

HEARING LOSS



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Objectives

- 1.To know the definition and severity of hearing loss.
- 2.To identify the clinical features of hearing loss.
- 3.To know the types and causes of hearing loss.
- 4.Out line the treatment of conductive hearing loss.
- 5.How to help a patient with sensori-neural hearing loss.

Hearing loss

Physiology of hearing:

The ear divided in to two parts.

1. Conductive apparatus. They magnify and transmit the sound wave in to inner ear fluid. Consist

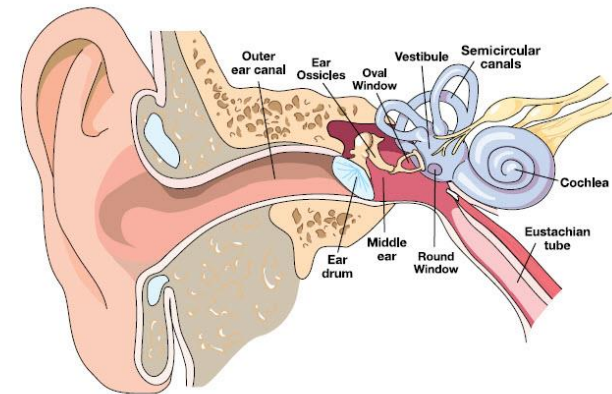
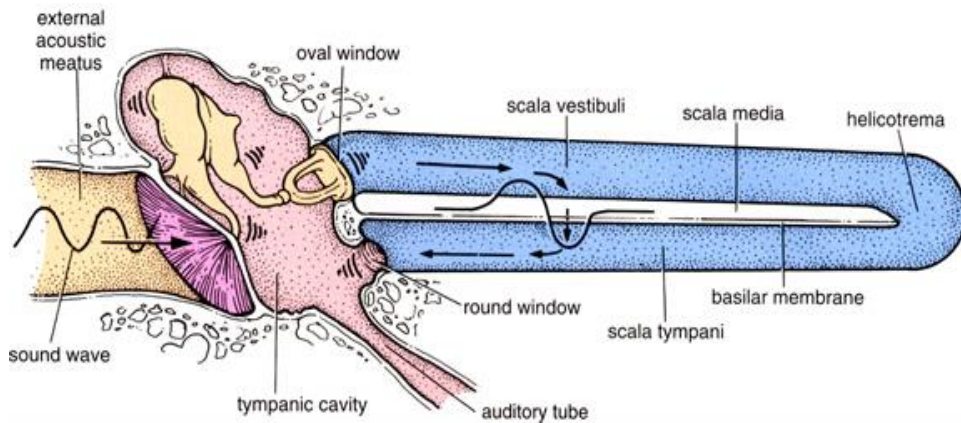
1. the Auricle ,and external auditory canal, they collect and direct the sound wave to the tympanic membrane .

2. The tympanic membrane, vibrate and transmit the wave to the ossicles.

3. The ossicles . Transmits sound wave to oval window, causes vibration of perilymph (fluid wave) and vibrate the basilar membrane .{The effective ratio of TM to footplate (oval window) is 14:1, and the ossicular lever ratio is 1.3:1, so $14 \times 1.3 = 18$ time increase sound pressure. others ($17 \times 1.3 = 22$)}.}

4. The Eustachian tube, Equalize the ext. and mid. ear pressure for maximum movement of the TM.

5. Labyrinthine fluid. the stapes move in rocking rather than a piston motion, so the inner ear fluid not compressed, causing the vibration transmitted to round window. This reciprocal movement of oval & round window is essential .

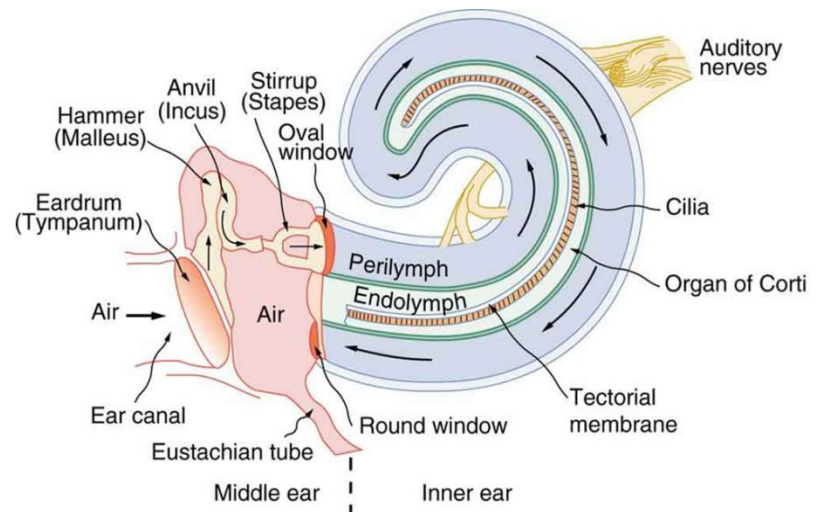
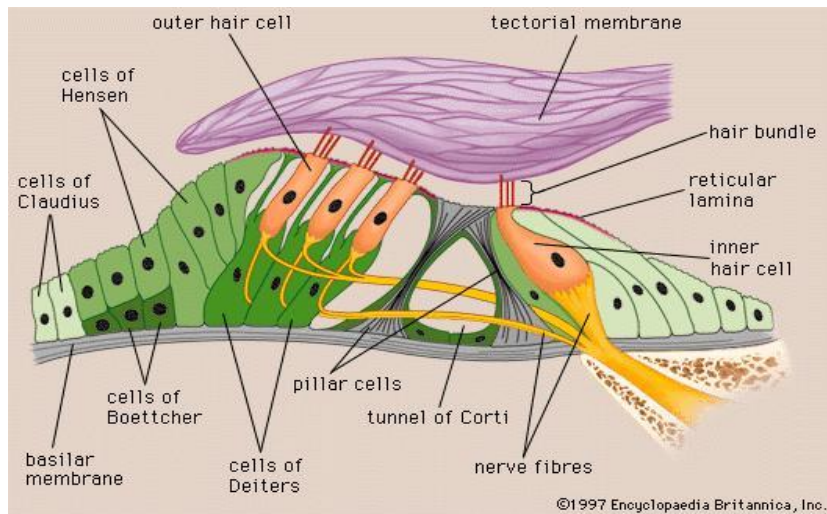


2.Perceptive (sensory) apparatus.

They convert the sound wave to electrical nerve impulse. Consist of

1.End organ of hearing (organ of corti), the vibration of the basillar membrane, and shearing movement between the hair cells and tectorial membrane initiate impulse in to fibers of auditory nerve .The greater degree of displacement ,the more hair cells and more fibers stimulated. So the basal portion of cochlear duct response for high frequency, and the apex for low frequency .

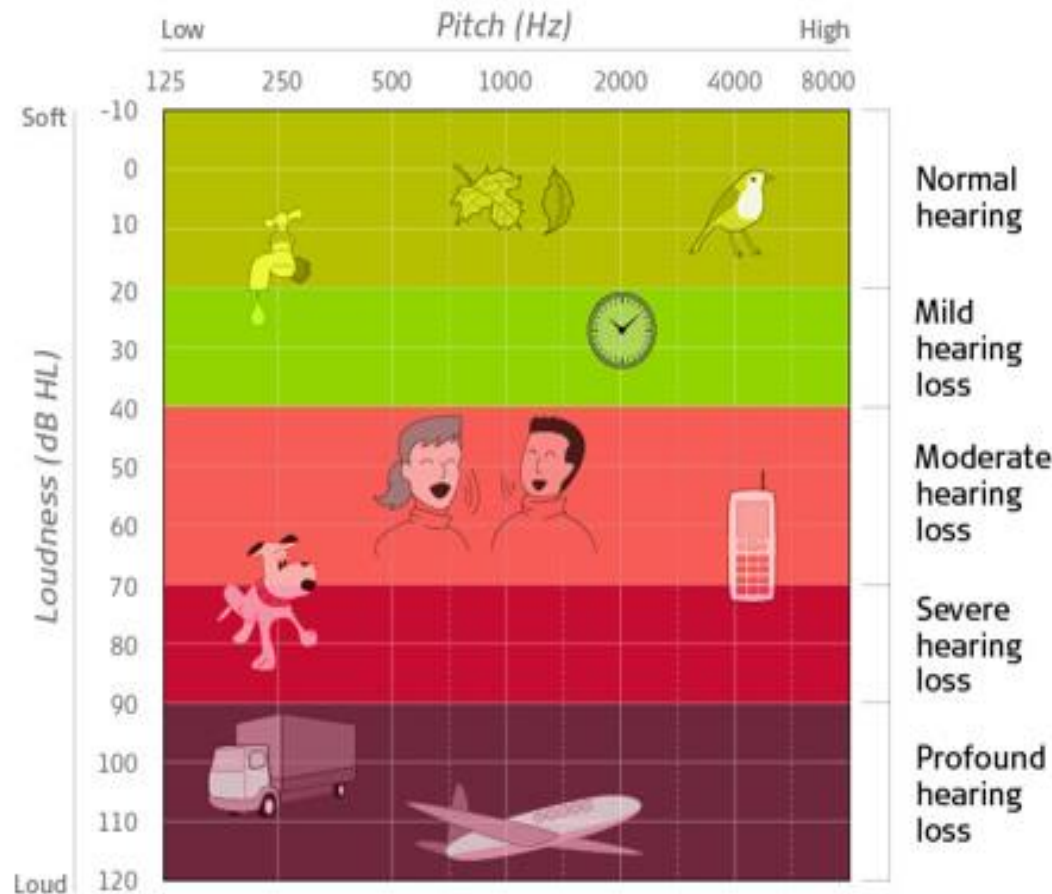
2.Cochlear division of vestibulocochlear N.,>Auditory nuclei in brain stem>midbrain>auditory cortex (superior temporal gyrus). where the impulses perceived as sound



Hearing loss: Is decrease person 's sensitivity to sound more than(20-25 dB HL) in 250-8000 Hz frequency rate.

Classification of hearing loss by severity.

Normal hearing.	20dB
Mild hearing loss	20-40dB HL
Moderate HL	40-70dB HL
Sever HL	70-90 dB HL
Profound HL	90-110db HL
Deafness	>110dB HL



Clinical features of hearing loss

*People with hearing loss suffer emotional, social, and communicative dysfunction.

*In children: *Delayed speech/language development.

* Difficulties in learning, playing and developing social skills.

*Persons with early hearing impairment can often compensate for it for a relatively long time, e.g., by

*Turning up the volume of the radio or television set or (in unilateral hearing impairment) by turning the healthy ear to the sound source.

*As hearing impairment worsens, vision is used as an additional aid to speech recognition, with an increasing reliance on lip-reading.

*Repeated questioning about things that have not been properly heard,

* Inappropriate answers to misheard questions.

*Excessively loud speaking voice in SNHL.

*Quiet speech in conductive HL

**Care must be taken for...

1* Unilateral sensori-neural hearing loss.,
with *vertigo* and tinnitus.)

2* Abnormalities of nerves (other than VIII)



Classifications (Types) and causes of hearing loss:

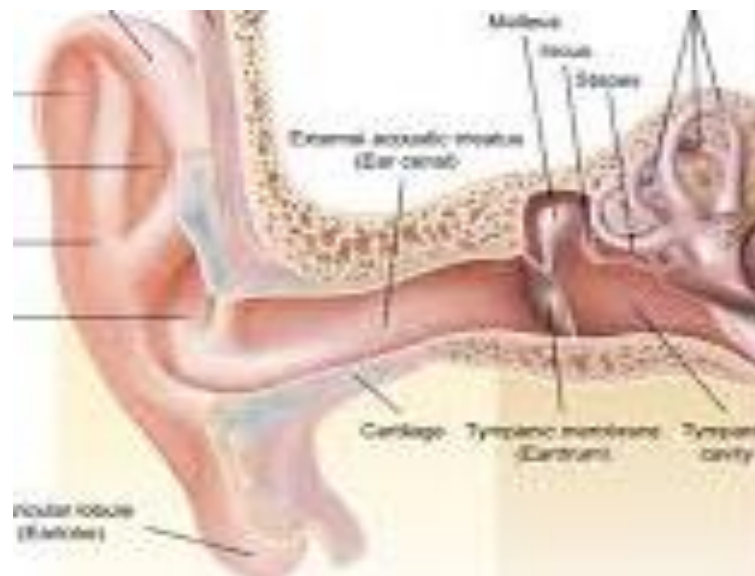
1. Conductive Hearing loss. (Due to external or middle ear pathology.)

1. External ear.

1. wax the commonest.
2. Congenital meatal atresia.
3. Acquired meatal stenosis & atresia.
4. Foreign body.
5. otitis externa.
6. Tumor *Osteoma, exostosis, papilloma.
**Malignant. squamous cell ca.

2. Middle ear.

1. Otitis media *(suppurative. acute & chronic),
**(non-suppurative otitis media with effusion, adhesive OM).
2. congenital middle ear defect (ex. fixation of footplate of stapes)
3. Otosclerosis.
4. Trauma. (perforated tympanic membrane, ossicular discontinuity, Haemotympanium).
5. Tumor *Glomus Tu. **squamous cell ca.



Treatment of conductive hearing loss

1* Wax removal :By probing, Suction, and ear syringing. •

2. Surgical treatment •

* Myringotomy +/- Grommet insertion.for Otitis media with effusion. •

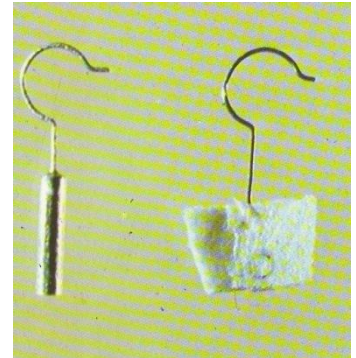
* Myringoplasty. Grafting of perforated TM.

* Ossiculoplasty. reconstruction of damaged ossicular chain.

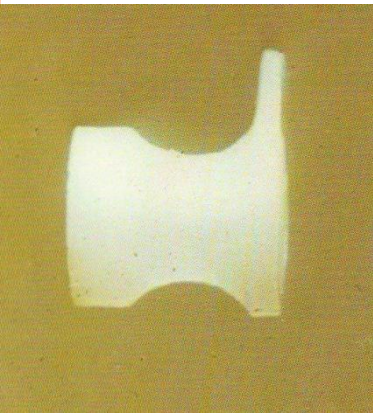
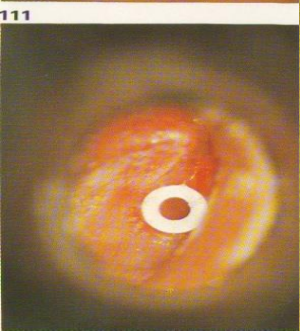
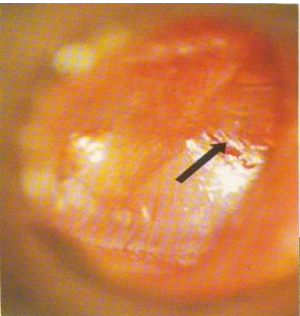
* Stapedectomy. For otosclerosis.

* Resection of tumor:like osteoma. Glomus Tu. etc.

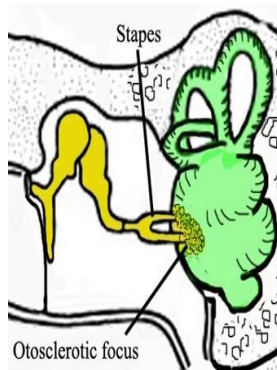
* Meatoplasty :For meatal atresia ,or stenosis.



3* Hearing Aids •



Stapedectomy



1-Otosclerosis



2-Removal of stapes supra-structures



3-Perform a whole in foot plate



4-Hang a prosthesis on incus to the whole in foot plate

II. Sensori-neural HL:

*Sensory (Damage of cochlea, organs of corti) .

*Neural (Damage of cochlear N. ,or neural pathway).

A. Congenital SNHL.

1.genetic. Like*.Waardenberg syndrome.* Pendred syndrome, and* Usher syndrome.

*B. Acquired SNHL.

1.Presbycusis. Degenerative changes of aging process is commonest causes Of SNHL .Bilateral symmetrical Progressive high frequency SNHL.

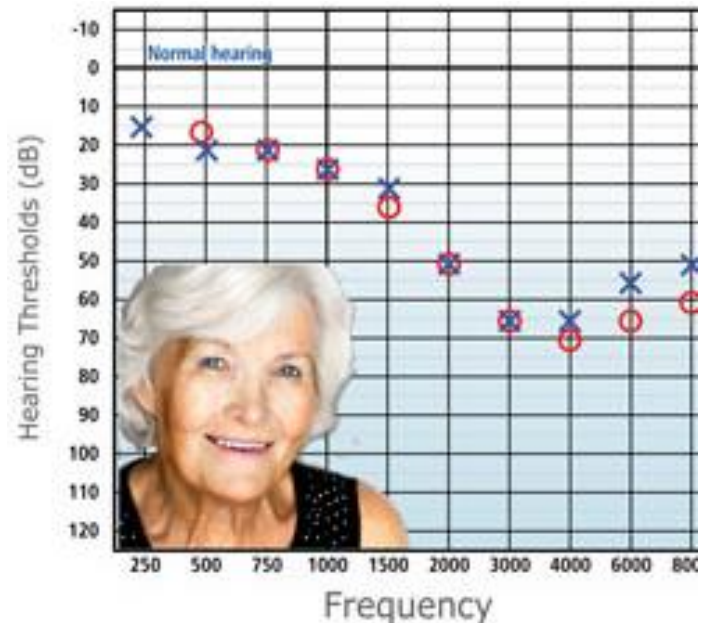
start 55-65 year

.Risk factor include

*Genetic susceptibility.

*Noise exposure,

*Metabolic and vascular diseases.



2.Noise-induced HL:

Damage of hair cells due to exposure to loud sound.

A . Acute acoustic trauma. Due to exposure to sudden intense sound >140 dB SPL, of short duration ,commonly Gunshot, and blast injury (explosion) when usually rupture of TM.

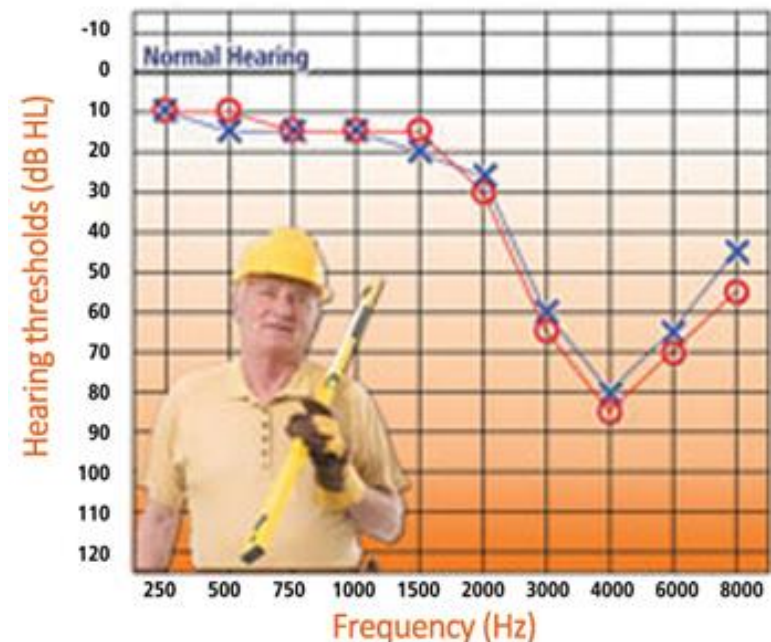
B. Chronic noise induced HL : When Exposure for >90 dB SPL >8hr per day ,for 5 days/in a week/ for 3months. commonly industrial noise. permanent hearing loss and tinnitus.

Audiogram show *drop hearing threshold between 3-6 KHz (dip at 4 KHz)* then deteriorate all frequency.

Treatment :

*Prevention Using ear muff for protection .

*Hearing Aids .



3.Ototoxicity:

Damage to the cochlea and/or Vestibular part of inner ear by drugs.

Rout of entry: 1.Parenteral(I.V,I.M)commonly.

2.Oral.(Lasix,Chloroquine.)

3.Local (ear drops ex. Garamycin, Neomycin)

Drugs.

1.Aminoglycosides antibiotic: Garamycin& streptomycin (mainly vestibulotoxic). Neomycin, Kanamycin, Tobramycin (mainly cochleotoxic). Permanent SNHL.

2.Loop diuretics: Frusemide, Bumitanide, Ethacrynic acid in high dose (reversible high freq. SNHL)

3. Cytotoxic drug. like Cisplatin, is cochleotoxic partially reversible on with drawls of drug,& Cyclophosphamide.

4. Salicylate. Aspirin in over dose causes tinnitus & flat SNHL, (reversible).

5.Antiprotozal agent.Quinine &chloroquine, (Cochleotoxic HL, permanent).

6.Anticonvulsant.Phenytoinðsuximide.Vestibulotoxic (acute vertigo& reversible.)

Treatment of Ototoxicity:

*Prevention.

1. Avoid or discontinue ototoxic drugs, when satisfactory alternative.
2. Monitor of drug by estimate serum level of drug & serum creatinin.
3. Monitor hearing by audiogram and vestibular function (caloric test).

*Therapeutic : Hearing Aids. (no medical or surgical treatment are Effective.)

4.Labyrinthitis: is an inflammation of the inner ear or labyrinth.
Clinically, vertigo, hearing loss, Tinnitus. Of varying degrees and may affect one or both ears.

I. viral *Mumps, *Measles, *Herpes Zoster, *HIV.

II.Bacterial *Otogenic (otitis media)

*Meningeal (commonly streptococcal pneumonia),

*Syphilis, *Tuberculosis.

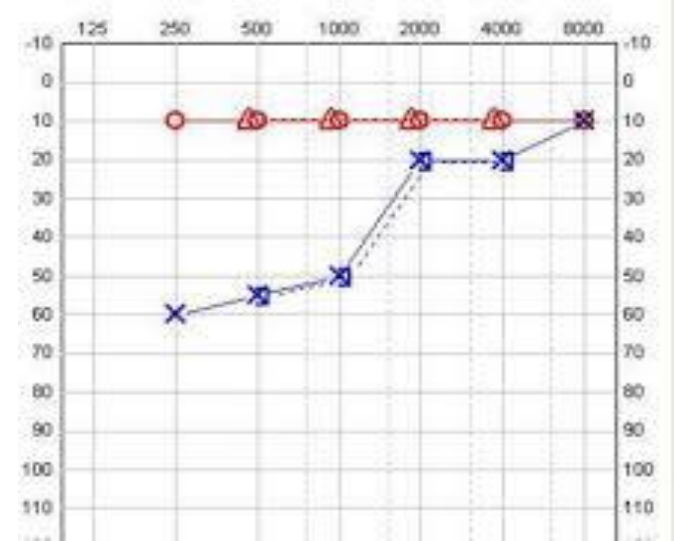
5.Meniere's disease: (Endolymphatic hydrops).

There are SNHL, vertigo & tinnitus).

*The hearing loss is Fluctuant (cochlear).

first low frequency HL. with repeated attack

become permanent, and involve high frequency.



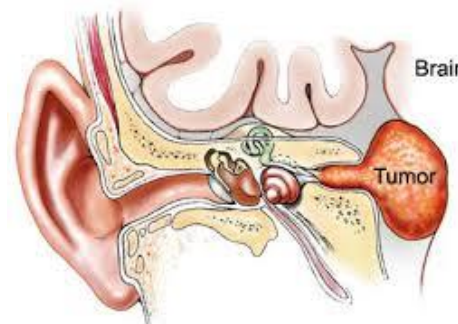
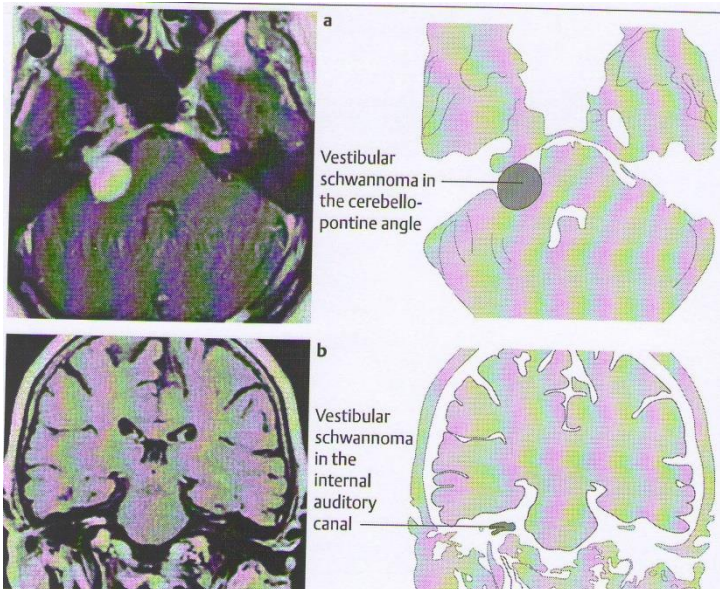
6.Acoustic neuroma: (vestibular shwanoma). Slow growing benign tumor ,arise from shwan cells of VIII n. (commonly from vestibular nerve) .in the internal auditory meatus then enlarged go to cerebello-pontine and enlarged

- *Usually unilateral progressive, or sudden SNHL (neural type), with tinnitus. Vertigo and disequilibrium less common.

Diagnosis:* MRI with gadolinium enhancement is diagnostic.

- * Auditory Brainstem response (ABR)

Treatment:Surgical excision .



a Post-gadolinium axial MRI shows a large, medial vestibular schwannoma in the cerebellopontine angle.
b Post-gadolinium coronal MRI demonstrates a vestibular schwannoma in the internal auditory canal.

7.Trauma:

- *Transverse fracture temporal bone,
- *Iatrogenic (Ear surgery) ,
- *Blast injury.

8.Psychological hearing loss.

Sudden sensorineural HL:

Is loss of 35 dB for three consecutive frequencies within three days or less. may be unilateral, or bilateral.

Causes;

1. Vascular (Hemorrhage, thrombosis).

2. Viral infection (Mumps, rubella, varicella zoster. CMV, Influenza virus) > labyrinthitis.

3. Rissener's membrane break. ionic fluid imbalance from mixing perilymph and endolymph results SNHL

Treatment. Vasodilator (to improve cochlear circulation) like

*inhalation of carbogen 5% Co₂.

*Low molec. Weight dextran 40%

*Beta- histidine(Beta serc tab.)

*Steroid important (prednisolon, hydrocortisone.)

Follow up by serial audiogram(P.T.A)

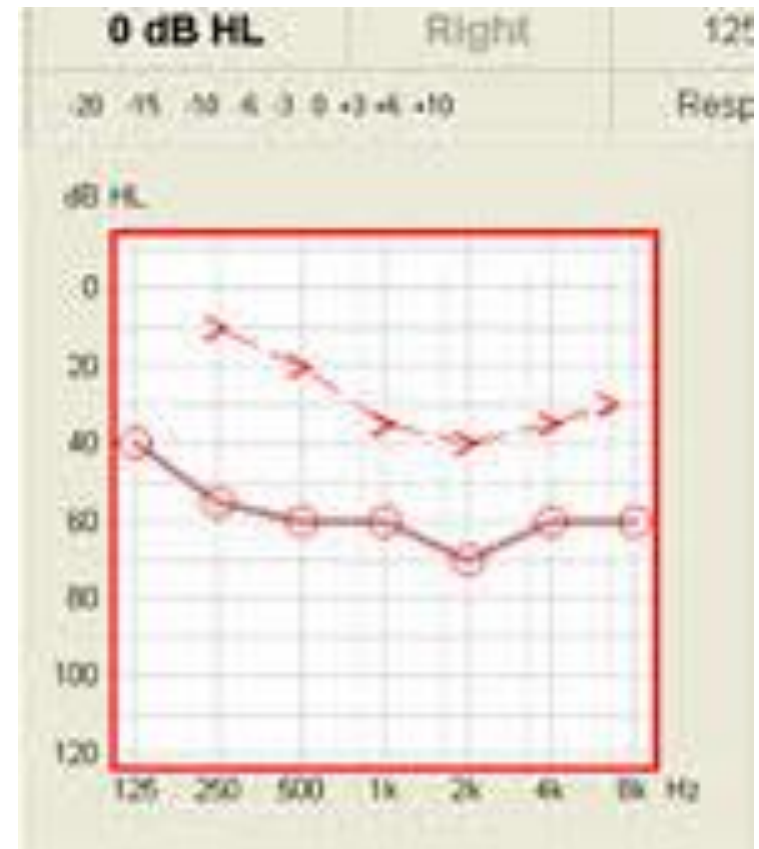
Prognosis. 40-70% improve, or get recovery

Bad prognosis: *Old age *Total deafness *High frequency HL.

*Vertigo *Delay treatment.

III. Mixed HL: (conductive & SNHL).

1. Advanced otosclerosis. (cochlear otosclerosis).
2. Chronic suppurative otitis media. Due to *Absorption of toxin , *use of systemic & local ototoxic drugs.
3. Glomus tumor.
4. Trauma. Ex. mixed fracture temporal bone.



Treatment of Sensori-neural hearing loss.

Aim of treatment is restoration as much as possible of hearing loss by

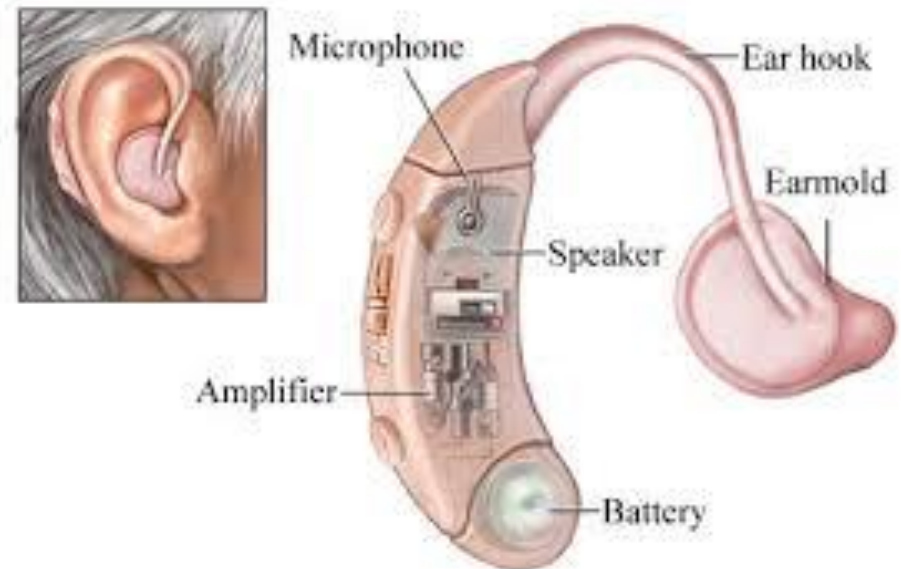
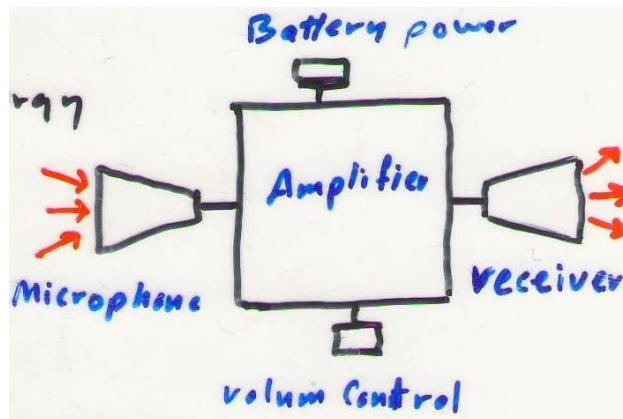
1. Hearing Aids. 2. Cochlear implant.

1. Hearing Aids: Consist of_ •

1. Microphone. Is electrical condenser to pickup sound. The diaphragm •
convert the acoustic energy (sound) to mechanical signal, then microphone
convert this mechanical energy to electrical energy.

2. Amplifier. A series of transistors. It boosts the electrical signals .The range of
amplification from 250-4000Hz.

3. Receiver (loudspeaker). It Covert the electrical signal back to acoustic
signal, Which then broadcast in to the ear.

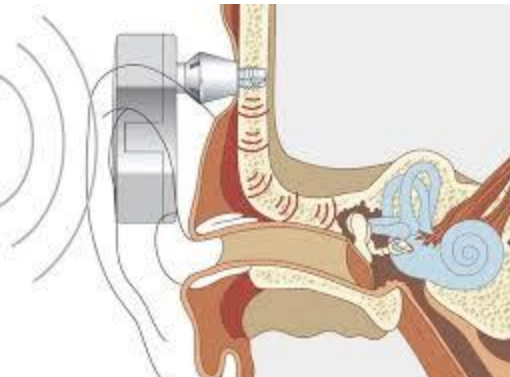


. The **receiver** are of three types.

1. Air conduction .the button attached to an ear mould (in the canal, or behind the ear.)

2. Bone conduction. when meatal atresia, or stenosis. Chronic suppurative otitis media (recurrent discharge) .Either headband to body worn H.A, or built in spectacle-type H.A.

3. Bone –anchored H.A. the aid directly anchored to the skull • percutaneous titanium



In-the-canal



Completely-in-the-canal

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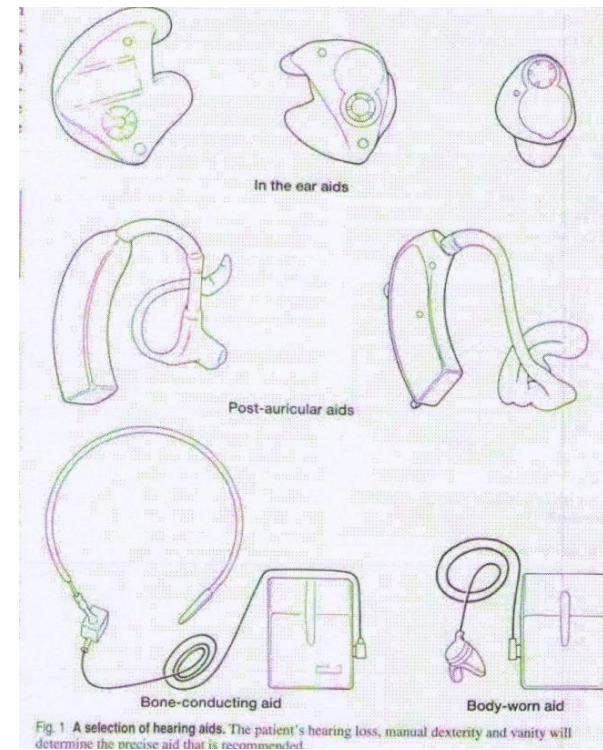
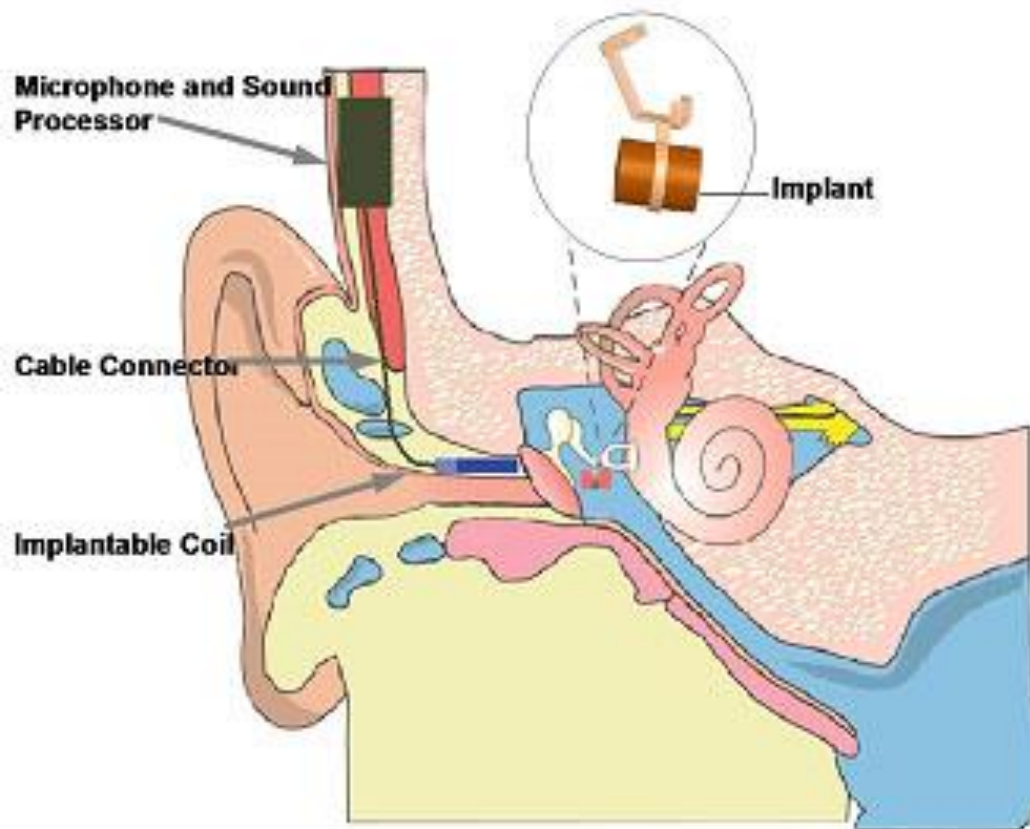
Models: * Body worn H.A, Of great power 120 dB amplification with minimum feed back. ** Behind the ear. *** In the ear (canal) **** Bone anchored.

Conventional technology.

1. Analogue H.A .

2. Digital H.A .

*** Middle ear implantable hearing devices**



Cochlear implantation:

An electronic device that generates electrical stimulation of auditory nerve directly.

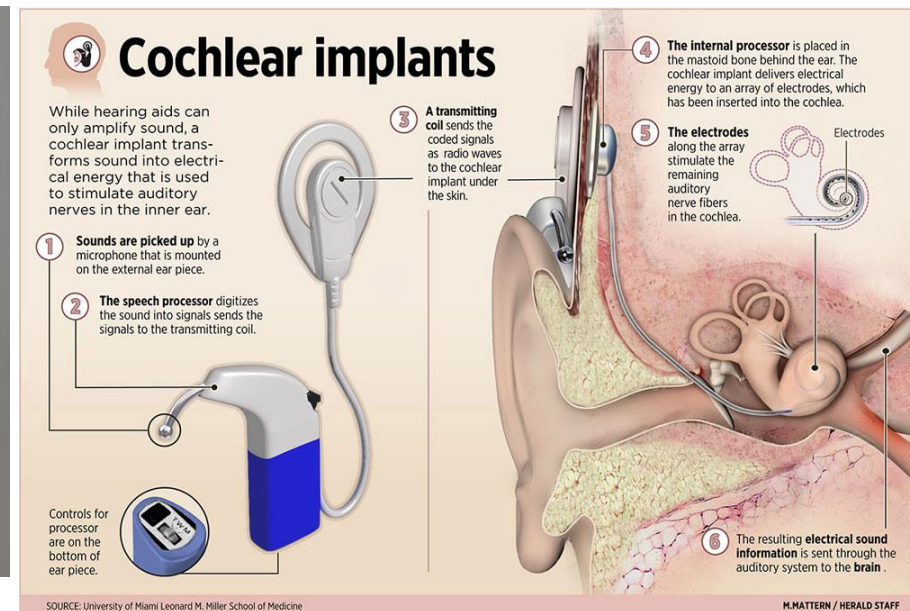
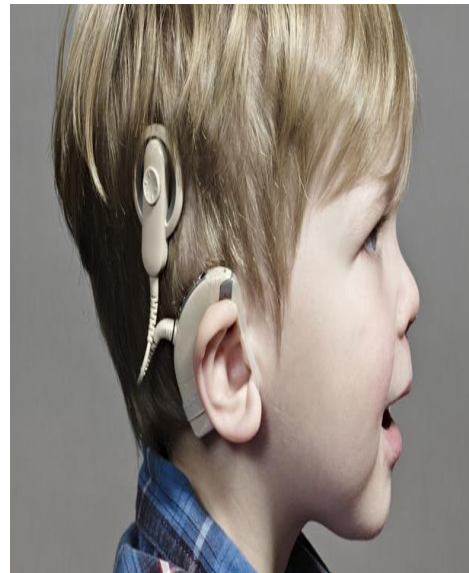
Indications.

_ Patient above 2 years of age with profound binaural cochlear (sensory) hearing loss with relatively intact cochlear N.

*Normal mentality

*No medical, surgical, or radiological contraindications. (ex. cochlear ossification.

- *Microphone
- *Speech processor
- *Transmitter
- *Receiver
- *Electrodes



Other meant to help the deaf patient

*Lip reading. for child with partial deafness

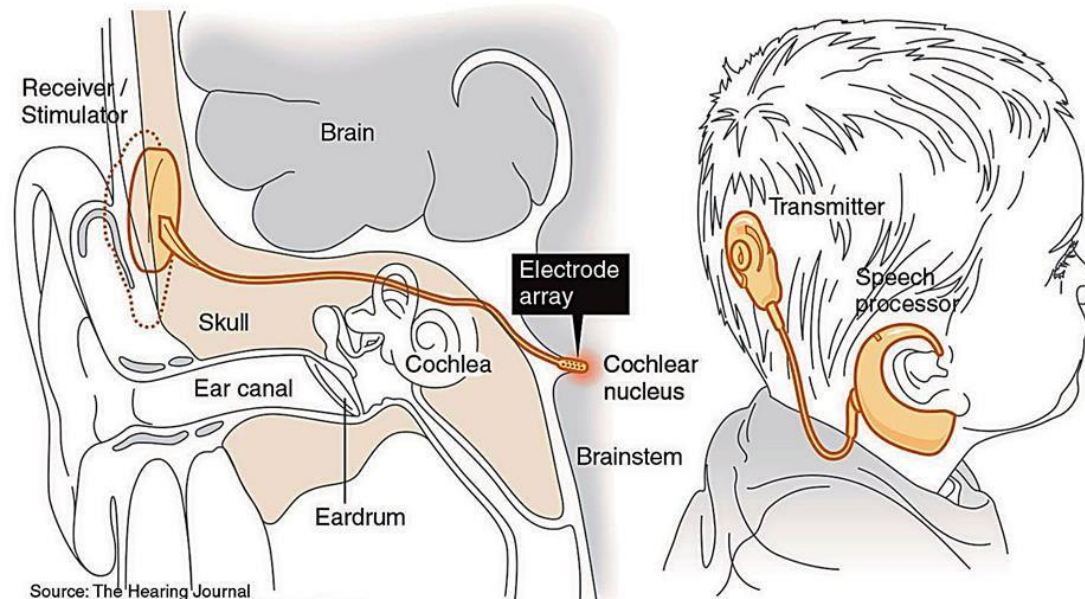
**Sign language. for profound deafness .

Brain stem implants

Patients who have had both acoustic nerves destroyed (eg, by bilateral temporal bone fractures or neurofibromatosis) can have some hearing restored by means of brain stem implants that have electrodes connected to sound-detecting and sound-processing devices similar to those used for cochlear implants.

A link to sound

The auditory brainstem implant, or ABI, is a surgically implanted device increasingly used to give children who were born deaf a chance to hear. The ABI helps patients whose auditory nerve, which ferries sound from the ear to the brain, is damaged or absent.



Source: The Hearing Journal
Graphic: Doug Stevens, Los Angeles Times

The End

Thank you