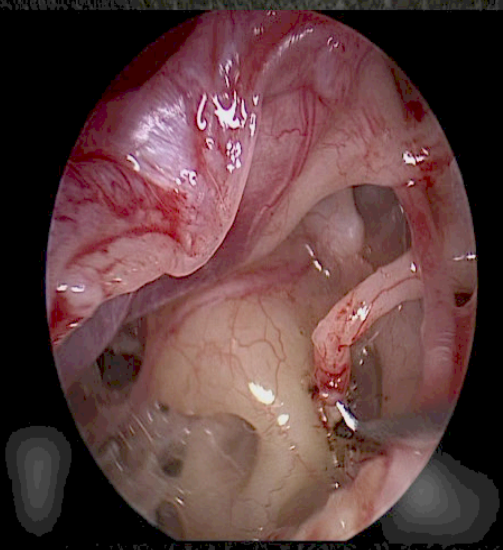
Diseases of Middle Ear



Balasubramanian Thiagarajan

Preface

The concept of authoring books as small modules has enticed the author to start writing taking advantage of this format. Otology is a highly developed science and post graduate students need to be trained in this field to deliver excellent services to the patient at large. This book covers all aspects of middle ear diseases with special emphasis on treatment.

While authoring this book the author had faced an enormous dilemma i.e where to start and where exactly to stop. The concept of writing a book as a water tight compartment really helped the author in surmounting this problem. The author has managed to confine himself only to middle ear anatomy, middle ear diseases and their management. Relevant topics covering basic anatomy, embryology, pathology and microbiology have been dealt with on a need to know basis.

This book is targetted towards the need of post graduate students in otolaryngology. Care is taken to ensure that current concepts and literature are included in this book. The chapter wise organization of this book will lead the student towards better understanding of this topic.

Illustrations and pictures have been included where ever necessary to provide better understanding. The author has taken enough care to make this book really simple and easy to understand.

The author would like to place on record his profuse thanks to his family members for have supported him tirelessly during this project.

About The Author

Dr Balasubramanian Thiagarajan served as professor of otolaryngology in Stanley Medical College Chennai India. He has never spared any effort in taking his knowledge to the student in which ever way possible. What started initially as well intended educational CDrom release, did not hesitate to take up internet revolution for efficient knowledge transmission. The author is hosting a number of sites catering to knowledge transfer in the field of otolaryngology.

Currently the author has started authoring books on various topics in otolaryngology. All these books take up a modular format for easy readability.

The author can be contacted at Email.

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Introduction

Non mammalian amniotes have only one middle ear ossicle known as columella auris in contrast to the mammalian middle ear which has three ossicles.

What are amniotes?

This term indicates a group of tetrapod vertebrates which include reptiles, birds and mammals that lay their egg on land or retain the fertilized egg within the mother. They are distinguished from anamniotes which lay their eggs in water.

Evolutionary biology attaches lots of importance to these evolutionary changes. Animals are unable to generate new anatomical elements de novo in evolution. According to the French anatomist Geoffroy Saint-Hilaire equivalent sets of skeletal elements are connected in an identical order in all animals. Changes if any are manifested by changes occurring to this common skeletal platform. This theory is known as theory of principe des connexions. As per this theory homologues for two of the three ossicles identified in a mammalian ear should be identified.

Fossil studies have proved that mammalian middle ear gradually transformed from the primary jaw joints. There are two evolutionary steps involved in Definitive Middle ear evolution. The first step is the medio lateral separation of the malleus and incus from the dentary and the second step being loss of anterior connection to the dentary resulting from absorption of Meckel's cartilage during later stages of evolution.

Reichert theory:

This theory proposed by Reichert proposed that malleus and incus dervied from first branchial arch cartilage.

Fuchs theory:
Fuchs suggested that based on studies involving congenital abnormalities that malleus and incus developed from second arch.

Vertebrates are unique because of the presence of ectodermally derived neural crest cells form the ectomesenchyme. Ectomesenchyme incidentally has been considered to be the source of craniofacial skeleton.

The pharyngeal arch ectomesenchyme originates from thee neural crest area ranging from midbrain to hind brain. Neural crest cells arising in this region are separated into three main streams that migrate into the mandibular, hyoid and branchial arches located posterior to the inner ear. Hox genes play a vital role in this phase of development.

Hox genes are a subset of homeotic genes. This group of genes control the body plan of an embryo along the head-tail axis. After the formation of embryonic segments the Hox proteins determine the type of appendages (like legs, antennae and wings in fruit flies or the different types of vertebrae in humans that will form on a segment). Hox proteins confer segmental identity, but are not known to form the actual segments themselves. The term Hox is actually a contraction of homeobox in the field of genetics. Hox genes can be considered to be akin to a play director who decides which scene the actors should carry out next.

The protein product of each Hox gene is a transcription factor. Each Hox gene contains a well conserved DNA sequence known as the homeobox. In majority of animals the organization of Hox genes in the chromosome is the same as the order of their expression along the anterior-posterior axis of the developing animal.

Drosophilia melanogaster is an important model for understanding body plan generation and evolution. The broad principles of Hox gene function and logic that occurs in these flies will apply nearly to all organisms. This insect like all other insects has eight Hox genes. These genes are clustered into two complexes. Both these complexes are located in chromosome 3.

Antennapedia complex:

This complex consists of 5 genes.
Labial (lab)
Proboscipedia (pb)
Deformed (Dfd)
Sex combs reduced (Scr)
Antennapedia (Antp)

Bithorax complex:

This complex consists of three genes.
Ultrabithorax (Ubx)
Abdominal A (abd-A)
Abdominal B (abd-B)

Labial:

The lab gene is the most anteriorly expressed gene. It is expressed in the head, primarily in the intercalary segment and also in the midgut. Loss of function of this gene results in the failure of Drosophilia embryo to internalize the mouth and head structures that initially develop on the outside of its body. This process is called head involution. Failure of head involution disrupts / deletes the salivary glands and pharynx.

Proboscipedia:

The pb gene is responsible for the formation of labial and maxillary palps.

Deformed:

The Dfd gene is responsible for the formation of the maxillary and mandibular segments in the larval head. Loss of function of Dfd in the embryo results in a failure of head involution with loss of larval head structures. Mutations in the adult have either deletions of parts of the head or transformations of head to thoracic cavity.

Sex combs reduced:

The scr gene is responsible for cephalic and thoracic development in drosophila embryo and adult.

Antennapedia:

The second thoracic segment (T2) develops a pair of legs and a pair of wings. The Antp gene specifies this identity by promoting leg formation and allowing wing formation. Abnormalities of this gene causes failure of development of antenna and development of leg coming out of the fly's head.

Ultrabithorax:

This gene is responsible for formation of third thoracic segment which includes a pair of legs and a pair of smaller wings known as halters.

Abdominal - A:

The abd-A gene is expressed along most of the abdomen. Expression of this gene is necessary to specify the identity of most of the abdominal segments. This gene also affects the pattern of cuticle generation in the ectoderm and pattern of muscle generation in the mesoderm.

Abdominal - B:

Gene abd-B is transcribed into two different forms i.e. a regulatory protein and a morphogenic protein. Regulatory abd-B suppresses embryonic ventral epidermal structures in the 8th and 9th segments of Drosophila abdomen. Both the regulatory and morphogenic protein are involved in the development of the tail segment.

The presence of middle ear is rather unique to terrestrial animals. The primary function of middle ear is to offset the decrease in acoustic energy that would occur if the low impedance ear canal air directly contacted with the high impedance cochlear fluid. When sound wave is transmitted from a low impedance medium (air) to one of high impedance (cochlear fluid containing water) considerable amount of energy is reflected back from the interface separating these two media. About 99% of sound gets reflected back at the leve of this interface if impedance matching function of the middle ear is absent.

Anatomy of Middle Ear

Synonyms: Middle ear cleft, Tympanum

The middle ear cleft includes the tympanum (middle ear cavity proper), the eustachean tube, and the mastoid air cell system. The tympanic cavity is an air filled irregular space contained within the temporal bone. It also contains the three auditory ossicles (malleus, incus and stapes) along with their attached muscles. For the purpose of description the tympanic cavity may be considered as a box with four walls, a roof and a floor. The corners of this hypothetical box is not sharp.

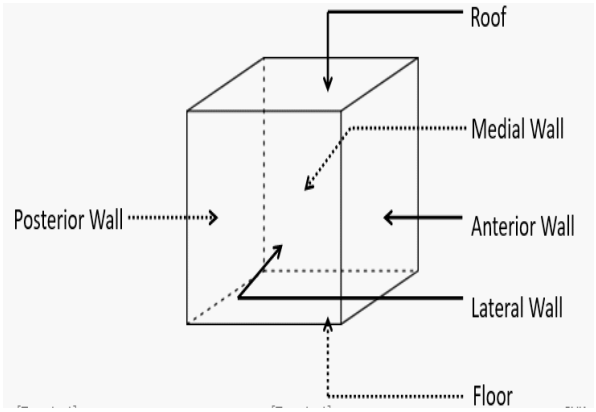
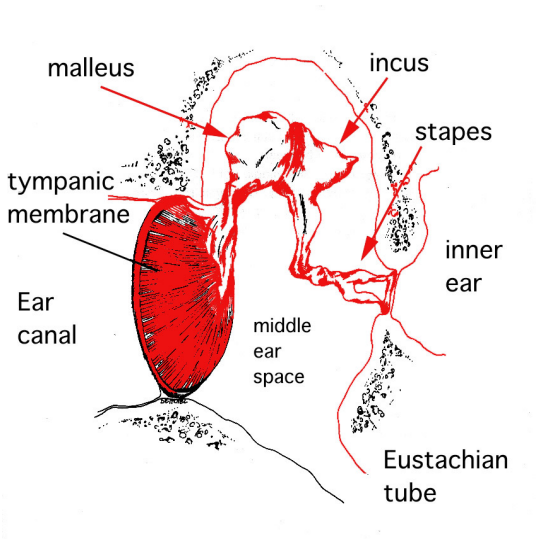


Diagram explaining the Box structure of middle ear cavity



Diagrammatic representation of middle ear cavity

Lateral wall:

The lateral wall of the tympanum / middle ear is partly bony and partly membranous. The central portion of the lateral wall is formed by the tympanic membrane, while above and below the tympanic membrane there is bone, forming the outer lateral walls of the epitympanum (attic) and hypotympanum respectively. The lateral wall of the epitympanum (attic) also includes that part of the tympanic membrane lying above the anterior and posterior malleolar folds - this portion of the ear drum is also known as pars flaccida.

This portion of the tympanic membrane lacks the middle fibrous layer, hence the name. The lateral attic wall (bony portion) is wedge shaped, its lower portion is also called the outer attic wall (scutum). Scutum actually means sheild in latin. This bony portion is thin and its lateral surface forms the superior portion of the deep portion of the external meatus.

Three openings are present in the bone of the medial surface of the lateral wall of the tympanic cavity. The first opening is the posterior canaliculus for the chorda tympani nerve. This opening is situated at the junction between the lateral and posterior walls of the tympanic cavity. This opening is usually present at the level of the upper end of the handle of the malleus. This opening leads to the bony canal which descends through the posterior wall of the tympanic cavity. Since chorda tympanic nerve traverses this canal it is also known as the canal for chorda tympani nerve. This canal also contains a branch from the stylomastoid artery which usually accompanies the chorda tympani nerve.

The second opening is the petrotympanic (Glaserian) fissure. This fissure opens anteriorly just above the attachment of the tympanic membrane. This opening is infact a small slit about 2 mm long. It receives the anterior malleolar ligament. It also transmits the anterior tympanic branch of the maxillary artery to the tympani cavity.

The third is the canal of Hugier. It lies medial to the Glaserian fissure. The chorda tympani nerve enters through this.

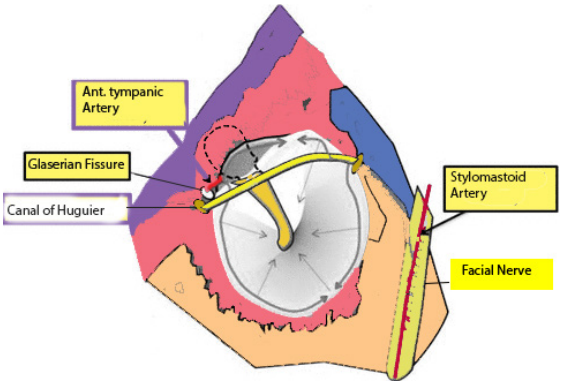


Diagram showing Glasserian fissure

Roof:

The roof of the middle ear cavity is formed by the tegmen tympani. It is this tegmen tympani which separates the middle ear cavity from the dura of the middle cranial fossa. This tegmen tympani is formed in part by the petrous portion of the temporal bone, and the squamous portion of the temporal bone. The suture line between these two components is known as the petrosquamous suture line. This suture line is unossified in the young, and does not close until adult life is reached. Through this suture veins from the middle ear may pass to the superior petrosal sinus.

Floor:

The floor is much narrow. In fact it is narrower than the roof of the middle ear cavity. This portion of the middle ear cavity lies in close relationship with the jugular bulb. The middle ear cavity is separated from the jugular bulb by a thin piece of bone. Rarely, the floor may be deficient and the jugular bulb in these patients is separated from the middle ear cavity only by fibrous tissue and mucous membrane. At the junction of the floor and the medial wall of the middle ear there is a small opening which allows the entry of tympanic branch of glossopharyngeal nerve to pass into the middle ear. This nerve takes an important part in the formation of tympanic plexus.

Anterior wall:

The anterior wall of the tympanic cavity is very narrow. This is because the medial and lateral walls converge anteriorly. The anterior wall can be divided into two portions; the upper and lower portions. The lower portion of the anterior wall is larger than the upper portion. It has a thin plate of bone which separates this portion from the internal carotid artery as it enters the skull. This plate has two openings for the carotico tympanic nerves. The upper opening transmits the superior carotico tympanic nerve and the inferior opening transmits the inferior carotico tympanic nerve. It is through these nerves that sympathetic nerves reach the tympanic plexus. The upper smaller part of the anterior wall has two tunnels placed one below the other. The upper tunnel transmits the tensor tympani muscle, and the lower tunnel transmits the bony portion of the Eustachian tube.

Medial wall:

The medial wall separates the middle ear from the inner ear. The most prominent portion of the medial wall of the middle ear cavity is the promontory. It is a rounded projection occupying most of the central portion of the medial wall of the middle ear. This projection is raised by the underlying basal turn of the cochlea. The promontory has numerous small grooves on its surface. These grooves contain the tympanic plexus of nerves. Behind and above the promontory is the oval window (fenestra vestibuli). This is a oval shaped opening connecting the tympanic cavity with the vestibule. In life this is closed by the foot plate of stapes and its surrounding annular ligament. The long axis of the fenestra vestibuli is horizontal. Its inferior border is concave. The size of the oval window varies, but on an average it is 3.25mm long and 1.75 mm wide. Above this fenestra vestibuli is the canal for facial nerve (horizontal portion) and below lies the promontory. Hence the fenestra vestibuli lies at the bottom of a depression also known as fossula that can be of varying depths depending on the position of the facial nerve and the prominence of the promontory.

The fenestra cochlea (round window) lies just below and behind the oval window. It is closed in life by a membrane known as the round window membrane (secondary tympanic membrane). The secondary tympanic membrane appears to be divided into an anterior and posterior portions by the presence of a transverse thickening. The diameter of the round window membrane is between 1.8 to 2.3 mm. It is made up of three layers; the outer mucosal, middle fibrous and an inner endothelial layer. The membrane of the fenestra cochleae does not lie at the end of the scala tympani but forms part of its floor.

The ampulla of the posterior semicircular canal is the closest vestibular structure to this membrane. The nerve supplying the ampulla of the posterior semicircular canal (singular nerve) lies close to this secondary tympanic membrane. The secondary tympanic membrane forms a landmark for the position of the singular nerve. This is useful during surgical procedures like singular neurectomy for treatment of intractable vertigo. These two windows (oval & round) are separated by the posterior extension of the promontory. This is known as the subiculum. Rarely a spicule of bone arises from the promontory above the subiculum and runs to the pyramid on the posterior wall of the middle ear cavity. This spicule of bone is known as the ponticulus. The round window faces inferiorly and a little posteriorly, lying completely under the cover of the promontory and hence usually is difficult to visualise.

The round window niche is usually triangular in shape, having anterior, posterosuperior and posteroinferior walls. The posterosuperior and posteroinferior walls meet posteriorly leading on to the sinus tympani. This sinus tympani is a difficult area to visualise.

Cholesteatoma may lurk in this area making it difficult to remove. This is one of the commonest causes of cholesteatoma recurrence after mastoidectomy. Small mirrors known as the zinne mirror can be used to visualise this area indirectly. Since sinus tympani lies under the pyramid, removal of the pyramid during surgery will bring the sinus tympani area into view.

The facial nerve canal is another important anatomical structure present in this wall. This nerve runs above the promontory and fenestra vestibuli in an anteroposterior direction. The canal may occasionally be deficient leaving an exposed facial nerve. This is a dangerous anatomical variant because this nerve can easily be traumatised during any surgical procedures in the middle ear cavity. Even infections of the middle ear mucosa can cause facial nerve palsy in patients with an exposed facial nerve. The anterior end of the facial nerve canal is marked by the presence of a bony process known as processus cochleariformis. This curved projection of bone is concave anteriorly and it houses the tendon of the tensor tympani muscle as it turns laterally to the handle of the malleus. Behind the fenestra vestibuli, the facial nerve turns inferiorly to begin its descent in the posterior wall of the tympani cavity.

The region above the level of the facial nerve canal forms the medial wall of the epitympanum or attic. The dome of the lateral semicircular canal extends a little lateral to the facial canal and is the major feature of the posterior portion of the epitympanum. In well pneumatised bones this dome of the lateral canal can be very prominent.

Posterior wall:

The posterior wall of the middle ear is wider above than below. In its upper part it has an important opening known as the aditus. This aditus helps the middle ear communicate with the mastoid air cell system. Aditus is a large irregular opening connecting the mastoid antrum to the middle ear cavity. Below the aditus is a small depression known as the fossa incudis. Fossa incudis houses the short process of the incus. Below the fossa incudis lies the pyramid.

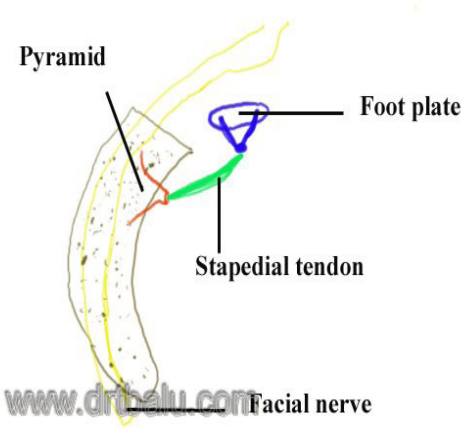


Figure showing Pyramid

Pyramid is a small conical projection which is hollow and its apex pointing anteriorly. It contains the stapedius muscle, the tendon of which passes forwards to insert into the neck of the stapes. The canal within the promontory curves downwards and backwards to join the descending portion of the facial nerve canal. Between the promontory and the tympanic annulus is the facial recess. The facial recess is bounded medially by the facial nerve and laterally by the tympanic annulus.

Running through the wall between the two with varying degrees of obliquity is the chorda tympani nerve. This nerve always run medial to the tympanic membrane. Drilling over the facial recess area between the facial nerve and the annulus in the angle formed by the chorda tympani nerve can lead into the middle ear cavity. This surgical approach to the middle ear cavity through this area is known as the facial recess approach. This approach is suitable for surgeries involving the round window niche like placement of electrodes during cochlear implant procedures. Hypotympanum can also be approached through this approach.

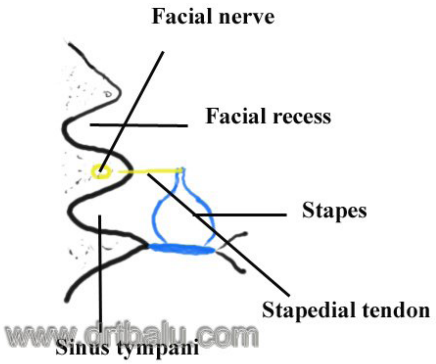


Figure showing sinus tympani of middle ear

Contents of the middle ear:

The most important content of the middle ear is air. The air flows into the middle ear through a patent Eustachian tube. The other contents are:

Chain of three ossicles which help in sound transmission; the malleus, incus and stapes. Two muscles, chorda tympani nerve and the tympanic plexus of nerves.

Malleus:

This bone is shaped like a hammer hence the name. This is the largest of the three ossicles of the middle ear cavity. It has a head, neck and three processes arising from below the neck. The overall length of the malleus ranges between 7.5 - 9 mm. Its head lies in the attic region of the middle ear effectively dividing the attic into an anterior portion and a posterior one. The anterior portion lie anterior to the handle of the malleus, while the posterior portion lie behind the handle of the malleus. During surgical procedures for attic cholesteatoma clipping of this head will improve the exposure in the attic region. The head of the malleus on its posteriomedial surface has an elongated saddle shaped cartilage covered facet for articulation with the incus. This articular surface is constricted near its middle dividing the articular facet into a larger superior and a smaller inferior portions. The inferior portion of the articular facet lies at right angles to that of the superior portion. This projecting lower portion is also known as the cog or spur of the malleus. Below the neck the bone broadens and gives rise to the following: the anterior process from which a slender anterior ligament arises to insert into the petrotympanic fissure; the lateral process which receives the anterior and posterior malleolar folds from the annulus tympanicum, and the handle which runs downwards, medially and slightly backwards between the mucous and fibrous layers of the tympanic membrane. On the deep medial surface of the handle there is a small projection into which the tendon of the tensor tympani muscle inserts. Additionally the malleus is supported by the superior ligament which runs from the head to the tegmen tympani.

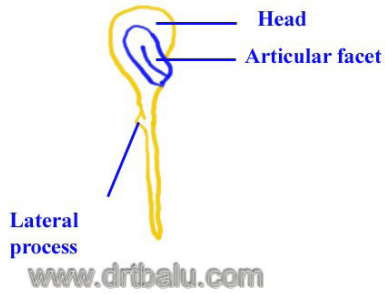


Diagram showing the Malleus

Incus:

This bone is shaped like an anvil. It articulates with the malleus and has a body and two processes. The body lies in the attic and has a cartilage covered articular facet corresponding to that of the malleus. The short process projects backwards from the body to lie in the fossa incudis. It is in fact attached to the fossa incudis by a short ligament. The long process of the incus descends into the mesotympanum behind and medial to the handle of the malleus. At its tip there is a small medially directed lenticular process which articulates with the stapes. The long process of the incus has precarious blood supply. This portion of the incus is prone for undergoing necrosis in disease conditions.

The stapes:

The stapes consists of a head, neck, two crura and a base (footplate). The head of the stapes points laterally and has a small cartilage covered depression for articulation with the lenticular process of the incus. The tendon of the stapedius muscle attaches to the posterior part of the neck and the upper part of the posterior crura. The neck of the stapes gives rise to two crura, the anterior crura is thinner and less curved than the posterior crura. The two crura join the foot plate which closes the oval window during life. The average dimensions of the foot plate is 3mm x 1.4mm. The long axis of the foot plate is almost horizontal, with the posterior end being slightly lower than the anterior.

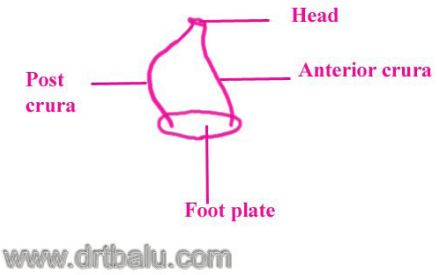


Diagram showing the Stapes

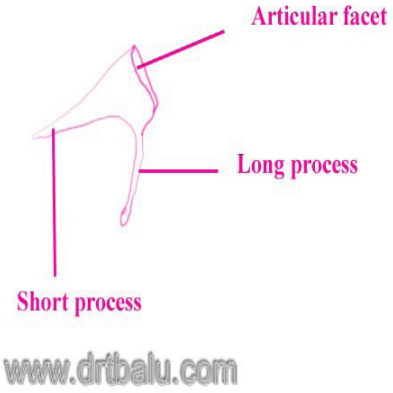


Figure showing incus

Muscles of the middle ear:

Stapedius muscle: arises from the walls of the conical cavity within the pyramid. A slender tendon emerges from the apex of the pyramid and inserts into the stapes. This muscle is supplied by a small branch from the facial nerve. The stapedial tendon is inserted into the neck of the stapes. On contraction this muscle rocks the stapes backwards holding it firm against the annular ligament preventing excessive transmission of sound into the inner ear. This muscle has a protective role to play. It protects the inner ear from insults caused by loud noise. Patients with facial nerve palsy have hyperacusis because of lack of action of this muscle.

Tensor tympani muscle: This long slender muscle arises from the walls of the bony canal which lie above the canal for the Eustachian tube. Parts of the muscle also arise from the cartilaginous portion of the Eustachian tube and the greater wing of sphenoid. From these origins the muscle passes backwards into the tympanic cavity lying on the medial wall of the middle ear just below the level of the facial nerve.

The bony covering of the canal is often deficient in its tympanic segment where the muscle is replace by its tendon. This tendon enters the processus cochleariformis, turns at right angles inserting into the medial aspect of the upper end of the handle of the malleus. This muscle is supplied by the mandibular nerve by way of a branch from the medial pterygoid nerve, which passes through the otic ganglion without synapsing. This muscle tenses the tympanic membrane by holding the handle of the malleus thus helping the middle ear in better sound perception.

Chorda tympani nerve:

This is a branch of the facial nerve. It enters the middle ear cavity through the posterior canaliculus which is present at the junction of the lateral and posterior walls. It runs across the medial surface of the tympanic membrane between the mucosal and fibrous layers passes medial to the upper portion of the handle of the malleus. Here it lies above the tendon of the tensor tympani muscle, continues forwards and leaves by way of the anterior canaliculus placed within the petrotympanic fissure. It joins the lingual branch of the V nerve with which it is distributed to the anterior 1/3 of the tongue.

Tympanic plexus:

Is found over the promontory. It is formed by the tympanic branch of the glossopharyngeal nerve, carotico tympanic nerves which supplies the sympathetic component. The tympanic plexus provide the following branches:

1. Branches to the mucous membrane lining the tympanic cavity, Eustachian tube, mastoid antrum and its air cells

2. A branch joining the greater superficial petrosal nerve.

3. The lesser superficial petrosal nerve, which contain all the parasympathetic fibers of the IX nerve. This nerve leaves the middle ear through a small canal below the tensor tympani muscle where it receives parasympathetic fibers from the VII nerve by way of a branch from the geniculate ganglion. The full nerve passes through the temporal bone to emerge lateral to the greater superficial petrosal nerve on the floor of the middle cranial fossa, outside the dura. It then passes through the foramen ovale with the mandibular nerve and accessory meningeal artery to the otic ganglion. Post ganglionic fibers from the otic ganglion supply secretomotor fibers to the parotid gland by way of the auriculotemporal nerve.

The mucosal lining of the middle ear cavity is varies according to the location. The attic or the epitympanum is lined by pavement epithelium, while the middle ear proper is lined by cuboidal epithelium and the hypotympanum is lined by ciliated columnar epithelium.

Anatomy of Chorda tympani nerve

Introduction:

The chorda tympani nerve is a branch of facial nerve. It exits the facial nerve just before it exits via the stylomastoid foramen. It is one of the three cranial nerves that is involved in transmission of taste fibers from the tongue. Chorda tympani nerve conveys taste fibers from the anterior 2/3 of tongue. Mechanism of taste sensation is rather unique in that it involves a complicated feed back loop with each nerve acting to inhibit the signal of other nerves. The chorda tympani exerts strong inhibitory influence on other taste fibers as well as pain fibers from the tongue. When chorda tympani nerve is damaged its inhibitory function is disrupted, causing the other taste fibers to act in an uninhibited manner.

The chorda tympani nerve carries with it two types of fibres which traverse via lingual nerve to reach their destination.

These fibers include:

Special sensory fibers providing taste sensation from anterior 2/3 of tongue.

Presynaptic parasympathetic fibers to submandibular ganglion providing secretomotor fibers to submandibular and sublingual salivary glands

Presynaptic parasympathetic fibers is also supplies the blood vessels of the tongue. When stimulated the chorda tympani nerve causes dilatation of blood vessels of the tongue.

Central connections:

Chorda tympani nerve contains fibers from two brain stem nuclei.

They are:

Superior salivatory nucleus: This nucleus houses cell bodies of secretomotor preganglionic parasympathetic neurons

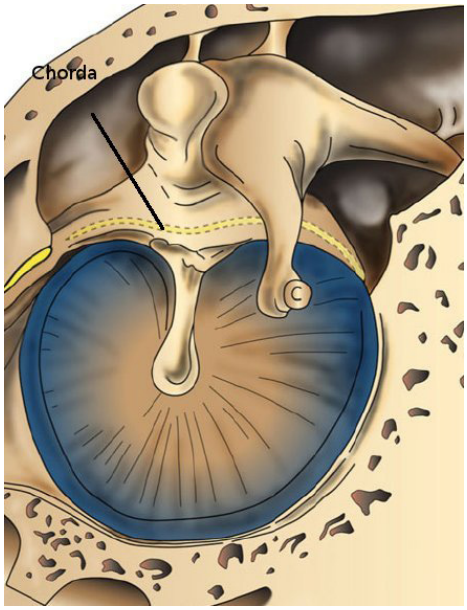
Nucleus of tractus solitarius: The superior portion contributes to chorda tympani fibers. It receives central processes of taste neurons which have their cell bodies in the ganglia of the three cranial nerves conveying taste sensation. After synapsing in this nucleus secondary axons ascend in the lateral lemniscus to relay in the thalamus. This pathway then passes through the posterior limb of internal capsule to reach the primary gustatory cortex.

Connections seen within the facial canal:

Sensory branch of facial nerve Nervus intermedius of Wrisberg joins the facial nerve here. It conveys special sensory fibers from taste buds present in the anterior 2/3 of tongue and soft palate. It also contains secretomotor fibers to salivary glands present below the oral cavity. Nerves intermedius exits the brain stem adherent to the vestibulo cochlear nerve. At the level of internal auditory meatus it leaves this nerve to merge with that of facial nerve.

Chorda tympani nerve exits from the facial nerve before the facial nerve exits via the stylomastoid foramen. It is the largest branch of facial nerve in its intrapetrous compartment. It arises below the nerve to stapedius. It traverses antero superiorly via the posterior canaliculus usually accompanied by posterior tympanic branch of stylomastoid artery.

This canaliculus opens into the middle ear cavity through an aperture situated at the junction of posterior and lateral walls of tympanic cavity. This opening lies just medial to the fibrocartilaginous annulus. The posterior canaliculus is roughly 0.5 mm in diameter. Chorda tympani nerve shows a large number of variations. In some patients the chorda tympani nerve may arise from more proximal portion of facial nerve, even close to the geniculate ganglion. The length of the posterior canaliculus is also highly variable ranging from 3 – 14 mm. In 10% of individuals there may not be a posterior canaliculus at all but could be replaced by a groove.



If the chorda tympani nerve originates outside the temporal bone then the posterior canaliculus will be separate from that of the facial nerve canal. In fetus and young infants the chorda tympani nerve leaves the facial nerve outside the skull, but the postnatal growth of mastoid process causes it to migrate to a more proximal position. Since the facial canal grows more than the mastoid segment of facial nerve the chorda tympani nerve typically diverges from the facial nerve in an infant of 1 yr of age.

Course of chorda tympani in the tympanum:

The chorda tympani arches across pars flaccida medial to the upper part of the handle of malleus and traverses above the insertion of tensor tympani. In patients with congenital anomalies of malleus chorda is also displaced laterally.

Chorda tympani nerve exits the middle ear via a separate bony canal, the anterior canaliculus also known as the canal of Hugier. This canal runs in the medial portion of petrotympanic fissure. Anterior tympanic branch of maxillary artery accompanies this nerve along this canal. Chorda exits the skull through a small foramen behind the base of spine of the sphenoid. At its exit it is closely related to the medial surface of temporomandibular joint.

4. Supplies secretomotor fibers to the parotid gland

5. Supplies efferent vasodilator fibers to the tongue

Embryology of Middle ear and its mucosal folds

Introduction:

Middle ear is anatomically not a single cavity, but a space containing different compartments. The middle ear cavity per se is divided into 5 compartments.

1. Mesotympnaum in the centre
2. Epitympanum superiorly
3. Protympanum anteriorly
4. Hypotympanum inferiorly
5. Retrotympanum posteriorly

Embryology:

In developing human the tympanomastoid system appears in the 3rd week of gestation from an outpouching of the first pharyngeal pouch called the tubotympanic recess. The endodermal tissue of the dorsal end of this pouch becomes the eustachean tube and the tympanic cavity. By the 7th week, a concomitant growth of the second branchial arch constricts the midportion of the tubotympanic recess, placing the primary tympanic cavity lateral to this constriction and the primordial eustachean tube medial to this construction. The future development of the eustachean tube is marked by lengthening, narrowing, and mesodermal chondrification establishing the fibrocartilaginous Eustachian tube.

The terminal end of the tubotympanic recess buds into four sacci:

Saccus anticus
Saccus medius
Saccus superior
Saccus posticus

Table showing the origin of different middle ear spaces is given in the next page.

These sacci expand progressively to replace middle ear mesenchyme and mastoid mesenchyme. The walls of the expanding sacci envelop the ossicular chain and line the walls of the middle ear cavity. The interface between two sacci gives rise to several mesentery like mucosal folds. These mucosal folds transmit blood vessels and ligaments to middle ear contents.

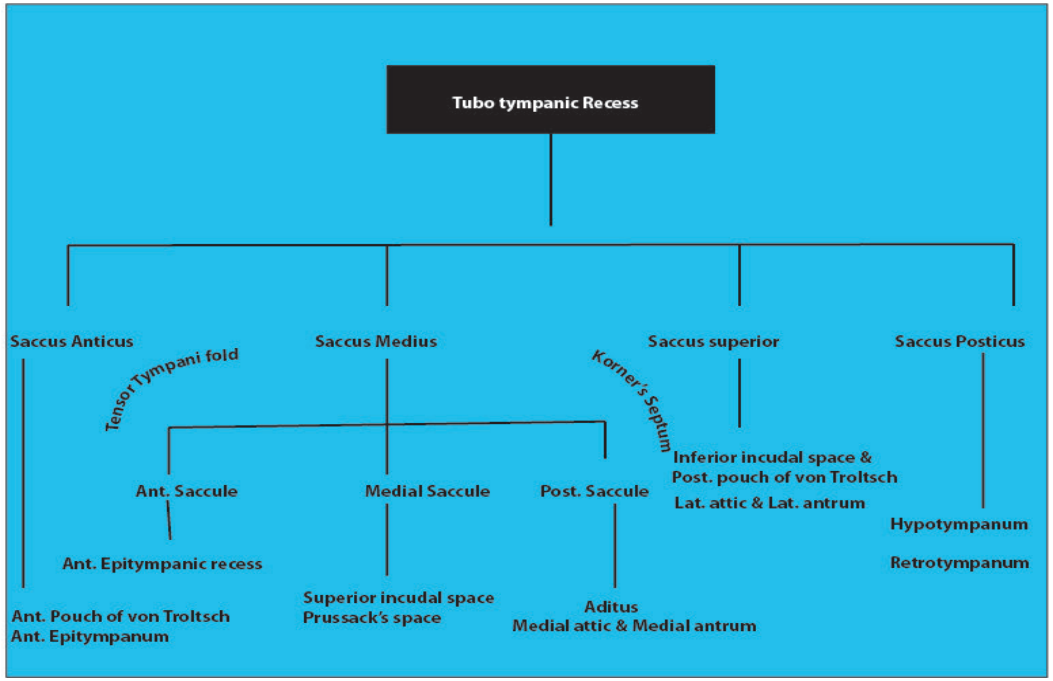
Saccus anticus:

This is the smallest of all saccus. It extends upward anterior to the tensor tympani tendon to form the anterior epitympanic recess and the anterior pouch of von Troltsch. At the level of the tensor tympani muscle canal, it fuses with anterior saccule of saccus medius to form an important mucosal fold, the tensor tympani fold. This fold separates the anterior epitympanic recess superiorly from the supratubal recess inferiorly.

Saccus Medius:

This saccus forms the attic. it extends upward and divides into three saccules:
Anterior saccule - It develops upward to form the anterior compartment of the attic.

Medial saccule - This saccule forms the superior incudal space by its growth over the incudo malleal bodies and the posterior incudal ligament. The medial saccule sends an offshoot forward to form the Prussak's space.


Tubo tympanic Recess
Saccus Anticus
Saccus Medius
Saccus superior
Saccus Posticus
Tensor Tympani fold
Korner's Septum
Ant. Saccule
Medial Saccule
Post. Saccule
Inferior incudal space &
Post. pouch of von Troltsch
Lat. attic & Lat. antrum
Hypotympanum
Retrotympanum
Ant. Epitympanic recess
Superior incudal space
Prussack's space
Aditus
Medial attic & Medial antrum
Ant. Pouch of von Troltsch
Ant. Epitympanum

Posterior saccule - It extends posteriorly between the long process of incus and the stapes to form the medial portion of mastoid antrum which is derived from the petrous portion of temporal bone.

Saccus superior:

This saccus forms posterior pouch of von Troltsch and the inferior incudal space. It extends posteriorly and laterally between the handle of malleus and long process of incus to form the posterior pouch of von troltsch, the inferior incudal space and lateral part of the antrum which derives from the squamous portion of temporal bone. The plane of fusion between the posterior saccule of saccus medius which forms the medial part of air cell system and the saccus superior which forms the lateral part of mastoid air cell system. This plane usually breaks down and the mastoid air cell system becomes unified. If this process of breakdown fails then a bony septum persists between these two parts and is known as the Korner's septum.

Saccus posticus:

This saccus extends along hypotympanum and rises up posteriorly to form the round window niche, oval window niche, the facial recess and the sinus tympani. The sinus tympani has variable sizes and depth posteriorly, this is dependent on the degree of extension of saccus posticus under the stapedial tendon during fetal development.

Protympanum:

This is actually a middle ear compartment that lies anterior to the frontal plane drawn through the anterior margin of the tympanic annulus. This the most anterior portion of the middle ear cavity. Superiorly it is bounded by the orifice of Eustachean tube. Anteriorly it is bounded by the canal for internal carotid artery.

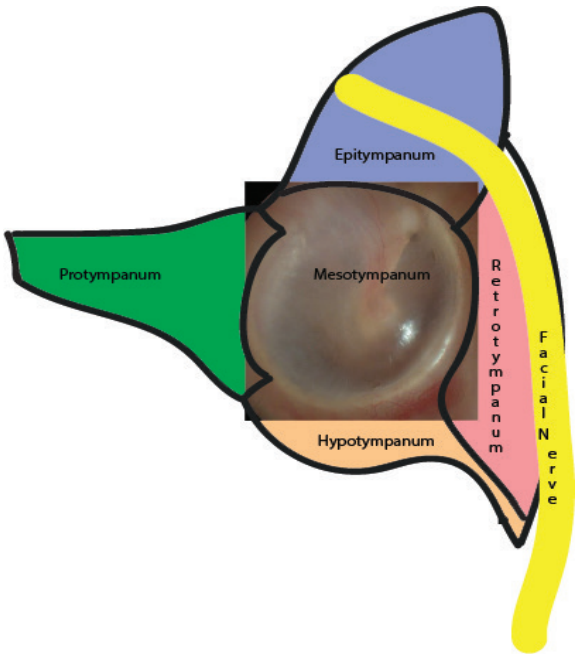


Diagram showing parts of middle ear

Some authors tend to include the entire bony portion of the Eustachean tube under protympanum. The bony portion of the Eustachean tube is nearly 1 cm long in adults.

The lateral wall of protympanum consists of a thin plate of the tympanic bone called the lateral lamina. This plate of bone separates protympanum from the mandibular fossa. Medial wall of protympanum is bounded by cochlea posteriorly and carotid canal anteriorly.

The roof is bounded by the bony canal for tensor tympani muscle and the tensor tympani fold which separates the protympanum from anterior attic.

Supra tubal recess:

This recess is a superior extension of protympanum. This recess corresponds to the space lying between the superior border of tympanic orifice of Eustachean tube and tensor tympani fold. It lies just below the anterior attic separated by the tensor tympani fold. This is also known as the anterior epitympanic recess.

This space lies anterior to malleus. It is partitioned from the attic proper by a coronally oriented bony septum known as the cog. This spicule of bone is suspended from the anterior petrosal tegmen.

Upward expansion of bony Eustachean tube to form the supra tubal recess begins at the late fetal age and continues throughout childhood. This is very much in contrast to the growth of tympanic cavity, the attic and the mastoid antrum which are fully developed at birth. It was also demonstrated that anterior epitympanic recess are found even in poorly pneumatized temporal bones suggesting that it develops independently of the temporal bone air cell system.

Tono et al described two types of supra tubal recess.

1. Single dome shaped supra tubal recess : This occurs as a single dome shaped extension of Eustachean tube.

2. Air cell shaped supra tubal recess. In this type petrosal cells surround the true anterior epitympanic recess.

Even though carotid canal and cochlea lie close to this recess medially the most important structures related are tympanic portion of facial nerve and the geniculate fossa. This portion of the facial nerve canal lie in the medial wall of this recess above the canal for tensor tympani muscle. Owing to this relationship even a mild enlargement of the facial nerve canal may fill or deform the anterior epitympanic recess. Common causes of enlargement of this segment of facial nerve include facial nerve schwannoma and small hemangiomas involving the facial nerve canal. Presence of persistent stapedial artery causes enlargement of tympanic segment of facial nerve canal causing narrowing of the anterior epitympanic recess.

Persistent stapedial artery is a rare vascular anomaly. Embryologically primitive second aortic arch gives rise to hyoid artery. Hyoid artery in turn gives rise to stapedial artery. Failure of stapedial artery to involute causes this situation. Since this artery is known to pass close to the foot plate of stapes, injury to this vessel during stapes surgery could cause troublesome bleeding.

Presence of cholesteatoma in this recess can easily be overlooked leading on to recurrence even after surgical removal.

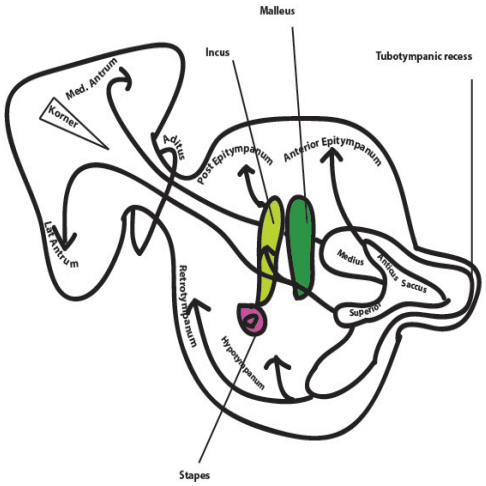
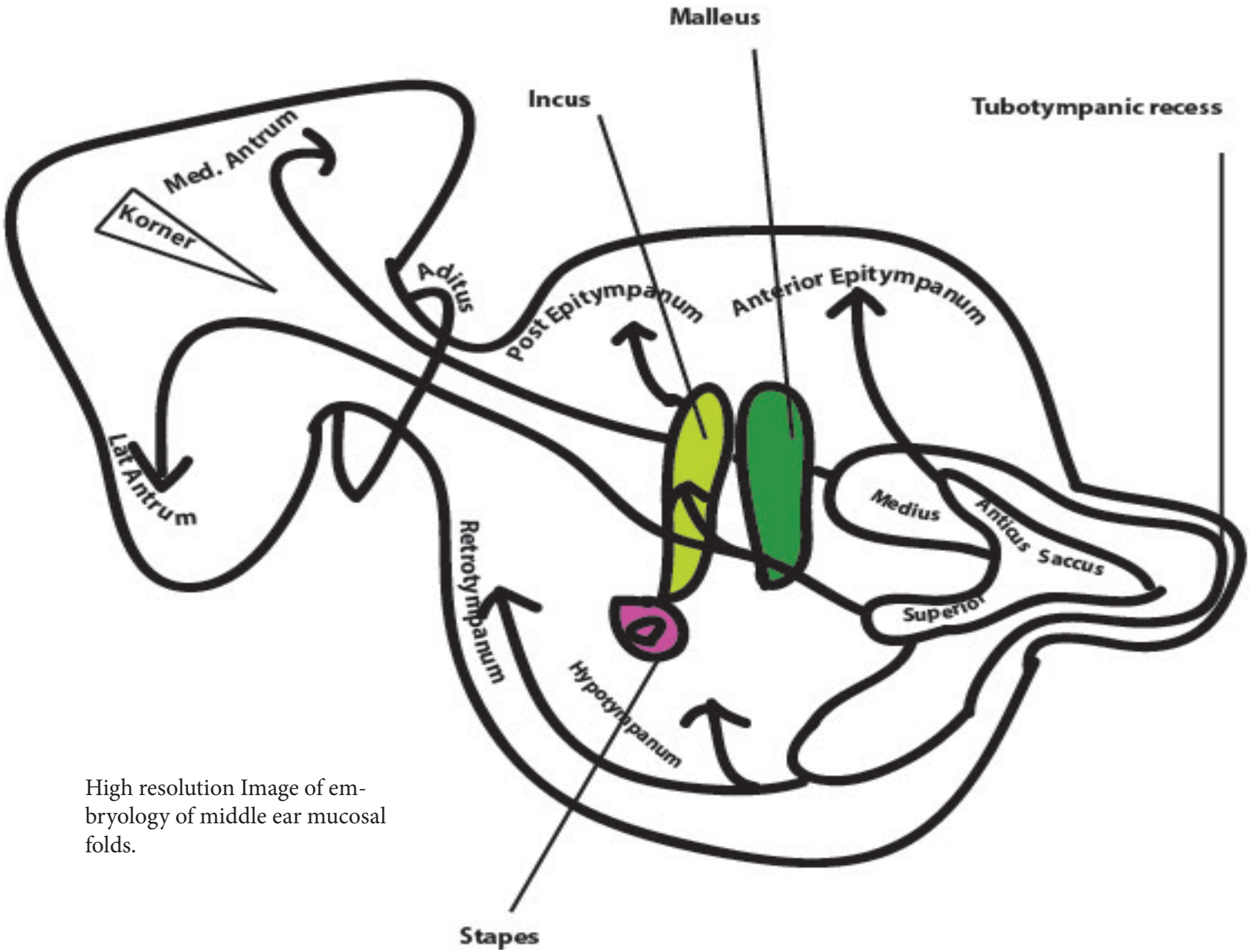


Figure showing embryology of middle ear mucosal folds



High resolution Image of embryology of middle ear mucosal folds.

Anatomy of Hypotympanum

This is a crescent shaped space located at the bottom of the middle ear cavity. It lies below the horizontal plane starting from the inferior margin of the fibrous annulus to the inferior margin of cochlear promontory.

Walls of hypotympanum:

Hypotympanum proper is surrounded by five walls.

Anterior wall - This wall is formed by carotid canal medially and dense bone laterally.

Posterior wall - This is formed by inferior part of styloid complex and the vertical segment of facial nerve canal. This wall is frequently pneumatized by retrofacial air cells that extends from the mastoid antrum to the hypotympanum medial to the course of facial nerve. This wall corresponds to a line drawn in a vertical plane from the posterior semicircular canal to the junction of sigmoid sinus with that of the jugular bulb.

Outer Wall - This wall is formed by the tympanic bone.

Medial wall - This is formed by lower part of promontory and a part of petrous bone that extends under the promontory. This wall is usually pneumatized. The air cell system extends beneath the cochlea (retrocochlear air cells) to reach the petrous apex air cells. Rarely the medial wall can also be compact bereft of any air cells.

Inferior wall - This is formed by a thin bony covering separating the hypotympanum from the jugular bulb. In cases of high jugular bulb hypotympanum would be reduced. In some cases of extremely highly placed jugular bulb it could even be absent.

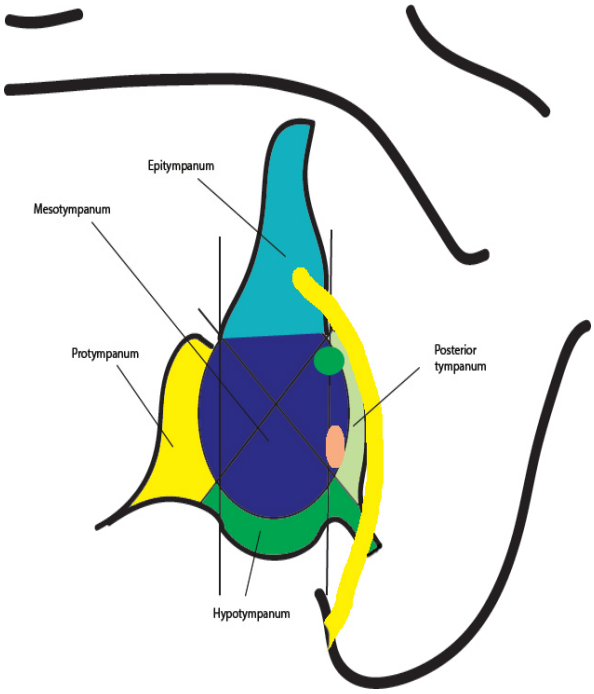


Diagram representing hypotympanum and its relations

Air cells of hypotympanum:

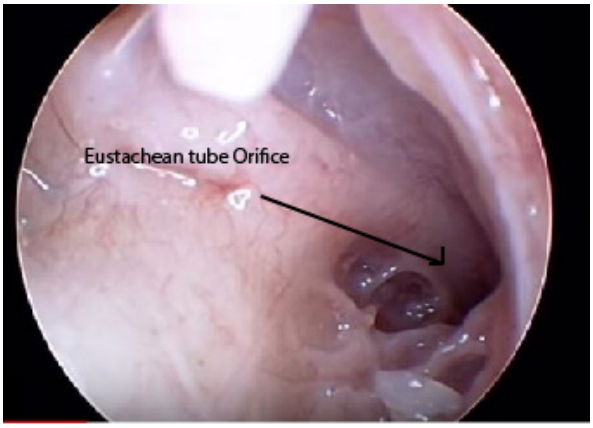
Air cells of hypotympanum is divided into:

Hypotympanic air cells

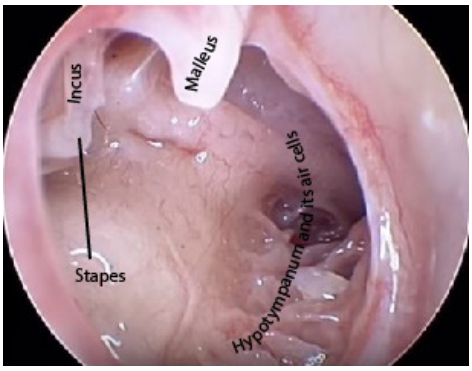
Retrofacial air cells

Hypotympanic air cells:
These air cells are seen in the medial and inferior walls of hypotympanum. These cells may also extend below the labyrinth to reach the petrous apex cells. If present these are known as infra labyrinthine tract.

Retrofacial cells:
These cells extend from the central mastoid tract always medial to the facial nerve draining into the hypotympanic space.



Photograph showing Eustachean tube orifice



Photograph showing hypotympanum and its air cells

Surgical importance of retrofacial air cells:

This is useful while preforming retrofacial hypotympanotomy where opening up these cells lying medial to the vertical portion of the facial nerve between jugular bulb inferiorly and posterior semicircular canal superiorly provides access to hypotympanum without the need to transpose to facial nerve.

The depth of hypotympanum varies from 1mm to 6 mm. The antero posterior diameter is 10 mm while medio laterally it is 4 mm. These values are not absolute and are highly variable.

Surgical importance of hypotympanum include:

1. It is the route of entry of major blood vessels

2. It is surgically the most neglected space also

3. It is highly variable in its dimensions

4. It contains trabeculi of various heights. These trabeculi could be 7 - 9 mm tall. The anteriorly placed long trabeculi is known as Trabeculi longa and the posteriorly placed another long trabeculi is known as Trabeculi Profunda. Sometimes trabeculi may also be entirely absent. Presence of these trabeculi should be considered to be safe for a surgeon because the jugular bulb in these patients is 6 mm deep and the sigmoid sinus lie posterior and hence they are protected.

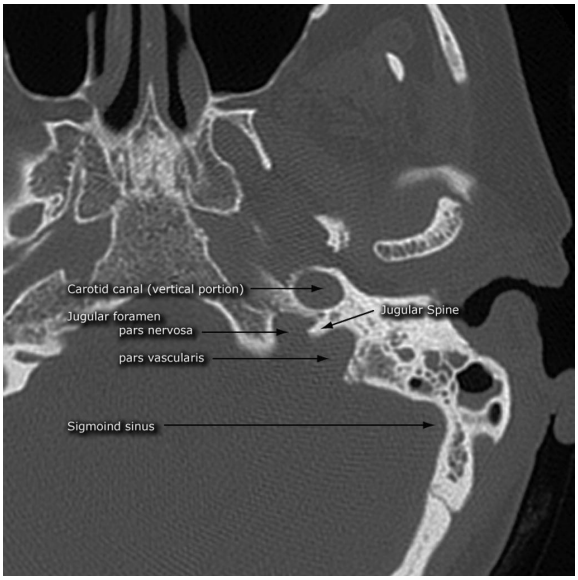
5. The air cells of hypotympanum is commonly a place for cholesteatoma. These air cells should be carefully followed during cholesteatoma surgery.

6. In nearly a quarter of temporal bones dissected a cavity known as anterior hypotympanic sinus is present in front of hypotympanum at the junction of its inner and outer walls. This is a common area for lurking cholesteatoma in hypotympanic area. This area is very difficult to access under microscopy. Use of otoendoscope would enable a surgeon to access and visualize this area of hypotympanum better.

7. Bony projection known as Finiculus / sustantaculum connects the anterior lip (postis anticus) of round window to hypotympanum.

8. Just under the cochlear capsule and finiculus there is a deep tunnel passing under internal carotid artery to reach the petrous apex. This is known as sub cochlear tunnel or tunnel of promontory. cholesteatoma can extend through this tunnel.

9. Jugulo carotid septum between the internal carotid artery and jugular vein is present in hypotympanum. This septum is also known as the crotch. Erosion of crotch can radiological be identified and is classic sign of glomus jugulare tumors. This is also known as jugular spine.



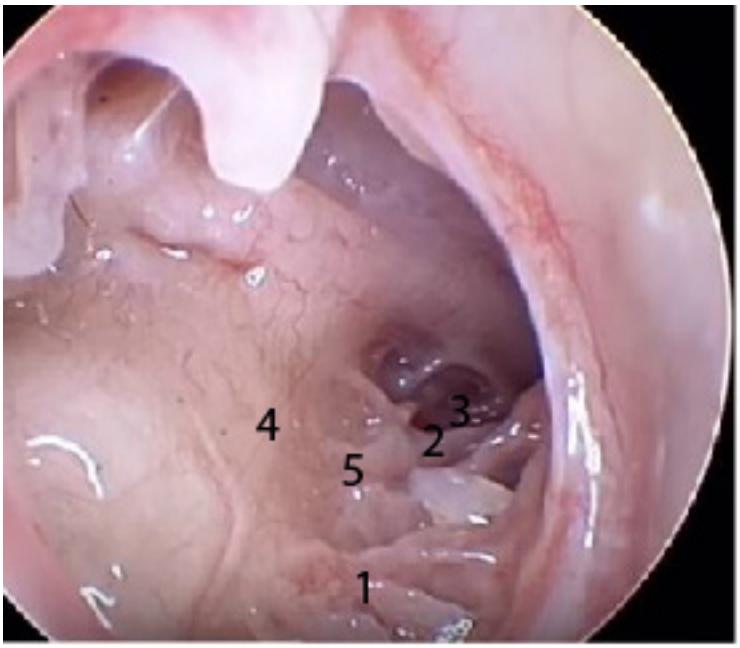
Axial cut CT skull base showing crotch

Case courtesy of A.Prof Frank Gaillard, Radiopaedia.org, rID: 35926

10. Inferior tympanic artery a branch of ascending pharyngeal artery along with Jacobson's nerve pass through the crotch. Ascending pharyngeal artery is the smallest branch of external carotid artery. Inferior tympanic artery is also known as the artery of trouble as it causes troublesome bleeding during surgery.

11. Finiculus serves as a rough surface marking for jacobson's nerve and Inferior tympanic artery in the hypotympanum. This is a reliable landmark for a surgeon in the hypotympanum.

12. Erosion of outer tympanic wall of hypotympanum sparing the annulus can occur in keratosis obturans. This is known as Hanging rope sign.



1. Trabeculi Profunda

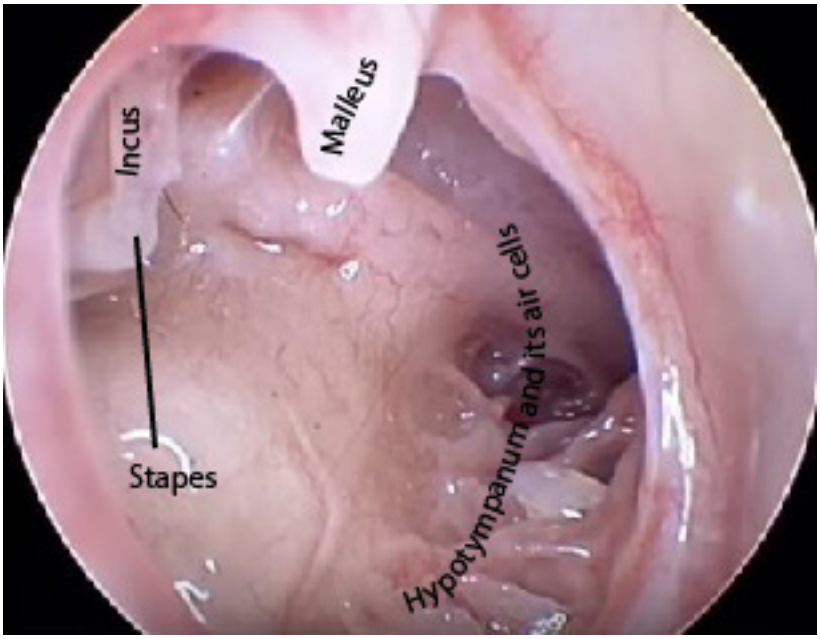
2. Trabeculi Longa

3. Anterior hypotymp sinus

4. Cochear capsule

5. Finiculus

Figure showing structures in the hypotympanum



Endo photograph of Hypotympanum

Anatomy of Retrotympanum

Introduction:

The posterior region of tympanic cavity is known as retrotympanum. This is a most complicated area to study both from anatomical / surgical point of view. Embryologically retrotympanum is derived from the second branchial arch. The ossification of Riechert's cartilage results in the formation of Proctor's styloid complex.

Important anatomic structures are lodged in this area. This area has assumed significance because of the difficulties encountered in clearing cholesteatoma from this area. This area is so narrow and has lot of crevices, it is very difficult to clear disease from this area.

The Proctor's complex comprises of three eminences:

1. The pyramidal eminence

2. Styloid eminence

3. Chordal eminence

Arising from these eminences, between them and the promontory and the posterior lip of the round window niche, lie numerous ridges and bridges.

This area is supposed to contain 4 important recesses. Each of these four recesses could hide cholesteatoma causing the surgeon to leave residual disease which could later recur. Precise knowledge of anatomy of this region is vital for the surgeon who wants to clear disease from this area. The recesses present in the retrotympanic area are:

1. Sinus tympani

2. Lateral tympanic sinus

3. Posterior tympanic sinus

4. Facial recess

Pyramidal eminence / ridge is the most prominent anatomical landmark of this area. This eminence hold the pyramidalis muscle. There are other prominences arising from this area projecting in various directions.

They include:

1. External: Chordal ridge

2. Inferior: Pyramidal ridge

3. Superior: Suprapyramidal ridge

4. Internal: Ponticulus

Types of sinus tympani:

Sinus tympani has been classified into three types depending on its depth. Note in type III it extends up to the level of lateral semicircular canal.

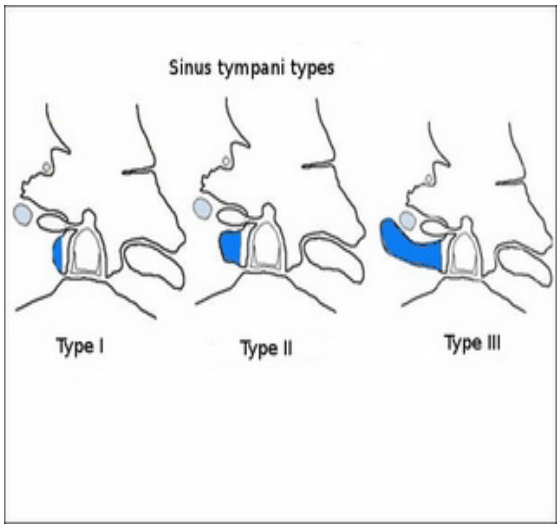


Diagram illustrating the types of sinus tympani

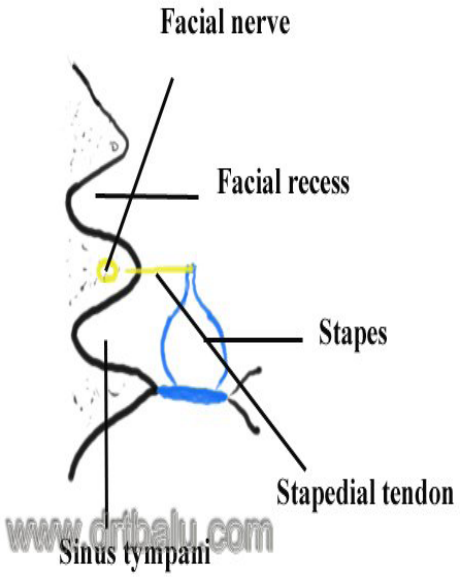


Illustration showing sinus tympani

Lateral tympanic sinus:

This sinus was first described by Proctor in 1969. This sinus lies between three eminences of styloid prominence.

Posterior tympanic sinus:

Posterior sinus of middle ear cavity is one of the recently identified anatomical sinus inside the middle ear cavity. Serial temporal bone dissections have shown that it is present in nearly 90% of dissected bones.

Position: It lies just posterior to the oval window.

Depth: 1mm or less
Width: 1.5 mm or less

In nearly 60% of dissected specimen a ridge of bone arising from the floor of middle ear cavity separates it from sinus tympani.

In 8% of dissected specimen, the sinus tympani and posterior sinus merged together to form one confluent sinus.

It has been demonstrated that cholesteatoma / granulation tissue may lie within this sinus making removal difficult leading on to residual disease.
Retraction pockets may also occur close to this area.

Facial recess:

This recess lies between the promontory and tympanic annulus. It is bounded medially by the facial nerve and laterally by tympanic annulus. Running between these two structures at varying angulations is the chorda tympani nerve. Superiorly it is bounded by the incudal buttress and incudal fossa which lodges the short process of incus. The incudal buttress seperates the facial recess from the aditus de antrum.

Inferiorly the facial recess is limited by the chordo facial angle (18 - 30 degrees), the distance between the origin of chorda tympani nerve and short process of incus (ranging between 5 -10 mm)

Chorda tympani nerve always runs medial to the ear drum. Drilling in this area between the facial nerve, annulus and the angle formed by the chorda tympani nerve will lead into the middle ear cavity without causing a breach in the ear drum. This approach is used in cochlear implant surgery to place the electrode in the round window area. Hypotympanum can also be approached through this approach.

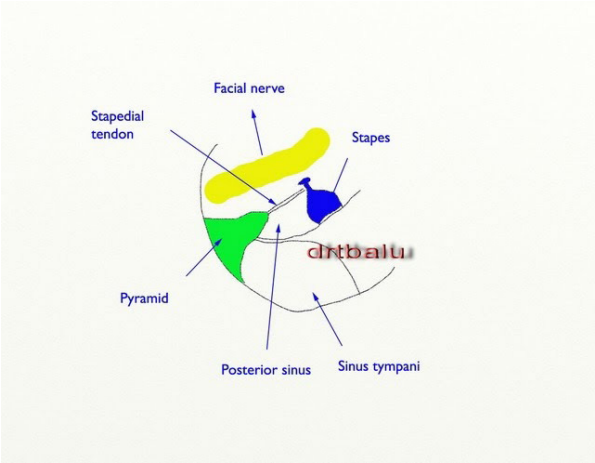


Figure showing the posterior sinus

The size of facial recess is variable among individuals. It is fully developed at birth and does not increase in size as growth occurs. It measures about 2 mm at the level of round window and 3 mm at the level of oval window.

The chordal ridge, which runs between the pyramidal eminence and the chordal eminence divides facial recess into facial sinus superiorly and lateral tympanic sinus inferiorly.

Subiculum:

This is the posterior extension of promontory separating oval and round windows.

Ponticulus:

Rarely a spicule of bone arises from the promontory above the subiculum and runs to the pyramid on the posterior wall of the middle ear cavity. This spicule of bone is known as the ponticulus.

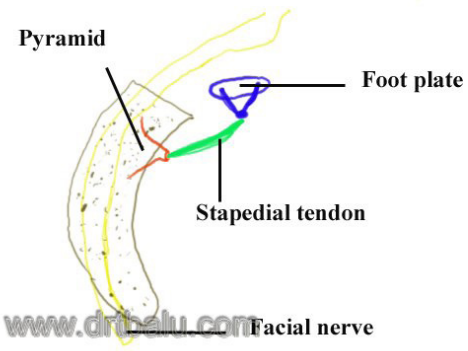


Illustration showing Pyramid and its relationships

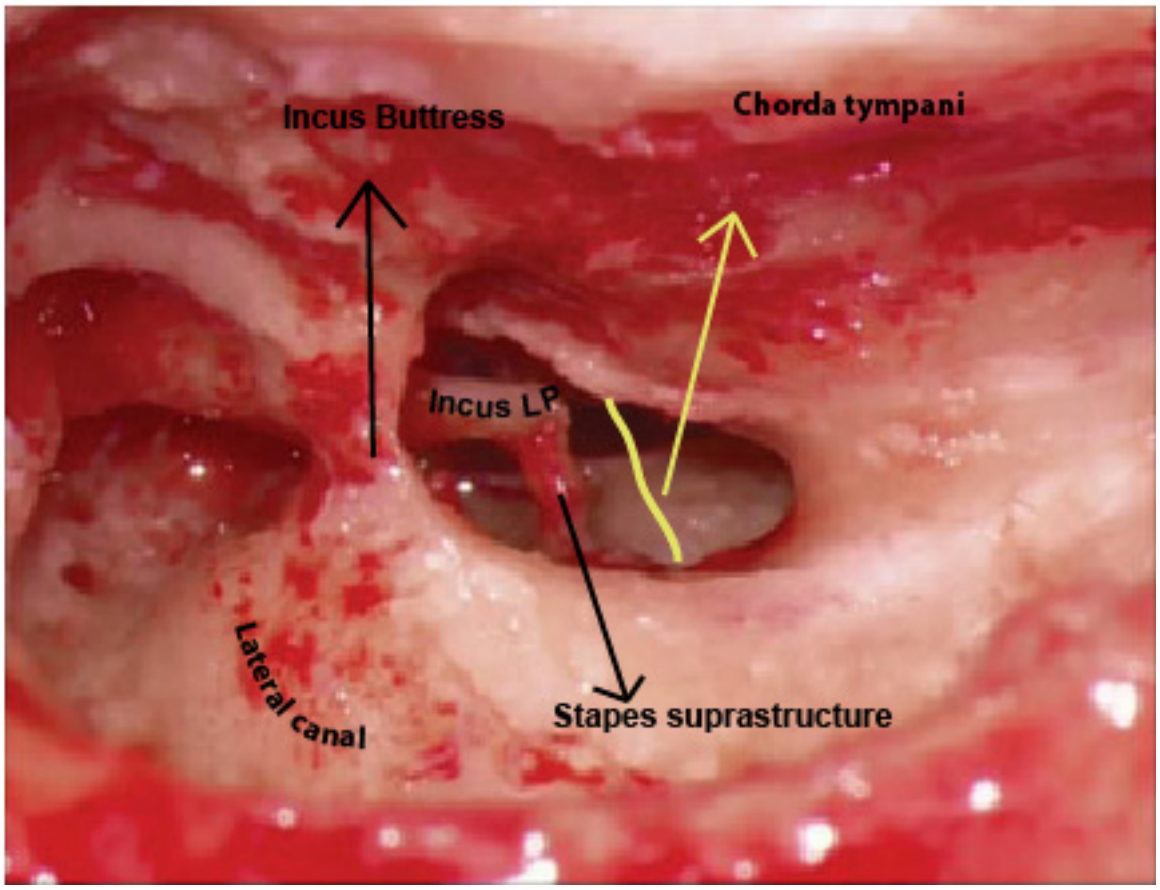
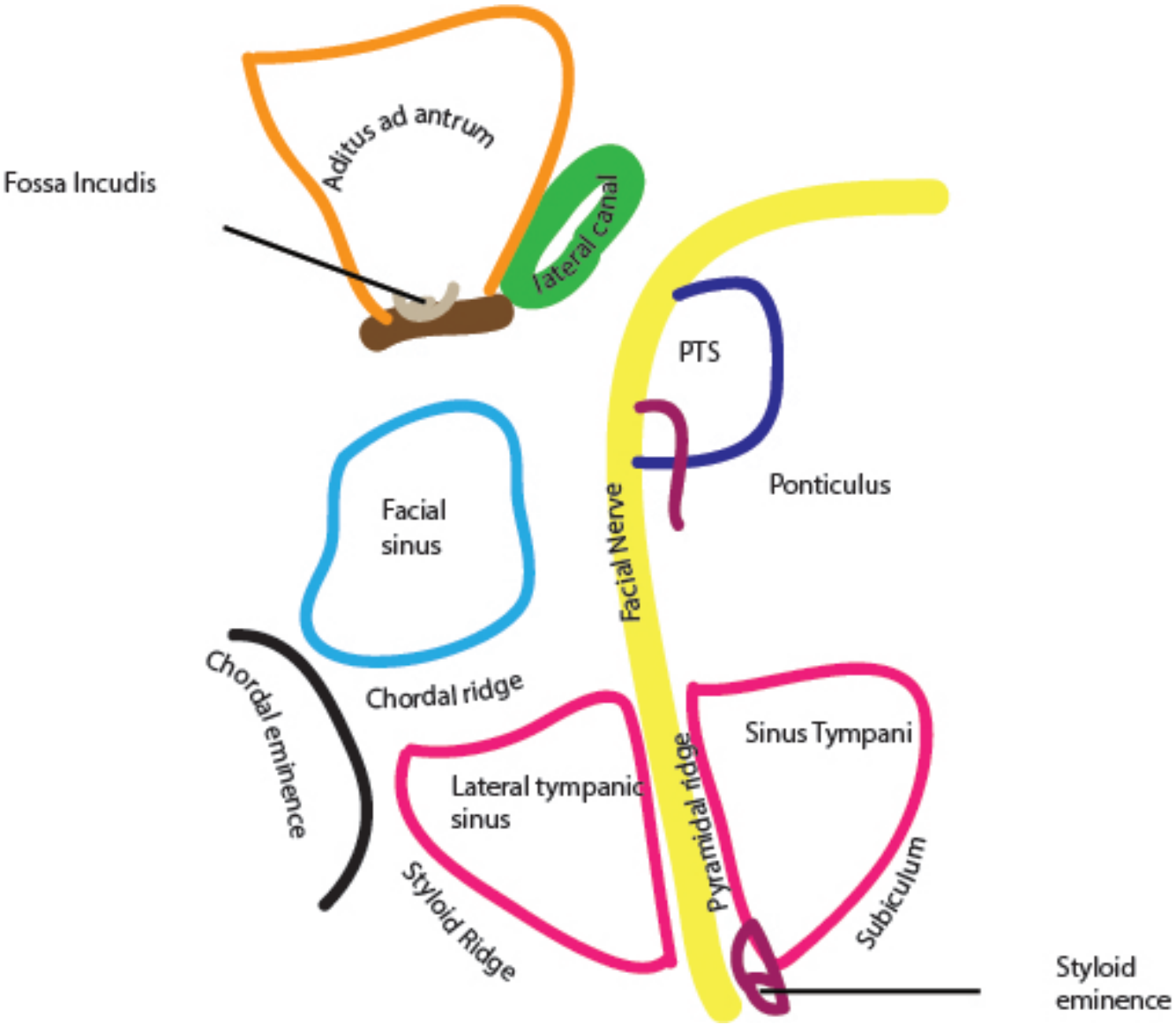


Figure showing facial recess dissected



Schematic representation of retrotympanic area

Facial sinus:
This forms the superior part of facial recess. It is a small pouch situated between the incudal buttress superiorly and chordal ridge inferiorly, the second genu of facial nerve medially. There is no communication between facial sinus and the air cells of attic and mastoid antrum.

Lateral tympanic sinus:

This is the inferior part of facial recess. it is also the most lateral and narrowest part of the retrotympanum. It represents the space among the three eminences of styloid complex, (pyramidal eminence, styloid eminence, and chordal eminence). It lies medial to the chordal eminence, inferior and lateral to pyramidal eminence and superior to the styloid eminence. The dimensions of lateral tympanic sinus varies from 1.5 - 2.5 mm and it has no connection with the attic or antrum.

Facial recess provides a posterior window to the middle ear cavity from the mastoid enabling a surgeon to visualize both oval and round windows. This surgical approach is known as posterior tympanotomy. In cases where this recess is anatomically narrow then extended facial recess approach may be resorted to in-order to access middle ear cavity through this route. In extended facial recess approach the chorda tympani nerve is sacrificed and drilling is proceeded between the annulus and facial nerve canal. This leads to a wider exposure into the middle ear cavity. The mean width of extended facial recess is about 5 mm. During posterior tympanotomy the decision to sacrifice chorda tympani nerve is dependent on the size of this recess. Pre operative study of thin section CT scans allows the surgeon to make a preop assessment of the size of facial recess enabling the surgeon to plan the procedure accordingly.

Medial spaces:

Medial spaces of retro tympanum include the tympanic sinus. These are depressions in the posterior wall of the middle ear cavity between the facial nerve and pyramidal eminence laterally and labyrinth medially. Bony ridge called ponticulus which runs between the promontory and pyramidal eminence divides the tympanic space into posterior tympanic sinus superiorly and the sinus tympani inferiorly. This has already been clearly stated in the previous pages and repeated for the sake of stressing their importance.

During middle ear surgery in-order to reach the posterior tympanic sinus, section of the stapedial tendon and drilling of pyramidal process is required.

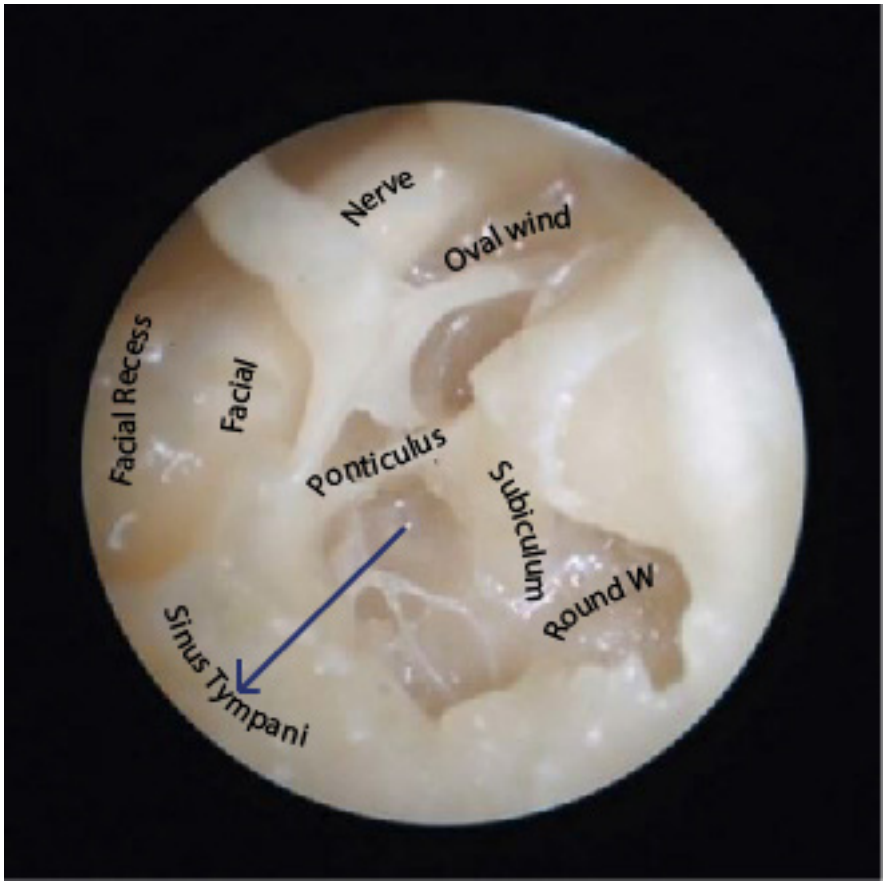
Applied anatomy of sinus tympani:

This is the largest sinus of retrotympanum. It lies medial to the mastoid portion of the facial nerve and lateral to the posterior semicircular canal. It is limited superiorly by Ponticulus and the pyramidal eminence. Inferiorly it is bounded by subiculum and styloid eminence. Sinus tympani shows great variation in size, shape and depth. Its posterior extension varies between 0.2 - 10 mm with an average of 2 mm. In about 10% of individuals sinus tympani and posterior tympanic sinus forms one single confluent recess.

Based on the depth sinus tympani is classified into three types occurring with equal frequency in the population. They include:

Type A:

This is shallow sinus tympani. It is shallow and does not reach up to the level of the vertical portion of facial nerve posteriorly. In these cases surgical transcanal approach to this area is very much possible.



Dissected specimen showing facial recess area and its relationships

Type B sinus tympani:

This type is intermediate in depth. It lies medial to the facial nerve (vertical portion) but does not extend posteriorly deeper than the level of facial nerve. In these individuals clear visualization of this area is not possible endomeatally using a microscope. Endoscope should be used to visualize this area. Blind dissection in this area should be avoided as this would leave behind residual cholesteatoma or could cause injury to facial nerve or jugular bulb if it is found to be high placed.

Type C sinus tympani:

This type is very deep. It extends posteriorly and deeper than that of the vertical portion of facial nerve. This type is commonly seen in well pneumatized mastoid bones. This type is difficult to access transmeatally even with the use of endoscope. Retrofacial approach should be combined with transmeatal approach to enable this area to be adequately visualized. In order to perform retrofacial approach there should be a minimum distance of 2 mm between the facial nerve and the posterior semicircular canal. If this space is narrow it would lead to complications like injury to facial nerve / posterior semicircular canal etc.

Anatomy of Epitympanum (Attic)

Epitympanum also known as the Attic is that part of the middle ear cavity which is situated above an imaginary line passing through the short process of malleus. This portion of the middle ear cavity occupies nearly 1/3 of the vertical dimension of the entire middle ear cavity.

It lodges the head and neck of malleus, body and short process of incus.

Boundaries of attic:

Lateral - Inferiorly this wall is formed by sharpnell's membrane. This membrane is the pars flaccida of ear drum. This portion of the ear drum lacks the middle fibrous layer. Superiorly it is bound by a bony wall known as the outer attic wall. It is this portion of the attic that gets commonly eroded in patients with cholesteatoma.

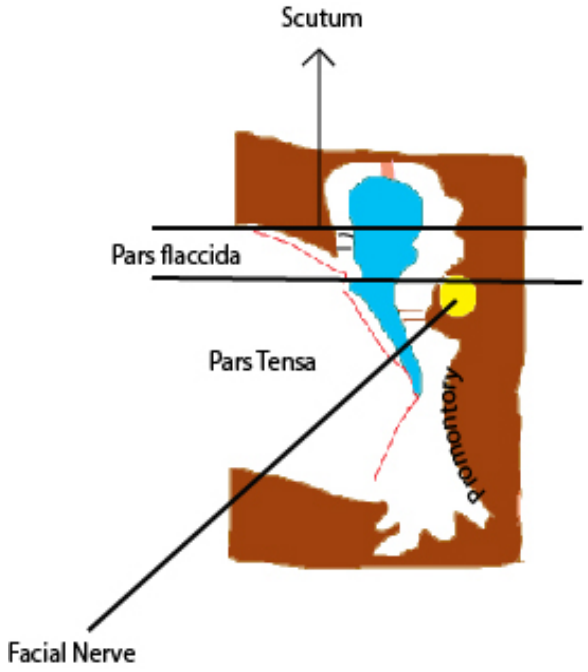
Medial - This is part of the medial wall of the middle ear cavity situated above the tympanic segment of facial nerve and tensor tympani muscle. It contains the lateral semicircular canal. This wall may be pneumatized by the supralabyrinthine tract.

Posterior - This wall is occupied by the aditus. It is 5-6 mm high and is usually larger above than below. The aditus provides communication between the antrum and the rest of the tympanic cavity.

Inferior - Tympanic diaphragm is present. It divides attic into an upper unit situated above the diaphragm and a lower unit of attic below the diaphragm known as the Prussak's space. Medially the tympanic diaphragm separates the upper unit of the attic from the underlying upper portion of mesotympanum (middle ear proper). The upper unit of attic and mesotympanum communicate with each other for ventilation purposes through two openings known as anterior and posterior isthmus tympani.

Upper attic:

This portion of attic lies above the tympanic diaphragm. Medially tympanic diaphragm separates the upper unit of attic completely from the upper mesotympanum. The communication between these spaces for the purpose of ventilation occurs via an opening in the tympanic diaphragm known as tympanic isthmus.



Diagrammatic representation of attic

The tympanic isthmus is situated between the tensor tympani muscle anteriorly and the posterior incudal ligament posteriorly. Laterally the tympanic diaphragm separates the upper unit of attic from its lower unit which happens to be the Prussack's space.
Posteriorly the upper unit of attic communicates with the mastoid cavity through the aditus. In addition to this separation in the horizontal plane by the tympanic diaphragm, there are many folds and ligaments dividing the space in a perpendicular plane.

The superior malleolar fold which is oriented coronally divides the upper unit of attic into two spaces the posterior attic (larger one) and anterior attic (smaller one).

Posterior attic (Posterior epitympanum):

This area is mostly occupied by the posterior part of the head of the malleus, the body and short process of incus. In an adult the distance from the tip of the incus to attic roof is about 6 mm. This area in turn is divided into Medial posterior attic and lateral posterior attic by superior incudal fold. It should be noted that the superior incudal fold is oriented in a saggital plane.

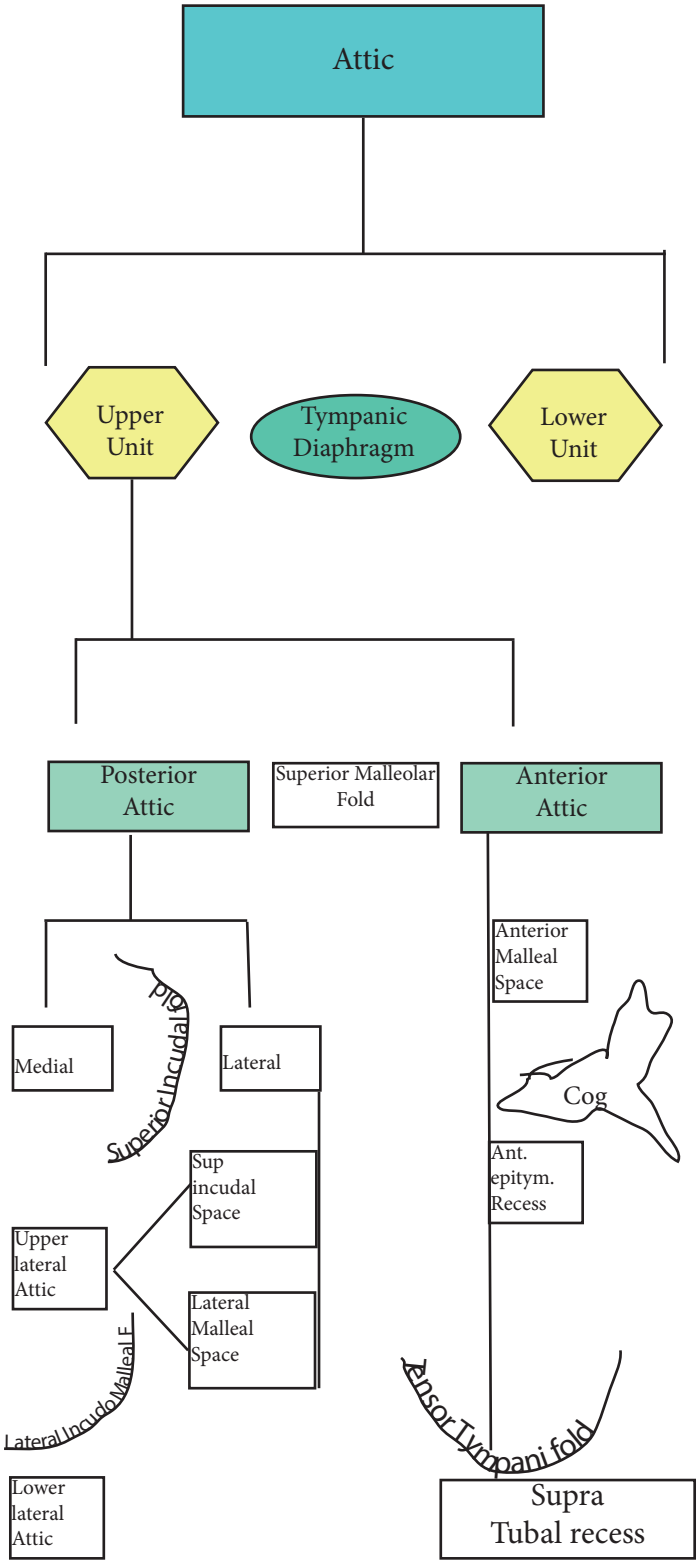
Medial posterior attic:

This space is also known as the medial incudal space and is the largest component of posterior attic. Its boundaries include:

Medial - lateral semicircular canal and fallopian canal.

Lateral - Ossicles and superior incudal fold.

The distance between the lateral canal and incus body is about 1.7 mm. This portion of the attic essentially contains the tympanic isthmus which is divided into anterior and posterior tympanic isthmus by the medial incudal fold. These openings are the only important aeration routes of epitympanum.



Lateral posterior attic:

This portion of attic is narrower than medial posterior attic. This space is located between the outer attic wall laterally and head of malleus, body of incus, and superior incudal fold medially. This space is subdivided into three spaces i.e.

Superior incudal space

Lateral malleal space

These two spaces form the upper lateral attic.

Inferior incudal space - This is also known as the lower lateral attic.

Upper lateral attic:

This space is composed effectively of two spaces. These two spaces open to each other at different levels. Posteriorly the space lying above lateral incudo malleal fold is known as the superior incudal space, and the more anteriorly lying space above the lateral malleal fold is known as the lateral malleal space.

Superior incudal space:

Lies in a more superior position than lateral malleal space. Inferiorly it is limited by incudomalleolar fold which separates it from inferior incudal space.

Lateral malleal space:

This space is part of lateral attic and lies above lateral malleal fold. It is limited medially by head and neck of malleus and laterally by the outer attic wall. Anteriorly it is bounded by anterior malleal fold which is a mucosal fold raised by the underlying anterior malleal ligament. Posteriorly it is bounded by the downward turning end of incudomalleal fold. This space is regularly open in its superior wall thereby establishing a free communication between this space and superior incudal space.

Rarely lateral malleal fold may be incomplete and hence a direct communication exists between the Prussack's space and lateral malleal space.

In some cases the incudomalleal fold may extend over the entire lateral malleal space (it slopes and joins the posterior malleal fold). In this scenario the lateral malleal space becomes isolated completely separated from the superior incudal space. It communicates with the inferior incudal space.

Lower lateral attic (Inferior incudal space):

This space lies below the lateral incudomalleal fold, hence this space lies inferior to the tympanic diaphragm. This space is located between the short process and body of incus medially and scutum laterally. A portion of mesotympanum ensures that this space is ventilated. The portion of mesotympanum that ensures this space is ventilated is limited medially by the medial incudal fold and anteriorly by the interossicular fold which lie between the long process of incus and upper 2/3 of the handle of malleus

Anterior Attic (Anterior epitympanum):

This cavity is separate. It varies in shape. It lies superior to the head of the malleus and the anterior malleal fold. This portion of the attic is divided into two portions by a bony eminence known as the cog. Cog is actually a bony crest extending downwards from the tegmen plate. This space lie superior to processes cochleariformis and antero superior to the head of the malleus. The two portions of anterior attic include:

Anterior malleal space (lie posteriorly and smaller).
Anterior epitympanic recess (lie anteriorly and somewhat larger than anterior malleal space).

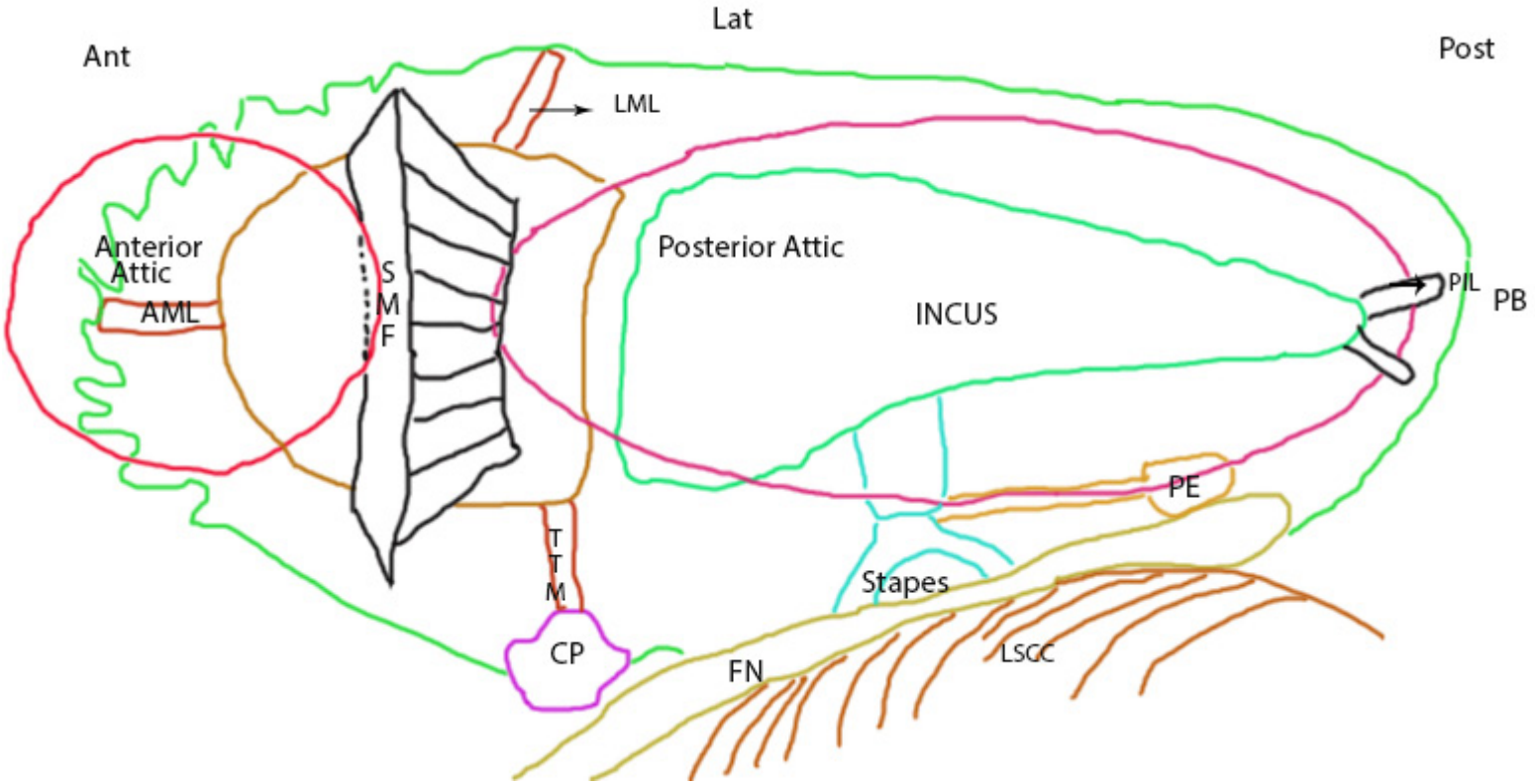


Figure showing superior view of attic. AML - Anterior malleolar ligament, SMF - Superior malleolar fold, LML - Lateral Malleolar ligament TTM - Tensor tympani muscle LSCC - lateral semicircular canal PB - Petrous bone, FN - Facial nerve, CP - Cochleariform process, PE - Pyramidal eminence PIL - posterior incudal ligament

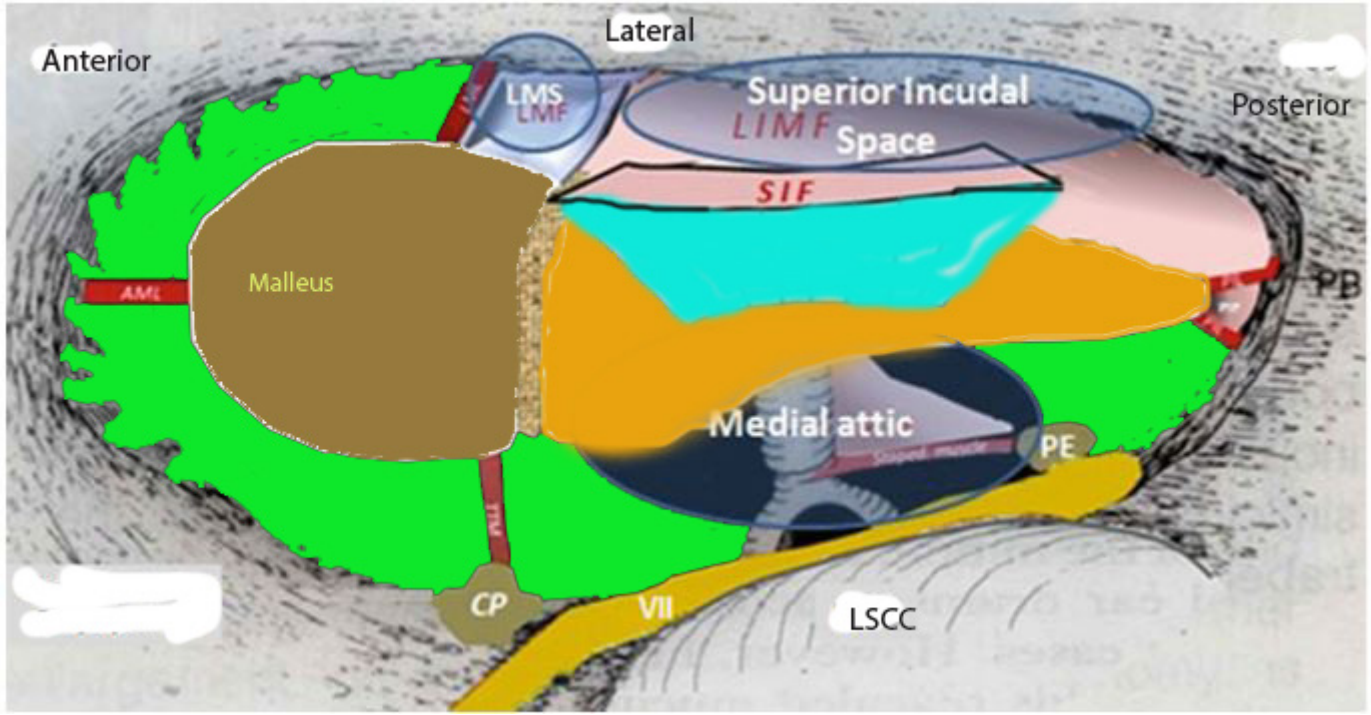


Diagram showing spaces around the attic

Anterior Malleal space:

The size of this space is highly variable. It is situated between the head of the malleus posteriorly and cog anteriorly.

Anterior Epitympanic recess:

This goes under different names. These include:

Anterior epitympanic sinus

Anterior epitympanic space

Sinus epitympani

Supratubal recess

Current anatomists tend to believe that supratubal recess and anterior epitympanic recess are two different spaces. These two spaces are separated by tensor tympani fold. The supratubal recess should be considered to be part of protympanum.

The term anterior epitympanum should be used to indicate the whole anatomical entity composed of anterior malleolar space and anterior epitympanic recess.

Boundaries of anterior epitympanic recess are as follows:

Superior - Anterior part of tegmen tympani

Anterior - Root of zygoma

Posterior - Cog

Lateral - Scutum

Medial - Anterior segment of tympanic portion of facial nerve and geniculate ganglion.

Floor - Processes Cochleariformis and Tensor tympanic fold.

The tensor tympanic fold is a part of tympanic diaphragm. When this fold is complete the anterior tympanic recess and supratubal recess form two separate spaces. In 15% of individuals there may be defect in the tensor tympani fold. In these individuals anterior epitympanic recess is in communication with supra tubal recess. This serves as an accessory route of aeration of attic area and it goes by the term anterior ventilatory channel. The posterior route of ventilation is represented by the anterior and posterior epitympanic isthmus.

Role of anterior epitympanic recess in chronic otitis media:

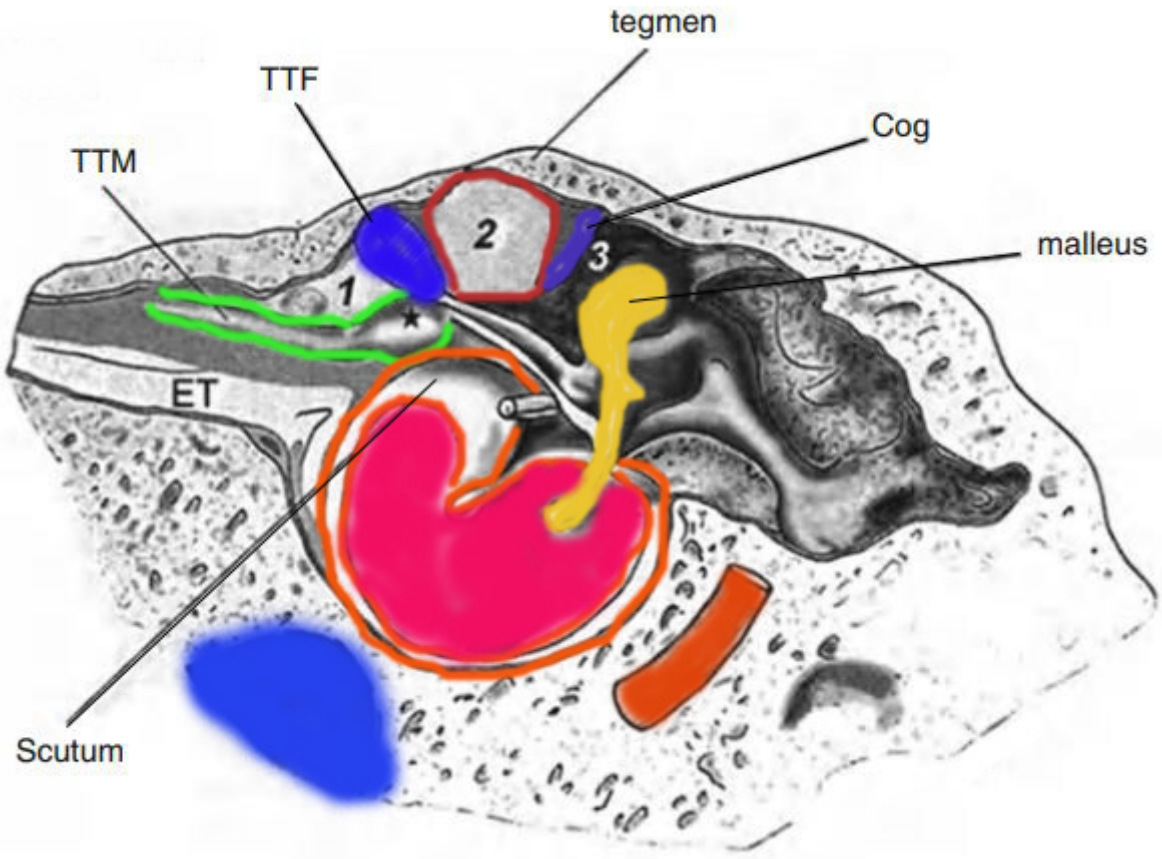
1. Infection involving this area should be considered in all cases with otorrohea and anterior central perforation.

2. Infections and effusions in this area is resistant to therapy and grommet insertion.

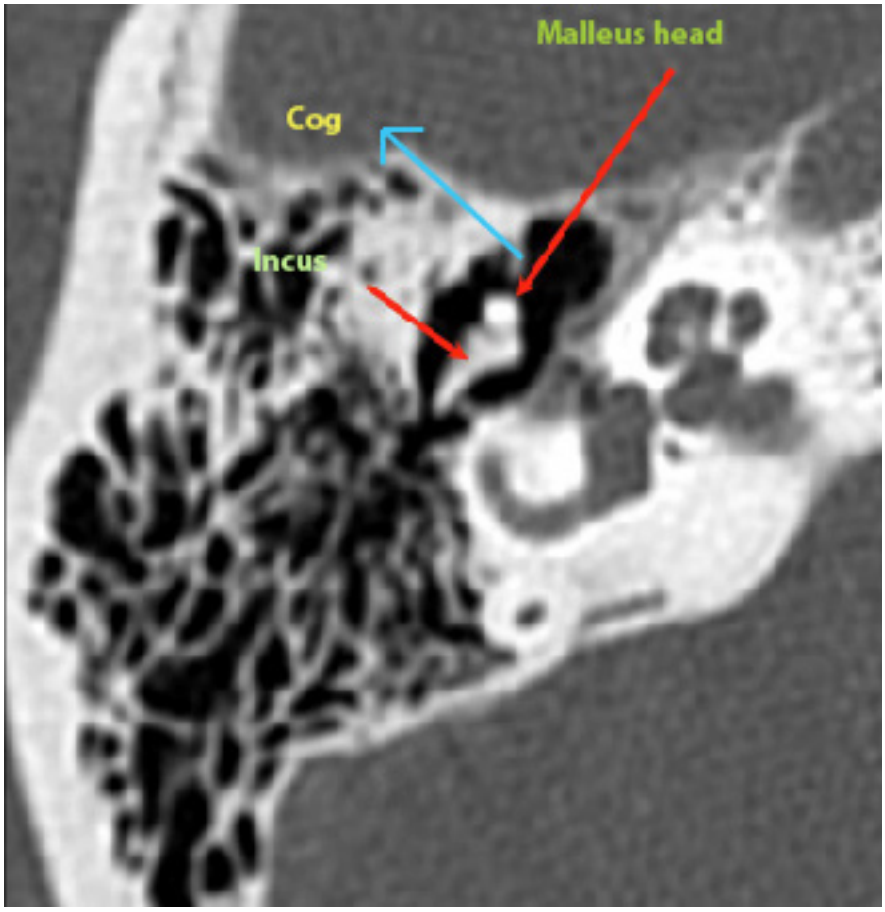
3. Presence of antero superior retraction pocket indicates pathology involving this area.

4. Resection of Cog and tensor tympani fold will help to ventilate this area better.

5. CT scan study of this area is mandatory not only to rule out involvement but also to ascertain the dimensions. This helps in selecting the ideal surgical approach.



1. Supra tubal recess
2. Anterior epitympanic recess
3. Anterior malleal space

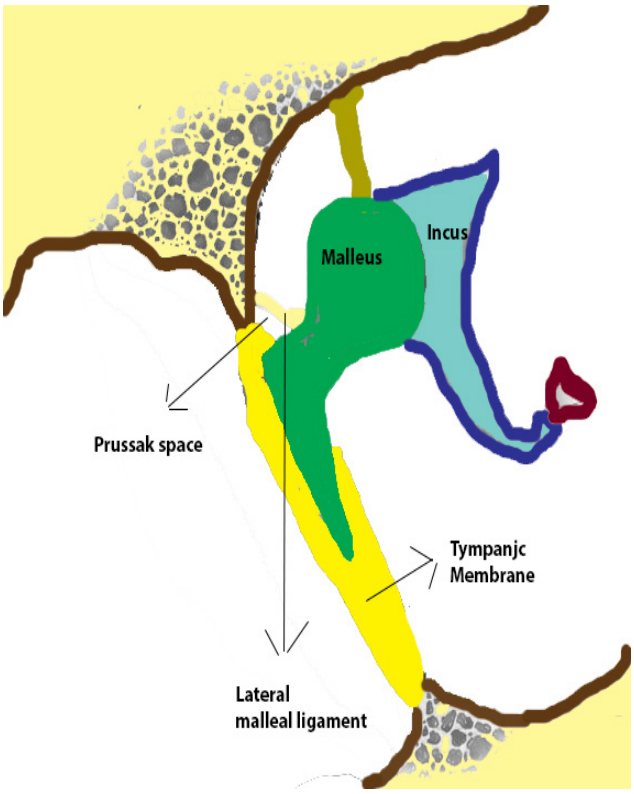


Axial CT image of anterior epitympanum displaying the Cog

Lower Attic Unit (Prussak's space):

This space was first described by Prussak in 1867. He initially described it as a superior pouch of the tympanic membrane. This space is located between Sharpnell's membrane and the neck of the malleus. This pouch was considered to be distinct from the anterior and posterior pouches of von Troltsch. Prussak's space is formed from the posterior pouch of von Troltsch. This occurs as a prolongation of either a low portion or high portion of superior saccus replacing the mesenchymal tissue that exists between the malleus and sharpnell's membrane.

Prussak's space is actually situated inferior to the tympanic diaphragm and actually represents the lower unit of attic. Laterally this space extends superior to the roof of the external auditory canal by about 0.4 mm. The largest cross sectional area of this space is at the level of roof of the external auditory canal measuring about 2.6 mm.



Diagrammatic representation of Prussak's space

Boundaries of Prussak's space include:

Roof - Is formed by the lateral malleolar fold which happens to be the lower portion of the tympanic diaphragm.

Floor - Is formed by the neck of the malleus.

Anterior - Is formed by the anterior malleal fold.

Lateral - Is formed by pars flaccida and the lower edge of outer attic wall (scutum).

Posterior - This wall opens into posterior pouch of von Troltsch and then into the mesotympanum. Ventilation of this space is independent of the upper unit of attic. Ventilation to this space occurs through the posterior pouch of von Troltsch. This ventilation channel is smaller than that of tympanic isthmus which ventilates the upper unit of attic.

Role of Prussak's space ventilatory dysfunction in pathogenesis of attic cholesteatoma:

Ventilation of Prussak's space is highly precarious. Ventilation can be blocked by the formation of thick tenacious mucous secretion in this area. This causes retraction of pars flaccida with adhesion to the neck of the malleus. This could occur without involvement of other compartments of the upper unit of attic situated above the tympanic diaphragm.

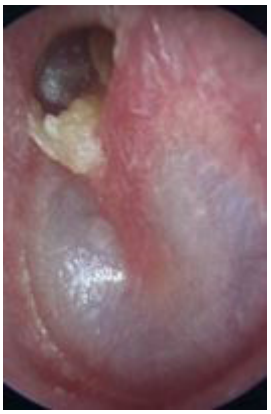
Initially the sac of retraction pocket remains small. As desquamated epithelium starts to accumulate the sac enlarges in size and expands involving pathways of least resistance.

The mucosal folds of attic directs the spread of cholesteatoma but in no way blocks the spread. A surgeon should always bear this aspect in mind while dealing with a patient suffering from chronic squamosal disease of middle ear.

Growth pathways of cholesteatoma in the attic could take these probable routes:

Pathway I:

Cholesteatoma from Prussak's space spreads through posterior pouch of von Troltsch. The posterior tympano malleolar fold directs the spread towards the inferior incudal space. From here cholesteatoma could extend medial to the long process of incus and then through the tympanic isthmus into the medial attic area.



Photograph showing attic cholesteatoma

Pathway II:

Cholesteatoma progresses through the thin part of the lateral malleal fold directly to the upper unit of attic and from there to the posterior attic, aditus and then to the antrum.

Pathway III:

The cholesteatoma passes from the lateral malleal space to the anterior attic, anterior epitympanic recess, and then it extends downwards to invade the supra tubal recess and protympanum.

Pouches associated with tympanic membrane:

Anterior pouch of von Troltsch : This pouch is situated between the anterior malleal fold and the pars tensa of ear drum. This space communicates with the supratubal recess and protympanum.

Posterior pouch of von Troltsch: This pouch is situated between the posterior malleal fold and the pars tensa of ear drum. It extends postero inferiorly opening into the most cranial portion of mesotympanum. This is supposed to be the main ventilation route of the Prussak's space.

SMF - Superior malleal fold

SIF - Superior incudal fold

SIS - Superior incudal space

AMLF - Anterior malleal ligament fold

LMS - Lateral malleal space

MIF - Medial incudal fold

IIS - Inferior incudal space

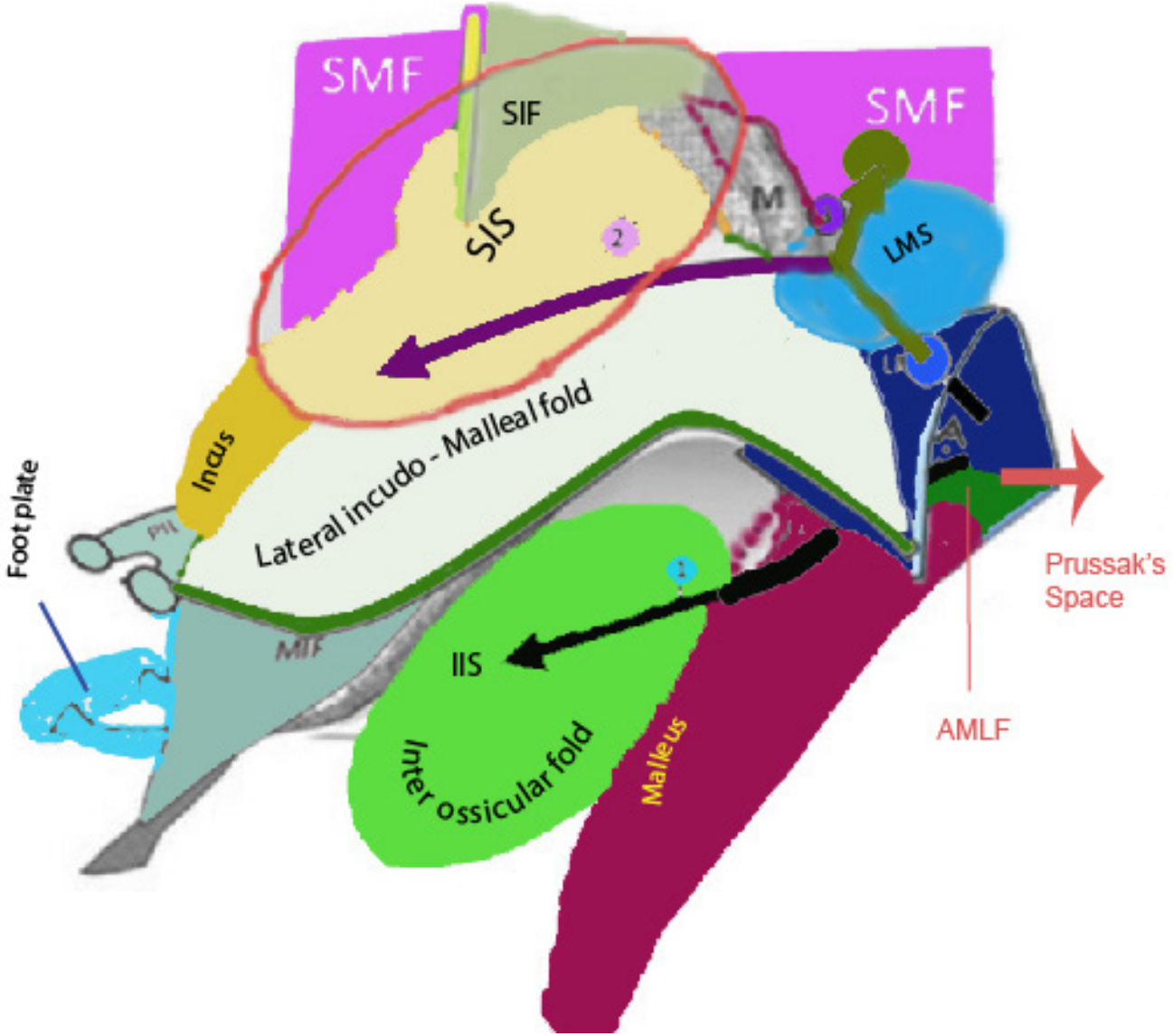


Figure showing the routes of spread of attic cholesteatoma from Prussack's space

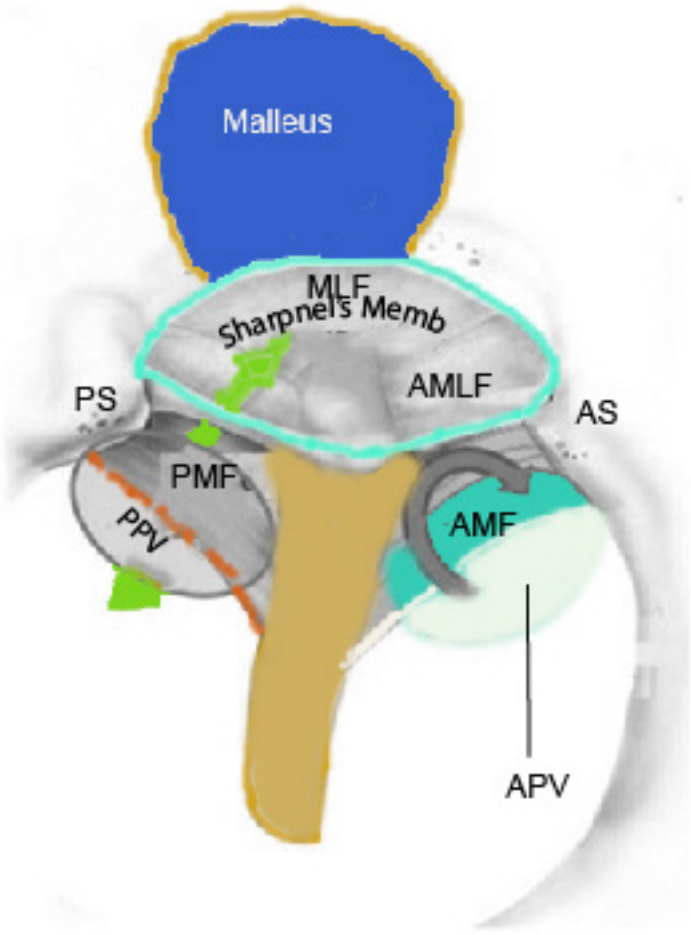


Diagram showing middle ear lateral wall after removal of tympanic membrane

APV - Anterior pouch of von Troltsch

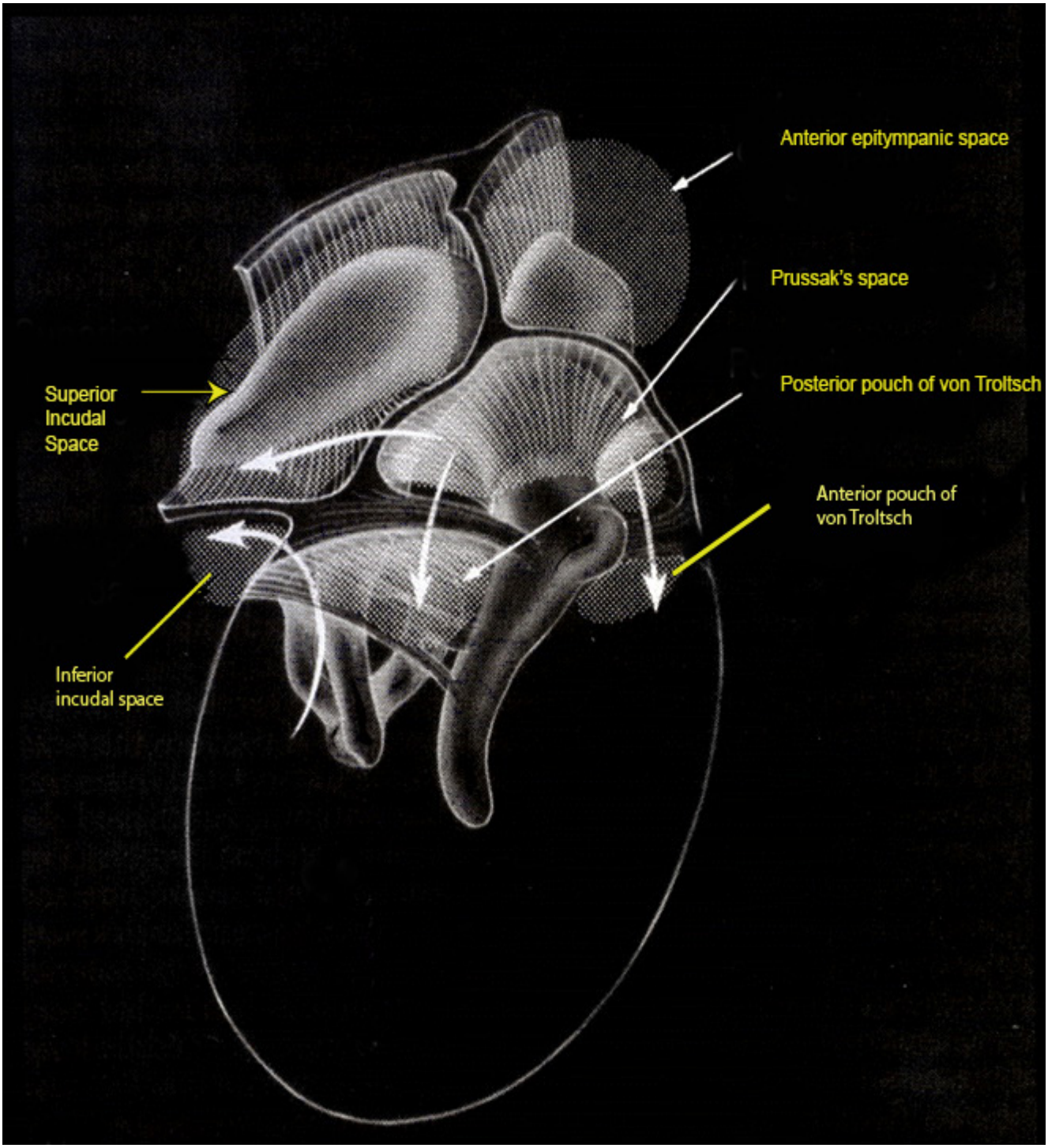
PPV - Posterior pouch of von Troltsch

AS - Anterior tympanic spine

PS - Posterior tympanic spine

AMF - Anterior malleal fold

PMF - Posterior malleal fold



Diagrammatic representation of various spaces in the middle ear cavity

Mastoid

The term Mastoid is derived from the Greek word mastos which means breast. The shape of mastoid bone resembles that of breast hence it is used to indicate the structure. This is actually a portion of temporal bone. It houses several important structures like facial nerve, sigmoid sinus and labyrinth.

This portion of temporal bone projects from the base of skull. It is situated behind the external auditory meatus at the inferior part of outer aspect of temporal bone. This process is a site of a number of air filled cavities known as mastoid air cell system. Thorough knowledge of anatomy of this area is important from surgical point of view as these air cells commonly get infected due to middle ear disease process. Mastoid serves as air reservoir for middle ear ventilation.

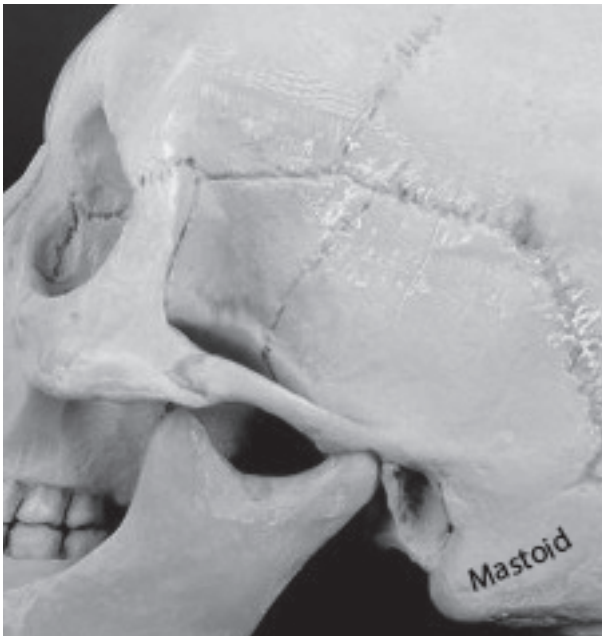


Figure showing mastoid process of temporal bone

Embryology:

The mastoid process appear during the 29th week of gestation. Embryologically this is the result of fusion of periosteal layers of otic capsule and the tympanic part of squamous bone.

During birth the mastoid process is underdeveloped. It becomes prominent by the age of 2 and continues to increase in size until the child reaches the age of 6. The expansion of mastoid process is an active phenomenon caused by increase in the size of mastoid air cell system.

During the process of pneumatization the bone of the mastoid process which contains bone marrow is invaded by expanding air filled sacs. The residual bone that has not been invaded by air cells become the septa between these existing air sacs.

Mastoid antrum:

This is the biggest air cell of mastoid air cell system. This air cell begins its development during the 22 week of intra uterine life. It reaches the adult size by the 35th week of intra uterine life. The antrum actually develops in the middle of mastoid process on either side of petrosquamous suture. The medial part of the antrum which is actually known as the petrous part develops from saccus medius while its lateral part also known as the squamous part develops from the saccus superior. The plane of fusion between these two parts is known as petrosquamous fissure.

Failure of complete fusion of these two sacci leads to septation of mastoid antrum by a bony partition known as Korner's septum.

Antrum is fully developed at birth. Its mean surface area is about 1 cm square. The mastoid antrum gets displaced medially because of the growth of mastoid process. The mastoid process continues to grow till puberty.

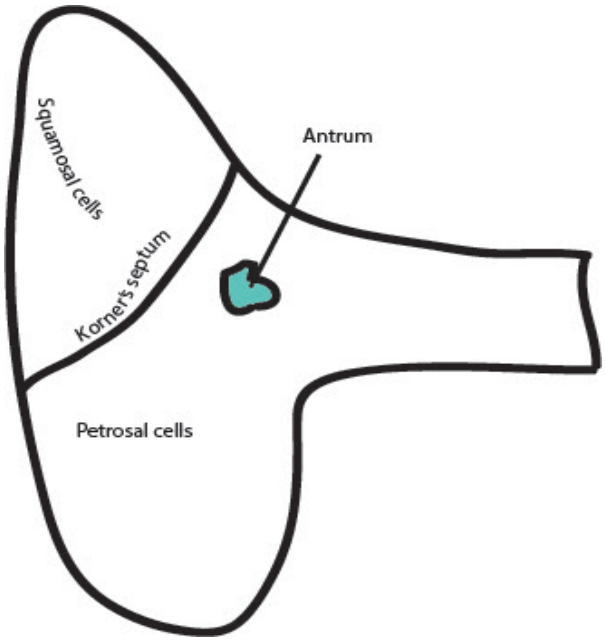


Figure showing korner's septum

At birth mastoid bone contains only one large air cell (the antrum). After birth mastoid air cells develop as an outgrowth of the antrum. Air cell tracts lined by epithelium buds off from the antrum and extend into adjacent areas of temporal bone to form mastoid air cells. This entire process is known as pneumatization. These buds arising from antrum to form well established tracts. The process of pneumatization is facilitated by differentiation of bone marrow into loose mesenchyme.

The process of mastoid pneumatization passes through a number of tracts. These tracts have been well established and documented. The main tracts involved in the process of pneumatization are as follows:

The posterosuperior cell tract:

This tract extends medially from the antrum at the junction of the posterior and middle cranial fossa dural plates. This tract traverses above the superior semicircular canal and the internal acoustic meatus. This tract pneumatizes the medial pyramid of temporal bone.

Posteromedial cell tract:

This tract is also known as superior retro-labyrinthine tract. This tract extends medially through the antrum slightly parallel and inferior to the posterosuperior tract and ends up pneumatizing the medial pyramid.

Subarcuate cell tract:

This is also known as translabyrinthine tract. This tract is situated more medially. Arising from the mastoid antrum it extends anteromedially passing under the superior semicircular canal and ends up pneumatizing petrous apex area.

Perilabyrinthine cell tract:

This tract arises from the mastoid antrum and pneumatizes the labyrinthine area. It is divided into supralabyrinthine and infra-labyrinthine tracts. This tract can extend up to the petrous apex.

Peritubal tract:

This tract arises from mastoid antrum and pneumatizes the tubal and peritubal area passing inferior to the labyrinth.

Phases of mastoid pneumatization:

Pneumatization of mastoid begins during the 33rd week of gestation. The process of pneumatization continues till the individual reaches puberty. The cells around petrous apex are the last to develop. Air cells around petrous apex are present in about 40% of normal individuals.

From birth to puberty three phases of mastoid pneumatization has been observed.

Phase I:

This takes place between 0-1 year. The antrum is adult size at birth (1cm²). During the first year of life there is rapid development of mastoid air cell system. These newly developing mastoid air cells add about 3cm² to the existing mastoid air cell area. At the end of the first year the surface area of mastoid air cell system becomes 4 cm². The mastoid process increases by 1 cm in length and width and 1cm in depth.

Phase II:

This phase takes place between 1-6 years of life. Mastoid pneumatization continues to increase in linear pattern increasing the air cell surface by 1cm² per year. By the age of 2 the mastoid tip covers the exit of facial nerve (stylomastoid foramen). The mastoid process grows at the rate of 0.5 cm in length and width while it increases by 0.25 cm in depth per year.

Phase III:

This phase occurs between 6 years and continues up to the puberty. This phase of pneumatization is rather slow when compared to the first and second phases. Pneumatization process continues till puberty when the surface area of mastoid air cells reach 12 cm².

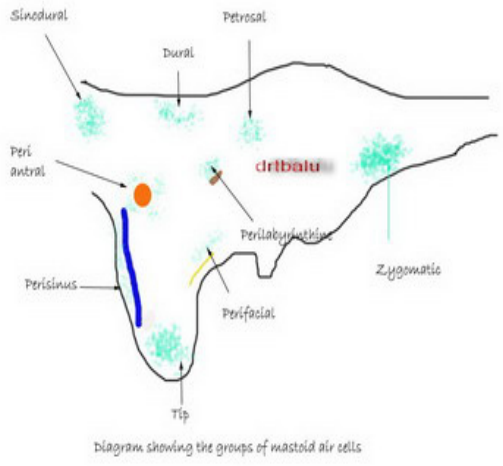
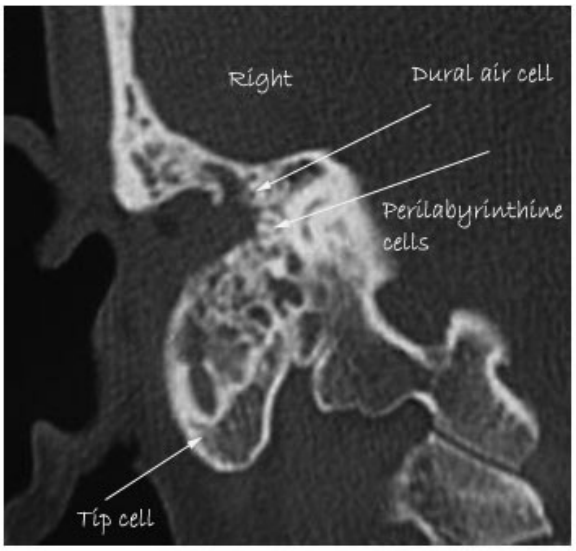


Diagram showing pneumatization process



HR CT of temporal bone showing the process of pneumatization

Pneumatization of mastoid show considerable amount of variations among individuals. These variations are related to factors like:

1. Heredity
2. Environment
3. Infections
4. Eustachean tube function

Various theories have been proposed to account for the pneumatization process. These are:

Environmental theory:

According to this theory prevalence of middle ear diseases in children play a huge role in determining the extent of pneumatization. Repeated infections involving the middle ear mucosa during childhood may halt / or prove to be detrimental to normal pneumatization process.

Genetic theory:

This theory suggests that amount of pneumatization is decided by genetic factors. Gene induced reduction of pneumatization process in a child may predispose to repeated middle ear infections.

Since the mastoid process is undeveloped / underdeveloped at birth the facial nerve lies superficial at the level of its exit out of stylomastoid foramen. At this level facial nerve can be injured during difficult forceps delivery. At the age of 2 development of mastoid process downwards and forwards protects the facial nerve as it exits from stylomastoid foramen.

Temporal Bone Pneumatization

"Success of middle ear surgery is dependent of the degree of mastoid pneumatization" Holmquist

Introduction:

Pneumatization of temporal bone is divided in to 5 compartments:
1. Middle ear
2. Mastoid
3. Perilabyrinthine
4. Petrous apex
5 Accessory: This region include Squamous, zygomatic, occipital and styloid cells.

Pneumatization of temporal bone follows definite cell tracts. These tracts are:
1. Posterosuperior cell tract
2. Posteromedial cell tract
3. Subarcuate cell tract
4. Perilabyrinthine cell tract
5. Peritubal cell tract

These tracts communicate with each other.

Posterosuperior and posteromedial cell tract:

These tracts extend medially through the antrum to pneumatize the medial pyramid. The posterosuperior tract lies at the level of above the level of Internal acoustic meatus.
Subarcuate tract:

This tract arises more medially from the mastoid antrum, extending anteromedially passing below the superior semicircular canal. This tract often participates in the formation of posterosuperior tract and may pneumatize the petrous apex.

Perilabyrinthine cell tract:

This tract pneumatizes the labyrinthine area. It divides into supralabyrinthine and infra labyrinthine tracts.

Peritubal cell tract:

This tract pneumatizes the tubal and peritubal area.

Functions of temporal bone air cells:

1. Sound reception

2. Resonance

3. Insulation

4. Supplementary air reservoir

5. Sound dissipation

6. Lightening the weight of skull

7. Protection against injury

Pneumatization of mastoid region is of three types:

1. Sclerotic mastoid - Absent pneumatization. The non pneumatized portion is covered with dense bone.

2. Diploic mastoid - Partial pneumatization. The non pneumatized area is filled with bone marrow.

3. Pneumatic mastoid - Complete pneumatization

The development of air cavities begin with the formation of bony cavities. This process is dependent on the normal periosteal activity. This cavity is known to contain primitive bone marrow. This bone marrow gets transformed into loose mesenchymal connective tissue. This cavity gets invaded by mucosa from the middle ear cavity.

Areas of temporal bone that are normally pneumatized:

1. Middle ear: Epitympanum, mesotympanum and hypotympanum

2. Squamomastoid: Antrum, central mastoid tract, and peripheral cells

3. Perilabyrinthine: Supralabyrinthine and infralabyrinthine

4. Petrous apex: Petrosal cells and apical cells

5. Accessory cells: Zygomatic cells, occipital cells, squamous cells and styloid cells

Temporal bone pneumatization can be best studied by High resolution CT scan.

Temporal bone pneumatization is symmetrical in 75% of normal individuals. Any asymmetrical pneumatization indicates middle ear disease.

Mastoid pneumatization can fail due to various causes leading to the formation of sclerosed mastoid. Various theories have been proposed to account for this failure of pneumatization process. These theories include:

Wittmaack theory: This theory is otherwise known as endodermal theory. This was first proposed by Wittmaack, who believed normal middle ear mucosa is a must for normal pneumatization to proceed. In the presence of infantile otitis media the pneumatization of temporal bone may get arrested causing a failure of the process of pneumatization. Infantile otitis media is common in premature infants due to meconium soiling of the middle ear cavity.

Tumarkin's theory: This theory proposed by Tumarkin states that failure of pneumatization occur due to failure of middle ear aeration due to eustachean tube dysfunction.

Diamant and Dahlberg: Suggested that dense bone is congenital and is a normal anatomical variant.

Ikarashi proved that long lasting inflammation increases bone mass thereby preventing normal pneumatization.

Factors determining the middle ear pressure:

1. Ventilation from eustachean tube

2. Passing of gases into circulation by diffusion

3. Thickness of middle ear mucosa

4. Elasticity of tympanic membrane

5. Size of mastoid pneumatization

Temporal bone anatomy

The temporal bones are situated at the sides and base of the skull. Each consists of five parts, viz., the squama, the petrous, mastoid, and tympanic parts, and the styloid process.

The Squama (squama temporalis)—The squama forms the anterior and upper part of the bone, and is scale-like, thin, and translucent.

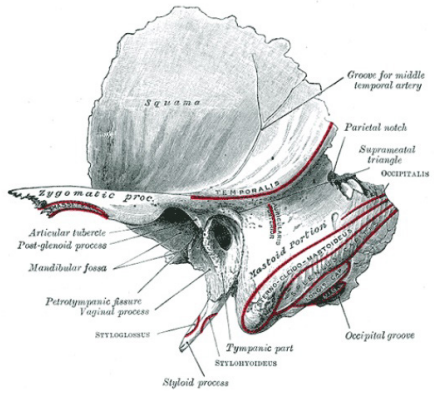


Figure showing outer surface of left temporal bone

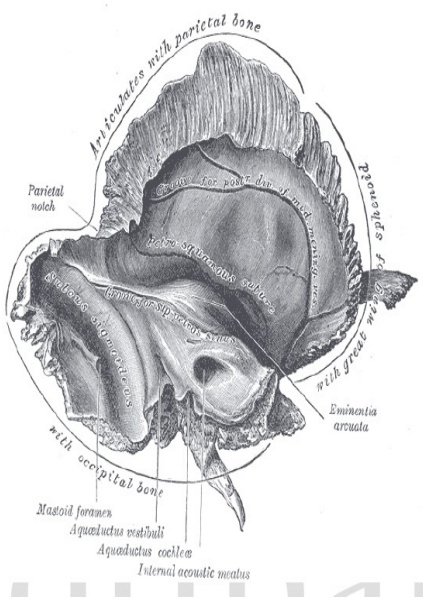
Surfaces — Its outer surface is smooth and convex; it affords attachment to the Temporalis muscle, and forms part of the temporal fossa; on its hinder part is a vertical groove for the middle temporal artery. A curved line, the temporal line, or supramastoid crest, runs backward and upward across its posterior part; it serves for the attachment of the temporal fascia, and limits the origin of the Temporalis muscle. The boundary between the squama and the mastoid portion of the bone, as indicated by traces of the original suture, lies about 1 cm. below this line. Projecting from the lower part of the squama is a long, arched process, the zygomatic process. This process is at first directed lateral ward, its two surfaces looking upward and downward; it then appears as if twisted inward upon itself, and runs forward, its surfaces now looking medial ward and lateral ward. The superior border is long, thin, and sharp, and serves for the attachment of the temporal fascia; the inferior, short, thick, and arched, has attached to it some fibers of the Masseter.

The lateral surface is convex and subcutaneous; the medial is concave, and affords attachment to the Masseter. The anterior end is deeply serrated and articulates with the zygomatic bone. The posterior end is connected to the squama by two roots, the anterior and posterior roots. The posterior root, a prolongation of the upper border, is strongly marked; it runs backward above the external acoustic meatus, and is continuous with the temporal line. The anterior root, continuous with the lower border, is short but broad and strong; it is directed medial ward and ends in a rounded eminence, the articular tubercle (eminentia articularis).

This tubercle forms the front boundary of the mandibular fossa, and in the fresh state is covered with cartilage. In front of the articular tubercle is a small triangular area which assists in forming the infratemporal fossa; this area is separated from the outer surface of the squama by a ridge which is continuous behind with the anterior root of the zygomatic process, and in front, in the articulated skull, with the infratemporal crest on the great wing of the sphenoid. Between the posterior wall of the external acoustic meatus and the posterior root of the zygomatic process is the area called the suprameatal triangle (Macewen), or mastoid fossa, through which an instrument may be pushed into the tympanic antrum.

At the junction of the anterior root with the zygomatic process is a projection for the attachment of the temporomandibular ligament; and behind the anterior root is an oval depression, forming part of the mandibular fossa, for the reception of the condyle of the mandible. The mandibular fossa (glenoid fossa) is bounded, in front, by the articular tubercle; behind, by the tympanic part of the bone, which separates it from the external acoustic meatus; it is divided into two parts by a narrow slit, the petrotympanic fissure (Glaserian fissure). The anterior part, formed by the squama, is smooth, covered in the fresh state with cartilage, and articulates with the condyle of the mandible. Behind this part of the fossa is a small conical eminence; this is the representative of a prominent tubercle which, in some mammals, descends behind the condyle of the mandible, and prevents its backward displacement. The posterior part of the mandibular fossa, formed by the tympanic part of the bone, is non-articular, and sometimes lodges a portion of the parotid gland.

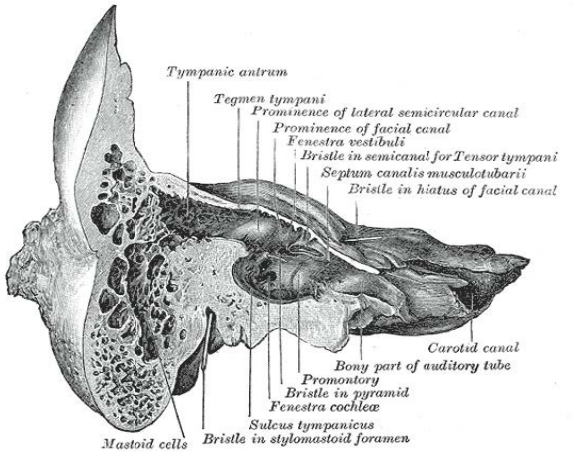
The petrotympanic fissure leads into the middle ear or tympanic cavity; it lodges the anterior process of the malleus, and transmits the tympanic branch of the internal maxillary artery. The chorda tympani nerve passes through a canal (canal of Huguier), separated from the anterior edge of the petrotympanic fissure by a thin scale of bone and situated on the lateral side of the auditory tube, in the retiring angle between the squama and the petrous portion of the temporal bone.



Inner surface of left temporal bone

The internal surface of the squama is concave; it presents depressions corresponding to the convolutions of the temporal lobe of the brain, and grooves for the branches of the middle meningeal vessels.

Borders: — The superior border is thin, and bevelled at the expense of the internal table, so as to overlap the squamous border of the parietal bone, forming with it the squamosal suture. Posteriorly, the superior border forms an angle, the parietal notch, with the mastoid portion of the bone. The antero-inferior border is thick, serrated, and bevelled at the expense of the inner table above and of the outer below, for articulation with the great wing of the sphenoid.
Mastoid Portion (pars mastoidea). — The mastoid portion forms the posterior part of the bone.



Coronal section of right temporal bone

Surfaces — Its outer surface is rough, and gives attachment to the Occipitalis and Auricularis posterior. It is perforated by numerous foramina; one of these, of large size, situated near the posterior border, is termed the mastoid foramen; it transmits a vein to the transverse sinus and a small branch of the occipital artery to the dura mater.

The position and size of this foramen are very variable; it is not always present; sometimes it is situated in the occipital bone, or in the suture between the temporal and the occipital. The mastoid portion is continued below into a conical projection, the mastoid process, the size and form of which very somewhat; it is larger in the male than in the female. This process serves for the attachment of the Sternocleidomastoideus, Splenius capitis, and Longissimus capitis. On the medial side of the process is a deep groove, the mastoid notch (digastric fossa), for the attachment of the Digastricus; medial to this is a shallow furrow, the occipital groove, which lodges the occipital artery.

The inner surface of the mastoid portion presents a deep, curved groove, the sigmoid sulcus, which lodges part of the transverse sinus; in it may be seen the opening of the mastoid foramen. The groove for the transverse sinus is separated from the innermost of the mastoid air cells by a very thin lamina of bone, and even this may be partly deficient.

Borders: The superior border of the mastoid portion is broad and serrated, for articulation with the mastoid angle of the parietal. The posterior border, also serrated, articulates with the inferior border of the occipital between the lateral angle and jugular process. Anteriorly the mastoid portion is fused with the descending process of the squama above; below it enters into the formation of the external acoustic meatus and the tympanic cavity.

A section of the mastoid process shows it to be hollowed out into a number of spaces, the mastoid cells, which exhibit the greatest possible variety as to their size and number. At the upper and front part of the process they are large and irregular and contain air, but toward the lower part they diminish in size, while those at the apex of the process are frequently quite small and contain marrow; occasionally they are entirely absent, and the mastoid is then solid throughout.

In addition to these a large irregular cavity is situated at the upper and front part of the bone. It is called the tympanic antrum, and must be distinguished from the mastoid cells, though it communicates with them. Like the mastoid cells it is filled with air and lined by a prolongation of the mucous membrane of the tympanic cavity, with which it communicates. The tympanic antrum is bounded above by a thin plate of bone, the tegmen tympani, which separates it from the middle fossa of the base of the skull; below by the mastoid process; laterally by the squama just below the temporal line, and medially by the lateral semicircular canal of the internal ear which projects into its cavity. It opens in front into that portion of the tympanic cavity which is known as the attic or epitympanic recess. The tympanic antrum is a cavity of some considerable size at the time of birth; the mastoid air cells may be regarded as diverticula from the antrum, and begin to appear at or before birth; by the fifth year they are well-marked, but their development is not completed until toward puberty.

Petrous Portion (pars petrosa [pyramis]).—The petrous portion or pyramid is pyramidal and is wedged in at the base of the skull between the sphenoid and occipital. Directed medialward, forward, and a little upward, it presents for examination a base, an apex, three surfaces, and three angles, and contains, in its interior, the essential parts of the organ of hearing.

Base: The base is fused with the internal surfaces of the squama and mastoid portion.

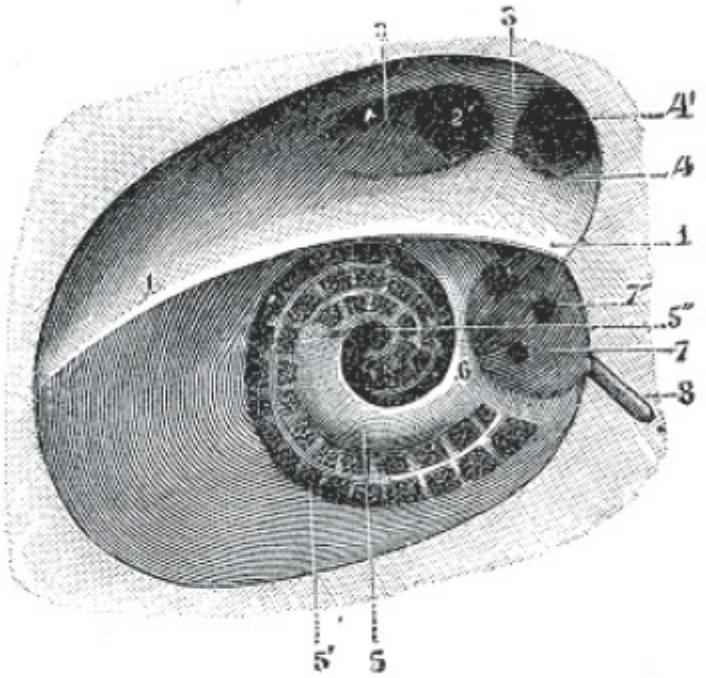
Apex: The apex, rough and uneven, is received into the angular interval between the posterior border of the great wing of the sphenoid and the basilar part of the occipital; it presents the anterior or internal orifice of the carotid canal, and forms the postero-lateral boundary of the foramen lacerum.

Surfaces: The anterior surface forms the posterior part of the middle fossa of the base of the skull, and is continuous with the inner surface of the squamous portion, to which it is united by the petrosquamous suture, remains of which are distinct even at a late period of life. It is marked by depressions for the convolutions of the brain, and presents six points for examination: (1) near the center, an eminence (eminentia arcuata) which indicates the situation of the superior semicircular canal; (2) in front of and a little lateral to this eminence, a depression indicating the position of the tympanic cavity: here the layer of bone which separates the tympanic from the cranial cavity is extremely thin, and is known as the tegmen tympani; (3) a shallow groove, sometimes double, leading lateralward and backward to an oblique opening, the hiatus of the facial canal, for the passage of the greater superficial petrosal nerve and the petrosal branch of the middle meningeal artery; (4) lateral to the hiatus, a smaller opening, occasionally seen, for the passage of the lesser superficial petrosal nerve; (5) near the apex of the bone, the termination of the carotid canal, the wall of which in this situation is deficient in front; (6) above this canal the shallow trigeminal impression for the reception of the semilunar ganglion.

The posterior surface forms the front part of the posterior fossa of the base of the skull, and is continuous with the inner surface of the mastoid portion. Near the center is a large orifice, the internal acoustic meatus, the size of which varies considerably; its margins are smooth and rounded, and it leads into a short canal, about 1 cm. in length, which runs lateralward. It transmits the facial and acoustic nerves and the internal auditory branch of the basilar artery.

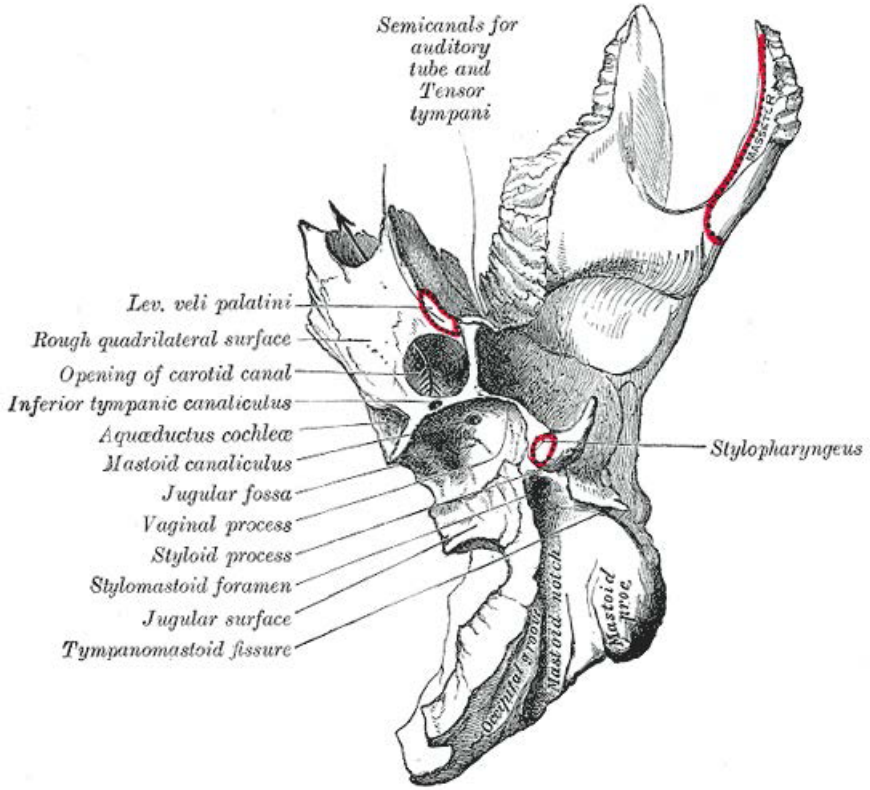
The lateral end of the canal is closed by a vertical plate, which is divided by a horizontal crest, the crista falciformis, into two unequal portions (Fig. 4). Each portion is further subdivided by a vertical ridge into an anterior and a posterior part. In the portion beneath the crista falciformis are three sets of foramina; one group, just below the posterior part of the crest, situated in the area cribrosa media, consists of several small openings for the nerves to the saccule; below and behind this area is the foramen singulare, or opening for the nerve to the posterior semicircular duct; in front of and below the first is the tractus spiralis foraminosus, consisting of a number of small spirally arranged openings, which encircle the canalis centralis cochleæ; these openings together with this central canal transmit the nerves to the cochlea. The portion above the crista falciformis presents behind, the area cribrosa superior, pierced by a series of small openings, for the passage of the nerves to the utricle and the superior and lateral semicircular ducts, and, in front, the area facians, with one large opening, the commencement of the canal for the facial nerve (aquæductus Fallopii). Behind the internal acoustic meatus is a small slit almost hidden by a thin plate of bone, leading to a canal, the aquæductus vestibuli, which transmits the ductus endolymphaticus together with a small artery and vein. Above and between these two openings is an irregular depression which lodges a process of the dura mater and transmits a small vein; in the infant this depression is represented by a large fossa, the subarcuate fossa, which extends backward as a blind tunnel under the superior semicircular canal.

The inferior surface is rough and irregular, and forms part of the exterior of the base of the skull. It presents eleven points for examination: (1) near the apex is a rough surface, quadrilateral in form, which serves partly for the attachment of the Levator veli palatini and the cartilaginous portion of the auditory tube, and partly for connection with the basilar part of the occipital bone through the intervention of some dense fibrous tissue; (2) behind this is the large circular aperture of the carotid canal, which ascends at first vertically, and then, making a bend, runs horizontally forward and medialward; it transmits into the cranium the internal carotid artery, and the carotid plexus of nerves; (3) medial to the opening for the carotid canal and close to its posterior border, in front of the jugular fossa, is a triangular depression; at the apex of this is a small opening, the aquæductus cochleæ, which lodges a tubular prolongation of the dura mater establishing a communication between the perilymphatic space and the subarachnoid space, and transmits a vein from the cochlea to join the internal jugular; (4) behind these openings is a deep depression, the jugular fossa, of variable depth and size in different skulls; it lodges the bulb of the internal jugular vein; (5) in the bony ridge dividing the carotid canal from the jugular fossa is the small inferior tympanic canaliculus for the passage of the tympanic branch of the glossopharyngeal nerve; (6) in the lateral part of the jugular fossa is the mastoid canaliculus for the entrance of the auricular branch of the vagus nerve; (7) behind the jugular fossa is a quadrilateral area, the jugular surface, covered with cartilage in the fresh state, and articulating with the jugular process of the occipital bone; (8) extending backward from the carotid canal is the vaginal process, a sheath-like plate of bone, which divides behind into two laminæ; the lateral lamina is continuous with the tympanic part of the bone, the medial with the lateral margin of the jugular surface; (9) between these laminæ is the styloid process, a sharp spine, about 2.5 cm. in length; (10) between the styloid and mastoid processes is the stylomastoid foramen; it is the termination of the facial canal, and transmits the facial nerve and stylomastoid artery; (11) situated between the tympanic portion and the mastoid process is the tympanomastoid fissure, for the exit of the auricular branch of the vagus nerve.



Diagrammatic view of the fundus of the right internal acoustic meatus.

1.Crista falciformis. 2. Area facialis, with (2′) internal opening of the facial canal. 3. Ridge separating the area facialis from the area cribrosa superior. 4. Area cribrosa superior, with (4′) openings for nerve filaments. 5. Anterior inferior cribriform area, with (5′) the tractus spiralis foraminosus, and (5″) the canalis centralis of the cochlea. 6. Ridge separating the tractus spiralis foraminosus from the area cribrosa media. 7. Area cribrosa media, with (7′) orifices for nerves to saccule. 8. Foramen singulare



Left temporal bone inferior surface

Angles: The superior angle, the longest, is grooved for the superior petrosal sinus, and gives attachment to the tentorium cerebelli; at its medial extremity is a notch, in which the trigeminal nerve lies. The posterior angle is intermediate in length between the superior and the anterior. Its medial half is marked by a sulcus, which forms, with a corresponding sulcus on the occipital bone, the channel for the inferior petrosal sinus. Its lateral half presents an excavation—the jugular fossa—which, with the jugular notch on the occipital, forms the jugular foramen; an eminence occasionally projects from the center of the fossa, and divides the foramen into two. The anterior angle is divided into two parts—a lateral joined to the squama by a suture (petrosquamous), the remains of which are more or less distinct; a medial, free, which articulates with the spinous process of the sphenoid.

At the angle of junction of the petrous part and the squama are two canals, one above the other, and separated by a thin plate of bone, the septum canalis musculotubarii (processus cochleariformis); both canals lead into the tympanic cavity. The upper one (semicanalis m. tensoris tympani) transmits the Tensor tympani, the lower one (semicanalis tubæ auditivæ) forms the bony part of the auditory tube.

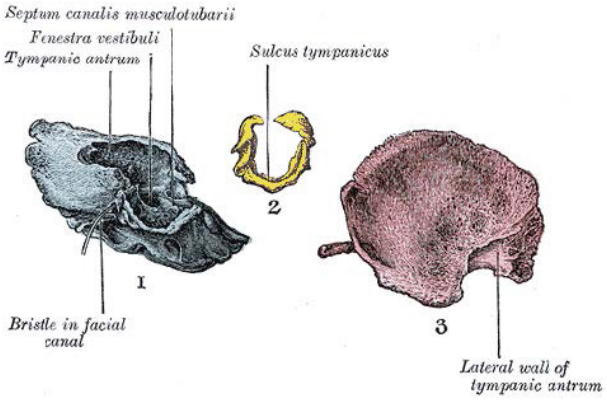
Tympanic Part (pars tympanica): The tympanic part is a curved plate of bone lying below the squama and in front of the mastoid process.
Surfaces: Its postero-superior surface is concave, and forms the anterior wall, the floor, and part of the posterior wall of the bony external acoustic meatus. Medially, it presents a narrow furrow, the tympanic sulcus, for the attachment of the tympanic membrane. Its antero-inferior surface is quadrilateral and slightly concave; it constitutes the posterior boundary of the mandibular fossa, and is in contact with the retromandibular part of the parotid gland.

Borders: Its lateral border is free and rough, and gives attachment to the cartilaginous part of the external acoustic meatus. Internally, the tympanic part is fused with the petrous portion, and appears in the retreating angle between it and the squama, where it lies below and lateral to the orifice of the auditory tube. Posteriorly, it blends with the squama and mastoid part, and forms the anterior boundary of the tympanomastoid fissure. Its upper border fuses laterally with the back of the postglenoid process, while medially it bounds the petrotympanic fissure. The medial part of the lower border is thin and sharp; its lateral part splits to enclose the root of the styloid process, and is therefore named the vaginal process. The central portion of the tympanic part is thin, and in a considerable percentage of skulls is perforated by a hole, the foramen of Huschke.

The external acoustic meatus is nearly 2 cm. long and is directed inward and slightly forward: at the same time it forms a slight curve, so that the floor of the canal is convex upward. In sagittal section it presents an oval or elliptical shape with the long axis directed downward and slightly backward. Its anterior wall and floor and the lower part of its posterior wall are formed by the tympanic part; the roof and upper part of the posterior wall by the squama. Its inner end is closed, in the recent state, by the tympanic membrane; the upper limit of its outer orifice is formed by the posterior root of the zygomatic process, immediately below which there is sometimes seen a small spine, the suprameatal spine, situated at the upper and posterior part of the orifice.
Styloid Procéss (processus styloideus): The styloid process is slender, pointed, and of varying length; it projects downward and forward, from the under surface of the temporal bone. Its proximal part (tympanohyal) is ensheathed by the vaginal process of the tympanic portion, while its distal part (stylohyal) gives attachment to the stylohyoid and stylomandibular ligaments, and to the Styloglossus, Stylohyoideus, and Stylopharyngeus muscles.

The stylohyoid ligament extends from the apex of the process to the lesser cornu of the hyoid bone, and in some instances is partially, in others completely, ossified.

Structure: The structure of the squama is like that of the other cranial bones: the mastoid portion is spongy, and the petrous portion dense and hard.



The three principal parts of the tempora bone at birth.
1. Outer surface of petromastoid part.
2. Outer surface of tympanic ring.
3. Inner surface of squama

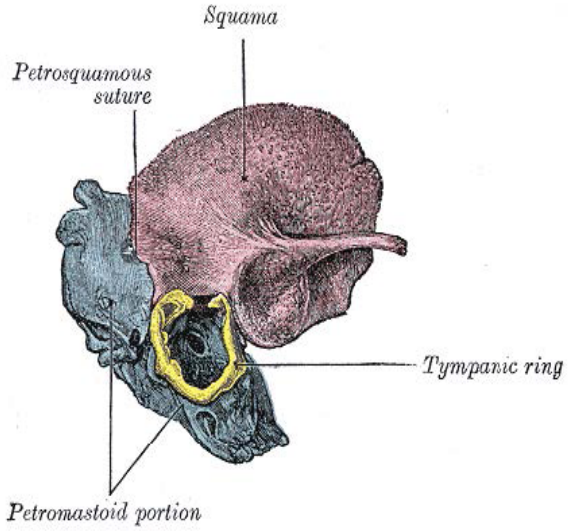


Figure showing temporal bone at birth inner aspect

Ossification—The temporal bone is ossified from eight centers, exclusive of those for the internal ear and the tympanic ossicles, viz., one for the squama including the zygomatic process, one for the tympanic part, four for the petrous and mastoid parts, and two for the styloid process. Just before the close of fetal life (Fig. 7) the temporal bone consists of three principal parts:

1. The squama is ossified in membrane from a single nucleus, which appears near the root of the zygomatic process about the second month.

2. The petromastoid part is developed from four centers, which make their appearance in the cartilaginous ear capsule about the fifth or sixth month.

One appears in the neighbourhood of the eminentia arcuata, spreads in front and above the internal acoustic meatus and extends to the apex of the bone; it forms part of the cochlea, vestibule, superior semicircular canal, and medial wall of the tympanic cavity. A second (opisthotic) appears at the promontory on the medial wall of the tympanic cavity and surrounds the fenestra cochleæ; it forms the floor of the tympanic cavity and vestibule, surrounds the carotid canal, invests the lateral and lower part of the cochlea, and spreads medially below the internal acoustic meatus. A third (pterotic) roofs in the tympanic cavity and antrum; while the fourth (epiotic) appears near the posterior semicircular canal and extends to form the mastoid process. 3. The tympanic ring is an incomplete circle, in the concavity of which is a groove, the tympanic sulcus, for the attachment of the circumference of the tympanic membrane. This ring expands to form the tympanic part, and is ossified in membrane from a single center which appears about the third month. The styloid process is developed from the proximal part of the cartilage of the second branchial or hyoid arch by two centers:
one for the proximal part, the tympanohyal, appears before birth; the other, comprising the rest of the process, is named the stylohyal, and does not appear until after birth. The tympanic ring unites with the squama shortly before birth; the petromastoid part and squama join during the first year, and the tympanohyal portion of the styloid process about the same time. The stylohyal does not unite with the rest of the bone until after puberty and in some skulls never at all.

The chief subsequent changes in the temporal bone apart from increase in size are:

(1) The tympanic ring extends outward and backward to form the tympanic part. This extension does not, however, take place at an equal rate all around the circumference of the ring, but occurs most rapidly on its anterior and posterior portions, and these outgrowths meet and blend, and thus, for a time, there exists in the floor of the meatus a foramen, the foramen of Huschke; this foramen is usually closed about the fifth year, but may persist throughout life.
(2) The mandibular fossa is at first extremely shallow, and looks lateralward as well as downward; it becomes deeper and is ultimately directed downward. Its change in direction is accounted for as follows. The part of the squama which forms the fossa lies at first below the level of the zygomatic process. As, however, the base of the skull increases in width, this lower part of the squama is directed horizontally inward to contribute to the middle fossa of the skull, and its surfaces therefore come to look upward and downward; the attached portion of the zygomatic process also becomes everted, and projects like a shelf at right angles to the squama.
(3) The mastoid portion is at first quite flat, and the stylomastoid foramen and rudimentary styloid process lie immediately behind the tympanic ring. With the development of the air cells the outer part of the mastoid portion grows downward and forward to form the mastoid process, and the styloid process and stylomastoid foramen now come to lie on the under surface. The descent of the foramen is necessarily accompanied by a corresponding lengthening of the facial canal.
(4) The downward and forward growth of the mastoid process also pushes forward the tympanic part, so that the portion of it which formed the original floor of the meatus and contained the foramen of Huschke is ultimately found in the anterior wall. (5) The fossa subarcuata becomes filled up and almost obliterated.
Articulations: The temporal articulates with five bones: occipital, parietal, sphenoid, mandible and zygomatic.

Surgical Anatomy of Temporal bone:

The extra cranial surface of the Temporal bone: After exposure of the mastoid process using the retroauricular approach or the endaural approach, the supra meatal spine (Henle's spine), situated posterosuperiorly at the entrance of the ear canal has to be located. The shape and position of the suprameatal spine varies. 4 variants of the suprameatal spine have been described.

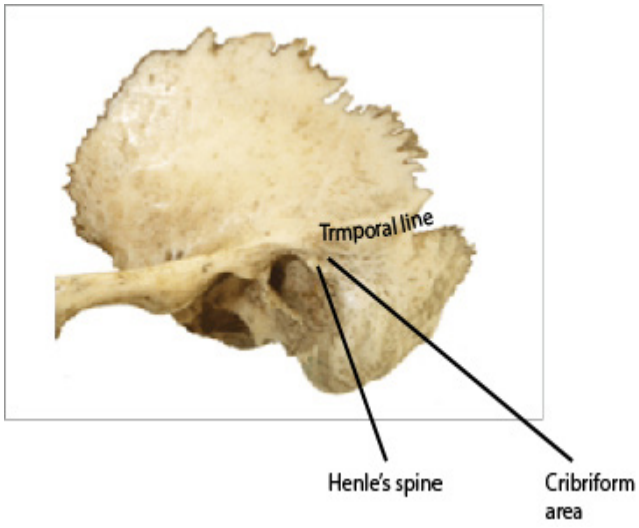
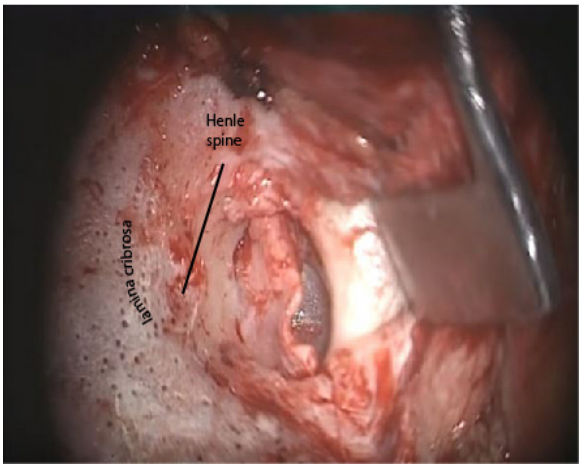
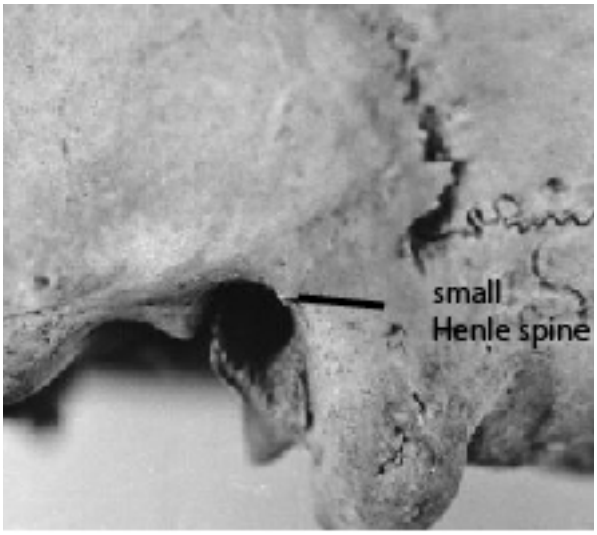


Image of temporal bone lateral surface showing Henle's spine and cribriform area



Henle's spine as seen during surgery

Variant 1: The suprameatal spine is small and smoothly contoured. In these cases, the tympanic bone, forming the anterior wall of the external acoustic meatus, is a vertical plate, and the styloid process is very short. A straight external acoustic meatus provides the best visualisation of the drum and tympanic cavity, and is the most favourable shape for tympanoplasty.



Surgeon's view - The suprameatal spine (SS) is small. The tympanosquamous suture is not prominent, and the anterior part of the tympanic bone is a vertical plate, allowing a relatively wide ear canal. The styloid process is very short.

Variant 2: The suprameatal spine is sharp and elongated. The deep tympanosquamous suture deludes a prominent anterior part of the tympanic bone, but the bone is thin and straight plate that provides good access to the middle ear.
The skin is difficult to elevate from the deep suture, and the bone itself is not prominent. The styloid process is short.

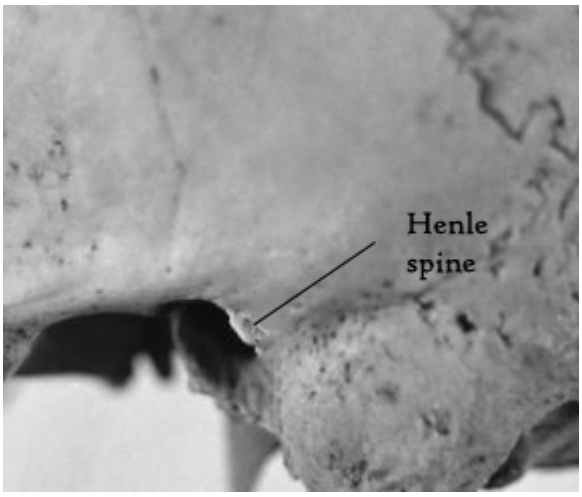


Photo showing variant 2 of Henle's spine

Variant 3: The aperture for vessels lies somewhat superoposteriorly, close to the temporal line, and there is no true suprameatal crest, only a cone shaped depression. The styloid process is relatively long. The anterior part of the tympanic bone is thick and prominent.

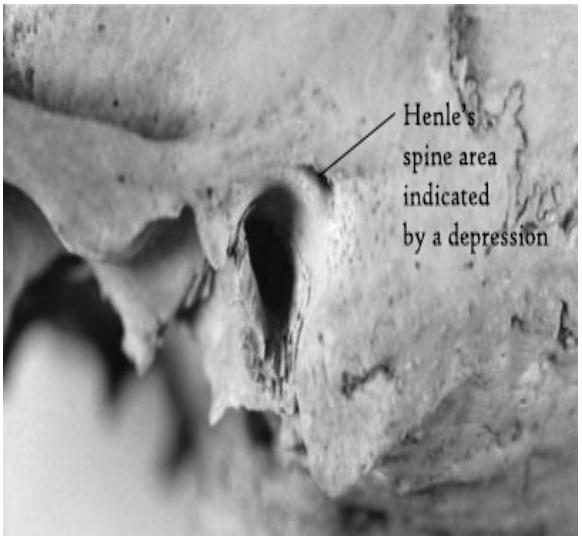
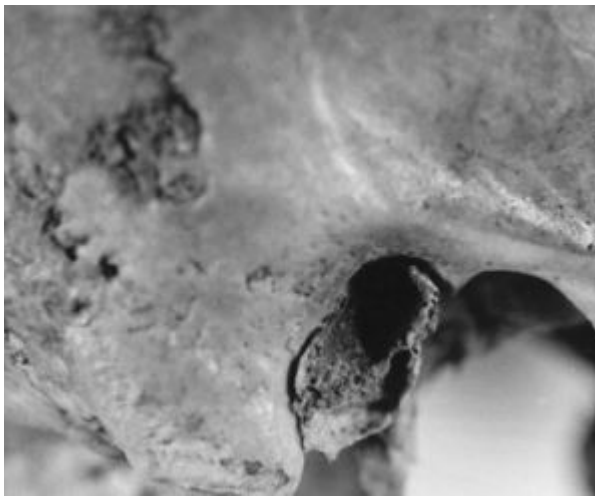


Photo showing third variant of Henle's spine

Variant 4: The suprameatal area is smooth, and there is no suprameatal crest. The external auditory meatus is oval in outline, and the tympanic part of the temporal bone, forming the anterior wall of the ear canal is prominent. The styloid process is very long.



Photograph showing variant 4 of Henle's spine which is absent spine

The suprameatal triangle also known as the Maceven's triangle is a depression that marks the lateral wall of mastoid antrum. This is the most valuable surgical landmark during mastoid surgeries to indicate the position of the antrum. Henle's spine forms the anterior boundary of the suprameatal triangle.

The temporal line is a horizontal ridge, continuing from the superior border of the zygomatic process posteriorly onto the mastoid cortex. This line approximately indicates the level of the middle cranial fossa dura. It is not always evident, sometimes it is a prominent sharp ridge, sometimes a broad prominence and in some other cases hardly recognizable. It is safest to start drilling the mastoid cortex a few millimetres below the temporal line and gradually proceed towards it.

Posterior to the suprameatal spine, a group of small holes in the mastoid cortex are occasionally seen, described as the cribriform area. Small vessels pass through these foramina to the mucosa of the underlying antrum in infants, and it is here that a Subperiosteal abscess forms in cases of acute coalescent mastoiditis. The cribriform area lies within Macewen's triangle an imaginary triangle defined by three lines. The first is the temporal line, the second is formed by the superior and posterior margins of the external bony meatus (this line goes through the suprameatal spine). The triangle is completed by a line drawn perpendicular to the first line and tangential to the second. This triangle roughly defines the mastoid antrum deep to the cortex.

Figure showing Maceven's triangle

Posteriorly, alongside the occipitomastoid suture, the mastoid foramen is visible, and this is the opening of the emissarium occipitale (santorini) (the occipital emissary vein) connecting the veins of the occipital region with the sigmoid sinus.

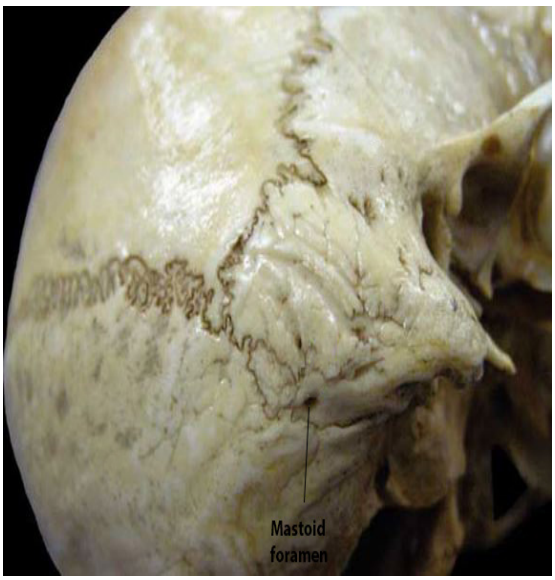


Figure showing mastoid foramen

The roof of the bony ear canal is an important landmark, and should be visualized in any type of Mastoidectomy except simple Mastoidectomy. The canal skin should be carefully elevated, and the tympanosquamous suture should be located at the 12-o'clock position and exposed. The skin is firmly attached at the suture by fibrous tissue, which can be cut with a sickle knife in order to expose the entire suture.
The size of the suture varies from a small, barely recognizable fissure to a deep notch, hampering the view of the Shrapnell's membrane region. Any irregular bone in the superior part of the ear canal should be drilled away in any type of Mastoidectomy.

The floor of the ear canal is formed by the tympanic bone, and should also be visualized before Mastoidectomy, at least as far as the tympanomastoid suture, which starts medially at the bony annulus at the 9-oclock position and continues inferolaterally towards the apex of the mastoid. Removal of the tympanic bone between the two sutures gives an excellent view of the entire hypotympanum.

The anatomy of the lateral part of the mastoid process varies widely in relation to the pneumatization, which can be almost non existent or extensive with full pneumatization. Towards the tip of the mastoid process the cells are usually larger.

Middle cranial fossa dural plate:
Superiorly, the middle cranial fossa dura plate has to be located. This is achieved by drilling superiorly in the mastoid process, towards the temporal line. The mastoid air cells superiorly lie just under the cortical bone, and end in a solid bony plate. This plate continues medially. When the bone at the middle cranial fossa dural plate is thinned, the dura becomes visible, initially pinkish due to the vascularity followed by the whitish color.
Laterally, the middle fossa dura plate is large. It extends from the zygomatic region anteriorly to the sinodural angle and the superior petrosal sinus posteriorly. Because the superior petrosal sinus runs in an anteromedial direction, the area of the middle fossa dura plate diminishes medially towards the labyrinth.

The middle fossa dura plate is the roof, or tegmen, of the pneumatized spaces of the temporal bone. The tegmen is divided into the tegmen mastoidei, the most lateral part; the tegmen antri at the level of the mastoid antrum; and the tegmen tympani at the level of epitympanum, extending medially as far as the superior semicircular canal and the epitympanic recess anteriorly.

Sigmoid sinus:
The posterior limit of the standard mastoid cavity is the sigmoid sinus, or the lateral sinus. It is a continuation of the transverse sinus, and passes through the deep part of the mastoid process, under the facial nerve, toward the jugular bulb. In a well pneumatized mastoid process, the lateral aspect of the sigmoid sinus is covered by the mastoid air cells. After removal of these cells, a slightly bluish discoloration of a smooth bony plate can be seen. With further thinning of the bone over the sinus, it becomes increasingly blue. The posterosuperior part of the sigmoid sinus is at the most superficial level. Inferiorly, the sinus lies gradually deeper, making an anterior curve and crossing the tip of the mastoid process at the level deep to the digastric crest. Adjacent to the vertical portion of the facial nerve before forming the jugular bulb, the sigmoid sinus lies the deepest.
The anatomy of the sigmoid sinus varies; it can be positioned anteriorly or posteriorly. It is more commonly anterior in poorly pneumatized bones.
Sinodural angle:
Is the angle between the middle fossa and posterior fossa dural plates and the superior part of the sigmoid sinus. In a well pneumatized ear, small air cells usually occupy the sinodural angle, and should be removed or opened in a mastoidectomy. Sometimes it is necessary to drill deep down into the angle, exposing the superior petrosal sinus. Laterally in this angle within the mastoid cortex, the emissarium mastoidium (citelli) is located. This emissary vein connects the sigmoid sinus with the veins draining the middle fossa dura and the temporal squama. The veins may lead to troublesome venous bleeding during the drilling of the cortical bone in the most lateral part of the sinodural angle. The sinodural angle is usually deep. In the depths of the angle, the superior petrosal sinus is located.

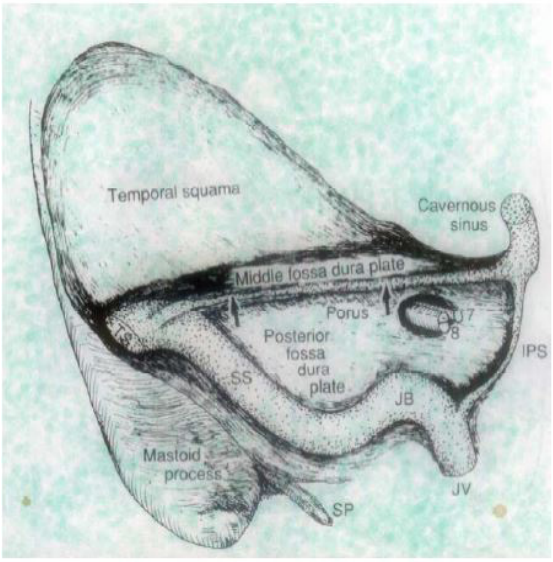


Figure showing inner surface of temporal bone – Superior petrosal sinus is indicated by arrows, IPS is inferior petrosal sinus, SS is sigmoid sinus TS is transverse sinus.

The superior petrosal sinus enters the sigmoid sinus on its medial surface, and runs in a bony sulcus along the edge of pars petrosal, dividing the middle fossa dura plate from the posterior fossa dura plate. It runs medially towards the petrous apex.

Korner's septum:
After exenteration of the superficial mastoid air cells, a thin bony plate can sometimes be seen at the bottom of the kidney shaped cavity. This plate is korner's septum, or petrosquamous lamina. In a well pneumatized mastoid process, the plate of Korner's septum is hardly recognizable. It is indicated by somewhat smaller cells in the upper part of the cavity. Korner's septum represents the site of embryonic fusion of the squamous and petrous portions of the temporal bone. Between the two components there is a suture line that is normally obliterated.

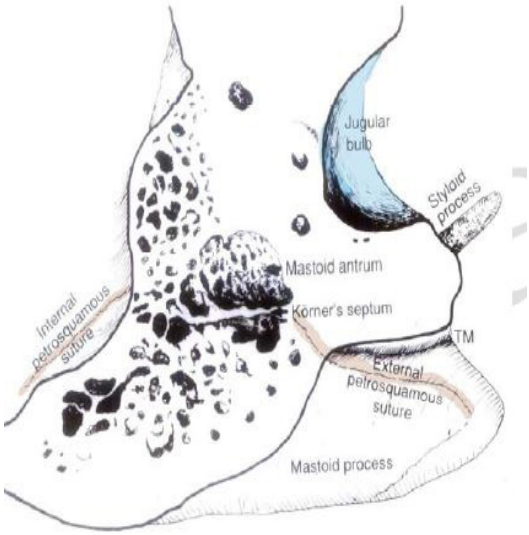


Image showing korner's septum continuing superiorly into the internal petrosquamous suture line and inferiorly into the external petrosquamous suture, dividing the superficial lateral mastoid
cells for these structures into squamomastoid cells and petromastoid cells lying medially to the
sutures.

The presence of korner's septum may give a false impression of having reached the antrum. When this bony septum is present it divides the mastoid process into a superficial squamous portion and a deep petrosal portion, both portions open separately into the antrum.

If the squamous portion is poorly pneumatized or sclerotic, there may be great difficulty finding the mastoid antrum, unless one is aware about the presence of korner's septum and its penetration is a must to reach the air cells in the deep portion of the temporal bone.

The presence of korner's septum must be contemplated if there is difficulty in approaching the antrum or if the antrum is small or constricted, or if it is felt that the antrum has an anomalous position.

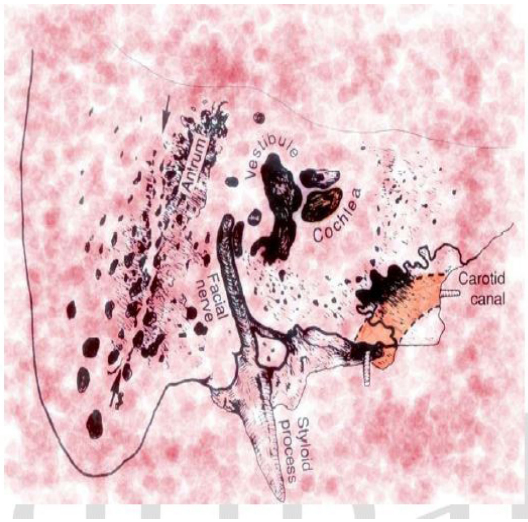


Figure showing Korner's septum (arrows) with the antrum and petromastoid cells medial to it and the squamomastoid cells lateral to it.

Whenever a persistent and dense korner's septum is found, it should be removed to eliminate a dual pneumatic system. Failure to recognize the presence of korner's septum may lead a surgeon to seek the antrum at a more superficial level. During the procedure, the surgeon may wrongly search for the antrum anteroinferiorly, and may expose and damage the facial nerve. In such cases, drilling along the middle fossa dura plate is therefore extremely important until the antrum and lateral semicircular canal are located

Mastoid antrum:

The antrum varies considerably in size; in a small sclerotic mastoid there is little or no pneumatization, the antrum is quite small. In ears with extensive pneumatization, it can be large. In ears with no petrosquamous lamina, there is no border between the large mastoid cells and the antrum. In such cases the antrum is large. Anterosuperiorly, the antrum continues along the tegmen to a narrow passage, the aditus ad antrum to the epitympanum.
The superior wall of the antrum is the tegmen antri. The medial wall of the antrum cavity is the labyrinth. Anteriorly, the lateral canal is immediately recognizable, and lateral to the canal, the incus body and short process of the incus are seen. The bone surrounding the antrum may be sclerotic, pneumatized with small air cells, or spongiotic in children.

The Labyrinth:
When the labyrinth is entered, the prominence of the lateral canal becomes visible and this is the most important landmark in the antrum. After removal of air cells the superior and posterior canals also become visible. The superior canal runs perpendicular to the lateral canal but is about 2 mm deeper. The anterior junction of the superior and lateral canals is at the anterior ampulla. The anterior crus then runs superiorly toward the tegmen tympani, and curves posteriorly to join with the posterior semicircular canal at the common crus. The posterior canal also runs perpendicular to the lateral canal. The posterior half of the posterior canal is located posterior to a line bisecting the lateral canal (Donaldson's line). The anterior half of the posterior canal runs anteriorly to the Donaldson's line and emerge deep to the facial nerve, to enter the vestibule.

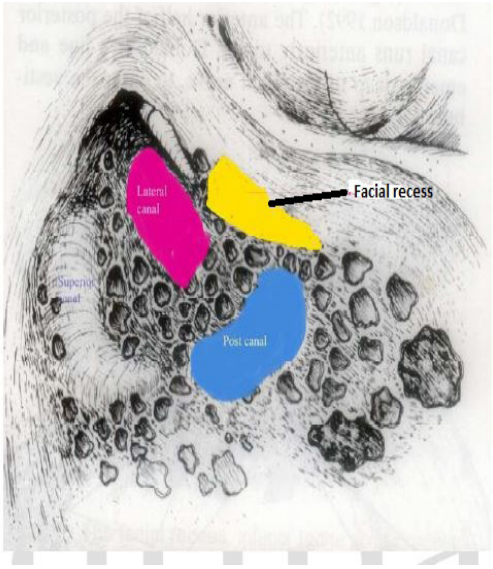


Figure demonstrating the three semicircular canals and facial recess.

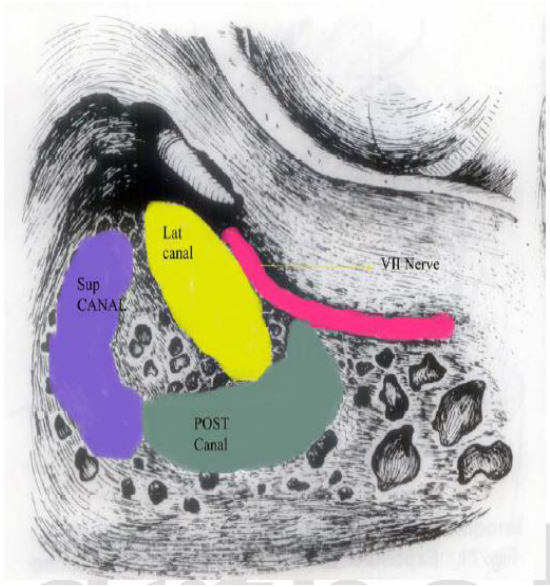


Image showing The supralabyrinthine, infralabyrinthine, and retro labyrinthine air cells are opened, the three semicircular canals are clearly seen, Facial nerve is also seen anteriorly.

Posterior fossa dural plate:

The posterior fossa dural plate is a large bony plate demarcated superiorly by the superior petrosal sinus, laterally and inferiorly by the sigmoid sinus, and medially by the posterior semicircular canal. This posterior fossa dural plate forms the posterior wall of the antrum and the posterior wall of the petrosal part of the mastoid process. Most of the posterior fossa dura is included in Trautmann's triangle, an imaginary triangle bounded by the tegmen mastoidei, superior petrosal sinus, sigmoid sinus, and the bony labyrinth. Inferior to the Donaldson's line is seen the endolymphatic sac resembling a reflection of posterior fossa dura. Donaldson's line is an imaginary line drawn perpendicular to the long axis of the lateral canal bisecting the posterior canal up to the sigmoid sinus.

The tip of the mastoid process in well pneumatized ears usually contains large air cells. At the same depth of the facial nerve there is a ridge known as the digastric ridge. On the external surface of the mastoid process, a corresponding depression occurs. This is known as the digastric fossa relating to the attachment of the digastric muscle.

Facial recess:

The facial recess lies immediately lateral to the facial nerve at the external genu of the nerve. It is usually a collection of small air cells continuing from the antrum towards the facial sinus, but there is no communication between these cells and the tympanic cavity. The facial recess is a surgical term. It is a triangle formed by the facial nerve medially, the chorda tympani laterally, and a bony buttress at the incudal fossa superiorly. Middle ear can be accessed by this route and this procedure is known as the posterior tympanotomy.

The primary aim of a posterior tympanotomy is to create a large facial recess during the canal wall up mastoidectomy in order to provide a new route for air flow from the middle ear into the antrum. However the facial recess is postoperatively often relatively closed off by fibrous tissue of adhesions. The other aim is to eradicate disease from the posterior tympanum, especially from the facial sinus and the lateral tympanic sinus, which are located directly lateral to the descending facial canal, it is also useful in facial nerve decompression and in cochlear implantation procedures.

The attic:

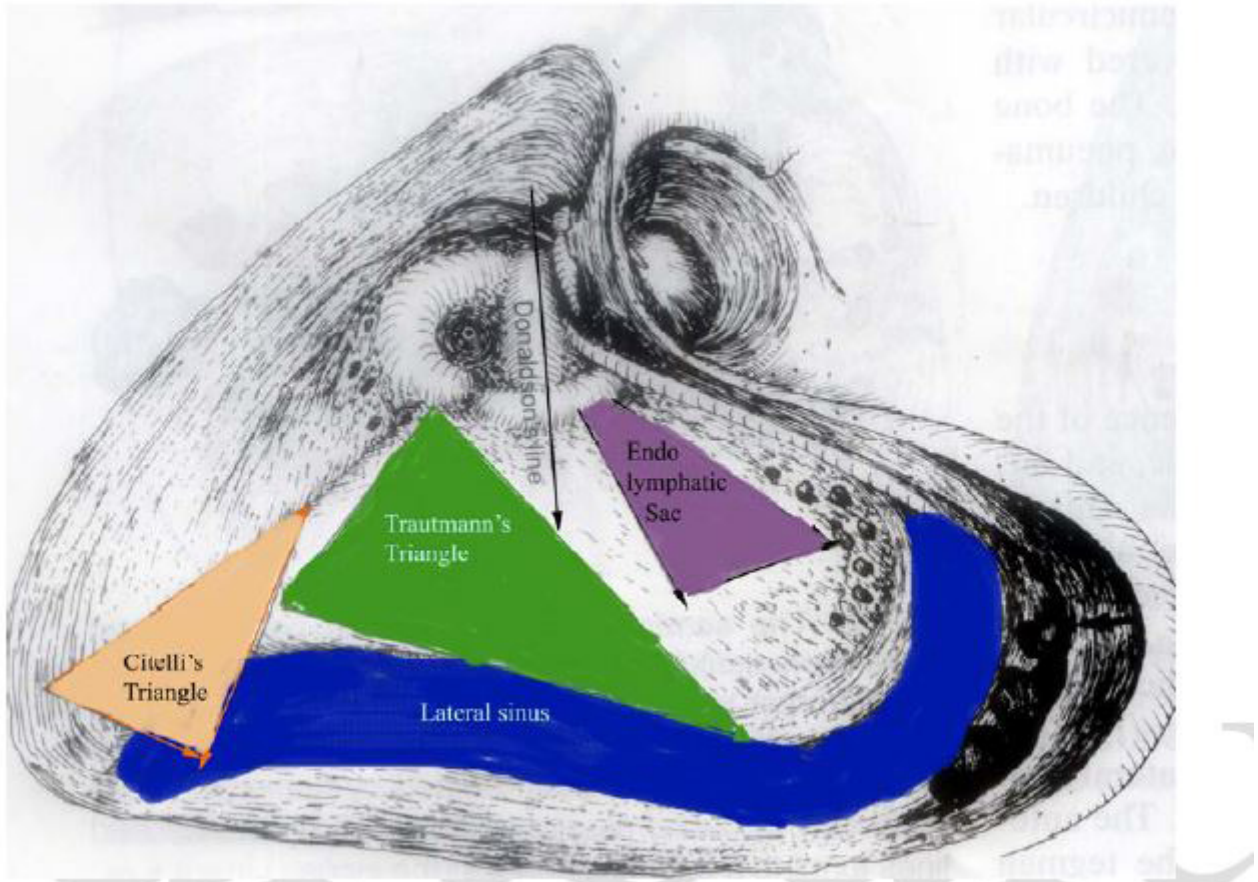
The attic or epitympanum is an important structure. It provides communication between the antrum and the rest of the tympanic cavity. Surgically it is difficult to reach, especially when a low dura is encountered, as is often the case in cholesteatoma surgery. The high frequency of postoperative attic retraction and recurrent cholesteatoma indicates that ventilation of the attic is difficult to maintain after surgery.
The superior border of the attic is the tegmen tympani, which is a thin bony wall, at times transparent or even dehiscent. There are often bony trabeculae present that resemble air cells. These cells connect with periantral cells posteriorly and perilabyrinthine cells medially, as well as with the cells of the meatal wall laterally.

The attic is divided into medial and lateral parts. The border between the two parts is formed by the head of the malleus and the body of the incus, the medial attic being larger. The distance between the prominence of the lateral canal and the body of incus is 1.7 mm, and slightly less at the level of the malleus head. The medial attic communicates with the mesotympanum through an opening - the isthmus tympani – bounded by the lateral semicircular canal and the prominence of facial nerve medially and the ossciles malleus and incus laterally.

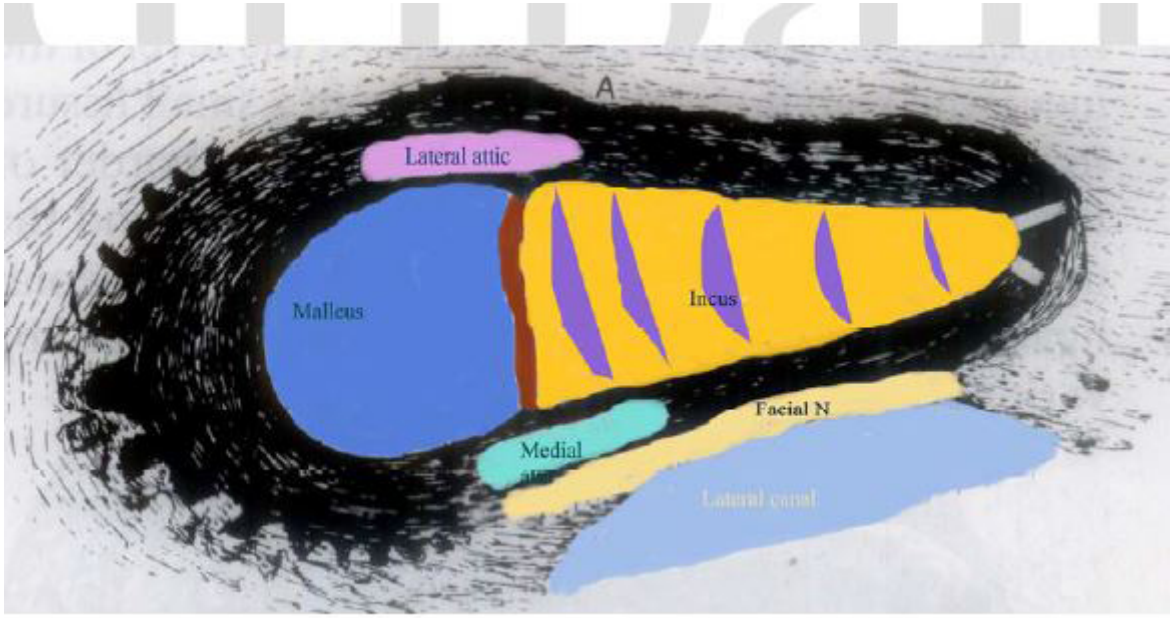
The isthmus tympani is a narrow elongated space, and is divided by the long process of incus into an anterior isthmus , with the tendon of the tensor tympani as its anterior border, and the posterior isthmus, bounded by the posterior wall of cavum tympani, the pyramidal eminence, and the buttress at the fossa incudis. The terms isthmus tympani posticus and isthmus tympani anticus are also used. Through the tympanic isthmus, and especially through the posterior isthmus, sinus cholesteatomas can spread to the attic. The tympanic isthmus is extremely important for attic ventilation.

The lateral attic is located between the outer attic wall laterally (the scutum) and the malleus head and incus body medially. Because of the outward projection of the ossicles, the lateral attic is smaller than the medial one. The outer attic wall (scutum) runs obliquely from the tegmen tympani to the upper edge of bony annulus, making the lateral attic even smaller. The communication between the lateral attic and the mesotympanum varies considerably. At the level of the malleus and the anterior to the malleus, it is virtually non existent. At the level of incus, the communication varies from a small fissure to a small, oval shaped cavity. The lower border of the communication is the chorda tympani, and the upper border is the annulus and the lateral malleolar ligament. The connection is often closed by various small mucosal folds or adhesions.

The anterior wall of the attic is narrow, and the malleus head is in very close proximity to the bony spicules of the tegmen tympani. The fact that this is a common site for a bony bridge to from between the spicules and the anterior surface of the malleus head is understandable and may be caused by simple mechanical irritation in that area or due to inflammation. But the most common cause of bony fixation is previous surgery in that area.



Completed extended mastoidectomy with intact canal wall, entire middle fossa dural plate seen, the sigmoid sinus, the three semicircular canals, and the three triangles clearly demonstrated.



Attic seen from above- Divided into medial and lateral attic

It is hence mandatory to avoid surgical disturbance to attic in simple mastoidectomy. If surgery in the attic region cannot be avoided, it is best to remove all bony spicules and to create a large space between the attic wall and the ossicles, in order to avoid post op bony fixation.

The epitympanic sinus:

Is a separate cavity of varying shape and size situated anterior to the attic, the two cavities being separated by a bony crest coming from the tegmen tympani. The crest is called the anterior attic plate or the cog. The superior wall of the epitympanic sinus is the tegmen tympani, and in some cases the anterior wall also because of the angulation of the tegmen towards the eustachean tube. The medial wall of the sinus is the bone covering the facial nerve at the geniculate ganglion. The lateral wall is formed by the tympanic ring. The inferior border is either the tensor tympani fold or the prominence of the canal of the tensor tympani muscle.

The shape of the epitympanic sinus varies. It can be divided into three main types.

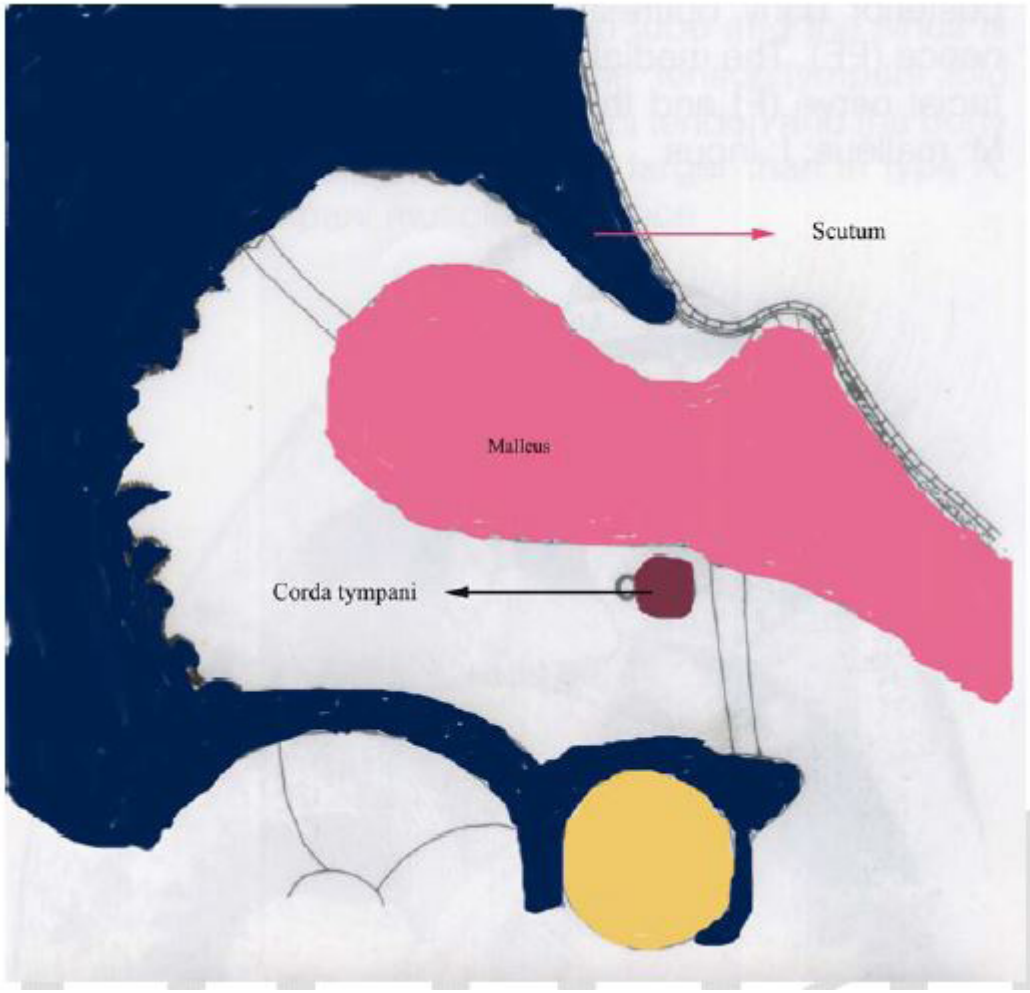
Type A: is a relatively deep sinus anterior to the bony prominence (cog). The sinus is entirely enclosed by bone, except for a narrow opening below the cog. The tensor tympani fold is attached to a relatively thick bony plate anteriorly separating the sinus from the eustachean tube. The tubal recess, also known as supratubal recess, is poorly developed. Found in 38% of cases.

Type B: is found in 40% of cases. The bony plate around the tube is poorly developed, and is connected with a long tensor tympani fold. The tubal recess is larger, but the epitympanic sinus is smaller.

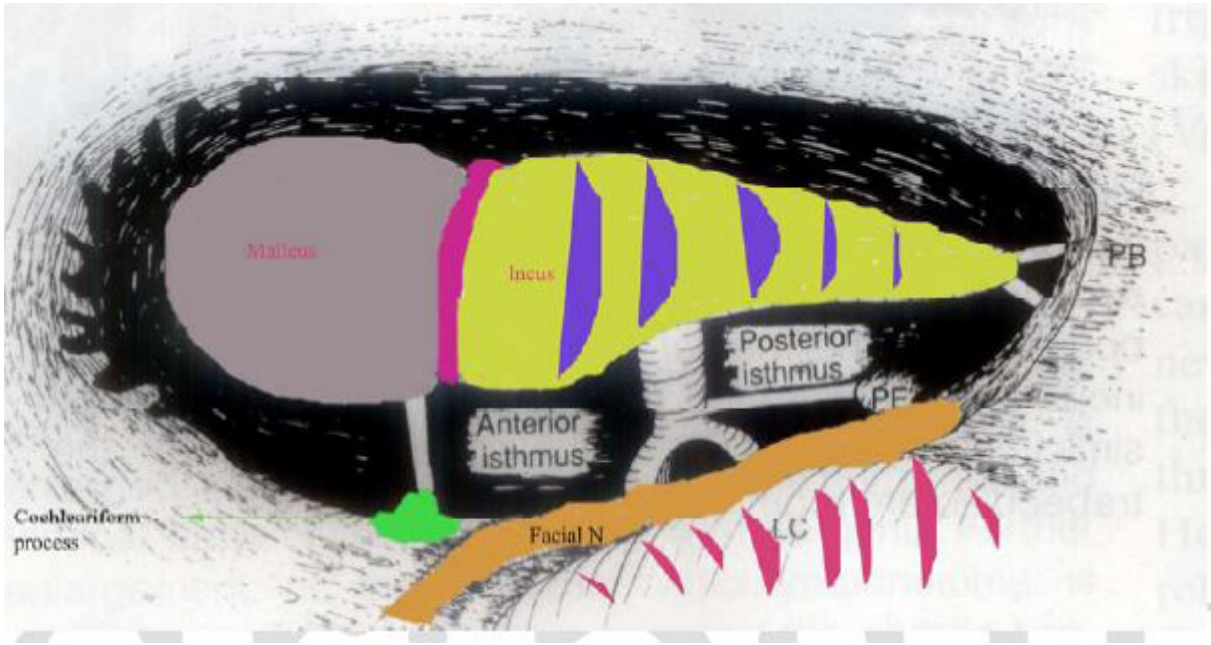
Type C: There are no well defined anterior and inferior boundaries. The sinus slopes down to the eustachean tube and creates a large tubal recess. Seen in 18% of cases.

In the surgical management of middle ear disease, the structural variation of the epitympanic sinus must be kept in mind. Adequate ventilation from the eustachean tube to the epitympanum can be provided by the removal of the cog and the tensor tympani fold in the type c pattern. In such circumstances, damage to the facial nerve and especially the geniculate ganglion is possible. The facial nerve on emerging at the cochleariform process runs anterior to the lateral and superior semicircular canals toward the geniculate ganglion. The nerve can form the medial wall of the epitympanic sinus or tympanic recess, and can be damaged in clearing disease from the attic in a canal wall down mastoidectomy. In both types A and B, the bony plate to which the tensor tympani fold attaches should be removed. Removal should be accomplished without division of the anterior malleolar fold or chorda tympani. Drilling of the cog should be performed medial to the chorda. Cholesteatoma in the epitympanic sinus may not be recognized, and this can lead to recurrences or even spread of the process towards the tip of the pyramid. Intact canal wall procedures provide poor access to the epitympanic sinus and the anterior attic.

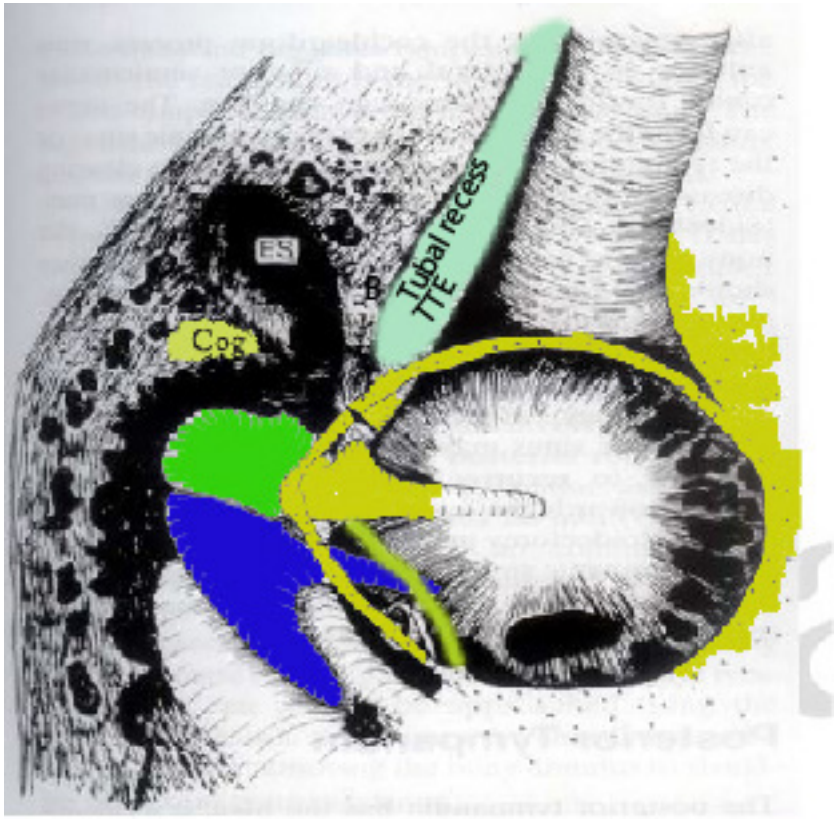
Posterior Tympanum:
The posterior tympanum has the highest incidence of middle ear pathology, especially retractions and cholesteatomas.
The anatomy of the posterior tympanum can be described a) through the ear canal, or b) through the facial recess.
Posterior tympanum through the ear canal:
The posterior tympanum can be visualized through the ear canal using endaural approach. It can be visualized when the bony annulus is drilled away and the patient's head is tilted backward. In the posterior tympanum there are four sinuses which become visible only when the annulus is partially drilled out and the head tilted backward. Two are located suprapyramidally, i.e. superior to the pyramidal eminence – the facial sinus and the posterior tympanic sinus. Two are infra pyramidal – the lateral tympanic sinus and the sinus tympani.



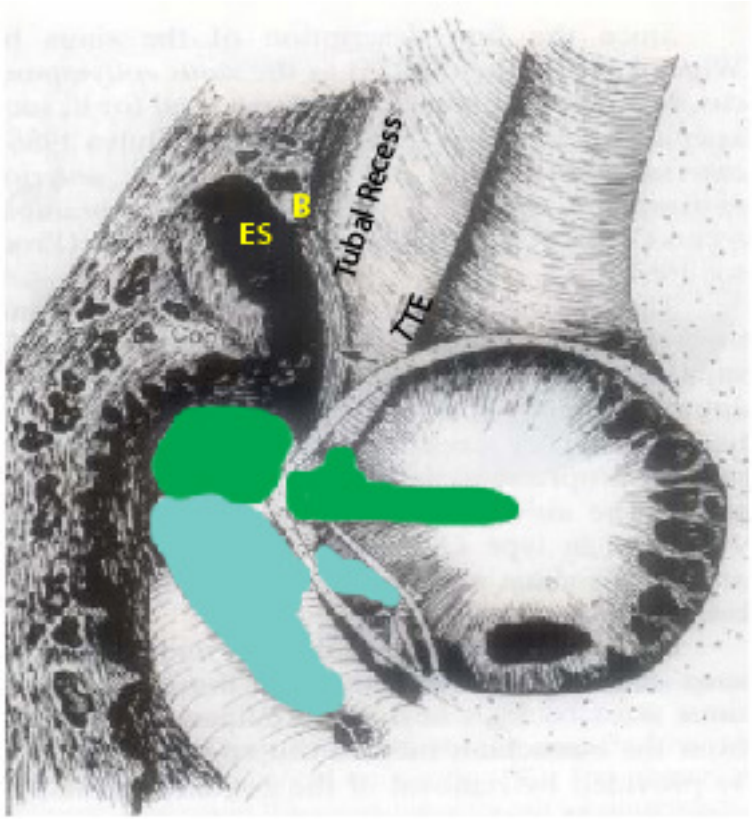
Attic seen from behind at the level of the malleus showing the superior malleolar ligament superiorly, medial malleolar ligament medially. The lateral attic is the space between the lateral attic wall and the bony annulus and the malleolar head. Medial attic is between the malleus and the lateral canal and is larger



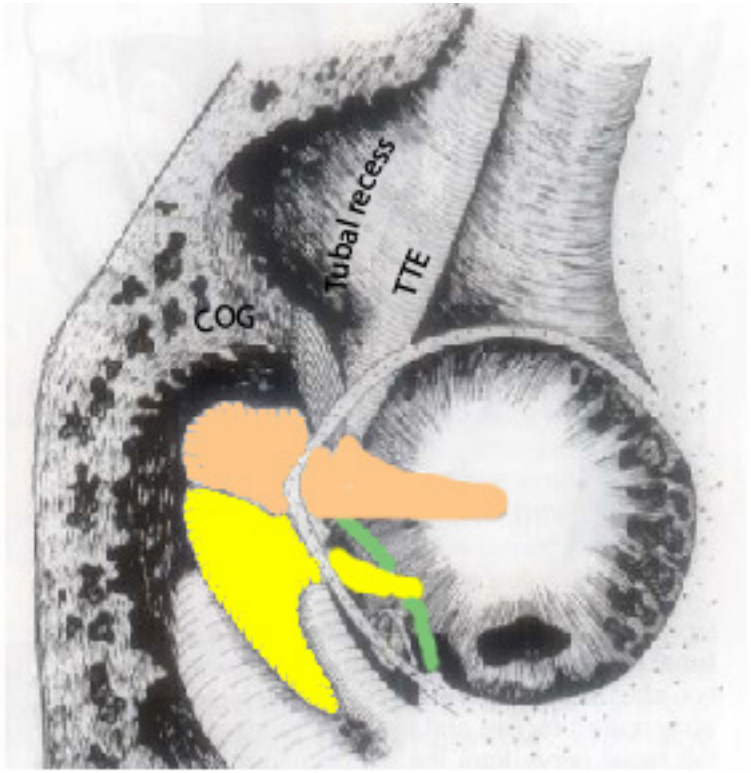
Lateral canal is seen (LC) anterior tympanic isthmus lies between the tendon of tensor tympani muscle with the cochleariform process, the long process of incus and the stapedial arch. The posterior tympanic isthmus lies between the long process of incus, the posterior bony buttress (PB), and the pyramidal eminence (PE), the medial borders of the two isthmi are the facial nerve and the lateral canal (LC).



Type A epitympanic sinus. The sinus (ES) is deep and totally surrounded by bone except for a narrow opening below the cog. The bony plate (B) separating the eustachean tube from the epitympanic sinus is long and prominent; the tensor tympani fold (arrow) is short. The tubal recess is poorly developed or not present.



Type B epitympanic sinus. The bony plate (B) between the eustachean tube and the sinus is poorly developed and short. The tensor tympani fold (arrow) between the tensor tympani tendon and the bony plate is long. The tubal recess is larger than in type A. TTE IS Tensor tympani eminence.



Type C epitympanic sinus

In relation to the facial nerve in the posterior tympanum, two sinuses are lateral and external to the facial nerve – the facial sinus and the lateral tympanic sinus, whereas the posterior tympanic sinus and the sinus tympani are medial to the nerve. The facial sinus is thus a superolateral sinus; the lateral tympanic sinus is the inferolateral sinus. The posterior tympanic sinus is the superomedial sinus, and the sinus tympani is the inferomedial sinus.

The lateral tympanic sinus: is the most lateral sinus. It lies between the three eminences of the styloid complex. The lateral tympanic sinus is hidden just medial to the bony annulus, but is often involved in middle ear diseases especially in the posterior retraction of the atrophic drum. There is no communication between the lateral tympanic sinus and the attic or antrum.
By carefully drilling the bony posterior meatal wall and annulus around the tympanic chorda, the lateral tympanic sinus can be accessed.

The facial sinus – is also a lateral sinus. It is situated at the genu of the facial nerve canal, medial to the bony annulus and chordal eminence, but superior to the chordal ridge and the pyramidal eminence. There is no connection between the facial sinus and the air cells of the attic or mastoid process. When approaching from behind through the facial recess, the facial sinus is entered directly. Failure to clear this area is the most common cause of residual cholesteatoma.

The posterior tympanic sinus is a medial sinus. It lies medial to the facial nerve and the pyramidal eminence, but superior to the ponticulus – a bridge located between the posterior tympanic wall and the promontory. In fact it is the ponticulus that divides the tympanic sinus into the smaller posterior tympanic sinus and the larger inferior sinus, called the sinus tympani.

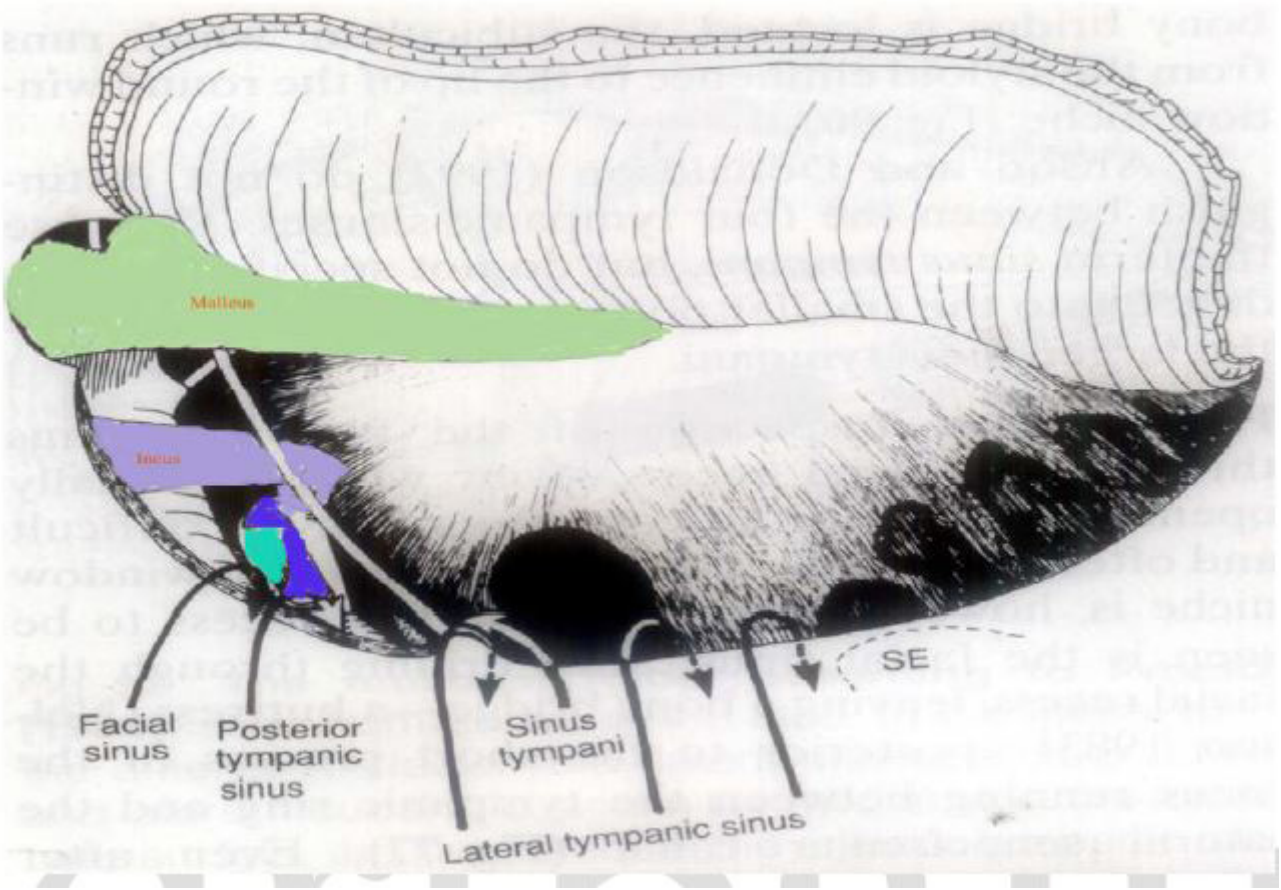
The sinus tympani is the largest sinus. It is a medial and infrapyramidal sinus. It lies deep to the descending part of the facial nerve, inferior to the ponticulus and the pyramidal eminence. At the inferior border of the tympanic sinus, a small bony bridge is located, the subiculum, which runs from the styloid eminence to the lip of the round window niche.

Facial recess:

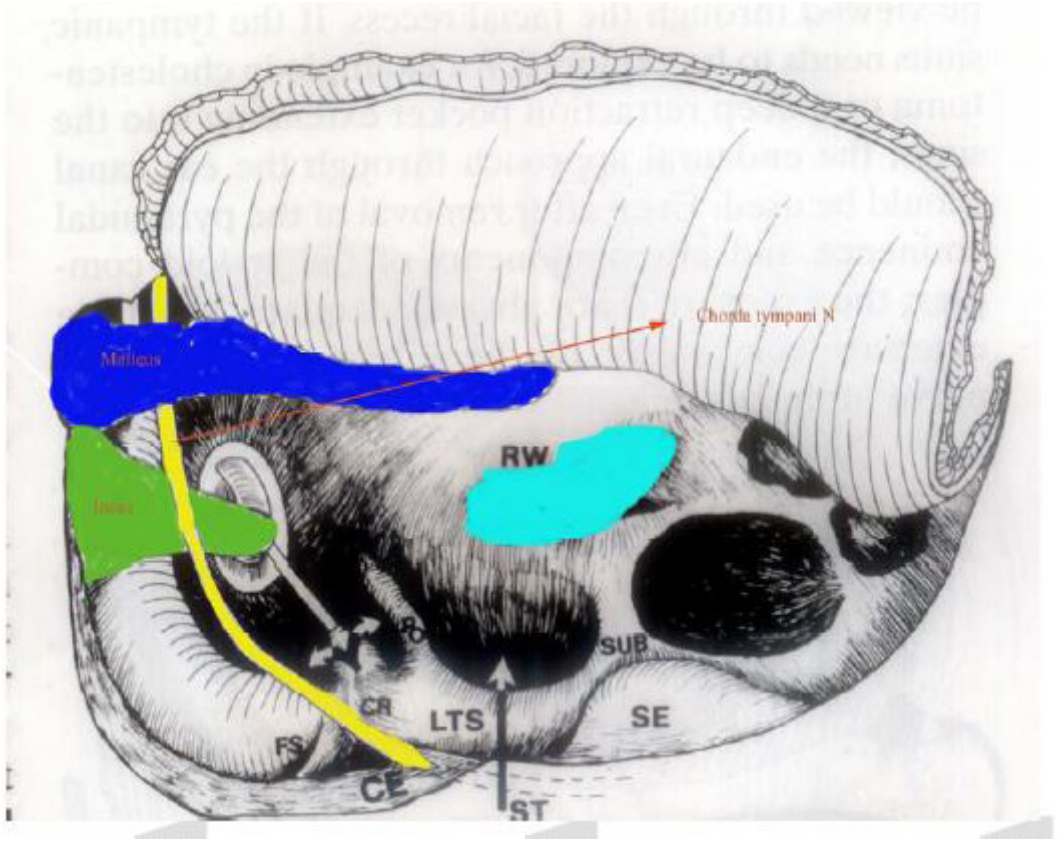
Inspection of the tympanic sinus through the facial recess, even when maximally opened in posterior atticotympanotomy, is difficult. The view to the round window is excellent. The first recess to be seen is the facial sinus, after drilling through the facial recess, leaving a bony ridge posterior to the short process of incus running between the tympanic ring and the lateral canal. If the tympanic sinus needs to be explored then an endaural approach is preferable.

Tympanic diaphragm:
Is a term first coined by Proctor in 1964. It defines the obstacles within the tympanic isthmus and the attic. These obstacles are the tympanic folds and ligaments running between the surrounding bony structures and the incus body and malleus head. It is a pretty crowded space. The tympanic diaphragm divides the attic from the mesotympanum. The tympanic diaphragm is a common site for impairment of ventilation to the antrum. Wullstein goes to the extent of describing this region as the second bottle neck of air flow, the first being the eustachean tube.

Mucosal folds:
These folds divide the attic space into various compartments. They are located both in the lateral and medial attic.



Sinuses of posterior tympanum – SE styloid eminence



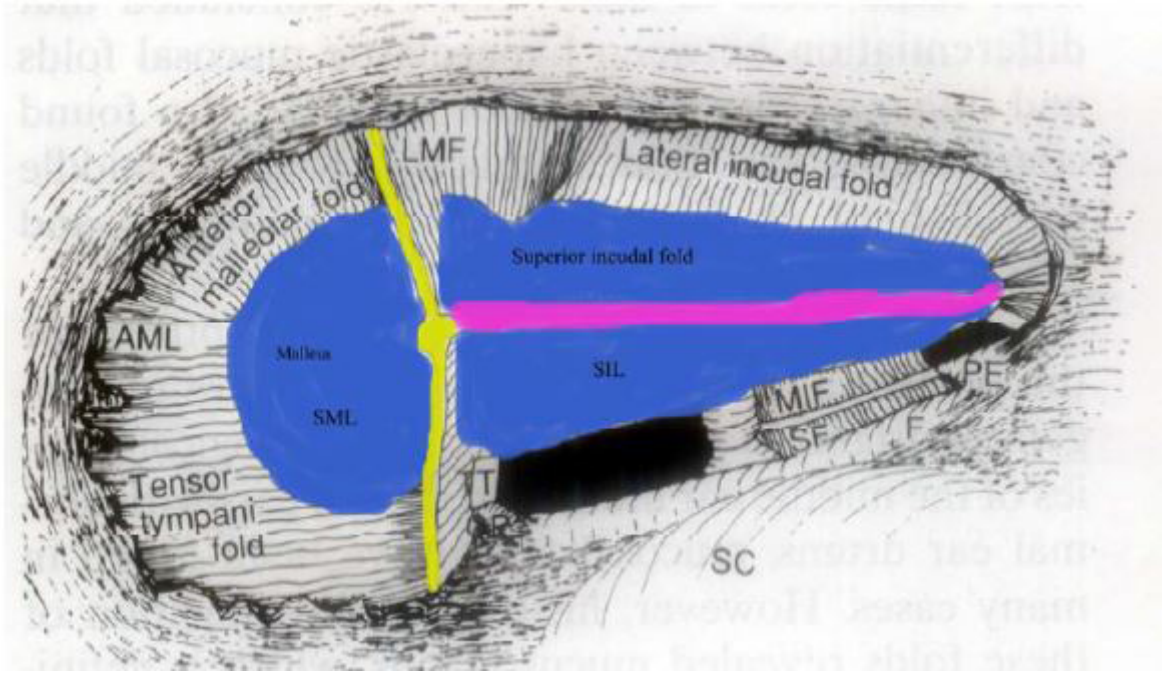
Postero superior bony annulus drilled completely – FS Facial sinus, RW round window, PE pyramidal eminence, PO ponticulus, CE chordal eminence, ST sinus tympani, SE styloid eminence, SUB subiculum, CR chordal ridge.

The lateral incudal fold connects the lateral attic wall and the body and the body of the incus. It extends posteriorly to the posterior incudal ligament.
The anterior malleolar fold of von Troltsch is located between the anterior surface of the malleus head, and the antero lateral bony wall of the attic, and the anterior malleolar ligament.
The superior malleolar fold extends between the superior surface of the malleus head and the superior attic wall, and in the same plane as the superior malleolar ligament.

The superior incudal fold extends, like the superior incudal ligament, between the superior aspect of the incus body and the superior attic wall.
The medial incudal fold is located between the long process of the incus and the tendon of the stapedial muscle, as far as the pyramidal eminence.
The lateral malleolar fold goes from the neck of the malleus up to the scutum forming the superior border of Prussak's space.
The interossicular fold extends between the malleus handle and the long process of incus.
The anterior malleolar ligament extends from the long process of the malleus toward the anterior attic wall.
The tensor tympani fold occupies the window between the tensor tympani tendon, the anterior bony plate of the attic wall, the tensor tympani eminence, and the anterior malleolar ligament.

The folds most commonly seen are 1. The tensor tympani fold, 2. The lateral incudal fold, 3. The medial incudal fold between the crura of the stapes and the incus 4. The lateral malleolar fold, 5. the stapedial fold between the posterior crus of the stapes and the posterior tympanic wall , following the stapedial tendon and the pyramidal eminence and 6. The obturator fold between the stapedial crura. The mucosal folds are very thin and carry blood vessels to the ossicles.

According to Proctor ventilation of the attic never goes through the anterior attic, but only through the tympanic isthmus, i.e. posterior to the tensor tympani tendon. The anterior attic space, according to proctor is blocked by the tensor tympani mucosal fold.
Recent studies show that these folds may be sequelae of previous infections or inflammation of the middle ear. Hence they can be considered as post inflammatory adhesions. Histology of these folds reveals mucosal glands which is a definite proof of inflammatory origin.



The mucosal folds forming the tympanic diaphragm LMF lateral malleolar fold, SIL superior incudal ligament, TT Tendon of the tensor tympani, SML superior malleolar ligament,
F facial nerve, AML anterior malleolar ligament.

Microbiology of middle ear

Introduction:

Middle ear communicates with the atmosphere via the eustachean tube. Ideally speaking middle ear is not accessible to microbes. Microbes to reach the middle ear should travel up via the eustachean tube or via a pre-existing ear drum perforation.

The outer ear is lined by skin and is in contact with the environment. Both the auricle and external auditory canal house a number of microbes even under healthy conditions. Since the external auditory canal is exposed to oxygen containing atmospheric air, the majority of microbes in the pinna and external auditory canal belong to aerobic species. The skin lining of external canal contains predominantly gram positive bacteria with gram negative bacteria less in number.

The main gram positive bacteria found in the skin of external auditory canal are:

1. Staphylococci

2. Coryneforms

3. Streptococci

4. Enterococci

5. Micrococci

Among the gram positive bacteria the predominant ones include:

Staph aureus
Staph capitis
Staph epidermidis
Micrococcus luteus

Gram negative bacteria inhabiting the external auditory canal skin include:

Ecoli

Pseudomonas aeruginosa

Fungal organism predominate in cerumen when compared to that of skin lining of the external auditory canal.

Bacteriological studies have proved that alpha hemolytic streptococci inhabit the middle ear cavity. This organism has a tendency to crowd out other organism. Lower incidence of alpha hemolytic streptococci has been observed in children with recurrent middle ear infections.

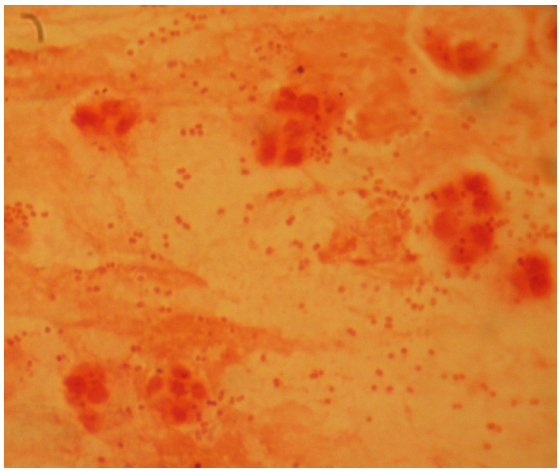
Normal middle ear is not accessible to external environment directly. It is connected to atmospheric air via the eustachean tube. In case of perforated ear drum bacteria from external ear and other contaminants could reach the middle ear via the perforation.

Alloiococcus otitis and Turicella otitis:
These organism are normal commensals found in the external auditory canal don't cause infections there. When they reach the middle ear cavity via the perforated ear drum can prolong the already existing middle ear infection.

Common microbes involved in acute otitis media include:

Streptococcus pneumonia
Haemophilus influenza
Moraxella catarrhalis
Streptococcus pyogenes

These common infecting organism are associated with upper respiratory tract infections in humans. They reach the middle ear via the eustachean tube.

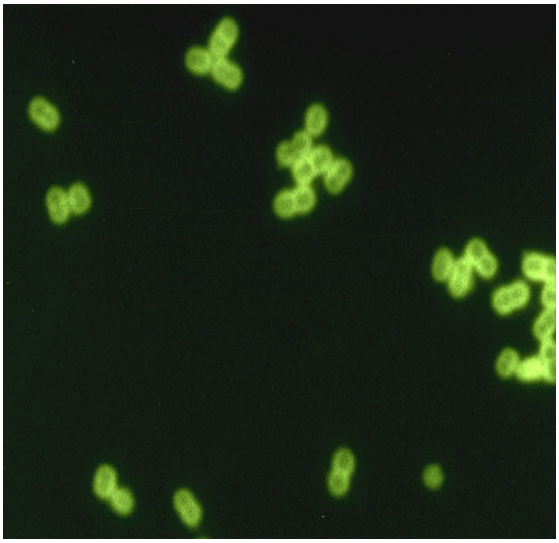


By Bobjgalindo - Own work, GFDL, https://commons.wikimedia.org/w/index.php?curid=7777288
H influenza in gram stain appearing as gram negative cocco bacilli

Haemophillus influenza is a gram negative cocco bacillary facultative anaerobe. Since this was isolated from sputum of patients during influenza epidemic it was erroneously postulated to be the cause for influenza and hence the name. Most strains of H influenza are opportunistic pathogens. Naturally occurring infections attributable to this organism is common in infants.
These organism are capable of secreting beta lactamases which could deactivate beta lactum producing antibiotics thereby adding to drug resistance.

Streptococcus pneumonia:

Also known as pneumococcus it is a gram positive and alpha hemolytic facultative anaerobic member of genus streptococcus. These are usually found in pairs and hence are also known as diplococci.



Streptococci Pneumoniae By Bobjgalindo - Own work, GFDL, https://commons.wikimedia.org/w/index.php?curid=7777288

Interaction between H influenza and streptococci pneumonia:

It is pertinent to note that both these organisms are found in the upper respiratory tract of humans. Studies reveal that there is competition between these two organism in which Streptococci pneumonia always manage to overpower H influenza by attacking it with hydrogen peroxide and stripping off the surface molecules that help H influenza to survive. This reaction stimulates immune system to attack streptococci pneumonia organism also.

Moraxella Catarrhalis:

This is a non motile gram negative aerobic organism causing infections of middle ear. These organism are resistant to penicillin group of antibiotics.

Microbiology in CSOM:

In the case of chronic inflammation of middle ear mucosa cultures yielded monomicrobical growth in nearly 60% of cases. Polymicrobical growth was observed in 30% of cases. No bacterial growth was demonstrable in 10% of cases.

Predominant organism isolated from ear discharge of patients with CSOM was staphylococcus aureus, next comes pseudomonas. Pseudomonas is not considered to be a normal commensals in the upper respiratory tract and hence cannot make an entry into middle ear via eustachean tube. Presence of pseudomonas organism in the middle ear cavity indicate that it could have entered only via perforation in the ear drum.

Coliforms like klebsiella and E coli have also been isolated from discharge in CSOM.

Anaerobes have been isolated only from ears with cholesteatoma / granulation tissue. Prominent anaerobes isolated include:

Clostridium

Peptococcus

Peptostreptococcus

Fungus have also been isolated from discharge of chronic ear disease. Fungus have been known to thrive in pus. Aspergillus and candida are the two common fungi isolated from chronic middle ear infections.

Viral infections have been implicated in acute middle ear infections especially among children. Invasion of middle ear by viruses from nasopharynx via eustachean tube is a real possibility. Most common virus isolated in acute middle ear infections is the respiratory syncytial virus. This virus has a strong ability to invade the middle ear cavity.

Some of the common viruses known to invade the middle ear cavity include:

Respiratory syncytial virus
Parainfluenza virus
Influenza virus
Adenovirus
Enterovirus

Role of Biofilms in chronic middle ear disease:

Biofilm formation has been demonstrated in the middle ear mucosa of patients with chronic otitis media. It would be more appropriate to state that chronic otitis media is a biofilm related disease. Biofilms are organized and complex bacterial communities in which bacteria communicate with each other thereby gaining tremendous advantages. Inside this biofilms bacteria can diffuse nutrients, gain resistance to antimicrobial agents and can also be immune to host defence mechanisms.

Even though bacteria are considered to be individual living organism they are known to organize together into complex communities and can attach themselves to mucosal surfaces. This is known as biofilm formation. This is a classic strategy of survival of the organism. Inside biofilms bacteria are embedded in a slime like extracellular matrix composed of proteins, polysaccharides and nucleic acids. These substances that form the matrix of biofilms are also known as extracellular polymeric substances. This could even be considered as bacterial community. In this community bacteria don't make random groups but arrange themselves into complex three dimensional structures.

Within the biofilm bacteria communicate with each other using signalling molecules and modulating gene expression. Nutrient delivery and waste disposal become highly organized within the biofilm. Being within the biofilm protects the organism from environmental changes like:

1. Temperature changes

2. Changes in the moisture levels

3. Changes in pH

4. Within the biofilms the metabolic activity of bacteria is reduced

5. Can escape host immune reactions

6. Can escape phagocytosis

7. Can gain resistance to common antibiotics

Biofilm as a survival mechanism of bacteria:

Biofilm formation is an old survival strategy employed by prokaryotic organism. The credit for being the first to observe biofilm should go to Antoni van Leeuwenhock who observed "animalcule" on dental surfaces. The term biofilm was coined in 1973 by Characklis during his classic studies involving microbes populating the sewage system. Biofilms can colonize any humid surface. Classically they can be observed in teeth, and as slippery slime seen over river stones, implanted medical devices and chronic infected tissue. These biofilms are not easily detached by rinsing. They are tightly adherent to the underlying surface.
Biofilms provide the ideal environment for exchange for exchange of extra chromosomal DNA (plasmids). conjugation or mechanical transfer of plasmid occur at a greater rate within bacterial cells inside Biofilm than otherwise.

The term biofilm is a misnomer because they are not a continuous monolayer of surface deposit. Biofilms are composed of heterogeneous containing components like water, polysaccharides and other macromolecules. Microcolonies of bacteria encased within the matrix are separated from each other by water channels which allow diffusion of nutrients, oxygen and antimicrobial agents. The composition of the matrix will change as the equilibrium between the members of the bacterial community is established.

Structures involved in the formation of biofilm:

Various structures like flagella, fimbriae, outer membranous proteins, curli (a protenaceous surface structure) and polysaccharides. Flagella plays an important role in the early stages of bacterial attachment to the substrate by overcoming the repulsive forces the substratum can demonstrate. Fimbriae are known to contribute to the cell surface hydrophobicity. These structures are known to contain high concentrations of hydrophobic aminoacid residues. These fimbriae helps in overcoming the initial electrostatic repulsive forces displayed by the substrate. Genes responsible for encoding mannose sensitive Type I and Type IV pili are required for host cell colonization and biofilm formation to occur.

Presence of outer membranous protein AG43 in E coli facilitates cell surface and cell to cell contacts. These proteins are generated in large quantities when E coli are cultured in a minimal medium. These proteins are not generated in such large amounts when the same organisms is cultured in a rich medium indicating that these proteins play a vital role in helping the organism to survive under hostile conditions.

The extracellular polymeric substances (EPS) seen in biofilms is distinct and is chemically and physically different from that of bacterial capsule which is supposed to be primarily composed of polysaccharides. The extracellular polymeric substances are highly hydrated because they can incorporate large amounts of water by hydrogen bonding. Different organisms produce differing amounts of EPS and the amount of EPS would vary with the age of the biofilm, showing an increase as the age of the biofilm increases. Chemically EPS secreted by E coli is colonic acid and that secreted by Pseudomonas is alginate.

Factors affecting Biofilm formation:

The ability to construct and maintain biofilm depends on the production of extracellular matrix components among which extracellular polysaccharides are the most important.

Extracellular polysaccharides secreted by microbes vary in composition, physical and chemical properties. These extracellular polysaccharides are categorized into aggregative, protective and architectural. These categories are self explanatory.

Aggregative polysaccharides:

The formation of biofilms occurs in various stages. These stages include:

Initial attachment
Microcolony formation
Macrocolony formation
Detachment

Aggregative polysaccharides play an important role in each of these steps. They aid in adhesion to surfaces, formation of complex structures by promoting microbial interactions. They can also facilitate detachment / dissolution of biofilm.

Polysaccharide intercellular adhesion is one of the most important property of polysaccharides secreted by Staph aureus and Staph epidermidis.

Predation:

Bacteria within the biofilm may be vulnerable to predators like free living protozoan, bacteriophages and polymorphs. Common free living protozoan that predates in the biofilm is Hartmanella vermiformis. Acanthamoeba is another predator belonging to protozoan family

Competition:

Competition between microbes within biofilm is common. Studies reveal that in biofilms containing K pneumonia and p aeruginosa both these organisms tend to coexist even though P aeruginosa growth rate is slower than that of K pneumonia.

Dispersal:

Biofilm cells may get dispersed due to any of the following causes:

1. Shedding of daughter cells from actively dividing cells

2. Detachment as a result of nutrient levels. Low nutrient levels cause detachment

3. Detachment as a result of quorum sensing

4. Shearing of biofilm aggregates because of flow effects

Surface hydrophobicity causes the newly divided daughter cells of E coli / P aeruginosa to be spontaneously be dispersed. Hydrophobicity gradually reduced on prolonged incubation hence promoting formation of biofilm aggregates.

Studies reveal that alginate is the major component of extracellular polysaccharides secreted by P aeruginosa. Increasing amounts of alginate promotes biofilm formation. An enzyme known as alginate lyase is also secreted by P aeruginosa. This enzyme causes destruction of alginase. Expression of this enzyme in P aeruginosa causes detachment of biofilm.

Physical forces involved in detachment of biofilm:

Physical forces like erosion, sloughing and abrasion are involved in detachment of biofilm. Sloughing causes massive and rapid detachment while abrasion causes detachment of biofilm due to collision of particles from the bulk fluid within the biofilm medium.

The rate of biofilm erosion increases with an increase in the thickness and the fluid shear at the biofilm - bulk liquid interface.

Quorum sensing:

Cell to cell signalling has been demonstrated to play a role in cell attachment and cell detachment from biofilms.

Significance of cellulose in biofilm:

Cellulose happens to be the most abundant of the polysaccharides in nature. It should be stated that the structure of cellulose remained unaltered across the species there by showing a tendency to be stationary even during the entire evolution process. Bacterial cellulose is virtually indistinguishable from fungal cellulose. The cellulose fibrils have a tensile strength equivalent to that of steel. Cellulose provides both structural support and protection to bacteria inside biofilms.

Role of Eustachean tube in Middle ear disease

Introduction:

Eustachean tube is otherwise known as pharyngotympanic tube, middle ear ventilation tube. It is bony cartilagenous in nature. It connects the middle ear with the nasopharynx. In adults it lies at an angle of 45 degrees to the horizontal plane. In infants this inclination is about 10 degrees. In adults its length is 38mm. For descriptive purposes it can be divided into posterior 1/3 which is osseus in nature and anterior 2/3 which is cartilagenous in nature.

This eustachean tube is shorter straighter and wider in infants predisposing middle ear infections through this tube. The osseus portion of the eustachean tube also known as protympanum lies completely within the petrous portion of the temporal bone. The lumen of the osseus portion of the eustachean tube is triangular and is open always in contrast to the fibrocartilageous portion which is kept closed at rest, and opens during swallowing, or during a valsalva manuver. The osseous and cartilagenous portion of the eustachean tube meet at an irregular bony portion and form an angle of about 160 degrees with each other. The cartilagenous tube courses anteromedially and inferiorly, angled between 30 and 40 degrees. The cartilagenous portion of the tube is not completely surrounded by cartilage, but is deficient inferolaterally where it is covered by a membrane. The cartilage is crook shaped covering the medial, lateral and superior walls of the cartilagenous portion of the tube. The tubal lumen is shaped like two cones joined at their apices. The junction of the cones is the narrowest portion of the lumen and is known as the isthumus, and is usually situated at the junction of the cartilagenous and bony portion of the tube.

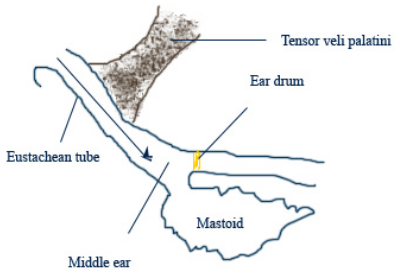
The cartilagenous portion of the eustachean tube doesnot follow a straight course in the adult but extends along a curve from the junction of the osseous and cartilagenous portions to the medial pterygoid plate, approximating the skull base during most of its course. The eustachean tube crosses the superior border of the superior constrictor muscle to enter the nasopharynx. The medial cartilagenous portion of the tube presses against the pharyngeal wall to form a prominent fold, the torus tubaris. The torus is the site of origin of the salpingopalatine muscle and is the point of origin of the salpingopharyngeal muscle.

The mucosal lining of the eustachean tube is continuous with that of the nasopharynx and middle ear (ciliated columnar epithelium). Certain differences in the mucosal lining is evident, mucous glands predominate at the nasopharyngeal orifice, and this gradually changes into a mixture of goblet cells at the tympanum.

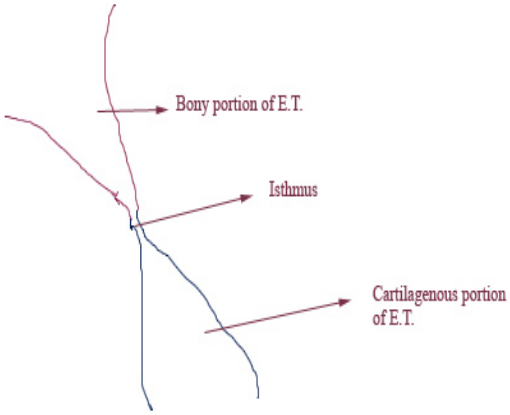
Muscles associated with eustachean tube: The muscles associated with the eustachean tube are 4 in number. They are tensor veli palatini, levator veli palatini, salpingopharyngeus, and tensor tympani.

Usually the eustachean tube is closed; it opens during such actions like swallowing, yawning thus equalising the middle ear pressure. Active dilatation of the tube is induced by the tensor veli palatini muscle. Closure of the tube has been attributed to passive reapproximation of tubal walls by extrinsic forces exerted by surrounding elastic fibres.
Blood supply: The eustachean tube is supplied by the ascending palatine artery, pharyngeal branch of internal maxillary artery, the artery of the pterygoid canal, ascending pharyngeal artery, and the middle meningeal artery. The venous drainage is via the pterygoid plexus.

Nerve supply: The pharyngeal orifice of the eustachean tube is supplied by a branch from the otic ganglion, the sphenopalatine nerve, and the pharyngeal plexus. The reminder of the tube receives its sensory supply from the tymapnic plexus and the pharyngeal plexus. The glossopharyngeal nerve has an important role in the innervation of the eustachean tube.

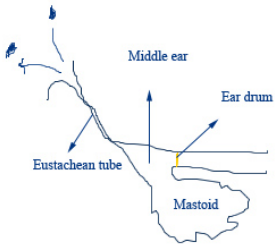


Ventilation function of eustachean tube



Protection: It protects the middle ear cavity from microbes of nasopharynx.

Figure showing various portions of Eustachean tube

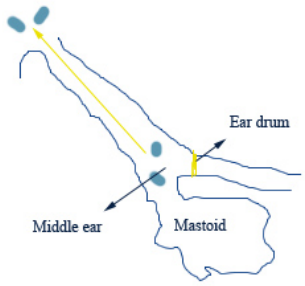


Protective function

Functions of the eustachean tube:

Ventilation: It ventilates the middle ear cavity via the nasopharyngeal airway.

Drainage: Drains the secretions from the middle ear cavity into the nasopharynx.



Drainage function

Features of Infant eustachean tube:

In infants the eustachean tube is about half as long as in the adults, averaging about 18 mm. The osseous portion is longer than the cartilagenous portion. It is shorter, straighter, and wider than that of adults. The tensor veli palatini muscle is less efficient in infants. The tube is also mostly horizontal in infants. Hence infants are more prone for middle ear infections arising from the eustachean tube.

Tests of Eustachean tube function:

For any middle ear surgical procedure to succeed a normal and functioning eustachean tube is a must.
It is always better to have a clear understanding of the functional status of ET before embarking on tympanoplasty / myringoplasty.

Tests of eustachean tube function:

Pneumatic otoscopy:
A normal appearing ear drum in otoscopy portends a normal eustachean tube function. A retracted ear drum indicates a blocked eustachean tube.
Fluid level seen behind the ear drum indicates secretory otitis media.
A normally moving ear drum on penumatic otoscopy indicates normal eustachean tube function.

Valsalva maneuver:

This maneuver is not popular now. It was first proposed by Antonio Maria Valsalva during 17th century.
This maneuver is performed
by exhaling forcibly exhaling against a closed airway.
This is a very difficult maneuver to perform. To overcome this difficulty a modified
valsalva Maneuver has been proposed. In this maneuver, the patient is made to expire against closed glottis.

Figure showing modified valsalva maneuver being performed.

Toyenbee maneuver: This maneuver is again used to subjectively / objectively test eustachean tube function. The patient
is instructed to swallow while pinching both the nostrils. A normally functioning eustachean tube will cause a popping
sound to occur inside the ear. If otoscopic examination is performed simultaneously the ear drum could be seen moving in and out.

Lowry technique: This is a combination of Valsalva and Toyenbee maneuvers. This technique involves pinching the nose while
attempting to blow and swallow at the same time.

Frenzel Maneuver: The patient is asked to pinch the nose closed. The back of the throat is closed (as it happens when one strains to lift a heavy weight). While performing this procedure the patient attempts to vocalize the letter "k". This forces the tongue backwards causing air to be pumped into the eustachean tube.

Yawning Maneuver: Attempting to open the mouth wide as if one is yawning will open the eustachean tube if it is patent.

Tympanometry:

This test is used to objectively assess eustachean tube function. Measurement of impedence of middle ear by this procedure
will help to assess its function. A normal middle ear pressure is associated with normal eustachean tube function, while a negative
middle ear pressure indicates a blocked eustachean tube.

Imaging:

CT scan / MRI scan of temporal bone will also help in assessing eustachean tube function.

Sonotubometry:

Sonotubometry is also known as acoustic tube endoscopy. This method of investigation tests the patency of eustachean tube by its ability to conduct sounds from the nasal cavity. A small speaker is placed over the nasal cavity.

This speaker is used to generate sounds at the level of nasal cavity. This sound if the eustachean tube is patent gets conducted to the middle ear cavity.

A microphone placed at the level of external canal picks up the sound. Eustachean tube function is one of the difficult functions to test clinically, leave alone objectively.

Eustachean tube block is one of the commonest postulated cause for middle ear inflammatory pathologies. Sonotubometry offers a very easy and versatile way of objectively assessing the function of eustachean tube. This test is also known as acoustic tube endoscopy. Sound usually 8 kHz is generated by a speaker which is placed close to the nasal cavity. This sound will reach the middle ear via the eustachean tube if it is patent. This sound can easily be recorded by placing a microphone in the external auditory canal.

Four common curves of sonotubometry have been identified by recording sound transmitted

at the level of external auditory canal:

1. Spike type (the most common type 60%)

2. Double peak type (17%)

3. Plateau type (17%)

4. Descendant curve (5%)

In patients with perforated ear drum the function of ET could be assessed by instilling ear drops into the affected ear. If the ET is patent

the patient will be able to sense the bitter taste of ear drops in the throat.

During normal circumstances the eustachean tube is in closed position. In order to open it muscles should actively act. In normal physiological circumstances opening of eustachean tube occurs transiently during swallowing. It could be kept open for longer duration during yawning. The closed eustachean tube protects the middle ear cavity from reflux of nasopharyngeal contents and sound. It should be pointed out that eustachean tube does not open with every swallow or yawn but it needs voluntary and involuntary effort for it to happen.

It has been postulated that there exists a unique homeostatic mechanism to regulate the strength of muscle contractions necessary to open up the eustachean tube. This mechanism is dependent on the changes in the middle ear environment like middle ear pressure / middle ear gas mixture. Closure of the eustachean tube is also dependent upon the relaxed soft tissue bulk within the lining of the tubal lumen. In the absence of dilatory action of the muscles, the elasticity of mucosa and submucosal tissues relax and increase in thickness occluding the eusachean tube entry. A pad of fat tissue Ostmann's fat pad also contributes to the closure mechanism of eustachean tube. The portion of the eustachean tube lumen that remains closed at rest and opens up during the dilatory process is known as the valve area. This area measures about 5 mm in length.

Dilatation of eustachean tube lumen is primarily by the activation and contraction of Tensor veli palatini muscle. The levator veli palatni muscle contraction serves to dilate principally the nasopharyngeal orifice of eustachean tube. The bony portion of the eustachean tube remains patentn and unchanged in its diameter throughout this opening and closing phases involving the cartilagenous portion. The process of dilatation involves only the cartilagenous portion and it begins begins at the nasopharyngeal orifice and continues to progress from distal to proximal portion. Whereas the closure of eustachean tube occurs in the reverse direction progressing from the proximal (adjacent to isthumus) to distal (nasopharyngeal orifice) of eustachean tube. The process of eustachean tube closure produces a pumping ation that forces secretions from the middle ear cavity to be expelled from the middle ear into the nasopharynx. This action ensures that low viscosity fluids are expelled from the middle ear cavity. High viscosity fluids are expelled through the mucociliary clearance mechanism.

Normal dilatation process of eustachean tube which takes place during normal swallow demonstrates 4 consisent phases i.e.

1. Elevation of soft palate and medial rotation of the posteromedial wall due to action of levator veli palatini muscle. This causes initial dilatation of the nasopharyngeal orifice of the eustachean tube.

2. Contraction and medial movement of lateral pharyngeal wall causing transient narrowing of the tubal end of nasopharyngeal orifice.

3. Lateral movement of the lateral pharyngeal wall further dilates the nasopharyngeal orifice and the Tensor veli palatini muscle contraction initiates dilatation of the proximal tubal lumen.

4. Maximal contraction of the Tensor veli palatini muscle combined with effacement of the tubal antero lateral wall and dilation of tubal valve distally brings the lumen to its fully dilated state. Usually the valve remains maximally dilated for approximately 50 msec.

Closure of the eustachean tube begins distally with closure of the valve followed by slow relaxation of the Tensor veli palatini muscle. Mucosal closure progressed from distal to proximal direction. The posterior cushion formed by the medial cartilaginous lamina returns to its resting closed position and the lateral pharyngeal wall retuns back to its normal position.

Disorders involving this opening and closing process of the eustachean tube have been known to cause chronic negative middle ear pressure / persistent otitis media with effusion. Obstructive disorders involving the eustachean tube may involve mucosal oedema, inflammation, mucosal or purulent secretions, mucosal projections due to mucosal oedema simulating small polypi. Dynamic dysfunctions are caused due to weakness involving the Tensor veli palatini muscle. Weakness involving the levator veli palatini muscle have also been documented. Similarly hyperactive contractions involving either the tensor veli palatini or levator veli palatini muscles create an excessive muscular bulk that can paradoxically block the eustachean tube lumen during the phase of swallowing.

Patients with patulous eustachean tube have a significant concave defect in the anterolateral wall throughout the valve area. This concavity occurs in the region where it would be convex in patients with a competent eustachean tube valve. Patients with patulous eustachean tube demonstrated loss of tissue volume involving mucosa, submucosa, Tensor veli palatini muscle, ostmann's pad of fat, lateral cartilaginous lamina or a combination of any of these entities.

Functional histology of middle ear cleft:

Middle ear mucosa is an extension of mucosa from the nose and nasopharynx. Attic area is lined by pavement epithelium while mesotympanum is lined by cuboidal cells. Hypotympanum is lined by ciliated columnar epithelium. Inflammatory process involving the antero inferior portion of middle ear cause mucociliary clearance problems. This causes accumulation of mucous causing serous or seromucinous otitis. Inflammation involving mucosa of the postero superior portion of middle ear cause problems with gas exchange causing the ear drum to retract in that area. These retraction pockets lead to cholesteatoma formation.

Under normal conditions gas transfer via the fibrocartilaginous eustachean tube is rather poor when compared to controlled gas exchanges between the mucosa of middle ear cleft and blood. This gas exchange that occurs via the mucosa is termed as mucosal gas exchange. The amount of gas reaching the middle ear cavity is very poor (1 microlitre 500-1000 times a day). The duration of opening of eustachean tubeis about 3-4 minutes a day.

In situations where the middle ear mucosa is healthy and normal altitudinal changes like flying / diving / accidental explosions cause sudden change in middle ear pressure. Pressure equalisation in this scenario occurs via the eustachean tube.

In situations where the middle ear mucosa is inflammed the fibrocartilaginous eustachean tube balances the middle ear pressure by gas exchange between the middle ear cleft and blood compartment.

Gas exchange between the middle ear cleft and blood compartment:

Gas exchange between middle ear cleft and blood compartment takes place via the middle ear mucosa. In the tympanic cavity oxygen and nitrogen are absorbed from the cavity. This absorption takes place via the mucosa into the blood compartment. Carbondioxide and water vapor are diffused from the blood compartment via the mucosa into the tympanic cavity.

In normal conditions when the middle ear mucosa is healthy, the tympanic cavity contributes to variations in middle ear pressure by two kinds of mechanisms:

1. In situations where the pressure changes in the middle ear take place all of a sudden (altitude, flight, diving, explosions etc) the compliance of tympanic membrane lamina propria is very important. The elasticity of the lamina propria and the flexibility of the incudo-malleal joint act as static pressure receptor for the tympanic membrane ensuring three dimensional movements into the malleus.

2. The second mechanism is the steady exchange of gas through the mucosa. This exchange of gas depends on the functional properties of the cells of the mucosa, diffusion rate of gas and the behavior of the vascular system. Exchange of gas in / out of the tympanic cavity depends on:

Relative speed of gas diffusion (This include both absorption and elimination)

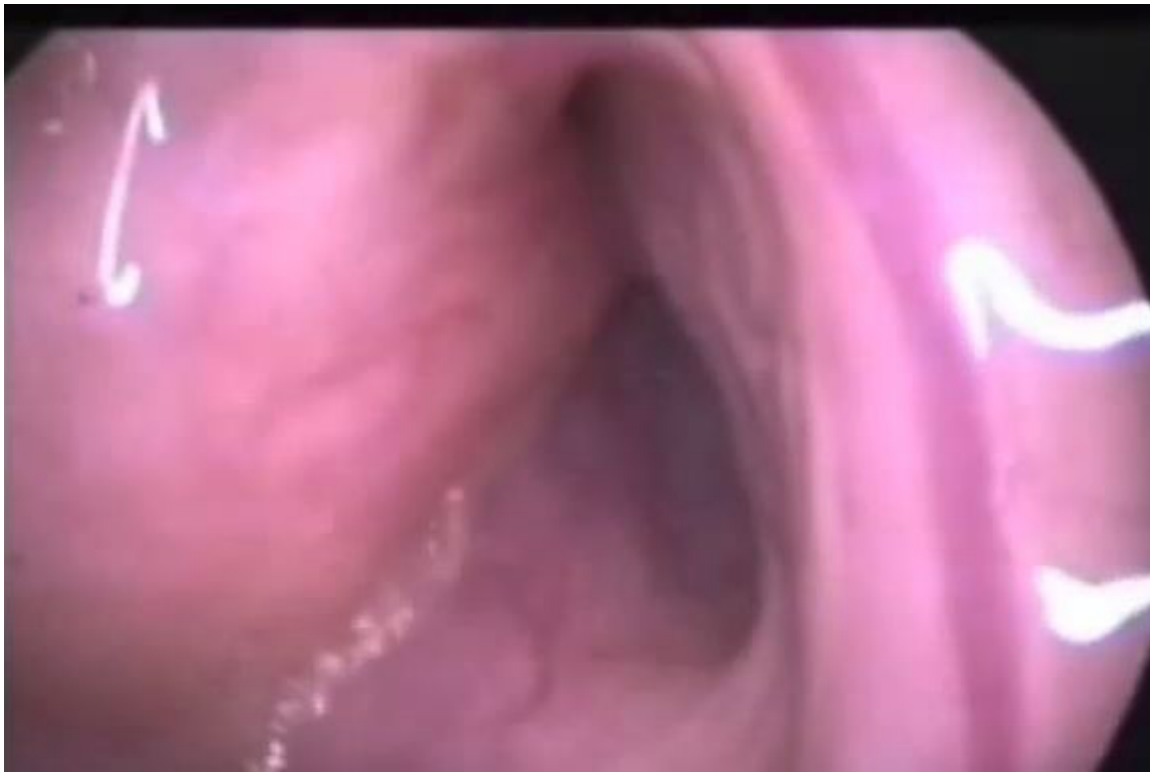
Diffusion rate of gas (This value is constant for each gas)

Characteristic of the particular gas concerned

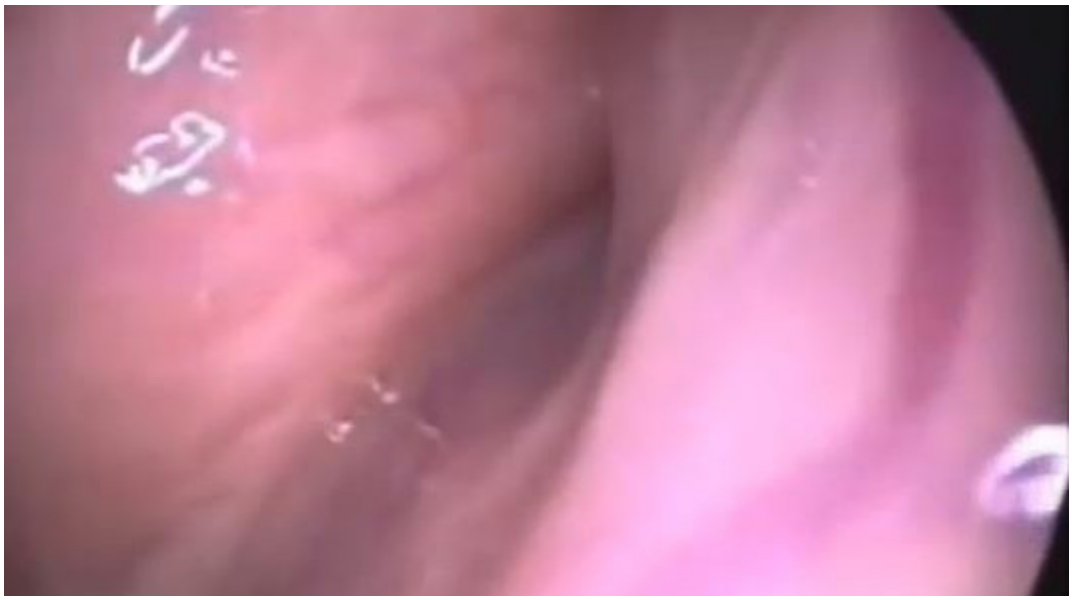
Variations in the middle ear cleft blood flow

Variations in permeability of blood vessels

Normal variations in middle ear pressure recorded during the 24 hour period is related to the vascular adaptations due to the body position and sleep.



Nasopharyngeal end of eustachean tube open



Discharge seen from nasopharyngeal end of eustachean tube

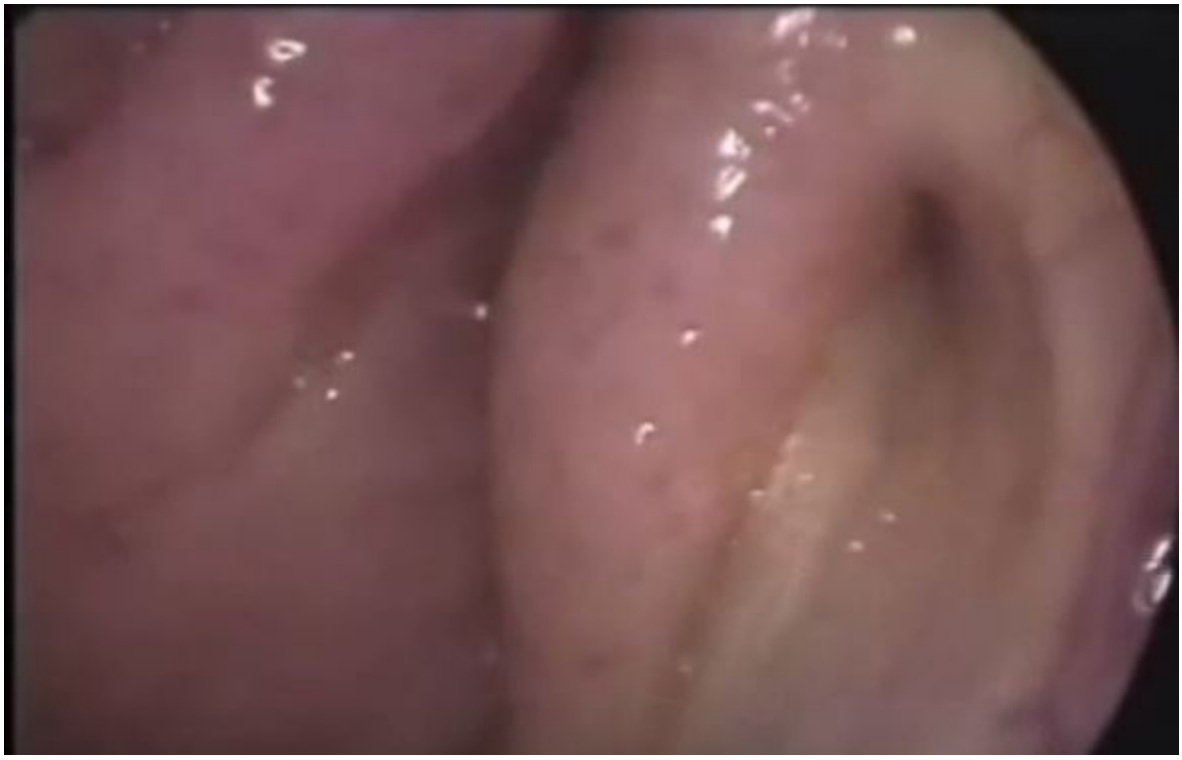


Image showing thick secretion from nasopharyngeal end of eustachean tube

The mastoid air cell system contributes to the variations in the gas composition of the middle ear cavity. It is much less than that of the contribution of the middle ear mucosal surface. The size of the mastoid air cell system varies between individuals. Small mastoid air cell system is associated with middle ear disease, because the smaller the system the more likely it is to develop middle ear pathology.

In normal conditions the slight negative pressure created by exhange of gas between the tympanic cavity and the blood compartment is compensated by the gas contained in the mastoid system. Mastoid air cell system acts as a first level buffer in these conditions allowing the recovery time for the normal middle ear ventilation mechanism. In inflammation the mucosal vascularity increases thereby increasing outflow of oxygen from the middle ear cavity to the blood compartment causing increased middle ear negative pressure.

Middle ear pressure regulating system:

Middle ear is equipped with sensors and nerves helping it to maintain middle ear pressure. The quality of gas in the middle ear cavity is maintained by chemosensitive sensors that act on the neurocapillary system. The eustachean tube manages the quantity of gas inside the middle ear cleft by the use of barosensitive receptors. these receptors act on the neuromuscular system.

In normal conditions the middle ear gas pressure is maintained close to atmospheric levels. Middle ear pathology can cause increase or reduction of middle ear pressure. These pressure variations are handled by pressure sensitive receptors present in the middle ear cavity.

Chemoreceptors in the middle ear cavity are part of glomus tissue in the middle ear cavity. These receptors respond to gas composition changes in the middle ear cleft by initiating middle ear ventilation reflexes. These reflexes are initiated by eustachean tube muscles.

Studies by Rockiley and Hawke reveal that normal ear is very sensitive to pressure changes in the middle ear cavity. This is possible because of the presence of stretch receptors in the tympanic membrane. Pars tensa portion of the ear drum is relatively stiff and it functioned as an efficient sound conducting tool, whereas the pars flaccida which is more flaccid and easily displaced is known to accomodate pressure differences in the middle ear cavity. This elasticity of pars flaccida is attributed to the presence of elastin fibers in contrast to that of pars tensa which is devoid of it. Studies reveal that eustachean tube function changed following anesthesia of the tympanic membrane.

Pressure receptors at the pharyngeal end of eustachean tube:
Fibro cartilaginous eustachean tube opens up when pressure imbalance occurs on both sides of the ear drum. This happens due to the presence of pressure receptors in the vicinity of the pharyngeal end of eustachean tube and the receptors present in the middle ear cleft and tympanic membrane.

Inflammation of middle ear and the mediators

Introduction:

Inflammation of the middle ear cavity mucosa is common during infancy. Otitis media with effusion is the common denominator. Inflammatory cellular infiltrate of submucosa combined with poor ventilation of middle ear are the features noted in serous otitis media. This results in hypersecretion of mucous and alteration of mucociliary clearance leading on to accumulation of fluid and cellular debris in the middle ear cavity. Studies have revealed that otitis media with effusion is the common origin for all types of chronic otitis media be it open mucous otitis (classic csom), fibro-adhesive otitis, atelectatic ear etc.

The middle ear epithelium serves two important functions:

1. Elimination of mucous and cellular debris to the pharynx by means of mucociliary clearance

2. Maintaining air filled fluid free middle ear cavity for efficient sound transmission.

These two functions are achieved due to the presence of Microvilli on the apical membranes of all cell types in the middle ear epithelium. These microvilli amplifies ion transport process.

Tracts of ciliated and mucous cells that actually converge towards the eustachean tube. The beating of these cilia propels the upper viscous layer of mucous (the gel layer) along these tracts. This is actually a low energy movement and is possible only if the ciliary bodies are bathed in a layer (sol) of fluid. If the sol layer is too deep, cilia will loose contact with the gel layer and the resulting uncoupling will leave the gel layer stagnant. Too shallow a sol layer will impede ciliary movement resulting in inefficient beating. It should be stressed that the ion transport process i.e. absorption of sodium and water controls the depth of this sol layer contributing to the efficient ciliary beat process.

It should also be remembered that the negative pressure which prevails in the middle ear of patients with otitis media always induce transudation of fluid which ion and fluid transport mechanism might counterbalance in order to prevent the filling of the middle ear cavity.

Role of PGE2 and Oxidants:

PGE2 belongs to prostaglandin group and is commonly involved in inflammation. It causes an increase in the intracellular cyclic AMP content. Cylcic AMP is an important biological molecule. It regulates ion transport and is considered as a secondary messenger for inflammatory mediators. This molecule would increase sodium influx into the middle ear cavity. This would naturally be followed by influx of chloride and water leading on to fluid accumulation in the middle ear cavity. This effect has been shown to be blocked experimentally by the presence of the drug amiloride which is a potent sodium channel blocker.

Reactive oxygen:

These include reactive metabolites derived from oxygen. These include hydrogen peroxide, superoxide anion and hydroxyl radical. These metabolites are produced in large quantities by phagocytic cells and can hence be considered as inflammatory mediators. Their effects on the epithelium include:

1. Impairment of epithelial ion transport
2. Decrease of sodium potassium ATPase activity
3. Direct interference with channel proteins
4. Increase of epithelial permeability
5. Finally cell death

Low concentrations of oxidants would cause just enough secretion of hydrogen peroxide to activate prostaglandin synthesis, increase in cellular cAMP without deleterious cellular effects. On the other hand higher concentrations of oxidants might produce enough hydorxyl radical to reveal its cellular toxic effects which could eventually lead to cellular death.

Nitric oxide in middle ear epithelium:

Nitric oxide is a free radical which has recently come to limelight. It is synthesized by nitric oxide synthase. Three major isoforms of nitric oxide have been indentified. They include:

Type I - Neuronal nitric oxide

Type III - Endothelial nitric oxide

Type II - Inducible nitric oxide. This is modulated in the presence of appropriate stimulus like interferon gamma.

Studies demonstrate that nitric oxide possesses potent anti-inflammatory property (direct action). Direct effects of nitric oxide are meant to be regulatory (antiinflammatory). This effect predominates under physiological conditions and when the rate of nitric oxide production is low. Indirect effects mediated by nitric oxide are inflammatory in nature and is caused by nitric oxide derived intermediates. This effect predominates during inflammation. Nitric oxide is continuously produced in the middle ear cavity and could be involved in homeostasis of middle ear cavity. It has also been shown to be involved in mucous secretion and regulation of ciliary beating. It could also contribute towards keeping the middle ear cavity pathogen free.

Excess nitric oxide secretion potentiates the development of inflammation. This is caused by means of peroxynitrite production. Use of ventilation tube has become the mainstay in secretary otitis media management. This device restores middle ear mucociliary clearance due to diffusion of nitric oxide out of middle ear cavities through the tube. Nitric oxide may play a part in tissue damage because it is cytostatic and cytotoxic not only for the invading microbe but also for the cells that produce it. It can also affect the neighboring cells. In certain situations it may interact with oxygen derived free radicals thereby generating molecules that could enhance its cytotoxic effects. Inhibitors of nitric oxide synthase and Nitric oxide donors in a way protect tissues from injury related issues. Nitric oxide has been demonstrated to have a multifaceted role during immune reactions. It can cause enhancement of vasodilatation, formation of oedema.

It can also modulate sensory nerve endings thereby creating pain and tenderness. It is also known to enhance leukocytic activity and can as well be cytotoxic. It is also known to trigger apoptotic pathways. It can also facililtate autoimmune reactions. Nitric oxide synthase II inhibitors have been proved to be of value in causing remissions from auto immune diseases. At low concentrations nitric oxide have known to protect cells from apoptosis by inactivation of CPP32 like protease and by increasing Bcl2 protein expression. High doses of nitric oxide induce thymocyte as well as splenic T cell apoptosis. These anti or proapoptotic effects of nitric oxide involves interaction of nitric oxide with simultaneously formed oxygen intermediates and are dependent on the redox state of the cell.
Studies demonstrate that Th1 cells are more susceptible to apoptosis than Th2 cells. Nitric oxide regulatesthe Th1/Th2 balance.

Nitric oxide is an important mediator of homeostatic processes and host defence. Any changes in its generation or actions contributes to pathologic states. Its discovery has thrown up numerous exciting developments in evolving new approaches for management and treatment of various diseases.

It has also been demonstrated that human cells are more reistant to cytotoxic actions of nitric oxide, hence its cytotoxic actions could be less relevant when compared to that of its regulatory effect in humans.
Nitric oxide production in the immune system:
Generation of nitric oxide is a feature of immune system cells which include:

Dendritic cells
NK cells
Mast cells
Phagocytic cells
Endothelial cells
Epithelial cells
Vascular smooth muscle cells
Fibroblasts
Keratinocytes
Chondrocytes
Hepatocytes
Mesangial cells
schwann cells

Mechanisms of regulation of Nitric oxide production:

Expression of i nitric oxide synthase (iNOS) is regulated by cytokines and are determined primarily by the synthesis and stability of i Nitric oxide synthase mRNA. In contrast nNOS (seen in neuronal cells) and (eNOS) seen in endothelial cells are preformed proteins. Activity of these preformed Nitric oxide isomers can be switched on by elevation of intracellular calcium concentration and the binding of calmodulin in response to neurotransmitters or vasoactive substances.

Regulators of Nitric oxide production by iNOS:

The iNOS isoform is regulated by cell - cell contact via adhesion and costimulatory molecules, cytokines, immune complexes, microbial and viral products.

Nitric oxide plays a role in the pathogenesis of otitis media with effusion by mediating intracellular and intercellular messengers that trigger middle ear pathology depending on the amount of nitric oxide radicals.

itric oxide and biofilm associated infections:

Chronic infections of middle ear cavity are commonly associated with biofilm formation. Nitric oxide signaling has been shown to elicit different responses in bacterial biofilms. Commonly it causes dispersal of organism from biofilm there by making the organims more susceptible to antibiotics and immune reactions.

Nitric oxide may play a role in arginine metabolism and biofilm development in s.pneumoniae. and in regulating growth in penumococci. Concentrations of nitric oxide modulate pneumoccal growth by making dormant bacteria within the biofilm to become more metabolically active, making them more susceptible to antibiotic killing.

Targetted therapy to manage biofilm infection:

Chronic pseudomonas infections involving the middle ear cavity are difficult to manage because they have a tendency to form biofilms. Use of nitric oxide as an adjunct can disrupt P. aeruginosa biofilms making them susceptible to antibiotics. Low dose nitric oxide cause disruption of biofilm through increased bacterial phosphodiesterase activity. Low dose nitric oxide have shown to increase motility of P. aeruginosa cells.

Excess nitric oxide secretion potentiates the development of inflammation. This is caused by means of peroxynitrite production. Use of ventilation tube has become the mainstay in secretary otitis media management. This device restores middle ear mucociliary clearance due to diffusion of nitric oxide out of middle ear cavities through the tube.

Nitric oxide in animal tissues is generated by enzymatic action of nitric oxide synthases which oxidise L-arginine to L-citrulline. There are 3 known isoforms of nitric oxide synthases. These include:

1. Nitric oxide synthase I (NOS I). This is also known as neuronal form found in neurons

2. Nitric oxide synthase II (iNOS). This is also known as inducible nitric oxide synthase and is present in various cell types and is generated on inflammatory stimulation

3. Nitric oxide synthase III (eNOS). This enzyme has been primarily isolated from endothelial cells.

All these three isoforms have a similar molecular structure and require multiple cofactors of activation.

The neuronal and endothelial nitric oxide synthase are activated by binding of the calcium regulatory protein calmodulin. The inducible isoforms of nitric oxide synthase are regulated at transcriptional level and is independent of intracellular calcium levels.

Functions of these three types of nitric oxide synthase have been studied using mice. The nNOS deficient mice develop preserved hippocampal long term potentiation, gastroparesis and muscle disorders. The eNOS deficient mice is repsonsible for maintaining low vascular tone and preventing leukocytes and platelets from adhering to the vascular wall. The nNOS is known to act as a neuromodulator or neuromediator. The iNOS stimulated release of nitric oxide are known to play multiple roles in eliciting inflammatory response.

Injuries to blood vessels and tissues that occur as a part of inflammatory reactions are caused by interaction between nitric oxide and oxygen. This interaction causes formation of potent cytotoxic oxidants $ONOO^-$ This provides rationale for the use of Nitric oxide synthase inhibitors like L-arginine analogs to prevent unplesant inflammatory reactions from proceeding further. Clearance of oxygen radicals will help in reducing inflammatory reactions. Studies reveal that simultaneous release of Nitric Oxide and Oxygen radicals potentiates inflammation and when release of one radical exceeded the other it protected the tissue form inflammatory reactions. A lot of work needs to be done in this area which offers promise in managing adverse reactions caused due to inflammation.

Studies demonstrate reduction in levels of nasal nitric oxide (nNitric oxide) in children with adenoid hypertophy predisposing them to chronic rhino sinus infections. Since middle ear mucosa is in continuity with the nasopharyngeal mucosa, infections involving the middle ear cavity is also common in these children.

Role of innate immune receptors (TLRs) in middle ear inflammation:

Toll like receptors (TLRs) are a class of proteins that play a key role in the innate immune system. These receptors are single, membrane spanning, and non-catalytic receptors usually expressed on sentinel cells like macrophages, and dendritic cells. They have the ability to recognize structurally conserved molecules derived from microbes. Once these pathogenic microbes manages to breach the physical barrier of middle ear mucosa they are recognised by these TLRs which in turn activate immune cell responses.

There are various types of TLRs i.e.

TLR1, TLR2, TLR3, TLR4, TLR5, TLR6, TLR7, TLR8, TLR9 TLR10 and TLR11.

These TLRs are pattern recognition receptors with an ability to recognize molecules that are broadly shared by pathogens but are distinguishable from host molecules. These molecules are collectively referred to as pathogen-associated molecular patterns.

Among these TLRs TLR2, TLR4, TLR5 and TLR9 were found at the level of RNA and proteins in middle ear mucousa of both otitis media and non otitis media patients. These TLRs in the middle ear starts off the inflammatory process in response to microbial infection.

Role of Hypoxia in middle ear inflammation:

Classically hypoxia is defined as insufficient oxygen level in blood / tissues. This causes the cells to reduce their metabolic rates in order to conserve oxygen. HIF 1alpha is a known biomarker for hypoxia and vascular endothelial growth factor another biomarker secreted by endothelial cells in response to hypoxia have been isolated from middle ear secretions of patients with otitis media with effusion. Hypoxia in the middle ear of patients with otitis media could be due to eustachean tube obstruction as well as excess oxygen consumption of inflammatory cells found in large numbers in the middle ear cavity of these patients. Hypoxia promotes production of reactive oxygen species causing more damage to middle ear mucosal epithelium thereby sustaining middle ear inflammation

Eustachean tube blockage demonstrated elevated expression of

HIF 1 apha
VEGF
IL-1 beta
TNF-alpha

Myringotomy not only serves to discharge the middle ear secretions but also improves oxygenation of the middle ear cavity.

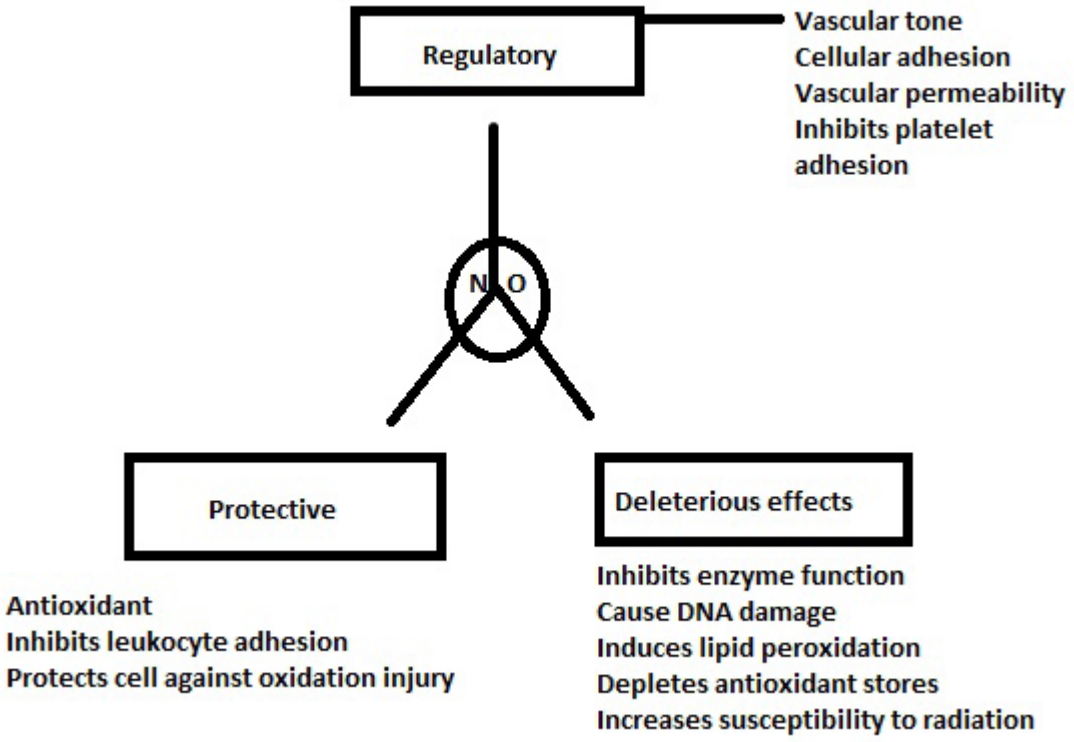


Figure showing effects of Nitric oxide

Acute Otitis Media

This is the most common disorder affecting infants and children. Statistics reveal nearly 2/3 of children have atleast one attack of acute otitis media before they become three years old necessitating atleast one course of antibiotic usage.

Nomenclature:

Acute otits media is used to describe all inflammatory disorders invoving middle ear mucosa. This includes both viral as well as bacterial induced otitis media. At this point it should be stressed that almost all cases of AOM are preceded by an episode of viral infection involving the upper respiratory tract. Suppuration following bacterial infections of middle ear cavity is known as Acute suppurative otitis media.

Acute suppurative otitis media is defined as suppurative infection involving the mucosa of the middle ear cleft. By convention it is termed acute if the infection is less than 3 weeks in duration.

Pathophysiology: Obstruction to the eustachean tube seem to be the most important antecendent event in the pathophysiology of acute suppurative otitis media. Majority of acute suppurative otitis media is triggered by upper respiratory infections which might find its way into the middle ear cavity through the eustachean tube orifice. Infections involving the nasopharynx may find its way into the middle ear through the pharyngeal end of eustachean tube. The infection is initially commonly viral in origin, allergy could also play an important role in the pathogenesis. Later the middle ear mucosa becomes secondarily infected by pathogenic bacteria. The bacteria commonly implicated in this disorder is S Pneumoniae, H. Influenza, and M Catarrhalis.

The majority of otitis media prone children have a patulous eustachean tube or an hypotonic eustachean tube. Children with neuromuscular disorders or with abnormalities of the first or second arch have a patulous eustachean tube leading on to this problem. To become pathogenic the bacteria must become adherent to the mucosa lining the middle ear cavity, this is made possible by prior infection of the middle ear mucosa by viruses.

Flask model explaining the role of eustachean tube in middle ear infections:

The eustachean tube, middle ear, and mastoid air cell system can be likened to a flask with a long narrow neck. The mouth of the flask represents the nasopharyngeal end, the narrow neck, the isthumus of the eustachean tube, and the bulbous portion, the middle ear and mastoid air chamber. The fluid flow through the neck of the flask would be dependent on the pressure at either end, the radius and length of the neck, and the viscosity of the liquid. When a small amount of liquid is instilled into the mouth of the flask, liquid flow stops somewhere in the narrow neck owing to capillarity within the neck and the relative positive air pressure that develops in the chamber of the flask.

The basic geometry is considered to be critical for the protective function of the eustachean tube - middle ear system. Reflux of liquid into the body of the flask occurs if the neck of the flask is excessively wide, or the length of the neck of the flask is too short as seen in children. Because infants have a shorter eustachean tube than adults, reflux is more likely to occur in the baby.

The position of the flask in relation to the liquid is another important factor. In humans, the supine position enhances flow of liquid into the middle ear; thus infants might be at risk for developing reflux otitis media because they are commonly supine. Reflux of liquid into the vessel can also occur if a hole is made in the bulbous portion of the flask, because this prevents the creation of positive pressure in the bulbous portion. This positive pressure is useful in the prevention of reflux of material from the neck of the flask.

If negative pressure is applied to the bulbous portion of the flask then this pressure is sufficient to cause aspiration of contents from the neck of the flask. This scenario is represented by high negative pressure in middle ear as it occurs in nose blowing, crying, closed nose swallowing, diving or airplane descent. The neck of the eustachean tube is supposed to be compliant hence compliance plays a vital role in prevention of reflux of secretions.

Clincial features:

Acute suppurative otitis media passes through 4 stages:
1. Stage of hyperemia

2. Stage of exudation

3. Stage of suppuration

4. Stage of resolution.

The progression of these stages depends on the virulence of the infecting organisms, resistance of the host, adequacy of antibiotic therapy. If the infecting organism is virulent or if the antibiotic treatment is not sufficient then the disease may progress to a stage of coalescent mastoiditis with its attendant complications.

Stage of hyperemia: Initial infection by infection results in hyperemia of the mucous membrane causing otalgia, fever and fullness in the affected ear. This stage is characterised by oedema of the mucoperiosteum due to vascular engorgement. Otoscopy show dilated vessels along the handle of malleus and along the rim of the tympanic membrane. Antibiotic therapy during this stage will help in resolution of the disease. Amoxycillin is the drug of choice.

Stage of exudation: Absence of treatment during the stage of hyperemia leads to the stage of exudation. In this stage there is outpouring of fluid from the dilated vessels of the mucoperiosteum. This fluid is serous in nature containing fibrin, red cells, and polymorphs. This exudate fills the tympanomastoid compartment really fast, and the whole middle ear cavity is under intense pressure due to this retained secretion. Pain is the most prominent feature of this stage.

The patients may have fever and fullness in the ear. Otoscopy shows a bulging ear drum with loss of all landmarks. The drum is reddish and bulging in nature. These patients have also coexistant mastoid tenderness due to mastoiditis.

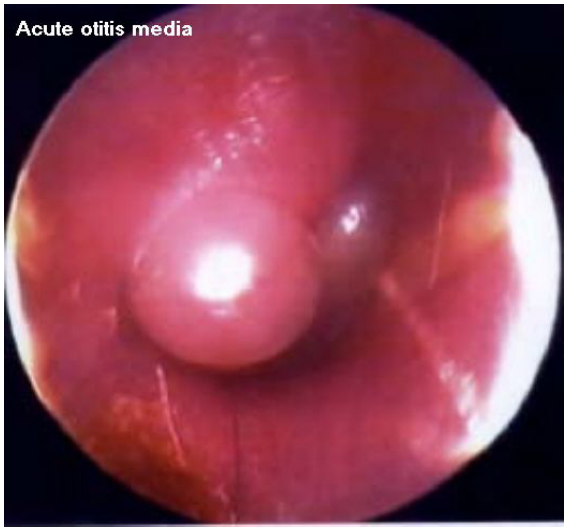
Stage of suppuration: Failure of treatment during the stage of exudation leads on to stage of suppuration. The exudate present in the middle ear cavity is a very good culture medium and hence there is secondary bacterial infection leading on to suppuration.

Stage of resolution: is preceded by either rupture of the ear drum leading on to a serous / serosanguinous / purulent discharge from the ear. When the middle ear is free from the exudate / pus the stage of resolution sets in. The patient has reduction in otalgia, fever subsides. The patient has considerable clinical improvement.
Stage of complication: If the infection persists beyond a period of 2 weeks then there is associated thickening of the mucoperiosteum especially in the air cells around the peri antral area leading to a block in the drainage from the antral cells. The pent up secretions in the mastoid air cell system causes intense pressure, venous stasis and local acidosis. This acidosis cause dissolution of calcium from the bone causing decalcification and coalescence of the mastoid air cell system. This condition is known as coalescent mastoiditis. This stage is characteristed by emergence of otalgia and low grade fever. Erosion of the outer cortex in the mastoid lead to the formation of abscess under the periosteum of the mastoid cortex. This condition is known as subperiosteal abscess.

Management:

Acute suppurative otitis media is a self limiting condition. If appropriate antibiotics are started early then it resolves. Amoxycillin is the drug of choice. Cephalosporins may also be started in refractive cases. Anti inflammatory drugs like ibuprofen is also prescribed in order to alleviate pain. Patients who are refractory to medical management may under go myringotomy in order to decompress the middle ear cavity. This procedure is done using a myringotome.

Coalescent otitis media and subperiosteal abscess are surgical complications. These patients must be taken up for surgery under adequate antibiotic cover.



Otoscopy showing bulging ear drum in a patient with otitis media

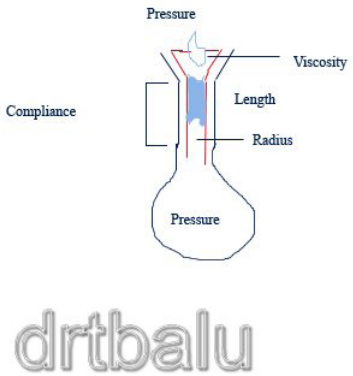


Figure explaining the normal eustachean tube functioning

Figure showing differences in eustachean tube between adult and child

Current trends:

Definition of acute otitis media has undergone lot of changes during recent times. According to the American Academy of Paediatrics the following criteria should be fulfilled before a diagnosis of acute otitis media could be made.

1. History of acute onset of signs & symptoms

2. Presence of middle ear effusion

3. Signs & symptoms of middle ear inflammation

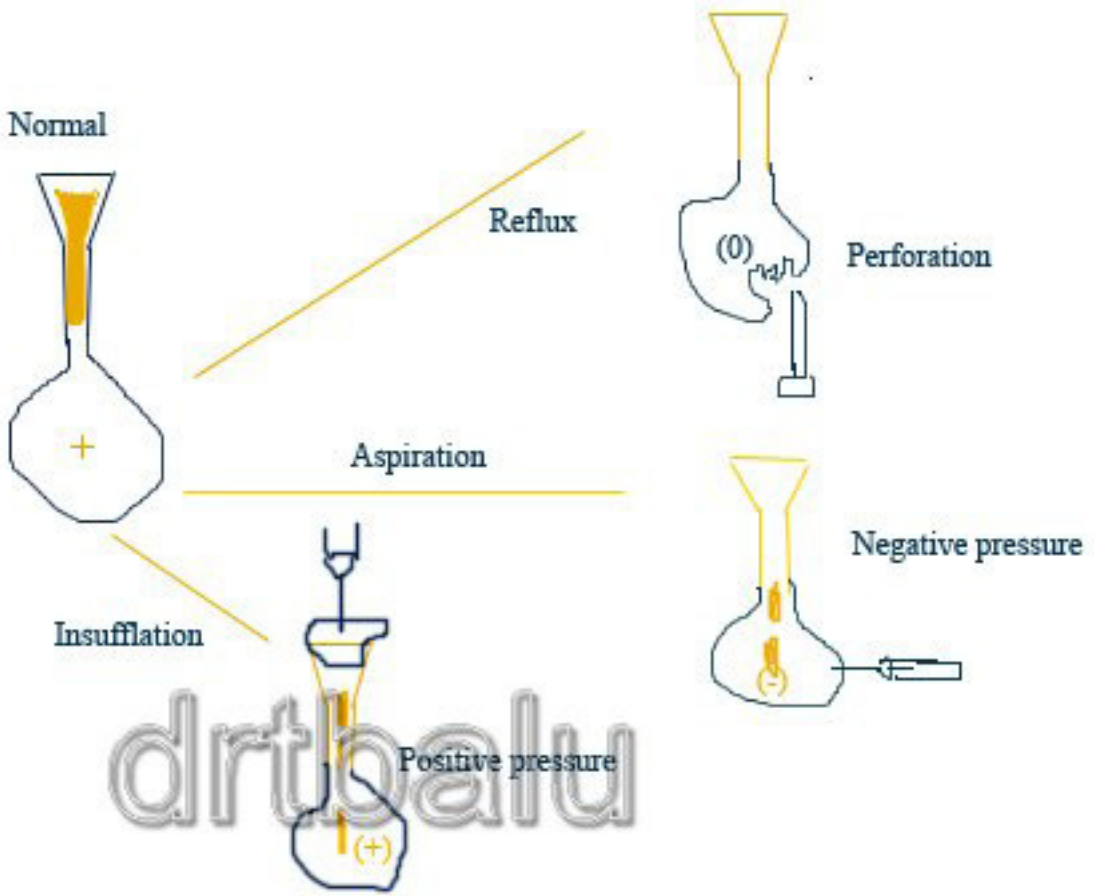


Figure showing pathogeneis of middle ear disease

The presence of middle ear infection is indicated by:

Bulging of ear drum
Limited / absent mobility of ear drum
Air / fluid level behind the ear drum
Otorrhoea
Signs & symptoms of middle ear infection include:

Erythema of ear drum
Otalgia with discomfort in the ear
According to American Academy of Paediatricians clinical history had a very poor predictive value in young children.

Role of Pneumatic otoscopy:

The committee constituted by American Academy of Paediatricians has stressed the important role of pneumatic otoscopy in the diagnosis of middle ear effusion. This is one of the most important clinical examination that could identify middle ear effusion. Tympanometry can atmost supplement pneumatic otoscopy because the external auditory canal of infants is highly pliable and the resultant graph would only produce a canalogram rather than tympanogram. The extreme pliability of external canal will cause the tympanometry readings to be normal even in the presence of middle ear effusion .

Before performing pneumatic otoscopy the external canal should be cleaned off cerumen / debris. The speculum used should be of proper dimensions to cause a proper seal of external auditory canal.

Pneumatic otoscopic findings to suggest the presence of middle ear effusion include:

Fullness / bulging of ear drum causing distortion to the normal cone of light
Reduced / absent mobility
Cloudiness of ear drum - caused by oedema in the mucosal layer
Redness of the ear drum - Could be seen even in a crying child
Presence if air fluid level as seen through the ear drum

Major challenge to the attending physician is to discriminate between Acute otitis media and otitis media with effusion. This discrimination is a must because otitis media with effusion may commonly be caused by viral infections of the upper respiratory tract. It should also be borne in mind that otitis media with effusion may be a prelude to acute otitis media / may be caused by acute otitis media. In patients with otitis media of viral etiology it is prudent to avoid unnecessary use of antibiotics.

Pathophysiology:

Eustachean tube has commonly been implicated. Eustachean tube has been attributed with three functions:

Protection
Ventilation
Drainage
In patients with acute otitis media it is the protective function of the eustachean tube that takes a beating.

Microbiology:

Causative organisms include:

Streptococcus Pneumoniae
H. Influenza
M. Catarrhalis
Pneumococci - common in infants

Role of antibiotics:

Observation of the patient without resorting to antibiotic use is an option to be considered in the following scenario:

Children above the age of 2
Aom is devoid of complications
Diagnosis is pretty certain
Patient is ready to attend periodic follow ups

Acute otitis media in a child of less than 2 years of age is likely to cause complications. These children should be immediatly treated with a course of antibiotics. The drug of choice being ampicillin in doses of 80 - 90 mg /day in three divided doses. Lack of symptomatic improvement within 48 hours after starting the antibiotic should alert the physician the need to change the antibiotic. Drug resistance is commonly caused by Beta lactamase producing H. Influenza / M. catharrhalis. In these patients potassium clavulanate can be prescribed in addition to amoxycillin in doses of 6.4 mg / kg / day in two divided doses.

Second line antibiotics include:

Cefdinir - 14mg/kg/day in two divided doses
Cefpodoxime - 10mg /kg/day single dose
Cefuroxime - 30mg/kg/day in two divided doses
Prevention:

Encourage breast feeding of infants
Pneumococcal / Influenza vaccines
Hygiene in day care centres

Otitis media with effusion:

Synonyms: Secretory otitis media, glue ear, serous otitis media, non purulent otitis media.

Definition: Otitis media with effusion is defined as chronic accumulation of mucus within the middle ear, and rarely this could involve the mastoid air cell system. This accumulation causes conductive hearing loss.

Histology and histopathology of eustachean tube: The pseudostratified ciliated columnar epithelium of respiratory tract extends up the eustachean tube as far as the anterior part of the middle ear cavity. These cells are capable of producing mucous. There are also goblet cells seen in their midst.

These cells are also capable of secreting mucous material. Otitis media with effusion is caused by inflammation of this epithelium in the eustachean tube and hypotympanum. In established cases of glue ear, the cuboidal epitheliumof middle ear and mastoid air cells gets replaced by thickened pseudostratified columnar epithelium. The cilia of these cells have also been found to be ineffective in propelling the secretions into the nasopharynx. The submucosa is found to be oedematous, inflammed with dilated blood vessels with increased number of macrophages and plasma cells.

Etiology:

1. In many children otitis media with effusion is preceded by an episode of acute otitis media. This is common in children who is more prone for upper respiratory infections. Common being viral infections which damages the eustachean tube epithelium.

2. Craniofacial abnormalities: Children with cleft palate have deficient palatal muscles causing a poor eustachean tube function leading on to Otitis media with effusion. This occurs despite a successful surgical repair of the cleft palate. Children with Down's syndrome are also more prone for OME.

Note: Children with bifid uvula donot appear to have higher incidence of OME

3. Allergy: Previously nasal allergy has been postulated as an important factor in the development of Otitis media with effusion. Studies have been unequivocal.

4. Gastrooesophageal reflux: GERDS has been commonly demonstrated radiologically in children with OME. Furthermore biochemical analysis of middle ear fluid have demonstrated significant amounts of pepsin (in 80% of cases).

5. Parental smoking has been attributed as an important predisposing factor for the development of OME.

Age of occurrence: OME shows classically a bimodal distribution. The first peak occurs around 2 years of age, and the second peak occurs at about 5 years of age. This distribution occurs roughly around the ages when the child goes to preschool and primary school.

Seasonal association: OME commonly occurs during winter season, when there is more likelihood of upper respiratory infections, and also because of the possibility of closer contact with affected children. This is seen in temperate zones. In non temperate zones it is commonly seen during rainy season.

Clinical features: A high index of suspicion is necessary to identify this condition. Every child with upper respiratory infection must be otoscopically examined. Otoscopic findings: The tympanic membrane may be bulging, or retracted with a distorted cone of light. The ear drum may appear yellow, blue or simply clear white. Pneumatic otoscopy will reveal a ear drum which has a restricted mobility.

Microbiology of OME: Commonly middle ear effusions due to glue ear is sterile. Rarely bacteria could be cultured. The incidence of these pathogens are higher in children under the age of 2, and in children with recurrent upper respiratory infections.

Investigations:

Puretone audiometry: Demonstrates mild to moderate conductive deafness.

Tympanograms (Type B) is commonly associated with OME. Type A is infrequently associated while Type C falls somewhere inbetween. Tympanometry can be used as a screening test to identify patients with OME.

Free field audiometry: Demonstrates deafness.

Management:

1. Antibiotics: Amoxycillin is the drug of choice followed by cephalosporins.

2. Nasal decongestants like oxymetazoline / xylometazoline may help in some cases.

3. Topical nasal steroids can be used in resistant cases.

4. Autoinflation of eustachean tube by performing valsalva maneuver. Balloon blowing may also help.

Surgical management:

1. Adenotonsillectomy

2. Myringotomy and insertion of ventilation tubes

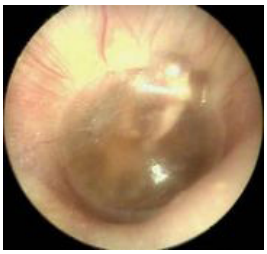


Figure showing appearance of ear drum in secretory otitis media

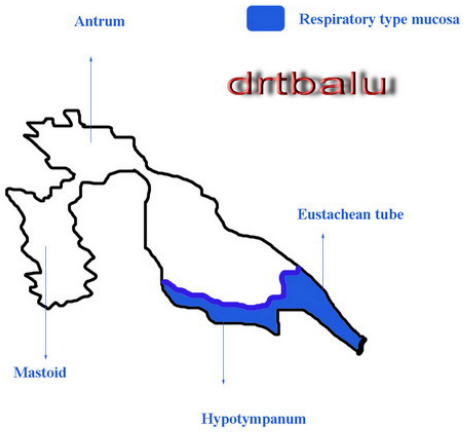


Figure showing the areas lined by respiratory type of epithelium

Eosinophilic otitis media:

Features of Eosinophilic otitis media:

Sudden deterioration of hearing

Bronchial asthma

Allergic rhinitis

Intractable otitis media

Persistent otorrhoea

Incidence:

Incidence of eosinophlic otitis media is not clearly known. Literature search puts it to be rather common cause of otitis media with effusion.

Managment:

Patients diagnosed with this condition should be warned of the possibility of sudden deterioration of hearing.

Administration of systemic / topical steroids 6 could be of benefit in these patients.

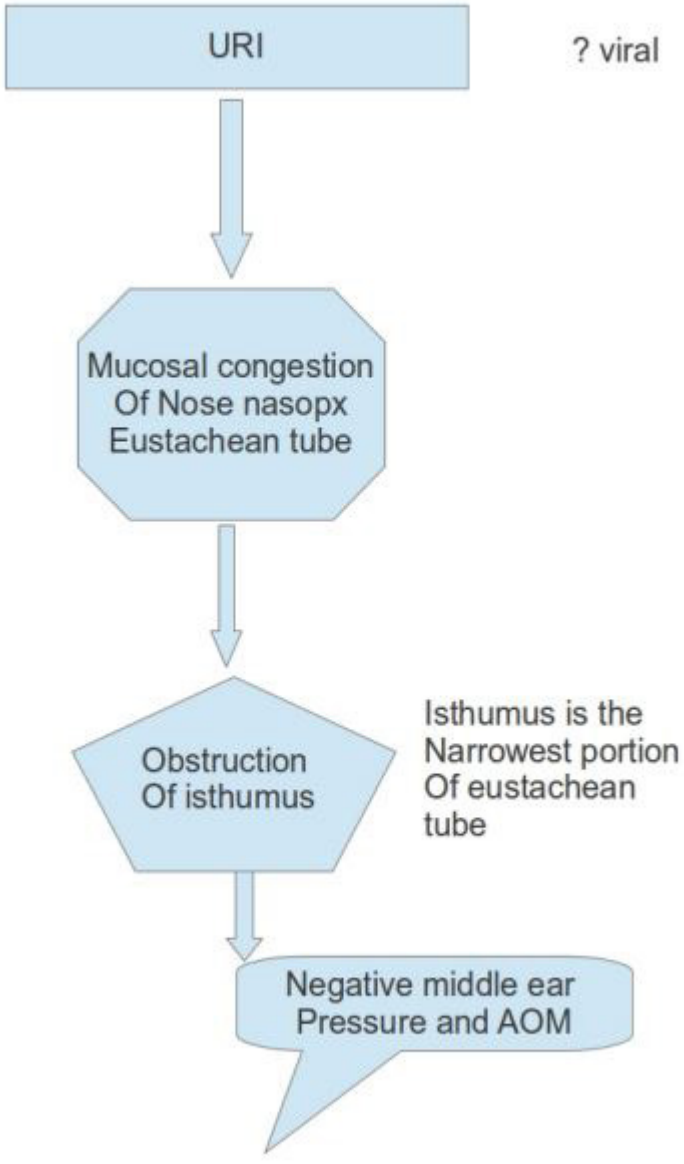
Antihistamines and leukotreine receptor antogonists can also be used with benefit.

Grommet insertion is indicated in patients with acute sudden hearing loss.

Role of pacifiers in causation of otitis media in children:

Acute otitis media is a common disorder in childhood. Role of pacifiers have been extensively studied as a causation of acute otitis media. Current concensus is that use of pacifiers are known to predispose development of acute otitis media in children. Children who use pacifiers are more prone for recurrent epidosdes of acute otitis media. This increase in risk could be due to increased incidence of reflux of nasopharyngeal contents into the middle ear cavity via the eustachean tube. It has also been postulated that pacifiers cause irreversible changes to dentition and eustachean tube function.

Pathophysiology of acute otitis media



Current concepts in diagnosis of Acute otitis media:

Acute otitis media and otitis media with effusion are very common childhood disorders. Studies reveal that most children have experienced atleast one episode of acute otitis media between ages 3 - 6. Nearly 15-20% of school going children suffer from middle ear effusion. It is imperative to make an accurate diagnosis before actually starting the treatment. There is actually no gold standard investigation / feature available to diagnose this condition. Diagnosis is usually made on history, symptoms and progression of the symptoms.

Middle ear cavity should be viewed carefully in these patients. In the absence of perforation of ear drum this becomes all the more difficult. The ear drum should be visualized in its toto for this to happen a large aural speculum should be used just to straighten the external auditory canal. It should be pointed out that the external canal is not a straight tube and a gentle posterior traction of the pinna would facilitate better visualization of the ear drum.

Use of otoendoscope have made the process of examination of ear that much easier. It can be inserted throuugh the perforation in order to visualize the middle ear cavity. For comforatable otoscopic examination proper positioning of the patient's head is of utmost importance.

The tympanic membrane is adult size even in infants. It is a three layered structure with the outer layer lined by stratified squamous epithelium, middle fibrous layer and inner mucosal layer.

Normal ear drum is translucent and pearly white in color. The handle of malleus and its short process can be clearly visualized through the ear drum.

Guidelines made by American academy of paediatrics to diagnose acute otitis media (2013) include:

1. This diagnosis should be made in children who present with moderate to severe bulging of the ear drum

2. Recent onset of otorrhoea (otitis externa to be ruled out)

3. AOM can be considered in the presence of mild bulging of ear drum associated with ear pain (crying child with constant tugging of ear) associated with intense erythema of ear drum

4. Diagnosis of AOM should not be considered in children with normal middle ear based on tympanometry / pneumatic otoscopy

On perusal of these new guidelines it could be inferred that great emphasis is placed on otoscopic examination of the patient in order to come to a correct diagnosis. Otoscopic examination should focus on the following features:

Color of the ear drum

Position of the ear drum

Mobility of ear drum

Normal ear drum is pearly white in color.

Red drum: This type of ear drum is seen in acute otitis media

Blue drum: Blue colored ear drum is seen in patients with secretory otitis media

Chalk white drum: This type of drum is seen in patients with tympanosclerosis which indicates previous episodes of middle ear infections

Yellow / creamy colored ear drum: Is seen in mucoid secretions filled middle ear cavity as seen in mucoid secretory otitis media.

Ear drum positions:

At birth ear drum is nearly horizontal in position but its orientation changes dramatically within the first few years of life. This is because the skull base grows in a vertical direction.

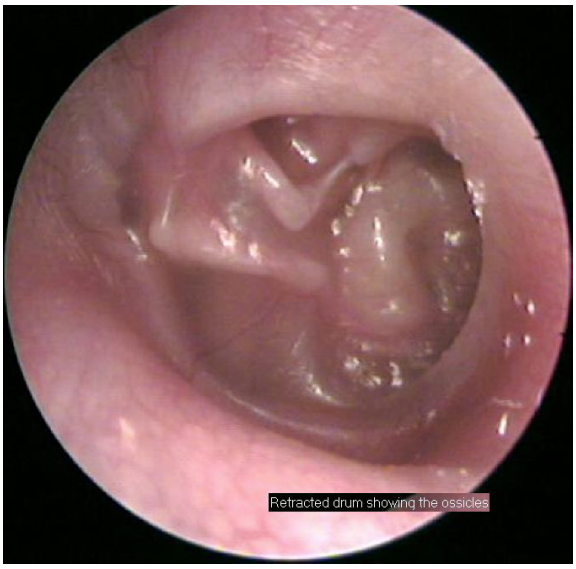


Image showing retracted ear drum clothing the middle ear structures

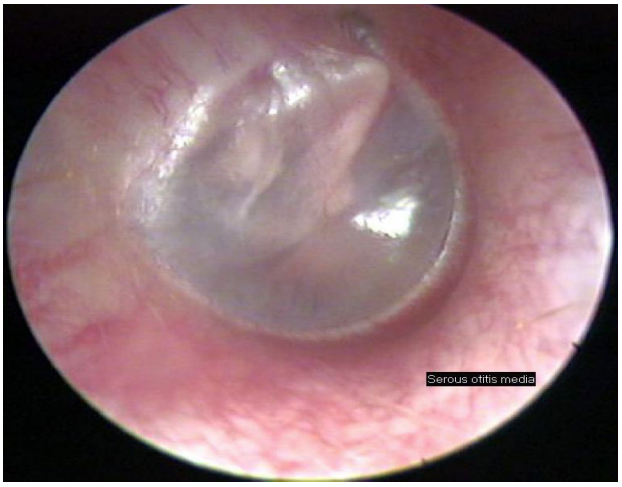


Image showing blue drum a feature of secretory otitis media

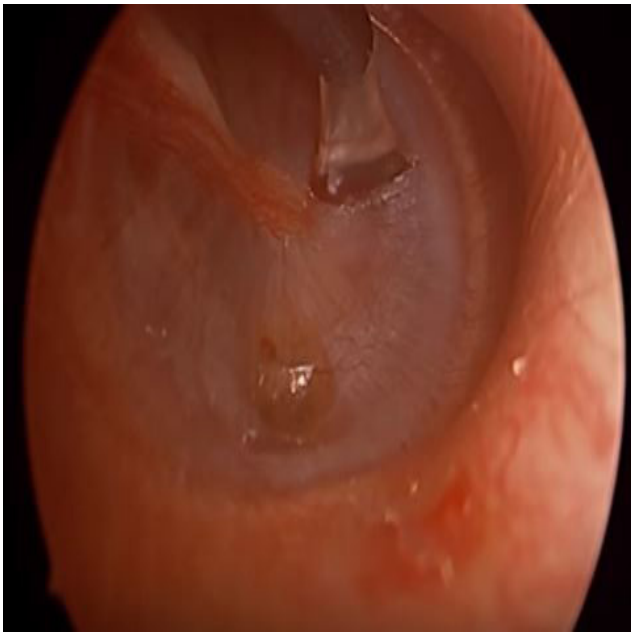


Figure showing glue ear being drained

Impaired mobility of ear drum had the highest sensitivity and specificity in the diagnosis of acute otitis media. It has been proved that bulging tympanic membrane with impaired mobility is the best predictor of acute otitis media. Moderate to severe bulging of ear drum is the most important characteristic in the diagnosis of acute otitis media.

As a symptom ear pain is a sensitive and specific predictor for acute otitis media. This is not as sensitive as bulging ear drum. Ear pain has been reported in only 50% of children with acute otitis media. Other signs which is of lesser relevance in the diagnosis of acute otitis media inlcude:

Restlessness
Ear rubbing
Fever
Non specific respiratory complaints

Pneumatic otoscopy really identifies restricted or loss of movement of ear drum which is a feature of acute otitis media.

Tympanometry:

This is used as an adjunct to penumatic otoscopy in the diagnosis of acute otitis media. Tympanometry is ideally performed using low frequency probe tones (220-226 Hz). The curves generated are classified as Jerger type A, B, or C curves. Type A represents normal middle ear compliance and Type B indicates no compliance while Type C indicates negative middle ear pressure.

Role of pure tone audiometry:

Pure tone audiometry testing is indicated in children when middle ear effusion is present for atleast 3 months or when there is delayed speech. The average hearing loss in middle ear effusion ranges between 25 - 35 dB.

Otitis media with effusion

Synonyms: Secretory otitis media, glue ear, serous otitis media, non purulent otitis media.

Definition: Otitis media with effusion is defined as chronic accumulation of mucus within the middle ear, and rarely this could involve the mastoid air cell system. This accumulation causes conductive hearing loss.

Histology and histopathology of eustachean tube: The pseudostratified ciliated columnar epithelium of respiratory tract extends up the eustachean tube as far as the anterior part of the middle ear cavity. These cells are capable of producing mucous. There are also goblet cells seen in their midst. These cells are also capable of secreting mucous material. Otitis media with effusion is caused by inflammation of this epithelium in the eustachean tube and hypotympanum. In established cases of glue ear, the cuboidal epitheliumof middle ear and mastoid air cells gets replaced by thickened pseudostratified columnar epithelium. The cilia of these cells have also been found to be ineffective in propelling the secretions into the nasopharynx. The submucosa is found to be oedematous, inflammed with dilated blood vessels with increased number of macrophages and plasma cells.

Etiology:

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2. Craniofacial abnormalities: Children with cleft palate have deficient palatal muscles causing a poor eustachean tube function leading on to Otitis media with effusion. This occurs despite a successful surgical repair of the cleft palate. Children with Down's syndrome are also more prone for OME.

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Age of occurrence: OME shows classically a bimodal distribution. The first peak occurs around 2 years of age, and the second peak occurs at about 5 years of age. This distribution occurs roughly around the ages when the child goes to preschool and primary school.

Seasonal association: OME commonly occurs during winter season, when there is more likelihood of upper respiratory infections, and also because of the possibility of closer contact with affected children. This is seen in temperate zones. In non temperate zones it is commonly seen during rainy season.

Clinical features: A high index of suspicion is necessary to identify this condition. Every child with upper respiratory infection must be otoscopically examined.
Otoscopic findings: The tympanic membrane may be bulging, or retracted with a distorted cone of light. The ear drum may appear yellow, blue or simply clear white. Pneumatic otoscopy will reveal a ear drum which has a restricted mobility.

Microbiology of OME: Commonly middle ear effusions due to glue ear is sterile. Rarely bacteria could be cultured. The incidence of these pathogens are higher in children under the age of 2, and in children with recurrent upper respiratory infections.

Investigations:

Pneumatic otoscopy

Puretone audiometry: Demonstrates mild to moderate conductive deafness.

Tympanograms (Type B) is commonly associated with OME. Type A is infrequently associated while Type C falls somewhere inbetween. Tympanometry can be used as a screening test to identify patients with OME.

Free field audiometry: Demonstrates deafness.

Lot of importance has been attached to pneumatic otoscopy in the diagnosis of otitis media with effusion. American Academy of Paediatrics in its guidlines for diagnosing otitis media with effusion has suggested routine use of pneumatic otoscopy by paediatricians in making a diagnosis of otitis media with effusion.

Tips for performing pneumatic otoscopy:

1. The size of the speculum chosen should be slightly wider than that of external auditory canal. This will enable the speculum to create a water tight seal with the external auditory canal. A speculum that is narrower than the size of the external auditory canal cannot create a proper seal and will cause false positive results.

2. After the speculum is attached the bulb is pressed and the tip of the speculum is occluded using index finger or middle finger. The bulb should stay compressed till the finger is removed from the tip of the speculum. This ensures that there is no air leak between the speculum and otoscope. If the bulb opens up even while the finger is occluding the tip of the speculum then it should be construed that there is leakage and the tubing of the pneumatic otoscope and the junction between the speculum and otoscope should be checked.

3. Before inserting the speculum into the external canal the bulb should be squeezed to its 50% volume and then it should be inserted. This maneuver allows the examiner to generate both negative and positive pressure (by releasing the bulb, or by further squeezing the bulb).

4. The speculum should be inserted deep into the ear canal so as to create an airtight seal, but not too tight since it could cause pain. Limiting insertion to the cartilaginous portion of the external canal is painless. Deeper insertion will involve the bony portion of the external canal thereby cause pain.

5. Mobility of tympanic membrane is observed by squeezing and releasing the bulb gently several times while observing for inward and outward movements of ear drum. Many children have negative middle ear pressure even under normal conditions and both positive and negative pressure application are needed to assess the drum mobility. Using slight and gentle pressure would allow the physician to perform a pain free pneumatic otoscopic examination.

6. Otitis media with effusion is diagnosed if the movement of ear drum is sluggish, dampened or restricted. Complete absence of movement is not a must for diagnosing otitis media with effusion. In ears without otitis media with effusion the movement of ear drum is rather brisk even when slight pressure is applied to the pneumatic bulb.

Tympanometry is not indicated in all patients. Specific situations where tympanometry is needed to make a diagnosis of middle ear effusion include:

1. Child intolerant to pneumatic otoscopy

2. Inability to perform pneumatic otoscopy because of difficulties in attaining proper seal between the external canal and the speculum

3. Difficulty in visualizing the ear drum due to the presence of cerumen

4. Difficulty in visualizing ear drum due to narrow external auditory canal

5. Desire to get objective confirmation of OME before embarking on surgical procedure

Management:

Majority of otitis media with effusion are self limiting and have a tendency to resolve spontaneously within a period of 3-6 weeks.

1. Antibiotics: Amoxycillin is the drug of choice followed by cephalosporins.

2. Nasal decongestants like oxymetazoline / xylometazoline may help in some cases.

3. Topical nasal steroids can be used in resistant cases.

4. Autoinflation of eustachean tube by performing valsalva maneuver. Balloon blowing may also help.

Studies reveal that antihistamines and nasal decongestants are totally ineffective in the management of middle ear effusion.

Surgical management:

1. Adenotonsillectomy

2. Myringotomy and insertion of ventilation tubes

Surgery is indicated when middle ear effusion remains unresolved even after a period of 3 months and is associated with Type B tympanogram (flat curve). Surgery is considered taking into consideration the hearing disability, developmental issues faced by the child due to hearing disability.

Children with associated risk factors like the presence of cleft palate should undergo grommet insertion.

Children with persistent otitis media with effusion who are not at risk should be re-examined between 3-6 month intravel till there is no fluid present inside the middle ear cavity.

Studies reveal that nearly 25-30% of children who underwent grommet insertion suffer from recurrence of symptoms after extrusion of grommet.

When a child needs repeat surgical procedure due to recurrence of otitis media with effusion even after grommet insertion then adenoidectomy along with grommet insertion should be resorted to. Recurrence rate is halved if both these surgeries are combined in the management of Otitis media with effusion.

Role of alternative medicine in the managment of otitis media with effusion is rather incomplete.

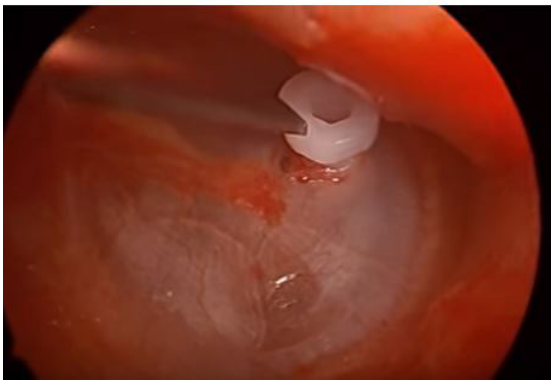
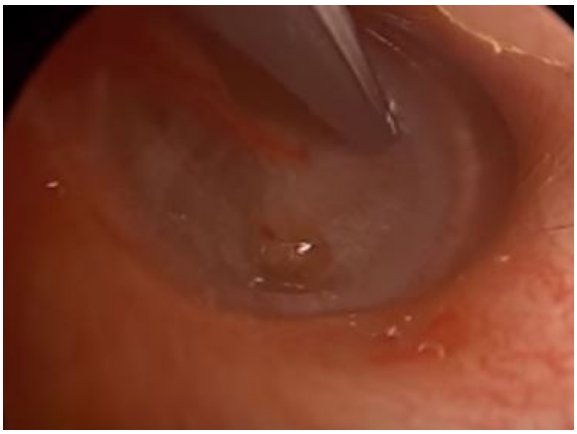
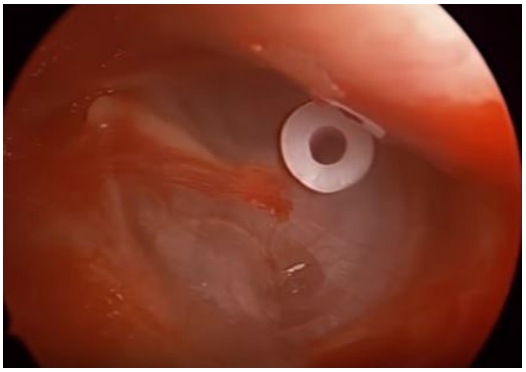


Figure showing Grommet being inserted

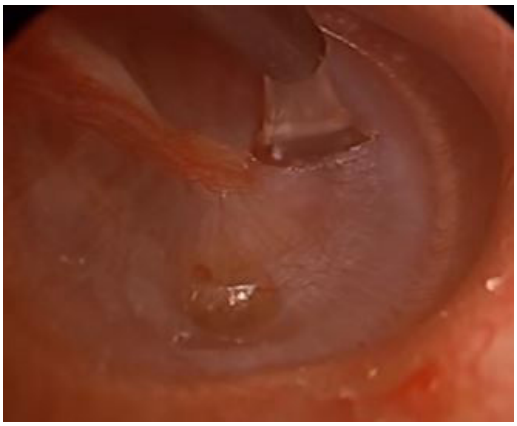
Antero inferior quadrant is preferred for grommet insertion because ventilation is ideal if performed closest to hypotympanum.



Incision given in the antero inferior quadrant for grommet insertion



Grommet seen inserted



Glue from middle ear being sucked out

CSOM

Definition:

Chronic suppurative otitis media is defined as a chronic infection of the mucosa lining the middle ear cleft. Middle ear cleft include the eustachean tube, hypotympanum, mesotympanum, epitympanum, aditus and mastoid air cell system.

Types of chronic suppurative otitis media:

Chronic suppurative otitis media is of two types:

1. Tubotympanic disease (safe type)

2. Atticoantral disease (unsafe type)

Tubotympanic disease: This is also known as safe disease because it is bereft of any serious complications. The infection is limited to the mucosa and the antero inferior part of the middle ear cleft, hence the name. This disease does not have any risk of bone erosion. The discharge any will flow through a perforation present in the pars tensa portion of the ear drum. This perforation is usually surrounded by a rim of remnant ear drum or atleast the annulus is intact. (Central perforation). The perforation is usually reniform (kidney shaped) because of poor blood supply to the affected portion of the tympanic membrane.

The infective activity of safe disease is related to the frequency of upper respiratory tract infections, the discharge tending to increase with increasing freqency of upper respiratory infections.

Aetiology:

1. Could be a sequelae to inadequately treated acute otitis media.

2. Acute suppurative otitis media causing persistant perforation which could be infected from bacteria in the external auditory canal. This condition is knonw as persistant perforation syndrome.

Microbiology of CSOM: In all varieties of CSOM the major organism found in the discharge are gram negative bacilli i.e. Ps. aeruginosa, E. coli, and B. proteus. These organisms are not commonly found in the upper respiratory tract, but they are found in the skin of external auditory canal.

Clinical features of tubotympanic disease:

1. The discharge in this condition is profuse and mucopurulent in nature.

2. The discharge is not foul smelling.

3. Since the infected area is open at both ends i.e. the eustachean tube end and the perforation in the ear drum, the discharge doesnot accumulate in the middle ear.

4. The ossicular chain is not at risk in this type of disorder, the conductive deafness caused is due to the presence of perforation in the tympanic membrane and thickening of the tympanic membrane.

5. Conductive deafness may also be accentuated by thickening of round window membrane due to the presence of secretions. Hearing loss is usually about 30 - 40 dB.

6. These patients have poorly pneumatised / sclerosed mastoid air cell system. This feature has been attributed to repeated attacks of middle ear infections during childhood causing inadequate pneumatisation of mastoid air cell system. In patients with pneumatised mastoid air cell system repeated middle ear infections can cause sclerosis with evidence of new bone formation. Mastoids in these patients may be sclerotic.

7. Pain in the ear when present is always associated with otitis externa. This commonly occurs when the patient attempts to clean the ear off the purulent secretions with a ear bud or cotton tipped applicator.

Pathology of tubotympanic disease:

Pathological changes depend on the stage of the disease. The stages are as follows:

Acute stage: This is where the ear is actively discharging. The mucosa of the middle ear cavity is hypertrophied, and congested.

Inactive stage: This condition is characterised by dry perfortion of ear drum, commonly in its antero inferior part, close to the eustachean tube orifice. The middle ear mucosa is normal.

Quiescent stage: Perforation of ear drum is present, the middle ear is dry and mucosa may be normal or hypertrophied.

Healed stage: Here the perforation of ear drum has healed by formation of thin scar. There may even be tympanosclerotic patches / chalky deposits on the ear drum. The ossicular chain is invariably intact.

Tuning fork tests show:

Rinne - Negative on the affected side

Weber - Lateralised to the good ear

Absolute bone conduction test - Not reduced

Pure Tone audiometry show conductive hearing loss. The hearing loss is invariably under 40 dB.

Management of tubotympanic disease:

Conservative management:

If the disease is active - with active ear discharge

Aural toileting - must be done using dry cotton swabs.

Suction method can be used to suck out secretions from the external canal and the middle ear cavity. The only disadvantage of this procedure is the risk of noise induced deafness.

Syringing the affected ear with warm saline mixed with acetic acid (1.5%) can be used to syringe the affected ear. This solution not only clears the ear of its purulent secretions, it also helps to remove crusts if present. The presence of weak acetic acid has bacteriostatic effect.

Role of antibiotics in the management of tubotympanic disease:

Antibiotics can be administered depending on the culture report. The best route of administration is topical because the presence of a large central perforation enables adequate concentration of antibiotics to reach the middle ear mucosa. Ototoxic drugs are to be avoided because the increased vascularity present in the middle ear mucosa will cause easy absorption of the drug into the inner ear fluids causing sensori neural hearing loss. Ciprofloxacillin can be administered topically.

Oral amoxycillin in adequate doses or penicillins in adequate doses may be beneficial.

Precautions:

1. The ear must be kept dry. This can be achieved by keeping the ears plugged when taking head bath. Swimming must be avoided till the perforation heals.

2. Pre existing sinus infections if any must be treated aggressively.

3. Presence of focal sepsis in the throat (tonsils commonly) must be ruled out.

Role of antihistamines and nasal decongestants: Is questionable. Their role is to decongest the nasal and naso pharyngeal mucosa, pharyngeal end of eustachean tube. Since there is associated perforation of tympanic membrane, secretions dont tend to accumulate inside the middle ear cavity. Topical nasal decongestants should not be used for more than a week, because of their propensity to cause rhinitis medicamentosa.

Surgical management:

Surgical management aims at correcting the causative problems if any.

The presence of deviated nasal septum must be corrected as this could predispose to chronic sinus infections.
If focal sepsis is identified in the tonsils and adenoid then adenotonsillectomy needs to be performed.

After eradicating the possible focal sepsis only attempt must be made to definitively treat the perforation. If the ear drum has managed to stay dry for more than 6 months myringoplasty can be performed. Temporalis fascia is used as grafting material because of its availability in close proximity, its thickness is more or less similar to that of normal ear drum. One other added advantage is its low basal metabolic rate.

If middle ear mucosa is wet and oedmatous then cortical mastoidectomy should be resorted to if conservative management fails. Mastoidectomy can always be combined with myringoplasty in the same sitting.

Atticoantral type of disease (Unsafe type of disease):

This is termed as unsafe because dangerous intra cranial and extra cranial complications can occur, proving fatal to the patient. This disease spreads by erosion of the bony wall of the attic. Cholesteatoma is commonly present in this condition. This disease is commonly seen in sclerosed mastoid cavities. Presence of granulation tissue is also common in this disorder.

This condition mainly affects the attic region of the middle ear. This region is pretty crowded, with the presence of the head of the malleus and incus. Any disease process involving crowded portions tend to cause more complications. Bone erosion occurs due to the presence of osteitic reaction in the bone tissue.

Definition of cholesteatoma: Cholesteatoma is defined as a cystic bag like structure lined by stratified squamous epithelium on a fibrous matrix. This sac contains desquamated squamous epithelium. This sac is present in the attic region. Cholesteatoma is also defined as 'skin in wrong place'. Cholesteatoma is known to contain all the layers of skin epithelium. The basal layer (germinating layer) is present on the outer surface of cholesteatoma sac in contact with the walls of the middle ear cleft.

Theories of bone invasion by cholesteatoma:

1. Pressure theory - states that increase in the pressure caused by enlarging cholesteatoma cause bone erosion. Ischemia has been attributed as the cause in this theory.

2. Enzymatic theory: Inside the cholesteatoma are present multinucleated osteoclasts and histiocytes. These cells release acid phosphatase, collagenase and other proteolytic enzymes. These enzymes are known to cause bone erosion.

3. Pyogenic osteitis: Pyogneic bacteria may release enzymes which could cause bone resorption.

Types of cholesteatoma:

1. Congenital cholesteatoma

2. Primary acquired cholesteatoma

3. Secondary acquired cholesteatoma

Congenital cholesteatoma: is known to arise from embryonic cell rests present in the middle ear cavity and temporal bone. These cell rests are known to commonly occur in cerebello pontine angle and petrous apex. Infact congenital cholesteatoma is seen as a whitish mass behind an intact tympanic membrane.

Derlacki and Clemis laid down the following as criteria to diagnose congenital cholesteatoma:

1. The patient should not have previous episodes of middle ear disease

2. Ear drum must be intact and normal

3. It is purely an incidental finding

4. If discharge and ear drum perforation is present then it should be contrued that congential cholesteatoma has managed to erode the tympanic membrane.

Clinical features:

The disorder is an incidental finding. The common location of congenital cholesteatoma is the antero superior quadrant of tympanic membrane, postero superior quadrant being the next common site of involvement. Anteriorly situated congenital cholesteatomas are known to affect the eustachean tube function causing conductive deafness due to middle ear effusion, where as posterior congenital cholesteatoma is known to cause conductive deafness due to impairment of ossicular chain mobility.

Staging of congenital cholesteatoma:

Staging as suggested by Derlacki and Clemis: They were the first to stage congenital cholesteatoma. They classified congenital cholesteatoma into

1. Petrous pyramid cholesteatoma

2. Cholesteatoma involving the mastoid cavity

3. Cholesteatoma involving the middle ear cavity.

Potsic suggested the following staging mechanism:

Stage I : Single quadrant involvement with no ossicular / mastoid involvement.

Stage II : Multiple quadrant involvement with no ossicular / mastoid involvement

Stage III : Ossicular involvement without mastoid involvement

Stage IV : Mastoid extension

Nelson's staging:

Type I : Involvement of mesotympanum without involvement of incus / stapes

Type II : Involvement of mesotympanum / attic along with erosion of ossicles without extension into the mastoid cavity

Type III : Involvement of mesotympanum with mastoid extension

Staging this disease will help in deciding the modality of treatment and in predicting the long term prognosis.

Acquired Cholesteatoma: can be divided into two types, primary acquired and secondary acquired cholesteatomas.

Primay acquired cholesteatoma: In this condition there is no history of preexisting or previous episodes of otitis media or perforation. Lesions just arise from the attic region of the middle ear.

Secondary acquired cholesteatoma: always follows active middle ear infection which manages to destroy the ear drum along with the annulus. This type of destruction is common in acute necrotising otitis media following exanthematous fevers like measles etc.

Theories to explain pathogenesis of cholesteatoma:

Various theories have been postulated to explain the pathogenesis of cholesteatoma. They are:

1. Cawthrone theory: This theory suggested by cawthrone in 1963 suggested that cholesteatoma always originated from congential embryonic cell rests present in various areas of the temporal bone.

2. Theory of immigration: This theory was suggested by Tumarkin. He was of the view that cholesteatoma was derived by immigration of squamous epithelium from the deep portion of the external auditory canal into the middle ear cleft through a marginal perforation or a total perforation of the ear drum as seen in acute necrotising otitis media.

3. Theory of invagination: This theory was suggested by Toss. He theorised that persistent negative pressure in the attic region causes invagination of pars flaccida causing a retraction pocket. This retraction pocket becomes later filled with desquamted epithelial debris which forms a nidus for the infection to occur later. Common organisms known to infect this keratin debris are Psuedomonas, E. coli, B. Proteus etc.

Toss also classified attic retraction pockets into 4 grades:

1. Grade I: The retracted pars flaccida is not in contact with the neck of the malleus.

2. Grade II: The retracted pars flaccida is in contact with the neck of the malleus to such an extent that it seems to clothe the neck of the malleus.

3. Grade III: Here inaddition to the retracted pars flaccida being in contact with the neck of the malleus there is also a limited erosion of the outer attic wall or scutum.

4. Grade IV: In this grade in addition to all the above said changes there is severe erosion of the outer attic wall or scutum.

4. Metaplastic theory: This theory was first suggested by Wendt in 1873. He took into consideration the histological changes seen in various portions of the middle ear cavity. The attic area of the middle ear cavity is lined by pavement type of epithelium. This epithelium undergoes metaplastic changes in response to subclinical infection. This metaplastic mucosa is squamous in nature there by forming a nidus for cholesteatoma formation in the attic region.

Of all the above mentioned theories, the theory of invagination appears to be the most plausible one currently explaining the various pathologic features of cholesteatoma.

Clinical features of acquired cholesteatoma:

Ear discharge: is scanty and foul smelling. Infact the odur is best described as musty in nature. This is due to the presence of saprophytic infection and osteitis.

Hearing loss: is commonly conductive in nature. Some patients may even surprisingly have a normal hearing despite the presence of a huge cholesteatoma. This normal hearing could be attributed to the bridging effects of cholesteatomatous mass.

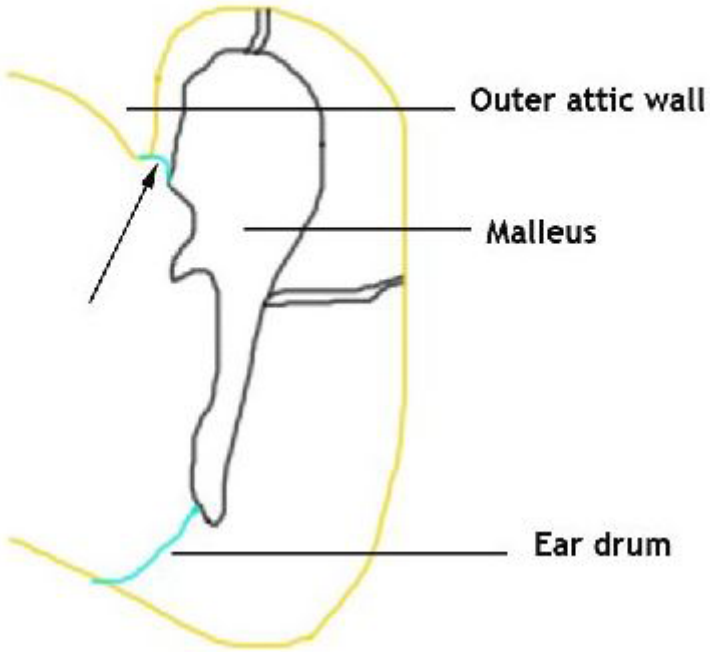
Sensorineural hearing loss if present could be attributed to the absorption of toxins through the round window membrane, or may be due to use of ototoxic antibiotics topically on a long term basis.

Ear ache: if present could be attributed to the presence of co existing otitis externa, or presence of extradural abscess.

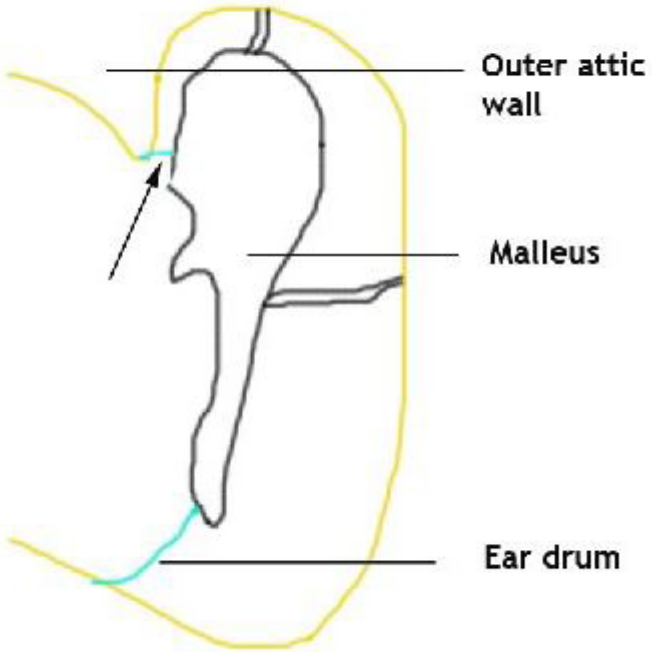
Tinnitus if present may indicate imminent sensorineural hearing loss.

Vertigo may be present if there is erosion of lateral semicircular canal by the cholesteatomatous matrix. Fistula test if performed is positive in these patient.

Grade I retraction pocket

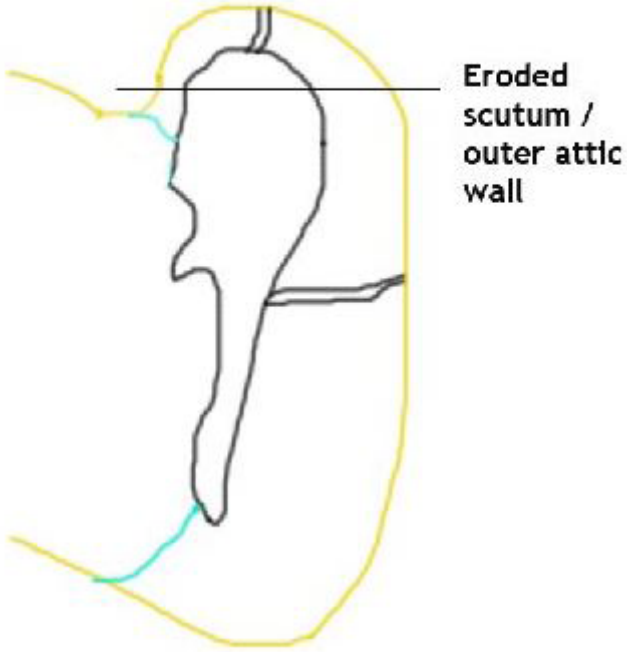


Grade II retraction pocket



Grade III retraction pocket

Grade IV retraction pocket



Fistula test: This test is positive if there is a third window is present in the laryrinth due to the erosion of the labyrinthine bone. This commonly occurs in the lateral semicircular canal area. This test is performed using a snugly fitting siegles pneumatic speculum and slowly applying pressure by compressing the pneumatic bulb. If labyrinthine fistula is present the patient will feel giddy and will have nystagmus.

Facial palsy may indicate erosion of facial nerve canal with involvement of facial nerve.

On examiantion:

There is destruction of the outer attic wall, with presence of attic perforation. Cholesteatomatous flakes may be seen through the perforation like cotton wool.

There is associated sagging of the posterior superior meatal wall.

Hearing tests indicate conductive deafness commonly if labyrinth is uninvolved. It may turn out to be sensorineural hearing loss if there is associated erosion of the labyrinth.

X ray mastoids may show slcerosis with presence of cavity.

Management:

Since this is a surgical problem modified radical mastoidectomy is advocated in almost all of these patients.

The aims of the surgical procedure is as follows:

1. To exteriorise the disease

2. To create adequate ventilation to the middle ear cavity

3. To create a permenant skin lined cavity exposed to the exterior.

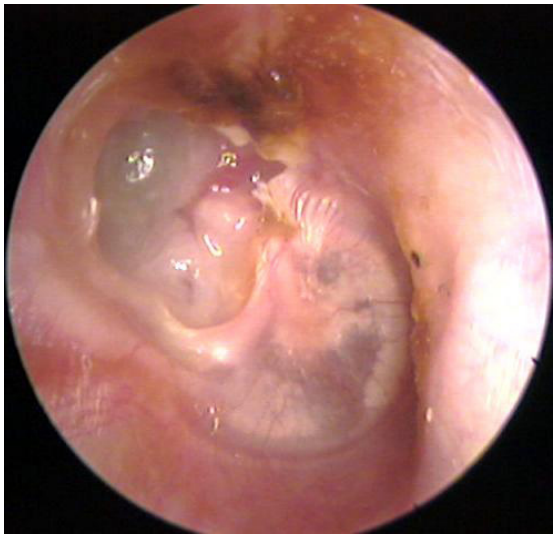


Image showing attic perforation

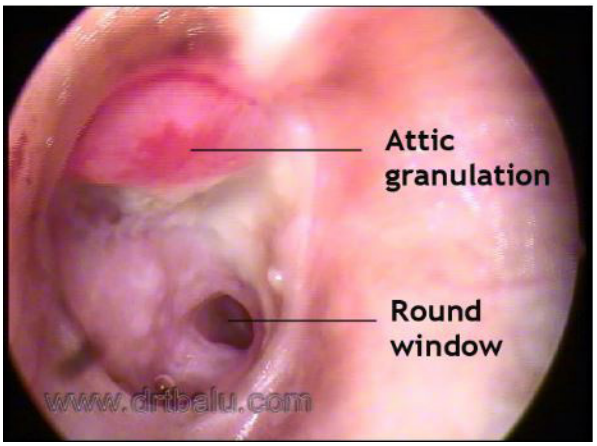


Image showing large central perforation with attic granulation

Cholesteatoma:

Definition of cholesteatoma: Cholesteatoma is defined as a cystic bag like structure lined by stratified squamous epithelium on a fibrous matrix. This sac contains desquamated squamous epithelium. This sac is present in the attic region. Cholesteatoma is also defined as 'skin in wrong place'. Cholesteatoma is known to contain all the layers of skin epithelium. The basal layer (germinating layer) is present on the outer surface of cholesteatoma sac in contact with the walls of the middle ear cleft.

Theories of bone invasion by cholesteatoma:

1. Pressure theory - states that increase in the pressure caused by enlarging cholesteatoma cause bone erosion. Ischemia has been attributed as the cause in this theory.

2. Enzymatic theory: Inside the cholesteatoma are present multinucleated osteoclasts and histiocytes. These cells release acid phosphatase, collagenase and other proteolytic enzymes. These enzymes are known to cause bone erosion.

3. Pyogenic osteitis: Pyogneic bacteria may release enzymes which could cause bone resorption.

Types of cholesteatoma:

1. Congenital cholesteatoma

2. Primary acquired cholesteatoma

3. Secondary acquired cholesteatoma

Congenital cholesteatoma:

Is known to arise from embryonic cell rests present in the middle ear cavity and temporal bone. These cell rests are known to commonly occur in cerebello pontine angle and petrous apex. Infact congenital cholesteatoma is seen as a whitish mass behind an intact tympanic membrane.

Derlacki and Clemis laid down the following as criteria to diagnose congenital cholesteatoma:

1. The patient should not have previous episodes of middle ear disease

2. Ear drum must be intact and normal

3. It is purely an incidental finding

4. If discharge and ear drum perforation is present then it should be contrued that congential cholesteatoma has managed to erode the tympanic membrane.

Clinical features:

The disorder is an incidental finding. The common location of congenital cholesteatoma is the antero superior quadrant of tympanic membrane, postero superior quadrant being the next common site of involvement. Anteriorly situated congenital cholesteatomas are known to affect the eustachean tube function causing conductive deafness due to middle ear effusion, where as posterior congenital cholesteatoma is known to cause conductive deafness due to impairment of ossicular chain mobility.

Staging of congenital cholesteatoma:

Staging as suggested by Derlacki and Clemis: They were the first to stage congenital cholesteatoma. They classified congenital cholesteatoma into

1. Petrous pyramid cholesteatoma

2. Cholesteatoma involving the mastoid cavity

3. Cholesteatoma involving the middle ear cavity.

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Congenital cholesteatoma presents very early in life as a pearly white mass behind an intact ear drum. Commonly it is seen in the anterior superior quadrant of the middle ear. As it increases in size it expands into other areas in a sequence which is almost predictable. Majority of these lesions sgtart as a spherical keratin pearl and grows like a round baloon inflating. Anterior expansion is towards the eustachean tube causing obstruction to the eustachean tube. Early sign of congenital cholesteatoma expanding anteriorly is development of otitis media secondary to eustachean tube blockage. Retraction of ear drum occurs as a result of negative pressure building up within the middle ear cavity consequent to bocked eustachean tube orifice. When the drum retracts due to negative pressure the cholesteatoma becomes easily visible as it drapes over the mass.

Inferior growth allows extension of congenital cholesteatoma towards the hypotymnpanum and the mass tends to assume more or less a spherical shape instead of assuming the irregular shape of hypotympanum.

Posterior growth is towards the handle of malleus and it generally follows the contour of the underside of the tympanic membrane developing an indendation under the handle of malleus. When the expansion crosses the handle of malleus it grows towards the incudostapedial joint and suprastructure of stapes. It then extends upwards towards towards the incudomalleolar joint, around the incus and into the attic. Ossicular destruction occurs when the cholesteatoma drapes the incudostapedial joint. It does not involve the foot plate of stapes.

Superior growth is concurrent with that of posterior growth and it extends upwards into the anterior epitympanum in front of the head of malleus. Since this space is rather crowded the cholesteatomatous mass loses its spherical shape and adapts to the contour of the anterior epitympanum.

Studies reveal that mastoid aircell system is fully developed in these patients with congenital cholesteatoma when compared to that of acquired ones where the middle ear pathology causes mastoid air cell sclerosis.

Currently acceptable classification:

Congenital cholesteatoma usually develops in the anterosuperior quadrant in young children, but does not remain there, but may extend to involve the middle ear and mastoid cavity causing varying degrees of ossicular erosion and deafness. The only structure that seems resistant to its erosion seems to be the foot plate of stapes. The current classification takes into account this spreading ability.

The sequence of spread involving three different anatomical areas form the basis of the current staging systtem, considering the fact that staging system should provide guidelines in deciding the proper treatment modality and also in determining the prognosis.

Type I lesion:
This type of lesion involves middle ear without contacting the ossicles

Type II lesion:
Involves the posterior superior quadrants of attic thereby coming into contact with the ossicles

Type III lesion:
This type of lesion involves the sites mentioned under type I and II lesions along with mastoid cavity.

As the progression from type I to type III occurs the incidence of recurrence also increases.

Type I lesions can be controlled by extended exploratory tympanotomy and do not require a second look surgery.

Type II lesions are approached by extended exploratory tympanotomy along with canal wall up tympanomastoidectomy. This procedure may be combined with facial recess approach if need arises. These lesions require second look operation as well as ossicular chain reconstruction.

Type III lesions may need the same procedure as described for type II but need canal wall down mastoidectomy also. Sound conduction mechanism needs to be reconstructed in these patients and the disease should be exteriorized using meatoplasty.

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Classification of CSOM Recent Nomenclature changes:

Chronic suppurative otitis media, its classification, and definition has undergone changes. Chronic suppurative otitis media has been rechristened to chronic otitis media. The term chronic otitis media implies a permanent abnormality of the pars tensa / pars flaccida as a result of acute otitis media, negative middle ear pressure or otitis media with effusion.
Chronic otitis media simply does not mean accumulation of pus in the middle ear cavity. It can be subdivided into active chronic otitis media (active com) and inactive chronic otitis media (inactive com).

Active COM: In this condition there is inflammation of middle ear mucosa associated with accumulation of pus. There may also be associated mastoiditis. Active COM can be subdivided into active mucosal COM and active squamosal COM.

Active mucosal COM:

Ear drum in these patients will be perforated. The middle ear mucosa may undergo polypoidal changes causing "aural polypi". It is also important to realize that inflammatory changes in this disorder is not confined to the middle ear alone, the whole of the middle ear cleft is involved. Simple closure of the perforation without removal of infected middle ear mucosa and granulations from the mastoid cavity is fraught with failure to control the disease.

Active mucosal COM is often associated with resorption of parts or whole of ossicular chain. This could be due to resorptive osteitis. The ossicles affected typically show hyperemia with proliferation of capillaries and prominent histiocytes. Long process of incus gets eroded commonly, followed by stapes crurae, body of incus and manubrium in that order.

Active squamous COM: This condition is otherwise known as unsafe ear or cholesteatoma. This condition is commonly associated with retraction of pars flaccida / tensa that has retained squamous epithelial debris. There is also associated inflammation of middle ear mucosa, production of pus, and erosion of ossicles. This condition is commonly associated with intracranial complications.

Inactive Chronic otitis media: In this condition the middle ear mucosa is relatively healthy. The mastoid cavity also appear healthy. These patients may slip into active phase rather easily because of the existing pathology. Inactive chronic otitis media can further be subdivided into Inactive mucosal chronic otitis media and Active squamous chronic otitis media.

Inactive mucosal chronic otitis media: This condition is always associated with dry perforation of the ear drum. There is permanent perforation of the pars tensa, but the middle ear and mastoid mucosa are not inflammed. The drum remnant around the perforation is always healthy. The rim of the perforation is thickened due to proliferation of fibrous tissue. Squamous epithelial cells from the external auditory canal does not migrate into the middle ear cavity in this stage because the annulus of the ear drum is intact and it prevents this migration. These patients benefit from myringoplasty.

Inactive squamous epithelial chronic otitis media: These include retraction pockets, atelectasis and epidermization. Negative middle ear pressure can cause retraction of tympanic membrane. A retraction pocket consists of an invagination into the middle ear space of part of the ear drum. These retraction pockets may be fixed when it is adherent to structures in the middle ear or free when it can move freely medially or laterally depending on the state of inflation of the middle ear. "Epidermization" is a more advanced type of retraction and it refers to replacement of middle ear mucosa by keratinizing squamous epithelium without retention of keratin debris. The area of epidermization may involve part or all of the middle ear cavity. Epidermization often remain quiescent and does not progress to cholesteatoma or active suppuration. So epidermization per se is not an indication for surgical intervention.

Healed chronic otitis media: In this stage the perforated ear drum has managed to heal itself. Loss of lamina propria or the tympanic membrane due to atrophy or failure of complete healing leads to a 'dimeric' membrane that consists of epidermis and mucosa only. Such thin membrane is more prone to retraction if there is negative middle ear pressure.

Tympanoslcerosis is also another form of healed ear drum. It refers to hyaline deposits of acellular material visible as whitish plaques in the tympanic membrane or as white nodular deposits in the submucosal layers of the middle ear on otoscopy. Tympanosclerosis is the end result of a healing process in which collagen in fibrous tissue hyalinizes, loses its structure and become fused into a homogenous mass. Calcification and ossification may occur to a variable extent.

Management of middle ear infections should be protocol driven. Eventhough the currently available protocol was devised and published in 2004 and modified in 2013 still it is considered to be relevant. As with any other protocol this can be modified according to local requirements and knowledge base.

Accumulating evidence has categorically ruled out use of antihistamines and nasal decongestants in the treatment protocol of acute otitis media.

Use of antibiotics again should be limited to more severe middle ear infections / bilateral middle ear infections in children.

Pain killers should be used to alleviate pain if it is causing too much discomfort to the child. Accompanying fever can be managed by using antipyretics (paracetomol) this too after tepid sponging.

Focus should be directed in the direction of preventing these episodes of acute middle ear infections developing in children. Accumulated evidence suggests that the following preventive steps need to be taken:

1. Compulsary breast feeding of the infant till the age of 6 months.

2. To avoid exposure to tobacco smoke exposure

3. Administration of influenza vaccine annualy to all children

4. Administration of pneumococcus conjugate vaccine to all children.

Ideal antibiotic of choice being amoxycillin in combination with clavulanic acid..

The following antibiotics should not be used in the treatment of acute otitis media:

1. Cephaliexin - It has no activity against H influenza / Morexella catarrhalis and its activity against pseudomonas aeruginosa is rather incomplete.

2. Cefaclor - It has very poor activity against H influenza / Morexella

3. Cefixime - Drug resistance is common

4. Clindamycin - Absolute zero activity against common organism causing AOM

5. Erythromycin - It has very poor activity against H influenza

Role of Myringotomy:

Should be used in very rare scenario. Indications for myringotomy include:

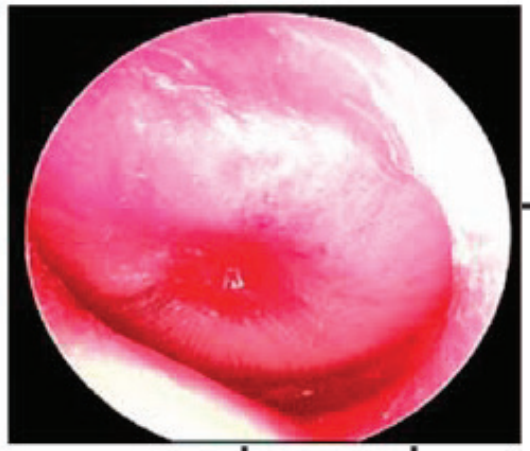
1. Otalgia persisting for more than 3 days and not releived after administration of pain killers

2. In patients suffering with recurrent episodes of otitis media atleast 3 attacks in a month or 4 attacks in a year in order to perform culture of middle ear secretions.

Acute otitis media treatment

No role for Prophylactic antibiotics even in cases of recurrent infections

No role for antihistamines / nasal decongestants



Grommet insertion
In case of recurrent otitis media
3 episodes in 6 months
or 4 episodes in a year

6 months / 24 months child If
Bilateral or
If severe and
Unilateral Antibiotics indicated.
Amoxycillin is the drug of choice

Not indicated if the child had received the same antibiotic within a span of 30 days

Febrile (Paracetomol)

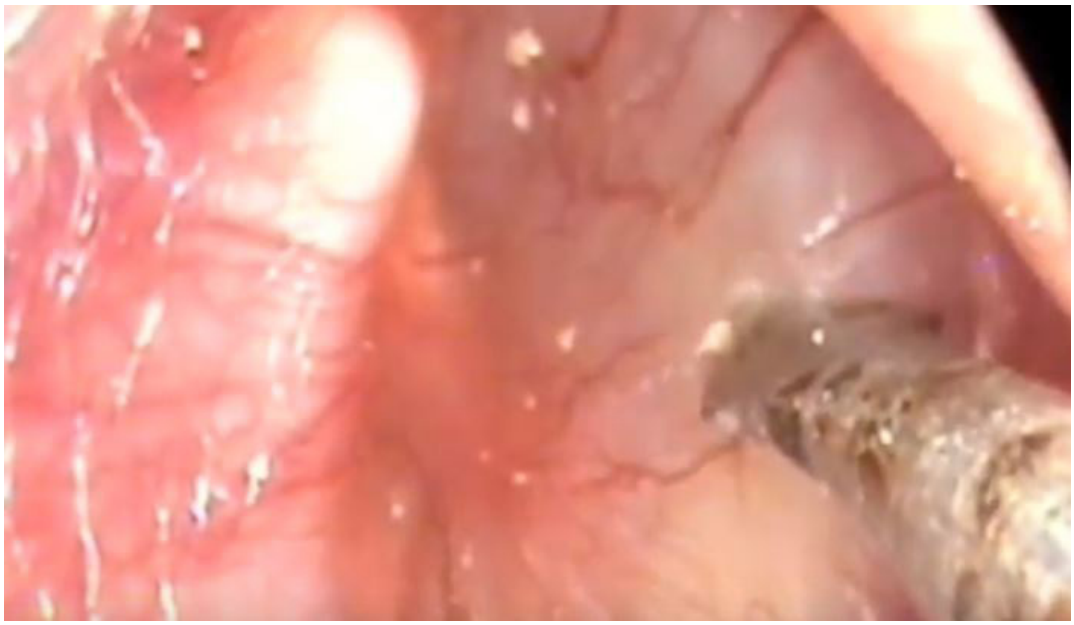
If Pain is prevalent (Pain killers are indicated) Ideally Ibuprofen

Role of Myringotomy

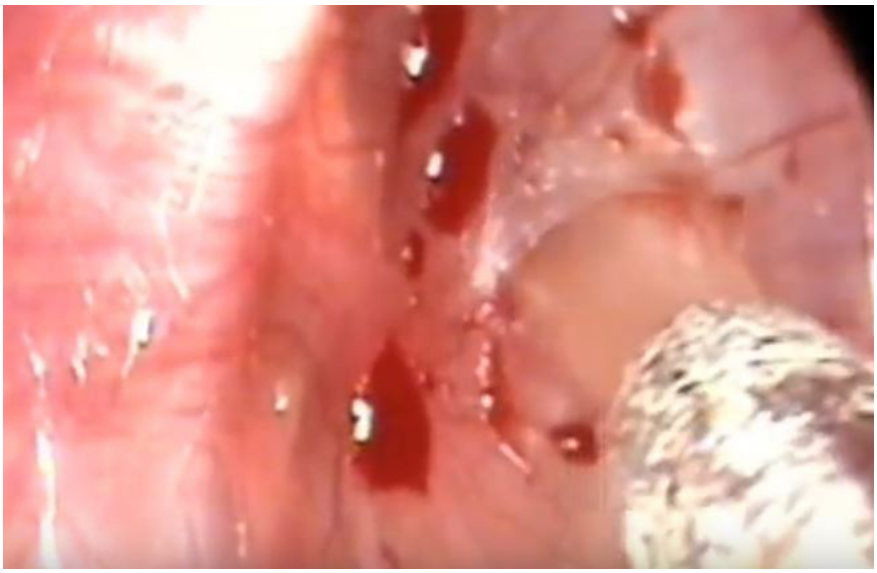
Secretions can be sent for culture and sensitivity

Incidence of complications reduced

Immediate pain relief



Myringotome seen being used to create incision in the antero inferior quadrant of ear drum



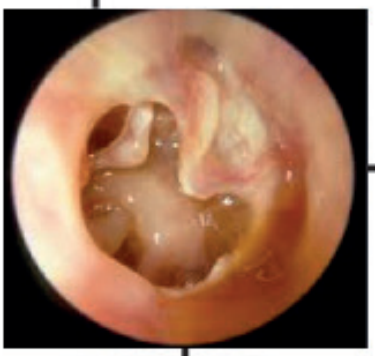
Glue seen aspirated

CSOM - Diagnosis

History - Duration of otorrhoea (more than 3 months)

Ear Pain elicited in patients with otitis externa associated with CSOM

Hard of hearing



Bacteriology:

Pseudomonas commonly isolated

Clinical features:

Central perforation
Attic perforation
Middle ear granulation
Ear discharge from middle ear
Foul smelling ear discharge (attico antral disease)

Management of CSOM:

Chronic suppurative otitis media with discharge should be managed by:

1. Regular mopping up of the secretions. This serves to keep the ear free of secretions and enables the topical antibiotics if used to act better. Mopping should be performed using cotton tipped applicators or ear buds. Patient should be cautioned about excessive mopping of the external ear as this could traumatize the skin lining of external auditory canal leading on to the formation of otitis externa.

2. Aural irrigation: Irrigation of ears with csom helps in clearing the discharge. Irrigation should always be followed by aural suction. The fluid used for irrigation should be warmed up to body temperature before being actually used. If not warmed then abnormally low or high temperature of the fluid used for irrigation may stimulate the vestibular apparatus causing unplesant sensation of giddiness to the patient. The following are some of the fluids used for irrigation purposes:

Half strength vinegar (white vinegar is used).

Half strength saline.

Half strength rubbing alcohol

Hydrogen peroxide

Half strength povidone iodine solution

Bulb syringes can be used to instill irrigation solution into the aural cavity. When bulb syringe is used the same syringe can also be used to suck out the irrigating fluid from the ear. If bulb syringe is not available then droppers can be used to instill irrigating fluid into the aural cavity.

3. Use of antibiotics

Antibiotics can be administered orally or topically.

In patients with chronic suppurative otitis media topical antibiotics are better suited because adequate concentrations of the drug can reach the target area without fear of systemic toxic effects. In addition in patients with CSOM there is certain amount of fibrosis involving the middle ear mucosa compromising the blood circulation. Systemic antibiotics will not be able to reach the target area when the vascularity is rather poor as in the case of CSOM.

Topical Quinolones are better than topical non quinolones. Topical ofloxacin is considered to be more effective than other topical preparations at the time of writing. Studies have also revealed that combining oral with topical antibiotics are not beneficial than using topical route of administration alone. Major drawback with topical antibiotic application is the propensity for the drug to cause ototoxic side effects. This is more common with gentamycin and streptomycin drops. In some countries like United kingdom these two drugs are not administered topically for the fear of their ototoxic side effects.

If CSOM is associated with otalgia and other syst emic signs like fever due to coexisting otitis externa then systemic antibiotics is preferred to topical antibiotics.

Studies have proved that use of topical antiseptics inlieu of antibiotics are also equally effective. This is more so in intractable infections involving the middle ear cavity. Some of the commonly used antiseptics include:

Boric acid
Zinc peroxide
Iodine powder
Dilute acetic acid drops
Alum acetate (Burow's solution)
Spirit ear drops containing methyl alcohol

Permanent perforation syndrome:

Patients with this type of CSOM manifest with a large central perforation without any ear discharge. This perforation does not have the ability to heal on its own because the edges of the perforation are thickened because squamous epithelium lines it. This squamous lining prevents normal healing process from proceeding any further. The middle ear mucosa in these patients dont show any sign of inflammation. Hearing loss in these patients is mild. Since there is no active infection / disharge myringoplasty alone would suffice in these patients.

In patients with persistent ear discharge inaddition to suction / mopping, topical antibiotics areas where focal infections could reside in Nose and throat needs to be addressed. Successful management of these sites of focal sepsis will render the ear dry thereby making normal healing process possible. If perforation in pars tensa is too large it is not possible for normal healing process to bridge the gap. These patients again will benefit from myringoplasty.

Persistant mastoiditis:

Patients suffering from persistent mastoiditis needs to under go mastoidectomy where the mastoid air cells are opened up and exenterated, clearing them off infection. The aditus needs to be widened to improve ventilation. These patients again will benefit from myringoplasty.

Complications of CSOM

Patients suffering from this type of infections where the squamlous epithelium has managed to migrate into the middle ear cavity are more prone for complications due to involvement of adjacent areas. The probable complications include:

1. Complications within the cranium

2. Complications within the temporal bone

Intracranial complications:

These can be further subclassified into extradural and intradural complications.

Extradural complications:

. Extradural abscess

. Meningitis

. Sigmoid sinus thrombosis

Intradural complications:

. Subdural abscess

. Brain abscess

. Otitic hydrocephalus

Intratemporal complications:

. Facial palsy

. Labyrinthitis

. Petrositis

. Subperiosteal abscess

. Internal carotid artery aneurysm

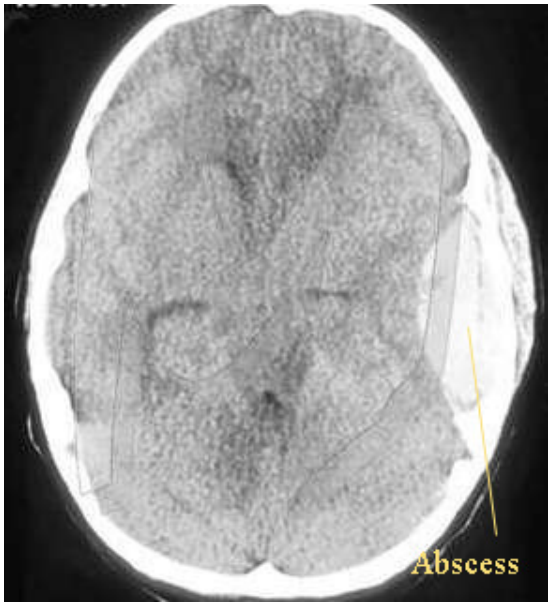
Extratemporal complications :

. Subclavian vein thrombosis

. Luc's abscess

. Citelli's abscess

. Bezold's abscess



CT image of brain showing extradural abscess

Routes of spread of infection from the ear:

Whether acute or chronic, the infection from the middle ear spreads via:

1. Extension through bone that has been demineralised during acute infections, or resorbed by cholesteatoma, or osteitis in chronic disease of the ear. Demineralisation is brought about by various enzymes that are released during the acute infections. Cholesteatoma causes bone erosion either due to pressure necrosis, or halisterisis. Halisterisis is also known as hyperimic decalcification. As the term itself suggests decalcification is caused by hyperaemia.

2. Spread through venous channels: Spreading of infected clot within small veins through the bone and dura into the dural venous sinuses. If spread via this route occurs then the infection may find its way into the brain without involving the bone or dura. Thrombophlebitis from the lateral sinus may spread to the cerebellum, and from the superior petrosal sinus may spread to the temproral lobe of the brain.

3. Spread through normal anatomical pathways: Spread may occur through oval / round windows into the internal auditory meatus. Spread may also occur through the cochlear and vestibular aqueducts. Certain areas may have dehiscent bone as a normal variant i.e. bony covering of the jugular bulb, dehiscent areas in the tegmen tympani, and dehiscent suture lines of the temporal bone.

4. Spread may occur through non anatomical bony defects like those caused due to trauma, (accident, surgical) or by erosion due to neoplasia.

5. Spread may occur through surgical defects as caused by fenestration of the oval window during stapedectomy procedures.

6. Spread may occur directly into the brain tissue through the peri arteriolar spaces of Virchow Robin. This spread does not affect the cortical arterioles perse, hence abscess occur in the white matter without the involvement of gray matter of brain.

Chronic middle ear disease cause complications by progressive and relentless erosion of the bone barriers, exposing the structures at risk to damage - the facial nerve, labyrinth and the dura. Acute infections cause early complications via the thrombophlebitis mechanism or extension through already available anatomical pathways.

Factors that determine the spread of infection:

I. Patient attributes: Patient's general condition and immunologic status play an important role in the spread of infections.

II. Bacterial attributes like the virulence of the infecting organism is also important. For example acute infections caused by Strep. pneumoniae type III, and H. Influenza type B have immense potential to spread.

III. Adequacy / Inadequacy of treatment of the middle ear condition may also play an important role.

Extradural abscess:

Is always associated with involvement of dura mater by the spreading disease, constituting pachymeningitis. This is commonly preceded by loss of bone, either through demineralisation in acute infection or erosion by cholesteatoma in chronic disease. If the cholesteatoma is non infected it may simply expose the dura without any inflammatory reaction. If cholesteatoma is infected it is associated with formation of granulation tissue over the dura. Dura is tough and resists infection. It attempts to wall off the infection, and collection of pus occur between the dura and the bone. This is known as extradural abscess and is the commonest of all intracranial complications.

A middle cranial fossa extradural mass may strip the dura from bone on the inner surface of squamous temporal bone.

Such an enlarging mass may cause increasing intracranial tension, causing focal neurological signs and papilloedema. Sometimes it could erode the skull from inside to the exterior causing a subperiosteal abscess i.e. the classic Pott's puffy tumor. Rarely an extradural abscess may develop medial to the arcuate eminence over the petrous apex. This irritates the Gasserian ganglion of the trigeminal nerve, and the 6th cranial nerve. This produces the classic Gradenigo's syndrome (includes facial pain, diplopia and aural discharge). Posterior fossa extradural abscess is limited by the attachments of the dura laterally to the sigmoid sinus. Posterior extension of this abscess around the sigmoid sinus produces the perisinus abscess. This could also extend to the neck through the jugular vein.

Clinical features:

Depends on the site of the abscess, its size, duration and rate of development. In most patients the symptoms are vague, and non specific. Sometimes it could be a incidental finding during mastoid surgery. The common complaint of the patient being headache accompanied by malaise. If the abscess communicates with the middle ear the patient may have interim relief following an episode of aural discharge.

CT scan is diagnostic. Surgery must be done as early as possible. Granulation tissue over the dura should not be disturbed because it could breach the only defence and the infection could spread to the brain.

Subdural abscess (Empyema): When spread of infection breaches the dura it exposes the subdural space to the perils of the infection. It may initially be associated with Leptomeningitis, or if the infection is contained as subdural effusions or subdural abscess. The rate of spread of the infection determines the clinical presentation. The dura is highly resistant to infection, the granulation tissue which develops on the inner side of the dura obliterates the subdural space. Initially seropurlent effusion develops in the subdural space, and eventually this becomes frankly purulent. The spread of this effusion is limited by the granulation tissue which attempts to obliterate the subdural space. The subdural pus tends to accumulate near the falx cerebri, that too particularly where it joins the tentorium cerebelli. Healing is always associated with fibrosis and obliteration of the subdural space in the area where granulation was present.

The cortical veins in the adjacent area may become involved by thrombophlebitis, this may be responsible for some of the clinical features. This may also produce multiple small abscess in the brain adjacent to the area of subdural infection. One or numerous multiloculated abscesses over the convex surface of the cerebral hemispheres may be seen. Commonly Non haemolytic streptococci have been implicated.

The subdural empyema can be suspected by the presence of headache and drowsiness. Focal neurological symptoms like irritative fits and paralysis may follow. Fits are usually of Jacksonian type, starting locally and spreading to affect one side of the body this is usually caused by cortical thrombophlebitis. Paralysis may start with one upper or lower limb and may gradually become hemiplegia. If dominant lobe is involved aphasia develops. The site of fits and the pattern of localising signs suggests the area of empyema. Papilloedema is highly uncommon, and similarly palsies involving individual cranial nerves are also rare.

Meningism may accompany headache, despite this feature this can be distinguished from meningitis by the presence of characteristic neurological localising signs. In children suspected of meningitis, subdural empyema should be considered if there is no response to treatment, or if motor seizures occur. CT scan is diagnostic. While CSF pressure may be elevated, the sugar contents are normal and the cultures are invariably sterile. In places where CT scan facilities are unavailable exploratory burr holes may be made to clinch the diagnosis.

Management: Must be done in close coordination with neurosurgeon. Massive doses of antibiotics (systemic) like penicillin and chloramphenicol must be given. The subdural abscess must be drained and the subdural space irrigated. Ear disease must be surgically treated only after the subdural empyema has been cleared or resolved. Acute ear infections may be treated with myringotomy and chronic infections can be managed with mastoidectomy. Neurosurgical management includes burr holing the skull thereby draining the abscess. Antiseizure drugs must be prescribed to supress seizures.

Lateral sinus thrombosis: Thrombophlebitis can develop in any of the veins adjacent to the middle ear cavity. Of these the lateral sinus, which comprise of the sigmoid and transverse sinuses is the largest and most commonly affected. Initially it is usually preceded by the development of an extradural perisinus abscess. The mural thrombus partly fills the sinus. The clot progressively expands and eventually occlude the lumen. The clot may later become organised, and partly broken down and may even be softened by suppuration. During this stage there is a release of infecting organism and infected material into the circulation causing bacteremia, septicemia and septic embolisation.

Extension / propagation of the thrombus upwards may extend to the confluence of the sinuses, and beyond that to the superior sagittal sinus. Invasion of the superior and inferior petrosal sinuses may cause the infection to spread to the cavernous sinus. This spread of venous thrombophlebitis into the brain substance accounts for the very high association of this complication with brain abscess. Downward progression of thrombus into and through the internal jugular vein can reach the subclavian vein.

The harmful effects are caused by the release of infective emboli into the circulation, and also from the haemodynamic disturbances caused to venous drainage from inside the cranial cavity. The use of antibiotics have greatly reduced the incidence of lateral sinus thrombosis these days.

Formerly it was commonly associated with acute otitis media in childhood; now it is commonly seen in patients with chronic ear disease. In the preantibiotic era the commonest infecting organism was beta hemolytic streptococci. This organsim was known to cause extensive destruction of red blood cells causing anaemia. Now a days the infection is by a mixed flora.

Clinical features:

The patients manifest with severe fever, wasting illness in association with middle ear infection. The fever is high and swinging in nature, when charted it gives an appearance of 'Picket fence'. It is always associated with rigors. The temperature rose rapidly from 39 - 40 degree Centigrade. Headache is a common phenomenon, associated with neck pain. The patient appear ematiated and anaemic. When the clot extended down the internal jugular vein, it will be accompanied by perivenous inflammation, with tenderness along the course of the vein. This tenderness descended down the neck along with the clot, and would be accompanied by perivenous oedema or even suppuration of the jugular lymph nodes. Perivenous inflammation around jugular foramen can cause paralysis of the lower three cranial nerves. Raised intracranial pressure produce papilloedema and visual loss. Hydrocephalus could be an added complication if the larger or the only lateral sinus is occluded by the thrombus, or if the clot reaches the superior sagittal sinus.

Extension to the cavernous sinus can occur via the superior petrosal sinus, and may cause chemosis and proptosis of one eye. If circular sinus is involved it could spread to the other eye. The propagation of the infected emboli may cause infiltrates in the lung fields, and may also spread to joints and other subcutaneous tissues.. These distant effects usually developed very late in the disease, these could be the presenting features if the disease is insiduous in onset. Masking by antibiotics could be one of the causes.

Patients always feel ill, and persisting fever is usual. The patients may have ear ache, in association with mastoid tenderness, and stiffness along the sterno-mastoid muscle. The presence of anaemia is rare now a days. Papilloedema is still a common finding. Other coexisting intracranial complications must be expected in more than 50 percent of patients.

Extension of infected clot along the internal jugular vein is always accompanied by tenderness and oedema along the course of the vein in the neck, and localised oedema over the thrombosed internal jugular vein may still be seen. One rare finding is the presence of pitting oedema over the occipital region, well behind the mastoid process, caused by clotting within a large mastoid emissary vein, this sign is known as the Griesinger's sign. Infact there is no single pathognomonic sign for lateral sinus thrombosis and a high index of suspicion is a must in diagnosing this condition.

Investigations:

A lumbar puncture must be performed, if papilloedema does not suggest that raised intracranial pressure may precipitate coning. Examination of CSFis the most efficient way of identifying meningitis. In uncomplicated lateral sinus thrombosis the white blood count in the CSF will be low when the cause is chronic middle ear disease, and somewhat raised in acute otitis media. The CSF pressure is usually normal. The variations in the level of CSF proteins and sugar are not useful.

Queckenstedt test: This is also known as Tobey - Ayer test. This is recommended whenever lumbar puncture for a possible intracranial infection is performed. The test involves measurement of the CSF pressure and observing its changes on compression of one or both internal jugular veins by fingers on the neck. In normal humans compression of each internal jugular vein in turn is followed by an increase in CSF pressure, of about 50 - 100mm above the normal level. When the pressure over the internal jugular vein is released then there is a fall in the CSF pressure of the same magnitude.

In patients with lateral sinus thrombosis pressure over the vein draining the occluded sinus cause either no increase, or a low slow rise in CSF pressure of 10 - 20 mm. Compression of the normal internal jugular vein produces a rapid pressure rise ranging from 2 - 3 times the normal level. This test is also prone for false negative results due to the presence of collateral channels draining the venous sinuses. False positives can occur if a normal lateral sinus is small or absent that creating an erroneous impression of lateral sinus thrombosis.

CT scanning: is an essential investigation in these patients. It may show filling defects within the sinus, and increased density of fresh clots. When contrast materials like Iothalamate (conray) is used failure of opacification of the affected lateral sinus may become evident. The presence of septic thrombosis shows intense inflammatory enhancement of the sinus walls and of the adjacent dura. This enhancement of the walls, but not of the contents of the sinus constitutes the empty triangle or 'delta' sign. It can also exclude accompanying complications like brain abscess and subdural empyema.

Angiography: is a definitive investigation of lateral sinus thrombosis. It helps to demonstrate the obstruction, its site and the anatomical arrangement of the veins. There is an impending risk of displacing the infected thrombus.

Arteriography: performed with radio opaque dye injected into the carotid artery can show the venous outflow during the venous phase. This can be clearly visualised in digital subtraction angiography. This technique involves precise superimposition of a negative arteriogram on a positive film of bone structures. This effectively cancels out the skeletal image thus clearly revealing the vascular pattern.

MRI: Is sufficiently diagnostic hence angiography can be avoided if MRI could be taken. Established thrombus shows increased signal intensity in both T1 and T2 weighted images. MRI can also be used to show venous flow. Gadolinum enhancement may show a delta sign comparable with that seen on CT scans

Management: Treatment involves administration of antibiotics, together with exposure of lateral sinus and incision of the sinus and removal of its contents. Anticoagulants are not advocated at present. Before exposing the lateral sinus and clearing its contents it is imperative to clear the ear of any infections by doing a cortical mastoidectomy. The involved sinus may feel firm, appear white and opaque thus suggesting occlusion of the lumen with clot. Dissemination of clot can be prevented by ligation of the affected internal jugular vein. Now a days the only indication of internal jugular vein ligation is the presence of septicemia which is resistant to antibiotics.

Meningitis:

It is also known as Leptomeningitis.(only the piamater and arachnoid are involved). This is a major and serious complication of middle ear infection. In the pre antibiotic era the sufferers invariably died. Nowadays, recovery is usual provided early diagnosis and prompt treatment is initiated.. In pre antibiotic era meningitis was a common complication of acute middle ear infections, but now it is a frequent complication of chronic middle ear disease. Childhood otogenic meningitis is commonly caused by acute middle ear infections, in adults it is commonly a complication of chronic middle ear disease. Spread to the meninges may occur via any of the dehicences in the bony barrier or preformed channels. The rate of development depends on the virulence of the organism and the resistance of the host.

Suppurative labyrinthitis can cause meningitis via access to the cerebrospinal spaces through internal auditory meatus, and through vestibular and cochlear aqueducts. Rarely rupture of brain abscess into the subarachnoid space may lead on to meningitis. Meningitis can develop within hours of the onset of acute otitis media. The organisms usually responsible to acute infection are H. Influenza type B, and Strep. pneumoniae type III. Infections from chronic ear diseases nay be caused by gram negative enteric organisms, proteus, and psuedomonas. Anaerobes and bacteriodes have also been reported.

The initial inflammatory response of the pia arachnoid to infection is an outpouring of fluid into the subarachnoid space, with a rise in CSF pressure. The CSF becomes permeated with white blood cells and rapidly multiplying bacteria. These bacteria feed on glucose present in the CSF reducing its level in CSF a characteristic finding in meningitis. Pus initially accumulates in the basal cisterns, and more rarely in the vertex. The free flow of CSF is impeded by the exudate obstructing the ventricular foramina to cause a non communicating hydrocephalus. Obstruction to CSF in the subarachnoid spaces may cause communicating hydrocephalus. Irritation of the upper cervical nerve roots by the exudate cause neck pain and neck stiffness which are the characteristic features of this condition. Exudates around the exit foramina of cranial nerves could cause nerve palsies during the late stage of the disease. Spread of infection through virchow robin spaces into the brain substance may lead to the formation of brain abscess.

Clinical features:

The most reliable clinical feature of this condition is the presence of headache and neck stiffness. At first the headache could be localised to the side of the affected ear but later it could become generalised and bursting in nature. There is also associated malaise and pyrexia. Initially neck stiffness shows resistance only to flexion, but later full rigidity or retraction may develop. During early stages the patient may have mental hyperactivity and restlessness. Tendon reflexes becomes exaggerated during this stage. Photophobia is another constant presenting feature, and the patient may be prompted to lie curled up away from the light. Vomiting projectile in nature is another important feature. As the condition worsens the symptoms also become progressively severe. When neck stiffness is marked the patient may manifest poitive kernigs sign. The stiffness may become more severe enough to cause opisthotonus.

Brudzinski's sign:

Brudzinski's sign is involuntary lifting of the legs in meningeal irritation when lifting a patient's head. Kernig's sign is resistance and pain when knee is extended with hips fully flexed. Patients may also show opisthotonus; spasm of the whole body that leads to legs and head being bent back and body bowed forward.

Diagnosis:

Is made by the examination of CSF. Any patient with suspected menigitis must undergo lumbar puncture. The CSF analysis show increased white cells and reduced glucose levels from 1.7-3 mmol/l to 0.. Chloride content may fall from 120 mmol/l to 80mmol/l. Bacteria may also be isolated from the CSF. Recently polymerase chain reaction have been used to detect bacterial DNA from CSF.

Management:

The mainstay in the medical management is large doses of systemic antibiotics. Penicillin is the drug of choice. Streptomycin may also be used as an adjunct. Chloramphenicol may also be used. Ceftrioxine a third generation cephalosporin is widely used these days in the treatment of meningitis. This has a broad spectrum activity. Metronidazole is also used because of its usefulness in treating anaerobes.

After the patient recovers from the acute problem, effort must be made to remove the middle ear pathology which was the cause for this problem. In chronic middle ear infections modified radical mastoidectomy is the procedure of choice, in acute middle ear infections cortical mastoidectomy is the preferred surgical procedure.

Brain abscess:

Otogenic brain abscess always develop in the temporal lobe or the cerebellum of the same side of the infected ear. Temporal lobe abscess is twice as common as cerebellar abscess. In children nearly 25% of brain abscesses are otogenic in nature, whereas in adults who are more prone to chronic ear infections the percentage rises to 50%.

The routes of spread of infection has already been discussed above, the commonest being the direct extension through the eroded tegment plate. Although dura is highly resistant to infection, local pachymeningitis may be followed by thrombophlebitis penetrating the cerebral cortex, sometimes the infection could extent via the Virchow - Robin spaces in to the cerebral white matter. Cerebellar abscess is usually preceded by thrombosis of lateral sinus. Abscess in the cerebellum may involve the lateral lobe of the cerebellum, and it may be adherent to the lateral sinus or to a patch of dura underneath the Trautmann's triangle.

Stages of formation of brain abscess:

Stage of cerebral oedema: This is infact the first stage of brain abscess formation. It starts with an area of cerebral oedema and encephalitis. This oedema increases in size with spreading encephalitis.

Walling off of infection by formation of capsule: Brain attempts to wall off the infected area with the formation of fibrous capsule. This formation of fibrous tissue is dependent on microglial and blood vessel mesodermal response to the inflammatory process. This stage is highly variable. Normally it takes 2 to 3 weeks for this process to be completed.

Liquefaction necrosis: Infected brain within the capsule undergoes liquefactive necrosis with eventual formation of pus. Accumulation of pus cause enlargement of the abscess.

Stage of rupture: Enlargement of the abscess eventually leads to rupture of the capsule containing the abscess and this material finds its way into the cerebrospinal fluid as shown in the above diagram.

Cerebellar abscess which occupy the posterior fossa cause raised intra cranial tension earlier than those above the tentorium. This rapidly raising intra cranial pressure cause coning or impaction of the flocculus or brain stem into the foramen magnum. Coning produces impending death.

If the walling off process (development of capsule) is slow, softening of brain around the developing abscess may allow spread of infection into relatively avascular white matter, leading to the formation of seconday abscesses separate from the original or connected to the original by a common stalk. This is how multilocular abscesses are formed. Eventually the abscess may rupture into the ventricular system or subarachnoid space, causing meningitis and death.

The mortality rate of brain abscess is around 40%, early diagnosis after the advent of CT scan has improved the prognosis of this disease considerably..

The bacteriological flora is usually a mixture of aerobes and obligate anaerobes. Anaerobic streptococci are the commonest organisms involved. Pyogenic staphylococci is common in children. Gram negative organims like proteus, E coli and Pseudomonas have also been isolated.

Clinical features:

The earliest stage where the brain tissue is invaded (stage of encephalitis) is marked by the presence of headache, fever, malaise and vomiting. Drowsiness eventually follow. These early features may be masked by the complications such as meningitis or lateral sinus thrombosis. If this stage progresses rapidly to generalised encephalitis before it could be contained by the formation of the capsule, drowsiness may progress to stupor and coma followed by death.. Usually the period of local encephalitis is followed by a latent period during which the pus becomes contained within the developing fibrous capsule. During this latent phase the patient may be asymptomatic.

During the next state (stage of expansion) the enlarging abscess first cause clinical features due to the alteration of CSF dynamics, and site specific features may also be seen due to focal neurological impairement. The pulse rate slows with rising intracranial pressure, the temperature may fall to subnormal levels. Drowsiness may alternate with periods of irritability. Papilloedema is also found due to elevated CSF pressure.

Clinical features also vary according to the site of involvement. Hence the differences that are seen between the cerebral and cerebellar abscess.

Cerebral (Temporo sphenoidal abscess):

A cerebral abscess in the dominant hemisphere often cause nominal aphasia, where in the patient has difficulty in naming the objects which are in day to day use. He clearly knows the function of these objects. Visual field defects arise from the involvement of optic radiations. Commonly there is quadrantic homonymous hemianopia, affecting the upper part of the temporal visual fields, more rarely it may also involve the lower quadrants. The visual field loss are on the side opposite to that of the lesion. This can be assessed by confrontation method. Upward development affects facial movements on the opposite side, and then progressively paralysis of the upper and lower limbs. If the expansion occur in inward direction then paralysis first affects the leg, then arm and finally the face.

Cerebellar abscess:

The focal features associated with cerebellar abscess is weakness and muscle incoordination on the same side of the lesion. Ataxia causes the patient to fall towards the side of the lesion. Patient may also manifest intention tremors which may become manifest by the finger nose test. This test is performed by asking the patient to touch the tip of the nose with the index finger first with the eyes open and then with the eyes closed. The patient may often overshoot the mark when attempted with the eyes closed in case of cerebellar abscess. The patient may also have spontaneous nystagmus. Dysdiadokinesis is also positive in these patients.

Investigations:

CT scan and MRI scans are the present modes of investigation. Scan is ideally performed using contrast media. These scans not only reveal the position and size of the abscess, the presence of localised encephalitis can be distinguished from that of an encapsulated abscess. Associated conditions such as subdural abscess, and lateral sinus thrombosis can also be seen.

Lumbar puncture:

Is frought with danger because of the risk of coning. Lumbar puncture must be performed in these patients only in a neurosurgical unit where immediate intervention is possible if coning occurs.

Treatment: involves use of large doses of antibiotics. Ideally the abscess should be controlled neurosurgically and with antibiotics. After the patient recovers mastoidectomy is performed to remove the focus of infection. Abscess can be drained by placement of burr holes, and excision of the necrotic tissue along with the capsule.

Otitic hydrocephalus:

Is one of the common complication of middle ear infection. It is a syndrome of raised intracranial pressure during or following middle ear infection. This condition is also known as Pseudotumor cerebri.

Pathogenesis:

The aetiology is unknown. The relationship of this condition with that of lateral sinus thrombosis has been documented. The inference is that obstruction of the lateral sinus affects cerebral venous outflow, or the extension of the thrombus into the superior sagittal sinus impedes CSF resorption by pacchionian bodies.

Clinical features:

The leading symptoms are

1. headache

2. drowsiness

3. blurred vision

4. nausea

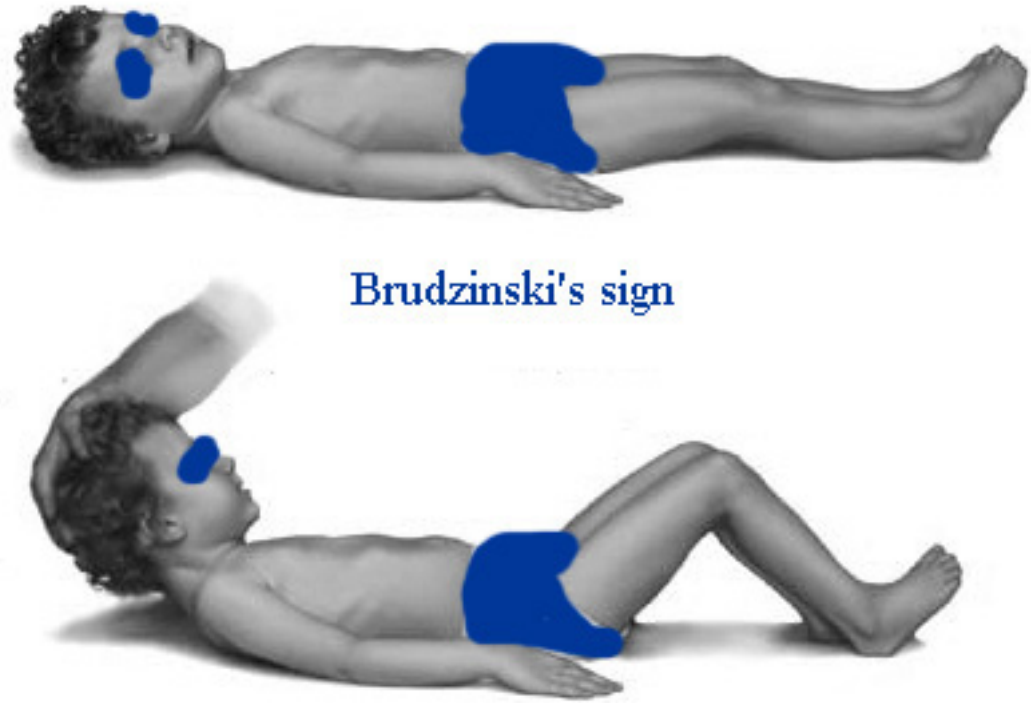
5. vomiting

6. diplopia (rarely)

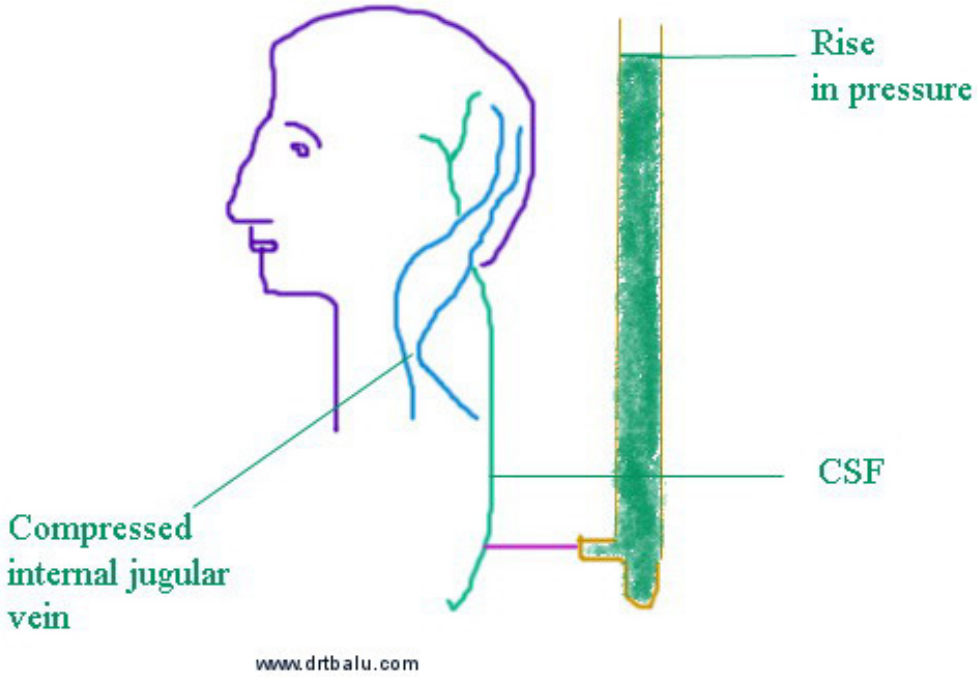
The onset may occur many weeks after acute otitis media, or many years after the start of the chronic middle ear disease. Clincial examination may show papilloedema. Lateral rectus palsy on one or both sides are also commonly seen. This occur due to the stretching of the 6th nerve due to increased intracranial pressure. CT scan is diagnostic.

Treatment:

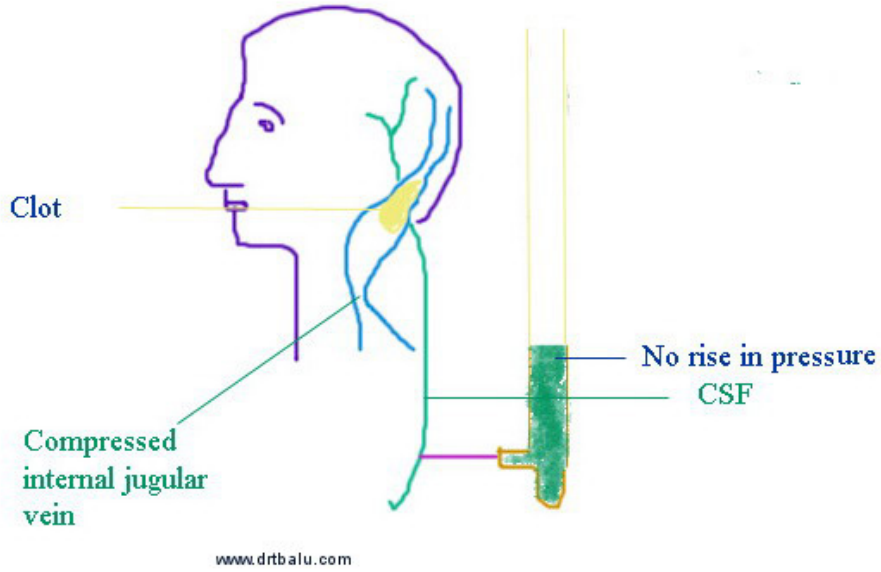
Revolves around the management of the elevated intra cranial tension. It includes use of steroids, diuretics and hyperosmolar dehydrating agents. Repeated lumbar punctures may also be used to reduce the tension. Surgical clearance of the infection of the middle ear should also follow.



Evolution of brain abscess



Negative Tobey Ayyar test



Positive Tobey Ayyar test

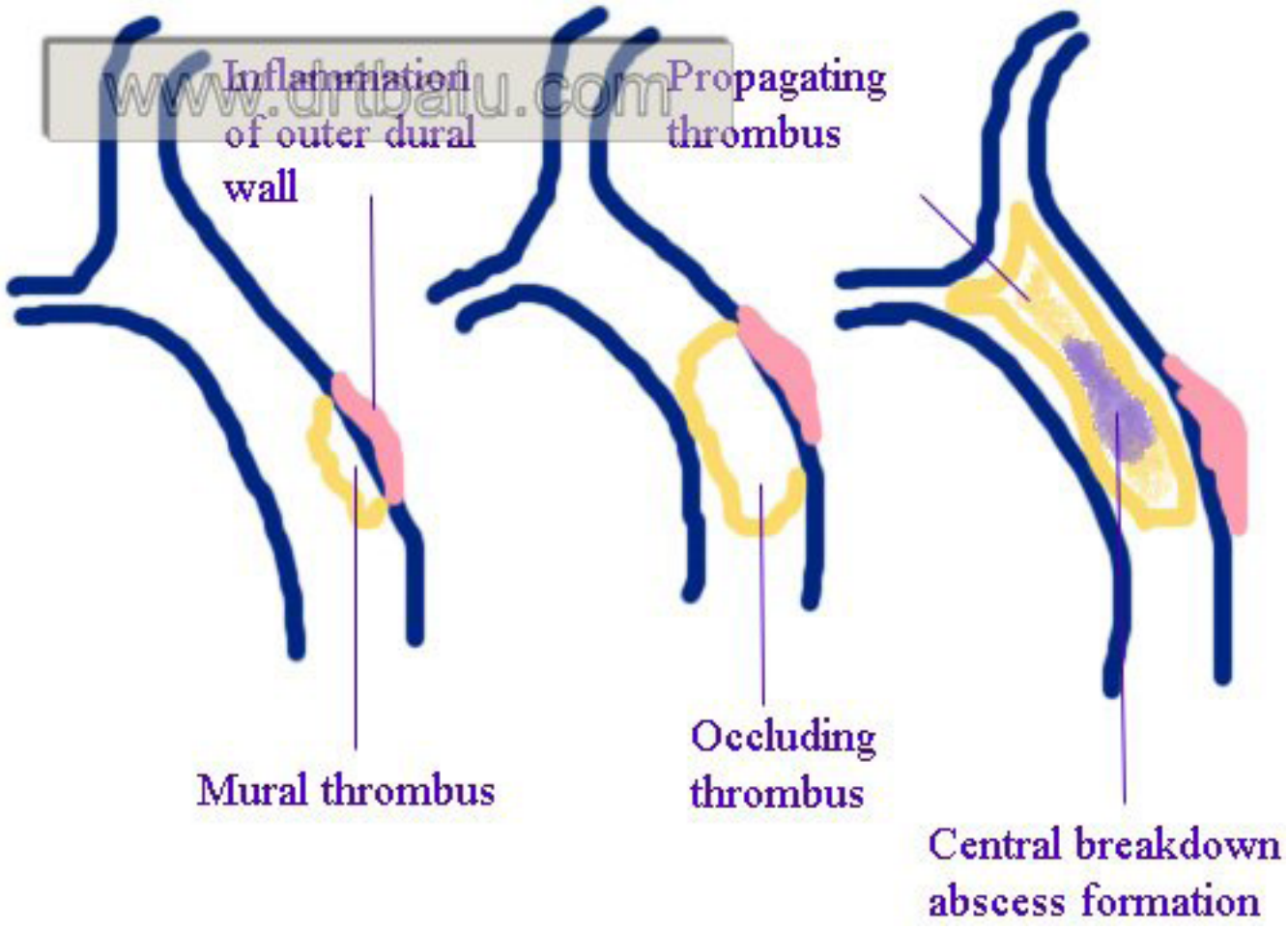


Figure showing various stages of lateral sinus thrombosis

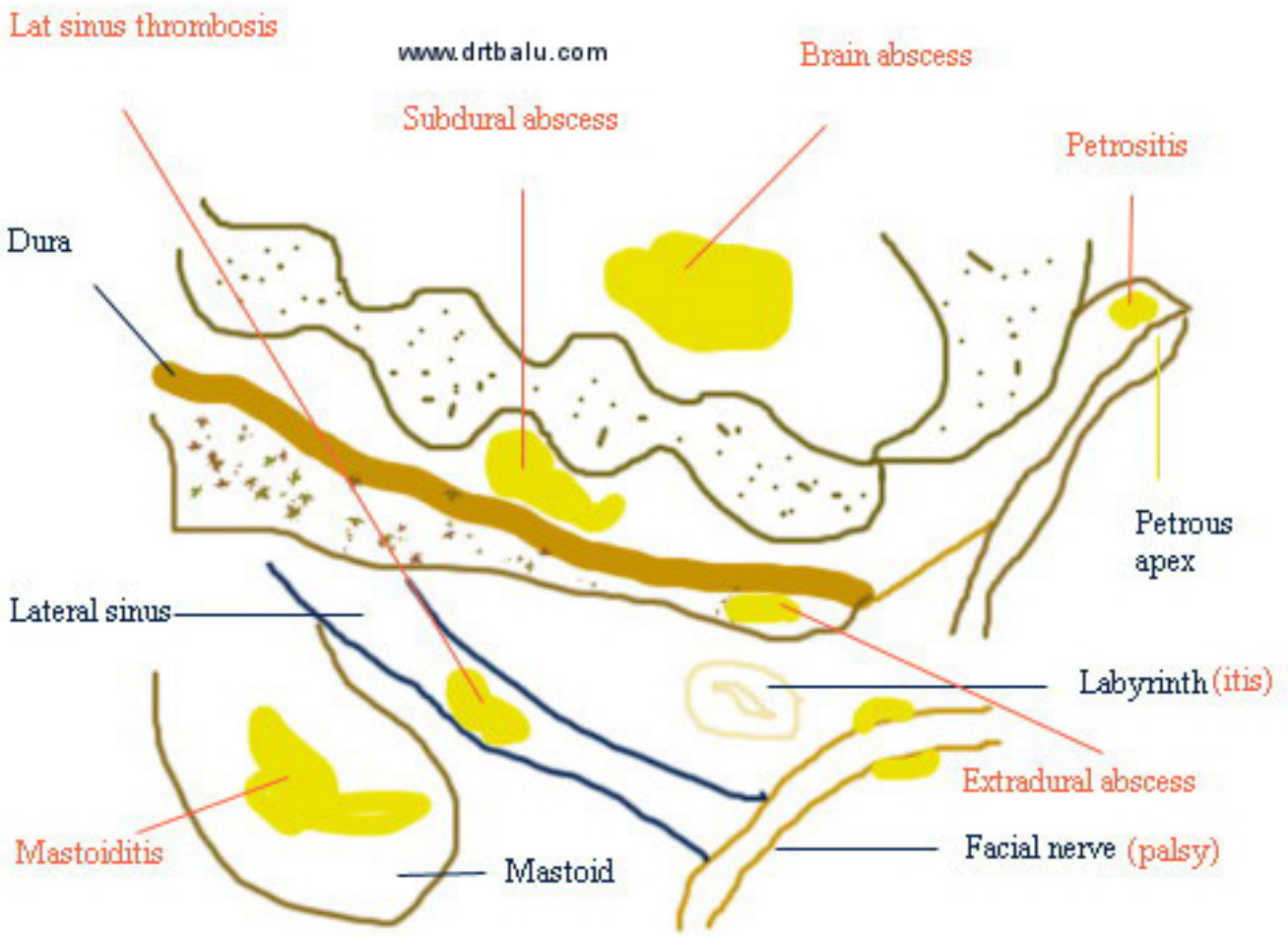


Figure showing various complications following otitis media

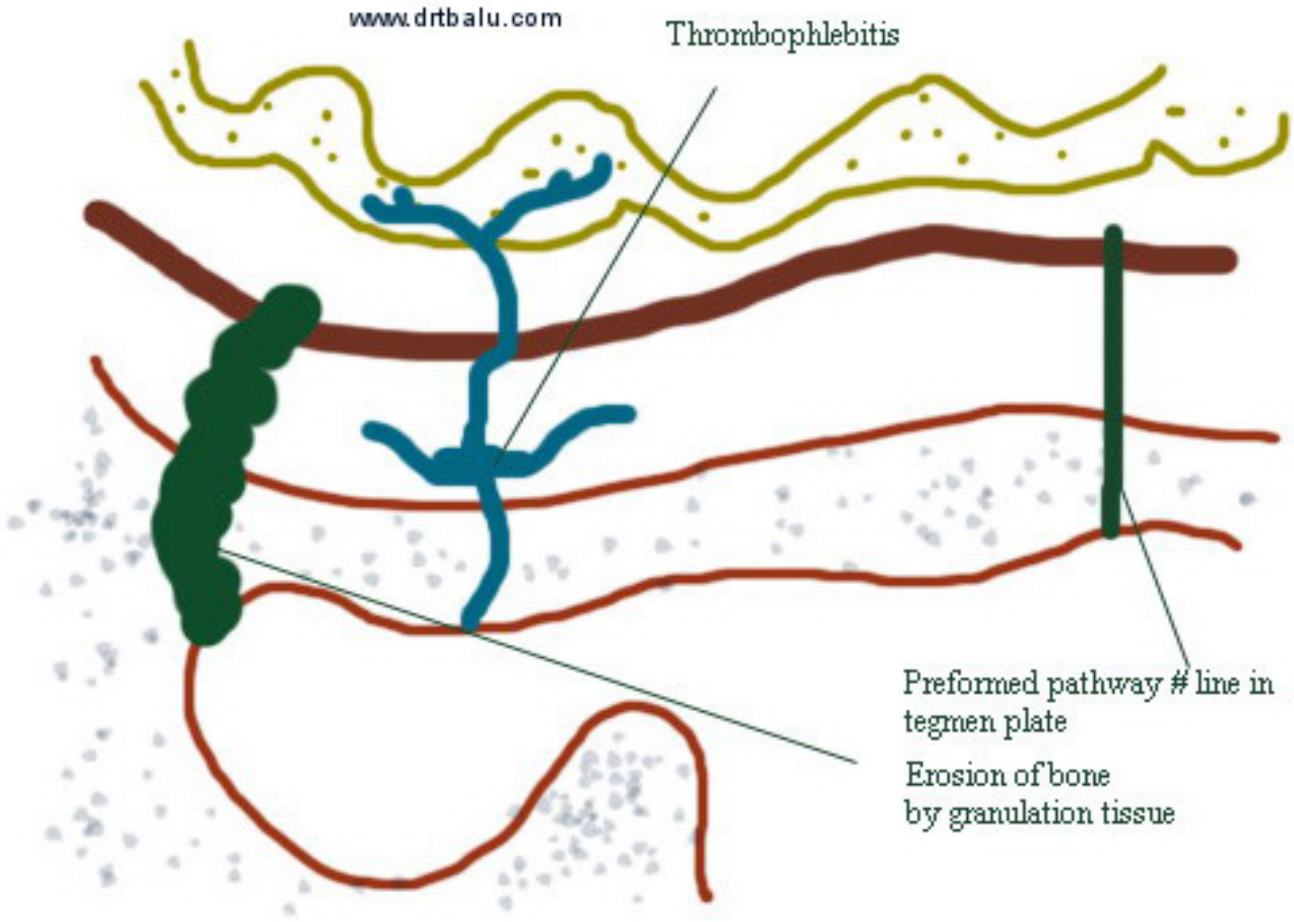


Diagram showing various route of spread of middle ear infections

Myringoplasty

Synonyms: Myringoplasty, Tympanoplasty.

Definition: Myringoplasty is a procedure used to seal a perforated tympanic membrane using a graft material.
Temporalis fascia is the commonly used graft material because:
1. It is an autograft with excellent chance of take
2. It is available close to the site of operation making its harvest easier
3. It has a low basal metabolic rate, brightening its success rate
4. Its thickness is more or less similar to that of tympanic membrane
There are two available methods of performing myringoplasty:
Overlay technique
Under lay technique
Overlay technique: This is a difficult technique to master. Here the graft material is inserted under the squamous (skinlayer) of the ear drum. It is a difficult task peeling only the skin layer away from the tympanic membrane, placing the graft over the perforation and redraping the skin layer.
Underlay technique: This is a simpler and commonly used technique. Here the graft is placed under the tympano meatal flap which has been elevated hence the name under lay. The major advantage of this procedure is that it is easy to perform with a good success rate.

Indications of Myringoplasty:

1. Central perforation which has been dry atleast for a period of 6 weeks.
2. As a follow up to mastoidectomy procedure to recreate the hearing mechanism

Prerequisites for myringoplasty:

1. Central perforation which has been dry for atleast 6 weeks
2. Normal middle ear mucosa
3. Intact ossicular chain
4. Good cochlear reserve

Procedure:

Firstly a temporalis fascia of adequate site must be harvested and allowed to dry.
The surgery is performed under local anesthesia. Temporalis fascia graft is harvested under local anesthesia conventionally and allowed to dry. The external auditory canal is then anesthetised using 2 % xylocaine mixed with 1 in 10,000 adrenaline injection. About 1/2 cc is infiltrated at 3 - o clock, 6 - o clock, 9 - o clock, and 12 - o clock positions about 3mm from the annulus. The patient is premedicated with intramuscular injections of 1 ampule fortwin and 1 ampule phenergan.

Step I:

Freshening the margins of perforation - In this step the margins of the perforation is freshened using a sickle knife of an angled pick. This step is very important because it breaks the adhesions formed between the squamous margin of the ear drum (outer layer) with that of the middle ear mucosa. These adhesions if left undisturbed will hinder the take up of the neo tympanic graft. This procedure will infact widen the already present perforation. There is nothing to be alarmed about it.

Step II: This step is otherwise known as elevation of tympano meatal flap. Using a drum knife a curvilinear incision is made about 3 mm lateral to the annulus. This incision ideally extends between the 12 - o clock, 3 - o clock, and 6 - o clock positions in the left ear, and 12 - o clock, 9 - o clock and 6 - o clock positions in the right ear. The skin is slowly elevated away from the bone of the external canal. Pressure should be applied to the bone while elevation. This serves two purposes:
1. It prevents excessive bleeding
2. It prevents tearing of the flap
This step ends when the skin flap is raised up to the level of the annulus.

Step III: Elevation of the annulus and incising the middle ear mucosa. In this step the annulus is gradually lifted from its rim. As soon as the annulus is elevated a sickle knife is used to incise the middle ear mucosal attachement with the tympano meatal flap. This is a very important step because the inner layer of the remnant ear drum is continuous with the middle ear mucosa. As soon as the middle ear mucosa is raised, the flap is pushed anteriorly till the handle of the malleus becomes visible.

Step IV: Freeing the tympano meatal flap from the handle of malleus. In this step the tymano meatal flap is freed from the handle of malleus by sharp dissection of the middle ear mucosa. Sometimes the handle of the malleus may be turned inwards hitching against the promontory. In this scenario, an attempt is made to lateralise the handle of the malleus. If it is not possible to lateralise the handle of the malleus, the small deviated tip portion of the handle can be clipped. The handle of the malleus is freshened and stripped of its mucosal covering.

Step V: Placement of graft (underlay technique). Now a properly dried temporalis fascia graft of appropriate size is introduced through the ear canal. The graft is gently pushed under the tympano meatal flap which has been elevated. The graft is insinuated under the handle of malleus. The tympano meatal flap is repositioned in such a way that it covers the free edge of the graft which has been introduced. Bits of gelfoam is placed around the edges of the raised flap.

One gel foam bit is placed over the sealed perforation. This gelfoam has a specific role to play. Due to the suction effect created it pulls the graft against the edges of the perforation thus perventing medialisation of the graft material.

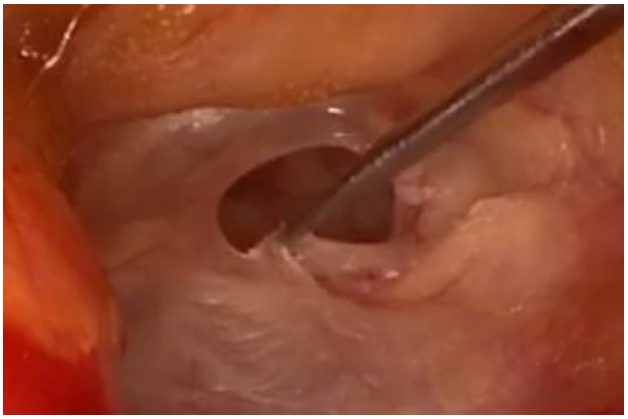


Figure showing eduges of perforation being freshened using a pick

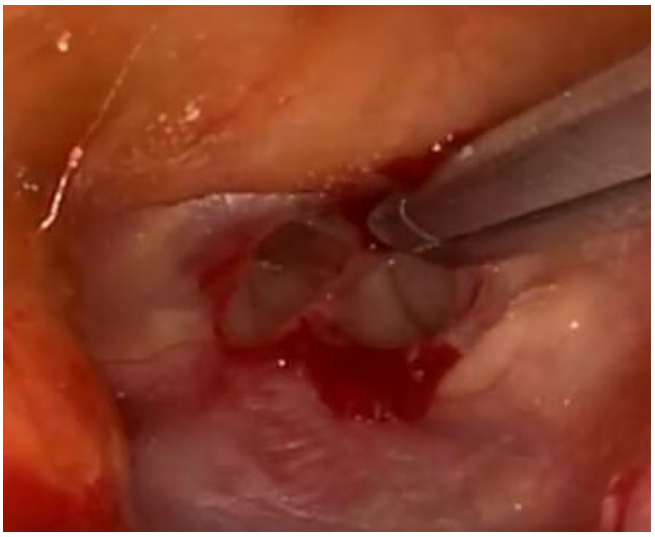
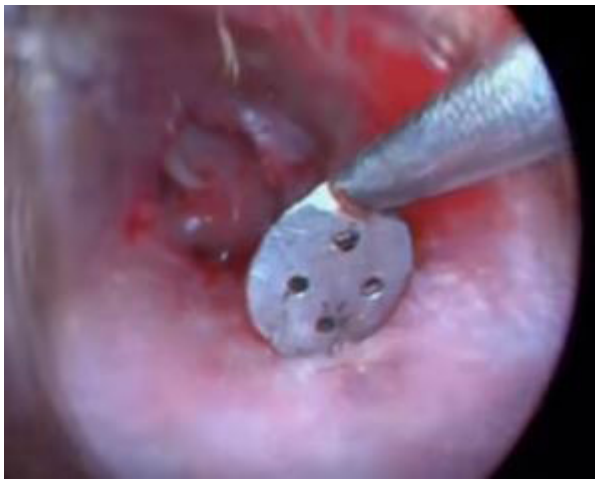


Figure showing tissue from the edge of perforation removed using forceps



Rosen's knife as seen while incision is being given for raising a tympano meatal flap

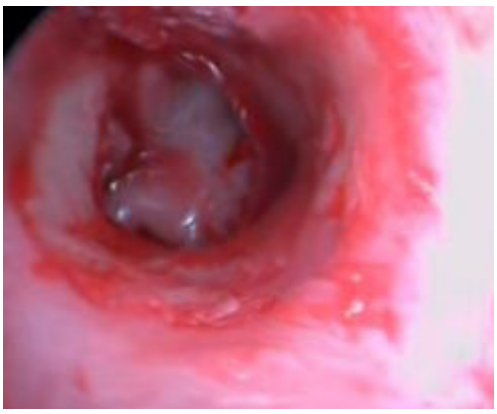
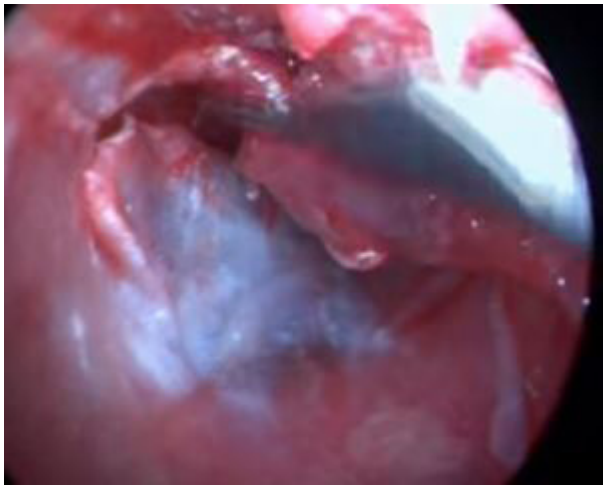
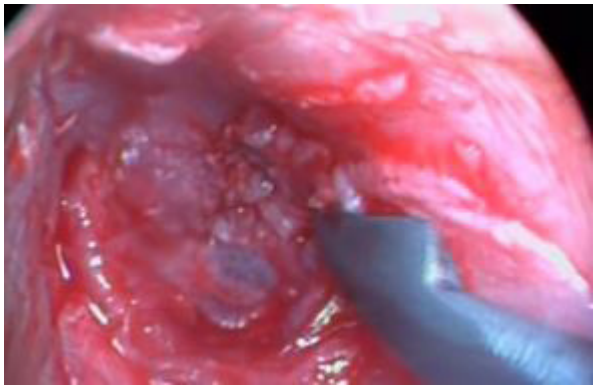


Figure showing tympanomeatal flap elevated and displaced antero superiorly



Temporalis fascia graft being inserted under the tympano meatal flap



TM flap being repositioned over the graft

Fat Myringoplasty:

In fat myringoplasty the graft material used is fat. ideally fat can be harvested from ear lobule or rarely abdomen. This surgical process depends on the fact that fat provides the base over which the healing epithelium would migrate. The graft used should ideally provide a matrix over which angiogenesis could take place and ultimately epithelial cells could cover that area. The source of neo epithelium is usually the basal cells of the squamous layer lining both the external canal skin and the tympanic membrane.

Suitability of fat as a graft material:

The effectiveness of fat as a useful graft material was extensively studied by Ringenberg. His classic study involved use of fat from three locations i.e.
Ear lobule
Abdomen
Buttock

Ringenberg concluded that among fat harvested from the three sites ear lobule fat was the best for myringoplasty as it was found to be the densest and more compact containing more number of fat cells. Fat is known to promote revascularization of even non vascular areas.

Criteria for using fat as a graft material:

1. Perforations involving pars tensa not more than 5 mm.
2. Non marginally located perforations not exposing the handle of malleus
3. Absence of calcific plaques or atrophic areas adjacent to the perforation
4. Absence of acute inflammation
5. No evidence of cholesteatoma
6. Absence of significant eustachean tube dysfunction
7. No ossicular reconstruction is planned

Use of Hyaluronic acid to the fat graft has shown to increase the success rate.
Advantages of use of hyaluronic acid along with fat graft include:

1. It prevents dehydration of perforation margins

2. It promotes centripetal migration of epithelial cell layer over the fat support

3. Itis safe to use and biodegradable. It tends to dissolve completely within 2 months of use

4. For fat hyaluronidase combination to be successful the margins of perforation should be clearly visible. Even if one edge is not visibible then this technique should not be used.

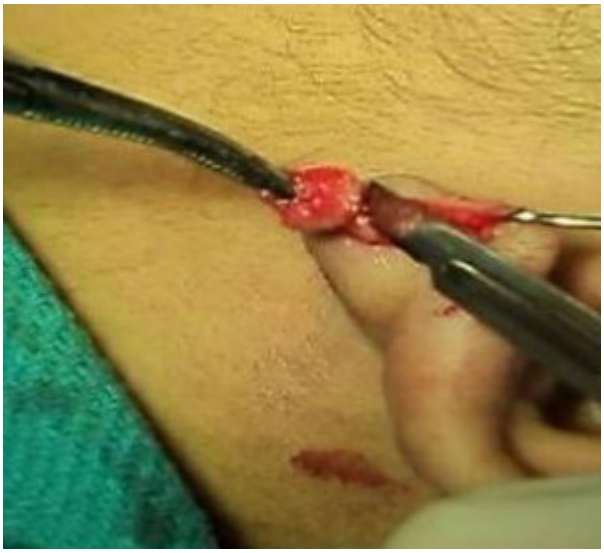


Figure showing ear lobule fat harvested

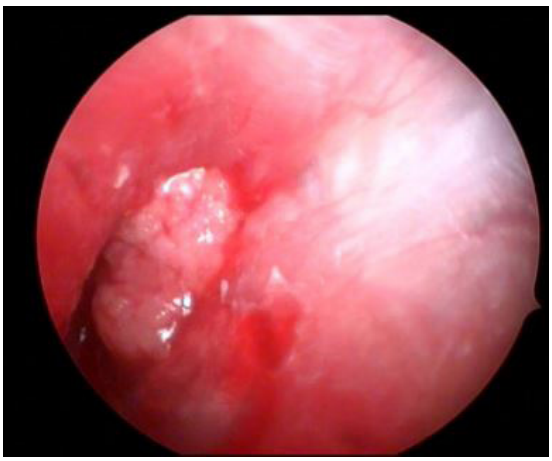


Image showing fat filling the perforation

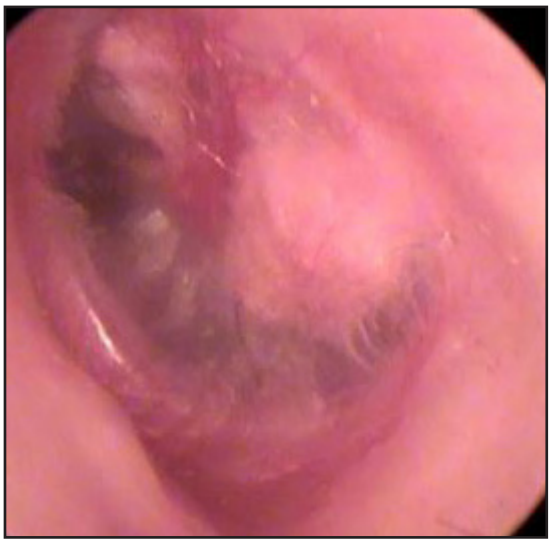


Image showing graft position on the 31st post operative day

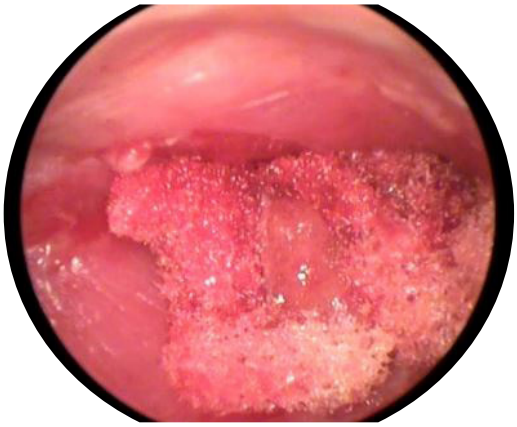


Image showing gelfoam placed over the fat graft as support

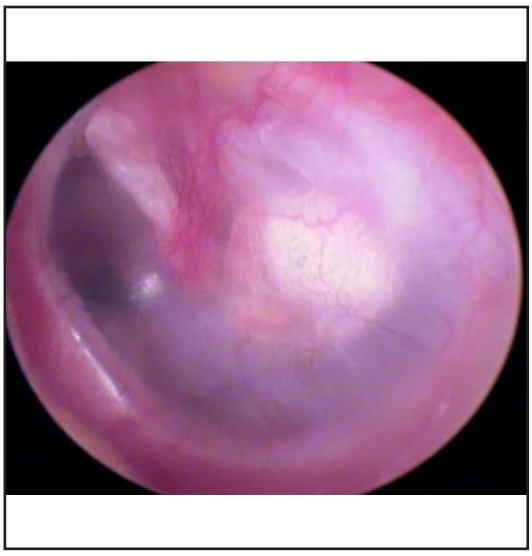


Image showing grafted TM using fat 60th Post operative day

An ideal myringoplasty technique should:

1. Restore sound protection to the round window

2. Restore the vibratory area of ear drum, this in turn improves hearing and reduces tinnitus

3. Restore sound pressure transmission mechanism to oval window via the ossicular chain

4. Reduce the susceptibility of infections of middle ear mucosa from microbes from external auditory canal

Cartilage Myringoplasty:

Myringoplasty in patients with:

1. Poor eustachean tube function

2. Adhesive otitis media

3. Total perforation involving the ear drum

4. Anterior quadrant perforations

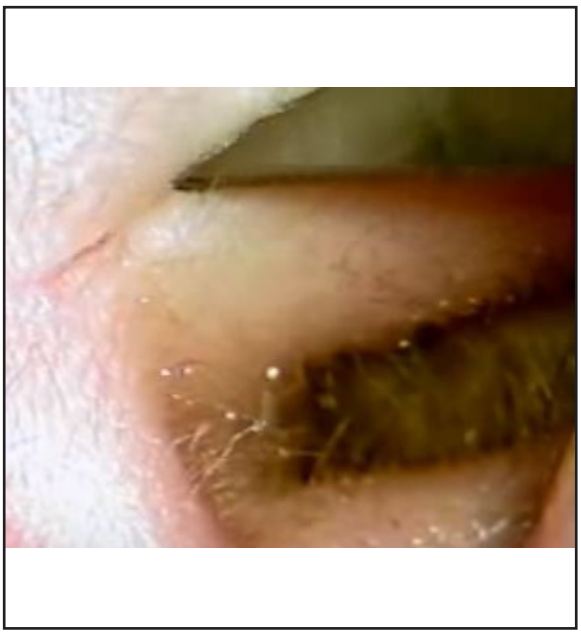
are a problem. Conventional techniques involving use of temporalis fascia as grafting material are bound to fail in these patients. Use of cartilage - perichondrium composite graft is an excellent alternative. This composite graft material provides stability against retraction.

This procedure can be carried out under local anesthesia. As a first step the edges of perforation is freshened using an angled pick.

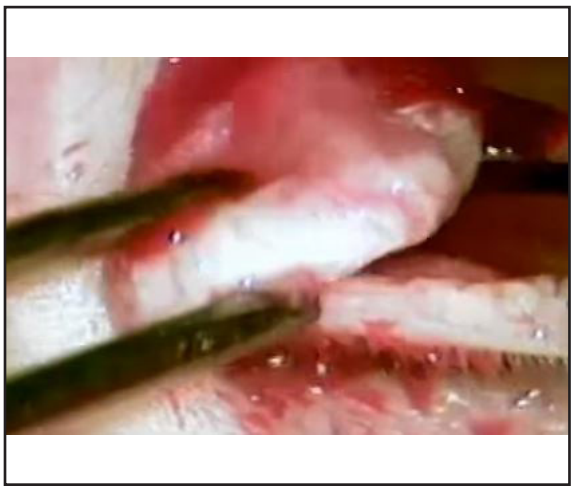
Harvesting tragal cartilage along with its perichondrium:

This is the next important step. A 4-5 mm incision should be sited about 3 mm medial to the tragal cartilage free border incising the skin and the cartilage. On the opposite side perichondrium is left attached to the cartilage. A 3 - 4 mm sized cartilage is harvested with perichondrium intact on one side while perichondrium from the opposite side is stripped. The site of the incision is sutured with an absorbable suture material.

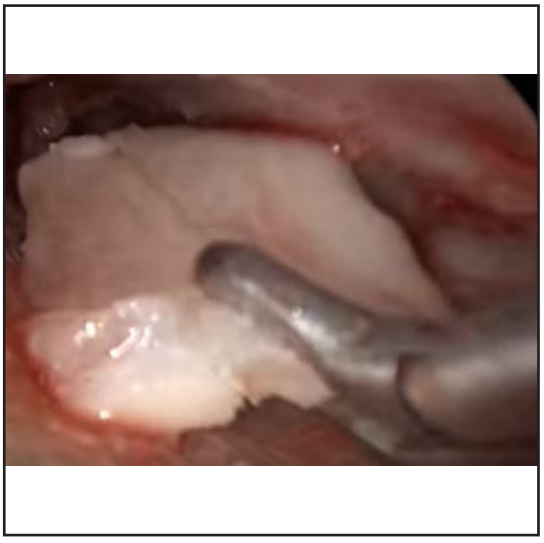
The ear canal side of conchal cartilage is concave and hence this side is placed facing the middle ear cavity. Under endoscopic vision the middle ear cavity is packed with gelfoam up to the level of the edges of the perforation. The tragal cartilage along with its perchondrial lining is not thinned but trimmed to a size just larger than that of the perforation and is inserted by pushing it under the perforation. It is held in place by its underlying gelfoam pack. Gelfoam is also placed on the outer surface of cartilage perichondrium composite graft.



Tragal cartilage being harvested



Tragal cartilage harvest completed



Cartilage being inserted

Mastoidectomy

Definition: Mastoidectomy is defined as a surgical procedure which opens up the mastoid cavity, cleans up the infected air cells, improves middle ear ventilation by widening of the aditus.

Indications of mastoidectomy:

1. Chronic mastoiditis not responding to conventional medical treatment

2. Chronic suppurative otitis media with cholesteatoma

3. Chronic suppurative otitis media not responding to medical management

4. As a preliminary step to other surgical procedures like:

a. Cochlear implants

b. Facial nerve decompression

c. Labyrinthectomy

d. Endolymphatic sac decompression

5. Subperiosteal abscess

6. Malignant lesions of middle ear

7. Benign tumors of middl ear i.e. Glomus jugulare

Types of mastoid surgeries:

1. Cortical mastoidectomy

2. Modified radical mastoidectomy

3. Radical mastoidectomy

Currently mastoidectomy surgeries are classified under two broad headings:

1. Canal wall up mastoidectomy

2. Canal wall down mastoidectomy

This classification depends on whether posterior canal wall is left intact or lowered.

Types of canal wall up procedures:

1. Simple cortical mastoidectomy (Schwartze's)

2. Classic canal wall up mastoidectomy

3. Combined approach tympanoplasty

Canal wall down mastoidectomy:

1. Modified radical mastoidectomy (Bondy's)

2. Atticotomy

3. Atticoantrostomy

4. Radical Mastoidectomy

5. Retrograde Mastoidectomy

Cortical Mastoidectomy:

Also known as simple mastoidectomy, Schwartz surgery, or complete mastoidectomy.

Indications:

1. Chronic suppurative otitis media not responding to medical management

2. As a preliminary step to other surgical procedures i.e. facial nerve decompression, cochlear implant etc.

Commonly cortical mastoidectomy is preformed for chronic suppurative otitis media which are resistant to medical managment.

The aims of cortical mastoidectomy when performed for infective conditions are:

1. To exenterate the infected mastoid air cells

2. To widen the aditus to facilitate better ventilation

3. To clear the middle ear of infections and hypertrophied mucosa

Procedure:

It is performed either under local anesthesia or general anesthesia. It is better to perform this surgery under general anesthesia in anxious patients. Whatever may be the choice of anesthesia, the following steps are more or less the same.

Infiltration: The post auricular area is infiltrated using 2% xylocaine with 1 in 80,000 units adrenaline. The whole of the post auricular sulcus is infiltrated. About 2 - 3ml of xylocaine can be used for this purpose. The infiltration serves two purposes:

1. It reduces bleeding due to local vasoconstriction

2. It elevates the periosteum from the mastoid cortex making it stripping easier.

Incision: Commonest incision used is William Wild's post auricular incision. It is a curviliner incision hugging the post auricular sulcus begining from the root of helix superiorly, extending up to the mastoid tip.

Gradual deepening of the skin incision exposes the periosteum. This is stripped away from the mastoid cortex using a sharp periosteal elevator. A post auricular skin flap is raised, and is pushed anteriorly to be held in place by a roller gauze tied through it. Now the external auditory canal, ear drum and the mastoid cortex becomes visible in the same view.

Cutting burrs are used to drill out the cortical bone from the mastoid cortex. Two incisions are made. One horizontal and one vertical. The horizontal cut is made just below the supra mastoid crest. This starts from the anterior portion of the Maceven's triangle extending posteriorly up to the sino dural angle. This line approximately indicates the level of dura and hence dissection should not go above this line. The second vertical cut is made along the external auditory canal starting from the Maceven's triangle up to the mastoid tip.

MacEven's triangle: is the surface marking for mastoid antrum in adults. The antrum lie about 1.5 cm below this triangle.

It is bounded above by the supra mastoid crest, antero inferiorly by posterior superior margin of external auditory canal and posteriorly by a tangential line drawn from the zygomatic arch. The spine of Henle lies within this triangle.

Antrum is entered by drilling the mastoid cortex. The antral and periantral air cells are exentrated. The aditus is identified. It is widened in the anterosuperior direction. It should not be widened in an inferior direction because the incus could become dislodged. After the aditus is widened, the posterior meatal wall is thinned out.

The middle ear is cleared off the infective material and oedematous mucosa after elevation of tympanomeatal flap. Ossicular chain is checked for functional continuity. If the incus is necrosed, ossicular prosthesis is introduced.

Wound is closed in layers.

Modified radical mastoidectomy: The initial steps are the same as for cortical mastoidectomy. After the aditus is widened, and posterior canal wall is thinned out, the Posterior canal wall is removed (removal of bridge). The facial ridge is lowered till the level of lateral semicircular canal. After the surgery is completed, a meatoplasty is performed making the external canal, middle ear cavity and mastoid cavity into one continuous self cleaning cavity lined by skin.

Complications of mastoid surgery:

1. Injury to ossicular chain

2. Injury to facial nerve

3. Injury to dura

4. Injury to lateral semicircular canal

5. Injury to lateral sinus

Atticotomy:

This procedure is ideal for limited attic disease, small attic retraction pockets. (Disease limited to attic area alone). The outer attic wall is removed. This helps in widening the access to attic area. Cholesteatoma can be mobilised from the attic area and removed via the space created by removing the outer attic wall. If cholesteatoma involves the anterior epitympanic recess area then the head of the malleus needs to be clipped to access this area. After completion of cholesteatoma clearance the outer attic wall and ossicular chain (if need be) should be repaired. Conchal cartilage is a favorite graft material for outer attic wall reconstruction. A PORP or TORP may be used to reconstruct ossicular chain. The temporalis facia graft is placed lateral to the outer attic wall cartilage. The major advantage of this procedure is that the middle ear depth is maintained facilitating proper ossicular chain reconstruction. Hearing results are excellent in this procedure. This is a conservative procedure.

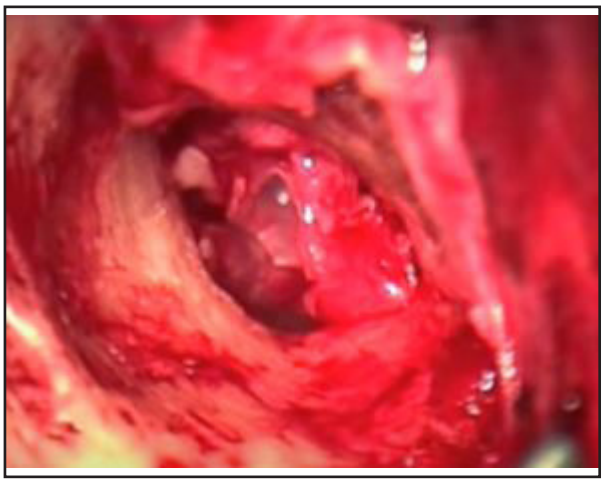


Figure showing tympanomeatal flap being elevated

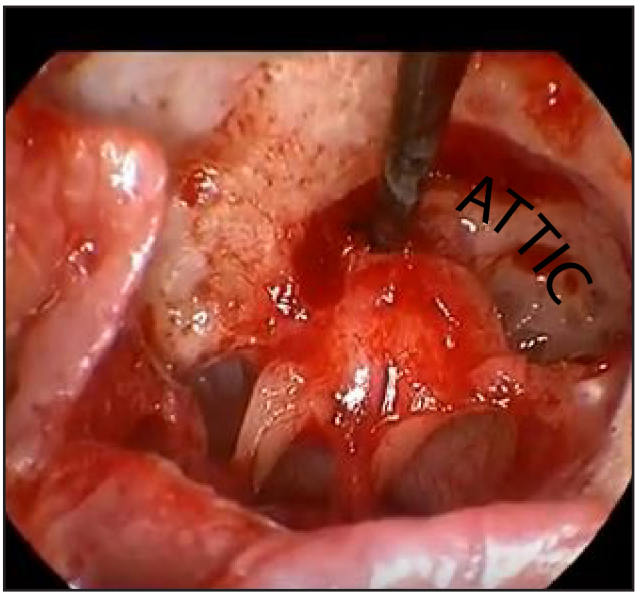
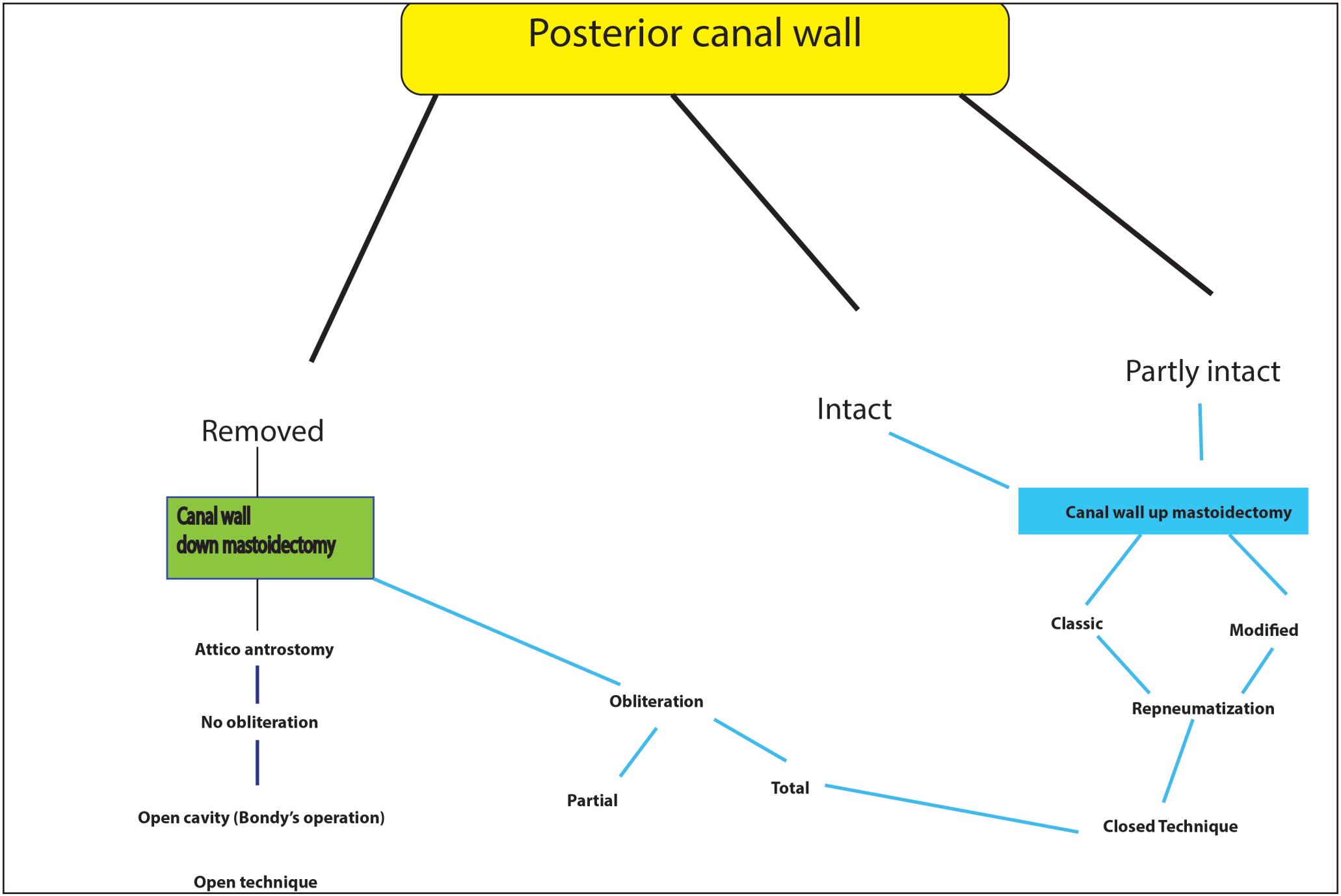


Figure showing attic exposed after elevating the tympanomeatal flap


Posterior canal wall
Removed
Intact
Partly intact
Canal wall down mastoidectomy
Canal wall up mastoidectomy
Attico antrostomy
No obliteration
Open cavity (Bondy's operation)
Open technique
Obliteration
Partial
Total
Classic
Modified
Repneumatization
Closed Technique

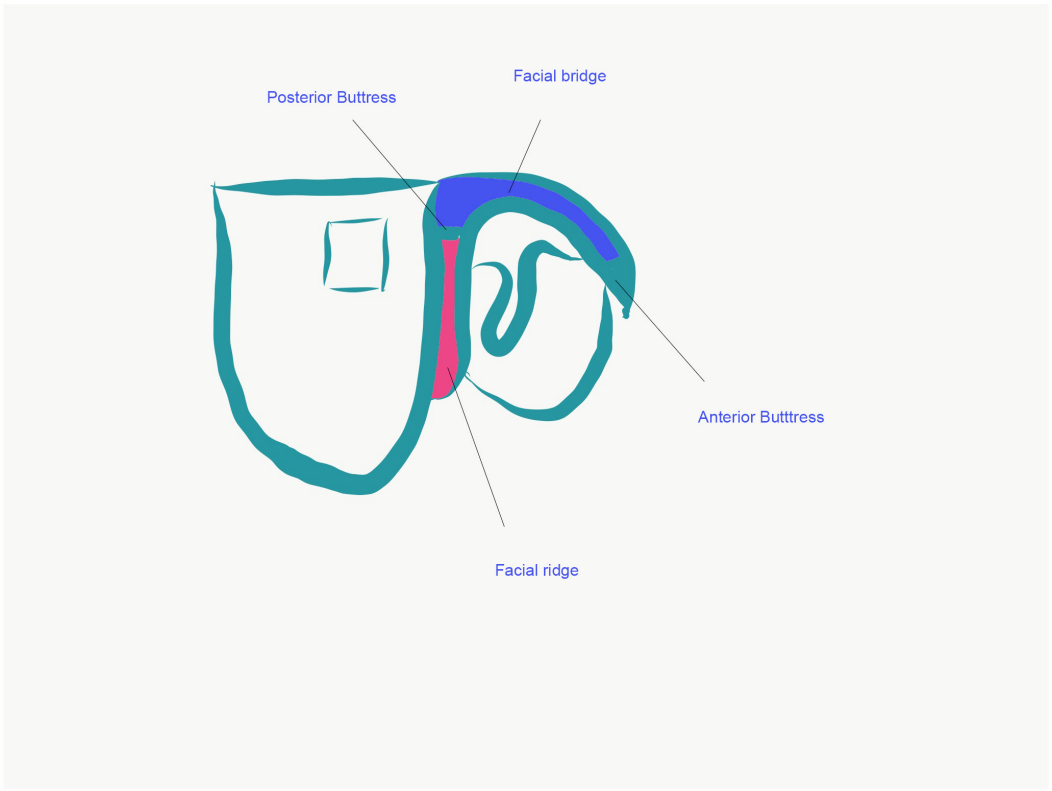


Diagram showing cortical mastoidectomy cavity

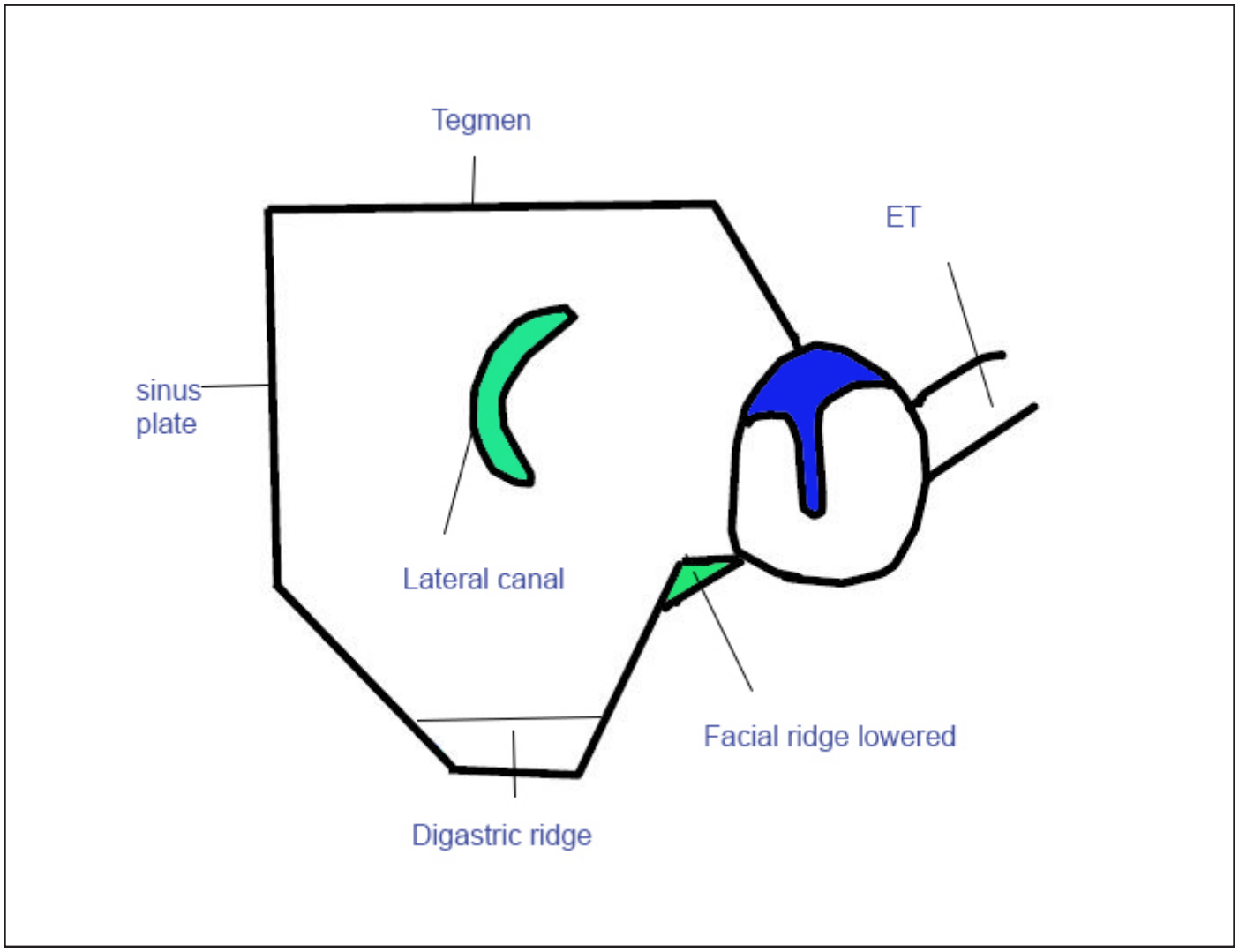


Diagram showing modified radical mastoidectomy

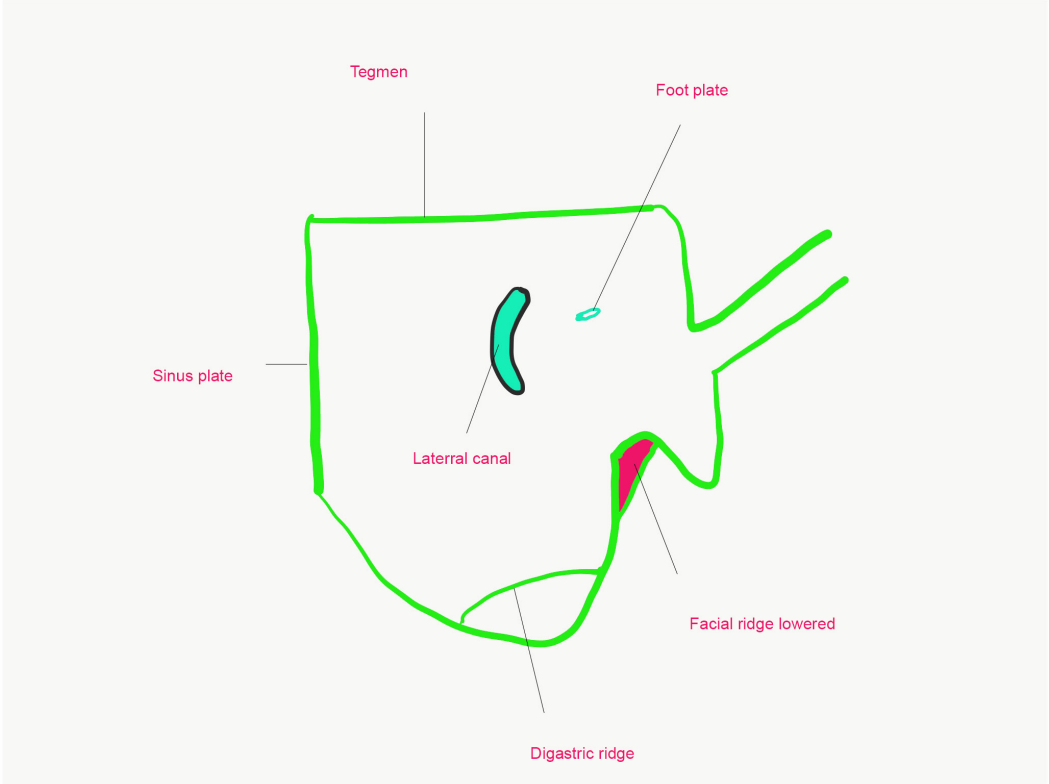
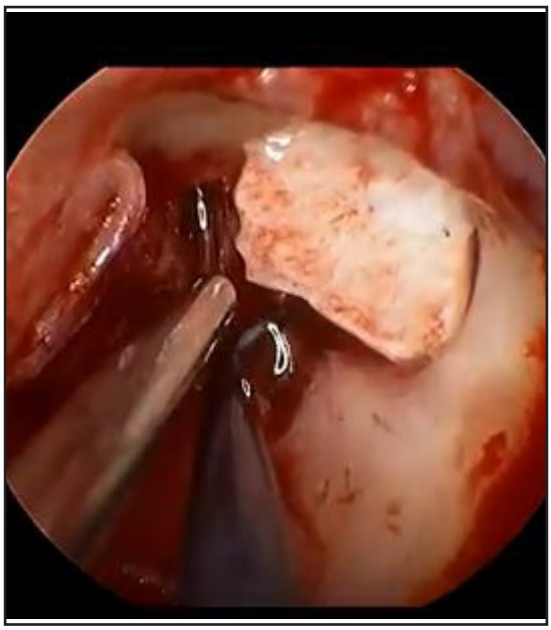


Figure showing radical mastoidectomy cavity



Conchal cartilage graft used to reconstruct outer attic wall defect following atticotomy

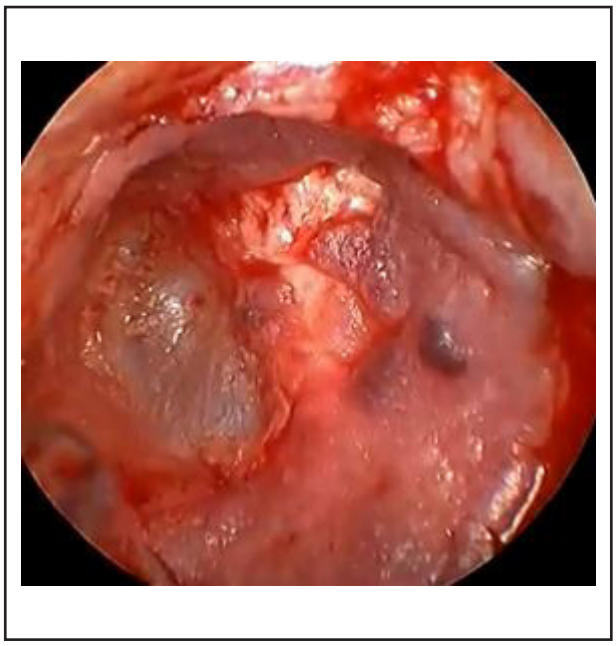


Figure showing the result of outer attic wall reconstruction

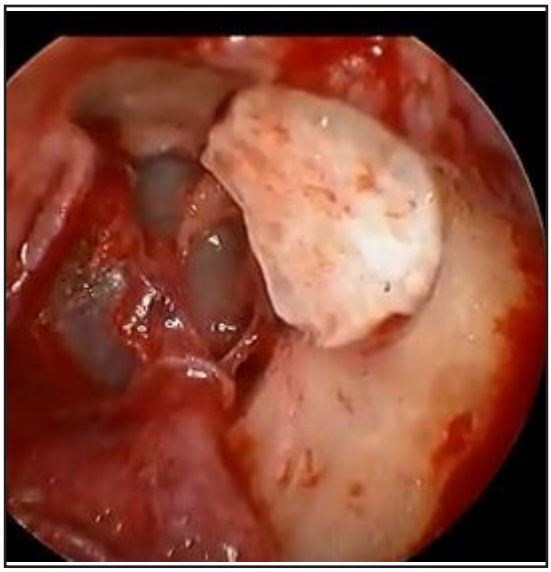


Figure showing conchal cartilage in place

Radical Mastoidectomy:

This procedure is rather rarely performed these days. It is mostly reserved for extensive cholesteatoma involving the middle ear with cochleo promontory fistula / middle ear malignancies. Hearing is not reconstructed in this procedure.

The incision used is the classic postaural incision of William wilde.

Differences between radical mastoidectomy and modified radical mastoidectomy:

Surgical steps involved is more or less similar to that of modified radical mastoidectomy. The following are the differences:

1. Posterior meatal wall is removed in toto

2. Entire middle ear is cleared of its contents and the mucosa is stripped till bare bone is visible

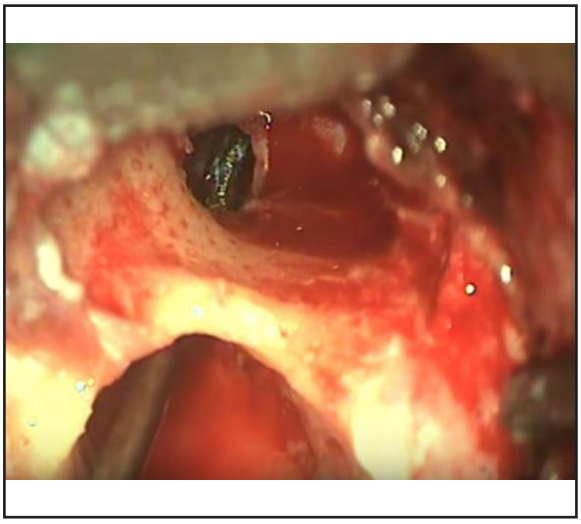
3. All ossicles are removed except the foot plate of stapes

4. Eustachean tube is obliterated using fat / cartilage in order to prevent middle ear becoming infected

5. Ear drum is removed

6. External auditory canal is obliterated using blind sac procedure

7. Mastoid cavity is obliterated using temporalis muscle graft.



First step in radical mastoidectomy is cortical mastoidectomy with widening of aditus as shown above

Indications for radical mastoidectomy:

1. When cholesteatoma cannot be removed completely for example from eustachean tube orifice, round window niche, perilabyrinthine cells or hypotympanic air cells.

2. Repeated attempts to surgically eradicate middle ear cholesteatoma has failed

3. As an approach to petrous apex

4. Removal of glomus tumor

5. Carcinoma of middle ear cavity

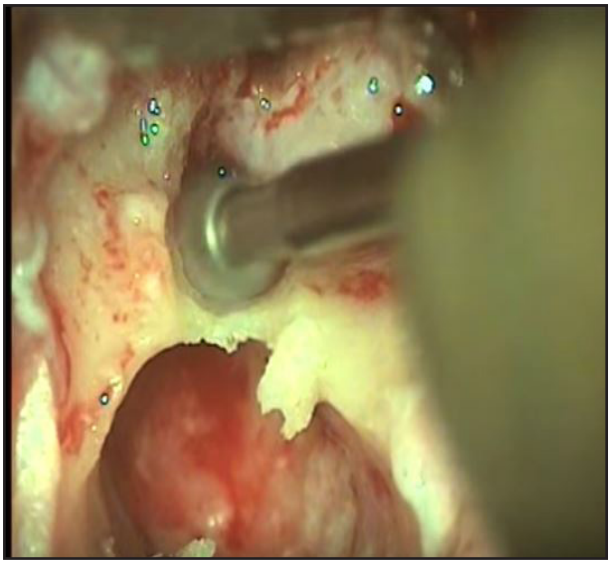


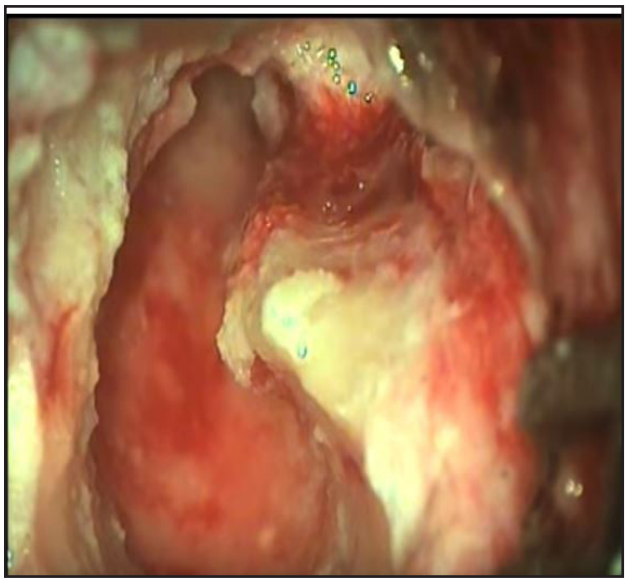
Image showing outer attic wall drilled out

Post operative care involves:

Wound dressing

Prevention of infection

Cavity care



Facial ridge seen lowered

Retrograde mastoidectomy:

This procedure was popularized by Dr Dornhoffer.

From time immemomorial the primary objective in the surgical management of cholesteatoma has been the eradication of the disease thereby ensuring a dry and safe ear free of complications. Hearing preservation / restoration was not given importance. In the current age of changing trends a lot of importance has been given to hearing preservation / reconstruction. With widespread availability of specialist services now more and more patients with cholesteatoma have been identified at a very early stage thereby kindling hopes of hearing preservation / reconstruction following mastoid surgery.

The main advantage of this procedure is to begin the surgery from where the disease (cholesteatoma) has started, i.e. from the epitympanic membrane. In this process instead of removing a significant amount of cortical mastoid bone to reach the disease, the surgeon prefers to follow the disease (retraction pocket, cholesteatoma) by initiating the surgery via the external auditory canal. The cortical bone removal is continued parallel to the lateral process of malleus and follows the disease pathology. In the next step the anterior epitympanic recess and superior malleolar ligament are identified. Subsequently bone removal is continued towards incudomalleal articulation and a clear view of tegmen and short process of incus is obtained. It is ideal to use a small diamond burr at a low speed of rotation to complete these critical steps.

The major advantage of this procedure is that the tegmen and facial nerve are identified right at the begining stage of the surgical procedure. The resulting mastoid cavity following this procedure is rather small. Reconstruction can be attempted using bone patte harvested from normal mastoid cortical bone and conchal cartilage as the need be.

This procedure involves mastoid obliteration combined wtih mastoid exclusion. These two are the major aims in reconstruction of posterior canal wall procedures. To enable this process of reconstruction to proceed flawlessly the posterior canal wall should not be fully removed during the procedure. Only those portions which obstruct the visibility of the middle ear cavity need to be removed leaving behind considerable portion of the posterior canal wall. The mastoid cavity need not be fully stripped off its mucosal lining as this procedure excludes the mastoid cavity from the middle ear cavity. Ideally more than 50 percent of posterior canal wall is retained.

This procedure is useful in patients with well pneumatized mastoid cavity. In sclerotic mastoid cavity there is associated eustachean tube dysfunction and middle ear infection and hence this procedure may not be successful.

The issue of reconstruction of posterior canal wall following mastoidectomy is rather controversial. Reconstructing the entire posterior canal wall using hard tissue reconstruction techniques has a tendency to cause retraction pockets at a later date. In this procedure more than 50 percent of the posterior canal wall is retained and only a small amount needs to be reconstructed. This can easily be done following the existing hard tissue reconstruction techniques (bone chips, bone patte, and temporalis muscle graft). This proves to be successful if the eustachean tube function and middle ear mucosal ventilation is functioning normally as in this case because the mucosa from the mastoid cavity is not stripped off completely.

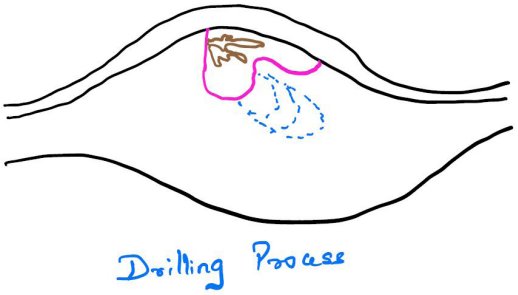


Diagram illustrating the drilling process for retrograde mastoidectomy

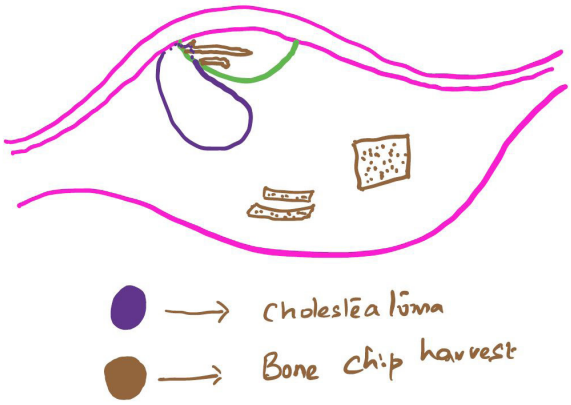


Diagram showing cholesteatoma for which retrograde mastoidectomy being performed. Bone chip and bone patte harvest has been diagrammatically represented

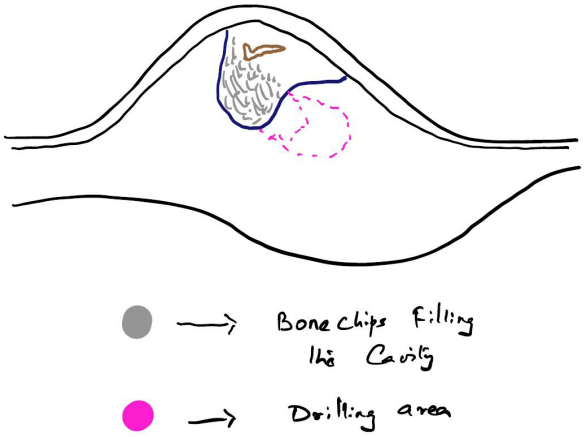


Diagram showing mastoid cavity filled with bone chips and pattee. Drillling area is indicated by dotted lines

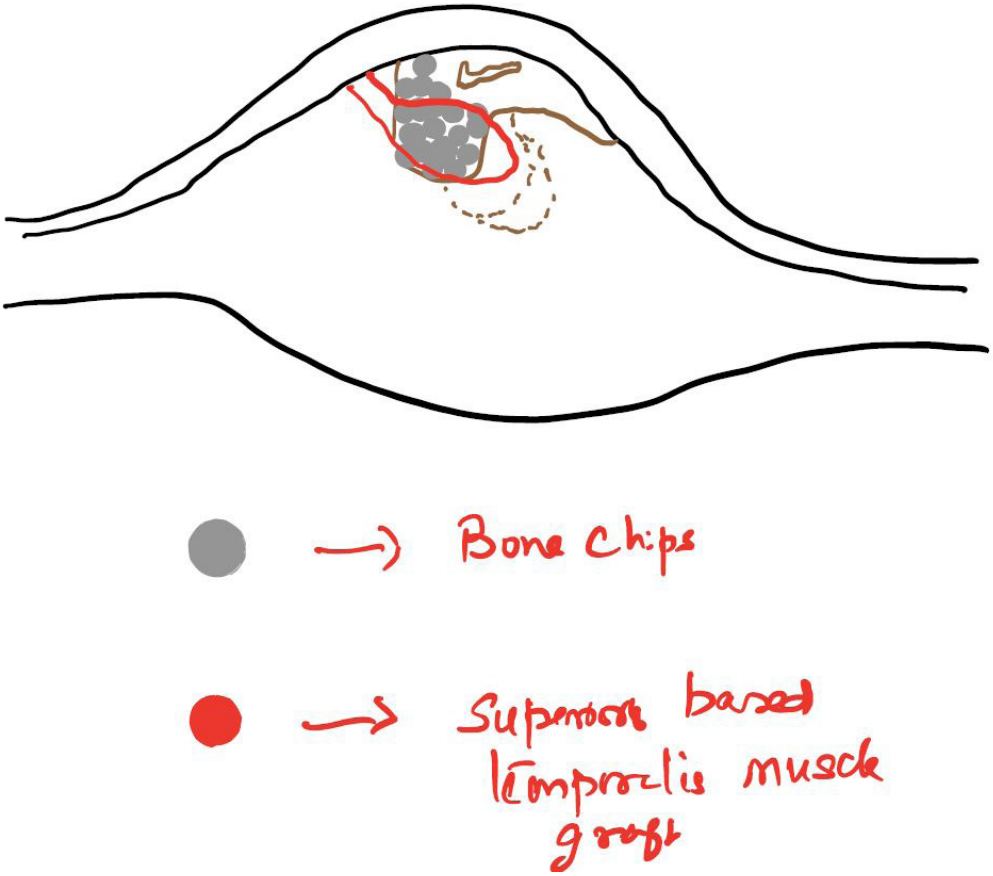


Figure showing superiorly based temporalis muscle flap rotated to reconstruct and fill the mastoid cavity

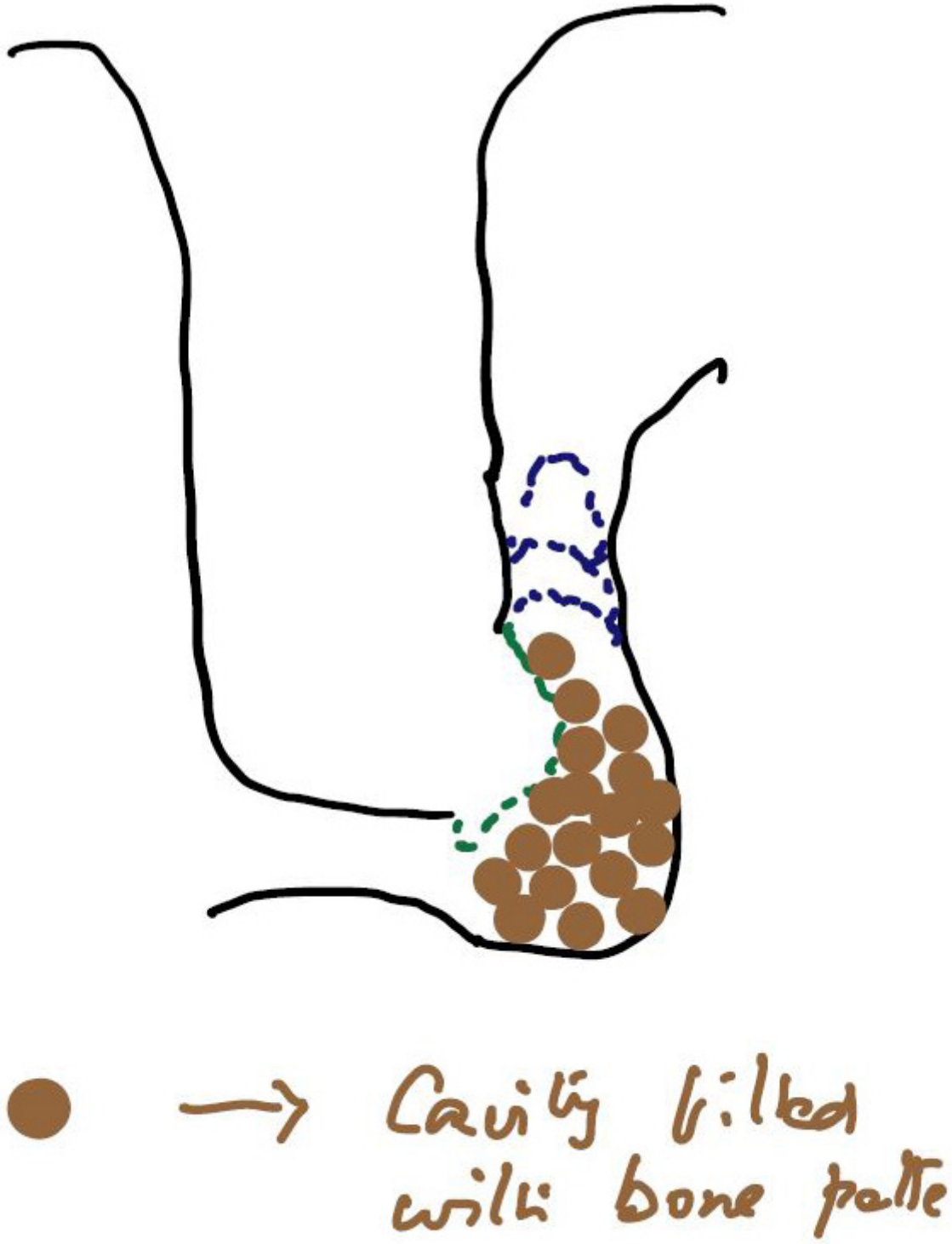
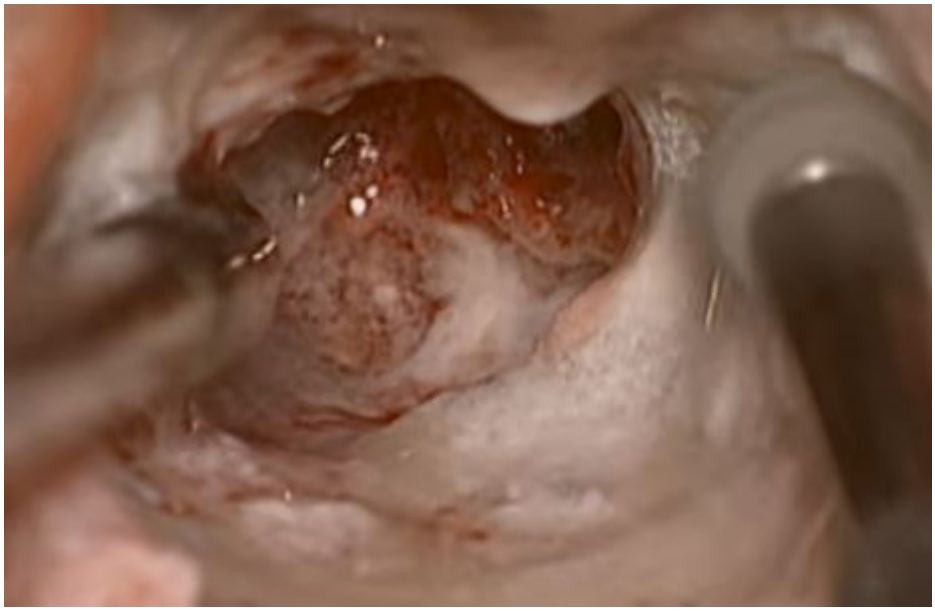


Figure showing cavity filled with bone chips and bone pattee. Drilling area is indiated by dotted lines. Note partial removal of posterior canal wall



Picture showing removal of outer attic wall

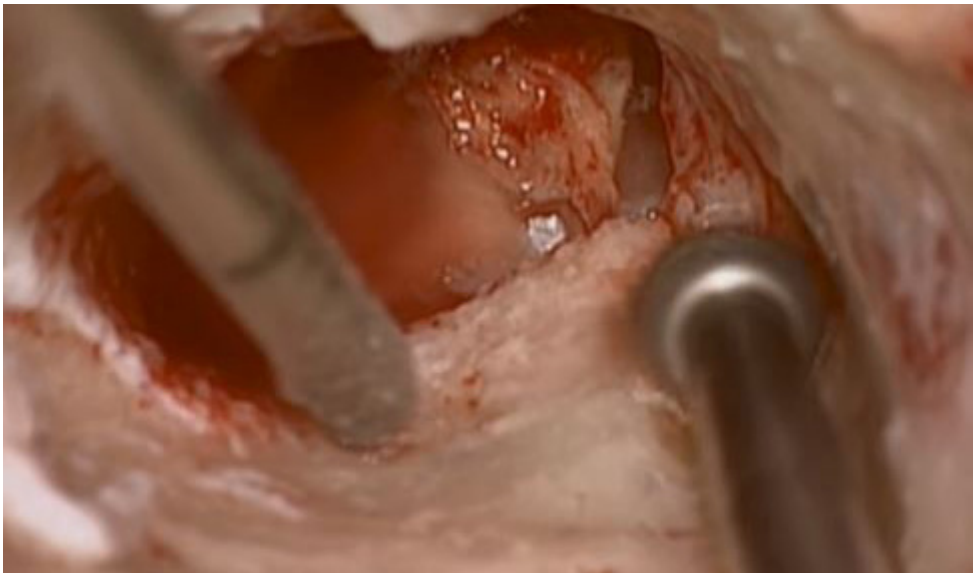


Figure showing reduction of posterior canal wall

History of Mastoidectomy

Introduction:

"One who ignores history would do so at his peril, to be condemned to repeat the same mistakes". A study of history of mastoid surgery and its instrumentation is important in a sense that they are the tombstones to our success today. Eighteenth century is characterized by advancement in instrument designs and sterilization techniques. Heat resistant metals were used to manufacture surgical instruments as they had to withstand extremely high sterilization temperatures. Our forefathers of 18th century were great innovators and to their credit even now majority of mastoid instruments in use were conceived and designed by them.

Mastoidectomy during different eras:

The art and craft of Mastoidectomy has evolved during the past 200 years. The process of this evolution can be studied under three different eras i.e.:

1. Era of trepan (18th century)
2. Era of chisel & gouge (Early 19th century)
3. Era of electrical drill (20th century)

Era of Trepan:

Trephination was performed to let out pus. This was extensively practiced during the 18th century to let out pus from skull bones. The first successful trephination of mastoid cavity was performed by Ambroise Pare during 16th century. Younger during 17th century devised a hand Trepan which he used extensively to perform this procedure. A hand held trepan was commonly used during this period.

The cutting head of trepan used could be circular (to cut a circular piece of bone), exfoliative head (to shed the superficial layer of bone), and perforative head (used to make a hole in the bone). In 1736 Jean Louis Petit performed the first mastoid opening for a patient with mastoid abscess. Pus His main aim was to create a hole through which pus from the mastoid cavity can drain. While using a Trepan it should be dipped in cold water often to reduce heat generated during the procedure.

In 1776 Jasser used a trocar to open up the mastoid cavity. He used the nozzle of a syringe to aspirate the contents from the mastoid cavity. This surgical procedure hence was aptly named as "Jasser procedure". The term "trocar" has its origin in French language. "Toris – quarts" is a French word to describe an instrument with three cutting sides used to make a hole. American otologist Fredreik White described this era of mastoid surgery as an experimental one. This experimental era proved that the concept of opening up the mastoid cavity and draining the secretions is a possibility. The instrumentation was of course woefully inadequate. The first catalogue of surgical instruments published in 1860's mentioned the various surgical and dental instruments in use. Mastoid instrumentation of course did not find a place in that catalogue.

Chisel & Gouge period: This period was characterized by the introduction of general anesthesia which facilitated a surgeon to operate leisurely on a patient. It was Amedee Forget a French surgeon who used a mallet and gouge to open the mastoid cavity and drain the accumulated pus. He performed this surgery during 1860.

Modern mastoid surgery was pioneered by the German otologist Scwartze during 1873. He and his assistant Adolf Eysell abandoned the use of Trepan in favour of chisel and gouge. He popularized Chisel and gouge as he was convinced that it was the safest way to open up the mastoid antrum.

His assistant had drawn up detailed illustrations of the various types of chisel and gouges used in this procedure. Buck introduced the small curette that could be used to widen the aditus. He also advocated continuous chiseling of the hard mastoid cortex till the soft bone is reached which could be curetted out rather easily using curettes of varying sizes.

Initially Volkmann sharp edged spoons were used as curette. Samuel Kopetzky, American otologist advised that one should become dexterous and elegant with the use of a set of instruments. Newer instruments (design wise) should be introduced only when they have distinct advantages over the tried out older ones. This observation holds good even today.

Electrically driven drill period: "Modern era Mastoidectomy"

Electrically driven drills were used to manage dental caries even way back in 1882. It was William McEwen who drew the attention of the world to this unique device. He believed that the safest instrument that can be used to drill the mastoid antrum is the rotating burr. It had better control and uniform rotator cutting ability. The size of the burr bits can vary according to the area of surgery. It was Julius Lempert in 1922 who really popularized the use of electrically driven drill in ear surgeries. William House introduced the suction irrigation system and retractors in mastoid surgery. He observed that while performing ear surgeries a surgeon needs to keep both hands useful.

Holmgren introduced the operating microscope which really made Mastoidectomy totally a safe procedure.

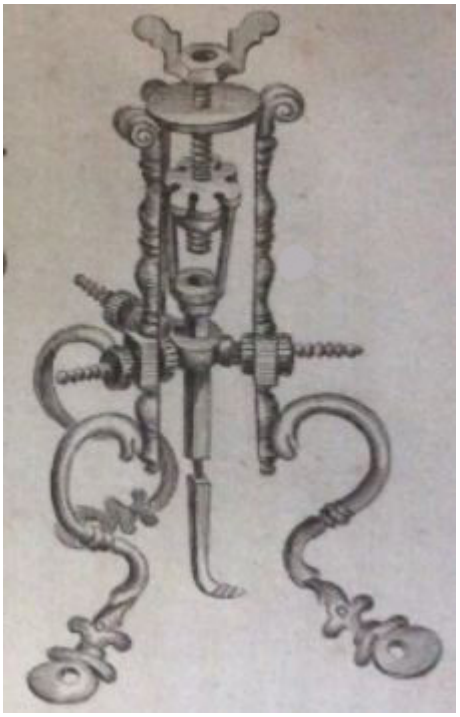


Image showing the Trephine used in the era of Trepan

Tympanoplasty

Definition:

Tympanoplasty is defined as the surgical procedure which enables reconstruction of middle ear cavity and the conducting ossicular system (tympano- ossicular system).

The following surgical procedures are components of tympanoplasty:

1. Canal plasty

2. Meatoplasty

3. Myringoplasty

4. Ossiculoplasty

Canalplasty: Is widening of the external auditory canal. It should be considered to be an integral part of myringoplasty. This procedure should be performed before grafting anterior perforations of tympanic membrane as it gives necessary surgical access for repair. This procedure facilitates healing, cleansing and if needed a second stage ossiculoplasty.

Meatoplasty: Is used to enlarge the opening of lateral cartilagenous portion of external auditory canal. This enlargement should be in proportion to the size of the medial bony external auditory canal.

Ossiculoplasty: This procedure is used to reconstruct the damaged middle ear sound conducting ossicular system.

Aims of this surgical procedure:

a. Disease eradication

b. Restoration of middle ear aeration

c. Reconstruction of sound transmission mechanism

d. Creation of self cleansing dry cavity

Desired Preoperative investigations:

Tubal function tests

Tubal funtion tests is very important for proper surgical planning and to assess the chance of a possible hearing improvement. If tubal function tests are negative, blockage if any at the tympanic end of the eustachean tube. If any scar tissue is present in that area it should be meticulously removed to pave way for better ventilation of the middle ear cavity.

Radiological investigation:

Xray of temporal bones / CT scan temporal bones will be a reliable indicator for pneumatization of temporal bone. A well pneumatized temporal bone indicates a normally functioning eustachean tube. Sclerosed mastoid indicates supressed due to infantile otitis media or blocked eustachean tube.

Temporary closure of perforation with wet gelfoam: This helps to assess the condition of ossicular chain / status of round and oval windows. If hearing improves when the perforation is temporarily occluded with a gelfoam plug then it indicates normally functioning middle ear sound conduction mechanism.

Fistula test: Should always be performed before surgery. If it is positive then surgery should be deferred as this would lead to dead ear.

Pre op preparation:
1. Surgery should be ideally performed in a dry ear. The ear must be cleaned before surgery. Systemic / topical antibiotics should be administered to reduce the likelihood of infection.

2. A persistently draining ear is a problem. If the secretion is purulent then it should be swabbed and sent for culture and sensitivity. This will help in prescription of drugs to which the microbes are sensitive to. A predominantly clear mucoid secretion from the ear is definitely related to hyperplastic changes of middle ear mucosa, hence ear swab for culture sensitivity is not necessary.

3. Hair is shaved above and behind the ear about 2 cms. The ear canal is plugged with sterile cotton when the area of surgery is disinfected with povidone iodine. The external auditory canal is not disinfected.

Anesthesia:

Local / General

Position: Supine. The head should be positioned slightly lower than the plane of the table. The angle between the head and shoulder should be between 100 - 130 degrees to allow adequate working space for the surgeon's hands.

Surgical approach:

Transcanal approach:

In this approach surgery is performed through the ear speculum inserted into the external auditory canal. This approach is indicated in:

a. Patients with wide external auditory canal

b. There is no overhanging bony wall obscuring the rim of the perforation

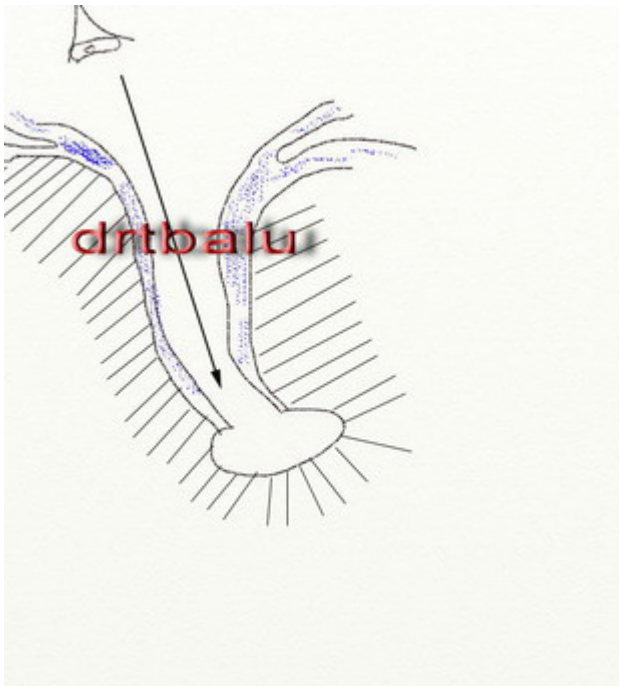


Figure showing transcanal approach

Endaural approach: In this approach the incision is made between the tragus and helix. An endaural speculum is introduced to widen the entrance into the external auditory canal. If posterior overhang is persent it can be easily burred out. If this approach is used better anterior visualization of the ear drum is possible.

Post aural approach:

Mastoid bone and the external auditory canal is approached via a post aural incision of William Wilde.

This approach is useful in cases of:

1. Narrow external auditory canal as it would improve exposure and facilitate canalplasty

2. In perforations involving the anterior portion of the ear drum

Figure showing transcanal view of ear drum

Figure showing endaural view of ear drum

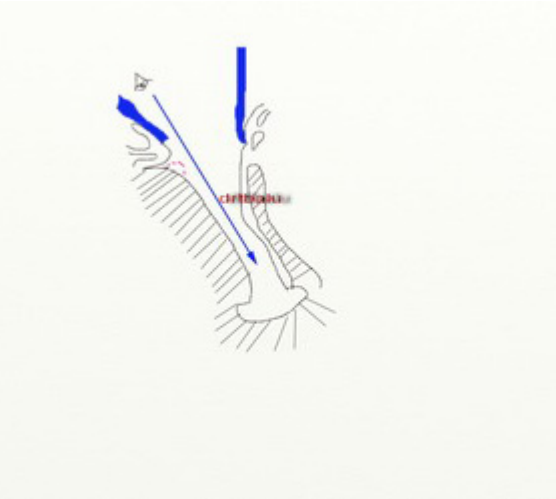


Figure showing endaural approach

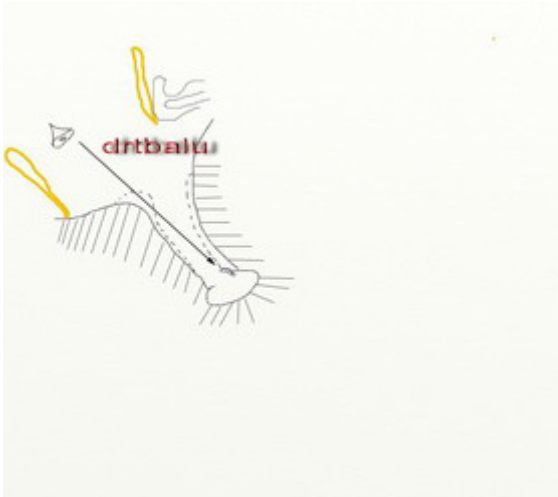


Figure showing post aural approach

Selection of approach:

Transcanal approach: is mostly used for repairing large acute traumatic perforations. The external canal should be fairly wide, and the rims of the perforation should be clearly visible.

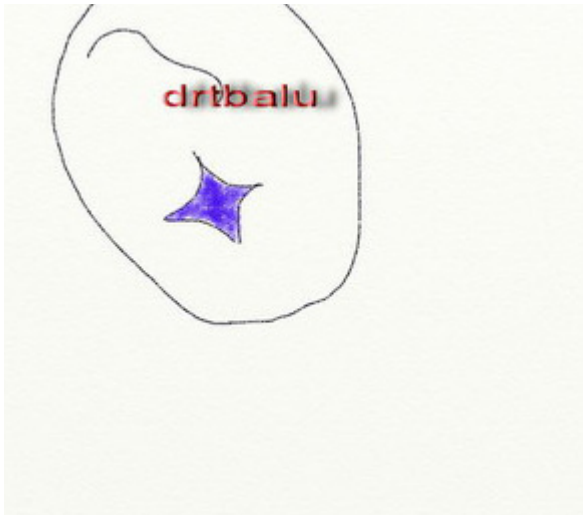


Figure showing large traumatic central perforation

End aural approach: Perforations involving the posterior portion of ear drum can be adequately visualized by endaural approach.

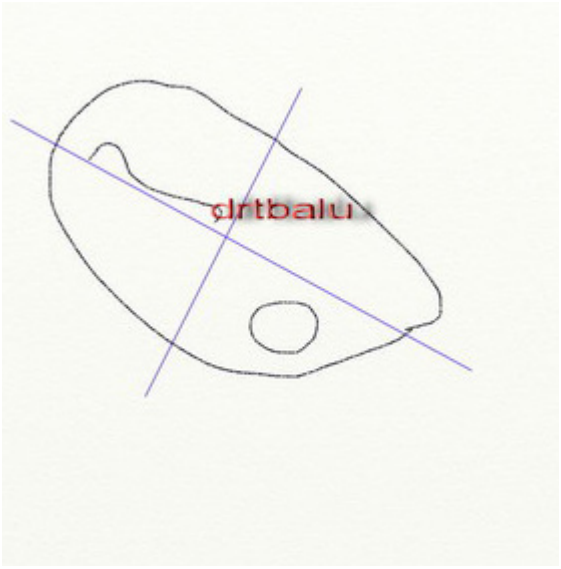


Image showing posterior central perforation

Postaural approach: Is reserved for those perforations whose margins could not be entirely visualised through intact external canal.

Grafting techniques:

Two types of grafting techniques are available i.e. overlay and underlay.

Overlay: The term overlay means that the graft has been placed over the bony tympanic sulcus, or over a bony ledge carved newly for this purpose when sulcus is absent. The overlaid fascia is supported by the presence of new / old annulus and is held in position by a remnant of tympanic membrane if still present.

Underlay: The term underlay indicates the position of the graft under the tympanic membrane and surrounding bone.

Combination of anterior underlay and posterior overlay: For this type of grafting procedure to succeed, there should be an anterior remnant of the tympanic membrane (atleast of the fibrous annulus). Anteriorly the graft is placed under the anterior remnant of the tympanic membrane and under the lateral wall of protympanum. Posteriorly, it is placed over the posterior tympanic sulcus and under the remnant of the tympanic membrane. With the exception of perforations limited to the anteroinferior quadrant, the graft lies under the malleus handle.

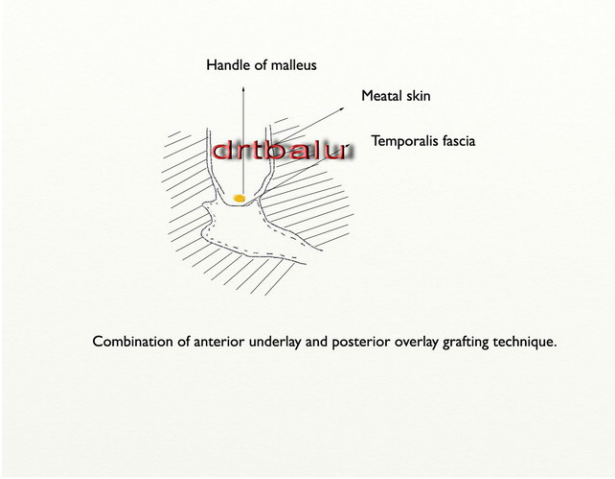
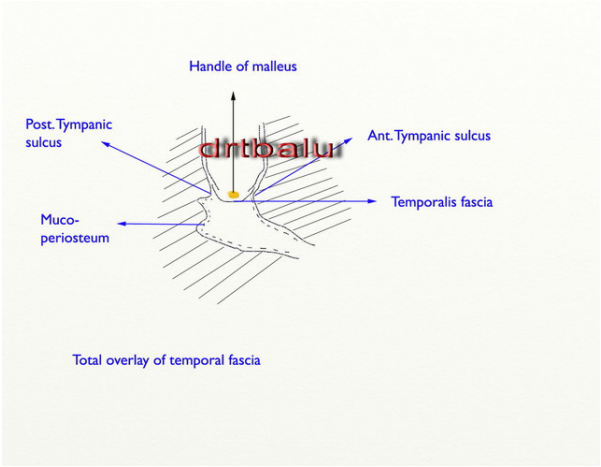
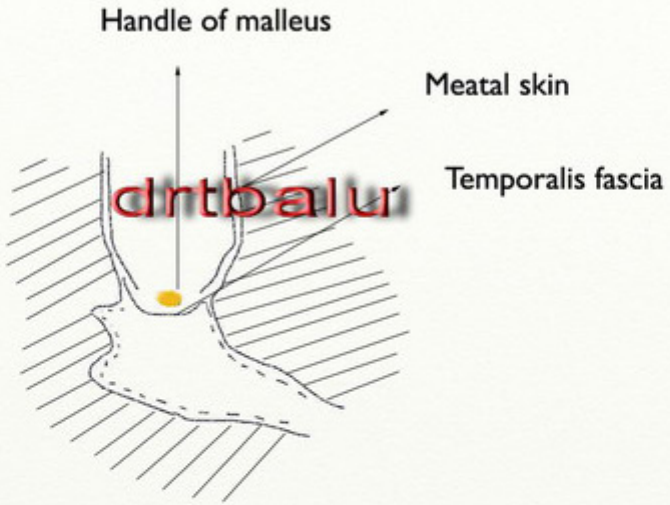


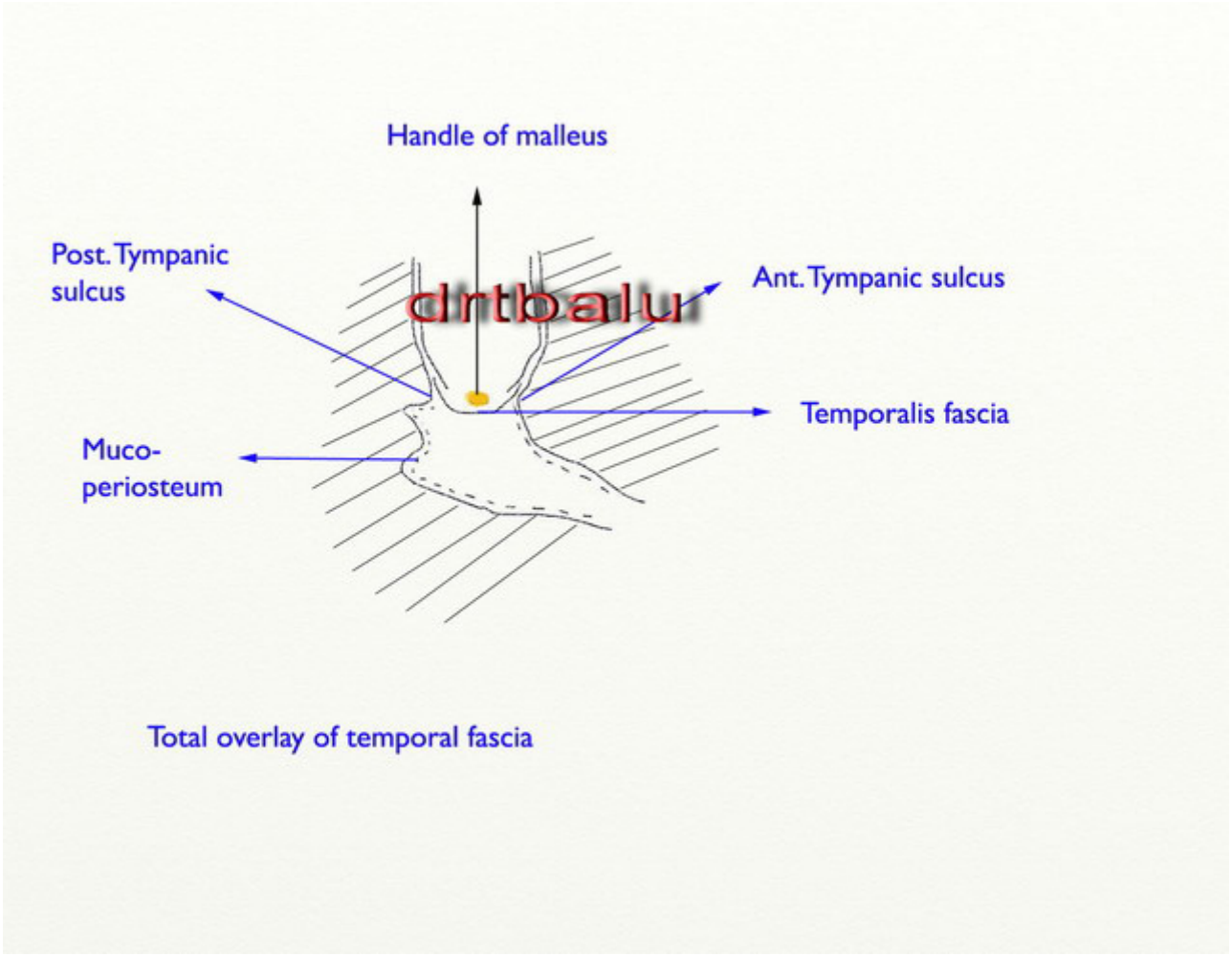
Figure showing large central perforation

Total overlay: This technique is useful when there is no remnant of the tympanic membrane is present. A new bony sulcus is drilled to support the fascia at the lateral opening of the tympanic cavity. The graft rests over the sulcus and underneath the malleus handle. The edges of the graft are covered along the canal wall by meatal skin.





Combination of anterior underlay and posterior overlay grafting technique.


Handle of malleus
Post. Tympanic sulcus
Ant. Tympanic sulcus
Temporalis fascia
Muco-periosteum
Total overlay of temporal fascia

Transcanal myringoplasty:

Indication:

1. Used to repair large acute traumatic perforations of ear drum

2. Used to repair dry central perforations.

Anesthesia: Ideally local

Ear speculum should be used to straighten the external auditory canal.

Perforated margins are outfolded.

Repositioned perforated margins are held in position by intra and extratympanic fixation using gelfoam.

Figure showing edges of perforation being outfolded

Procedure:

1. Perforation is visualized using aural speculum. Use of aural speculum straightens the external canal.

2. The speculum is fixed by holding it in the left hand

3. Outfolding of perforated margins: The edges of the perforation are outfolded using a 1.5mm, 90 degree hook.

4. Intratympanic fixation of perforation margins: Outfolded perforation margins are kept in position by intratympanic gelfoam pledgets soaked in Ringer's solution.

5. Extratympanic fixation of perforation margins: A piece of gelfoam is wet with Ringer lactate solution is used to stabilize the outer surface of lacerated tympanic membrane.

Endaural approach:

This technique is used for repairing posterior perforations, whose margins can be clearly seen through external auditory canal.

Anesthesia: Local

Incision: Endaural Lempert's incision

Tympanomeatal flap elevation and freshening of perforation margins: The skin incision for tympanomeatal flap is carried from the tympanic annulus at 7 'o clock and 12 'o clock in an ascending spiral fashion to meet the endaural incision. The anterior limb of the incision extends just anterior to the lateral process of the malleus. The edges of the perforation is freshened before elevation of the flap.

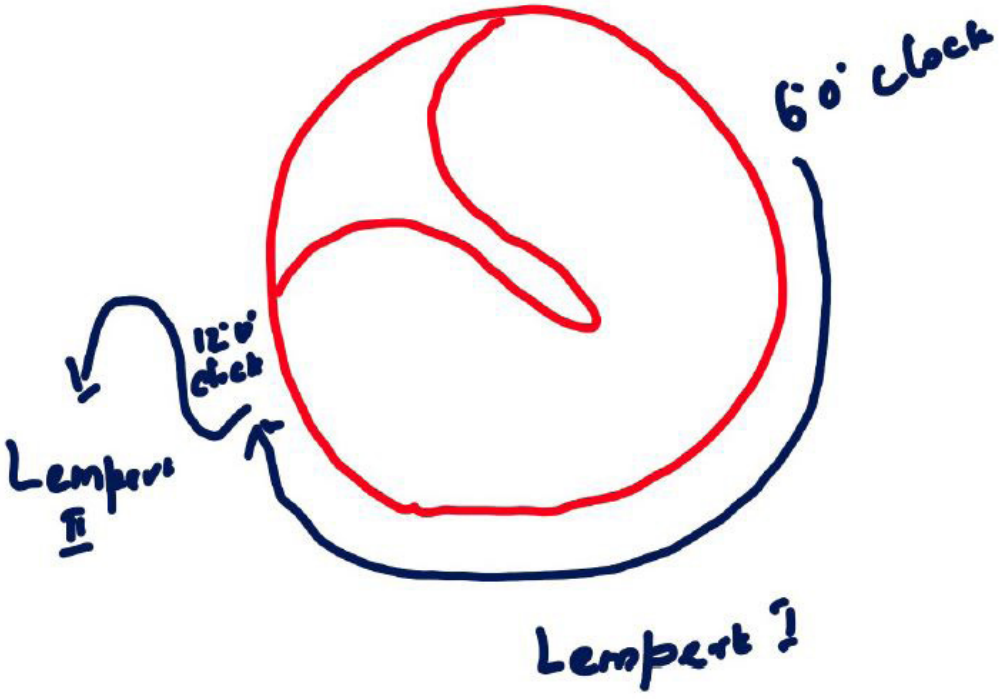


Figure showing Lempert incision

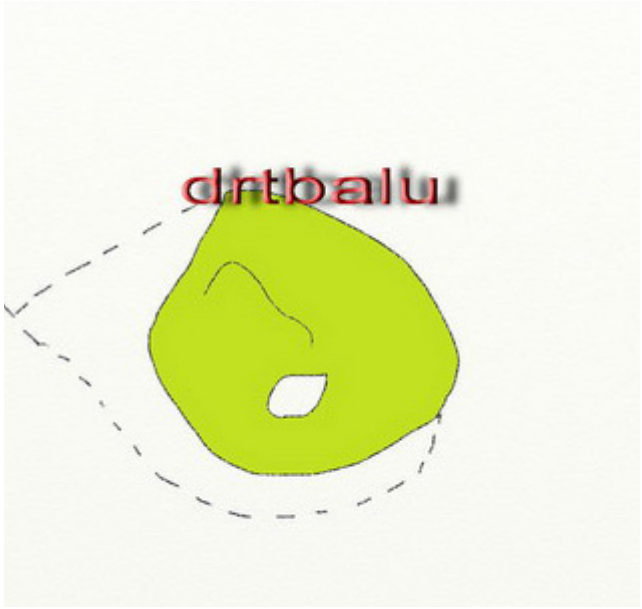


Figure showing elevation of tympanomeatal flap

After elevation of tympanomeatal flap, gelfoam pledgets soaked in Ringer's lactate is placed in the hypotympanum. Mobility of ossicular chain is checked.

Figure showing gelfoam inserted into the hypotympanum

Graft is inserted beneath the perforated drum and above the posterior tympanic sulcus.

Figure showing graft being inserted under the tympanomeatal flap.

Figure showing tympanomeatal flap replaced.

Post aural approach:

Indication: This approach is mainly used for anterior perforations of ear drum whose margins are obscured by bony overhang. In most of these patients canal plasty need to be performed.

Anesthesia:

General anesthesia.

Incision:

Post aural sking incision of William wild is used. It is carried out along the hairline and behind the mastoid tip. The incision is initially made only skin deep perserving underlying fascia and periosteum.

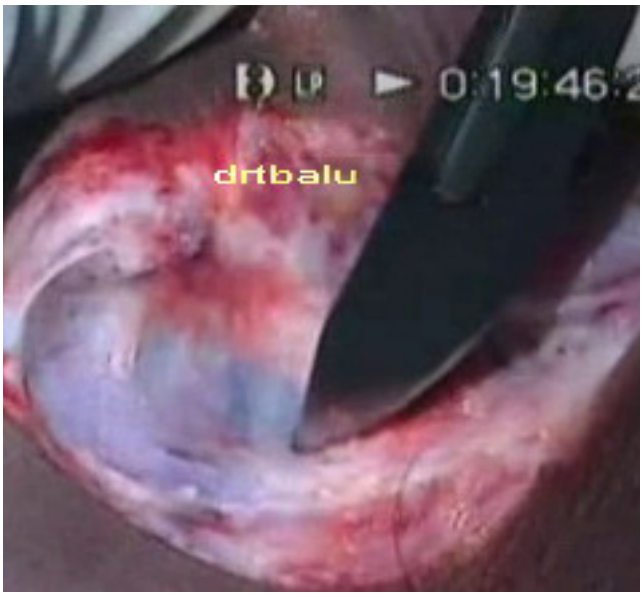


Figure showing post aural incision

Elevation of periosteal flap: Periosteal flap is then elevated from the bone using a periosteal elevator.

Figure showing periosteal flap being elevated

Exposure of external auditory canal:

While the periosteal flap is being elevated, the external auditory canal can be entered.

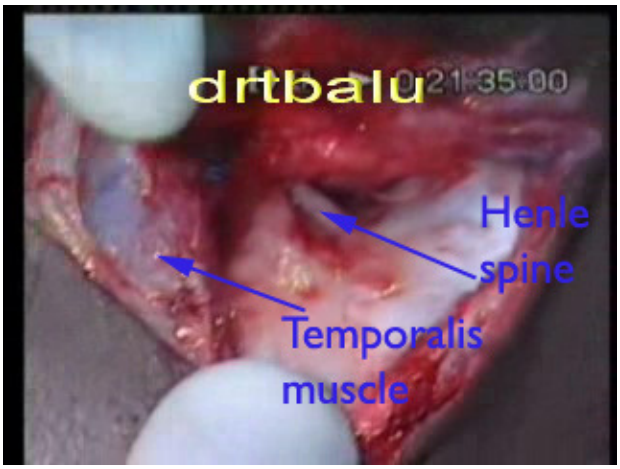
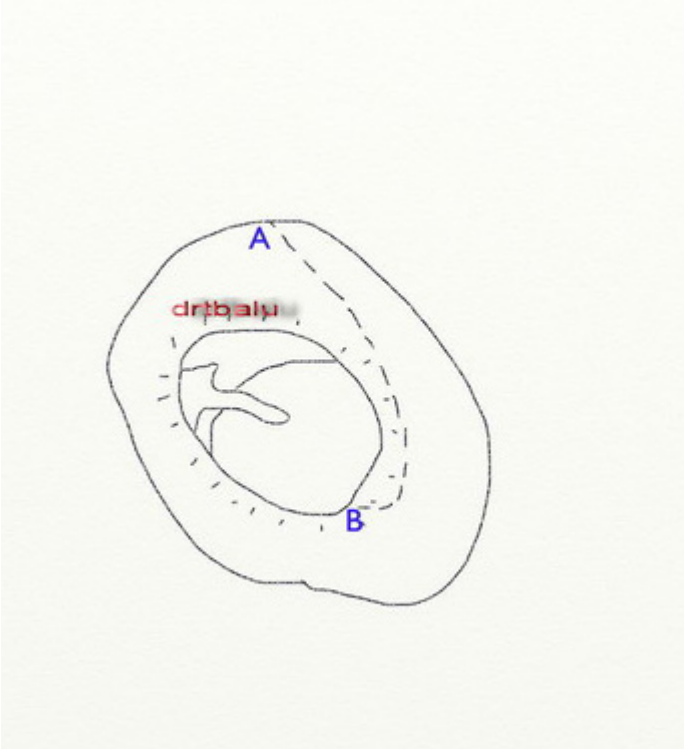


Figure showing external canal being exposed



Meatal skin flap is elevated as shown in figure below, extending from A to B.

If proper visualization could not be achieved canalplasty needs tobe performed.

Canalplasty: In this procedure the external bony canal is enlarged using diamond burrs, eliminating all bony overhangs. Care should be taken not to breach the temporomandibular joint situated anteriorly.

The rim of the perforation is freshened. Graft inserted, using underlay technique. Flap repositioned.

Types of tympanoplasty:

The fundamental principles of tympanoplasty were introduced by Zollner and Wullstein. These principles were directed towards restoration of middle ear function as well as ensured trouble free and stablized ear. Wullstein and Zollner classified tympanoplasty according to the type of ossicular reconstruction needed. Six types of tympanoplasty have been classified.

Type I tympanoplasty:

This is indicated in patients with presence of all the middle ear ossicles with normal mobility. Ossicular chain reconstruction is not needed in these patients. Efforts are made to close the perforated ear drum using temporalis fascia graft which is also known as (Hong-kong flap). This procedure is also known as myringo-plasty.

Advantages of using temporalis fascia as graft material:

1. It is an autograft with excellent chance of take

2. It is available close to the site of operation making the harvest easier

3. It has a low basal metabolic rate there by brightening its success rate

4. Its thickness is more or less similar to that of tympanic membrane

There are two available techniques for performing myringoplasty / Type I tympanoplasty:

1. Overlay technique

2. Underlay technique

Overlay technique:

This is actually a difficult technique to master. Here the graft material is inserted under the squamous layer of the ear drum. It is a difficult task peeling only the skin layer away from the ear drum and placing the graft over the perforation and then redraping the skin layer.

Underlay technique:

This is a simpler and commonly used technique. Here the graft is placed under the tympanomeatal flap which has been elevated and hence the name underlay technique. The major advantage of this procedure is that it is easy to perform with a good success rate.

Indications of myringoplasty:

1. Central perforation which has been dry atleast for a period of 6 weeks.

2. As a follow up to mastoidectomy procedure to recreate the hearing mechanism

Prerequisites for myringoplasty:

1. Central perforation which has been dry for atleast a period of 6 weeks

2. Presence of normal middle ear mucosa

3. Intact ossicular chain

4. Good cochlear reserve

Procedure:

Firstly a temporalis fascia of adequate size must be harvested and allowed to dry. The surgery is performed under local anesthesia.

The external auditory canal is then anesthetized using 2% xylocaine mixed with 1 in 10,000 adrenaline injection. About 1/2 cc is infiltrated at 3-oclock, 6-oclock, 9 -oclock and 12-o clock positions about 3 mm from the annulus. The patient should also be premedicated with intramuscular injections of 1 ampoule of fortwin and 1 ampoule of phenergan.

Step I:

Freshening the margins of perforation.

In this step the margins of the perforation is freshened using a sickle knife or an angled pick. This step is very important because it breaks the adhesions formed between the squamous margin of the ear drum with the middle ear mucosa. These adhesions if left undisturbed will hinder the take up of the neo tympanic graft. This procedure will infact widen the already present perforation. There is nothing to be alarmed about it.

Step II:

Elevation of tympano meatal flap.

Using a drum knife a curvilinear incision is made about 3 mm lateral to the annulus. This incision ideally extends between 12-o clock, 3-o clock, and 6-o clock, positions in the left ear and 12 -o clock, 9 -o clock and 6 - o clock positions in the right ear. The skin is slowly elevated away from the bone of the external canal. Pressure should be applied to the bone while elevating this flap. This serves two purposes:

1. It prevents excessive bleeding

2. It prevents tearing of the flap

This step ends when the skin flap is raised up to the level of the annulus.

Step III:

Elevation of the annulus and incising the middle ear mucosa. In this step the annulus is gradually lifted from its rim. As soon as the annulus is elevated a sickle knife is used fo incise the middle ear mucosal attachment with the tympano meatal flap. This is a very important step because the inner layer of the remnant ear drum is continuouswith the middle ear mucosa. As soon as the middle ear mucosa is raised, the flap is pushed anteriorly till the handle of the malleus becomes visible.

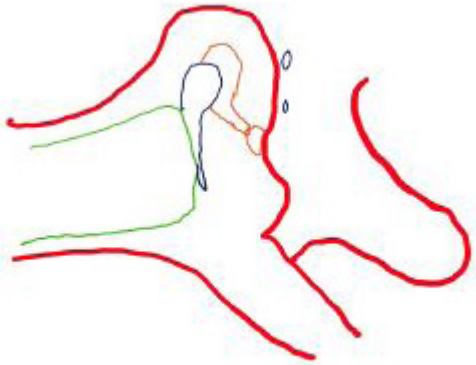


Diagram showing tympanoplasty type I

Step IV:

Freeing the tympano meatal flap from the handle of malleus.

In this step the tympano meatal flap is freed from the handle of malleus by sharp dissection of the middle ear mucosa. Sometimes the handle of the malleus may be turned inwards hitching against the promontory. In this scenario an attempt is made to lateralize the handle of malleus. If it is not possible to lateralize the handle of the malleus, the small deviated tip portion of the handle can be clipped. The handle of the malleus is freshened and stripped of its mucosal covering.

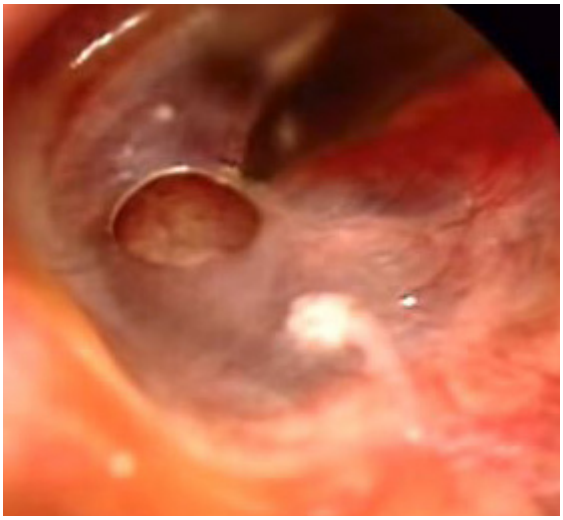
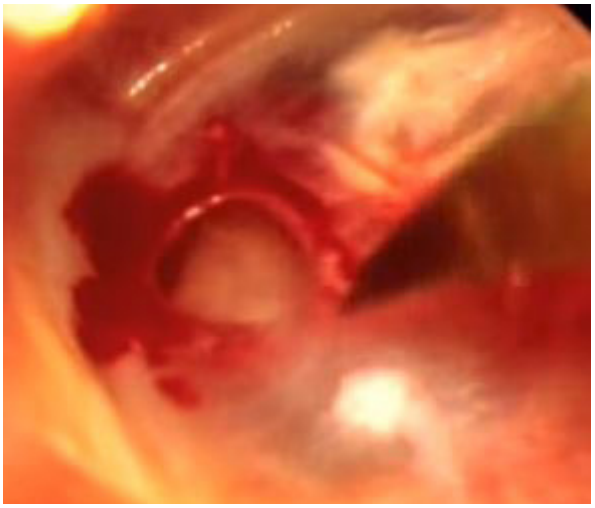


Figure showing freshening of edges of perforation using a sickle knife

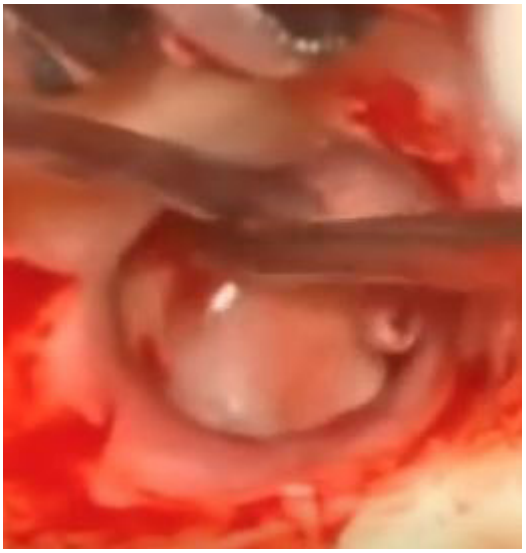
Step V:

Placement of graft (underlay technique).

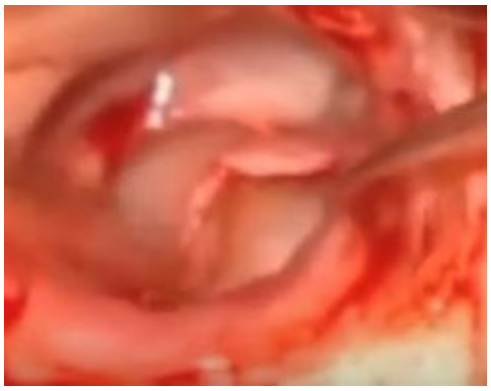
The properly dried temporalis fascia graft of appropriate size (ideally it should be twice the size of the perforation). The graft is gently pushed under the tympano meatal flap which has been elevated. The graft is insinuated under the handle of the malleus. The tympano meatal flap is repositioned in such a way that it covers the free edge of the graft which has been introduced. Bits of gelfoam are placed around the edges of the raised flap. One gelfoam bit is placed over the sealed perforation. This gelfoam has a specific role to play. Due to the suction effect created it pulls the graft against the edges of the perforation thus preventing medialization of the graft material.



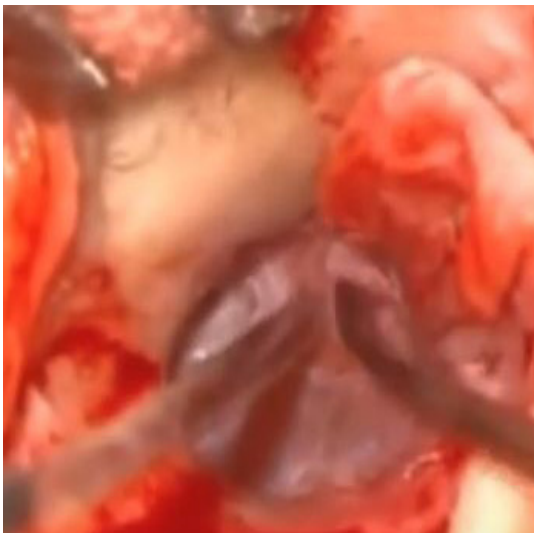
Freshening of perforation edges completed



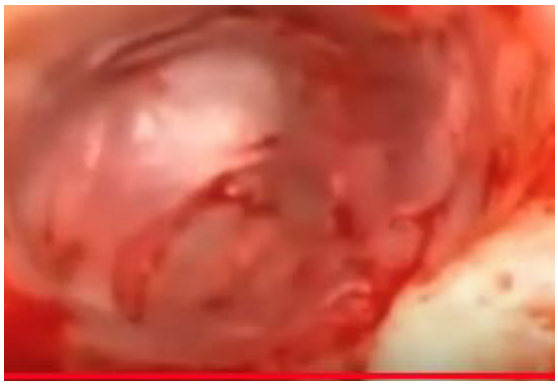
Flap elevation completed



Typanomeatal flap seen being elevated



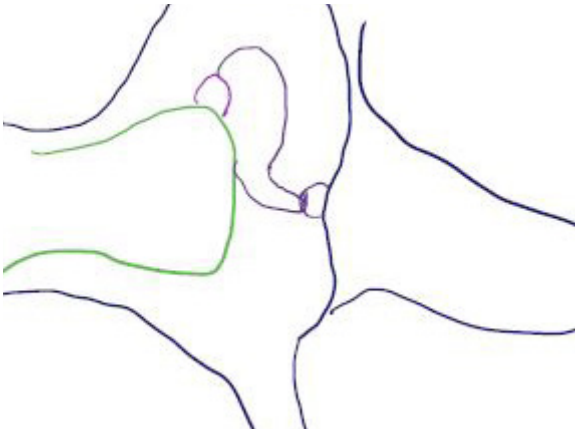
Graft shown positioned



Tympano meatal flap shown repositioned

Type II tympanoplasty:

In this procedure the graft is placed over the intact incus and stapes. This procedure is rarely used because it is rare for erosion of the handle of malleus to occur alone without involvement of other ossicles. The neo-tympanum is draped over the existing incus and stapes. This involves a certain amount of obliteration of middle ear space. Since the ossicular lever ratio is normally maintained after this procedure these patients tend to have atleast 30 dB conductive hearing loss even after a successful surgery.



Type II tympanoplasty

Type III tympanoplasty:

This technique is used only when a mobile suprastructure of stapes alone is present. In this procedure the tympanic membrane graft is draped over the mobile suprastructure of stapes. This is also known as Columella effect. This type of middle ear is commonly seen in birds.

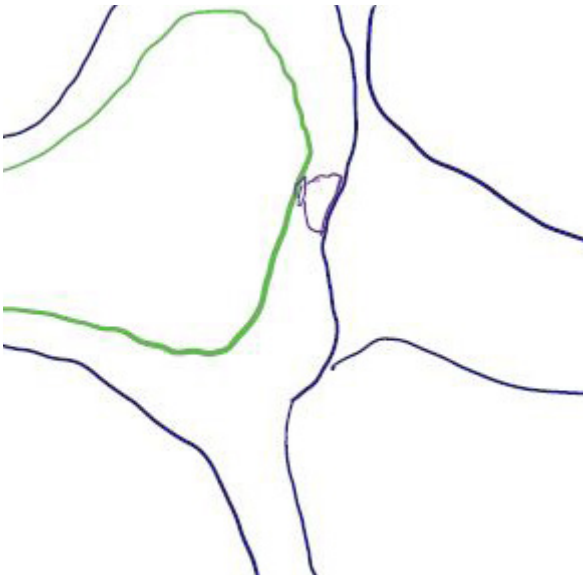


Figure showing Type III tympanoplasty

The middle ear space is really non existant. Even after successful surgery these patients still manifest 30 - 40 dB conductive hearing loss. This procedure is useful in patients without malleus and incus. It should be stated that the incus has the most precarious blood supply among the three ossicles.

Type IV tympanoplasty:

This procedure is performed in patients only with a mobile foot plate of stapes. The grafted ear drum is draped over the mobile foot plate. In these patients there is virtually no middle ear space at all. The grafted ear drum virtually drapes the promontory. Even after successful surgery these patients still have abovt 40 - 50 dB hearing loss. In this procedure the round window is protected from the incoming sound waves. This helps in preserving the round window baffle effect.

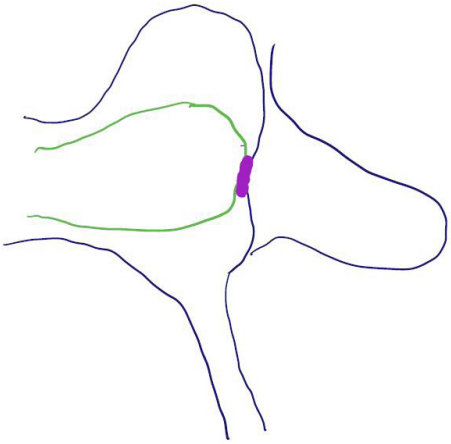
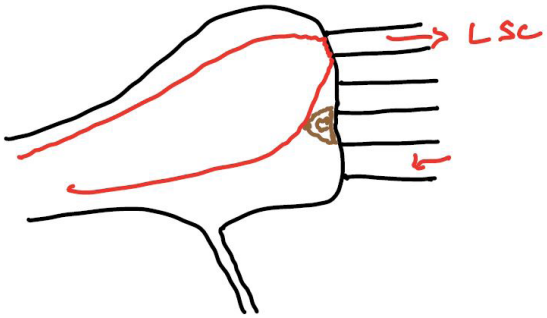


Figure showing Type IV tympanoplasty

Type V tympanoplasty:

In this surgical procedure a third window is created over the lateral canal (Lateral canal fenestration). This procedure is rather outdated these days.



Type V tympanoplasty

Belluci's prognostic classification:

Belluci used the status of the middle ear cavity in determining the prognostic features of tympanoplasty. He grouped those under 4 heads.

Group I:

Patients with dry ear for a period of atleast 6 months fall in this category.

Group II:

Patients with occasionally draining ear were included in this group

Group III:

Patients with persistent ear drainage associated with mastoiditis were included in this group

Group IV:

Patients with persistent ear discharge associated wtih palatal malformations (cleft palate) were included in this group

Ossicular grafts have revolutionized tympanoplasty procedure these days. These grafts help in the preservation of middle ear space thereby producing excellent improvement in hearing.

Implants used for ossiculoplasty should satisfy four basic requirements:

1. They should be biocompatible and should not extrude / cause severe tissue reaction

2. They should improve / maintain hearing

3. They should be technically easy to use

4. They should maintain results over time

Austin in 1971 classified the anatomical defects found in the ossicular chain due to chronic suppurative otitis media. Isolated losses of handle of malleus and stapes suprastructure were not included in this classification due to their rarity.

Type I Normal = M+I+S

Type II = M+S Absent incus - Good prognosis

Type III = Malleus + Foot plate of stapes - Poor prognosis

The forerunner of partial and total ossicular replacement prosthesis was Dr Austin's Polyethylene malleus to foot plate strut. He designed the "Sunflower columella" designed out of teflon. Teflon and polyethylene have the advantage of excellent air bone closure.

The following are the various categories of biomaterials used in ossiculoplasty:

1. Polyethylene tubing

2. Polytetrafluoroethylene (Teflon)

3. Gelatin foam (Gelfoam)

4. Silastic (Dimethyl silicone polymer)

5. Platinum - This material is very ductile, non magnetic and biocompatible

6. Titanium alloy

7. Polycel and plastipore

8. Capsel - Hydroxyapatite

9. Otocel - Clear bioactive bioglass (ceramic material)

Selection of prosthesis:

Factors to be considered while selecting an optimal prosthetic design are:

1. Status of ear drum

2. Status of residual ossicles

3. Severity of eustachean tube dysfunction

4. Stability of prosthesis

5. Ease of placement

6. Sound conductivity

Comparison of prosthetic materials

Parameter	Cartilage	Autologous bone	Homologous bone	Plastic	Hydroxy apatitie
Biocompatibility	++	+++	+++	+	+++
Storage	+++	+++	+	+++	+++
Sound conduction	+	+++	+++	++	+++
Ease of preparation	+	+	++	+++	++
Transmission of disease	-	-	Possible	-	-
Interposition graft	-	-	-	Required	-

Stapes to malleus reconstruction:

When malleus is present it can be used to help to stabilize the prosthesis and reduce the possibility of extrusion. The malleus is never directly aligned to the underlying stapes (M-S offset). A variety of implants have been designed to take advantage of the stabilizing effect of malleus.

Incus interposition:

Guilford transposed the residual incus autograft on to its side so that it lies on the stapes capitulum and beneath the manubrium. Hearing results could be excellent if the middle ear anatomy is favorable. The incus remnants could be too short orlong. Too long incus prosthesis could lead to ankylosis. Revision surgery is difficult in such patients owing to fixation of the prosthesis to the stapes and fallopian canal.

Zollner's sculpted incus: Zollner popularized the sculpturing of Autologous incus. This helps in obtaining a better fit. It also reduces the incidence of subsequent ankylosis. Weher's refined this technique to include homograft ossicles. This technique could be time consuming. Remnant Autologous incus could harbor cholesteatoma.

Grote Hydroxyapatite assembly: Grote developed the first commercial Hydroxyapatite prosthesis. Its configuration attempted to accommodate the M-S offset. This prosthesis should be placed lateral to the malleus necessitating dissection of the ear drum away from the malleus. There is also the associated risk of iatrogenic perforation of the ear drum.

Wehr's Hydroxyapatite prosthesis: Wehr's advocated sculpted homograft for incus interposition. He also developed Hydroxyapatite incus prosthesis in order to reduce the preparation time inside the operation theatre during ossiculoplasty procedures. This prosthesis had an anterior extension which was created to cradle the malleus. Biocompatibility of this material was really superior.

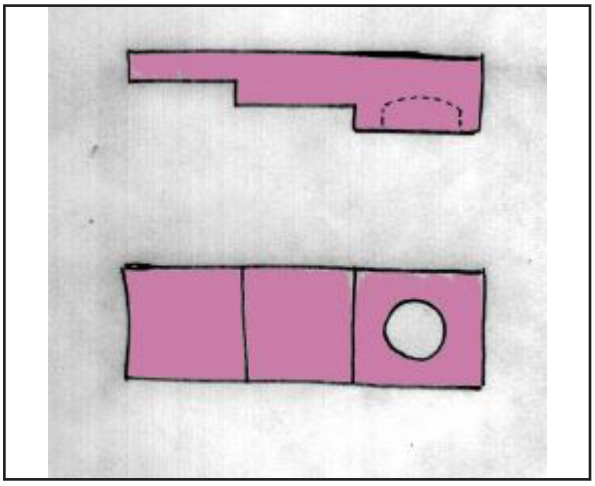


Figure showing Grote prosthesis

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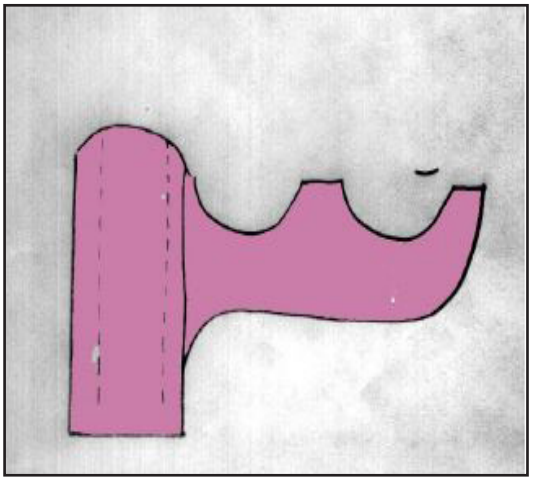

Figure showing the Wehr's prosthesis. The anterior cradle supports the malleus

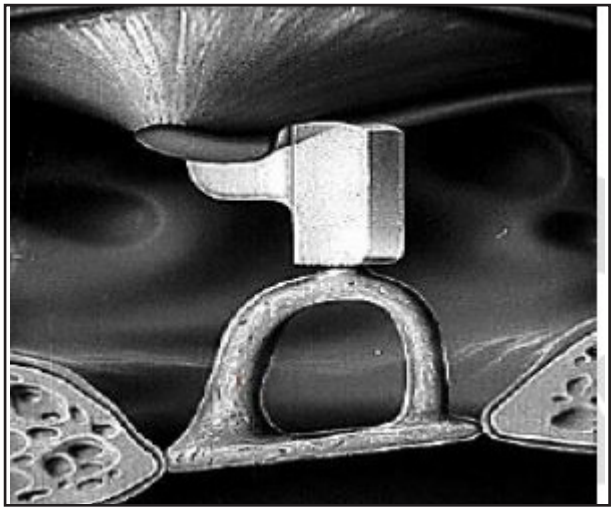

Figure showing Weher prosthesis in position

There are two types of Weher's prosthesis:

1.Incus replacement prosthesis

2.Incus – Stapes replacement prosthesis

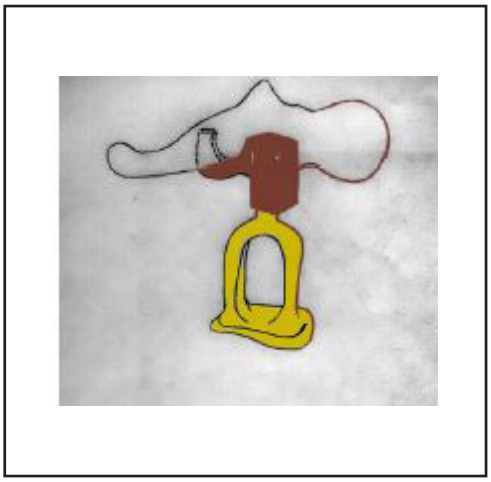

Figure showing incus replacement prosthesis

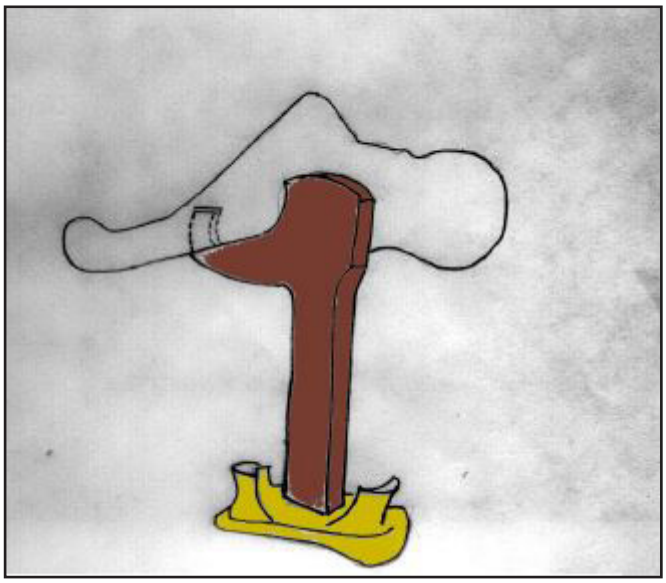

Figure showing incus – stapes replacement prosthesis

Kartush Hydroxyapatite struts: These struts were designed to function as either a TORP or PORP. Hydroxyapatite was used. This prosthesis has a self locking mechanism. It has very low displacement and extrusion rates.

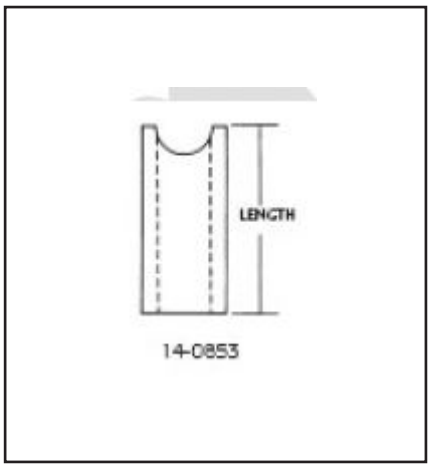

Figure showing Kartush prosthesis

Incus interposition ossiculoplasty: Incus due to its precarious blood supply commonly undergoes necrosis, especially its long process. Homograft incus was shaped and placed between the malleus and stapes head. A notch was created in the short process of the incus that fit under the malleus handle. This is done to stabilize the ossicle. If the stapes suprastructure was intact in the patient, the long process of incus was amputated. A small cup was made in the amputated long process of incus. The head of the stapes fits into this cup. The notch prevented the prosthesis from being displaced anteriorly / posteriorly. The spring in the patient's malleus would keep the prosthesis from being displaced inferiorly. Superiorly its position is maintained by the contraction of tensor tympani tendon.

When the stapes superstructure is absent, the long process of incus could be placed over the foot plate of stapes.

Pitfalls: With AID's being common these days, incus homograft has given way to artificially designed prosthesis. Hydroxyapatite was commonly used to design these prosthetic incus replacements.

Factors that should be taken into consideration before designing the optimal prosthesis:

1. Proper tension is very important. A prosthesis that makes tension adjustment easy for the surgeon should be advantageous.
2. Prosthesis with masses less than 40mg is best for overall acoustic performance.
3. For improved high frequency performance, rigid low mass prosthesis (less than 10g) is the best choice.
4. Longer prosthesis produces excellent high frequency function at the expense of low frequencies.
5. Prosthesis that connects malleus to stapes appears to have no acoustic advantage over prosthesis that connects the ear drum to the stapes.
6. If the ear drum is conical, prosthesis with the head angulated at about 30° appears to be beneficial because the angulation increases the surface area in contact with the ear drum.

These prostheses may be used to reconstruct the ossicular chain during Tympanoplasty, in patients in whom erosion and discontinuity of ossicular chain has occurred. Long process of incus gets frequently eroded because of its precarious blood supply. In these cases the lenticular process of incus is still attached to the head of stapes. The incudo stapedial joint in these patients should be separated and the long process of incus removed. This is done because squamous debris could still be attached to the incus fragment. It is also preferable to remove the body of the incus, because it could also have squamous ingrowth. It can also have scar tissue blocking the antrum.

Surgical procedure:

The prosthesis is laid on its side on the promontory. The cup of the prosthesis is near the stapes and its notched portion close to the tip of the handle of malleus. With the help of right-angle pick held in the surgeon's left hand, the malleus is elevated, and with a gently curved pick in the surgeon's right hand, the prosthesis is brought up under the manubrium of the malleus. As it is brought to an upright position, the cup engages the head of stapes.

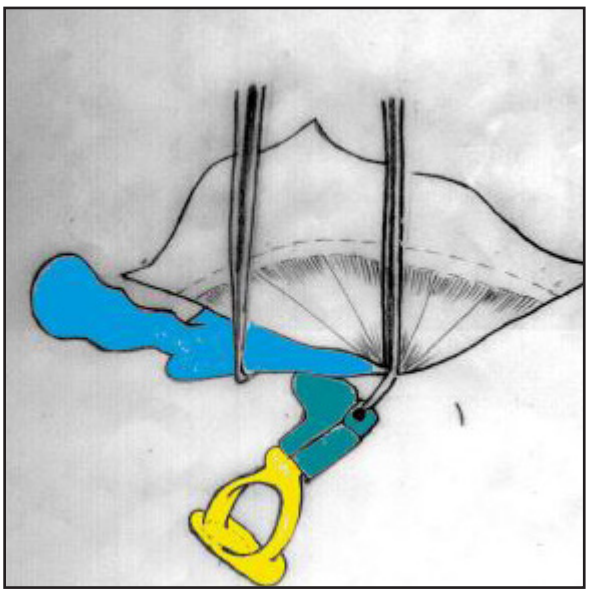

Figure showing the prosthesis being positioned

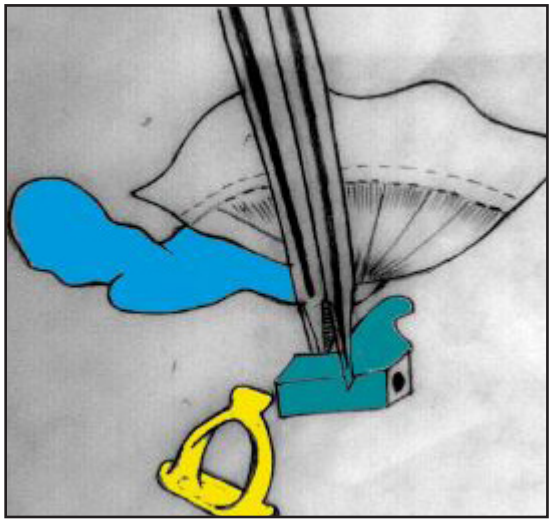

Figure showing the prosthesis laid on its side on the promontory

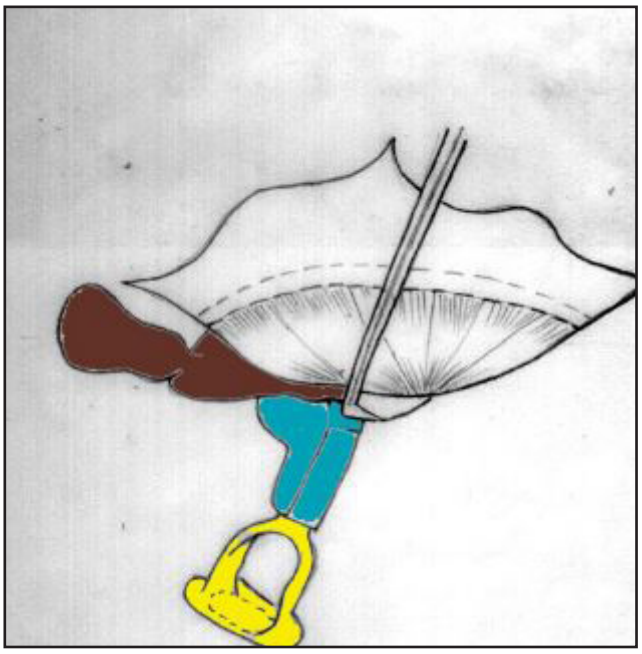

Figure showing the prosthesis in its final position

Ossicular reconstruction with prosthesis of Hydroxyapatite should not be attempted in cases of acute trauma / traumatic perforation of ear drum. It should be performed only after the drum has healed and stabilized.

Complications:
Owing to the biocompatibility of this prosthesis, the incidence of complications is rare.
1.Extrusion of the prosthesis.
2.Too short / Too long prosthesis could lead to increased extrusion rates
3.Failure to improve hearing

The success or failure of ossiculoplasty procedure could be assessed by calculating the Middle Ear Risk (MER) Index. In this index a value is assigned for each risk factor, and these values are added to determine the MER index.

According to MER:

0 – Best prognosis
2 – Mild risk
5 – Moderate risk
7 – Severe risk
12 – Worst prognosis

Ossiculoplasty using presculptured banked cartilage: Homologous cartilage can be sculptured prior to surgery into TORP / PORP configuration. They can easily be stored by a tissue bank for use at a later date. It is configured in a self stabilizing manner with a disk shaped upper surface.

Donors should be screened serologically for Hepatitis and HIV antigens. Costal cartilage is ideal for this purpose. Graft material is harvested from the costochondral cartilages. These cartilages are fashioned into TORP type implants. The classic TORP configuration is about 8 mm long. It has a disk like head of about 4 mm diameter. The diameter of the shaft should be 2 mm in diameter.

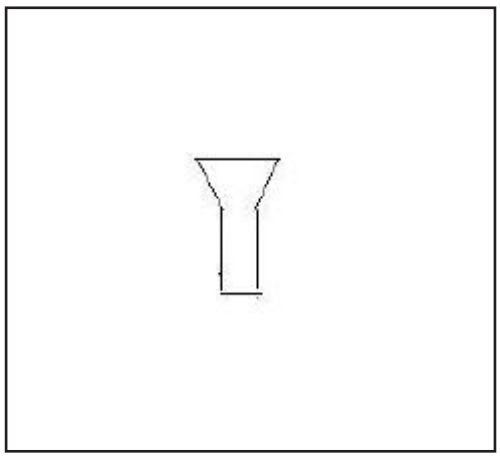

Figure showing PORP configuration to be used when malleus is absent

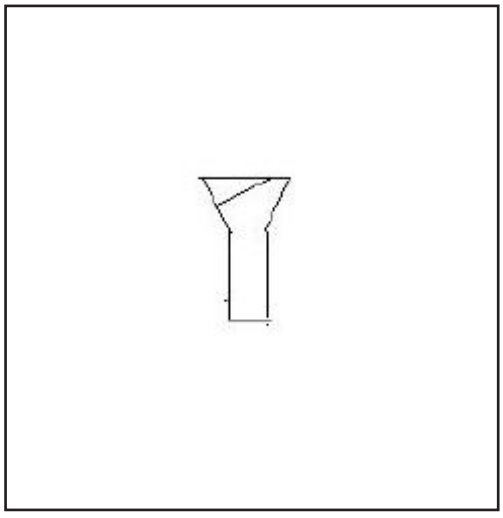

Figure showing PORP configuration to be used when malleus is present

MER Index

Risk Factor	Risk value
Otorrhoea (Belluci criteria)	
1. Dry	0
2. Occasionally wet	1
3. Persistently wet	2
4. Wet ear with cleft palate	3
Perforation	
Absent	0
Present	1
Cholesteatoma	
Absent	0
Present	1
Ossicular status (Austin)	
M+I+S	0
M+S	1
M+S-	2
M-S+	3
M-S-	4
Ossicle head fixation	2
Stapes fixation	3
Middle ear: Granulations / Effusion	
No	0
Yes	1
Previous surgery	
None	0
Staged	1
Revision	2

The disk like top of the implant can be placed in contact with the posterior bony annulus for added stabilization. It is better to thin the cartilage in the area of contact with the annulus, thereby minimizing the potential for dense adhesions.

TORP configuration: Ossicular reconstruction in the absence of stapes suprastructure is technically more demanding. Cartilaginous homografts are effective if the patient has a wide oval window niche. Measurements are taken as described for PORP configuration. The length of the shaft should be trimmed and contoured as per requirements.

If there is a perforation in the tympanic membrane that corresponds with the location of the disk shaped head of the reconstruction prosthesis, the head of the prosthesis itself can be used as a graft for the perforation. The surface of the TORP readily epithelializes.

Advantages of presculptured homograft cartilage as prosthesis:

1. The incidence of graft extrusion is rare
2. Contact of the implant with adjacent bony walls of middle ear can be consistent with excellent hearing results, because the cartilage remains flexible.
3. Hearing improvement is excellent
4. Operating technique is less demanding when presculptured cartilage homograft is used.

Ossiculoplasty with composite prosthesis: PORP's and TORP's designed out of composite materials was first popularized by Sheehy and Shea. Major advantage of using synthetic graft is there is no fear of transmission of diseases like HIV and Hepatitis.

Composite prosthesis has two distinct portions: a Hydroxyapatite head and a plastipore or fluoroplastic shaft. The Hydroxyapatite head is a universal design, and no modification or intraoperative reshaping is required. The plastipore shaft is manufactured in such a way that it can be precisely trimmed to within a 0.5 mm variance on the basis of intraoperative measurements.

The type of Hydroxyapatite head that should be used in the prosthesis depends upon whether malleus is present or absent. In cases where malleus is present, the head of the prosthesis used should be in the form of a delicate hook. It is designed in such a way that the hook is positioned under the handle of the malleus. The Hydroxyapatite head to be used when the malleus is absent has a flat, egg shaped design, with gently rounded edges. This design facilitates easy insertion under the ear drum without the need for cartilage interposition. This prosthesis is best used when the middle ear is healthy and free of disease.

The plastipore shaft is of two types:

1. Type I: The shaft has a hollow sleeve to accommodate the head of stapes
2. Type II: The shaft is more slender, wire reinforced. This design helps the shaft to rest directly on the foot plate of stapes / oval window.

There are 4 types of composite prosthesis designed to solve the four basic problems encountered during ossicular reconstruction. These situations include:

- Malleus present, stapes present
- Malleus present, stapes absent
- Malleus absent, stapes present
- Malleus absent, stapes absent

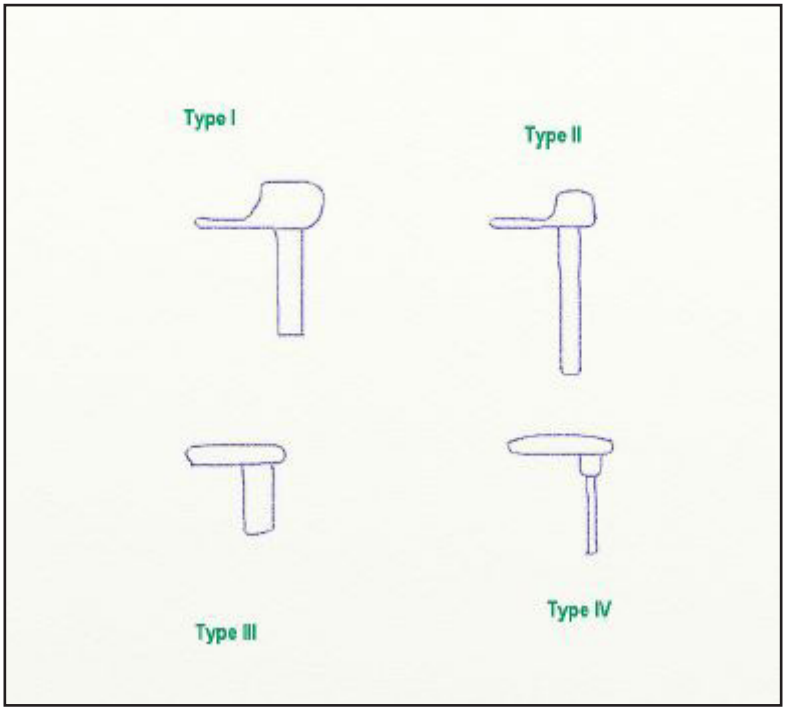

Figure showing the types of composite prosthesis

Contraindications for composite prosthesis:

1. Should not be used in patients with severe Eustachian tube function
2. Should not be used in patients with an obliterated middle ear space
3. Middle ear mucosa should be healthy and free of any disease

Cartilage harvested from rib is cut into 8 mm sections. They are then placed over sterile hard surface. Using a 4mm disposable dermal punch cylinders of cartilage are created each with 4 mm diameter and 8 mm long. From these cylindrical grafts, appropriately shaped TORP's can be prepared. Cartilage material can be placed in sterile saline and put in glass specimen sterile bottles and sealed with a plastic seal.

PORP configuration: When stapes is present and mobile, a measurement is taken from the lateral most part of the capitulum of the stapes to the ear drum. 1 mm should be added to this value, and the TORP blank cartilage is trimmed to this measurement. A depression is made in the end of the shaft of the trimmed blank to accommodate the head of the stapes. The depth of this indentation could be about 0.5 – 1 mm. The 4 mm disk of the top of the implant should be in complete contact with the ear drum. If an intact malleus handle is present, the anterior most portion of the head of the implant can be trimmed to fit the handle. If the malleus handle is absent, a more flat configuration can be used.

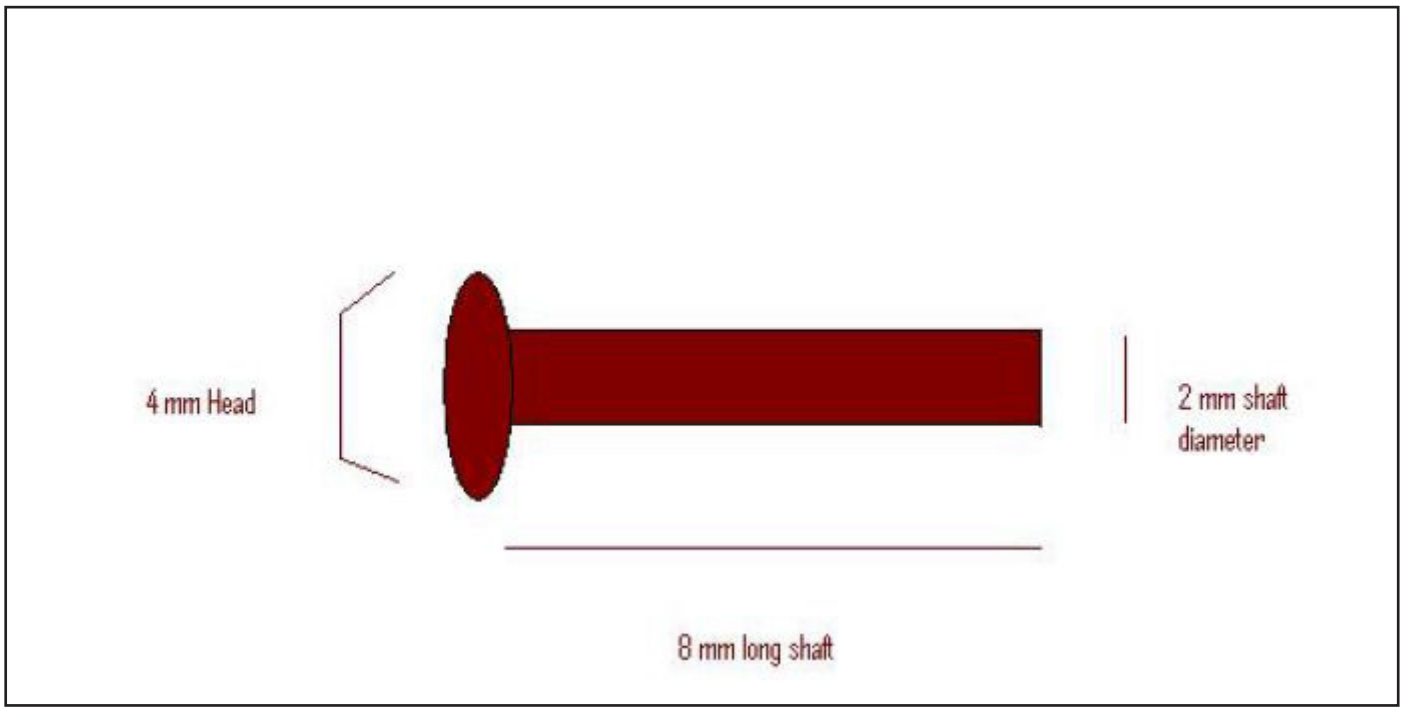

Figure showing standard TORP configuration

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Spandrel: This is a type of TORP. It has a wide head which can be slid under the ear drum and a narrow shaft. The length of the shaft can be reduced by cutting it. The shaft rests over the foot plate of stapes. Parts of spandrel: It has a perforated shoe to allow protrusion of the wire core. It has a thin flange on the prosthesis head to avoid possible damage induced by a sharp edge of the Polycel disk.

Before assembling the prosthesis, air is removed from the Polycel casing by connecting the prosthesis and its shoe to a syringe containing Ringer's solution and antibiotic.
This prosthesis ensures better closure of air bone gap.

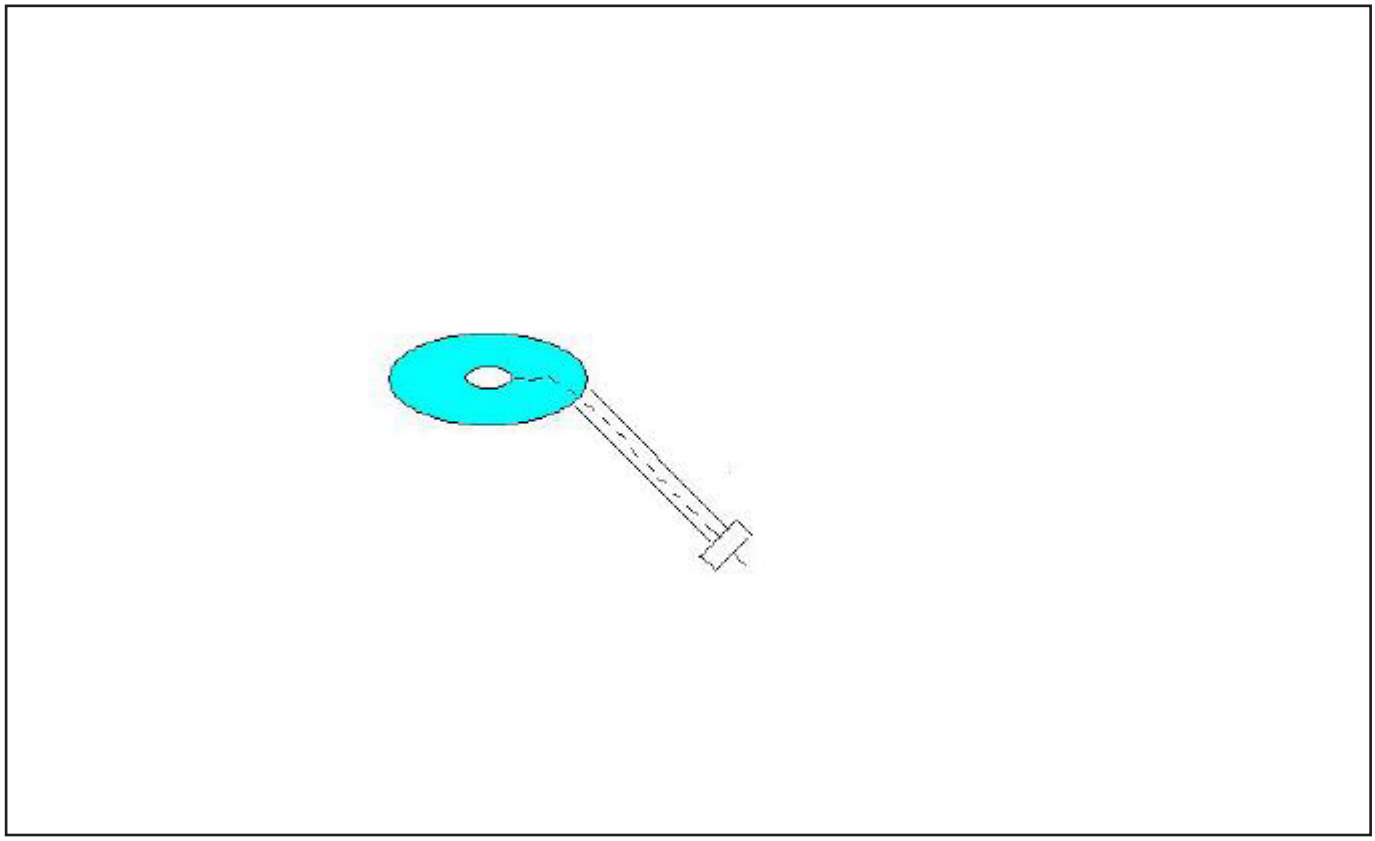


Figure showing a Sprandel

Complications of Mastoidectomy

Many vital and delicate structures are contained in the ear. Many more vital and delicate structures are in close relationship with the ear within the temporal bone. All these vital structures are at risk during surgeries of temporal bone.

The advent of operating microscope and precision drilling machines has reduced the number of complications drastically. Still iatrogenic complications do occur, most of these are caused by anatomical anamolies which the surgeon had failed to appreciate. These complications do have an impact on patient's lives as well as medico legal issues.

Severe iatrogenic major complications could be:

1. Injury to facial nerve

2. Injury to labyrinth

3. Injury to Dura

4. Injury to critical blood vessels

Injury to facial nerve:

This could be the most devastating complication following mastoid surgery. This is commonly encountered when associated with congenital malformation of the ear. The tympanic segment of facial nerve, mastoid segment of facial nerve and the second genu of facial nerve are could be injured commonly in this order.

Vulnerability of tympanic segment of facial nerve is attributable to the fact that it is common for the nerve to be dehiscent in this area. Somtimes the nerve can herniate via the dehiscence simulating a polyp in its appearance.

The location of mastoid segment of facial nerve is highly variable especially with regard to its lateromedial extension.

Mastoid drilling or simple uncovering of the facial nerve from granulations can lead to facial palsy. Bony uncovering of the nerve can cause transient facial palsy. Complete transection of facial nerve is rather rare and should immediatly be repaired by using nerve transposition graft. Nerve transposition generally does not require nerve suturing when performed in the mastoid or typanic segment areas because the fallopian canal provides a cradle for proper seating of the nerve graft. Fibrin glue can be used to stabilize approximating surfaces.

Commonly iatrogenic facial palsy is identified only post operatively after the effects of local anesthesia has completely worn out. If the surgeon is fairly confident that the facial nerve was identified during the course of surgery and was not damaged then the best option would be to wait. It could eventually turn out to be neuropraxia. It should be stressed that the fallopian canal is highly restricting and hence gives very little space to accomodate nerve swelling due to oedema. In the event of a small segement of fallopian canal getting deroofed during the course of surgery it would be prudent on the part of the surgeon to deroof a large segment of fallopian canal so that the facial nerve will be provided with more space for expansion and neuropraxia will not occur. If this is not done swelling involving facial nerve in a small segment will cause neuropraxia due to compromise of blood circulation to the nerve due to compression caused by swelling within the confines of the fallopian canal.
Majority of nerve injuries are known to occur when the facial nerve has not been identified during the surgical procedure.

Injuries to facial nerve during surgery may be caused due to errors of ommission and commission. Errors of ommission include inadequate patient preparation for surgery leading on to excessive bleeding on the table causing difficulities for the surgeon to identify the facial nerve. Errors of commission include inadequate exposure of surgical field.

The neural landmarks should be identified early during surgery if facial nerve is to be preserved. Landmarks could be difficult to identify in revision surgery as they could have very well have been damaged during the earlier surgical procedure. Surgeon should always ensure that neural landmarks are left undisturbed during surgery.

Wide exposure by removing large amounts of bone would help in identification of neural landmarks like:

1. Cog

2. Cochleariform process

3. Oval window

4. Lateral canal

5. Digastric ridge

Cog is one useful lankmark which can be reliably used to identify the location of facial nerve even in diseased ears as it is not eroded even in cholesteatomatous ears.

On identification of the nerve the drill should always be used parallel to the course of the nerve and never perpendicular to the nerve.

Cog is as stated a useful landmark for the position of facial nerve. It is a bony projection that detaches from the tegmen tympani cranially coming down vertically towards the cochleariformis. When fully formed it is a constant landmark for geniculate ganglion.

Anatomical importance of Cog:

1. It is the medial projection of transverse crest

2. The transverse crest can cause changes to attic dimensions due to its varying positions. The transverse crest starts from the anterior tympanic spine and crosses the tegmen transversely 1-2 mm in front of the head of malleus. Its medial leg can continue up to the cochleariformis and is also known as the cog. This transverse crest divides anterior epitympanum into two portions i.e. anterior and posterior. Both these spaces merge inferior to this crest to form one large space.

3. Cog when fully formed is a reliable landmark for the geniculate ganglion of facial nerve

4. Cog is considered to be a junction between saccus anticus and saccus medius

5. Commonly vertical tensor fold is attached to cog. This fold can be complete or partial. If vertical tensor fold and horizontal tensor folds are complete then it would lead to ventilation block to anterior attic. In this scenario cog will have to be removed in order for anterior epitympanic space to be ventilated.

6. Cog roughly indicates Fisch plane or meatal plane

7. Cog is a rough landmark for internal acoustic canal on medial side

Cutting burrs are less dangerous than diamond burrs because cutting burrs need less pressure to cut and hence generate less hear whereas diamond burrs need more pressure to remove bone and there by generate that much more heat causing damage to nerve sheath. When nerve sheath needs to be decompressed then diamond burrs can be used in that area combined with profuse irrigation with saline in order to dissipate generated heat.

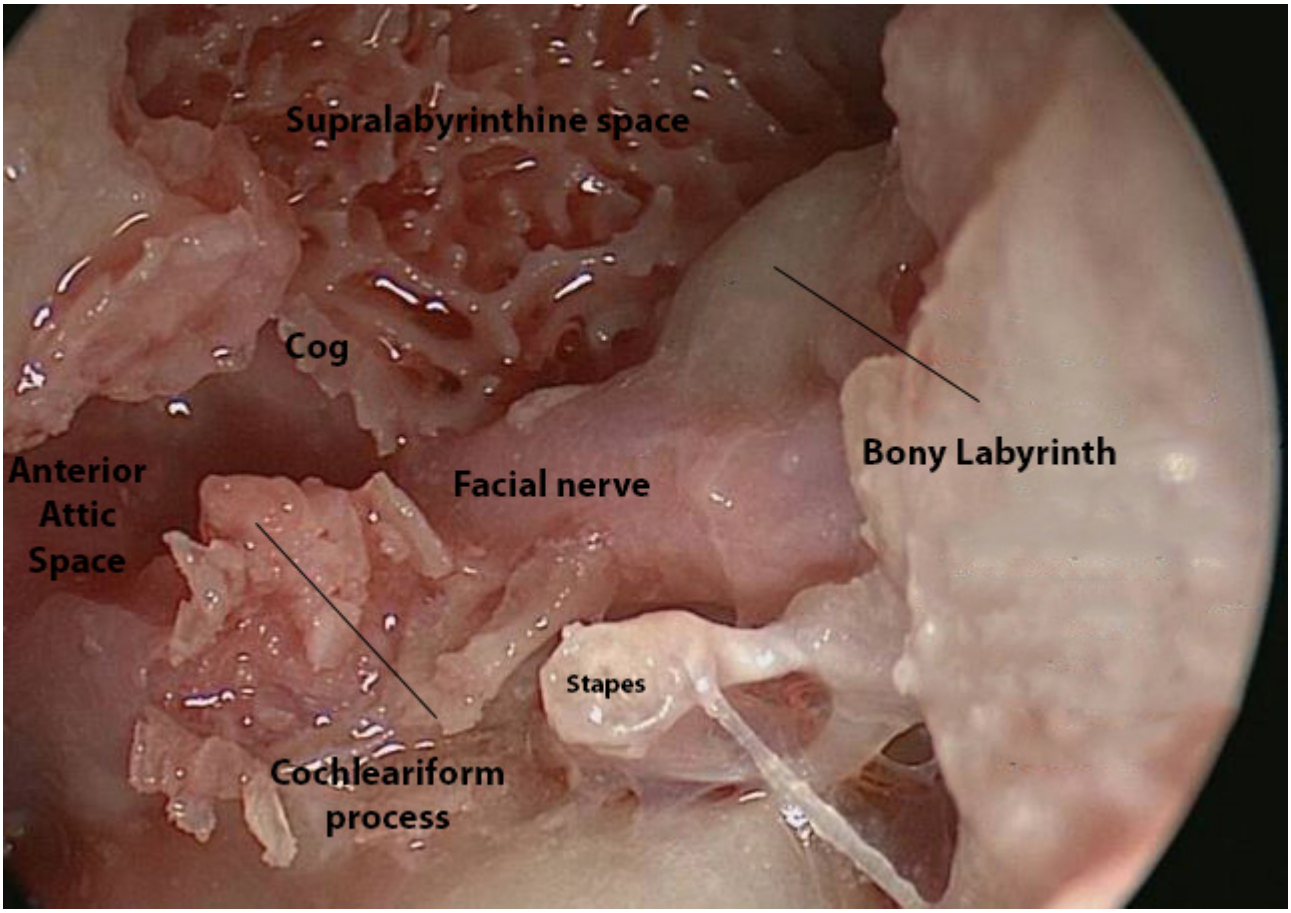


Figure showing cog and its relationship to facial nerve

While drilling in the facial nerve area the size of the burr used should be larger than that of the nerve.

Art of avoiding damage to facial nerve during mastoid surgery:

Identifying the facial nerve is fundamental to performing a safe mastoid surgery. In intact canal wall mastoidectomy the space between the facial nerve and chorda tympani nerve (facial recess) provides access into the hypotympanic portion of middle ear cavity. This is the commonly preferred approach for cochlear implant surgery. In canal wall down mastoidectomy identification of facial nerve allows the surgeon to lower the facial ridge adequately creating more space which could facilitate complete removal of cholesteatoma.

Various anatomial fixed landmarks can be used to identify the position of facial nerve. The various landmarks to identify the intra temporal portion of facial nerve can be divided into major and minor landmarks.

Major landmarks:

1. Lateral semicircular canal

2. Short process of incus

3. Posterior bony external auditory canal

Minor landmarks:

1. Digastric ridge

2. Processus cochleariformis

3. Oval window

It would be confusing for a surgeon to locate the facial nerve in patients with:

1. Recurrent disease in whom revision surgery is contemplated. In these cases the landmarks could be destroyed.
2. Brittle mastoid bone as in the case of fibrous dysplasia.
3. Contracted mastoid.
4. Huge cholesteatoma.
5. Anatomical variations of facial nerve

Surgeon should remember that the key landmark while performing mastoid surgery is identification of antrum with the dome of lateral semicircular canal forming its floor. In well pneumatized mastoid cavity the location of antrum is fairly straight forward. This becomes rather tricky in sclerosed mastoid bones. Locating antrum during surgery is vital not only for disease control but also to identify the location of facial nerve.

The total length of facial nerve inside the temporal bone is 30 mm. Within the temporal bone the facial nerve is divided into three segments i.e.

Labyrinthine segment:

This segment runs laterally from the fundus of the internal auditory canal until it reaches the geniculate ganglion. The geniculate ganglion is located superior to the cochlea beneath the middle cranial fossa dural plate.

Tympanic segment:

This segment of facial nerve can be identified along the inferior margin of lateral semicircular canal. Hence the dome of the lateral canal is an important landmark. The nerve here runs from antero superior to postero inferior direction. This segement of facial nerve canal may be dehiscent leaving the nerve naked within the tympanic cavity. Close to the posterior edge of oval window and medial to the short process of incus the nerve follows a gentle curve in an inferior direction. This is known as the second genu. This is where the nerve is commonly traumatized during mastoid surgery.

Mastoid segment:

This portion of the facial nerve segment traverses in a vertical direction towards the stylomatoid foramen. Uncapping this segment of the nerve should be done in a retrograde manner starting from the stylomastoid foramen.

Ths digastric ridge and stylomastoid periosteum should be exposed in order to identify the course of mastoid segment of facial nerve. Another important anatomical landmark for identifying the mastoid segment of facial nerve is the inferior edge of posterior semicircular canal. The pyramidal segment of facial nerve is situated 2mm anterior and lateral to the inferior edge of posterior semicircular canal.

Two grooves are drilled along the superior and posterior external auditory canal wall in order to find the antrum. Antrum is ideally located at the intersection of both these grooves. The initial burr cut is made along th linea temporalis which happens to be the lowest points of the middle fossa dura in most cases. The second burr cut is made along a line perpendicular to the one described above and tangent to the posterior margin of the external auditory canal. The apex of this triangle is directly over the lateral canal. Drilling directly over Maceven's triangle and enlarging it progressively towards the mastoid tip will ensure easy identification of antrum with minimal risk of damage to facial nerve.

The mastoid segment of facial nerve which is about 15 mm long is highly vulnerable during mastoid surgeries as its course is highly variable. This segment of facial nerve lies lateral to stapedius muscle and the sinus tympani. The mean distances between the posterior borders of oval and round windows from that of facial nerve is about 4 mm. This anatomical fact should always be borne in mind while working on this portion of the mastoid bone. The lower third of mastoid segment of facial nerve lie antero medial to digastric ridge and the distance could range between 2 - 5 mm.

The course of the mastoid segment of facial nerve showed variations in its descent towards stylomastoid foramen. In close to 60% of cases the descent is vertical towards the stylomastoid foramen and in 20% of cases it is known to descend slightly laterally or medially. This segment courses near the line between the short process of incus and the digastric ridge in the posterior wall of tympanic cavity.

The surgeon should be able to identify the facial nerve at any segment using the available landmarks. Anatomical variations should also be taken into consideration.

In the oval window area commonly facial nerve lies inferiorly. This should always be remembered while working in this area.

Two things stand out while one is close to decapping the facial nerve canal:

1. Pilot bleeding - Frequent troublesome bleeding while drilling in this area. These blood vessels are intimately associated with facial nerve canal.

2. Before actually uncapping the nerve as such the surgeon would notice whitish nerve sheath showing through thin bony lining of fallopian canal.

In order to avoid injury to facial nerve the operating surgeon should be fairly clear about the normal anatomical course of facial nerve. Any anatomical abnormalities should be anticipated well in advance before the surgery. Intraoperative facial nerve monitoring should be used in difficult anatomical situations and while performing revision surgeries. Intraoperative facial nerve monitoring should not be considered to be a replacement of through anatomical knowlege of the nerve course. Utmost it can be considered to add one more level of safety.

Injury to labyrinth and sensorineural hearing loss:

Iatrogenic trama to labyurinth associated with sensorineural hearing loss is quite a possibility following mastoid surgery. It is rather infrequent with wide spread use of operating microsope and micro drills. Studies reveal that three sites are most vulnerable to injury during mastoid surgical procedures. These include:

Lateral semicircular canal

Promontory

Oval window

Iatrogenic breach of membranous labyrinth is rather treacherous and hearing is invariably lost. Sometimes hearing may also be preserved despite breach of membranous labyrinth. Injury to lateral semicircular canal in the absence of cholesteatoma has somewhat better prognosis. These patients have stable hearing. A permanent mild, high frequency hearing loss was common. Speech discrimination scores were within normal range in these patients. Vertigo and nystagmus are also seen.

If the lateral canal is injured close to its posterior limb, then symptoms could vary from hemorrhage and serous labyrinthitis.

Injuries near the vestibule would cause contamination of perilymph by endolymph causing permanent damage to hearing. Cochlear preservation could occur in these patients possibly due to collapse of the walls of membranous labyrinth due to loss of fibrovascular support and sudden loss of endolymph sealing off the breached area. Jahrsdoerfer et al has different theory to account for hearing preservation in these patients. According to them there is a closure of pars superior from pars inferior near the utriculoendolymphatic valve. This closure prevents excessive loss of endolymph and admixture of endolymph with perilymph. Sometimes acute loss of endolymph from the vestibule couyld cause the untricular wall to collapse closing off the valve area thereby protecting the cochlea from acute decompression.

Sealing the fistula with fat, bone wax, temporalis fascia could help in saving hearing in these patients. If trauma is identified on the table and sealing is done immediatly then the chances are brighter for complete recovery to occur.

Accidental opening of oval window is another area of labyrinthine injury. This could be caused due to inadvertant avulsion of foot plate of stapes during the surgical procedure or due to drilling in the area. These patients are better managed on the table by sealing the oval window using fat and temporalis fascia graft.The following steps may be taken to protect the oval window while removing cholesteatoma from that area:

1. Cholesteatoma should be removed by dissecting parallel to stapedius tendon to steady the stapes. Manipulating stapes in a superoinferior direction or by depressing the foot plate would damage annular ligament causing disruption of stapes.

2. Granulation tissue / inflammed mucosa from foot plate need not be removed as a routine and would heal when the middle ear heals. This would minimize damage to foot plate area.

3. If dissectiom in the area of oval window gets difficult then it should be stopped and the tympanic membrane if any closed with temporalis fascia graft. A second look operation can be performed after 6 months during which time epithelial pearl formation should have taken place over remnant cholesteatoma. It is easy to remove epithelial pearl from the foot plate area.

4. Opening if created should immediatly be covered with temporalis fascia. Suction should not be applied in this area.

Other causes of sensorineural hearing loss following mastoidectomy:

1. Excessive manipulation of ossicular chain during surgery

2. Transmission of vibrations from burr bit to inner ear via foot plate of stapes

Dural injury:

Dural exposure could occur during mastoid surgery. This usually occurs when the the tegmen tympani and tegmen mastoideum are being thinned. When dura is exposed it causes bleeding. Dural bleeding can be arrested by cautery or by temporarily packing the area. When a small area of dura is exposed it can be ignored after arresting the bleeding. Laceration of dura causes CSF leak and herniation of brain into the mastoid cavity. Breach of tegmen does not does not perse induce meningeal / menigoencephalic herniation unless it is associated with dural breach. Exposure of dura and CSF leak can be avoided by using diamond burr while drilling the tegmen plate. In some cases the disease process i.e. cholesteatoma can thin out the tegemen plate.

Dural tear / abrasion can lead to:

1. CSF leak

2. Meningitis

3. Encephalocele

4. Meningoencephalocele

5. Cerebral abscess

6. Pneumocephalocele

7. Fungus cerebri

A tegmen breach cannot by itself induce meningeal / menigoencephalic herniation. For herniation to occur it should be associated with dural breach.

Dural breach should immediatly be sealed on the table using sutureless technique using Fat plug and fascia lata

Fungus cerebri:

Fungus cerebri is protrusion of brain and dura out of the cranial cavity into the mastoid and middle ear cavity. This is a rare complication.

During early days this complication occurred when brain abscess complicating mastoiditis was drained through mastoidectomy. In these patients cerebral / cerebellar herniation developed either behind the ear or into the external auditory meatus depending on the approach used.

Predisposing factors:

1. Previous ear surgery

2. Meningitis

3. Erosion of tegmen plate by cholesteatoma

4. Active chronic otitis media

Brain herniation may be associated with CSF leak.

This condition is managed by wide surgical exposure, amputation of necrotic herniated brain tissue, water tight closure of dural defect using fat and facia lata graft.

Bleeding:

Bleeding following mastoidectomy commonly occurs due to injury to dura, sigmoid sinus. Rarely fatal bleeding could occur in patients with aberrant carotid artery.

Bleeding can be controlled by using packs and cautery.

Otosclerosis

Synonyms: Otospongiosis, Ankylosis of foot plate of stapes.

Definition: Otosclerosis is a hereditary localised disease of the bone derived from the otic capsule characterised by alternating phases of bone resorption and new bone formation. The mature lamellar bone is removed by osteoclasis and replaced by woven bone of greater thickness, cellularity and vascularity.

History: In 1741 Antonio Valsalva described ankylosis of stapes while doing a postmortem on the body of a deaf patient.
In 1894, Adam Politzer introduced the term "otosclerosis" and described the histopathology of the disease for the first time.
In 1912 Siebenmann introduced the term otospongiosis to denote active otosclerotic foci.

Pathophysiology:

The primary pathological change occurs in the bony labyrinth with secondary effects upon middle ear and inner ear function. The otosclerotic focus may be asymptomatic, or if present in the area of foot plate of stapes it may give rise to ankylosis of foot plate with resultant conductive deafness. Otosclerotic foci may involve other portions of labyrinth causing sensori neural hearing loss and vestibular abnormalities.

A combination of effects are possible in otosclerosis. They are:
Histological otosclerosis: Otosclerotic foci doesnot cause any symptoms and hence known as histological otosclerosis.
Stapedial otosclerosis: is the classical otosclerosis with fixation of stapedial foot plate causing conductive deafness.
Cochlear otosclerosis: The foci involves the cochlea causing sensorineural deafness.
Combined otosclerosis: Here in addition to fixation of foot plate of stapes there is also associated sensorineural hearing loss due to involvement of cochlea.

Otospongiosis: European otologists prefer to use this term to indicate the active phase of otosclerosis.

Incidence: Otosclerosis is common in caucasian races. It is rarely found in Mongoloid and Negro population.

Sex incidence: In clinical practice otosclerosis is seen more often in women than in men. The ratio was found to be 2:1. Nowadays the authors believe the apparent female preponderance may be due to the fact that unilateral otosclerotic deafness is less common in women than in men. Noticeable deterioration in hearing also occur during pregnancy due to hormonal changes. Deafness due to otosclerosis may be initiated or made worse by pregnancy.
Causative factors / etiology: Many theories have been proposed to explain the etiological factors of otosclerosis. They are:

1. Metabolic

2. Immune disorders

3. Vascular disease

4. Infection (Measles) currently accepted

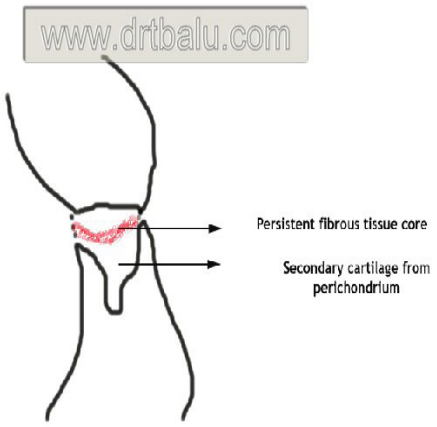
5. Trauma : The petrous bone doesnot have regenerative capacity. This is because of the fact that the enzymes released during reparative phase are very toxic to the inner ear hair cells.
Pockets of tissue capable of regeneration may be sequestered in various portions of labyrinthine bone. These tissue could be activated by the presence of regenerative enzymes in the blood following bone fracture elsewhere in the body.

6. Temporal bone abnormalities (congenital)

Genetic factors predisposing to otosclerosis: The tendency for otosclerosis to run in families has been documented. Authors have postulated an autosomal dominant mode of inheritance with varying degrees of penetration.
Otosclerosis is associated with osteogenesis imperfecta in 0.15 % of cases. This is known as Van der Hoeve syndrome or Adair - Dighton syndrome.

Sites affected by otosclerosis: The commonest site for apperance of otosclerotic bone is fissula ante fenestrum. This fissula is constantly seen in the labyrinthine capsule lying in front of the oval window. This area may contain unossified cartilage persisting even in adults. This area was referred to as Cozzolino's zone by Perozzi in memory of his teacher. Otosclerosis may occur in this area due to bony ossification of the cartilage.
Residual cartilage may be present in the following areas of labyrinth:
1. Fissula ante fenestram
2. Fissula post fenestram
3. Intracochlear
4. Round window
5. Semicirular canals
6. Petrosquamous suture
7. Base of styloid process

In normal development the fissula appears as fibrous connective tissue bundle joining the vestibule with the tympanic cavity. This fibrous tissue is encased in primary cartilage which later gets replaced by bone. From the fissula the bone acquires a connective tissue lining which later becomes converted into perichondrium. The fissula is reduced in size by the production of new secondary cartilage from the perichondrium. These changes are completed by birth. The secondary cartilage remains throughout life as a stable, dormant cartilage and hence may even be considered as normal structure. It is only when this secondary cartilage gets ossified otosclerosis occur (Bast & Anson).



Diagramatic representation of fissula ante fenestram

Otosclerotic changes may appear as a result of interaction between bone growth promoting substances circulating in the blood stream, and the unstable cartilagenous elements in the capsule of the labyrinth. Otosclerosis is often seen at times when the bone growth promoting substances are circulating in the blood as in pregnancy and following fractures of other bones.

Histopathology of otosclerosis:

The normal endochondral bone of labyrinthine capsule in which otosclerotic focus begins is compact in type. Ultrastructurally, lamellae composed of fine fibrils lying in a ground substance are concentrically disposed around haversian canals containing blood vessels and connective tissue. In otosclerosis there is sharply defined new bone formations that could be differentiated from normal bone by their deep carmine stain and by marked enlargement of bone spaces and haversian canals.

The following are the changes which occur in a otosclerotic foci:

1. Focal / diffuse replacement of normal compact bone by irregular, loose cancellous bone with more deeply staining lamellae.
2. There is an associated increase in size of Haversian canals, cell spaces and marrow spaces with corresponding increase in vascularity. The blood vessels are frequently surrounded by a narrow margin of blue staining material that Manassee described first as Blue Mantle zone.
3. Increase in osteocytes, and appearance of osteoblasts and osteoclast cells.

Histologically otosclerosis may be classified into:

1. Early focal otosclerosis
2. Diffuse active otosclerosis
3. Quiescent otosclerosis
4. Cochlear otosclerosis

Early focal otosclerosis: In this type the abnormalities are localised to one or two small areas of an otherwise normal foot plate section. The abnormal areas show an enlarged marrow space surrounded by a blue staining area on H&E staining.

Diffuse active otosclerosis: In this type there is abnormal vascularity with a great increase in size and number of marrow spaces. Most of these spaces are lined by osteoblasts. In places around the circumference of the marrow spaces there is a scalloped appearance where bone has been recently absorbed. The number of osteocytes are greatly increased.

Quiescent otosclerosis: Here eventhough there may be some increase in the size and number of marrow spaces there is no evidence of bone formation or bone destruction. Osteoblasts and osteoclasts are only occasionally seen. This could be considered as a burnt out phase of the disease spectrum.

Cochlear otosclerosis: This condition causes pure sensorineural deafness without stapes fixation. Otosclerotic foci may occur in the otic capsule without the involvement of stapedial foot plate. The process of bone erosion and new bone formation which occur in otosclerosis releases enzymes like amylase, SGOT, SGPT etc which can enter into the endolymph via the round window membrane. These enzymes are toxic to the sensitive hair cells of the cochlea causing sensorineural hearing loss.

Clinical types of otosclerosis: Classification of various clinical types of otosclerosis is based on microscopic appearances of the diseased foot plate.

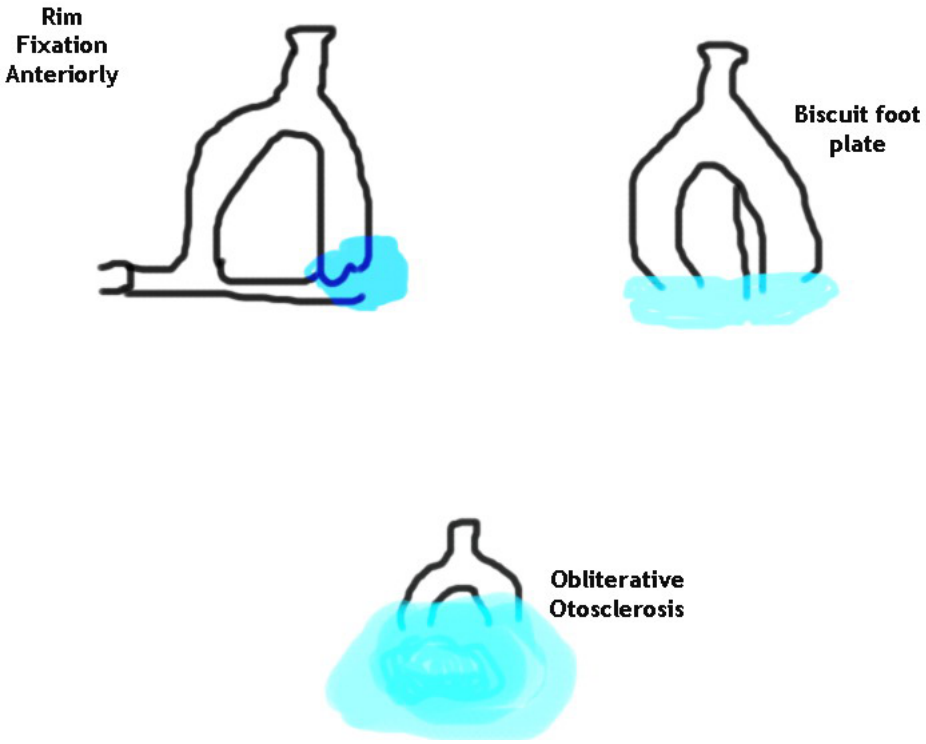
Rim fixation: Here the otosclerotic foci starts from the anterior portion of the oval window niche. It gradually expands to involve the anterior portion of the foot plate causing fixation of the anterior portion of the foot plate only leaving the centre of the plate free.

Biscuit foot plate: This type occur less frequently. The focus originates in the foot plate itself and as it expands it gives rise to the biscuit or rice grain foot plate with delineated margins.

Obliterative otosclerosis: Rarely a large mass of otosclerotic new bone fills up the oval window niche obscuring the entire foot plate. This condition is known as obliterative otosclerosis. It is a difficult condition to manage surgically.

Clinical features:

Deafness: Typically deafness in otosclerosis is bilateral and gradually increasing in nature. Unilateral otosclerosis occur in 15% of patients. Frequently it occurs between third and fifth decades of life. In majority of cases the deafness is conductive in nature. The deafness will not be noticed by the patient till the loss reaches 30 dB level. This is the deafness level in which understanding speech becomes difficult. These patients may hear better in noisy environment because the speaker has a tendency to raise his voice because of excessive ambient noise.



Diagramatic representation of various clinical types of otosclerosis

This phenomenon a feature of otosclerosis is known as Paracusis Willisii.
In cochlear otosclerosis the deafness is purely sensorineural in nature. Some patients may have both conductive and sensorineural hearing loss (mixed deafness) because of the tendency of bone reparative enzymes to damage the inner hair cells.

Patients with otosclerosis have characteristically quiet voice with good tone and the change in speech pattern may be detected only by close relatives.

Tinnitus: is a common symptom and occasionally could be the only presenting feature. The presence of tinnitus should alert the physician about the presence of cochlear otosclerosis. It could also be seen in some patients without cochlear degeneration due to abnormally increased vascularity of the otosclerotic bone. Mostly tinnitus indicates sensorineural degeneration. Tinnitus may be unilateral or bilateral. It is usually roaring in nature.

Vertigo: Transient attacks of vertigo is not uncommon in patients with otoslerosis. This could be due to the action of toxic enzymes released by the lesion into the vestibular labyrinth. These patients may even have coexisting Meniere's disease.

Clinical examination: The ear drum in these patients is normal (mint condition). Rarely during active phase of the disease the increased vascularity of the promontory may be seen through the ear drum. This sign is known as Flemingo's flush sign or Schwartz's sign. This indicates otospongiosis (active otosclerosis).

Hearing assessment can be done using tuning forks. For detailed description of tuning fork tests read the chapter titled clinical examination of the ear.

Pure tone auditometry will show precisely the amount and type of hearing loss. The presence of Carhart's notch is a classic audiometric feature in these patients. This Carhart's notch is present in bone condution. There is a dip centered around 2000 Hz. This is actually an artifact. In cochlear otosclerosis audiometry reveals sensorineural hearing loss.

Stapes fixation causes an elevation in the bone conduction thresholds of 5dB at 500Hz, 10dB at 1000 Hz, 15 dB at 2000 Hz, and 5 dB at 4000 Hz. In the audiogram this creates a peculiar pattern known as Cookie bite audiogram. The bone conduction audiogram appears like a cookie having been bitten.

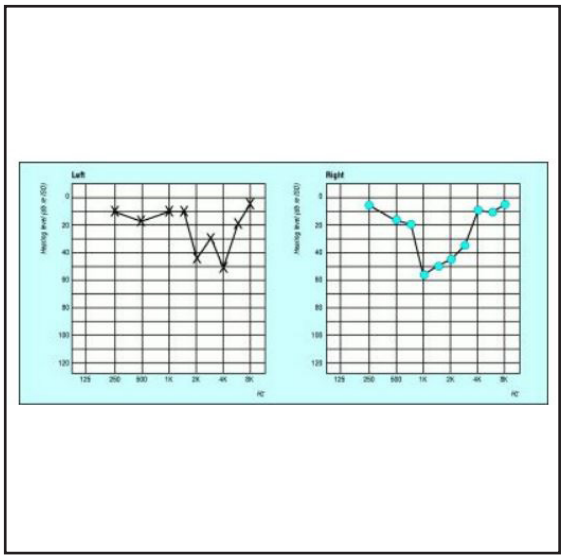


Figure showing cookie bite audiogram

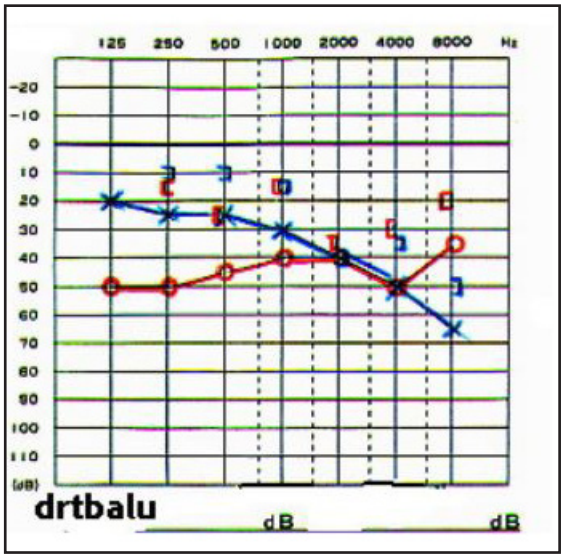


Figure showing carhart's notch

Impedence audiometry is an useful investigation to diagnose otosclerosis. Middle ear compliance is often reduced. When stapes is fixed stapedial reflex is absent. The typical impedence curve is As curve.

All these patients with pure conductive deafness have excellent speech discrimination thresholds.

Management:

Medical: The aim of medical management is to convert an active otosclerotic foci into an inactive or quiscent foci. Fluride is the drug of choice.

Indications of fluride therapy:

1. Patients with surgically confirmed otosclerosis who show progressive sensorineural deafness disproportionate to age.

2. Patients with pure sensorineural loss with family history, age of onset, audiometric pattern and good auditory discrimination indicate the possibility of cochlear otosclerosis.

3. Patients with radiological demonstration by CT scan of spongiotic changes in the cochlear capsule

4. Patients with positive Schwartz sign.

5. Post op treatment: If patients are found to have an active focus during surgery, fluride therapy is prescribed for 2 years.

Contraindications of fluride therapy:

1. Patients with chronic nephritis and nitrogen retention

2. Patients with chronic rheumatoid arthritis

3. Patient who are pregnant / lactating

4. In children before skeletal growth has been completed

5. Patients who show allergy for the drug

6. Patients with skeletal flurosis

Flurides act on otosclerotic foci by reducing osteoclastic bone resorption with a corresponding increase in osteoblastic bone formation. Fluride also has antienzymatic action thereby it can neutralise the toxic enzymes released from the otospongiotic foci.

Dose: A daily dose of 50 mg of sodium fluride is given for a period of 2 years. In patients with positive Schwartz's sign the dose can be increased up to 75 mg per day.

Adverse effects of sodium fluride therapy:

1. Gastric disturbance

2. Arthritis

3. Skeletal flurosis

Surgical treatment: Stapedectomy

Hearing aids: These patients will benefit from the use of hearing aids if surgery is not acceptable to the patient or if it is risky. There is always a 1% risk of producing a dead ear during surgery even in the best of hands.

Shambaugh's criteria to identify patients suffering from Sensorineural hearing loss due to otosclerosis:

1. Schwartze sign in either ear

2. Family history of otosclerosis

3. Unilateral conductive hearing loss consistent with otosclerosis and bilateral
   symmetric snhl

4. audiogram with a flat / "cookie - bite" curve with excellent speech discrimination

5. Progressive pure cochlear loss begining at the usual age of onset for otosclerosis

6. CT scan showing demineralization of the cochlea typical for otosclerosis

7. Stapedial reflex demonstrating the biphasic "on-off effect" seen before stapedial fixation

Stapedectomy

This surgical procedure is performed to treat deafness due to otosclerosis. Otosclerosis is caused by fixation of the foot plate of stapes which prevents efficient sound transmission to the oval window. The deafness caused is conductive in nature.

The surgical procedure is performed under local anesthesia. Advantages of performing this surgery under local anesthesia are:

1. Improvement in hearing can be ascertained on the table.

2. Bleeding is minimal under local anesthesia.

Indications for stapedectomy:

1. Conductive deafness due to fixation of stapes.

2. Air bone gap of atleast 40 dB.

3. Presence of Carhart's notch in the audiogram of a patient with conductive deafness.

4. Good cochlear reserve as assessed by the presence of good speech discrimination.

Contraindications for stapedectomy:

1. Poor general condition of the patient.

2. Only hearing ear.

3. Poor cochlear reserve as shown by poor speech discrimination scores

4. Patient with tinnitus and vertigo

5. Presence of active otosclerotic foci (otospongiosis) as evidenced by a positive flemmingo sign.

Since a patient with otosclerosis is also an ideal candidate for hearing aid and surgery, the patient must be properly counselled regarding the advantages and disadvantages of both.

Anaesthesia:

Xylocaine with adrenaline mixed in concentration of 1:1000 is used to infiltrate the external auditory canal. 0.25 ml of the solution is infiltrated using a 27 gauge needle. Infiltration is given as illustrated in the diagram.

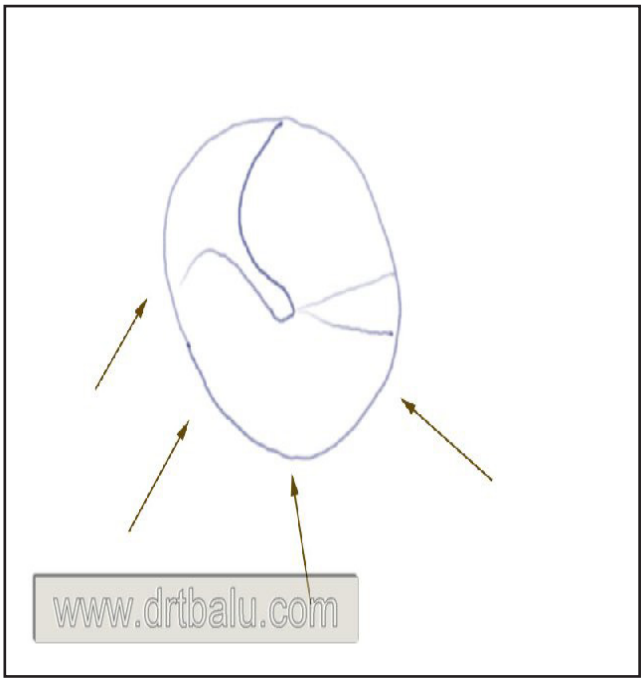


Figure showing injection sites for infiltration with local anesthetic agent

Exposure: A large speculum is used to straighten the external auditory canal. A curved or triangular incision is made in the external canal skin begining at 2mm away from the annulus. The incision extends from 11 o clock position to 6 o clock position as viewed in the right ear. The tympano meatal flap is elevated up to the annulus. Using a sharp pick the annulus is slowly lifted from its groove, the middle ear mucosa is exised and the middle ear proper is entered.

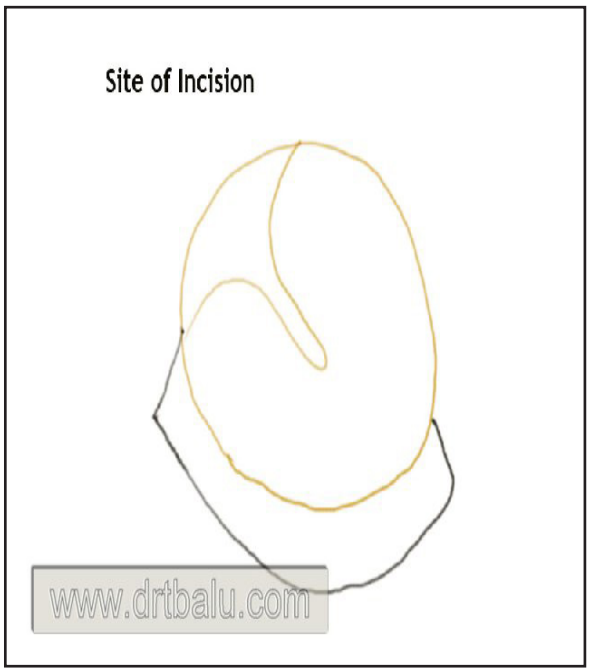


Figure showing incision made in the right ear for stapedectomy

In most patients the posterior superior bony overhang must be curetted using a curette (designed by House). The long process comes into view. Curetting is continued till the base of the pyramidal process is visualised. Oval window is visualised. At this point round window reflex is tested by moving the handle of malleus and looking for movement of round window membrane. In otosclerosis this reflex is absent.

Using a hand burr a small fenestra about 0.6mm in diameter is made over the foot plate. The stability of the incus is left intact because the stapedial tendon is not cut at this point. From now on the steps may vary according to the surgeon's viewpoint. Some surgeons would like to insert the piston at this stage without disturbing the stability of the incus. The distance between the long process of incus and the foot plate is measured using a measuring rod. Appropriate size teflon piston is introduced and humg over the long process of the incus and is crimped after ascertaining whether its lower end is inside the fenestra. The stapedial tendon is cut at this point and the supra structure of the stapes is disarticulated and removed. The Tympanomeatal flap is repositioned.

Complications of stapedectomy:

1. Facial palsy

2. Vertigo in the immediate post op period

3. Vomiting

4. Perilymph gush

5. Floating foot plate

6. Tympanic membrane tear

7. Dead labyrinth

8. Perilymph fistula

9. Labyrinthitis

Wordsworth criteria for second ear stapedotomy in otosclerosis:

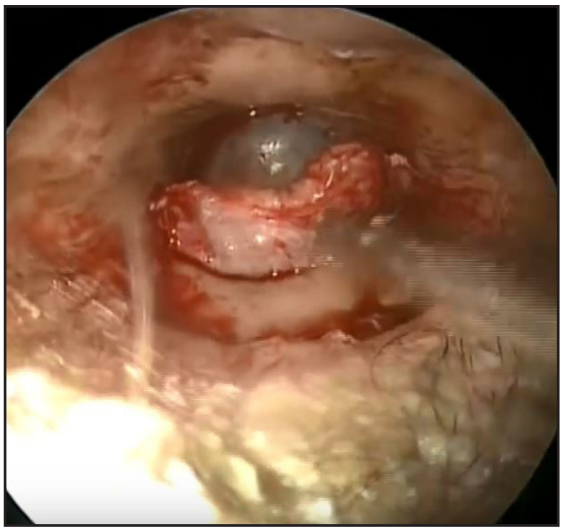
1. Patient request

2. Two year period between operations with a good result from the first operation

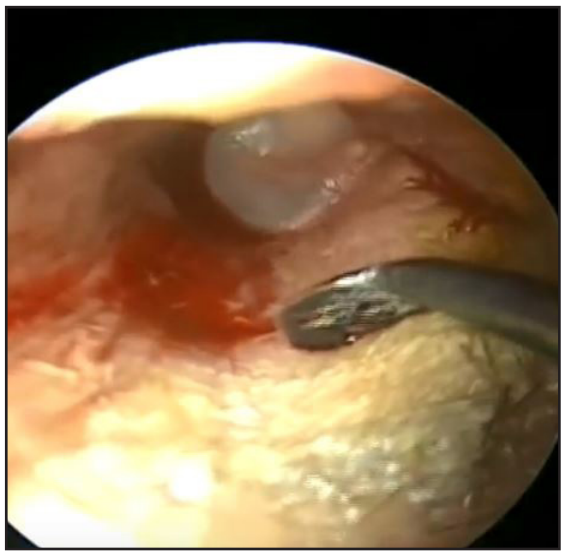
3. No evidence of significant snhl

4. Vestibular function test shows no e/o vestibular dysfunction

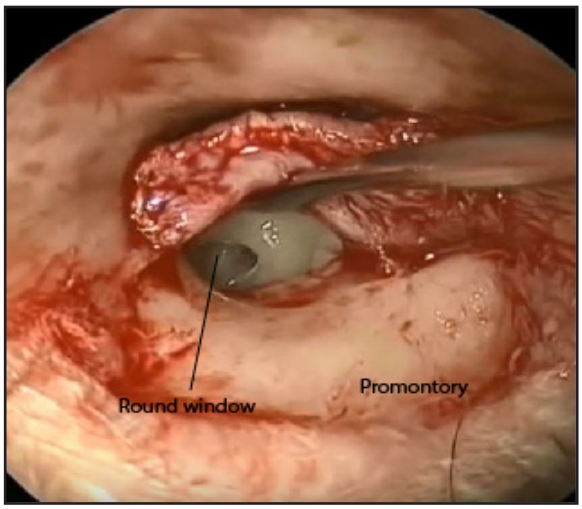
5. Absence of technical difficulties during the first operation 6.the surgeon must be experienced



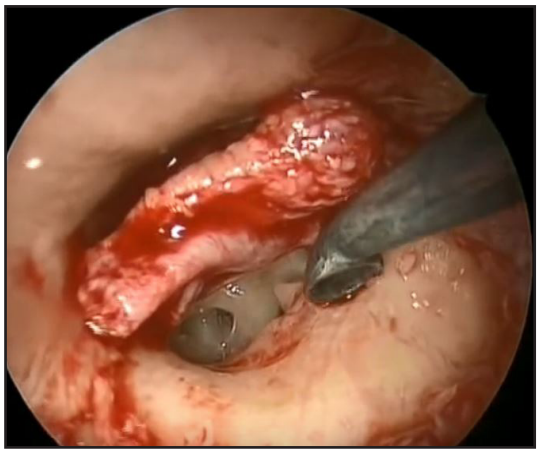
Picture showing tympanomeatal flap being elevated



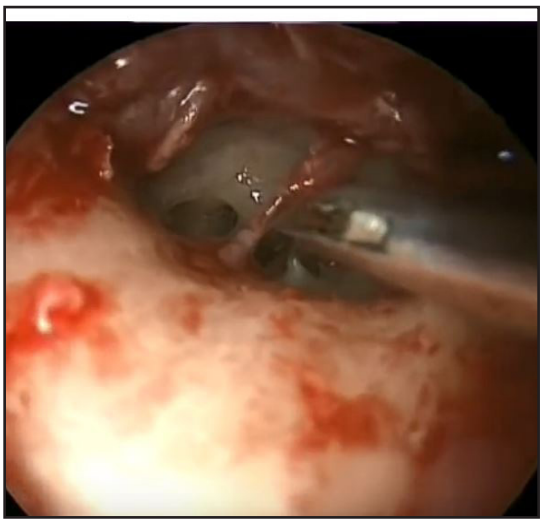
Picture showing incision made to elevate tympanomeatal flap



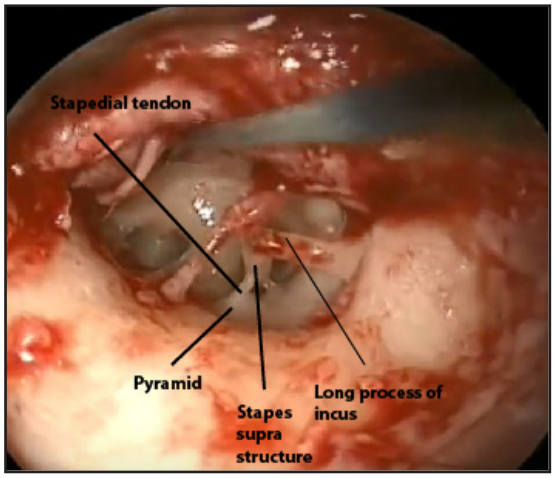
Picture showing middle ear entered



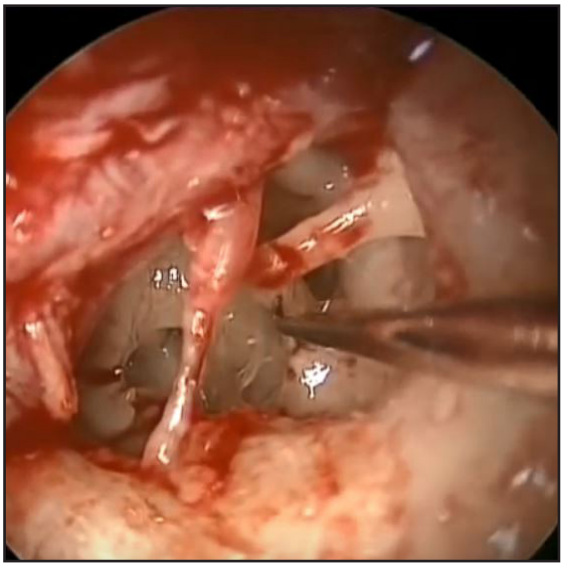
Picture showing bony overhang being curretted



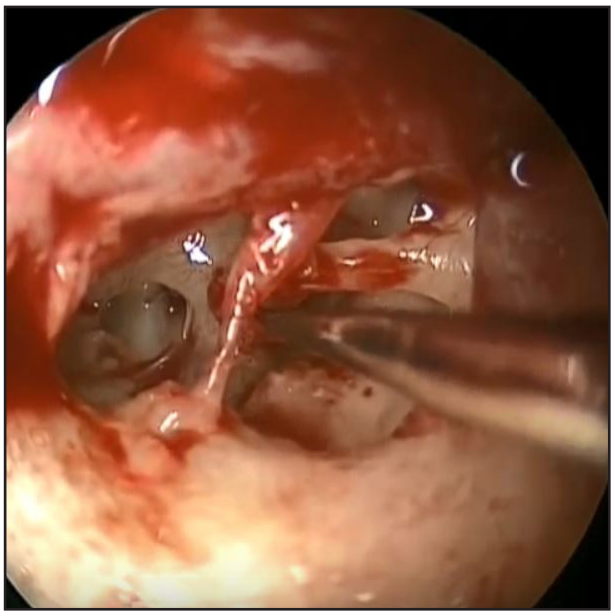
Picture showing stapedial tendon being cut



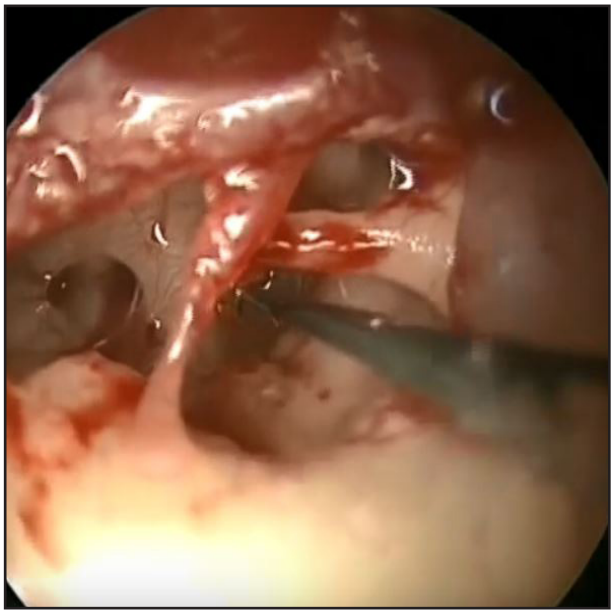
View of middle ear cavity after removal of bony overhang



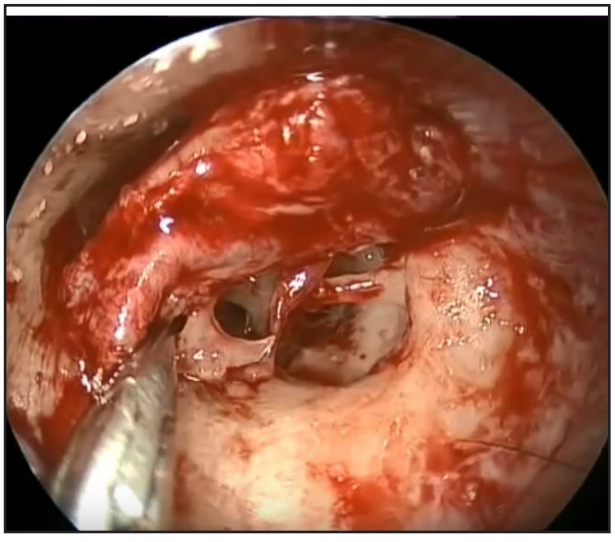
Picture showing disarticulation of supra structure of stapes



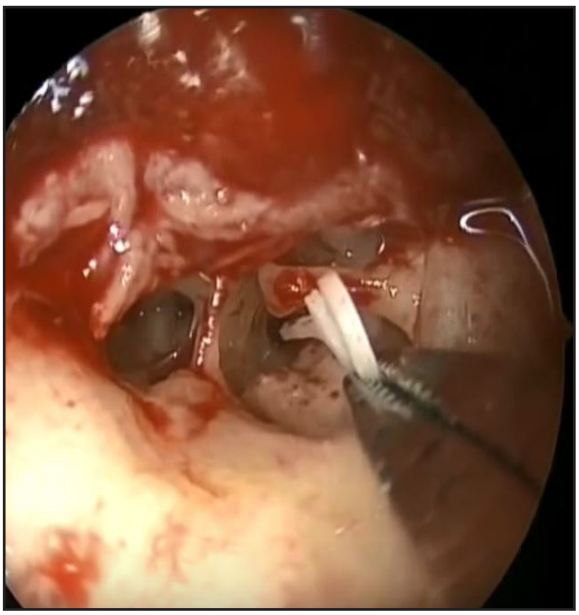
Suprastructure of stapes being removed



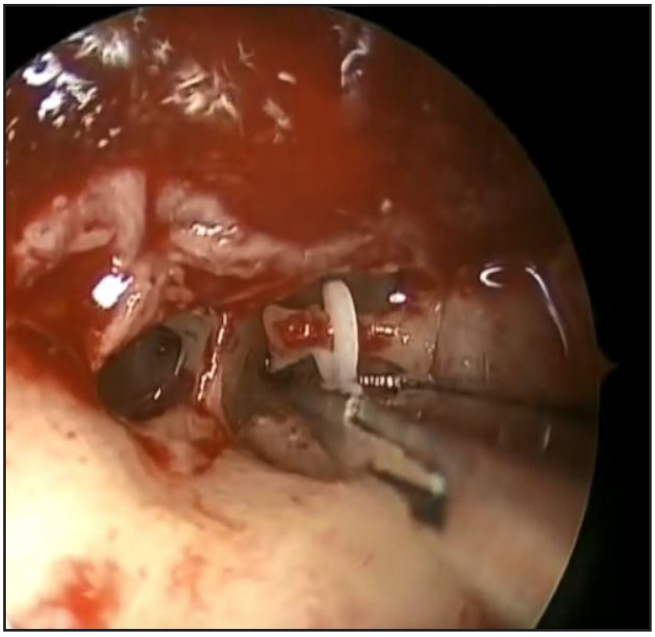
Footplate being fenestrated



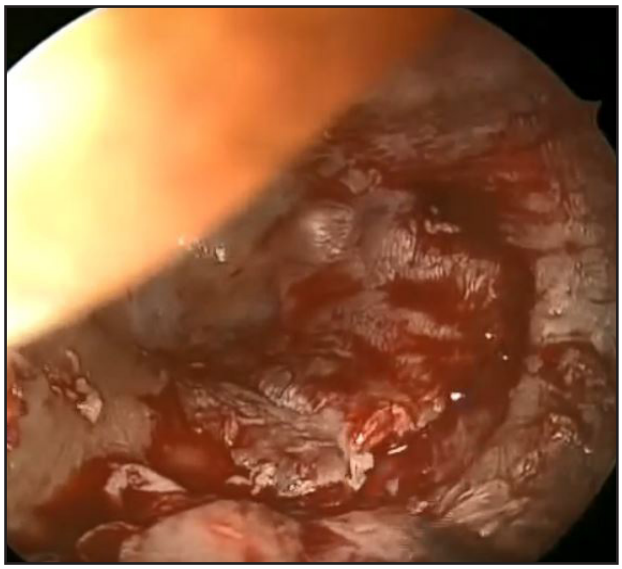
Stapes suprastructure pulled out



Picture showing piston being introduced



Picture showing piston insertion completed



Picture showing tympanomeatal flap repositioned after completion of surgery

Glomus Jugulare

Synonyms: Paraganglioma, Chemodectoma, Ganglia tympanica, Vascular tumors of middle ear.

Definition: Glomus jugulare is defined as a collection of ganglionic tissue within the temporal bone in close relationship with the jugular bulb. The jugular bulb is closely related to the floor of the middle ear cavity (i.e. Hypotympanum).

History: Valentine in 1840 described this condition as ganglia tympanica. Guild recognised its histological relationship with the carotid body. Lattes and Waltner suggested that the ideal generic term for these structures is non-chromaffin paraganglioma.

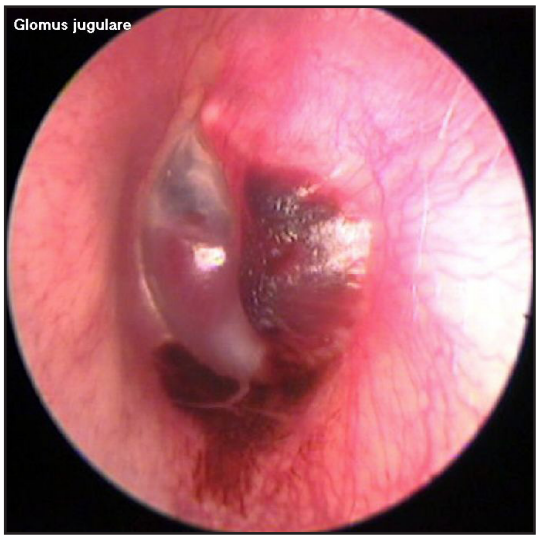
Paraganglia cells are derived from the neural crest and are found widely distributed in the autonomic nervous system. Paraganglia having negative chromaffin reaction are termed non - chromaffin paraganglia. Guild in his anatomical studies on temporal bones found that on an average three glomus bodies were found in them. They were usually found in close relationship with the tympanic branch of glossopharyngeal nerve or the auricular branch of vagus. These bodies were supplied with non medullated sensory fibers from the adjacent nerves. They are supplied by branches from the ascending pharyngeal artery.

Eventhough the paraganglia cells are closely related to either the tympanic branch of glossopharyngeal nerve or the auricular branch of vagus, their position in the temporal bone is highly variable. Commonly they are found in the adventitial layer of the jugular bulb. In about 25% of cases they may be found over the mucosa of the promontory. Histologically, they resemble carotid body. It contains epitheloid cells interspaced in a highly vascular stroma of capillary and precapillary vessels. The proportion of the cellular and stromal components vary.

Guild classified glomus tumors into two types depending on the amount of cellular and stromal components:

1. Cellular glomus bodies - when the cellular component is predominant
2. Vascular glomus bodies - when the vascular stromal component predominates.

Their sizes could be variable, but mostly they are ovoid in shape.



Otoscopic image of glomus jugulare

Paragangliomas of the temporal bone are generally divided into those that originate within the middle ear, glomus tympanicum tumors, and those that originate within the jugular fossa, glomus jugulare tumors. This latter term, however, is often used to refer to large tumors where the origin is difficult to determine. The predominance of the paraganglia within the jugular fossa likely accounts for the increased frequency of tumors with this origin. Classification systems that have been developed for temporal bone paragangliomas are used for staging purposes. surgical planning, and comparison among different therapeutic modalities.

Incidence: Glomus jugulare occurs in about 1 in 100000 patients. It is 6 times more common in females when compared to males.

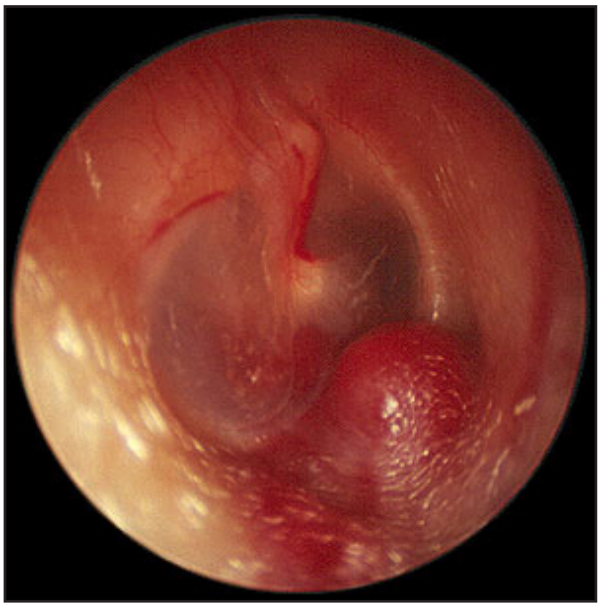
Hereditary pattern: It shows autosomal dominant inheritance with variable penetrance.

Endocrine activity: Eventhough these tumors are considered non chromaffin paragangliomas with no endocrine activity, some cases with endocrine activity by these tumors have been reported. It is hence important to look for evidence of endocrine activity by urine estimation of VMA (Vanillylmandelic acid).

Glomus tumors sometimes may show multicentric presentation i.e. present in both ears, or in conjunction with other paragangliomas. The carotid body being commonly the second site.

Pathophysiology: Glomus tumors are encapsulated, highly vascular, and locally invasive tumors. Inside the temporal bone they tend to expand along the pathway of least resistance such as air cells, vascular lumen, skull base foramina and eustachean tube. They also invade and erode bone in a lobular fashion. The middle ear ossicles are commonly spared. Initially skull base erosion occur in the region of jugular fossa and postero inferior part of petrous bone. Later on extension occurs to the mastoid and adjacent occipital bone.
The parenchyma of the paraganglia consists of 2 primary cell types. Type I cells are more common and are typically round with indistinct cell borders. Type II cells are smaller and irregularly shaped.

Presentation: These tumors are slow growing, with very little symptoms. The diagnosis may easily be missed. Infact the average delay between the onset of symptoms and diagnosis varied from 6 years to 15 years. The first symptoms generally follow middle ear involvement is easily overlooked. Pulsatile tinnitus and conductive deafness are the common presenting symptoms. A red mass behind an intact ear drum (rising sun sign) may also be seen. In some 30% of cases cranial nerve palsies are common. Facial nerve is affected most commonly.



Picture showing Rising sun sign a classic feature of glomus jugulare

Presenting features of Gomus jugulare:

1. Deafness - 69%
2. Middle ear mass - 75%
3. Pulsatile tinnitus - 55%
4. Imbalance - 8%
5. Otorrhoea - 5%

6. Facial palsy - 8%
7. Endocrine syndrome - 3%
8. Cranial nerve deficits
    Hoarseness - 16%
    Dysphagia - 16%
9. Headache - 15%
10. Visual disturbance - 6%
11. Presence of headache indicates intracranial extension
12. Dural sinuses may be involved may mimic sinus thrombosis

Clinical features: Otoscopic examination reveals a characteristic, pulsatile, reddish-blue tumor behind the tympanic membrane that often is the beginning of more extensive findings (ie, the tip of the iceberg). When the drum is examined under a microscope will show a pulsation of the reddish mass behind the drum. On seigalisation the mass blanches. This sign is known as Brown's sign. This is pathognomonic of glomus tumor.
Audiologic examination reveals mixed conductive and sensorineural hearing loss. The sensorineural component tends to be more significant with larger tumors.

Classification:

Glasscock - Jackson classification of temporal bone paraganglioma:

1. Type I : Small tumor involving the jugular bulb, middle ear and mastoid.
2. Type II: Tumor extending under the internal auditory canal. There may be intracranial extension.
3. Type III: Tumor extending into the petrous apex. There may be intracranial extension.
4. Type IV: Tumor extending beyond the petrous apex into the clivus and infratemporal fossa. There may be intracranial extension.

The Fisch classification of glomus tumors is based on extension of the tumor to surrounding anatomic structures and is closely related to mortality and morbidity.

Fisch classification:

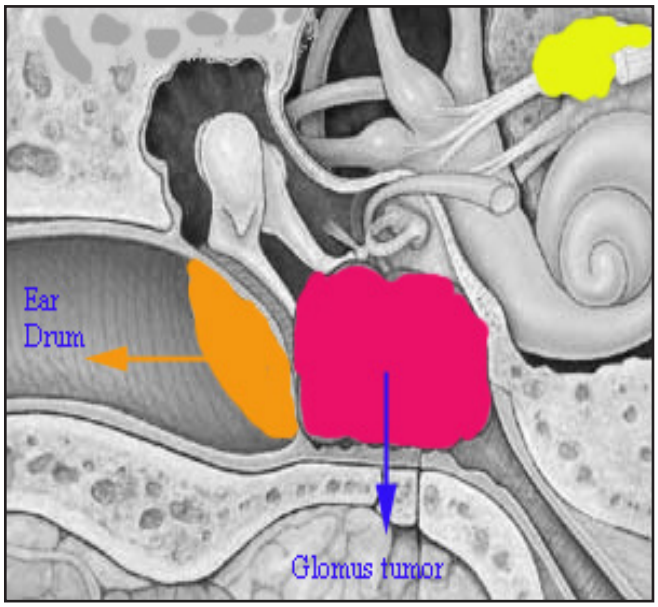
1. Type A tumor - Tumor limited to middle ear (carries the best prognosis)
2. Type B tumor - Tumor limited to the tympanomastoid area with no infralabyrinthine compartment involvement
3. Type C tumor - Tumor involving the infralabyrinthine compartment of temporal bone with extension to petrous apex
   This is divided into three types: C1, C2 and C3.
   Type C1 - Tumor with limited involvement of the vertical portion of the carotid canal
   Type C2 - Tumor invading the vertical portion of the carotid canal
   Type C3 - Tumor invasion of the horizontal portion of the carotid canal
4. Type D tumor has 2 types

   Type D1 - Tumor with an intracranial extension less than 2 cm in diameter
   Type D2 - Tumor with an intracranial extension greater than 2 cm in diameter

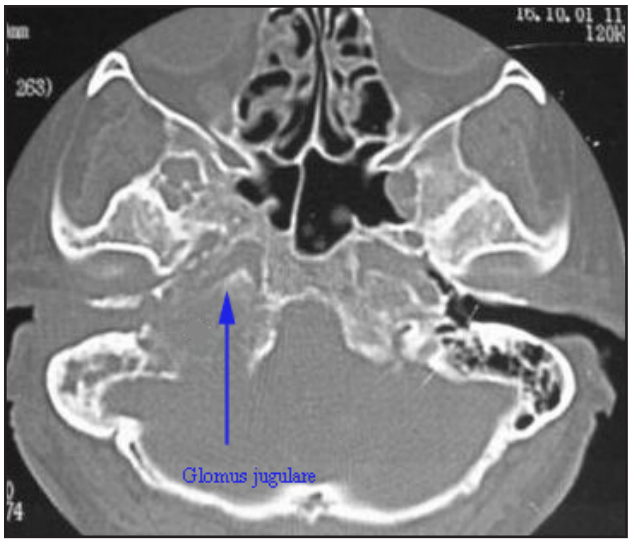
Investigations: Radiological investigations help in the diagnosis.

Plain X ray skull: May show enlargement of lateral jugular foramen and jugular fossa.

CT scan and Contrast MRI using Gadolinum enhancement is very helpful in delineating tumor extension.



Diagrammatic representation of glomus tumor



CT scan image showing glomus tumor

Applied anatomy of jugular bulb area:

The posterolateral portion of the foramen (pars venosa) contains the jugular bulb, posterior meningeal artery, and cranial nerves X and XI. The anteromedial portion (pars nervosa) contains the inferior petrosal sinus and cranial nerve IX. The jugular bulb is situated between the sigmoid sinus and the internal jugular vein. The lower cranial nerves are situated medial to the medial wall of the jugular bulb. The inferior petrosal sinus enters the medial aspect of the jugular bulb via several channels anterior to cranial nerves IX, X, and XI.
Many important structures are in proximity to the jugular bulb, including the internal auditory canal, the posterior semicircular canal, the middle ear, the medial external auditory canal, the facial nerve (posterolaterally), and the ICA (anteriorly) within the carotid canal. At the extracranial end of the jugular foramen, the ICA, internal jugular vein, and cranial nerves VII, X, XI, and XII are within a 2-cm area.

Treatment:

Treatment is mainly surgical. Complete resection of the mass is curative. Since it is a highly vascular tumor pre op intravascular embolisation may help to reduce bleeding during surgery.
The particular surgical approach used to resect temporal bone paragangliomas depends on the location and extent of the tumor. Paragangliomas originating from the promontory of the middle ear and isolated to the mesotympanum can be resected by elevating the tympanic membrane and removing the tumor using microdissection techniques. If the tumor extends into the hypotympanum or the mastoid, a tympanomastoidectomy is performed and the tumor resected.

In extensive Fisch type 3 tumors the mass can be approached with help from neurosurgeons. The skull base approach ensures better exposure of the mass and facilitates complete resection.

Management of Fisch type 4 tumors is highly controversial. Irradiation of the mass has been tried with very little effect. Considering the slow growth rate of these tumors with a very long doubling time, these patients are best left alone with symptomatic treatment of the complications.

Complications of surgery:

Complications of surgery include death, cranial nerve palsies, bleeding, cerebrospinal fluid (CSF) leak, meningitis, uncontrollable hypotension/hypertension, and tumor regrowth.