Bhuvaneshwari A | 711073 | 29 | F | - | C | - | - | - | 1y | - | - | - | - | - | - | - | N | ST | - | N | 46.6 | P | S | Ad | P | - | N | Intact &mobile |
| 54 | Manjula | 715649 | 30 | F | - | C | - | - | - | 1y | - | - | - | - | - | - | - | N | ST | - | N | 38.3 | D | S | Ad | P | - | N | Intact & mobile |
| 55 | Venkatamma G | 739226 | 40 | F | - | 1y | - | - | - | - | - | - | - | - | + | - | - | N | Lg | - | N | 55 | P | S | Poor | Bl | H mucosa | N | Mal tip erod, i & m |
| 56 | Murthy M | 612369 | 28 | M | 17y | - | - | - | 18m | - | - | - | - | - | + | - | - | ST | N | - | N | 25 | S | S | Ad | Bl | H mucosa | N | Intact & mobile |
| 57 | Mrithyunjay singh | 743559 | 25 | M | 10y | 10y | - | - | - | - | - | - | - | - | - | - | - | Mm | Sm | - | N | 26.6 | P | P | Ad | P | - | N | Intact & mobile |
| 58 | Venkatamma | 710789 | 30 | F | 2m | - | - | - | 2m | - | - | + | + | - | - | - | - | Sm | N | - | N | 55 | S | S | Poor | Bl | Oe muco | N | Intact & mobile |
| 59 | Jayamma KA | 803103 | 50 | F | - | 8y | - | + | - | 1y | - | - | - | - | - | - | - | N | Mm | - | N | 31.6 | P | D | Ad | P | - | N | Intact & mobile |
| 60 | Rajagopal | 773535 | 52 | M | - | C | - | - | - | 2y | - | - | - | - | + | - | - | N | Lg | - | N | 35 | P | S | Ad | P | - | N | Mal tip erod, i & m |