Anatomy:

The external ear: Auricle, External auditory canal and Tympanic membrane.

The middle ear: Communicates with Eustachian tube and Mastoid air cells.

The inner ear: Bony labyrinth and membranous labyrinth.

I- **External Ear**

1- **The auricle** - Developed from 6 tubercles around first branchial arch
   - Cupped structure formed of cartilage covered by skin.
   - It shows following features.

2- **External auditory canal (EAC):** - Developed from first branchial arch
   - S shaped, 24mm length - Lateral 1/3 cartilaginous, medial 2/3 bony
   - Lined by skin: In cartilaginous part: contains hairs with sebaceous & ceruminous glands that secretes cerumin (wax).
   - In bony part: no hairs or glands.

3- **Tympanic membrane (Drum)**
- Separates EAC from middle ear
- Lies in a bony groove called tympanic sulcus deficient above
- Oval (10x8mm), 0.1mm thickness, semitransparent, pearly Grey, it lies obliquely so it shows a cone of light anteroinferiorly.
- Concave laterally.
- Composed of 2 parts:
  a- Pars tensa
  b- Pars flaccida
- Formed of 3 layers
  a- Outer squamous layer
  b- Middle fibrous layer
  c- Inner mucosal layer

**II- Middle Ear**

1- **Middle ear cavity (Tympanic cavity):**
- 6 walled box, developed from first and second branchial arches.
- 15mm in height, 15mm anteroposterior, but narrow from side to side.

  **Parts:**
  a- Mesotympanum .......... opposite tympanic membrane
  b- Epitympanum (attic)......... above tympanic membrane
  c- Hypotympanum .......... below tympanic membrane

  **Walls:**
  1- Roof: thin plate of bone (tegmen tympani) separating the attic from dura of MCF
  2- Floor: thin plate of bone separating ME from the jugular bulb
  3- Anterior wall:
    a- canal for tensor tympani muscle
    b- opening of Eustachain tube
    c- bony plate separating ME from ICA
  4- Posterior wall:
a- Aditus ad antrum (the opening connecting antrum to attic)
b- Pyramid: bony process containing stapedius m.
c- Vertical part of facial bony canal

5- Medial wall:

a- Promontory: elevation by basal turn of cochlea
b- Oval window: above & behind the promontory, closed by foot plate of stapes
c- Round window: below & behind the promontory, closed by 2nd T.M

d- Sinus tympani between b & c

e- Processes cochleariformis: bony projection, landmark for FN.
f- Horizontal part of facial bony canal

2- Eustachian Tube

Site: connect middle ear with nasopharynx

Shape:
- 36 mm
- Directed downwards, forwards & medially
- In children it is shorter, wider, more horizontal

Structure:
- Lateral 1/3 is bony
- Medial 2/3 is cartilaginous
- Lined with respiratory mucosa
**Function:** Closed at rest, opens only during swallowing & yawing to allow inflow of air to ME so equalize air pressure on both sides the T.M. It is opened by contraction of tensor palati muscle.

3- **Mastoid air cells**

- Lie within mastoid process which is 3 sided pyramidal bone. (part of temporal bone).
- Air filled cavities lined by m.m. and arranged in groups.
- Mastoid process is divided according to degree of cellularity into:
  1- Pneumatic
  2- Diploic
  3- Sclerotic

**III- Inner Ear (Labyrinth)**

( Neural in origin)

- Consists of a membranous laryrinth surrounded by a bony labyrinth
- The membranous labyrinth is surrounded by perilymph, filled with endolymph, it contains the labyrinthine sensory end organ

A- **Bony labyrinth**

- Bony cochlea: coiled canal resembles snail shell, 2½ turns
- 3 bony semicircular canals: (lat, sup. & post) each is 2/3 of circle lie in 3 different planes
- Vestibule: between the cochlea & S.C.C

B- **Membranous labyrinth**

- Membranous cochlea
- Membranous semicircular canals
- Utricle & saccule within the vestibule
- Endolymphatic duct & sac

Labyrinth is divided into:
1- Cochlear portion for hearing (organ of corti)

2- Vestibular portion for equilibrium

   Sensory end organ of utricle & saccule: macula

   Sensory end organ of S.C.C: crista

**Nerve supply of the ear**

**A- Sensory**

1- Auricle

2- EAC ant. Part, auriculo temporal ............... mandibular ........ V

   Post part, auricular branch of vagus (arnold’s)

3- Drum like E.A.C

4- middle ear tympanic branch of IX (jacobson)

5- mastoid antrum & cells: trigeminal

**B- Motor**

1- Auricular muscles: VII. 2- Tensor tympani: V. 3- Stapedius VII

**Arterial supply:**

EAC: auriculotemporal branch of superficial temporal artery

   Postauricular branch of ECA

Middle ear: tympanic branches

Labyrinth: internal auditory artery from the AICA (anterior inferior cerebellar artery)

**Functions of the ear**

**A- Hearing**

1- Auricle collects sounds 2- EAC transmits sounds, protect T.M
3-ME conduct sound from TM to OW via ossicles, magnify sound about 20 times due to:

a- Areal ratio between vibrating surface of T.M & oral window is 17:1
b- Lever ratio between the arm of malleus and arm of incus 1.3:1

Over all amplification is \(17 \times 1.3 = 22\) (middle ear transformer mechanism)

4- Inner ear

Converts the mechanical sound vibration to electrical impulses: Vibration of the footplate of stapes in oval window ……… vibration of the cochlear fluids

Vibration of the basilar membrane …………. stimulation of cochlear hair cells which lie on the basilar membrane (in the organ of corti) conversion of the mechanical sound vibration into electrical impulses.

Cochlear nerve transmits the electrical impulses ………. cochlear nuclei………… higher hearing centers in the brain

**B- Equilibrium**

Maintenance of equilibrium occurs in 3 steps:

1- the brain receives sensory informations from

   a- vestibular part of inner ear which is stimulated by head movement
      - Cristae of SCC stimulated by angular acceleration
      - Maculae of utricle & saccule stimulated by linear acceleration

   b- Visual impulses

   c- proprioceptors of muscles, joints & tendons

2- the brain integrates sensory information

3- the brain sends motor orders to

   a- Extra occular muscles

   b- Spinal muscles
**Symptoms & Examination**

**Symptoms of ear diseases**

1- Deafness unil. or bil.  
2- Tinnitus usually with deafness  
3- Discharge: character  
4- Pain  
5- Vertigo  
6- Facial asymmetry  

N.B.: symptoms of auricular diseases: pain and swelling

**Exam:**

a- **Auricle** Pre & post auricular regions  
   - Inspection: swelling, deformity, scar  
   - Palpation: tenderness, fistula sign  

b- **EAC & Drum**  
   * Ear speculum illuminated by  
     Head mirror, head light, otoscope, oto-microscope  
   * Pneumatic otoscope (Siegle’ speculum): Assess mobility of T.M  
   **C-Eustachian tube patency** Valsalva’s maneuver  

d- **Tuning fork tests**  
   - We use forks 64, 128, 256, 512, 1024, 2056 Hz ,best is 512 as it is equally heard & felt
**Rinne test**

- it compares hearing by air conduction (AC) with hearing by bone conduction (BC) in the same ear
- for BC put the tuning fork on the mastoid
- for AC put the tuning fork at 8mm distance from EAC & patient is asked to tell which position sounds louder
  - in normal hearing AC is better than BC due to amplification, it is called R +ve
  - C.D: BC is better than AC (if > 15dB loss): R –ve
  - P.D: AC is better than BC, but both are reduced: reduced R +ve
  - Severe P.D: BC is heard due to transcranial hearing : by other side : false R –ve

**Weber test**

- it compares hearing by bone conduction in both ears
- base of vibrating tuning fork is placed over midline of forehead or central incisors
  - normal: sound is heard equally (weber central)
  - P.D: sound is heard in normal ear if bil P.D sound is heard in better ear
  - C.D: sound is heard in diseased ear if bil CD sound is heard in the ear with more C.D

**E- Clinical examination of equilibrium**

**Investigations**

1- Culture & sensitivity: if discharge  2-Hearing tests:  PTA
3-Radiology:  Xray or CT  4-Vestibular tests
# Diseases of the Auricle

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<th>Deformities</th>
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<td>Traumatic</td>
<td>Haematoma, laceration, frost bite</td>
</tr>
<tr>
<td>Inflammatory</td>
<td>Perichondritis</td>
</tr>
<tr>
<td>Neoplastic</td>
<td>Benign – malignant</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>----</td>
</tr>
</tbody>
</table>

## I- Congenital anomalies:

a- **Auricular atresia (aplasia)**: Varied from microtia to anotia
   
   ttt plastic surgery

b- **Pre auricular fistula, cyst, sinus, appendages (accessory auricle)**
   
   ttt excision

c- **Macrotia: large auricle**: ttt plastic surgery

d- **Protruding ear (bat ear)**: ttt plastic surgery

## II- Traumatic conditions

### A- Haematoma

**Def:** blood collection between cartilage & perichondrium

**Aet:** blunt trauma

**Incid:** common as auricle is exposed

**Symptoms:** pain, swelling

**Signs:** bluish, smooth cystic swelling

**Complications:** perichondritis

**Ttt:** aspiration if early or evacuation if clotted.

  - Pressure.
  - Antibiotics.
**B- Laceration**

**Def:** auricular wound  
**Aet:** sharp trauma  
**Incid:** common  
**Path:** varies from mild, to avulsion  
**Symptom:** pain, bleeding  
**Signs:** as pathology  
**Ttt:** surgical repair - Antibiotics

**III- Perichondritis**

**Def:** infection of auricular cartilage  
**Aet:** infected haematoma or laceration  
- Furunculosis of EAC  
- Following surgery if septic  
- Burns  
**Organism:** usually strept pyogenes  
**Signs:** red, hot, tender, auricle  
**Complication:**  
- cartilage necrosis ............. Cauliflower ear  
**Ttt:** 1- systemic & local antibiotics  
- analgesics  
- 3- Small incision (s) ...... removal of necrosed cartilage, drainage.

**IV- Tumors of the auricle**

1- Basal cell carcinoma (rodent ulcer)  
Rolled in beaded edge, indurated base  
**Ttt:** Excision with safety margin

2- Squamous cell carcinoma  
Ulcer é raised everted edge, necrotic floor, indurated base ± LN metastasis  
**Ttt:** excision &/or radio therapy ± RND
Diseases of the External Auditory Canal

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<tr>
<th>Congenital</th>
<th>Atresia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Traumatic</td>
<td>F.B – fracture base</td>
</tr>
<tr>
<td>Inflammatory</td>
<td>Otitis externa</td>
</tr>
<tr>
<td>Neoplastic</td>
<td>Benign – malignant</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>Wax – wash</td>
</tr>
</tbody>
</table>

**I- Congenital meatal atresia**

*Def:* congenital obstruction of external auditory canal.

*Aet:* failure of canalization of part of 1st branchial arch

*Incid:* unilateral or bilateral

*Path:* bony obstruction (usually) or thin membrane.

*Symptoms:* conductive deafness ± P.D ± microtia (usually)

*Signs:* obstructed EAC ± microtia

*Investigations:*

1- CT to assess inner ear, mastoid pneumatization(facial nerve)

2- Hearing tests

*Ttt:* - Unilateral: elective canaloplasty,and auriculoplasty(school age)
   - Bilateral: - at least one side - Hearing aid

*NB:* bone anchored hearing aid

**II- F.B**

*Incid:* children – mentally retarded

*Types:* animate: flies

Inanimate: Vegetable eg beans, seeds

Non vegetable eg buttons, stones
**Symptoms:** - deafness
  - Tinnitus (noise is case of animate F.B)
  - pain and discharge, if infected

**Signs:** F.B is seen by exam.

**Ttt:** - Animate: killed by oily drops, then wash
  - Inanimate: instrument or wash
  - In uncooperative patients use GA & microscopic removal

### III- Otitis externa

**Def:** inflammation of skin of EAC

**Types:**

<table>
<thead>
<tr>
<th></th>
<th>Infective</th>
<th>Non infective</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Bacterial</td>
<td>Fungal</td>
</tr>
<tr>
<td>Furunculosis</td>
<td>Otomycosis</td>
<td>H zoster</td>
</tr>
<tr>
<td>Diffuse O.E</td>
<td></td>
<td>H. simplex</td>
</tr>
<tr>
<td>Malignant O.E</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Furunculosis** (Localized otitis externa)

**Def:** Acute staphylococcus infection of a hair follicle

**Aet:** staphylococcus aureus

**Incide:** more in D.M

**Path:** folliculitis ………. furuncle / outer part only

**Symptoms:** 1- Severe pain (as skin is tightly attached to perichondrium) on mastication

2- Deafness, if large.

3 discharge, if rupture (scanty, purulent)
**Signs:** 1-Tenderness on moving auricle, pressure on tragus
2-Localized red tender swelling
3-Discharge
4-Pre & post auricular lymphadenopathy

**Investigations:** blood sugar

**D.D:** acute mastoiditis

**Ttt:** - systemic: antibiotics & analgesics
   - Local: - aural toilet (suction or dry mopping)
   - packing the EAC with gauze strip soaked with glycerine icthyol or steroids & antiseptic
   - Local heat
   - Incision if abscess forms.

**Diffuse otitis externa**

**Def:** acute diffuse inflammation of skin of EAC.

**Aet:** predisposing factors:
1- Decreased immunity eg D.M
2- Hot humid atmosphere
3-Moisture, from bathing or swimming
4- Mild trauma from scratching

**Organisms:** staph, strept, pseudomonas, proteus

**Symptoms:** 1- Severe pain on mastication
2-Deafness due to edema
3-Scanty purulent discharge

**Signs:** 1-Tenderness on moving auricle, tragal
2-Diffuse redness & edema
3-Scanty purulent discharge
4-Possible adenopathy

**Investigations:** blood sugar C & S

**ttt:** Systemic: antibiotics & analgesics

Local: aural toilet (suction or dry mopping)

Packing EAC with gauze strip soaked with antibiotic & steroid.

**Malignant otitis externa**

**Def:** persistent severe infection that extends to involve bone & soft tissues of skull base

**Aet:** predisposing factors: - D.M -low immunity (AIDS, chemotherapy)
**Organisms:** pseudomonas, proteus

**Incid:** old, D.M

**Path:** - micro angiopathy ....... osteomyetitis of temporal bone

**Symptoms** 1-Severe deep seated pain, worse by night

2-Scanty purulent discharge.

**Signs** 1-Granulations in the bony-cartilagenous junction of EAC

2-Manifestation of extension of infection

**Investigations:**

- Blood sugar
- C & S
- biopsy

- Renal functions
- PTA
- Bone scan

- ESR
- CT (Gallium, technicium)

**Treatment:**

1- Hospitalization – control D.M

2- Antibiotics for 6 weeks

   a- Quinolones eg ciprofloxacin

   b- Aminiglycosides + carpenicillin

   c- 3rd generation cephalosporins

3- local: a- aural toilet          b- antibiotic

4- surgical debridment

**Otomyosis**

**Def:** Fungal infection of the EAC

**Aet:** Aspergillus (niger, fumigatus)          - Candida albicans

Predisposing factors: general:          - Local: antibiotics drops
Symptoms: 
1- Itching: .......... pain
2- Deafness, if obstruct EAC
3- Creamy or grayish white discharge

Signs: 
1- Skin erythema
2- Fungal mass black, gray .......wet newspaper
   White .......... fluffy cotton

Ttt: 
1-remove fungal mass (suction, wash)
2-Local antifungal:
   Nystatine alcohol + salicylic acid (keratolytic)
3-pack with antifungal cream

Viral otitis externa

a- Herpes simplex
   Occurs in lowered resistance; Bullous erythema of the canal

b- Herpes zoster
   - Caused by varicella zoster virus
   - Unilateral pain and vesicles (affects cyma conchae)
   - Ramsy-Hunt syndrome otalgia + VII palsy + SNHL
   - ttt: - Analgesics
   - Topical & systemic acyclovir
   - Steroids in VII palsy

c- Bullous otitis exerna
Viral infection of the EAC & outer layer of the TM, usually post influenzal
- pain and serous bloody discharge
- Bluish haemorrhagic bullae on EAC & T.M

Ttt: - systemic & local antibiotics
   - Analgesics

Allergic otitis externa
   Acute: itching, redness,& vesicles  Chronic: itching, fissuring,&scalling
Ttt: remove the cause  local steroids

Seborrhoic otitis externa
   Occurs with scalp seborrhea,Itching & scaling of EAC
Ttt: of seborrhoic dermatitis  local steroids
IV- **Tumors**

1- **Benign:** Exostosis
   
   **Def:** benign neoplasm of bone
   
   **Aet:** diving & swimming in cold water
   
   **Path:** diffuse or localized ivory osteoma covered by intact skin
   
   **Symptoms:** silent - deafness if large (wax impaction)
   
   **Sings:** multiple, bilateral, smooth, bony swelling
   
   **Invest:** CT
   
   **ttt:** excision by drill if symptomatic

2- **Malignant:**
   
   a- **Basal cell carcinoma**
   
   Common in fair people, exposed to sun
   
   Inverted beaded edge, necrotic floor & hard base, no LN
   
   TTt: local excision with safety margin
   
   b- **Squamous cell carcinoma**
   
   See carcinoma of middle ear

V- **WAX**

**Def:** accumulation of wax in EAC

Wax is the normal brownish secretion of ceruminous & sebaceous glands. Normally it is cleared off by epithelial migration

**Symptoms:** deafness: (after bathing) Tinnitus

**Signs:**

- Hard wax (brown)
- Soft wax (yellow)

**Ttt:**

- soft ............ wash.

  Hard: softened by glycerin bicarbonate drops ............ wash.
**Ear wash**

**Indication:**

1. wax
2. F.B
3. Otomycosis !
4. Caloric test

**Contraindications:**

- vegetable or impacted F.B
- Dry perforation
- CSOM
- Post – operative

**Complication:**

1. Injury to: a- The T.M : perforation: pain, bleeding, water in the throat 
   b- EAC
2. Infection: otitis externa.
3. Vertigo: caloric stimulation
4. Vasovagal attack or cough due to stimulation of X

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**Middle ear diseases**

<table>
<thead>
<tr>
<th>Category</th>
<th>Description</th>
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</thead>
<tbody>
<tr>
<td>congenital</td>
<td>Aplasia, Ossicular anomalies &amp; Wall dehiscence</td>
</tr>
<tr>
<td>traumatic</td>
<td>Rupture drum, Otitic barotraumas &amp; Fracture base</td>
</tr>
<tr>
<td>inflammatory</td>
<td>Otitis media</td>
</tr>
<tr>
<td>Neoplastic</td>
<td>Glomus &amp; Sq. cell carcinoma</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>Otosclerosis Facial nerve</td>
</tr>
</tbody>
</table>

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**Congenital anomalies**

1. Aplasia or hypoplasia: may be associated with meatal atresia.
2. Ossicular anomalies: adherent to the roof or together.
3. Congenital dehiscence: one of the walls, or facial canal.
**Otitis media**

<table>
<thead>
<tr>
<th>Acute</th>
<th>Chronic</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Non specific</td>
</tr>
<tr>
<td><strong>Suppurative:</strong></td>
<td><strong>Non suppurative:</strong></td>
</tr>
<tr>
<td>Tubotympanic</td>
<td>Secretory</td>
</tr>
<tr>
<td>Cholesteatoma</td>
<td>Atelectatic</td>
</tr>
<tr>
<td></td>
<td>Adhesive</td>
</tr>
<tr>
<td></td>
<td>Tympanosclerosis</td>
</tr>
</tbody>
</table>

**Acute otitis media**

**Def:** acute inflammation of mucosal lining of middle ear cleft.

**Aet:** Organisms: strept.pneumonia, haemophilus influenza & moraxilla catarhalis

**Routes of spread**

a- Via eustachian tube

Infection: common cold, tonsillitis, sinusitis

Infected material: vomitus, water

b- via T.M perforation or ventilation tube

c- blood spread: very rare

**Incidence:** more in infant & children .......... see later

**Pathology & clinical picture:**

1- *stage of eustachian tube obstruction (tubal catarrh)*

**Path:** tubal obstruction due to congestion, then air is absorbed.

**Symptoms:** 1- fullness in the ear 2-deafness 3-tinnitus

**Signs:** - Retracted T.M

Prominent lat. process of malleus

Exaggerated malleolar folds

Shortened handle of malleus

Distorted cone of light

Limited mobility by siegle

- C.D
2- **stage of hyperaemia & congestion : (catarrhal O.M)**

**Path:** oedema & congestion of ME & serous exudate.

**Symptoms:**
1- starting general symptoms  
2- deafness  
3- tinnitus  
4- pain

**Signs:**
- T.M is congested & prominent vascularity, lost cone of light  
- C.D

3- **stage of suppuration**

**Path:** pus accumulates under tension.

**Symptoms:**
1- more severe general symptoms  
2- Deafness increase  
3- Tinnitus increase  
4- Pain: severe, throbbing

**Signs:**
- T.M is congested markedly & bulging with lost cone of light  
- C.D

4- **Stage of perforation**

**Path:** necrosis of part of T.M

**Symptoms:**
1- general symptoms start to subside  
2- aural discharge  
3- deafness  
4- pain starts to subside

**Signs:**
- Mucopurulent bloody pulsating discharge  
- Perforated T.M: usually anteroinferior, if large ME mucosa appears congested  
- C.D

**Investigations:**
1- C & S  
2- Hearing tests: C.D
**Treatment:** depends on stage

I- before perforation:

a- *medical:* for 10-14 days
   1- Rest, light diet, fluids       2- Systemic antibiotics
   3- Analgesics                    4- Nasal decongestants
   5- Local warm glycerin phenol drops decrease edema & pain

b- *surgical:* myringotomy
   
   Indications: 1- failed medical ttt for 48 hours
                 2- bulging T.M (pus under tension)
                 3- complications
   
   After myringotomy:
   - Antibiotics according to C & S   - Local drops
   - Avoid water entry                - Aural toilet

II- after perforation

a- *medical:* as after myringotomy

b- *surgical:* myringotomy if perforation is small or high

**Prognosis:**

1- Cure: middle ear mucosa & hearing return normal

2- Chronicity: suppurative or non.

3- Complications

**Acute suppurative otitis media in children**

**Incidence:** It is much commoner in children, because:

1- Route of infection is easier in children, because the Eustachian tube is shorter, wider & more horizontal.

2- Sources on infection are commoner in children, because:
   a- Upper respiratory infections are commoner.
   b- Infected materials passing through the tube are commoner.
      - Milk: it is commoner in bottle – fed than breast – fed infants due to supine position and contamination
      - Vomitus: gastro-enteritis is commoner in infants.

3- General resistance is lower in children because of teething, frequent gastro-enteritis & artificial feeding.
Symptoms:
1- Fever is higher in children. It may be accompanied by convulsions.
2- Vomiting & diarrhea. It may be mistaken for gastro-enteritis.
3- Continuous crying & the child do not sleep well.
4-The child pulls or rubs his ear & moves his head .

Signs: The infantile tympanic membrane is thicker than in adults so bulging occurs late

Treatment: Similar to adults but myringotomy is done early.

NB: Necrotizing otitis media:
Sever form follow exanthemata specially measles, massive necrosis of the TM

1- Secretory otitis media

Def: chronic non suppurative otitis media with accumulation of fluid in ME.

Aet: 1-Incomplete resolution of acute otitis media
2-Eustachian tube obstruction: dysfunction, adenoid, nasopharyngeal tumors, cleft palate, sinusitis, rhinitis
3-Allergy. 4-Barotrauma. 5- Radiotherapy

Incid: commonest cause of deafness in children

Path: fluid is either mucoid: exudate by glands serous transudate.

Symptoms: unilateral or bilateral. 1- Deafness main & may be only
2-bubbling in the ear 3-tinnitus

Signs: - retracted T.M see before
- color: amber yellow to dull gray
- may be fluid level (hair line) é air bubbles

Investigations:
1-Hearing test: C.D, tympanogram: type B
2-Radiology: Xray or CT nasopharynx
3- Nasopharyngoscopy, in adults with unilateral S.O.M
**Ttt:**

**I- Conservative:**

1- ttt of predisposing factors  
2- Systemic antibiotics  
3- Corticosteroids  
4- Mucolytics  
5- Nasal decongestants  
6- Valsalva maneuver

**II- Surgical**  
Myringotomy & insertion of ventilation tube:

- If medical ttt for 3 months fails.
- Complications: risk of atelectatic or adhesive OM
- Nasopharyngeal tumors; before radiotherapy

To drain & ventilate M.E

Types  
grommet (temporarily)  
T tube (permanent)

<table>
<thead>
<tr>
<th></th>
<th>2- Atelectatic O.M</th>
<th>3- Adhesive O.M</th>
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<tr>
<td><strong>Def:</strong></td>
<td>T.M retraction onto promontory &amp; ossicles</td>
<td>Retraction &amp; adhesions</td>
</tr>
<tr>
<td><strong>Aet:</strong></td>
<td>E.T dysfunction, S.O.M</td>
<td>Improper ttt of S.O.M, and A.O.M</td>
</tr>
<tr>
<td></td>
<td>Recurrent A.O.M</td>
<td></td>
</tr>
<tr>
<td><strong>Symptoms</strong></td>
<td>Deafness, tinnitus</td>
<td>Deafness, tinnitus</td>
</tr>
<tr>
<td><strong>Signs</strong></td>
<td>T.M is retracted. C.D</td>
<td>T.M retracted &amp; adherent C.D</td>
</tr>
<tr>
<td><strong>investigations</strong></td>
<td>PTA: C.D</td>
<td>Similar</td>
</tr>
<tr>
<td></td>
<td>Tympanogram: type C</td>
<td></td>
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<td></td>
<td>Xray mastoid: poor pneumatization</td>
<td></td>
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<tr>
<td><strong>Ttt</strong></td>
<td>Proper ttt of S.O.M, A.O.M, insertion of T.tube</td>
<td>Early T.tube, late cartilage tympanoplasty</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hearing aid</td>
</tr>
</tbody>
</table>
4- Tympanosclerosis

**Def:** white area on the TM  
**Path:** hyaline degeneration of collagen & calcification (end point of healing), affect mainly TM & may affect ossicles  
**Aet:** CSOM (25%) complication of ventilation tubes (50%)  
**Symptoms:** History of discharge or ventilation tube, hearing loss  
**Signs:** white patches on the TM, may be perforation  
**Invest:** PTA : CD Tympanometry: reduced compliance  
**Ttt:** Medical: of discharge. Surgical: removal & TM grafting

**C.S.O.M**

**Def:** chronic inflammation of mucosal lining of middle ear cleft, characterized by irreversible pathological changes, intermittent or persistent discharge & perforation in T.M

**Types:**

1- Mucosal chronic suppurative otitis media (CSOM):

- It is also called “tubo – tympanic disease”, because it starts in the middle ear & Eustachian tube.
- It also called “safe CSOM”, because it is much less liable to cause complications, as the infection is mucosal.

2- Cholesteatoma:

- It is also called “attico – antral disease”, because it starts in the attic (epitympanum) & mastoid antrum.
- It is also called “unsafe CSOM”, because it is much more liable to cause complications, as it causes bone erosion.

**Tubotympanic CSOM**

**Aetiology**

It occurs usually secondary to A.S.O.M due to:

1- Inadequate treatment: inadequate antibiotic therapy, or inadequate drainage of discharge (small or high perforation).

2- Repeated middle ear infection through the Eustachian tube or through a persistent T.M. perforation.

3- High virulence of the organism  
4- Low resistance of the patient.

5- Persistent predisposing factor e.g. E.T. dysfunction.
**Symptoms:**
1- Persistent or intermittent aural discharge, profuse, mucoid or mucopurulent.
2- Deafness (mild).
3- Tinnitus.

**Signs:**
1- Aural discharge: it is mucopurulent or mucoid, usually odourless, and profuse in amount.
2- T.M. perforation: the perforation is usually central in the pars tensa. It may attain any size or shape.
3- Middle ear mucosa: if visible through the perforation may look:
   - Oedematous, congested and velvety pink during active infection.
   - Thin pale during the inactive phase
4- Tuning fork tests: variable degree of CD, due to: perforation, granulations

N.B.: the ossicular chain is usually intact, but it may be eroded and the commonest is the long process of incus.

**Investigation:**
1- Audiogram (PTA): conductive hearing loss.
2- Plain X-ray of mastoid: usually well pneumatised.
3- Culture and sensitivity of the discharge

**Treatment:**

a- Conservative treatment

The aim of conservative treatment is to obtain a safe dry ear to allow spontaneous healing of the perforation, or before surgery.

1- Systemic antibiotics:
   - Best according to culture and sensitivity, avoid ototoxic drugs
   - They may not be effective as fibrosis doesn’t allow antibiotics to reach tissues in a proper concentration.

2- Topical application of antibiotic ear drops but avoid ototoxic ones
3- Aural toilet: repeated local cleaning and removal of discharge by suction or dry mopping.
4- Avoidance of reinfection:
   o Avoid forcible blowing of the nose during rhinitis.
   o Avoid wetting the ear.
   o Control upper respiratory tract infection e.g.; sinusitis
   o Adenoidectomy, in cases of enlarged adenoids.

b- Surgical treatment:
   Tympanoplasty, with or without cortical mastoidectomy.

• Aim:
  1- Eradication of irreversible changes in the middle ear mucosa and to clear it from infection.
  2- Reconstruction of the conductive hearing mechanism (ossicular reconstruction).

• Cortical mastoidectomy is combined with tympanic membrane grafting, if there is persistent aural discharge (mastoid reservoir) to eradicate the mastoid pathology.

• The grafting material commonly used to repair the defect in the T.M. is temporalis fascia.

**Cholesteatoma (atticoantral disease)**

**Definition:**
The presence of a squamous epithelium within the middle ear (i.e. skin in an abnormal site).

Cholesteatoma is a misnomer because it is neither a tumor, nor it necessarily contains cholesterol crystals.

**Aetiology:**

A- Congenital cholesteatoma.
Develops from persistent embryonic squamous epithelial cells.
May be at different sites related to petrous bone
It is sterile so long as it is not connected with the external canal (i.e. cholesteatoma behind an intact T.M.)
Presents usually in adult life, with facial palsy

B- Acquired cholesteatoma
1- Primary acquired cholesteatoma (no previous history of O.M)
   Retraction pocket theory (Attic retraction): most accepted. Prolonged intratympanic negative pressure causes invagination of part of T.M. into the middle ear with formation of retraction pocket which becomes filled with keratin.
2 - Secondary acquired cholesteatoma (previous history of O.M.)

a - Migration therapy: direct migration of stratified squamous epithelium from surface of T.M. into the middle ear through a marginal perforation

b - Metaplasia theory: due to chronic irritation of the middle ear mucosa by chronic infection

Pathology:

A cholesteatoma is a sac lined by keratinizing stratified squamous epithelium (matrix) and is filled with concentric sheets of white-yellow keratin flakes, in which the cholesterol crystals may be embedded. It has an onion-like appearance on cut section.

Bone erosion is due to:

1- Osteolytic enzymes and collagenases

2- Secondary bacterial infection, so, the cholesteatoma becomes activated to secrete demineralizing and osteolytic enzymes.

3- Increase osteoclastic activity.

4- Pressure necrosis by the cholesteatoma sac (doubtful).

Symptoms:

1- Deafness (usually severe).

2- Aural discharge: scanty, purulent, & offensive.

3- Tinnitus.

4- Symptoms of complications if they arise.

Signs:

1- Aural discharge: occurs due to secondary infection of the cholesteatoma. It is purulent (never mucoid or mucopurulent), and may be bloody due to formed granulations and polypi.

   It is scanty and has a foul odour (characteristic) which is due to bone necrosis (osteitis) and anaerobic infection.

   It may contain cheesy white epithelial flakes (debris).

2- T.M. perforation: it is a marginal perforation (destruction of the bony annulus), in the posterosuperior quadrant of pars tensa or attic perforation.

3- A retraction pocket may be seen posterosuperiorly without perforation in the T.M (inactive).
4- Cholesteatoma itself may be seen as pearly white sheets or cheesy white masses of keratin (active).

5- Granulations are frequent and appear as sessile, fleshy red projections that bleed easily on touch, or polyp.

6- Signs of complications when they arise.

7- Tuning fork tests:
   - Variable degrees of conductive hearing loss, but may be mixed
   - Sometimes, there is normal or mild conductive hearing loss, and this occurs with early retraction pocket or when the cholesteatoma itself “bridges” the gap in the ossicular chain.

**Investigation:**

1- Audiogram (PTA): Variable degrees of conductive or mixed hearing loss.

2- Plain X-ray mastoid: The cholesteatoma appears as an irregular area surrounded by sclerosed bone, bone erosion.

3- CT scan of petrous bone: to detect the cholesteatoma mass, bone erosion and condition of the ossicular chain. It should always be done if complication is suspected.

4- Culture and sensitivity of discharge.

**Treatment:** (surgical)

The main line of treatment is surgery, aiming at safe & dry ear.

Two techniques are used to eradicate the cholesteatoma:

A. **Open (canal wall down) technique**
   
   This entails removal of all or a part of the posterosuperior bony meatal wall and includes:
   
   1- Radical mastoidectomy; this is the most common procedure performed
   
   2- Modified radical mastoidectomy: in limited cholesteatoma with good hearing.
   
   3- Atticotomy; in limited attic cholesteatoma

B. **Closed (Canal wall up) technique**
   
   - An opening is done in the posterior meatal wall (posterior tympanotomy), in a site called facial recess, via which small cholesteatoma could be removed
• A second look operation is mandatory after 6 months for detection of residual or recurrent cholesteatoma.

_Reconstruction of the radial cavity:_ may be needed including reconstruction of hearing after being sure of complete eradication of the cholesteatoma for a year or later on.

_Uses of endoscopy in cholesteatoma_

A 1.9mm endoscope is recently introduced

It is used to detect residual or recurrence 6 months after performing the closed technique.

_DD of aural polyp:_

1-inflamatory: A- CSOM (more with cholesteatoma)….explore and do polypectomy then manage according to the case

   B- Polyp arising from the canal

2-Neoplastic: a-Benign: glomus b- malignant:carcinoma

3-Miscellaneous: esinophilic granuloma

_Species of suppulsive otitis media_

_Def:_ spread of infection beyond mucoperiosteal lining of middle ear cleft

_Aet:_

1- A.S.O.M

   2- Acute exacerbation on top of CSOM

   3- Cholesteatoma

_Classification:_

1- Cranial (within temporal bone)

   a- Mastoiditis  b-Petrositis

   c-Labyrinthitis  d-Facial paralysis

2- Intra cranial

   a- Extradural abscess (& subdural)

   b- Meningitis  c-Brain abscess

   c- Lat. Sinus thrombosis

3- Extra cranial: otitis externa
**Routes of spread:**

1. Bone erosion by cholesteatoma & granulations.
2. Pre-formed pathway as fracture lines & congenital dehiscence.
3. Retrograde thrombo-phlebitis along venous drainage of the middle ear.

**Acute mastoiditis**

**Def:** Acute inflammation of mucosal lining of mastoid air cells

**Aet:** see before

**Incid:** the commonest complication

**Path:** osteoporosis of bony wall (hyperaemic decalcification) ..........ischaemic necrosis ..........mastoid forms large cavity filled pus extension of infection leads to:

a- subperiosteal abscess:
   - Sagging of postero sup. meatal wall
   - Post auricular abscess
   - Zygomatic abscess.
   - Bezold abscess: sheath of sternomastoid
   - Citteli abscess: lat. Pharyngeal space

b- mastoid (post auricular) fistula

**Symptoms:**

Symptoms of O.M becomes more sever

1. general: fever, headache, malaise, anorexia
2. deafness: increase
3. tinnitus
4. discharge: increase
5. Pain: Dull aching: Retro auricular radiates down & back
   - Increase in recumbent position
   - Throbbing if abscess
6. Swelling
**Sings:**

1. **general:** fever, tachycardia

2. **external exam:**
   - Acute mastoiditis:
     - tenderness over antrum (cymba conchae), posterior border, tip
     - edema over mastoid
   - mastoid abscess: external fluctuant swelling
     - a- post auricular: pushing the auricle downwards & forwards é preserved retro auricular sulcus
     - b- zygomatic: above & infront of the auricle
     - c- bezold’s: upper part of the neck
   - mastoid fistula

3. **Ear exam:**
   - discharge : M.P, profuse, recurs rapidly (reservoir sign)
   - Sagging of postero sup. meatal wall (diagnostic)
   - T.M usually perforated or congested

4. **tuning pork:** C.D

**Investigations:**

1. Hearing tests: C.D
2. C & S
3. X-ray mastoid: clouding & blurring
4. C.T: exclude other complications

**Treatment:**

*Medical:*  
- systemic antibiotics according to C & S
- analgesics & anti inflammatory
- local antibiotics -Avoid reinfection

*Surgical:*  
Cortical mastoidectomy if:  
- Failed medical ttt for 48h,
  - Mastoid abscess
  - Other complications
Myringotomy in acute otitis media
Radical mastoidectomy if cholesteatoma
**D.D** - Acute otitis media: never tip tenderness, shorter history, & normal X ray
- Post auricular lymphadenitis
- Lipoma, hematoma over mastoid

**Petrositis**

**def:** inflammation of air cells in petrous apex

**Aet:** as mastoiditis    **Incid:** rare, only 30% have cellular apex, usually in diabetics

**Path:** as mastoiditis

**Clinical picture** (Gradenigo triade)

1- Aural discharge
2- Fcial pain due to affection of V ganglion
3- Diplopia & squint due to VI paralysis

**Investigations:**

1- hearing tests    2- C & S    3- C.T
**Treatment:**

a- medical as mastoiditis

b- surgical:  
1- Treat ear disease
2- Drainage of infected petrous air cells

---

**Labyrinthitis (otitic)**

**Def:** Inflammation of inner ear as complication of middle ear suppuration

**Aet:** as mastoiditis

**Path:**

1- Labyrinthine fistula (circumscribed labyrinthitis): localized erosion of the bony labyrinth by cholesteatoma producing a fistula between ME & IE

2- Diffuse serous labyrinthitis: accumulation of serous fluid in membranous labyrinth, still no pus

3- Diffuse suppurative labyrinthitis: accumulation of pus in membranous labyrinth é degeneration of hair cells then:
   - healing by fibrosis (dead ear)  
   - intra cranial extension of infection

**Clinical picture**  Sppurative otitis media  
- Clinical picture depends on stage:

1- **labyrinthine fistula**
   - silent
   - vertigo: mild, transient, induced by sudden movement, tragal pressure, , no nausea or vomiting
   - nystagmus, rapid phase……….diseased ear
   - positive fistula test (diagnostic)
     - A test to detect labyrinthine fistula
     - Brief vertigo ± nystagmus
     - On: siegle, tragal pressure, manipulation of aural polyp
     - Positive test :vertigo ± nystagmus
     - False negative: - very small  
       - Non functioning ear - inadequate sealing - Cholesteatoma mass prevents pressure transmission

2- **Serous labyrinthitis**
   - vertigo: severe, continuous, spontaneous with nausea & vomiting
   - nystagmus: spontaneous ,directed to diseased ear
   - reversible SNHL
3- suppurative labyrinthitis

- Similar to serous labyrinthitis but more severe
- Nystagmus with rapid phase …….. normal side
- irreversible SNHL (dead labyrinth)
- no or minimal general manifestation
- distinction between 2 & 3 is retrospective

Investigations:
1- hearing tests 2- C & S 3- C.T

Treatment
A- medical:
1- hospitalization, rest, fluids
2- sedatives, antivertigo drugs
3- systemic massive antibiotics specially that crosses BBB: sulpha, chloramphenicol & cephalosporins
4- treat middle ear infection

B- surgical
- in fistula: mastoidectomy, remove the cholesteatoma & cover fistula è graft
- in suppurative labyrinthitis è SNHL & vertigo surgical labyrinthectomy used to be done but rarely needed nowadays due to powerful antibiotics

Facial paralysis (Complicating suppurative O.M)

Aet: ASOM è dehiscence in bony canal
CSOM è cholesteatoma or granulations eroding the canal

Clinically: LMNL facial paralysis (acute in ASOM and gradual in CSOM)
In A.S.O.M: fever, earache, deafness, congested or pulging drum
In C.S.O.M: deafness, discharge, usually cholesteatoma

Investigations: Audiogram C & S C.T Tests for facial nerve
**Treatment:**
1- In A.S.O.M: myringotomy, antibiotics, and steroids.
2- In mastoiditis: cortical mastoidectomy, antibiotics, and steroids.
3- In cholesteatoma: radical mastoidectomy, antibiotics, and steroids.

**Extradural abscess**

*Def:* collection of pus between the dura & adjacent bones  
*Aet:* cholesteatoma  
*Path:* middle or post cranial fossae  

**Clinical picture:**
1- Asymptomatic  
2- Ipsilateral temporal headache  
3- Low grade lever  
4- CSOM: ± pulsating discharge.  

**Investigations**  
- C.T - audiogram - C & S  

**Treatment**  
*Medical:* antibiotics  

*Surgical:* mastoidectomy with abscess drainage till healthy dura is exposed all around  

----------bone should be removed  

**Lateral sinus thrombophlebitis**

*Def:* infection & thrombosis of lat. venous sinus  
*Aet:* usually cholesteatoma  

**Path:**
1- Periphlebitis  
2- Endophlebitis: mural thrombus which occludes sinus lumen  
3- Infection & progression:  
   - septic emboli & systemic pyaemia  
   - extension of thrombosis  

**Clinical picture:**  
May be asymptomatic & discovered accidentally during surgery being masked by antibiotics (sterile)
A- manifestation of blood infection

1- Intermittent fever: attacks of fever 39-40 °C rigors (septic emboli) followed by fall of temperature, attacks are irregular & in-between patient seems well & mild fever
2- Severe occipital headache.
3- Malaise, anorexia, anemia, and toxic face.

B- Manifestation of thrombus extension

- upwards: to superior sagittal sinus (which absorbs CSF): increase ICT (otitis hydrocephalus): headache, projectile vomiting, papilloedema.
- downwards: internal jugular V.: neck pain & vein felt as tender cord like structure
- Forwards: via sup. Petrosal sinus: cavernous sinus thrombosis: ptosis, proptosis, chemosis & ophthalmoplegia
- laterally: mastoid emissary vein: tender edematous swelling of the skin over mastoid (Griessinger sign)

Investigations:

1- Blood picture: leucocytosis, anaemia, high ESR
2- Blood culture: +ve in bacteraemia (fever)
3- CT petrous bone & brain with contrast
4- MRA (best one)
5- Audiogram
6- C&S
7- Positive Queckenstedt’s (Tobey – Ayers)
   Pressure on IJV on normal side ……… increase CSF pressure
   Pressure on IJV on affected side ……… no change in CSF pressure
8- Fundus examination: papilloedema & engorged retinal veins

DD

1- Malaria: regular fever, leucopenia, +ve blood film for parasite
2- Typhoid: step ladder fever, leucopenia, +ve widal test, splenomegaly
3- Meningitis & brain abscess (by CT & MRI)
Treatment

a- Medical: Parentral antibiotics, antipyretics Anti coagulants in C.S.T

b- surgical

mastoidectomy with exposure of the sinus which is incised & evacuated till free blood flow is obtained ± ligatin of I.J.V

thromboosed sinus: grey, firm, pulsating, covered by granulations

normal one bluish, cystic, not pulsating smooth

Meningitis

Def: inflammation of the subarachnoid CSF space, which surrounds the brain & spinal cord

Aet: A.S.O.M or CSOM

Path: meningeal irritation........ serous fluid in CSF with oedema & congestion ........ cellular exudate & Bacterial invasion ........ pus formation.

Clinical picture: Infection of meninges & CSF

a- Manifestation of infection
  - High fever - headache, malaise, anorexia
  - tachycardia, toxic face

b- manifestation of meningeal irritation:
  - irritability & photophobia
  - neck rigidity - opsthotonus position
  - stretch signs:
    +ve Kernig’s: inability to extend knee when thigh flexed
    +ve brudziniski’s: flexion of knee & hip when neck flexed
    +ve babinski’s: dorsiflexion on stimulation

c- Manifestation of high ICT
  1- Severe headache
  2- Projectile vomiting
  3- Papilloedema...........blurring of vision
  4- Drowsiness............coma (late)
Investigations:

1- C.T
2- Lumber puncture & CSF analysis

<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>Meningitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>CSF</td>
<td>Clear</td>
<td>Turbid</td>
</tr>
<tr>
<td>Color</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pressure</td>
<td>100-150 mm H₂O</td>
<td>increased</td>
</tr>
<tr>
<td>Proteins</td>
<td>10-40 mg%</td>
<td>increased</td>
</tr>
<tr>
<td>Chlorides</td>
<td>700-750mg %</td>
<td>decreased</td>
</tr>
<tr>
<td>Sugar</td>
<td>40-80 mg %</td>
<td>decreased</td>
</tr>
<tr>
<td>Organisms</td>
<td>Absent</td>
<td>Present</td>
</tr>
<tr>
<td>cells</td>
<td>1-5 lymphocytes HPF</td>
<td>Specially PNLS</td>
</tr>
</tbody>
</table>

Treatment:

1- systemic antibiotics I.V cross BBB
   a: sulphonamides 2gm/4h
   b: chloramphenicol 500mg/6h
   c: metronidazol 100mL/8h
   d: 3rd generation cephalosporins
   e: crystalline penicillin / million /4h I.M intrathecal (not done)

2- Decrease ICT
   Mannital 20% - glucose 25% - lasix IV – steroids, repeated L.P

3- supportive measures
   - quiet dark room
   - adequate fluids & electrolytes
   - analgesics & antipyretics
   - care of comatosed

4- treatment of ear disease when general condition allows (cortical or radical)

Brain abscess

Def: collection of pus within brain substance.

Aet: Routes: as usual + perivascular space of Virchow Robin

   Organisms: anaerobic strept, staph, pneumococci

incid: commonest cause for brain abscess age: 10-30 years
Path:
- site temporal or cerebellar

- Stages:
a- Encephalitis in localized area
b- Localization: abscess formation surrounded by glial capsule
c- Enlargement: increased manifestation
d- Terminal: abscess rupture è fatal meningitis

Clinical picture:
a- stage of encephalitis (cerebral dysfunction)
   Apathy, loss of concentration   Headache, fever, malaise, anorexia
   Irritability drowsiness
b- Stage of localization (latent stage)
   General symptoms decreased   Mild fever & headache
c- Manifest stage (due to enlargement)
   1- manifestations of ICT
      • severe generalized headache
      • projectile vomiting
      • papilloedema…….. blurring of vision
      • Decreased temperature, pulse, respiratory rate, cerebration
      • abducent palsy – photophobia
   2- Toxemia:   Anorexia, malaise, foetid breath, toxic facies
   3- Focal neurologic manifestation
      I- temporal lobe abscess
         Nominal aphasia (speech center)
         Contralateral hemiplegia (motor center)
         Contralateral UMNL VII
         Contralateral hemianaesthesia (sensory area)
         Homonymous hemianopia (optic radiation)
II-cerebellar abscess

- nystagmus: slow spontaneous, pendular
- stacatto speech
- Ipsilateral hypotonia
- Intention tremors
- Positive rebound asymetria
- Disdiadokokinesi
- Ataxia

d- terminal stage (rupture): bradycardia, coma, death

**Investigations:**

1- CT è contrast (hypotense area surrounded by an enhancing ring) (ring sign)
2- MRI
3- C&S
4- audiogram

**Treatment:**

a- medical

1- systemic antibiotics I.V cross B.B.B (see before)
2- supportive measures
3- care of comatosed (if present)

b- surgical

1- abscess drainage

I- via craniotomy (burr holes)

II-transmastoid: Tegmen tympani for temporal

Trautman’s triangle for cerebellar

2- surgical ttt of ear disease

Radical mastoidectomy usually 10-14 days after abscess resolution

**Otitic hydrocephalus**

Raised CSF pressure due to thrombosed superior sagittal sinus
**Trauma to middle ear**

1- **Traumatic T.M rupture**

_Aet:_ self induced as during cleaning during ear wash
- During F.B removal
- Slap on the ear
- Blast injuries; compression and rarefaction waves

**Symptoms:**
1- Deafness
2- Otalgia
3- Bleeding per ear (does not clot if mixed with CSF)

**Signs:**
1- T.M perforation (see the table)
2- Blood clots in EAC
3- Tuning fork: C.D

**D.D**

<table>
<thead>
<tr>
<th></th>
<th>Traumatic</th>
<th>Pathological</th>
</tr>
</thead>
<tbody>
<tr>
<td>History</td>
<td>trauma</td>
<td>O.M</td>
</tr>
<tr>
<td>Discharge</td>
<td>absent</td>
<td>P or MP</td>
</tr>
<tr>
<td>Bleeding</td>
<td>present</td>
<td>if present: scanty</td>
</tr>
<tr>
<td>Perforation Site</td>
<td>pars tensa</td>
<td>anywhere</td>
</tr>
<tr>
<td>Type</td>
<td>nearly always central</td>
<td>central or marginal</td>
</tr>
<tr>
<td>Size</td>
<td>usually small</td>
<td>any size</td>
</tr>
<tr>
<td>Edge</td>
<td>sharp irregular</td>
<td>smooth, regular</td>
</tr>
<tr>
<td>ME mucosa</td>
<td>normal</td>
<td>usually congested</td>
</tr>
</tbody>
</table>

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Treatment:

a- conservative

Keep ear sterile, no water entry or ear drops
Systemic antibiotics, nasal decongestants & avoid nose blowing
In most cases it heals

b- Surgical: myringoplasty in persistent perforation (after 6 months).

2- Otitic barotrauma

Def: middle ear trauma due to rapid & marked decrease in middle ear pressure.

Aet: During ascent: atmospheric pressure decreases so middle ear pressure becomes relatively positive this is corrected by passive outflow of air from middle ear along E.T.

- During diving & airplane descent: atmospheric pressure increases so middle ear pressure becomes relatively negative this can be corrected only by active opening of E.T (by swallow) to allow air inflow to middle ear.

So otitic barotrauma occurs when E.T fails to open, this results in retraction, rupture, and effusion

Symptoms:

1- earache, discomfort & fullness in ear 2- deafness – tinnitus

Signs:

1- retracted congested T.M
2- middle ear effusion (may be haemotympanum)
3- T.M rupture 4- Tuning fork C.D

Investigations: Audiogram: C.D

Treatment:

A- Prophylactic

1- avoid flying è common cold
2- avoid sleep during descent
3- chewing gums
4- nasal & oral decongestants

B- Curative

In retraction: valsalva’s maneuver
In severe retraction or effusion: myringotomy
In rupture: as usual
Local & systemic decongestants
3- **Traumatic ossicular disruption**

*Sites:* incudo stapedial joint dislocation, long process of incus, crura of stapes

*Symptoms:* history of trauma, unilateral deafness& tinnitus, vertigo

*Signs:* Intact or perforated TM, marked CD, & positive fistula test

*Invest:* PTA: CD more than 50 dB, tympanometry: AD curve

*DD:* CD with intact drum

*Ttt:* ossiculoplasty

4- **Fracture Temporal Bone**

<table>
<thead>
<tr>
<th>Incidence</th>
<th>Longitudinal</th>
<th>Transverse</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cause</td>
<td>80% Trauma to side of head passes across longitudinal axis of petrous bone.</td>
<td>20% Trauma to occiput perpendicular to long axis of petrous bone.</td>
</tr>
<tr>
<td>Fracture line</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Symptoms</td>
<td>C.D = rupture T.M ossicles dislocation inner ear is free. Bleeding per ear. CSF otorrhea. Mastoid ecchymosis (battle sign) Tear in T.M, laceration of EAC Bleeding. - less common 15-20% usually partial</td>
<td>Sudden P.D as fracture crosses inner ear Sudden vertigo, nausea vomiting &amp; nystagmus. Usually patient is comatosed. Haemotympanum (bluish intact drum)</td>
</tr>
<tr>
<td>Signs</td>
<td></td>
<td>More common 50% torn or compressed.</td>
</tr>
<tr>
<td>Facial</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Investigations:*

1- Radiology X ray – CT – MRI. 2- Audiogram.

3- Vestibular tests 4- Electrical tests for facial nerve
**Treatment:**
- Avoid cough & straining
- Bed rest in semi sitting position.
- Care of comatosed
- Dressing to cover the ear no local ttt.
- Systemic antibiotics cross BBB
- Hearing aid for SNHL


Ossicles : ossiculoplasty.

Facial n.: see facial n.

Management of associated neurosurgical problems.

---

**Tumors of the Middle Ear**

(A) **Benign** glomus tumors: paraganglioma: chemodectoma.

**Def.:** locally invasive, highly vascular tumor.

**Incid:** 40 – 50 y. more in females.

**Path.:** arise from non chromaffin paraganglionic cells in relation to nerves.

Glomus jugular: in relation to Arnold’s nerve on the dome of jugular bulb.

Glomus tympanicum: in relation to jacobson’s nerve on the promontory.
Clinical picture

1 Otological

Symptoms:
1- Unilateral progressive deafness C → P.
2- Unilateral pulsatile tinnitus (characteristic).
3- Bloody otorrhea.
4- Deep seated otalgia.
5- ± Vertigo.

Signs:
a) Reddish mass behind intact drum (rising sun appearance) blanches by sieglization (Brown’s sign).
b) Vascular polyp that bleeds on touch. ± bruit on mastoid.

II- Cranial nerve palsies
- LMNL facial palsy. 9, 10, 11, 12 palsies in G j.

III- Increased ICT: see before.

Investigations:
1- CT: widening of jugular foramen, enhancing soft tissue mass destroying bone.
2- Angiography: tumor blush & feeding vessels.
3- MRI, MR angiography.
4- Audiogram & tympanometry.

Treatment:
(A) Surgery: tumor excision.
G.T: transcanal, post. tympanotomy, radical mastoidectomy.
G.J: infratemporal fossa approach type A.

Preoperative embolization 2-3 days before to ↓ bleeding.

(B) Inoperable cases: radiotherapy or embolization or both.

(B) Malignant: Sq. cell carcinoma.

Def.: malignant tumor of EAC or ME.
Aet.: CSOM – irradiation Incid.: rare, more in males.

Symptoms:
1- Discharge: blood stained, foul smelling, profuse.
2- Deafness: increased → SNHL later.
3- Deep seated pain. 4- Tinnitus increase.
5- Vertigo. 6- Facial paralysis due to erosion.

**Signs:**

1- Granulation & polypi friable, bleeds on touch.
2- Manifestation of spread.
   - Skin ulceration, TM J & parotid affection.
   - Enlarged hard fixed parotid or UDCLN.
   - Last 4 cranial n. palsies due to:

Involvement in jugular foramen.
Involvement in parapharyngeal space by metastasis to retropharyngeal LN.

**Investigations:**

1- X ray: bone erosion.
   C.T enhancing soft tissue mass e bone destruction carotid angiography.
2- Biopsy 3- Audiogram 4- Metastatic work up.

**Treatment:**

A- Surgical resection + post operative radiotherapy + RND.
B- Palliative radiotherapy.

**Otosclerosis**

**Def.:** Primary disease of otic capsule, characterized by replacement of normal compact lamellar bone by abnormal spongy bone of greater thickness, cellularity & vascularity with progressive fixation of stapes.

**Aet.:** may be due to:

1- Developmental defect. 2- Enzymatic abnormalities.

**Predisposing factors:**

a- Hereditary tendency: +ve family history in 50%.

b- Endocrinal:↑ by thyrotoxicosis & puberty.

c- Pregnancy: accelerates the process.

**Incid.:** Onset 15 – 45 y. Female = male 2/1.

Commonest cause of bil C.D in adults.
Bilateral, but unilateral in 10%. 
**Pathology:**

Replacement of normal compact lamellar bone by abnormal spongy bone of greater thickness, cellularity & vascularity with progressive fixation of stapes.

**Sites:** Ant. margin of oval window (fissula antfenstam) 85% of cases. Fissula post fenstram → Round w → cochlear capsule.

**Types:**
A: Stapedial CHL (commonest).
B: Pure cochlear: affection of endosteal layer of labyrinth → SNHL (rare).
C: Mixed: mixed H.L.

**Symptoms:** Insidious onset, progressive course.

1- Deafness: bilateral, progressive.
2- Tinnitus.
3- Paracusis willsii: hearing better in noisy places due raising of friends voice.

**Signs:**
- Quiet voice.
- Intact normal T.M, in active otosclerosis dilated B.V on promontory produce flamingo red tinge (schwartze sign).
- Tunning fork: bilateral C.D variable degree (rarely SNHL or mixed).

**Investigations:**
1- Hearing tests PTA: see before. Tympanometry: type As.
2- Radiology CT shows otosclerotic foci.
**Treatment:**

(A) **No ttt:** in mild cases AB gap < 20dB.

(B) **Surgical ttt:** stapedectomy: ttt of choice, done on worse ear 1\textsuperscript{st}.

(C) **Medical ttt:** Sodium fluoride therapy.

   **Indication:** Progressive SNHL.
   **Action:** Regress process of otosclerotic.
   **Dose:** 30mg enteric coated tab T.D.S. after meals up to 2 y with ca & vit.D.
   **Contraindication:** pregnancy, young age, renal dysfunction, rheumatoid.

(D) **Hearing aid.**

Good alternative to surgery if operation is contraindicated, refused, failed.

**Facial nerve**

**Anatomy:**

(A) **Intracranial part.** Facial nerve has 3 nuclie

1-Motor nucleus lies in pons.

2-Superior salivatory nuclies: in pons, parasympathetic secretomotor to lacrimal, submandibular, and sublingual glands

3-Nucleus solitarius: in medulla, sensory, carries taste sensation from the tongue

Motor fibers form the motor root, while parasympathetic and sensory fibers join to form the sensory root (nervus intermedius)

The 2 roots emerge on the side of the brain stem, at junction between pons & medulla to cross CPA to I.A.M. Controlled by pyramidal & extrapyramidal fibers.
(B) **Cranial (intratemporal) part**: 37-45mm.

- Labyrinthine segment: runs in the I.A.C superior to VIII & anterior to superior vestibular nerve, runs laterally to medial wall at geniculate ganglion.

- Tympanic (horizontal) segment: starts at geniculate ganglion, curves to form 1st genu, runs backwards in medial wall till the posterior wall.

- Mastoid (vertical) segment: Runs inferiority (2nd genu), lies antero inferior to lat. SCC, then vertically downwards to leave skull at stylomastoid foramen.

(C) **Extra cranial part**

Enters parotid gland → divides into terminal motor branches.

---

1-superior salivatory nucleus(14)
2-nucleus solitarius
3-pyramidal tract
4-motor nucleus
5-facial nerve

1-facial nerve 2-geniculate ganglion
3-greater sup. Petrosal 4-nerve to stapedius
5-chorda tympani 6-parotid gland
7-sensory to EAC 8-nerve to stylohyoid
9-nerve to post.belly of digastric 10-stylomastoid foramen
11-middle ear 12-second genu

**Branches**

(1) *In temporal bone.*
a) Greater superficial petrosal: arises at geniculate ganglion → secreto motor parasympathetic to lacrimal, nasal, palatine glands.

b) Nerve to stapedius: motor supply to stapedius.

c) Chorda tympani: exit just above stylo mastoid foramen to enter ME → carries taste from ant. 2/3 of tongue & secretomotor parasympathetic to sub lingual & sub mandibular glands.

(2) After exit from skull:

Two motor branches to stylohyoid & post belly of digastric. 

Posterior auricular nerve: motor to occipital belly of occipitofrontalis

(3) Five terminal branches in parotid:

Temporal, zygomatic, buccal, mandibular & cervical supply muscles of scalp, face, auricles & platysma.

(4) Sensory fibers to small part in post wall of EAC.

**Aetiology of facial paralysis**

I- **Supra nuclear (UMNL)**

a) Trauma to head.

b) Meningitis, encephalitis, and abscess.

c) Brain tumors.

d) Stroke Hge, thrombosis, embolism.

II- **Peripheral (LMNL)**

(A) Intracranial.

1) In pons: Congenital nuclear aplasia.

   - Basal meningitis
   - Pontine Hge.
   - Pontine tumors.
   - Multiple sclerosis.

2) In CPA: congenital cholesteatoma meningioma, vestibular schwannoma

(B) Cranial (intratemporal)

1) Traumatic.

   A: Birth trauma: forceps delivery.
B: Fracture especially transverse type.
C: Surgical: mastoidectomy, stapedectomy.

2) Inflammatory.
   A: A.O.M with dehiscence in facial canal
   B: CSOM, specially cholesteatoma.
   C: Herpes zoster oticus (Ramsy – Hunt syndrome).
   D: Malignant otitis externa

3) Neoplastic.
   A: glomus
   B: Sq. cell carcinoma
   C: Acoustic neuroma.

4) Idiopathic Bell’s palsy commonest cause.

(C) Extra cranial
   1- Trauma: cut wound in parotid Parotid or face surgery.
   2- Sarcoidosis of the parotid.
   3- Tumor: malignant parotid tumors.

(D) Miscellaneous
   - Polyneuritis (Guillane – Barre Syndrome).
   - DM – Lyme disease – T.B.
   - Milkersson Rosenthal Syndrome.

Pathology of facial paralysis.

1- Neuropraxia: functional conduction block, spontaneous complete recovery in one month.

2- Axonotmesis: damaged axons with intact myelin tube, axon grows at a rate 1mm/day, recovery in 2-3 month & may be incomplete.

3- Neurotmesis: degeneration of axons & myelin tubes, spontaneous recovery may occur in 1 y & always incomplete.

Clinical picture of facial paralysis

Depends on site of lesion: in LMNL.

(A) Paralysis of muscles of the face:
1- Inability to raise eye brow (occipito frontalis).
2- Inability to close eyes firmly (orbicularis oculi).
3- Inability to whistle (orbicularis oris).
4- Food collects beneath cheek (buccinator).
5- Deviation of angle of mouth to healthy side upon smiling, drooping of angle of month on affected side, dripping of saliva & loss of nasolabial fold (levator anguli oris)

(B)Sensory and parasympathetic affection
Affection of GSP: decreased lacrimation
Affection of nerve to stapedius: hyperacusis (disturbed hearing)
Affection of chorda tympani: metallic taste

Determine:
Tone: by comparing both sides at rest.
Power: by comparing both sides during movement.
Degree: partial or complete paralysis.

Differentiation between UMN and LMN paralysis.

<table>
<thead>
<tr>
<th>ULMN</th>
<th>LMNL</th>
</tr>
</thead>
<tbody>
<tr>
<td>* Paralysis of lower ½ of face.</td>
<td>* Total facial paralysis.</td>
</tr>
<tr>
<td>* Emotional movements (e.g.</td>
<td>* Emotional movements are lost.</td>
</tr>
<tr>
<td>Laughing) are intact.</td>
<td></td>
</tr>
<tr>
<td>* Associated hemiplegia.</td>
<td>* No hemiplegia.</td>
</tr>
<tr>
<td>* Hypertonia (spastic).</td>
<td>* Hypotonia (flaccid).</td>
</tr>
<tr>
<td>* No fasiculation.</td>
<td>* Fasiculations are present.</td>
</tr>
<tr>
<td>* No muscle atrophy.</td>
<td>* Muscle atrophy occurs later.</td>
</tr>
</tbody>
</table>

N.B: In UMN facial paralysis only the lower half of the face is paralyzed, this is because the upper part of the motor facial nucleus supplying the upper part of the face is bilaterally represented in the cerebral cortex.

Leveling of LMN facial paralysis: (according to presentation)
Investigations

A- To identify the cause.

1- CT scans of brain and petrous bone to show fractures, cholesteatoma, or tumors.

2- MRI petrous bone to show tumors especially facial neuroma.

3- Audiogram (PTA).

B- To detect the level of the lesion.

1- Schirmer’s test (test for lacrimation): It is significant when the differences between the lacrimal flow of both sides exceed 30% of the total bilateral lacrimation, indicating a lesion at or above the geniculate ganglion.

2- Stapedial reflex: lost in any affection above the nerve to stapedius.

3- Taste sensation:
   a- Qualitative: compare taste of different staff applied to the lateral edge of the anterior 2/3 of the tongue.
   b- Quantitative: electrogustometry.

4- Submandibular salivary flow test.

C- Electrodiagnostic studies:

1- Nerve excitability test

Determine the minimal electrical current in milliamperes required to produce a just visible muscle contraction, and compare both side.

   a- A difference more than 3-5 mAmp between both sides indicates degeneration.

   b- A difference less than 3 mAmp between both sides indicates neuropraxia. It is of no value if done before 3 days of injury because the degenerated fibers can continue to respond to stimuli during this period.

2- Electroneuronography (ENO), evoked electromyography.

   - This is the most important test
- Measurement of the amplitude of the summation action potentials of the muscles when a supramaximal stimulus is applied to the nerve and compare both sides.

- This is important to detect the percentage of degeneration, which is important to decide the way of management and prognosis.

- It is done after 2-3 days.

3- Electromyography

a- Fibrillation potentials indicate degeneration.

b- Polyphasic potentials indicate regeneration, and these are detected earlier than clinical recovery.

**General management of facial paralysis.**

1- **Reassurance** of the patient

2- **Care of the eyes:** To prevent exposure keratitis and corneal ulceration due to lack of Bell’s phenomenon (frequent blinking).

   a- Artificial tears during the day. B-Eye ointment by night.

   C-Use of eyeglasses outdoors.

   D-In prolonged cases, lateral tarsorrhaphy or gold weight implantation

**Care of the paralyzed facial muscles:** to prevent disuse atrophy and fibrosis.

   a- Physiotherapy and gentle massage in a circular manner.

   b- Infrared heat and galvanic stimulation.

   c- When voluntary movement starts, the patient should start active exercises.

3- **Treatment of facial paralysis according to the cause**

4- **Rehabilitation**

   a- Dynamic: to improve the function of the nerve during movement provided there is good status of muscles.

   - End to end anastmosis of the nerve
- Nerve graft: The cable graft is obtained from the great auricular nerve in the neck, or from the sural nerve in the leg behind the lateral meleolus.

- Cross facial anastomosis: This is an anastomosis between the facial nerves of both sides, through a supralabial tunnel, using the sural nerve.

- Hypoglosso – facial anastmosis.

- Static: to improve the appearance of the face at rest.

- Implantation of fascia lata slings.

- Regional muscle transplantation e.g. temporalis muscle in the cheek.

Results of facial paralysis.

1- Contractures due to fibrosis of denervated muscles.

2- Tics and spasms.

3- Cross innervation due to misdirection of the regenerating fibers, resulting in:

   a- Synkinesis:

   b- Crocodile tears: This is lacrimation while eating.

Important causes of facial paralysis.

- Bell’s palsy.

- Traumatic facial paralysis.

- Herpes zoster oticus.

- Facial paralysis complicating C.S.O.M.

**Bell’s palsy**

**Def.**: LMNL facial palsy.

**Aet.**: Idiopathic (several theories).

1- Vascular ischaemia: local vasospasm of vasa nervosa → oedema of nerve sheath → nerve compression → secondary ischaemia & more damage.

   1ry ischaemia may be due to exposure to cold draughts.
2- Viral theory: isolated viral neuritis, single manifestation of polyneuritis, or reactivation of herpes simplex.

3- Auto immune.

**Incid.:** The commonest cause of LMNL  80 – 90 %.

**Clinical picture:**

It affects mainly middle aged adults, and affects both sexes equally. It may be precipitated by exposure to cold air draughts, emotional stress, or pregnancy. The diagnosis of Bell’s palsy is made by exclusion of all other etiologies of facial paralysis.

It presents as:

1- Unilateral LMN facial paralysis of sudden onset, which may be partial or complete, and reaches a maximum in few days.

2- Retroauricular pain may occur several hours before the onset of the paralysis.

3- A reddish chorda tympani nerve may be visible through the posterosuperior part of the T.M.

4- Metallic taste and hyperacusis

**Investigations**

1- In prolonged, or recurrent cases CT & MRI to exclude facial neuroma.

2- To detect the level.

3- Electrodiagnostic tests.

**Treatment** most cases recover spontaneously.

1- General measures.

2- Medical ttt as early as possible.

a) **Vasodilators** in 1st few hours to relieve 1ry ischaemia e.g. nicotinic acid, histamine, beta histine.

b) **Steroids** (Medical nerve decompression):

   - To ↓ oedema & inflammation.
   - Should be given early in tapering dose.
   - Start with prednisolone 80mg / day.
   - If no response after 2w give 2nd course.

C) **Acyclovir** (Zovirax): 200 mg 5 times daily for 10 days

3- Surgical decompression
Indication: > 90% degeneration after 2w. Decompression by exposing bony canal & splitting the sheath from stylo mastoid foramen to level of compression.

4- Late cases: facial rehabilitation.

**Traumatic facial paralysis**

**Fracture temporal bone** (stretch & ischaemia, compression, contusion or cut).

<table>
<thead>
<tr>
<th>Incidence</th>
<th>Longitudinal</th>
<th>Transverse</th>
</tr>
</thead>
<tbody>
<tr>
<td>Facial palsy</td>
<td>Common 80%</td>
<td>Rare 20%</td>
</tr>
<tr>
<td></td>
<td>- rare 15-20 %</td>
<td>- Common 30-50%.</td>
</tr>
<tr>
<td></td>
<td>- delayed, partial +CHL.</td>
<td>- Immediate, complete +SNHL</td>
</tr>
</tbody>
</table>

**Treatment:**

1- If paralysis is immediate & complete (>90%) → surgical exploration.
   
   - Decompress bone specule. -If cut with no gap: end to end anastomosis.
   
   - If cut e gap: nerve graft.

2- If partial or delayed start conservative (Antibiotics & steroids) & follow up by ENOG if gets worse (90% degeneration in 2w) → surgical exploration.

**II- Surgical trauma (iatrogenic):** Due to:

Paralysis is either:

A) Immediate, usually complete (cut nerve): exploration

B) Delayed after several hours or days usually partial (compression, heat): conservative (steroids, antibiotics, remove tight pack), follow up →

**Diseases of the Inner Ear**

<table>
<thead>
<tr>
<th>Congenital</th>
<th>A plasia or Hypoplasia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Traumatic</td>
<td>Transverse temporal bone fracture</td>
</tr>
</tbody>
</table>
surgical trauma perilymph fistula
Acoustic trauma.
Concussion

<table>
<thead>
<tr>
<th>Inflammatory</th>
<th>Labyrinthitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vascular</td>
<td>Ischaemia, Hge.</td>
</tr>
</tbody>
</table>
| Miscellaneous      | Presbyacusis (degenerative).
                     | Menier’s               |
                     | Pure cochlear otosclerosis |

Congenital, inflammatory, traumatic & vascular are discussed with causes of P.D.

**Menier’s disease**

(Endolymphatic hydrops)

**Def.**: Disorder of vestibular labyrinth characterized by triad of paroxysmal vertigo, deafness & tinnitus due to increased volume & pressure of endolymph.

**Aet.**: Several theories.


2. Salt & water retention.

3. Histamine sensitivity.

4. Autoimmune disease.

5. Endocrinal theory.

**Incid:**

- Common cause of peripheral vertigo.
- More common in males.
- More before 50.
- Unilateral or bilateral (in 50%).

**Path:**

Increased volume and pressure of endolymph → injury to the cells in inner ear.

**Symptoms:**
Attacks of vertigo, deafness & tinnitus with remission in between.

1- Vertigo: Sudden, severe, lasts from minutes to hours, may be with nystagmus (rapid phase to opposite side).

2- Deafness SNHL.
   Early  Fluctuant, affect low tones, reversible.
   Later  Progressive, affect all tones, permanent.

3- Tinnitus increased during or before the attack.

4- In severe cases: vagal stimulation: nausia, vomiting, sweating, colics & bradycardia.
   - In 50% may aura: ear fullness, otalgia & increase tinnitus.

**Signs:** Normal T.M - Tunning fork: P.D

**Investigations:**

1- PTA: SNHL (see above).

2- Speech audiogram: poor discrimination matching PTA.

3- Electro cochleography: diagnostic.

4- Dehydration (Glycerol) test: during the attack PTA → 1.5 mg/kg glycerin + equal saline → PTA after 3h.: if improved by 10-15 dB (+ve).

5- Caloric test: hypoactive labyrinth (canal paresis).

6- CT to exclude retrocochlear pathology.

**Treatment:**

**I - Medical: during the attacks:**

1- Sedatives diazepam (Valium).

2- Relaxation & reassurance

3- Avoid dangerous activities, alcohol, smoking, and coffee

4- Bed rest with head supported.

**Between attacks:**

1- Salt restriction.

2- Vestibular suppressants: Sinnarezine.
3- Antiemetics: chlorpromazine (largactil).
4- Vaso dilators: nicotinic acid, betahistine.
5- Diuretics: lasix.

Treatment for symptoms
1- Vestibular rehabilitation.
2- Hearing aid.
3- Masking the tinnitus: masker.

II - Surgical
- Failed prolonged medical ttt. - Progressive hearing loss.
  A) Good (serviceable) hearing.
    - Intratympanic injection of selective vestibulotoxic drugs.
    - Endolymphatic sac decompression (Saccus decompression).
    - Selective section of vestibular nerve.
  B) Bad (non serviceable) hearing.
    - Surgical labyrinthectomy (not done).
    - Chemical labyrinthectomy (gentamycin).

Diseases of the cochlea – vestibular nerve

<table>
<thead>
<tr>
<th>Inflammatory</th>
<th>Vestibular neuritis (see vertigo).</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neoplasmatic</td>
<td>Acoustic neuroma.</td>
</tr>
</tbody>
</table>

Acoustic Neuroma

**Def.**: benign tumor arises from schwann cells of vestibular n. (vestibular schwannoma).

**Incid.**: 40-50y age of presentation. 8% of brain tumors, 80% of CPA

**Path.**: Arise from glial neurilemmal junction at IAM (or CPA).

Grossly: slowly growing, encapsulated, smooth, firm.

MP: Fasiculated type (Antoni A), Reticular (Antoni B).
**Clinical picture**

A) **otological**
1- Unilateral slowly progressive SNHL.
2- Unilateral tinnitus.
3- Vertigo is not marked as condition is slowly progressive, allows for central compensation.

B) **Neurological**
V: Lost corneal reflex.  VII: rare.

C) **Cerebellar:** (see brain abscess).

D) **Terminal** ↑ ICT coma, death.

**Investigations:**
1. PTA SNHL.
2. Speech audiogram: poorer speech discrimination than PTA (retrocochlear lesion).
3. ABR delay of wave V.
4. CT with contrast.  5-MRI with contrast: the best.

**DD:** other CPA lesions.
Meningioma, congenital cholesteatoma, arachnoid cyst, pontine glioma.

**Treatment:**
- Surgery: approach depends on size, hearing
- Small intracanalicular tumor with good hearing….middle cranial fossa
- Large CPA tumors with good hearing………retrosigmoid
- Bad hearing ….translabrynthine.
- Gamma knife (stereotactic radio surgery).

**NB.:** difference between cochlear & retro cochlear.

| Cochlear | Retro cochlear |
Symptoms of Ear Diseases

Deafness

Def.: It means diminution of hearing up to complete loss.


II - Hysterical

A - Conductive hearing loss.

Interference of sound transmission along the conductive apparatus (External & middle ear).

Causes:

I - EAC

- Congenital: meatal atresia or stenosis.
- Trauma: F.B or traumatic acquired atresia.
- Inflammatory: large furuncle, Diffuse OE, otomycosis, polyp.
- Neoplastic: Large exostosis – malignant tumors.
- Miscellaneous: wax (commonest cause).

II - T. M

- Traumatic: rupture of T. M. Inflammatory: bullous myringitis

III - Middle ear.

- Congenital: middle ear atresia or hypoplasia, ossicular fixation or deformity.
  - Longitudinal temporal bone fracture
- Inflammatory: - Acute suppurative otitis media.
- CSOM.
- Chronic non suppurative otitis media.

- **Neoplastic:** Glomus tumors, Carcinoma.
- **Miscellaneous:** Otosclerosis, Tympanosclerosis.

IV - Eustachian tube

*Congenital:* cleft palate.

*Traumatic:* post adenoidectomy scarring, barotrauma.

*Inflammatory:* tubal catarrh.

*Neoplastic:* nasopharyngeal tumors.

*Miscellaneous:* hypertrophy of the adenoid.

**B - Sensorineural hearing loss**

Defect in conversion of sound energy to electrical impulses (cochlea) or transfer of impulses along cochlear nerve & central connections to auditory cortex.

**I - Cochlear Causes (Sensory).**

1 - **Congenital:** Hearing loss dating since birth or shortly after

1 - **Hereditary** due to genetic aberrations:

- Deafness alone:
  
  Mickel’s: total lack of inner ear development.
  
  Mondini’s: partial aplasia of labyrinth cochlea makes 1 ½ turns.

- Deafness with other abnormalities.
  
  Usher’s Syndrome                    SNHL + retinitis pigmentosa.
  
  Pendred’s Syndrome                  SNHL + goitre.
  
  Alport’s Syndrome                   SNHL + nephritis.

2 - **Prenatal:**

  a- Maternal infections as rubella in the 1st trimester.
  
  b- Drug intake in the 1st trimester e.g. quinine, aminoglycosides and salicylates.

3 - **Natal:** (during labour).

  a- Hypoxia or anoxia of the fetus.
b- Birth trauma as in forceps delivery.

4 - Postnatal:
   a- Neonatal infections.
   b- Erythroblastosis foetalis (Rh incompatibility).

(2) Traumatic:
   1- Transverse fracture of temporal bone involving the labyrinth
   2- Labyrinthine membrane rupture (perilymph fistula)

   Aet:  1- Operative trauma: mastoidectomy & stapedectomy
         2- Head injury (with or without fracture)
         3- Blast injury
         4- Diving & straining

   Predispositions:  1- Congenital round window weakness
                    2- Absent stapedial tendon
                    3- Large cochlear aqueduct

   Sites: Oval window or round window

   Clinical picture: history of trauma

   Small fistula: fluctuant SNHL & tinnitus, vertigo & nystagmus
   Large fistula: sudden profound SNHL, intense vertigo

   Signs: +ve fistula sign  +ve positional testing
          Gait disturbance  SNHL

   Invest: SNHL, canal paresis on caloric test, exploration

   DD: Meniere

   Management: conservative in small, recent cases: bed rest, semi sitting, avoids straining.

   Exploration in: large fistula or failed conservative ttt

   Results: relief of vertigo, cessation of hearing deterioration, variable for tinnitus

3- Acoustic (noise) trauma:
   a- Acute: exposure to single severe noise as in explosions and fire shots causes permanent SNHL of the high tones in the exposed ear.
b- Chronic: prolonged exposure to noise as in industries and air crafts.

4- During ear surgery as in stapedectomy.

5- Concussion without fracture.

(3) **Inflammatory (Labyrinthitis):**

1- *Infective:*

a- Viral: measles, mumps, influenza, the deafness occurs after the febrile stage. It may be unilateral or bilateral, asymmetric and affects more the high tones.

b- Syphilitic labyrinthitis: deafness is progressive, asymmetric and may be associated with vestibular symptoms (i.e. Vertigo).

c- Bacterial:
   - Labyrinthitis secondary to suppurative otitis media.
   - Meningitis: deafness is bilateral and profound.

2- *Toxic*
   a- Ototoxic drugs: quinine, aminoglycosides, salicylates, lasix.
   b- Metabolic: uraemia, diabetes, thyrotoxicosis.

(4) **Vascular:**

Internal auditory artery occlusion due to spasm, thrombosis or embolism.

It causes sudden hearing loss (treated by large dose of steroids).

(5) **Miscellaneous:**

a- Presbycusis (senile deafness).

b- Meniere’s disease.

c- Pure cochlear otosclerosis.

d- Perilymph fistula.

**II - Retrocochlear causes:**
Due to lesion either in the vestibulocochlear nerve, or in the auditory pathway.

1- **Vestibulocochlear nerve affection:**
   
   A-Cerebellopontine angle lesions as in acoustic neuroma, and congenital cholesteatoma.  
   B-Meningitis.  
   c-Vascular loop.

2 - **Central:** due to lesion anywhere in the auditory pathway. It is rare.
   
   a- Multiple sclerosis.  
   b- Meningitis, encephalitis.  
   c- Cerebrovascular accidents e.g. thrombosis, haemorrhage or embolism.  
   d- Brain tumours.

3- **Mixed hearing loss**
   
   1- Congenital meatal atresia with inner ear anomaly.  
   2- Fracture base of skull.  
   3- Complicated CSOM with labryinthitis.  
   4- Combined otosclerosis i.e. (footplate fixation, cochlear otosclerosis).

### Deafness in children

Good hearing is important for development of language & speech, considerable hearing loss in early childhood will interfere with proper speech & child may not be able to talk (deaf – mute).

**Causes:**

(A) Conductive: mainly congenital, traumatic & inflammatory lesions.

(B) Perceptive: mainly congenital, traumatic & inflammatory lesions.

Most common causes are **mumps, measles, and meningitis**.

### Psychogenic deafness

- Sudden deafness or unexplained fluctuation, related to emotional stress.
- Marked disparity between PTA & speech audiometry.
- Improve by psychotherapy.

**Tests of hearing**

** (A) **Clinical speech testing.**

The patient is asked to repeat whispered words at different intensities (other ear is masked by Barany’s box).

** (B) **Tuning fork tests**  
See before.
(C) **Audiological assessment**

Sound has two physical characters:

a- **Frequency**: oscillation per second, in Hertz (HZ)

   Normal human ear can perceive 16-20000 HZ.

b- **Intensity**: amplitude of sound energy measured in dB

   Normal person hears as low as 10 dB, 140dB produces pain.

**Audiometry**

This is the measurement of hearing by the use of a special apparatus (audiometer) for studying the degree of hearing at different intensities and different frequencies. The resultant data are recorded as an audiogram.

**Value**

1- To detect the hearing threshold of the patient.

2- Detect the type of hearing loss (conductive, sensorineural, or mixed).

3- Detect the degree of hearing loss (mild, moderate, severe, or profound).

4- Selection of a hearing aid if needed.

**Types**

A- **Pure tone audiometry: (PTA)**

   1- It is the measurement of the patient’s hearing threshold by using pure tones of a single frequency.

   2- The test is done once with the ear phone to determine AC curve and once with a B.C. vibrator over the mastoid to determine B.C. curve. The hearing threshold is obtained at 8 frequencies (250, 500, 2000, 4000, 6000, 8000 HZ).

   3- The hearing threshold is the minimum intensity of sound that the person can hear. Normally it varies from 0-20 dB at all frequencies.

   4- The resultant two curves (A.C. curve and B.C. curve) are plotted on a graph (audiogram) and this will show the type of hearing loss.

**Results**

1- Conductive hearing loss: elevated A.C. threshold, while B.C. threshold is normal (i.e. Air/bone gap).
2- Sensorineural hearing loss: Both A.C. and B.C. threshold are elevated without air / bone gap.

3- Mixed hearing loss: Both A.C. and B.C. thresholds are elevated but with an air / bone gap (e.g. A.C. threshold is elevated more.

To determine the degree of hearing loss:

The average of A.C. threshold at 500, 1000, and 2000 HZ is obtained then the degree of hearing loss is obtained as follows:

1- Normal: 0 – 20 dB
2- Mild hearing loss: 20- 40 dB.
3- Moderate hearing loss: 40 – 60 dB
4- Severe hearing loss: 60 – 80 dB.
2- Profound hearing loss: more than 80 dB.

B- Speech audiometry

This is the hearing assessment using spoken words presented to the patient through earphones, and he is asked to repeat those words. It provides an idea about the ability to communicate:

Speech tests include:

1. Speech reception threshold (SRT)
   It is the level (in dB) at which the patient can correctly repeat 50% of the presented speech material. It should match with the hearing threshold level obtained by PTA.

2. Speech discrimination
   It is the percentage of the correctly repeated speech material by the patient to that presented to him. Scores of 90- 100% are normal. In SNHL it is poor than expected from the PTA.

Values of speech audiometry:

Confirms results of PTA. Selection of a hearing aid.

Detects malingerers. Differentiates between cochlear and retrocochlear.
C- **Impedence audiometry**

(1) **Tympanometry**

This is the measurement of middle ear pressure, through measuring the mobility (compliance) of the T.M.:

1. **Type A tympanogram: (normal)**
   
The pressure is around 0 (-100 to + 150) and the compliance is between 0.5 – 1.75 mm H₂O.

2. **Type As tympanogram:**
   
The pressure is normal but the compliance is reduced below 0.5 mm H₂O. It occurs in cases of ossicular fixation as in otosclerosis, and tympanosclerosis.

3. **Type AD tympanogram: (Hypermobile or flail)**
   
The pressure is normal, but the compliance is increased above 1.75 mm H₂O & it may exceed the limits of the machine. This occurs in ossicular disruption or dislocation.

4. **Type B Tympanogram: (Flat curve)**
   
This occurs in secretory otitis media.

5. **Type C Tympanogram:**
   
Normal compliance with negative pressure. This occurs in ET dysfunction.

*N.B*: Oscillating tympanogram occurs with glomus tumours.

(2) **Acoustic reflex**

Usually stapedius muscle contracts 70 – 90 dB above hearing threshold level.

D- **Evoked response audiometry**

This is recording of the action potentials anywhere in the auditory pathway from the cochlea up to the auditory cortex. They include:

1. **Electocochleography.**

2. **Auditory brain stem response audiometry.**
   
It records the electrical activity in the auditory pathway (from wave I to V.)
Value:
1. Objective detection of hearing threshold level.
2. It differentiates between cochlear and retrocochlear, a delay in latency of 0.4 m.sec. between the wave number V of both sides is suggestive of a retrochlear pathology (e.g. Acoustic neuroma).

3. Cortical evoked response.

E- Otoacoustic emission (OAES)

These are low intensity waves produced in the cochlear and recorded in the EAC. They are classified into:

1. Spontaneous OAES: recorded in the E.A.C. without provoking stimulus
2. Evoked OAES: recorded in response to a provoking stimulus (tones or clicks).

They are very sensitive to any cochlear abnormality and can detect and cochlear affection very early. It is used in hearing threshold detection especially in infants and children.

Assessment of hearing in infants and children

Special hearing tests are needed as they are uncooperative.

1. Under 2 Years: distraction method.
2. 2-5 years: Play audiometry.
3. Above 5 years: The same as adults.
4. Objective tests.

Hearing aid

Def: Sound amplification system, that increase the level of surrounding sounds to make them audible to hearing aid users

Goals: 1-Amplify normal speech 2-Hear warning signals

3-Help in education & language development

Components: 1-Microphone: pick up sounds & transfer to electric energy
2-Amplifier: amplify the electric energy
3-Receiver: converts electric energy to sounds
4-Power supply 5-Controls: gain & tone control

Types: Air conduction: -body worn (pocket type) -behind the ear
-Spectacle hearing aid -in the ear hearing aid

Bone conduction hearing aid
**Cochlear Implant**

Cochlear implant is a device that by pass the damaged hair cells & directly stimulate the auditory nerve fibers electrically after transforming sound energy to electrical impulses.

- It has two components internal – external.

  **A: External component**

  Microphone, speech process or & transmitting coil.

  **B: Internal component**

  Internal coil & multi channel electrodes.

- **Patient selection**
  - Done in bilateral profound SNHL.
  - Post lingual gives better results than pre.
  - A child hears more rapid than adult.

- **Results**

  In 60% allows non visual discrimination of speech in 40 % enhances lip reading.

**Otalgia (Earache)**

<table>
<thead>
<tr>
<th>1-local</th>
<th>(a) auricle</th>
<th>(b) EAC</th>
<th>(c) TM &amp; ME</th>
</tr>
</thead>
<tbody>
<tr>
<td>Traumatic</td>
<td>Haematoma. Laceration.</td>
<td>F. B</td>
<td>Traumatic Rupture Barotrauma</td>
</tr>
<tr>
<td>Inflammatory</td>
<td>Perichondritis Gouty tophi</td>
<td>Furunculosis Diffuse OE Malignant OE Otomycosis Herpes zoster Bullous O.E</td>
<td>Bullous myringitis ASOM Complicated CSOM (mastoiditis, LST) Petrositis</td>
</tr>
<tr>
<td>Neoplastic (malignant)</td>
<td>Rodent ulcer Carcinoma</td>
<td>Rodent ulcer carcinoma</td>
<td>Carcinoma</td>
</tr>
</tbody>
</table>
(2) Referred Otalgia

To determine the cause of earache when the ear appears normal, the areas supplied by 5th, 7th, 9th, and 10th cranial nerves, and the 2nd and 3rd cervical nerves should be examined.

A. Oral cavity: (along the 5th nerve).
   1. Dental carries, dental infections, un-erupted or impacted wisdom tooth.
   2. Glossitis, stomatitis (particularly herpetic).
   3. Malignant tumours of the tongue, and oral cavity.
   4. Calculi of the period (wharton’s) duct.
   5. Temporomandibular joint arthritis, or dislocation.

B. Nose: (Along the 5th nerve).
   1. Sinusitis.
   2. Barotrauma of sinuses.
   3. Tumours of the nose and paranasal sinuses.

C. Pharynx: (Along the 9th nerve).
   1- Tonsillitis, Quinsy, pharyngitis and retropharyngeal abscess.
   2- Malignant tumours especially of the tonsils, hypopharynx and nasopharynx.
   3- Postadenoidectomy and post tonsillectomy.

D. Larynx: (Along the 10th nerve).
   - Non specific laryngitis.
   - Epiglottitis.
   - T.B. Laryngitis.
   - Perichondritis.
   - Malignant laryngeal tumors.

E. Oesophagus: (Along the 10th nerve)
   - F.B. - Oesophagitis.
   - Malignant tumors.

F. Cervical: (along the 2nd and 3rd cervical nerves).
   1. Spondylosis
   2- Osteoarthritis of cervical spine.

G. Miscellaneous:
   - Great vessel aneurysm.
   - Acute thyroiditis.
   - Migraine.
   - Angina pectoris.
   - Long styloid process.
(3) Neuralgias

1. Trigeminal neuralgia (5\textsuperscript{th} nerve).
2. Geniculate ganglion neuralgia (7\textsuperscript{th} nerve).
3. Glossopharyngeal neuralgia (9\textsuperscript{th} nerve).
4. Occipital neuralgia (C\textsubscript{2} and C\textsubscript{3} nerves).
5. Superior laryngeal neuralgia (10\textsuperscript{th} nerve).

(4) Psychogenic: this is diagnosed by exclusion of all other etiologies.

Otorrhoea

Types

A. Watery: Cerebrospinal otorrhoea, due to:
   1- Trauma either base of the skull or after stapedectomy.
   2- Tumours.

B. Serous:
   1- Bullous myringitis.
   2- Allergic dermatitis of the external canal.

C. Mucopurulent:
   1- Acute suppurative otitis media.
   2- Tubo-tympanic type of C.S.O.M.

D. Purulent:
   1- Cholesteatoma.
   2- Furunculosis and otitis externa.

E. Bloody:
   1- Trauma: - Foreign body or lacerations of the E.A.C.
      - Fracture base of the skull.
      - Traumatic rupture of the T.M.
   2- Bullous myringitis.
   3- Tumors: - Glomus. - Malignant tumors.
Vertigo

Def:
- Subjective sensation of motion usually in the form of rotation of patient or his surroundings.
- It is hallucination of movement. May be alone or with deafness & tinnitus.

Causes:

Physiological: Due to rotation or thermal stimulation to labyrinth (caloric test).

Pathological

1 - Peripheral

1- Labyrinthine

a. Traumatic
   - Head trauma with transverse skull base fracture involving the labyrinth.
   - Perilymph fistula.
   - Post operative: Stapedectomy.

b. Inflammatory: labyrinthitis (see before).

c. Vascular: spasm, thrombosis, embolism of I.A.A.

d. Degenerative: Benign paroxysmal positional vertigo (B.P.P.V).
   - Very common.
   - May occur after head trauma, infection or spontaneously.
   - Vertigo related to certain position, no deafness, or neurological deficit.
   - Self limiting within 6 month
   - Improves with vestibular rehabilitation (Epley maneuver).
   - In resistant cases singular n. section (the nerve supplying post. SCC).

e. Miscellaneous: Menier’s

2- Vestibular.

a. Inflammatory (vestibular neuronitis):
   It is characterized by sudden severe vertigo without deafness.
Lasting for a few days, with associated nystagmus.

A viral cause is suspected.

It is treated by antivertiginous drugs and steroids.

In rare instances, vestibular nerve section can be beneficial.

- Basal meningitis.

b. Neoplastic


II - Central

Due to affection of the vestibular nuclei or the vestibular connections in the brain stem or cerebellum. It is associated with other neurologic manifestations.

- Vertebrobasilar insufficiency (brainstem ischaemia).
- Cerebrovascular strokes: e.g. thrombosis, embolism.
- Basilar artery migraine
- Lateral medullary syndrome (occlusion of the posterior inferior cerebellar artery).
- Cerebellar tumours, and abscess. -Multiple sclerosis.
- Epilepsy -Meningitis, encephalitis.

Clinical approach to a case of vertigo.

(A) History

1. Make sure that the patient is actually describing vertigo (sense of rotation).
2. Timing of vertigo; occurs in attacks or persistent.
3. It should be determined whether vertigo provoked by certain positions, as in benign positional vertigo.
4. Associated symptoms of nausea, vomiting and diarrhea indicating the severity of vertigo.
5. Loss of consciousness should raise the possibility of epilepsy.
6. Symptoms of ear disease: deafness, tinnitus, earache, and discharge.
7. Neurological symptoms: Headache, weakness, paraesthesia, diplopia, ataxia and in co-ordination, may suggest a central cause.
(B) Examination

A. General exam: pulse B.P. for atherosclerosis, pallor for anemia.

B. Full neurological examination:

C. Cranial nerves examination.

D. Vestibular examination:

1- **Examination of eye movements** (observation for nystagmus).

   - Nystagmus is an involuntary deviation of the eyes away from the direction of the gaze followed by return of the eyes to the central resting position.

   - **Classification**:

     a. Physiologic (optokinetic): the rapid phase is towards the center.

     b. Pathologic:

        (1) Occular: rotatory or pendular.

        (2) Vestibular: has a rapid phase and a slow phase.

        (3) Central: vertical.

           - **Detection**

              a. Direct observation by looking into the eyes.

              b. Observation using Frenzel glasses to abolish the optic fixation.

              c. Electronystagmography and videonystagmography.

2- **Examination of balance and co-ordination**:

   Evaluation of the vestibular system, vision, proprioception and cerebellar function

   - Romberg test: the patient is asked to stand with feet together and arms in front of the chest, first with eyes open, then closed. If obvious uncontrollable falling occurs this indicates positive test (i.e. vestibular or cerebellar cause).

   - Gait test: for ataxic gait.

   - Tests for cerebellar affection.
(C) **Investigations**

A. **Vestibular assessment**: (observation for evoked systagmus).

1. **Positional testing:**
   
   The patient is seated with the head in 7 different positions, and observe for nystagmus in each position to detect canal paresis.

2. **Caloric test:**
   
   The patient lies supine with the head flexed 30°, the ear is washed with cold water (30°C) and then with warm water (44°C), for 30–40 seconds each time.
   
   - Normally: Vertigo occurs, with nystagmus lasting about 90 – 120 seconds.
   
   - Nystagmus is detected by direct looking in the eyes or using Frenzel glanes or by electronystagnagraphy.

   - According to a certain formula we can detect normal functioning, hypofunctioning (canal paresis) or dead labyrinth (no response).

3. **Rotation test:** Stimulation of the semicircular canals by rotating the patient on a chair, and observe for nystagmus as in caloric test.

4. **Posturography.**

B. **Audiological assessment:**

C. **Other investigations:**

   - CT scan: brain and CPA.
   
   - Serology for syphilis

   - FBS,CBC, T₃, T₄, TSH, and serum cholesterol

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**Tinnitus**

It is a subjective sensation of noise in the ear and / or the head, without an acoustic stimulation from outside.
**Causes:**

Subjective: all causes of deafness

   Hemodynamic disturbance (anemia, hyper or hypotension)

Objective: 1. Contraction of muscles in middle ear or around ET

   2. Glomus tumor

   3. Internal carotid artery or occipital artery stenosis or compression

**Characters:** Could be whistling, ringing, noises, or pulsations.

   All increases at bed time

**Invest:** General: personal & occupational history

   Blood pressure & blood picture

   Auscultation of the neck

   Special: full ENT examination

   Audiometry & tympanometry

   CT & MRI of temporal bone

   Doppler & angiography of carotids & vertebrobasilar arteries

   Nystagmus measurement

**Ttt:** mainly of the cause, if no obvious cause:

   Acute: IV vasodilators & plasma expanders. IV steroids

   Chronic & subacute: tinnitus masker, psychological support, sedatives, antidepressant, Ca antagonists & betahistine

**Ear Operations**

**Myringotomy**

Incision of the tympanic membrane.

**Indications**

1. Acute suppurative otitis media with:
2. Secretory otitis media: myringotomy is done followed by insertion of ventilation tubes in:

3. Otitis barotrauma.

**Anesthesia**: general anesthesia, local anesthesia can be used in adults.

**N.B.**: In cases of grommet tube insertion for secretory otitis media the incision is done in the anterosuperior quadrant as it has the least migratory epithelium, so there is no early extrusion of the tube.

**Postoperative care**

1. Repeated local cleaning and suction of discharge.
2. Antibiotics according to culture and sensitivity, analgesics,
3. Observe the patient until the drum heals.

**Advantages**

1. Immediate relief of pain and toxic manifestations in uncomplicated cases.
2. Allows rapid resolution of residual deafness.
3. The incision heals better (Primary intention) than that of spontaneous perforation with less scarring.

**Complications**

1. Ossicular disruption (dislocation of incudo-stapedial joint).
2. Injury of a high jugular bulb (dehiscent floor). It appears as bluish colored inferior portion of the TM
3. Residual perforation.
4. Injury of a dehiscent facial nerve.

**Cortical mastoidectomy (Schwartz operation)**

This is a drainage operation with exentration of all the mastoid air cells.

**Indication**

1. Acute matoiditis with:
   a. Persistent pain, tenderness, and fever more than 48 hours despite medical treatment.
   b. Development of complication e.g. facial paralysis, labyrinthitis and other cranial or intracranial complications.
2. Mastoid abscess, belzold’s abscess, zygomatic abscess.
3. As an approach to various ear operations:
- Facial exploration.  
- Saccus decompression in Menier’s diseases
- Labyrinthectomy.  
- Posterior tympanotomy approach.

**Anesthesia:** general.

**Technique**
- Post auricular incision
- Elevate mastoid periosteum
- Remove mastoid cortex
- Exentration of all mastoid air cells
- Opening & cleaning of mastoid antrum (McEwens triangle)
  
  A. superiorly: supramastoid crest  
  B. Anteriorly: posterosuperior meatal wall & spine of Henle
  C. Posterior: tangent to posterior meatal wall

In adults the antrum is at a depth of half an inch, but more superficial in children

Closure of the wound in two layers; periostium and skin.

N.B.: If an abscess is encountered a specimen of pus is obtained and sent for culture and sensitivity.

**Complications**

1. Injury of the: dura, lateral venous sinus, incus, facial nerve
2. Persistent discharge

**Radical mastoidectomy**

This is exentration of the mastoid air cells, and all contents of middle ear except the stapes (if present). Thus the middle ear and the mastoid are converted into a single cavity.

**Indications**

1. Cholesteatoma.
2. Tumours:
   - glomus tympanicum.  
   - Carcinoma.
3. Tuberculous otitis media.
**Procedure**

- **Anesthesia:** General.

- **Technique:** As cortical mastoidectomy with the following procedures.
  
a. Removal of the posterosuperior bony metal wall (bridge) and the outer attic mass.
  
b. Lowering the facial ridge (posterior meatal wall).
  
c. Removal of all cholesteatoma, polypi, granulations, mucous membrane of the middle ear and remnants of the T.M.

- At the end of the operation meatoplasty is done to widen the external auditory meatus, so as to accommodate the large mastoid cavity.

**Complications**

1. Injury of the: as cortical
2. Mastoid cavity problems: discharge, wax accumulation
3. Recurrence of cholesteatoma.

**Tympanoplasty**

It is an operation to

- Eradicate irreversible pathological changes in the middle ear and
- Reconstruct the middle ear conductive hearing mechanism.

It may be combined with mastoidectomy

**Indications**

1. Chronic suppurative otitis media.
2. Congenital middle ear anomalies causing conductive deafness.
3. Middle ear trauma causing conductive deafness.

**Anaesthesia:** Either general or local anesthesia.

**Technique:**

1. Incision: either postauricular or endaural incision. It may be done through the external auditory canal (permeatal), if mastoidectomy will not be performed.
2. Eradication of all diseased mucosa, or any granulation in the middle ear and mastoid.
3. Reconstruction of the hearing mechanism by:
A. **Myringoplasty**: to repair a tympanic membrane perforation; using a graft material which acts as a supporting framework for regeneration of the outer epithelial and inner mucosal layers of the tympanic membrane. The graft may be placed under the T.M. and the annulus (underlay) or between the epithelial and mucosal layers of the T.M. (on lay).

The graft material used may be: temporalis fascia, tragal perichondrium

B. **Ossiculoplasty**: which entails reconstruction of fixed, damaged or absent ossicular chain.

Reconstruction of the ossicular chain is performed either by:

a. Cartilage grafts.

b. Bone grafts.

c. Prosthesis; TORP (total ossicle replacement prosthesis) PORP (partial ossicle replacement prosthesis), or Teflon.

**Wullestine types tympanoplasty**

Type I: Myringoplasty only, for reconstruction of a T.M. perforation.

Type II: The T.M. graft is placed on the incus (i.e. absent malleus).

Type III: The T.M. graft is placed on the head of stapes (absent malleus and incus).

Type IV: The T.M. graft is placed on the footplate of the stapes (absent stapes supra structures).

Type V: The T.M. graft is placed over an artificial fenestra on the lateral canal, or stapedectomy due to fixed foot plate of the stapes.
Stapedectomy

- **Principle:** Removal of stapes suprastructure, making a hole in foot plate then linking ling process of incus to oval window by prosthesis.

- **Prosthesis** may be Teflon, wire, Tefplatinum, Tefwire, gold.

- **Anesthesia** G or L.

- **Indication:** otosolerosis with A-B gap more than 20 – 25 dB.

- **Technique.**
  1. End aural or permeatal incision.
  2. Elevate tympanomeatal flap.
  3. Remove arch of stapes after cutting stapedius m.
  4. Create a fenestrum in foot plate (stapedotomy).
  5. Connect incus to oval window by prosthesis.

- **Contraindications:**
  - Medical contra indications! Old age, above 50-60.
  - Young age (still active). Pregnancy
  - Occupations requiring strain, balance. Active otosclerosis.
  - Severe SNHL. Only hearing ear.
  - Associated menier’s disease, (high risk of P.D).

- **Complications:**
  - SNHL & dead ear 3%.
  - Persistent or recurrent C.D.
  - Vertigo.
  - Perilymph fistula.
  - Facial paralysis.
  - Injury of chorda tympani nerve: loss of taste
  - Infection, granuloma.

**NB:** Tinnitus may not improve after surgery.