**COM**

**DEFINITION:**
- The diagnosis of chronic otitis media (COM) implies a permanent abnormality of the pars tensa or flaccida, most likely a result of earlier acute otitis media, negative middle ear pressure or otitis media with effusion.
- COM equates with the classic term chronic 'suppurative' otitis media that is no longer advocated as COM is not necessarily a result of 'the gathering of pus'.

**TYPES**
- **Active COM** ➔ where there is inflammation and the production of pus
- **Inactive COM** ➔ where this is not the case though there is the potential for the ear to become active at some time.
- **Healed COM** ➔ where there are permanent abnormalities of the pars tensa, but the ear does not have the propensity to become active because the pars tensa is intact and there are no significant retractions of the pars tensa or flaccida.
- 'Healed COM' can also be the end result of successful surgery.

**CLASSIFICATION OF COM**
- Healed COM
- Inactive Mucosal (perf)
- Inactive Squamosal (retraction)
- Active Mucosal
- Active squamousal (Retraction of the pars flaccida or tensa that has retained squamous epithelial debris and is associated with inflammation and the production of pus, often from the adjacent mucosa)

**PATHOLOGY OF VARIOUS TYPES OF COM**

**Inactive mucosal COM (dry perforation)**
- There is a permanent perforation of the pars tensa, but the middle ear and mastoid mucosa is not inflamed.
- The mucocutaneous junction is usually located at the margin of the perforation, but not necessarily. Squamous epithelium can migrate medially into the middle ear.
- Chances of medial epithelial migration highest if perforation extends till the annulus.(velvety appearance under operating microscope)

**Active mucosal COM (perforation with otorrhoea)**
- There is chronic inflammation within the mucosa of the middle ear and mastoid, with varying degrees of oedema, submucosal fibrosis, hypervascularity and infiltration with lymphocytes, plasma cells and histiocytes. Ulceration may finally lead to granulation tissue formation.
Production of mucopurulent discharge
The mucosal changes may progress and coalesce to form 'aural polyps'
These processes occur in complete middle ear cleft including mastoids
Resorptive osteitis i.e. resorption of part or all of ossicular chain can occur.
The ossicles thus affected typically show hyperemia with proliferation of capillaries and prominent histiocytes
Long process of incus >>> Stapes Crurae >>> Body of incus are involved in frequency (The reason that the long process of the incus and stapes superstructure are most frequently affected is likely to be due to their delicate structure and location rather than their tenuous blood supply.)
So bony resorption occurs in Active mucosal and active squamous both.
This resorption is mediated by osteoclasts activated by a no of factors including IL1, IL6, TNF, PG's, NO, neurotransmitters.
Some ears with active COM (both mucosal and cholesteatomatous subtypes) demonstrate focal areas of cholesterol granuloma, which is a histologic term applied to an area of giant cell reaction around cholesterol crystals.
Cholesterol crystals can either be break down products of haemorrhage OR can be derived from middle ear effusion.

Inactive squamous epithelial COM (retraction, atelectasis and epidermization)
Negative static middle ear pressure can result in retraction (atelectasis) of the tympanic membrane.
Retraction may be fixed also
'Epidermization' is a more advanced type of retraction and refers to replacement of middle ear mucosa by keratinizing squamous epithelium without retention of keratin debris.
Epidermization often remains quiescent and does not progress to cholesteatoma or active suppuration.

Active squamous epithelial COM (cholesteatoma)
The hallmark of a cholesteatoma is its retention of keratinous debris. Thus, a 'keratoma' would be histologically a more correct term. Histologically, the squamous epithelial lining or 'matrix' of a cholesteatoma is similar to that of skin.
A cholesteatoma can be dry or asso with malodourous discharge.
Cholesteatomas are potentially dangerous because of their potential to incite resorption of bone, leading to intratemporal or intracranial complications.

HEALED PERFORATION (DIMERIC MEMBRANE)
Loss of the lamina propria of the tympanic membrane due to atrophy or failure to reform during healing of a perforation leads to a 'dimeric' membrane that consists of epidermis and mucosa. Such a thin membrane is prone to retraction if there is negative static middle ear pressure.

TYMPANOSCLEROSIS
Tympanosclerosis refers to hyaline deposits of acellular material visible as white plaques in the tympanic membrane and as white nodular deposits in the submucosal layers of the middle ear on otoscopy.
Pathologically, tympanosclerosis is the end result of a healing process in which collagen in fibrous tissue hyalinizes, loses its structure and becomes fused into a homogeneous mass. Thereafter, calcification and ossification may occur to a variable extent. Theories of pathogenesis of tympanosclerosis include local immunologic hypersensitivity, increased oxygen concentration in the middle ear with exposure to oxygen radicals and local inflammatory activity. A prerequisite for Tympanosclerosis appears to be COM followed by healing. Tympanosclerosis involving the tympanic membrane is usually of little consequence to middle ear sound transmission. Tympanosclerosis surrounding the ossicles in the epitympanum and the stapes superstructure or footplate in the oval window causes varying degrees of immobility of the ossicular chain and is a well-recognized adverse factor in tympanoplasty.

FIBROCYSTIC AND FIBRO-OSSEOUS SCLEROSIS

Some cases of healed COM result in end-stage pathology characterized by fibrosis and cyst formation. Obliterates large portions of the middle ear cleft. The cystic spaces become lined by mucosal epithelium that has been overrun by proliferation of connective tissue. There is often deposition of new bone in the mastoid antrum and surrounding mastoid cell tracts (‘fibro-osseous sclerosis’), eventually resulting in a sclerotic mastoid. It represents a non progressive end stage pathology that is a contraindication for tympanoplasty.

PATHOLOGY OF COMPLICATIONS OF COM

Routes of spread of infection

- Natural routes include Oval window, round window (round window membrane and annular ligament which otherwise are natural barriers for spread to labyrinth might be breached)
- Direct erosion of bone.
- Abnormal preformed pathways (either congenital or acquired, including aberrant arachnoid granulations, meningo-encephalocoeles and temporal bone fractures.
- Vascular channels (Progressive thrombophlebitis of small venules may result in spread of infection through intact bone.

Labyrinthine fistula

- MC in Lateral SCC and one of MC complication of COM (4-13% cases)
- Histologically, cholesteatoma matrix or granulation tissue becomes apposed to the endosteum of the inner ear, or in larger fistulae, directly to the membranous labyrinth.
- If the natural barrier posed by the endosteum or membranous labyrinth is overcome either by overwhelming infection or by surgery, then the fistula would allow rapid dissemination of infection throughout the inner ear.
- The margins of many labyrinthine fistulae show evidence of new bone formation.
- Many fistulae show spontaneous bony closure after removal of the offending cholesteatoma or infection.

Labyrinthitis

- Inflammation of the labyrinth has traditionally been classified as 'serous or toxic' and 'suppurative'.

Labyrinthitis

- **Serous**: Sterile inflammatory response of labyrinth to bacterial toxins, acidophilic staining of perilymphatic fluid, some showing staining of endolymph and endolymphatic hydrops also.
- **Severe forms can lead to degeneration of Organ of Corti.**
- **Suppurative labyrinthitis**: is assumed to be caused by bacterial invasion of the inner ear and is characterized histologically by collections of PMN leukocytes in the perilymphatic spaces at the site of bacterial invasion.
- Acidophilic staining of the inner ear fluids, endolymphatic hydrops and, eventually, necrosis of the membranous labyrinth.
- Both types early stage may lead to SNHL
- Inflammatory cytokines released in both types can definitely disrupt integrity of spiral ligament (maintains ion and fluid homeostasis within the cochlea).
- It is the host inflammatory response and not the offending bacteria that is responsible for the hearing loss.
- Serous labyrinthitis reversible hearing loss whereas suppurative is permanent.

Facial nerve paralysis

- Occurs by direct erosion of bone of the facial canal by cholesteatoma or granulation tissue.
- Histologic changes within the nerve can include infiltration of the nerve by inflammatory cells, oedema and fragmentation of myelin sheaths and degeneration of axons.

Petrositis

- **GRADINEGO's triad of persistent otorrhoea, severe periorbital pain and abducent nerve paralysis** is characteristic of infection of the petrous apex.
- **Acute petrositis** shows the characteristic changes of acute inflammation in the mucous membrane of petrous apex cells and it may progress to an abscess and osteitis.
- **Chronic petrositis** is a complication of COM where, in addition to inflammatory changes, there is resorption of bone as well as formation of new bone. Chronic petrositis may be an indolent infection that can persist for months or years.
- However, petrositis is a potentially serious complication because of the anatomical proximity of the petrous apex to the meninges, trigeminal nerve and carotid artery.
- So can lead to meningitis, extradural abscess, brain abscess, multiple cranial nerve paralyses and carotid artery thrombosis.
- Otogenic intracranial complications
- These complications include bacterial meningitis, extradural or subdural abscess, cerebellar or temporal lobe brain abscess and sigmoid sinus thrombophlebitis.

SURGICAL PATHOLOGY

**Tympanic membrane grafts**

- Commonly used grafts include autologous temporalis fascia, perichondrium, cartilage and adipose tissue.

**Ossicular grafts and implants**

- A wide variety of autograft, homograft and synthetic materials has been used for reconstructing the ossicular chain.
- Autologous grafts can work for >25yrs
- The general availability, low cost, ease of handling and time-tested functional results make autologous ossicles the material of choice for ossiculoplasty, as long as they are available.
- Histologically homologous ossicles are also well tolerated though **Creeping bony substitution** (They undergo slow replacement of nonviable bone by new bone formation through a process of 'creeping substitution'.) occurs less rapidly than with autologous ossicles.
- Cartilage grafts for ossicular recons. ⊗ as chondromalacia develops and they resorb
- Synthetic porous high density polyethylene, gold, titanium, hydroxlapattite, bioactive glasses, hydroxylapatite polyethylene combo.

Mastoidectomy cavities
Dry and well healed is characterized histologically by a lining of keratinized squamous epithelium on a layer of subepithelial fibrous tissue and underlying sclerotic mastoid bone.

A chronically draining or infected mastoid cavity can arise as a result of:

1. Recurrent or residual cholesteatoma
2. Mechanical factors promoting retention of debris such as a high facial ridge, meatal or canal stenosis.
3. Suppurative in unexenterated cells of the mastoid and middle ear.

If part of a cholesteatoma matrix is left within a closed middle ear cleft, one of two things may happen. The squamous epithelium may undergo metaplasia to middle ear type mucosa.

The other alternative is that the matrix will develop into a squamous epithelial retention cyst or cholesteatoma pearl.

The natural history of such cholesteatoma pearls is unknown: some may disappear spontaneously, some remain static and others progressively enlarge to become a cholesteatoma.

**BIOLICAL AND PATHOLOGICAL FACTORS DETERMINING SUCCESS OF TYPANOMASTOID SURGERY FOR COM**

The primary objective of surgery for COM is to eradicate infection and disease and make the ear safe and dry.

A second objective of surgery for COM is to restore hearing to serviceable levels by means of tympanoplasty.

Hinderance to achieving these objectives are:

A number of pathological mucosal changes can occur within the middle ear as a healing response to COM or as a sequel to surgical trauma. The changes include deposition of fibrous tissue, formation of adhesions and neo-osteogenesis, fixation of the stapes footplate, ankylosis or displacement of an ossicle strut, immobilization of the round window, immobilization of the tympanic membrane, as well as more subtle interference with the mechanics of the tympanic membrane or ossicles.

Another factor leading to failure of tympanoplasty is total or partial non-aeration of the middle ear and development of negative static middle ear pressure due to ET dysfunction and can lead to severe tympanic membrane atelectasis, middle ear effusion, fibrocystic sclerosis of the middle ear or a combination of these changes. Can also lead to recurrent cholesteatomas.

**ETIOLOGY:**

- M=F, 18-40yrs, Low socio economic groups

**AOM and OME**

These changes may reduce the elastic properties of the tympanic membrane, making it more susceptible to chronic perforation or retraction.

**Genetics and race**

- highest in Eskimos, native Americans

**Environment**

- COM is higher in lower socioeconomic groups.

**Eustachian tube dysfunction**

- It is not known however, if the Eustachian tube dysfunction is the initiating factor in COM or whether it is a result of COM.

**Gastro-oesophageal reflux**

**Craniofacial abnormalities**
The incidence of COM in cleft palate patients followed up to ten years of age is around 20 percent, with 2 percent of them having a cholesteatoma.

Autoimmune disease and immunodeficiency are implicated but not proved.

**FACTORS INFLUENCING ACTIVITY OF COM**

**Infection**

- Proteus >> Staph aureus >> Pseudomonas
- Pseudomonas aeruginosa and Staphylococcus aureus are the most commonly reported pathogens, most of the other organisms being gram negative coliforms

Upper respiratory tract infections

- Upper respiratory tract infections produce transient Eustachian tube dysfunction in healthy individuals and also can lead to ascending infection.

**AETIOLOGY OF CHRONIC MUCOSAL OTITIS MEDIA**

- Single measles episode (preimmunization era)
- AOM
- Ventilation tube

**AETIOLOGY OF CHRONIC SQUAMOUS EPITHELIAL OTITIS MEDIA (CHOLESTEATOMA)**

**Sades theory of Squamous metaplasia**

- One theory suggests that cells from which a cholesteatoma arise originate from metaplasia of the middle ear mucosa.
- The best evidence suggests that cholesteatoma arises from skin cells of the tympanic membrane.
- Middle ear cholesteatoma has a cytokeratin pattern typical of skin and closely resembling skin of the external auditory meatus. Cholesteatoma sacs are found in close proximity to the tympanic membrane and are not encapsulated but are connected to the tympanic membrane by a neck of invaginated squamous epithelium, and the tympanic membrane appears to follow progressive changes from normal to retraction pockets and then to cholesteatoma.

**Whittmaack’s theory of Misplaced epithelium**

- Cholesteatomas are most likely to arise from a retraction pocket in the pars flaccida or the posterosuperior part of the pars tensa and the initiating factor is probably dysfunction of the Eustachian tube resulting in negative middle ear pressure.
- Because of their greater blood supply compared with the rest of the tympanic membrane, the pars flaccida and the postero-superior quadrant of the pars tensa are more affected by inflammatory cell infiltration in acute otitis media and in otitis media with effusion.
- This may leave the fibrous layer in these areas thinner than the remainder of the tympanic membrane.

**Haberman's Ingrowth of epithelium**
Through tympanic membrane perforation

Ruedi suggested that papillary ingrowth of squamous epithelium through its own basement membrane, in the pars flaccida, could result in a cholesteatoma.

Retraction pocket formation and papillary ingrowth of squamous epithelium may coexist.

Bone destruction of cholesteatoma is explained by Pressure Theory, Enzymatic Theory And Pyogenic Osteitis Theory.

**DIAGNOSIS AND ASSESSMENT**

- History and clinical examination
- Look for any previous ear surgeries
- **Otoscopy** with the aid of a microscope is the 'gold standard' for the diagnosis of COM.
- Facilitates aural toilet, which is almost always required to some extent to fully visualize all areas.
- It is also helpful in viewing the anterior recess of the tympanic membrane.
- After any aural toilet, the clinician has to assess all areas of the external ear and the tympanic membrane to decide if there is pathology and if so what the pathology is.

**SITE OF PATHOLOGY**

- Quadrant involved, % of involvement preferred rather than small, medium large perforation.
- Three quadrant preferred to 4

**OPEN MASTOID CAVITIES**

- These are usually created surgically but can occur spontaneously

**ACTIVE OR INACTIVE?**

**INVESTIGATIONS**:

- Pus C/S
- PTA
- The degree of air-bone gap depends on:
  - the size of the perforation in the tympanic membrane;
  - erosion of the ossicular chain, most commonly the long process incus and sometimes also the stapes superstructure;
  - significant granulation tissue around the ossicular chain which can reduce its mobility.
- Vestibular Assessment
  - This will be performed on those with episodes of peripheral-type vertigo and some patients undergoing revision surgery.
  - A fistulae test, if positive with the production of acute vertigo accompanied by a nystagmus, is suggestive of a semicircular canal fistulae created
  - Computed tomography (CT) scanning with 1.5-mm sections in both the coronal and axial planes of the temporal bone is preferred if intracranial complications suspected

**INACTIVE COM**

**PROGRESSION TOWARDS HEALING**

- This is usually recurrent episodes of acute infection with perforation of the drum, which initially heals successfully within a few days, but after a variable number of attacks the tympanic membrane fails to heal. This is regarded as result of failure of the blood supply to the perforation edges due to endarteritis.
- Natural healing is a relatively rare event in adults though it appears to occur more often in children.
MANAGEMENT

Management options are surgery, a hearing aid or no treatment.

OBJECTIVES OF SURGERY

- The aim of middle ear surgery for hearing is reduction in the patient's hearing disability, not just closure of the airbone gap.
- Dry perforations that are symptom free do not usually require closure.

MYRINGOPLASTY:

- The success rate in achieving an intact tympanic membrane in expert hands is often quoted as around 95 percent.
- The closure rate is reported to be higher in small perforations
- **Failure rate in anterior perforations is higher.** Can be reduced by tucking technique of tympanoplasty (anchoring the anterior margin of the graft beneath the annulus)
- Prophylactic antibiotics do not influence closure rate
- Only 60 percent of tympanic membranes were intact after revision surgery which is a much lower success rate than usually claimed for primary surgery
- Successful closure of the tympanic membrane usually gives only a small improvement in hearing.
- Rare complications (rarely facial palsy)

OSSICULOPLASTY:

- The most common pathology in the middle ear is erosion of the long process incus.
- Mills reported a mean hearing improvement after ossiculoplasty of 14 dB when the stapes arch was intact and 6 dB when it was eroded.
- It is generally accepted that the success rate of ossiculoplasty is higher if the procedure is staged.

MANAGEMENT OF ACTIVE MUCOSAL COM

- **Aural toilet** ➔ This is most effectively carried out with a microscope and suction.
- However, some clinicians use gentle syringing with saline or antiseptic agents.
- **TOPICAL MEDICATION** ➔
  - Topical antibiotics with steroids are significantly better than dry-mopping alone, dry mopping in combination with a topical saline solution or topical aluminium acetate solution.
  - There is no conclusive evidence that topical aminoglycosides are ototoxic when used in treatment of COM
- Quinolones are preferred.
- Myringoplasty ➔
  - Role of adjuvant cortical mastoidectomy ➔
  - It should be remembered that polyps can be attached to the stapes superstructure or to the facial nerve, and that damage to these structures can occur.
  - Removal of polyps usually causes bleeding in the ear that interferes with surgical access, but this can be greatly reduced by the use of a laser in the removal of the polyp
- **RETRACTION OF THE PARS TENSA**
  - Is this retraction pocket thought to be self cleansing or not? What is the relationship of the tympanic membrane to the incudo-stapedial joint? Is there partial or complete erosion? What effect has this had upon hearing thresholds? Has the bony ear canal at the level of the tympanic membrane been eroded by longstanding retraction of the pars tensa and become wider? This can be evaluated because structures not normally visible, such as the stapedius tendon and facial nerve, becoming visible.
  - There is good case series evidence that pars tensa and pars flaccida retractions coexist.
  - Retraction may lead to histological changes in the tympanic membrane with loss of elasticity and rigidity so that the tympanic membrane no longer 'drives' the ossicular chain or areas of
the tympanic membrane may be eroded leaving a perforation. Tympanic retraction may also damage middle ear structures, for example erode the long process of incus.

- There is evidence from follow-up of pars tensa retractions in children with otitis media with effusion that it is a dynamic condition with around 70 percent resolving spontaneously.
- Indeed the insertion of a ventilation tube significantly increases the chances of retractions and atrophy of the pars tensa
- Children have asso btw pars tensa and flaccida retractions and adults have pars flaccida and cholesteatoma association

**MANAGEMENT OF INACTIVE SQUAMOSAL**

- MANAGEMENT OF NASAL DISEASE
- AURAL TOILET

**Management of Tympanic Membrane**

**Excision, no graft**
- Some have argued that the retracted tympanic membrane is abnormal, should be excised and a new 'normal' eardrum will naturally grow in its place without any need of grafting 😊😊
- Others have combined excision with insertion of a ventilation tube in the remaining tympanic membrane.
- The evidence is not of good quality so it is difficult to make out a case for inserting a ventilation tube at the same time as excising the retraction pocket

**Excision, myringoplasty**
- Use of temporalis fascia grafts was associated with recurrence of retraction at levels varying from 5 to 25 percent.
- The cartilage, usually prepared from tragus or concha, can be used in a number of ways. They can be composite perichondrial-chondrial grafts. Alternatively 'thin sheets' or 'pallisades' (multiple small fragments) of cartilage can be prepared.
- These may be appropriately positioned by being placed underneath a conventional temporalis fascia graft or sandwiched between two temporalis fascia grafts. The use of tissue glues to maintain a stable position is common
- BUT 😊 recurrent retraction may be more reliably prevented than with temporalis fascia grafts alone.
- **Excision, myringoplasty with cortical mastoidectomy** seems to suggest that no further benefit is gained

**VENTILATION TUBES**
- Not so useful
- Indeed, the use of ventilation tubes in otitis media with effusion seems to increase the prevalence of pars tensa atelectasis and attic retraction
- The apparent lack of success of ventilation tubes in the management of tympanic retraction may be because histological changes may have taken place in the tympanic membrane rendering the situation irreversible.
- If the patient is an adult (over the age of 12) the Eustachian tube function is likely to be normal and the retraction has a substantial chance of being stable. If there is no significant hearing loss and the retraction pocket is self-cleansing, then follow-up on an occasional basis would be appropriate.
- The cleansing, then it may be managed with regular microscopic suction clearance.
- If the retraction pocket is not self In well-pneumatized temporal bones, such as in children, the disease is frequently extensive, expanding down the well-formed air cell tracts. Squamous epithelial disease is more commonly found in poorly pneumatized sclerotic bones, but whether the sclerosis is relevant to the aetiology of the disease or is caused by it has also not been fully resolved.
- The evidence suggests that the most important factor in the development of mastoid sclerosis is poor Eustachian function.
CASES reported where the patient has a well-formed 'atticotomy' or 'mastoidectomy' cavity, but denies ever having surgery. In these cases presumably the disease process has selectively resulted in bone erosion of the outer attic wall and in some cases the whole posterior meatal wall

- It is well recognized that hearing is often preserved until a very late stage in ears containing cholesteatoma in spite of the ossicular chain being disrupted.
- Patient, hearing preservation occurs because the cholesteatoma sac bridges the gap between the functioning part of the ossicular chain and the inner ear.
- The unfortunate consequence is that removal of the disease surgically may reduce the hearing.
- Sometimes there is an aural polyp obscuring the attic or posterior pars tensa; such a case should be assumed to be a cholesteatoma until proven otherwise.
- most experienced surgeons do not consider radiology necessary, unless intracranial complications of disease are suspected.
- Surgical removal is the only effective treatment for cholesteatoma.

THE AIMS OF SURGERY FOR ACTIVE SQUAMOUS COM ARE:

1. Eradication Of Disease;
2. An Epithelialized, Self-Cleaning Ear;
3. Hearing Improvement

- There are many different surgical techniques for managing active squamous COM, which can largely be categorized as open cavity (canal wall down) and closed cavity (intact canal wall) mastoidectomy.

CANAL WALL DOWN MASTOIDECTOMY

- Large cavities can be problematic: many continue to discharge and, even if well epithelialized, they often do not self-clean so regular clinic attendance is required for removal of squamous debris and wax from the cavity.
- Small cavity mastoidectomy, or attico-antrostomy, the anterior to posterior approach, has become more popular.
- The cholesteatoma is identified in the epitympanum or posterior mesotympanum and followed backwards.
- The problems of mastoid cavities can be reduced by good surgical technique and by partial obliteration of cavities, either with cartilage or prosthetic materials. The cavity must be rounded and smoothly contoured with no overhanging ridges and no facial ridge in order to allow migration of epithelium.
- Canal wall down surgery has lower rates of recurrence of cholesteatoma (5-15 percent).

INTACT CANAL WALL MASTOIDECTOMY

- incidence of recurrence of cholesteatoma is high (20-50 percent), therefore second look operations after 12-18 months are necessary in almost all cases, and some cases require further procedures subsequently.
- It has been claimed at times that postoperative hearing results are better following intact canal wall mastoidectomy.
- Cholesteatoma arising from the pars tensa may be confined to the middle ear, most commonly growing into the facial recess and sometimes spreading Anteriorly beneath the pars tensa and malleus handle.
- OSSICULOPLASTY IN ACTIVE SQUAMOUS COM
- The incus is probably the most commonly used prosthesis for ossicular reconstruction.
- There is thus the theoretical risk that the use of such an incus could cause a recurrent cholesteatoma BUT NONE REPORTED YET
However, many surgeons nowadays prefer to obliterate, at least partially with bone wax or hydroxapatite granules.

HEARING AIDS

- The person with the conductive hearing impairment will, however, derive more speech recognition benefit from hearing aids than the person with the sensorineural hearing impairment.
- **IN INACTIVE COM** ➔ A sensible management plan in such a patient would be to try a hearing aid. If the ear becomes active, then the tympanic membrane should be repaired.
- The only presenting symptom in patients with inactive squamous disease is hearing impairment. If they have a hearing disability then a hearing aid is often the most effective management.
- **Hearing Aid in active ears may exacerbates Otorrhoea** the Otorrhoea should be managed first and the preferred option is usually surgical repair of the tympanic membrane.
- These patients can often be effectively managed with a boneanchored hearing aid (BAHA).
- Facial Nerve Complications: More common in squamous disease more so in presence of granulations where granulations can form over the nerve sheath itself.
- The matrix should then be carefully lifted and the fistula identified. It is then peeled off the membranous labyrinth and the fistula is immediately sealed with fascia and bone dust. The fascial graft for the tympanic membrane provides a further layer of closure.
- Facial nerve complications
- In the presence of a facial nerve palsy, active COM should be managed urgently and almost always surgically.